Journal of Cognitive Psychotherapy: An International Quarterly
Volume 22, Number 4 • 2008

Physical Exercise as Interoceptive Exposure Within a Brief Cognitive-Behavioral Treatment for Anxiety-Sensitive Women

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A brief cognitive-behavioral treatment intervention that included an interoceptive exposure (IE) component was previously demonstrated effective in decreasing fear of anxiety-related sensations in high anxiety-sensitive (AS) women (see Watt, Stewart, Birch, & Bernier, 2006). The present process-based study explored the specific role of the IE component, consisting of 10 minutes of physical exercise (i.e., running) completed on 10 separate occasions, in explaining intervention efficacy. Affective and cognitive reactions and objective physiological reactivity to the running, recorded after each IE trial, were initially higher in the 20 high-AS participants relative to the 28 low-AS participants and decreased over IE trials in high-AS but not in low-AS participants. In contrast, self-reported somatic reactions, which were initially greater in the high-AS participants, decreased equally in both AS groups over IE trials. Findings were consistent with the theorized cognitive and/or habituation pathways to decreased AS.

Keywords: anxiety sensitivity; physical exercise; interoceptive exposure; cognitive behavioral approach

Anxiety sensitivity (AS) is defined as the fear of anxiety-related bodily sensations arising from beliefs that these sensations have harmful physical, psychological, and/or social consequences (Reiss, 1991). AS is implicated in the development and maintenance of

DOI: 10.1891/0889-8391.22.4.303

anxiety-related psychopathology, particularly panic disorder (Schmidt, Zvolensky, & Maner, 2006). AS is also a risk factor for other mental health disorders, such as posttraumatic stress disorder (Feldner, Lewis, Leen-Feldner, Schnurr, & Zvolensky, 2006), depression (Taylor, Koch, Woody, & McLean, 1996), and hypochondriasis (Watt & Stewart, 2000).

AS is also associated with physical inactivity. High-AS individuals, compared with low-AS individuals, report a lower frequency of strenuous exercise (McWilliams & Asmundson, 2001), more negative attitudes toward exercise, and a lower likelihood of exercising to cope with stress (T. MacDonald & Watt, 2003). McWilliams and Asmundson proposed two explanations that are not necessarily mutually exclusive for the observed association between AS and physical inactivity. First, high-AS individuals might avoid physical exercise because it produces physiological sensations similar to those feared by these individuals (e.g., elevated heart rate). Alternatively, or in addition, infrequent exercise might lead to higher levels of AS by limiting exposure to arousal-related sensations.

Cognitive-Behavioral Therapy and Interoceptive Exposure in Anxiety Treatment

Previous studies suggest that cognitive-behavioral therapy (CBT) interventions that include an interoceptive exposure (IE) component (i.e., exposure to feared anxiety-related bodily sensations by practicing brief and harmless exercises) are successful in decreasing AS levels in patients with panic disorder (Arntz, 2002; Beck & Shipherd, 1997; Penava, Otto, Maki, & Pollack, 1998; Telch, Schmidt, Jaimez, Jacquin, & Harrington, 1995) and in nonclinical samples of high-AS individuals (Harrington, Telch, Abplanalp, Hamilton, & Austin, 1995). Examples of IE exercises used in these trials have included chair spinning to induce dizziness and breathing through a straw to induce breathlessness.

Several studies have also examined the effects of the individual components of CBT plus IE interventions in patients with panic disorder (Bouchard et al., 1996; Hecker, Fink, Vogeltanz, Thorpe, & Sigmon, 1998). In these studies, cognitive restructuring was conducted without behavioral experiments, or, alternatively, IE exercises were performed without cognitive restructuring. In fact, Bouchard et al.'s IE participants were told that cognitive techniques were ineffective in the treatment of panic disorder. Results suggested that the IE component alone produced outcomes that were equally favorable to cognitive restructuring, with no differences in dropout rates. The studies, however, did not examine the processes through which these benefits were achieved.

Theoretically, IE exercises might decrease AS in one of at least two ways. First, habituation might occur via a learning process where IE practice leads to extinction of conditioned fearful responding to arousal sensations (Bouton, 2002). That is, through repeated exposure to the feared arousal-related sensations (i.e., the conditioned stimulus), anxiety (i.e., the learned alarm response) resulting from these sensations diminishes. This may occur via a process of stimulus–response dissociation and/or new more positive stimulus–response associations being formed (Bouton, 2002). Alternatively, repeated IE to feared stimuli might serve to alter how arousal sensations are interpreted (i.e., a cognitive explanation for decreases in AS levels). More precisely, with exposure to the feared arousal sensations, individuals have an opportunity to learn to reappraise these threatening cues (e.g., learning that the sensations are harmless)—an opportunity they missed when they were avoiding arousal-inducing activities (Beck & Shipherd, 1997; Beck, Shipherd, & Zebb, 1997). A first step in determining whether habituation and/or cognitive mechanisms of action underlie therapeutic effects involves determining whether a particular treatment (e.g., IE) affects process variables (e.g.. changes in cognitions and/or affective responses to the IE) consistent with each potential underlying mechanism.

Physical Exercise in Anxiety Treatment

In addition to the efficacy of IE, studies also show that physical exercise alone is effective in decreasing anxiety and panic symptoms in patients with panic disorder (Broocks et al., 1998) and in decreasing AS in nonclinical samples (Broman-Fulks, Berman, Rabian, & Webster, 2004; Smits et al., 2008). Broman-Fulks et al. (2004) examined the effects of low- and high-intensity aerobic exercise on AS levels in university students with elevated AS at baseline. High-intensity exercise consisted of either brisk walking or running on a treadmill at a speed that elevated participants' heart rates to between 60% and 90% of their predetermined age-related maximal heart rates. Low-intensity exercise consisted of walking at a speed (i.e., approximately 1 mile per hour) at which heart rates would remain below 60% of maximal heart rate. The exercise interventions consisted of six 20-minute sessions conducted over a 6-week period. Both exercise intensity levels resulted in decreases in AS. However, high-intensity exercise was more effective in decreasing AS than low-intensity exercise, demonstrating a dose–response relationship between exercise intensity and AS reduction. It is likely that the ability of high-intensity exercise to better induce the physiological arousal feared by high-AS individuals (i.e., to activate the "fear network"; Foa & Kozak, 1986) was responsible for the corresponding larger decreases in AS levels in the high- versus low-intensity-exercise group.

