2018

Aerobic Exercise Exposure Targeting Anxiety Sensitivity: Effects on Associated Health Behaviors in Young Adults

Autumn Lanoye

Virginia Commonwealth University

AEROBIC EXERCISE EXPOSURE TARGETING ANXIETY SENSITIVITY: EFFECTS ON ASSOCIATED HEALTH BEHAVIORS IN YOUNG ADULTS

A dissertation submitted in partial fulfillment of the requirements for the degree of Doctor of Philosophy at Virginia Commonwealth University

By: AUTUMN LANOYE
B.A., University of Rochester, May 2009
M.S., Virginia Commonwealth University, August 2014

Co-Director: Jessica G. LaRose, Ph.D.
Associate Professor
Department of Health Behavior and Policy, Virginia Commonwealth University

Co-Director: Bruce D. Rybarczyk, Ph.D.
Professor
Department of Psychology, Virginia Commonwealth University

Virginia Commonwealth University
Richmond, VA
July 27, 2018

Acknowledgements

I owe my sincere gratitude to the many people who helped to make this project possible. Dr. Jessica LaRose, I cannot thank you enough for your guidance, insight, reassurance, and especially your unwavering patience. Dr. Bruce Rybarcyzk, thank you for your mentorship over the past 7 years—your advice and encouragement kept me on track throughout my thesis, internship, and dissertation. To Drs. Melanie Bean, Caroline Cobb, Ron Evans, and Scott Vrana, thank you for serving on my committee and providing your expertise and feedback in shaping this investigation. Dr. Tricia Leahey, your instruction and guidance regarding HLM was invaluable and is truly appreciated. To the OPT for Health team members who continued data collection efforts in my absence while I was on internship, thank you for allowing me to rest easy knowing that this project was in good hands. Lastly, I would also like to acknowledge those who participated in this study, without whom this endeavor would not be possible.

I would be remiss if I did not also express my love and appreciation for all those who encouraged and supported me throughout the entirety of graduate school. To my friends in the program, thank you for late nights in the library, for good times that got me through the hard times, and for understanding the unique frustrations and victories that are part and parcel of being a doctoral student in clinical psychology. Mom, Dad, Rich, Bri, and Lys—your unwavering faith in me was and is a constant source of strength; thank you for always believing in me. Marshall, thank you for remaining calm despite my stress, for getting excited about opportunities to teach me about statistics, and for being the best partner I could ever imagine.

Table of Contents

List of Tables

List of Figures

Abstract

Anxiety sensitivity (AS) is associated with health behaviors such as low rates of physical activity, overeating, alcohol use, and poor sleep; however, interventions targeting AS via exercise-based interoceptive exposure have not assessed these as outcomes. In addition, previous studies are limited by brief follow-up periods. This study aimed to replicate previous aerobic exercise interoceptive exposures with an extended (6-week) follow-up and measurement of health behaviors.

Participants were 44 sedentary young adults with elevated AS randomized to intervention (6 20-minute sessions of moderate-intensity treadmill walking) or assessment-only control. Assessments took place at baseline, week 2 (post-treatment), week 4, and week 8 with measurements of AS (ASI-3), physical activity (7-Day PAR), sleep (ISI), binge eating, alcohol use, depression (PHQ-8), anxiety (GAD-7), and stress (PSS-4).

The intervention condition demonstrated a marginally significant reduction in AS compared to control at week 4 which eroded by week 8. There were no significant between-group differences for health behavior change. The intervention condition demonstrated decreases in depression, general anxiety, and perceived stress compared to control, but these effects eroded by week 4. There was no difference in findings for participants with BMI<25 vs. those with BMI>=25.

Findings indicate that a brief intervention might not be sufficient to produce lasting changes in AS without additional treatment. Intervention effects were not as strong in this study compared to previous reports, which may be due to the size and greater racial/ethnic diversity of the current sample. Future research should objectively measure physical activity and explore individual variability in treatment response.

Aerobic Exercise Exposure Targeting Anxiety Sensitivity: Effects on Associated Health Behaviors in Young Adults

Anxiety sensitivity was first proposed by Reiss and McNally in 1985 as a critical component in their expectancy model of fear. This model explains fear behavior (i.e., avoidance) as the result of two factors: the extent to which a person expects the situation to cause harm and the extent to which a person expects the situation to cause anxiety. This latter factor is conceptualized as the product of both learned anticipation of anxiety for the specific situation (anxiety expectancy) and beliefs about negative consequences of experiencing anxiety (anxiety sensitivity). While expectancies about danger and anxiety are specific to the situation, anxiety sensitivity is an individual factor determined by learning experiences and, to some extent, biological predisposition (Reiss & McNally, 1985; Stein, Jang, & Livesley, 1999). Anxiety sensitivity serves to amplify the experience of anxiety above and beyond a delimited situation: a specific situation cues an individual to respond anxiously; for those who are high in anxiety sensitivity, this sparks a cascade of secondary anxiety due to anticipation of additional negative effects. Thus, anxiety sensitivity can be thought of as an individual's tendency to globally believe that the experience of anxiety itself results in adverse cognitive, physical, and/or social consequences (Reiss, Peterson, Gursky, & McNally, 1986).

Anxiety sensitivity is thought to be conceptually distinct from trait anxiety despite its relative stability over time, having demonstrated meaningful contribution to the prediction of anxiety-related phenomena such as panic attacks above and beyond the contribution of trait anxiety (McNally, 1989; Reiss, 1991; Reiss, 1997). Anxiety sensitivity has also been shown to be distinct from other potentially overlapping constructs such as negative affect (Reiss, 1991) and neuroticism

(Naragon-Gainey, 2010). In experimental settings, anxiety sensitivity has been shown to predict greater fear response and shorter duration (i.e., lower tolerance) of hyperventilation and CO_2 inhalation challenges (Brown, Smits, Powers, & Telch, 2003; Rapee, Brown, Antony, & Barlow, 1992). Similarly, individuals high in anxiety sensitivity have been shown to respond with greater negative affect and subjective distress in response to a caffeine intake challenge (Telch, Silverman, & Schmidt, 1996).

Structure and Measurement

The evolution of thought regarding the structure of anxiety sensitivity is inherently tied to the history of its measurement. The development of an initial self-report measure—the 16-item Anxiety Sensitivity Index (ASI; Reiss et al., 1986)—was predicated on the assumption that anxiety sensitivity is a unitary construct. Subsequent factor analyses of the ASI across research groups returned conflicting results, with some supporting a unidimensional structure and others supporting a multidimensional structure. A hierarchical model consisting of one higher-order factor—global anxiety sensitivity—and several lower-order factors resolves these discrepancies and is now the most common conceptualization of anxiety sensitivity (Lilienfeld, Turner, & Jacob, 1996; Zinbarg, Barlow, & Brown, 1997).

In response to these findings, a 36-item revised version of the ASI was created in order to better assess potential lower-order factors (ASI-R; Taylor & Cox, 1998), given that approximately half of the items on the original ASI mapped onto concerns regarding physical symptoms of anxiety. However, the factor structure of the ASI-R is generally considered to be unreliable (Deacon, Abramowitz, Woods, & Tolin, 2003; Armstrong, Khawaja, & Oei, 2006; Taylor et al., 2007). Across studies examining the ASI and ASI-R, the three most commonly-replicated orthogonal lower-order factors are cognitive, physical, and social concerns; as such, these have

come to be accepted as the major components of anxiety sensitivity (Zinbarg, Mohlman, & Hong, 1999; Deacon et al., 2003; Naragon-Gainey, 2010). The ASI-3 (Taylor et al., 2007) was designed specifically to improve the psychometric properties of these three dimensions and assess them in a more robust manner than either the original ASI or the ASI-R. Psychometric analyses of the 18-item ASI-3 reflect a stable factor structure across both clinical (i.e., diagnosed anxiety disorders) and nonclinical (i.e., college undergraduate) samples (Taylor et al., 2007; Wheaton, Deacon, McGrath, Berman, & Abramowitz, 2012).

Anxiety Sensitivity and Psychopathology

In the relatively brief time since its initial formulation, the topic of anxiety sensitivity has amassed an impressive amount of scientific interest due to its potential clinical applications with respect to the development, exacerbation, and maintenance of psychological disorders. Research has most commonly linked high levels of anxiety sensitivity to diagnoses of panic disorder, generalized anxiety disorder (GAD), post-traumatic stress disorder (PTSD), social anxiety disorder, agoraphobia, obsessive-compulsive disorder, and depression. Results of meta-analyses suggest that anxiety sensitivity manifests differently across diagnoses—with respect to both higher order and lower order factors (Rector, Szacun-Shimizu, & Leybman, 2007; Naragon-Gainey, 2010; Olatunji & Wolitzky-Taylor, 2009). For example, Naragon-Gainey (2010) found that as a global factor, anxiety sensitivity was highest among those diagnosed with PTSD, GAD, and panic disorder when compared to other anxiety disorders. However, the constellation of lower-order factors differed across these diagnoses: PTSD was characterized by a stronger association with cognitive concerns compared to physical and social concerns; GAD was characterized by stronger associations with both cognitive and social concerns compared to physical concerns; panic

disorder was characterized by stronger associations with both physical and cognitive concerns compared to social concerns.

To date, the majority of research regarding the relationship between anxiety sensitivity and psychopathology has been cross-sectional in nature; however, preliminary longitudinal evidence suggests that high anxiety sensitivity serves as a risk factor for developing additional anxiety symptomatology and diagnoses. In two large, nonclinical samples, high anxiety sensitivity at baseline was found to predict panic attack incidence, other anxiety symptomatology, and anxiety-related functional impairment five weeks following a discrete stressful situation: military basic training (Schmidt, Lerew, & Jackson, 1997; 1999). In prospective studies with nonclinical samples, high anxiety sensitivity was significantly associated with the development of anxiety disorders over the course of two years (Schmidt, Zvolensky, & Maner, 2006) and with the experience of panic attacks over the course of 11 years (Plehn & Peterson, 2002). Similar findings are present in adolescent samples, where those who demonstrated high or escalating trajectories of anxiety sensitivity were more likely to experience panic attacks (Weems, Hayward, Killen, & Taylor, 2002) as well as broader symptoms of anxiety and depression (Allan, Felton, Lejuez, MacPherson, & Schmidt, 2016) compared to those demonstrating a low, stable trajectory.

Treatments for Anxiety Sensitivity

Intervention-related changes in anxiety sensitivity have been assessed mainly among clinical samples seeking treatment for panic disorder. In this context, a variety of treatment programs have been found to produce reductions in anxiety sensitivity, including cognitive therapy (Beck, Stanley, Baldwin, Deagle, & Averill, 1994), relaxation training (Beck et al., 1994), non-prescriptive treatment (Shear, Pilkonis, Cloitre, & Leon, 1994), and pharmacotherapy (Romano, van Beek, Cucchi, Biffi, & Perna, 2004; Simon et al., 2004). However, cognitive-behavioral

therapy (CBT) remains the most commonly implemented treatment approach for panic disorder; as such, most evidence supporting treatment-related reductions in anxiety sensitivity is derived from CBT intervention studies. Cognitive-behavioral treatment components include psychoeducation, cognitive restructuring, diaphragmatic breathing, and interoceptive exposure: the induction of feared physiological sensations mimicking anxiety. Standard implementation of CBT—i.e., therapist-led treatment spanning 8-12 sessions—has been shown to produce decreases in anxiety sensitivity in both group (Telch et al., 1993; Penava, Otto, Maki, & Pollack, 1997; Carter, Sbrocco, Gore, Marin, & Lewis, 2003) and individual (McNally & Lorenz, 1987; Clark et al., 1994) treatment formats. These reductions in anxiety sensitivity are specifically associated with improvement in clinical symptoms (Hazen, Walker, & Eldridge, 1996), with preliminary evidence supporting the notion that reductions in anxiety sensitivity mediate the relationship between CBT and improvements in panic-related symptomatology along with associated functional impairment (Smits, Powers, Cho, & Telch, 2004).

In light of longitudinal research suggesting that anxiety sensitivity may precede the development of more clinically significant psychological symptoms, more recent efforts have focused on developing and evaluating preventive treatments specifically targeting this construct. In fact, the relationship between anxiety sensitivity and a broad range of psychological disturbances have led some to propose that it may represent a powerful trans-diagnostic treatment target across mood and anxiety disorders (Boswell et al., 2013; Olthuis, Watt, Mackinnon, & Stewart, 2014). While medications such as benzodiazepines are effective in the management of panic disorder, evidence suggests that medication alone is insufficient to decrease anxiety sensitivity with the same efficacy as CBT (Simon et al., 2004), and may actually contribute to its long-term maintenance due to the suppression of anxiety sensations, preventing habituation and

corrective learning experiences (Otto, Pollack, & Sabatino, 1996; van Balkom, de Beurs, Koele, Lange, & can Dyck, 1996). Thus, research has focused mainly on the development of non-pharmacological treatments for anxiety sensitivity.

Due to the success of CBT in reducing anxiety sensitivity among clinical samples, many interventions for "at-risk" samples—i.e., those high in anxiety sensitivity—include both cognitive restructuring and exposure components (Abplanalp, 2001 [as cited in Smits, Berry, Tart, & Powers, 2008]; Maltby, 2001 [as reported in Maltby, Mayers, Allen, & Tolin, 2005]; Gardenswartz & Craske, 2001; Kenardy, McCafferty, & Rosa, 2003). Initial treatments were largely unsuccessful in achieving intervention effects compared to control groups (Gardenswartz & Craske, 2001; Maltby, 2001; Kenardy, McCafferty, & Rosa, 2003), which might be due to a variety of factors. One such consideration is the finding that conducting a detailed diagnostic evaluation prior to randomization appears to be associated with improvements in anxiety sensitivity among control group participants—perhaps due to perceived normalization of anxiety symptoms (Maltby et al., 2005).

An apparent lack of emphasis on guided interoceptive exposure may also have contributed to the absence of significant treatment effects reported in early CBT interventions for anxiety sensitivity. For example, Gardenswartz and Craske (2001) conducted large-group workshops of up to 20 participants, allowing little room for individualized attention while potentially providing ample opportunity for participants to engage in experiential avoidance; further, the authors describe the intervention as largely didactic rather than experiential in nature. Similarly, Kenadry, McCafferty, and Rosa (2003) implemented an internet-delivered, self-guided intervention for anxiety sensitivity, which again may have resulted in poor participant engagement in interoceptive exposure.

The importance of interoceptive exposure. Interoceptive exposure has been identified as an essential component of effective CBT treatment for panic and other anxiety disorders (Barlow, Craske, Cerny, & Klosko, 1989; Smits, Powers, Berry, & Otto, 2007; Stewart & Watt, 2008), as it allows for corrective learning experiences. Individuals high in anxiety sensitivity hold beliefs that the experience of anxiety results in negative social, cognitive, and/or physical consequences. Inducing physiological sensations resembling anxiety via hyperventilation, physical activity, chair spinning, or other means allows these individuals to experience feared stimuli and reinterpret their experience in a less catastrophic manner. For those especially high in anxiety sensitivity, however, it may be particularly difficult to engage in these exercises due to strong motivation to avoid such experiences. Experiential avoidance of physiological sensations associated with anxiety is possible even in the context of interoceptive exposure—for example, by means of distraction and/or rationalization (e.g., "I'm feeling out of breath, my face is flushed, and my heart is racing—but that's because my therapist is asking me to run in place, not because I'm anxious). Thus, it is important that those in treatment for anxiety sensitivity understand the purpose of interoceptive exposure, and are instructed to focus on their experience and allow themselves to attribute bodily sensations to anxiety rather than to the exposure (Craske & Barlow, 2007).

An examination of interoceptive exposures conducted at varying intensities found that greater reductions in anxiety sensitivity were produced by the highest intensity interoceptive exposure condition (Deacon et al., 2013), underscoring the importance of this approach for treatment of anxiety sensitivity in nonclinical samples. Indeed, later CBT interventions for high anxiety sensitivity with perhaps a greater focus on interoceptive exposure have produced intervention effects: Watt, Stewart, Lefaivre, & Uman (2006) designed and implemented a three-session in-person small group intervention and found significant reductions in anxiety sensitivity

compared to a contact-matched non-specific control group. After receiving content regarding the nature of anxiety and the role of avoidance (Session 1, psychoeducation), and strategies for changing interpretation of anxiety experiences (Session 2, cognitive restructuring), the third session was dedicated to conducting interoceptive exposures via a running challenge. Olthuis and colleagues (2014) reported similar results following an 8-week telephone-delivered intervention with instruction in repeated running exposures. Single-session CBT interventions have also produced reductions in anxiety sensitivity with hyperventilation exposure (Schmidt, Capron, Raines, & Allan, 2014) and personalized interoceptive exposure based on participants' most feared bodily sensations (Keough & Schmidt, 2012). Of note, these interventions with an emphasis on interoceptive exposure have been found to produce clinically significant effects at lower intensities than standard CBT interventions conducted with clinical samples.

Physical activity interventions for anxiety sensitivity. The importance of interoceptive exposure for the treatment of anxiety sensitivity has led some researchers to design exposure-only interventions without a cognitive restructuring component. Physical activity is often chosen as the interoceptive exposure in these instances, as it produces several physiological sensations that overlap with anxiety (e.g., increased heart rate, sweating, shortness of breath, muscle tension) and can be easily conducted in a variety of settings (Smits et al., 2007). Broman-Fulks, Berman, Rabian, and Webster (2004) conducted an exploration of an exercise-only intervention targeting anxiety sensitivity in which participants with high levels of anxiety sensitivity were randomly assigned to high- or low-intensity treadmill conditions for six 20-minute sessions conducted over 2 weeks. While participants in both high- and low-intensity conditions exhibited declines in anxiety sensitivity at one-week follow-up, the high-intensity condition produced greater reductions and reductions in fear of anxiety-related bodily sensations. Broman-Fulks and Storey extended

this inquiry in 2008 by including a non-active control condition, which yielded treatment effects consistent with previous findings.

In order to examine the potential additive effects of cognitive restructuring, Smits and colleagues (2008) randomly assigned participants to one of three conditions: interoceptive exposure (IE; using the Broman-Fulks et al. paradigm described above), interoceptive exposure plus cognitive restructuring (IE+C), and waitlist control (WC). Both active intervention arms were matched on treatment contact, with cognitive restructuring taking place periodically as participants completed their treadmill exposures. The authors found that both IE and IE+C conditions produced significant reductions in anxiety sensitivity compared to WC, which were maintained following 3 weeks of no further treatment contact. However, results produced by the IE+C condition were not significantly different than results produced by the IE condition, indicating that therapist-led cognitive restructuring is not necessary to effect changes in anxiety sensitivity in a nonclinical sample. These results are compatible with findings from comparisons of interoceptive exposure to cognitive restructuring among samples with panic disorder, which demonstrate no significant differences between approaches (Arntz, 2002; Bouchard et al., 1996; Hecker, Fink, Vogeltanz, Thorpe, & Sigmon, 1998).

Extensions of this physical activity intervention for anxiety sensitivity have explored dosage, type of physical activity, and delivery method in order to optimize intervention effects. LeBouthillier and Asmundson (2015) found that a single 30-minute aerobic exercise exposure produced reductions in anxiety sensitivity at post-intervention assessment, with additional declines continuing over the next several days. However, analyses revealed that anxiety sensitivity began to trend upward following a week with no further intervention. In a single-session comparison of aerobic exercise interoceptive exposures to strength training interoceptive exposures, Broman-

Fulks, Kelso, and Zawilinski (2015) found that both produced reductions in anxiety sensitivity compared to control, with no significant differences between intervention conditions. Between-group differences did emerge with respect to performance on a post-intervention CO_2 challenge, where participants in the aerobic exercise condition responded with less fearful arousal than did participants in the strength training condition. Extant research indicates that these interventions should be conducted with supervision from research staff in order to ensure adherence; self-guided running exposures assigned as homework have been shown to be completed at rates of 30%-43% (Sabourin et al., 2008; Sabourin, Stewart, Watt, & Krigolson, 2015). Taken together, the results of these studies suggest that aerobic exercise conducted over multiple sessions in a supervised setting optimizes reductions in anxiety sensitivity. The most standard implementation is six 20-minute sessions over 2 weeks; however, lack of long-term follow-up in these studies precludes conclusions regarding sufficient dosage to produce long-term change in anxiety sensitivity.

