Running head: EXERCISE AND ANXIETY SENSITIVITY

Exercise and anxiety sensitivity: An examination of dose response, credibility, expectancy, and perceived effort.

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Abstract

Despite ongoing research examining exercise and its anxiolytic effects (Petruzello, Landers, Hatfield, Kubitz, & Salazar, 1991), our understanding of this relationship is limited. The present study evaluated the effects of exercise on anxiety sensitivity (AS), and sought to evaluate the effects of various exercise intensities as well as several potential moderating variables including perceived effort, credibility, and expectancy. It was hypothesized that exercise at both mild and moderate intensities would result in a reduction of AS compared to a no-exercise control condition. In addition, it was hypothesized that perceived effort, credibility, and expectancy would moderate the relationship between exercise, and AS. Fifty-five participants (37 females and 18 males) were randomly assigned to one of three groups: 1) a no exercise control condition (n=18), 2) a mild exercise condition (n=19), and 3) a moderate exercise condition (n =18). All participants attended the lab a total of six times over a two week period, and completed measures for perceived effort, credibility, expectancy, and AS. Results indicate that participants in the two exercise conditions experienced a similar decrease in AS while those in the no exercise condition experienced no significant change in AS. This finding suggests that a very mild dose of exercise intervention is sufficient to improve scores of AS in an at risk population. Furthermore, the variables of expectancy, credibility, and perceived effort did not moderate the effects of exercise on AS.

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To all the people who made a difference, you already know who you are.

Thanks! Take Luck!

Table of Contents

List of Appendices

List of Tables

List of Figures

Exercise and anxiety sensitivity: An examination of dose response, credibility, expectancy, and perceived effort

The Greek philosopher Plato wrote, "Lack of activity destroys the good condition of every human being, while movement and methodical physical exercise save it and preserve it", while Edward Stanley, a 19th century British statesman opined, "those who think they have not time for bodily exercise will sooner or later have to find time for illness" (Quotations Book, n.d.). Despite the many centuries that separate these two thinkers, their beliefs regarding the importance of exercise and the consequences of inactivity are virtually identical. Interestingly the idea of 'exercise' as definitive and repetitive movement for the sake of getting in shape, losing weight, or improving health (Caspersen, Powell, & Christenson, 1985), would have been foreign to our ancestors only three or four generations ago. That is not to say that they did not value physical activity, but rather physical activity was simply an ingrained part of everyday life and not one that required special attention. Physical activity several generations ago was attained through chores such as chopping and stacking firewood for the winter or walking several miles into town as the only, rather than an alternative, form of transportation. However, today's society views exercise as an often optional add-on to our already busy lives. As work, leisure, and the population in general become more sedentary (Jebb, & Moore, 1999), the idea of exercise as definitive and repetitive movement for the sake of maintaining and improving health, has become much more important.

The degree of importance that current society has attributed to the idea of exercise is revealed through the most basic examination of the internet or analysis of mainstream advertising. A cursory internet search on topics such as 'exercise', 'physical activity', or 'weight loss' will provide millions of sites claiming (for the right price), to be able to help shed pounds, increase fitness, or tone abs. Additionally, television advertising and infomercials offer a

plethora of gadgets, pills, machines, and short cuts all designed to provide the consumer with a fast, and easy way to exercise, lose weight, and improve health. Not surprisingly, exercise research has mirrored the trends of mainstream society and has put forth considerable effort to understand the role that exercise plays in maximizing, and maintaining our health.

If one were to have the time and inclination to browse all of the websites or watch all of the infomercials broadcast, it would soon become apparent that the primary concern of this advertising and general information focuses on the physical health benefits associated with exercise (Berry, & Howe, 2004; Sabiston, & Chandler, 2009). Some of these benefits include weight loss, improved cardio-vascular health and decreased risk for diseases such as diabetes (Walker, O'Dea, Gomez, Girgis, & Colagiuri, 2010). However, along with the well reported physical benefits associated with exercise, there is also a vast body of literature that highlights the importance of regular exercise with regards to mental health (Conn, 2010; Deslandes et al., 2009; Stein, Collins, Daniels, Noakes, & Zigmond, 2007). This research includes findings supporting the beneficial effects of exercise for psychotic disorders (Ellis, Crone, Davey, & Grogan, 2007), general quality of life (Gillison, Skevington, Sato, Standage, & Evangelidou, 2009), mood disorders (Reed & Buck, 2009), anxiety disorders (Long & van Stavel, 1995; Stich, 1999; Strohle, 2009) as well as executive functioning and degenerative diseases such as Alzheimer's (Perez, Cancela, 2008; Radak et al., 2010). Despite the varied domains in which exercise has a therapeutic effect, the majority of research conducted has been primarily focused on mood disorders and to a lesser extent, anxiety disorders (Salmon, 2001; Stathopoulou, Powers, Berry, Smits, & Otto, 2006).

Exercise and Mental Health

In the past four decades, research evaluating the mental health benefits of exercise has been steadily increasing (Roman, 2010). Early case reports have given way to more refined observational data, population based studies, and experimental research. As noted, the majority of this research has concerned itself with the therapeutic effects of exercise on mood (i.e., clinically depressed individuals), with less attention being given to anxiety and its related disorders (Conn, 2010). Given the surfeit of research available, the conclusions regarding the effects of exercise on mood are reasonably consistent and essentially positive (Mago, & Mahajan, 2010). Both clinical and epidemiological studies have found that individuals who exercise are less likely to feel down (Sylvia et al., 2009), have a greater overall quality of life, feel a greater sense of self efficacy (Kelley, Kelley, Hootman, & Jones, 2009), and experience decreased levels of stress (Ritvanen, Louhevaara, Helin, Halonen, & Hanninen, 2007). In addition, experimental studies and meta-analyses report exercise can have a direct antidepressant effect (Reed, & Buck, 2009). Indeed, many of these studies report findings which indicate that in some instances exercise has been found to be as or more effective than some established pharmacological or psychotherapeutic interventions (Smith, 2006). Physical inactivity, on the other hand, has been linked not only to detriments to our physical health, but may have contribute to disordered mental health as well (Callaghan, 2004).

Although not as extensive as the research on exercise and depression, the anxiolytic effects of exercise have become well documented (Long, & van Stavel, 1995; Stich, 1999). Studies indicate that decreases in anxiety occur after exercise in both clinical (i.e., those diagnosed with an anxiety disorder), and non-clinical (i.e., those experiencing increased general state or trait anxiety or sub threshold levels of anxiety) populations (Salmon, 2001). Furthermore,

these improvements in symptoms and disorders can occur quite quickly and have long lasting effects (Peluso, & de Andrade, 2005). As with exercise and depression studies, some anxiety studies have found that exercise treatments are able to achieve results equal or greater to many standard therapeutic interventions (Byrne, & Byrne, 1993; Strohle, 2009).

Exercise Modalities

In order to gain a better understanding of the beneficial effects of exercise on mental health, researchers have examined both anaerobic and aerobic exercise modalities. Anaerobic exercise generally lasts less than three minutes (i.e., interval training or weightlifting repetition) and results in the consumption of glycogen without oxygen (ACSM, 2011). These exercises, while generally short in duration, can be adjusted to provide low, moderate, or high levels of intensity (i.e., based on amount of weight lifted). Conversely, aerobic exercise is described as any activity which requires elevating and sustaining an increased heart rate (Smits, Berry, Powers, Greer, & Otto, 2008). The American College of Sports Medicine (ACSM, 2011), further defines aerobic exercise as any physical activity during which glycogen is consumed with oxygen and lasts longer than three minutes. This type of exercise activates large muscle groups and can include activities such as walking, swimming, running, or cycling (Pang, 2006). Although both types of exercise are important for health, psychological research has predominantly focused on aerobic exercise. It is possible that this is because anaerobic exercises such as weightlifting may be more technically difficult, require more equipment, and may potentially be intimidating to novice exercisers, whereas aerobic exercises such as walking or jogging, are familiar and require little training or equipment.

Studies evaluating aerobic exercise have examined the anxiolytic effects of low, moderate, and vigorous levels of intensity (Broman-Fulks, & Storey, 2008; Felts, 1989; Oeland,

Laessoe, Olesen, & Munk-Jorgensen, 2010). Moderate intensity exercise has been defined by the Canadian Centre for Disease Control and Prevention (CDC) as 50%-70% of maximal heart rate, while mild and vigorous exercise are considered to be less than 50% and 70%-85% respectively. These levels of intensity are used to help guide individuals in determining appropriate exercise exertion. For example, mild intensity exercise may be indicated for individuals new to an exercise program, recovering from injury, or for individuals with health concerns that would contraindicate more intense levels of exercise. Moderate exercise would be utilized for individuals who want to improve cardiovascular fitness and endurance, are familiar with exercise, and have no injuries or other factors which would prevent safe exercising. Finally, vigorous exercise may be utilized to maximize performance and peak cardiovascular fitness. In order to maintain health and fitness, the CDC guidelines recommend 150 minutes of moderate physical activity every week for an adult.

Although the recommendations for physical activity are often presented as percentages of maximal heart rate, there are many ways to measure exercise intensity. These techniques include ratings of perceived effort, percentage of maximum oxygen uptake, as well as percentage of maximum heart rate reserve. Of these methods the most accurate measure of exercise intensity is the amount of oxygen utilized or VO_2 (Levine, 2008). Although the ACSM recommends utilizing reserve VO_2 (calculate by subtracting resting VO_2 from maximum VO_2) the equipment necessary to measure VO_2 is not readily available to most amateur exercisers and fitness institutions. As an alternative, researchers have found that utilizing heart rate reserve as calculated by the Karvonen formula ([(max HR – resting HR) x % Intensity] + resting HR) provides an exercise intensity that is equivalent to the desired percentage of reserve VO_2 (Swain, & Leutholtz, 1997). This method is useful as it requires no special equipment to calculate and

can be monitored through the checking of one's pulse by hand or with the use of a heart rate monitor.

Evaluation of the anxiolytic effects of exercise has included both anaerobic and aerobic studies. Researchers have primarily evaluated weightlifting as an anaerobic exercise intervention, while aerobic exercise studies typically utilize walking, running, cycling, and swimming interventions (Petruzello et al., 1991). In addition to examining exercise modalities (i.e., aerobic vs. anaerobic), other factors associated with exercise have been evaluated to determine their impact on mental health. For example, researchers have conducted studies to examine: exercise environments such as indoors versus outdoors (Kerr et al., 2006; McAuley, Mihalko, Shannon, & Bane, 1996; Pretty, Peacock, Sellens, & Griffin, 2005), exercising alone or with others (Floyd, 2010), and exercise selection (i.e., self-selected intensity and type of exercise vs. imposed intensity and type of exercise; Mackay, & Neill, 2010). These studies have provided somewhat mixed results with some studies reporting significant effects and others not (Chu, Bushman, & Woodward, 2008; Kruisselbrink, Dodge, & Swanburg, 2004; Strong, Martin-Ginis, Mack, & Wilson, 2006). For example, a 1985 study by Morgan and Raglin, indicated that warming the body (i.e. through exercise or sauna) resulted in a decrease in state anxiety. Conversely in another study published that same year, researchers found that when increasing body temperature through exercise and heat insulating clothing, anxiety actually increased (Reeves et al., 1985). These types of discrepancies have been attributed to the many variations and factors that can confound the findings associated with exercise and anxiety (Petruzello et al., 1991).

Anxiety Sensitivity

Anxiety disorders are one of the most prevalent forms of psychopathology in the United States (Kessler, Chiu, Demler, Merkangas, & Walters, 2005). These disorders can result in an

inability to work, decreased life satisfaction and are responsible for substantial economic costs resulting from loss of productivity and health care expenses. Common anxiety disorders include panic disorder, generalized anxiety disorder, social/specific phobias, and post-traumatic stress disorder. Furthermore, the American Psychological Association (2000) notes that these anxiety disorders are typically chronic, recurrent and often co-morbid with other disorders (i.e., mood and substance use disorders). Understandably, research in this area has sought to identify the factors associated with anxiety in an attempt to prevent or alleviate these disorders and their associated costs.

Within the literature, one identified factor that has a significant impact on anxiety disorders is the construct of anxiety sensitivity (AS). Although AS is not the only important factor with regards to anxiety disorders, this variable has been recognized as being part of the etiology of, and having a mediating effect on many anxiety disorders (Keough, Riccardi, Timpano, Mitchell, & Schmidt 2010). In addition, AS has also been found to play a role in both mood disorders (Otto, Pollack, Fava, Uccello, & Rosenbaum, 1995; Taylor, Koch, and McNally, 1992), and substance use disorders (Forsyth, Parker, & Finlay 2003; Howell, Leyro, Hogan, Buckner, & Zvolensky, 2010; Schmidt, Zvolensky, & Maner 2006). Anxiety sensitivity is defined as the fear of anxiety and anxiety-related sensations arising from the belief that these sensations can have harmful physical, psychological, or social consequences (Arnau, Broman-Fulks, Green, & Berman 2009). As a result, individuals who score high on measures of AS are more likely to be hyper-vigilant for somatic sensations and threatening environmental triggers. Furthermore, those who are high in AS will subsequently interpret those sensations as dangerous, catastrophic, or intolerable (Smits, Berry, Tart, & Powers, 2008).

For example, someone with low AS is not likely to notice a slightly elevated heart rate and would interpret it as something benign if it was noticed (i.e., a result of climbing some stairs). On the other hand an individual with high AS is much more likely to perceive a rapid heartbeat and is more apt to interpret that increased heart rate as something catastrophic (i.e., a symptom of an impending heart attack or stroke). In addition, people with high AS would potentially view their somatic complaints as being noticeable to others, exceptionally embarrassing, or never ending (Stewart, Peterson, & Pihl, 1995). However, it is important to note that all people experience somatic complaints at some point. Nevertheless not everyone reacts with a heightened state of fear or anxiety. Thus, it is the high level of sensitivity and distress as a result of misinterpretation of anxiety related symptoms which are the crucial components of AS (Reiss, 1991). This misinterpretation is fundamental to the attribution theory of anxiety which posits that humans will always attempt to identify and explain causes of behaviour and triggering events. Nevertheless, when an attempt is made to identify the cause of a physiological sensation (i.e., rapid heart rate), without an obvious trigger (i.e., vigorous exercise), there is a potential to misattribute the physiological sensation as harmful, or negative (i.e., I'm having a heart attack). It is this misattribution and subsequent misinterpretations that lead to the fear of physiological symptoms and can develop into panic or generalized anxiety disorder. Finally, AS is considered to be a dimensional construct as opposed to a categorical one (Broman-Fulks, Berman, Rabian, & Webster, 2004). Consequently, rather than being identified as either having AS or not having AS, individual levels of AS can fall along a continuum with some individuals experiencing more fear, distress, and impairment as a result of their AS than others.

The etiology of AS is thought to occur primarily as a result of a combination of genetic factors and learned experiences which shape how an individual perceives and understands

themselves and their environment (Smits et al., 2008; Schmidt et al., 2010). Twin studies estimate the heritability of AS to be as high as 45% (Stein, Jang & Livesley, 1999). However, AS is considered to be distinct from trait anxiety as it has been found to predict fear responses to bodily sensations greater than what is predicted by trait anxiety alone (Rabian, Embry, & MacIntyre 1999). Anxiety sensitivity is also thought to be extremely reliable and stable over time. For example, studies have indicated that high AS in youth and adolescents is a significant predictor of developing an anxiety disorder later in life (Joiner et al., 2002). Anxiety sensitivity differs from trait anxiety due to its focus on metacognitions with respect to fears of anxiety related symptoms (Taylor, Koch & Crockett, 1991). Whereas trait anxiety is related to feelings of worry or discomfort that an individual experiences in general across various situations, AS is the specific metacognitive fears about the symptoms associated with experiencing anxiety.

In terms of clinical usefulness, AS is considered to play an important role in the development and maintenance of panic attacks and panic disorder (Ehlers, 1995; Schmidt, Lerew, & Jackson, 1997; Schmidt et al., 2006). This perspective is supported by studies indicating that people with higher levels of AS have a greater likelihood of experiencing a panic attack and perceiving that panic attack as more threatening (Cox, Enns, Freeman, &Walker 2001; Simon, et al., 2005). Additionally, significant improvements in panic disorder (i.e., decrease in number of panic attacks) have been found to occur when individuals receive therapeutic interventions that cause an amelioration of AS symptomology (Smits, Powers, Cho, &Telch, 2004). As will be discussed below, one such therapeutic intervention that has been hypothesized to be effective in the reduction of AS is physical activity (Broman-Fulks, & Storey, 2008; Smits et al., 2008).

Proposed Mechanisms of Change

Although the mental health benefits associated with exercise are undeniable, exercise is not seen as a panacea, nor is it effective for all individuals. Despite the growing body of literature on exercise and mental health, there are still many questions regarding the cause of its efficacy. Importantly, although exercise is able to have a positive effect on a variety of mental health concerns, there is limited consensus on exactly what the mechanism of action(s) is (Weir, 2011). Thus, gaining an increased understanding into the mechanism of action with respect to exercise and anxiety is an important step for furthering the use of exercise as a treatment. Over the past several decades, a variety of both physical and psychological mechanisms of action have been hypothesized. Physiological mechanisms are thought to improve mood and anxiety by changing physical attributes of the body such has increasing body temperature, regulating sleep, inducing the release of various neurotransmitters (i.e., serotonin), and increased endorphin release. Conversely, psychological mechanisms are thought to impact mood and anxiety by cognitive means such as distraction, improving self-efficacy, or by altering thought patterns (i.e., reducing fear of bodily sensations).

Proposed Physiological Mechanisms.

Thermogenic hypothesis. The thermogenic hypothesis posits that raising the core body temperature (i.e., via sauna or warm bathing) of an individual is able to enhance mood and decrease anxiety (Petruzzello, Landers, & Salazar, 1993). One reason that warming the body is thought to improve affect is due to high rates of muscle tension which often accompany anxiety and mood disorders. Thus, it is thought that as exercise intensity increases, core body temperature will also increase, resulting in an improvement in affect by reducing muscular tension (deVries, 1987). However, this hypothesis has received little support from empirical

studies. For example, Petruzzello et al., (1993) report that studies examining exercise and body temperature have found that moderate exercise for as long as 20 minutes only results in small increases in body temperature (i.e., less than 1°C). In addition, in a study examining the anxiolytic effects of exercise with respect to body temperature, Petruzzello, Landers, and Salazar (1993), had participants run for 30 minutes at 75% of VO_2max, in three different temperature conditions (normal, cooler, and warmer). In the normal temperature condition, participants experienced a typical temperature change associated with exercising. In the cool temperature condition, participants experienced an attenuated increase in temperature change. While in the warmer condition, participants experienced an accentuated increase in temperature change. In contrast to the thermogenic hypothesis, the results of this study showed that although anxiety was reduced following exercise after a 30 minute recovery, the temperature conditions (and subsequently body temperature) explained only a small percentage of the change in variance. Interestingly, this study also found that anxiety actually initially increased in participants in the warmer running condition before decreasing during the recovery phase.

A similarly designed study utilized by Reeves, Levinson, Jestesen, and Lubin (1985), utilized 20 minutes of moderate exercise with participants wearing either comfortable work out attire or vapour barrier and insulated clothing to prevent passive heat loss. The results of this study also refuted the thermogenic hypothesis with participants in the control group experiencing no significant temperature change or an increase in anxiety, while those in the insulated clothing only experienced a temperature change of 1.2°C, and an increase in anxiety.

Although the thermogenic hypothesis in general has not been widely supported some researchers have proposed that increasing the temperature of the brain rather than the core body temperature may play a more important role in improving affect through exercise (DeBoer,

Powers, Utschig, Otto, & Smits, 2012). Since the hypothalamus is associated with emotion regulation (Carlson, 1988), as well as body temperature (Zajonc, & McIntosh, 1992), it is thought that increasing the temperature of the hypothalamus through exercise may produce an anxiolytic effect. Unfortunately, the inability to measure the temperature of the hypothalamus and the brain may preclude further assessment of the thermogenic hypothesis (Yeung, 1996).

Sleep hypothesis. Individuals experiencing mood and anxiety disorders often report disturbances in sleep patterns, such as insomnia, difficulty staying asleep, and reduction in REM sleep (Mellinger, Balter, & Uhlenhuth, 1985; Paffenbarger et al., 1994; Weissman et al., 1997). Furthermore, as many as one third of adults report having sleep difficulties on an ongoing basis (Ohayon & Caulet, 1996). It is thought that sleep serves to conserve energy, restore body tissue, and down regulate body temperature (Wong, Halaki, & Chow, 2013). As a result, research suggests that increasing catabolic activity (i.e., exercising) prior to sleep should increase the need for sleep (as the body recovers from the demands of the exercise), and result in longer duration of sleeping as well as increased REM and slow wave sleep (Taylor, Rogers & Driver, 1997). Thus, the sleep theory proposes that the beneficial aspects of exercise arises from the ability of exercise to regulate the distorted sleep cycle commonly associated with anxiety and mood disorders (Mellman, 2006; Morin & Edinger, 1999).

However, this proposed mechanism has not found extensive support in the literature as the relationship among sleep, exercise, and mental health concerns is still uncertain. Despite the widespread beliefs about the benefits of exercise for improving sleep, the actual effects of exercise on sleep patterns are inconsistent. While meta-analyses have found that in general exercise is correlated with improvements in sleep, findings suggest that the positive effects are not as significant as previously thought (Kubitz, Landers, Petruzzello, & Han, 1996). For

example, during a 12 month exercise program in which participants exercised at a moderate intensity five days per week, no significant differences in slow wave sleep, duration of sleep, sleep efficiency, or sleep onset latency were found between the exercise group and the control group (King et al., 2008). In addition, a study by Yu et al., (2013) found that moderate to large amounts of exercise in the evening did not have a significant effect on sleep quality in college students and that vigorous exercise may actually be a detriment to improved sleep quality.