Whereas research has established both CBT including IE and physical exercise alone as effective interventions for reducing elevated AS, only recently has the specific use of physical exercise as the IE component of a CBT intervention been examined empirically. A brief group-based intervention consisting of CBT including an IE component in the form of physical exercise (i.e., running) was developed to target high levels of AS in undergraduate women (see review in Watt, Stewart, Conrod, & Schmidt, 2008). Female undergraduates were eligible if they scored either one standard deviation (*SD*) above or below the female mean on an established measure of AS—the Anxiety Sensitivity Index (ASI; Peterson & Reiss, 1992)—during a mass screening of first-year psychology students. After three group sessions (anxiety psychoeducation, cognitive restructuring, and introduction to IE, respectively), participants who partook in the brief CBT intervention were instructed to perform the IE exercises (i.e., running) individually on 10 occasions between the final day of the intervention and the 10-week follow-up. After each running trial, they were instructed to record their affective (i.e., anxiety-related feelings), cognitive (i.e., catastrophizing about arousal sensations) and somatic (i.e., a self-report measure of physiological arousal) reactions to running and to record their pulse rate before running and after 5 and 10 minutes of running (i.e., an objective measure of physiological reactivity).

Outcome measures (e.g., ASI scores) were taken immediately prior to and following the intervention plus at 10 weeks postintervention; these have been reported previously (see Watt, Stewart, Birch, & Bernier, 2006; Watt, Stewart, Lefaivre, & Uman, 2006; Watt et al., 2008). Preintervention to postintervention changes in ASI scores for participants in the CBT condition were 6.7 units for high anxiety-sensitive (HAS) and 3.0 units for low anxiety-sensitive (LAS) participants. For the nonspecific therapy (NST) control condition, change scores were 4.9 and 3.9 units for HAS and LAS participants, respectively. As predicted, there was a significant interaction between AS group (HAS, LAS) and treatment condition (CBT, NST) on changes in ASI scores, $F(3, 160) = 3.54$, $p < .05$. Simple effects revealed a significant difference in change scores between HAS and LAS participants only in the CBT condition, $F(1, 79) = 8.75$, $p < .01$, but not in the NST condition, $F(1, 81) = 1.68$, $p > .05$ (see Watt, Stewart, Lefaivre, et al., 2006). If regression to the mean had been responsible for the AS group effect in the CBT condition, then an AS group effect in the NST condition would also have been observed. Thus, the CBT intervention clearly had the intended effects in reducing AS levels in high-AS individuals. While the overall efficacy of the intervention has been established, data on the IE component (i.e., the recorded reactions to the running trials) have much to offer in demonstrating the utility of repeated IE exposures in the form of physical exercise when treating elevated levels of AS.

Objectives and Hypotheses for the Present Study

Research on the previously mentioned intervention (see Watt et al., 2008) has thus far used traditional data-analytic strategies (e.g., analysis of variance) to examine a relatively narrow slice of change (i.e., participants' scores at preintervention, postintervention, and 10-week follow-up). The present study extends this research by focusing in on the IE intervention component by assessing a somewhat broader spectrum of change (involving 10 measurement occasions) and by using growth curve analyses to estimate individual- and group-level growth trajectories in affective reactions, cognitive reactions, somatic reactions, and physiological reactivity over 10 IE trials (i.e., 10 minutes of running on 10 occasions).

Growth curve models involve an intercept (i.e., the criterion at IE trial 1) and a slope (i.e., the rate of change in the criterion from IE trial 1 to IE trial 10). An intercept and a slope are estimated for each individual and then averaged across groups (e.g., to create a group of individuals high in AS). Building on past work (e.g., Rapee & Medoro, 1994), it was hypothesized that individuals high in AS, relative to individuals low in AS, would evidence (a) higher levels of affective and cognitive reactions at IE trial 1 and (b) steeper rates of change (i.e., decreases) in affective and cognitive reactions from IE trial 1 to IE trial 10. Low-AS participants' affective and cognitive reactions to running were not expected to change over subsequent trials, suggesting that any observed change in these measures would be specific to high-AS individuals. Moreover, high-AS participants' reactions were expected to approach those of low-AS participants by the end of the 10 IE trials. Consistent with prior research (e.g., study 1 in Rapee & Medoro, 1994), it was also hypothesized that individuals high in AS, compared to individuals low in AS, would display (a) comparable somatic reactions (i.e., self-report physiological arousal) and physiological reactivity (i.e., objective pulse rate) at IE trial 1 and (b) comparable levels of somatic reactions and physiological reactivity across IE trial 1 to IE trial 10.

In sum, both cognitive and affective reactions were expected to decrease for high-AS but not low-AS participants over repeated IE trials. However, because IE is believed to decrease distress associated with somatic sensations in high-AS individuals but not necessarily to decrease the somatic sensations themselves (Stathopoulou, Powers, Berry, Smits, & Otto, 2006), self-report somatic reactions and objective pulse rate reactivity to running were not expected to decrease as a result of repeated running trials for either AS group.

Method

Participants

A total of 221 undergraduate women from two universities in eastern Canada participated in the brief CBT intervention (i.e., the larger outcome trial of which this process study is a part; Watt, Stewart, Birch, et al., 2006). Psychology students were selected for inclusion in the study based on their scores on the ASI, a screening measure they completed during class time. Potential participants who screened positive for any physical or health concern (e.g., hypertension) that would preclude them from participating in the exercise program were excluded from the trial. Only women were selected as participants to control for the effects of sex and because women have been found to have higher levels of AS than men (Stewart, Taylor, & Baker, 1997). Participants in the high- and low-AS groups scored at least one *SD* above or below the mean ASI screening score for females (i.e., 17.9 ± 8.7). Mean (*SD*) ASI screening scores for the high- and low-AS groups were 34.16 (6.37) and 8.33 (3.58), respectively (as reported in Watt, Stewart, Birch, et al., 2006). The two groups did not differ significantly on age, year of study, or ethnicity (see Watt, Stewart, Lefaivre, et al., 2006). Participants within each AS group were randomly assigned to either the

CBT or NST conditions to form four groups: high AS/CBT ($n = 51$), low AS/CBT ($n = 61$), high AS/NST ($n = 56$), and low AS/NST ($n = 53$).

