

ANXIETY SENSITIVITY MEDIATES THE RELATIONSHIP BETWEEN EXERCISE
FREQUENCY AND ANXIETY

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Abstract

ANXIETY SENSITIVITY MEDIATES THE RELATIONSHIP BETWEEN EXERCISE FREQUENCY AND ANXIETY

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The anxiolytic effects of regular exercise have been well documented, though the mechanisms through which exercise leads to reductions in anxiety remain unclear. In recent years, there has also been mounting research indicating that exercise reduces anxiety sensitivity, or the fear of anxiety sensations, a known vulnerability factor for the development and maintenance of anxiety disorders. The purpose of the present study was to examine whether anxiety sensitivity mediates the relation between exercise frequency and anxiety symptoms in a large community sample of 954 western North Carolina residents. Results indicated that exercise frequency was negatively correlated with anxiety sensitivity and anxiety, and anxiety sensitivity significantly mediated the relation between exercise frequency and anxiety symptoms. These findings provide additional evidence supporting the inverse association between exercise and anxiety, and suggest that anxiety sensitivity is one mechanism through which exercise reduces anxiety. The implications of these findings for clinicians and future research are discussed.

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Table of Contents

Abstract	iv
Acknowledgments.....	v
List of Tables	vii
List of Figures	viii
Introduction.....	1
Method	9
Results.....	13
Discussion.....	15
References.....	20
Appendix A.....	31
Vita.....	32

List of Tables

Table 1. Demographic Characteristics.....	10
Table 2. Descriptive Statistics.....	13
Table 3. Total, Direct, and Indirect Effects of All Study Measures	15

List of Figures

Figure 1. Mediation Model	14
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Anxiety Sensitivity Mediates the Relationship between Exercise Frequency and Anxiety

Anxiety is a future-oriented emotional state characterized by a feeling of helplessness and a lack of perceived ability to predict, control, or obtain desired results (Barlow, 2002). Anxiety and related disorders are often accompanied by physiological arousal (Barlow, 2002). Although anxiety is a ubiquitous human emotion, some individuals experience persistent anxious apprehension that results in high levels of distress and/or functional impairment, and are thus labeled as having an anxiety disorder. Anxiety disorders are the most prevalent category of psychiatric illnesses in the United States, affecting approximately 40 million adults nationwide, or more than 18% of the population each year (Kessler, Chiu, Demler, & Walters, 2005). The national costs associated with anxiety disorders are estimated to be over \$42 billion annually, totaling approximately a third of the country's total mental health costs (Greenberg et al., 1999). Individuals with anxiety disorders tend to seek treatment for symptoms that emulate physical illness. In fact, over half of the country's costs related to anxiety disorders are associated with the repeated utilization of healthcare services (Greenberg et al., 1999).

Etiology of Anxiety Disorders

Although dozens of theories have been put forth to explain the etiology and maintenance of anxiety disorders, cognitive-behavioral models have developed the strongest empirical support to date. Modern cognitive-behavioral theories generally propose that anxiety disorders result from a complex interplay of biological vulnerabilities, generalized psychological vulnerabilities, and specific psychological vulnerabilities (e.g., Abramowitz, Deacon, & Whiteside, 2011; Barlow, 2002; Mineka & Cook, 1986; Nebel-Schwalm & Davis, 2013). For example, Mineka and Zinbarg (2006) propose a diathesis-stress model of anxiety

that suggests anxious individuals inherit biological vulnerabilities that influence temperament (e.g., behavioral inhibition), the intensity with which emotions are experienced, and fear conditioning. In addition, various life experiences influence the development and perpetuation of anxiety problems. For example, early learning experiences that promote a general sense of environmental uncontrollability and unpredictability, contextual variables associated with specific learning experiences during or following stressful or traumatic events, and individual differences in threat perception and coping strategies (e.g., avoidance) appear to influence the extent to which one will experience anxiety-related distress and impairment. In addition, the individual's specific learning experiences are thought to determine the type of anxiety problem experienced. For example, individuals who experience panic disorder are thought to experience an initial panic attack, or false alarm, after which any of the physiological sensations experienced during the panic attack (i.e., interoceptive conditioning) or environmental stimuli present before or during the attack (i.e., exteroceptive conditioning) can serve as conditioned stimuli and can elicit subsequent fear responses.