An additional concern regarding the efficacy of exercise in improving sleep is that a large proportion of the positive effects of sleep have been found in participants who are already categorized as good sleepers. As a result, the positive effects of exercise on sleep may not be generalizable to those with sleep disturbances (Youngstedt, O'Connor, & Dishman, 1997). Studies evaluating the effects of exercise on sleep in participants with disturbed sleep rather than those who are already good sleepers are important given the relationship between sleep disturbances and mental health concerns. For example, an epidemiological survey by Ohayon and Roth (2003) found that insomnia was able to significantly predict psychiatric history. Furthermore, with regards to anxiety disorders specifically, insomnia was found to have occurred prior to the onset of the disorder 38% of the time (Ohayon, & Roth, 2003). However, the relationship between anxiety and sleep disturbance can be bidirectional. Johnson, Roth and Breslau (2006) examined the directionality of risk associated with sleep disturbances and mood disorders and found that while the onset of insomnia preceded depressive disorders, insomnia occurred after the commencement of an anxiety disorder. Furthermore, Papadimitrou and Linkowski (2005) note that although sleep disturbance is a symptom of many anxiety disorders and many individuals with anxiety disorders report difficulties with sleep, objective sleep data

collected in a laboratory indicate that most individuals with anxiety disorders do not demonstrate the same severity of sleep disturbances as they subjectively describe.

In an attempt to directly assess the effects of exercise on sleep and anxiety, Kupfer et al., (1985), utilized a three week protocol, where participants exercised at three different intensities (none, mild, and moderate), and then slept in a laboratory setting for two nights following each exercise session. Results of this study, based on self-report measures for somatic and psychobiological symptoms related to depression and anxiety as well as objective measures of sleep found that there were no significant differences in sleep patterns or somatic/psychobiological symptoms (Kupfer et al., 1985). In another study which focused on insomnia in adult females, Baron, Reid, and Zee (2013), had participants engage in 30 minutes of aerobic exercise three times per week. At the conclusion of 16 weeks, the results suggested that sleep had a greater influence on subsequent exercise rather than exercise affecting sleep. Findings such as this, coupled with the disparity in research findings related to exercise, and sleep, limit the likelihood that improved sleep is the mechanism by which exercise improves anxiety.

Serotonin hypothesis. The serotonin hypothesis posits that exercise interventions impact mental health by altering the release or uptake of the neurotransmitter serotonin (Callaghan, 2004; Stathopoulou et al., 2006). It is hypothesized that an increase in stress can lead to increased levels of cortisol being produced, which in turn impairs serotonin transmission (Heinz et al., 2002; Tafet el al. 2001). This impairment in serotonin transmission is thought to be correlated with depression and anxiety (Charney et al., 2004; Anderson, & Shivakumar, 2013). Consequently, a primary function of many medications designed to treat depression and anxiety has been to increase the amount of serotonin available in the brain (Reus, & Rawitscher, 2000).

Neurotransmitters were first thought to be important as a result of animal studies. For example, researchers noted that the use of electric shock in rats resulted in rats exhibiting behaviours associated with depression and anxiety as well as a concurrent decrease in brain serotonin (Miller et al., 1975). Further studies with animals noted that when rats exercise (e.g., running on a wheel), a corresponding increase in serotonin levels occurs (Dunn, & Dishman, 1991). This increase in serotonin levels, measured with both direct (brain autopsy), and indirect (measuring serotonin levels in blood plasma) methods, is similar to what occurs when the rats are given antidepressants (Dunn et al., 1996; Dishman, 1997).

With respect to studies involving humans, researchers hypothesized that exercise results in an "adaptive down-regulation of postsynaptic serotonin receptors" (Broocks, Schweiger, & Pirke, 1991; p. 387). This assertion is supported by studies comparing participants with various levels of activity. Researchers compared marathon runners to a control group of sedentary participants (Broocks et al., 1998). In the study, researchers gave participants metachlorphenylpiperazine (m-CPP), which is an agonist for the 5-HT receptor (a receptor site for serotonin), and typically results in an increase in cortisol being produced. Results from the study showed that while the sedentary control group experienced the expected increase in cortisol levels, marathon runners exhibited diminished levels of cortisol in response to the m-CPP (Broocks et al., 1999). This finding suggests that the reduction in hormonal response to m-CPP is mediated by postsynaptic 5HT receptors, which may indicate that beneficial effects of exercise with respect to anxiety and depression may be mediated by the down-regulation of the 5-HT receptor (Broocks et al., 1998).

In order to directly assess the effect of exercise on serotonin levels, depression, and anxiety, Wipfli, Landers, Nagoshi, and Ringenbach (2011), randomly assigned 72 non-exercising

participants to either an aerobic exercise condition or a stretching control group for seven weeks. In the exercise condition participants completed three 30 minute sessions of cycling on a stationary bike at 70% HR_{max} three times per week, while those in the stretching group participated in light stretching and yoga. Upon completion of the seven weeks, researchers found that participants in the exercise condition reported lower scores on measures of depression than those in the control group, while participants in both groups experienced a similar decrease in anxiety. With respect to serotonin, it was found that participants in the cycling group experienced a significantly greater decrease in blood serum serotonin compared to the stretching group. The relevance of this finding is predicated on research findings that serotonin levels in platelets, plasma, serum, and whole-blood are significantly decreased when SSRIs are given (Hughes, Petty, Sheikha, & Kramer, 1996; Moreno et al., 2006). This decrease in serotonin levels in the blood is thought to reflect changes in serotonin levels in the brain as evidenced in rat studies (Svenningsson et al., 2006).

However, there are some significant limitations associated with the study of exercise and serotonin. With respect to study design, accurate tools to measure brain serotonin in live participants are currently unavailable. As a result, only indirect methods of measuring serotonin (i.e., blood tests for serum serotonin), which are hypothesized to be related to brain serotonin levels but not yet supported, are utilized (Wipfli et al., 2011). In addition, studies also use primarily healthy individuals and thus may not provide information on the antidepressant or anxiolytic effects of exercise that is generalizable to a clinical population. Thus, an unfit and highly anxious individual may experience greater gains associated with exercise than the healthy individuals being studied (Martinsen, Hoffart, & Solberg, 1989). Finally, much of the research examining the relationship between neurochemical imbalances in the brain and mood/anxiety

disorders is based on the idea that medications such as SSRIs are effective treatments. However, a new analysis of antidepressant research, including previously unpublished studies, indicates that much if not all of these findings are actually placebo effects in action (Kirsch, 2011). As a result, the antidepressant and anxiolytic effects of neurotransmitters such as serotonin, whether influenced by medication or exercise, are called into question.

Endorphin hypothesis. Another theory proposed to explain the beneficial effects of exercise is the endorphin hypothesis. Endorphins are endogenous opioids that occur naturally within the body, and it is the Beta Endorphins that have been the most widely researched in terms of mood, anxiety, and mental health (Sforzo, 1988). The role of endorphins in the human body is an analgesic one that serves to decrease pain and regulate emotion (Bodnar, & Klein, 2005). Furthermore, both animal research, and human clinical studies suggest that there is a strong correlation between low levels of beta endorphins present in the body, and increased rates of depression and anxiety (Scarone et al., 1990; Darko, Risch, Gillin, & Golshan, 1992; Hegadoren, O'Donnell, Lanius, Coupland, & Lacaze-Masmonteil, 2009). Thus, it is posited that exercise acts to increase the release and binding of β-endorphins (endogenous opioids) to their receptor sites in the brain. Subsequently, the greater availability of endorphins is thought to create a more positive and euphoric state that can also alleviate the unpleasantness of anxious mood states (Wipfli et al., 2011).

For example, when opioid agonists are administered to rats in an acute stress environment, anxiety related behaviours (e.g., avoidance) are decreased (Asakawa et al., 1998; Zarrindast, Lahmi, & Ahamadi, 2008). Conversely, when an opioid antagonist such as naloxone is given, the anxiety related behaviours are increased (Koks, Bourin, Voikar, Soosaar, & Vasar., 1999). The naloxone has also been used extensively as an opioid antagonist in human research.

For example, researchers found that an initial administration of naloxone (4mg/kg) in healthy participants resulted in higher self-ratings of anxiety and anger (Pickar, Cohen, Naber, & Cohen, 1982). Additionally, researchers have found that when naloxone was administered to patients scheduled for minor surgery, the anxiolytic effects of diazepam were mitigated (Duka et al., 1982). In a related study by Esquivel, Fernandez-Torre, Schruers, Wijnhoven, and Griez, (2009), the researchers utilized a double blind randomized crossover design to test the effect of an opioid antagonist and placebo during carbon dioxide induced panic conditions. The findings from this study indicate a significant increase in discomfort and anxiety, including significant panic symptoms in some participants when given the opioid antagonist (Esquivel et al., 2009). In addition, Pickar et al., found that when compared with a group of normal participants, psychiatric patients showed a decrease in levels of opioids which correlated with an increase in both manic-depressive symptoms and anorexia (1982).

Although there seems to be some degree of support for the endorphin hypothesis, several limitations prevent its full acceptance as the mechanism of action for the mental health benefits of exercise. First, as with serotonin, it is impossible to directly measure endorphin levels in the brain. As a result, indirect methods of measurement are utilized (i.e., blood plasma levels of endorphins). Therefore, although it is thought that blood plasma endorphin levels reflect changes in endorphin levels in the brain, it is impossible to be certain what changes are actually occurring in the brain (Goldfarb, & Jamurtas, 1997). It is also evident that the majority of the research in this area is conducted using healthy participants. As a result, it is difficult to predict how the effects of exercise would affect endorphin levels of those who are sedentary or obese or who are suffering from anxiety or depression.

Second, the endorphin producing effects of exercise seem to occur primarily at high intensities or long durations of exercise. If this is the case, it would conflict with evidence that short bouts of mild to moderate intensities of exercise have been found to positively impact mental health. For example, in a study examining the relationship of exercise and endorphins, Donevan and Andrew (1987), had participants exercise on a cycle ergonometer for 8 minute sessions at 25%, 50%, 75%, and 95% of VO_2max. Blood samples collected before, during, and after the exercise indicated that plasma β-endorphins, did not change significantly for the 25% and 50% of VO_2max, but did experience a significant increase for the 75% and 95% VO_2max exercise, with elevations being maintained for 30 minutes post exercise (Donevan, & Andrew, 1987). In a similar study, researchers measured β-endorphin levels after participants cycled at 85% and 100% of their individual anaerobic threshold (Gabriel, Schwarz, Steffen, & Kindermann, 1997). The results of this study indicated that β-endorphin levels increased at 100% IAT but not 85%. Goldfarb, Hatfield, Armstrong, and Potts (1990), also found that when participants exercised at 60%, 70%, and 80% of VO_2max for 30 minutes on a cycle ergonometer, β-endorphin levels increased for the 70% and 80% intensities but not for the 60% intensity (Goldfarb et al., 1990).

The third limitation to accepting the endorphin hypothesis is the conflicting results in studies utilizing opioid antagonists such as naxolone. If endorphins are primarily responsible for the anxiolytic and mood enhancing effects of exercise, opioid antagonists should consistently mitigate this effect. However, several studies have shown that this is not always the case (Carr et al., 1981; Thoren et al., 1990; Yeung, 1996). For example, Markoff, Ryan, and Young (1982), utilized a double blind, counter balanced method to compare the effects of naxolone and placebo on the Profile of Mood States (POMS) scores of long distance runners. The results of this study

found no significant effect of naxolone on POMS scores compared with placebos (Markoff et al., 1982). Results such as this imply that mood changes associated with exercise may not be mediated by endorphins.

Proposed Psychological Mechanisms

Along with the potential physiological explanations for exercise interventions, several psychological possibilities have also been proposed. Of these mechanisms, the most commonly studied have been the distraction hypothesis, the self-efficacy/mastery hypothesis, and the exposure hypothesis. However, many of these theories have not been tested extensively (Craft, & Perna, 2004).

Distraction hypothesis. The distraction hypothesis was first suggested by Barhke and Morgan, in their study comparing exercise, meditation, and quiet rest. In the study, the researchers randomly assigned participants to either a 20 minute exercise condition (70% of self-imposed HRmax), 20 minutes of meditation (Benson's relaxation response), or 20 minutes of quiet rest sitting in a recliner (with an option to read a magazine). The results of this study found that state anxiety was significantly decreased in all three conditions (Bahrke, & Morgan, 1978). In their discussion, the authors suggest that the benefits of exercise may not be a result of physiological changes but rather in how "diversional" it is (p. 331). In essence, it is argued that exercise allows individuals to become distracted from most everything else except the activity they are engaged in. Thus, anxious or depressing thoughts or distressing somatic complaints are diverted from conscious attention, and a relaxing affect (relative to the disordered mood or anxious state) occurs (Morgan, 1985).

These findings were supported by Breus and O'Connor (1998), who tested the 'time-out' hypothesis in high anxious females. In this study, participants completed four conditions in a

random order: Exercise Only (20 min of cycling at 40% VO_2max, followed by 20 minutes of recovery), Study Only (40 minutes of studying), Exercise/Study (20 minutes of cycling while studying followed by 20 minutes of studying only), and Control (sitting for 40 minutes). Results from this study indicate that a significant change in state anxiety only occurred for the exercise only condition. Thus, the authors assert that the use of a studying protocol during exercise prevented the participants from getting a 'time-out' which prevented a reduction in state anxiety normally associated with exercise (Breus, & O'Connor, 1998). Breus and O'Connor also found that participants did not experience a decrease in state anxiety while in the quiet rest condition, which supports the assertion that changes are not simply due to a passage of time. However, as other studies have reported that quiet rest can result in a decrease in state anxiety, the authors suggest that sitting on an uncomfortable cycle ergonometer and lack of reading material (as in the Bahrke and Morgan study), may have contributed to these differences.

Despite these findings, acceptance of distraction as the primary mechanism of change with respect to exercise does not seem fully supported (Anderson, & Shivakumar, 2013). One finding which challenges the distraction hypothesis is that exercise interventions generally produce results that last significantly longer than other forms of distraction (Morgan, & O'Connor, 1989). For example, Raglin and Morgan (1987) conducted two experiments comparing exercise and quiet rest. In the first study, 40 minutes of quiet rest was compared with 40 minutes of self-selected exercise activity and intensity in normotensive participants. The results indicated that blood pressure (BP) and state anxiety decreased for both conditions. However, improvements resulting from the exercise conditions were maintained for 2-3 hours post exercise while improvements resulting from the quiet rest condition returned to baseline after only 20 minutes (Raglin, & Morgan, 1987). A similar experiment was then conducted

utilizing participants who were receiving pharmacotherapy for hypertension. The results of this study indicated that improvements in BP only occurred in the exercise group, while both the exercise group and quiet rest group experienced a reduction in state anxiety. Moreover, as with the first experiment, anxiolytic improvements after exercise again lasted significantly longer than improvements after quiet rest.

Another limitation to the distraction hypothesis is that comparable findings between exercise and other forms of distraction (e.g., meditation, quiet rest, etc.) are generally associated with state anxiety (Stathopoulou, Powers, Berry, & Smits, 2006). In a meta-analysis of acute and chronic exercise, Petruzzello, Landers, Hatfield, Kubitz, and Salazar (1991), found that exercise exhibited a greater effect of change on trait anxiety than for state anxiety, with the greatest changes occurring in exercise protocols lasting several weeks. Furthermore, although other forms of distraction are able to temporarily reduce state anxiety, they do not have a similar effect on trait anxiety.

Self-Efficacy hypothesis. A second proposed psychological mechanism to explain the effects of exercise is the Self Efficacy hypothesis. Self-efficacy refers to the belief in one's ability to achieve goals, complete tasks in demanding situations or successfully manage threats (Bandura, 1997). Self-efficacy with respect to exercise suggests that achieving target goals (e.g., running a certain distance or improving cardiovascular fitness) improves mental health by increasing self-confidence, and providing an enhanced ability to meet challenges (Paluska, & Schwenk, 2000). In principal, as fitness levels increase through repeated exercise, feelings of fatigue and pain will attenuate and self-efficacy should increase. Subsequently, if the exercisers believe that their abilities have increased (e.g., self-mastery), anxiety about future 'threatening'

or challenging situations should be decreased as well (Anderson, & Shivakumar, 2013; DeBoer, Powers, Utschig, Otto, & Smits, 2012; Petruzzello et al., 1991).

The mediating effects of self-efficacy with respect to exercise and mental health have been evaluated by relatively few studies in the literature. For example, Bodin and Martinsen (2004), compared mood changes in clinically depressed participants after engaging in two different forms of exercise. It was hypothesized that an exercise intervention with high and stable self-efficacy (e.g. riding a bike), would be less likely to improve affect, then exercise that was initially low elf-efficacy but increased over time (martial arts). It is posited that activities which are continuous, have no specific beginning or end, and do not change in experience, provide less feedback and fewer sources of self-efficacy. Conversely, activities which are more complicated and require a series of discrete skills being combined provide the greatest potential for feedback and self-efficacy (Schmidt, 1999). Thus, a repeated measures counterbalanced design was utilized with participants participating in 45 minutes of either cycling or martial arts on separate days. Results indicate that when participants participated in the martial arts exercise, they experienced a significantly greater increase in positive affect and self-efficacy, as well as a significant decrease state anxiety. However, no significant changes were noted for the cycling exercise (Bodin, & Martinsen, 2004).

Additionally, a study of low active women utilized a self-efficacy manipulation to determine its effects on subsequent self-efficacy and anxiety (Marquez, Jerome, McAuley, Snook, & Canaklisova, 2002). In this study, participants were randomly assigned to either a high efficacy (HE), or low efficacy (LE) group. Each participant participated in an initial graded exercise test on a treadmill and was then given either high or low feedback regarding their cardiovascular health. Participants in the HE group were informed that their results placed them

in the top 20% in relation to their peer group, while participants in the LE group were informed that their results placed them in the bottom 20% in relation to their peer group. All participants returned to complete a 20 minute acute exercise session several days later. Results from this study indicated that although the participants did not initially differ in self-efficacy, after the false feedback was given participants in the HE group reported greater feelings of self-efficacy than those in the LE group. Furthermore, participants in the HE group also reported significantly less anxiety than those in the LE group after the graded exercise test and after the acute exercise session (Marquez et al., 2002).

In spite of evidence of studies such as these, self-efficacy has similar limitations to some of the other mechanism proposed. It is clear that engaging in new and challenging activities (e.g., martial arts), intense physical exercise (e.g., high VO_2max), or prolonged exercise programs may provide opportunity for the development of self-efficacy and subsequent improvement in mood or decrease in anxiety. However, these findings do not support research findings which indicate anxiolytic or mood improvement after exercise sessions of short duration and mild intensity. These studies often utilize exercise conditions that are highly stable in terms of skill development and self-efficacy such as walking or cycling and thus provide limited opportunities for increasing self-efficacy.

As an example, one such study examined the effects of exercise at varying intensities on changes in self-efficacy and anxiety (Katula, Blissmer, & McAuley, 1999). In this study, researchers randomly assigned eighty older participants to a mild (self-selected walking intensity –approximately 30% of Heart Rate max), moderate (timed completion of a 1-mile fitness test – approximately 50% of Heart Rate Max), or high intensity (graded exercise test –approximately 96% of HRR) exercise condition. The results of this study indicate that state anxiety significantly

decreased for the light exercise group while the moderate exercise group experienced no change and the high exercise group experienced an increase in state anxiety. However, when State Anxiety Inventory items for arousal were controlled for, all groups experienced a significant decrease in anxiety. As self-efficacy is not thought to be related to mild intensity stable exercises, this result would seem to suggest that another mechanism may be at work with respect to the changes in anxiety in the mild and moderate exercise groups.

Exposure hypothesis. The physiological symptoms associated with panic and anxiety are often identical to the physiological sensations associated with exercise (e.g., rapid heart rate, sweating, changes in breathing, etc.). Anxiety Sensitivity itself is a fear of these bodily sensations and researchers have found that there is a negative correlation between AS and amount of exercise (McWilliams, & Asmundson, 2001). This may occur due to individuals with anxiety mistakenly misinterpreting the physical sensations associated with exercise as anxiety. Thus, the exposure hypothesis proposes that exercising provides a controlled exposure to potentially fearful physiological symptoms and results in an increased tolerance for such symptoms, as the individual learns they may be uncomfortable but are not threatening (Antony, Ledley, Liss, & Swinson, 2006; Strohle, 2009).

Evidence for the exposure hypothesis has been derived from studies which have utilized experimentally induced panic attacks. In one study, experimenters compared participants diagnosed with panic disorder to age matched controls (Strohle, 2009). All participants completed both a quiet rest experimental condition and a 30 minute walking exercise session at 70% VO_2max. Immediately following the experimental condition, participants were injected with cholecystokinintetrapeptide (CCK-4), which induces panic symptoms. Results of this study found that administration of CCK-4 after the quiet rest session resulted in 75% of the participants

with panic disorder and 50% of the healthy control participants experiencing a panic attack. However, administration of CCK-4 after the exercise condition resulted in only 33.3% of the panic disorder participants and 8.3% of the control participants experiencing a panic attack (Strohle, 2009). A similar study randomly assigned patients with panic disorder to either a light (cycling at 1 watt per kg weight -20), or moderate (cycling at 100 -150 watts) exercise condition. Upon completion of the exercise a 35% CO_2 panic provocation procedure was induced. As with the previous study, panic reactions as measured by the Panic Symptom List (PSL), and the Visual Analogue Anxiety Scale (VAAS), were significantly smaller in patients in the moderate exercise group compared with the light exercise group (Esquivel et al., 2009).

Dose Response

One of the most important questions studied by pharmacological treatment researchers is the notion of dose response. Essentially, the dose response question attempts to determine how little of a treatment is necessary to be able to achieve the desired results. As an example, if a new antidepressant medication is approved for testing, researchers can begin treating depressed individuals, and slowly titrate the dose over time until the desired effect (i.e., increased positive mood) is attained. Once the desired effect is achieved, researchers now have a basic idea of what minimum level of this particular medication is necessary for treatment efficacy (Schatzberg, Cole, & DeBattista, 2007). This understanding is crucial in order to minimize any possible negative side-effects associated with the proposed treatment, ensure treatment adherence, and to provide the most cost effective treatment possible. With regards to exercise and mental health, the question of dose response is as critically important as it is for pharmacological medication trials.

