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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.

Keywords

Exercise; Treatment outcome; Treatment moderators; Anxiety sensitivity; Gender differences; Anxiety

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.

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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 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 > 2 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 ≥ 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., 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-intensity exercise (e.g., 60–80% of maximum age-predicted heart rate) is superior to low-intensity exercise (Wipfli et al., 2008). Accordingly, the intervention consisted of thrice-weekly 20-min moderate intensity aerobic exercise sessions over two weeks (six sessions total), which were designed to elicit sensations of physiological arousal as a type of interoceptive exposure targeting their anxiety-related fears. Along with direct verbal instruction from the experimenter for the participant to attend to the sensations experienced, participants were asked by the protocol therapist to rate their level of subjective distress (assessed on a scale of “0 = not intense at all” to “100 = extremely intense”) every 3 min during exercise. At the first session, participants were provided with the rationale behind exercise for anxiety (Smits, Berry, Rosenfield, et al., 2008). Exercise was completed and supervised by CPR-certified study personnel at the laboratory in a room furnished with treadmills and appropriate safety equipment (e.g., defibrillator); the exercise room featured a computer-controlled exercise training management system (Smooth® 7.1 HR Pro, InternetFitness.com, Inc., Mt. Laurel, NJ). The system allows for input of relevant data for each participant (resting and maximal heart rate, target heart rate, and session duration). The computer receives a heart rate (HR)

signal from a Polar® transmitter (chest strap), allowing the experimenter to adjust speed and incline of the treadmill to target specific levels of effort as assessed by HR. Exercise intensity was pre-set at the high-moderate level, which was determined by the standard calculation (70% of HR_{max} [(220 – age)*.70]). Supervising staff were responsible for checking the computerized HR feedback system every 5 min persession in order to verify participants were exercising within their target HR zone. Additional elements included the review of session procedures and accomplishments at the end of each session.

1.3.2. Waitlist [WL]—Participants assigned to the WL condition completed assessments at the same time intervals as the two active conditions, but did not participate in exercise.

1.4. Data analysis strategy

We performed a repeated-measures ANOVA using mixed-effects models (MRMs) in SPSS 21.0. The MRM approach to repeated measures ANOVA is advantageous because it allows inclusion of all participants regardless of missing data (which improves power and generalizability), can model the covariance matrix of the repeated measures more flexibly than ANCOVA, and is the recommended method for longitudinal data analysis (Hamer & Simpson, 2009). The independent variables in our $2 \times 2 \times 4$ mixed-effects ANOVA were Gender, Treatment Condition (EX vs. WL), and Time (pre-treatment, mid-treatment, post-treatment, and the three week follow-up). We treated Time as a categorical variable in an ANOVA model rather than as a continuous variable in a growth curve model because it allows the change over time to be free of constraints that would be required in a functional growth curve model. This approach is recommended when one performs multiple between-group comparisons over a small number of assessment points (Blackwell, Mendes De Leon, & Miller, 2006).

Pattern mixture modeling (Hedeker & Gibbons, 2006) was used to test the effects of missing data on the model. In pattern mixture modeling one investigates whether missing data patterns moderate the results. Accordingly, we created a variable coding each participant's dropout status (i.e., identifying each participant as either a dropout [coded 1] or completer [coded 0]). This variable was then entered as a moderator of the effects in our model (Hedeker & Gibbons, 2006). Analyses included all randomized individuals ($N = 60$; 45 Female).