Anxiety Sensitivity

One individual difference variable that has been shown to be particularly relevant to the development of anxiety disorders generally and is often included in cognitive-behavioral models of anxiety disorders is anxiety sensitivity. Anxiety sensitivity refers to the fear of anxiety-related sensations thought to arise from beliefs that such sensations precipitate harmful outcomes (e.g., Reiss, Peterson, Gursky, & McNally, 1986; Taylor, Jang, Stewart, & Stein, 2008). Anxiety sensitivity is conceptualized as a trait-like variable that serves as a specific vulnerability for anxiety-related problems by amplifying fear in response to anxiety-eliciting stimuli (Reiss, 1991). The fear of anxiety sensations increases the intensity of fear

responses because the individual experiences a combination of anxiety generated by a feared stimulus plus the additional fear of their arousal sensations. Individual differences in anxiety sensitivity are believed to develop in response to various learning experiences, such as experiencing (or observing another individual experience) a catastrophic physical event (e.g., heart attack), observing others respond fearfully to their physical sensations, or receiving misinformation regarding the dangerousness of certain sensations (e.g., Craske & Rowe, 1997).

To date, a large body of research has accumulated to support the association between anxiety sensitivity and anxiety disorders. For example, individuals who meet criteria for various anxiety disorders tend to exhibit elevated levels of anxiety sensitivity, with anxiety sensitivity being highest among individuals with panic disorder (Olatunji & Wolitzsky-Taylor, 2009). In addition, research has consistently indicated that anxiety sensitivity predicts the future development of anxiety disorders among individuals without a history of panic (e.g., Maller & Reiss, 1992; Schmidt, Lerew, & Jackson, 1997) and the maintenance of anxiety disorders among those who have been previously diagnosed (e.g., Schmidt, Lerew, & Jackson, 1999). Fortunately, anxiety sensitivity has been shown to be malleable in response to intervention. For example, various treatment outcome studies have noted that reductions in anxiety symptoms appear to be accompanied by changes in anxiety sensitivity (e.g., Nowakowski, Rowa, Antony, & McCabe, 2016), and cognitive-behavioral therapy appears to result in large reductions in anxiety sensitivity scores (e.g., hedges $g = 1.40$; Smits et al., 2008). Of particular note, studies suggest that interventions that have been shown to be effective in treating anxiety disorders may operate at least partially through their effects on anxiety sensitivity. For example, Smits, Powers, Cho, and Telch (2004) found that changes in

anxiety sensitivity mediated the relation between cognitive-behavior therapy and anxiety disorder treatment outcomes. Thus, although anxiety sensitivity increases risk for anxiety disorders, it appears to be highly malleable, and reductions in anxiety sensitivity appear to be associated with reduced anxiety symptomology and risk for anxiety disorders.

Exposure Therapy for Anxiety Disorders

Modern cognitive-behavioral models of anxiety disorders propose that learned associations, once developed, cannot be unlearned (Craske, Treanor, Conway, Zbozinek, & Vervilet, 2014). Rather, cognitive-behavioral interventions aimed at treating anxiety disorders generally focus on helping the individual to develop new, or strengthen previous, non-threat associations via the presentation of information or experience that is incompatible with threat associations (e.g., Abramowitz, et al., 2011). For example, cognitive-behavior therapy for panic disorder commonly involves repeatedly exposing the individual to feared physiological sensations (i.e., interoceptive exposure) and environmental stimuli (i.e., exteroceptive exposure) in the absence of feared outcomes so the individual can learn such stimuli do not necessarily indicate threat, and that they are capable of tolerating the experience of aversive physical sensations and environmental conditions without having to engage in avoidant coping strategies (Barlow, 2002). Given that anxiety sensitivity is a core vulnerability factor for anxiety disorders, and repeated exposure to feared cues is associated with reductions in anxiety symptomology, some have suggested that interoceptive exposure may be effective for treating anxiety disorders largely through its effects on anxiety sensitivity (e.g., Broman-Fulks, Berman, Rabian, & Webster, 2004).

Several meta-analyses have examined the effectiveness of exposure therapy for anxiety disorders both as a stand-alone treatment and in combination with other

interventions. In general, exposure-based interventions have been shown to be highly effective in reducing anxiety symptomology and improving quality of life. More specifically, exposure therapy has been shown to generate large effect sizes in the treatment of specific phobia (ES = 1.05; Wolitzky-Taylor, Horowitz, Powers, & Telch, 2008), panic disorder (ES = 1.55; Westen & Morrison, 2001), obsessive-compulsive disorder (ES = 1.46; van Balkom et al. 1994), post-traumatic stress disorder (ES = 1.89; Van Etten & Taylor, 1998), social anxiety disorder (ES = .89; Gould, Buckminster, Pollack, Otto, & Yap, 1997) and generalized anxiety disorder (ES = .82; Mitte, 2005). Overall, meta-analytic findings provide consistent support for the effectiveness of exposure-based treatments for anxiety disorders.