Only participants in the CBT condition participated in the IE activities and were requested to complete the 10 physical exercise trials. Of these, 20 of the 51 (39%) high-AS/CBT participants and 28 of the 61 (46%) low-AS/CBT participants handed in homework assignments.[1] Of those who handed in homework, participants in the high-AS group handed in a mean of 9.30 ($SD = 1.63$) of the 10 homework assignments, whereas those in the low-AS group handed in a mean of 8.71 ($SD = 2.61$) homework assignments. There were no differences between the high- and low-AS groups in the proportion of participants who handed in homework assignments, $\chi^2(1, N = 221) = 0.91$, $p > .05$, or in the number of homework assignments completed and handed in, $F(1, 46) = 0.79$, $p > .05$. An AS group (high, low) × homework (handed in, did not hand in) between-subjects ANOVA on initial ASI scores revealed that only the main effect of AS group was significant, $F(1, 107) = 286.18$, $p < .001$. Neither the main effect of homework, $F(1, 107) = 1.94$, $p > .05$, nor the interaction, $F(1, 110)$ 0.02, $p > .05$, was significant, revealing that neither participants in the high-AS group nor those in the low-AS group who handed in the questionnaires differed in pretreatment AS from those who failed to hand in questionnaires.

The 48 participants who handed in any homework assignments are considered the participants for the present process study. The mean age of these participants was 18.91 ($SD = 2.40$) years of age. The majority were first-year undergraduates (89%) and White (91%). There were no difference between high- and low-AS participants on age, $F(1, 44) = 0.21$, $p > .05$; year of study, $F(1, 44) = 3.33$, $p > .05$; or ethnicity, $\chi^2(1, N = 112) = .48$, $p > .05$.

Measures

ASI. The ASI (Peterson & Reiss, 1992) is a 16-item self-report questionnaire that assesses the amount of fear an individual experiences in regard to bodily sensations commonly associated with anxiety and arousal. Participants are asked to rate each item on a 5-point Likert-type scale ranging from *very little* (scored as 0) to *very much* (scored as 4). Ratings on the 16 items are summed for a total that can range from 0 to 64. Psychometric studies have found support for the ASI's test–retest reliability, criterion validity, and construct validity (e.g., support for a distinction between AS and trait anxiety; Peterson & Reiss, 1992). The pretreatment ASI score for the 48 participants in the present process study showed excellent internal consistency (Cronbach's alpha = .91).

Hyperventilation Questionnaire. The Hyperventilation Questionnaire (HVQ; Rapee & Medoro, 1994) is a self-report questionnaire that measures responses to physiological arousal induction challenges. The version of the HVQ scale used in this study contains six items assessing affective reactions (e.g., nervousness), six items assessing cognitive reactions (e.g., feeling of losing control), and 18 items assessing somatic reactions (e.g., breathlessness) to the running exercises.[2] Each symptom is rated on a 4-point scale with anchors of *not at all* (scored as 0) and *markedly* (scored as 3). Subscale scores were obtained by adding scores on each scale's respective items. Thus, the range of possible values was 0 to 18 for the affective and cognitive subscales and 0 to 54 for the somatic subscale. The three subscales have all been shown to possess good to excellent internal consistency (Cronbach's alphas = .88 for affective, and .80 for cognitive, .92 for somatic; Rapee & Medoro, 1994). In addition, the HVQ has been shown to discriminate between high- and low-AS participants when used as a measure of response to hyperventilation challenge (i.e., another way of inducing physiological arousal in IE exercises; A. B. MacDonald, Baker, Stewart, & Skinner, 2000; A. B. MacDonald, Stewart, Hutson, Rhyno, & Loughlin, 2001; Rapee & Medoro, 1994). While labeled the "Hyperventilation Questionnaire" because it was developed for use with lab-based hyperventilation challenge research (Rapee & Medoro, 1994), its item and

subscale content make it appropriate for use in assessing reactions to a wide range of IE activities, including running.

Procedure

High- and low-AS participants were randomly assigned to an active CBT or an NST control condition. The NST condition consisted of a discussion about ethics in psychology (cf. Harrington et al., 1995) and was designed to control for degree of contact with the therapists, group format, and degree of group interaction.

The CBT program consisted of three 50-minute sessions conducted in a small-group format (6–10 participants) over 3 consecutive days. During the first session, participants learned about anxiety, panic attacks, AS, and the role of cognitions in the anxiety cycle (psychoeducational session). They learned that an overfocus on arousal-related physical sensations leads to more ready detection of these sensations, which can then trigger negative cognitions (e.g., catastrophizing), resulting in escalations in anxiety. During the second session, they were taught strategies to identify, challenge, and restructure their dysfunctional thoughts, consistent with cognitive therapy for panic disorder (Clark, 1994; Craske & Barlow, 2001).

The third session consisted of a group introduction to the novel IE component of aerobic exercise (i.e., running), conducted in a venue that was conducive to group physical activity. Running was chosen as the aerobic exercise, as it was an activity that could be easily performed in a group format and could be readily assigned as homework to be performed prior to follow-up. Moreover, some have argued that running is a more ecologically valid IE exercise as compared to activities such as chair spinning and breathing through a straw (e.g., Otto, 2008). Finally, assigning physical exercise, which is known to have positive mental health effects (Stathopoulou et al., 2006), had the potential of benefiting participants even beyond the 10-week trial, especially for high-AS individuals who tend to avoid exercising (McWilliams & Asmundson, 2001). Participants were first taught to find their pulse and calculate their pulse rate in beats per minute by counting the number of beats for 15 seconds and multiplying by four. They calculated a prerunning (i.e., resting) pulse rate, ran together as a group for 5 minutes, and then calculated their pulse rate for a second time. They immediately ran again for an additional 5 minutes, after which they calculated their pulse rate for a third time. Immediately after the 10 minutes of running, participants discussed reactions to the running and were taught how to apply the cognitive restructuring skills learned in the last session to challenge catastrophic cognitions around the interpretation of sensations. They also completed the HVQ (Rapee & Medoro, 1994), which assessed their somatic, affective, and cognitive reactions to the running. Participants were then instructed to complete ten 10-minute running trials between the third day of the intervention and the 10-week follow-up. As on the third day of the intervention, they were asked to record their pulse rate before running, after 5 minutes of running, and again after the full 10-minute running period and then to complete the HVQ after each running trial in reference to their reactions while they were running. The pulse rate calculations and HVQ completion during the third day of the intervention were intended as practice for participants to learn how to complete the process measures. Participants were given a chance to ask questions of group leaders before engaging in the 10 homework running trials where these same measures would be completed for process data collection purposes. Only participants in the CBT and not those in the NST condition performed the IE component of the intervention.