Anxiety Sensitivity and Health Behaviors

The primary outcome in each of the above-described exercise exposure interventions conducted with nonclinical samples was change in anxiety sensitivity, with secondary outcomes occasionally including psychological correlates such as depression, panic attacks, distress intolerance, etc. None of the reported outcomes included change in health behaviors associated with anxiety sensitivity, which include physical activity, eating patterns, alcohol use, and sleep habits—all of which are associated with overweight and obesity. Thus, just as anxiety sensitivity may represent a transdiagnostic mental health treatment target, anxiety sensitivity may also function as a "transdiagnostic" risk factor for health behaviors associated with overweight and obesity. While much cross-sectional work has been conducted with respect to these associations, little is known regarding the effects of anxiety sensitivity interventions on related health behaviors.

Physical activity. Many of the physiological sensations associated with exercise (e.g., increased heart rate, shortness of breath, sweating) mimic somatic symptoms of anxiety, and are likely to be interpreted as such by those high in anxiety sensitivity. In fact, it is this similarity that makes exercise exposure such an effective intervention for anxiety sensitivity by allowing for the opportunity to re-interpret these sensations as non-threatening (Smits et al., 2007). In a naturalistic setting, however, the experiential overlap between physical activity and anxiety may result in exercise avoidance for those high in anxiety sensitivity.

Reported relationships between physical activity and anxiety sensitivity vary based on assessed dimensions of physical activity and measurement of anxiety sensitivity itself, sometimes resulting in conflicting conclusions. For example, McWilliams and Asmundson (2001) found that higher anxiety sensitivity regarding physical concerns (measured continuously) was associated with infrequent exercise (defined as structured bouts of moderate-intensity activity of at least 30 minutes duration) among male but not female undergraduates. However, when assessing physical activity in undergraduate women (measured in total across a variety of types and intensity levels) Sabourin, Hilchey, Lefaivre, Watt, and Stewart (2011) did find a significant difference in exercise frequency when comparing high v. low anxiety sensitivity (measured categorically) groups. Of note, those with high anxiety sensitivity perceived more barriers to physical activity than those with low anxiety sensitivity, which mediated the relationship between anxiety sensitivity status and exercise frequency. Moshier and colleagues (2013) argue that exercise intensity is particularly important to take into account, as higher-intensity activities are more likely to evoke feared physiological sensations than those of lower-intensity.

In order to better understand the role of anxiety sensitivity in relation to exercise behavior, Moshier, Szuhany, Hearon, Smits, and Otto (2016) recruited participants interested in increasing

their frequency of physical activity and asked them to set an exercise goal for the following week. In addition, the authors assessed anxiety sensitivity and potential predictors of behavior change including perceived behavioral control, impulsivity, and perseverance. At 1-week follow-up, anxiety sensitivity emerged as the sole significant predictor of exercise goal attainment above and beyond baseline physical activity level—with high anxiety sensitivity associated with lower rates of exercise increases.

Weight status also appears to play a role in the relationship between anxiety sensitivity and exercise avoidance. Hearon, Quatromoni, Mascoop, and Otto (2014) found a significant interaction effect for anxiety sensitivity x BMI predicting moderate intensity activity (measured objectively) over a 2-week period in which high anxiety sensitivity was associated with *greater* participation in physical activity among normal weight individuals while high anxiety sensitivity was associated with *less* physical activity among individuals with overweight/obesity. In a similar experimental paradigm, Smits, Tart, Presnell, Rosenfield, and Otto (2010) found that peak subjective distress during 20 minutes of moderate/vigorous treadmill activity was highest for those with high anxiety sensitivity *and* high BMI. These findings suggest that compared to normal weight individuals with high anxiety sensitivity, those with overweight/obesity and high anxiety sensitivity may experience more frequent or more intense somatic sensations during exercise, prompting anxious arousal and appraisal.

The inverse relationship between anxiety sensitivity and exercise frequency among higher BMIs is particularly concerning given the benefits of regular physical activity with respect to sustained weight loss and prevention of related medical comorbidities. Establishing an exercise routine is a key component of behavioral weight loss interventions, but these findings suggest that individuals with high anxiety sensitivity may find this to be difficult and aversive. Indeed, physical

discomfort and negative affect are often cited as a barrier to physical activity among those with overweight/obesity (Egan et al., 2013; Ekkekakis & Lind, 2006; Grubbs & Carter, 2002; Leone & Ward, 2013), which may be amplified or explained in part by anxiety sensitivity for some individuals.

Eating. It has also been proposed that those high in anxiety sensitivity may turn to food as either a maladaptive coping response to aversive internal states or an experiential avoidance strategy to escape from and/or reduce anxiety sensations (Dave, 2015; Fulton et al., 2012). Higher anxiety sensitivity has been associated with greater endorsement of problematic eating behaviors (e.g., "I think about bingeing," "I eat when I am upset," "I stuff myself with food") on the Bulimia subscale of the Eating Disorder Inventory in a nonclinical sample of undergraduate students after controlling for both depression and anxiety (Anestis, Holm-Denoma, Gordon, Schmidt, & Joiner, 2008). In a naturalistic assessment of calorie consumption, individuals high in anxiety sensitivity consumed more calories following strong negative affect than did individuals low in anxiety sensitivity (Hearon et al., 2014). Mediation analyses suggest that the pathway between anxiety sensitivity and excess eating may be explained by poor distress tolerance (Anestis, Selby, Fink, & Joiner, 2007). Interestingly, interoceptive sensitivity—awareness of bodily sensations and cues—has been linked to adaptive eating (i.e., eating in response to hunger rather than emotion) but is also a necessary facet of anxiety sensitivity (Herbet, Blechert, Hautzinger, Matthias, & Herbert, 2013); thus it may be the specific negative appraisal of these somatic sensations that contributes to eating for reasons other than hunger.

The association between anxiety sensitivity and eating is of particular concern among those with overweight and obesity, given that this population is already susceptible to problematic eating behavior (Burton, Smit, & Lightowler, 2007; Geliebter & Aversa, 2003). In a community sample

of individuals with overweight/obesity, anxiety sensitivity was correlated with eating expectations (affect regulation and loss of control) and eating in response to emotions such as anger/frustration, anxiety, and depression (Hearon, Utschig, Smits, Moshier, & Otto, 2013). Similarly, DeBoer and colleagues (2012) found a significant association between binge frequency and anxiety sensitivity in a community sample; further, this relationship was moderated by physical activity, such that high anxiety sensitivity was associated with binge eating among those who did not routinely engage in moderate-intensity exercise. These findings lend further support to the potential role of anxiety sensitivity in behaviors associated with weight gain, overweight, and obesity.

Alcohol use. A large body of literature contributes to our understanding of the relationship between anxiety sensitivity and alcohol use, building upon the established relationship between anxiety variables (e.g., clinical diagnosis, panic attacks, trait anxiety) and problematic alcohol use. In examinations of college students without diagnosed alcohol use disorder, high anxiety sensitivity has been found to be associated with greater weekly alcohol use and more frequent episodes of drinking to excess (Stewart, Peterson, & Pihl, 1995; Stewart, Zvolensky, & Eifert, 2001). Evidence suggests that this relationship is mediated by negative reinforcement alcohol expectations (Chandley, Luebbe, Messman-Moore, & Ward, 2014; DeMartini & Carey, 2011; Lawyer, Karg, Murphy, & McGlynn, 2002; Stewart & Zeitlin, 1995; Stewart et al., 2001). In addition, these same and other studies have found gender to be a significant moderator of the relationship between anxiety sensitivity and drinking motives (Lawyer et al., 2002; O'Connor, Farrow, & Colder, 2008; Stewart et al., 2001; Zack, Poulos, Aramakis, Khamba, & MacLeod, 2007).

Alcohol outcomes have been assessed in the context of CBT interventions for anxiety sensitivity; though again, none of the exercise exposure interventions reported on these outcomes.

Implementation of the three-session CBT protocol designed by Watt and colleagues (2006) has been found to produce reductions in anxiety-related motives for alcohol use among a community sample, with this relationship mediated by reductions in anxiety sensitivity (Olthuis, Watt, Mackinnon, & Stewart, 2015). This same intervention conducted in an undergraduate sample yielded significant reductions in rates of alcohol-related consequences but not frequency of alcohol use (Watt et al., 2006).

Sleep. Results of several studies reflect a link between anxiety sensitivity and symptoms of insomnia or other sleep disturbances. Anxiety sensitivity may play a role in amplifying anxious arousal at night, resulting in delayed sleep onset (Babson, Trainor, Bunaciu, & Feldner, 2008; Hoge et al., 2011; Weiner, Meredith-Elkins, Pincus, & Comer, 2015), and thus representing a potential predisposing factor for the development of chronic insomnia (Short, Allan, Raines, & Schmidt, 2015). During the day, the awareness of bodily sensations associated with anxiety sensitivity may contribute to a hyperawareness of sleep-related sensations such as the perception of fatigue or difficulty concentrating, and interpret these experiences as threatening or undesirable—again, predisposing these individuals to insomnia (Short et al., 2015).

High anxiety sensitivity has the potential to worsen the course of both insomnia and anxiety disorders by contributing to sleep disturbances. Anxiety sensitivity is associated with sleep-related impairment and medication use among patients with chronic insomnia (Vincent & Walker, 2001). In a cross-sectional survey of participants with diagnosed anxiety disorder, anxiety sensitivity was found to mediate the relationship between diagnosis and sleep dysfunction (Baker et al., 2016). In general, the cognitive and physical components of anxiety sensitivity have been most reliably linked to sleep disturbance (Vincent & Walker, 2001; Calkins, Hearon, Capozzoli, & Otto, 2013; Short et al., 2015).

Providing further elucidation of the relationship between anxiety sensitivity and sleep dysfunction, Short and colleagues (2015) conducted an intervention targeting anxiety sensitivity in a community sample. Participants were randomized to either a health education (control) condition or a cognitive-behavioral intervention for anxiety sensitivity, consisting of psychoeducation and interoceptive exposure; treatment contact was equivalent across groups (45-minute computerized intervention). Results at 1-month post-intervention indicated that the intervention was successful in reducing anxiety sensitivity symptoms—in total and across all subfactors. Anxiety sensitivity and each of its components were also found to mediate the relationship between intervention completion and reduction in insomnia symptoms, providing longitudinal support for the role of anxiety sensitivity in sparking and maintaining anxious arousal regarding sleep and related consequences.

The Critical Period of Young Adulthood

The majority of anxiety sensitivity investigations have been performed among samples of undergraduate students, representing a convenience sample for researchers at academic institutions, but also representing an important population to consider given the relationship between anxiety sensitivity and mental health concerns. Young adults (typically defined as ages 18-35; National Institutes of Health, 2008) are at high risk for of anxiety and depressive disorders, subclinical emotional disturbances, and high rates of perceived stress. One third of U.S. adults age 18-29 reported lifetime incidence of anxiety disorder (Kessler et al., 2005), and 18-to-25-year-olds report the greatest 12-month prevalence of major depressive episode compared to other adult age groups (Center for Behavioral Health Statistics and Quality, 2015). Young adults also report greater rates of perceived stress than other age groups, with these rates increasing from year to year (American Psychological Association, 2016).

Similarly, the association between anxiety sensitivity and weight-related health behaviors is also particularly important to investigate in this population. Young adulthood is associated with increased alcohol use, changes in eating patterns, declines in physical activity, and poor sleep quality, which are associated with weight gain in this age group (Coren, 1994; Duffey, Gordon-Larsen, Jacobs, Williams, & Popkin, 2007; Gordon-Larsen, Nelson, & Popkin, 2004; Spiegel, Tasali, Penev, & van Cauter, 2004). Furthermore, this developmental period is marked by transitions such as enrolling in college, getting married, and beginning a family—all of which are also associated with weight gain and represent sources of stress that may heighten anxious responses among those high in anxiety sensitivity (Anderson, Shapiro, & Lundgren, 2003; Gunderson & Abrams, 2000; The & Gordon-Larsen, 2009).

Despite the problem of weight gain throughout young adulthood, young adults represent a particularly challenging group to recruit, retain, and engage in standard behavioral weight loss trials (Gokee-LaRose et al., 2009). As such, recent efforts have focused on developing weight control interventions specifically targeting young adults (National Institutes of Health, 2008), including conducting formative work in order to learn more about their needs and preferences with respect to participation in such programs. Preliminary results of this body of literature indicate that young adults are receptive to physical activity as a tool for weight management, and that engagement around physical activity is central to their interest in participating in a weight loss program (Corsino et al., 2013; LaRose et al., *in press*). In the context of such programs, young adults high in anxiety sensitivity may be less likely to follow-through on exercise recommendations, which could affect their weight loss success and contribute to lack of program engagement. Thus, heightened anxiety sensitivity among young adults enrolled in a weight management program may contribute to both variability in weight loss outcomes in addition to

amplifying documented difficulties with intervention engagement in an already difficult-to-engage population.

Statement of the Problem

An emerging literature of exercise exposure interventions has demonstrated robust short-term effects on anxiety sensitivity; however, these reports suffer from a lack of long-term follow-up assessments, limiting our ability to assess maintenance of treatment effects. Given that effects of single-session exercise exposures begin to erode by 1-week post-intervention (LeBouthillier & Asmundson, 2015), it is possible that a similar trajectory occurs following implementation of the 2-week, six-session paradigm. However, the longest post-intervention follow-up in such studies is 3 weeks (Smits et al., 2008), which may not be sufficient length to capture rebounds in anxiety sensitivity if they occur. Further, independent continuation of exercise exposures (i.e., participants' increases in real-world moderate-to-vigorous intensity physical activity) would be expected to mitigate such post-intervention increases in anxiety sensitivity; however, this has not been assessed in previous studies.

Changes in post-intervention physical activity behavior are important to consider not only with respect to understanding maintenance of intervention effects, but also as these may represent improvements in an important health behavior known to diminish across adolescence and emerging adulthood (Gordon-Larsen et al., 2004). Previous studies have not examined whether this brief exercise-exposure intervention produces real-world change with respect to other anxiety sensitivity-related health behaviors—not only physical activity, but also eating patterns, alcohol use, and sleep habits. This is a particularly important question to pose in a young adult population given documented declines in physical activity, problematic eating patterns, increased alcohol use, and poor sleep habits in this age group (Coren, 1994; Gordon-Larsen et al., 2004; Niemeier,

Raynor, Lloyd-Richardson, Rogers, & Wing, 2006; Center for Behavioral Health Statistics and Quality, 2015). Moreover, rates of overweight and obesity exceed 50% in this population (Behavioral Risk Factor Surveillance System, 2010), and data indicate that exercise avoidance among those high in anxiety sensitivity is stronger at higher BMIs, which could pose a significant barrier to weight loss success and/or effective weight gain prevention.

Interestingly, formative work conducted with young adults suggests that they identify exercise as a hook to weight loss program participation (Corsino et al., 2013; LaRose et al., (*in press*), yet those high in anxiety sensitivity may need additional intervention in the form of guided interoceptive exposure in order to successfully establish an exercise routine. The degree of supervised physical activity instruction varies across such programs, but this factor is important to consider given previous research demonstrating that simply setting an exercise goal is insufficient to elicit behavior change for those high in anxiety sensitivity (Moshier et al., 2016). Further, while intensive in-person lifestyle interventions designed specifically for young adults have been effective in producing clinically significant weight losses (Gokee-LaRose, Gorin, & Wing, 2009; Jakicic et al., 2015), there remains a great degree of individual variability in weight loss outcomes; thus anxiety sensitivity may represent an important baseline variable to assess as a predictor of treatment response. If the proposed study is effective in producing maintained reductions in anxiety sensitivity, it can easily be delivered as a low-cost, pre-treatment adjunct to extant behavioral weight loss programs for young adults.

Specific Aims and Hypotheses

This study aims to replicate and extend findings from an established exercise-exposure intervention for anxiety sensitivity (i.e., six 20-minute treadmill sessions conducted over 2 weeks) by conducting a 2-arm randomized controlled trial with follow-up assessments at 2 weeks (post-

intervention), 4 weeks, and 8 weeks following baseline. The length of no-intervention follow-up spans 6 weeks and allows for an examination of maintenance versus erosion of treatment effects. In addition, this study examines intervention effects on key weight-related health behaviors among young adults that are also associated with anxiety sensitivity. Participants in the proposed study will be randomized to either the intervention condition or an assessment-only control condition, with matched assessment schedule between conditions.

1. The first aim of the proposed study is to extend the follow-up period of an established exercise exposure paradigm for anxiety sensitivity in order to determine whether reductions in anxiety are maintained over time. Previous implementations of exercise exposure interventions have conducted follow-ups ranging from 1 to 3 weeks post-intervention (Broman-Fulks et al., 2004; Broman-Fulks & Storey, 2008; Smits et al., 2008); the current study proposes a follow-up period of 6 weeks post-intervention, thereby doubling the length of follow-up.

   Hypothesis 1a: There will be significant group by time interaction such that participants in the intervention condition will demonstrate a greater reduction in anxiety sensitivity than participants in the control group over the 8-week study period.

   Hypothesis 1b: There will be significant group by time interaction such that participants in the intervention group will demonstrate a greater reduction in anxiety sensitivity than participants in the control group from baseline to week 2 (post-treatment).

   Hypothesis 1c: Anxiety sensitivity will remain stable from week 2 to week 4 for participants in the intervention group with no significant group by time interaction; i.e., treatment effects will be maintained.

   Hypothesis 1d: There will be significant group by time interaction such that anxiety sensitivity will increase from week 4 to week 8 at a greater rate for participants in the

intervention group than for those in the control group; i.e., treatment effects will begin to erode.

2. The current study also aims to assess whether an exercise-exposure intervention for anxiety sensitivity produces changes in moderate-to-vigorous physical activity in a sample of largely inactive young adults.

   Hypothesis 2a: There will be a significant group by time interaction such that moderate-to-vigorous physical activity will increase from baseline to week 4 among participants in the intervention group at a greater rate than among participants in the control group.

   Hypothesis 2b: There will be a significant group by time interaction such that behavioral intention to increase physical activity will increase from baseline to week 4 for participants in the intervention group at a greater rate than for participants in the control group.

3. A third aim of the proposed study is to assess whether an exercise-exposure intervention for anxiety sensitivity produces changes in other health behaviors important in young adulthood: binge eating, alcohol use, and sleep quality.

   Hypothesis 3: If participants in the intervention condition demonstrate significant change from baseline to week 4 with respect to these health behaviors compared to those in the control condition, changes will represent improvements in these areas (i.e., decreased frequency of binge eating, decreased quantity/frequency of alcohol use, and improved sleep quality/duration).

4. An exploratory aim is to assess change in associated psychological variables (depression, general anxiety, and perceived stress) from baseline to week 4.

4. Another exploratory aim is to assess differences in these findings with respect to young adults with overweight/obesity compared to those of normal weight.

5. Lastly, the current study will explore whether any demonstrated behavior changes are associated with changes in specific ASI subfactors (physical, cognitive, & social concerns).

Method

Participants

Participants were recruited via digital advertisements, print fliers, email announcements, and radio spots from May 2017 through November 2017. This study was advertised as one aiming to "learn more about the relationship between anxiety, health, and mood." Print and digital ads were displayed throughout the VCU campus in high-visibility areas. Email announcements were distributed to VCU faculty and staff via the university's daily email system. The radio campaign was broadcast over local Richmond stations with the highest concentration of 18-35 year-old listeners. In addition, inquiries regarding this study were received via its listing on clinicaltrials.gov.

Recruitment materials directed interested individuals to the "Current Studies" section of our research lab website, which provided an overview of the study and a link to a secure online screening questionnaire in order to determine eligibility. Participants provided electronic consent for this screening measure by clicking a checkbox indicating their consent to provide data including personal health information. Eligibility criteria were consistent with those in previous brief exercise-based interventions for anxiety sensitivity (Broman-Fulks & Storey, 2008; Medina et al., 2004; Smits et al., 2008), and are outlined in Table 1. A recruitment goal of 45 participants was chosen in order to account for approximately 20% attrition in order to yield the 36 participants required to detect a small effect size [$f = .20$] at 80% power according to an a priori power analysis conducted in G*Power. This sample size is also consistent with similar studies in this body of

literature, which typically randomize between 18-25 participants per group (e.g., Broman-Fulks & Storey, 2008; Smits et al., 2008).