Like many pharmacological treatments, physical activity as an intervention is prone to side effects. Fortunately, the majority of the side effects associated with exercise are positive (i.e., increased cardiovascular health, weight loss, improved biological rhythms), and not likely to dissuade someone from adhering to treatment (Johnsgard, 2004). However, there are certain situations in which exercise may result in negative side effects. For example, individuals with extreme panic disorder may experience a panic attack at the first sign of a somatic trigger (many of which are associated with exercise) such as increased heart rate or sweating (Broocks et al., 1998). In addition, for individuals with extremely low body weight, any weight loss as a result of exercise may be perceived as a negative side effect rather than a positive one. These situations, although rare, require careful prescription, and implementation of exercise treatments in order to minimize any potential harm.

In addition to potential negative side effects, treatment adherence is potentially one of the biggest barriers for exercise interventions (Jones, Harris, & McGee, 1998; Whiteley, Williams, & Marcus, 2006). Unlike a pharmacological intervention which generally only requires swallowing a pill, exercise interventions require much more time and effort on the part of the participant. Reasons for not exercising can include lack of time, lack of desire to engage in physical activity or other factors that preclude someone from exercising (i.e., difficulty in beginning or maintaining an exercise regime). In a study implementing a 30 minute exercise program five days a week, researchers found that 22% of participants dropped out early in the study and of the 78% who completed the study, only 4.7% completed the full 150 minutes of prescribed exercise per week (Arikawa, O'Dougherty, Kaufman, Schmitz,, & Kurzer, 2011). In a study of 248 elderly Australians, researchers reported an attrition rate of 35% and noted loneliness, low socioeconomic status, and higher obesity rates as predictors of attrition (Jancey et

al., 2007). However, determining minimal doses of exercise necessary for positive treatment may overcome these barriers. For example, studies have found that although some conventions for a healthy lifestyle include moderate exercise for 30 minutes each day, the 30 minutes can be broken up into several shorter exercise sessions throughout the day (DeBusk, Stenestrand, Sheehan, & Haskell, 1990; Schmidt, Biwer, & Kalscheuer, 2001). This type of evidence is important when prescribing exercise for individuals who lack time and motivation to exercise for longer periods. Furthermore, minimum dose requirements which minimize any potential negative side effects (e.g., exposure to somatic symptoms) are likely to increase treatment adherence.

Finally, although the costs of pharmacological and psychotherapeutic treatments can be quite high, exercise treatment does not share this disadvantage. Obtaining a pair of supportive shoes for walking are all that are necessary to engage in moderate levels of physical activity (i.e., walking or jogging). On the other hand, if more varied exercises are required or preferred, a gym membership providing access to swimming pools, weights, and cardiovascular machines can be obtained for a nominal fee. However, much like the various medications available to treat single disorders, there are myriad exercise modalities that may have varying effects on the mental health outcome being pursued. For this reason, comparative studies may find that 10 minutes of one particular exercise may be equivalent to 20 minutes of a different type. Traditionally, most exercise studies have focused on walking or jogging, exercise bicycles, and swimming. In addition, it is necessary for researchers to establish norms and minimum doses of these activities in order to provide a comparison for other activities (e.g., Yoga, Tai Chi, Ultimate, Volleyball, etc.).

Ratings of Perceived Exertion

Once the minimal dose has been established, there are many factors which may influence the actual dose of a treatment prescribed to a patient. With regards to medications, body mass may play a role in determining an effective dose. In addition, other variables such as drug-drug interaction with other health concerns, health concerns which may contraindicate the administration of certain medications (e.g., low blood pressure), and treatment resistance may require that adjustments be made to the prescribed dose (Schatzberg et al., 2007). Given the positive physical health benefits associated with exercise the lack of negative side effects, the above concerns may not be as relevant to exercise dose response research. However, one aspect of exercise treatment that may in fact be quite important for determining the optimal amount of each exercise activity is the concept of perceived effort. Perceived effort simply refers to how much effort participants perceive they have put forth to engage in a particular activity (Lagally, & Amorose, 2007). Therefore, two individuals exercising on identical treadmills at an identical intensity level for an identical amount of time may have widely different perceptions of how much effort it took to complete that exercise session. Thus, individuals who are physically fit and enjoys exercising may perceive their overall effort as lower than someone who is overweight and loathes exercising. At this point it is not clear how perceived effort may impact the effects of exercise. Although perceived effort may impact an individual's expectancies about the effects of exercise, it may also be that perceived effort has a unique contribution to the outcome of exercise (Dunn, Trivedi, & O'Neal, 2001).

When individuals are asked about the reasons they exercise, two common answers given are: a) positive feelings experienced by meeting personal challenges and achieving goals, and b) the positive feelings experienced after exercising referred to as 'afterglow' (Johnsgard, 2004).

These two motivators are intrinsically linked with perceived effort in that, without expending effort one will not be able to meet exercise goals and feelings of afterglow may not be experienced (Johnsgard, 2004). Given the increased positive feelings associated with increased effort, increasing effort should also have a positive impact on other beneficial aspects associated with exercise (e.g., reduction in anxiety or depression). Thus, greater reductions in anxiety or mood symptoms may be related to increases in perceived effort.

Credibility and Expectancy

The Latin origin of the placebo effect is translated as meaning 'I shall please' (Wampold, Minami, Tierney, Baskin, & Bhati, 2005), which highlights an early conceptualization which postulated that patients want to improve for their doctors. Thus, whatever was prescribed (regardless of efficacy) had a healing effect (Price, Finniss, & Benedetti, 2008). However, after decades of research into the topic, the placebo effect is understood to be a much more complex participant. Indeed, the placebo effect and the nocebo effect (when negative expectations result in negative results: Hahn, 1997) have been found to have a great impact across gender, culture, age, and both clinical, and non-clinical populations (Kirsch, 1997; Stewart-Williams, 2004). These effects include, but are not limited to, decreasing blood pressure, inducing tranquilizing or analgesic effects, and inducing affects associated with substance use (Kirsch, 1997).

By definition, a placebo is considered to be any inert substance, treatment or procedure that has a therapeutic effect (Cheyne, 2005). Furthermore, a placebo effect is said to have occurred only when a change is solely the result of the placebo administered (Stewart-Williams, 2004). Two factors are vitally important to be able to identify a true placebo effect. First, the pill, treatment, or procedure must lack any ingredient which may actually have an effect on the condition or problem being investigated. Secondly, the individuals who receive the placebo must

have an expectation that the placebo will actually have an effect (Frank, 1973; Parloff, 1986). Although it is difficult to control for all potential confounds, ensuring that a pill or treatment is inert is generally a straightforward task. However, many factors impact the expectations of individuals and must be carefully considered when conducting research in this area. Two important components that have been found to influence placebo are a participant's expectations about the treatment (what they expect will happen as a result of taking the placebo) and their perceived credibility of the treatment (how credible the treatment seems).

With respect to expectancy, two prominent theories on why the placebo effect occurs are that of classical conditioning and the expectancy effect (Cheyne, 2005; Parloff, 1986). In brief, the classical conditioning hypothesis posits that, much like Pavlov's bell experiments, the placebo effect occurs after an individual has been conditioned to receive and expect a certain response (Ader, 1997). For example, if taking a small white pill to alleviate the pain from a headache is effective and produces similar results on subsequent trials, then an individual may begin to feel pain relief just by seeing the white pill since they have learned to associate pain relief with the small white pill. However, this theory does not satisfactorily account for the placebo effects since results can occur as a result of the first administration of a placebo without the opportunity for learning to occur (Montgomery, & Kirsch, 1997).

Conversely, the expectancy hypothesis proposes that simply expecting an outcome to occur will cause that outcome to occur (Stewart-Williams, 2004). For example when individuals expect that a particular beer will taste bitter, when they drink the beer, they are more likely to perceive the beer as bitter (Kirsch, 1997). Unlike classical conditioning which requires specific learning opportunities directly with the stimulus and outcome, researchers have found that expectancies can be acquired in various ways including personal experience, observational

learning, and verbal information (Cheyne, 2005). Personal experience, much like classical conditioning gives rise to expectancies when an individual experiences a stimulus and subsequently associates a particular outcome with that stimulus. Observational learning on the other hand, can occur when an individual merely observes another individual experience the outcome of some explicit stimulus. Finally, verbal information can induce expectancies by providing information which causes an individual to believe that not only is the information credible, but that it is relevant to his/her personal self.

With respect to credibility, research has found that higher scores on measures of credibility are also positively associated with greater reduction in symptom severity (Smeets et al., 2008). For example, studies have found that in pill placebo conditions, two pills or a larger pill is believed to be more effective or credible than one pill or a smaller pill thus increasing the effect of the placebo (Buckalew, & Ross, 1981; Stewart-Williams, 2004). In addition, injected treatments are generally believed to be more credible or effective than capsule forms of treatment (Kaptchuk, 2000; Kaptchuk et al. 2006).

With regards to cognitive placebos (i.e., psychotherapeutic treatment or information designed to increase or decrease expectancy), research indicates that in addition to the actual information being provided, clinician or facilitator factors play the largest role in impacting the beliefs of participants (Lundh, 1987). For example, if a clinician or facilitator behaves in a way that conveys trustworthiness and expertise, the patient or participant is more likely to believe the information given by the clinician and perceive it as more credible. In addition, the more optimistic and enthusiastic a facilitator or a clinician is regarding the treatment or information, the more likely it will be that the beliefs of the individuals will be affected (Lundh, 1987). These examples are important as they underscore the necessity for sound methodology when

conducting research evaluating expectancy. Without careful consideration of the effects of expectancy and credibility a dual danger exists. First, researchers may unwittingly consider change that has occurred to be a direct result of their treatment rather than as a result of the expectations of the participants. Second, researchers may ascribe the change that has occurred to expectancies of the participants when in reality it was actually a result of their intervention or some other uncontrolled for confound (Stewart-Williams, 2004).

Previous Research

Traditionally, research studies evaluating placebos have been developed to compare the effectiveness of pharmacological interventions. This is due to the ease with which pharmacological placebos can be created and administered (i.e., inert sugar pills that look identical to the treatment pill). However, in recent years, researchers have begun attempting to determine the placebo effects associated with other psychotherapeutic interventions such as cognitive behavioural therapy and eye movement desensitization and reprocessing. Unfortunately, with regards to expectancy and credibility, exercise remains a largely unstudied area. Part of the reason for this is due to the fact that unlike pill or cognitive placebos it is extremely difficult to produce a satisfactory exercise related placebo (Ojanen, 1994). To date, most studies examining expectancy credibility of exercise interventions have focused on the role that these variables play in program adherence rather than as an active agent of change (Jones, Harris, Waller, & Coggins 2005; Resnick 2004; Desharnais, Bouillon, & Godin 1986). Similar to the amount of research on exercise and AS, very little research has been conducted in this area and only two studies have been conducted which examine the role of expectations in exercise outcome.

Despite the difficulties inherent in research evaluating the placebo effect in exercise, an early attempt to answer this question sought to modify the expectations of participants prior to exercise induction (Desharnais, Jobin, Cote, Levesque, & Godin, 1993). In this novel study 48 participants were randomized into either an experimental or control condition. Both groups participated in identical training programs which occurred three times per week over the course of 10 weeks. All sessions were led by the same two facilitators and were 90 minutes in length, including a 10 minute warm-up period, 70 minute activity, and 10 minute cool-down period. The nature of these activities was primarily group-based and consisted of activities such as jogging through city streets, aerobic dancing, playing soccer, and so on. The difference between the two groups consisted of information and feedback provided by the facilitators. For the control condition, participants were informed by the facilitators that their training program was intended to improve aerobic capacity. On the other hand, the participants in the experimental condition were informed that the intention of their exercise program was to improve aerobic capacity as well as psychological well-being. Throughout the program participants were reminded of the purpose of the exercise and were encouraged by the facilitators to attend to any biological and in the case of the experimental group, psychological improvement. Participants were subsequently assessed for physical and psychological well-being (as measured by the Rosenberg Self Esteem Scale) over a 12 week period (i.e., one week prior to and one week after completion of the exercise program).

As expected, the findings from this study indicated that participant expectations may be one of the factors involved in the psychological improvement associated with exercise. Although both groups achieved similar levels of aerobic improvement, the experimental group reported significantly higher levels of self-esteem than the control group. Unfortunately, methodological

limitations within this study serve to mitigate the findings. The authors acknowledge that a true expectancy effect can only be attributed when the control group truly does not expect any significant improvement to occur. However the participants in the control group in this study reported high expectations for psychological benefits from physical activity even without the information provided to the experimental group (Desharnais et al., 1993).

The only other study to this author's knowledge that has attempted to evaluate exercise and the expectancy effect examined the impact of participant expectations on physical health (Crum, & Langer, 2007). In this particular study, participants were female room attendants working at several different hotels (n = 84). The rationale behind this study stems from research indicating that the recommendation of 30 minutes of physical activity per day (i.e., walking or climbing stairs) can be met through the demands of housework, or in this case, 'hotel work'. On average, room attendants clean 15 rooms per day, with each room taking approximately 20-30 minutes to clean. This task requires a variety of physical exertions including heavy lifting, walking, pushing, and carrying heavy loads. However, the researchers hypothesized that room attendants may not see their work as exercise and would thus, have lowered expectations about the benefits of their work. The researchers attempted to increase the expectation of room attendants by providing the experimental group of room attendants with information describing the benefits of exercise and how their daily tasks met and in fact exceeded daily exercise requirements. In addition, information regarding the amount of calories burned through various activities was included. Conversely, the control group received no such information. In order to measure the impact of this information on physical health, the researchers measured each participant's weight, percentage of body fat, waist to hip ratio, and blood pressure.

After four weeks, during which time the attendants continued work as usual, the same physiological measures were recorded. As hypothesized, the participants in the informed group lost an average of 2 pounds, lowered their blood pressure, and had lower body fat percentages and waist to hip ratios. All of these gains were significantly greater than changes in the control group. Although all participants reported no significant changes in lifestyle over the course of the study it is possible that other factors contributed to the weight loss and other health improvements (i.e., change in working style such as taking the stairs more and improved eating habits). As a result, more study is necessary to understand the effects of exercise on physiological and psychological well-being, with specific emphasis on the mechanisms of change such as the placebo effect (Crum, & Langer, 2007).

Previous Anxiety Sensitivity Research

As one of the risk factors for panic disorder is AS, it is not surprising that CBT techniques such as interoceptive exposure, which serve to reduce panic symptoms, also result in a decrease in AS. To date, only three studies appear to have experimentally examined the impact of exercise on AS. The first such study to attempt to investigate this relationship in an experimental design was conducted by Broman-Fulks and colleagues in 2004. This initial study utilized 54 participants who met all inclusion criteria including an elevated score on the Anxiety Sensitivity Index (ASI; i.e., greater than 25). These individuals were then randomly assigned to either a high or mild intensity exercise condition which consisted of six 20-minute treadmill sessions completed over a two week period. The high intensity group was required to exercise on a treadmill (the speed of which was controlled by the experimenter who was monitoring heart rate) at a rate between 60-90% of their maximal heart rate. The mild intensity group on the other hand was asked to walk no faster than one mile per hour for the 20 minute session. The

researchers measured AS prior to the beginning of the first exercise session, immediately following the final exercise session, and at one week follow-up. Results indicated that both exercise groups experienced a decrease in AS as evidenced at the one-week follow up. It is interesting to note that although the individuals in the high intensity group initially responded faster (i.e., had earlier decreases in AS), at one week follow up, results between the two groups were similar. It is also important to highlight the extremely moderate pace of one mile per hour in the mild intensity group given that a key component of many effective treatments for panic and panic symptoms is interoceptive exposure (Forsyth, Fuse, & Acheson, 2008). Interoceptive exposure involves mimicking panic symptoms in a therapy session so that clients can be exposed to the panic inducing somatic complaint so that they experience a decrease in anxiety over time (Forsyth et al., 2008). Examples include spinning in a chair to induce dizziness, running on the spot or up and down stairs to induce a rapid heartbeat, or breathing through a straw to mimic shortness of breath. Since individuals with panic disorder fear these somatic symptoms they will avoid any and all behaviours that may cause them to experience such physiological responses.

For example, if an individual feels that an increased heart rate is a symptom of a heart attack he/she may avoid running, climbing stairs, or other behaviours that may induce an increased heart rate. As a result, avoidance behaviours reinforce irrational cognitions and fears on which panic disorder is based. The intent of interoceptive exposure is to provide opportunities for individuals to experience the feared physiological symptoms without avoiding or attempting to neutralize the symptom. As a result, individuals are able to experience the symptom, challenge the irrational fear or cognition and decrease their panic (Schmidt, & Trakowski, 2004). For interoceptive exposure to be effective, the activity (i.e., breathing through a straw) must be effective at inducing the feared physiological symptoms (i.e., shortness of

breath). However, the mild level of exercise participants in the mild intensity group experienced (walking one mile per hour) is significantly slower than the average human walking speed of approximately three miles per hour (Anderson, & Pandy, 2001). Given this extremely slow speed (relative to everyday movement) it is theoretically unlikely that many somatic symptoms such as increased heart rate or shortness of breath would occur or be noticeable to any individual regardless of level of AS. As a result, it does not seem likely that interoceptive exposure is the sole mechanism for the anxiolytic effect experienced by the mild intensity group, although it may play a role in alleviating anxiety/AS more rapidly as seen in the quicker response reported by the high intensity aerobic exercise group.

Although the findings from this initial study were promising, Broman-Fulks et al., (2004), identified several methodological flaws to be improved upon for future studies. First, the researchers highlighted the possibility of a regression to the mean effect occurring as a result of selecting individuals based on extreme scores (i.e., elevated ASI scores). Since both experimental groups produced a similar effect at follow-up, these results could indicate either a statistical regression or a delayed effect of the mild intensity exercise group. Although the researchers do not believe the results are solely due to statistical regression, they recommend further study utilizing an assessment-only group as a control condition. Other limitations noted by the authors are the lack of information gathered regarding participants previous exercise history and lack of ratings of perceived effort by the participants. These information deficits may have bearing on the outcome of similar studies. As an example, although the exclusion criteria required that participants not be engaging in a regular exercise program during the course of the study, it is possible that individuals may have had extensive experience with exercise in the past.

As a result, these individuals may have different expectations and perceptions of exercise not shared by individuals who seldom or never exercise. In addition, despite the researcher's attempts to ensure equal intensities of exercise for each group, it is likely that variations in group members may have impacted the results. For example, it is possible that someone who is not physically fit, overweight, or has other health concerns, may perceive the effort required to walk at one mile per hour similarly to how an extremely fit person finds jogging at 60% of their maximal heart rate. Although the probability of this occurrence happening is unlikely, the researchers acknowledge that an evaluation of perceived exertion is warranted for future studies (Broman-Fulks et al., 2004). Furthermore, sedentary and/or overweight individuals may have been less likely to participate in the study in the first place.

In an attempt to address some of the noted concerns, Broman-Fulks and Storey (2008) conducted a subsequent study utilizing a similar research design protocol. For this study, participants were randomly assigned to either an experimental exercise condition, or a control condition. Participants in the experimental group, as in the previous study, were asked to engage in 20 minutes of moderate aerobic treadmill exercise (i.e., 60-90% of maximal heart rate) six times over a two week period. The control group was asked to only come to the research lab and fill out the required measures (i.e., ASI). As hypothesized, after six sessions of aerobic exercise, the results indicated that the experimental group exhibited significant decreases in AS, while the control group did not experience any such decrease. As a result, the authors conclude that the effect of physical activity on AS measures is not accounted for by statistical regression (Broman-Fulks, & Storey, 2008). Given the relatively small sample size (n=24) the authors note that additional studies are necessary to replicate the findings in order to provide the necessary evidence to establish exercise as a therapeutic intervention for AS. In addition, the authors

highlight the need to evaluate exercise as a stand-alone intervention compared with other standardized treatments or as an adjunct to these therapies. Due to the limited negative side effects of exercise interventions, as well as its ease of administration to almost any client, it is possible that combining treatments may in fact provide the most effective treatment for anxiety disorders and related psychopathologies (Broman-Fulks, & Storey, 2008).

The final experiment designed to evaluate the reduction of AS through exercise utilized a similar exercise intervention program and added a cognitive restructuring component as well (Smits, et al., 2008). In this study, researchers randomly assigned participants to one of two experimental conditions or a wait-list control group. Participants in the first exercise group were asked to exercise on a treadmill at a rate sufficient to achieve a heart rate of 70% HR_{max} for six sessions over a two week period. These individuals were provided a rationale for the study, including information regarding AS, avoidance, anxiety of bodily sensations, and evidence supporting the effects of interoceptive exposure and its ability to decrease AS. These participants were then asked to briefly summarize the information and intervention rational to ensure that they understood the purpose of the study and procedures. During the exercise session, the participants were asked every three minutes to provide their subjective units of distress (SUDS) of the intensity of their bodily sensations and anxiety. At this time, they would also be reminded to focus on their bodily sensations by the experimenter. This was done in order to ensure participants were fully exposed to the interoceptive sensations and that distraction did not occur. At the conclusion of each session the experimenter would help the participant identify any changes in anxiety ratings as a result of the exercise intervention. In the exercise plus cognitive restructuring group, the procedure was identical with two exceptions. First, the information given to participants in this group emphasized the efficacy of cognitive restructuring for maximizing

the effect of interoceptive exposure (i.e., through exercise). This emphasis was provided by highlighting that repeatedly experiencing a situation that an individual perceives as threatening (i.e., increased heart rate), can provide contradictory evidence that the expected catastrophic event (i.e., increased heart rate indicates I'm having or will have a heart attack) will not occur. Secondly, participants in this group were asked to provide SUDS not only for the intensity of their bodily sensations and anxiety but also to their fear about the sensations. Finally, participants in the wait-list condition completed the assessments at similar intervals to the exercise groups but did not engage in any exercise or evaluation.