Participants completed the ASI at pretreatment, at posttreatment, and at the 10-week follow-up. For the purpose of the present process study, only the pretreatment and follow-up ASI questionnaires were examined in participants who actually passed in the homework exercises (i.e., the process study sample) to measure the effects of the brief CBT plus the IE component on AS levels.

Results

Intervention Effects on AS Levels

To determine whether the CBT treatment was effective in reducing AS levels for the subset of 48 participants who completed the homework exercises, a 2 (AS group: high, low) × 2 (time: preintervention, follow-up) mixed-model ANOVA with AS group as the between-subjects factor and time as the within-subjects factor was performed on ASI scores, revealing main effects of time $F(1, 45) = 53.84$, $p < .001$, and AS group, $F(1, 45) = 118.47$, $p < .001$, and a significant time × group interaction, $F(1, 45) = 20.24$, $p < .001$. Simple effects analysis confirmed that the treatment led to greater reductions in ASI scores for high-AS participants, $F(1, 19) = 40.82$, $p < .001$, $\eta_p^2 = .68$, than for low-AS participants, $F(1, 26) = 7.40$, $p < .05$, $\eta_p^2 = .22$.[3]

Physiological Arousal Manipulation Check

Intent-to-treat analyses were conducted using a conservative last-value carry-forward method for replacing missing values from participants who failed to hand in all 10 homework trials. To test whether running actually induced increases in physiological arousal, changes in pulse rate (averaged over the 10 trials) from prerunning, after 5 minutes, and after 10 minutes of running were analyzed using a 2 (AS group: high, low) × 3 (phase: prerunning, after 5 minutes, after 10 minutes) mixed-model ANOVA, with AS group as the between-subjects factor and phase as the within-subjects factor. There was a significant main effect of phase, $F(2, 92) = 187.11$, $p < .001$, and a significant AS group × phase interaction, $F(2, 92) = 4.963$, $p < .01$. While simple effects analyses revealed that running resulted in increased pulse rates relative to resting baseline for both high-AS participants, $F(2,18) = 119.95$, $p < .001$, $\eta_p^2 = .86$, and low-AS participants, $F(2,26) = 73.86$, $p < .001$, $\eta_p^2 = .73$, the effect size was somewhat larger for the high-AS than for the low-AS participants. Pairwise post hoc tests were conducted for both high- and low-AS participants. All pairwise comparisons were significant at $p < .001$[4]. Further, high- and low-AS groups did not differ on prerunning pulse rates or on pulse rates after 5 minutes of running, but after 10 minutes of running, pulse rates were higher in high-AS participants than in low-AS participants, reflecting a higher level of physiological reactivity for high-AS compared with low-AS participants (see Table 1). This set of analyses provided a check that the IE was effective in inducing physiological arousal (in terms of elevated pulse rate) in both AS groups and that this elevation was sustained over the 10 minutes of running.

Growth Curve Analyses

Growth curve analyses were conducted with multilevel modeling and HLM 6.04 software (Raudenbush, Bryk, & Congdon, 2007). All participants who completed at least one IE trial were included in analyses regardless of attrition or missing data. HLM 6.04 uses restricted maximum-likelihood

TABLE 1. Means (and Standard Deviations) of Pulse Rates Measured in Beats per Minute

	LAS	HAS	*F*
Prerunning	72.61 (13.95)	69.59 (12.47)	0.68
Post 5 minutes running	118.30 (32.09)	130.93 (31.67)	1.83
Post 10 minutes running	125.66 (34.15)	144.25 (28.28)	3.97*

Note. HAS = high anxiety sensitivity; LAS = low anxiety sensitivity.
*$p = .05$.

estimation (Raudenbush & Bryk, 2002), an estimation approach that provides efficient estimates and allows for use of all potential data so that any participant with at least one IE trial may be included in growth curve analyses (Raudenbush, 2002). Growth curve analysis is also a flexible data-analytic strategy that accommodates variability in both the spacing and the number of repeated observations (Raudenbush, 2002).

In the present study, growth curve analyses were used to analyze the rate and the pattern of change in outcome variables (i.e., subjective affective, cognitive, and somatic reactions and objective physiological reactivity) over IE trials. Specifically, growth curve analyses examined whether outcome variables for the high-AS group (coded as 1) were different from outcome variables for the low-AS group (coded as 0) over IE trials. Additionally, growth curve analyses examined linear rates of change in outcome variables over IE trials as a function of AS group. Changes in outcome variables were mostly constant across IE trials, suggesting that linear growth models would adequately represent these changes.

Growth curve analyses consisted of two levels. At level 1, within-person variability in a given outcome variable (e.g., affective reactions) was modeled as a function of time. Number of IE trials was used to demarcate time and was centered so that trial 1 was set to 0. The intercept thus represents the value of a given outcome variable in response to the first IE trial for the low-AS group. At level 2, between-persons variability in intercepts and in slopes was modeled as a function of AS group. Growth curve analyses thus examined if AS group influenced outcome variables in response to the first IE trial and if AS group moderated linear rates of change in outcome variables over IE trials (i.e., a cross-level interaction of IE trials × AS group). Significant interactions were probed using simple slopes analyses (Preacher, Curran, & Bauer, 2006).

Separate analyses were conducted for each level 1 outcome variable. A sample equation follows:

level 1 (within-person) model:

$$\text{affective reactions}_{ti} = \pi_{0i} + \pi_{1i}\,(\text{IE trials}) + e_{ti}$$

level 2 (between-persons) model:

$$\pi_{0i} = \beta_{00} + \beta_{01}\,(\text{AS group}) + r_{0i}$$

$$\pi_{1i} = \beta_{10} + \beta_{11}\,(\text{AS group}) + r_{1i}$$

Growth curve analyses are presented in Table 2, where it may be seen that variance components (representing individual variability) for slopes and for intercepts were significant in all four models. Slopes and intercepts were thus allowed to vary randomly across participants, and person-specific parameters for slopes and for intercepts were computed for each participant at level 1. Person-specific parameters were pooled when estimating level 2 parameters.

