



Gender moderates the effect of exercise on anxiety sensitivity



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ABSTRACT

A moderate to vigorous intensity exercise program is emerging as a promising strategy for reducing anxiety sensitivity (AS). Initial evidence suggests that the effects of exercise on mental health outcomes may vary as a function of gender, with men benefitting more than women. Building upon this evidence, the present study tested the hypothesis that the effect of exercise on AS would vary as a function of gender, such that the effect would be stronger for men than for women. We tested this hypothesis using the data from a published study (Smits, Berry, Rosenfield, et al., 2008). In this study, participants ($N = 60$) with elevated levels of AS were randomly assigned to a two-week exercise intervention [EX] or a waitlist control condition [WL]. Results revealed that males showed significantly greater initial AS reductions relative to females (following 1 week of exercise). However, these gender differences were no longer evident at the end of the intervention. Possible mechanisms for the observed findings and directions for future research are discussed.

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Accruing empirical evidence supports exercise as a therapeutic strategy for improving mood and anxiety disorders (Asmundson et al., 2013; DeBoer, Powers, Utschig, Otto, & Smits, 2012), especially for mild to moderate major depressive disorder (MDD; Dunn, Trivedi, Kampert, Clark, & Chambliss, 2005; Singh et al., 2005; Veale et al., 1992). A meta-analysis aggregating the results of randomized, controlled trials for MDD revealed a large effect ($d = 1.42$; Stathopoulou, Powers, Berry, Smits, & Otto, 2006) supporting the overall effectiveness of exercise interventions for improving clinical depression.

Though less extensively studied, exercise has also shown initial efficacy for treating anxiety disorders (O'Connor, Raglin, & Martinsen, 2000; Petruzello et al., 1991; Stathopoulou et al., 2006; Wipfli, Rethorst, & Landers, 2008). A recent quantitative review evaluated the effectiveness of exercise for anxiety in both clinical and non-clinical adult samples. The 49 RCTs included exercise interventions at a moderate-to-vigorous intensity ranging

from an acute bout to five times per week for a duration of 30–90 min per session. Results showed exercisers to fare significantly better than those in no-treatment control groups (*Hedge's* $g = -.48$), and either comparable to or better than those in other active treatments commonly used to treat anxiety (*Hedge's* $g = -.19$), such as cognitive behavioral therapy (CBT), relaxation therapies (e.g., meditation, light exercise, yoga), group psychotherapy, and stress management education. Moreover, their results indicated that exercise can yield outcomes comparable to medication (Wipfli et al., 2008).

Similarly, because exercise can induce somatic arousal in a repeated, systematic, and prolonged fashion, it may effectively serve as fear extinction training. Indeed, within standard evidence-based cognitive-behavioral interventions for panic disorder (PD), this type of interoceptive exposure appears to be critical for symptom improvement (Smits, Powers, Cho, & Telch, 2004), likely by way of reducing anxiety sensitivity (AS; fear of anxiety sensations and their consequences; McNally, 2002; Smits, Berry, Tart, & Powers, 2008; Smits, Julian, Rosenfield, & Powers, 2012; Smits, Powers, Berry, & Otto, 2007). Given the identified role of AS as a cognitive-affective risk and maintenance factor for anxiety disorders (e.g., panic disorder; McNally, 2002; Olatunji & Wolitzky-Taylor, 2009) as well as for related problems characterized by

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maladaptive coping behaviors and poor emotion regulation (e.g., substance use, binge eating, PTSD; Taylor, 1999), a moderate-to vigorous-intensity exercise program may prove beneficial, not only for those with a PD diagnosis, but also for reducing AS in at-risk populations more broadly.

Our group extended previous work by Broman-Fulks, Berman, Rabian, and Webster (2004) by demonstrating that a brief (2-week) moderate-intensity aerobic exercise intervention in adults with elevated scores (≥ 25 indicating possible clinical problems; Peterson & Plehn, 1999, p. 70) on the Anxiety Sensitivity Index (ASI; Reiss & McNally, 1985) led to clinically significant changes in AS from pretreatment through 3-week follow-up (Smits, Berry, Rosenfield, Powers, Behar, & Otto, 2008). Indeed, clinically significant change (requiring reduction of scores \geq two standard deviations below the baseline sample mean) was observed in 88% of exercising participants (Smits, Berry, Rosenfield, et al., 2008), with mean ASI decreasing from 33 at pretreatment to 14.5 at post-treatment and to 11.5 by 3-week follow-up. Beyond building additional support for a brief exercise regimen for reducing AS, we showed that these clinically meaningful reductions in AS resulting from exercise mediated subsequent improvements in self-reported depressive and anxiety symptoms (see Smits, Berry, Rosenfield, et al., 2008). This trial was the first to directly evaluate altered-AS as a causal mechanism of the anxiolytic and antidepressant effects of exercise. Identifying moderators of exercise's efficacy for reducing AS may further elucidate the nature of the AS-exercise relation, and thus, the potential utility of exercise as treatment for anxiety disorders and related aforementioned problems associated with elevated AS.

