THE RELATIONSHIP BETWEEN EXERCISE AND ANXIETY SENSITIVITY AND THE ROLE OF RUNNING AS INTEROCEPTIVE EXPOSURE IN A BRIEF COGNITIVE BEHAVIOURAL TREATMENT FOR DECREASING ANXIETY SENSITIVITY

by

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The undersigned hereby certify that they have read and recommend to the Faculty of Graduate Studies for acceptance a thesis entitled "THE RELATIONSHIP BETWEEN EXERCISE AND ANXIETY SENSITIVITY AND THE ROLE OF RUNNING AS INTEROCEPTIVE EXPOSURE IN A BRIEF COGNITIVE BEHAVIOURAL TREATMENT FOR DECREASING ANXIETY SENSITIVITY" by Brigitte Colette Sabourin in partial fulfilment of the requirements for the degree of Doctor of Philosophy.

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ABSTRACT

Anxiety sensitivity (AS; fear of anxiety-related bodily sensations) is a risk factor for anxiety and related psychological disorders. Preliminary evidence also associates high AS with reduced levels of physical exercise and fitness. The primary objectives of the five studies comprising this dissertation were 1) to further explore the relationships between AS levels and exercise/fitness levels, and 2) to evaluate outcomes and processes of a brief group cognitive behaviour therapy (CBT) that included a novel exercise-based interoceptive exposure (IE; exposure to feared anxiety-related sensations) component of running, with female undergraduate students. High AS female undergraduate participants endorsed more barriers to exercise than low AS participants, and these accounted for the inverse relationships between AS group and exercise/fitness levels (Study 1). The brief CBT/IE led to decreases in AS levels (Studies 2 and 4) and in symptoms of stress, depression, and anxiety (Study 4) for high AS participants. Processes involved in the brief CBT's therapeutic effects were explored by examining cognitive (i.e., catastrophic thoughts), affective (i.e., feelings of anxiety), and somatic (i.e., physical sensations) reactions to the running IE component with an existing measure, the hyperventilation questionnaire (HVQ; Study 2), and a brief version of the measure, the HVQ-B, developed and validated in Study 3 (Study 5). Changes in cognitive and affective reactions to running were most closely associated with the brief CBT/IE's therapeutic benefits, underlying the importance of changing the meaning of and emotional reaction to physiological arousal. Surprisingly, a health education control (HEC) intervention consisting of an interactive discussion on exercise, nutrition and sleep for health, including problem-solving barriers to health behaviours, also led to decreases in AS levels and in symptoms of depression and anxiety (Study 4). Physical exercise, the common link between the two interventions, may be partially driving the interventions' therapeutic benefits. More specifically, perhaps both interventions addressed barriers to exercise, either by altering the meaning of and emotional reaction to exercise (CBT/IE), or through problem-solving (HEC). Encouraging physical exercise in high AS individuals by acknowledging and addressing barriers to exercise might help decrease these individuals' AS levels and improve their overall mental health.

LIST OF ABBREVIATIONS AND SYMBOLS USED

ANOVA	Analysis of variance
AS	Anxiety sensitivity
ASAT	Anxiety sensitivity amelioration training
ASI	Anxiety Sensitivity Index
ASI-R	Anxiety Sensitivity Index – Revised
ASI-3	Anxiety Sensitivity Index - 3
B	Unstandardized multiple regression coefficient
BAI	Beck Anxiety Inventory
CBT	Cognitive behaviour therapy
CFA	Confirmatory factor analysis
CFI	Comparative fit index
CO_2	Carbon dioxide
d	Cohen's d; measure of effect size
df	Degrees of freedom
DASS-21	Depression Anxiety Stress Scales – 21 item version
DB	Decisional balance
DBS	Decisional Balance Scale
EAST	Extrinsic affective Simon task
EQS	Structural equation modeling service
EX	Exercise condition
EX + C	Exercise plus cognitive behaviour therapy condition
F	Computed value of ANOVA
GAD	Generalized anxiety disorder
HEC	Health education control
HLM	Hierarchical Linear and Nonlinear Modeling
HK	Heterogeneous kurtosis
HVQ	Hyperventilation Questionnaire
HVQ-B	Hyperventilation Questionnaire-Brief
IE	Interoceptive Exposure
IFI	Incremental fit index
M	Mean
N	Sample size
NS	Nonsignificant
NST	Non specific treatment
p	Probability of Type I error
PAM	Physical Activity Measure
PCA	Principal component analysis
PTSD	Post traumatic stress disorder
r	Pearson product-moment correlation
R^2	Measure of strength of association in regression
RMSEA	Root mean square error of approximation
SD	Standard deviation
SE	Standard error
SL	Standardized factor loading

SSRI	Selective serotonin re-uptake inhibitors
t	Computed value of *t* test
TTM	Transtheoretical model
VH	Voluntary hyperventilation
WLC	Wait list control
z	Computed value of Sobel test
α	Alpha co-efficient; index of internal consistency
β	Beta weight; standardized multiple regression coefficient
$\chi 2$	Computed value of a chi-square test
$\eta_p 2$	Partial eta squared; measure of effect size
ΔR^2	Change in value of strength of association in regression

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Chapter 1 . INTRODUCTION

"So first of all let me assert my firm belief that the only thing we have to fear is fear itself—nameless, unreasoning, unjustified terror which paralyzes needed efforts to convert retreat into advance" (Roosevelt 1965, p. 274).

Anxiety sensitivity (AS) is an individual difference variable referring to the fear of arousal-related sensations. This fear arises from beliefs that these sensations have harmful physical, social, or psychological consequences (Reiss & McNally, 1985). For example, an individual with high levels of AS might fear heart palpitations, believing them to portend a heart attack, fear trembling in anticipation of public ridicule, and/or fear nervousness believing it to be a sign of mental illness. In contrast, a person with low levels of AS would perceive these sensations to be perhaps unpleasant but transient and harmless consequences of being in an anxious state.

Measuring Anxiety Sensitivity: The Anxiety Sensitivity Index

Since the introduction of AS as a concept by Reiss and McNally in 1985 (Peterson & Plehn, 1999), the 16-item Anxiety Sensitivity Index (ASI; Peterson & Reiss, 1992) has been the most widely used measure to assess AS. Items on the ASI are intended to describe an individual's perceptions of the possible negative consequences associated with the experience of anxiety (Reiss, Peterson, Gursky, & McNally, 1986). Items (e.g., "It scares me when my heart beats rapidly") are rated on a 5-point Likert scale ranging from "Very Little" (scored as 0) to "Very Much" (scored as 4). Total scores range from 0 to 64. The mean ASI level in a normative sample of the general population has been reported at 19.01 (*SD* = 9.11; Peterson & Reiss, 1992). A study examining sex differences in university students found a mean of 17.4 (*SD* = 9.4) for women and 14.6

(*SD* = 8.7) for men (Stewart, Taylor, & Baker, 1997). In general, women score slightly but significantly higher on the ASI than men (Peterson & Plehn, 1999).

In the first thirteen years after the ASI was developed, there were already over 100 published, peer-reviewed studies examining its reliability and validity. (Zinbarg, Mohlman, & Hong, 1999). The ASI has demonstrated excellent psychometric properties in both clinical and non-clinical samples (Peterson & Reiss, 1992; Rector, Szacun-Shimizu, & Leybman, 2007). The ASI is the instrument that was used in the present dissertation to measure AS (see Appendix A for selected items of the ASI).

Anxiety Sensitivity and Trait Anxiety

AS differs from trait anxiety both conceptually and empirically (McNally, 1989). Conceptually, trait anxiety refers to a higher order trait denoting a tendency to respond fearfully to stressors in general. On the other hand, AS refers to a lower order trait denoting a general tendency to respond to one's own arousal sensations fearfully. Individuals with high trait anxiety might experience frequent intense episodes of state anxiety, with accompanying physiological sensations; however, they do not necessarily respond fearfully to the sensations themselves. Alternatively, individuals with high AS would fear the actual physiological sensations associated with being in an anxious state; however, they do not necessarily react fearfully to a wide variety of situations (McNally 2002). In other words, it is possible to be high AS without being high trait anxious, and vice versa.

Anxiety Sensitivity, Trait Anxiety, and Biological Challenges.

Numerous studies have been able to differentiate between trait anxiety and AS. Arousal induction procedures, also called biological challenges (throughout this dissertation, the terms arousal induction and biological challenge will be used interchangeably), involve experimentally inducing arousal sensations that mimic anxiety reactions. The two most common forms of arousal induction procedures are voluntary hyperventilation (VH) and inhalation of carbon-dioxide-enriched air, ranging anywhere from 4% to 65% (CO_2 inhalation; Zvolensky & Eifert, 2001). An early study by Rapee, Brown, Antony, and Barlow (1992) examined responses to both VH and inhalation of 5.5% CO_2 in 198 participants with any anxiety disorder and 25 non-anxious control participants. All participants exhibited similar objective physiological responses to the challenges, as assessed by respiratory rate, heart rate, and CO_2 measures. Participants with panic disorder, however, exhibited the highest degree of subjective distress, followed by participants with other anxiety disorders. As expected, non-anxious control participants had the lowest levels of subjective distress. AS level, but not trait anxiety level, was a significant predictor of subjective response to both the hyperventilation and CO_2 challenge procedures. These findings suggest that distress to biological challenge procedures are not necessarily related to physiological responsivity to these types of challenges but, rather, depend on participants' fear of the sensations (i.e., AS levels).

Rapee and Medoro (1994) conducted three studies examining the relationships between AS, trait anxiety, and reactions to VH. They used the Hyperventilation Questionnaire (HVQ; Rapee & Medoro, 1994) to evaluate responses to VH. The HVQ is divided into three subscales that assess cognitive (i.e., catastrophic thoughts; e.g., "Worrying that your actions are damaging to your health"), affective (i.e., feelings of anxiety; e.g., "Nervousness"), and somatic (i.e., physical sensations; e.g., "Breathlessness") reactions to arousal. In Study 1, high and low AS participants who were selected to be similar on trait anxiety levels engaged in a VH task. Both groups had similar physiological reactivity to the task, as revealed by non-significant differences in heart rate reactivity, and respiratory rate and depth. HVQ scores were collected before and immediately following the VH task. Before the VH task, participants rated their current symptoms to use as a baseline measure. Following VH, participants rated the maximum symptoms experienced during the task. High AS participants had greater increases in scores from baseline to VH on the cognitive subscale of the HVQ than low AS participants. There were no differences on the affective or somatic scales. Analyzing pre-post change in biological challenges, however, produces a conservative marker of responses to the arousal, as high AS individuals might have elevated scores before the challenge due to greater levels of anticipatory anxiety. In fact, pre-VH scores on the affective subscale were elevated in high AS individuals compared with low AS individuals.

Study 2 was designed to correct for the possibility of meaningfully elevated pre-VH scores on the HVQ-B. In addition, Study 2 specifically examined the influence of both trait anxiety and AS, as continuous measures, on responses to the VH task. Participants completed the HVQ before, immediately following (again assessing maximum symptoms during VH), and five minutes after the VH task. Scores on all three HVQ subscales were higher before the VH task than five minutes after the task, suggesting some degree of anticipatory anxiety. Thus, post-VH scores were used as the baseline measure for the analyses. Trait anxiety did not significantly account for differences from baseline to mid-VH scores on any of the HVQ subscales. AS levels, on the other hand, significantly predicted changes in scores on both the cognitive and affective, but not the somatic, subscales, even after controlling for trait anxiety levels.

Study 3 was similar to Study 2 except that trait anxiety, AS levels, and baseline HVQ scores were collected on a different day than the day participants performed the VH task. Trait anxiety did not predict baseline to VH increases in any of the HVQ subscales. On the other hand, AS levels predicted changes in all three subscales of the HVQ, even after controlling for trait anxiety. Additionally, in both Studies 2 and 3, AS levels and trait anxiety levels were moderately but significantly correlated (*r*s = .31 - .55), demonstrating an association between the two constructs. Results from these studies suggest that AS levels are important determinants of reactions generally, but especially of cognitive and affective reactions, to a VH task, over and above levels of trait anxiety.

Later studies (e.g., Carter, Suchday, & Gore, 2001; Eke & McNally, 1996; Sturges, Goetsch, Ridley, & Whittal, 1998) also found AS levels to be better predictors of responses to arousal induction than measures of general trait anxiety. Heightened responses to arousal induction procedures, therefore, can be considered a distinguishing feature of AS.

Anxiety Sensitivity, Trait Anxiety, and Anxiety Disorders.

AS also differentiates itself from trait anxiety by its ability to distinguish between different types of anxiety disorders. Individuals with generalized anxiety disorder (GAD) and panic disorder exhibit equivalent levels of trait anxiety but differ in their cognitions and fears of the sensations themselves (Taylor, Koch, & McNally, 1992; Rapee, 1985). Not surprisingly individuals with panic disorder display higher levels of AS and more negative cognitions about the potential effects of experiencing anxiety sensations, than those with GAD. These results provide further evidence that trait anxiety and AS measures assess distinct constructs.

Anxiety Sensitivity and Psychopathology

High AS has been implicated in the development and maintenance of anxiety disorders, particularly panic disorder (Reiss, Peterson, Gursky, & McNally, 1986). The tendency for high AS individuals to catastrophize arousal-related sensations amplifies anxiety reactions, thereby increasing the risk of panic attacks and panic disorder. Indeed, AS levels have been found to be elevated in panic disorder patients (Stewart, Knize, & Pihl, 1992; Taylor et al, 1992).

Longitudinal studies also have found that AS constitutes a pre-morbid vulnerability factor for the development of panic attacks and panic disorder (Maller & Reiss, 1992; Schmidt, Lerew, & Jackson, 1997; 1999). Maller and Reiss (1992) found that high AS individuals were five times more likely to have developed one or more anxiety disorders than low AS individuals when retested at a three year follow-up. High AS also was associated with the development and frequency of panic attacks over this same time period. Finally, the study found that AS levels, compared with trait anxiety levels, were a better predictor of panic attacks during the year preceding the three-year follow-up assessment. Schmidt, Lerew, and Jackson (1997; 1999) assessed new U.S. Air Force cadets before and after attending an intense 5-week basic training “boot camp”. Pre-boot camp ASI scores predicted the onset of panic attacks following boot camp, even after controlling for trait anxiety levels and prior history of panic attacks. Collectively, these studies provide compelling evidence that high AS is a robust longitudinal predictor of panic attacks and panic disorder.

Elevated AS levels also are associated with other mental health disorders, such as posttraumatic stress disorder (Taylor et al., 1992), other anxiety disorders (Maller & Reiss, 1992; Taylor et al., 1992), substance use disorders (Stewart, Samoluk, & MacDonald, 1999), hypochondriasis (Watt & Stewart, 2000; Watt, Stewart, & Cox, 1998) and depression (Taylor, Koch, Woody, & McLean, 1996). Additionally, AS has been implicated as playing a role in physical health issues such as chronic pain (Ocañez, McHugh, & Otto, 2010), smoking behaviours (Zvolensky, Bonn-Miller, Bernstein, & Marshall, 2006), and physical inactivity (Goodin et. al., 2009; Smits & Zvolensky, 2006). In summary, a large body of evidence has accumulated in the past 25 years linking high AS with a wide range of psychological and physical difficulties. This evidence also speaks to the importance of research targeted at decreasing AS levels.

Anxiety Sensitivity and Physical Exercise

Countless studies have shown that physical exercise confers important mental and physical health benefits (for reviews see Penedo & Dahn, 2005; Stathopoulou, Powers, Berry, Smits, & Otto, 2006; Warburton, Nicol, & Bredin, 2006). In fact, research suggests that for high AS individuals, the benefits of exercise might be even more pronounced than for low AS individuals (Smits, Tart, Rosenfield, & Zvolensky, 2011). Smits et al. (2011) conducted a study with healthy community volunteers with no history of Axis I psychopathology or panic attacks. They examined the relationships between AS, physical activity levels, and responsivity to a biological challenge consisting of inhalation of CO_2-enriched air. AS levels were found to moderate the relationship between physical activity levels and fearful responding to arousal. That is, for low AS participants, there was no relationship between physical activity levels and fearful responding to the challenge. On the other hand, high AS participants who exercised regularly had lower levels of distress following the arousal procedure than high AS participants who did not exercise regularly. Thus, it appears that exercising confers some protective effects specifically for high AS individuals.

There is growing evidence, however, suggesting that elevated AS levels are associated with lower levels of physical exercise, particularly strenuous exercise, and lower physical fitness levels (Goodin et al., 2009; McWillams & Asmundson, 2001; Smits & Zvolensky, 2006). Goodin et al. (2009) found that AS levels were significantly inversely related to weekly strenuous exercise bouts in a sample of healthy young adults (r = -.26). McWilliams and Asmundson (2001) found partial support for the hypothesized negative relationship between exercise and AS in a sample of undergraduate students. In undergraduate males, there was a negative association between self-reported frequency of strenuous exercise and AS, and also between self-reported physical fitness levels and AS. For females, there was a significant inverse relationship but only between AS and self-reported fitness levels. Finally, Smits and Zvolensky (2006) noted that several studies have demonstrated that patients with panic disorder have lower levels of cardiovascular fitness. For example, one earlier study showed that participants with panic disorder and/or agoraphobia self-reported avoidance of moderate or vigorous exercise and performed more poorly on objective tests of physical fitness than untrained (i.e., engaging in no regular aerobic exercise for at least two years prior to the study) healthy participants (Broocks et al., 1997). Smits and Zvolensky postulated that AS might be a contributing factor to this relationship. In order to test this, they conducted a study with community volunteers with panic disorder. They found that 33% of the sample were currently inactive (i.e., self-reporting that they exercised “0” times per week). In contrast, in 2007, only 21.5% of the general population were inactive (Physical Activity Council, 2012). Inactive participants did not differ from active participants in medical health, but they did have higher ASI scores than active participants.

Theoretically, it is possible that high AS individuals avoid physical exercise due to a fear of the physiological sensations associated with exercise (e.g., perspiration, elevated heart rate), which are similar to those induced by anxiety. Alternatively, infrequent exercise might limit exposure to arousal sensations and, thus, lead to higher AS levels. These two possible explanations are not mutually exclusive, and might indeed work in conjunction with each other in maintaining both exercise avoidance and elevated AS levels.

Although there is some evidence of an inverse relationship between AS levels and exercise participation, some studies (e.g., MacWilliams & Asmundson, 2001) have found only partial support for this relationship. Additional research is needed to further confirm, or refute, the growing evidence that high AS individuals do indeed tend to avoid physical exercise. Furthermore, there have been no published studies to date exploring some of the factors which might be involved in explaining this possible exercise avoidance in high AS individuals.

Factors Affecting Decisions to Exercise

When determining potential reasons why AS individuals might avoid participating in physical exercise, it is useful to examine which factors influence decisions to exercise in the general population. The transtheoretical model (TTM; Prochaska & DiClemente, 1982) of behaviour change has been useful in understanding participation in health behaviours more generally, and physical exercise participation in particular (Marshall & Biddle, 2001). According to the TTM, individuals can be classified in different stages of "readiness" to engage in physical exercise. Theoretically, earlier stages of readiness would be associated with physical inactivity, whereas later stages would be associated with physical activity. A meta-analysis performed by Marshall and Biddle (2001) confirmed that physically active individuals were indeed more likely to be classified in later stages of readiness, and that physically inactive individuals were more likely to be classified in earlier stages of readiness. One important factor in determining a particular individual's stage classification is the decisional balance (DB) construct (Marcus, Rakowski, & Rossi, 1992). DB involves weighing the pros (i.e., benefits) and cons (i.e., barriers) of engaging in exercise. If the benefits outweigh the barriers, it is expected that an individual is more likely to exercise. Studies have shown that individuals who are physically active have higher net DB scores (i.e., benefits - barriers) than those who are physically inactive (Marshall & Biddle, 2001; Williams, Lewis, et al., 2008). Also, DB scores have been shown to prospectively predict exercise participation at six months follow-up (Williams, Lewis, et al., 2008).

Because higher net DB scores are related to physical activity participation (e.g., Williams, Lewis, et al., 2008), and high AS individuals engage in *less* physical activity than low AS individuals (e.g., Goodin et al., 2009), it would be expected that high AS individuals would have *lower* net DB scores than low AS individuals. There has been no research to date, however, that has applied the DB construct in explaining differences in exercise behaviours between high and low AS individuals. Because of the many benefits of exercise that appear to be amplified in high AS individuals, better knowledge of the factors affecting exercise participation in these individuals is paramount. Understanding how high and low AS individuals differ in their perceptions of the benefits and barriers to exercise marks an important first step in addressing potential exercise avoidance in this at-risk group.

Cognitive Behaviour Treatment and Anxiety Sensitivity

Evidence suggests that AS is a personality-like trait. Maller and Reiss (1992) documented a 3-year test-retest reliability of 0.71 for ASI scores. This level is comparable with the two-week test-retest reliability of .71 for men and .74 for women found in previous research (Reiss et al., 1986), and as such constitutes a sign of stability over a relatively long period of time. Although AS has been considered a dispositional trait-like concept (McNally, 1999), results from several studies suggest that AS is indeed malleable with cognitive behavioural treatment (CBT), including brief, single-session CBT-based interventions (e.g., Keough & Schmidt, 2012; Schmidt et al., 2007).

CBT aims to alter cognitions related to anxiety sensations and decrease avoidance of feared stimuli. Typically, CBT treatments for anxiety include most or all of the following components: psychoeducation, cognitive restructuring (i.e., changing maladaptive thought patterns), relaxation training (e.g., diaphragmatic breathing), in-vivo exposure, and interoceptive exposure (IE). In-vivo exposure refers to exposure to feared or previously avoided places or situations. IE, on the other hand, refers to exposure to feared arousal-related sensations, through procedures such as breathing through a straw, hyperventilation, and chair spinning.

Studies with Clinical Populations

Results from earlier treatment studies revealed that when participants underwent CBT for panic disorder and/or agoraphobia, they also experienced decreases in AS. One of the first studies to examine this effect was conducted by McNally and Lorenz (1987), who evaluated the effects of a 10-12 session treatment for agoraphobia consisting of psychoeducation, cognitive restructuring, diaphragmatic breathing training, and in vivo exposure. As expected, treatment participants experienced a reduction in agoraphobic avoidance behaviours following treatment. The treatment also resulted in pronounced decreases in AS levels from the clinical to non-clinical range, where they remained at 6-month follow-up. Similar results were found for CBT interventions delivered in group format (Penava, Otto, Maki, & Pollack, 1998; Telch et al., 1993). Penava et al. (1998) tested a clinic-based group CBT, emphasizing mostly cognitive restructuring and IE, but also including the other CBT components. A total of 26 individuals diagnosed with panic disorder, with or without agoraphobia, participated in 12 90-minute group sessions, and completed the ASI before starting treatment, and following sessions 4, 8, and 12. ASI scores decreased following the first four sessions, decreasing further after sessions 8 and 12. Furthermore, improvements in ASI scores were significantly correlated with improvements in panic symptoms (r = .62).

In a treatment study by Schmidt, Trakowski, and Staab (1997), individuals with panic disorder were randomly assigned to either a 12-session group-based CBT intervention with or without a diaphragmatic breathing retraining component, or a wait-list condition, forming three separate groups. At the 12-week follow-up, both intervention groups, but not the control group, experienced decreases in panic-related symptoms and in AS levels. Schmidt et al. also assessed response to a 35% CO_2 inhalation challenge. At pre-treatment, all participants experienced similar levels of anxious responding to the challenge and 74% of participants experienced a panic attack as a result of the challenge. At post-treatment, on the other hand, participants in both treatment conditions responded with lower levels of anxiety to the challenge than those in the control condition. Also, only 20% of participants in the treatment conditions, versus 64% of participants in the control condition experienced a panic attack as a result of the challenge. AS levels moderated treatment effects: participants with high ASI levels post-treatment (ASI >14) reported more distress, and were eight times more likely to experience a panic attack from the CO_2 challenge, than those with low ASI levels. This study suggests that a reduction in AS levels is an important determinant of improvements in anxious responding to physiological arousal following CBT treatment for panic.

A meta-analysis (Smits, Berry, Tart, & Powers, 2008), that included 16 studies, revealed a large AS reduction effect size (mean Hedges' g [comparable to Cohen's D for interpretation] = 1.40) for CBT interventions in individuals seeking treatment for panic disorder and other Axis I pathology. Additional analyses revealed that the average participant receiving CBT improved more than 92% of control group participants. These results suggest that CBT treatments aimed at ameliorating panic and other related disorders also lead to reductions in AS levels.

Changes in AS levels, in fact, have been found to mediate improvements in anxiety disorders and symptoms. Smits, Powers, Cho, and Telch (2004) delivered a 12-session CBT over an 8-week period to 90 individuals with panic disorder with agoraphobia. An additional 40 participants were randomly assigned to a wait-list condition. Treatment consisted of psychoeducation, cognitive restructuring, training in diaphragmatic breathing, in-vivo exposure, and IE. Participants in the treatment condition experienced greater decreases in symptoms of panic disorder, including anxiety, frequency of panic attacks, agoraphobic avoidance, and global disability, than those in the control condition. Between-group effect sizes (i.e., difference in effects between treatment and control participants) ranged from d = 0.55 to 1.30. Treatment participants also experienced a decrease in AS, with a between-subjects effect size of d = 1.72. Mediation analyses revealed that changes in AS fully mediated changes in global disability, and partially mediated changes in anxiety, frequency of panic attacks, and agoraphobic avoidance. The study provides further evidence of the central role of AS reduction in improvements in clinical anxiety syndromes.

Studies with Non-Clinical Populations

It has become evident that AS plays an important role in the development and maintenance of psychopathology. Furthermore, changes in AS have been found to be related to and in some cases, even mediate, improvements in psychopathological symptoms. As a result, a number of treatment studies have tested variants of CBT aimed specifically at reducing AS levels as a way to target this underlying risk factor for psychopathology. One of the first reported treatment studies evaluating an AS reduction intervention was conducted by Telch and colleagues (Harrington, Telch, Abplanalp, Hamilton, & Austin, 1995). The intervention consisted of a brief CBT which included psychoeducation, IE, and diaphragmatic breathing. There was some evidence that, compared with a control group, the treatment significantly reduced AS levels.

Gardenswartz and Craske (2001) compared a 5-hour CBT-based prevention workshop to a wait-list control condition with 121 at risk college students who had at least moderate levels of AS (ASI scores ≥ 16), and had experienced at least one unexpected panic attack in the past 12 months. Participants were excluded if they had a diagnosis of panic disorder, were undergoing psychological treatment for any anxiety disorder, or had been prescribed psychotropic medication. Participants in both the workshop and wait-list conditions experienced similar decreases in ASI scores. The workshop, however, appeared to have an additional protective effect against the development of panic disorder: nine participants in the control condition (13.6%), but only one participant in the workshop condition (1.8%) developed panic disorder six months following the workshop.

More recently, Schmidt and colleagues (2007) delivered a primarily computer-based, brief Anxiety Sensitivity Amelioration Training (ASAT) to high school students (n = 46), university undergraduates (n = 263), and community individuals (n = 96). Interested individuals were eligible if they had at least moderately elevated levels of AS (ASI scores ≥ 17), and no current or past 12-month psychiatric illness. Participants were randomly assigned to the ASAT condition or to a control condition. Both conditions consisted of a 30-minute computer-based audio-visual presentation followed by 10 minutes with an experimenter. The goal of the intervention was to educate participants about the benign nature of the immediate effects of stress on the body. In addition, the presentation taught participants that they may have developed a conditioned fear to arousal-related sensations and then went on to describe IE exercises designed to decrease the conditioned fear. The control condition focused on nutrition and health, without directly addressing AS levels.

There were no between-groups differences in AS levels prior to the intervention; however participants in the experimental group had lower scores on the ASI following the single-session intervention. On average, ASI scores had decreased by 30% for participants who attended the ASAT compared with 17% for those in the control condition, a significant difference. There was also some evidence for reduced incidences of psychopathology at 2-year follow-up for participants in the ASAT condition compared with those in the control condition. Also, reaction to a 20% CO_2 inhalation challenge was included as an outcome measure. Participants in the intervention condition responded with less distress to the challenge compared with participants in the control condition. It appears that the brief one-session intervention resulted in decreases in both self-report and behavioural markers of AS, and helped to prevent Axis I pathology.

A follow-up study (Keough & Schmidt, 2012) was conducted with university students with more elevated ASI scores ($M = 29.6$). This study did not exclude those with psychiatric disorders. For this study, a homework component was added to the single-session intervention. Following the brief audio-visual presentation, participants in the treatment condition were exposed to a number of IE procedures. The IE exercise that produced the highest level of distress was performed until distress became minimal, and then assigned to be performed for homework. The control condition consisted of the same health presentation as the previous (Schmidt et al., 2007) study, with the addition of assigning daily monitoring of health habits as homework. Participating in the intervention resulted in substantially larger reductions in ASI scores than participating in the control condition. The authors noted that the 58% reduction in total ASI scores achieved at the one-month follow up in their study was greater than reductions achieved in other similar prevention studies (e.g., Gardenswartz & Craske, 2001; Schmidt et al., 2007). Surprisingly, homework adherence did not influence outcomes.

A recent review and meta-analysis by Smits, Berry, Tart, et al. (2008) examined the efficacy of AS-reduction CBT interventions in non-treatment seeking, at-risk, high AS individuals. A total of eight studies were included, and revealed a pooled effect size that was in the moderate to large range (Hedges' $g = 0.74$). The average participant in the treatment conditions improved more than 76% of participants in the control conditions. Collectively, these studies suggest the useful role of AS reduction as a preventative measure against the development of psychopathology. CBT is a promising means of achieving substantial reductions in AS levels. Furthermore, results from some of the studies reported above (e.g., Schmidt, Trakowski, & Staab, 1997; Schmidt et al., 2007) suggest that responses to biological challenge exercises can be useful clinical markers of intervention-based improvements.

Aerobic Exercise as Treatment for Anxiety Sensitivity

Aerobic exercise produces many of the same bodily sensations that are associated with anxiety (e.g., increased heart rate, respiration, perspiration). Thus, repeated exposure to feared arousal-related sensations through aerobic exercise can act as another type of IE, similar to IE procedures such as hyperventilation or breathing through a straw.

Broman-Fulks, Berman, Rabian, and Webster (2004) conducted the first documented aerobic exercise intervention specifically targeting AS levels. High AS individuals (i.e., ASI scores > 25) who were in good general health and not currently engaging in aerobic exercise were recruited for the study. Participants were randomly assigned to either a high-intensity aerobic exercise condition, as defined by brisk walking or jogging on a treadmill at 60-90% of age-adjusted predicted maximal heart rate, or a low-intensity exercise condition, consisting of treadmill walking at one mile per hour. Both conditions included six 20-minute sessions over a two-week period. Both high- and low-intensity exercise resulted in decreases in AS. High-intensity participants, however, experienced decreases in AS more quickly than low-intensity participants. Also, more high-intensity than low-intensity participants experienced a significant decrease in AS. Finally, only participants in the high-intensity group experienced decreases in fear of bodily sensations as assessed by the Body Sensations Questionnaire (Chambless, Caputo, Bright, & Gallagher, 1984). Results suggest that a brief intervention consisting of six sessions of high-intensity exercise decreased fears of arousal-related sensations.

A follow-up study conducted by Broman-Fulks and Storey (2008) replicated and extended the group's original study. High AS, non-active, participants were randomly assigned to six 20-minute exercise sessions or to a no-exercise control group. A newer version of the ASI, the ASI-Revised (ASI-R), which consists of four subscales, was used as the outcome measure. Scores on three of the four subscales (i.e., Fear of Publicly Observable Anxiety Reactions, Fear of Cardiovascular Symptoms Scores, Fear of Cognitive Dyscontrol) decreased only for the exercise group, and not for the control group. Scores on the fourth subscale, Fear of Respiratory Symptoms, on the other hand, decreased in both groups. Total ASI-R scores for participants in the exercise group decreased following the first bout of exercise, and stabilized at the lower level throughout the rest of the sessions. For participants in the control condition, there was a slight (non-significant) decrease in total ASI-R scores, but a rebound to pre-contact scores from the third session onward.

Smits, Berry, Rosenfield, et al. (2008) conducted a similar study examining the effects of aerobic exercise on decreasing AS levels. High AS (i.e., ASI score ≥ 25) individuals were selected if they were physically inactive (i.e., exercising less than once per week), in general good health, not involved in psychotherapy, and on stable dosage of psychotropic drugs. They were randomly assigned to one of three conditions. The first condition consisted of aerobic exercise (EX), the second of aerobic exercise plus cognitive restructuring (EX + C), and the third of a wait-list control group (WLC). Exercise consisted of six 20-minute sessions over a 2-week period, at a prescribed intensity of 70% of age-adjusted maximal heart rate. In contrast to the Broman-Fulks et al. (2004) study, Smits et al. provided participants with a treatment rationale prior to beginning the exercise participation. A videotape was shown to participants explaining AS and the role of aerobic exercise in decreasing AS levels. In addition, participants were reminded to focus on the physiological sensations throughout the exercise sessions. The EX+C condition consisted of all of the aspects of the EX condition plus a specific explanation on the role of cognitive restructuring in decreasing AS. Additionally, participants in the EX+C condition were exposed to Socratic questioning during the exercise sessions, based on their highest scoring ASI items, which formed the cognitive restructuring portion of the intervention. Both active interventions, but not the WLC condition, resulted in decreases in AS and in depression and anxiety symptoms. Unexpectedly, there were no differences between the EX+C and the EX conditions. The study also demonstrated that reductions in AS preceded and mediated reductions in anxiety and depression symptoms. The three above studies demonstrate the promising role of aerobic exercise as a potential stand-alone treatment for decreasing AS levels. None of the studies, however, included a follow-up assessment that was longer than three weeks in order to ascertain if changes could be maintained over a longer term.

Cognitive Behaviour Therapy with Exercise as Interoceptive Exposure

A recent study conducted at two eastern Canadian universities was the first to use physical exercise (i.e., running) as the IE component of a brief CBT, AS reduction intervention (Watt, Stewart, Birch, & Bernier, 2006; Watt, Stewart, Conrod, & Schmidt, 2008; Watt, Stewart, Lefaivre, & Uman, 2006). The intervention followed a treatment manual that had been adapted from previous interventions (Conrod et al., 2000; Harrington & Telch, 1994). A total of 221 undergraduate women were recruited based on scoring one SD above or below a normative mean for female university students on the ASI (i.e., 17.9 ± 8.7). Only female participants were selected for the study to control for gender effects in the groups and because women have higher AS levels than men (Stewart, Taylor, & Baker, 1997). Potential participants with any health condition or concern (e.g., cardio-vascular problems) that might preclude safe participation in aerobic exercise were excluded from the study. High AS (mean [*SD*] ASI = 34.2 [6.4] and low AS (mean [*SD*] ASI = 8.3 [3.6] participants were randomly assigned to either the treatment condition, or a control condition to form four groups: high AS/CBT, high AS/control, low AS/CBT, low AS/control. The control condition, consisting of a group discussion on ethics in psychology, was included in order to control for non-specific effects that could influence outcome (e.g., therapist effects, group exposure).

The intervention consisted of three 50-minute group sessions on three consecutive days. The first session focused on psychoeducation. Participants learned about anxiety, AS, panic, and the anxiety cycle. During the second session, participants practiced identifying, challenging, and restructuring dysfunctional cognitions related to anxiety, consistent with cognitive therapy for panic disorder (Craske & Barlow, 2001). During the third session, participants engaged in the novel IE portion of the intervention. They ran together as a group for 10 minutes in order to induce arousal-related sensations. Running was selected because it is an activity that can be easily performed in a group format and assigned as homework to be completed prior to follow-up. Participants were instructed to focus on the sensations produced by running, and compare these with sensations experienced when in an anxious state. Then, participants were instructed to complete ten 10-minute running sessions between the last therapy session and the 10-week follow-up assessment. Participants were instructed to complete the HVQ (Rapee & Medoro, 1994) immediately following every running trial.