The results of this study found that both exercise conditions significantly decreased AS as compared to the waitlist condition. Contrary to the hypothesized outcome, adding procedures intended to help participants reappraise the threats relating to bodily sensations did not increase efficacy of exercise treatment. Indeed, this additional intervention may have in fact been harmful to outcome as the attrition rate trend of the exercise plus cognitive restructuring group was slightly higher than the exercise only condition. Although this study did not compare a true exercise only condition with alternative forms of therapy its findings continue to support the use of exercise as a therapeutic intervention for anxiety. However, the authors of this study recognized that the lack of a placebo condition is a methodological flaw that remains to be addressed. Although this concern was not raised in the previous two studies, it is possible that expectancy may play a role in the anxiolytic effects of exercise. As a result, possible placebo effects with regards to exercise need to be confirmed or disconfirmed in order to effectively identify appropriate dose, intensity, and duration of any prescribed exercise intervention.

Limitations of Prior Research

Support for the use of exercise as a mental health intervention for anxiety and its related disorders is unambiguous and widely supported. Exercise has been found to be effective for improving mood (Byrne, & Byrne, 1993; De Moor, Boomsma, Stubbe, Willenseb, & de Geus, 2008), decreasing anxiety symptoms (Landers & Petruzzello 1994; Long & van Stavel, 1995; Petruzzello, Landers, Hatfield, Kubitz, & Salazar, 1991) and improving overall quality of life (Courneya, Friedenreich, Arthur, & Bobick, 1999). These improvements are apparent across various subtypes of anxiety disorders, exercise modalities, and population groups (Wipfli, Rethorst, & Landers, 2008). However, although few would argue the potential effectiveness of physical activity as a treatment intervention, there are four significant shortcomings within the exercise, and anxiety literature that must be overcome before physical activity can be fully utilized as an empirically validated treatment.

First, critics have noted that the use of non-clinical participants limits the generalizability of it to the clinical population (Dunn et al., 2001). Although recruiting participants from universities or the general population is convenient and provides valuable information, this type of recruitment does not guarantee that the findings will be similar for individuals from a clinical population such as those diagnosed with an anxiety disorder. As a result, future studies in this area must use either clinical participants or else non-clinical control participants with similar characteristics to the clinical population (i.e., participants with sub-threshold symptom clusters or similar prodromes such as AS).

Second, the majority of the research conducted in this area is comprised primarily of correlational or observational evidence (Wipfli et al., 2008). To gain empirical evidence, the effects of exercise on anxiety must be evaluated in randomized controlled trials (RCTs) that

result in significant effects. Reviews of the literature reveal that only a small percentage of studies examining the effects of exercise on anxiety are RCTs (Petruzzello et al., 1991). Due to the small number of RCT studies conducted, and empirical concerns associated with these concerns (e.g., lack of control groups), further studies and additional evidence is required. Additional RCT studies which replicate and expand on previous research will help to provide the necessary evidence to support the use of exercise as an empirically based treatment (EBT; Wipfli et al., 2008).

Third, associated with the limited number of randomized controlled experiments, there is limited understanding of the dose response necessary to achieve desired results. Studies have found that varying degrees of intensity and duration of exercise are able to produce anxiolytic effects (See Dunn et al., 2001, for a review). However, these studies often provide incomplete methodologies regarding intensity and duration of exercise. For example, some studies have used a range of intensities (e.g.,60-90% of maximal heart rate; Broman-Fulks et al., 2004; Cox, Thomas, Hinton, & Donahue, 2004), while others have used duration (e.g.,20 minutes of self-selected intensity; Szabo, 2003). As a result, it is difficult to compare studies or determine minimum requirements necessary to decrease anxiety (Cox et al., 2004). Furthermore, employing a wide range of intensity such as 60-90% does not provide enough information to determine the effects of exercise since there may be significant differences between individuals exercising at the lower end and those exercising at the upper end. In order to explicitly define how much physical activity of a given intensity is necessary to achieve an anxiolytic effect, researchers have argued that studies must better control for the intensity of exercise utilized (i.e., using a more specific intensity rather than a wide range; Wipfli et al., 2008). These precise intensities can then be directly compared in an experimental setting.

Fourth, a key factor limiting the use of exercise as a treatment intervention is the lack of consensus regarding the actual mechanisms of change (Petruzzello et al., 1991; Wipfli et al., 2008). Understanding the mechanisms of change for a treatment is integral to understanding why a particular intervention works (Foley et al., 2008). In order to help identify why exercise has an anxiolytic effect, studies must examine the variables which moderate this relationship (Petruzzello et al., 1991; Wipfli et al., 2008).

Potentially important moderating variables include credibility, expectancy, and perceived effort (Dunn et al., 2001; Ojanen, 1994), and few studies have examined the role these moderators may play with regards to physical activity interventions. Although the literature does not seem to have evaluated the effect of credibility on exercise interventions, placebo trials for pharmacological interventions have indicated that a participant's expectations can induce effects which mimic the effects of the active medication (Pacheco-Lopez, Engler, Niemi, & Schedlowski, 2006). Thus, it is plausible that one's expectations regarding physical activity may also impact the efficacy of any exercise intervention (see Crum, & Langer, 2007; Desharnais et al., 1993). However, given the difficulty of blinding participants to the fact that they are exercising, only one study to this author's knowledge has attempted to use a true placebo exercise group when examining the anxiolytic effects of aerobic exercise (Newman, 2009). Although some studies have attempted to control for attention effects (Daley, Mutrie, Crank, Coleman, & Saxton, 2004) future experimental studies must consider the effect of expectations on all findings by utilizing true placebo groups.

Like expectancy, some researchers posit that the perceived effort for an exercise intervention may have a moderating effect on the ability of exercise to decrease anxiety (Dunn et al., 2001). Thus, it is hypothesized that individuals who perceive they have expended a greater

amount of energy will report greater improvements in anxiety. Consequently, in order to aid in determining appropriate levels of exercise intensity for treatment, future studies examining the efficacy of exercise would find it useful to examine the perceived effort of the participant. By understanding the key components necessary for exercise interventions to produce a positive effect (e.g. if higher levels of perceived effort or higher levels of expectations are necessary for improved outcome) researchers will be able to refine and improve treatment protocols as well as help develop new interventions (Foley et al., 2008).

Present Study

The purpose of the present study was to expand the current understanding of the effects of exercise on AS and was designed to address the limitations noted in the literature. These limitations include lack of an empirical control group with regards to the non-exercise conditions, exercise interventions utilizing different measures of intensity (e.g., absolute vs. relative intensity), and a lack of analysis for mechanisms of action. As a result, four main objectives were considered. First, since AS has been correlated with the onset of future anxiety disorders, it is possible that exercise may have a preventative effect in individuals who are at risk (e.g., those high in AS). Thus, in order to better understand this relationship, recruitment criteria utilized a minimum ASI cut-off score to be included in the study.

The second objective of this study was to evaluate the possibility of a dose effect utilizing two relative intensity exercise conditions in addition to an empirical control group. As previous studies have not used an empirical control group (i.e., participants who attend the same experimental setting for an equivalent amount of time as the exercise conditions) the question of whether previous findings are a result of a regression to the mean effect remain unanswered. In addition the control group helps control for potential experimenter effects by having participants

in the current control group sit quietly for the same amount of time as the exercise groups. Furthermore, it is posited that the implementation of two narrower ranges of exercise intensity will provide increased understanding of dose response. As noted, a relative method of gauging exercise intensity is preferred to an absolute method (e.g., setting a treadmill at a specific setting for the duration of the session) as it takes into consideration the individual variability of physical capability of the participant (Cox et al., 2004). It is also anticipated that the comparison of the mild exercise condition (which is unlikely to result in a significant release of endorphins given its mild intensity), with the moderate exercise condition, will provide insight into the endorphin hypothesis as a potential mechanism of action.

The third objective of this study was to assess three potential psychological factors which may be related to the mechanism of action for change with respect to exercise and AS. These factors include credibility and expectancy as measured by the CEQ, and perceived effort, as measured by Borg's RPE. It is possible that the participants perceived credibility of the exercise intervention, their expectations regarding the intervention, or their perceived effort while exercising may impact the efficacy of exercise to reduce AS.

The fourth objective of this study was to examine the effects of fitness, as related to BMI and resting heart rate on the reduction in AS through exercise. These two indices were utilized because resting heart rate is a direct component of calculating heart rate reserve and BMI, although not as accurate as hip to waist ratio, is considered to be an adequate indicator of fitness in non-exercisers (Chinapaw, Proper, Brug, van Mechelen, & Singh, 2011). Thus, heart rate and BMI were examined to determine if physiological variables interact with psychological variables. For example, if fitness levels are poor (e.g., high BMI and resting HR), it is possible

that participants may have higher levels of perceived effort or differing levels of expectancy or credibility for the exercise conditions.

Hypotheses

The hypotheses for this study were as follows: 1) participants in the exercise conditions will experience a similar, significant decrease in AS scores, while those in the control condition will not experience a decrease in AS scores: 2) it is also hypothesized that credibility, expectancy, and perceived effort will positively predict changes in AS due to exercise:, and 3) it was of interest to evaluate if any physiological characteristics (i.e., resting heart rate and BMI) could predict ASI change.

Method

Power Analysis

Prior to conducting the experiment a power analysis using 'G*Power © (Faul, Erdfelder, Buchner, & Lang, 2009) was conducted. It was found that 14-21 participants in each group was required to obtain .8-.95 power for detecting a medium effect size (.5) when using the .05 level for statistical significance.

Participants

Three-hundred-and-ninety-two potential participants were recruited from Lakehead University and from the Thunder Bay community. Recruitment at Lakehead occurred via presentations given by the experimenter to various undergraduate classes (i.e., engineering, psychology, business) where individuals were informed of the rationale for the study and provided with a questionnaire package. Recruitment from the Thunder Bay community occurred via online advertisements on 'kijiji' ™. The online advertisement provided the rationale for the study, a link to survey monkey where they would be able to complete the screening and

demographic questionnaires and the researchers contact information for any potential questions. These questionnaires were administered to screen for AS, physical activity readiness, current involvement in an exercise program, age, recent changes in psychotropic medications, and current involvement in psychotherapy. Participants were excluded from the study based on the following criteria: (a) a score lower than 25 on the ASI (to reduce floor effects and identify an at risk group), (b) an affirmative response to any item on the PAR-Q (as a health safety screen), (c) body mass index greater than 35, (d) current involvement in an exercise program (to reduce the possibility of independent exercise masking the effects of the study), (e) any recent changes in psychotropic medications, and (f) currently receiving any form of psychotherapy (to reduce the possibility of progress in treatment or medication masking the effects of the study). Based on their responses to the questionnaires, 337 participants were excluded from participating in the study. In total 207 individuals were excluded based on the ASI criteria, 73 individuals were excluded based on their current involvement in an exercise program, and 38 individuals were excluded based on the PAR-Q criteria. After contacting potential participants who met eligibility requirements, an additional 16 individuals either declined to participate further in the study (n=5), or did not complete all phases of the study (n=11).

In total, 58 individuals (40 females and 18 males) completed all phases of the study (Table 1). At the completion of data collection all of the names were written on equal sized pieces of paper and placed into a container. A colleague with no affiliation to the study, or awareness of participants then drew a name at random for the gift card and the prize was delivered to the winner. In addition, all participants who were currently enrolled in an introductory psychology courses were credited with one bonus percentage point toward their final mark. The participants ranged in age from 18 to 51 with a mean of 22.81 years (SD = 7.04).

Of the 58 participants, the sample was predominantly Caucasian (72.4%) with the remainder being European (10.3%), Native Canadian (5.2%), African Canadian (3.4%), South Asian 1.7%), and Unspecified (6.9%). In addition, data related to the physiological characteristics is provided (Table 2).

Table 1.
Demographic characteristics by group.

Variable	No Exercise (n = 18)		Mild Exercise (n = 21)		Moderate Exercise (n =19)	
	n	*M (SD)*	*n*	*M (SD)*	*n*	*M (SD)*
Age		21.94 (4.30)		23.86 (8.73)		22.42 (7.03)
Gender						
Men	8		4		6	
Women	10		17		13	
Completers						
Yes	18		21		19	
No	5		2		4	

Table 2.
Height and Weight characteristics by group.

Variable	No Exercise (n = 18)	Mild Exercise (n = 21)	Moderate Exercise (n =19)
	M (SD)	*M (SD)*	*M (SD)*
Height (inches)	69.22 (2.73)	67.37 (2.61)	68.72 (3.01)
Weight (lbs)	170.83 (24.87)	152.37 (27.15)	164.72 (36.52)
BMI	25 (2.61)	23.53 (3.53)	24.56 (3.96)

Materials

Anxiety Sensitivity Index. Given its demonstrated validity, internal reliability (α .80-.90), and test-retest reliability, (r ranging from .71-.75), as well as its ease of administration, the ASI (Appendix A) is one of the most widely used measures of AS (Broman-Fulks, & Storey, 2008; Taylor, & Cox, 1998). For this study, the internal reliability was found to be high

(α .= .84). In addition, the ASI has been found to predict the development of panic disorder and provides some indication of the nature of someone's fears, such as fear of panic attacks, mental illness, or heart attacks (Reiss and McNally, 1985).

It has been used extensively to identify individuals likely to experience panic in stressful situations such as military personnel during basic training (Reiss, & Peterson, 1992). In addition to discriminating between clinical and non-clinical populations, the ASI is useful for measuring changes in AS in research applications (Smits et al., 2008). Two revised versions of the ASI have been developed subsequent to the original, the 36 item ASI-R (Taylor, & Cox, 1998), and the 18 item ASI-3 (Taylor et al., 2007). The original version of the ASI was utilized for this study due to the long research history that is available relative to the ASI-R and ASI-3.

The ASI is comprised of 16 items which address questions about how distressing anxiety symptoms are for an individual (e.g., It scares me when I feel shaky, or When I am nervous, I worry that I might be mentally ill). The responses utilize a 5-point rating scale ranging from 0 (very little) to 4 (very much). Along with the excellent psychometric properties, the ASI was employed in this study since it can be completed in a relatively brief amount of time (approximately 2 minutes), and can be completed by individuals aged 12 and up (Deacon, Abramowitz, Woods, & Tolin, 2003). Interpretation of the ASI is based on total score whereby a score greater than 25 is seen as an indicator for potential clinical problems and scores of 30 and above may be a possible indication of a psychological disorder such as panic disorder or post-traumatic stress disorder (Smits et al., 2008). In addition, the ASI has several subscales related to different areas of AS including, cardiovascular concerns (i.e., "It scares me when my heart beats rapidly"), physical concerns (i.e., "It scares me when I feel "shaky" (trembling)."), cognitive

concerns (i.e., When I cannot keep my mind on a task, I worry that I might be going crazy.), and social concerns, (i.e., Other people notice when I feel shaky.).

Credibility and Expectancy Questionnaire. The Credibility and Expectancy Questionnaire (CEQ; Appendix B) was used to measure the participant's credibility and expectations of the exercise intervention. The CEQ consists of two subscales defined as (a) Credibility (e.g., "At this point, how logical does the intervention offered to you seem?"), and (b) Expectancy (e.g., "How much improvement in your symptoms do you really feel will occur?"), (Smits et al., 2008). The CEQ utilizes a 9-point rating scale with anchoring points of 0 (not at all) to 9 (extremely). The CEQ also has high internal consistency ($\alpha = .84$), and good test-retest reliability (*r* ranging from .75 to .82; Devilly, & Borkovec, 2000). With respect to this study, the internal consistency for the credibility ($\alpha = .76$), and expectancy ($\alpha = .84$), were both found to be high. In order to match the content of the questions with the current study a minor modification to the CEQ was deemed appropriate. For example "reduce stress and trauma symptoms" was replaced with "impact your general mood" (G. Devilly, personal communication, Nov. 21, 2010).

Borg's Rating of Perceived Effort Scale. Perceived exertion is a subjective construct which refers to how hard you feel your body is working during physical activity. Ratings of Perceived Effort are used in a variety of settings for estimating effort and exertion and may take into account factors such as breathlessness and fatigue (Borg, 1998). In order to measure participants' perceptions of effort in this study, Borg's Rating of Perceived Effort Scale (RPE; Appendix C) was utilized. The RPE uses a 15 point scale ranging from 6-20, where a score of 6 indicates "no exertion at all", and a score of 20 indicates "maximal exertion" (Borg, 1998). The anchor points of '6' and '20' were utilized to reflect the typical heart rate of a healthy adult when

divided by 10. For example, a resting heart rate of an adult is approximately 60 beats per minute or a '6' on the scale (no exertion). While a score of 10 is thought to reflect a heart rate of approximately 100 bpm. Ratings between 12 and 14 on the RPE suggest that a moderate intensity of physical activity has been reached, while a score lower than 10 indicates a very light intensity has been reached. Subjective scales of perceived exertion have been used in a variety of settings for exercise prescription and fitness monitoring although it is commonly used in conjunction with other measures of exertion such as heart rate reserve or oxygen consumption (Heyward & Gibson, 2014). In particular, Borg's RPE is one of the most widely used subjective measure of perceived effort and has been found to be a valid and reliable tool for this purpose (Birk & Birk, 1987; Robertson, 2004). However, for the purpose of this study, the RPE scale was utilized to determine if perception of effort would impact the effects of exercise on anxiety rather than to determine the level of exercise intensity for the participants.

Physical Activity Readiness Questionnaire. The Physical Activity Readiness Questionnaire (PAR-Q; Appendix D) is a 7-item measure which can be used for individuals between the ages of 15 and 69. The items on the PAR-Q pertain to any health concerns which may preclude or prevent them from engaging in physical activity prior to consulting with a clinician (e.g., "Do you feel pain in your chest when you do physical activity?"). Recommendations on the PAR-Q suggest that if individuals answer affirmatively to any question on the scale, they should consult with a physician before engaging in any new physical activities or becoming much more physically active.

Demographic Questionnaire. A questionnaire (Appendix F) was utilized to collect basic demographic data, including age, height, and weight. All information was self-report and BMI was determined from this information using appropriate gender BMI tables.

Heart Rate Monitor. An e-pulse heart monitor worn by all participants measured heart rate during each session. The monitor enabled adjustments in the speed of the treadmill to be made in order to ensure participants in the mild and moderate exercise conditions were exercising at their target heart rate for the duration of their sessions. In order to ensure continuity across all groups, participants in the quiet rest group also wore a heart rate monitor.

Treadmill. A Trackmaster (model 500AC/A) treadmill was used for participants in both the mild and moderate exercise groups. The settings on the treadmill were selected by the experimenter prior to beginning the exercise session. To ensure that the target heart rate was achieved and maintained, the heart rate monitor was noted and speed of the treadmill adjusted every two minutes as in the Broman-Fulks and Storey experiment (2008).

Procedure

Participants were randomly assigned to one of the three treatment conditions prior to their first appointment. Each exercise intervention included a total of six 20-minute sessions completed over a two week period, with no more than four sessions being completed in a single week. All participants were asked to measure and provide resting heart rate information and were instructed to dress comfortably for exercising. Target heart rate reserve range for each treatment condition was calculated for all participants using the Karvonen formula (Percent Heart Rate Reserve = [((max HR 220-age)-resting HR) x % Intensity + resting HR]. As noted, heart rate reserve has been found to be strongly related to oxygen uptake reserve. This method was utilized rather than a percentage of maximum heart rate as it has been found to be more indicative of exercise intensity with calculations based on percentage of maximum heart rate tending to be lower and requiring less effort than calculations based on reserve VO_2 or heart rate reserve.

Participants in the exercise conditions exercised at their prescribed intensity for the duration of the session, with a two minute warm up and cool down, while those in the control group sat quietly for the 20 minute duration. All participants wore a heart rate monitor for the duration of their experimental sessions with an output display attached to the treadmill where it was easily observed by the experimenter. In order to ensure the target heart rate range was maintained, heart rate was monitored throughout the exercise session and treadmill speed adjusted accordingly. At the beginning of the first session participants were reminded of the rationale for the study (to investigate the effect of exercise on general mood states), and asked to provide their resting heart rate (measured by the participants immediately upon waking in the morning by taking their pulse for 60 seconds). Procedures were also reviewed and participants were then asked to complete the CEQ and ASI (Table 3).

At the completion of the session participants were then asked to circle the number on the RPE that believed best reflected their perceived level of exertion. Participants were then reminded about their next scheduled session and were also sent reminder e-mails if requested. Participants were asked to complete all measures again at the fourth session and the sixth session. At one week follow-up participants were asked to complete the ASI for a final time. After the completion of this questionnaire, participants were thanked for their participation and informed that they would receive an e-mail detailing the debriefing and results of the study upon completion of the experiment (Appendix G). All participants were reminded that they would be entered into the draw at the completion of the data collection. In addition, participants taking introductory psychology courses were given a receipt of their participation for their records to ensure they received their eligible course credit. The appropriate documentation was then provided to the psychology department to ensure that bonus points were awarded appropriately.

Participants were debriefed at the conclusion of the study and were provided with contact information should they wish to discuss any of the findings further or to address any potential concerns they may have had.

Table 3
Measurement points for each questionnaire

Measure	Screening	Exercise Session 1	Exercise Session 4	Exercise Session 6	Follow Up
ASI	X	X	X	X	X
RPE		X	X	X	
EXPECTANCY		X	X	X	
CREDIBILITY		X	X	X	

"Change-Post" scores are calculated as the difference between Exercise Session 1 and Exercise Session 6.
"Change-Follow Up" scores are calculated as the difference between Exercise Session 1 and Follow-Up

Experimental Conditions

Experimental condition 1 - Moderate intensity exercise. Participants in the moderate intensity condition exercised at 65% to 75% of their heart rate reserve during each of their scheduled exercise sessions.

Experimental condition 2 - Mild intensity exercise. Participants in the mild intensity condition exercised at 30% to 40% of their maximum heart rate during each of their scheduled exercise sessions.

Experimental condition 3 - Resting control. Participants in the resting control condition did not exercise but were instead asked to sit quietly for the duration of the session. They were not provided with any activities (e.g., listening to music or reading). They also filled out identical questionnaires in a similar time structure as the two exercise conditions at the first, fourth, and sixth exercise sessions, as well as at one week follow up.