Exercise

Relation between Exercise and Anxiety

Physical exercise is another intervention that has been shown to be associated with significant reductions in anxiety and related disorders. Since 1995, the results of at least five population-level cross-sectional studies have indicated that regular physical activity is associated with lower rates of anxiety and serves a protective function against the negative effects of anxiety-provoking stimuli (De Moor, Beem, Stubbe, Boomsma, & De Geus, 2006; Goodwin, 2003; Strine, Chapman, Kobau, & Balluz, 2005; Taylor, Pietrobon, Pan, Huff, & Higgins, 2004; Thorsen et al., 2005). In addition, regular exercise is associated with reduced risk for anxiety disorders. For example, Goodwin (2003) found that regular exercise was associated with significantly lower prevalence rates of panic attacks, generalized anxiety disorder, social anxiety disorder, specific phobia, and agoraphobia in a large nationally representative sample of American adults ($N = 8,098$). Specifically, individuals who engaged in regular physical activity were 28-43% less likely to develop an anxiety disorder than those

that did not exercise. In addition, regular physical activity was associated with decreased likelihood of an anxiety disorder regardless of age, sex, or medical condition (e.g., Goodwin, 2003; Herring, O'Connor, & Dishman, 2010). Taken together, existing data suggests that regular exercise may act as a protective factor against anxiety disorders.

Longitudinal research has also indicated that individuals with high levels of anxiety tend to experience significant reductions in anxiety symptoms following initiation of an exercise regimen. Although meta-analytic studies estimate the anxiolytic effect of physical activity to be small but significant among non-clinical populations (Rebar et al., 2015), the therapeutic effects among individuals with a diagnosis of an anxiety disorder or those who present with elevated anxiety symptoms appears to be moderate to large (e.g., Conn, 2010; Petruzzello, Landers, Hatfeild, Kubitz, & Salazar, 1991; Wang et al., 2014; Wipfli, Rethorst, & Landers 2008). For example, in a meta-analytical review of the anxiolytic effects of physical activity among clinical populations, Wipfli, Rethorst, and Landers (2008) found an overall effect size of -0.48, and a more recent systematic review of 12 randomized control trials and 5 meta-analyses concluded that exercise is comparable to other established treatments for anxiety, including cognitive behavioral therapy and medication, and is more effective than placebo or waitlist control (Stonerock, Hoffman, Smith, & Blumenthal, 2015). Overall, empirical findings support the therapeutic benefits of exercise for reducing anxiety among both clinical and non-clinical populations.

Possible Mechanisms for the Anxiolytic Effects of Exercise

Although the anxiolytic effects of exercise are well-documented, less is understood regarding the mechanisms through which exercise functions to reduce anxiety related symptoms and disorders. One mechanism that has received recent attention is the notion that

physical activity acts as a form of interoceptive exposure, or the repeated exposure to anxiety-related sensations (e.g., rapid heart rate, sweating, shortness of breath). As noted above, interoceptive exposure is a common component of cognitive-behavioral therapies for anxiety disorders and the preferred method of treatment in cases where a person fears anxious arousal itself (Abramowitz et al., 2011).

Interoceptive exposure has been proposed to lead to therapeutic change via two pathways. The first pathway involves habituation to anxiety-related sensations, or a decline in fearful reactions over time as a result of repeated encounters with fear-provoking stimuli (e.g., Stewart & Watt, 2008; Watts, 1979). The second pathway involves cognitive re-evaluation of the dangers that could arise from somatic cues (e.g., Beck, Shipherd, & Zebb, 1997; Clark, 1994; Stewart & Watt, 2008). This theory is based on the idea that some individuals misinterpret anxiety-related sensations to be threatening and exercise-based exposure may serve to provide corrective information about feared physiological states (e.g., Ströhle, 2009; Ströhle et al., 2005). In other words, through exercise, individuals may learn that anxious arousal may be uncomfortable, but feared catastrophic outcomes are unlikely, thereby strengthening non-threat associations with anxiety sensations (e.g., Beck et al., 1997; Craske et al., 2008).