Table 1. Eligibility criteria and measurement

Criterion	Measurement
Inclusion	
Age 18-35	Self-report at screening
Elevated anxiety sensitivity	ASI-3 $\geq$ 23
Able to safely participate in moderate-intensity physical activity	PAR-Q
Exclusion	
Current aerobic exercise routine	L-CAT > 2
Receiving ongoing psychotherapy or counseling	Self-report at screening
Recent change in psychotropic medication for anxiety	Self-report at screening
Psychiatric hospitalization in past 6 months	Self-report at screening
Current pregnancy	Self-report at screening

Procedures

Consent and Randomization. After completing the initial online screening survey, interested and eligible individuals were invited via phone or email to schedule an individual in-person meeting with a trained research assistant in order to complete the informed consent process. As part of consent procedures, participants received both a verbal and written explanation of study purpose and procedures, and had the opportunity to ask questions regarding their participation. Following provision of informed consent, research staff verified that participants had enough time to complete the baseline session and had availability to complete treadmill sessions within the next 2 weeks if randomized to intervention condition. If these conditions were met, participants then completed in-person baseline measures administered by the research assistant and received randomization allocation (exercise intervention v. assessment-only control); otherwise, baseline session was rescheduled and randomization allocation was delayed until after completing baseline measures. Consenting participants assigned to the intervention condition scheduled their exercise appointments at baseline, with the option to complete the first session immediately following

pretreatment assessment. Randomization allocation was stratified on weight status (BMI < 25 kg/m^2 or BMI $\geq$ 25 kg/m^2) and biological sex.

Intervention Condition. Participants randomized to the intervention condition received a brief rationale for exposure procedures, including psychoeducation regarding the role of interoceptive exposures for the treatment of anxiety sensitivity. Aerobic exercise exposures were completed as described in previous reports (Broman-Fulks et al., 2004; Broman-Fulks & Storey, 2008; Smits et al., 2008) and guided by recommendations outlined in a meta-analysis conducted by Wipfli, Rethorst, and Landers (2008). Other than check-ins with research staff at 5-minute intervals, care was taken to minimize potential distractions while participants were on the treadmill. Exposures took place in a private room arranged such that the treadmill faced a blank wall and the research assistant was seated behind the participant in order to further minimize opportunities for distraction. Participants were asked to remain focused on their internal experience of the treadmill exercise.

One day prior to each scheduled exposure session, participants received a reminder email with instructions regarding wearing or bringing appropriate clothing (i.e., tennis shoes/sneakers with laces tied, nothing dragging or dangling from pants, clothing that allows full range of motion). This email also included the dates and times of all future exposure sessions in order to promote attendance. Upon arrival for each treadmill session, participants received instructions on heart rate monitor placement, a review of intervention rationale and instructions including treadmill safety, and orientation to subjective units of distress (SUDs) ratings. Baseline heart rate and SUDs were recorded prior to the participant stepping on the treadmill.

Treadmill activity began with a 5-minute warm-up period, during which participants were instructed to slowly increase their treadmill speed until their heart rate reached their target range

of 60-80% of maximum age-predicted heart rate. This range was calculated using the formula: (220-age) x (0.60 [lower bound] or 0.80 [upper bound]), and was provided to participants so that they could monitor their heart rate using the wristwatch monitor and adjust the treadmill speed accordingly to remain within their target heart rate range. Once participants' heart rate reached this range, they were informed that the 20-minute session would begin. Research staff checked in with participants at 5-minute intervals in order to record their heart rate, SUDs, and treadmill speed. After 20 minutes, participants were instructed slowly decrease their treadmill speed over the course of 5 minutes to cool down prior to stopping the treadmill.

Control Condition. Participants randomly assigned to the control condition completed assessments at the same time intervals as the intervention condition, but did not participate in exercise exposure sessions. They received no psychoeducation regarding interoceptive exposures for anxiety sensitivity. In addition, they received no instructions regarding their level of physical activity.

Assessments. Assessments took place at baseline, week 2 (post-intervention), week 4, and week 8 (see Table 2 for measurement schedule). Measures were administered in-person at baseline for all participants. All other assessments were completed via telephone by a trained research assistant and via a secure, HIPAA compliant online survey platform (REDCap) for participant ease and accessibility. Participants received $5 for each completed assessment, with a $15 bonus awarded if they completed all assessments (maximum total of $35). In addition, participants in the intervention condition received $5 for each completed treadmill session, with a $15 bonus awarded if they completed all treadmill sessions (maximum total of $45).

Measures

See Appendix for full measures included for use in the current study.

Demographics. Data regarding age, sex, race, ethnicity, student status, educational attainment, weight, height, and mental health diagnosis/treatment history were collected as part of the screening process.

Appropriateness to safely engage in physical activity. The Physical Activity Readiness Questionnaire (PAR-Q) is a commonly-used screening measure for determining whether beginning a physical activity routine is appropriate based on the American College of Sports Medicine guidelines (Warburton et al., 2011). Participants who answer "yes" to any of the seven items were not eligible for the current study.

Physical inactivity. To assess whether participants were physically inactive and therefore eligible for the study, the Stanford Leisure-Time Activity Categorical Item (L-Cat; Kiernan et al., 2013) was administered at screening. Six categorical responses comprise this single-item measure, which was designed to be face-valid and easy to use. The L-CAT has demonstrated strong test-retest validity and adequate criterion validity when compared to pedometer data (Kiernan et al., 2013) and armband activity monitor (Ross, Leahey, & Kiernan, 2018).

Anxiety sensitivity. The ASI-3 (Taylor et al., 2007) was used as a measure of anxiety sensitivity. This 18-item self-report questionnaire was developed in response to psychometric analyses of the ASI and ASI-R, which reflected unstable factor structures. Relevant to the current study, the ASI-3 was created and normed in a nonclinical sample of college undergraduates, making it an appropriate measure to assess anxiety sensitivity in the population of interest. The ASI-3 yields individual subscale scores (physical concerns, social concerns, and cognitive concerns) in addition to a total score reflecting overall anxiety sensitivity ranging from 0 to 72, which higher scores indicating greater anxiety sensitivity.

Heart rate. Participants randomized to the intervention condition wore the Polar H7 Bluetooth heart rate monitor for the duration of the treadmill exposure. This device provides real-time heart rate measurements via electrodes located on an adjustable chest strap, then transmits these data via Bluetooth to a wrist-worn monitor for ease of tracking.

Health behaviors.

Physical activity: In order to assess participants' self-reported moderate-to-vigorous physical activity throughout the study period, trained research assistants administered the Seven-Day Physical Activity Recall (PAR; Sallis et al., 1985) in-person at baseline assessments and via telephone at follow-up assessments. The PAR is a semi-structured interview designed to assess time spent in various types of physical activity, and is widely used in studies where objective collection of physical activity data is not feasible. An advantage of this measure is that it does not rely on participants to categorize the intensity of their reported physical activity, thus reducing the likelihood of obtaining significantly inflated physical activity estimates (Hagstromer, Ainsworth, Oja, & Sjostrom, 2010). Instead, the interviewer classifies participant responses as "moderate," "hard," or "very hard" physical activity. The timeline follow-back approach guides participants through each morning, afternoon, and evening of their past week to increase accuracy of responses. In addition to reporting their actual physical activity, participants also rated their behavioral intention to increase physical activity on a 5-point Likert scale at each timepoint.

Binge eating frequency: Consistent with methods reported in Deboer et al., (2012), binge eating frequency was assessed continuously using item 8 of the Eating Disorder Diagnostic Scale (EDDS; Stice, Telch, & Rizvi, 2000): "How many times have you eaten an unusually large amount of food and experienced a loss of control?" This question was integrated into the semi-structured interview procedures as described above in order to increase accuracy of responses.

Sleep: In addition to assessing physical activity, the PAR also collects information regarding sleep and wake time; thus, sleep duration can be estimated from this measure. The Insomnia Severity Index (ISI; Bastien, Vallieres, & Morin, 2001) was also administered in order to capture more nuanced information about participants' sleep quality. Seven items assessing severity of insomnia symptoms comprise the ISI, with higher scores indicating more significant sleep disturbances. This measure has been successfully administered in samples of nonclinical young adults (Gress-Smith, Roubinov, Andreotti, Compas, & Luecken, 2013), and is sensitive to change over time (Bastien et al., 2001).

Alcohol use: Quantity and frequency of alcohol use was assessed using the Timeline Follow-back method (Alcohol TLFB; Sobell & Sobell, 1992). This semi-structured interview approach is consistent with that of the PAR, asking participants to retrace their behavior and activities over a given timespan in order to assess daily alcohol use. The TLFB method yields a more accurate assessment of individual alcohol consumption compared to standard quantity-frequency measures, as it prompts participants to respond based on specific days rather than general patterns. This method has been used extensively with nonclinical samples of young adults (e.g., Rueger, Trela, Palmeri, & King, 2012; Sobell, Sobell, Klajner, Pavan, & Basian, 1986) and can be administered either in-person or via telephone (Rueger et al., 2012).

Psychosocial variables.

Depression symptoms. Designed as an efficient tool for use in clinical practice and research, the 8-item Patient Health Questionnaire (PHQ-8) is a brief, 8-item self-report measure of depression severity (Kroenke et al., 2009). Possible scores range from 0 to 24, with higher scores indicating more severe symptoms of depression; a score of 10 or higher has been established as a cut-point to reflect clinically significant depression (Kroenke et al., 2009). Psychometric properties

of this measure including reliability, internal consistency, and criterion validity have been found to be adequate (Kroenke, Spitzer, & Williams, 2001; Kroenke & Spitzer, 2002).

Anxiety symptoms. The Generalized Anxiety Disorder 7-item scale (GAD-7) is a self-report measure of generalized anxiety symptoms (Spitzer, Kroenke, Williams, & Lowe, 2006). Possible scores range from 0 to 21, with higher scores reflecting greater symptom severity; cut-points of 5, 10, and 15 correspond to mild, moderate, and severe levels of anxiety. This measure demonstrates adequate convergent validity with clinician diagnosis and measures of functional status; in addition, the GAD-7 has strong internal consistency and test-retest reliability (Spitzer et al., 2006).

Perceived stress. Similar to the PHQ-9 and GAD-7, the 4-item Perceived Stress Scale (PSS-4) was designed for quick and efficient administration to a broad range of community respondents and demonstrates adequate internal consistency and test-retest reliability (Cohen, Kamarck, & Mermelstein, 1983). The PSS-4 measures the extent to which respondents perceive recent life circumstances to be stressful.

Table 2. Measurement schedule

	Screening	**Baseline**	**Week 2**	**Week 4**	**Week 8**
Anxiety sensitivity (ASI-3)	X	X	X	X	X
Physical activity readiness (PAR-Q)	X				
Physical inactivity (L-CAT)	X				
Demographics	X				
Height & weight (self-report)	X		X	X	X
Height & weight (objective)		X			
Alcohol use (Alcohol TLFB)		X	X	X	X
Binge eating (Single item)		X	X	X	X
Physical activity (7-Day PAR)		X	X	X	X
Sleep (ISI)		X	X	X	X
Perceived stress (PSS-4)		X	X	X	X
Anxiety symptoms (GAD-7)		X	X	X	X
Depression symptoms (PHQ-8)		X	X	X	X

Shaded cells represent intervention period.

Analytic Strategy

This study implemented a two-arm randomized controlled trial with assessments at weeks 0 (baseline), 2 (post-treatment), 4, and 8. Preliminary analyses (i.e., independent samples *t*-test and/or chi-square test for continuous and categorical variables, respectively) were conducted to detect potential differences with respect to demographic characteristics between participants in the intervention condition compared to those in the control condition. Similarly, between-group analyses with respect to study attrition were conducted in order to determine differential drop-out between randomization arms. Prior to conducting analyses, variables were assessed to ensure they met assumptions of the statistical test to be employed. A level of $p \leq .05$ was selected to reflect statistical significance. Due to small sample size, we also pre-specified that $p > .05$ but $< .10$ would be interpreted as marginal statistical significance; in these instances, an effect size was also calculated in order to facilitate interpretations regarding clinical significance of the effect.

Aims 1 through 4 concerned the effect of the intervention on continuous outcomes (i.e., anxiety sensitivity, physical activity, binge eating, alcohol use, sleep quality) when compared to control. In order to account for nested structure of the data—i.e., within-participant repeated measurements (4 timepoints) and between-participant group analyses (treatment condition or weight status)—a two-level hierarchical linear modeling (HLM) approach was implemented. Advantages of HLM include maximizing statistical power, accounting for irregular time intervals between measurements, allowing for inclusion of dynamic covariates, and the flexible handling of missing data within longitudinal designs (Kwok et al., 2008; Shin et al., 2009; Smits et al., 2008). All analyses were pre-specified as follows:

Aim 1: Modeling change in anxiety sensitivity over time.

Anxiety sensitivity was estimated as a function of time within individuals, with level 1 equation: $AnxietySensitivity_{ij} = b_{0i}+b_{1i}Time_{ij}+e_{ij}$, where *i* represents each individual participant and *j* represents the four assessment time points; b_{0i} represents the outcome value for each individual at time 0 (i.e., intercept); b_{1i} represents the slope of change across time; and e_{ij} represents the error in predicting outcome *j* for participant *i*. At level 2, individual differences in intercept and slope were determined as functions of arm assignment with the equations $b_{0i} = \gamma_{00}+ \gamma_{01}RandomizationArm_j+u_{0j}$ and $b_{1i} = \gamma_{10}+ \gamma_{11}RandomizationArm_j+u_{1j}$. In order to assess change in anxiety sensitivity between timepoints—i.e., baseline to week 2, week 2 to week 4, week 4 to week 8, these same equations were run in databases including only those timepoints of interest.

Aims 2-3: Modeling change in health behaviors over time.

Separate analyses were performed to assess the change in each health behavior outcome from baseline to week 4. Level 1 equations for these analyses were: $HealthBehavior_{ij} = b_{0i}+b_{1i}Time_{ij}+e_{ij}$, where *i* represents each individual participant and *j* represents the three assessment time points; b_{0i} represents the outcome value for each individual at time 0 (i.e., intercept); b_{1i} represents the slope of change across time; and e_{ij} represents the error in predicting outcome *j* for participant *i*. At level 2, individual differences in intercept and slope were determined as functions of arm assignment with the equations $b_{0i} = \gamma_{00} + \gamma_{01}RandomizationArm_j+u_{0j}$ and $b_{1i} = \gamma_{10}+ \gamma_{11}RandomizationArm_j+u_{1j}$.

Aim 4: Modeling change in psychological variables over time.

Separate analyses were performed to assess the change in depression, generalized anxiety, and perceived stress from baseline to week 4. Level 1 equations for these analyses were: $PsychologicalVariable_{ij} = b_{0i}+b_{1i}Time_{ij}+e_{ij}$, where *i* represents each individual participant and *j*

represents the three assessment time points; b_{0i} represents the outcome value for each individual at time 0 (i.e., intercept); b_{1i} represents the slope of change across time; and e_{ij} represents the error in predicting outcome *j* for participant *i*. At level 2, individual differences in intercept and slope were determined as functions of arm assignment with the equations $b_{0i} = \gamma_{00} + \gamma_{01}\text{RandomizationArm}_j + u_{0j}$ and $b_{1i} = \gamma_{10} + \gamma_{11}\text{RandomizationArm}_j + u_{1j}$.

Aim 5: Assessing differences in anxiety sensitivity reduction and health behavior change as a function of weight status.

These analyses were conducted in the subsample of participants randomized to the intervention group. Level 1 equations remained as written above in Aims 1-3. Level 2 equations determined individual differences in intercept and slope as a function of weight status dichotomized as normal weight v. overweight/obesity: $b_{0i} = \gamma_{00} + \gamma_{01}\text{WeightStatus}_j + u_{0j}$ and $b_{1i} = \gamma_{10} + \gamma_{11}\text{WeightStatus}_j + u_{1j}$. In addition to total ASI-3 score, subfactor scores were also modeled as a function of weight status.

Aim 6: Exploring the relationship between change in anxiety sensitivity components (physical, social, cognitive concerns) and change in health behaviors.

First, each subfactor was modeled as a function of time within individuals with randomization arm added as a level 2 variable in order to assess whether there were any significant differences between groups with respect to rate of change in physical, social, and cognitive concerns related to anxiety sensitivity. In addition, change scores were calculated for each ASI-3 subscale from baseline to week 2 and for each health behavior from week 2 to week 4. Regression analyses were performed in order to determine whether change in health behaviors could be predicted from change in anxiety sensitivity. Lastly, change in ASI-3 from baseline to week 2 was

also correlated with change in health behaviors during that same time period in order to detect potential simultaneous change in these constructs.

Results

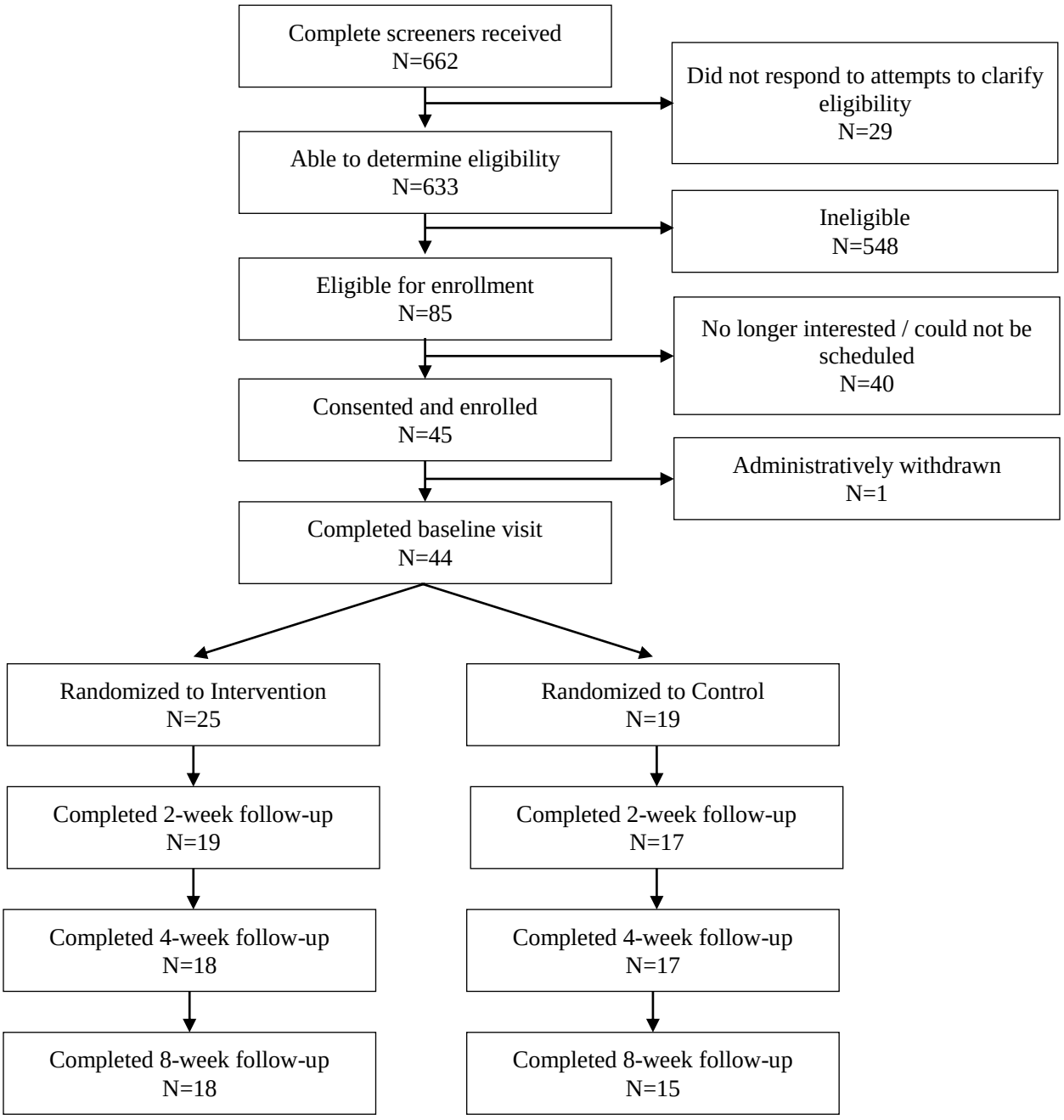
Sample Characteristics

Of 662 complete online screening questionnaires received, 85 individuals (12.8%) were eligible for the study. Figure 1 depicts participant flow throughout the course of the study from screening to 8-week follow-up. Reasons for ineligibility are listed in Table 3; 40.7% of those ineligible for the study were excluded based on multiple criteria. Of those eligible at screening, 45 (52.9%) provided informed consent for participation and completed their baseline visit. One participant was administratively withdrawn from the study following baseline visit due to adverse event at baseline; thus, 44 participants were randomized: 25 were assigned to the intervention condition and 19 were assigned to the control condition.