Ethical Considerations

Prior to data collection, the present study was reviewed and approved by the Lakehead University Research Ethics Board. The voluntary nature of participation was explained in the informed consent form. Participants were also reminded at their first session that their participation was voluntary and that they could withdraw at any time without penalty. Participants were also informed that the purpose of the study was to examine general mood states. This was done in an attempt to reduce attention to anxiety symptoms. It is possible that had the participants been sensitized to the focus of the study (anxiety symptoms), it may have elevated ASI scores. Thus, the title of the study was omitted on all documentation, as well as the removal of the title on the CEQ. Participants were informed prior to giving consent that the study involved either six sessions of exercising at a mild or moderate pace on a treadmill for 20 minutes, or six quiet rest sessions. Participants received instructions on how to operate, safely stop, and get off of the treadmill and were informed that they could discontinue participating if they desired to do so in the middle of an exercise session.

Analyses

In order to determine if there were differences between groups and over time, ANOVAs were utilized. These analyses assessed differences in ASI scores, CEQ scores, as well as RPE scores. Furthermore, ASI change scores were analyzed. Correlation analysis was utilized to examine regression to the mean effects by analyzing the relationship of ASI scores at screener and Time 1 to ASI scores at Post Treatment and Follow-Up. Finally, regression analysis was utilized to explore the relationship between ASI change scores and the covariates.

Results

Data Screening

Data gathered from the 58 participants was entered into SPSS 21. Frequency distributions were calculated for each variable and checked for missing values. All data were present for all participants. To determine the presence of univariate outliers, z scores were calculated for all variables as well as change scores. According to Tabachnik and Fidell (2007), a data point is considered to be an outlier if its standardized z score is in excess of ± 3.29 (p. 73). Using this criteria two participants were found to each have an individual outlier: one was noted at ASI time 2 and a second participant had an outlier noted for ASI Follow-Up. The impact of these outliers was reduced by replacing them with a raw score that was one unit larger than the next highest score in the distribution (Tabachnik and Fidell, 2007). In addition three participants (two from the mild exercise group and one from the moderate exercise group) were found to have significant outliers with respect to age and were thus excluded from the study to ensure a homogenous demographic sample. Thus, the no exercise group maintained 18 participants, while the mild exercise group decreased to 19 participants and the moderate exercise group decreased to 18 participants. Additionally, to check for multivariate outliers, Mahalanobis D^2 (a multidimensional version of a z-score) were calculated based on demographic variables (including Age, Gender, BMI, Ethnicity, and Amount of Exercise), psychological variables at session 1 (including credibility, expectancy, and perceived effort), and ASI scores at session 1. A set of data points is considered to be a multivariate outlier if the probability associated with its D^2 is less than 0.001. Using this criterion, no multivariate outliers were observed.

An identical process was utilized to screen for univariate and multivariate outliers for each specific group. With respect to the No Exercise condition, z-scores indicated univariate

outliers for three scores. One outlier was noted at ASI screener, one at ASI time 2, and one at ASI-follow up. For the Mild Exercise condition, no univariate outliers were noted for any variables. Finally, z-scores for the Moderate Exercise condition indicated a single univariate outlier at ASI screener and a single univariate outlier for ratings of perceived exertion at time 3. As before, the impact of these outliers was reduced by replacing them with a raw score that was one unit larger than the next highest score in the distribution (Tabachnik and Fidell, 2007), and subsequent analysis for univariate outliers indicated no further outliers. For Multivariate outliers, Mahalanobis D^2, was calculated for each group for the demographic variables, psychological variables, and ASI total scores at time 1. Using the .001 criteria, no multivariate outliers were observed[1].

After correcting for outliers, the data was analyzed for assumptions of homoscedasticity. Except for perceived effort, all Levene tests for homogeneity of variance were not significant thus indicating that the data had equality of variance. With respect to perceived effort, because the no exercise group did not vary in their response (all participants indicated no perceived effort), there was no variance. However, when the no exercise group was excluded, the Levene test for homogeneity of variance was not significant for the two exercise conditions thus indicating that there was homogeneity of variance. Furthermore, because the no exercise group did not experience any significant change in ASI scores at post or follow-up, this group was not utilized in the analysis of the predictor variables. Thus the lack of homogeneity of variance for perceived effort for the no exercise group did not impact analysis of this variable for subsequent analyses.

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[1] Examination of the data with the outliers included indicated that the removed or adjusted variables did not alter the overall results of the analysis, but did affect assumptions and were thus removed from the study (as per Tabachnik and Fidell, 2007) in a conservative approach.

Computing Variables

The means and standard deviation scores for the ASI are provided in Table 4 for the five sessions separately for all three groups. Two change scores (ASI Change Post and ASI Change Follow-Up) were computed for ASI. ASI Change Post was calculated by subtracting the participants scores on the ASI at time 1 (their initial exercise session) from their score on the ASI at time 4 (at the completion of their exercise sessions). While ASI Change Follow-Up was calculated by subtracting their score at ASI time 1, from their score at ASI follow-up. Change scores between time 1 and time 3 were also calculated for credibility, expectancy, and perceived effort.

Table 4.
Descriptive statistics for AS total scores

	Anxiety Sensitivity Index Total Score		
	No Exercise	Mild Exercise	Moderate Exercise
Session	M (SD)	M (SD)	M (SD)
Screener	27.50 (2.75)	32.00 (6.49)	27.00 (2.61)
Session 1	26.67 (5.08)	31.21 (6.71)	27.06 (3.30)
Session 4	26.72 (3.06)	29.68 (6.15)	24.94 (4.37)
Session 6	27.78 (5.73)	28.84 (6.53)	24.94 (4.58)
Follow-Up	26.11 (3.28)	28.00 (5.92)	24.67 (4.69)

Anxiety Sensitivity Analysis

Analysis of variance indicates a significant difference over time F (4, 49) = 8.99, p <. 001, as well as between groups F(2, 52) = 4.46, p = .016. Results also indicate a significant Time by Group interaction, F(8, 100) = 2.19, p =.03 (Figure 1). ASI scores were significantly different at screener, F (2, 52) = 7.34, p = .002, session 1, F(2, 52) = 4.29, p = .019, and Session 4, F (2, 52) = 5.37, p = .008. At the screener session, participants in the mild exercise group reported higher ASI scores than the no exercise,

$t(35) = -2.72$, $p = .01$, $d = .66$ and moderate exercise groups, $t(35) = -3.04$, $p = .004$, $d = .84$. At session 1, participants in the mild exercise group continued to report higher ASI scores than the no exercise group, $t(35) = -2.31$, $p = .027$, $d = .76$, and the moderate exercise group, $t(35) = -2.37$, $p = .024$, $d = .78$. However, at measurement 2, the mild exercise group indicated significantly higher scores than the moderate exercise group, $t(35) = -2.69$, $p = .01$, $d = .89$, but there was no longer a significant difference between the mild, and no exercise groups. In addition, the ANOVA indicated a significant group by session interaction $F(8, 100) = 2.33$, $p = .03$.

To evaluate if changes occurred in response to the exercise interventions, a series of ANOVAs were conducted to analyze the differences between groups on ASI change scores at post, $F(2, 52) = 4.30$, $p = .019$ (difference between Exercise Session 1 and Exercise Session 6), as well as at follow-up, $F(2, 52) = 3.89$, $p = .027$, (difference between Exercise Session 1 and Follow-Up; Figure 2). With regards to Post Change, t-tests indicate that participants in the Mild Exercise group, $t(35) = -2.55$, $p = .015$, $d = .84$, and the Moderate Exercise group, $t(34) = -2.32$, $p = .027$, $d = .77$, reported greater reductions in AS than the No Exercise group. However, there was no significant difference between the Mild and Moderate groups, $t(34) = .30$, $p =$ n.s..

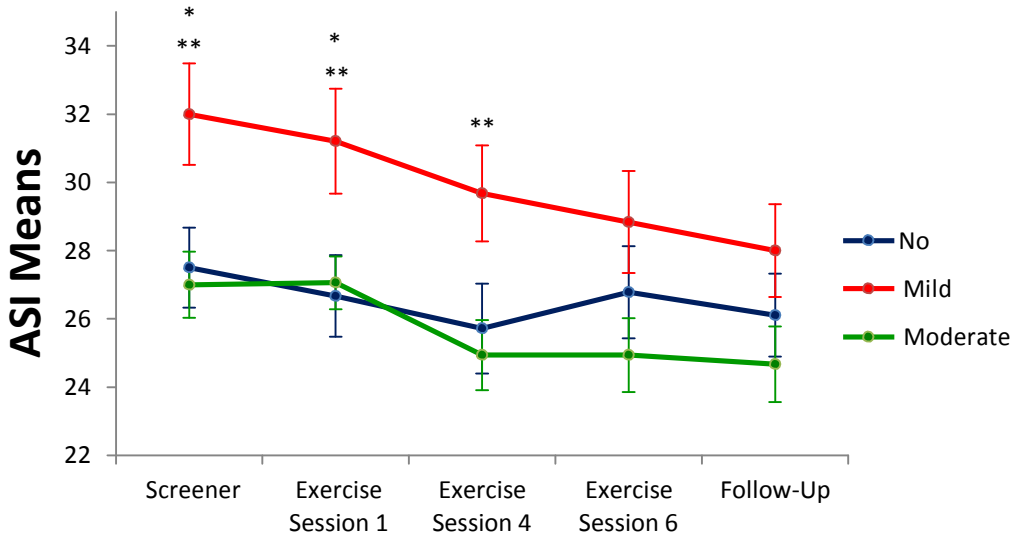

Figure 1. ASI Total scores (Means) at each assessment session by exercise group. Error bars represent standard error.
* No Exercise vs. Mild Exercise, p < .05, ** Mild Exercise vs. Moderate Exercise, p < .05.

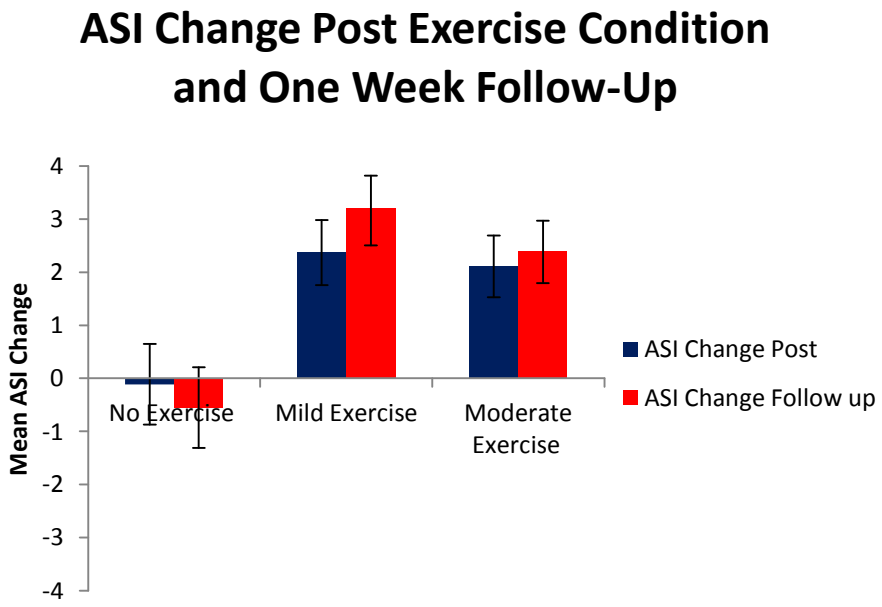

Figure 2: Mean ASI Total change post and follow-up. Error bars represent standard errors.
* ASI Change Post: No vs. Mild, $p < .05$, No vs. Moderate: $p < .05$
** ASI Change Follow-Up: No vs. Mild, $p < .05$, No vs. Moderate: $p < .05$

Similarly, with regards to Follow-Up change, both the Mild exercise group $t(35) = -2.57$, $p = .015$, $d = 1.22$, and the Moderate Exercise Group $t(34) = -1.9$, $p = .006$, $d = 1.03$ reported greater reductions of AS than the No Exercise Group. There were no significant ASI change score differences between the Mild and Moderate Exercise Groups at follow up, $F(35) = .88$, $p =$ n.s..

Covariate Analysis

Univariate ANOVAs were conducted separately for credibility, expectancy, and perceived effort (See Table 5 for Means and SD). For credibility, it was found that there was no significant change in scores across sessions, $F(2, 51) = .95$, p = n.s., nor was there a significant group by session, $F(4, 104) = .20$, $p =$ n.s. interaction. Conversely, there was a significant difference in credibility between the three groups, $F(2, 52) = 18.70$, p = < .001. Collapsing across sessions, it was found that the no exercise group reported lower credibility then the mild group, $t(35) = -4.97$, $p = < .001$, $d = 1.63$, and the moderate group, $t(34) = -5.41$, $p < .001$, $d = 1.80$. However, there were no significant differences between the mild, and moderate groups $t(35) = .77$, $p =$ n.s. (Figure 3).

With regards to expectancy, it was also found that there was no significant change over sessions, $F(2, 51) = .052$, $p =$ n.s., nor was there a significant group by session $F(4, 104) = 1.76$, $p=$ n.s., interaction. However, there was a significant group effect $F(2, 52) = 28.09$, $p < .001$. Collapsing across sessions, it was found that participants in the no exercise group reported lower expectancy scores than the mild group, $t(35) = -6.09$, $p = < .001$, $d = 2.00$. Participants in the no exercise group also reported lower expectancy scores than the moderate exercise group, $t(34) = -6.83$, $p < .001$, $d = .2.27$. On the

other hand, there were no significant differences between the participants in the mild and moderate groups $t(35) = .95$, p = n.s. (Figure 4).

As the no-exercise group did not change with respect to perceived effort over time, ANOVA results for perceived effort only included the two exercise conditions and indicated that there was no significant change over session, $F(2, 34) = .14$, p = n.s, nor a significant group by session $F(2, 70) = 1.56$, p = n.s., interaction. However, there was a significant group effect, $F(1, 35) = 54.63$, $p < .001$. Collapsing across sessions found that participants in the mild exercise group reported significantly lower perceived effort than those in the moderate exercise group, $t(35) = -7.39$, $p < .001$, $d = .2.25$. Furthermore, a one sample t-test comparing the mild and moderate exercise groups to a constant score of six (the lowest score possible, which all members of the no exercise group endorsed), the mild exercise group was significantly different, $t(18) = 9.69$, $p < .001$, as was the moderate exercise group, $t(17) = 20.49$, $p < .001$ (Figure 5).

Table 5.
Means and Standard Deviations of Credibility, Expectancy, and Perceived Effort Scores

	No Exercise	Mild Exercise	Moderate Exercise
	M(SD)	M(SD)	M(SD)
Credibility			
Session 1	9.39(5.66)	18.05(5.02)	19.56(5.32)
Session 3	9.17(5.65)	17.84(5.01)	19.28(5.68)
Session 6	9.11(5.88)	17.79(4.97)	18.78(6.05)
Mean of Sessions	9.22(5.66)	17.89(4.94)	19.20(5.41)
Expectancy			
Session 1	6.67(4.72)	15.92(4.18)	16.97(4.94)
Session 3	6.31(4.61)	15.39(4.92)	17.75(4.74)
Session 6	6.51(4.69)	15.63(4.74)	17.44(5.50)
Mean of Sessions	6.49(4.62)	15.65(4.52)	17.39(4.95)
Perceived Effort			
Session 1	6.00*	10.11(1.79)	14.00(1.72)
Session 3	6.00*	9.95(1.96)	14.06(2.24)
Session 6	6.00*	9.68(1.83)	14.22(1.40)
Mean of Sessions	6.00*	9.91(1.76)	14.09(1.67)

* All participants chose the minimum value for Perceived Effort "6.0"

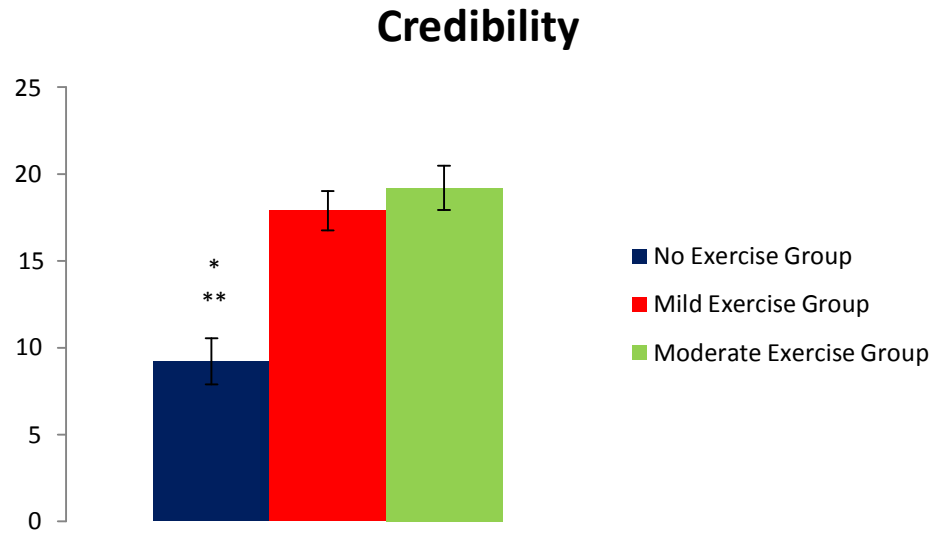

Figure 3. Average credibility scores for each group. Error bars represent standard errors.
* No Exercise vs. Mild Exercise, $p < .05$, ** No Exercise vs. Moderate Exercise, $p < .05$.

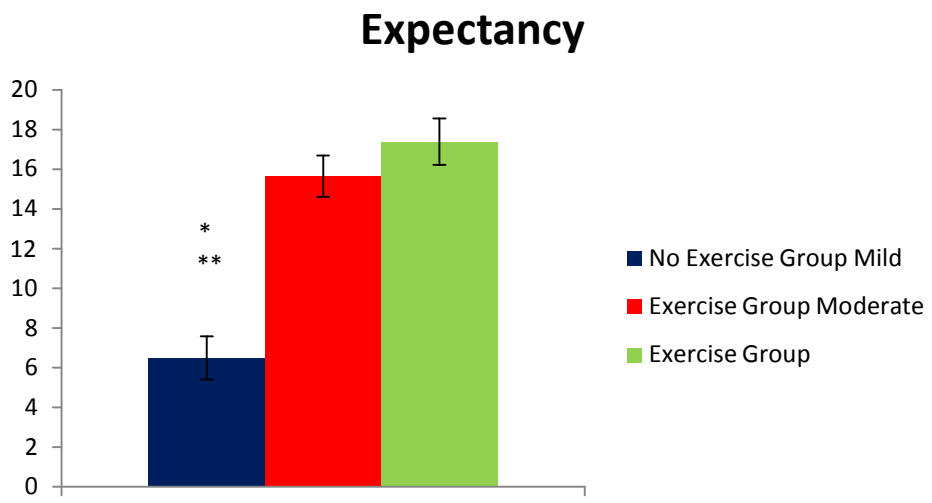

Figure 4. Average Expectancy scores (Means) for each group. Error bars represent standard errors.
* No Exercise vs. Mild Exercise, p < .05, ** No Exercise vs. Moderate Exercise, p < .05.

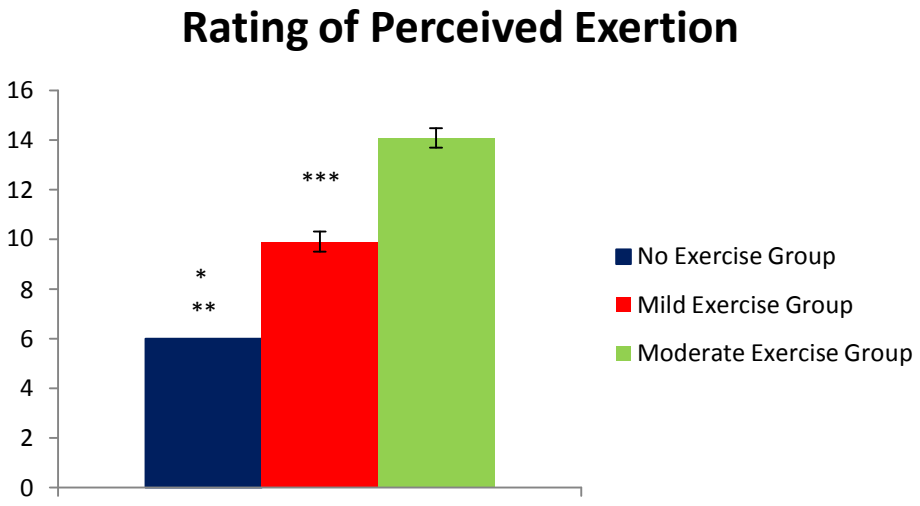

Figure 5. Average Ratings of Perceived Exertion scores (Means) for each group. Error bars represent standard errors. (Note: 6 is the minimum possible score on this measure, with all participants in the no exercise group indicating 6 or no perceived effort)
* No Exercise vs. Mild Exercise, $p < .05$, ** No Exercise vs. Moderate Exercise, $p < .05$,
*** Mild Exercise vs. Moderate Exercise, $p < .05$

Correlations

In order to assess for potential regression to the mean effects, correlation analysis was examined to see if there was a significant relationship between starting ASI scores and ASI change scores. Results from this analysis indicate that ASI Screener Total Scores were significantly correlated with ASI Time 1 Scores and ASI Change at Post was significantly correlated with ASI Change at Follow-Up. However, neither ASI Screener nor ASI Time 1 were significantly correlated with ASI Change Post or ASI Change Follow-Up (Table 6).

Correlation analysis for the covariates utilized only the two exercise groups as the no exercise group did not have variability with respect to perceived effort. This analysis indicated that credibility was significantly correlated with expectancy, $r(35) = .82$, $p < .001$. However, perceived effort was not significantly correlated with credibility, $r(35) = .03$, p = n.s., or expectancy, $r(35) = .13$, p = n.s..