Relation between Anxiety Sensitivity and Exercise

Given that exercise elicits many of the physiological sensations often experienced during an anxiety response, some researchers have suggested that individuals with high anxiety sensitivity may experience exercise as aversive and employ an avoidant coping strategy (e.g., McWilliams & Asmundson, 2001). Indeed, several self-report and laboratory-based studies have indicated that individuals with heightened anxiety sensitivity rate exercise

as more distressing than their low anxiety sensitivity counterparts, and they are less likely to engage in physical exertion (e.g., McWilliams & Asmundson, 2001; Moshier et al., 2013; Sabourin, Hilchey, Lefaiivre, Watt, & Stewart, 2001; Smits, Tart, Presnell, Rosenfield, & Otto, 2010). Research has also suggested that repeated exposure to feared physiological sensations in the context of exercise leads to significant reductions in anxiety sensitivity (e.g., Broman-Fulks et al., 2004; Broman-Fulks & Storey, 2008; Broman-Fulks, Kelso, & Zawilinski, 2015; Smits et al., 2008). For example, Broman-Fulks and colleagues (2004) conducted a study in which individuals with high anxiety sensitivity were randomly assigned to participate in six 20-minute sessions of either moderate-intensity aerobic exercise or non-aerobic activity over the course of two weeks. Individuals who engaged in aerobic exercise exhibited significant reductions in anxiety sensitivity compared to those in the non-aerobic condition. This finding has been replicated several times with aerobic and anaerobic forms of exercise, and reductions in anxiety sensitivity have been noted in as little as one session (e.g., Broman-Fulks et al., 2015; Broman-Fulks & Storey, 2008; Smits et al., 2008).

Although not directly assessed using exercise-based interventions, research has suggested that treatment-generated reductions in anxiety sensitivity can lead to reduced risk for the future development of anxiety disorders (Schmidt et al., 2007). Thus, exercise-induced reductions in anxiety sensitivity have the potential to also decrease anxiety symptomology and risk for the development or maintenance of anxiety disorders. However, researchers have yet to directly assess whether the observed reductions in anxiety symptomology following exercise may be at least partially attributable to exercise-induced reductions in anxiety sensitivity.

Summary

Empirical evidence suggests that exercise frequency is inversely related to anxiety symptoms (e.g., De Moor et al., 2006; Goodwin, 2003; Strine et al., 2005; Taylor et al., 2004; Thorsen et al., 2005), and anxiety sensitivity serves as a vulnerability factor for anxiety-related psychopathology (e.g., Olatunji & Wolitzky-Taylor, 2009; Maller & Reiss, 1992). Cognitive-behavioral interventions for anxiety disorders generally include exposure-based components, which have demonstrated efficacy for reducing anxiety symptoms (e.g., Wolitzky-Taylor et al., 2008; Westen & Morrison, 2001; Gould et al., 1997; Mitte, 2005), and research suggests that reductions in anxiety sensitivity mediate the relation between cognitive-behavioral therapy and treatment outcomes (e.g., Smits et al., 2004). Although the mechanisms through which exercise exerts its anxiolytic effects are not well-understood, exercise has been shown to decrease anxiety sensitivity (e.g., Broman-Fulks et al., 2004; Broman-Fulks & Storey, 2008; Broman-Fulks et al., 2015; Smits et al., 2008), which may, in turn, reduce anxiety and related symptomology. However, researchers have yet to directly test this possibility. Thus, the purpose of the present study is to assess whether anxiety sensitivity mediates the relation between exercise frequency and anxiety. Based on a review of the literature, it was hypothesized that anxiety sensitivity would mediate the relation between exercise frequency and anxiety symptoms.

Methods

Participants

Participants consisted of 955 western North Carolina residents (60.80% women) ages 18 to 85 ($M = 45.80$, $SD = 16.22$) who were recruited via mass advertising to be a part of a larger clinical trial examining the effects of an antioxidant on physical and psychological

health between January and August 2008 (Broman-Fulks, Canu, Trout, & Nieman, 2012).

Participants were stratified by age during recruitment to ensure diverse age representation, including 40% young adults (18-40), 40% middle age (41-65), and 20% older (66-85) adults. Participants were also stratified by body mass index to include 33% normal body mass index (18.5-24.9), 33% overweight (25-29.9), and 33% obese (30 or more). Due to the nature of the clinical trial, women who were pregnant or lactating were excluded from the study.

Table 1

Demographic Characteristics.

Respondents' details (<i>n</i> = 955)	Mean (SD)	Frequency (<i>s</i>)	Percentage (%)
Age	45.80 (16.22)	951	
Missing		4	
Gender			
Male		374	39.2
Female		580	60.8
Missing		1	
Race			
White, not of Hispanic origin		906	95.2
Black, not of Hispanic origin		17	1.8
American Indian/Alaskan native		5	.5
Pacific Islander		4	.4
Asian		4	.4
Hispanic		5	.5
Other		8	.8
Missing		3	

Measures

Exercise Frequency. Respondents were asked: "Outside of your normal work or daily responsibilities, how often do you engage in exercise that at least moderately increases your breathing and heart rate, and makes you sweat for at least 20 minutes (such as brisk walking, cycling, swimming, jogging, aerobic dance, stair climbing, rowing, basketball,

racquetball, vigorous yard work, etc.).” Response choices were: 5 or more times per week, 3 to 4 times per week, 1 to 2 times per week, less than 1 time per week.