Affective Reactions. Figure 1A displays the trajectories of affective reactions to IE trials for individuals within both high-AS (depicted by black broken lines) and low-AS (depicted by gray solid lines) groups. As can be seen in the figure, most of the high-AS participants' reactions to IE were initially higher than reactions of low-AS participants and decreased gradually over time. By the last running trial, high-AS participants' reactions to running were approaching those of low-AS participants. Reactions to running for most low-AS participants, on the other hand, did not appear to experience any systematic changes over subsequent running trials.

High-AS participants had higher levels of affective reactions in response to the first IE trial than low-AS participants. AS group also significantly moderated the linear rate of change in affective reactions across IE trials (see panel 1 in Table 2 and Figure 2A). Simple slope analyses suggested that the linear rate of change in affective reactions for low-AS participants was not significantly different from zero, $t(46) = 0.10$, $p > .05$. However, for high-AS participants, the linear

TABLE 2. Growth Curve Analyses Predicting Changes in Affective, Cognitive, and Somatic Reactions, and Physiological Reactivity in High-AS and Low-AS Participants

	Fixed Effects		Random Effects
Predictor	Unstandardized Coefficient	*SE*	Variance Component
Predicting affective reactions			
Intercept	0.49	0.41	4.10****
Interoceptive exposure trials	0.00	0.04	0.02****
AS group	1.98***	0.63	—
Interoceptive exposure trials × AS group	–0.13**	0.06	—
Predicting cognitive reactions			
Intercept	0.36	0.29	2.06****
Interoceptive exposure trials	0.01	0.03	0.02****
AS group	1.33***	0.45	—
Interoceptive exposure trials × AS group	–0.10*	0.05	—
Predicting somatic reactions			
Intercept	12.01****	1.45	53.74****
Interoceptive exposure trials	–0.29***	0.10	0.08**
AS group	4.88**	2.24	—
Interoceptive exposure trials × AS group	–0.01	0.16	—
Predicting physiological reactivity			
Intercept	48.86****	5.50	817.89****
Interoceptive exposure trials	0.09	0.37	2.24****
AS group	23.19***	8.51	—
Interoceptive exposure trials × AS group	–1.03*	0.56	—

Note. Growth curve analyses predicting changes in affective, cognitive, and somatic reactions are based on 430 responses from 48 participants. Growth curve analyses predicting changes in physiological reactivity are based on 428 responses from 48 participants. *SE* = standard error. For AS group, low AS = 0 and high AS = 1.
*$p < .10$. **$p < .05$. ***$p < .01$. ****$p < .001$.

rate of change in affective reactions was negative and significant, $t(46) = –2.69$, $p < .01$, suggesting a decline in affective reactions across IE trials.

Cognitive Reactions. Figure 1B displays the trajectories of cognitive reactions to IE trials for individuals within both high- and low-AS groups. As can be observed in the figure, the majority of high-AS participants' reactions to the IE were initially higher than reactions of low-AS participants and decreased gradually over time. By the last running trial, observable differences between high- and low-AS participants appear to have decreased substantially. Reactions of low-AS participants did not appear to follow any consistent pattern, with some participants experiencing slight decreases and other participants experiencing slight increases in reactions over subsequent running trials.

High-AS participants had higher levels of cognitive reactions in response to the first IE trial compared to low-AS participants. Moreover, the interactive effect of AS group on the linear rate

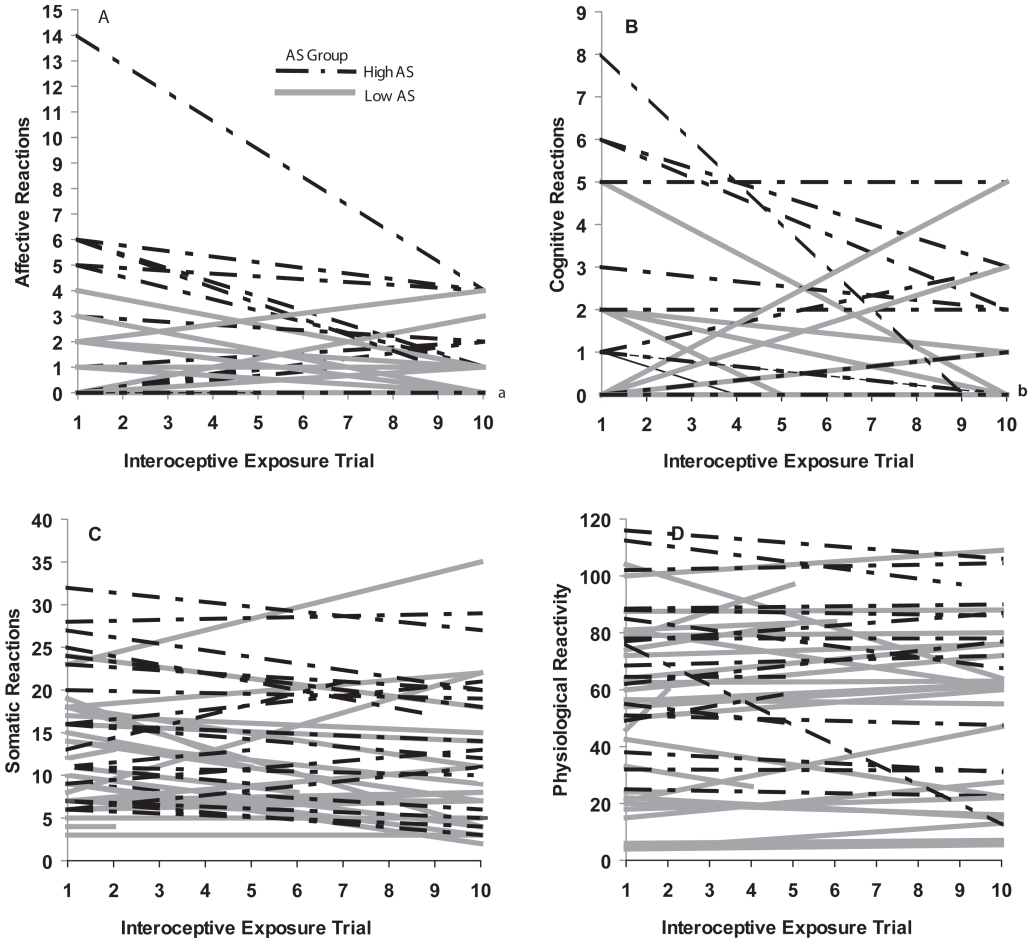

Figure 1. Individual trajectories for affective, cognitive, and somatic reactions, and physiological reactivity for high- and low-AS participants across interoceptive exposure trials.
[a]The line that spans the horizontal axis, reflecting scores of 0 on interoceptive trials, represents seven high-AS participants and 18 low-AS participants. [b]The line that spans the horizontal axis, reflecting scores of 0 on interoceptive trials, represents nine high-AS participants and 20 low-AS participants.