Table 6
Correlations Among ASI Total and Change Scores

	ASI Screener Total	ASI Time 1 Total	ASI Change Post	ASI Change Follow-Up
ASI Screener Total	___			
ASI Time 1 Total	.842**	___		
ASI Change Post	-.099	.120	___	
ASI Change Follow-Up	.181	.291	.596**	___

** Correlation is significant at the 0.01 level

Regression Analysis of only Exercisers

To explore the relationship between exercise and change in AS with respect to potential moderator effects from expectancy, credibility, and perceived effort, regression analysis was used. Multiple regression analysis can be used to test whether or not there are significant

associations amongst variables and allows for the controlling of potential confounds (Hoyt et al., 2006). The first analysis focused on whether the ASI change of the exercisers was predicted by the covariates. As the two exercise groups did not differ significantly with respect to change, for this analysis the two groups were combined and the no exercise group (which did not experience any exercise intervention or change in ASI) was excluded. Thus, a series of regressions examined ASI change at Post and at Follow up for both the psychological covariates (credibility, expectancy, and perceived effort), as well as the physiological covariates (BMI, resting HR, and age).

The regression equation including credibility, expectancy, and perceived effort did not significantly predict ASI change at post $R = .39$, $F\ (3, 33) = 1.98$, p = n.s.. Nor did credibility, expectancy, or perceived effort significantly predict change at Follow-Up, $R = .45$, $F\ (3, 33) = 2.84$, p = n.s.. Additionally, a regression equation with Age, Gender, BMI, and Resting HR, also failed to significantly predict ASI change at post, $R = .44$, $F\ (4, 32) = 1.88$, p = n.s., and at follow up, $R = .31$, $F\ (4, 32) = .83$, p = n.s..

Discussion

Anxiety disorders are considered to be the most common of all mental illnesses, and it is predicted that anxiety disorders affect 12% of Canadians annually (Stewart, Lips Lakaski, & Upshall, 1999). These high rates of anxiety can result in high costs associated with health care, work productivity, and quality of life. Unfortunately, despite a variety of psychological and pharmacological interventions available, many individuals either have difficulties accessing these resources (e.g., due to financial, or time limitations), or find them ineffective (Salmon, 2001). Given these concerns, alternative forms of treatment that are more accessible are being examined in the literature. Specifically, there is a plethora of literature which supports the

anxiolytic effects of exercise (Broocks et al., 1998; Petruzzello et al., 1991; Yeung, 1996), although our current understanding of dose response and the mechanism(s) of action is limited. Despite the many physiological and psychological hypotheses proposed to explain the anxiolytic effects of exercise, conclusive empirical evidence is lacking and does not provide a clear consensus in the literature. Furthermore, although psychological factors such as credibility and expectancy have been shown to affect final outcome for many forms of treatment (Smeets, Beelen, Goossens, Schouten, & Knottnerus, 2008), they have not yet been examined with respect to anxiety reduction through exercise in order to examine potential moderating effects.

In an attempt to better understand the relationship between exercise and anxiety, new research has begun exploring the effects of exercise on AS. Anxiety sensitivity refers to the degree to which a person fears the behaviours, or symptoms associated with anxiety. As a person with high anxiety sensitivity will be more likely to notice physiological symptoms and interpret them as harmful (Reiss, 1991), AS is seen as a risk factor for the development of future anxiety diagnoses such as generalized anxiety disorder and panic disorder (Schmidt et al., 2006). This misperception of symptoms is explained by the attribution theory of anxiety which proposes that unexpected symptoms may be interpreted as harmful or dangerous (i.e., 'I'm having a heart attack' or 'I'm going crazy'), and are thus feared (Austin, & Richards, 2001). Consequently, it is believed that a reduction in AS may prevent the onset of future disorders (Schmidt et al., 2006). To date, only three studies have evaluated the efficacy of AS reduction through exercise. These studies have provided some support for the assertion that exercise interventions (i.e., treadmill walking/running), are able to decrease AS. However, these studies have not addressed the mechanisms involved in this relationship, nor have they utilized adequate control groups, or effectively assessed how much or little exercise is required to achieve these effects.

The present study sought to add to the existing literature on the anxiolytic effects of exercise and replicate findings of specific studies (Broman-Fulks, Berman, Rabian, & Webster, 2004; Broman-Fulks, & Storey, 2008; Smits et al., 2008). Specifically, this study made an attempt to improve upon existing methodologies to provide a clearer picture of how exercise may be able to reduce AS in participants. Furthermore, as few studies have attempted to study the role of credibility, expectations (Newman, 2009), and perceived effort (Dunn et al., 2001), this study was also designed to provide insight into the role that these variables may play in moderating or mediating the relationship between exercise and AS. These goals were achieved by utilizing specific mild and moderate exercise intensities, as well as an empirical control group. In addition, in an attempt to identify a potential mechanism of action, measures of credibility, expectancy, and perceived effort were utilized, and physical fitness characteristics such as BMI, and resting HR were considered.

ASI Change Due to Exercise

The primary finding from this study was that scores of AS were significantly reduced following an exercise intervention compared to a no-exercise control group. These findings support the initial hypothesis that a two week, six session, exercise intervention will result in a reduction in AS compared with no intervention. Furthermore, the decrease in AS for both the mild and moderate exercise groups was maintained at one week follow-up. These findings replicated those found in previous studies (Broman-Fulks et al., 2004, Broman-Fulks, & Storey, 2008, Smits et al., 2008) which also found that AS decreased as a result of a six session over two week exercise intervention. However, previous studies have not controlled for a regression to the mean effect nor have they adequately controlled for experimenter effects. Of the three previous studies to examine exercise and AS, one study did not utilize any control group (Broman-Fulks

et al., 2004), and two studies had 'control' participants fill out the same questionnaires as the exercising participants although the control participants were not required to remain in the lab or have the same degree of interaction with the experimenter for the same amount of time (Broman-Fulks, & Storey, 2008; Smits et al., al., 2008). The comparison of any intervention to an empirical control group is an important step in the process of defining empirically supported treatments (Chambless, & Hollon, 1998). By comparing the intervention to an empirical control, placebo effects and experimenter effects are ruled out as potential mechanisms of action.

For the current study, in order to control for experimenter effects as well as initial high and/or low values regressing to the mean over repeated tests, a control group was utilized in which participants matched the exercise conditions with respect to experimenter contact and time spent in the exercise setting. Thus, participants in the control group were asked to come to the lab for six sessions and wear a heart rate monitor for the duration of the session. During this time heart rate was monitored by the experimenter in an identical manner to the exercising groups. As this control condition did not experience a reduction in ASI despite having the same interactions with the experimenter and time spent in the lab, the possibility of an experimenter effect causing the reduction in ASI is ruled out for this study.

Due to the randomization of participants to the study, an initial significant difference in ASI scores was noted, with the mild exercise group reporting significantly higher ASI scores than the moderate and no exercise groups. However, a correlation analysis of initial scores at screener and Time 1, were not found to predict ASI change scores. The correlations indicate that while ASI Screener and Time 1 are significantly correlated, these scores are not correlated with either change score. This result, in addition to the finding that only participants in the exercise conditions experienced a change in ASI scores, indicates that the change that occurred for the

exercisers is a true exercise effect rather than a regression to the mean effect. If a regression to the mean effect was in fact responsible for the differences in ASI scores, it would be expected that higher scores on ASI at Screener or Time 1, would be correlated with the change scores for post and follow-up.

Dose Effect

With respect to a dose effect, the analysis of ASI change scores indicated that both the mild and moderate exercise groups experienced a significant decrease in AS when compared to the no exercise group. Furthermore, there was no significant difference in the amount of change between the participants who exercised at 30%-40% of their heart rate reserve and those who exercised at 65%-75% of their heart rate reserve. This lack of difference between the two exercise conditions suggests that exercising at a mild level of intensity may be as effective as exercising at a higher intensity. While other studies have compared relative and absolute intensities or utilized exercise interventions with a wide range (e.g., 60-90%), this study used a much narrower range of intensities (e.g., 30%-40%, and 65% to 75%). The concern with allowing a wide range of exercise intensity such as 60%-90% is that the results of participants who exercise at the lower end of the range may be very different from those who exercise at the higher end of the range, yet all would be included in the same group for analysis. This is problematic for the question of dose response as it cannot determine whether 60% of exercise intensity is necessary for change or 90%. In addition, exercise studies using an absolute intensity such as having participants walk at a speed of four kilometres per hour, are problematic due to the potential for vastly different exercise experiences. For example, people with shorter legs or greater body weight may experience four kilometers per hour as very strenuous while someone

with longer legs or lower body weight may experience four kilometers per hour as extremely mild.

The finding that both exercise conditions were equivalent in terms of ASI change is relevant to the discussion of exercise prescription. Some of the most common reasons for not exercising include lack of time, energy, or perceived ability, with many individuals believing that benefits are only experienced with prolonged, high intensity exercise. However, the duration and intensity of the mild exercise group for this study was brief (20 minutes), and of a mild intensity (30%-40% HRR). Thus, at risk individuals (i.e., high in AS) for developing an anxiety disorder, may be able to prevent the onset of an anxiety disorder with very little time or effort. As with other studies which have examined AS longitudinally and found that early interventions have a significant impact over time (Schmidt et al., 2000), future studies could further examine the relationship of mild exercise and AS to determine efficacy over time as well as secondary considerations such as treatment adherence.

In addition, the similarity in reduction in AS between the two exercise groups also constrains the endorphin hypothesis as the primary mechanism of action. As noted, endorphins are thought to be released during moderate to high levels of physical activity (Donevan, & Andrew, 1987), with moderate levels of exertion being indicated by scores of 12-14 on the RPE (Borg, 1998). However, the mean score for RPE of the mild group was 9.91 while the mean RPE score for the moderate exercise group was 14.09. Thus while the participants in the moderate group may have experienced an increase in Beta Endorphin availability, it is improbable that participants in the mild group experienced a level of intensity high enough to result in a significant change in Beta Endorphin levels. Thus, any change in AS scores are unlikely a direct result of an increase in endorphin levels.

It is also important to note that there was no significant difference between ASI change scores at post, and ASI change scores at one week follow-up. Participants in this study were screened out if they engaged in regular physical activity more than once per week. Furthermore, participants were instructed to not change their exercise habits during the one week follow-up. Given that increases in endorphins due to exercise are temporary (Babyak et al., 2000), it is likely that any improvement resulting from an increase in endorphins would have been attenuated during the one week post exercise phase where the participants did not engage in regular physical activity. However, future studies may directly measure Beta-Endorphin levels before, during, and after each exercise session and one week follow-up to directly assess the impact of endorphin levels.

Prospective Explanation of Exercise Effects

The utilization of a comparable control group as well as narrowly-defined exercise intensities provides important support for the assertion that exercise is able to reduce AS in an at risk population. This finding is significant as research has indicated that AS, (the fear of arousal symptoms), is related to an increased risk of anxiety disorders (Schmidt et al., 2007). Thus exercise may act as a preventative measure for the onset of future anxiety disorders. Given the lack of difference between the changes in ASI for the two exercise conditions, it seems likely that despite differences in exercising, the participants from both conditions are having a similar cognitive experience.

A prospective explanation for the ability of exercise to reduce scores of AS for both the mild and moderate exercise groups may be related to the level of attention directed towards internal signals by the participants. In a study evaluating the effects of attention, researchers found that when participants' attention was focused internally (i.e., on breathing), they reported

greater fatigue and experienced lower performance on a jogging task than participants whose attention was focused externally (i.e., on a cognitive task: Pennebaker, James, & Lightner, 1980). Similarly, exposure therapy for anxiety disorders such as Panic Disorder (which is significantly related to AS), consists of repeated exposures to a feared stimulus without avoidance, distraction or safety behaviours until the anxiety subsides. This may include interoceptive exposure to physiological sensations associated with Panic (i.e., rapid heart rate, increased respiration, etc.). However, in order for the exposure to be effective, individuals must focus on their feared stimulus as well as on their anxious symptoms in order to learn that a) the feared stimuli is not harmful, and b) their anxiety will go away on its own over time. As fear of physiological sensations is a significant part of AS, it is possible that exercising is operating as an exposure to feared somatic sensations and the conditions associated with the experimental design served to increase internal attention toward physiological sensations. Thus, although the mild group exercised at a very low pace (akin to walking) it is possible that by being in the laboratory setting, as well as wearing a heart rate monitor, enhanced the level of attention they paid to their physiological sensations. This attention is potentially much greater than the attention that would normally be paid while simply walking outside where there are likely many distractions (i.e., other people, scenery, attention on destination, etc.). As a result, it is possible that participants in both the mild and moderate groups experienced an exposure effect due to increased attention towards internal sensations. Thus, as participants exercised, they a) experienced physiological symptoms (i.e., increased heart rate and respiration), b) paid attention to those symptoms, and c) interpreted those symptoms as less threatening or distressing. Essentially, the exposure to the feared stimuli (i.e., heart rate) without a negative consequence (i.e., heart attack or fainting) may have reflected habituation of psychological concerns. Consequently, due to the exercise

interventions, participants have potentially altered their negative misattributions about physiological symptoms (i.e., rapid heart rates are dangerous) to more benign interpretations (i.e., a rapid heart rate is uncomfortable but harmless).

Moderators

A moderator, as defined by Baron and Kenney is a "variable that affects the direction and/or strength of the relation between an independent or predictor variable and a dependent or criterion variable." (p. 1174, 1986). While a variable is considered to be a mediator if, (a) variation in the independent variable (in this case exercise intensity), accounts for variations in the mediator, (b) this variation in the mediator now accounts for variations in the dependent variable (in this case AS), and finally (c) the significance of the relationship between the independent and dependent variables becomes non-significant when the relationship between the independent variable and mediator variable and the mediator variable and dependent variable are controlled (Baron, & Kenney, 1986).

The majority of the research examining factors such as credibility, expectancy, and perceived effort with respect to exercise has focused on treatment adherence rather than on outcome. These findings posit that if participants have greater expectations and view the intervention as more credible, they will be more inclined to continue participating or put forth more effort during treatment. The present study controlled for participant participation (by limiting the amount of exercise), and effort (by controlling exercise intensity) to look at whether these factors moderate the effects of exercise independent of their effects on compliance to treatment or effort.

In order to evaluate these factors the two exercise conditions were combined (as they were not statistically different from each other with respect to change), and the no exercise group

was excluded, (as it did not experience any statistically significant change). For this analysis the three covariates were used to predict change in AS. The purpose of this analysis was to examine, within the range of change in AS, whether credibility, expectancy, and perceived effort moderated the prediction. The results of this analysis were not significant indicating that none of the covariates significantly predicted the change in AS scores. However, it is likely that there was limited variability to detect a difference in this group as the combination of the two exercise conditions resulted in a very homogenous sample. Essentially, since the two exercise groups did not differ significantly with respect to credibility, expectancy, or total change, the analysis was unable to indicate any significant differences. Although perceived effort was found to be significantly different between the two exercise groups it also did not predict AS change at post or follow up. Nevertheless, if as suggested, exposure is a primary cause of a reduction in AS, then the level of perceived effort needs only be small enough to result in an exposure to noticeable somatic sensations.

With regards to expectancy, treatment expectations refer to improvements that clients believe will occur (Kazdin, 1979). However, although people often have specific expectations regarding exercise, it is possible that the nature of the exercise intensities (mild or moderate), or modality (treadmill), did not live up to those expectations. For example, if someone is asked if he/she believes exercising at a mild or moderate intensity will be effective, he/she may have varying beliefs about what 'mild' or 'moderate' exercise will entail. Therefore, if their beliefs about mild or moderate exercise were vastly different than the actual requirements of this study, the expectation or placebo effect may be muted.

In addition, since participants were not screened or matched based on physical fitness, it is possible that fitness levels may have confounded the effects of expectancy. For example, if

individuals had extremely poor physical fitness levels, they may have expectations that exercise can, and will be beneficial but still experience increased anxiety based on beliefs about their ability to complete the exercise requirements. Furthermore, if individuals were more physically fit, they may believe that exercise is effective at reducing anxiety, but require extremely high intensities of exercise to have their expectations met. As a result, it will be important for future studies in this area to recruit participants who are of the same physical fitness levels, and/or attempt to match participants across groups. Furthermore, as noted previously, expectancy has been found to increase treatment adherence as well as engagement in treatment. Therefore, it is possible that the current intervention was too regimented to allow for factors such as expectancy and credibility to have an enhancing effect on the treatment.

Limitations

In addition to the limitations already noted, there are three additional limitations that are important to note. The first limitation to this study is related to the factors associated with utilizing an accurate control group. In order to ensure that participants in the control group had an identical experience to the participants in the exercising groups, they were required to remain in the lab for an equivalent amount of time, wear a heart monitor, and fill out all of the same questionnaires. However, despite the care taken to ensure the control group was identical in every way except for the amount of exercise, it is possible that the factor of 'engagement' was not equivalent for both exercisers and non-exercisers. While in the lab, participants in the exercise conditions had a task to complete (running on a treadmill) whereas the control group had no such task. Thus, it is possible that the differences in ASI were a result of engagement in an activity rather than for any specific reason associated with exercise. Due to the experience of sitting quietly for 20 minutes without any obvious purpose, it is possible that the participants in

the control group were bored, distracted or experiencing thoughts, worry or rumination related to AS. This process may have served to facilitate the observed lack of change in AS scores for this group.

In order to prevent the limitation of engagement, participants in the control group could have been asked to participate in some activity or task (i.e., do a puzzle or read an article). However, although an activity would mitigate the issue of engagement, it would have engendered concerns about the type of engagement. That is, if the no exercise group were engaged in a cognitive task, it still would not have been identical to the physically engaging task completed by those in the exercise conditions. Thus, despite the concerns associated with potential engagement, it is difficult to imagine a physiologically equivalent engaging activity for the control group that would not constitute exercise itself, at which point it would no longer be a control group but would rather be an alternative exercise condition.

The second potential limitation to this study occurred as a result of the random assignment to groups. Although group membership was randomly assigned, participants in the mild exercise condition reported significantly higher ASI scores at the outset of the study compared with the no exercise and moderate exercise condition. While the overall decline in ASI scores were similar for both the mild and moderate exercise groups, the initial high scores of the mild group provides some limitations for interpretation and generalizability. For example, perhaps moderate exercise would have a greater impact on individuals who are higher in AS to begin with or conversely, mild exercise may not be effective whatsoever for individuals with lower AS initially. Thus future studies will need to utilize a matched group design to determine if initial scores on measures of AS impact potential outcome.

Finally, the third limitation to this study is a product of the exercise design itself in that the experimental design utilized removed the majority of the control from the participants in terms of how intensely they exercised. Considering that most physical exercise is at self-selected intensities, removing this control from participants may be reducing the effectiveness of exercise to ameliorate anxiety symptoms. This may occur as a result of distraction (i.e., participants may have been thinking about other exercise they may have engaged in and been comparing it to the required exercise), fear (i.e., participants may have actually experienced an increase in fear or anxiety, based on their beliefs about their ability to complete the required exercise), or other emotional responses (i.e., participants may have experienced resentment, anger or frustration at their selected intensity and ruminated on this fact).

Future Directions

In order to advance this area of research, future studies are necessary to evaluate effects of alternate forms of exercise as well as minimal doses of exercises necessary to obtain an effect. Given the goal of determining the function that expectancy and credibility play in reducing anxiety through exercise, as well as the concerns with having different initial scores of AS it would be beneficial to conduct a similar study using a matched participants design. Although a matched design would have its own limitations, this type of participant assignment would allow researchers to screen for both high and low levels of expectancy and credibility, as well as for initial scores of AS. Consequently, equal distribution across groups would allow for a more refined interpretation of the effects of exercise on AS.

Future studies should also be of a longitudinal design to determine if the reduction in AS is maintained and if a corresponding reduction in the onset of anxiety disorders is noted. By following at risk participants for months and potentially years and comparing rate of onset of

future anxiety disorders with population base rates, a more definitive conclusion could be made with respect to the ability of exercise to prevent anxiety disorders.

A further important area for future studies to focus on is the examination of fitness levels to determine if factors such as fitness are directly related to credibility and expectancy. Specific measures of fitness such as oxygen consumption or fit tests could be used to more accurately determine if there is a difference in outcome in efficacy for individuals with high or low levels of cardiovascular health.

Future directions for study should also include more diverse populations including clinical participants. Participant sampling using individuals suffering from diagnosed anxiety disorders would provide a much clearer picture of the ability of exercise to treat anxiety disorders as opposed to prevent.

Finally, other possible considerations for future areas of study include manipulating variables such as expectations and credibility, (i.e., by providing altered information), current anxiety levels (i.e., through anxiety induction), and perceived effort (i.e., increasing ambient temperature to induce sweating). By creatively controlling these variables researchers can better understand how to maximize or minimize their impact with respect to the anxiolytic effects of exercise.

Conclusion

Overall, the results indicate that exercise is associated with a reduction of AS. This study replicated the findings of three previous studies examining this construct. However, this study was unique from other studies examining this relationship due to the utilization of a control group, narrow ranges of exercise intensities and an evaluation of perceived effort, expectancy, and credibility. The use of an empirical control group was significant as it controlled for time

spent in the laboratory setting as well as time spent with the experimenter. Thus, the finding that these participants did not experience significant change in ASI lends strength to the conclusion that the findings are not a result of experimenter effects or regression to the mean effects. Furthermore, this study found that there was no support of a dose effect as the two exercise conditions experienced equal amounts of change in AS. The assertion that mild forms of exercise (30%-40% of HR_{max}) are effective at decreasing AS may indicate that the question of dose response may not be how much exercise is necessary for a reduction in AS but how little. This finding is especially important to the discussion of exercise prescription in determining how much is necessary, and how to maximize treatment adherence and effectiveness.

To date, research evaluating the relationship between exercise and AS has been consistent. Brief sessions of mild to moderate exercise utilizing a treadmill two to three times a week over a two week period reliably reduces scores of AS over time. Despite the necessity to further expand the literature on exercise and AS, the positive change associated with exercise is a significant one. Due to the high rates of anxiety disorders in North America and the costs associated with such disorders, finding effective and accessible treatments to prevent the onset of these disorders is an important goal. By identifying the factors which enhance or mitigate the effects of exercise on AS it is possible that effective treatment strategies can be developed which would have a significant impact on decreasing AS and potentially decrease the onset of future psychological disorders.