Anxiety Sensitivity Index-3. The Anxiety Sensitivity Index-3 (ASI-3; Taylor et al., 2007) is an 18-item self-report instrument used to measure a respondent’s fear of anxiety-related sensations. Items are rated on a 5-point Likert Scale ranging from 0 (very little) to 4 (very much), and the total score represents a summation of scores across all 18 items (range = 0 to 72). The ASI-3 has demonstrated good psychometric properties, including excellent test-retest reliability ($\alpha = 0.93$; Wheaton, Deacon, McGrath, Berman, & Abramowitz, 2012), positive alpha scores (ranging from .78 to .91 among North American samples), and excellent convergent, discriminant, and criterion-related validity (Taylor et al., 2007). Cronbach’s alpha in the present sample was .91.

Brief Symptom Inventory, Anxiety Subscale. The Brief Symptom Inventory (BSI; Derogatis, 1975) is a 53-item self-report instrument designed to measure clinically relevant psychological symptoms corresponding with nine symptom dimensions: anxiety, somatization, obsessive-compulsive disorder, inter-personal sensitivity, depression, hostility, phobic anxiety, paranoid ideation, and psychoticism. The anxiety subscale (ANX) of the BSI is made up of six self-report items (items 1, 12, 19, 38, 45, and 49 of the BSI) designed to measure clinically relevant symptoms of anxiety. Items are ranked on a 5-point Likert Scale ranging from 0 (not at all) to 4 (extremely) and responses reflect the level of distress experienced over the past week. Scores are interpreted in comparison to both clinical and non-clinical age-based norms (Derogatis, 1993; Derogatis & Spencer, 1982). Good internal reliability and validity has been reported for the BSI and each of its nine symptom

dimensions (Derogatis, 1993), and the anxiety subscale exhibited acceptable internal consistency in the present sample (Cronbach's alpha = .79).

Procedure. After informed consent was obtained, and two weeks prior to the initiation of the clinical trial, participants completed an online battery of questionnaires administered via surveymonkey.com, which included the anxiety subscale of the Brief Symptom Inventory, Anxiety Sensitivity Index-3, and a demographic questionnaire that contained an item regarding exercise frequency. The present study, which involved the secondary analysis of existing data, complied with the standards of the American Psychological Association's ethical guidelines for human research (American Psychological Association, 2002) and was approved by Appalachian State University's Institutional Review Board on October 25, 2016 (see Appendix E).

Planned Analysis

To test the hypothesis that anxiety sensitivity would mediate the relation between exercise frequency and anxiety symptoms, the PROCESS procedure for SPSS (Hayes, 2013) was used. Exercise frequency was entered as the independent variable (X), anxiety was entered as the outcome variable (Y), and anxiety sensitivity was entered as the mediator variable (M). It was predicted that the total effect of X (exercise frequency) on Y (anxiety) would be comprised of a direct effect of X on Y and an indirect effect of X on Y through a predicted mediator, anxiety sensitivity.

The PROCESS procedure produced total, direct, and indirect effects. Rather than outputting a t-statistic or p-value for the indirect effect, PROCESS generated a 95% bootstrapped confidence interval computed from 50,000 resamples. In a PROCESS analysis,

if zero does not occur between the upper and lower limits of the interval, researchers can conclude (with 95% confidence) that the indirect effect is significant.

Results

Demographic details including information about the age, race, and sex of respondents can be found in Table 1. Of the 955 participants, 22.4% ($n = 214$) endorsed having high anxiety or phobias either during the study or prior to the study, and 5% ($n = 43$) reported regularly taking medication to treat anxiety at the time of the study. Frequencies, correlations, means, and standard deviations are reported in Table 2. Bivariate correlations revealed significant associations among all variables (exercise frequency, ANX, and ASI-3). As expected, exercise frequency was negatively correlated with ANX, $r = -.19, p < .001$ and ASI-3, $r = -.18, p < .001$. In addition, ANX was significantly positively correlated with ASI-3, $r = .44, p < .001$.

Table 2

Descriptive Statistics.