2. Results

2.1. Baseline equivalence and attrition

As can be seen in Table 1, the study conditions (EX [$n = 40$], WL [$n = 20$]) did not differ on any demographic or clinical (i.e., ASI) measures at pre-treatment, suggesting that random assignment produced groups with comparable demographic and clinical characteristics (Smits, Berry, Rosenfield, et al., 2008). Further, a one-way ANOVA revealed that pre-treatment ASI scores did not vary as a function of Gender ($F(1,58) = .18, p = .67$). One-way ANOVAs also indicated neither mean participant age ($F(1,58) = .71, p = .40$) or mean pre-treatment ASI ($F(1,58) = .66, p = .42$) differed significantly between the two study sites. Our study had a dropout rate of 21.7% ($n = 13$; 10 in EX and 3 in WL), although all participants

were included in the mixed effects analysis. Chi-square analyses revealed that there was not a significant difference between study condition ($\chi^2(1) = .78, p = .51$), gender ($\chi^2(1) = .33, p = .56$), or study site ($\chi^2(1) = .29, p = .59$) in dropout rate. Lastly, a one-way ANOVA revealed that there was no difference between dropouts and completers, $F(1,58) = .92, p = .34$, on pre-treatment ASI scores.

2.2. Hypothesis testing

Pattern mixture modeling was used to test the effects of dropout status on outcome (ASI). Results showed the dropout variables did not affect outcome (i.e., $p > .20$ for all dropout main effects and interactions). Thus, dropout variables and their interactions were removed from the final model. In our final model, we observed a significant main effect for Time, $F(3,87) = 50.21, p = .000$, and Treatment Condition, $F(1,55) = 46.19, p = .000$, indicating a general improvement over time and lower scores in EX vs. WL, respectively. These main effects were qualified by a significant Time $\times$ Treatment Condition interaction, $F(3,87) = 20.67, p = .000$, suggesting greater improvement in AS for those in EX compared to WL. Additionally, we found a significant Gender $\times$ Treatment Condition interaction, $F(1,55) = 4.53, p = .04$, such that, collapsed across time points, in EX compared to WL, men evidenced lower mean ASI than women.

Finally, consistent with our hypothesis, we observed a significant Time $\times$ Treatment Condition $\times$ Gender interaction, $F(3,87) = 3.37, p = .02$, such that the relative improvement over Time in EX compared to WL was different for males than for females (see Fig. 1). To further understand this three-way interaction, we decomposed the Time variable, which consisted of 4 levels (hence 3 degrees of freedom), into three simple contrasts: the change from pre-to mid-treatment, from pre-to post-treatment, and from pre to follow up. We then investigated whether these contrasts interacted with Treatment Condition and Gender. These analyses showed that the change from pre- and mid-treatment interacted with Treatment Condition and Gender, $b = -11.61, t(125) = -2.99, p = .003$, such that the relative improvement in Exercise compared to WL was greater for men than for women (see Fig. 1). The other two contrasts (pre-to-post and pre-to-follow-up changes), contrary to our hypothesis, were not moderated by Treatment Condition and Gender (p 's $> .30$). In particular, the lack of a significant Treatment Condition $\times$ Gender $\times$ pre-to post-treatment contrast ($b = -3.56, t(98) = -.80, p = .43$) suggests that, at post-treatment, the overall improvement in EX vs. WL in response to an exercise intervention was not different for men and women.

3. Discussion

Overall, our findings add to the growing body of literature indicating that the effects of exercise on anxiety and related constructs may be stronger for males than for females. Additionally, our study expands previous work by finding that gender also moderates the effects of a brief exercise intervention on AS, an anxiety vulnerability trait. Specifically, our findings demonstrated that two weeks of moderate intensity exercise can effectively reduce ASI across gender. However, as indicated by a significant gender by time by treatment condition interaction, the relative improvement over time in EX compared to WL was

different for males than for females. This was driven by greater relative ASI reduction for men between pre- and mid-treatment. Despite their more-robust early treatment gains, however, men did not show greater relative improvements in EX compared WL than women at post-treatment nor at follow-up.