The treatment resulted in greater decreases in AS levels for high AS than for low AS participants, but only in the CBT intervention group (Watt, Stewart, Birch et al., 2006). The treatment also resulted in decreased problematic drinking behaviours (Watt, Stewart, Birch, et al., 2006), and reduced pain anxiety levels (Watt, Stewart, Lefaivre, et al., 2006), for high AS participants in the CBT intervention group.

Although outcome data were analyzed and reported, data from the HVQ completed following every homework running trial were not analyzed. Because the HVQ assesses affective, cognitive and somatic responses to arousal, analyzing patterns of change in these different dimensions can provide useful information regarding the mechanisms driving the intervention's therapeutic benefits. Also, because of the theoretical importance of IE in decreasing AS levels, a more extensive IE component might help increase the intervention's efficacy and maintain treatment gains. Finally, the control condition consisted of a group discussion on ethics in psychology. Although this might have controlled for the effect of contact with therapists and other group members, a more stringent, believable, and relevant control condition would help control for other non-specific factors (e.g., the effects of discussing topics relevant to participants' own health), thereby strengthening the evidence supporting the brief intervention's efficacy. Although the brief CBT/IE appeared promising, additional research was needed to further explore both outcomes and processes of this novel intervention, and compare it to other, more stringent, control conditions.

A Further Examination of the Relationship between AS and Reactions to Physiological Arousal

As mentioned above, fearful reactions to physiological arousal is a key feature of AS. Biological challenge exercises induced in a laboratory setting are a useful way of inducing physiological arousal in order to better understand AS and its clinical correlates. Additionally, treatments aimed at decreasing AS and clinical conditions associated with AS (e.g., panic disorder) commonly incorporate an IE component. Thus, it appears that inducing physiological arousal can be used both experimentally (i.e., as biological challenge procedures) and therapeutically (i.e., as IE exercises).

There has been a recent surge in the interest of IE as a therapeutic tool, as evidenced by a series of articles devoted to novel uses of IE (introduced by Stewart & Watt, 2008). IE was described as helpful for treating conditions as diverse as post traumatic stress disorder (Wald, 2008), hypochondriasis (Walker & Furer, 2008), and smoking addiction (Zvolensky, Yartz, Gregor, Gonzalez, & Bernstein, 2008).

Additionally, research in the treatment of anxiety and related disorders has moved beyond simply investigating *which* treatments work, and has proceeded to elucidating *how* treatments work (e.g., Meuret, Rosenfield, Seidel, Bhaskara, & Hofmann, 2010; Hofmann et al., 2007; Smits, Rosenfield, McDonald, & Telch, 2006; Woody, Whittal, & McLean, 2011). That is, there is increasing emphasis on researching the mechanisms through which interventions exert their therapeutic effects (Moscovitz, Antony, & Swinson, 2009; Stewart & Watt, 2008). In the case of IE for AS and associated clinical difficulties, repeated exposure to physiological arousal can lead to fear reduction through at least two mechanisms.

First, a cognitive model posits that arousal-related sensations are misinterpreted as dangerous or portending catastrophic consequences. This appraisal heightens apprehensive arousal, and thereby increases the risk of developing panic attacks and panic disorder (D. M. Clark, 1988). Repeated exposure to arousal sensations in the absence of the accompanying feared catastrophic consequences might result in a re-evaluation of the sensations as less threatening (Chambless & Goldstein, 1981).

Second, a conditioning model posits that fear of physiological arousal is a learned response (Bouton, Mineka, & Barlow, 2001). New learning occurs when feared stimuli (e.g., physiological arousal), is repeatedly presented without the feared consequences (e.g., panic attack; Bouton, 2004). In the case of high AS individuals, repeated exposure to arousal sensations through IE exercises provides precisely such an opportunity for new learning to occur, leading to reduction in the fear of arousal sensations through a process of extinction learning (Moscovitch et al., 2009).

Empirical evidence favouring either a cognitive process, a learning (i.e., fear reduction) process, or both processes, has yet to emerge (Moscovitch et al., 2009; Stewart & Watt, 2008). Well-designed longitudinal studies are needed in order to test these two potential mechanisms, or possibly develop alternative mechanisms of action. In order to conduct these, measures are needed that assess different dimensions of responding to arousal sensations.

Measuring Reactions to Physiological Arousal: The Hyperventilation Questionnaire

One useful tool in measuring reactions to physiological arousal is the HVQ, which was described briefly above. The scale contains 33 items, each rated on a 4-point Likert scale ranging from 0 (“Not at all”) to 4 (“Markedly”). Although labelled the “Hyperventilation Questionnaire” because it was first used to assess reactions to a lab-based hyperventilation task, the HVQ’s items are appropriate to measure reactions to arousal sensations in general. The HVQ’s usefulness lies in the ability of its three separate subscales to distinguish between affective, cognitive, and somatic reactions to physiological arousal. This allows researchers to further evaluate cognitive and conditioning models of fearful responding to arousal. Furthermore, the HVQ’s three subscales can also be helpful in further teasing out the mechanisms of change resulting from IE exercises. In fact, the HVQ has been used in both laboratory and treatment studies. In laboratory studies, it has been able to distinguish between high and low AS participants’ responses to arousal induction procedures (Carter, Suchday, & Gore, 2001; A. B. MacDonald, Baker, Stewart, & Skinner, 2000; A. B. MacDonald, Stewart, Hutson, Rhyno, & Loughlin 2001; Rapee & Medoro, 1994). In treatment studies, responses on the HVQ have been shown to decrease over time following cognitive restructuring and IE (e.g., Carter, Marin, & Murrell, 1999).

The HVQ has demonstrated strong psychometric properties, including internal consistency and concurrent validity (Rapee & Medoro, 1994). With a total of 33 items, however, the HVQ can be taxing when completed within a battery of measures, or repeatedly as would be done as part of a treatment process assessment. Developing an abbreviated measure that maintains the original's strong psychometric properties would increase the ease with which this measure could be used for both laboratory and treatment studies.

The Current Dissertation

The current dissertation consists of five stand-alone manuscripts. Aims of the current dissertation include: 1) to expand on the existing body of knowledge regarding AS and exercise behaviours, including determining the roles of benefits and barriers to exercise in explaining differences in exercise behaviours in high and low AS individuals, 2) to examine the process(es) through which an existing brief group-based CBT intervention that includes running as a novel IE component results in decreases in AS levels, by examining changes in cognitive, affective, somatic, and physiological reactions to IE, 3) to create and evaluate a brief version of the HVQ, and 4) to replicate and extend an existing treatment study to further explore the (a) outcomes and (b) process(es) of the intervention.

Study 1 further explores the relationships between AS and exercise/perceived fitness levels to replicate and extend previous research in this area. First, physical exercise and fitness levels are compared in high versus low AS female undergraduates. Next, factors involved in decisions to engage in physical exercise are examined. Specifically, high and low AS individuals' perceptions of the benefits and barriers to exercise are compared. Finally, mediation analyses are performed to determine if benefits and/or barriers to exercise account for the relationships between AS levels and physical exercise/fitness levels.

Study 2 examines the processes through which the recent CBT intervention reported in Watt et al. (2008) resulted in decreases in AS levels for high AS female undergraduates. Subjective, self-report reactions recorded by participants engaging in the homework portion of the intervention are analysed to determine changes in cognitive, affective, and somatic reactions of running for high and low AS participants. Also, changes in recordings of participants' heart rates before, during, and after the 10-minute running trials are examined.

Study 3 describes the development and psychometric properties of a brief version of the HVQ, the HVQ-Brief (HVQ-B). Data from three separate studies are combined to select the items to be retained for the HVQ-B. Next, the newly-created HVQ-B is used in a study and its psychometric properties are examined. Finally, data from all studies are combined and a confirmatory factor analysis is performed on the 18-item HVQ-B.

Study 4 describes outcomes of a replication and extension study of Watt et al.'s (2008) CBT plus running as IE intervention. The treatment is compared to a more stringent control condition consisting of interactive discussions on health, focusing on the roles of exercise, nutrition, and sleep on optimal health. The effects of the treatment on AS levels and on symptoms of stress, depression, and anxiety are examined. As the original study (Watt et al., 2008) examined the effects of the intervention until only a 10-week follow-up period, the fourth manuscript also examines the treatment's therapeutic effects, in terms of decreases in AS levels, stress, depression, and anxiety, at a longer-term, 14-week follow-up.

Study 5 is a replication and extension of Study 2, examining the process(es) through which the CBT/IE intervention results in decreases in AS. For this study, the IE component of the study is expanded, from 10 IE running trials over 10 weeks, to 42 IE trials over a 14-week period. Changes in the cognitive, affective, and somatic responses to the IE are examined for high and low AS participants. The relationship between changes in reactions to the IE and changes in AS levels is also examined to determine whether changes in the intervention's process indicators are indeed related to changes in treatment outcomes.

Chapter 2 . STUDY 1: WHY DO THEY EXERCISE LESS? BARRIERS TO EXERCISE IN HIGH ANXIETY SENSITIVE WOMEN[1]

Abstract

Anxiety sensitivity (AS; fear of arousal sensations) is a risk factor for mental and physical health problems, including physical inactivity. Because of the many mental and physical health benefits of exercise, it is important to better understand why high AS individuals may be less likely to exercise. The present study's aim was to understand the role of barriers to exercise in explaining lower levels of physical exercise in high AS individuals. Participants were undergraduate women who were selected as high (N = 82) or low (N = 72) AS. High AS women participated in less physical exercise and perceived themselves as less fit than low AS women. Mediation analyses revealed that barriers to exercise accounted for the inverse relationships between AS group and physical exercise / fitness levels. Findings suggest that efforts to increase physical exercise in at-risk populations, such as high AS individuals, should not focus exclusively on benefits to exercise, but should also target reasons why these individuals are exercising less.

Introduction

Anxiety sensitivity (AS) is the fear of arousal-related sensations that arises from beliefs that these sensations have harmful physical, psychological, or social consequences (Reiss, 1991). For example, individuals with high levels of AS are more likely to believe that a racing heartbeat is an indicator that they are going to have a heart attack.

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[1]Adapted from *Cognitive Behaviour Therapy, 40,* Sabourin, Hilchey, Lefaivre, Watt, & Stewart "Why do they exercise less? Barriers to exercise in high anxiety sensitive women" 206-215, Copyright (2011) with permission from Taylor & Francis (see Appendix K). As the first author of this article, I contributed to the design of the study, participated in mass screening data collection, organized and managed participant recruitment, collected data, conducted the data analyses, wrote the manuscript, and revised the manuscript in accordance with suggestions from my co-authors, the peer reviewers, and the journal editor.

Conversely, individuals with lower levels of AS may perceive these sensations to be unpleasant, but are able to recognize their generally harmless and transient nature. High AS is a risk factor for anxiety disorders, including panic disorder and other mental and physical health issues, such as substance use disorders and chronic pain (Asmundson, 1999; Stewart, Knize, & Pihl, 1992). Research also suggests that elevated AS levels are associated with lower amounts of physical exercise, especially strenuous exercise, and lower physical fitness levels (Goodin et al., 2009; McWilliams & Asmundson, 2001; Smits & Zvolensky, 2006). Given the physical (for reviews, see Penedo & Dahn; 2006; Warburton, Nicol, & Bredin, 2006) and mental (for review see Stathopoulou, Powers, Berry, Smits, & Otto, 2006) health benefits associated with physical activity participation, it is important to identify factors that may deter high AS individuals from participating in physical exercise.

Previous research identified the decisional balance construct as useful in understanding determinants of health behaviour change (Prochaska et al., 1994). The decisional balance process involves weighing the advantages (i.e., pros, benefits) and disadvantages (i.e., cons, barriers) of a particular behaviour (e.g., exercise involvement) as a way to predict an individual’s adoption of the behaviour (Prochaska & Marcus, 1994). Applying the decisional balance process to exercise reveals that individuals who are physically active have higher decisional balance scores (i.e., benefits minus barriers) for exercise relative to those who are physically inactive (Marshall & Biddle, 2001; Williams, Lewis, et al., 2008). Furthermore, when measured longitudinally, decisional balance scores at baseline predict exercise status six months later (Williams, Lewis, et al., 2008). The decisional balance construct is therefore relevant in understanding exercise behaviours.

Because high AS individuals tend to engage in less exercise than low AS individuals (e.g., Goodin et al., 2009), and because exercise levels are positively associated with net decisional balance scores (e.g. Williams, Lewis, et al., 2008), it would be expected that high AS individuals would have a lower net decisional balance score for exercise than low AS individuals, as they would endorse fewer benefits and more barriers to exercise as compared to low AS individuals. There has been very little research, however, examining the relationship between these decisional balance constructs, AS levels, and exercise.

Goals and Hypotheses of the Present Study

The present study was conducted to better understand why high AS women engage in less physical activity and are less physically fit than low AS women by examining the role of the decisional balance process (i.e., evaluating benefits versus barriers to exercise). Specifically, we sought to determine whether net decisional balance scores (i.e., benefits - barriers), and also barriers and benefits separately, accounted for (i.e., mediated) the expected inverse relationships between AS and both physical activity and fitness levels. Women only were selected for study based on past research that suggests that a) women report higher levels of AS than men (Stewart, Taylor, & Baker, 1997), b) AS levels are more closely associated with psychopathology for women than for men (Olatunji & Wolitzky-Taylor, 2009), and c) women tend to report lower exercise levels than men (Huang et al., 2003).

First, it was hypothesized that high AS women would self-report lower levels of physical activity and fitness than low AS women. Second, women with high AS were expected to report lower net decisional balance scores, more barriers, and fewer benefits to exercise than low AS women. Third, higher net decisional balance scores and benefits would be associated with higher levels of exercise and fitness, whereas barriers would be associated with lower levels of exercise and fitness. Finally, it was expected that net decisional balance scores, barriers, and benefits to exercise, would each account for (i.e., mediate) the expected inverse relationships between AS and self-reported exercise and fitness levels.

Method

Participants

Female participants (N = 154) were recruited from three undergraduate psychology participant pools in eastern Canada, based on Anxiety Sensitivity Index (ASI; Peterson & Reiss, 1992; see Appendix A for selected items) scores collected at mass screenings. Women who scored one standard deviation above or below the mean ASI score for university women (i.e., 18±8; Watt, Stewart, Lefaivre, & Uman, 2006) were invited to participate in a larger AS reduction intervention study, of which this study was a part. Participants were categorized as high AS (N = 82; M [SD] ASI = 35.0 [7.3]) and low AS (N = 72; M [SD] ASI = 8.5 [2.0]). In order to qualify for the larger AS intervention study, women had to be available to attend three 1-hour sessions. In addition, women were screened to exclude all who were not able to participate in the running component of the experimental arm of the study based on physical or health reasons (e.g., cardiac disease). Participants' mean age was 19 years old (*SD* = 2.2); 86% were Caucasian.

Measures

Anxiety Sensitivity Index (ASI; Peterson & Reiss, 1992). The ASI is a 16-item self-report questionnaire designed to assess levels of AS (see Appendix A for selected items). Each item is rated on a 5-point Likert scale ranging from 0 (very little) to 4 (very much). The measure has strong test-retest reliability, construct validity, and criterion-related validity (Peterson & Reiss, 1992). Internal consistency of the ASI was excellent in the present study (alpha = .91).

Physical Activity Measure (PAM; Lefaivre, 2010). The PAM is a 14-item questionnaire that assesses participation in a range of physical activities, including low intensity (e.g., walking), moderate (e.g., swimming) and vigorous (e.g., running) physical activity (see Appendix B). The distinction between these three levels of physical activity was based on definitions used in the literature (see Godin & Shepherd, 1985; Health Canada & Public Health Agency of Canada, 1998). Participants self-report their frequency and duration of each category of physical activity during the past three months. The three self-report categories were combined for the present study in order to assess total exercise participation. Average monthly frequency was multiplied by average duration of a typical exercise session to yield a total monthly exercise participation score.

Perceived Fitness Scale. Participants were asked to rate their perceived level of physical fitness on an author-compiled single-item scale ranging from 0 (no activity) to 10 (Olympic calibre fitness level; see Appendix C). Scores on the measure were normally distributed (skewness = -.06, *SE* = .19, N.S.; kurtosis = -.646, *SE* = .14, N.S.) with endorsed values ranging from 0 to 10. Also, the scale demonstrated good convergent validity, correlating moderately to strongly (Cohen, 1992) with physical activity in both low AS, $r = .47$, $p < .001$, and high AS, $r = .43$, $p < .001$, participants. Other studies (e.g., McWilliams & Asmundson, 2001) have also used single-item scales to assess physical fitness levels.

Decisional Balance Scale (DBS; Marcus, Rakowsky, & Rossi, 1992). The DBS is a 16-item self-report questionnaire designed to assess the perceived benefits and barriers associated with physical activity participation (see Appendix D). Participants are asked to rate the importance of each statement in their decision of whether or not to be physically active using a 5-point scale ranging from 1 (not at all important) to 5 (extremely important). The scale consists of ten benefits (e.g., “I would feel more confident if I were regularly physically active”; “I would have more energy for my family and friends if I were regularly physically active”) and six barriers (e.g., “I feel uncomfortable when I am physically active because I get out of breath and my heart beats very fast”; “Regular physical exercise would take too much of my time”) to exercise. A net score on the DBS was calculated by subtracting average barriers from average benefits. The DBS was found to have satisfactory to high internal consistency values, and to correlate with stage of change (Marcus et al., 1992). The present study also found good to excellent internal consistency for the DBS (alphas = .91 for benefits and .80 for barriers).

Procedure

Participants completed the aforementioned measures as part of a larger AS reduction intervention study’s pre-treatment testing battery. After participants read and signed informed consent forms, two clinical psychology graduate students administered the questionnaires. The study was approved by the Research Ethics Boards of all three universities involved.

Data Analytic Plan

Mediation analyses tested whether DBS constructs (i.e., the net scores [benefits minus barriers]; benefits, and barriers) accounted for the hypothesized inverse relationships between AS and physical exercise / physical fitness. Figure 2-1 depicts one of the hypothesized mediational pathways with barriers to exercise mediating the inverse relationship between AS group and physical exercise. For each outcome, three separate mediation analyses were conducted examining net scores, benefits, and barriers to exercise as mediators, respectively. In order to show mediation, four conditions must be met, which were tested with a series of linear regressions (Baron & Kenny, 1986). First, a significant relationship must exist between the predictor (i.e., AS group) and the outcome (i.e., exercise or fitness). Second, a significant relationship must exist between the predictor (i.e., AS group) and the mediator (i.e., DBS constructs). Third, a significant relationship must exist between the mediator (i.e., DBS constructs) and the outcome (i.e., exercise or perceived fitness) even when accounting for the predictor (i.e., AS group). Fourth, when accounting for the mediator (i.e., DBS constructs), the relationship between the predictor (i.e., AS group) and the outcome (i.e., exercise or fitness) is no longer significant when full mediation occurs. When partial mediation occurs, the relationship between the predictor and the outcome is attenuated without being fully eliminated. Sobel tests were also conducted, which tested whether the indirect effects of AS on exercise and fitness via DBS constructs were significantly different from zero (Sobel, 1982).

Results

Descriptive statistics and effect sizes for the differences between high and low AS participants are presented in Table 2-1. The results of mediation analyses are presented in Table 2-2. For mediation analyses, AS was entered as a dichotomous (i.e., high AS vs. low AS) variable. First, mediation analyses were performed on the relationship of AS group to physical exercise. Fulfilling the first requirement of mediation, a regression analysis revealed that high AS group membership predicted lower levels of physical exercise, $R^2 = .04$, $p = .01$ (small to medium effect size).

Next, fulfilling the second requirement of mediation, linear regression analyses revealed that, as expected, high AS group predicted both lower net DBS scores $R^2 = .03$, $p = .03$ (small effect), and increased barriers, $R^2 = .21$, $p < .001$ to exercise (large effect). However, interestingly, high AS group also predicted *increased* benefits, $R^2 = .06$, $p = .002$, to exercise (medium effect). Fulfilling the third requirement of mediation, three linear regression analyses revealed that even after accounting for AS group, higher barriers to exercise were related to decreased levels of exercise, $\Delta R^2 = .08$, $p < .001$, and higher benefits to exercise were related to increased levels of exercise, $\Delta R^2 = .03$, $p < .05$. Also, after accounting for AS group, DBS net scores were related to increased levels of exercise $\Delta R^2 = .11$, $p < .001$.

Finally, to fulfill the fourth requirement of mediation, the linear regression testing whether the DBS net score mediated the relationship between AS group and physical exercise revealed that the relationship was no longer significant, $p > .05$. A Sobel test indicated that the indirect effect was significant, $z = -1.99$, $p < .05$, suggesting that net scores fully mediated the relationship between AS group and exercise. A second regression analysis revealed that after accounting for barriers to exercise, the relationship between AS group and exercise was no longer significant, $p > .05$ The Sobel test also revealed a significant indirect effect, $z = -3.22$, $p = .001$, indicating that barriers to exercise also fully mediated the relationship between AS group and exercise.

AS group was positively associated with benefits of exercising. However, conceptually, one would not test whether it is *because* high AS individuals endorse more benefits to exercise that they exercise less. Therefore, no mediation analyses were performed for benefits.

A separate mediation analysis was performed with fitness levels as the criterion variable (see Table 2-2). AS group was significantly related to lower fitness levels, $R^2 = .09$, $p < .001$ (medium effect). Even after accounting for AS group, higher net scores on the DBS were associated with increased fitness levels $\Delta R^2 = .24$, $p < .001$ and benefits also were associated with increased fitness levels, $\Delta R^2 = .10$, $p < .001$. Barriers, on the other hand were associated with decreased fitness levels, $\Delta R^2 = .14$, $p < .001$. After accounting for DBS net scores, AS group still predicted fitness levels, $p = .003$. The Sobel test conducted with DBS net scores was significant, however, suggesting partial mediation, $z = -2.08$, $p < .05$. When accounting for barriers, AS group no longer predicted fitness, $p = .21$. The Sobel test assessing the indirect effect of AS group on fitness levels through barriers was also significant, $z = -3.95$, $p < .001$, suggesting full mediation. As was the case with exercise, mediation analyses were not conducted for benefits in the case of fitness.

Discussion

The goal of the present study was to examine the role of decisional balance constructs in explaining lower levels of exercise in high AS individuals. First, as expected, AS group was negatively associated with exercise participation, extending to women a previous study's (McWilliams & Asmundson, 2001) finding of this relationship in men. In an attempt to understand this relationship, many forms of exercise (e.g., swimming, running) are likely to induce physiological sensations associated with anxiety (e.g., elevated heart rate, rapid breathing). Previous research suggests that elevated AS levels are also associated with difficulties with another health behaviour, smoking cessation, in part because of the arousal-related sensations produced by nicotine withdrawal (Zvolensky, Bernstein, et al., 2007). Furthermore, it is noteworthy that lower levels of physical exercise in our high AS participants cannot simply be attributed to a higher likelihood of their having health-related problems that preclude them from exercising, as potential participants were excluded if they could not engage in physical exercise for health reasons. Instead, lesser involvement in exercise by high AS versus low AS individuals may represent a form of behavioural avoidance given high AS individuals' fear of arousal sensations.

AS was negatively related to net DBS scores (i.e., benefits minus barriers), and positively related to barriers to exercise. Interestingly, AS was also *positively* related to benefits to exercise. A similar positive relationship between AS levels and perceived benefits to exercise was also found in a recent dissertation study with a mixed gender sample of high school students (Lefaivre, 2010). Moreover, parallels can be drawn from research on smoking cessation where high AS individuals have been shown to endorse more benefits to quitting smoking than low AS individuals (Zvolensky, Vujanovic, et al., 2007). On the other hand, they experience more difficulties quitting and have higher relapse rates than low AS individuals (R. A. Brown, Kahler, Zvolensky, Lejuez, & Ramsey, 2001; Zvolensky, Bernstein, et al., 2007). High AS individuals also report higher levels of barriers to smoking cessation than low AS individuals. It is believed that high AS individuals are more likely to be reactive to the negative consequences associated with quitting smoking, including the bodily sensations (e.g., rapid breathing, dizziness, perspiring palms) associated with nicotine withdrawal that often mimic sensations produced by anxiety and arousal (Zvolensky, Schmidt, & Stewart, 2003). Perhaps because of the high levels of barriers associated with both smoking cessation and exercising for high AS individuals, the acknowledged benefits fail to influence actual behaviour. As net DBS scores for exercising were lower in high AS than in low AS individuals, it can be argued that high AS women actually see fewer benefits to exercise after adjusting for their high levels of barriers to exercise.

Also, as expected, benefits to exercise were positively related to both exercise and fitness levels and barriers to exercise were negatively related to both exercise and fitness levels. Net scores (benefits minus barriers) were also positively related to both exercise and fitness levels. These results are also consistent with previous research (Marshall & Biddle, 2001; Williams, Lewis, et al., 2008).

Mediation analyses revealed that the net decisional balance score, derived by subtracting the barriers from the benefits, mediated the inverse relationship between AS and exercise (full mediation), and between AS and fitness levels (partial mediation). That is, high AS women are less active and less fit than low AS women, in part, because they see relatively few benefits to exercising after considering barriers to exercise. Mediation analyses also revealed that barriers to exercise fully mediated the relationships between AS and exercise avoidance / fitness levels. Thus, for high AS individuals, it appears to be barriers that play a dominant role in decision making regarding exercising. The role of barriers in driving lower exercise levels in high AS (compared with low AS) individuals has several implications for prevention and intervention of anxiety and other mental health difficulties. First, high AS individuals may be missing some of the many mental health benefits of exercising (Stathopoulou et al., 2006). Because this group of individuals is already at increased risk for psychopathology (Naragon-Gainey, 2010), the importance of obtaining such benefits is even greater. Thus, health promotion efforts for high AS individuals should consider incorporating exercise.

Second, efforts to increase physical activity participation in these individuals should focus more on addressing barriers to exercise rather than on promoting the benefits of exercising. High AS individuals are even more aware than their low AS counterparts of the benefits of exercising, yet they still engage in less physical exercise. One of the potential barriers to address is the fear of the arousal related sensations experienced by high AS individuals.

Previous studies suggest that repeated exposure to arousal-related sensations (i.e., interoceptive exposure; Stewart & Watt, 2008) decreases AS levels in patients with panic disorder (Arntz, 2002) and in non-clinical university students with high AS (Forsyth, Lejuez, & Finlay, 2000). Exercise, particularly higher-intensity exercise, is also effective in decreasing AS levels (Broman-Fulks et al., 2004). More recently, running, which also produces arousal-related sensations, was used as the interoceptive exposure component of a brief CBT for reducing AS in high AS undergraduate women (Watt, Stewart, Birch, & Bernier, 2006). The combination of the brief CBT and interoceptive exposure component of ten 10-minute sessions of running led to decreases in AS (Watt, Stewart, Birch, & Bernier, 2006) and in fearful responses to arousal sensations elicited by exercise (Sabourin et al., 2008; see Study 2 in Chapter 4 of this dissertation). Thus, exposure strategies that incorporate physical exercise appear to be viable and effective for reducing AS levels.

In order to further explore the types of barriers that are most likely to affect high AS individuals, future studies could include more extensive measures of barriers than the DBS. For example, the Benefits and Barriers to Exercise scale (Myers & Roth, 1997) contains 24 barrier items that are classified into four separate factors: time-effort, physical effects, social, and specific obstacles. It is possible that high AS individuals' exercise avoidance is driven more by internal types of barriers, such as feeling uncomfortable with the physical effects of exercise (e.g., elevated heart rate), or being embarrassed of displaying these physical effects in public (e.g., perspiring), than by other types of barriers (e.g., no convenient place to exercise; see Lefaivre, 2010). More refined measures of barriers to exercise would help determine which specific types of barriers to address when targeting intervention efforts for high AS individuals.

One of the limitations of the present study was its cross sectional design, which prevented determination of the direction of the AS / exercise relationship. That is, the present study's design precludes distinguishing whether higher levels of AS result in lower levels of exercise participation, or whether low exercise participation results in higher levels of AS through missed opportunities to experience arousal-related sensations. Regardless of the order of origin, however, exercise avoidance in high AS individuals is likely to serve as a maintenance factor for elevated AS levels.

The retrospective, self-report nature of the measures is another limitation of the present study. Participants relied on memory of the previous three months to estimate the amount of exercise they engaged in, introducing potential memory bias. In addition, although the PAM (Lefaivre, 2010) is similar to other measures of physical activities (e.g., Godin & Shepherd, 1985), the psychometric properties of this measure remain unknown. Unfortunately, there is currently no gold standard to measure physical exercise participation over an extended period of time. Fitness levels were also assessed subjectively, instead of objectively (e.g., maximal oxygen consumption; cf. Broocks et al., 1997); however, previous research suggests that individuals, especially young women, are fairly accurate at self-reporting fitness levels (Germain & Hausenblas, 2005). Future research could benefit from more in-the-moment assessments of physical activity and exertion levels, possibly through ecological momentary assessment methodologies using heart rate monitors and accelerometers (c.f., Dunton, Whalen, Jamner, Henker, & Floro, 2005), to validate retrospective reporting. More objective assessments of exertion levels could also elucidate whether high AS individuals specifically avoid higher intensity exercise (e.g., resulting in more pronounced heart rate elevations), which possibly more closely mimics anxiety-related sensations. Obtaining more specific information about exercise avoidance patterns in high AS individuals could also help tailor exercise promotion efforts.

Finally, because this study was conducted with undergraduate women, results may not be generalizable to men or to clinical samples. On the other hand, the mean AS level observed in the present high AS sample (ASI score = 35.5) is comparable to AS levels found in individuals with panic disorder (e.g., Meuret, Rosenfield, Seidel, Bhaskara, & Hofmann, 2010; *M* = 35-36). Also, because women experience higher levels of AS (Stewart et al., 1997) and participate in less exercise than men (Huang et al., 2003), and because AS levels are more closely related to psychopathology in women than in men (Olatunji & Wolitzky-Taylor, 2009), focusing on women was a good starting point for the present study. Future research could further examine sex differences in the relationships between decisional balance constructs, AS, and exercise participation/fitness. Future research could also explore this relationship specifically for high AS individuals with panic disorder or other forms of anxiety-related psychopathology.

In conclusion, the present study found that high AS women engage in less physical exercise and are less physically fit than low AS women. The present study also established that their lower exercise / perceived fitness levels can be attributed, in part, to high AS women's elevated levels of perceived barriers to exercise. Given the well-documented mental (Stathopoulou et al., 2006) and physical (Penedo & Dahn, 2005; Warburton et al., 2006) health benefits of exercise, it is important to find interventions that encourage exercise participation. The present study provided an important step in this direction by gaining a better understanding of why certain individuals might avoid exercising.

Table 2-1.

Means and standard deviations (SD) for exercise variables and Decisional Balance Scale (DBS)

	High AS Mean (SD)	Low AS Mean (SD)	Cohen's *d*
Physical exercise	104.0(115.8)	164.7 (175.9)**	0.4
Perceived fitness	4.6 (2.0)	5.8 (2.0)***	0.6
DBS net scores	1.5 (1.0)	1.9 (1.0)*	0.4
DBS barriers	2.6 (0.8)	1.8 (0.6)***	1.0
DBS benefits	4.1 (0.6)	3.7 (0.9)**	0.5

Note. AS = anxiety sensitivity; DBS net scores = benefits minus barriers. Effect sizes were calculated using Cohen's *d* and categorized as small (d = 0.2), medium (d = 0.5) and large (d = 0.8), respectively (Cohen, 1992).
* $p < .05$; ** $p \leq .01$; *** $p < .001$.

Table 2-2.

Unstandardized regression coefficients (and standard errors) for mediation tests of the effect of AS group on exercise behaviours and fitness levels.

Pred	Med	Outcome	$c(se_c)$	$a(se_a)$	$b(se_b)$	$c'(se_{c'})$
AS	net	exer	-60.74(23.89)**	-0.37(.17)**	49.03(11.09)***	-43.39(22.85)
AS	barriers	exer	-60.74(23.89)**	0.74(0.12)***	-60.61(16.03)***	-15.04(25.89)
AS	benefits	exer	-60.74(23.89)**	0.38(0.12)**	37.08(16.15)*	
AS	net	fit	-1.23(0.32)***	-0.37(.17)**	0.99(.14)***	-0.86(.28)**
AS	barriers	fit	-1.23(0.32)***	0.74(0.12)***	-1.08(0.21)*	-0.43(.34)
AS	benefits	fit	-1.23(0.32)***	0.38(0.12)**	0.87(0.21)***	

Note. AS = anxiety sensitivity group; Pred = predictor; Med = mediator; se = standard error; barriers = barriers subscale of DBS; benefits = benefits subscale of DBS; net = net score (i.e., benefits – barriers) of the DBS; exer = physical exercise; fit = fitness level; path a is the effect of AS group on the DBS subscales; path b is the effect of the DBS subscales with AS group included as predictor on the dependent variable (i.e., exercise / fitness); path c is the unmediated effect of AS group on the dependent variable; path c' is the effect of AS group on the dependent variable as mediated by DBS subscales
* $p < .05$; ** $p \leq .01$; *** $p < .001$

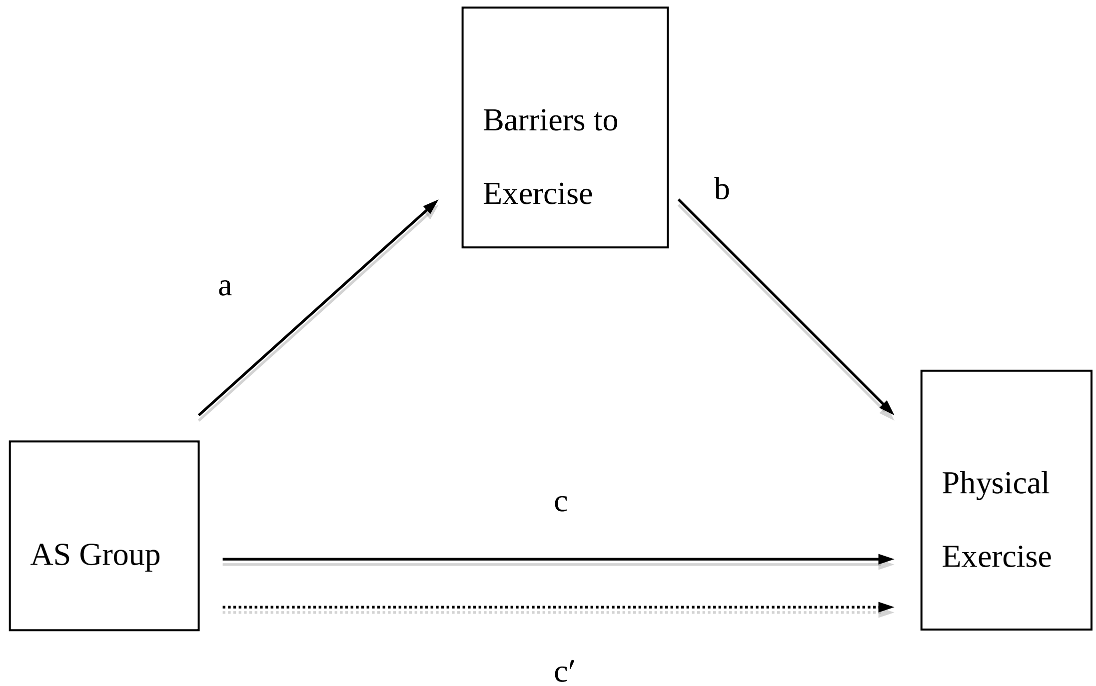

Figure 2-1. A model of the mediational effect of barriers to exercise on the relationship between AS group and physical exercise.
The relationship between AS group and physical exercise is represented by c; the relationship between AS group and barriers to exercise by a; and the relationship between barriers to exercise and physical exercise (controlling for AS group) by b. Path c′ is the effect of AS group on physical exercise as mediated by barriers to exercise. AS = anxiety sensitivity

Chapter 3 . PROLOGUE TO STUDY 2

Study 1 demonstrated that high AS women participate in less physical exercise than low AS women, a difference that is at least partly due to higher levels of barriers to exercise in high versus low AS women. One candidate barrier to exercise lies in the fear of physiological arousal that characterizes AS. That is, it is possible that high AS women avoid exercising in part because they feel uncomfortable with the sensations induced by physical exercise.