of change in cognitive reactions across IE trials was marginally significant (see panel 2 in Table 2 and Figure 2B). Simple slopes analyses indicated that the linear rate of change in cognitive reactions for low-AS participants was not significantly different from zero, $t(46) = 0.30$, $p > .05$. For high-AS participants, however, the linear rate of change in cognitive reactions was negative and significant across IE trials, $t(46) = -2.26$, $p < .05$.

Somatic Reactions. Figure 1C displays the trajectories of subjective somatic reactions to IE trials for individuals within both high- and low-AS groups. Somatic reactions appear to be higher for the high-AS group than for the low-AS group; however, unlike the trajectories for affective and cognitive reactions, trajectories for somatic reactions do not appear to differ systematically between AS groups. The figure also reveals that, unexpectedly, somatic reactions decreased with subsequent IE trials for the majority of participants, regardless of AS group.

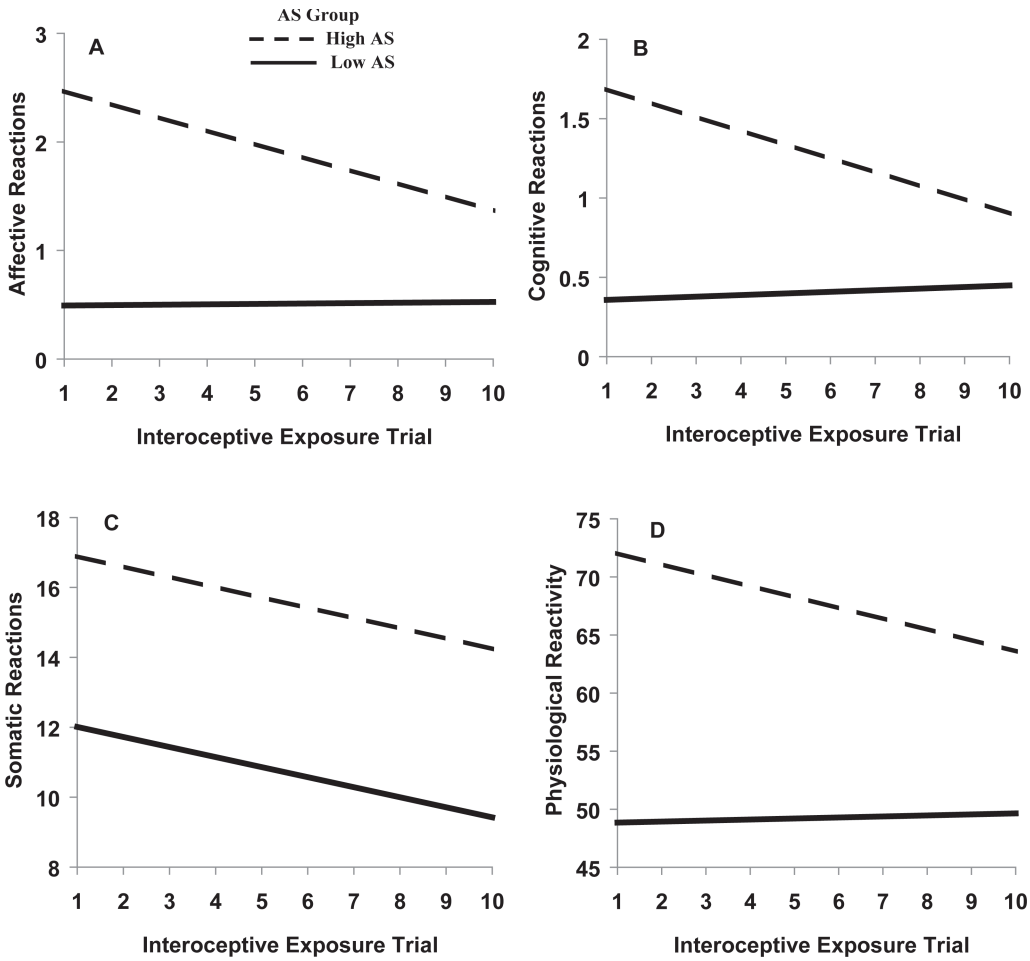

Figure 2. Trajectories for affective, cognitive, somatic reactions, and physiological reactivity for high- and low-AS participants across interoceptive exposure trials.

High-AS participants also had significantly higher levels of somatic reactions in response to the first IE trial than low-AS participants. However, AS group did not significantly moderate the linear rate of change in somatic reactions across IE trials (see panel 3 in Table 2 and Figure 2C). This suggests that the linear rate of change in somatic reactions across IE trials was similar for high- and low-AS participants. In both groups, a negative and significant decline in somatic reactions was observed across IE trials.

Physiological Reactivity. Figure 1D displays the trajectories of physiological reactivity to IE trials for individuals within both high- and low-AS groups. Again, high-AS participants appear to have higher physiological reactivity initially than low-AS participants. Further, physiological reactivity appears to decrease for high-AS participants over subsequent running trials. This pattern over running trials is not apparent for low-AS participants.

High-AS participants had higher levels of physiological reactivity in response to the first IE trial relative to low-AS participants. Moreover, the interactive effect of AS group on the linear rate of change in physiological reactivity (i.e., difference between resting pulse rate and the average

pulse rate while running) across IE trials was significant (see panel 4 in Table 1 and Figure 2D). Simple slope analyses showed that the linear rate of change in physiological reactivity for low-AS participants was not significantly different from zero, $t(46) = 0.24$, $p > .05$. However, for high-AS participants, the linear rate of change in physiological reactivity was negative and significant across IE trials, $t(46) = -2.25$, $p < .05$.