References

ACSM (2012, June 15). ACSM Issues New Recommendations on Quantity and Quality of Exercise. Retrieved from http://acsm.org/.

Ader, R. (1997). The role of conditioning in pharmacotherapy. In A. Harrington (Ed.), *The placebo effect: An interdisciplinary exploration.* (pp. 138-165). Cambridge, MA, US: Harvard University Press.

Anderson, E., & Shivakumar, G. (2013). Effects of exercise and physical activity on anxiety. *Frontiers in Psychiatry, 4* doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.3389/fpsyt.2013.00027

Anderson, F. C., & Pandy, M. G. (2001). Static and dynamic optimization solutions for gait are practically equivalent. *Journal of Biomechanics, 34*(2), 153-161. doi:10.1016/S00219290(00)00155-X

Antony, M. M., Ledley, D. R., Liss, A., & Swinson, R. P. (2006). Responses to symptom induction exercises in panic disorder. *Behaviour Research and Therapy, 44*(1), 85-98. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1016/j.brat.2004.12.005

Arikawa, A. Y., O'Dougherty, M., Kaufman, Schmitz, K. H., & Kruzer, (2011). Adherence to a strength training intervention in adult women. *Journal of Physical Activity, & Health, 8*(1), 111-118.

Arnau, R. C., Broman-Fulks, J. J., Green, B. A., & Berman, M. E. (2009). The anxiety sensitivity Index—Revised: Confirmatory factor analyses, structural invariance in Caucasian and african american samples and score reliability and validity. *Assessment, 16*(2), 165-180. doi:10.1177/1073191108328809

Asakawa, A., Inui, A., Momose, K., Ueno, N., Fujino, M. A., & Kasuga, M. (1998). Endorphins have exigenic and anxiolytic activities in mice. *Neuroreport: An International Journal for the Rapid Communication of Research in Neuroscience, 9*(10), 2265-2267. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1097/00001756-199807130-00022

Atlantis, E., Chow, C., Kirby, A., & Singh, M. F. (2004). An effective exercise-based intervention for improving mental health and quality of life measures: A randomized controlled trial. *Preventive Medicine: An International Journal Devoted to Practice and Theory, 39*(2), 424-434. doi:10.1016/j.ypmed.2004.02.007

Austin, D. W., & Richards, J. C. (2001). The catastrophic misinterpretation model of panic disorder. *Behaviour Research and Therapy, 39*(11), 1277-1291. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1016/S0005-7967(00)00095-4

Babyak, M., Blumenthal, J. A., Herman, S., Khatri, P., Doraiswamy, M., Moore, K., Krishnan, K. R. (2000). Exercise treatment for major depression: Maintenance of therapeutic benefit at 10 months. *Psychosomatic Medicine, 62*(5), 633-638.

Bahrke, M. S., & Morgan, W. P. (1978). Anxiety reduction following exercise and meditation. *Cognitive Therapy and Research, 2*(4), 323-333. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1007/BF01172650

Bandura, A. (1997). *Self-efficacy: The exercise of control*. New York, NY, US: W H Freeman/Times Books/ Henry Holt, & Co, New York, NY.

Berk, M. (2007). Should we be targeting exercise as a routine mental health intervention? *Acta Neuropsychiatrica, 19*(3), 217-218.

Berry, T. R., & Howe, B. L. (2004). Effects of health-based and appearance-based exercise advertising on exercise attitudes, social physique anxiety and self-presentation in an exercise setting. *Social Behavior and Personality, 32*(1), 1-12. doi:10.2224/sbp.2004.32.1.1

Bodin, T., & Martinsen, E. W. (2004). Mood and self-efficacy during acute exercise in clinical depression. A randomized, controlled study. *Journal of Sport, & Exercise Psychology, 26*(4), 623-633.

Borg, G. (1998). *Borg's perceived exertion and pain scales*. Champaign, IL, US: Human Kinetics, Champaign, IL.

Breus, M. J., & O'Connor, P. J. (1998). Exercise-induced anxiolysis: A test of the "time out" hypothesis in high anxious females. *Medicine, & Science in Sports, & Exercise, 30*(7), 1107-1112. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1097/00005768-199807000-00013

Broman-Fulks, J., Deacon, B., Olatunji, B., Bondy, C., Abramowitz, J., & Tolin, D. (2010). Categorical or dimensional: A reanalysis of the anxiety sensitivity construct. *Behaviour Therapy, 41*(2), 154-171.

Broman-Fulks, J. J., & Storey, K. M. (2008). Evaluation of a brief aerobic exercise intervention for high anxiety sensitivity. *Anxiety, Stress, & Coping: An International Journal, 21*(2), 117-128. doi:10.1080/10615800701762675

Broman-Fulks, J. J., Berman, M. E., Rabian, B. A., & Webster, M. J. (2004). Effects of aerobic exercise on anxiety sensitivity. *Behaviour Research and Therapy, 42*(2), 125-136. doi:10.1016/S0005-7967(03)00103-7

Broocks, A., Bandelow, B., Pekrun, G., George, A., Meyer, T., Bartmann, U., Hillmer-Vogel, U., & Rüther, E. (1998). Comparison of aerobic exercise, clomipramine and placebo in the treatment of panic disorder. *The American Journal of Psychiatry, 155*(5), 603-609.

Broocks, A., Schweiger, U., & Pirke, K. M. (1991). The influence of semistarvation-induced hyperactivity on hypothalamic serotonin metabolism. *Physiology & Behavior, 50*(2), 385-388. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1016/0031-9384(91)90082-Y

Buckalew, L. W., & Ross, S. (1981). Relationship of perceptual characteristics to efficacy of placebos. *Psychological Reports, 49*(3), 955-961.

Byrne, A., & Byrne, D. G. (1993). The effect of exercise on depression, anxiety and other mood states: A review. *Journal of Psychosomatic Research, 37*(6, pp. 565-574), September. doi:10.1016/0022-3999(93)90050-P

Callaghan, P. (2004). Exercise: A neglected intervention in mental health care? *Journal of Psychiatric and Mental Health Nursing, 11*(4), 476-783. doi:10.1111/j.1365-2850.2004.00751.x

Caspersen, C. J., Powell, & Christenson. (1985). Physical activity, exercise and physical fitness: Definitions and distinctions for health-related research. *Public Health Reports Hyattsville, 100*(2), 126.

Charney, D. S., Stein, M. B., Bienvenu, O. J., Crowe, R. R., Fuchs, E., Flügge, G., . . . Gorman, J. M. (2004). *Part V: Anxiety disorders*. New York, NY, US: Oxford University Press, New York, NY.

Cheyne, C. (2005). Exploiting placebo effects for therapeutic benefit. *Health Care Analysis, 13*(3), 177-188. doi:10.1007/s10728-005-6444-x

Chu, H., Bushman, B. A., & Woodard, R. J. (2008). Social physique anxiety, obligation to exercise and exercise choices among college students. *Journal of American College Health, 57*(1), 7-13. doi:10.3200/JACH.57.1.7-14

Conn, V. S. (2010). Anxiety outcomes after physical activity interventions: Meta-analysis findings. *Nursing Research, 59*(3), 224-31.

Conn, V. S. (2010). Depressive symptom outcomes of physical activity interventions: Meta-analysis findings. *Annals of Behavioral Medicine, 39*(2), 128-138. doi:10.1007/s12160-010-9172-x

Courneya, K. S., Friedenreich, C. M., Arthur, K., & Bobick, T. M. (1999). Physical exercise and quality of life in postsurgical colorectal cancer patients. *Psychology, Health, & Medicine, 4*(2), 181-187. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1080/135485099106315

Cox, B. J., Enns, M. W., Freeman, P., & Walker, J. R. (2001). Anxiety sensitivity and major depression: Examination of affective state dependence. *Behaviour Research and Therapy, 39*(11), 1349-1356. doi:10.1016/S0005-7967(00)00106-6

Cox, R. H., Thomas, T. R., Hinton, P. S., & Donahue, O. M. (2006). Effects of acute bouts of aerobic exercise of varied intensity on participantive mood experiences in women of different age groups across time. *Journal of Sport Behavior, 29*(1), 40-59.

Crum, A. J., & Langer, E. J. (2007). Mind-set matters: Exercise and the placebo effect. *Psychological Science, 18*(2), 165-171. doi:10.1111/j.1467-9280.2007.01867.x

Daley, A. J., Mutrie, N., Crank, H., Coleman, R., & Saxton, J. (2004). Exercise therapy in women who have had breast cancer: Design of the sheffield women's exercise and well-being project. *Health Education Research, 19*(6), 686-697. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1093/her/cyg094

Darko, D. F., Irwin, M. R., Risch, S. C., & Gillin, J. C. (1992). Plasma beta-endorphin and natural killer cell activity in major depression: A preliminary study. *Psychiatry Research, 43*(2), 111-119. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1016/0165-1781(92)90125-M

Darko, D. F., Risch, S. C., Gillin, J. C., & Golshan, S. (1992). Association of b-endorphin with specific clinical symptoms of depression. *The American Journal of Psychiatry, 149*(9), 1162-1167.

de Geus, E., (2008). Testing causality in the association between regular exercise and symptoms of anxiety and depression. *Archives of General Psychiatry, 65*(8), 897-905.

De Moor, M. H. M., Beem, A. L., Stubbe, J. H., Boomsma, D. I., & de Geus, E. J. C. (2006). Regular exercise, anxiety, depression and personality: A population-based study. *Preventive Medicine: An International Journal Devoted to Practice and Theory, 42*(4), 273-279. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1016/j.ypmed.2005.12.002

De Moor, M. H. M., Boomsma, D. I., Stubbe, J. H., Willemsen, G., & de Geus, E. J. C. (2008). Testing causality in the association between regular exercise and symptoms of anxiety and depression. *Archives of General Psychiatry, 65*(8), 897-905. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1001/archpsyc.65.8.897

Deacon, B. J., Abramowitz, J. S., Woods, C. M., & Tolin, D. F. (2003). The anxiety sensitivity index - revised: Psychometric properties and factor structure in two nonclinical samples. *Behaviour Research and Therapy, 41*(12), 1427-1449. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1016/S0005-7967(03)00065-2

DeBoer, L. B., Powers, M. B., Utschig, A. C., Otto, M. W., & Smits, J. A. J. (2012). Exploring exercise as an avenue for the treatment of anxiety disorders. *Expert Review of Neurotherapeutics, 12*(8), 1011-1022. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1586/ern.12.73

DeBusk, R. F., Stenestrand, U., Sheehan, M., & Haskell, W. L. (. (1990). Training effects of long versus short bouts of exercise in healthy participants. *The American Journal of Cardiology, 65*(15), 1010-1013. doi:10.1016/0002-9149(90)91005-Q

Desharnais, R., Bouillon, J., & Godin, G. (1987). Participants' early impressions of a supervised exercise program as a determinant of their subsequent adherence. *Perceptual and Motor Skills, 64*(3), 847-850.

Desharnais, R., Jobin, J., Côté, C., & Lévesque, L. (1993). Aerobic exercise and the placebo effect: A controlled study. *Psychosomatic Medicine, 55*(2), 149-154.

Deslandes, A., Moraes, H., Ferreira, C., Veiga, H., Silveira, H., Mouta, R., Pompeu, F. A. M. S., Coutinho, E. S. F., & Laks, J. (2009). Exercise and mental health: Many reasons to move. *Neuropsychobiology, 59*(4), 191-198. doi:10.1159/000223730

Devilly, G. J., & Borkovec, T. D. (2000). Psychometric properties of the credibility/expectancy questionnaire. *Journal of Behavior Therapy and Experimental Psychiatry, 31*(2), 73-86. doi:10.1016/S0005-7916(00)00012-4

deVries, H. A. (1987). *Tension reduction with exercise*. Washington, DC, US: Hemisphere Publishing Corp, Washington, DC.

Diagnostic and statistical manual of mental disorders: DSM-5TM (5th ed.) (2013). . Arlington, VA, US: American Psychiatric Publishing, Inc., Arlington, VA.

Dishman, R. K. (1997). Brain monoamines, exercise and behavioral stress: Animal models. *Medicine, & Science in Sports, & Exercise, 29*(1), 63-74. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1097/00005768-199701000-00010

Dunn, A. L., & Dishman, R. K. (2005). Anxiety and performance on the tour de france and tour de france feminin. *International Journal of Sport and Exercise Psychology, 3*(4), 410-427.

Dunn, A. L., Reigle, T. G., Youngstedt, S. D., Armstrong, R. B., & Dishman, R. K. (1996). Brain norepinephrine and metabolites after treadmill training and wheel running in rats. *Medicine & Science in Sports & Exercise, 28*(2), 204-209. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1097/00005768-199602000-00008

Dunn, A. L., Trivedi, M. H., & O'Neal, H. A. (2001). Physical activity dose–response effects on outcomes of depression and anxiety. *Medicine, & Science in Sports, & Exercise, 33*(6, Suppl), S587-S597. doi:10.1097/00005768-200106001-00027

Ehlers, A. (1995). Cardiac perception, panic attacks and phobias. In D. Vaitl, & R. Schandry (Eds.), *From the heart to the brain: The psychophysiology of circulation – brain interaction.* (pp. 299-313). New York, NY, US: Peter Lang Publishing.

Ellis, N., Crone, D., Davey, R., & Grogan, S. (2007). Exercise interventions as an adjunct therapy for psychosis: A critical review. *British Journal of Clinical Psychology, 46*(1), 95-111. doi:10.1348/014466506X122995

Esquivel, G., Fernández-Torre, O., Schruers, K. R. J., Wijnhoven, L. L. W., & Griez, E. J. L. (2009). The effects of opioid receptor blockade on experimental panic provocation with CO2. *Journal of Psychopharmacology, 23*(8), 975-978. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1177/0269881108093844

Felts, W. M. (1989). Relationship between ratings of perceived exertion and exercise-induced decrease in state anxiety. *Perceptual and Motor Skills, 69*(2), 368-370.

Floyd, A. (2010). Group versus individual exercise interventions for women with breast cancer: A meta-analysis. *Health Psychology Review, 4*(1), 22.

Foley, L. S., Prapavessis, H., Osuch, E. A., De Pace, J. A., Murphy, B. A., & Podolinsky, N. J. (2008). An examination of potential mechanisms for exercise as a treatment for depression: A pilot study. *Mental Health and Physical Activity, 1*(2), 69-73. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1016/j.mhpa.2008.07.001

Forsyth, J. P., Fusé, T., & Acheson, D. T. (2009). Interoceptive exposure for panic disorder. In W. T. O'Donohue, & J. E. Fisher (Eds.), *General principles and empirically supported techniques of cognitive behavior therapy.* (pp. 394-406). Hoboken, NJ, US: John Wiley, & Sons Inc.

Forsyth, J. P., Parker, J. D., & Finlay, C. G. (2003). Anxiety sensitivity, controllability and experiential avoidance and their relation to drug of choice and addiction severity in a residential sample of substance-abusing veterans. *Addictive Behaviors, 28*(5), 851-870. doi:10.1016/S0306-4603(02)00216-2

Frank, J. D. (1973). *Persuasion and healing: A comparative study of psychotherapy*. Oxford, England: Johns Hopkins U. Press.

Gillison, F. B., Skevington, S. M., Sato, A., Standage, M., & Evangelidou, S. (2009). The effects of exercise interventions on quality of life in clinical and healthy populations; a meta-analysis. *Social Science, & Medicine, 68*(9), 1700-1710. doi:10.1016/j.socscimed.2009.02.028

Goodwin, R. D. (2003). Association between physical activity and mental disorders among adults in the united states. *Preventive Medicine, 36*(6), 698-703. doi:10.1016/S0091-7435(03)00042-2

Hahn, R. A. (1997). The nocebo phenomenon: Concept, evidence and implications for public health. *Preventive Medicine, 26*(5), 607-611. doi:10.1006/pmed.1996.0124

Hegadoren, K. M., O'Donnell, T., Lanius, R., Coupland, N. J., & Lacaze-Masmonteil, N. (2009). The role of β-endorphin in the pathophysiology of major depression. *Neuropeptides, 43*(5), 341-353. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1016/j.npep.2009.06.004

Heinz, A., Jones, D. W., Bissette, G., Hommer, D., Ragan, P., Knable, M., . . . Weinberger, D. R. (2002). Relationship between cortisol and serotonin metabolites and transporters in alcoholism. *Pharmacopsychiatry, 35*(4), 127-134. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1055/s-2002-33197

Howell, A. N., Leyro, T. M., Hogan, J., Buckner, J. D., & Zvolensky, M. J. (2010). Anxiety sensitivity, distress tolerance and discomfort intolerance in relation to coping and conformity motives for alcohol use and alcohol use problems among young adult drinkers. *Addictive Behaviors,* doi:10.1016/j.addbeh.2010.07.003

Hughes, C. W., Petty, F., Sheikha, S., & Kramer, G. L. (1996). Whole-blood serotonin in children and adolescents with mood and behavior disorders. *Psychiatry Research, 65*(2), 79-95. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1016/S0165-1781(96)02932-0

Jancey, J., Lee, A., Howat, P., Clarke, A., Wang, K., & Shilton, T. (2007). Reducing attrition in physical activity programs for older adults. *Journal of Aging and Physical Activity, 15*(2), 152-165.

Jebb, S. A. (1999). Contribution of a sedentary lifestyle and inactivity to the etiology of overweight and obesity: Current evidence and research issues. *Medicine and Science in Sports and Exercise, 31*(11), S534.

Johansson, M., Hassmén, P., & Jouper, J. (2008). Acute effects of qigong exercise on mood and anxiety. *International Journal of Stress Management, 15*(2), 199-207. doi:10.1037/1072-5245.15.2.199

Johnson, E., Roth, T., & Breslau, N. (2006). The Association of insomnia with anxiety disorders and depression: Exploration of the direction of risk. *Journal of Psychiatric Research, 40*(8), 700-708.

Joiner, T. E., Jr., Schmidt, N. B., Schmidt, K. L., Laurent, J., Catanzaro, S. J., Perez, M., & Pettit, J. W. (2002). Anxiety sensitivity as a specific and unique marker of anxious symptoms in youth psychiatric inpatients. *Journal of Abnormal Child Psychology, 30*(2), 167-175. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1023/A:1014757300294

Jones, F., Harris, P., & McGee, L. (1998). Adherence to prescribed exercise. In L. B. Myers, & K. Midence (Eds.), *Adherence to treatment in medical conditions.* (pp. 343-362). Amsterdam, Netherlands: Harwood Academic Publishers.

Jones, F., Harris, P., Waller, H., & Coggins, A. (2005). Adherence to an exercise prescription scheme: The role of expectations, self-efficacy, stage of change and psychological well-being. *British Journal of Health Psychology, 10*(3), 359-378. doi:10.1348/135910704X24798

Kaptchuk, T. J. (2000). Do medical devices have enhanced placebo effects? *Journal of Clinical Epidemiology, 53*(8), 786.

Kaptchuk, T. J., Stason, W. B., Davis, R. B., Legedza, A. T. R., Schnyer, R. N., Kerr, C. E., . . . Goldman, R. H. (2006). Sham device v inert pill: Randomised controlled trial of two placebo treatments. *BMJ: British Medical Journal, 332*(7538), 391-397. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1136/bmj.38726.603310.55

Katula, J. A., Blissmer, B. J., & McAuley, E. (1999). Exercise and self-efficacy effects on anxiety reduction in healthy, older adults. *Journal of Behavioral Medicine, 22*(3), 233-247. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1023/A:1018768423349

Kelley, G. A., Kelley, K. S., Hootman, J. M., & Jones, D. L. (2009). Exercise and health-related quality of life in older community-dwelling adults: A meta-analysis of randomized controlled trials. *Journal of Applied Gerontology, 28*(3), 369-394. doi:10.1177/0733464808327456

Keough, M. E., Riccardi, C. J., Timpano, K. R., Mitchell, M. A., & Schmidt, N. B. (2010). Anxiety symptomatology: The association with distress tolerance and anxiety sensitivity. *Behavior Therapy,* doi:10.1016/j.beth.2010.04.002

Kerr, J. H., Fujiyama, H., Sugano, A., Okamura, T., Chang, M., & Onouha, F. (2006). Psychological responses to exercising in laboratory and natural environments. *Psychology of Sport and Exercise, 7*(4), 345-359. doi:10.1016/j.psychsport.2005.09.002

Kessler, R. C., Chiu, W. T., Demler, O., Merikangas, K. R., & Walters, E. E. (2005). Prevalence, severity and comorbidity of 12-month DSM-IV disorders in the national comorbidity survey replication. *Archives of General Psychiatry, 62*(6), 617-627.

King, A. C., Pruitt, L. A., Woo, S., Castro, C. M., Ahn, D. K., Vitiello, M. V., . . . Bitwise, D. L. (2008). Effects of moderate-intensity exercise on polysomnographic and participantive sleep quality in older adults with mild to moderate sleep complaints. *The Journals of*

Gerontology: Series A: Biological Sciences and Medical Sciences, 63A(9), 997-1004. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1093/gerona/63.9.997

Kirsch, I. (1997). Specifying nonspecifics: Psychological mechanisms of placebo effects. In A. Harrington (Ed.), *The placebo effect: An interdisciplinary exploration.* (pp. 166-186). Cambridge, MA, US: Harvard University Press.

Kirsch, I. (2009). Antidepressants and the placebo response. *Epidemiology and Psychiatric Sciences, 18*(4), 318-322.

Kõks, S., Bourin, M., Võikar, V., Soosaar, A., & Vasar, E. (1999). Role of CCK in anti-exploratory action of paroxetine, 5-HT reuptake inhibitor. *International Journal of Neuropsychopharmacology, 2*(1), 9-16. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1017/S1461145799001297

Kramer, A. F., & Erickson, K. I. (2007). Effects of physical activity on cognition, well-being and brain: Human interventions. *Alzheimer's, & Dementia, 3*(21), S45-S51. doi:10.1016/j.jalz.2007.01.008

Kruisselbrink, L. D., Dodge, A. M., & Swanburg, S. L. (2004). Influence of same-sex and mixed-sex exercise settings on the social physique anxiety and exercise intentions of males and females. *Journal of Sport, & Exercise Psychology, 26*(4), 616-622.