A recent study (Watt et al., 2008) combined a brief group CBT intervention for decreasing AS levels with a novel IE component of physical exercise (i.e., running). The goal of the IE portion of the intervention was to decrease fear of physiological arousal. Participants first ran as a group for 10 minutes, and then were instructed to run individually for 10 minutes, 10 times over the following 10 weeks. They were also instructed to complete the HVQ immediately following every running trial, focusing on the sensations they were experiencing while they were running. They were also taught to measure their pulse rate, and instructed to record their pulse rates before starting to run, after 5 minutes of running, and again at the end of the 10-minute run. Watt et al. (2008), however, did not examine changes in reactions to running over time, to determine whether reactions to the running trials were indeed initially higher for high AS individuals, and if these decreased over time.

Study 2 of the current dissertation consists of an analysis of the data collected during the IE portion of the intervention. One of the main goals of Study 2 was to examine patterns of change in participants' cognitive catastrophizing about, fearful reactions to, and somatic sensations arising from, the running trials. Changes in heart rate responsivity across trials were also examined. Specifically, Study 2 aimed to determine if high AS participants had higher reactivity to running than low AS participants, and if changes in reactivity were specific to high AS participants. Reducing negative reactivity to physiological arousal would potentially address one of the barriers preventing high AS women from experiencing the benefits associated with participating in physical exercise.

Chapter 4 . STUDY 2: PHYSICAL EXERCISE AS INTEROCEPTIVE EXPOSURE WITHIN A BRIEF COGNITIVE BEHAVIOURAL TREATMENT FOR ANXIETY SENSITIVE WOMEN[2]

Abstract

A brief cognitive behavioural 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.

Introduction

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). High AS is implicated in the development and

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[2]Adapted from *Journal of Cognitive Psychotherapy: An International Quarterly, 22,* Sabourin, Stewart, Sherry, Watt, Wald, & Grant, "Physical exercise as interoceptive exposure within a brief cognitive behavioural treatment for anxiety sensitive women", (303-320). Copyright (2008) with permission from Springer Publishing Company (see Appendix K). As first author of this article, I contributed to the data analytic plan, participated in data analysis, wrote most of the manuscript, and revised the manuscript in accordance with suggestions from my coauthors, the peer-reviewers, and the journal editor.

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

High 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, which 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 Behavioural Therapy and Interoceptive Exposure in Anxiety Treatment

Previous studies suggest that cognitive behavioural 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; J. G. Beck, & Shipherd, 1997; Penava, Otto, Maki, & Pollack, 1998; Telch, Schmidt, Jaimez, Jacquin, & Harrington, 1995) and in non-clinical 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 also have 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 behavioural 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 favourable to cognitive restructuring, with no differences in drop-out 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 re-appraise these threatening cues (e.g., learning that the sensations are harmless) – an opportunity they missed when they were avoiding arousal-inducing activities (J. G. Beck & Shipherd, 1997; J. G. 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 non-clinical samples (Broman-Fulks, Berman, Rabian, & Webster, 2004; Smits et al., 2008). Broman-Fulks et al. 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 60-90% of their pre-determined age-related maximal heart rates. Low intensity exercise consisted of walking at a speed (i.e., approximately one 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 six-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 five 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 ten weeks post-intervention; these have been reported previously (see Watt, Stewart, Birch, & Bernier, 2006, Watt, Stewart, Lefaivre, & Uman, 2006; Watt et al., 2008). Pre- to post-intervention changes in ASI scores for participants in the CBT condition were 6.7 units for high AS participants and 3.0 units for low AS participants. For the non-specific treatment (NST) control condition, change scores were 4.9 and 3.9 units for high AS and low AS participants, respectively. As predicted, there was a significant interaction between AS group (high AS, low AS) 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 high AS and low AS 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, & Uman, 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) has 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 above 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 pre-intervention, post-intervention, 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-level 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, & Bernier, 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 AS 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 AS and low AS groups were 34.16 (6.37) and 8.33 (3.58), respectively (as reported in Watt, Stewart, Birch, & Bernier, 2006). The two groups did not differ significantly on age, year of study, or race (see Watt, Stewart, Lefaivre, & Uman, 2006). Participants within each AS group were randomly assigned to either the CBT or non-specific treatment (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[3]. 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) x 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 , $p > .05$, were significant, revealing that neither participants in the high AS group nor in the low AS group who handed in the questionnaires differed in pre-treatment 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 Caucasian (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 race, $\chi^2(1, N = 112) = .48$, $p > .05$.

[3] 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.

Measures

Anxiety Sensitivity Index. (ASI; Peterson & Reiss, 1992). The ASI 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 (see Appendix A for selected items). Participants are asked to rate each item on a 5-point Likert 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 pre-treatment ASI score for the 48 participants in the present process study showed excellent internal consistency (Cronbach's alpha = .91).

Hyperventilation Questionnaire (HVQ; Rapee & Medoro 1994). The HVQ is a self-report questionnaire that measures responses to physiological arousal induction challenges (see Appendix E). The version of the HVQ scale used in this study contains 6 items assessing affective reactions (e.g., nervousness), 6 items assessing cognitive reactions (e.g., feeling of losing control), and 18 items assessing somatic reactions (e.g., breathlessness) to the running exercises[4]. Each symptom is rated on a four-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 to18 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 alpha's = .88 for affective, .80 for cognitive, .92 for somatic;

[4] One reverse-scored item from the original 7 item affective scale (i.e., relaxation) was removed as it negatively impacted internal reliability.

Rapee & Medoro, 1994). Also, 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 three 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 over-focus 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 (D. M. 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., 2007), 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 pre-running (i.e., resting) pulse rate, ran together as a group for five minutes, and then calculated their pulse rate for a second time. They immediately ran again for an additional five 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) that 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 five 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 pre-treatment, post-treatment, and at the 10-week follow-up. For the purpose of the present process study, only the pre-treatment 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) x 2 (time: pre-intervention, 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 x group interaction, $F(1, 45) = 20.24$, $p < .001$. Simple effects analyses 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 low AS participants, F (1, 26) = 7.40, $p < .05$, $\eta_p^2 = .22$.[5]

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 pre-running, after five minutes, and after 10 minutes of running were analyzed using a 2 (AS group: high, low) x 3 (phase: pre-running, 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 x 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 for 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. Pair-wise post hoc tests were conducted for both high AS and low AS participants. All pair-wise comparisons were significant at $p < .001$. Further, high AS and low AS groups did not differ on pre-running 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

[5] 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, 2006). 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: pre-treatment 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).

physiological reactivity for high AS compared with low AS participants (see Table 4-1). 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 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 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 x 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 is provided below.

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 4-2, 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 4-1A 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 grey 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 4-2 and Figure 4-2A). 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 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[6].

Cognitive reactions. Figure 4-1B displays the trajectories of cognitive reactions to IE trials for individuals within both high AS 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

[6] Figure 4-1A indicates the presence of a potential outlier in the high AS group. Appendix F contains a re-analysis of affective reactions to running after removal of this participant. The findings reported in the main body of the thesis persist following removal of this single participant.

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 of change in cognitive reactions across IE trials was marginally significant (see panel 2 in Table 4-2 and Figure 4-2B). 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 4-1C displays the trajectories of subjective somatic reactions to IE trials for individuals within both high AS 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.

High AS participants also had significantly higher levels of somatic reactions in response to the first IE trial than low AS participants. AS group, however, did not significantly moderate the linear rate of change in somatic reactions across IE trials (see panel 3 in Table 4-2 and Figure 4-2C). 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 4-1D displays the trajectories of physiological reactivity[7] to IE trials for individuals within both high AS 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 4-2 and Figure 4-2D). 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, & Bernier, 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

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[7] Physiological reactivity refers to changes in pulse rate as a result of engaging in the running trial. Specifically, the resting pulse rate calculated prior to beginning the running IE trial was subtracted from the average of the two pulse rates taken after five and 10 minutes of running, respectively.

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 (J. G. Beck & Shipherd, 1997; J. G. Beck, Shipherd, & Zebb, 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. Also, 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 re-interpretation 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 re-interpret 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, nor would it 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. Also, 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, 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 which is 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, the above possibilities can only be 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, 1986). 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-Caucasians, non-university students, and clinical populations.

Results from the current study are consistent with both the theorized cognitive and habituation pathways (J. G. Beck & Shipherd, 1997; J. G. Beck, Shipherd, & Zebb, 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 that includes a larger number of IE trials, and a larger sample, would help tease out these alternative theoretical explanations. With additional IE exposures it would 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, 2006), 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 processes of change during therapeutic interventions (e.g., Bouchard et al., 2007). Additional IE trials and assessing participants' engagement in other physical activities could also assess for 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 now to have far-reaching mental health benefits (e.g., Folkins & Sime, 1981; see Stathopoulou et al. 2006 for a review), and running is a cost-effective and flexible form of physical exercise.

Table 4-1.

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

	Low AS	High AS	*F*
Pre-running	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. AS = anxiety sensitivity
*$p = .05$

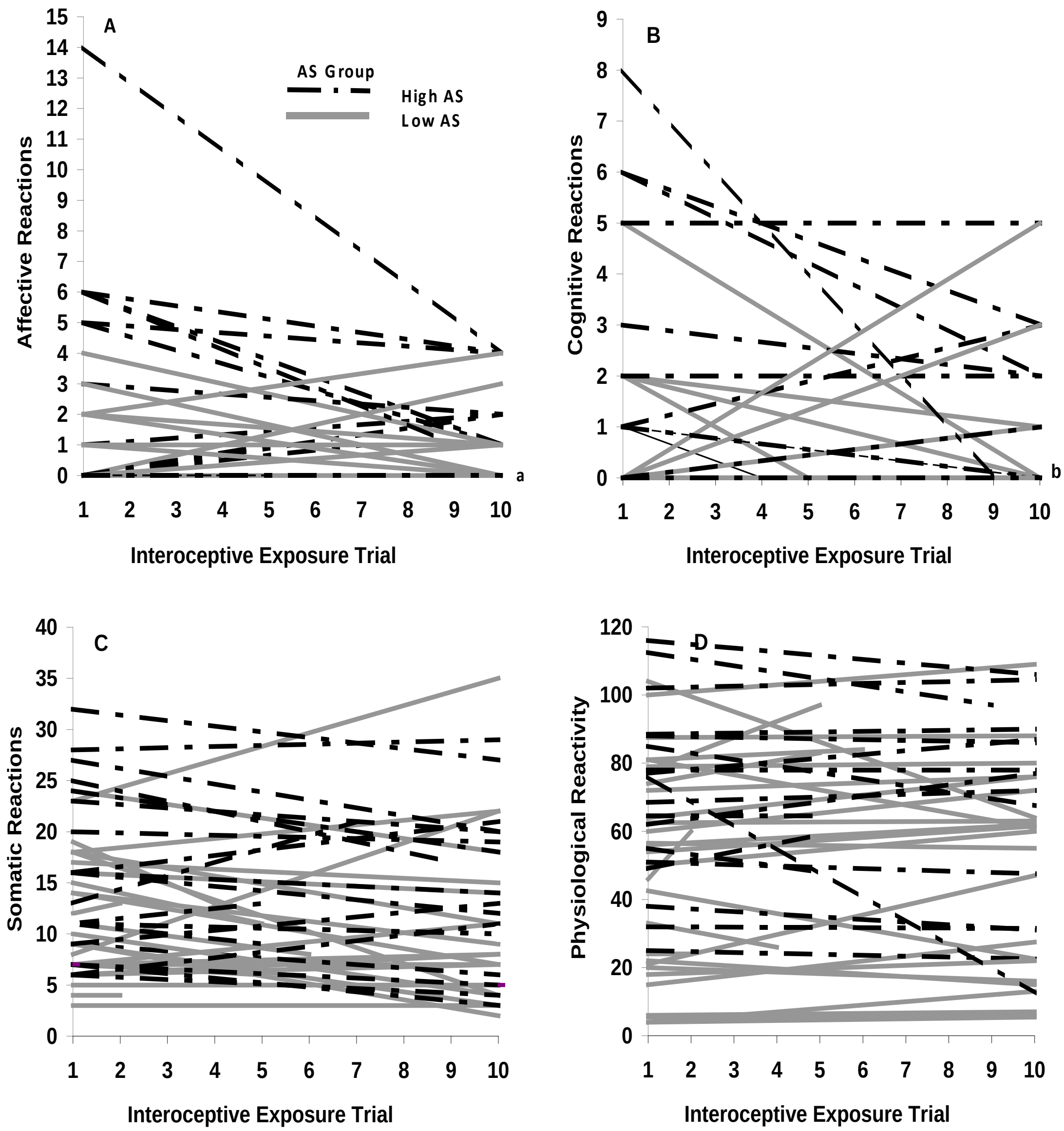
Table 4-2.

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

Predictor	Fixed effects		Random effects
	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 x 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 x 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 x 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 x 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.

†$p < .10$. *$p < .05$. **$p < .01$. ***$p < .001$.



[a] The line that spans the horizontal axis, reflecting scores of "0" on interoceptive trials represents 7 High AS participants and 18 Low AS participants

[b] The line that spans the horizontal axis, reflecting scores of "0" on interoceptive trials represents 9 High AS participants and 20 Low AS participants

Figure 4-1. Individual trajectories for affective, cognitive, and somatic reactions, and physiological reactivity for high AS and low AS participants across interoceptive exposure trials. Physiological reactivity was calculated by subtracting the resting pulse rate from the average of the two pulse rates taken after five and 10 minutes of running, respectively.

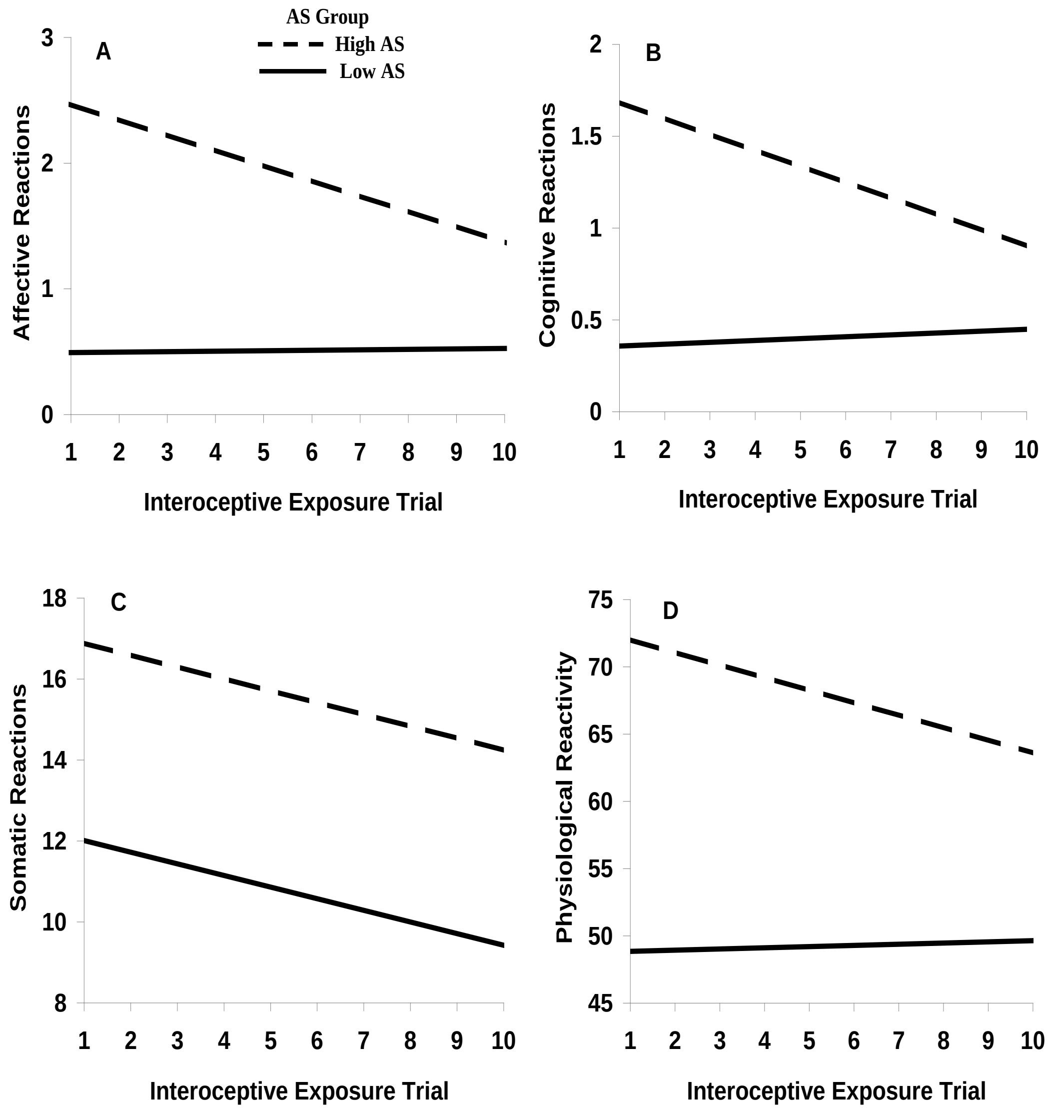


Figure 4-2. Trajectories for affective, cognitive, somatic reactions, and physiological reactivity for high and low AS participants across interoceptive exposure trials

Chapter 5 . PROLOGUE TO STUDY 3

Study 2 demonstrated that repeated exposure to running as an IE component of a brief CBT, AS reduction intervention resulted in decreases in affective and cognitive reactions to running specifically for high AS participants. Because of the theoretical importance of IE in reducing AS levels, a follow-up CBT/IE intervention study (Studies 4 and 5) included an expanded IE component, from 10 running trials over a 10-week period, to 42 running trials over a 14-week period.

The HVQ used to assess reactions to running in Study 2 is a valuable tool to measure different dimensions of reactivity to physiological arousal. At 33 items, however, the measure can be lengthy when completed repeatedly. The goal of Study 3 of the current dissertation was to develop an abbreviated form of the HVQ, the HVQ-Brief (HVQ-B) in order to facilitate its use in Study 5. Additionally, assessing reactions to physiological arousal can provide vital information on the processes involved in the development, maintenance, and/or treatment of AS and related conditions. Having a brief version of the HVQ increases the feasibility of examining different dimensions of responses to physiological arousal across basic research, treatment research, and/or clinical treatment settings.

Chapter 6 . STUDY 3: DEVELOPMENT AND PSYCHOMETRIC EVALUATION OF A BRIEF VERSION OF THE HYPERVENTILATION QUESTIONNAIRE: THE HVQ-B[8]

Abstract

The fear of arousal sensations characterizes some anxiety disorders and is a core feature of an established risk factor for anxiety and related disorders (i.e., anxiety sensitivity; Taylor, 1999). Anxiety sensitivity (AS) refers to a fear of anxiety-related bodily sensations stemming from beliefs that these have catastrophic consequences. Interoceptive exposure (IE; repeated exposure to feared arousal sensations) has been shown to decrease AS. The 33-item Hyperventilation Questionnaire (HVQ; Rapee & Medoro, 1994) measures state levels of cognitive, affective, and somatic responses to IE and arousal induction exercises more generally. The aim of the present set of studies was to develop and evaluate a brief version of the HVQ, the HVQ-B, in order to facilitate its use in research and clinical settings. In Study 3A, three existing data sets that used the long version of the HVQ were combined to select the items to be retained for the HVQ-B. In Study 3B, the 18-item HVQ-B was administered and its psychometric properties were evaluated. Finally, in Study 3C, a confirmatory factor analysis (CFA) was performed on the 18-items of the HVQ-B. The HVQ-B demonstrated excellent psychometric properties, and accounted for most of the variance of the questionnaire's longer version. CFA indicated a reasonably good fit of the three-factor measurement model. Finally, the HVQ-B was able to distinguish between responses to arousal induction exercises by high versus low AS participants. The HVQ-B is a useful tool to

[8] Adapted from Sabourin, Stewart, Watt, & MacDonald (2012) "Development and Psychometric Evaluation of a Brief Version of the Hyperventilation Questionnaire: The HVQ-B". Manuscript re-submitted for publication. As the first author of this article, I collected the data for Study B, conducted all data analyses, wrote the manuscript, and revised the manuscript in accordance with suggestions from my co-authors, the peer reviewers, and the journal editor.

assess cognitive, affective, and somatic responsivity to arousal sensations in both research and practice.

Introduction

The fear of arousal sensations characterizes some anxiety disorders (e.g., panic disorder; Barlow, 2002) and is a core feature of a well studied risk factor for anxiety and related disorders (i.e., anxiety sensitivity; Taylor, 1999). Anxiety sensitivity (AS) refers to a fear that anxiety-related bodily sensations portend catastrophic physical, social, or psychological consequences. To better understand AS, panic disorder, and their clinical correlates, research studies use specific procedures that induce physical arousal (i.e., arousal induction exercises) to study fearful responding to these types of sensations (A. B. MacDonald, Baker, Stewart, & Skinner, 2000; Rapee & Medoro, 1994). Arousal sensations have been induced through procedures such as hyperventilation, carbon dioxide inhalation, chair spinning, and physical exercise. In addition, treatment techniques aimed at decreasing fearful responding to arousal-related sensations by exposing individuals repeatedly to these sensations (i.e., interoceptive exposure; IE; Goldstein & Chambless, 1978), have been used successfully with panic-disordered individuals and in non-clinical high AS samples (e.g., Craske, Brown, & Barlow, 1991; Forsyth, Lejuez, & Finlay, 2000).

Traditionally, IE procedures were used in the treatment of panic-related conditions, such as panic disorder. More recently, there has been a surge of interest in novel uses of IE in clinical practice (see Stewart & Watt, 2008 for a discussion on the history of IE). In a recent series of empirical research articles, IE was described for use in treating difficulties as diverse as post traumatic stress disorder, hypochondriasis, and smoking cessation (Wald, 2008; Walker & Furer, 2008; Zvolensky, Yartz, Gregor, Gonzalez, & Bernstein, 2008). There is also an increasing emphasis on better understanding the mechanisms of action driving the benefits of IE, and in better assessing changes derived from repeated exposures to feared sensations (Meuret, Ritz, Wilhelm, & Roth, 2005; Stewart & Watt, 2008).

Theoretically, exposure to feared anxiety sensations might exert its fear-reducing effects through one of at least two mechanisms. First, a conditioning model posits that fear is a learned response. Repeated exposure to the feared stimuli (e.g., arousal sensations), without the accompanying feared catastrophic consequences (e.g., panic attack, heart attack), would lead to extinction of the feared stimuli through a process of new learning (Moscovitch, Antony, & Swinson, 2009). Alternatively, a cognitive model posits that exposure to feared sensations might serve to alter how these sensations are interpreted, leading to a re-evaluation of the meaning of these sensations as less threatening (Chambless & Goldstein, 1981). Empirical evidence confirming one of these potential mechanisms over the other, however, has yet to emerge (Moscovitch et al., 2009; Stewart & Watt, 2008). Future research employing well-designed longitudinal studies specifically examining potential process markers is needed to further discern mechanisms of action of exposure-based therapy for fear of arousal sensations.

Given the recent diversification of IE uses and increased interest in better understanding processes associated with IE, measuring different dimensions of responses (i.e., cognitive, affective, somatic) to arousal induction has become more essential than ever. Also, because changes in AS levels have been postulated as important drivers of clinical improvements in panic and other anxiety-related disorders (Smits, Powers, Cho, & Telch, 2004), responses on these measures should be sensitive to varying AS levels.

The Hyperventilation Questionnaire (HVQ; Rapee & Medoro, 1994) is a 33-item self-report measure designed to assess an individual's responses to arousal induction exercises. Participants respond to each item (e.g., "dizziness", "feeling trapped or helpless") on a 4-point Likert scale ranging from "not at all" (scored as 0) to "markedly" (scored as 3). The HVQ is divided into three subscales: cognitive (i.e., catastrophic thoughts), affective (i.e., feelings of anxiety), and somatic (i.e., physiological sensations). Although called the "Hyperventilation Questionnaire" because its original use was to assess responses to a lab-based hyperventilation challenge (Rapee & Medoro, 1994), items on the HVQ are appropriate for assessing reactions to a wide range of arousal induction exercises.

The HVQ has successfully differentiated reactions to arousal induction exercises in individuals with varying levels of AS (e.g., Carter, Suchday, & Gore, 2001; A. B. MacDonald et al., 2000; Rapee & Medoro, 1994; Sabourin et al., 2008; see Study 2 in Chapter 4 of the current dissertation). Rapee and Medoro (1994; Study 3) found that high AS participants had more pronounced cognitive, affective, and somatic reactions than low AS participants following hyperventilation. In addition, the HVQ has been used to assess differential responses to arousal after individuals undergo certain experimental manipulations. A. B. MacDonald et al. (2000) used the HVQ to study the effects of alcohol on responses to a hyperventilation challenge in high and low AS university students. Sober high AS participants scored higher on the cognitive and affective subscales than sober low AS participants. In addition, high AS, but not low AS, participants who consumed alcohol scored lower than those who did not on the affective and cognitive subscales following hyperventilation. Thus, responses on the HVQ indicated a cognitive-emotional response dampening effect of alcohol specifically for high AS participants.

Intervention studies have also used the HVQ to track changes following treatments aimed at decreasing fearful responding to arousal. Carter, Marin, and Murrell (1999) investigated the effects of a treatment consisting of repeated exposure to hyperventilation and cognitive restructuring in decreasing fearful responding to arousal in high AS university students. Scores on all three subscales of the HVQ decreased following this intervention. Our own research (Sabourin et al., 2008; see Study 2 in Chapter 4 of the current dissertation) also found that, following a brief cognitive behavioural intervention with an IE component consisting of ten 10-minute running trials, scores on all subscales of the HVQ decreased in a sample of high AS women. On the other hand, only scores on the somatic subscale (e.g. “breathlessness”), but not on the affective (e.g., “nervousness”) and cognitive (e.g., “worrying that your actions are damaging your health”) subscales, decreased for low AS women. These findings suggest that therapeutic effects of IE, in terms of decreasing catastrophic interpretations of and fearful responding to arousal, were specific to high AS women. In summary, the HVQ has proven to be a useful tool for basic and treatment research to assess both initial reactions to arousal induction exercises, as well as changes in these reactions over time with treatment.

The original HVQ, consisting of 33 items, can be taxing, however, when completed several times in treatment outcome research or clinical settings, or within a battery of other measures. The aim of the present set of studies was to develop and evaluate a briefer, and thus less taxing, version of the HVQ (i.e., the HVQ-Brief; HVQ-B), while retaining the original scale's strong psychometric properties (Rapee & Medoro, 1994). In Study 3A, we merged three existing data sets that used a long form of the HVQ to determine the items to retain for the HVQ-B. In Study 3B, we evaluated the psychometric properties of the HVQ-B when administered as an 18-item scale. Finally, in Study 3C, we combined the data from studies one and two to perform a confirmatory factor analysis of the HVQ-B's 18 items.

Study 3A

Method

Participants and Procedure. Three existing data sets that used a long form of the HVQ were combined in developing the HVQ-B. First, A. B. MacDonald et al. (2000; A. B. MacDonald, Stewart, Hutson, Rhyno, & Loughlin, 2001) used the HVQ in two separate studies to assess the effects of alcohol on responses to a hyperventilation task in high and low AS participants. For the present study, only the non-alcohol conditions were used (N = 63) to avoid the confounding effects of alcohol on responses to hyperventilation. Undergraduate students were eligible to participate and categorized as high or low AS if they scored one SD above or below the mean Anxiety Sensitive Index (ASI; Peterson & Reiss, 1992) score for their respective sex (see A. B. MacDonald et al., 2000; 2001 for additional details on participants and design). Scores assessing participants' reactions during the hyperventilation task were used for the present study.

Second, Sabourin et al. (2008; see Study 2 in Chapter 4 of the current dissertation) used the HVQ to assess female high and low AS participants' reactions to another type of arousal-inducing exercise – ten minutes of running. Running was used as the IE component of a brief cognitive behavioural intervention for AS. Participants (N = 41) were recruited from undergraduate psychology subject pools at two universities, and categorized as high or low AS based on scoring one SD above or below the mean female ASI score (see Sabourin et al., 2008, Study 2 in Chapter 4 of the current dissertation for additional information on participants and design). Scores on the HVQ assessing participants' reactions during the initial running trial were used for the present study.

A total of 104 participants (39 low AS / 65 high AS; 18 men / 86 women) were included in the present study. Mean (*SD*) ASI scores were 33.30 (7.36) for high AS and 6.82 (2.89) for low AS participants. Participants ranged in age from 17 to 40 years (*M* = 20.6, *SD* = 3.7).

Measures. *Anxiety Sensitivity Index (ASI; Peterson & Reiss 1992).* The ASI is a widely used 16-item questionnaire designed to assess trait AS levels (see Appendix A for selected items). Participants rate each item (e.g., "It scares me when I feel shaky") on a 5-point Likert scale ranging from "very little" (scored as 0) to "very much" (scored as 4). The measure has shown strong psychometric properties, including internal consistency, test-retest reliability, and criterion and construct validity (Peterson & Reiss, 1992).

Hyperventilation Questionnaire (HVQ; Rapee & Medoro, 1994). The HVQ was described above (see Appendix E). In addition to the measure's construct validity as described above, the three subscales also have good-to-excellent internal consistency (Rapee & Medoro, 1994).

Statistical Analysis. In determining items to retain for the brief measure's three subscales, internal consistency and inter-item correlations were assessed for each subscale separately. Because the cognitive and affective subscales contained only six and seven items respectively, the majority of items were retained to maintain good subscale internal consistency. On the other hand, the longer, 18-item somatic subscale was shortened more substantially. Criteria used in retaining items for this subscale were 1) internal consistency and 2) construct validity. More specifically, to maximize internal consistency, items with higher corrected item-subscale correlations were favoured (cf., von Baeyer, Chambers, & Eakins, 2011). To determine criterion validity, items were subjected to between-subjects t-tests to assess their ability to differentiate between responses in high versus low AS participants. As described above, a measure assessing reactions to physiological arousal should be able to distinguish between responses from individuals who are believed to have a dispositional tendency to respond fearfully to arousal sensations (i.e., high AS individuals) versus individuals who are not (i.e., low AS individuals; Reiss & McNally, 1985).

Results

The original cognitive subscale possessed good internal consistency with an alpha coefficient of .82. Items correlated moderately-to-strongly with each other (rs = .22-.81). Deleting items made little difference to internal consistency, with the deletion of one item increasing the alpha coefficient negligibly to .83. Therefore the six-item cognitive subscale remained unchanged.

The original affective subscale's internal consistency was also good with an alpha coefficient of .84. The reverse-scored "relax" item demonstrated weak correlations with other subscale items (rs = .16-.30). When this item was removed, the alpha coefficient increased noticeably to .88. Other items correlated moderately-to-strongly with each other (*r*s = .40-.69). The item "relaxed" was thus removed, resulting in a six-item affective subscale[9].

The somatic subscale, at 18 items, was reduced to six items to make it consistent in length with the other two subscales. Items with the highest item-subscale correlation that also showed the ability to distinguish between high and low AS participants' responses to the arousal induction exercises were retained (Table 6-1). In addition, the item "Racing heart" was retained, as heart rate has long been considered an important physiological marker of anxiety (e.g., Foa & Kozak, 1986; Liebowitz et al., 1985)[10]. The briefer six-item somatic subscale possessed good reliability with an alpha coefficient of .83. Items correlated moderately to strongly with each other (*r*s = .28 - .62).

The revised 18-item HVQ-B scale accounted for most of the original scale's variance, with correlations between the original and shortened somatic and affective subscales, $r = .93$, $p < .001$, and $r = .97$, $p < .001$, respectively. The cognitive subscale remained unchanged. In addition, univariate ANOVAs revealed that all three HVQ-B subscales differentiated between high and low AS participants' reactions to the arousal induction procedures (Table 6-2).[11]

Study 3B

The HVQ-B was administered in a recent follow-up study to Sabourin et al. (2008; see Study 2 in Chapter 4 of the current dissertation; Sabourin, Stewart, Watt, &

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[9] The item "relaxed" was also removed in the previous study of the current dissertation, which also used the HVQ (i.e., Study 2 in Chapter 4).

[10]The item differentiated between high and low AS participants' responses for one of the tasks (running). Also, the two other items with higher item-total correlations, "Numbness in extremities" and "Buzzing in the head", did not differentiate between high and low AS participants' responses for either task.

[11]Individuals were selected based on scoring one SD above or below mean ASI scores, creating two distinct groups. Upon visual inspection of histograms, however, scores on the HVQ-B appeared to be uni-modally distributed. This is most likely because the HVQ-B assesses a construct similar to, but distinct from, the ASI.

Krigolson, 2012; see study 5 in Chapter 10 of the current dissertation). Whereas in Study 3A, we used the long version of the scale to determine which items to retain for the HVQ-B, in Study 3B we assessed the psychometric properties of the HVQ-B when administered as a stand-alone 18-item measure.

Method

Participants. Participants in the present study consisted of 50 female undergraduate students who were part of a larger follow-up AS intervention study (Sabourin et al., 2012; see Study 5 in Chapter 10 of the current dissertation). Women were selected based on mass screening ASI scores that were one standard deviation above or below mean female ASI scores. Mean (*SD*) scores were 35.8 (6.8) for high AS women (N = 27) and 7.7 (2.4) for low AS women (N = 23). Participants ranged in age from 17 to 34 years (*M* = 18.9, *SD* = 2.5).

Materials and Procedure. Participants ran for a total of 10 minutes on the third day of the brief intervention. Scores on the HVQ-B completed immediately following the IE running trial were used for the current study's analyses.

Results

All three subscales of the HVQ-B possessed good internal consistency with alpha coefficients of .82 for the cognitive, .82 for the affective, and .84 for the somatic subscales. Each item correlated moderately-to-highly with other items on its subscale: inter-item *r*s ranged from .23 - .71 for the cognitive subscale, from .23 - .74 for the somatic subscale and from .24 - .71 for most items of the affective subscale. The items "fear" and "rising agitation" correlated at .09. Both of these items, however, showed moderate to strong correlations with all other items of the affective subscale. Item-subscale correlations were also moderate to high (Table 6-3). As an indication of construct validity, each subscale was able to differentiate between high and low AS participants' reactions to running (Table 6-2). Thus, the HVQ-B scale demonstrated good reliability and construct validity.

Study 3C

Scores on the 18 items that were retained for the HVQ-B were combined from the A. B. MacDonald et al. (2000, 2001) and Sabourin et al. (2008; see Study 2 in Chapter 4 of the current dissertation; Sabourin et al., 2012; see Study 5 in Chapter 10 of the current dissertation) datasets to conduct a confirmatory factor analysis (CFA) of the HVQ-B's hypothesized three-factor structure.