	Mean	SD	Correlation		
			1	2	3
1. Exercise Frequency	2.56	1.05			
2. ANX	.43	.49	-.19**		
3. ASI-3	11.88	10.07	-.18**	.44**	

Note:

**Significant at the .001 significance level.

Mediation Analysis

A simple mediation analysis conducted using the Hayes (2013) PROCESS tool for SPSS revealed that anxiety sensitivity indirectly influenced anxiety symptomology through its effect on exercise frequency. As can be seen in Figure 1 and Table 3, participants who endorsed heightened anxiety sensitivity reported less frequent physical activity than those

who endorsed lower levels of anxiety sensitivity ($a = -1.724$), and participants who reported less frequent physical exercise endorsed more anxiety symptoms ($b = .021$). The indirect effect ($ab = -.036$) of anxiety sensitivity accounted for approximately 40% of the total effect ($c = -.089$) of exercise frequency on anxiety symptoms, and bias-correct bootstrap confidence interval for the indirect effect based on 50,000 bootstrap samples did not include zero (-.052 to -.023).

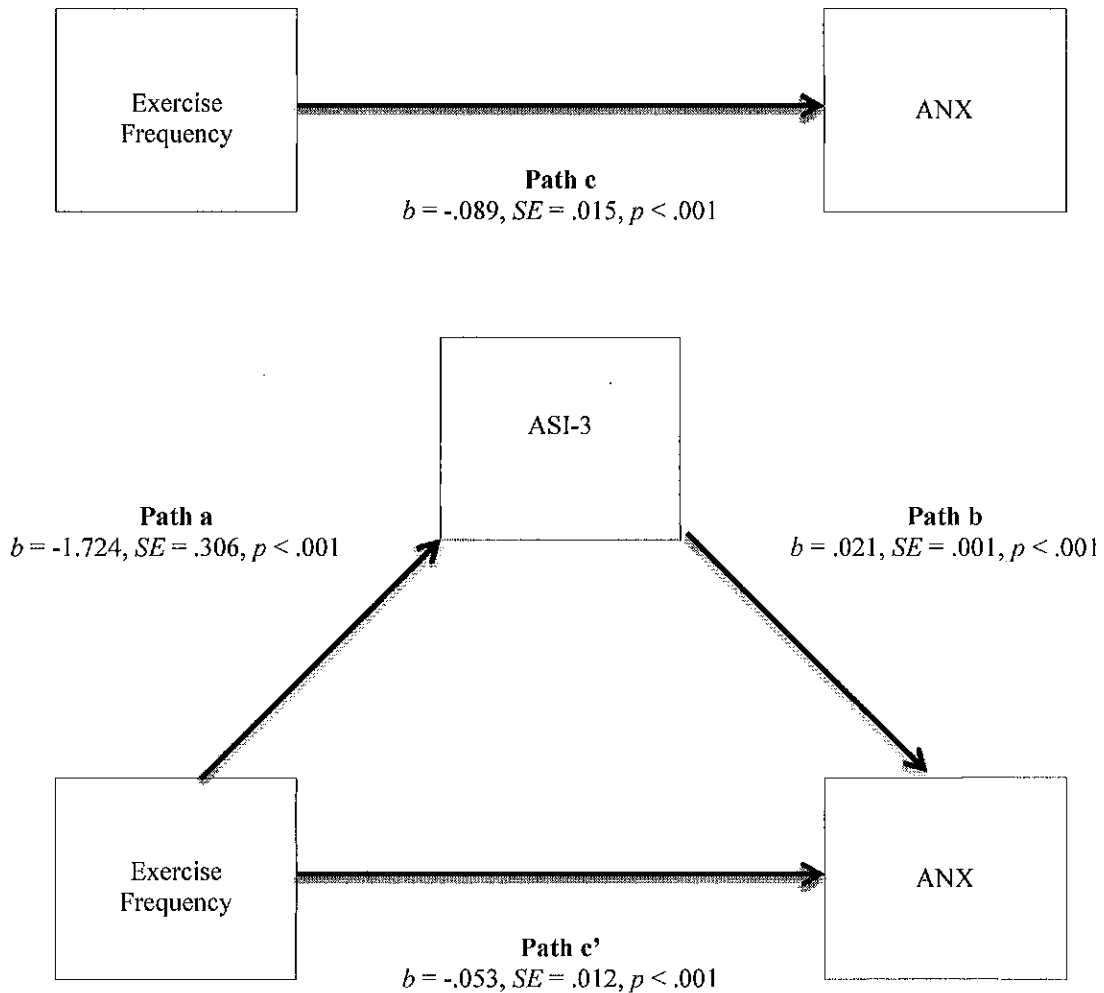


Figure 1. Mediation Model. This figure illustrates the mediating role of ASI-3 scores in the relationship between exercise frequency and ANX.

Table 3

Total, Direct, and Indirect Effects of All Study Measures.