Table 3. Reasons for ineligibility at screening.

	Redundant		Non-Redundant	
	N	%	N	%
Unsafe to engage in physical activity (PAR-Q)	204	37.2	78	14.2
Reported routine physical activity (L-CAT)	354	64.6	167	30.5
Low anxiety sensitivity (ASI-3)	121	22.1	38	7.0
Currently in counseling or psychotherapy	117	21.4	34	6.2
Recent change in psychotropic medication	7	1.3	7	1.3
Psychiatric hospitalization within past 6 months	26	4.7	1	0.2
Multiple reasons for ineligibility	---		223	40.7

Figure 1. Participant Flow.



The majority of randomized participants were female (90.9%), racial/ethnic minority (63.6%), and reported a previous diagnosis of anxiety or depression (61.4%). Half (47.7%) of the participants were full-time students, 15.9% were part-time students, and 36.4% were not students at the time of enrollment. Chi-square analyses for categorical variables and independent sample t-

tests for continuous variables reflected no significant differences between participants in the control v. intervention group with respect to baseline characteristics (Table 4).

Table 4. Baseline characteristics.

	Intervention (n=25) N(%) or $\bar{x}$(SD)	Control (n=19) N(%) or $\bar{x}$(SD)	p
Age	25.1 (4.8)	27.3 (5.2)	0.15
Sex			0.44
Male	3 (12.0%)	1 (5.3%)	
Female	22 (88.0%)	18 (94.7%)	
Race			0.95
Non-Hispanic white	9 (36.0%)	7 (36.8%)	
Racial/ethnic minority	16 (64.0%)	12 (63.2%)	
Student Status			0.17
Full-time	12 (48.0%)	9 (47.4%)	
Part-time	6 (24.0%)	1 (5.3%)	
Not a student	7 (28.0%)	9 (47.4%)	
Weight Status			0.82
Normal weight	14 (56.0%)	10 (52.6%)	
Overweight/obesity	11 (44.0%)	9 (47.4%)	
Diagnosis of anxiety/depression	14 (56.0%)	13 (68.4%)	0.40
Previous psychotherapy	16 (64.0%)	12 (63.2%)	0.95
Current psychotropic medications	9 (36.0%)	5 (26.3%)	0.50
ASI-3 score	33.0 (10.2)	39.5 (15.5)	0.11
ISI score	12.4 (4.6)	15.4 (5.6)	0.70
Average hours sleep/night	8.0 (1.1)	8.4 (1.4)	0.41
Loss of control eating (episodes/wk)	0.1 (0.2)	0.3 (0.8)	0.19
Alcoholic drinks per hour during drinking episodes	0.7 (0.9)	1.3 (1.6)	0.11
Physical activity (minutes/wk)	157.0 (90.5)	159.6 (147.6)	0.94
Intention to increase physical activity	3.8 (0.9)	3.4 (1.0)	0.18

Though differential retention rates between intervention and control groups did not reach statistical significance (p=0.10), 6 participants did not complete assessments beyond baseline in the intervention group compared to 1 participant in the control group. There were no significant differences between groups with respect to follow-up timepoints: week 2 χ^2=3.38, p=0.19, week 4 χ^2=2.92, p=0.23, week 8 χ^2=1.03, p=0.60. Participants in the intervention group completed an

average of 5.2(SD=1.8) of 6 treadmill sessions. Only 5 participants completed fewer than the full 6 sessions, with none of these 5 participants providing data beyond baseline.

Manipulation Check

With 512 heart rate measurements taken across the 128 treadmill sessions completed, participants in the intervention condition remained within their target heart rate 99.0% of the time. There were 3 instances of heart rates falling below the target range and 2 instances of heart rate surpassing the target range. In all 5 cases, participants were asked to adjust the speed of their treadmill accordingly; in all cases, their next heart rate reading was within the target range.

Change in Anxiety Sensitivity over Time

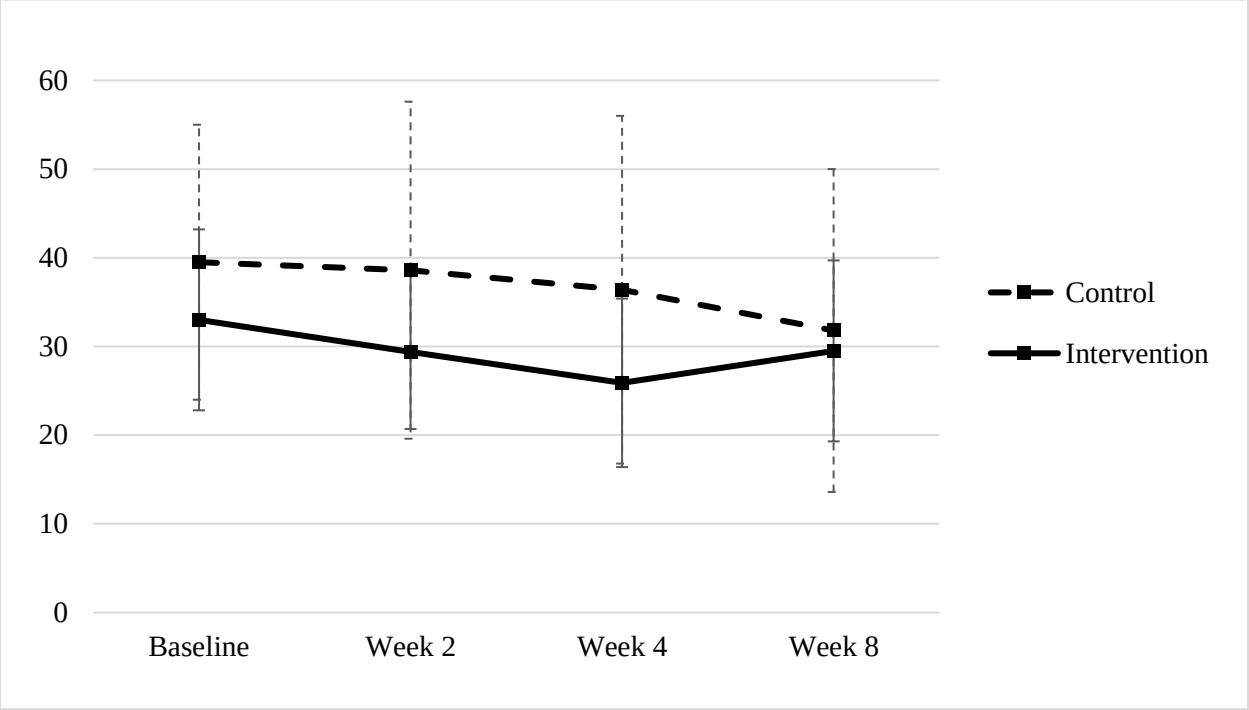
ASI-3 scores were normally distributed at all timepoints with scatterplots reflecting equivalent variances between groups and normally distributed, linear residuals. There were no significant outliers in scores; all data were included in analyses. The overall repeated measures model including between-participant analyses (randomization assignment) and within-participant analyses (ASI-3 at weeks 0, 2, 4, and 8) indicated a non-significant group-by-time interaction (Coeff=0.05, SE=0.34, t=0.15, p=0.90). This is depicted in Figure 2 below, with markers representing mean ASI-3 scores for each group at each of the 4 timepoints. In order to approximately replicate previous findings of significant differences in anxiety sensitivity reduction at shorter follow-up, the model was run using data from weeks 0, 2, and 4. Results were marginally significant and reflected greater ASI-3 reductions among participants in the intervention group compared to those in the control group (Coeff=-1.31, SE=0.84, t=-1.57, p=0.06). The change in ASI-3 scores from baseline to week 4 among participants in the intervention group represents a medium effect size, Cohen's d=0.40.

Next, group-by-time interactions were examined for each time segment: weeks 0 to 2, weeks 2 to 4, and weeks 4 to 8 in order to assess patterns of change over time throughout the study period. At week 2, participants in the intervention condition experienced greater reductions in anxiety sensitivity compared to those in the control condition at a level of marginal significance (Coeff=-2.34, SE=1.26, t=-1.86, p=0.07) and small effect size, Cohen's d=0.20. From weeks 2 to 4, there was no significant difference in rate of change between intervention and control (Coeff=-0.84, SE=1.10, t=-0.77, p=0.45). Similarly, there was no significant difference in rate of anxiety sensitivity change between intervention and control from week 4 to week 8 (Coeff=0.64, SE=0.47, t=1.35, p=0.19).

Analyses were repeated excluding participants (n=5) who were randomized to the intervention condition but did not complete all 6 sessions. Results were similar to those outlined above, with a non-significant group-by-time interaction (Coeff=0.03, SE=0.34, t=0.11, p=0.91) for the full model. The 4-week follow-up model was again marginally significant such that participants in the intervention group experienced greater reductions in anxiety sensitivity than did participants in the control group (Coeff=-1.36, SE=0.70, t=-1.95, p=0.06).

Time segment analyses using this completers subsample were also similar to those of the full sample. From weeks 0 to 2, participants in the intervention condition demonstrated larger reductions in anxiety sensitivity compared to those in the control condition at a marginal significance level (Coeff=-2.41, SE=1.26, t=-1.92, p=0.06). There was no group-by-time interaction from weeks 2 to 4 (Coeff=-0.84, SE=1.10, t=-0.77, p=0.45) or weeks 4 to 8 (Coeff=0.64, SE=0.47, t=1.35, p=0.19).

Figure 2. Change in Anxiety Sensitivity by Randomization Condition



Change in Health Behaviors over Time

Physical Activity. There were 3 cases with extreme outliers with respect to self-reported moderate-to-vigorous physical activity (PAR); these were not included in the analyses. Resultant PAR scores were normally distributed at all timepoints with scatterplots reflecting equivalent variances between groups and normally distributed, linear residuals. Analysis of change from week 0 to week 4 yielded a significant time effect such that physical activity declined over time for both groups (Coeff=164.88, SE=25.44, t=6.48, p<0.01); rate of this decline did not significantly differ between groups (Coeff=6.18, SE=9.74, t=0.63, p=0.53). Repeating this analysis including only those participants who had completed all 6 treadmill sessions yielded a similar pattern of results. Analyses were also conducted assessing change in hard or very hard physical activity from baseline to week 4; results were consistent with those including all intensities of physical activity: decline over time (Coeff=17.10, SE=7.43, t=2.30, p=0.03) but no significant interaction (Coeff=-

2.20, SE=3.31, t=-0.66, p=0.51). Once again, analysis of intervention completers yielded this same pattern of effects.

Self-reported intention to increase physical activity was normally distributed at all timepoints with scatterplots reflecting equivalent variances between groups and normally distributed, linear residuals. There were no significant outliers in scores; all data were included in analyses. When looking at change in intention to increase physical activity from baseline to week 4, results were similar to those found for physical activity with a significant time effect reflecting decreased intention (Coeff=3.42, SE=0.22, t=15.88, p<0.01), but no significant interaction (Coeff=-0.02, SE=0.08, t=-0.35, p=0.73).

Sleep. ISI scores were normally distributed at all timepoints with scatterplots reflecting equivalent variances between groups and normally distributed, linear residuals. There were no significant outliers in scores; all data were included in analyses. Analysis of change from week 0 to week 4 yielded a significant time effect such that ISI scores decreased over time for both groups (Coeff=15.4, SE=1.24, t=12.39, p<.01); rate of this decrease did not vary as a function of randomization group (Coeff=-0.58, SE=0.40, t=1.44, p=0.16).

With respect to average hours of sleep per night, one case represented an outlier and was removed from the analyses. Resultant values were normally distributed at all timepoints with scatterplots reflecting equivalent variances between groups and normally distributed, linear residuals. Results reflected those found in ISI scores: between baseline and week 4, average hours slept per night increased over time for participants across both groups (Coeff=8.36, SE=0.27, t=31.56, p<0.01), with no significant difference in rate of increase between groups (Coeff=0.05, SE=0.07, t=0.69, p=.50).

Binge Eating. Frequency of binge eating was extremely low over the course of the study period, with only 13 episodes reported by 8 participants across all timepoints. As such, these data deviated significantly from a normal distribution. Rather than examining binge eating as a continuous outcome, this variable was recoded as dichotomous (yes/no) for each timepoint, and between-group differences were assessed using chi-square tests. There were no significant differences between groups at any timepoint with respect to reports of binge eating, all *p*s > 0.05.

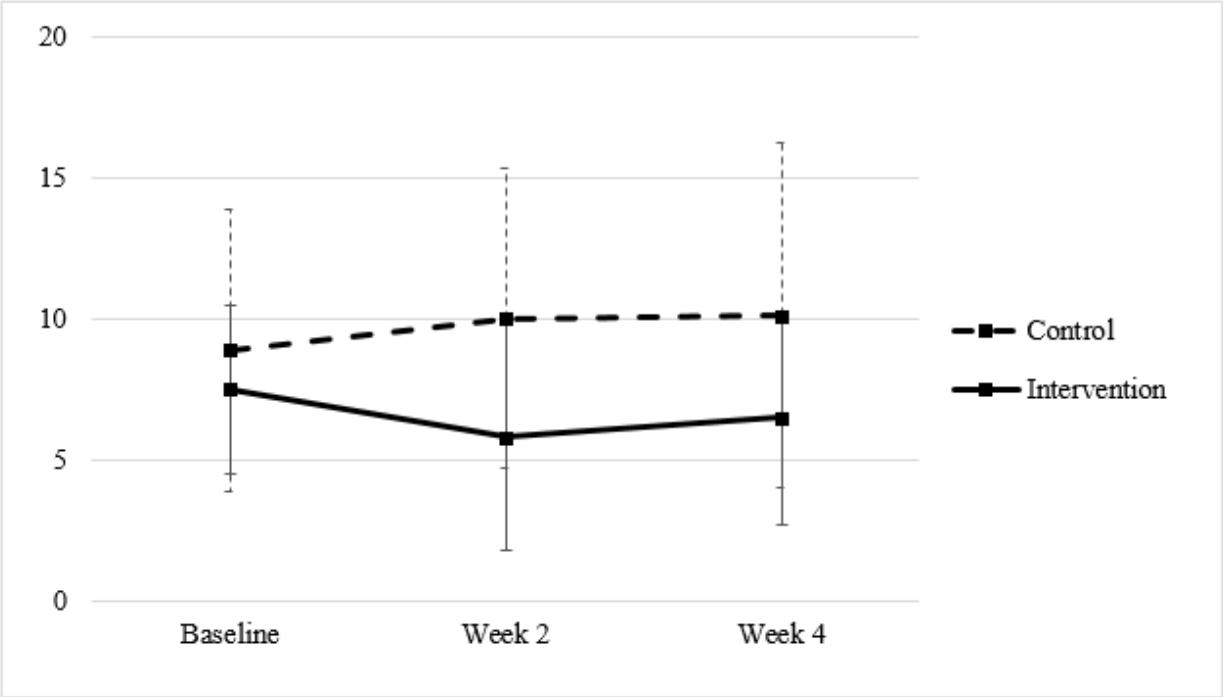
Alcohol Use. There was 1 case with an extreme outlier with respect to number of alcoholic drinks per hour when drinking; this case was removed from these analyses. Resultant data were normally distributed at all timepoints with scatterplots reflecting equivalent variances between groups and normally distributed, linear residuals. Consistent with previous results, analysis of change from week 0 to week 4 yielded a significant time effect (Coeff=1.03, SE=0.19, t=5.37, *p*<.01) reflecting fewer drinks per hour; there was no group-by-time interaction (Coeff=0.03, SE=0.07, t=0.36, p=0.72). Rates of binge drinking were extremely low in this sample, with only 10 episodes reported by 8 participants across all timepoints. This variable was recoded as dichotomous (yes/no) for each timepoint, and between-group differences were assessed using chi-square tests. There were no significant differences between groups at any timepoint with respect to reports of binge drinking, all *p*s > 0.05.

Change in Other Psychological Variables over Time

Depression. PHQ-8 scores were normally distributed at all timepoints with scatterplots reflecting equivalent variances between groups and normally distributed, linear residuals. There were no significant outliers in scores; all data were included in analyses. Examining the change between baseline and week 2 did yield a significant interaction effect such that PHQ-8 scores decreased among participants in the intervention group at a greater rate than those among

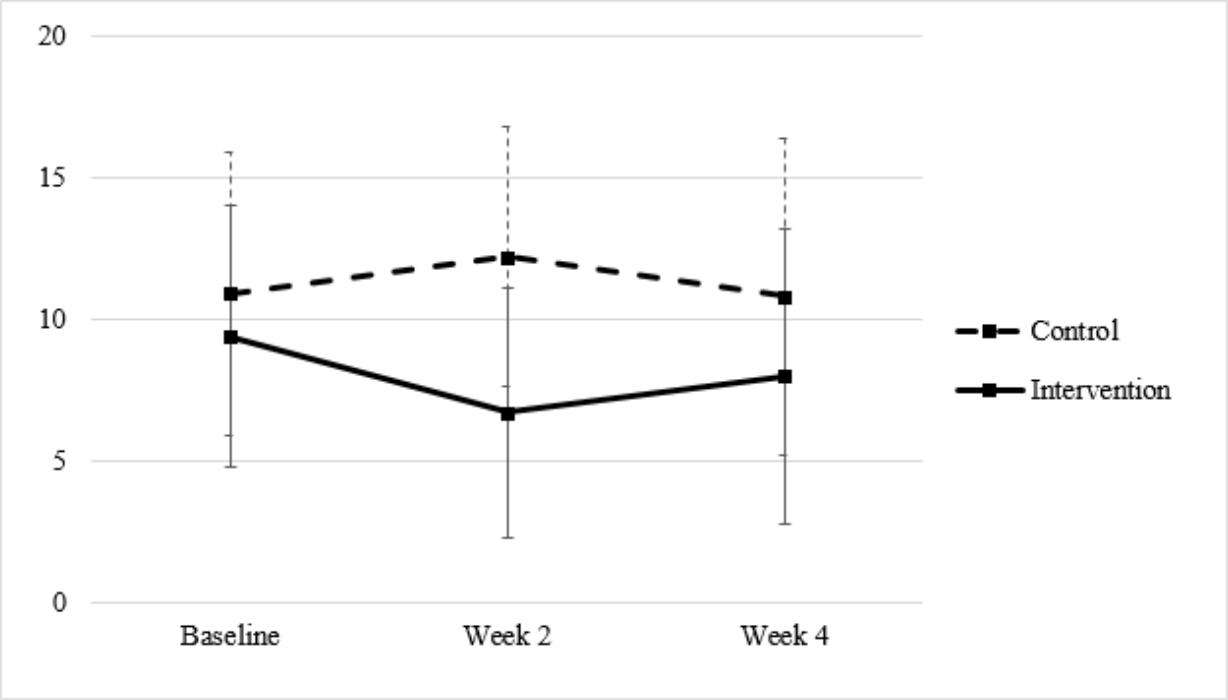
participants in the control group (Coeff=1.43, SE=0.68, *t*=2.10, *p*=0.04). The model including PHQ-8 scores at weeks 0, 2, and 4 indicated a non-significant group-by-time interaction (Coeff=0.57, SE=0.35, *t*=1.61, *p*=0.11).