Method

Participants and Measures. The participants for Study 3C have been described in Studies 3A and 3B. Briefly, the study included a total of 156 participants, of which 64 were low AS and 92 were high AS. Most (89%) were women. The 18 items retained for the HVQ-B were used in the present study's analyses.

Results

All CFAs were conducted using EQS (Version 6.1) software (Bentler, 1995). Mardia's normalized coefficient of multivariate kurtosis (Mardia, 1970) indicated violation of multivariate normality. In addition, item kurtoses varied significantly (range = -1.19 to 21.41). Therefore, the geometric mean approach (Bentler, Berkane, & Kano, 1991) to the heterogeneous kurtosis (HK) model estimation was used.

Multiple fit indices were used to determine model goodness of fit. The fit indices used for the present study were: 1) chi-square (χ^2); 2) chi-square divided by degrees of freedom ratio (χ^2/df); 3) comparative fit index (CFI; Bentler, 1990); 4) incremental fit index (IFI; Bollen, 1989); and 5) root mean square error of approximation (RMSEA; Steiger, 1990). There are no absolute cut-off scores to indicate good fitting models. Larger χ^2 values represent poorer fit. A good fitting model should result in a non-significant χ^2, although significance tests are highly dependent on assumption of multivariate normality (e.g., Bentler & Bonett, 1980; McIntosh, 2007). As an alternative indication of fit, the χ^2/df ratio should not exceed three (Kline, 1998). According to traditional rules of thumb, values above .90 for the CFI and IFI indicate an adequate fit (Hu & Bentler, 1995). RMSEA values below .08 represent a good fit, those between .08 - .10 represent an adequate fit, and those above .10 represent a poor fit (MacCallum, Browne, & Sugawara, 1996). Recently, more stringent criteria propose cut-off values of close to .95 for CFI and IFI and close to .06 for RMSEA (Hu & Bentler, 1999) to indicate good fitting models.

Results from the CFA on the three-factor HVQ-B suggest an adequate to good fit of the model (χ^2 = 301.77, $p < .001$, χ^2/df = 2.29, CFI = .93, IFI = .93, RMSEA = .09). Each item loaded significantly on its respective factor (Table 6-4). The LeGrange Multiplier test indicated that: 1) cross-loading the indicator variable “feel like passing out” on both cognitive (its original latent factor) and somatic factors would decrease χ^2 by 23.6 points, and 2) cross-loading this indicator variable on both cognitive and affective factors would decrease χ^2 by 20.4 points. However, no factor items were altered due to the possibility that these findings were attributable to error variance that is unique to the current study’s data. Future research could examine the utility of cross-loading this item.

Factors correlated highly with each other, with *r*s of .77 between the affective and somatic factors, .88 between the cognitive and affective factors, and .97 between the cognitive and somatic factors. Because of concerns that correlations above .90 may be indicative of multicollinearity and redundancy (Tabachnick & Fidell, 2006), the cognitive and somatic factors, were combined to form a 2-factor model which was tested against the original 3-factor model. As the alternative model is nested within the original 3-factor model, a χ^2 difference test was used to assess whether the 3-factor model is a significantly better fit than the more parsimonious 2-factor model. The 2-factor model, consisting of a combined cognitive/somatic factor and the original affective factor, was a significantly poorer fit than the original 3-factor model, $\chi^2_{diff}(2) = 8.65$, $p < .05$. Thus, even given the high correlations between these two factors, it appears that the 3-factor model remains superior to this alternative 2-factor model.[12]

General Discussion

The goal of the present paper was to develop a brief measure to assess state level responses to arousal sensations as they occurred in real time. As Study 3A demonstrated, the 18-item HVQ-B accounted for most of the variance of the original long form HVQ scale from which it was derived. As was shown in Studies 1 and 2, the HVQ-B demonstrated good psychometric properties, with high alpha coefficients and subscale items that were moderately correlated with each other. In addition, each subscale was

[12] As an additional test of the optimal number of factors to retain for the HVQ-B, a principal components analysis (PCA; cf., Woicik, Stewart, Pihl, & Conrod, 2009) was performed. Analysis revealed three factors with eigenvalues greater than one (i.e., 8.4, 1.6, 1.3). Visual examination of the scree plot also indicated a 3-factor solution. Finally, the 3-factor solution accounted for 62.6% of the variance in item scores. These findings further indicate a 3-factor solution for the HVQ-B, consistent with the original HVQ.

able to differentiate responses to arousal induction between low versus high AS individuals.

Confirmatory factor analysis performed in Study 3 indicated that the 3-factor model provides an adequate to good fit with the data. A few potential problematic findings may inform future evaluations and use of the HVQ-B. One of the indicators, the RMSEA, was indicative of a slightly poorer fit than other indicators, and fell just outside of the newer, more conservative acceptable range. RMSEA, however, is susceptible to rejecting good-fitting models when sample size is under 250 (Hu & Bentler, 1999) as with the present data. The other fit indices (e.g., CFI, IFI) at just below .95, suggest a good fitting model.

The three factors of the HVQ-B were highly correlated, with one of the correlations above .90, potentially indicating multicollinearity and redundancy between factors (Tabachnick & Fidell, 2006). On the other hand, the alternative 2-factor model combining the highly correlated cognitive and somatic factors resulted in a less well-fitting model. In addition, a number of previous studies using various experimental manipulations with the original HVQ have shown that participants' response patterns on the cognitive and somatic scales tend to differ, suggesting that they are tapping separable constructs (e.g., Rapee & Medoro , 1994, Study 1; Sabourin et al., 2008; see Study 2 in Chapter 4 of the current dissertation). Furthermore, three highly overlapping but distinct factors is consistent with the cognitive behavioural model of anxiety, which postulates very strong associations between the three components (i.e., physiological sensations, thoughts, feelings) of anxiety-related experiences (e.g. D. A. Clark & Beck, 2010). These three components, although conceptually distinct, might be difficult to separate empirically. Finally, in future work, some of the items from the cognitive subscale could be re-examined, and words such as “feel” or “fear” could be substituted with words that more clearly assess cognitions, such as “worry” or “think”. Such substitutions might decrease the correlations between subscales.

For the current study, two arousal induction tasks (i.e., hyperventilation and running) were used to evaluate the properties of the HVQ-B. Sample sizes in the current study, however, were not sufficient to directly compare the two tasks. Evaluating the scale by comparing these and other types of arousal induction exercises (e.g., chair spinning) will be useful to further establish the reliability and validity of the HVQ-B.

The current study’s participants scored either one SD above or below the ASI mean. Future research could examine the scale’s performance using a broader sample that includes those in the normative or moderate AS range. In the future, the HVQ-B could be compared with other measures (e.g., a measure of trait anxiety or neuroticism) to further examine the HVQ-B’s concurrent and discriminant validity. These types of analyses would be useful to further evaluate the scale’s psychometric properties. Additionally, the HVQ-B was used with mostly female, non-clinical undergraduates. Future research using the HVQ-B should be conducted with more diverse populations, including clinical populations and males. Given the high level of correlation between the original and brief version of the HVQ, it is expected that the HVQ-B would perform similarly to its original version across diverse samples, but this remains to be tested empirically.

There has been increasing interest in using IE in research and clinical settings, as well as in further understanding how IE works (Lejuez et al., 2011; Stewart & Watt, 2008). This trend parallels a broader growing emphasis on the need to further discern possible mechanisms of action for different types of treatment (Kraemer, Wilson, Fairburn, & Agras, 2002; Stout, 2007). In addition, it has become more feasible, with modern statistical techniques (e.g., hierarchical linear modeling), to incorporate a larger number of time-points in order to better measure dynamic processes as they occur throughout treatment (Stout, 2007). In treatment research and clinical practice, the HVQ-B can be informative as either a treatment outcome or process measure. In fact, a recent study compared changes over 20 running IE trials in high versus low AS participants for the three HVQ-B subscales, finding significant differences in patterns of change between these two groups (Sabourin, Stewart, Watt, & Krigolson, 2012; see Study 5 in Chapter 10 of the current dissertation). Furthermore, changes in HVQ-B scores over time can be used in clinical practice as an educational tool for clients to learn about their own responses to arousal. In fact, Meuret et al. (2005) recommend that self-report measures such as the HVQ be used in treatments that include an IE component before, during, and following the exposure. With just over half of the items of the original HVQ, the HVQ-B increases the feasibility of implementing this recommendation.

In summary, the HVQ-B is a brief, 18-item measure to assess individuals' cognitive, affective, and somatic responses to physiological arousal. Given the importance of better understanding the processes of anxiety development, maintenance, and recovery, the HVQ-B can be a useful tool in both research and clinical practice.

Table 6-1.

Corrected Item-Subscale Correlations of Original Somatic Subscale Items and Mean (SD) Ratings by Low and High AS Participants

Item	Corrected Item-Subscale Correlation	Low AS Mean (SD)	High AS Mean (SD)
Dizziness	.674	0.8(0.9)	1.3(1.0)**
Feeling distant	.655	0.3(0.6)	0.9(0.9)*
Breathlessness	.652	1.0(0.8)	1.5(1.0)*
Weakness	.637	0.5(0.7)	0.9(1.0)*
Blurred vision	.577	0.2(0.6)	0.5(0.8)*
Racing heart	.578	0.9(1.0)	1.2(1.0)
Numbness in extremities	.647	0.5(0.7)	0.7(0.9)
Tingling in extremities	.499	0.7(0.8)	0.7(1.0)
Pounding heart	.492	1.1(1.0)	1.3(1.1)
Hot or flushed	.417	1.4(1.1)	1.2(1.1)
Nausea	.334	0.1(0.3)	0.3(0.7)*
Buzzing in the head	.662	0.6(0.9)	0.9(1.1)
Feeling unreal	.549	0.2(0.5)	0.7(0.9)**
Headache	.552	0.2(0.5)	0.6(0.8)*
Band across head	.556	0.1(0.3)	0.4(0.9)*
Fatigue	.545	1.0(0.9)	1.4(1.0)
Tingling in the face	.560	0.5(0.7)	0.7(0.9)
Tight or stiff muscles	.520	0.4(0.6)	0.6(0.8)

Note. Items above the solid black line were retained to form the Somatic Subscale of the HVQ-B; AS = anxiety sensitivity
* $p \leq .05$; ** $p \leq .01$.

Table 6-2.

Means and Standard Deviations (SD) for Hyperventilation Questionnaire-Brief Subscales in Low and High AS Participants

	Study 3A		Study 3B	
	Low AS N = 39 M (SD)	High AS N = 65 M (SD)	Low AS N = 23 M (SD)	High AS N =27 M (SD)
Cognitive	0.59 (1.14)	2.20 (3.18)**	0.48 (1.16)	2.11 (2.71)**
Affective	0.72 (1.36)	2.92 (3.30)***	0.52 (0.85)	2.85 (3.39)**
Somatic	3.72 (3.04)	6.18 (4.27) **	3.65 (2.87)	6.78 (3.37)***

Note. AS = anxiety sensitivity
**$p \leq .01$; ***$p \leq .001$.

Table 6-3.

Items on the Hyperventilation Questionnaire-Brief (HVQ-B) and Correlations with their Respective Subscales from Study 3B

HVQ-B	Item-Subscale *r*s
Breathlessness[s]	.69
Feeling distant[s]	.47
Fear[a]	.43
Feeling trapped or helpless[c]	.66
Anxiety[a]	.77
Rising Agitation[a]	.43
Feeling of suffocation[c]	.78
Dizziness[s]	.61
Feeling of losing control[c]	.70
Worrying that your actions are damaging to your health[c]	.54
Blurred vision[s]	.54
Weakness[s]	.73
Nervousness[a]	.70
Racing heart[s]	.70
Feel like passing out[c]	.59
Fear of heart attack[c]	.34
Tension[a]	.55
Feel like panicking[a]	.75

Note. Subscales denoted as (c) = cognitive, (a) = affective, (s) = somatic.

Table 6-4.

Standardized Factor Loadings and Standard Errors for the Three-Factor Solution of the Hyperventilation Questionnaire-Brief

Item	Cognitive SL (SE)	Affective SL (SE)	Somatic SL (SE)
Breathlessness			.68 (.06)
Feeling distant			.61 (.06)
Fear		.61 (.05)	
Feeling trapped or helpless	.48 (.05)		
Anxiety		.72 (.06)	
Rising agitation		.66 (.07)	
Feeling of suffocation	.68 (.06)		
Dizziness			.73 (.06)
Feeling of losing control	.64 (.06)		
Worrying that your actions are damaging your health	.44 (.06)		
Blurred vision			.62(.06)
Weakness			.77 (.06)
Nervousness		.70 (.06)	
Racing heart			.53 (.07)
Feel like passing out	.82 (.07)		
Fear of a heart attack	.34 (.04)		
Tension		.67 (.07)	
Feel like panicking		.73 (.05)	

Note. SL = standardized factor loadings; SE = standard errors; all factor loadings significant at .05.

Chapter 7 . PROLOGUE TO STUDY 4

Study 3 demonstrated that the HVQ-B is a viable brief version of the HVQ, accounting for most of the original scale's variance and distinguishing between high and low AS participants' reactions to physiological arousal. Furthermore, a CFA provided evidence of a reasonably good fit of the 3-factor model to the HVQ-B item level data. Thus, the HVQ-B was used in Study 5 to assess reactions to the running IE component of the follow-up brief CBT intervention tested in Study 4.

In addition to an expanded IE component, the follow-up study described in Studies 4 and 5 included a more credible control condition, consisting of an interactive discussion on the roles of physical exercise, nutrition, and sleep for optimal health. In Study 4, the main outcomes of the follow-up intervention study are examined, comparing the effects of the CBT/IE intervention condition and the health education control condition on levels of AS and emotional distress.

Chapter 8 . STUDY 4: TWO INTERVENTIONS INVOLVING PHYSICAL EXERCISE DECREASE ANXIETY SENSITIVITY AND DISTRESS AMONG HIGH ANXIETY SENSITIVE WOMEN[13]

Abstract

A recent study found that a brief group-based cognitive behaviour therapy (CBT) which included running as a novel interoceptive exposure (IE) component was effective in reducing anxiety sensitivity (AS) levels in undergraduate women (Watt et al., 2008). The present replication and extension study's aim was to determine whether the CBT/IE intervention would result in decreases in AS and in emotional distress, and if these would be maintained over a 14-week follow-up. Female undergraduate students selected to be high (n = 81) or low (n = 73) in AS were randomized to receive either a 3-day CBT intervention plus 42 ten-minute running IE trials (n = 83), or a 3-day health education control (HEC) intervention consisting of interactive discussions on exercise, nutrition, and sleep (n = 71). The CBT/IE intervention led to decreases in AS and in symptoms of depression, stress, and anxiety for high AS participants, which were maintained over the 14-week follow-up. Unexpectedly, participants in the HEC condition experienced similar and lasting decreases in AS, and in depression and anxiety symptoms. As expected, low AS participants experienced few sustained changes. Clinical implications and the possible role of aerobic exercise in accounting for the efficacy of both interventions are discussed.

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[13] Adapted from Sabourin, Stewart, Watt, & Krigolson (2012) "Two Interventions Involving Physical Exercise Decrease Anxiety Sensitivity and Distress Among High Anxiety Sensitive Women". Manuscript submitted for publication. As the first author of this article, I contributed to the design of the study, participated in mass screening data collection, organized and managed participant recruitment, collected data, conducted the data analyses, wrote the manuscript, and revised the manuscript in accordance with suggestions from my co-authors.

Introduction

A growing body of empirical evidence links anxiety sensitivity (AS; fear of arousal-related sensations) with mental health symptoms and disorders (Naragon-Gainey, 2010; Olatunji & Wolitzky-Taylor, 2009; Schmidt, Zvolensky, & Maner, 2006; Zvolensky, Schmidt, Bernstein, & Keough, 2006). Cross-sectional studies have found elevated AS levels in individuals with panic disorder (Stewart, Knize, & Pihl, 1992), posttraumatic stress disorder (PTSD; Taylor, Koch, & McNally, 1992), other anxiety disorders (Taylor et al., 1992; Zinbarg, Barlow, & Brown, 1997), and depression (Taylor, Koch, Woody, & McLean, 1996). In addition, longitudinal studies demonstrate that elevated AS levels predict the development of Axis I psychopathology (Maller & Reiss, 1992; Schmidt et al., 2006), even after controlling for trait anxiety. In non-clinical samples, AS acts as a pre-morbid vulnerability factor for the development of panic attacks, again after controlling for trait anxiety (Hayward, Killen, Kraemer, & Taylor, 2000; Maller & Reiss, 1992; Schmidt, Lerew, & Jackson, 1997; 1999; Schmidt et al., 2006). Also in non-clinical samples, AS levels prospectively predict emotional distress, after controlling for higher-order traits such as neuroticism and trait anxiety (Cox, Taylor, Clara, Roberts, & Enns, 2008; Weems, Hayward, Killen, & Taylor, 2002). In fact, AS has been found to mediate the relationships between the higher-order trait of neuroticism and both anxiety symptoms and general distress (Ho et al., 2011). In short, the wide array of both cross-sectional and longitudinal studies, in both clinical and community samples, supports AS as a general risk factor for the development and maintenance of anxiety and related disorders.

Elevated AS levels are also associated with lower levels of physical exercise and lower physical fitness levels (Goodin et al., 2009; McWilliams & Asmundson, 2001; Sabourin, Hilchey, Lefaivre, Watt, & Stewart, 2011; see Study 1 in Chapter 2 of the current dissertation; Smits & Zvolensky, 2006). Epidemiological studies suggest that individuals who engage in regular physical exercise are less likely to experience anxiety and depressive disorders and symptoms (for review see Biddle, 2000). Furthermore, exercise has been found to be a promising tool for alleviating symptoms of depression and anxiety (for review, see Stathopoulou, Powers, Berry, Smits, & Otto, 2006). Due to the association between physical exercise and mental health, high AS individuals, through physical inactivity, may also be at increased risk for poorer mental health than their lower AS counterparts.

Treatment studies provide additional evidence supporting the importance of AS in anxiety disorders. Treatments aimed at ameliorating anxiety disorder symptoms have found that decreases in AS accompanies, and in some cases even mediates, improvements in anxiety disorders and symptoms (e.g., Smits, Powers, Cho & Telch, 2004; for review see Otto & Reilly-Harrington, 1999). As a result of these promising findings, recent treatment studies have aimed specifically at decreasing AS levels as a means of targeting this underlying risk factor for mental health difficulties. These types of treatments have resulted in decreases in AS, as well as decreases in symptoms of the associated disorders, in individuals with panic disorder (Craske, Maidenberg, & Bystritsky, 1995), PTSD (Wald, 2008; Wald & Taylor, 2005), and social phobia (Plotkin, 2001). Also, AS reduction treatments show promise in preventing future occurrences of panic disorder for at-risk, high AS individuals (e.g., Gardenswartz & Craske, 2001).

Cognitive behaviour treatments (CBT) for decreasing AS levels commonly include an interoceptive exposure (IE) component (e.g., Carter, Sbrocco, Gore, Marin, & Lewis, 2003; Craske, Lang, Aikins, & Mystkowski, 2005; Gardenswartz & Craske, 2001; Telch et al., 1993). IE consists of repeated exposure to feared arousal-related sensations with the goal of habituating to these sensations. That is, by experiencing the sensations, without the imagined accompanying catastrophic consequences (e.g., embarrassment, heart attack), fear of the sensations eventually subsides. IE exercises include activities such as hyperventilation, breathing through a straw, or chair spinning. A recent meta-analysis (Smits, Berry, Tart, & Powers, 2008) found that interventions consisting of CBT plus IE resulted in decreases in AS in both treatment-seeking samples and high AS at-risk samples.

Repeated exposure to aerobic exercise has also shown promise as a viable treatment for decreasing AS levels (Broman-Fulks, Berman, Rabian, & Webster, 2004; Broman-Fulks & Storey, 2008; Smits, Berry, Rosenfield, & Powers, 2008). Because aerobic exercise produces many of the same bodily sensations as acute anxiety (e.g., increased heart rate, perspiration), repeated exposures to aerobic exercise may serve to decrease fear of arousal sensations in the same way as other IE exercises. In addition to providing high AS individuals with corrective learning experiences, aerobic exercise may exert its therapeutic effects via a number of additional mechanisms, such as improved affect or neurochemical changes brought about by exercise (Smits, Tart, Rosenfield, & Zvolensky, 2011).

A recent study conducted with female undergraduates at two eastern Canadian universities pioneered the use of aerobic exercise (running) as the IE component of a brief AS-reduction CBT intervention (Watt, Stewart, Conrod, & Schmidt, 2008). The brief CBT/IE consisted of three 50-minute sessions on three consecutive days. During the first session, participants learned about anxiety, panic attacks, AS, and the anxiety cycle. They learned how their interpretations of arousal sensations could affect their reaction to the sensations (e.g., avoidance behaviours). During the second session, participants were taught strategies to identify, challenge, and restructure their dysfunctional thoughts consistent with accepted cognitive therapy for panic disorder (Craske & Barlow, 2001). The third session included the novel IE component of running. Participants ran as a group for 10 minutes, during which they were instructed to pay attention to the physical sensations and reflect on how these paralleled anxiety sensations. Participants were then instructed to complete ten 10-minute running trials between the last therapy session and the 10-week follow-up assessment.

The treatment was effective in decreasing AS levels in high AS female university students (Watt et al., 2008). The treatment also resulted in decreases in pain anxiety (Watt, Stewart, Lefaivre, & Uman, 2006), and in risky drinking behaviours (Watt, Stewart, Birch, & Bernier, 2006). The study did not assess whether changes in AS levels were maintained beyond a 10-week follow-up period. Moreover, the study did not assess whether the intervention that was aimed at decreasing AS also led to improvements in general emotional distress. Finally, the study's design included a control condition consisting of a group discussion on ethics in psychology to control for non-specific effects that could influence outcome (e.g., group exposure, therapist effects). The control condition's content, however, bore no similarity to the intervention's content, and thus could not control for other factors that could potentially affect outcomes (e.g., discussions that were directly relevant to the participants' own health).

The Present Study

The present study consisted of a replication and extension of Watt et al. (2008) to increase our understanding of the benefits of the brief CBT/IE intervention. Specifically, we aimed to examine the effects of the intervention on changes in AS levels, extending the assessment period to a 14-week follow-up. Additionally, we aimed to examine the benefits of the intervention on general emotional distress, by assessing changes in stress, depression, and anxiety symptoms. We also used a more stringent control condition – a health education control (HEC) comprised of a group discussion on health, focusing on the importance of exercise, diet, and sleep on participants' optimal health. Our HEC was partially derived from a control condition used in a randomized controlled trial conducted by Schmidt et al. (2007). The CBT/IE intervention remained mostly unchanged from the previous study other than the IE component. Because of the theoretical importance of IE in reducing AS levels, we believed that increasing the frequency and duration of the IE trials would help maintain benefits and enhance the intervention's efficacy. IE trials were therefore expanded from a total of 10 trials over a period of 10 weeks, to three IE trials per week for 14 weeks.

Based on previous findings with the brief CBT/IE, it was hypothesized that only high AS participants in the brief CBT/IE condition, but not those in the HEC condition, nor low AS participants in either condition, would experience significant reductions in AS levels. Second, it was expected that participating in the CBT/IE intervention, but not in the HEC condition, would result in general improvements in stress, depression, and anxiety levels for high AS participants specifically. Improvements in AS levels and in general emotional distress were hypothesized to persist at the 14-week follow-up. High AS participants randomized to the CBT/IE condition were expected to differ from low AS participants randomized to the CBT/IE intervention on all outcome measures at pre-treatment, but these AS group differences were expected to become *non-significant* following the CBT/IE intervention and at follow-up.

Methods

Participants

Undergraduate women were recruited for the present study based on scoring one standard deviation above or below the mean for university women (i.e., 18 +/- 7; see Watt et al., 2008) on the ASI (Peterson & Reiss, 1992), a widely-used measure of AS (see Appendix A for selected items). The ASI was completed as part of a screening battery either online or using paper-and-pencil format during an introductory psychology class. Potential participants who screened positive for any physical or health problem (e.g., hypertension) that might prevent them from participating in physical exercise were excluded from the study. Only women were selected to participate for the present study for several reasons. First, we wanted to control for sex effects during groups. Second, women have been found to have higher AS levels than men (Stewart, Taylor, & Baker, 1997). Third, AS has been shown to be more closely related to psychopathology in women than in men (Olatunji & Wolitzky-Taylor, 2009). Finally, we were attempting to replicate findings from the previous study (Watt et al., 2008), which used only women.

High and low AS participants were randomly assigned to the CBT or HEC group, to form four separate groups: high AS/CBT (n = 44), high AS/HEC (n = 37), low

AS/CBT (n = 39), low AS/HEC (n = 34). A 2 (AS group: high AS, low AS) x 2 (condition: CBT, HEC) between subjects ANOVA on initial ASI scores revealed a main effect of AS group, $F(1,146) = 1018.62$, $p < .001$, but a non-significant main effect of condition, $F(1,146) = 0.21$, p = NS, and a non-significant interaction, $F(1,146) = 0.74$, p = NS. These results suggest that the high AS group had a higher ASI score than the low AS group, but that there were no differences in ASI scores between the two treatment conditions (see Figure 8-1, pre-scores). Participants ranged from 17 to 34 years of age (*M* = 18.8; *SD* = 2.2). A 2 (AS group: high AS, low AS) x 2 (condition: CBT, HEC) between subjects ANOVA revealed no significant effects (*F*s < 2.31, *p*s > .10), suggesting that none of the conditions differed significantly in participant age. Most (90%) participants self-identified as Caucasian. A 2 (race: Caucasian, other) x 4 (group: high AS/CBT, low AS/CBT, high AS/HEC, low AS/HEC) chi-square analysis confirmed that race did not differ between groups, $\chi^2(3) = 1.08$, $p > .10$.

Of the original 154, a total of 125 participants completed all three days of treatment (i.e., 81% completion rate). Participants were lost to the study for a variety of reasons, including snow storms, time change due to return to Standard Time, conflicting participant work schedules, and illness. Sixty seven participants completed both the intervention and the 14-week follow-up. Participants who completed the intervention and the follow-up did not differ from those who either did not complete the intervention or were not available for follow-up on initial ASI scores, $F(1,148) = 1.11$, p = NS, on age, $F(1,150) = 1.08$, p = NS, or on race, $\chi^2(1) = 0.28$, p = NS.

Measures

Anxiety Sensitivity Index (ASI; Peterson & Reiss 1992). The ASI is a widely used 16-item questionnaire which assesses trait AS levels (see Appendix A for selected items). Participants rate each item (e.g., “It scares me when I become short of breath”) on a 5-point Likert scale ranging from “very little” (scored as 0) to “very much” (scored as 4). The measure has shown strong psychometric properties (Peterson & Reiss, 1992; Rector, Szacun-Shimizu, & Leybman, 2007). For the present study, pre-intervention internal consistency was high with an alpha coefficient of .93.

Depression Anxiety Stress Scales - 21 (DASS-21; Lovibond & Lovibond, 1995). The DASS-21 is a 21-item self report measure assessing participants’ current experiences of psychological distress (see Appendix G). For the current study, the 7-item stress and depression subscales were used: 1) stress (e.g., “I found myself getting upset over quite trivial things”), and 2) depression (e.g., “I felt that I had lost interest in just about everything”). Participants rate the extent to which they experienced the negative emotional state over the past week using a 4- point Likert scale (scored as 0-3). The DASS has good internal consistency, and convergent and discriminant validity (Lovibond & Lovibond, 1995). Pretreatment internal consistency was high in the current study’s sample, with alpha coefficients of .89 for the stress subscale, and .91 for the depression subscale.

Beck Anxiety Inventory (BAI; A. T. Beck & Steer, 1993). The BAI is a 21-item self-report measure used to assess both cognitive and somatic symptoms of anxiety (see Appendix H for selected items). Participants rate each item (e.g., “nervous”, “hands trembling”) on a 4-point Likert scale ranging from “Not at all” to “Severely” (scored as 0-3) based on how much they have been bothered by each symptom over the past month. The measure has shown excellent psychometric properties, including high internal consistency, test-retest reliability, and concurrent validity with other measures of anxiety (A. T. Beck & Steer, 1993; Steer, Ranieri, Beck, & Clark, 1993). The alpha coefficient in the current study was .90 at pretreatment.

Procedure

The brief CBT and the HEC conditions consisted of three one-hour group sessions over a period of three consecutive days. For the CBT group, the manual for the intervention was adapted by Watt and colleagues from manuals that were originally developed by Conrod et al. (2000) and Harrington and Telch (1994), and used in the previous Watt et al. (2008) study. As explained above, the first two days consisted of psychoeducation and cognitive reframing of dysfunctional thinking. On the third day of the intervention, participants engaged in the IE running component. They were instructed to run as a group for 10 minutes, after which they participated in a debriefing and completed the HVQ-B. Participants were then instructed to complete the homework assignment comprised of 10 minutes of running three times per week for a period of 14 weeks, for a total of 42 running trials.

The HEC condition focused on the importance of exercise, nutrition, and sleep on optimal health. Each topic was introduced with a brief video presentation used in a recent study by Schmidt et al. (2007). The video was supplemented by an interactive discussion further elaborating on the topic as it applied to participants. The interactive discussion, which was not included in the Schmidt et al. control condition, was added in order to make the two groups comparable in format (i.e., group interactive discussion) and time spent in group.

The baseline measures of interest for the current study were collected prior to participants taking part in the intervention (i.e., 'pre'). Outcome measures were collected again after participants in the experimental group took part in the running component for 10 weeks or after an equivalent time had elapsed for those in the control group (i.e., 'post'), and again at the 14-week follow-up (i.e., 'follow-up').

Results

Completer analyses were used in the present study in order to determine the effects of the intervention for participants who completed the treatment.[14] Outcome variables (i.e., ASI, DASS-21 Stress and Depression subscales, BAI) were analyzed in relation to study hypotheses by decomposing the full 3 (time: pre, post, follow-up) x 2 (condition: CBT, HEC) x 2 (AS group: high AS, low AS) table of means into a series of *a priori* planned comparisons (cf., Birch et al., 2008; Stewart, Hall, Wilkie, & Birch, 2002). Planned comparisons were used in order to examine the comparisons of primary interest first, using conventional alpha levels (Tabachnick & Fidell, 2006). Specifically, paired samples t-tests were used to compare pre scores with both post and follow-up scores for the CBT/IE and HEC conditions in high and low AS participants separately. Next, independent samples t-tests were used to compare high AS and low AS participants in the CBT/IE group at each time point. For comparisons that had a significant Levene's test,

[14] Nine participants were not available for completion of post measures but did complete the 14-week follow-up measures. These participants were retained in the current analyses and post scores were imputed using a conservative carry-forward method that assumes no change from pre to post. Deleting these nine participants from the analyses did not affect results, other than three minor changes in significance: 1) DASS stress: the pre/follow-up significance value for low AS participants in the HEC condition changed from $p = .05$ to $p < .05$, 2) DASS depression: the pre/follow-up significance value for high AS participants in the CBT condition changed from $p = .07$ to $p < .05$, and 3) DASS depression: the pre/follow-up significance value for high AS participants in the HEC condition changed from $p = .05$ to $p = .06$.

independent samples t-tests that did not assume homogeneity of variances were performed.

Intervention effects on AS levels

As expected, the intervention resulted in significant decreases in AS levels for high AS participants in the CBT/IE group, which were maintained at the 14-week follow-up (pre/post: $t[17] = 2.75$, $p = .01$; pre/follow-up: $t[17] = 4.14$, $p = .001$). Unexpectedly, participating in the HEC condition also resulted in significant decreases in AS levels for high AS participants, which were also maintained at the 14-week follow-up (pre/post: $t[21] = 4.84$, $p < .001$; pre/follow-up, $t[21] = 6.86$, $p < .001$). There were no changes in ASI scores for low AS participants in the CBT/IE condition (pre/post: $t[10] = 0.25$, p = NS; pre/follow-up: $t[10] = 0.54$, p = NS). There was an unexpected *marginally significant increase* in ASI scores for low AS participants in the HEC condition following the intervention, $t(14) = -2.07$, p = .06. However, this increase was not evident by the 14-week follow-up, $t(14) = -0.29$, p = NS. Finally, analyses of ASI scores indicated that high AS and low AS participants in the CBT/IE group differed at pre-treatment, as expected (pre: $t[25.82] = 20.90$, $p < .001$; see Figure 8-1), but also, unexpectedly, at the two follow-up assessments (post: $t[23.50] = 6.75$, $p < .001$; follow-up: $t[25.5] = 5.75$, $p < .001$; see Figure 8-1). Although ASI scores differed at each measurement time, the magnitude of differences in AS levels between high and low AS participants appeared to decrease at the two time points following the CBT/IE intervention (see also Figure 8-1).

Changes in stress, depression, and anxiety symptoms

As expected, high AS participants in the CBT/IE condition experienced a decrease in stress symptoms following the intervention, which was maintained at the 14-week follow-up (pre/post: $t[14] = 2.93$, $p = .01$; pre/follow-up: $t[14] = 3.02$, $p < .01$). Also as expected, neither high AS participants in the HEC group (pre/post: $t[18] = 1.00$, p = NS; pre/follow-up: $t[18] = 1.02$, p = NS), nor low AS participants in the CBT/IE condition (pre/post: $t[10] = 1.65$, p = NS; pre/follow-up: $t[10] = 0.93$, p = NS) experienced changes in stress scores. Unexpectedly, low AS participants in the HEC condition experienced a slight decrease in stress scores (pre/post: $t[14] = 2.18$ $p < .05$), which was somewhat maintained at follow-up (pre/follow-up: $t[14] = 2.11$, $p = .05$). Finally, high AS and low AS participants in the CBT/IE group differed significantly from each other prior to the intervention, $t(25) = 2.61$, $p = .01$, and at post intervention, $t(25) = 3.11$, $p < .01$. By the 14-week follow-up period, however, there was no longer a significant difference between the two groups, $t(25) = 1.71$, p = NS (see Figure 8-2A).

As expected, high AS participants in the CBT/IE condition experienced a decrease in depression scores (pre/post: $t[14] = 2.50$, $p < .05$), which was, to some extent, maintained over time (pre/follow-up: $t[14] = 2.00$, $p = .07$). Unexpectedly, high AS participants in the HEC condition also experienced a decrease in depression scores which was also, to some extent, maintained over time (pre/post: $t[18] = 2.65$, $p < .05$; pre/follow-up: $t[18] = 2.08$, $p = .05$). Low AS participants did not experience a decrease in depression scores over time (CBT/IE, pre/post: $t[10] = 0.74$, p = NS; pre/follow-up: $t[10] = 0.08$, p =NS; HEC, pre/post: $t[14] = 0.00$, p = NS; pre/follow-up: $t[14] = 1.54$, p = NS). Finally, high AS and low AS participants in the CBT/IE group differed significantly from each other prior to the intervention, $t(18.22) = 2.96$, $p < .01$, and at post intervention, $t(17.60) = 2.80$, $p = .01$. However, by the 14-week follow-up period, the groups no longer differed on depression scores, $t(25) = 1.72$, p = NS (see Figure 8-2B).