Although speculative, we offer one potential mechanism accounting for greater AS reductions observed in men relative to women over the first week of exercise that can be drawn from the broader AS and exercise physiology literature. Namely, gender differences in physiological response to exercise appear to contribute to differing degrees of comfort during, and in-response-to, exercise performance. Specifically, women exhibit differential metabolic, pulmonary, and cardiovascular patterns compared to men of comparable fitness, including greater indices of physiological stress and autonomic arousal (Harms, 2006; Harms et al., 1998; Spina et al., 1993; Tarnopolsky, MacDougall, Atkinson, Tarnopolsky, & Sutton, 1990). For example, during moderate-intensity long-duration exercise, while males utilize lipids and metabolize carbohydrates more efficiently than females, females exhibit lower insulin and higher epinephrine concentrations (Tarnopolsky et al., 1990). Indeed, these data parallel women reporting greater stress and exhaustion following exercise (Gavin, Sequin, & McBrearty, 2006). This suggests a potential for more aversive somatic responses to exercise in women, which among inactive women with elevated AS, is likely to be particularly pronounced within the beginning stage of initiating an exercise regimen.

A number of limitations deserve mention. First, although individuals with elevated AS are at risk for clinical anxiety disorders, our use of a relatively small sample of young at-risk students, as opposed to a large treatment-seeking sample, limits the generalizability of our results. For example, we cannot infer that the effects of exercise on AS is moderated by gender in older or clinical anxiety-disordered samples. The small number of males in our sample also limits generalizability and reduces our power to detect gender differences. However, a post-hoc Monte Carlo study suggests that we have sufficient power (greater than .80) to a detect medium effect size for our gender $\times$ study condition $\times$ pre-to post-treatment (and pre-to follow-up) interactions. Replication of gender differences in AS reduction in response to aerobic exercise by future studies must be evidenced before firm conclusions can be drawn.

As another limitation, our intervention was only two weeks in length and was compared only to a waitlist control condition. Further, because we did not assess exercise continuation of participants after the assessment period, we cannot speak to gender differences in response to long-term exercise. Additionally, evaluating the effects of aerobic exercise on rates of AS relative to other brief active control conditions (e.g., strength training, mind-body exercise, health education course) may yield differing between-gender comparisons. Though we speculate the smaller AS-reduction observed in women by the end of the first week of exercise to reflect elevated perceived distress relative to men, we cannot confirm this with the given data. Though participants were cued by experimenters to provide subjective distress ratings during exercise, this procedure was intended solely as a clinical exposure technique (i.e., attention manipulation); in future studies, such ratings should be formally recorded to evaluate their role in the effectiveness of exercise.

In conclusion, our findings point to the possibility that responses to exercise interventions for anxiety pathology vary by gender. This may have implications for the delivery of these interventions and highlight the possibility that the mechanisms of change of exercise interventions, at least as it relates to anxiety reduction, may be different in women than men. We hope that this finding encourages further research aimed at understanding differences between men and women in terms of their responding to exercise interventions. In addition to considering mechanisms at multiple levels (e.g., psychological, physiological), studies that compare types of exercise modalities (e.g., yoga, resistance training) and doses (e.g., intensities, frequencies, and duration) may yield findings that can help refine the knowledge needed to develop individually tailored exercise interventions.

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Fig. 1. Anxiety sensitivity reduction as a function of the interaction between gender and exercise.

Table 1

Baseline demographic and outcome variables.

Variable	Exercise condition (<i>N</i> = 40)		Waitlist condition (<i>N</i> = 20)	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
Age	19.7	2.3	22.7	9.4
ASI	33.0	6.4	34.6	6.6
ASI by gender				
Male	31.0	5.7	35.0	8.4
Female	33.4	6.5	34.3	5.8
	<i>n</i>	<i>%</i>	<i>n</i>	<i>%</i>
Gender				
Male	8	20.0	7	35.0
Female	32	80.0	13	65.0
Race				
White	20	50.0	16	80.0
African American	4	10.0	0	.0
Asian	3	7.5	0	.0
Other	2	5.0	1	.0
Missing/not reported	11	27.5	3	15.0
Hispanic or Latino ethnicity	2	5.0	1	5.0