Variable	<i>B</i>	SE	<i>t</i>	<i>p</i>
<i>Total and direct effects</i>				
Exercise frequency on ANX	-0.089	.015	-5.923	< .001
Exercise frequency on ANX, controlling for ASI-3	-0.053	.014	-3.853	< .001
Exercise frequency on ASI-3	-1.724	.306	-5.632	< .001
ASI-3 on ANX	0.021	.001	14.306	< .001
Variable	<i>B</i>	SE	LLCI	ULCI
<i>Indirect effect and bootstrap confidence intervals</i>				
Exercise frequency on ANX through ASI-3	-.036	.008	-.052	-.023

Note: *N* = 954. Bootstrap sample size = 50,000, LL = lower limit, UL = upper limit, CI = 95% confidence interval.

Discussion

The present study represents the first examination of whether anxiety sensitivity, a core vulnerability factor of anxiety disorders, is a mechanism through which exercise relates to anxiety symptomology. Consistent with previous research, exercise frequency was correlated with anxiety symptoms (e.g., Goodwin, 2003; McWilliams & Asmundson, 2001) and anxiety sensitivity (e.g., McWilliams & Asmundson, 2001). Results of the PROCESS mediation analysis revealed that anxiety sensitivity accounted for approximately 40% of the total effect of exercise frequency on anxiety. These findings provide evidence that exercise affects anxiety in part via its impact on anxiety sensitivity, which is consistent with the results of previous research demonstrating that cognitive behavioral protocols lead to anxiety symptom amelioration through their effects on anxiety sensitivity (e.g., Norr, Allan, Macatee, Keough, & Schmidt, 2014; Olthuis, Watt, Mackinnon, Stewart, 2014; Schmidt et al., 2007).

Thus, the present findings extend prior research supporting the role of anxiety sensitivity as a mediator of exposure-based therapies for anxiety disorders.

Anxiety sensitivity accounted for nearly half of the variance of exercise frequency on anxiety, though it did not account for the total effect, suggesting that exercise relates to anxiety outcomes via other mechanisms as well. This finding is consistent with previous research proposing a variety of potential mechanisms through which exercise may exert anxiolytic effects. For example, exercise has been proposed to affect anxiety by leading to an increased sense of mastery and enhanced self-efficacy, reductions in catastrophic thinking, and increases in perceived ability to persist in activities despite aversive emotional or somatic experiences (Asmundson et al., 2013; Gallagher, Schoemann, & Pressman, 2011; Moses, Steptoe, Mathews, Edwards, 1989; Stathopoulou, Powers, Berry, Smits, & Otto, 2006). Alternatively, some have suggested that exercise reduces anxiety by creating a diversion from worrisome thoughts and daily stressors (Yeung, 1996). Several physiological changes have also been observed, which may relate to the anxiolytic effects of exercise. For example, exercise has been proposed to lead to reductions in anxiety through improvements in the central serotonergic systems (Chaouloff, 1997), increases in endorphin secretion (Hoffman, 1997), the activation of thermoregulatory responses (Koltyn, 1997), and adaptations of the neuroendocrine system (Dishman, 1997). Given the relatively large number of proposed mechanisms, additional mediation research is needed to further clarify those mechanisms responsible for the well-documented anxiolytic effects of exercise. An understanding of alternative mechanisms may be particularly relevant for individuals with anxiety disorders who endorse low anxiety sensitivity, as other avenues for symptom reduction may be more pertinent to address in clinical interventions.

Clinical implications

Theoretical frameworks consistently emphasize the influence of interoceptive exposure in explaining the effects of exercise on anxiety sensitivity and anxiety disorders (e.g., Broman-Fulks et al., 2004; Sabourin et al., 2008). As previously noted, interoceptive exposure involves repeated exposure to anxiety-related sensations (e.g., rapid heart rate, sweating, shortness of breath). Exposure is believed to lead to therapeutic change through inhibitory learning, or the development of new, or strengthening of existing, non-threat associations (e.g., Craske et al., 2008; Craske et al., 2014; Deacon et al., 2013). This is consistent with the Pavlovian conditioning model, which posits that original fear associations are neither erased nor unlearned during extinction; rather a parallel but inhibitory association develops (e.g., Bouton, 1988; Bouton, 1993; Bouton & King, 1983). Thus, repeated exposure to anxiety-related sensations in a healthy context, such as during exercise, may serve to strengthen non-threat associations with anxiety sensations.