High AS participants in the CBT/IE condition did not experience a decrease in anxiety scores following the intervention (pre/post: $t[14] = 1.43$, p = NS). However, by the 14-week follow-up, scores had decreased significantly from pre-levels, $t(14) = 3.08$. p < .01. Unexpectedly, this pattern was also evident for high AS participants in the HEC condition (pre/post: $t[20] = 1.30$, p = NS; pre/follow-up: $t[20] = 2.96$, $p < .01$), as well as for low AS participants (CBT/IE, pre/post: $t[10] = 1.43$, p = NS; pre/follow-up: $t[10] = 2.29$, $p < .05$; HEC, pre/post: $t[11] = 1.76$, p = NS; pre/follow-up: $t[11] = 3.04$, $p = .01$). High AS participants' scores on the BAI differed significantly from low AS participants', but this was unexpectedly found to be true at all three time points (pre: $t[16.23] = 4.63$, p < .001; post: $t[18.11] = 4.83$, p < .001; follow-up: $t[22.56] = 3.49$, $p < .005$; see Figure 8-2C) [15].

Discussion

The current study's aim was to replicate and extend findings from a previous study (Watt et al., 2008) demonstrating the therapeutic effects of a brief CBT/IE AS reduction intervention. As hypothesized, the intervention resulted in significant decreases in AS for high AS participants, which were maintained over the 14-week follow-up

[15] Supplementary analyses of the current study's data were also conducted using a mixed model repeated measures ANOVA (see Appendix I). This supplemental set of analyses replicated the conclusions drawn for two of four outcome variables (AS and depression). Although the results were slightly different for anxiety and stress variables, more weight would be placed in the results of the original planned comparison approach because the study was inadequately powered to detect the interactions of interest within an omnibus ANOVA. Nonetheless, the results of the omnibus ANOVA are presented for the interested reader.

period. These results confirm and extend those found by the Watt et al. (2008) study, which did not examine longer-term benefits of the intervention.

Unexpectedly, high AS participants in the HEC (i.e., control) condition, which consisted of a group discussion on health behaviours, also experienced a decrease in AS. These results differ from those of a study by Schmidt et al. (2007) from which the video portion of our HEC was derived; Schmidt et al. found no decrease in AS for high AS participants in their health control condition. As the current study supplemented the video with an interactive, personalized discussion, it is possible that this additional component produced the observed changes in AS. Discussion of the role of exercise, nutrition, and sleep on health included topics related to mental health. In particular, both the physical and mental health benefits of aerobic exercise were discussed. Furthermore, the discussion included problem-solving various barriers to engage in health behaviours, including physical exercise (e.g., how to fit exercise into one's schedule). Perhaps these discussions encouraged participants to appreciate the role of aerobic exercise, and the sensations associated with exercise, in improving mental health. Consequently, if they began to think of the sensations brought about by aerobic exercise as a sign of improving health, they perhaps gained a new perspective on physiological arousal in general. In addition, barriers to exercise have been shown to account for lower levels of exercise participation in high AS women (Sabourin et al., 2011; see Study 1 in Chapter 2 of the current dissertation). The interactive discussion and problem solving on barriers to engaging in health behaviours like exercise might have therefore led to increased exercise participation for high AS participants in the HEC condition, contributing to decreases in AS.

Results from another measure of psychological distress, the DASS-21, indicated that high AS participants in the CBT/IE, but not the HEC, condition experienced a sustained decrease in general stress, as was hypothesized. Thus, it appears that although both groups experienced decreases in AS, additional therapeutic benefits were conferred from participating in the brief CBT/IE intervention, compared with participating in the HEC condition, for high AS women. It is possible that high AS participants in the CBT/IE group learned from the cognitive restructuring portion of the intervention more adaptive ways to deal with stressful situations, thereby decreasing levels of general stress in their lives.

Participation in both interventions resulted in sustained decreases in depression scores for high AS participants. Cognitive restructuring in the CBT/IE condition, and a focus on physical and mental health in the HEC condition, might have each led to improvements in mood for high AS participants. Because exercise has been shown to be effective in decreasing symptoms of depression (Craft & Landers, 1998), it is possible that both groups' improvements resulted in part from increased exercise participation.

Anxiety scores, as assessed by the BAI, decreased for all participants by the 14-week follow-up. It is unclear why even low AS participants experienced a decrease in BAI scores. Because AS and trait anxiety are believed to be related but distinct constructs (McNally, 1989), it is possible that both interventions had anxiolytic effects even for low AS participants. It is also unclear why decreases in anxiety were only apparent at the 14-week follow-up, and not immediately following the intervention. It is possible that earlier changes in ASI scores mediated later decreases in overall anxiety levels, at least in high AS participants. It is also possible that the BAI's longer time frame relative to the DASS (past month vs. past week, respectively) explains why therapeutic effects were not evident until the follow up for anxiety symptoms.

As hypothesized, there were few effects of the interventions on any of the outcomes in low AS participants. As expected, neither intervention led to decreases in AS or depression at either post-treatment or follow-up for the low AS participants. Unexpectedly, the HEC treatment did lead to a marginally significant *increase* in low AS participants' AS levels but these were short lived (i.e., not persisting at follow-up). Furthermore, the increases did not bring low AS participants' scores to high AS levels, but rather brought them closer to normative AS levels. It is not clear why the HEC control condition had this short lived effect. Perhaps discussing health issues made them somewhat more aware of certain physiological states in the short term. Also unexpectedly, low AS participants in the HEC control condition experienced a slight decrease in stress scores. It is possible that the interactive discussion on health helped low AS participants better manage stress by improving their health behaviours.

As expected, high AS and low AS participants in the CBT/IE condition differed on their levels of AS, stress, depression, and anxiety symptoms before beginning the intervention. These AS group differences on depression and stress scores were attenuated by the 14-week follow-up. Thus, it appears that participating in the CBT/IE intervention brought levels of general stress and depression down to those experienced by individuals who, based on ASI scores, would be considered at lower risk of emotional disorders. Unexpectedly, anxiety scores remained significantly different at follow-up. This might be due to the unexpected decrease in symptoms of anxiety even for participants in the low AS group. Also unexpectedly, ASI scores remained significantly different between the two groups even at post and follow-up. However, because low AS participants were selected specifically based on scoring one standard deviation *below* the mean on the ASI, it was not entirely surprising that high AS participants' ASI scores did not reach the low AS group's levels following the brief CBT/IE treatment.

As the previous (Watt et al., 2008) study did not assess the impact of the brief CBT/IE intervention on general psychological distress, results from the current study provided new information regarding the potential benefits of the intervention. It also provided new information on potential psychological benefits of interactive discussions on health behaviours, including physical exercise.

Implications

The current study lends further support for the CBT/IE intervention's efficacy in targeting a known risk-factor for various anxiety and anxiety-related disorders. The current study also appears to indicate that an interactive discussion on health confers similar benefits. The fact that both of the 3-hour group-based interventions confer such benefits is encouraging in an era when health care systems are asked to do more with fewer resources. The longer-term effects beyond 14 weeks of both of these interventions, however, are still unknown. Future research could follow participants in the longer-term (i.e., several years) to compare rates of psychological disorders in high AS individuals who participate in one of the two interventions or in a no-treatment control condition. This would lend further support to the potential preventative effects of the interventions. It would also help elucidate whether there are potential additional longer-term benefits to participating in the CBT/IE intervention, compared with either health education or no-treatment.

Based on previous research demonstrating the known beneficial effects of exercise (e.g., Broman-Fulks et al., 2004; Broman-Fulks & Storey, 2008; Smits, Berry, Rosenfield, et al., 2008), it is possible that physical exercise might be the underlying link between both interventions' therapeutic effects. In fact, Smits et al. found that adding a CBT component to an exercise intervention did not confer additional therapeutic benefit. Smits et al.'s rationale consisted of the usefulness of exercise as a means of inducing physiological arousal to decrease AS levels, whereas the current study's HEC group's discussion focused on global physical and mental health benefits of exercise and included problem-solving exercise barriers. Future research could explore the additional benefits of adding a rationale to an exercise "prescription" that includes a focus on the role of exercise in re-interpreting physiological arousal sensations, over and above one that focuses on its role in improving overall health. In addition, future research could assess change in total exercise participation in both interventions to determine whether physical exercise is indeed a common mediator of these interventions' therapeutic benefits.

The implications and potential benefits of using physical exercise as part of an intervention for AS are manifold. Encouraging individuals who might otherwise shy away from exercise to develop a better tolerance and acceptance of the sensations associated with exercise might pave the way for adoption of physical exercise for the longer term. Given the numerous physical and mental health benefits of increasing exercise participation (Stathopoulou et al., 2010; Warburton, Charlesworth, Ivey, Nettlefold, & Bredin, 2010), becoming more open to engaging in physical exercise might lead to life-long additional benefits, including lower incidence of mental illness and increased longevity. Furthermore, it appears that physical exercise provides even more pronounced protective effects in responding to physiological stressors for high AS than for low AS individuals (Goodin et al., 2009; Smits et al., 2011). Thus, it appears that it is even more crucial to encourage high AS individuals to engage in physical exercise.

Anecdotally, several high AS participants in the CBT/IE condition expressed that the running IE exposure exercises had eventually become a positive experience, and that they began to feel good about the fact that they were running. It has been suggested that a potential tool for increasing exercise adoption is a focus on the immediate positive affective experience post-exercise (Smits & Otto, 2009). In fact, acute positive affective response to exercise has been shown to prospectively predict increases in exercise participation 6- and 12- months later (Williams, Dunsiger, et al., 2008). Future research could examine positive affectivity specifically, in order to ascertain whether, with repetition, the IE running trials indeed became a positive experience for high AS participants.

Limitations and Future Directions

The current investigation was not without its limitations. First, the IE trials were performed unsupervised. Although participants were instructed to focus on sensations during the running, and to abstain from listening to music while running, it is possible that participants did use music or other distractions, which might have decreased the exposure's potential for producing fear habitation (Foa & Kozak, 1986).

Unfortunately, changes in physical activity participation were not assessed in the current dissertation as a manipulation check. Therefore, it was not possible to ascertain whether participants in the CBT condition had increased their physical exercise participation moreso than those in the HEC condition as had been originally intended, or whether the discussion and problem solving around barriers to exercise in the HEC condition resulted in equivalent increases in exercise participation to that expected in the CBT condition as we have suggested. Future research should measure changes in physical exercise participation to determine whether these indeed occurred and were related to therapeutic outcomes.

The study used the ASI, a self-reported measure of AS. Future research could supplement the ASI with other indicators of AS, such as psychophysiological responsivity to biological challenge exercises (e.g., CO_2 inhalation; cf., Richey, Schmidt, Hofmann, & Timpano, 2010).

Participants in both the experimental and control conditions of the current study experienced significant decreases in ASI scores. Without an additional control condition (e.g., no-treatment control, other non-specific treatment) that showed no change in ASI scores, it is not possible to rule out the possibilities that observed decreases in the current study represented either a regression to the mean or some non-specific therapeutic effect (e.g., therapist attention). On the other hand, the magnitude of decrease experienced in the current study was similar to the magnitude in the previous Watt et al. (2008; Sabourin et al., 2008; see Study 2 in Chapter 4 of the current dissertation) study. Sabourin et al. reported pre-to immediate post-intervention changes in ASI scores for high AS participants in the CBT/IE intervention condition of 6.7 ASI units. Changes were reported to be maintained at the 10-week follow-up (Watt, Stewart, Birch, et al., 2006). The current study's high AS CBT/IE participants' scores decreased by 7.8 ASI units from pre- to post-treatment (i.e., 10-weeks), and by 10.6 units from pre-treatment to follow-up (i.e., 14-weeks). It is unlikely that this similar magnitude of change experienced in the current study represents only a regression to the mean. And our previous study (Watt et al., 2008), which controlled for non-specific therapy factors, suggests that changes are not simply secondary to therapist attention, for example. Future research, however, should incorporate a no-treatment or non-specific therapy control group to completely rule out these possibilities[16] .

The current study incorporated additional running trials not assigned in the original study. Although the previous study did not assess participants beyond the 10-week follow-up, the larger magnitude of change seen at follow-up in the current study suggests the possibility of a dose-response relationship. Because exercise dose was not manipulated experimentally, however, it is not possible to directly attribute the change in response to the larger dose of IE trials. Future research could compare the effects of experimentally varying the number of running trials to determine whether additional psychological benefits are indeed conferred with additional running trials.

Several participants did not complete the intervention or were lost to follow-up. The fact that the intervention consisted of a group-based, 3-day intervention provided once to twice per year prevented the possibility that participants could reschedule if they had to miss one of the three days due to conflicting work schedule or illness. In addition, previous intervention studies consisting of unsupervised physical exercise or follow-up assessments several months after the end of treatment have also had a significant amount of non-completion (e.g., Broocks et al, 1998, Craske et al., 2011). Although the final study participants did not differ from those participants who dropped out on variables

[16] The HEC condition in the current study was intended to be a non-specific therapy control group. The HEC, however, included a discussion on exercising and problem solving around barriers to exercise. Due to this theoretically important inclusion, and the HEC's apparently beneficial effects, future research needs to compare the brief CBT against a more theoretically neutral control group.

such as pre-intervention AS levels, age, and race, it is possible that they systematically differed on other relevant unmeasured variables

The current study's 3-day intervention included one 1-hour session focusing on cognitive restructuring, with no follow-up or booster sessions following the group intervention. Future research could incorporate longer interventions in order to determine whether individuals would benefit from continued practice with cognitive restructuring and/or review of the running IE exercises. Regular check-ins with participants could also be incorporated to assess for potential added benefits of problem-solving for barriers to engage in the IE portion of the intervention and/or reviewing comparisons between physical sensations associated with running and with anxiety.

Participants in the current study were comprised of undergraduate non-clinical women. Thus, it is unclear whether these results would generalize to men and/or to clinical samples. On the other hand, the pre-intervention ASI scores in high AS women (i.e., 35.5) in the present study reflect levels that are comparable to individuals with panic disorder (Meuret, Rosenfield, Seidel, Bhaskara, & Hofmann, 2010; *M* range = 35-36)[17]. Also, because AS levels are higher and more closely related to psychopathology in women than in men (Olatunji & Wolitzky-Taylor, 2009; Stewart et al., 1997), the current study's focus on women provides a good starting point for future research.

Finally, as a newer version of the ASI, the ASI-3 (Taylor et al., 2007) has shown stability in separating AS into three components (i.e., social, psychological, physical), future research might examine the potential benefits of targeting the intervention based on participants' scores on each of the three components. For example, focusing the

[17] Additionally, although there were no specific assessments of psychopathology, almost half (39/81) of the high AS participants had experienced at least one panic attack. Of these, fifty percent had experienced at least one unexpected panic attack.

interventions on the meaning of publicly displaying physiological signs of anxiety might be of particular benefit for individuals scoring high on the social subscale of the ASI-3.

Conclusion

In conclusion, the present replication and extension study provided additional support for the efficacy of a brief group-based, CBT/IE intervention in reducing AS levels, and improving symptoms of general stress, depression, and anxiety in high AS women. Additionally, the study showed that providing a rationale and problem-solving barriers to engaging in health behaviours, including physical exercise, also results in improvements in AS and depression and anxiety symptoms. Given the evidence linking high AS levels with emotional disorders (Naragon-Gainy, 2010), decreasing AS levels provides an important preventive-type intervention. Finally, as both the IE component and the health discussion encouraged physical exercise, these interventions also have the potential of increasing exercise participation in high AS individuals, conferring additional mental and physical health benefits for this at-risk population.

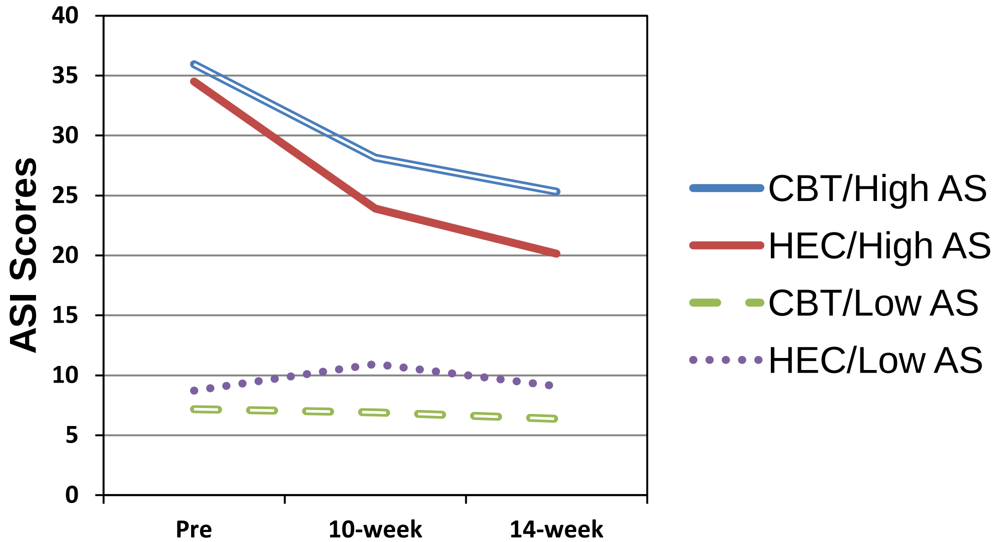

Figure 8-1. Scores on the Anxiety Sensitivity Index over time for high AS and low AS participants in the CBT and HEC groups. CBT = Cognitive behavioural treatment; HEC = Health education control; AS = anxiety sensitivity.

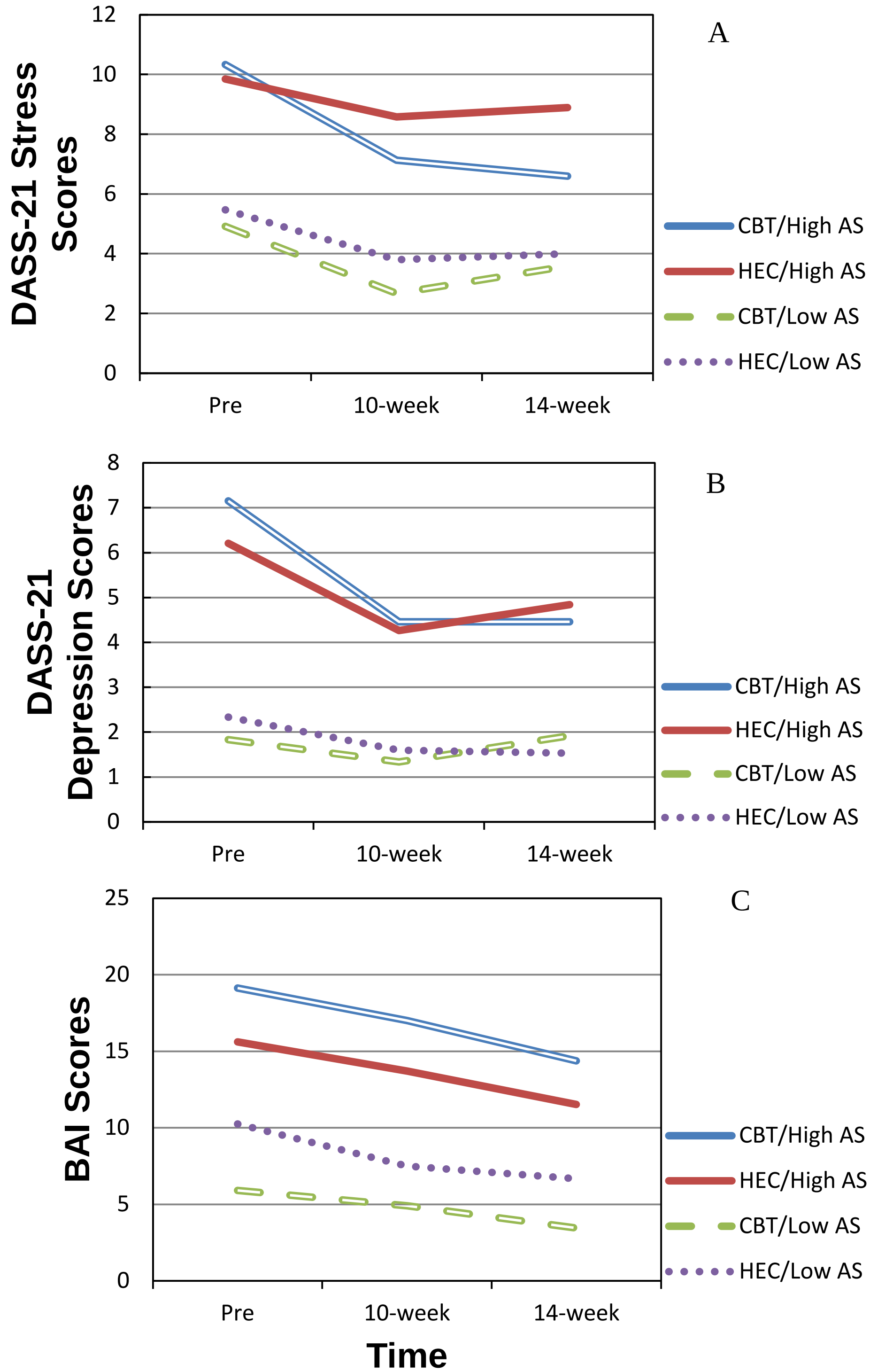

Figure 8-2. Change in DASS-21 subscale and BAI scores over time. AS= anxiety sensitivity; DASS-21 = Depression, Anxiety, Stress Scales-21; BAI = Beck Anxiety Inventory

Chapter 9 . PROLOGUE TO STUDY 5

Study 4 demonstrated that the CBT/IE intervention and the health education intervention both resulted in decreases in AS levels and in symptoms of emotional distress in high AS participants. Study 4 also described the IE component of the brief CBT/IE intervention. Reactions to the IE component, as measured by responses to the three subscales of the HVQ-B, however, were not analysed in Study 4.

In Study 5, changes in reactions to the IE component across running trials are examined in both high and low AS participants to further determine the processes through which the CBT/IE intervention exerted its therapeutic benefits. The primary goal of Study 5 was to replicate and extend findings from Study 2. Specifically, Study 5 examined whether initial reactions to running differed between high and low AS participants. Further, Study 5 sought to determine whether affective and cognitive changes would decrease only for high AS, but not for low AS participants. Finally, Study 5 sought to extend findings from Study 2 by determining whether changes in cognitive and affective reactions to running were indeed related to changes in AS levels, providing further evidence for the role of altering cognitive catastrophizing of, and fearful responding to, physiological arousal in decreasing AS levels.

Chapter 10 . STUDY 5: RUNNING AS INTEROCEPTIVE EXPOSURE FOR DECREASING ANXIETY SENSITIVITY: REPLICATION AND EXTENSION[18]

Abstract

A recent brief, group cognitive behavioural therapy (CBT) with running as the interoceptive exposure (IE; exposure to physiological sensations) component was effective in decreasing anxiety sensitivity (AS; fear of arousal sensations) levels in female undergraduates (Watt et al., 2008). A process study based on the CBT intervention found that repeated exposure to running led to decreases in cognitive (i.e., catastrophic thoughts) and affective (i.e., feelings of anxiety) reactions over time for high AS, but not low AS, participants (Sabourin et al., 2008; see Study 2 in Chapter 4 of the current dissertation). A follow-up study that included the brief CBT intervention with an expanded IE component also resulted in decreases in AS levels (Sabourin et al., 2012; see Study 4 in Chapter 8 of the current dissertation). The present process study's goal was to replicate the original process study, with the expanded IE component. An additional goal was to determine whether decreases in cognitive, affective, and/or somatic (physiological sensations) reactions to running would be related to decreases in AS. Eighteen high AS and 10 low AS participants completed 20 IE running trials following the 3-day group intervention. Whereas for high AS participants, there were decreases in cognitive, affective, and somatic reactions to running over time, in low AS participants there were only marginal decreases in affective and somatic, and no decrease in cognitive reactions over time. Furthermore, decreases in cognitive and affective, but not in somatic reactions

[18] Adapted from Sabourin, Stewart, Watt, & Krigolson (2012) "Running as Interoceptive Exposure for Decreasing Anxiety Sensitivity: Replication and Extension". Manuscript submitted for publication. As the first author of this article, I contributed to the design of the study, participated in mass screening data collection, organized and managed participant recruitment, collected data, conducted the data analyses, wrote the manuscript, and revised the manuscript in accordance with suggestions from my co-authors.

to running were related to decreases in AS levels. The study lends further support to the roles of reducing fears of and maladaptive beliefs about physiological arousal in decreasing AS levels.

Introduction

Anxiety sensitivity (AS; fear of arousal-related sensations) is a well-documented risk factor for anxiety and other related disorders (Naragon-Gainy, 2010; Olatunji & Wolitzky-Taylor, 2009; Schmidt, Zvolensky, & Maner, 2006). Individuals with high levels of AS are inclined to catastrophize the possible consequences of arousal-related sensations (e.g., public ridicule, heart attack, going crazy), which can cause the sensations to spiral, leading to a vicious cycle of heightened fear and amplified anxiety-related physiological sensations. As such, AS has been described as both a cognitive (i.e., beliefs) and an affective (i.e., fear) construct (McNally, 1999).

Previous research supports cognitive behaviour therapy's (CBT) efficacy in decreasing AS levels in both clinical populations and in at-risk high AS individuals (for review, see Smits, Berry, Tart, & Powers, 2008). CBT interventions for AS commonly incorporate an interoceptive exposure (IE; exposure to physiological sensations) component (e.g., Carter, Sbrocco, Gore, Marin, & Lewis 2003; Craske, Lang, Aikins, & Mystkowski, 2005).

Although CBT/IE interventions have been shown to be efficacious in reducing AS levels, research in the treatment of anxiety and related disorders has moved beyond simply identifying *which* treatments work, and has become more interested in ascertaining *how* treatments work (e.g., Meuret, Rosenfield, Hofmann, Seidel, & Bhaskara, 2010; Hofmann et al., 2007; Smits, Rosenfield, McDonald, & Telch, 2006;

Woody, Whittal, & McLean, 2011). That is, there is increasing emphasis on elucidating the processes through which interventions exert their therapeutic benefits. In the case of IE for AS and associated conditions, theoretically, repeated exposure to physiological arousal can lead to decrease in fearful responding by way of at least two separate mechanisms.

First, from a cognitive perspective, arousal or anxiety-related sensations are misinterpreted as signifying imminent danger. These catastrophic thoughts lead to a heightened state of apprehensive arousal, culminating in an increased risk of developing a panic attack (D. M. Clark, 1988). Exposure to the feared sensations without the accompanying feared consequences, therefore, might lead to a re-evaluation of the *meaning* of arousal sensations as less threatening (Chambless & Goldstein, 1981). Second, from a learning perspective, fear of physiological arousal (the conditioned stimulus; CS), arises due to a learned association with a feared consequence, such as a heart attack (the unconditioned stimulus; US). IE exercises act to present the CS repeatedly in the absence of the US, such that new learning occurs accompanied by a reduction in fear of the CS (Bouton, 2004), resulting in what has been called *extinction learning* (Moscovitz, Antony, & Swinson, 2009). Empirical evidence, however, has yet to emerge that clearly supports these theoretical processes (Moscovitch et al., 2009; Stewart & Watt, 2008). That is, it is still unclear whether one or both of the following mechanisms are indeed driving IE's therapeutic effects: 1) alterations in the meanings of anxiety sensations drive reductions in fear (i.e., a *cognitive* mechanism), or 2) reductions in fear of anxiety sensations, which are produced via extinction learning, drive alterations in meanings (i.e., an *affective* mechanism).

Anxiety Sensitivity and Physical Exercise

High AS is associated with lower levels of physical exercise and physical fitness (Goodin et al., 2009; McWilliams & Asmundson, 2001; Sabourin, Hilchey, Lefaivre, Watt, & Stewart, 2011; see Study 1 in Chapter 2 of the current dissertation; Smits & Zvolensky, 2006). It is possible that high AS individuals avoid physical exercise due to fears of the associated physiological sensations. Alternatively, the limited exposure to arousal-related sensations in individuals who do not engage in physical exercise might contribute to elevated AS levels. It is also possible that these two mechanisms work in conjunction in maintaining both exercise avoidance and high AS levels.

Aerobic exercise is a promising treatment for improving mental health, including risk factors such as AS levels (Broman-Fulks, Berman, Rabian, & Webster, 2004; Broman-Fulks & Storey, 2008; Smits, Berry, Rosenfield, et al., 2008; for review see Stathopoulou, Powers, Berry, Smits, & Otto, 2006). Studies have found that six supervised sessions of moderate intensity aerobic exercise performed over a 2-week period has led to decreases in AS, and in symptoms of depression and anxiety (Broman-Fulks et al., 2004; Broman-Fulks & Storey, 2008; Smits, Berry, Rosenfield, et al., 2008). Because aerobic exercise induces physiological sensations that are similar to those experienced while anxious (e.g., racing heart, perspiration), aerobic exercise can be considered as an alternate form of IE.

Running as Interoceptive Exposure

A recent study by Watt and colleagues (Watt, Stewart, Conrod, & Schmidt, 2008) pioneered the use of combining a brief group-based CBT with an IE component comprised of aerobic exercise (i.e., running). Female undergraduate participants attended three one-hour group sessions on three consecutive days. The program was designed by Watt and colleagues based on manuals that were originally developed by Conrod et al. (2000) and Harrington and Telch (1994). The first day of the intervention consisted of psycho-education, focusing on learning about anxiety, AS, panic, and the anxiety cycle. On day two participants acquired cognitive restructuring skills to better deal with anxiety sensations, consistent with established cognitive treatment for panic disorder (Craske & Barlow, 2001). On the third day, participants engaged in a 10-minute group run. They were instructed to focus on the physiological sensations while running, and to compare these to anxiety-related sensations. Participants were then instructed to complete ten 10-minute running trials individually during the following 10 weeks. Following every running trial, participants completed a measure assessing cognitive (i.e., catastrophic thoughts), affective (i.e., feelings of anxiety) and somatic (i.e., physiological sensations) reactions during running.

The intervention was successful in decreasing AS levels (Watt et al., 2008). In addition, high AS, but not low AS, participants experienced decreases in cognitive and affective reactions to the running (Sabourin et al., 2008; see Study 2 in Chapter 4 of the current dissertation). On the other hand, somatic reactions decreased for both the high and low AS women. Because decreases in cognitive and affective reactions, as well as in AS levels, were unique to high AS individuals, both a cognitive and an affective mechanism of action can be postulated for the intervention’s therapeutic effects. Although the study found decreases in both responsivity to the IE trials and in AS levels, the study did not assess whether these changes were in fact related.

The Present Study

The present process study was part of a larger outcome study (Sabourin, Stewart, Watt, & Krigolson, 2012; see Study 4 in Chapter 8 of the current dissertation) that sought to replicate and extend results from the previous Watt et al. (2008) study. The larger outcome study's intervention remained unchanged from the original study. However, because of the theoretical importance of IE in decreasing AS levels, we reasoned that a more extensive running IE homework would help maintain gains and enhance the intervention's efficacy. The IE portion was therefore expanded from 10 running trials over a period of 10 weeks, to three trials per week over a period of 14-weeks, for a total of 42 trials. High AS female undergraduates who participated in the CBT plus IE intervention arm experienced decreases in AS, and in stress, depression, and anxiety symptoms (Sabourin et al., 2012; see Study 4 in Chapter 8 of the current dissertation). The present study's aim was to further elucidate the processes through which these changes occurred.

First, we sought to replicate and extend findings of changes in cognitive and affective reactions to running specifically for high AS participants using results from the additional running trials. Second, we aimed to discover whether changes in reactions to running were related to changes in AS levels, in order to further strengthen the evidence that cognitive and affective changes resulting from repeated IE trials are instrumental in decreasing AS levels.

Consistent with Sabourin et al. (2008; see Study 2 in Chapter 4 of the current dissertation), it was hypothesized that there would be decreases over time in affective and cognitive reactions to the IE trials for high AS, but not low AS, participants. Also consistent with Sabourin et al., it was hypothesized that both high and low AS participants would experience changes in somatic sensations as a result of repeated exposure to running. Finally, previous findings from Sabourin et al. support cognitive and affective changes in reactions to IE as driving forces for reductions in AS levels. Additionally, AS has been conceptualized both as an affective construct denoting fear, and as a cognitive construct denoting beliefs regarding arousal sensations (McNally, 1999). Because high levels of AS are associated with elevated levels of fear and of maladaptive beliefs about arousal sensations, it was expected that decreases in both affective and cognitive reactions to the IE exercises would be correlated with decreases in AS. However, decreases in somatic reactions to IE were not expected to be correlated with decreases in AS.

Method

Participants

A total of 154 female undergraduate students participated in the larger outcome study (Sabourin, Stewart, Watt, & Krigolson, 2012; see Study 4 in Chapter 8 of the current dissertation), of which this process study is a part. Only women were recruited for the present study to control for sex effects, and because women have been found to have higher AS levels than men (Stewart, Taylor, & Baker, 1997). Also, we were replicating a previous study that had also used only female participants (i.e., Sabourin et al., 2008; see Study 2 in Chapter 4 of the current dissertation). Participants were recruited based on scoring one SD above or below the mean score for university women (i.e., 18 +/- 7; see Watt et al., 2008) on the Anxiety Sensitivity Index (ASI; a widely used measure of AS; Peterson & Reiss, 1992; see Appendix A for selected items). Any potential participants with health conditions or concerns (e.g., chest pains) that might preclude them from safely engaging in the running portion of the intervention were excluded from the study. Participants were randomly assigned to either the CBT intervention or a health education control (HEC) condition, resulting in four separate groups: high AS/CBT (n = 44), high AS/HEC (n = 37), low AS/CBT (n = 39), low AS/HEC (n = 34). Participants in the two conditions did not differ significantly in AS level or age (see Sabourin, Stewart, Watt, & Krigolson, 2012; Study 4 in Chapter 8 of the current dissertation). A total of 125 participants completed all three days of the interventions (i.e., 81% completion rate). There were no differences between participants who completed the three day-intervention (N=125) and those who did not (N=29) on initial ASI scores, F (1,148) = 0.10, p = NS, nor on age, F (1,150) = 3.55, p = NS.

Only participants in the CBT group who completed the three-day intervention (n = 63) were instructed to engage in the IE running trials. Ten participants completed all 42 trials. A visual inspection of HVQ-B scores over time revealed that minimal decreases in scores occurred after the first 20 trials. Because nearly all of the benefits from IE occurred during the first 20 trials, and few participants completed all 42 trials, treatment completion for the current study was determined by having completed at least 20 running trials.

A total of 28 participants (10 low AS and 18 high AS) handed in completed HVQ-Bs for 20 running trials; these were the participants for the current process study[19]. Participants ranged in age from 17-23 years (M =18.89, SD = 1.59). Most (82.1%) were Caucasian. A 2 (AS group: high AS, low AS) x 2 (completed 20 trials: yes, no) between-

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[19] Although the number of participants who handed in at least 20 HVQ-B sheets is relatively low, it is possible that more participants completed the IE trials but did not hand in the completed questionnaires.

subjects ANOVA performed on pre-intervention ASI scores revealed a main effect of AS group, $F(1,56) = 385.51$, $p < .001$, but no significant main effect of completion, $F(1,56) = 0.12$, p = NS, and no significant interaction, $F(1,56) = 0.12$, p = NS. Thus, there were no differences in initial ASI scores between participants who completed all 20 trials and those who did not. Another 2 (AS group: high AS, low AS) x 2 (completed 20 trials: yes, no) between-subjects ANOVA was performed on participant age. There were no significant main effects or interaction (ps > .10), demonstrating that there were no age differences between groups. A 2 (race: Caucasian, other) x 4 (group: high AS/completed 20 trials, high AS/did not complete 20 trials, low AS/completed 20 trials, low AS/did not complete 20 trials) chi-square analysis demonstrated that there were no race differences between groups, $\chi^2(3) = 4.08$, p = NS.