Discussion

The goal of the present study was to further examine the IE component of a brief CBT previously shown to decrease AS levels in high-AS individuals (Watt, Stewart, Birch, et al., 2006). Specifically, the present study sought to determine whether IE operates differently for individuals who are high and low in AS in terms of impact on subjective affective, cognitive, and somatic reactions and objective physiological reactivity to physical exercise (i.e., running) over repeated exposures.

Cognitive and Affective Reactions to IE Trials

First, high-AS participants initially experienced higher levels of catastrophic cognitive reactions to IE than low-AS participants. Further, high-AS but not low-AS participants experienced decreases in catastrophic cognitions over subsequent trials of IE. Although the current analyses did not specifically test the mechanism of action of IE's treatment efficacy, results are consistent with the theorized cognitive pathway to decreases in AS levels (Beck & Shipherd, 1997; Beck et al., 1997). Second, anxious affect in response to IE was also initially higher for high-AS than for low-AS participants. Anxious affect also decreased for high-AS but not for low-AS participants over subsequent trials of IE. Again, although the current analyses did not specifically test mechanism of action, results are consistent with the theorized habituation pathway to decreases in AS levels (Bouton, 2002). That cognitive and affective reactions to the IE trials were initially higher in high-AS individuals and decreased only for this group provides preliminary evidence of a link between AS and these reactions and of the potential influence of decreases in these reactions to decreases in ASI scores for high-AS individuals. In addition, given the decreases in affective and cognitive reactions observed for high-AS individuals across IE trials, the present results are consistent with the possibility that running may be an effective IE strategy for achieving decreases in fear of arousal sensations.

Previous studies found that IE, without cognitive restructuring, was effective in alleviating panic-related symptoms (Arntz, 2002; Bouchard et al., 1996; Hecker et al., 1998). These studies, however, provided some rationale to participants regarding the purpose and role of IE in reducing symptoms. For example, Hecker et al.'s participants were taught that panic attacks were a learned fear of normal physical sensations and, based on this model, were provided an explanation of the rationale behind IE. On the other hand, another study (Carter, Marin, & Murrell, 1999) found that IE without any accompanying explanation of treatment rationale was not effective in decreasing AS levels in high-AS university students. It is possible, then, that even in the absence of a specific cognitive restructuring component, in the studies where an IE treatment rationale was provided (i.e., Arntz, 2002; Bouchard et al., 1996; Hecker et al., 1996), the rationale encouraged participants to nonetheless engage in some type of conscious reinterpretation of anxiety-related symptoms. In fact, Bouchard et al. (1996) found that IE interventions resulted in changes in participants' beliefs about the catastrophic consequences of anxiety-related sensations that were equivalent in magnitude to those changes resulting from cognitive restructuring, consistent with an underlying cognitive mechanism for even the IE intervention. Similarly, in the present study, participants were specifically instructed to reinterpret the arousal sensations when engaging in their assigned running trials based on the cognitive restructuring training they had

received prior to the IE component. Thus, even a habituation-focused explanation of the role of IE in decreasing participants' fear of anxiety cannot preclude the potential role of altered cognitions in treatment efficacy.

Somatic Reactions and Physiological Reactivity to IE Trials

The third hypothesis, that physiological reactivity to IE would not differ between high- and low-AS groups initially or decrease over subsequent trials, was not supported. High-AS participants did report higher initial somatic reactions than low-AS participants. Increased awareness of the arousal-related sensations stemming from greater fear of these sensations in high-AS individuals might account for these unexpected initial differences. Alternatively, it is possible that lower fitness levels in high-AS individuals due to physical inactivity (T. MacDonald & Watt, 2003; McWilliams & Asmundson, 2001) led to greater somatic reactions (e.g., breathlessness, pounding heart) to running. Although Rapee and Medoro (1994) did not find such AS group differences in somatic reactions to a hyperventilation challenge (i.e., another form of physiological arousal induction) between high- and low-AS participants in the first of three studies using a large sample of undergraduate students ($N = 450$), in their subsequent two studies, AS levels were significantly positively correlated with somatic reactions to the challenge. In addition, other studies have reported higher levels of self-reported somatic reactions to hyperventilation challenges for high-AS participants compared to low-AS participants (e.g., A. B. MacDonald et al., 2000). Further, Rapee, Brown, Antony, and Barlow (1992) found that self-report measures of somatic reactions to a hyperventilation challenge differed between participants with an anxiety disorder and controls.

The second unexpected result in the present study pertained to parallel decreases in high- and low-AS participants in somatic reactivity over IE trials. It is possible that both AS groups paid progressively less attention to the somatic sensations over trials and thus that scores decreased for all participants. Alternatively, all participants might have become more physically fit by the end of the trials, resulting in less somatic sensations (e.g., heart pounding, breathlessness). This fitness-based explanation, however, is unlikely, as 10 minutes of running falls below the threshold of a minimum of 20 minutes of continuous high-intensity exercise recommended by the American College of Sports Medicine to achieve any fitness benefits (Pollock et al., 1998).

Similarly, and also unexpectedly, pulse rate reactivity to IE differed between high- and low-AS participants. The high-AS group was somewhat more physiologically reactive to the IE exercise than the low-AS group, consistent with either a fear-based explanation (i.e., fear of arousal sensations increased pulse rate further on top of the arousal induced by physical exercise per se) or a decreased fitness explanation (i.e., greater pulse rate reactivity to exercise due to lower physical fitness secondary to exercise avoidance). However, unlike parallel decreases in self-reported somatic reactions over trials in both high- and low-AS participants, pulse rate reactivity, an objective measure of physiological arousal, decreased only for high-AS participants. A few possibilities exist in explaining these unexpected findings. First, if running resulted in higher pulse rates in high-AS participants because of decreased fitness levels, it may be possible that for unfit individuals (as opposed to moderately fit individuals), some fitness benefit resulted from even the minimal physical activity involved in the current study (i.e., 10 minutes of running on 10 occasions). Second, decreased anxiety over the exercises specifically for high-AS individuals might have led to decreases in pulse rate reactivity for this group. In fact, some characterize heart rate as a physiological expression of anxious affect (e.g., Foa & Kozak, 1986). Also consistent with an anxiety-based explanation is the fact that pulse rate reactivity data followed a pattern that was more similar to self-reported affective reactions than to self-reported physiological reactivity data (i.e., the somatic subscale of the HVQ). At this time however, these possibilities can be only speculative without the use of an objective measurement of fitness level to rule out fitness-based explanations of reduced pulse rate reactivity to running in high-AS individuals.