Figure 3. PHQ-8 Scores by Randomization Condition.



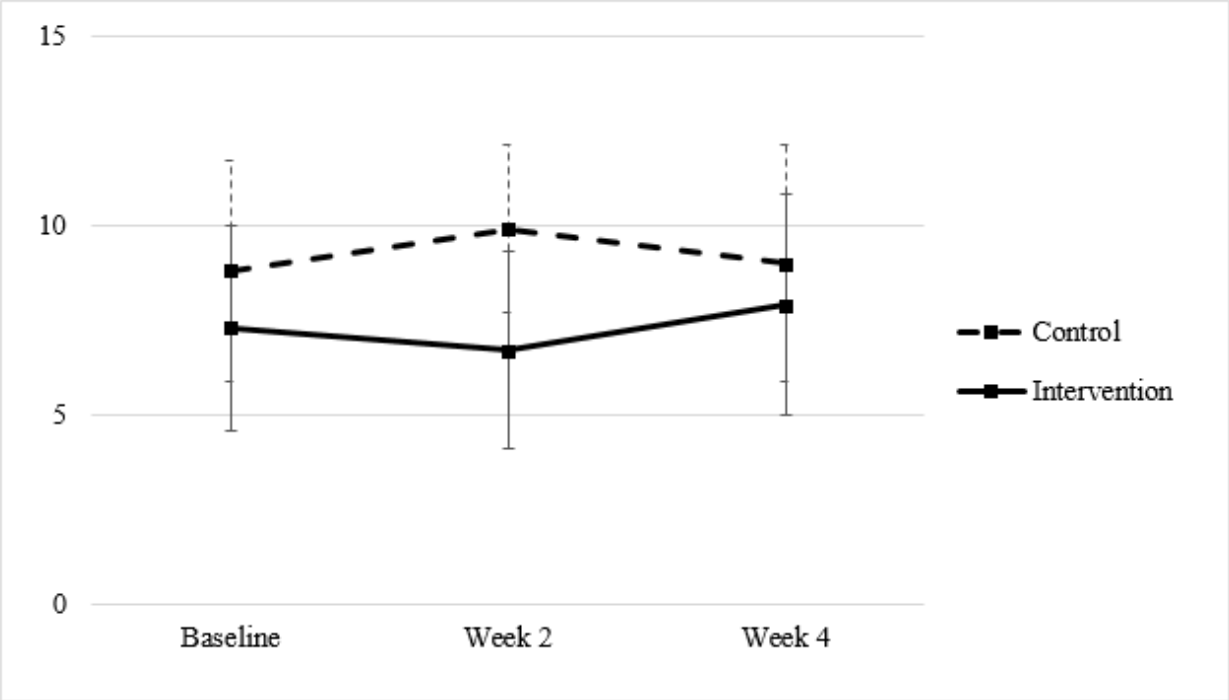
Anxiety. GAD-7 scores were normally distributed at all timepoints with scatterplots reflecting equivalent variances between groups and normally distributed, linear residuals. There were no significant outliers in scores; all data were included in analyses. Rates of change in GAD-7 scores from baseline to week 2 differed significantly between intervention and control conditions such that participants in the intervention condition demonstrated greater decreases in general anxiety symptoms than did participants in the control condition (Coeff=2.29, SE=0.69, *t*=3.33, *p*=.02). The model including GAD-7 scores at weeks 0, 2, and 4 reflected a non-significant group-by-time interaction (Coeff=0.49, SE=0.37, *t*=1.30, *p*=0.20).

Figure 4. GAD-7 Scores by Randomization Condition.



Perceived Stress. PSS-4 scores similarly met assumptions for normal distribution, homoscedasticity, and linear, normally distributed residuals. There were no significant outliers; thus, all data were included in analyses. Similar to findings for symptoms of depression and general anxiety, there was a significant group-by-time interaction effect such that PSS-4 scores decreased in the intervention group at a greater rate than for the control group (Coeff=0.77, SE=0.45, t=1.72, p=0.04). The model including PSS-4 scores at weeks 0, 2, and 4 indicated that there was not a significant group-by-time interaction (Coeff=0.13, SE=0.22, t=0.60, p=0.56).

Figure 5. PSS-4 Scores by Randomization Condition.



Role of Weight Status

There were no significant differences in rate of ASI-3 change between participants with normal weight compared to those with overweight/obesity across the 8-week study period (Coeff=0.62, SE=0.51, t=1.21, p=0.23). Similar findings emerged when running the model including data from weeks 0, 2, and 4 only (Coeff=1.31, SE=1.05, t=1.25, p=0.22). There were no significant differences with respect to changes in ASI-3 subscales as a function of weight status for physical (Coeff=0.27, SE=0.20, t=1.33, p=0.19) or cognitive (Coeff=0.08, SE=0.17, t=0.47, p=0.64) domains of anxiety sensitivity. There was a marginally significant group-by-time effect for ASI-3 social concerns (Coeff=0.43, SE=0.26, t=1.70, p=0.08) such that scores on this subscale decreased for participants of normal weight but remained stable for those with overweight/obesity. Whether or not participants completed all 6 treadmill exposures did not significantly differ as a function of weight status (χ^2=0.65, p=0.42).

Neither self-reported physical activity nor intention to increase physical activity varied as a function of weight status from baseline to week 4 (Coeff=7.36, SE=15.12, t=0.49, p=0.64 and Coeff=0.18, SE=0.13, t=1.35, p=0.20, respectively). There were no differences in rates of change for insomnia symptoms (Coeff=0.44, SE=0.66, t=0.68, p=0.50) or hours of sleep per night (Coeff=0.03, SE=0.08, t=0.46, p=0.65) between participants of normal weight compared to those with overweight/obesity. There were no significant differences between participants of normal weight compared to those with overweight/obesity in terms of binge eating at any timepoint, all ps >0.10. Rate of change for number of alcoholic drinks per hour during drinking episodes did not significantly vary by weight status (Coeff=0.02, SE=0.10, t=0.21, p=0.84), and there were no differences between weight statuses at any timepoint with respect to reports of binge drinking (all ps >0.10). Lastly, differences in rates of change in depression, general anxiety, and stress were examined by weight status. There were no significant differences between participants of normal weight and those with overweight/obesity throughout the 8-week study period (all ps >.10), nor when examining change over weeks 0 to 4 (all ps >0.10).

ASI Subscales

Physical. ASI-3 physical subscale scores were normally distributed at all timepoints with scatterplots reflecting equivalent variances between groups and normally distributed, linear residuals. There were no significant outliers in scores; all data were included in analyses. Over the course of the 8-week study period, there was a significant time effect (Coeff=10.23, SE=1.10, t=9.26, p<.01) such that physical concerns related to anxiety sensitivity decreased across both randomization conditions. These declines did not vary as a function of randomization condition, i.e., there was no significant group-by-time interaction (Coeff=0.04, SE=0.15, t=0.30, p=0.77). Change in ASI-3 physical subscale scores from baseline to week 2 did not significantly predict

change in reported physical activity (t=0.92, p=0.37), behavioral intention to increase physical activity (t=0.98, p=0.34), ISI score (t=1.96, p=0.83), hours of sleep (t=0.95, p=0.70), or alcoholic drinks per hour (t=0.15, p=0.62) from week 2 to week 4. Additional correlational analyses were conducted assessing change within time period (i.e., change in ASI-3 physical concerns from baseline to week 2 correlated with change in health behaviors from baseline to week 2) in order to identify any potential simultaneous changes in these constructs. Decreases in physical concerns related to anxiety sensitivity from baseline to week 2 were associated with decreases in ISI scores (r=0.40, p=0.03) and increases in behavioral intention to increase physical activity (r=0.36, p=0.05). There were no significant associations between change in ASI-3 physical concerns scores and change in health behaviors from weeks 2 to 4 (all ps >0.11).

Cognitive. ASI-3 cognitive subscale scores were normally distributed at all timepoints with scatterplots reflecting equivalent variances between groups and normally distributed, linear residuals. There were no significant outliers in scores; all data were included in analyses. Over the course of the 8-week study period, there was a significant time effect (Coeff=13.11, SE=1.54, t=8.50, p<.01) such that cognitive concerns related to anxiety sensitivity decreased across both randomization groups. These declines did not vary as a function of randomization condition, i.e., there was no significant group-by-time interaction (Coeff=0.06, SE=0.13, t=0.05, p=0.96). Change in ASI-3 cognitive subscale scores from baseline to week 2 did not significantly predict change in self-report physical activity (t=0.62, p=0.54), behavioral intention to increase physical activity (t=0.14, p=0.89), ISI score (t=0.09, p=0.93), hours of sleep (t=0.40, p=0.70), or alcoholic drinks per hour (t=1.54, p=0.13) from week 2 to week 4. There were no significant associations between change in ASI-3 cognitive subscale scores and change in health behaviors from baseline to week

2 (all *p*s >0.12). Similarly, there were no significant correlations from week 2 to week 4 (all *p*s >0.27).

Social. ASI-3 social subscale scores similarly met assumptions for normal distribution, homoscedasticity, and linear, normally distributed residuals at all timepoints. There were no significant outliers; thus, all data were included in analyses. Over the course of 8 weeks, there was a significant time effect (Coeff=16.56, SE=1.33, t=12.48, p<.01) such that social concerns related to anxiety sensitivity decreased across both groups. These declines did not vary as a function of randomization condition, i.e., there was no significant group-by-time interaction (Coeff=0.04, SE=0.16, t=0.27, p=0.79). Change in ASI-3 social subscale scores from baseline to week 2 did not significantly predict change in physical activity (t=0.92, p=0.37), intention to increase physical activity (t=0.20, p=0.84), ISI score (t=0.25, p=0.93), hours of sleep (t=0.40, p=0.70), or alcoholic drinks per hour (t=0.02, p=0.98). When examining the relationship between changes in ASI-3 social concerns and change in health behaviors within the same time period (i.e., baseline to week 2 and week 2 to week 4), no significant correlations emerged (all *p*s >0.17).

Discussion

Consistent with the first hypothesis of this study, reductions in anxiety sensitivity produced by the intervention eroded by 6-week post-treatment follow-up (week 8 assessment point). This represents the longest no-intervention follow-up for this intervention paradigm; thus, this finding represents a novel contribution and indicates that a brief, low-intensity intervention might not be sufficient on its own to produce long-lasting changes in anxiety sensitivity. Findings of previous studies reporting reductions in anxiety sensitivity at post-treatment and short-term no-treatment follow-up were reproduced in the current study with marginal statistical significance. Lack of statistical significance in this case is likely a power issue, especially given effect sizes signifying

some degree of clinical importance in the current study. At the same time, the effects produced by the intervention described here are weaker than those previously reported in the literature, which may also contribute to lack of statistically significant findings. Broman-Fulks & Storey (2008) found anxiety sensitivity reductions of medium-to-large magnitude, Cohen's *d*=0.75, at 2-week (post-treatment) follow-up; in the current study, the effect size for the intervention group over this same time period was *d*=0.20. In their 2008 investigation of this same paradigm, Smits and colleagues found post-treatment anxiety sensitivity reductions reaching an effect size of *d*=1.46. The post-treatment effect size produced by the intervention in the current study was more consistent with that found in a 6-session low-intensity (1-mph treadmill walking) exercise exposure for anxiety sensitivity, *d*=0.23 (Broman-Fulks et al., 2004), despite participants' heart rates remaining in a range that would indicate moderate intensity at 99.0% of readings.

There are several potential explanations for the relatively small intervention effects seen in the current study. First, despite demographic similarities between samples with respect to heightened anxiety sensitivity, young adult age range, and majority female gender, the sample in the current study was predominately of racial/ethnic minority background while non-Hispanic White participants comprised the majority of samples in the other studies cited above. It is possible that cultural differences in the experience of anxiety may limit the effectiveness of aerobic exercise as an exposure for anxiety sensitivity. For example, in a study examining differences in panic disorder symptoms between Whites and African Americans, Friedman and Paradis (2002) found that African Americans were more likely to report numbness/tingling in their extremities than Whites. Aerobic exercise-based treadmill exposures would not necessarily allow for habituation to this and other culturally-influenced experiences of anxiety; instead, this would require a more tailored intervention to allow for targeting of specific symptoms through other means.

Cultural differences might play a role in the measurement of anxiety sensitivity as well, with some studies (Carter, Miller, Sbrocco, Suchday, & Lewis, 1999; Hunter, Keough, Timpano, & Schmidt, 2012) concluding that the three components of anxiety sensitivity (social, physical, and cognitive concerns) do not adequately capture the experience of anxiety sensitivity for African Americans. Instead, these authors propose a four-factor solution to the ASI: cognitive, emotional control, fear of cardiovascular problems, and unsteadiness. Hunter and Schmidt (2010) raise the possibility that African Americans are more likely to interpret physiological sensations of anxiety as symptoms of physical illness and pursue medical explanations or interventions rather than place stock in a psychological explanation. Despite these suggestions of cultural variations in experiences of anxiety sensitivity, there is little research regarding how individuals of different racial/ethnic backgrounds respond to interventions targeting anxiety sensitivity. This represents an important area for future research, particularly given the overlap between anxiety sensitivity, weight promoting behaviors, and higher risk for cardiovascular disease among African Americans.

The unexpectedly high rates of physical activity reported at baseline may also have contributed to the weak intervention effects seen in the current study. Despite screening procedures meant to exclude participants engaging in regular physical activity, mean baseline levels of reported moderate-to-vigorous physical activity were relatively high. The intent in including only sedentary participants was to capitalize on the novelty of this combination of physiological sensations (e.g., increased heart rate, shortness of breath, sweating, muscle tension/fatigue) as an exposure to symptoms of anxiety. If participants were having this experience in a naturalistic setting shortly before beginning the study, however, this may have reduced the effectiveness of the intervention. As this is the first anxiety sensitivity aerobic exercise exposure study to measure physical activity, it is unknown how this finding compares to the behavior of previous samples.

Another unexpected finding was that a small percentage of participants exhibited a decrease in anxiety sensitivity between screening and baseline. Differences in ASI scores between screening and baseline were not included in other reports of similar interventions, making it difficult to determine whether this is to be expected, or whether this represents an idiosyncrasy of the current sample.

Lastly, there may have been study design issues that contributed to the anxiety sensitivity outcomes reported here. For instance, though all research staff was thoroughly trained in all intervention and data collection procedures, those running treadmill sessions varied greatly with respect to prior training and expertise in psychology. This may have weakened the impact of the psychoeducation provided to the intervention group, particularly if participants had nuanced questions regarding the exposures. Again, previous reports of this intervention paradigm provide few details regarding who delivered the psychoeducation components.

The reductions in anxiety sensitivity exhibited by control group participants in the current study are also worthy of comment, as this was an unexpected finding. The lack of an active comparison or waitlist condition may have inadvertently produced threats to internal validity due to potential regression to the mean of elevated ASI-3 scores. Previous studies vary in their approach to control group, with many choosing to include an active intervention or wait-list comparison group (e.g., Broman-Fulks et al., 2004; Smits et al., 2008; Broman-Fulks et al., 2015; LeBouthiller & Asmundson, 2015). While yet other studies chose an assessment-only control condition (e.g., Broman-Fulks & Storey, 2008; Broman-Fulks et al., 2015), the follow-up periods were significantly shorter than that of the current study and reductions in anxiety sensitivity for control group participants were not seen. It is possible that because participants in the current study

knew they were not receiving treatment for anxiety sensitivity as part of this trial, they engaged in alternative strategies for coping with anxiety sensitivity which were not measured here.

Contrary to the pre-specified hypotheses, reported moderate-to-vigorous intensity physical activity decreased from baseline to week 4. This decline was seen across both participants in the intervention group and those in the control group with no interaction effect—that is, this change occurred at a similar rate for participants regardless of randomization assignment. The unexpectedly high baseline rates despite screening for sedentary lifestyles may play a role here such that this decrease in physical activity represents regression to participants' typical levels of exercise. Rolling—rather than cohort-based—recruitment and enrollment makes it unlikely that these time effects are due to seasonal factors such as change in weather or the beginning/end of the semester.

In addition, decreases in moderate-to-vigorous physical activity might also be a function of decreases in anxiety sensitivity over this same period of time. Heightened anxiety sensitivity at baseline may have confounded participants' ability to accurately report moderate-to-vigorous physical activity due to these individuals' heightened reactivity to internal physiological cues. This may have resulted in their classifying activity as "moderate" when it would objectively be measured as "light" due to their increased awareness of physiological changes in sensations such as shortness of breath and increased heart rate. As this reactivity decreased over time via reductions in anxiety sensitivity, participants might have recalibrated their interpretation of physical activity intensity, leading to more accurate classification and an apparent decrease in moderate-to-vigorous physical activity.

Reassuring participants that their responses regarding physical activity will not be judged and that accuracy is the goal of the interview is a critical aspect of administering the 7-Day PAR.

Interviewers were trained to be mindful of their reactions to participant reports in order to avoid responding differentially to reports of activity versus reports of no activity, thereby inadvertently reinforcing one or the other. However, because the first administration of the 7-Day PAR took place in-person, participants may have experienced stronger social desirability demands to respond with inflated reports of baseline physical activity compared to future administrations via telephone. Indeed, both high social desirability and need for social approval have been linked to over-reporting of physical activity on the PAR compared to physical activity energy expenditure assessed via doubly-labeled water and physical activity intensity/duration assessed via accelerometer (Adams et al., 2005). Though validation analyses found no significant differences in accuracy of reporting between in-person and telephone PAR interviews (Hayden-Wade, Coleman, Sallis, & Armstrong, 2003), it is possible that social desirability and approval factors were activated in a highly anxious sample as in the current study, leading to differences between administration modalities.

Participants' high levels of physical activity at baseline despite seeming to be largely sedentary at screening raises a potential discrepancy between measurement of physical activity using the L-CAT versus the PAR. The L-CAT is a single-item measure that asks about general patterns of behavior, whereas the PAR is a semi-structured interview designed to capture bouts of moderate-to-vigorous physical activity. Both the L-CAT (Kiernan et al., 2013; Ross et al., 2018) and the 7-Day PAR (Leenders, Sherman, & Nagaraja, 2000; Sloane, Snyder, Demark-Wahnefried, Lobach, & Kraus, 2009) have been validated against objective measures of physical activity (e.g., pedometer and accelerometer) with a sufficient level of agreement between these methods. Of note, however, there are mixed reports as to whether the PAR underestimates (Sloane et al., 2009) or overestimates (Leenders et al., 2000) physical activity compared to objective measurement

using accelerometer. To date, there has been no comparison between the L-CAT, PAR, *and* an objective measure of physical activity, which would be helpful in interpreting the results of the current study.

An examination of the relationship between the L-CAT and the PAR would also be useful when considering how a future study might best approach screening for sedentary participants. Previous studies of aerobic exercise exposure (e.g., Broman-Fulks et al., 2004; Broman-Fulks & Storey, 2008; Smits et al., 2008; LeBouthillier & Asmundson, 2017) do not describe their methodology for assessing level of physical activity in their screening process. Thus, the inclusion of the L-CAT as a brief screening measure of typical physical activity in the current study represented a potential improvement upon prior methodologies in this respect. However, if this measure is not in fact appropriately categorizing participants as sedentary versus non-sedentary, other methods for screening must be considered. Implementing the PAR at screening would be extremely burdensome for both research staff and participants given the volume of screening questionnaires received and is likely not a viable solution. One option would be to continue using the L-CAT at screening, but determine final eligibility for the study at baseline after participants have completed the PAR or—ideally—accelerometry. This would ensure that participants are both typically sedentary (L-CAT) and specifically sedentary within the past week (PAR/accelerometry), preserving the exposure as a novel task. This would potentially require a much larger volume of participants presenting for in-person baseline assessment, as 21 of 44 participants in the current study (12 of 25 in the intervention arm) reported engaging in > 150 minutes of moderate-to-vigorous physical activity at baseline despite being categorized as sedentary at screening.