Measures

Anxiety Sensitivity Index (ASI; Peterson & Reiss, 1992). The ASI is a widely used measure assessing fear of arousal sensations (see Appendix A for selected items). It consists of 16-items (e.g., “It scares me when my heart beats rapidly”), each scored on a 5-point Likert scale ranging from “very little” (scored as 0) to “very much” (scored as 4). The ASI has shown excellent psychometric properties (Peterson & Reiss, 1992). Pre-intervention internal consistency was high in the current study ($\alpha = .93$).

Hyperventilation Questionnaire-Brief (HVQ-B; Sabourin, Stewart, Watt, & MacDonald, 2012; see Study 3 in Chapter 6 of the current dissertation). The HVQ-B is an eighteen item measure that assesses reactions to physiological arousal (see Appendix J). It consists of three separate subscales: 1) cognitive (e.g., “worried about damaging health”), 2) affective (e.g., “anxiety”), and 3) somatic (e.g., “dizziness). Each item is rated on a 4-point Likert scale ranging from “not at all” (scored as 0) to “markedly” (scored as 4). The HVQ-B is a brief version derived from the HVQ (Rapee & Medoro, 1994). Both scales have shown strong psychometric properties in university samples, including strong internal consistency, test-retest reliability, and construct validity (Rapee & Medoro, 1994; Sabourin, Stewart, Watt, & MacDonald, 2012; see Study 3 in Chapter 6 of the current dissertation). Internal consistency as assessed during the first running trial in the present study was high with alpha coefficients of .84 for the cognitive subscale, .83 for the affective subscale, and .89 for the somatic subscale.

Procedure

Participants in the CBT plus IE condition participated in three one-hour interactive group sessions on three consecutive days. The CBT intervention protocol was identical to the one described above that was used in the previous Watt et al. (2008) study. Briefly, the intervention consisted of psychoeducation, cognitive restructuring, and the group IE running component. Following the group run, participants discussed how running sensations were similar to anxiety-related sensations, and completed the HVQ-B. Participants were then instructed to run on their own, three times per week, for a period of 14 weeks, and to complete the HVQ-B after every running trial.

Results

Decreases in AS Levels

Scores on the ASI collected prior to the intervention and 10-weeks following the 3-day intervention were analysed separately for those high and low AS participants in the present study using paired-sample t-tests. As expected, high AS participants experienced a significant decrease in ASI scores, $t(17) = 2.25$, $p < .05$. Low AS participants, on the other hand, did not experience a significant decrease in ASI scores, $t(9) = 1.71$, p = NS.

Changes in Reactions to IE Running Trials

To confirm that there was a difference in initial responses to the running trials between high AS and low AS participants, between subjects t-tests were performed on reaction scores from the first IE homework trial for each subscale. As expected, high AS participants had higher initial reactions than low AS participants for the cognitive subscale, t(26) = 2.99, $p < .01$, the affective subscale, $t(26) = 3.26$, $p < .005$, and the somatic subscale, $t(26) = 2.70$, $p = .01$ (see Figure 10-1, trial 1 scores).

The primary goal of the present study was to replicate and extend previous research findings (Sabourin et al., 2008; see Study 2 in Chapter 4 of the current dissertation) demonstrating that high AS participants, but not low AS participants, experienced decreases in cognitive and affective reactions to the running trials over time. The present study also sought to replicate the finding that both high and low AS participants experienced decreases in somatic sensations over time. Therefore, trend analyses were conducted separately for high and low AS participants for the three HVQ-B subscales.

Results indicated that high AS participants experienced a decrease in cognitive reactions to running over time, as reflected by a significant linear effect of time on the cognitive subscale, $F(1,17) = 9.90$, $p < .01$. On the other hand, cognitive reactions did not decrease over time for low AS participants, as reflected by a non-significant linear effect of time on the cognitive subscale, $F(1,9) = 0.14$, p = NS (see Figure 10-1A). High AS participants also experienced a significant decrease in affective reactions to running over time, as reflected by a significant linear effect of time on the affective subscale, $F(1,17) = 4.67$, $p < .05$. Unexpectedly, there was a marginally significant linear effect of time on the affective subscale for low AS participants, $F(1,9) = 4.81$, $p = .06$ (see Figure 10-1B). High AS participants experienced a significant decrease in somatic reactions over the 20 trials, as reflected by a significant linear effect of time on the somatic subscale, $F(1,17) = 9.12$, $p < .01$. There was a marginally significant linear effect of time on the somatic subscale for low AS participants, $F(1,9) = 3.57$, $p = .09$ (see Figure 10-1C).

Changes in Reactions to IE Running Trials and in AS Levels

In order to determine whether changes in HVQ-B subscales were related to changes in ASI scores, three separate HVQ-B subscale change scores and an ASI change score were computed. First, HVQ-B scores from the first three running trials were combined as an index of early scores, and scores from the last three trials (i.e., trials 18-20) were combined as an index of later scores. These combinations also helped correct for potential random variability arising from any one particular running trial. The later scores were then subtracted from the earlier scores, resulting in a change score. The first and 20^{th} running trials ranged from 23 to 65 days apart, with a mean of 44 ($SD = 10$). Next, post-treatment ASI scores, which were collected between 70 and 80 days following the beginning of the intervention, were subtracted from pre-treatment ASI scores. The 10-week post-treatment ASI scores were used for the current analyses as they were the first ASI assessment following the 20^{th} running trial. HVQ-B cognitive change scores and affective change scores were both significantly associated with ASI change scores. On the other hand, HVQ-B somatic change scores were not associated with ASI change scores (see Table 10-1). Furthermore, cognitive and affective changes were strongly related with a correlation of *r* = .71, much higher than correlations between these two dimensions and the somatic dimension. These results suggest that decreases in cognitive and affective, but not in somatic, reactions may play a part in the resulting decreases in AS.

Discussion

The current study's aim was to further explore the role of running as the IE component of a brief CBT for high AS women, by replicating and extending results from a previous intervention (Sabourin et al., 2008; see Study 2 in Chapter 4 of the current dissertation). As a result of repeated exposure to the running IE component of the intervention, high AS participants, but not low AS participants, experienced a decrease in catastrophic cognitions about running. That is, AS moderated the relationship between IE exposure and decreases in cognitive reactions. This result is consistent with results from our previous study (Sabourin et al., 2008; see Study 2 in Chapter 4 of the current dissertation). As expected, high AS participants also experienced decreases in both affective and somatic reactions to running over time. Unexpectedly, marginal decreases in affective scores suggest that low AS participants may have experienced a decrease in affective reactions over time. Also unexpectedly, there was only a marginal decrease in low AS participants' scores on the somatic subscale. These results are partly consistent with results from our previous study (Sabourin et al., 2008; see Study 2 in Chapter 4 of the current dissertation), which found decreases in affective reactions to running only in high AS participants, and decreases to somatic reactions to running in both high and low AS participants. The marginal decrease in affective reactions for low AS participants in the current study may be due in part to the expanded IE component. By extending the running trials from 10 to 20, low AS participants may have had an opportunity to experience a small decrease in even the slightest fearful reaction to running. Also, perhaps marginal effects occurred for both affective and somatic reactions due to insufficient power to detect significant changes in reactions to running in the low AS group, which consisted of only 10 participants.

The current study also extended results from the previous study by revealing that changes in cognitive and affective responses to, but not in somatic sensations arising from, the IE exercises, were related to changes in AS. This result suggests that the therapeutic effects of repeated exposure to running in decreasing sensitivity to anxiety-related sensations are not dependent on decreasing the experience of somatic sensations themselves. Rather, they depend on altering the meaning of (i.e., cognitive) and emotional reaction to (i.e., affective) these sensations.

Results from the current study and the Watt et al. (2008; Sabourin et al., 2008; see Study 2 in Chapter 4 of the current dissertation) studies lend further support to the possibility that repeated exposure to physiological sensations may result in decreases in AS through two possible mechanisms. First, decreases in cognitive catastrophizing related to arousal sensations drive AS reduction. Second, the repeated exposure itself leads to a decrease in fear, through a process of extinction learning. These two mechanisms are not mutually exclusive, and could in fact be operating in tandem. Alternatively, there might be two distinct types of high AS individuals who experience the therapeutic benefits of exposure through different processes. Future research could further examine these alternative explanations.

Limitations and Future Directions

The current study's limitations included having participants complete the running IE component of the intervention unsupervised. Although participants were explicitly instructed to focus on the physiological sensations and to refrain from listening to music while engaging in the IE component, it is possible that they used some sort of distraction. Distractions, such as music, limit opportunities for learning that the sensations are in fact harmless (Foa & Kozak, 1986). Also, it was not possible to control the intensity of the running IE homework. It is possible that some participants, especially high AS participants, engaged in subtle avoidance by running at an intensity that would not induce a sufficient level of physiological arousal to be effective exposure. Future research could have participants run supervised, using individualized measures of intensity (e.g., measures of ventilator or lactate threshold), in order to objectively assess physical exertion (e.g., Parfitt, Rose, & Burgess, 2006; Welch, Hulley, Ferguson, & Beauchamp, 2007).

A substantial number of participants did not complete all homework IE trials. Perhaps more individualized contact, including supervised or group-based running trials, after the initial intervention, would have increased adherence to the homework portion of the study. Previous studies that have included an intervention consisting of unsupervised physical exercise have also experienced a significant amount of non-completion (e.g., Broocks et al, 1998). Although non-completers did not differ from participants who did complete the trials on initial ASI scores, on age, or on race, it is possible that they differed on other theoretically relevant variables that were not assessed in the current study.

The current study's participants were a fairly homogeneous community sample of mostly young, Caucasian, female undergraduate students. Future research could test the brief CBT plus IE intervention on diverse groups, including clinical populations, men, and individuals from other ethnic or racial groups. On the other hand, mean ASI scores for high AS participants in the current study were in the clinical range, comparable to scores of individuals with panic disorder (e.g., Meuret et al., 2010).

Conclusion

In conclusion, the current study strengthened the evidence base for the efficacy of repeated, brief, physical exercise trials as an IE component of a brief group-based CBT for decreasing AS. Moreover, study findings suggest that decreases in maladaptive cognitions about the meaning of physiological arousal, as well as extinction learning resulting from repeated exposure to physiological arousal, are instrumental in decreasing both aversion to arousal sensations and AS.

Table 10-1.

Correlations between Hyperventilation Questionnaire-Brief (HVQ-B) and ASI Change Scores

	Cog Change	Aff Change	Som Change
Cog Change	-----		
Aff Change	.71**	-----	
Som Change	.46*	.44*	-----
ASI Change	.46*	.42*	.04

Note. ASI = Anxiety Sensitivity Index; Cog Change = Change on HVQ-B Cognitive subscale between first three and last three trials; Aff Change = Change on HVQ-B Affective subscale between first three and last three trials; Som Change = Change on HVQ-B subscale between first three and last three trials; ASI Change = Change in ASI scores from Pre-treatment to the 10-week follow-up.
* $p < .05$; ** $p < .001$.

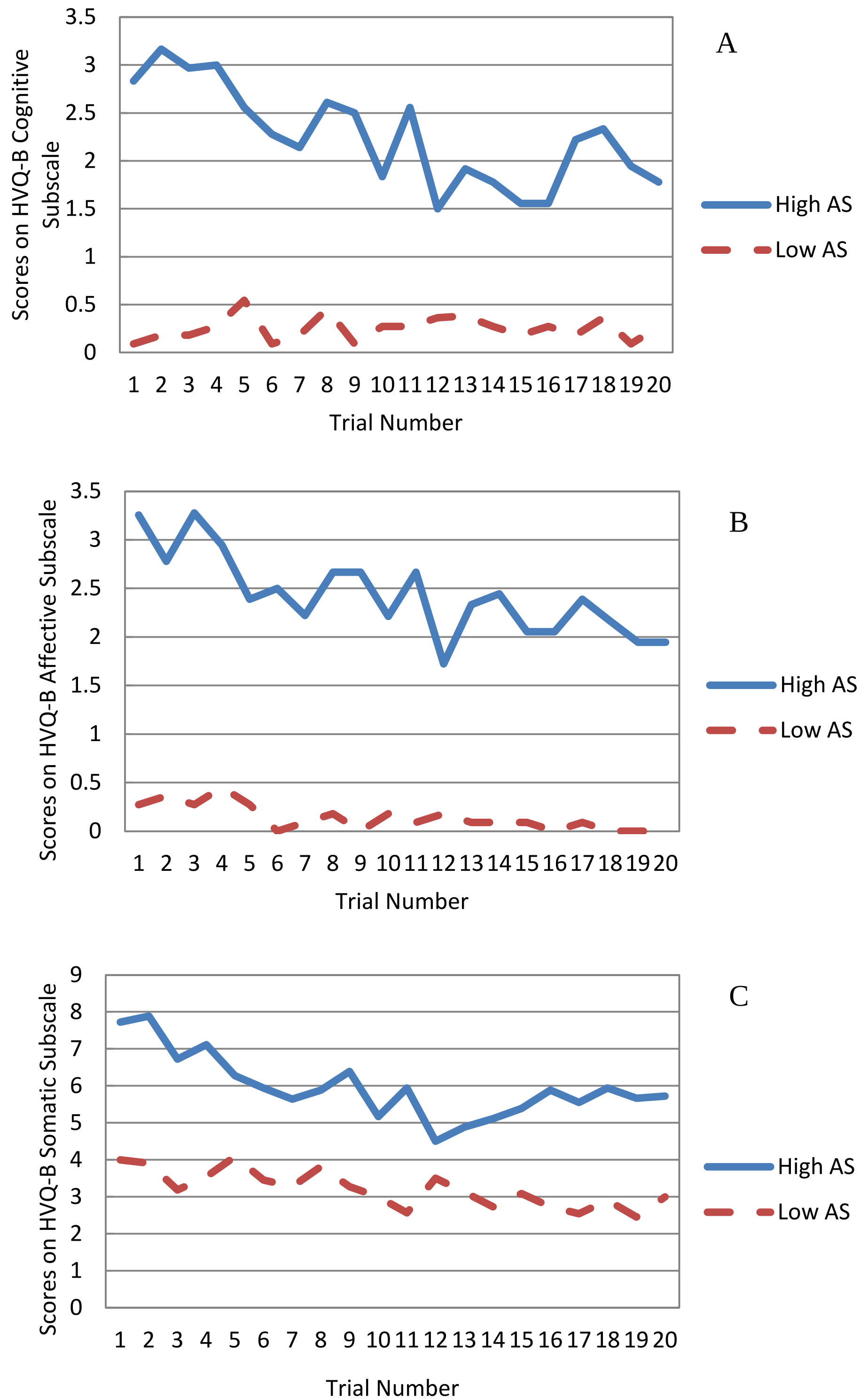

Figure 10-1. HVQ-B Cognitive, Affective, and Somatic subscale scores over time. AS = anxiety sensitivity; HVQ-B = Hyperventilation Questionnaire-Brief

Chapter 11 . GENERAL DISCUSSION

Review of the Objectives of the Dissertation

The current dissertation research had four main objectives. These were: 1) to further explore the relationships between AS levels and exercise/ fitness levels, including examining perceived benefits of and barriers to exercise in high and low AS individuals (Study 1), 2) to investigate the possible mechanisms of action of a brief CBT plus running IE intervention's AS reducing effect, by examining changes in reactions to the IE portion of the CBT/IE intervention (Study 2), 3) to create and evaluate a brief version of a measure assessing state-level reactions to physiological arousal, the HVQ-Brief (HVQ-B), in order to increase the ease with which the measure can be used for research and/or clinical practice (Study 3), and 4) to replicate and extend an existing CBT/IE intervention study by creating a more stringent control condition and expanding the running IE portion of the intervention, examining both the longer-term effects on AS levels and emotional distress (Study 4), and the mechanisms of action for the intervention (Study 5). Each of the five stand-alone publication-style manuscripts included its own discussion section. The general discussion section below will include a brief overview of the main novel findings of the current dissertation research, followed by an examination of some of the key clinical implications of this research. Finally, strengths and limitations of the dissertation, along with some areas for future research will be discussed.

Main Novel Findings of this Program of Research

Study 1 found that high AS female undergraduate students self-reported engaging in less physical exercise, and perceived themselves as less fit, than low AS female undergraduates. This finding supports previous research in the area (Smits & Zvolensky, 2006; Goodin, 2009). It also strengthens the evidence demonstrating an inverse relationship between AS levels and exercise, given that some studies (e.g., McWilliams & Asmundson, 2001) had only found partial support for the inverse AS level/physical exercise relationship. In addition, Study 1 found that high AS individuals endorsed more barriers to exercising than low AS individuals. Surprisingly, they also endorsed *more benefits* of exercising. On the other hand, barriers to exercise mediated the relationships between AS group (i.e., high versus low AS) and exercise participation / perceived fitness levels. Thus it appears that high AS individuals are well aware of the benefits of exercising, but are engaging in less physical exercise in part because of elevated perceived barriers to exercise.

Study 2 examined changes in reactions to a running IE component of a brief CBT intervention (Watt et al., 2008). First, initial cognitive, affective, and somatic reactions and initial heart rate reactivity to running were higher in high AS versus in low AS participants. Over the course of ten 10-minute running IE trials, cognitive and affective reactions to the running IE, as well as heart rate reactivity, decreased only for high AS, but not for low AS female undergraduates. In contrast, somatic reactions to the running IE trials decreased over time for both high and low AS individuals. Findings suggest that changes in somatic reactions following repeated exposure to running may not account for decreases in AS levels for high AS individuals. Rather, it appears that changes in the meaning of (i.e., cognitive reactions) and emotional response to (i.e. affective reactions) physiological arousal may be associated with the brief intervention's therapeutic benefits, in terms of decreases in AS for high AS participants. Furthermore, decreases in heart rate reactivity specifically for high AS participants may be another sign of decreasing anxiety levels or it may also be a sign of an increase in fitness levels from participating in the running IE exercises.

Study 3 demonstrated that a brief 18-item version of the HVQ, the HVQ-B, accounted for most of the variance of the original scale. The scale had good internal consistency and construct validity as demonstrated by the ability of its three subscales to distinguish between high and low AS participants' responses to physiological arousal procedures. Furthermore, a confirmatory factor analysis (CFA) provided evidence of a reasonably good fit of the 3-factor measurement model to the HVQ-B item-level data.

Study 4 demonstrated that a replication of a brief CBT with an expanded IE component was efficacious in decreasing AS levels in high AS undergraduate women. Surprisingly, a control intervention (i.e., health education intervention) consisting of an interactive discussion on the roles of exercise, nutrition, and sleep on health also led to decreases in AS levels. Both interventions also led to decreases in depression symptoms in high but not low AS participants. Decreases in anxiety symptoms were not evident post-intervention but reached significance for all study participants by the 14-week follow-up. Only the brief CBT/IE intervention led to decreases in symptoms of general stress for high AS participants. All changes were also evident at a 14-week follow-up assessment showing persistence of the effects. Because physical exercise is the common link between the two interventions, it is possible that physical exercise is one of the driving factors behind the therapeutic effects of both interventions.

In Study 5, decreases in cognitive reactions following repeated trials of the IE portion of the brief CBT/IE intervention were specific to high AS individuals. This result was consistent with results from Study 2. High AS participants also experienced decreases in affective and somatic reactions to running, consistent with Study 2 results. Unexpectedly, there were marginally significant decreases in affective reactions for low AS participants as well. It is possible that additional IE trials resulted in some changes in affective reactions even in low AS participants. There were also marginally significant decreases in somatic sensations in low AS participants. Furthermore, both cognitive and affective, but not somatic, changes to the IE component were correlated with changes in AS levels. The study further supports the hypothesis that decreases in catastrophizing the meaning of (i.e., cognitive responses), and in experiencing a negative emotional reaction to (i.e., affective responses) IE trials are associated with the brief CBT/IE's therapeutic effects in decreasing AS levels.

Taken together, it appears that perceived barriers are important deterrents to exercising for high AS individuals. A brief CBT intervention that incorporates physical exercise (i.e., running) as the IE component decreases AS levels, as well as levels of stress, depression, and anxiety in high AS undergraduate women. When engaging in physical exercise as an IE component of a brief CBT intervention, changes in affective and cognitive responses to IE appear to be related to the therapeutic benefits of the intervention. Additionally, an alternative health education intervention that includes promoting health behaviours, including physical exercise, and addressing potential barriers to engaging in health behaviours like exercise, also results in decreases in AS levels and in depression and anxiety symptoms. Both interventions address, in very separate ways, barriers to engaging in physical exercise. That is, the brief CBT targets high AS individuals' fear of arousal sensations, whereas the health education intervention allows participants to openly engage in problem-solving various barriers to exercise.

Irrespective of these differences, it is possible that the therapeutic effects of the interventions lie, at least in part, in the interventions' effects on changing participants' attitudes toward, and participation in, aerobic exercise. These changes might be partly responsible for decreases in the fear of physiological arousal generally, and also decreases in symptoms of depression and anxiety.

Clinical Implications of this Program of Research

Clinical implications of this program of research have been discussed in each of the publication-style manuscripts. A few of the more salient clinical implications will be discussed here. First, the current program of research increased our understanding of the complicated relationship between AS levels and physical exercise. Given the fact that AS is a risk factor for mental health difficulties (Schmidt et al., 2006), and that physical exercise confers numerous mental health benefits (Stathopoulou et al., 2006), it is even more critical for high AS, compared with low AS, individuals to engage in physical exercise. Furthermore, it appears that for high AS individuals, exercise confers even more pronounced protective effects than for low AS individuals (e.g., Smits et al., 2011). Unfortunately, the first study of the dissertation found that high AS individuals are more likely to avoid engaging in physical exercise than low AS individuals. High AS individuals, therefore, may be missing some of the many mental health benefits of exercising. Reasons behind lower levels of physical exercise in high AS individuals have been relatively speculative. Because aerobic exercise commonly produces some of the physiological sensations feared by high AS individuals, it is possible that high AS individuals avoid physical exercise in order to avoid experiencing these sensations.

Alternatively, it is also possible that individuals who avoid exercising are more likely to develop high levels of AS due to lack of exposure to physiological arousal.

Study 1 of the current dissertation examined possible reasons why high AS individuals might avoid engaging in exercise, by exploring perceived benefits of and barriers to exercising in high and low AS individuals. First, it appears that high AS individuals do not engage in less exercise because they fail to see the benefits of exercising. In fact, they acknowledge the benefits of exercising even more than their low AS counterparts. Rather, they appear to perceive high levels of barriers to exercise. Consequently, touting the benefits of exercise may not be the most effective way to increase physical exercise participation in high AS individuals. Rather, addressing barriers to exercise is paramount in promoting physical exercise in this population. Barriers to exercise can exist in a number of different domains, such as cognitive, social environment, physical environment, and demographics (S. A. Brown, 2005). Cognitive variables might be an appropriate domain to target, as cognitions are more amenable to change on an individual basis than other types of domains such as demographics or the physical environment (S. A. Brown, 2005).

Targeting fear of physiological arousal may be one way to address some of the cognitive barriers to exercise. The brief CBT/IE intervention delivered in Studies 2, 4 and 5 of this dissertation was a combination of a traditional CBT intervention used for anxiety, panic disorder and related difficulties plus the novel and promising intervention of physical exercise as IE. Having an IE component comprised of physical exercise can provide valuable learning in more than one way. First, participants were asked to focus on the physiological sensations they were experiencing while running and compare these with the sensations associated with being in an anxious state. The fact that they experienced physiological arousal sensations repeatedly, without experiencing adverse effects, may have helped them learn that physiological arousal sensations, in general, are not harmful. The goal of this was to reduce AS levels. Second, although this was not a direct goal of the intervention, participants also learned that the physiological arousal sensations specifically related to exercise are not harmful, but might in fact have some positive results, such as improved affect following the exercise. This might also indirectly target cognitive barriers to exercise. Thus, pairing a CBT/IE intervention with exercise, by making the IE portion of the CBT intervention physical exercise, has the potential to target both AS levels and physical inactivity.

Study 4 provided unexpected and new findings regarding the benefits of an interactive discussion on health in decreasing AS and emotional distress. The health education intervention also included a focus on decreasing barriers to health behaviours, including physical exercise. As opposed to focusing specifically on potential discomfort of physiological sensations, as was done in the CBT/IE intervention, the health education intervention discussed more general types of barriers. For example, topics such as finding time to exercise despite busy schedules, and dealing with adverse weather conditions, were discussed. Thus, one common link between both interventions was addressing barriers to exercise, either indirectly with the CBT/IE, or directly with the health education intervention. Future studies could follow participants for an extended period of time in order to determine whether high AS individuals participating in either a health education or a CBT/IE intervention are indeed more likely to increase their participation in aerobic exercise.

Concluding Statement on Clinical Implications

According to Health Canada, 20% of Canadians will experience mental illness in their lifetime, resulting in a yearly economic cost estimated at over $7 billion (Public Health Agency of Canada, 2002). Furthermore, it has been suggested that targeting a common risk factor for prevention of multiple forms of psychopathology is likely more cost-effective than targeting different risk factors for each form of psychopathology (Feldner, Zvolensky, & Schmidt, 2004). Because AS has been associated with a range of Axis I psychopathology, and other health-related issues, targeting AS levels would certainly follow this recommendation.

The accumulated evidence supporting both the mental and physical health benefits of exercise has led to recent physical activity guidelines in Canada and across a number of other countries (Colley et al., 2011; Warburton, Nicol, & Bredin, 2006). The new guidelines recommend that adults engage in at least 150 minutes per week of moderate or vigorous physical activity, accumulated in stretches of at least 10 minutes (Warburton, Nicol, & Bredin, 2006). A recent epidemiological study conducted by Statistics Canada used accelerometers to measure physical activity participation in adult Canadians (Colley et al., 2011). Results revealed that only 15% of Canadians meet the guidelines. Furthermore, as Canadians get older, they tend to exercise less, and are less likely to meet the guidelines (Colley et al., 2011). Thus, interventions such as the CBT/IE or health education intervention whose aims are to 1) target emotional distress, 2) prevent future anxiety and other disorders, and 3) increase physical exercise participation, have immense potential benefits.

Strengths and Limitations of Current Program of Research

Strengths

One of the strengths of the current dissertation research was the fact that there were few exclusion criteria for potential participants. Other than scoring either one SD above or below mean AS levels, the only exclusion criterion was a medical condition precluding safe participation in the exercise portion of the intervention. No participant was excluded for the presence of psychopathology. Participants' AS levels (*M*s = 34.2 for Study 2 and 35.5 for Study 4) were also similar to levels seen in individuals with panic disorder (Meuret, Rosenfield, Seidel, Bhaskara, & Hofmann, 2010). Also, almost half (39/81) of the high AS participants in Study 4 had experienced at least one panic attack, and fifty percent of these had experienced at least one unexpected panic attack. Thus, it appears that a number of high AS participants may have been experiencing symptoms in the clinical range, increasing the generalizability of the current dissertation's results to the general population where high AS individuals are more likely to experience panic attacks and Axis I psychopathology (e.g., Taylor et al., 1992).

Another strength of the dissertation was that it was the first to specifically identify perceived benefits and barriers to exercise in high versus low AS individuals. It is important to determine factors affecting health behaviours, including exercise, in specific at-risk populations, especially ones that are at risk of both exercise avoidance and psychopathology. This helps better target interventions to these populations. Extrapolating information from general populations and applying it to specific at-risk groups might provide erroneous information about that group. For example, one study conducted with 398 college undergraduate students examined perceived benefits and barriers to exercise using a slightly different, but conceptually similar, scale, the Exercise Benefits and Barriers scale (S. A. Brown, 2005). The author found that benefits were associated with physical exercise levels, but that barriers were not associated with exercise levels, even for women. Furthermore, a number of research studies have found that benefits to exercise are significant predictors of exercise participation (Marshall & Biddle, 2001). The unique role of barriers to, but not benefits of, exercising, in predicting exercise participation for high AS individuals, therefore, might have been overlooked had results from general populations been extrapolated to this specific group.

An additional strength of the dissertation study was the inclusion of a longer, 14-week follow-up to the interventions in Study 4. The fact that benefits from both of the 3-day interventions were maintained over several months is encouraging. Future research should extend the follow-up period even more in order to determine if benefits are sustained over even longer periods of time (e.g., one year or more).

University-aged individuals are an important target population for research focused on both prevention of mental illness and physical exercise. First, late adolescence and early adulthood are the ages at which individuals are most at risk of developing mental illness (Public Health Agency of Canada, 2002). Second, university is a time of many transitions, one of which is the end of mandatory high-school physical activity. Furthermore, it has been shown that participation in high school sports has little influence on later participation in physical exercise. On the other hand earlier *adult* leisure physical exercise predicts later adult leisure physical exercise participation (Dishman, Washburn, & Heath, 2004). Additionally, epidemiological data reveal that individuals are less likely to engage in physical exercise as they become older (Colley et al., 2011). Thus, interventions that target university-age individuals at a time when they are beginning to form life-long habits, and are at increased risk of developing mental illness, can be an effective way to influence these individuals' long-term health. Although future research needs to be conducted with different populations as well in order to generalize the findings from the current dissertation, there is merit to examining these interventions' effects on individuals at this important junction in their lives.

Furthermore, studying AS levels and their associations with emotional distress and physical exercise in women specifically is beneficial for a number of reasons. First women tend to have higher AS levels than men (Stewart, Taylor, & Baker, 1997). Second, AS levels are more closely associated with psychopathology in women than in men (Olatunji & Wolitzky-Taylor, 2009). Third, women are less physically active than men (S. A. Brown, 2005; Huang et al., 2003). Fourth women experience a greater prevalence of anxiety and depression than men (Kessler, McGonagle, & Zhao, 1994; Regier, Narrow, & Rae, 1990). Thus, testing an intervention for women not only provides a good starting point, but is also an important target in and of itself.

Limitations

The present dissertation research used self-report measures of AS, emotional distress, physical exercise participation, and fitness levels. Future research should use different markers of these constructs to further strengthen the current dissertation's results. Because of past research implicating responsivity to biological challenge tasks as a distinguishing feature of AS, behavioural measures of fear of arousal sensations could include responsivity to a biological challenge task as an outcome measure (cf., Schmidt et al., 2007). Additionally, implicit measures of AS, such as the Stroop task (cf., Stewart, Conrod, Gignac, & Pihl, 1998), dot-probe task (cf., Keogh, Dillon, Georgious, & Hut, 2001,), or an implicit association task (cf., Lefaivre, Watt, Stewart, & Wright, 2006) provide complimentary information to self-report measures by assessing earlier stages of information processing. In fact, a previous study (Lefaivre et al., 2006) found that high AS individuals responded faster to trials on an implicit association task (i.e., the extrinsic affective Simon task; EAST; De Houwer, 2003) that paired anxiety-related symptoms (e.g., breathless) with harmful (e.g., suffocate), versus harmless (e.g., benign) consequences. These results suggest that an implicit association exists in memory between anxiety-related symptoms and harmful consequences for high AS individuals. Future research could employ these types of implicit tasks as outcome measures for the interventions from Study 4 of the current dissertation (cf., Teachman & Woody, 2003). This would help determine whether the interventions are also are able to weaken automatic associations in memory between anxiety-related symptoms and negative consequences, for high AS individuals.

Future research could also employ objective methods to assess physical fitness levels and physical activity participation. Physical fitness is most commonly associated with cardiovascular fitness, which is defined as the maximal oxygen consumption while exercising at maximum capacity ($V0_2$ max; Shephard et al., 1968). $V0_2$ max can be assessed either directly, or inferred through maximal or submaximal effort tests using protocols that involve either walking, running, or cycling on bicycle ergometers. Because high AS individuals may be disinclined to exercise until maximal effort due to discomfort with physiological arousal, submaximal rather than maximal exercise tests would most likely be recommended.

Physical activity participation could be measured using ecological momentary assessments with instruments such as heart rate monitors and accelerometers, which measure both the frequency and intensity of movement (cf. Dunton, Whalen, Jamner, Henker, & Floro, 2005). Objective measures of the intensity of physical exercise participation would help elucidate whether high AS individuals are more likely to avoid participating in higher intensity exercise specifically, which is more likely to mimic the types of arousal sensations associated with being in an anxious state. For example, perhaps high AS individuals are as likely as low AS individuals to engage in leisurely walking, but would be less likely to engage in vigorous running.

If using retrospective self-report measures of physical exercise, future research could employ one or more of several ways to reduce retrospective bias. Participants could be instructed to record their activity levels at the end of each day for a period of time. Alternatively, it might be possible to adapt a tool such as the Time-Line Follow-Back (TLFB), which has been used extensively in alcohol research (Sobell, Maisto, Sobell, & Cooper, 1979). The TLFB has also been adapted to other behaviours such as gambling (i.e., Gambling Timeline Followback; G-TLFB; Weinstock, Whelan, & Meyers, 2004). Similar to the way the TLFB and G-TLFB are used with alcohol consumption and gambling, participants could be presented with a calendar and asked to recall their estimates of daily physical exercise over a specified period of time in relation to specific memory anchor points to improve recall.

Changes in exercise participation were not assessed in Study 4 of the current dissertation. Future research could use the methods described above to determine whether the interventions were indeed effective in increasing exercise participation in high AS participants. Additionally, mediation analyses could be performed to assess whether changes in exercise participation might account for some of the therapeutic effects of the interventions in terms of decreases in AS and in psychological distress in high AS participants.

Finally, because the current dissertation was conducted with non-clinical female undergraduate students, generalizability to other populations cannot be assumed. Future research should be conducted with samples comprised of men, participants of different ages, and clinical populations, to confirm that findings from the current dissertation would apply to these diverse populations.

Areas for Future Research

Barriers to Exercise

The current dissertation ascertained that high AS women endorse more barriers to exercise than low AS women. It would be useful to better understand the kinds of barriers most applicable to high AS individuals. Because high AS individuals fear arousal-related sensations, barriers might be related to discomfort with physiological arousal associated with exercise in particular. Alternatively, or additionally, because AS is related to negative emotionality (e.g., Cox et al., 2008), it is possible that high AS individuals are more prone to see the negative aspects to exercise, resulting in higher endorsement of barriers in general. As was discussed above, barriers to exercise can exist in a number of domains, such as individual, social, or environmental (S. A. Brown, 2005). Future research could further explore barriers, either using qualitative types of research or more extensive quantitative measures. For example, the longer 48-item Benefits and Barriers to Exercise scale (Myers & Roth, 1997) includes 24 barrier items, which are classified into four distinct subscales: time-effort, physical effects, social, and specific obstacles. It would be expected that high AS individuals would score higher on the physical effects subscale, but this remains to be tested empirically. Better understanding barriers to exercise in high AS individuals is an important step towards developing and refining interventions, such as the CBT/IE and the health education intervention from the current dissertation, to address the barriers that exert the most influence on high AS individuals' exercise behaviours.

Prevention of Mental Health Disorders

Previous studies have shown that a single CBT treatment session with at-risk high AS individuals resulted in a reduced incidence of panic disorder diagnoses at follow-up (Gardenswartz & Craske, 2001; Schmidt et al., 2007). Future research examining the current dissertation's two interventions needs to be conducted with longer term follow-ups to assess their capacity for preventing panic disorder, and other Axis I psychopathology. Although both interventions resulted in significant decreases in emotional distress and AS levels, their effectiveness as preventative interventions for clinical pathology remains to be investigated.

Dose Response Relationships

Cognitive Restructuring. The CBT/IE was a very brief intervention, with the cognitive restructuring portion lasting only one hour. Future research could vary the length of the CBT/IE intervention to determine whether giving participants more of a chance to practice cognitive restructuring skills confers additional benefits. Alternatively, or in addition, occasional "booster sessions", either via telephone or in person, where participants can re-visit and further practice cognitive restructuring could also theoretically enhance the intervention’s effects. During these booster sessions, participants could also review the running homework portion with a facilitator, and discuss and trouble-shoot any compliance issues. Adding either booster sessions or review sessions might help participants consolidate learning for longer-term sustenance of gains.