Limitations and Future Directions

The current study was not without its limitations. First, participants completed the IE homework trials in the absence of the investigators, and therefore running conditions might have been inconsistent between participants. For example, some participants might have been listening to music during the running trials, providing a distraction from arousal sensations. Distraction may in fact deter from habituation across exposure trials (Foa & Kozak, 1988). Similarly, although the self-report measure of physiological arousal and the noted increases in pulse rates suggested that participants were engaging in strenuous exercise, it is possible that some high-AS individuals were continuing avoidance of arousal sensations by engaging in less strenuous running (i.e., relatively low intensity exercise). Future research could use individually determined measures of exercise intensity (e.g., measures of ventilatory or lactate threshold) to objectively measure physical effort (e.g., Parfitt, Rose, & Burgess, 2006; Welch, Hulley, Ferguson, & Beauchamp, 2007).

Second, exposure to running without the anticipated catastrophic consequences might have decreased these individuals' aversion to physical exercise and led them to participate in exercise over and above the instructed IE trials. Engagement in additional physical exercise might have had an effect on reactions to the IE over trials but was not monitored in the present study. Future studies could use physiological measures (e.g., maximal oxygen consumption, blood lactate levels) to determine fitness levels before and after the prescribed period of physical exercise (cf. Broocks et al., 1997).

Third, as the HVQ was administered through a paper-and-pencil format, it is possible that participants failed to complete the questionnaire on the actual self-reported dates (e.g., participants might have completed them well after the running exercise was performed). Electronic diaries with time stamps could be used in future studies to ensure such compliance. Nonetheless, paper-and-pencil diaries have yielded similar compliance to electronic diaries in terms of reported and actual questionnaire completion dates (e.g., Green, Rafaeli, Bolger, Shrout, & Reis, 2006). Some argue that rapport with participants and study design can have an equally strong effect on compliance as the format of the questionnaire itself (Green et al., 2006; Piasecki, Hufford, Solhan, & Trull, 2007).

Fourth, although participants who failed to hand in the IE homework did not differ on any of the measured variables from those who did, it is possible that they differed systematically on other potentially confounding characteristics that were not assessed in the current study. Thus, it is not clear to what degree the present sample is representative of those who participated in the larger treatment outcome study (and thus to what degree the present results are generalizable). Finally, the homogeneity of the current study's sample potentially limits generalizability to men, non-Whites, nonuniversity students, and clinical populations.

Results from the current study are consistent with both the theorized cognitive and habituation pathways (Beck & Shipherd, 1997; Beck et al., 1997; Foa & Kozak, 1986) in explaining the role of IE in decreasing fear of anxiety sensations in high-AS individuals. However, because of the limited number of IE trials, it was not possible to verify whether and how the two proposed pathways interact with each other in achieving decreases in AS and whether it is possible that one pathway is actually "driving" the other. Perhaps decreases in cognitive catastrophizing over IE trials lead to subsequent decreases in anxious affect or, alternatively, decreases in anxious affect resulting from habituation drive subsequent decreases in the tendency to catastrophize potential consequences of physiological arousal. It is also possible that these two alternative explanations apply in different ways across individuals.

A study is presently under way in Nova Scotia, Canada, that includes a larger number of IE trials that will provide sufficient data to tease out these alternative theoretical explanations. With additional IE exposures, it will be possible to use the types of analytic tools, such as multivariate time-series analyses (i.e., a technique that requires a minimum of 50 repeated observations;

Tabachnick & Fidell, 2007), that can analyze the relationships between two or more series of data (e.g., cognitive and affective reactions to the IE trials). These types of analyses have been used in other studies to determine process of change during therapeutic interventions (e.g., Bouchard et al., 2007). Additional IE trials and assessing participants' engagement in other physical activities will also allow us to provide a better test of a possible fitness-based mechanism for explaining treatment efficacy.

In conclusion, the current study found that an IE component of a brief CBT intervention for reducing AS led to decreases in both cognitive and affective reactions to physiological arousal, induced through physical exercise, exclusively for high-AS individuals. Finding these positive effects from an intervention that includes a running component is especially appealing, as physical exercise has been shown for several decades to have far-reaching mental health benefits (e.g., Folkins & Sime, 1981; for a review, see Stathopoulou et al. 2006) and running is a cost-effective and flexible form of physical exercise.

Notes

1. Although the participation rate for handing in the homework assignments was relatively low, it is possible that more participants performed the homework but failed to hand in the completed questionnaires.

2. One reverse-scored item from the original seven-item affective scale (i.e., relaxation) was removed as it negatively impacted internal reliability.

3. η_p^2 = partial eta squared is a measure of effect size. η_p^2 represents the proportion of the effect + error variance (i.e., the total variance minus the variance due to other factors in the analysis) that is accounted for by the effect itself (Tabachnick & Fidell, 2007). η_p^2 gives the contribution of each factor separately, taken as if it were the only variable, so that it is not masked by any more powerful variable. For example, in the simple effects analysis, because there is only one factor (time: pretreatment vs. follow up), the η_p^2 represents the proportion of *total* variance that is accounted for by time. A η_p^2 of .68, then, signifies that 68% of the variance in ASI scores can be attributed to time (i.e., to treatment).

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Acknowledgments. *The authors are grateful to the Dalhousie doctoral students who served as study therapists: Denise Bernier, Cheryl Birch, Marie-josée Lefaivre, and Lindsay Uman. The authors also wish to thank Maureen Balcom for her research assistance in data coding and entry. This research was supported by an operating grant from the Nova Scotia Health Research Foundation (NSHRF) to the second and fourth authors (SHS and MCW). The first author (BCS) is supported by an NSHRF graduate studentship. The second author (SHS) was supported through an Investigator Award from the Canadian Institutes of Health Research at the time this study was conducted; she is currently supported through a Killam Research Professorship from the Faculty of Science at Dalhousie University. The fifth author (JW) is supported by a Career Investigator Award from the Michael Smith Foundation for Health Research. The final author (VVG) is supported by a doctoral fellowship from the Social Sciences and Humanities Research Council of Canada.*

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