Future investigations of the relationship between anxiety sensitivity and physical activity could benefit from using both self-report and objective measures of physical activity at baseline

and follow-up timepoints in order to increase construct validity. Within the framework of behaviors that may contribute to maintenance of a healthy weight, it might also be of interest to assess changes in bouts of *intentional* exercise in addition to moderate-to-vigorous physical activity overall. Administering two sources of measurement for physical activity would allow for a semi-structured interview such as the PAR to be used as a querying tool for data collected via accelerometer in order to separate intentional exercise from lifestyle activity. This distinction might be particularly important because while lifestyle physical activity may reach the threshold of moderate-to-vigorous intensity for bouts of 10 minutes or more, it is less likely than intentional physical activity to occur on a regular basis over sustained periods of time. With current guidelines recommending 250 minutes of exercise per week to facilitate clinically significant weight loss (Donnelly et al., 2009), behavioral strategies such as goal-setting and self-monitoring of physical activity are critical to success in meeting this benchmark. Indeed, planning and monitoring of physical activity was significantly associated with greater levels of physical activity in a sample of adults who had successfully lost weight (Fuglestad, Jeffery, & Sherwood, 2012). Compared to those who have regained weight following initial weight loss, those who have sustained clinically significant weight loss report higher levels of physical activity, highlighting the importance of exercise not only for weight loss itself—but also successful maintenance of progress (McGuire, Wing, Klem, & Hill, 1999).

Improvements with respect to sleep and alcohol use among participants in both randomization conditions from baseline to week 4 could be driven by the fact that reductions in anxiety sensitivity were seen among participants in both conditions over this time period. Further, despite reaching the level of statistical significance, it remains questionable whether these changes represent clinically meaningful improvements. For example, mean hours of sleep per night

increased from 8.10 at baseline to 8.40 at 4-week follow-up, representing an increase of approximately 18 minutes of sleep per night. In this sample, there were very few (*n*=3) participants who reported poor sleep to the extent that this would be associated with increased risk for overweight/obesity among young adults, i.e., < 6 hours per night (Hart, LaRose, Fava, James, & Wing, 2013). In fact, only 5 participants averaged below the recommended 7-9 hours of sleep per night (Hirshkowitz et al., 2015).

Similarly, the mean rate of alcoholic drinks per hour during drinking episodes decreased from 0.83 at baseline to 0.63 at 4-week follow-up. There were very few participants who met the threshold at any timepoint for binge drinking, thus limiting our ability to conclude whether an interoceptive exposure for anxiety sensitivity might be effective for reducing this behavior. The same is true for reports of binge eating among participants in the current sample, which occurred at a very low frequency. Despite the documented associations between anxiety sensitivity and alcohol use/binge eating, these behaviors did not appear to be particularly problematic in the current sample; however, it is possible that they did occur at higher rates compared to those with low anxiety sensitivity, who were not included in this study. Alternatively, it is possible that for some individuals with high anxiety sensitivity, the sense of being out of control associated with alcohol use and/or binge eating is reminiscent of anxiety and therefore aversive; there may be moderating factors at play here that warrant additional research. Future studies investigating the effects of exercise-based anxiety sensitivity treatments on these specific health behaviors should select for high rates of these behaviors in their eligibility criteria.

Participants in the intervention condition demonstrated statistically significant decreases in symptoms of anxiety, depression, and perceived stress during the active exposure period compared to participants in the assessment-only condition. These findings are consistent with those of Smits

and colleagues (2008), who reported significant reductions in depression (measured with the BDI) and anxiety (measured with the BAI) for participants assigned to aerobic exercise compared to those assigned to waitlist-control. The authors were able to conduct mediation analyses demonstrating that reductions in anxiety sensitivity mediated the relationship between exercise and mood improvements. Results of the current study are also partially consistent with the findings of LeBouthillier and Asmundson (2017), who extended the 2-week intervention to 4 weeks and found that aerobic exercise was associated with significant reductions in self-reported stress and general anxiety; however, the authors did not find a significant effect with respect to symptoms of depression. In the current study, these improvements were not sustained beyond the end of the intervention; however, it is unknown whether a greater magnitude of reduction in anxiety sensitivity might have been associated with prolonged symptom reductions in depression, generalized anxiety, and stress.

Among those randomized to the intervention condition, there were no significant differences between participants of normal weight and those with overweight/obesity with respect to changes in anxiety sensitivity, associated psychological variables, or related health behaviors. While Hearon and colleagues (2014) found an inverse relationship between BMI and physical activity among those with elevated anxiety sensitivity, this was not replicated in the current sample, where there was no significant correlation between BMI and baseline physical activity (r=0.14, p=0.40). This may be an issue related to physical activity measurement, as Hearon and colleagues used accelerometers to assess these data. In addition to revising study methods to include objective means of capturing physical activity, future explorations of the relationship between anxiety sensitivity and weight status might also expand the breadth of health behaviors assessed. For example, those high in anxiety sensitivity might avoid beverages such as soda and

energy drinks due to physiological effects of caffeine that resemble anxiety. In this case, anxiety sensitivity may contribute to weight gain prevention through avoidance of these high-calorie drinks.

There was a marginally significant finding reflecting greater change in social concerns related to anxiety for participants of normal weight than for those with overweight/obesity. Though a previous investigation (Smits et al., 2008) found that cognitive restructuring did not enhance the effects of interoceptive exposure, adding this component to the intervention may be helpful in clarifying and reducing cognitive distortions regarding social perceptions of anxiety specifically among those with overweight/obesity. This remains an area for additional investigation, as Smits and colleagues did not include data regarding BMI or weight status of their sample. Alternatively, supplementing the exercise exposure paradigm with exposure to anxiety-provoking social situations might also help to decrease anxiety sensitivity social concerns among those with overweight/obesity. Dixon, Kemp, Farrell, Blakey, and Deacon (2015) developed and tested interoceptive exposure tasks to specifically target social concerns related to anxiety sensitivity (e.g., blushing or trembling in the presence of others) which might be of interest to incorporate into an aerobic exercise paradigm.

Another potential explanation for this finding is that the items on the social concerns subscale of the ASI-3 may tap into constructs other than anxiety sensitivity for those with overweight/obesity. For example, items such as "it is important for me not to appear nervous" and "when I begin to sweat in a social situation, I fear people will think negatively of me" may also reflect internalized experiences of weight-based discrimination or greater social vigilance due to weight rather than due to anxiety sensitivity. This may be especially true among African Americans, for whom social vigilance is particularly salient (Hicken, Lee, & Hing, 2018). If the

items on the ASI-3 social concerns subscale overlap with powerful constructs such as vigilance in anticipation of discrimination, it is unlikely that an intervention targeting anxiety sensitivity would produce changes in this domain.

With respect to other findings regarding subfactors of anxiety sensitivity, change in ASI-3 subscales during the intervention period (baseline to week 2) did not predict change in any health behaviors over the following 2 weeks. When examining simultaneous change within time periods (i.e., baseline to week 2 and week 2 to week 4), decreases in physical concerns related to anxiety sensitivity were significantly associated with decreases in symptoms of insomnia and increases in behavioral intention to increase physical activity. Conclusions regarding this finding are limited due to small sample size and inability to speak to causation, but may indicate that reinterpretation of the physiological experiences associated with anxiety promotes improved sleep quality and bolsters motivation to increase physical activity. These associations warrant additional exploration in a larger sample in order to better ascertain the nature of these relationships, but are consistent with previous reports that reductions in the physical domain of anxiety sensitivity are associated with reduced sleep disturbance (Short et al., 2015). If change in physical concerns related to anxiety sensitivity are found to produce changes in sleep and physical activity, the tailoring of the interoceptive exposure to participants' individual physiological experiences of anxiety might become even more important.

Strengths of the current study include a randomized design and no-treatment follow-up period that doubles those currently reported in the literature. These specifications allowed for testing and confirmation of the hypothesis that reductions in anxiety sensitivity produced by a 2-week exercise-based exposure were not maintained at 6-weeks post-treatment. In addition, this study used the ASI-3 to measure anxiety sensitivity, which has been updated from the original ASI

and ASI-R to more robustly capture the factors comprising this construct: physical concerns, social concerns, and cognitive concerns (Taylor et al., 2007). We also collected data regarding physical activity and examined these as outcomes of this exercise-based intervention, which represents a novel contribution to the literature in this area. Lastly, this study recruited and enrolled a diverse sample of young adults with respect to student status, weight status, and race/ethnicity, which increases confidence in generalizability of these findings to young adults with high anxiety sensitivity in the population.

At the same time, conclusions of this study were limited by several notable factors. The power estimate for determining sample size was based on the primary aim of assessing change in anxiety sensitivity over four timepoints; thus, analyses for subsequent aims and hypotheses may have been underpowered to detect an effect. This is compounded by the fact that attrition was greater than anticipated at follow-up timepoints; however, HLM is robust in its handling of missing data as analyses do not require or assume equal number of observations (Kwok et al., 2008; Shin et al., 2009; Smits et al., 2008). In addition, the compensation structure designed to enhance retention (i.e., $15 bonus for completing all assessment timepoints) may have actually undermined this goal; it is possible that once participants missed an assessment, they perceived little incentive to resume participation because they had already lost out on earning the bonus. Lastly, it appears as though reliance on the PAR for collecting physical activity data is a limitation of the current study. This might be due in part to under-reporting of moderate-to-vigorous physical activity due to reinterpretation of physiological cues as anxiety sensitivity decreased throughout the study period. It is possible that the 7-Day PAR is not a valid instrument for assessing physical activity in a population with heightened anxiety—particularly in the context of a study aiming to produce changes in anxiety, which could lead to changes in the way physical activity intensity is perceived.

This overlap in reliance on interpretation of physiological sensations produces a significant confound, making it difficult to interpret results with respect to physical activity in the current study.

This study advances the landscape of our knowledge regarding anxiety sensitivity, its treatment, and its relationship with health behaviors; in addition, it highlights several areas for next steps in this area. First, future explorations of the relationship between anxiety sensitivity and physical activity would benefit from multidimensional assessment of physical activity. Pairing a self-report instrument with objective measurement such as accelerometry would provide several avenues for contextualizing the findings of the current study and advancing our knowledge regarding the association between anxiety sensitivity and physical activity. Determining whether the 7-Day PAR is a valid instrument for assessing physical activity in a population highly sensitive to anxiety is a critical to continued work in this area. In addition, multidimensional assessment would provide opportunities for querying specific types of physical activity such as intentional exercise. This would also allow investigators to ensure capture of physical activity that might not be captured by a wrist-worn accelerometer—or, alternatively, rule out movement detected by the accelerometer that would not be classified as physical activity.

The finding that physical activity decreased among participants assigned to both randomization conditions may be an artifact of measurement using the PAR, but may also suggest that the intervention produced insufficient reductions in anxiety sensitivity in order to produce the expected ripple effect. As yet another alternative, this finding might indicate that experiencing reductions in anxiety sensitivity while exercising is not sufficient to produce increases in physical activity in a naturalistic setting. A 3-arm randomized controlled trial testing the role of psychoeducation regarding physical activity would be interesting in order to examine what might

be necessary to increase exercise in this population. Randomizing to 1) the exposure paradigm as executed in the current study; 2) the exposure paradigm paired with psychoeducation regarding increasing physical activity; or 3) psychoeducation regarding increasing physical activity alone, would allow for a better understanding of what is necessary to decrease exercise avoidance due to anxiety sensitivity and promote sustained physical activity after the intervention period. Psychoeducation in this design might entail providing normative feedback regarding physical activity level in comparison to national recommendations and teaching effective behavioral strategies for increasing physical activity such as goal-setting and problem-solving.

In addition, more work is needed in order to determine individual attributes that may influence the effectiveness of exercise-based interventions for anxiety sensitivity. Though the results of the current study suggest that those of normal weight and those with overweight/obesity experience similar reductions in overall anxiety sensitivity with this exposure, the finding that changes in social concerns regarding anxiety did not change for those with overweight/obesity indicates that certain dimensions of anxiety sensitivity may be differentially responsive to the intervention based on weight status. Exploring the role of race/ethnicity also warrants further attention, as some evidence suggests that cultural factors may influence how anxiety is experienced. Future investigations might focus on collecting qualitative data regarding the experience of anxiety sensitivity among racial/ethnic minorities in order to determine whether this intervention paradigm appropriately targets sensations associated with anxiety. Alternatively, a design similar to that of the current study with a larger sample size and randomization stratified by race would be well-positioned to investigate differential effects of an exercise-based anxiety sensitivity intervention.

Another factor to consider when examining what works best for whom is the type of physical activity assigned as interoceptive exposure, as a recent study (LeBouthillier & Asmundson, 2017) found that aerobic exercise and resistance training produced differential effects in terms of anxiety-related constructs such as disorder-specific symptoms, distress tolerance, anxiety sensitivity, and stress. Furthermore, we know very little about the dose-response effects of exercise-based exposures. Recent investigations have examined 1-session exposures (Broman-Fulks et al., 2015) and 12-session exposures (i.e., 3 sessions per week over 4 weeks, LeBouthillier & Asmundson, 2017), but variations in implementation and assessment limit our understanding of magnitude and sustainability of the effects. While varying intensities of physical activity have been compared within a single trial (Broman-Fulks et al., 2004), no studies to date have randomly assigned participants to varying doses or schedules of moderate intensity aerobic physical activity. These designs would provide insight regarding whether "more is better," or whether intervention effects reach a ceiling effect after a certain dosage. In addition, such studies might also shed light on how best to sustain intervention effects—perhaps through the provision of maintenance sessions or by providing participants with instructions for self-guided exposures following the initial intervention period.

Conclusion

Taken together, the primary findings of this trial suggest that effects of a 6-session aerobic exercise exposure for anxiety sensitivity are not sustained for more than 4 weeks following completion of the intervention. The self-report nature of physical activity in the current study limit conclusions regarding the effects of this intervention on patterns of physical activity, but suggest that additional psychoeducation or coaching might be necessary in order to produce behavioral changes in a naturalistic setting. The intervention was not associated with significant changes in

sleep, alcohol use, or binge eating compared to control. Strengths include randomized design, extended no-treatment follow-up period, and a diverse sample with respect to weight status and race/ethnicity. Limitations include physical activity measurement and small sample size to test secondary aims. Future research in this area should assess physical activity using multidimensional methods, explore individual variability in treatment response, and examine dose-response relationship of exercise-based interventions. Continued research regarding the role of anxiety sensitivity in health behaviors has the potential to allow for improved tailoring and personalization in the context of health promotion programs.

References

Adams, S.A., Matthews, C.E., Ebbeling, C.B., Moore, C.G., Cunningham, J.E., Fulton, J., Hebert, J.R. (2005). The effect of social desirability and social approval on self-reports of physical activity. *American Journal of Epidemiology, 161*(4), 389-398.

Allan, N.P., Felton, J.W., Lejuez, C.W., MacPherson, L., & Schmidt, N.B. (2016). Longitudinal investigation of anxiety sensitivity growth trajectories and relations with anxiety and depression symptoms in adolescence. *Development and Psychopathology, 28*(2), 459-469.

American Psychological Association (2016). Stress in America: The impact of discrimination. Retrieved from http://www.apa.org/news/press/releases/stress/2015/impact-of-discrimination.pdf.

Anderson, D.A., Shapiro, J.R., Lundgren, J.D. (2003). The freshman year of college as a critical period for weight gain: An initial evaluation. *Eating Behaviors, 4*(4), 363-367.

Anestis, M.D., Holm-Denoma, J.M., Gordon, K.H., Schmidt, N.B., & Joiner, T.E. (2008). The role of anxiety sensitivity in eating pathology. *Cognitive Therapy and Research, 32*(3), 370-385.

Anestis, M.D., Selby, E.A., Fink, E.L., & Joiner T.E. (2007). The multifaceted role of distress tolerance in dysregulated eating behaviors. *International Journal of Eating Disorders, 40*(8), 718-726.

Armstrong, K.A., Khawaja, N.G., & Oei, T.P.S. (2006). Confirmatory factor analysis and psychometric properties of the Anxiety Sensitivity Index—Revised in clinical and normative populations. *European Journal of Psychological Assessment, 22*(2), 116-125.

Arntz, A. (2002). Cognitive therapy versus interoceptive exposure as treatment of panic disorder without agoraphobia. *Behavior Research and Therapy, 40*(3), 325-341.

Babson, K.A., Trainor, C.D., Bunaciu, L., & Feldner, M.T. (2008). Sleep anticipatory anxiety and sleep onset latency. *Journal of Cognitive Psychotherapy, 22*, 258-270.

Baker, A.W., Keshaviah, A., Goetter, E.M., Bui, E., Swee, M., Rosencrans, P.L., & Simon, N.M. (2016). Examining the role of anxiety sensitivity in sleep dysfunction across anxiety disorders. *Behavioral Sleep Medicine, 20*, 1-12.

Barlow, D.H., Craske, M.G., Cerny, J.A., & Klosko, J.S. (1989). Behavioral treatment of panic disorder. *Behavior Therapy, 20*, 261-282.

Bastien, C.H., Vallieres, A., & Morin, C.M. (2001). Validation of the Insomnia Severity Index as an outcome measure for insomnia research. *Sleep Medicine, 2*(4), 297-307.

Beck, J.G., Stanley, M.A., Baldwin, L.E., Deagle, E.A., & Averill, P.M. (1994). Comparison of cognitive therapy and relaxation training for panic disorder. *Journal of Consulting and Clinical Psychology, 62*(4), 818-826.

Behavioral Risk Factor Surveillance System (2010). Overweight and obesity prevalence and trends data retrieved from http://apps.nccd.cdc.gov/brfss/age.asp?cat=OB&yr=2013&qkey=8261&state=UB.

Boswell, J.F., Farchione, T.J., Sauer-Zavala, S., Murray, H.W., Fortune, M.R., & Barlow, D.H. (2013). Anxiety sensitivity and interoceptive exposure: A transdiagnostic construct and change strategy. *Behavior Therapy, 44*(3), 417-431.

Bouchard, S., Gauthier, J., Laberge, B., French, D., Pelletier, M.H., & Godbout, C. (1996). Exposure versus cognitive restructuring in the treatment of panic disorder with agoraphobia. *Behaviour Research and Therapy, 34*(3), 213-224.

Broman-Fulks, J.J., Berman, M.E., Rabian, B.A., & Webster, M.J. (2004). Effects of aerobic exercise on anxiety sensitivity. *Behaviour Research and Therapy, 42*(2), 125-136.

Broman-Fulks, J.J., Kelso, K., Zawilinski, L. (2015). Effects of a single bout of aerobic exercise versus resistance training on cognitive vulnerabilities for anxiety disorders. *Cognitive Behaviour Therapy, 44*(4), 240-251.

Broman-Fulks, J.J., & Storey, K.M. (2008). Evaluation of a brief aerobic exercise intervention for high anxiety sensitivity. *Anxiety, Stress, and Coping, 21*(2), 117-128.

Brown, M., Smits, J.A., Powers, M.B., & Telch, M.J. (2003). Differential sensitivity of the three ASI factors in predicting panic disorder patients' subjective and behavioral response to hyperventilation. *Journal of Anxiety Disorders, 17*(5), 583-591.

Burton, P., Smit, H.J., & Lightowler, H.J. (2007). The influence of restrained and external eating patterns on overeating. *Appetite, 49*(1), 191-197.

Calkins, A.W., Hearon, B.A., Capozzoli, M.C., & Otto, M.W. (2013). Psychosocial predictors of sleep dysfunction: The role of anxiety sensitivity, dysfunctional beliefs, and neuroticism. *Behavioral Sleep Medicine, 11*(2), 133-143.

Carter, M.M., Miller, O., Sbrocco, T., Suchday, S., Lewis, E.L. (1999). Factor structure of the Anxiety Sensitivity Index among African American college students. *Psychological Assessment, 11*(4), 525.

Carter, M.M., Sbrocco, T., Gore, K.L., Marin, N.W., & Lewis, E.L. (2003). Cognitive-behavioral group therapy versus a wait-list control in the treatment of African American women with panic disorder. *Cognitive Therapy and Research, 27*(5), 505-518.

Center for Behavioral Health Statistics and Quality (2015). Behavioral health trends in the United States: Results from the 2014 National Survey on Drug Use and Health. Retrieved from http://www.samhsa.gov/data/.