Prior evidence from outside the AS literature suggest that the effects of exercise on mental health may vary as a function of gender, with men benefitting more than women (Bhui & Fletcher, 2000; Elliot, Kennedy, Morgan, Anderson, & Morris, 2012; Hunt-Shanks, Blanchard, & Reid, 2009). For example, a recent investigation revealed a stronger inverse relation for males more than for females between physical activity frequency and frequency of depressive symptoms and consideration of suicide (Elliot et al., 2012). In addition, the meta-analysis by Wipfli et al. (2008) revealed significant effect sizes in mixed-gender and male-only samples, but not in female-only samples, suggesting that there may be meaningful gender differences in the efficacy of exercise interventions for reducing anxiety symptoms. Such differences could have important implications for clinical practice (e.g., matching strategies, need for additional strategies) and help guide research on the understanding of the mechanisms of action of exercise for mental health conditions.

Building upon the aforementioned work, the present study tested the hypothesis that the effect of exercise on AS would vary as a function of gender, such that the effect would be stronger for men than for women. We tested this hypothesis using data from our previously published study (Smits, Berry, Rosenfield, et al., 2008). In this study, participants with elevated levels of anxiety sensitivity ($N = 60$) were randomly assigned to a 2-week exercise intervention [EX] or a waitlist control condition [WL]. Smits, Berry, Rosenfield, et al. (2008) found no significant difference between the two slightly different exercise conditions in reductions of AS (with both showing greater decreases in ASI scores from pre-treatment to follow-up compared to WL), we collapsed across exercise conditions to form a single EX group for the present study. Using this data set, we investigated whether there were gender differences in the effects of exercise on AS in response to a brief, 2-week programmed exercise. More specifically, we predicted a significant 3-way interaction, such that the change in ASI over time in EX relative to WL would be greater for males than for females.

1. Method

1.1. Participants

Study participants ($N = 60$; 45 Female; $M_{Age} = 20.68$, $SD = 5.80$) consisted of (1) undergraduate students at a medium-sized private university in the Southwest of the United States of America ($N = 50$; 38 Female; $M_{Age} = 20.40$, $SD = 5.03$) and (2) community individuals in the Boston area ($N = 10$; 7 Female; $M_{Age} = 22.10$, $SD = 8.95$). Undergraduate participants were recruited from classroom survey screenings in introductory psychology courses. In the Southwest sample, 182 of the 286 potentially eligible participants (i.e., with surveys indicating clinically elevated $AS \geq 25$) indicated interest in participating and were contacted by study staff to complete a phone interview. Of the 91 deemed eligible, 50 participants enrolled, and were given course credit for participation. Participants from the Boston area site were recruited from community advertisements near a large public university. Of the 91 surveyed at the Boston site, 30 completed a phone interview, and 10 interested participants were enrolled (9 students and 1 person from larger community area; see Smits, Berry, Rosenfield, et al., 2008).

All participants met the following entry criteria: elevated anxiety sensitivity (indexed by ASI score ≥ 25 ; Peterson & Reiss, 1992); absence of physical conditions that could be exacerbated by exercise; body mass index < 35 ; no current involvement in an exercise program (i.e., \leq one aerobic exercise session per week); no recent change in psychotropic medications (e.g., stabilized for at least four weeks for antidepressants and two weeks for benzodiazepines); and no current psychotherapy. The study sample consisted primarily of students (98%) who were Caucasian (71%). Only 1 participant reported taking current psychotropic medication (prescribed Adderall for ADHD symptoms).

1.2. Measures

1.2.1. Demographics

Following study enrollment and one week prior to the intervention (i.e., *pre-treatment*), self-reported demographic information was collected from each participant.

1.2.2. Anxiety sensitivity

The Anxiety Sensitivity Index (ASI) is a 16-item validated measure of anxiety sensitivity, defined as the fear of anxiety-related sensations and their consequences (Peterson & Reiss, 1992). On the ASI, respondents indicate on a 5-point Likert-type scale (0 = *very little* to 4 = *very much*) the degree to which anxiety symptoms are distressing (e.g., "It scares me when my heart beats rapidly") and the concern about negative consequences of anxiety symptoms (e.g., "When I notice that my heart is beating rapidly, I worry that I might have a heart attack"). Normative studies indicate that scores above 25 indicate possible clinical problems (Peterson & Plehn, 1999). The ASI has sound psychometric properties in both clinical and non-clinical samples, including adequate construct validity (McNally & Lorenz, 1987; Reiss, Peterson, Gursky, & McNally, 1986; Taylor, Koch, & McNally, 1992; Telch, Shermis, & Lucas, 1989). The ASI was administered to all participants at four separate time points: pre-treatment, mid-treatment (1 week after exercise initiation), post-treatment (end of 2-week exercise program), and 3-week follow-up (i.e., three weeks following completion of exercise intervention).

1.3. Intervention procedures

1.3.1. Exercise [EX]

The exercise dose for this intervention was guided by previous meta-analyses, which documented that moderate-to-high-

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