Relatedly, Craske and colleagues (2008) have suggested several ways in which clinicians may be able to enhance exposure outcomes by optimizing inhibitory learning (Craske et al., 2008), many of which can be easily applied to exercise-based interventions. For example, an important part of exposure includes presenting clients with information that violates their fear expectancies, which forms the basis of inhibitory learning (e.g., Craske et al., 2008; Craske et al., 2014; Deacon et al., 2013). Thus, clinicians may consider debriefing after exercise sessions to highlight the mismatch between perceived danger and actual outcomes. Further, researchers have proposed that variations in the delivery of exposure increase the storage strength of exposure-based learning, and therefore enhance long-term gains (Bjork & Bjork, 1992, 2006; Craske et al., 2008; Magill & Hall 1990). Based on this

view, the therapeutic effects of exercise on anxiety, and particularly anxiety sensitivity, may be enhanced by varying the type and intensity of physical exercise. Finally, exercising in multiple contexts (e.g., outside, the gym) may also optimize exposure by offsetting context renewal, or the recovery of an extinguished response when a phobic stimulus is confronted in context that differs from the context in which exposure-based learning occurred (e.g., Bouton, 1988; Craske et al., 2008; Craske et al., 2014).

Strengths and limitations

The present study has several notable strengths. For example, this study is novel in that it provides the first examination of the potential mediating role of anxiety sensitivity in the relation between exercise frequency and anxiety symptoms. In addition, the data examined was collected from a large community sample of adults ranging in age from 18 to 85, many of whom endorsed having been or currently being diagnosed with or treated for high levels of anxiety. The prevalence of anxiety amongst the sample population was fairly representative of the adult general population in the United States. However, the present study also had several methodological limitations. For example, a single item was used to assess exercise frequency, and measures of exercise type, duration, and intensity were not administered. Given that previous research has suggested that exercise intensity affects anxiety sensitivity (Broman-Fulks et al., 2004), future research should include additional assessment of exercise characteristics. Further, although the correlations between exercise frequency and anxiety ($r = -.19$) and exercise frequency and anxiety sensitivity ($r = .18$) were significant, they were relatively small and somewhat lower than some previous research. For example, McWilliams and Asmundson (2001) reported significant negative associations between males' self-ratings of exercise frequency and trait anxiety ($r = -.37$) and anxiety

sensitivity ($r = -.31$). However, it should be noted that exercise frequency was not significantly associated self-ratings of trait anxiety or anxiety sensitivity among females (McWilliams & Asmundson, 2001). This finding, along with the fact that the sample population of the present study was predominately female, may explain why only weak correlations were found among exercise frequency and anxiety and exercise frequency and anxiety sensitivity. Additional research regarding the association between exercise frequency, anxiety and anxiety sensitivity is warranted, and future research may benefit from analyzing the potential moderating role of gender.

The cross-sectional design of this study also inhibits the ability to draw conclusions regarding causation. Mediation is a causal model, which assumes that the independent variable produces changes in the mediating variable, which in turn leads to changes in the dependent variable. Although previous research has clearly established that exercise leads to reductions in anxiety symptomology, the absence of longitudinal data in the present study prevents a direct examination of the extent to which exercise-generated reductions in anxiety are influenced by changes in anxiety sensitivity. Thus, it will be important for future research to replicate and extend the results of the present study using data collected from the same sample at multiple points in time.

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Appendix A

To: Chelsea Abraham
Psychology , Psychology
CAMPUS EMAIL

From: IRB Administration

Date: 10/25/2016

RE: Determination that Research or Research-Like Activity does not require IRB Approval

STUDY #: 17-0096

STUDY TITLE: Anxiety Sensitivity Mediates the Relationship Between Exercise Frequency and Anxiety

The IRB determined that the activity described in the study materials does not constitute human subject research as defined by University policy and the federal regulations [45 CFR 46.102 (d or f)] and does not require IRB approval.

Study Specific Notes:

Secondary analysis of existing de-identified data, that currently resides in a de-identified database.

This determination may no longer apply if the activity changes. IRB approval must be sought and obtained for any research with human participants.

If you have any questions about this determination, please contact Robin Tyndall at 262-2692; or irb@appstate.edu. Thank you.

CC:

Joshua Broman-Fulks, Psychology

James Denniston, Psychology

Lisa Grizzard, Psychology

Vita

Chelsea Marie Abraham was born in Laurinburg, North Carolina, to David and Stacey Abraham. Ms. Abraham graduated cum laude from University of North Carolina Wilmington in May 2010 and was awarded a Bachelor of Arts degree in Psychology. During her time at UNCW, she was a member of Alpha Delta Pi Sorority. Ms. Abraham commended work toward a Master of Arts degree in Clinical Psychology at Appalachian State University in the fall of 2014. She completed her graduate externship at The Counseling and Psychological Services Center at Appalachian State University during the fall of 2016 and was awarded the Master of Arts in December 2016.