Chandley, R.B., Luebbe, A.M., Messman-Moore, T.L., & Ward, R.M. (2014). Anxiety sensitivity, coping motives, emotion dysregulation, and alcohol-related outcomes in college women: A moderated-mediation model. *Journal of Studies on Alcohol and Drugs, 75*(1), 83-92.

Clark, D. M., Salkovskis, P. M., Hackmann, A., Middleton, H., Pavlos, A., & Gelder, M. (1994). A comparison of cognitive therapy, applied relaxation and imipramine in the treatment of panic disorder. *British Journal of Psychiatry, 164*, 759-769.

Cohen, S., Kamarck, T., & Mermelstein (1983). A global measure of perceived stress. *Journal of Health and Social Behavior, 24*(4), 385-396.

Coren, S. (1994). The prevalence of self-reported sleep disturbances in young adults. International *Journal of Neuroscience, 79*(1-2), 67-73.

Corsino, L., Lin, P.H., Batch, B.C., Intille, S., Grambow, S.C., Bosworth, H.B., …, Volis, C.I. (2013). Recruiting young adults into a weight loss trial: Report of protocol development and recruitment results. *Contemporary Clinical Trials, 35*(2), 1-7.

Craske, M.G., & Barlow, D.H. (2007). *Mastery of your anxiety and panic: Therapist guide*. New York, NY: Oxford University Press.

Dave, P.N. (2015). The role of experiential avoidance in the association between anxiety sensitivity and emotional eating in non-eating disordered overweight/obese individuals seeking weight loss treatment. *ProQuest Dissertations and Theses*.

Deacon, B.J., Abramowitz, J.S., Woods, C.M., & Tolin, D.F. (2003). The Anxiety Sensitivity Index—Revised: Psychometric properties and factor structure in a nonclinical sample. *Behavior Research and Therapy, 41*(12), 1427-1449.

Deacon, B.J., Kemp, J.J., Dixon, L.J., Sy, J.T., Farrell, N.R., & Zhang, A.R. (2013). Maximizing the efficacy of interoceptive exposure by optimizing inhibitory learning: a randomized controlled trial. *Behavior Research and Therapy, 51*(9), 588-596.

DeBoer, L.B., Tart, C.D., Presnell, K.E., Powers, M.B., Baldwin, A., & Smits, J.A. (2012). Physical activity as a moderator of the association between anxiety sensitivity and binge eating. *Eating Behaviors, 13*, 194–201.

DiMartini, K.S., & Carey, K.B. (2011). The role of anxiety sensitivity and drinking motives in predicting alcohol use: A critical review. *Clinical Psychology Review, 31*(1), 169-177.

Dixon, L.J., Kemp, J.J., Farrell, N.R., Blakey, S.M., & Deacon, B.J. (2015). Interoceptive exposure exercises for social anxiety. *Journal of Anxiety Disorders, 33*, 25-34.

Donnelly, J.E., Blair, S.N., Jakicic, J.M., Manore, M.M., Rankin, J.W., Smith, B.K., American College of Sports Medicine. American College of Sports Medicine position stand. Appropriate physical activity intervention strategies for weight loss and prevention of weight regain for adults. *Medicine and Science in Sports and Exercise*, 41(2), 459-471.

Duffey, K.J., Gordon-Larsen, P., Jacobs, D.R., Williams, O.D., & Popkin, B.M. (2007). Differential associations of fast food and restaurant food consumption with 3-y change in body mass index: the Coronary Artery Risk Development in Young Adults Study. *American Journal of Clinical Nutrition, 85*, 201-208.

Egan, A.M., Mahmood, W.A., Fenton, R., Redziniak, N., Kyaw, T.T., Sreenan, S., & McDermott, J.H. (2013). Barriers to exercise in obese patients with type 2 diabetes. *QJM: Monthly Journal of the Association of Physicians, 106*(7), 635-638.

Ekkekakis, P., & Lind, E. (2006). Exercise does not feel the same when you are overweight: The impact of self-selected and imposed intensity on affect and exertion. *International Journal of Obesity, 30*(4), 652-660.

Friedman, S., & Paradis, C. (2002). Panic disorder in African-Americans: Symptomatology and isolated sleep paralysis. *Culture, Medicine, and Psychiatry, 26*(2), 179-198.

Fuglestad, P.T., Jeffery, R.W., & Sherwood, N.E. (2012). Lifestyle patterns associated with diet, physical activity, body mass index, and amount of recent weight loss in a sample of successful weight losers. *International Journal of Behavioral Nutrition and Physical Activity*, 9(79).

Fulton, J.J., Lavender, J.M., Tull, M.T., Klein, A.S., Muehlenkamp, J.J., & Gratz, K.L. (2012). The relationship between anxiety sensitivity and disordered eating: The mediating role of experiential avoidance. *Eating Behaviors, 13*, 166-169.

Gardenswartz, C.A., & Craske, M.G. (2001). Prevention of panic disorder. *Behavior Therapy, 32*, 725-737.

Geliebter, A., & Aversa, A. (2003). Emotional eating in overweight, normal weight, and underweight individuals. *Eating Behaviors, 3*(4), 341-347.

Gokee-LaRose, J., Gorin, A.A., Raynor, H.A, Laska, M.N., Jeffery, R.W., Levy, R.L., & Wing, R.R. (2009). Are standard behavioral weight loss programs effective for young adults? *International Journal of Obesity, 33*(12), 1374-1380.

Gokee-LaRose, J., Gorin, A.A., & Wing, R.R. (2009). Behavioral self-regulation for weight loss in young adults: A randomized controlled trial. *International Journal of Behavioral Nutrition and Physical Activity, 6*(10).

Gordon-Larsen, P., Nelson, M.C., & Popkin, B.M. (2004). Longitudinal physical activity and sedentary behavior trends: Adolescence to adulthood. *American Journal of Preventive Medicine, 27*(4), 277-283.

Gress-Smith, J.L., Roubinov, D.S., Andreotti, C., Compas, B.E., & Luecken, L.J. (2013). Prevalence, severity and risk factors for depressive symptoms and insomnia in college undergraduates. *Stress and Health, 31*(1), 63-70.

Grubbs, L., & Carter, J. (2002). The relationship of perceived benefits and barriers to reported exercise behaviors in college undergraduates. *Family & Community Health, 25*(2), 76-84.

Gunderson, E.P., & Abrams, B. (2000). Epidemiology of gestational weight gain and body weight changes after pregnancy. *Epidemiologic Reviews, 22*(2), 261-274.

Hagstromer, M., Ainsworth, B.E., Oja, P., & Sjostrom, M. (2010). Comparison of a subjective and an objective measure of physical activity in a population sample. *Journal of Physical Activity and Health, 7*, 541-550.

Hart, C.N., LaRose, J.G., Fava, J.L., James, B., & Wing, R.R. (2013). The association between time in bed and obesity risk in young adults. *Behavioral Sleep Medicine, 11*(5).

Hayden-Wade, H.A., Coleman, K.J., Sallis, J.F., Armstrong, C. (2003). Validation of the telephone and in-person interview versions of the 7-day PAR. *Medicine and Science in Sports and Exercise, 35*(5), 801-809.

Hazen, A.L., Walker, J.R., & Eldridge, G.D. (1996). Anxiety sensitivity and treatment outcome in panic disorder. *Anxiety, 2*(1), 34-39.

Hearon, B.A., Quatromoni, P.A., Mascoop, J.L., & Otto, M.W. (2014). The role of anxiety sensitivity in daily physical activity and eating behavior. *Eating Behaviors, 15*(2), 255-258.

Hearon, B.A., Utschig, A.C., Smits, J.A., Moshier, S.J., & Otto, M.W. (2013). The role of anxiety sensitivity and eating expectancy in maladaptive eating behavior. *Cognitive Therapy and Research, 37*(5), 923-933.

Hecker, J.E., Fink, C.M., Vogeltanz, N.D., Thorpe, G.L., & Sigmon, S.T. (1998). Cognitive restructuring and interoceptive exposure in the treatment of panic disorder: A crossover study. *Behavioural and Cognitive Psychotherapy, 26*, 115-131.

Herbert, B.M., Blechert, J., Hautzinger, M., Matthias, E., & Herbert, C. (2013). Intuitive eating is associated with interoceptive sensitivity. Effects on body mass index. *Appetite, 70*, 22-30.

Hicken, M.T., Lee, H., & Hing, A.K. (2018). The weight of racism: Vigilance and racial inequalities in weight-related measures. *Social Science & Medicine, 199*, 157-166.

Hirshkowitz, M., Whiton, K., Albert, S.M., Alessi, C., Bruni, O., DonCarlos, L., …Ware, J.C. (2015). National Sleep Foundation's updated sleep durations: Final report. *Sleep Health*, 1(4), 233-243.

Hoge, E.A., Marques, L., Wechsler, R.S., Lasky, A.K., Delong, H.R., Jacoby, R.J., & Simon, N.M. (2011). The role of anxiety sensitivity in sleep disturbance in panic disorder. *Journal of Anxiety Disorders, 25*, 536–538.

Hunter, L.R., Keough, M.E., Timpano, K.R., Schmidt, N.B. (2012). Ethnoracial differences in anxiety sensitivity: examining the validity of competing anxiety sensitivity index subscales. *Journal of Anxiety Disorders, 26*(4), 511-516.

Hunter, L.R. & Schmidt, N.B. (2010). Anxiety psychopathology in African American adults: Literature review and development of an empirically informed sociocultural model. *Psychological Bulletin, 136*(2), 211-235.

Jakicic, J.M., King, W.C., Marcus, M.D., Davis, K.K., Helsel, D., Rickman, A.D., …, Belle, S.H. (2015). Short-term weight loss with diet and physical activity in young adults: The IDEA Study. *Obesity, 23*(12), 2385-2397.

Kenardy, J., McCafferty, K., & Rosa, V. (2003). Internet-delivered indicated prevention for anxiety disorders: A randomised controlled trial. *Behavioural and Cognitive Psychotherapy, 31*, 279-289.

Keough, M.E, & Schmidt, N.B. (2012). Refinement of a brief anxiety sensitivity reduction intervention. *Journal of Consulting and Clinical Psychology, 80*(5), 766-772.

Kessler, R.C., Berglund, P., Demler, O., Jin, R., Merikangas, K.R., & Walters, E.E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry, 62*(6), 593-602.

Kiernan, M., Schoffman, D.E., Lee, K., Brown, S.D., Fair, J.M., Perri, M.G., & Haskell, W.L. (2013). The Stanford Leisure-Time Activity Categorical Item (L-Cat): a single categorical item sensitive to physical activity changes in overweight/obese women. *International Journal of Obesity, 37*(12), 1597-1602.

Kroenke, K., Spitzer, R.L., & Williams, J.B. (2001). The PHQ-9: validity of a brief depression severity measure. *Journal of General Internal Medicine, 16*(9), 606-613.

Kwok, O.M., Underhill, A.T., Berry, J.W., Luo, W., Elliott, T.R., & Yoon, M. (2008). Analyzing longitudinal data with multilevel models: An example with individuals living with lower extremity intra-articular fractures. *Rehabilitation Psychology, 53*(3), 370-386.

LaRose, J.G., Guthrie, K.M., Lanoye, A., Tate, D.F., Robichaud, E., Caccavale, L.J., & Wing, R.R. (in press). A mixed methods approach to improving recruitment and engagement of emerging adults in behavioural weight loss programs. *Obesity Science & Practice*.

Lawyer, S.R., Karg, R.S., Murphy, J.G., & McGlynn, F.D. (2002). Heavy drinking among college students is influenced by anxiety sensitivity, gender, and contexts for alcohol use. *Journal of Anxiety Disorders, 16*(2), 165-173.

LeBouthillier, D.M., & Asmundson, G.J. (2015). A single bout of aerobic exercise reduces anxiety sensitivity but not intolerance of uncertainty or distress tolerance: A randomized controlled trial. *Cognitive Behaviour Therapy, 44*(4), 252-263.

LeBouthillier, D.M., & Asmundson, G.J. (2017). The efficacy of aerobic exercise and resistance training as transdiagnostic interventions for anxiety-related disorders and constructs: A randomized controlled trial. *Journal of Anxiety Disorders, 52*, 43-52.

Leenders, N.Y.J.M., Sherman, W.M., Nagaraja, H.N. Comparisons of four methods of estimating physical activity in adult women. *Medicine and Science in Sports and Exercise*, *32*(7), 1320-1326.

Leone, L.A., & Ward, D.S. (2013). A mixed methods comparison of perceived benefits and barriers to exercise between obese and nonobese women. *Journal of Physical Activity & Health, 10*(4), 461-469.

Lillienfeld, S.O., Turner, S.M., & Jacob, R.G. (1996). Further comments on the nature and measurement of anxiety sensitivity: A reply to Taylor (1995b). *Journal of Anxiety Disorders, 10*(5), 411-424.

Maltby, N., Mayers, M.F., Allen, G.J., & Tolin, D.F. (2005). Anxiety sensitivity: Stability in prospective research. *Journal of Anxiety Disorders, 19*(6), 708-716.

McGuire, M.T., Wing, R.R., Klem, M.L., & Hill, J.O. (1999). Behavioral strategies of individuals who have maintained long-term weight loss. *Obesity Research, 7*(4), 334-341.

McNally, R.J. (1989). Is anxiety sensitivity distinguishable from trait anxiety? Reply to Lilienfeld, Jacob, and Turner (1989). *Journal of Abnormal Psychology, 98*(2), 193-194.

McNally, R.J., & Lorenz, M. (1987). Anxiety sensitivity in agoraphobics. *Journal of Behavior Therapy and Experimental Psychiatry, 18*(1), 3-11.

McWilliams, L.A., & Asmundson, G.J. (2001). Is there a negative association between anxiety sensitivity and arousal-increasing substances and activities? *Journal of Anxiety Disorders, 15*(3), 161-170.

Medina, J.L., DeBoer, L.B., Davis, M.L., Rosenfield, D., Powers, M.B., Otto, M.W., & Smits, J.A. (2014). Gender moderates the effect of exercise on anxiety sensitivity. *Mental Health and Physical Activity, 7*(3), 147-151.

Moshier, S.J., Hearon, B.A., Calkins, A.W., Szuhany, K.L., Utschig, A.C., Smits, J.A., & Otto, M.W. (2013). Clarifying the link between distress intolerance and exercise: Elevated anxiety sensitivity predicts less vigorous exercise. *Cognitive Therapy and Research, 37*(3), 476-482.

Moshier, S.J., Szuhany, K.L., Hearon, B.A., Smits, J.A., & Otto, M.W. (2016). Anxiety sensitivity uniquely predicts exercise behaviors in young adults seeking to increase physical activity. *Behavior Modification, 40*(1-2), 178-198.

Naragon-Gainey, K. (2010). Meta-analysis of the relations of anxiety sensitivity to the depressive and anxiety disorders. *Psychological Bulletin, 136*(1), 128-150.

National Institutes of Health. (2008, April 8). Targeted approaches to weight control for young adults. Retrieved from http://grants.nih.gov/grants/guide/rfa-files/RFA-HL-08-007.html.

Niemeier, H.M., Raynor, H.A, Lloyd-Richardson, E.E., Rogers, M.L., & Wing, R.R. (2006). Fast food consumption and breakfast skipping: Predictors of weight gain from adolescence to adulthood in a nationally representative sample. *Journal of Adolescent Health, 39*(6), 842-849.

O'Connor, R.M., Farrow, S., & Colder, C.R. (2008). Clarifying the anxiety sensitivity and alcohol use relation: Considering alcohol expectancies as moderators. *Journal of Studies on Alcohol and Drugs, 69*(5), 765-772.

Olatunji, B.O., & Wolitzky-Taylor, K.B. (2009). Anxiety sensitivity and the anxiety disorders: A meta-analytic review and synthesis. *Psychological Bulletin, 135*(6), 974-999.

Olthuis, J.V., Watt, M.C., Mackinnon, S.P., & Stewart, S.H. (2014). Telephone-delivered cognitive behavioral therapy for high anxiety sensitivity: A randomized controlled trial. *Journal of Consulting and Clinical Psychology, 82*(6), 1005-1022.

Olthuis, J.V., Watt, M.C., Mackinnon, S.P., & Stewart, S.H. (2015). CBT for high anxiety sensitivity: Alcohol outcomes. *Addictive Behaviors, 46*, 19-24.

Otto, M.W., Pollack, M.H., & Sabatino, S.A. (1996). Maintenance of remission following cognitive behavior therapy for panic disorder: Possible deleterious effects of concurrent medication treatment. *Behavior Therapy, 27*, 473-482.

Penava, S.J., Otto, M.W., Maki, K.M., & Pollack, M.H. (1998). Rate of improvement during cognitive-behavioral group treatment for panic disorder. *Behaviour Research and Therapy, 36*(7-8), 665-673.

Plehn, K., & Peterson, R.A. (2002). Anxiety sensitivity as a predictor of the development of panic symptoms, panic attack, and panic disorder: A prospective study. *Journal of Anxiety Disorders, 16*(4), 455-474.

Rapee, R.M., Brown, T.A., Antony, M.A., & Barlow, D.H. (1992). Response to hyperventilation and inhalation of 5.5% carbon dioxide enriched air across the DSM-III-R anxiety disorders. *Journal of Abnormal Psychology, 101*, 538-552.

Rector, N.A., Szacun-Shimizu, K., & Leybman, M. (2007). Anxiety sensitivity within the anxiety disorders: Disorder-specific sensitivities and depression comorbidity. *Behavior Research and Therapy, 45*(8), 1967-1975.

Reiss, S. (1991). Expectancy model of fear, anxiety, and panic. *Clinical Psychology Review, 11*(2), 141-153.

Reiss, S. (1997). Trait anxiety: It's not what you think it is. *Journal of Anxiety Disorders, 11*(2), 201-214.

Reiss, S., & McNally, R.J. (1985). Expectancy model of fear. In S. Reiss & R.R. Bootzin (Eds.), *Theoretical issues in behavior therapy*. (pp. 107-121). New York, NY: Academic Press.

Reiss, S., Peterson, R.A., Gursky, D.M., & McNally, R.J. (1986). Anxiety sensitivity, anxiety frequency, and the prediction of fearfulness. *Behavior Research and Therapy, 24*(1), 1-8.

Romano, P., van Beek, N., Cucchi, M., Biffi, S., & Perna, G. (2004). Anxiety sensitivity and modulation of the serotonergic system in patients with PD. *Journal of Anxiety Disorders, 18(*3), 423-431.

Rueger, S.Y., Trela, C.J., Palmeri, M., & King, A.C. (2012). Self-administered web-based timeline followback procedure for drinking and smoking behaviors in young adults. *Journal of Studies on Alcohol and Drugs, 73*(5), 829-833.

Sabourin, B.C., Hilchey, C.A., Lefaivre, M.J., Watt, M.C., & Stewart, S.H. (2011). Why do they exercise less? Barriers to exercise in high-anxiety-sensitive women. *Cognitive Behaviour Therapy, 40*, 206–215.

Sabourin, B.C., Stewart, S.H., Sherry, S.B., Watt, M.C., Wald, J., & Grant, V.V. (2008). Physical exercise as interoceptive exposure within a brief cognitive-behavioral treatment for anxiety-sensitive women. *Journal of Cognitive Psychotherapy, 22*, 303–320.

Sabourin, B.C., Stewart, S.H., Watt, M.C., & Krigolson, O.E. (2015). Running as interoceptive exposure for decreasing anxiety sensitivity: Replication and extension. *Cognitive Behaviour Therapy, 44*(4), 264-274.

Sallis, J.F., Haskell, W.L., Wood, P.D., Fortmann, S.P., Rogers, T., Blair, S.N., & Paffenbarger, R.S. (1985). Physical activity assessment methodology in the Five-City Project. *American Journal of Epidemiology, 121*(1), 91-106.

Schmidt, N.B., Capron, D.W., Raines, A.M., & Allan, N.P. (2014). Randomized clinical trial evaluating the efficacy of a brief intervention targeting anxiety sensitivity cognitive concerns. *Journal of Consulting and Clinical Psychology, 82*(6), 1023-1033.

Schmidt, N.B., Lerew, D.R., & Jackson, R.J. (1997). The role of anxiety sensitivity in the pathogenesis of panic: Prospective evaluation of spontaneous panic attacks during acute stress. *Journal of Abnormal Psychology, 106*(3), 355-364.