Interoceptive Exposure. Because of the theoretical importance of IE in AS reduction interventions, the intervention used in Studies 4 and 5 of the current dissertation expanded the IE component from 10 running trials over a period of 10 weeks, to three trials per week over a period of 14 weeks. Few participants, however, completed all running trials, but a substantial number completed 20 of the trials. Although participants completed twice the amount of running IE trials in the current versus the previous (Watt et al., 2008) study, it was not possible to evaluate a dose-response relationship between the number of IE trials and AS reduction for a number of reasons. First, the differing assessment periods between the two studies precluded a direct comparison in intervention effects. The Watt et al. (2008; Sabourin et al., 2008) study provided information on the magnitude of effects immediately following the intervention, whereas Study 4 of the present dissertation assessed participants at 10- and 14-weeks following the intervention. Second, the number of running trials was not experimentally manipulated so any relationship between number of trials completed and therapeutic benefits could be due to extraneous factors. Future research could experimentally vary the number of IE trials to determine whether there are additional benefits conferred with expanding the IE component of the intervention.

A recent meta-analysis (Wipfli, Rethorst, & Landers, 2008) examined the possibility of a dose-response relationship between weekly exercise volume and anxiety reduction. The meta-analysis included randomized controlled trials comparing exercise interventions with either other treatments or no treatment, revealing that exercise interventions were efficacious in decreasing anxiety levels when compared with no treatment (effect size = -0.48). Exercise interventions also resulted in significantly larger decreases in anxiety than alternative treatments (e.g., yoga, meditation; effect size = -0.19). There was some indication of a quadratic relationship between exercise volume and benefits, with benefits increasing as exercise increased until a dose of 12.5kcal/kg/week, after which benefits began to decrease again. This relationship, however, was not significant. In short, the meta-analysis was unable to provide evidence for a dose-response relationship. It is possible that too few of the studies in the meta-analysis included information on dose-response (12/49), or that exercise interventions were too homogeneous in terms of dose applied, which might have prevented the meta-analysis from finding a dose-response relationship.

A recent study by Schmidt et al. (2012), described in the general introduction of the current dissertation, also failed to find evidence of a dose-response relationship between number of IE trials and AS reduction. The study found that a single session of AS reduction intervention, coupled with homework IE exercises, significantly reduced AS levels. Although the IE exercises were not comprised of physical exercise, the study did examine the dosage of IE trials in terms of the number of days the IE homework was completed, and the number of IE trials completed. Neither index of dosage was related to decreases in AS levels. Because the authors did not provide information on homework completion other than its relationship with AS levels, it is possible that there was insufficient variability in homework completion to find a relationship. Furthermore, because dose was not manipulated experimentally, the study only provided indirect evidence in support of the absence of a dose-response relationship.

Stemming from findings of the above two studies, an alternative and useful approach could be to determine the *minimum* number of trials needed to bring about meaningful changes. The studies conducted by Smits, Berry, Rosenfield, et al. (2008), and Broman-Fulks et al. (2004; Broman-Fulks & Storey, 2008), for example, suggest that considerable benefits can be accrued after only six aerobic exercise sessions. The studies, however, did not include any longer-term follow-up. Thus, future research should examine whether six exercise sessions (or possibly even less) are sufficient for longer-term benefits, or whether additional sessions over an extended time-frame are needed in order to maintain gains in AS levels and other indices of emotional distress.

Mechanisms of Action for Health Education and CBT/IE Interventions

Health Education Intervention. The health education intervention used in Study 4 was developed as a control condition, and as such, not expected to produce the observed therapeutic effects. Because of this, efforts were not made in the current dissertation research to examine possible mechanisms of action driving the health education intervention's therapeutic benefits. Future research should further explore the processes through which the intervention results in decreases in AS levels and emotional distress. For example, it is unclear whether the interactive discussion about physical exercise actually resulted in increases in exercise. This would be an important determination to make in future research. Also, it is unclear whether the intervention also produced improvements in maladaptive cognitions concerning anxiety-related sensations.

Previous research conducted with individuals with panic disorder (Bouchard et al., 1996) found that IE exercises without a specific cognitive restructuring component resulted in reductions in cognitions about the catastrophic consequences of anxiety-related sensations, as assessed by the Agoraphobic Cognitions Questionnaire (Chambless et al., 1984). It is possible that in Study 4, the discussion about exercise in the HEC intervention also produced cognitive changes in terms of the meanings attributed to physiological sensations. Future research could employ more direct tests of cognitions about physiological arousal, either by assessing cognitive reactions to a biological challenge task (cf., A. B. MacDonald, Stewart, Baker, & Skinner, 2000) or by using an implicit measure of arousal-related cognitions (e.g., EAST task; cf., Lefaivre et al., 2006). Alternatively an explicit interpretative bias measure such as the Body Sensations Interpretation Questionnaire (D. M. Clark et al., 1997; cf., Olthuis, Stewart, Watt, Sabourin, & Keogh, in press) would also be useful in assessing cognitions related to body sensations. These measures / tasks could be administered before the beginning of the intervention and then either at post-intervention and/or at longer-term follow-up assessment periods to assess cognitive changes resulting from the HEC intervention.

IE Component of CBT Intervention. An additional line of research that could be pursued would be to further explore *how* the running IE trials resulted in decreases in AS and emotional distress. The current dissertation explored the role of running specifically as an exposure technique, with resulting changes in cognitive appraisals to arousal and extinction in fear of arousal as possible mechanisms of change. On the other hand, physical exercise that has been performed *without* a specific focus on its role as exposure to feared sensations has also been effective in bringing about therapeutic changes (Broman-Fulks et al., 2004; Broman-Fulks & Storey, 2008). There have been a number of psychological and physiological theories for how exercise might confer its therapeutic benefits for anxiety and related conditions. Psychologically, exercise-induced increases in self-esteem and self-efficacy might be partially responsible for mental health benefits (Wipfli et al., 2008). Physiologically, it is possible that changes in neurotransmitter levels (e.g., serotonin, norepinephrine, endorphins), partially account for exercise's mental health benefits (Wipfli et al., 2008). One study (Wipfli, Landers, Nagoshi, & Ringenbach, 2011) found that decreases in serum blood serotonin levels partially mediated mood improvements following seven weeks of aerobic exercise compared with a stretching control condition (Wipfli et al., 2011). Decreases in blood serotonin levels are also evident following treatment with selective serotonin re-uptake inhibitors (SSRIs; e.g., Figueras et al., 1999). Alternatively, it is possible that benefits are a result of improvements in sleep patterns (Stathopoulou et al., 2006). It is still unclear to what extent these possible mechanisms might be involved in the AS-reduction effects of physical exercise. Future research could further explore these hypotheses with the current dissertation's AS-reduction CBT/IE intervention. For example, changes in self-efficacy, self-esteem, or sleep patterns could be assessed in future research. Additionally, indirect measurements of neurotransmitter levels (e.g., changes in blood serotonin levels) could further explore the roles of various neurotransmitters in exercise-induced mental health improvements.

Necessary Components for Therapeutic Benefits

The fact that both the CBT/IE and the health education interventions were found to be efficacious in the current dissertation research raises the question about the necessary or essential components of these types of interventions. The health education intervention did not include a specific cognitive restructuring component. Because interventions have induced cognitive changes even in the absence of a specific cognitive restructuring component (Bouchard et al., 1996), perhaps cognitive restructuring is unnecessary for AS-reduction interventions.

Smits, Berry, Rosenfield, et al.'s (2008) study revealed that no additional benefits were incurred as a result of adding a cognitive restructuring component to an exercise only intervention that already included a rationale for using exercise to decrease fear of arousal. The rationale given to participants about the role of exposure in decreasing AS may have been enough to induce cognitive changes in participants. In addition, after each of the six exercise sessions, participants in the exercise only condition were asked to reflect on changes in distress over the sensations throughout the exercise session. They discussed with a facilitator what they had learned from these experiences. The cognitive restructuring addition to the intervention consisted of Socratic questioning during the exercise portion addressing the items on the ASI measure most endorsed by each participant. As such, it did not appear to be much different from the exercise only arm of the study. It is possible that the brief and limited cognitive restructuring portion used in the Smits et al. study was not potent or extensive enough to produce additional benefits over and above benefits produced by the exercise plus rationale condition. Future research should examine adding more extensive cognitive restructuring components to these types of exercise interventions.

In Study 4 of the current dissertation, the CBT/IE condition included an “AS reduction” rationale for exercising, whereas the health education intervention included a “physical and mental health promotion” rationale. Future research could be conducted that would compare cognitive restructuring with these two rationales. For example, future research could include three experimental conditions: a cognitive restructuring component, an AS reduction rationale for exercise, and a health promotion rationale for exercise. Additionally, it is also possible that these different interventions are most efficacious for certain types of high AS individuals; therefore, potential moderating variables (e.g., levels of social anxiety, depression, or suffocation fear; history of panic attacks) could also be explored. Because cognitive changes would be of interest in this proposed future research, self-report measures should be supplemented by other measures of cognitive change (e.g., behavioural or implicit measures) as discussed above.

Dimensions of Anxiety Sensitivity

A newer version of the ASI, the ASI-3 (Taylor et al., 2007) separates the AS construct into its three dimensions (i.e., physical, social, cognitive) with three separate stable and psychometrically sound subscales (Osman et al., 2009; Taylor et al., 2007; Wheaton, Deacon, McGrath, Berman, & Abramowitz, 2012). Future research could employ the ASI-3 to determine the subscale most applicable to certain individuals, and tailor the interventions based on these findings. For example, for individuals who score highest on the social subscale of the ASI-3, perhaps having participants exercise in public could enhance the exposure’s effects.

Other At-Risk or Clinical Populations

Finally, future research could examine the brief CBT/IE intervention with other at-risk populations, or with individuals experiencing difficulties that are related to AS. One promising avenue to explore in future research is adapting the brief CBT/IE for individuals with chronic pain. AS has been conceptualized and validated as a risk factor for chronic pain (Asmundson, 1999; Stewart & Asmundson, 2006; Ocañez,, McHugh, & Otto, 2010). High AS individuals have reported more cognitive anxiety related to pain, more fearful appraisals of pain, and more pain-related escape/avoidance behaviours than low AS individuals (Asmundson & Norton, 1995; Ocañez et al., 2010). The brief CBT/IE intervention has previously been shown to decrease pain-related anxiety in a sample of high-AS non-clinical female undergraduates (Watt, Stewart, Lefaivre, & Uman, 2006). It is possible that targeting AS levels with an adapted brief CBT/IE intervention with individuals with chronic pain could improve these individuals' pain experiences as well.

Concluding Remarks

AS is a risk factor for mental and physical health difficulties. AS is also associated with lower levels of physical exercise and lower fitness levels. There is ample and convincing evidence spanning several decades supporting the mental and physical benefits of physical exercise (Colley et al., 2011; Folkins & Sime, 1981; Penedo & Dahn, 2005; Stathopoulou et al., 2006; Warburton et al., 2006), which unfortunately, at-risk high AS individuals may be missing. The current dissertation found that high AS individuals are influenced to avoid exercise by perceived barriers to exercise, possibly some of these arising from their discomfort with and fear of physiological arousal. A novel brief CBT/IE intervention that included repeated exposure to running, and a health education intervention that focused on the importance of physical exercise and problem-solved barriers to being physically active, were both efficacious in decreasing AS levels and emotional distress. A closer analysis of the CBT's IE component revealed that changes in cognitions about, and affective reactions to, running were related to decreases in AS. Because the exercise portion brought up physiological sensations akin to those experienced when anxious, these results suggest that altering high AS individuals' perception of exercise specifically, and physiological arousal more generally, might be key in the intervention's efficacy. Similarly, the health education intervention promoted and problem-solved engaging in physical exercise, and may have lead to decreases in AS and in emotional distress via increased exercise participation.

Given that only approximately 15% of Canadians engage in the recommended amount of physical exercise (Colley et al., 2011), developing interventions that increase the appeal of physical exercise, especially for individuals who are at risk for both exercise avoidance and mental health difficulties, is paramount. The current dissertation's findings provided an important step in this endeavour towards improving the health and well-being of high AS individuals. Future studies should build on the results of the current dissertation to better understand the role of CBT/IE and physical exercise in decreasing AS levels in high AS individuals, to help alleviate current and future difficulties experienced by this at-risk group.

References

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

Asmundson, G. J. G. (1999). Anxiety sensitivity and chronic pain: Empirical findings, clinical implications, and future directions. In S. Taylor (Ed.), *Anxiety sensitivity: Theory, research, and treatment of the fear of* anxiety (pp. 269-285). Mahwah, NJ: Lawrence Erlbaum Associates.

Asmundson, G. J. G., & Norton, G. R. (1995). Anxiety sensitivity in patients with physically unexplained chronic back pain: A preliminary report. *Behaviour Research and Therapy, 33,* 771-777.

Barlow, D. H. (2002). *Anxiety and its disorders: The nature and treatment of anxiety and panic (2nd ed.)*. New York, NY: The Guilford Press.

Baron, R. M., & Kenny, D. A. (1986). The moderator-mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology, 51*, 1173-1182.

Beck, A. T., & Steer, R. A. (1993). *Beck Anxiety Inventory Manual.* San Antonio, TX: The Psychological Corporation.

Beck, J. G., & Shipherd, J. (1997). Repeated exposure to interoceptive cues: Does habituation of fear occur in panic disorder patients? *Behaviour Research and Therapy, 35*, 551-557.

Beck, J. G., Shipherd, J. C., & Zebb, J. (1997). How does interoceptive exposure for panic disorder work? An uncontrolled case study. *Journal of Anxiety Disorders, 11*, 541-556.

Bentler, P. M. (1990). Comparative fit indices in structural models. *Psychological Bulletin, 107*, 238−246.

Bentler, P. M. (1995). *EQS 6 structural equations program manual*. Encino, CA: Multivariate Software Inc.

Bentler, P. M., Berkane, M., & Kano, Y. (1991). Covariance structure analysis under a simple kurtosis model. In E. M. Keramidas (Ed.), *Computing science and statistics (pp. 463−465)*. Fairfax Station, VA: Interface Foundation of North America.

Bentler, P. M., & Bonett, D. G. (1980). Significance tests and goodness of fit in the analysis of covariance structures. *Psychological Bulletin, 88,* 588-606.

Biddle, S. (2000). Exercise, emotions, and mental health. In Y. L. Hanin (Ed.), *Emotions in sport (pp. 267-291)*. Champaign, IL: Human Kinetics.

Birch, C. D., Stewart, S. H., Wiers, R. W., Klein, R. M., MacLean, A. D., & Berish, M. J. (2008). The mood-induced activation of implicit alcohol cognition in enhancement and coping motivated drinkers. *Addictive Behaviors, 33*, 565-581.

Bollen, K. A. (1989). A new incremental fit index for general structural models. *Sociological Methods & Research, 17*, 303−316.

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

Bouchard, S., Gauthier, J., Nouwen, A., Ivers, H., Vallières, A., Simard, S, & Fournier, T. (2007). Temporal relationship between dysfunctional beliefs, self-efficacy and panic apprehension in the treatment of panic disorder with agoraphobia. *Journal of Behavior Therapy, 38,* 275-292.

Bouton, M. E. (2002). Context, ambiguity, and unlearning: Sources of relapse after behavioral extinction. *Biological Psychiatry, 52,* 976–986.

Bouton, M. E. (2004). Context and behavioral processes in extinction. *Learning & Memory, 11*, 485-494.

Bouton, M. E., Mineka, S., & Barlow, D. H. (2001). A modern learning theory perspective on the etiology of panic disorder. *Psychological Review, 108,* 4-32.

Broman-Fulks, J. J., Berman, M. E., Rabian, B. A. & Webster, M. J. (2004). Effects of aerobic exercise on anxiety sensitivity. *Behaviour Research &Therapy, 41,* 125-126.

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

Broocks A., Bandelow, B., Pekrun, G., George, A., Meyer, T., Bartmann, U., . . . Rüther, E. (1998). Comparison of aerobic exercise, clomipramine, and placebo in the treatment of panic disorder. *American Journal of Psychiatry 155,* 603–609.

Broocks, A., Meyer, T. F., Bandelow, B., George, A., Bartmann, U., Rüther, E., & Hillmer-Vogel, U. (1997). Exercise avoidance and impaired endurance capacity in patients with panic disorder. *Neuropsychobiology, 36,* 182-187.

Brown, S. A. (2005). Measuring perceived benefits and perceived barriers for physical activity. *American Journal of Health Behavior, 29*, 107-116.

Brown, R. A., Kahler, C. W., Zvolensky, M. J., Lejuez, C. W., & Ramsey, S. E. (2001). Anxiety sensitivity: Relationship to negative affect smoking and smoking cessation in smokers with past major depressive disorder. *Addictive Behaviors, 26,* 887–899.

Carter, M. M., Marin, N. W., & Murrell, K. L. (1999). The efficacy of habituation in decreasing subjective distress among high anxiety-sensitive college students. *Journal of Anxiety Disorders*, *13*, 575-589.

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*, 505-518.

Carter, M. M., Suchday, S., & Gore, K. L. (2001). The utility of the ASI factors in predicting response to voluntary hyperventilation among nonclinical participants. *Journal of Anxiety Disorders, 15*, 217-230.

Chambless, D. L., Caputo, G. C., Bright, P., & Gallagher, R. (1984). Assessment of fear of fear in agoraphobics: The Body Sensations Questionnaire and the Agoraphobic Cognitions Questionnaire. *Journal of Consulting and Clinical Psychology, 52*, 1090-1097.

Chambless, D. L., & Goldstein, A. J. (1981). Clinical treatment of agoraphobia. In M. Mavissakalian & D. H. Barlow (Eds.) *Phobia: Psychological and pharmacological treatment (pp. 103-144)*. New York, NY: The Guilford Press.

Clark, D. A., & Beck, A. T. (2010). *Cognitive therapy of anxiety disorders: Science and practice*. New York, NY: The Guilford Press.

Clark, D. M. (1988). A cognitive model of panic attacks. In S. J. Rachman & J. D. Maser (Eds.). *Panic: Psychological* perspectives (pp. 71-89). Hillsdale, NJ: Lawrence Erlbaum Associates, Inc.

Clark, D. M. (1994). Cognitive therapy for panic disorder. In B.E.Wolfe & J.D. Maser (Eds.), *Treatment of panic disorder: A consensus development conference (pp. 121-132)*. Washington, DC: American Psychological Association.

Clark, D. M., Salkovskis, P. M., Öst, L., Breitholtz, E., Koehler, K. A., Westling, B. E., ... Gelder, M. (1997). Misinterpretation of body sensations in panic disorder. *Journal of Consulting and Clinical Psychology, 65*, 203-213.

Cohen, J. (1992). A power primer. *Psychological Bulletin*, *112*, 155-159.

Colley, R. C., Garriguet, D., Janssen, I., Craig, C. L., Clarke, J., & Tremblay, M. S. (2011). Physical activity of Canadian adults: Accelerometer results from the 2007-2009 Canadian Health Measures Survey. Retrieved from Statistics Canada website: statcan.gc.ca/pub/82-003-x/2011001/article/11396-eng.htm

Conrod, P. J., Stewart, S. H., Pihl, R. O., Côté S., Fontaine, V., & Dongier, M. (2000). Efficacy of brief coping skills interventions that match different personality profiles of female substance abusers. *Psychology of Addictive Behaviors, 14*, 231-242.

Cox, B. J., Taylor, S., Clara, I. P., Roberts, L., & Enns, M. W. (2008). Anxiety sensitivity and panic-related symptomatology in a representative community-based sample: A 1-year longitudinal analysis. *Journal of Cognitive Psychotherapy, 22*, 48-56.

Craft, L. L., & Landers, D. M. (1998). The effects of exercise on clinical depression and depression resulting from mental illness: A metaregression analysis. *Journal of Sport and Exercise Psychology, 20*, 339-357.

Craske, M. G., & Barlow, D.H. (2001). Panic disorder and agoraphobia. In D.H. Barlow (Ed.), *Clinical handbook for psychological disorders: A step-by-step treatment manual (3rd ed.)*. New York, NY: The Guilford Press.

Craske, M. G., Brown, T. A., & Barlow, D. H. (1991). Behavioral treatment of panic disorder: A two-year follow-up. *Behavior Therapy*, *22*, 289-304.

Craske, M. G., Lang, A. J., Aikins, D., & Mystkowski, J. L. (2005). Cognitive behavioral therapy for nocturnal panic. *Behavior Therapy*, *36*, 43-54.

Craske, M. G., Maidenberg, E., & Bystritsky, A. (1995). Brief cognitive-behavioral versus nondirective therapy for panic disorder. *Journal of Behavior Therapy and Experimental Psychiatry*, *26*, 113-120.

Craske, M. G., Wolitzky-Taylor, K. B., Labus, J., Wu, S., Frese, M., Mayer, E. A., & Naliboff, B. D. (2011). A cognitive-behavioral treatment for irritable bowel syndrome using interoceptive exposure to visceral sensations. *Behaviour Research and Therapy, 49*, 413-421.

De Houwer, J. (2003). The extrinsic affective Simon task. *Experimental Psychology, 50,* 77-85.

Dishman, R. K., Washburn, R. A., & Heath, G. W. (2004). *Physical activity epidemiology.* Champaign, IL: Human Kinetics.

Dunton, G. F., Whalen, C. K., Jamner, L. D., Henker, B., & Floro, J. N. (2005). Using ecologic momentary assessment to measure physical activity during adolescence. *American Journal of Preventive Medicine, 29,* 281-287.

Eke, M., & McNally, R. J. (1996). Anxiety sensitivity, suffocation fear, trait anxiety, and breath-holding duration as predictors of response to carbon dioxide challenge. *Behaviour Research and Therapy, 34*, 603-607.

Feldner, M. T., Lewis, S. F., Leen-Feldner, E. W., Schnurr, P. P., & Zvolensky, M. J. (2006). Anxiety sensitivity as a moderator of the relationship between trauma exposure frequency and posttraumatic stress symptomatology. *Journal of Cognitive Psychotherapy, 20*, 201–213.

Feldner, M. T., Zvolensky, M. J., & Schmidt, N. B. (2004). Prevention of anxiety psychopathology: A critical review of the empirical literature. *Clinical Psychology: Science and Practice, 11*, 405-424.

Figueras, G., Pérez, V., San Martino, O., Alvarez, E., Grupo de Trastornos Afectivos, & Artigas, F. (1999). Pretreatment platelet 5-HT concentration predicts the short-term response to paroxetine in major depression. *Biological Psychiatry, 46*, 518-524.

Foa, E. B., & Kozak, M. J. (1986). Emotional processing of fear: Exposure to corrective information. *Psychological Bulletin, 99*, 20–35.

Folkins, C. H., & Sime, W. E. (1981). Physical fitness training and mental health. *American Psychologist, 36*, 373–389.

Forsyth, J. P., Lejuez, C. W., & Finlay, C. (2000). Anxiogenic effects of repeated administration of 20% CO_2-enriched air: Stability within sessions and habituation across time. *Journal of Behavior Therapy and Experimental Psychiatry, 31*, 103-121.

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

Germain, J. L., & Hausenblas, H. A. (2006). The relationship between perceived and actual physical fitness: A meta-analysis. *Journal of Applied Sport Psychology, 18*, 283-296.

Godin, G., & Shepherd, R. J. (1985). A simple method to assess exercise behaviour in the community. *Canadian Journal of Applied Sports Science, 10*, 141-146.

Goodin, B. R., McGuire, L. M., Stapleton, L. M., Quinn, N. B., Fabian, L. A., Haythornthwaite, J. A., & Edwards, R. R. (2009). Pain catastrophizing mediates the relationship between self-reported strenuous exercise involvement and pain ratings: Moderating role of anxiety sensitivity. *Psychosomatic Medicine, 71*, 1018-1025.

Goldstein, A. J., & Chambless, D. L. (1978). A reanalysis of agoraphobia. *Behavior Therapy, 9*, 47-59.

Green, A. S., Rafaeli, E., Bolger, N., Shrout, P. E., & Reis, H. T. (2006). Paper or plastic? Data equivalence in paper and electronic diaries. *Psychological Methods, 11*, 87–105.

Harrington, P. J., & Telch, M. J. (1994). *Lowering anxiety sensitivity in nonclinical subjects: Preliminary evidence for a panic prevention program*. Paper presented at the 28th annual meeting of the Association for Advancement of Behavior Therapy, San Diego, CA, November.

Harrington, P. J., Telch, M. J., Abplanalp, A. C., Hamilton, A. C., & Austin, U. T. (1995). *Lowering anxiety sensitivity in nonclinical subjects: Preliminary evidence for a panic prevention program* [Abstract]. Proceedings of the 29th annual meeting of the Association for Advancement of Behavior Therapy, Washington, DC, p. 192.

Hayward, C., Killen, J. D., Kraemer, H. C., & Taylor, C. B. (2000). Predictors of panic attacks in adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry, 39*, 207-214.

Health Canada & Public Health Agency of Canada (1998). Canada's Physical Activity Guide to Healthy Active Living. Retrieved November 28, 2006 from http://www.phac-aspc.gc.ca/pau-uap/fitness/downloads.html.

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.

Ho, M-H. R., Auerbach, R. P., Jun, H. L. J., Abela, J. R. Z., Zhu, X., & Yao, S. (2011). Understanding anxiety sensitivity in the development of anxious and depressive symptoms. *Cognitive Therapy and Research, 35*, 232-240.

Hofmann, S. G., Meuret, A. E., Rosenfield, D., Suvak, M. K., Barlow, D. H., Gorman, J. M., … Woods, S. W. (2007). Preliminary evidence for cognitive mediation during cognitive-behavioral therapy of panic disorder. *Journal of Consulting and Clinical Psychology, 75*, 374-379.

Hu, L.-T., & Bentler, P. (1995). Evaluating model fit. In R. H. Hoyle (Ed.), *Structural equation modeling. Concepts, issues, and applications (pp. 76–99).* London, United Kingdom: Sage.

Hu, L., & Bentler, P. M. (1999). Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. *Structural Equation Modeling, 6*, 1−55.

Huang, T. T.-K., Harris, K. J., Lee, R. E., Nazir, N., Born, W., & Kaur, H. (2003). Assessing overweight, obesity, diet, and physical activity in college students. *Journal of American College Health*, *52*, 83-86.

Keogh, E., Dillon, C., Georgious, G., & Hut, C. (2001). Selective attentional biases for physical threat in physical anxiety sensitivity. *Journal of Anxiety Disorders, 15,* 299-315.

Keough, M. E., & Schmidt, N. B. (2012). Refinement of a brief anxiety sensitivity reduction intervention. *Journal of Consulting and Clinical Psychology*. Advance online publication. doi: 10.1037/a0027961

Kessler, R. C., McGonagle, K. A., & Zhao, S. (1994). Lifetime and 12-month prevalence of DSM-III-R psychiatric disorders in the United States: Results from the National Comorbidity Survey. *Archives of General Psychiatry, 51*, 8-19.

Kline, R. B. (1998). *Principles and practice of structural equation modeling.* New York, NY: The Guilford Press.

Kraemer, H. C., Wilson, T., Fairburn, C. G., & Agras, S. (2002). Mediators and moderators of treatment effects in randomized clinical trials. *Archives of General Psychiatry, 59*, 877-883.

Lefaivre, M.-j. (2010). Physical activity participation and exercise beliefs in adolescents and young adults with high and low anxiety sensitivity. *Dissertation Abstracts International: Section B: Sciences and Engineering, 71(2-B),* 1346.

Lefaivre, M-j, Watt, M. C., Stewart, S. H., & Wright, K. D. (2006). Implicit associations between anxiety-related symptoms and catastrophic consequences in high anxiety sensitive individuals. *Cognition and Emotion, 20,* 295-308.

Lejuez, C. W., Olatunji, B., Schmidt, N. B., Smits, J., Otto, M., & Stewart, S. H. (2011, November). Panelists. In A. C. McLeish (Chair), *Transdiagnostic cognitive vulnerability factors: Where have we been and where do we go from here?* Clinical round table discussion conducted at the meeting of the Association of Behavioral and Cognitive Therapies, Toronto, ON, Canada.

Liebowitz, M. R., Gorman, J. M., Fyer, A. J., Levitt, M., Dillon, D., Levy, G., . . . Klein, D. F. (1985). Lactate provocation of panic attacks: Biochemical and physiological findings. *Archives of General Psychiatry, 42,* 709-719.

Lovibond, P. F., & Lovibond, S. H. (1995). The structure of negative emotional states: Comparison of the Depression Anxiety Stress Scales (DASS) with the Beck Depression and Anxiety inventories. *Behaviour Research and Therapy, 33*, 335–343.

MacCallum, R. C., Browne, M. W., & Sugawara, H. M. (1996). Power analysis and determination of sample size for covariance structure modeling. *Psychological Methods, 1*, 130–149.

MacDonald, A. B., Baker, J. M., Stewart, S. H., & Skinner, M. (2000). Effects of alcohol on the response to hyperventilation of participants high and low in anxiety sensitivity. *Alcoholism: Clinical and Experimental Research, 24*, 1656-1665.

MacDonald, A. B., Stewart, S. H., Hutson, R., Rhyno, E., & Loughlin, H. (2001). The roles of alcohol and alcohol expectancy in the dampening of response to hyperventilation among high anxiety sensitive young adults. *Addictive Behaviors, 26,* 841-867.

MacDonald, T., & Watt, M. C. (2003, November). *An investigation of relations between anxiety sensitivity and physical activity in adolescence.* Poster session presented at the Child and Adolescent Anxiety Special Interest Group Poster Exposition at the 37th Annual Convention of the Association for Advancement of Behavior Therapy (AABT), Boston, MA.

Maller, R. G., & Reiss, S. (1992). Anxiety sensitivity in 1984 and panic attacks in 1987. *Journal of Anxiety Disorders, 6,* 241-247.

Marcus, B. H., Rakowski, W., & Rossi, J. S. (1992). Assessing motivational readiness and decision making for exercise. *Health Psychology, 11,* 257–261.

Mardia, K. V. (1970). Measures of multivariate skewness and kurtosis with applications. *Biometrika*, *57*, 519−530.

Marshall, S. J., & Biddle, S. J. (2001). The transtheoretical model of behavior change: A meta-analysis of applications to physical activity and exercise. *Annals of Behavioral Medicine, 23,* 229-246.

McIntosh, C. (2007). Rethinking fit assessment in structural equation modelling: A commentary and elaboration on Barrett (2007). *Personality and Individual Differences*, *42*, 859–67.

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

McNally, R. J. (1999). Theoretical approaches to the fear of anxiety. In S. Taylor (Ed.), *Anxiety sensitivity: Theory, research, and treatment of the fear of anxiety (pp. 3-16)*. Mahwah, NJ: Lawrence Erlbaum Associates.

McNally, R. J. (2002). Anxiety sensitivity and panic disorder. *Biological Psychiatry, 52,* 938-946.

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

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

Meuret, A. E., Rosenfield, D., Seidel, A., Bhaskara, L., & Hofmann, S. (2010). Respiratory and cognitive mediators of treatment change in panic disorder: Evidence for intervention specificity. *Journal of Consulting and Clinical Psychology, 78*, 691-704.

Meuret, A. E., Ritz, T., Wilhelm, F. H., & Roth, W. T. (2005). Voluntary hyperventilation in the treatment of panic disorder – functions of hyperventilation, their implications for breathing training, and recommendations for standardization. *Clinical Psychology Review, 25*, 285-306.

Moscovitch, D. A., Antony, M. M., & Swinson, R. P. (2009). Exposure-based treatments for anxiety disorders: Theory and process. In M. M. Antony & M. B. Stein (Eds.) *Oxford handbook of anxiety and related disorders (pp. 461-475)*. New York, NY: Oxford University Press.

Myers, R. S., & Roth, D. L. (1997). Perceived benefits of and barriers to exercise and stage of exercise adoption in young adults. *Health Psychology, 16*, 277–283.

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

Ocañez, K. S., McHugh, R. K., & Otto, M. W. (2010). A meta-analytic review of the association between anxiety sensitivity and pain. *Depression & Anxiety, 27*, 760-767.

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

Olthuis, J. V., Stewart, S. H., Watt, M. C., Sabourin, B. C., & Keogh, E. (in press). Anxiety sensitivity and negative interpretation biases: Their shared and unique associations with anxiety symptoms. *Journal of Psychopathology and Behavioral Assessment.*

Osman, A., Gutierrez, P. M., Smith, K., Fang, Q., Lozano, G., & Devine, A. (2009). The Anxiety Sensitivity Index-3: Analyses of dimensions, reliability estimates, and correlates in nonclinical samples. *Journal of Personality Assessment, 92,* 45-52.

Otto, M.W. (2008). *Interoceptive exposure in the treatment of panic and co-morbid disorders: Novel applications and mechanisms of action*. Discussant for symposium presented at the 28th Anxiety Disorders Association of America (ADAA) Annual Conference (Chair: Dr. Margo C. Watt), Savannah, GA, March.

Otto, M. W., & Reilly-Harrington, N. A. (1999). The impact of treatment on anxiety sensitivity. In S. Taylor (Ed.), *Anxiety sensitivity: Theory, research, and treatment of the fear of anxiety* (pp. 321-336). Mahwah, NJ: Lawrence Erlbaum Associates.

Parfitt, G., Rose, E. A., & Burgess, W. M. (2006). The psychological and physiological responses of sedentary individuals to prescribed and preferred intensity exercise. *British Journal of Health Psychology, 11,* 39–53.

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*, 665-673.

Penedo, F. J., & Dahn, J. R. (2005). Exercise and well-being: A review of mental and physical health benefits associated with physical activity. *Current Opinion in Psychiatry, 18*, 189-193.

Peterson, R. A., & Plehn, K. (1999). Measuring anxiety sensitivity. In S. Taylor (Ed.), *Anxiety sensitivity: Theory, research, and treatment of the fear of anxiety (pp. 61-81)*. Mahwah, NJ: Lawrence Erlbaum Associates.

Peterson, R.A., & Reiss, S. (1992). *Anxiety Sensitivity Index Manual (2nd ed.).* Worthington, OH: International Diagnostic Services.

Physical Activity Council. (2012). *2012 participation report: The Physical Activity Council's annual study tracking sports, fitness and recreation participation in the USA.* Retrieved from: http://www.physicalactivitycouncil.com/PDFs/2012PACReport.pdf

Piasecki, T. M., Hufford, M. R., Solhan, M., & Trull, T. J. (2007). Assessing clients in their natural environments with electronic diaries: Rationale, benefits, limitations, and barriers. *Psychological Assessment, 19*, 25-43.

Plotkin, D. P. (2001). *The effects of interoceptive exposure on fear reduction and return of fear in individuals with public speaking anxiety*. University of California, Los Angeles. *ProQuest Dissertations and Theses*, 130 p. http://ezproxy.library.dal.ca/login?url=http://search.proquest.com/docview/275855323?accountid= 10406

Pollock, M. L., Gaesser, G. A., Butcher, J. D., Despres, J. P., Dishman, R. K., Franklin, B. A., & Garber, C. E. (1998). American College of Sports Medicine position stand: The recommended quantity and quality of exercise for developing and maintaining cardiorespiratory and muscular fitness, and flexibility in healthy adults. *Medicine and Science in Sports and Exercise, 30*, 975–991.

Preacher, K. J., Curran, P. J., & Bauer, D. J. (2006). Computational tools for probing interaction effects in multiple linear regression, multilevel modeling, and latent curve analysis. *Journal of Educational and Behavioral Statistics, 31*, 437-448.

Prochaska, J. O., & DiClemente, C. C. (1982). Transtheoretical therapy: Toward a more integrative model of change. *Psychotherapy: Theory, Research, & Practice, 19,* 276-288.

Prochaska, J.O., & Marcus, B.H. (1994). The transtheoretical model: Applications to exercise. In R.K. Dishman (Ed.) *Advances in exercise adherence* (pp. 161-180). Champaign, IL: Human Kinetics.

Prochaska, J. O., Velicer, W. F., Rossi, J. S., Goldstein, M. G., Marcus, B. H., Rakowski, W., . . . Rossi, S. R. (1994). Stages of change and decisional balance for 12 problem behaviours. *Health Psychology, 13*, 39-46.

Public Health Agency of Canada (2002). *A Report on Mental Illnesses in Canada*. Retrieved from http://www.phac-aspc.gc.ca/publicat/miic-mmac/chap_1-eng.php

Rapee, R. M. (1985). Distinctions between panic disorder and generalized anxiety disorder: Clinical presentation. *Australian and New Zealand Journal of Psychiatry, 19*, 227-232.

Rapee, R. M., Brown, T. A., Antony, M. M., & 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.

Rapee, R. M. & Medoro, L. (1994). Fear of physical sensations and trait anxiety as mediators of the response to hyperventilation in nonclinical subjects. *Journal of Abnormal Psychology, 103*, 693-699.

Raudenbush, S. W. (2002). Alternative covariance structures for polynomial models of individual growth and change. In D. S. Moskowitz & S. L. Hershberger (Eds.). *Modeling intraindividual variability with repeated measures data: Methods and applications (pp. 25-57)*. Mahwah, NJ: Lawrence Erlbaum Associates.

Raudenbush, S., & Bryk, A. (2002). *Hierarchical linear models*. London: Sage.

Raudenbush, S., Bryk, A., & Congdon, R. (2007). *HLM 6.04: Hierarchical linear and non-linear modeling.* Lincolnwood, IL: Scientific Software International.

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

Regier, D. A., Narrow, W. E., & Rae, D. S. (1990). The epidemiology of anxiety disorders: The epidemiologic catchment area (ECA) experience. *Journal of Psychiatric Research, 24*, 3-14.

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

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)*. San Diego, CA: Academic Press.

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

Richey, J. A., Schmidt, N. B., Hofmann, S. G., & Timpano, K. R. (2010). Temporal and structural dynamics of anxiety sensitivity in predicting fearful responding to a 35% CO_2 challenge. *Journal of Anxiety Disorders, 24*, 423-432.

Roosevelt, F. D. (1965). First inaugural address. In R.D. Heffner (Ed.) *A documentary history of the United State (Rev. ed., pp. 274-278).* New York, NY: Mentor Books.

Schmidt, N. B., Eggleston, A. M., Woolawy-Bickel, K., Fitzpatrick, K. K., Vasey, M. W., & Richey, J. A. (2007). Anxiety Sensitivity Amelioration Training (ASAT): A longitudinal primary prevention program targeting cognitive vulnerability. *Journal of Anxiety Disorders, 21*, 302-319.

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*, 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,* 532-537.

Schmidt, N. B., Trakowski, J. H., & Staab, J. P. (1997). Extinction of panicogenic effects of a 35% CO_2 challenge in patients with panic disorder. *Journal of Abnormal Psychology, 106,* 630-638.

Schmidt, N. B., Zvolensky, M. J., & Maner, J. K. (2006). Anxiety sensitivity: Prospective prediction of panic attacks and Axis 1 pathology. *Journal of Psychiatric Research, 40,* 691-699.

Shephard, R. J., Allen, C., Benade, A. J., Davies, C. T., Prampero, D., Hedman, P. E., . . . Simmon, R. (1968). The maximum oxygen intake. An international reference standard of cardiorespiratory fitness. *Bulletin of the World Health Organization, 38*, 757-764.

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

Smits, J. A. J., 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,* 1047-1054.

Smits, J. A. J., & Otto, M. W. (2009). *Exercise for Mood and Anxiety Disorders: Therapist Guide*. New York, NY: Oxford University Press.

Smits, J. A. J., 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*, 646-652.

Smits, J. A. J., Rosenfield, D., McDonald, R., & Telch, M. J. (2006). Cognitive mechanisms of social anxiety reduction: An examination of specificity and temporality. *Journal of Consulting and Clinical Psychology, 74*, 1203-1212.

Smits, J. A. J., Tart, C. D., Rosenfield, D., & Zvolensky, M. J. (2011). The interplay between physical activity and anxiety sensitivity in fearful responding to carbon dioxide challenge. *Psychosomatic Medicine, 73*, 498-503.

Smits, J. A., & Zvolensky, M. J. (2006). Emotional vulnerability as a function of physical activity among individuals with panic disorder. *Depression and Anxiety, 23*, 102-106.

Sobel, M. E. (1982). Asymptotic intervals for indirect effects in structural equations models. In S. Leinhart (Ed.), *Sociological methodology (pp. 290-312).* San Francisco, CA: Jossey-Bass.

Sobell, L. C., Maisto, S. A., Sobell, M. B., & Cooper, A. M. (1979). Reliability of alcohol abusers' self-reports of drinking behavior. *Behaviour Research and Therapy, 17*, 157-160.

Stathopoulou, G., Powers, M. B., Berry, A.C., Smits, J. A. J., & Otto, M. W. (2006). Exercise interventions for mental health: A quantitative and qualitative review. *Clinical Psychology: Science and Practice, 13*, 179-193.

Steiger, J. H. (1990). Structural model evaluation and modification: An internal estimation approach. *Multivariate Behavioral Research, 25*, 173−180.

Steer, R. A., Ranieri, W. F., Beck, A. T., & Clark, D. A. (1993). Further evidence for the validity of the Beck Anxiety Inventory with psychiatric outpatients. *Journal of Anxiety Disorders, 7*, 195-205.

Stewart, S. H., & Asmundson, G. J. G. (2006). Anxiety sensitivity and its impact on pain experiences and conditions: A state of the art. *Cognitive Behaviour Therapy, 35*, 185-188.

Stewart, S. H., Conrod, P. J., Gignac, M. L. & Pihl, R. O. (1998). Selective processing biases in anxiety-sensitive men and women. *Cognition and Emotion, 12*, 105-133.

Stewart, S. H., Hall, E., Wilkie, H., & Birch, C. (2002). Affective priming of alcohol and enhancement motivated drinkers. *Cognitive Behaviour Therapy, 31*, 68-80.

Stewart, S. H., Knize, K., & Pihl, R. O. (1992). Anxiety sensitivity and dependency in clinical and non-clinical panickers and controls. *Journal of Anxiety Disorders, 6*, 119-131.

Stewart, S. H., Samoluk, S. B., & MacDonald, A. B. (1999). Anxiety sensitivity and substance use and abuse. In S. Taylor (Ed.), *Anxiety sensitivity: Theory, research, and treatment of the fear of anxiety (pp.287-319).* Mahwah, NJ: Lawrence Erlbaum Associates.

Stewart, S. H., Taylor, S., & Baker, J. M. (1997). Gender differences in dimensions of anxiety sensitivity. *Journal of Anxiety Disorders*, *11*, 179-200.

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*, 291-302.

Stout, R. L. (2007). Advancing the analysis of treatment process. *Addiction, 102*, 1539-1545.

Sturges, L. V., Goetsch, V. L., Ridley, J., & Whittal, M. (1998). Anxiety sensitivity and response to hyperventilation challenge: Physiologic arousal, interoceptive acuity, and subjective distress. *Journal of Anxiety Disorders, 12*, 103-115.

Tabachnick, B. G., & Fidell, L. S. (2006). *Using multivariate statistics (5th ed)*. Boston, MA: Allyn & Bacon.

Taylor, S. (Ed.). (1999). *Anxiety sensitivity: Theory, research and treatment of the fear of anxiety*. Mahwah, NJ: Lawrence Erlbaum Associates.

Taylor, S., Koch, W. J., & McNally, R. J. (1992). How does anxiety sensitivity vary across the anxiety disorders? *Journal of Anxiety Disorders, 6*, 249-259.

Taylor, S., Koch, W. J., Woody, S., & McLean, P. (1996). Anxiety sensitivity and depression: How are they related? *Journal of Abnormal Psychology, 105,* 474–479.

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

Teachman, B. A., & Woody, S. R. (2003). Automatic processing in spider phobia: Implicit fear associations over the course of treatment. *Journal of Abnormal Psychology, 112,* 100-109.

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. *Behaviour Research and Therapy, 31*, 279-287.

Telch, M. J., Schmidt, N. B., Jaimez, T., Jacquin, K. M., & Harrington, P. J. (1995). Impact of cognitive behavioral treatment on quality of life in panic disorder patients. *Journal of Consulting and Clinical Psychology, 63*, 823-830.

Von Baeyer, C. L., Chambers, C. T., & Eakins, D. M. (2011). Development of a 10-item short form of the Parents' Postoperative Pain Measure: The PPPM-SF. *The Journal of Pain, 12*, 401-406.

Wald, J. (2008). Interoceptive exposure as a prelude to trauma-related exposure therapy in a case of posttraumatic stress disorder with substantial comorbidity. *Journal of Cognitive Psychotherapy, 22*, 331-345.

Wald, J. & Taylor, S. (2005). Interoceptive exposure therapy combined with trauma-related exposure therapy for post-traumatic stress disorder: A case report. *Cognitive Behaviour Therapy, 34*, 34-40.

Walker, J. R., & Furer, P. (2008). Interoceptive exposure in the treatment of health anxiety and hypochondriasis. *Journal of Cognitive Psychotherapy, 22*, 366-378.

Warburton, D. E. R., Charlesworth, S., Ivey, A., Nettlefold, L., & Bredin, S. S. D. (2010). A systematic review of the evidence for Canada's Physical Activity Guidelines for Adults. *International Journal of Behavioral Nutrition and Physical Activity, 7*, 39-259.

Warburton, D. E. R., Nicol, C. W., & Bredin, S. S. D. (2006). Health benefits of physical activity: The evidence. *Canadian Medical Association Journal, 174*, 801-809.

Watt, M. C., & Stewart, S. H. (2000). Anxiety sensitivity mediates the relationships between childhood learning experiences and elevated hypochondriacal concerns in young adulthood. *Journal of Psychosomatic Research*, 49, 107-118.

Watt, M. C., Stewart, S. H., Birch, C. D., & Bernier, D. B. (2006). Brief CBT for high anxiety sensitivity decreases drinking and drinking problems: Evidence from a randomized controlled trial. *Journal of Mental Health*, 15, 683-695.

Watt, M. C., Stewart, S. H., Conrod, P. J., & Schmidt, N. B. (2008). Personality-based approaches to treatment of co-morbid anxiety and substance use disorder. In S. H. Stewart & P. J. Conrod (Eds.), *Anxiety and substance use disorders: The vicious cycle of comorbidity (pp.201-219).* New York, NY: Springer.

Watt, M. C., Stewart, S. H., & Cox, B. J. (1998). A retrospective study of the learning history origins of anxiety sensitivity. *Behaviour Research and Therapy, 36*, 505-525.

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*, 248-256.

Welch, A. S., Hulley, A., Ferguson, C., & Beauchamp, M. R. (2007). Affective responses of inactive women to a maximal incremental exercise test: A test of the dual-mode model. *Psychology of Sport and Exercise, 8,* 401-423.

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

Weinstock, J., Whelan, J. P., & Meyers, A. W. (2004). Behavioral assessment of gambling: An application of the Timeline Followback method. *Psychological Assessment, 16,* 72-80.

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*, 401-408.

Williams, D. M., Dunsiger, S., Ciccolo, J. T., Lewis, B. A., Albrecht, A. E., & Marcus, B. H. (2008). Acute affective response to a moderate-intensity exercise stimulus predicts physical activity participation 6 and 12 months later. *Psychology of Sport and Exercise, 9*, 231-245.

Williams, D. M., Lewis, B. A., Dunsiger, S., Whiteley, J. A., Papandonatos, G. D., Bock, B. C., . . . Marcus, B. H. (2008). Comparing psychosocial predictors of physical activity adoption and maintenance. *Annals of Behavioral Medicine, 36*, 186-194.

Williams, S. L., & Falbo, J. (1996). Cognitive and performance-based treatments for panic attack in people with varying degrees of agoraphobic disability. *Behaviour Research and Therapy*, *34,* 253–264.

Wipfli, B., Landers, D., Nagoshi, C., & Ringenbach, S. (2011). An examination of serotonin and psychological variables in the relationship between exercise and mental health. *Scandinavian Journal of Medicine & Science in Sports*, *21,* 474-481.

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 and Exercise Psychology*, *30,* 392-410.

Woicik, P. A., Stewart, S. H., Pihl, R. O., & Conrod, P. J. (2009). The Substance Use Risk Profile Scale: A scale measuring traits linked to reinforcement-specific substance use profiles. *Addictive Behaviors, 34*, 1042-1055.

Woody, S. R., Whittal, M L, & McLean, P. D. (2011). Mechanisms of symptom reduction in treatment for obsessions. *Journal of Consulting and Clinical Psychology, 79*, 653-664.

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,* 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.81-114)*. Mahwah, NJ: Lawrence Erlbaum Associates.

Zvolensky, M. J., Bernstein, A., Cardenas, S. J., Colotla, V. A., Marshall, E. C., & Feldner, M. T. (2007). Anxiety sensitivity and early relapse to smoking: A test among Mexican daily, low-level smokers. *Nicotine & Tobacco Research, 9,* 483–491.

Zvolensky, M., Bonn-Miller, M., Bernstein, A., & Marshall, E. (2006). Anxiety sensitivity and abstinence duration to smoking. *Journal of Mental Health, 15,* 659-670.

Zvolensky, M. J., & Eifert, G. H. (2001). A review of psychological factors/processes affecting anxious responding during voluntary hyperventilation and inhalations of carbon dioxide-enriched air. *Clinical Psychology Review, 21,* 375-400.

Zvolensky, M. J., Schmidt, N. B., Bernstein, A., & Keough, M. E. (2006). Risk-factor research and prevention programs for anxiety disorders: A translational research framework. *Behaviour Research and Therapy, 44*, 1219-1239.

Zvolensky, M. J., Schmidt, N. B., & Stewart, S. H. (2003). Panic disorder and smoking. *Clinical Psychology: Science and Practice, 10*, 29-51.

Zvolensky, M. J., Vujanovic, A.A., Miller, M. O. B., Bernstein, A., Yartz, A. R., Gregor, K. L., et al. (2007). Incremental validity of anxiety sensitivity in terms of motivation to quit, reasons for quitting, and barriers to quitting among community-recruited daily smokers. *Nicotine and Tobacco Research, 9*, 965-975.

Zvolensky, M. J., Yartz, A. R., Gregor, K., Gonzalez, A., & Bernstein, A. (2008). Interoceptive exposure-based cessation intervention for smokers high in anxiety sensitivity: A case series. *Journal of Cognitive Psychotherapy, 22,* 346-365.

APPENDIX A

Selected Items from the Anxiety Sensitivity Index

(Peterson & Reiss, 1992)

Participants are to respond to the items by circling the phrase that best represents the extent to which they agree with each item.

1. It is important to me not to appear nervous.
VERY LITTLE A LITTLE SOME MUCH VERY MUCH

9. When I notice that my heart is beating rapidly, I worry that I might have a heart attack.
VERY LITTLE A LITTLE SOME MUCH VERY MUCH

10. It scares me when I become short of breath.
VERY LITTLE A LITTLE SOME MUCH VERY MUCH

12. It scares me when I am unable to keep my mind on a task.
VERY LITTLE A LITTLE SOME MUCH VERY MUCH

13. Other people notice when I feel shaky.
VERY LITTLE A LITTLE SOME MUCH VERY MUCH

15. When I am nervous, I worry that I might be mentally ill.
VERY LITTLE A LITTLE SOME MUCH VERY MUCH

APPENDIX B

Physical Activity Measure Modified Version

(Lefaivre, 2010)

Low-intensity Exercise

1. Think of the last 3 months: how *often* have you participated in *low-intensity exercise* (For example: yoga, pilates, leisure walking, etc)? You may include low-intensity physical activity that is performed as a mode of transportation (for example, a 15 minute leisurely walk to go to school)

Please place a number beside one of the following:

_____ time(s) per day
_____ time(s) per week (if less than once per day)
_____ time(s) per month (if less than once per week)
_____ time(s) per year (if less than once per month)

What type of exercise did you do?

____

____

____

2. How long do you normally exercise during a typical low-intensity exercise session? (Please select one of the following.)

_____ 0 to 15 minutes
_____ 16 minutes to 30 minutes
_____ 31 minutes to 45 minutes
_____ 46 minutes to 60 minutes
_____ 61 minutes to 75 minutes
_____ 76 minutes to 80 minutes
_____ 81 minutes to 95 minutes
_____ more than 95 minutes (please specify:___________________)

3. Are you more likely to do low-intensity physical exercise on your own or with others?

_____ ALONE _____ WITH OTHERS _______ BOTH EQUALLY

Moderate Physical Activity

4. Think of the last 3 months: how *often* have you participated in *moderate physical activity* (for example: brisk walking, skating, bike riding, swimming, playing outdoors, etc)? You may include moderate physical activity that is performed as a mode of transportation (for example, a 15 minute brisk walk to go to school)

Please place a number beside one of the following:

_____ time(s) per day
_____ time(s) per week (if less than once per day)
_____ time(s) per month (if less than once per week)
_____ time(s) per year (if less than once per month)

What type of exercise did you do?

____

____

____

5. How long do you normally exercise during a typical moderate physical activity? (Please select one of the following.)

_____ 0 to 15 minutes
_____ 16 minutes to 30 minutes
_____ 31 minutes to 45 minutes
_____ 46 minutes to 60 minutes
_____ 61 minutes to 75 minutes
_____ 76 minutes to 80 minutes
_____ 81 minutes to 95 minutes
_____ more than 95 minutes (please specify:___________________)

6. Are you more likely to do moderate physical activity on your own or with others?

_____ ALONE _____ WITH OTHERS _______ BOTH EQUALLY

Vigorous Physical Activity

7. Think of the last 3 months: how *often* have you participated in *vigorous physical activity* (for example: running, soccer, etc)?

Please place a number beside one of the following:

_____ time(s) per day
_____ time(s) per week (if less than once per day)
_____ time(s) per month (if less than once per week)
_____ time(s) per year (if less than once per month)

What type of exercise did you do?

____

____

____

8. How long do you normally exercise during a typical vigorous physical activity? (Please select one of the following.)

_____ 0 to 15 minutes
_____ 16 minutes to 30 minutes
_____ 31 minutes to 45 minutes
_____ 46 minutes to 60 minutes
_____ 61 minutes to 75 minutes
_____ 76 minutes to 80 minutes
_____ 81 minutes to 95 minutes
_____ more than 95 minutes (please specify:___________________)

9. Are you more likely to do vigorous physical activity on your own or with others?

_____ ALONE _____ WITH OTHERS ______ BOTH EQUALLY

Organized Sports

10. Think of the last 3 months: how *often* have you participated in organized sports at school or in the community (e.g., exercise class, sports team, intramural sports, running clinics, etc)?

Please place a number beside one of the following:

_____ time(s) per day
_____ time(s) per week (if less than once per day)
_____ time(s) per month (if less than once per week)
_____ time(s) per year (if less than once per month)

What type of organized sport did you do?

____

____

____

11. How long do you normally exercise during a typical organized sport? (Please select one of the following.)

_____ 0 to 15 minutes
_____ 16 minutes to 30 minutes
_____ 31 minutes to 45 minutes
_____ 46 minutes to 60 minutes
_____ 61 minutes to 75 minutes
_____ 76 minutes to 80 minutes
_____ 81 minutes to 95 minutes
_____ more than 95 minutes (please specify:___________________)

12. Are you more likely to do organized sports on your own or with others?
_____ ALONE _____ WITH OTHERS ______BOTH EQUALLY

Active Life Style

13. What is your main mode of transportation? Please rate the options below from "1" to indicate most frequently used, "2" for next most frequently used etc. Please leave blank those options that do not at all apply to you.

_____ motorized vehicle
_____ public transportation
_____ biking
_____ rollerblading/skateboarding
_____ walking
_____ other (please specify:______________________________________)

14. When given the choice, do you typically go up the elevator or use the stairs?
_____ ELEVATOR or _______STAIRS

APPENDIX C

Perceived Fitness Measure

HOW FIT WOULD YOU DESCRIBE YOURSELF? (Circle the number that best represents your answer)

0 = No activity
1-3 = Occasional activity
4-6 = Casual Sports
7-9 = Competitive Recreational Sports
10 = Olympic calibre fitness level

0 1 2 3 4 5 6 7 8 9 10

APPENDIX D

Decisional Balance Scale

(Marcus, Rakowsky, & Rossi, 1992)

Physical activity or exercise include activities such as walking briskly, jogging, bicycling, swimming, or any other activity in which the exertion is at least as intense as these activities.

Please rate how important each of these statements is in your decision of whether to be physically active. In each case, think about how you feel **right now**, not how you have felt in the past or would like to feel.

Circle only one number per item:

	1 = not at all important	**2 = slightly important**	**3 = moderately important**	**4 = very important**	**5 = extremely important**
1. I would have more energy for my family and friends if I were regularly physically active	1	2	3	4	5
2. Regular physical activity would help me relieve tension.	*1*	*2*	*3*	*4*	*5*
3. I think I would be too tired to do my daily work after being physically active.	1	2	3	4	5
4. I would feel more confident if I were regularly physically active.	*1*	*2*	*3*	*4*	*5*
5. I would sleep more soundly if I were regularly physically active.	1	2	3	4	5
6. I would feel good about myself I kept my commitment to be regularly physically active.	*1*	*2*	*3*	*4*	*5*
7. I would find it difficult to find a physical activity that I enjoy that is not affected by bad weather.	1	2	3	4	5
8. I would like my body better if I were regularly physically active.	*1*	*2*	*3*	*4*	*5*
9. It would be easier for me to perform routine physical tasks if I were regularly physically active.	1	2	3	4	5
10. I would feel less stressed if I were regularly physically active.	*1*	*2*	*3*	*4*	*5*

	1 = not at all important	**2 = slightly important**	**3 = moderately important**	**4 = very important**	**5 = extremely important**
11. I feel uncomfortable when I am physically active because I get out of breath and my heart beats very fast.	1	2	3	4	5
12. I would feel more comfortable with my body if I were regularly physically active.	*1*	*2*	*3*	*4*	*5*
13. Regular physical activity would take too much of my time.	1	2	3	4	5
14. Regular physical activity would help me have a more positive outlook on life.	*1*	*2*	*3*	*4*	*5*
15. I would have less time for my family and friends if I were regularly physically active.	1	2	3	4	5
16. At the end of the day, I am too exhausted to be physically active.	*1*	*2*	*3*	*4*	*5*

APPENDIX E

Hyperventilation Questionnaire

(Rapee & Medoro, 1994)

<u>Pulse frequency per minute</u>

-Before running:

-After 5 minutes of running:

-After 10 minutes of running:

INSTRUCTIONS: Please rate the MAXIMUM degree to which you experienced the following feelings during the running exercise by placing a circle around the appropriate number.

<u>Feeling</u>	**Not at all**			**Markedly**
-Numbness in extremities	0	1	2	3
-Breathlessness	0	1	2	3
-Buzzing in the head	0	1	2	3
-Feeling distant	0	1	2	3
-Feeling unreal	0	1	2	3
-Fatique	0	1	2	3
-Fear	0	1	2	3
-Pounding heart	0	1	2	3
-Feeling trapped or helpless	0	1	2	3
-Hot or flushed	0	1	2	3
-Anxiety	0	1	2	3
-Headache	0	1	2	3

Feeling	Not at all			Markedly
-Rising agitation	0	1	2	3
-Feeling of suffocation	0	1	2	3
-Dizziness	0	1	2	3
-Feeling of losing control	0	1	2	3
-Worrying that your actions are damaging to your health	0	1	2	3
-Tingling in the face	0	1	2	3
-Tight or stiff muscles	0	1	2	3
-Blurred vision	0	1	2	3
-Weakness	0	1	2	3
-Relaxation	0	1	2	3
-Nervousness	0	1	2	3
-Racing heart	0	1	2	3
-Feeling like passing out	0	1	2	3
-Nausea	0	1	2	3
-Tingling in extremities	0	1	2	3
-Fear of heart attack	0	1	2	3
-Band across head	0	1	2	3
-Tension	0	1	2	3
-Feel like panicking	0	1	2	3

APPENDIX F

Results of Growth Curve Analyses from Study 2

Predicting Changes in Affective Reactions in High AS and Low AS Participants, after Removal of Potential Outlier

Predictor	Fixed effects		Random effects
	Unstandardized Coefficient	*SE*	Variance component
Predicting affective reactions			
Intercept	0.50	0.29	1.87***
Interoceptive exposure trials	0.00†	0.03	0.18
AS group	1.45**	0.46	--
Interoceptive exposure trials x AS group	-0.08†	0.05	--

†$p < .10$. *$p < .05$. **$p < .01$. ***$p < .001$.

Growth curve analyses predicting changes in affective reactions are based on 422 responses from 47 participants. *SE* = standard error. For AS group, low AS = 0 and high AS = 1.

Simple slope analyses suggested that the linear rate of change in affective reactions for low AS participants was not significantly different from zero, $t(45) = 0.06$, $p > .05$. However, for high AS participants, the linear rate of change in affective reactions was negative and significant, even after removal of potential outlier, $t(45) = -2.22$, $p < .05$.

APPENDIX G

Depression Anxiety Stress Scales

(Lovibond & Lovibond, 1995)

DASS *Name:*
Date:

Please read each statement and circle a number 0, 1, 2 or 3 that indicates how much the statement applied to you ***over the past week***. There are no right or wrong answers. Do not spend too much time on any statement.

The rating scale is as follows:

0 Did not apply to me at all
1 Applied to me to some degree, or some of the time
2 Applied to me to a considerable degree, or a good part of time
3 Applied to me very much, or most of the time

1	I found myself getting upset by quite trivial things	0	1	2	3
2	I was aware of dryness of my mouth	0	1	2	3
3	I couldn't seem to experience any positive feeling at all	0	1	2	3
4	I experienced breathing difficulty (eg, excessively rapid breathing, breathlessness in the absence of physical exertion)	0	1	2	3
5	I just couldn't seem to get going	0	1	2	3
6	I tended to over-react to situations	0	1	2	3
7	I had a feeling of shakiness (eg, legs going to give way)	0	1	2	3
8	I found it difficult to relax	0	1	2	3
9	I found myself in situations that made me so anxious I was most relieved when they ended	0	1	2	3
10	I felt that I had nothing to look forward to	0	1	2	3
11	I found myself getting upset rather easily	0	1	2	3
12	I felt that I was using a lot of nervous energy	0	1	2	3
13	I felt sad and depressed	0	1	2	3
14	I found myself getting impatient when I was delayed in any way (eg, elevators, traffic lights, being kept waiting)	0	1	2	3
15	I had a feeling of faintness	0	1	2	3
16	I felt that I had lost interest in just about everything	0	1	2	3
17	I felt I wasn't worth much as a person	0	1	2	3
18	I felt that I was rather touchy	0	1	2	3
19	I perspired noticeably (eg, hands sweaty) in the absence of high temperatures or physical exertion	0	1	2	3
20	I felt scared without any good reason	0	1	2	3
21	I felt that life wasn't worthwhile	0	1	2	3

APPENDIX H

Selected Items from the Beck Anxiety Inventory

(A. T. Beck & Steer, 1993)

Participants rate each item according to how much they have been bothered by the symptom of anxiety during the past month, including the day they are completing the measure.

	Not At All	Mildly but it didn't bother me much.	Moderately - it wasn't pleasant at times	Severely – it bothered me a lot
Numbness or tingling	0	1	2	3
Unable to relax	0	1	2	3
Fear of worst happening	0	1	2	3
Dizzy or lightheaded	0	1	2	3
Heart pounding/racing	0	1	2	3
Terrified or afraid	0	1	2	3
Nervous	0	1	2	3
Fear of losing control	0	1	2	3
Difficulty in breathing	0	1	2	3
Fear of dying	0	1	2	3
Scared	0	1	2	3

APPENDIX I

Results of ANOVAs from Study 4

A series of 3x2x2 omnibus mixed model ANOVAs were conducted as supplementary analyses for the four outcome variables of the current study. Time (pre-intervention, post-intervention, follow-up) was the within subjects factor and condition (CBT, HEC) and AS group (Low AS, High AS) were the two between subjects factors. Significant main (or simple main) effects of time were further analysed using post hoc contrasts examining differences between pre- and post-intervention and between post-intervention and follow-up.

First, for changes in AS levels, there was a significant main effect of time, $F(2,124) = 21.23$, $p < .001$, and a significant main effect of AS group, $F(1,62) = 145.85$, $p < .001$. The main effect of condition was non-significant, $F(1,62) = 0.07$, $p > .10$. The main effects of time and AS group were qualified by a significant time x AS group interaction, $F(2,124) = 21.92$, $p < .001$. Simple effects analyses revealed that for low AS participants, the effect of time was non-significant, $F(2,50) = 1.49$, $p > .10$. High AS participants, on the other hand, experienced a significant decrease in AS levels over time, $F(2,78) = 39.45$, $p < .001$. Although both the linear, $F(1,33) = 46.41$, $p < .001$ and quadratic $F(1,33) = 8.78$, $p = .006$, trends were significant, results suggested that a linear trend better represented the data. This result was consistent with further post-hoc analyses of the significant simple effect of time for high AS participants. There was a significant decrease from pre- to post-intervention, $t(39) = -5.35$, $p < .001$. ASI scores decreased further for high AS participants from post-intervention to follow-up, $t(39) = -3.65$, $p = .001$. The condition x AS group interaction was marginally significant, $F(1,62) = 3.81$, p

= .06. Simple effects analyses revealed that, for low AS participants, mean scores on the ASI were higher in participants in the HEC condition than those in the CBT condition, $F(1,24) = 4.82$, $p < .05$. However, for high AS participants, there were no differences in mean ASI scores between participants in the HEC and CBT conditions, $F(1,38) = 2.12$, $p > .10$. The time x condition interaction was non-significant, $F(2,124) = 0.25$, $p > .10$. Finally, the three-way interaction was also non-significant, $F(2,124) = 1.11$, $p > .10$. These interactions remained non-significant when examining linear and quadratic trends (all *p*s > .10).

For changes in stress levels, there was a significant main effect of time, $F(2,114) = 9.93$, $p < .001$. Trend analyses revealed that linear, $F(1,57) = 11.96$, $p = .001$ and quadratic, $F(1,57) = 7.46$, $p = .008$ trends were both significant; however, the linear trend was a slightly better representation of the data's pattern. Planned contrasts revealed that participants experienced a decrease in stress scores from pre- to post-intervention, $t(60) = -3.55$, $p = .001$. A non-significant difference between post-intervention and follow-up scores, $t(60) = 0.50$, $p > .10$ indicated that these gains were maintained at follow-up. There was also a significant main effect of AS group, $F(1,57) = 18.87$, $p < .001$. The main effect of condition was non-significant, $F(1,57) = 0.74$, $p > .10$. The time x condition interaction was non-significant, $F(2,114) = 1.05$, $p > .10$. All other two-way interactions and the three-way interaction were also non-significant, *F*s ≤ 1.00, *p*s > .10, even when examining linear and quadratic trends.

For changes in depression levels, there was a significant main effect of time, $F(2,114) = 8.14$, $p < .001$. There was also a significant main effect of AS group, $F(1,57) = 12.76$, $p = .001$. The main effect of condition was non-significant, $F(1,57) = 0.00$, $p >$

.10. The main effects of time and AS group were qualified by a marginally significant time x AS group interaction, $F(2,114) = 2.86$, $p = .06$. Decomposing the time x AS group interaction into simple effects of time for high and low AS participants, respectively, revealed that for low AS participants, there was no significant simple effect of time, $F(2,52) = 1.02$, $p > .10$. However, high AS participants experienced a significant decrease in depression scores over time, $F(2,66) = 9.45$, $p < .001$. Trend analyses revealed that both linear, $F(1,33) = 8.20$, $p = .007$ and quadratic, $F(1,33) = 12.31$, $p = .001$ trends were significant. Results suggested that quadratic trends represented a better characterization of the data's pattern. This was consistent with further post hoc analyses of the simple effect of time for high AS participants. A significant decrease occurred from pre- to post-intervention, $t(60) = -3.88$, $p < .001$. This was maintained at follow-up, as revealed by a non-significant change from post-intervention to follow-up, $t(60) = 1.21$, $p > .10$. All other two-way interactions and the three-way interactions were non-significant, Fs $\leq$ 0.01, ps $> .10$, even when examining linear and quadratic trends.

For changes in anxiety levels, there was a significant main effect of time, $F(2,126) =$ 8.07, $p = .001$. Trend analyses revealed that the data was better represented as following a linear, $F(1,63) = 14.11$, $p < .001$, rather than a quadratic, $F(1,63) = 0.74$, $p = .391$ trend. Participants experienced a significant decrease in anxiety scores between pre- and post-intervention, $t(26) = -2.69$, $p = .01$. The decrease was sustained at follow-up, as indicated by a non-significant difference between post-intervention and follow-up, $t(26) = 0.63$, $p >$ .10. There was also a significant main effect of AS group, $F(1,63) = 13.92$, $p < .001$. The main effect of condition was non-significant, $F(1,63) = 0.08$, $p > .10$. There was a significant condition x AS group interaction, $F(1,63) = 5.70$, $p = .02$. Simple effects analyses revealed that for low AS participants, there was a significant difference in mean BAI scores for participants in the CBT versus HEC condition, $F(1,25) = 6.90$, $p = .02$, with those in the HEC condition exhibiting higher scores than those in the CBT/IE condition. However, for high AS participants, this difference was only marginally significant, $F(1,38) = 3.00$, $p = .09$, and high AS participants in the CBT/IE condition exhibited higher scores than those in the HEC condition. All other interactions were non-significant, Fs < 1.00, ps > .10, even when examining both linear and quadratic trends.

APPENDIX J

Hyperventilation Questionnaire-Brief

Date ____________ Participant Number___________

Running Session # ___________

Instructions: Please rate the maximum degree to which you are experiencing the following feelings at the present time by placing a circle around the appropriate number.

Feeling	**Not at all**			**Markedly**
Breathlessness	**0**	**1**	**2**	**3**
Feeling distant	**0**	**1**	**2**	**3**
Fear	**0**	**1**	**2**	**3**
Feeling trapped or helpless	**0**	**1**	**2**	**3**
Anxiety	**0**	**1**	**2**	**3**
Rising agitation	**0**	**1**	**2**	**3**
Feeling of suffocation	**0**	**1**	**2**	**3**
Dizziness	**0**	**1**	**2**	**3**
Feeling of losing control	**0**	**1**	**2**	**3**
Worrying that your actions are damaging to your health	**0**	**1**	**2**	**3**
Blurred vision	**0**	**1**	**2**	**3**
Weakness	**0**	**1**	**2**	**3**
Nervousness	**0**	**1**	**2**	**3**
Racing heart	**0**	**1**	**2**	**3**
Feel like passing out	**0**	**1**	**2**	**3**
Fear of a heart attack	**0**	**1**	**2**	**3**
Tension	**0**	**1**	**2**	**3**
Feel like panicking	**0**	**1**	**2**	**3**

APPENDIX K

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