Schmidt, N.B., Lerew, D.R., & Jackson, R.J. (1999). Prospective evaluation of anxiety sensitivity in the pathogenesis of panic: Replication and extension. *Journal of Abnormal Psychology, 108*(3), 532-537.

Schmidt, N.B., Zvolensky, M.J., & Maner, J.K. (2006). Anxiety sensitivity: Prospective prediction of panic attacks and Axis I pathology. *Journal of Psychiatric Research, 40*(8), 691-699.

Shear, M.K., Pilkonis, P.A., Cloitre, M., & Leon, A.C. (1994). Cognitive behavioral treatment compared with nonprescriptive treatment of panic disorder. *Archives of General Psychiatry, 51*, 395-401.

Shin, J.H. (2009). Application of repeated-measures analysis of variance and hierarchical linear model in nursing research. *Nursing Research, 58*(3), 211-217.

Short, N.A., Allan, N.P., Raines, A.M., & Schmidt, N.B. (2015). The effects of an anxiety sensitivity intervention on insomnia symptoms. *Sleep Medicine, 16*(1), 152-159.

Simon, N.M., Otto, M.W., Smits, J.A., Nicolaou, D.C., Reese, H.E., & Pollack, M.H. (2004). Changes in anxiety sensitivity with pharmacotherapy for panic disorder. *Journal of Psychiatric Research, 38*(5), 491-495.

Sloane, R., Snyder, D.., Demark-Wahnefried, W., Lobach, D., & Kraus, W.E. (2009). Comparing the 7-Day PAR with a Triaxial Accelerometer for Measuring Time in Exercise. *Medicine and Science in Sports and Exercise, 41*(6), 1334-1340.

Smits, J.A., Berry, A.C., Rosenfield, D., Powers, M.B., Behar, E., & Otto, M.W. (2008). Reducing anxiety sensitivity with exercise. *Depression and Anxiety, 25*(8), 689-699.

Smits, J.A., Berry, A.C., Tart, C.D., & Powers, M.B. (2008). The efficacy of cognitive-behavioral interventions for reducing anxiety sensitivity: A meta-analytic review. *Behaviour Research and Therapy, 46*(9), 1047-1054.

Smits, J.A., Powers, M.B., Berry, A.C., & Otto, M.W. (2007). Translating empirically supported strategies into accessible interventions: The potential utility of exercise for the treatment of panic disorder. *Cognitive and Behavioral Practice, 14*, 364-374.

Smits, J.A., Powers, M.B., Cho, Y., & Telch, M.J. (2004). Mechanism of change in cognitive-behavioral treatment of panic disorder: evidence for the fear of fear mediational hypothesis. *Journal of Consulting and Clinical Psychology, 72*(4), 646-652.

Smits, J.A., Tart, C.D., Presnell, K., Rosenfield, D., & Otto, M.W. (2010). Identifying potential barriers to physical activity adherence: Anxiety sensitivity and body mass as predictors of fear during exercise. *Cognitive Behaviour Therapy, 39*(1), 28-36.

Sobell, L.C. & Sobell, M.B. (1992). Timeline Follow-back: A technique for assessing self-reported ethanol consumption. In J. Allen & R. Z. Litten (Eds.), *Measuring Alcohol Consumption: Psychosocial and Biological Methods* (pp. 41-72). Totowa, NJ: Humana Press.

Sobell, L.C., Sobell, M.B., Klajner, F., Pavan, D., & Basian, E. (1986). The reliability of a timeline method for assessing normal drinker college students' recent drinking history: Utility for alcohol research. *Addictive Behaviors, 11*(2), 149-161.

Spiegel, K., Tasali, E., Penev, P., & van Cauter, E. (2004). Brief communication: Sleep curtailment in healthy young men is associated with decreased leptin levels, elevated ghrelin levels, and increased hunger and appetite. *Annals of Internal Medicine, 141*(11), 846-850.

Spitzer, R.L., Kroenke, K., Williams, J.B., & Lowe, B. (2006). A brief measure for assessing generalized anxiety disorder: The GAD-7. *Archives of Internal Medicine, 166*(10), 1092-1097.

Stein, M.B., Jang, K.L., & Livesley, W.J. (1999). Heritability of anxiety sensitivity: A twin study. *The American Journal of Psychiatry, 156*(2), 246-251.

Stewart, S.H., Peterson, J.B., & Pihl, R.O. (1995). Anxiety sensitivity and self-reported alcohol consumption rates in university women. *Journal of Anxiety Disorders, 9*, 283-292.

Stewart, S.H., & Watt, M.C. (2008). Introduction to the special issue on interoceptive exposure in the treatment of anxiety and related disorders: Novel applications and mechanisms of action. *Journal of Cognitive Psychotherapy: An International Quarterly, 22*(4), 291-302.

Stewart, S.H., Zvolensky, M.J., & Eifert, G.H. (2001). Negative-reinforcement drinking motives mediate the relation between anxiety sensitivity and increased drinking behavior. *Personality and Individual Differences, 31*, 157–171.

Stewart, S.H., & Zeitlin, S.B. (1995). Anxiety sensitivity and alcohol use motives. *Journal of Anxiety Disorders, 9*, 229-240.

Stice, E., Telch, C.F., & Rizvi, S.L. (2000). Development and validation of the Eating Disorder Diagnostic Scale: A brief self-report measure of anorexia, bulimia, and binge-eating disorder. *Psychological Assessment, 12*(2), 123-131.

Taylor, S., & Cox, B.J. (1998). An expanded Anxiety Sensitivity Index: Evidence for a hierarchic structure in a clinical sample. *Journal of Anxiety Disorders, 12*(5), 463-483.

Taylor, S., Zvolensky, M.J., Cox, B.J., Deacon, B., Heimberg, R.G., Ledley, D.R., …, Cardenas, S.J. (2007). Robust dimensions of anxiety sensitivity: Development and initial validation of the Anxiety Sensitivity Index—3. *Psychological Assessment, 19*(2), 176-188.

Telch, M.J., Lucas, J.A., Schmidt, N.B., Hanna, H.H., Jaimez, T.L., & Lucas, R.A. (1993). Group cognitive-behavioral treatment of panic disorder. *Behavior Research and Therapy, 31*, 279-287.

Telch, M.J., Silverman, A., & Schmidt, N.B. (1996). Effects of anxiety sensitivity and perceived control on emotional responding to caffeine challenge. *Journal of Anxiety Disorders, 10*(1), 21-35.

The, N.S., & Gordon-Larsen, P. (2009). Entry into Romantic Partnership is Associated with Obesity. *Obesity (Silver Spring), 17*(7), 1441-1447.

van Balkom, A.J., de Beurs, E., Koele, P., Lange, A., & can Dyck, R. (1996). Long-term benzodiazepine use is associated with smaller treatment gain in panic disorder with agoraphobia. *The Journal of Nervous and Mental Disease, 184*(2), 133-134.

Vincent, N., & Walker, J. (2001). Anxiety sensitivity: Predictor of sleep-related impairment and medication use in chronic insomnia. *Depression and Anxiety, 14*(4), 238-243.

Warburton, D.E., Gledhill, N., Jamnik, V.K., Bredin, S.S., McKenzie, D.C., Stone, J., …Shephard, R.J. (2011). Evidence-based risk assessment and recommendations for physical activity clearance: Consensus Document 2011. *Applied Physiology, Nutrition, and Metabolism, 36*(Suppl 1), S266-S298.

Watt, M.C., Stewart, S.H., Birch, C., & Bernier, D. (2006). Brief CBT for high anxiety sensitivity decreases drinking problems, relief alcohol outcome expectancies, and conformity

drinking motives: Evidence from a randomized controlled trial. *Journal of Mental Health, 15*(6), 683-695.

Watt, M.C., Stewart, S.H., Lefaivre, M.J., & Uman, L.S. (2006). A brief cognitive-behavioral approach to reducing anxiety sensitivity decreases pain-related anxiety. *Cognitive Behaviour Therapy, 35*(4), 248-256.

Weems, C.F., Hayward, C., Killen, J., & Taylor, C.B. (2002). A longitudinal investigation of anxiety sensitivity in adolescence. *Journal of Abnormal Psychology, 111*(3), 471-477.

Weiner, C.L., Meredith-Elkins, R., Pincus, D., & Comer, J. (2015). Anxiety sensitivity and sleep-related problems in anxious youth. *Journal of Anxiety Disorders, 32*, 66-72.

Wheaton, M.G., Deacon, B.J., McGrath, P.B., Berman, N.C., & Abramowitz, J.S. (2012). Dimensions of anxiety sensitivity in the anxiety disorders: Evaluation of the ASI-3. *Journal of Anxiety Disorders, 26*(3), 401-408.

Wipfli, B.M., Rethorst, C.D., & Landers, D.M. (2008). The anxiolytic effects of exercise: A meta-analysis of randomized trials and dose-response analysis. *Journal of Sport & Exercise Physiology, 30*(4), 392-410.

Zack, M., Poulos, C.X., Aramakis, V.B., Khamba, B.K., & MacLeod, C.M. (2007). Effects of drink-stress sequence and gender on alcohol stress response dampening in high and low anxiety sensitive drinkers. *Alcohol, Clinical, and Experimental Research, 31*(3), 411-422.

Zinbarg, R.E., Barlow, D.H., & Brown, T.A. (1997). Hierarchical structure and general factor saturation of the Anxiety Sensitivity Index: Evidence and implications. *Psychological Assessment, 9*(3), 277-284.

Zinbarg, R.E., Mohlman, J., & Hong, N.N. (1999). Dimensions of anxiety sensitivity. In S. Taylor (Ed.), Anxiety sensitivity: *Theory, research, and treatment of the fear of anxiety* (pp. 83-113). Mahwah, NJ: Erlbaum.

Appendix. Measures

Demographics

1. Current age: _____
2. Sex
   ☐ Male ☐ Female
3. Highest level of education completed
   ☐ Grade School (6 years or less)
   ☐ Junior High School (7-9 years)
   ☐ High School (10-12 years)
   ☐ Vocational Training (Beyond high school)
   ☐ Some College (Less than 4 years)
   ☐ College/University Degree
   ☐ Graduate or Professional Education
4. Are you currently in school?
   ☐ Yes, attending part-time ☐ Yes, attending full-time ☐ No
5. Race (check all that apply)
   ☐ American Indian/Alaskan Native
   ☐ Asian
   ☐ Black/African American
   ☐ White/Caucasian
   ☐ Native Hawaiian/Pacific Islander
6. Ethnicity
   ☐ Hispanic/Latino ☐ Not Hispanic/Latino
7. Weight: _____ pounds
8. Height: _____ feet, _____ inches
9. Are you currently pregnant?
   ☐ Yes ☐ No
10. Have you ever been diagnosed with depression or anxiety?
    ☐ Yes ☐ No
11. Have you ever taken medication for depression or anxiety?
    ☐ Yes ☐ No
    IF YES: Are you currently taking medication for depression or anxiety?
    ☐ Yes ☐ No
    IF YES: _____ [Name of medication and dosage, if known]
12. Have you ever received therapy or counseling for depression or anxiety?
    ☐ Yes ☐ No

IF YES: Are you currently in therapy or counseling for depression or anxiety?

☐ Yes ☐ No

13. Have you been hospitalized for depression or another psychiatric disorder within the past year?

☐ Yes ☐ No

Physical Activity Readiness Questionnaire (PAR-Q)

1. Has your doctor ever said that you have a heart condition and that you should only perform physical activity recommended by a doctor?
   ☐ Yes ☐ No

2. Do you feel pain in your chest when you perform physical activity?
   ☐ Yes ☐ No

3. In the past month, have you had pain in your chest when you were not performing any physical activity?
   ☐ Yes ☐ No

4. Do you lose your balance because of dizziness or do you ever lose consciousness?
   ☐ Yes ☐ No

5. Do you have a bone or joint problem that could be made worse by a change in your physical activity?
   ☐ Yes ☐ No

6. Is your doctor currently prescribing any medication for your blood pressure or for a heart condition?
   ☐ Yes ☐ No

7. Do you know of any other reason why you should not engage in physical activity?
   ☐ Yes ☐ No

Stanford Leisure-Time Activity Categorical Item (L-CAT)

During the past month, which statement best describes the kinds of physical activity you usually did? Do not include the time you spent working at a job. Please read all six statements before selecting one.

1. I did not do much physical activity. I mostly did things like watching television, reading, playing cards, or playing computer games. Only occasionally, no more than once or twice a month, did I do anything more active such as going for a walk or playing tennis.

2. Once or twice a week, I did light activities such as getting outdoors on the weekends for an easy walk or stroll. Or once or twice a week, I did chores around the house such as sweeping floors or vacuuming.

3. About three times a week, I did moderate activities such as brisk walking, swimming, or riding a bike for about 15–20 minutes each time. Or about once a week, I did moderately difficult chores such as raking or mowing the lawn for about 45–60 minutes. Or about once a week, I played sports such as softball, basketball, or soccer for about 45–60 minutes.

4. Almost daily, that is five or more times a week, I did moderate activities such as brisk walking, swimming, or riding a bike for 30 minutes or more each time. Or about once a week, I did moderately difficult chores or played sports for 2 hours or more.

5. About three times a week, I did vigorous activities such as running or riding hard on a bike for 30 minutes or more each time.

6. Almost daily, that is five or more times a week, I did vigorous activities such as running or riding hard on a bike for 30 minutes or more each time.

Anxiety Sensitivity Index—3 (ASI-3)

How much do you agree with each of the following statements?

	Very little	A little	Some	Much	Very much
1. It is important for me not to appear nervous	0	1	2	3	4
2. When I cannot keep my mind on a task, I worry I might be going crazy	0	1	2	3	4
3. It scares me when my heart beats rapidly	0	1	2	3	4
4. When my stomach is upset, I worry that I might be seriously ill	0	1	2	3	4
5. It scares me when I am unable to keep my mind on a task	0	1	2	3	4
6. When I tremble in the presence of others, I fear what people might think of me	0	1	2	3	4
7. When my chest feels tight, I get scared that I won't be able to breathe properly	0	1	2	3	4
8. When I feel pain in my chest, I worry that I'm going to have a heart attack	0	1	2	3	4
9. I worry that other people will notice my anxiety	0	1	2	3	4
10. When I feel "spacey" or spaced out, I worry that I may be mentally ill	0	1	2	3	4
11. It scares me when I blush in front of other people	0	1	2	3	4
12. When I notice my heart skipping a beat, I worry that there is something seriously wrong with me	0	1	2	3	4
13. When I begin to sweat in a social situation, I fear people will think negatively of me	0	1	2	3	4
14. When my thoughts seem to speed up, I worry that I might be going crazy	0	1	2	3	4
15. When my throat feels tight, I worry that I could choke to death	0	1	2	3	4
16. When I have trouble thinking clearly, I worry that there is something wrong with me	0	1	2	3	4
17. I think it would be horrible for me to faint in public	0	1	2	3	4
18. When my mind goes blank, I worry there is something terribly wrong with me	0	1	2	3	4

7-Day Physical Activity Recall (PAR)

1. Were you employed in the past 7 days? 0. No (Skip to Q4) 1. Yes
2. How many days of the last 7 did you work? ___ days
3. How many total hours did you work in the last 7 days? ___ hours last week
4. What two days do you consider your weekend days? _____
(mark days below with a squiggle)

	SLEEP	1___	2___	3___	4___	5___	6___	7___
MORNING	Moderate							
	Hard							
	Very Hard							
AFTERNOON	Moderate							
	Hard							
	Very Hard							
EVENING	Moderate							
	Hard							
	Very Hard							
Total Min/ Day	Strength:							
	Flexibility:							

4a. Compared to your physical activity over the past three months, was last week's physical activity more, less, or about the same? 1. More 2. Less 3. About the same

INTERVIEWER QUESTIONS:

5. Were there any problems with the PAR interview? 0. No 1. Yes (explain on back)
6. Do you think this was a valid PAR interview? 0. No 1. Yes
7. Please list any activities reported by the subject which you don't know how to classify:
_____
8. Please provide any other comments in the space below:

Intention to Increase Physical Activity

To what extent do you intend to increase your physical activity over the next 2 weeks?

Definitely not	Probably not	Possibly	Very likely	Definitely
0	1	2	3	4

Insomnia Severity Index (ISI)

Please the severity of the following sleep-related problems over the **past 2 weeks**.

	None	Mild	Moderate	Severe	Very Severe
1. Difficulty falling asleep	0	1	2	3	4
2. Difficulty staying asleep	0	1	2	3	4
3. Problems waking up too early	0	1	2	3	4

4. How satisfied are you with your current sleep pattern?

Very satisfied	Satisfied	Moderately satisfied	Dissatisfied	Very dissatisfied
0	1	2	3	4

5. How noticeable to others do you think sleep problems are in terms of impairing the quality of your life?

Not at all noticeable	A little	Somewhat	Much	Very much noticeable
0	1	2	3	4

6. How worried/distressed are you about current sleep problems?

Not at all worried	A little	Somewhat	Much	Very much worried
0	1	2	3	4

7. To what extent do you consider sleep problems to interfere with your daily functioning (e.g., daytime fatigue, mood, ability to function at work/daily chores, concentration, memory, mood, etc.)?

Not at all interfering	A little	Somewhat	Much	Very much interfering
0	1	2	3	4

Alcohol Timeline Follow-back (Alcohol TLFB)

	1___	2___	3___	4___	5___	6___	7___
Number of drinks							
Number of hours drinking							

Patient Health Questionnaire—Depression Module (PHQ-8)

Over the past 2 weeks, how often have you been bothered by the following problems?	Not at all	Several days	More than half the days	Nearly every day
1. Little interest or pleasure in doing things	0	1	2	3
2. Feeling down, depressed, or hopeless	0	1	2	3
3. Trouble falling asleep, staying asleep, or sleeping too much	0	1	2	3
4. Feeling tired or having little energy	0	1	2	3
5. Poor appetite or overeating	0	1	2	3
6. Feeling bad about yourself—that you're a failure or have let yourself or your family down	0	1	2	3
7. Trouble concentrating on things, such as reading the newspaper or watching television	0	1	2	3
8. Moving or speaking so slowly that other people could have noticed. Or, the opposite—being so fidgety or restless that you have been moving around a lot more than usual	0	1	2	3

Generalized Anxiety Disorder 7-item Scale (GAD-7)

Over the past 2 weeks, how often have you been bothered by the following problems?	Not at all	Several days	More than half the days	Nearly every day
1. Feeling nervous, anxious, or on edge	0	1	2	3
2. Not being able to stop or control worrying	0	1	2	3
3. Worrying too much about different things	0	1	2	3
4. Trouble relaxing	0	1	2	3
5. Being so restless it's hard to sit still	0	1	2	3
6. Becoming easily annoyed or irritable	0	1	2	3
7. Feeling afraid as if something awful might happen	0	1	2	3

4-item Perceived Stress Scale (PSS-4)

The questions in this scale ask you about your feelings and thoughts during the **past 2 weeks**. In each case, please indicate with a check how often you felt or thought a certain way.

	Never	Almost Never	Sometimes	Fairly Often	Very Often
1. In the past 2 weeks, how often have you felt that you were unable to control the important things in your life?	0	1	2	3	4
2. In the past 2 weeks, how often have you felt confident about your ability to handle your personal problems?	0	1	2	3	4
3. In the past 2 weeks, how often have you felt that things were going your way?	0	1	2	3	4
4. In the past 2 weeks, how often have you felt difficulties were piling up so high that you could not overcome them?	0	1	2	3	4

Vita

Autumn Lanoye was born on October 28, 1987 in Jacksonville, NC and grew up in Williamsville, NY. She graduated magna cum laude from the University of Rochester in 2005, earning a Bachelor of Arts with honors and distinction in Psychology. She worked for 2 years as a research assistant at Syracuse University's Center for Health and Behavior prior to acceptance into the Clinical Psychology doctoral program at Virginia Commonwealth University. Autumn received her Master of Science degree in Clinical Psychology in August 2014, and completed her clinical internship at West Virginia University Health Sciences Center in Charleston, WV in June 2018. She will graduate with her doctorate degree in August 2018.