Kupfer, D. J., Sewitch, D. E., Epstein, L. H., Bulik, C., McGowen, C. R., & Robertson, R. J. (1985). Exercise and subsequent sleep in male runners: Failure to support the slow wave sleep-mood-exercise hypothesis. *Neuropsychobiology, 14*(1), 5-12. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1159/000118193

Lagally, K. M., & Amorose, A. J. (2007). The validity of using prior rating pf perceived exertion to regulate resistance exercise intensity. *Perceptual and Motor Skills, 104*(2), 534-542. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.2466/PMS.104.2.534-542

Long, B. C., & van Stavel, R. (1995). Effects of exercise training on anxiety: A meta-analysis. *Journal of Applied Sport Psychology, 7*(2), 167-189. doi:10.1080/10413209508406963

Lundh, L. (1987). Placebo, belief and health: A cognitive-emotional model. *Scandinavian Journal of Psychology, 28*(2), 128-143. doi:10.1111/j.1467-9450.1987.tb00747.x

Mackay, G., & Neill, J. (2010). The effect of "green exercise" on state anxiety and the role of exercise duration, intensity and greenness: A quasi-experimental study. *Psychology of Sport and Exercise, 11*(3), 238-245.

Mago, R., & Mahajan, R. (2010). Exercise in the treatment of depressive disorders. In D. A. Monti, & B. D. Beitman (Eds.), *Integrative psychiatry.* (pp. 22-34). New York, NY, US: Oxford University Press.

Marquez, D. X., Jerome, G. J., McAuley, E., Snook, E. M., & Canaklisova, S. (2002). Self-efficacy manipulation and state anxiety responses to exercise in low active women. *Psychology, & Health, 17*(6), 783-791. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1080/0887044021000054782

Martinsen, E. W., Hoffart, A., & Solberg, Ø. (1989). Comparing aerobic with nonaerobic forms of exercise in the treatment of clinical depression: A randomized trial. *Comprehensive Psychiatry*, 30(4), 324-331. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1016/0010-440X(89)90057-6

McAuley, E., Mihalko, S. L., & Bane, S. M. (1996). Acute exercise and anxiety reduction: Does the environment matter? *Journal of Sport, & Exercise Psychology, 18*(4), 408-419.

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*(3), 161-170. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1016/S0887-6185(01)00056-1

Mellinger, G. D., Balter, M. B., & Uhlenhuth, E. H. (1985). Insomnia and its treatment: Prevalence and correlates. *Archives of General Psychiatry, 42*(3), 225-232. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1001/archpsyc.1985.01790260019002

Mellman, T. A. (2006). Sleep and anxiety disorders. *Psychiatric Clinics of North America, 29*(4), 1047-1058. doi:10.1016/j.psc.2006.08.005

Meyer, B., Pilkonis, P. A., Krupnick, J. L., Egan, M. K., Simmens, S. J., & Sotsky, S. M. (2002). Treatment expectancies, patient alliance and outcome: Further analyses from the national institute of mental health treatment of depression collaborative research program. *Journal of Consulting and Clinical Psychology, 70*(4), 1051-1055. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1037/0022-006X.70.4.1051

Montgomery, G. H., & Kirsch, I. (1997). Classical conditioning and the placebo effect. *Pain, 72*(1-2), 107-113. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1016/S0304-3959(97)00016-X

Moreno, J., Campos, M. G., Lara, C., López, G., Pavón, L., Hernández, M. E., Torner, C. (2006). Tryptophan and serotonin in blood and platelets of depressed patients: Effect of an antidepressant treatment. *Salud Mental, 29*(4), 1-8.

Morgan, W. P. (1985). Affective beneficence of vigorous physical activity. *Medicine, & Science in Sports, & Exercise, 17*(1), 94-100. doi:10.1249/00005768-198502000-00015

Morin, C. M., & Edinger, J. D. (1999). Sleep/wake disorders. (pp. 390-409). New York, NY, US: Oxford University Press, New York, NY.

Newman, T.C. (2009). Walking Through Anxiety: Examing the role of expectancy in exercise. (Unpublished Thesis). Lakehead University, Thunder Bay.

O'Connor, P. J., Raglin, J. S., & Martinsen, E. W. (2000). Physical activity, anxiety and anxiety disorders. *International Journal of Sport Psychology.Special Issue: Exercise Psychology, 31*(2), 136-155.

Oeland, A., Laessoe, U., Olesen, A. V., & Munk-Jørgensen, P. (2010). Impact of exercise on patients with depression and anxiety. *Nordic Journal of Psychiatry, 64*(3), 210-217. doi:10.3109/08039480903511373

Ohayon, M. M., & Caulet, M. (1996). Psychotropic medication and insomnia complaints in two epidemiological studies. *The Canadian Journal of Psychiatry / La Revue Canadienne De Psychiatrie, 41*(7), 457-464.

Ohayon, M. M., & Roth, T. (2003). Place of chronic insomnia in the course of depressive and anxiety disorders. *Journal of Psychiatric Research, 37*(1), 9-15. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1016/S0022-3956(02)00052-3

Ojanen, M. (1994). Can the true effects of exercise on psychological variables be separated from placebo effects? *International Journal of Sport Psychology, 25*(1), 63-80.

Oliver, D. P. (2008). Conquering depression and anxiety through exercise. *Journal of Gerontological Social Work, 51*(1-2), 179-181.

Otto, M. W., Pollack, M. H., Fava, M., & Uccello, R. (1995). Elevated anxiety sensitivity index scores in patients with major depression: Correlates and changes with antidepressant treatment. *Journal of Anxiety Disorders, 9*(2), 117-123. doi:10.1016/0887-6185(94)00035-2

Pacheco-López, G., Engler, H., Niemi, M., & Schedlowski, M. (2006). Expectations and associations that heal: Immunomodulatory placebo eVects and its neurobiology. *Brain, Behavior and Immunity, 20*(5), 430-446. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1016/j.bbi.2006.05.003

Paffenbarger, R. S., Kampert, J. B., Lee, I., Hyde, R. T., Leung, R. W., & Wing, A. L. (1994). Changes in physical activity and other lifeway patterns influencing longevity. *Medicine, & Science in Sports, & Exercise, 26*(7), 857-865. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1249/00005768-199407000-00008

Paluska, S. A., & Schwenk. (2000). Physical activity and mental health: Current concepts. *Sports Medicine, 29*(3), 167.

Pang, M. Y. C. (2006). The use of aerobic exercise training in improving aerobic capacity in individuals with stroke: A meta-analysis. *Clinical Rehabilitation, 20*(2), 97.

Papadimitriou, G. N., & Linkowski, P. (2005). Sleep disturbance in anxiety disorders. *International Review of Psychiatry, 17*(4), 229-236. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1080/09540260500104524

Parloff, M. B. (1986). Placebo controls in psychotherapy research: A sine qua non or a placebo for research problems? *Journal of Consulting and Clinical Psychology, 54*(1), 79-87. doi:10.1037/0022-006X.54.1.79

Peluso, M. A. M. (2005). Physical activity and mental health: The association between exercise and mood. *Clinics, 60*, 61.

Petruzzello, S. J., Landers, D. M., & Salazar, W. (1993). Exercise and anxiety reduction: Examination of temperature as an explanation for affective change. *Journal of Sport, & Exercise Psychology, 15*(1), 63-76.

Petruzzello, S., Landers, D., Hatfield, B., Kubitz, K., & Salazar, W. (1991). A meta-analysis on the anxiety-reducing effects of acute and chronic exercise. *Sports Medicine, 11,* 143-182.

Pickar, D., Cohen, M. R., Naber, D., & Cohen, R. M. (1982). Clinical studies of the endogenous opioid system. *Biological Psychiatry, 17*(11), 1243-1276.

Plante, T. G. (1999). Could the perception of fitness account for many of the mental and physical health benefits of exercise? *Advances in Mind-Body Medicine, 15*(4), 291-295.

Polivy, J., & Herman, C. P. (2000). The false-hope syndrome: Unfulfilled expectations of self-change. *Current Directions in Psychological Science, 9*(4), 128-131. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1111/1467-8721.00076

Pretty, J., Peacock, J., Sellens, M., & Griffin, M. (2005). The mental and physical health outcomes of green exercise. *International Journal of Environmental Health Research, 15*(5), 319-337. doi:10.1080/09603120500155963

Price, D. D., Finniss, D. G., & Benedetti, F. (2008). A comprehensive review of the placebo effect: Recent advances and current thought. *Annual Review of Psychology, 59*, 565-590. doi:10.1146/annurev.psych.59.113006.095941

Rabian, B., Embry, L., & MacIntyre, D. (1999). Behavioral validation of the childhood anxiety sensitivity index in children. *Journal of Clinical Child Psychology, 28*(1), 105-112. doi:10.1207/s15374424jccp2801_9

Radak, Z., Hart, N., Sarga, L., Koltai, E., Atalay, M., Ohno, H., & Boldogh, I. (2010). Exercise plays a preventive role against Alzheimer's disease. *Journal of Alzheimer's Disease, 20*(3), 777-783.

Raglin, J. S., & Morgan, W. P. (1987). Influence of exercise and quiet rest on state anxiety and blood pressure. *Medicine, & Science in Sports, & Exercise, 19*(5), 456-463. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1249/00005768-198710000-00006

Reed, J., & Buck, S. (2009). The effect of regular aerobic exercise on positive-activated affect: A meta-analysis. *Psychology of Sport and Exercise, 10*(6), 581-594. doi:10.1016/j.psychsport.2009.05.009

Reeves, D. L., Justesen, D. R., Levinson, D. M., & Riffle, D. W. (1985). Endogenous hyperthermia in normal human participants: I. experimental study of evoked potentials and reaction time. *Physiological Psychology, 13*(4), 258-267.

Reiss, S. (1991). Expectancy model of fear, anxiety and panic. *Clinical Psychology Review. Special Issue: Applied Learning Theory: Research Issues for the 1990s, 11*(2), 141-153. doi:10.1016/0272-7358(91)90092-9

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), 1-8. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1016/0005-7967(86)90143-9

Resnick, B. (2004). A longitudinal analysis of efficacy expectations and exercise in older adults. *Research and Theory for Nursing Practice: An International Journal, 18*(4), 331-344. doi:10.1891/rtnp.18.4.331.64093

Reus, V. I., & Rawitscher, L. (2000). Possible interaction of tramadol and antidepressants. *The American Journal of Psychiatry, 157*(5), 838.

Ritvanen, T., Louhevaara, V., Helin, P., Halonen, T., & Hänninen, O. (2007). Effect of aerobic fitness on the physiological stress responses at work. *International Journal of Occupational Medicine and Environmental Health, 20*(1), 1-8. doi:10.2478/v10001-007-0005-5

Roman, M. (2010). Physical exercise as psychotherapeutic strategy: How long? what will it take? *Issues in Mental Health Nursing. Special Issue: Violence Exposure in Children, Adolescents and Women: The Lived Experience, 31*(2), 153-154. doi:10.3109/01612840903497610

Ryan, M. P. (2008). The antidepressant effects of physical activity: Mediating self-esteem and self-efficacy mechanisms. *Psychology, & Health, 23*(3), 279-307. doi:10.1080/14768320601185502

Sabiston, C. M., & Chandler, K. (2009). Effects of fitness advertising on weight and body shape dissatisfaction, social physique anxiety and exercise motives in a sample of healthy-weight females. *Journal of Applied Biobehavioral Research, 14*(4), 165-180. doi:10.1111/j.1751-9861.2010.00047.x

Salmon, P. (2001). Effects of physical exercise on anxiety, depression and sensitivity to stress: A unifying theory. *Clinical Psychology Review, 21*(1), 33-61. doi:10.1016/S0272-7358(99)00032-X

Scarone, S., Gambini, O., Calabrese, G., Sacerdote, P., Bruni, M., Carucci, M., & Panerai, A. E. (1990). Asymmetrical distribution of beta-endorphin in cerebral hemispheres of suicides: Preliminary data. *Psychiatry Research, 32*(2), 159-166. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1016/0165-1781(90)90082-G

Schatzberg, A. F., Cole, J. O., & DeBattista, C. (2007). *Manual of clinical psychopharmacology (6th ed.)*. Arlington, VA, US: American Psychiatric Publishing, Inc.

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

Schmidt, N. B., & Trakowski, J. (2004). Interoceptive assessment and exposure in panic disorder: A descriptive study. *Cognitive and Behavioral Practice, 11*(1), 81-92. doi:10.1016/S1077-7229(04)80010-5

Schmidt, N. B., Eggleston, A. M., Woolaway-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*(3), 302-319. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1016/j.janxdis.2006.06.002

Schmidt, N. B., Keough, M. E., Mitchell, M. A., Reynolds, E. K., MacPherson, L., Zvolensky, M. J., & Lejuez, C. W. (2010). Anxiety sensitivity: Prospective prediction of anxiety among early adolescents. *Journal of Anxiety Disorders, 24*(5), 503-508. doi:10.1016/j.janxdis.2010.03.007

Schmidt, N. B., Schmidt, K. L., Laurent, J., Catanzaro, S. J., Perez, M., & Pettit, J. W. (2002). Anxiety sensitivity as a specific and unique marker of anxious symptoms in youth psychiatric inpatients. *Journal of Abnormal Child Psychology, 30*(2), 167-175.

Schmidt, N. B., Zvolensky, M. J., & Maner, J. K. (2006). Anxiety sensitivity: Prospective prediction of panic attacks and axis I pathology. *Journal of Psychiatric Research, 40*(8), 691-699. doi:10.1016/j.jpsychires.2006.07.009

Sears, S. R., & Stanton, A. L. (2001). Expectancy-value constructs and expectancy violation as predictors of exercise adherence in previously sedentary women. *Health Psychology, 20*(5), 326-333. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1037/0278-6133.20.5.326

Simon, N. M., Otto, M. W., Fischmann, D., Racette, S., Nierenberg, A. A., Pollack, M. H., & Smoller, J. W. (2005). Panic disorder and bipolar disorder: Anxiety sensitivity as a potential

mediator of panic during manic states. *Journal of Affective Disorders, 87*(1), 101-105. doi:10.1016/j.jad.2005.02.004

Smeets, R. J. E. M., Beelen, S., Goossens, M. E. J. B., Schouten, E. G. W., Knottnerus, J. A., & Vlaeyen, J. W. S. (2008). Treatment expectancy and credibility are associated with the outcome of both physical and cognitive-behavioral treatment in chronic low back pain. *The Clinical Journal of Pain, 24*(4), 305-315. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1097/AJP.0b013e318164aa75

Smith, C. D. (2006). *A comparison of the effects of anaerobic and aerobic exercise on mood*. *Dissertation Abstracts International: Section B: The Sciences and Engineering, 67*(1-B), 561-561. (Electronic; Print). (2006-99014-153)

Smits, J. A. J., Berry, A. C., Powers, M. B., Greer, T. L., & Otto, M. W. (2008). The promise of exercise interventions for the anxiety disorders. In M. J. Zvolensky, & J. A. J. Smits (Eds.), *Anxiety in health behaviors and physical illness.* (pp. 81-104). New York, NY, US: Springer Science + Business Media. doi:10.1007/978-0-387-74753-8_4

Smits, J. A. J., Berry, A. C., Rosenfield, D., Powers, M. B., Behar, E., & Otto, M. W. (2008). Reducing anxiety sensitivity with exercise. *Depression and Anxiety, 25*(8), 689-699. doi:10.1002/da.20411

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*(9), 1047-1054. doi:10.1016/j.brat.2008.06.010

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*(4), 646-652.

Smits, J. A. J., Tart, C. D., Presnell, K., Rosenfield, D., & Otto, M. W. (2010). Identifying potential barriers to physical activity adherence: Anxiety sensitivity and body mass as predictors of fear during exercise. *Cognitive Behaviour Therapy, 39*(1), 28-36. doi:10.1080/16506070902915261

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*(2), 179-193. doi:10.1111/j.1468-2850.2006.00021.x

Stein, D. J., Collins, M., Daniels, W., Noakes, T. D., & Zigmond, M. (2007). Mind and muscle: The cognitive-affective neuroscience of exercise. *CNS Spectrums, 12*(1), 19-22.

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

Stewart, S. H., Peterson, J. B., & Pihl, R. O. (1995). Anxiety sensitivity and self-reported alcohol consumption rates in university women. *Journal of Anxiety Disorders, 9*(4), 283-292. doi:10.1016/0887-6185(95)00009-D

Stewart-Williams, S. (2004). The placebo puzzle: Putting together the pieces. *Health Psychology, 23*(2), 198-206. doi:10.1037/0278-6133.23.2.198

Stich, F. A. (1999). A meta-analysis of physical exercise as a treatment for symptoms of anxiety and depression. *Dissertation Abstracts International: Section B: The Sciences and Engineering, 59*(8-B), 4487-4487. (Electronic; Print). (1999-95004-037)

Ströhle, A. (2009). Physical activity, exercise, depression and anxiety disorders. *Journal of Neural Transmission, 116*(6), 777-784. doi:10.1007/s00702-008-0092-x

Strong, H. A., Martin Ginis, K. A., Mack, D. E., & Wilson, P. M. (2006). Examining self-presentational exercise motives and social physique anxiety in men and women. *Journal of Applied Biobehavioral Research, 11*(3-4), 209-225. doi:10.1111/j.1751-9861.2007.00006.x

Svenningsson, P., Chergui, K., Rachleff, I., Flajolet, M., Zhang, X., El Yacoubi, M., . . . Greengard, P. (2006). Alterations in 5-HT1B receptor function by p11 in depression-like states. *Science, 311*(5757), 77-80. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1126/science.1117571

Sylvia, L. G., Kopeski, L. M., Mulrooney, C., Reid, J., Jacob, K., & Neuhaus, E. C. (2009). Does exercise impact mood? exercise patterns of patients in a psychiatric partial hospital program. *Journal of Psychiatric Practice, 15*(1), 70-78. doi:10.1097/01.pra.0000344923.81898.53

Szabo, A. (2003). The acute effects of humor and exercise on mood and anxiety. *Journal of Leisure Research, 35*(2), 152-162.

Tabachnik, B.G., & Fidell, L.S. (2007). *Using multivariate statistics (5th ed.)*. Boston, MA: Pearson.

Tafet, G. E., Idoyaga-Vargas, V. P., Abulafia, D. P., Calandria, J. M., Roffman, S. S., Chiovetta, A., & Shinitzky, M. (2001). Correlation between cortisol level and serotonin uptake in patients with chronic stress and depression. *Cognitive, Affective, & Behavioral Neuroscience, 1*(4), 388-393. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.3758/CABN.1.4.388

Taylor, S. R., Rogers, G. G., & Driver, H. S. (1997). Effects of training volume on sleep, psychological and selected physiological profiles of elite female swimmers. *Medicine, & Science in Sports, & Exercise, 29*(5), 688-693. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1097/00005768-199705000-00016

Taylor, S., & Cox, B. J. (1998). Anxiety sensitivity: Multiple dimensions and hierarchic structure. *Behaviour Research and Therapy, 36*(1), 37-51. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1016/S0005-7967(97)00071-5

Taylor, S., Koch, W. J., & Crockett, D. J. (1991). Anxiety sensitivity, trait anxiety and the anxiety disorders. *Journal of Anxiety Disorders, 5*(4), 293-311. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1016/0887-6185(91)90030-W

Taylor, S., Koch, W. J., & McNally, R. J. (1992). How does anxiety sensitivity vary across the anxiety disorders? *Journal of Anxiety Disorders, 6*(3), 249-259. doi:10.1016/0887-6185(92)90037-8

Taylor, S., Zvolensky, M. J., Cox, B. J., Deacon, B., Heimberg, R. G., Ledley, D. R., . . . Cardenas, S. J. (2007). Robust dimensions of anxiety sensitivity: Development and initial validation of the anxiety sensitivity index-3. *Psychological Assessment, 19*(2), 176-188. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1037/1040-3590.19.2.176

Thorén, P., Floras, J. S., Hoffmann, P., & Seals, D. R. (1990). Endorphins and exercise: Physiological mechanisms and clinical implications. *Medicine, & Science in Sports, & Exercise, 22*(4), 417-428.

Walker, K., O'Dea, K., Gomez, M., Girgis, S., & Colagiuri, R. (2010). Diet and exercise in the prevention of diabetes. *Journal of Human Nutrition and Dietetics, 23*(4), 344-352.

Wampold, B. E., Minami, T., Tierney, S. C., Baskin, T. W., & Bhati, K. S. (2005). The placebo is powerful: Estimating placebo effects in medicine and psychotherapy from randomized clinical trials. *Journal of Clinical Psychology, 61*(7), 835-854. doi:10.1002/jclp.20129

Weissman, M. M., Bland, R. C., Canino, G. J., Faravelli, C., Greenwald, S., Hwu, H., . . . Yeh, E. (1997). The cross-national epidemiology of panic disorder. *Archives of General Psychiatry, 54*(4), 305-309. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1001/archpsyc.1997.01830160021003

Whiteley, J. A., Williams, D. M., & Marcus, B. H. (2006). Adherence to exercise regimens. In W. T. O'Donohue, & E. R. Levensky (Eds.), *Promoting treatment adherence: A practical handbook for health care providers.* (pp. 307-320). Thousand Oaks, CA, US: Sage Publications, Inc.

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

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*(3), 474-481. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1111/j.1600-0838.2009.01049.x

Wolff, E., & Strohle, A. (2010). Causal associations of physical activity/exercise and symptoms of depression and anxiety. *Archives of General Psychiatry, 67*(5), 540(2)-542.

Wong, S. N., Halaki, M., & Chow, C. (2013). The effects of moderate to vigorous aerobic exercise on the sleep need of sedentary young adults. *Journal of Sports Sciences, 31*(4), 381-386. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1080/02640414.2012.733823

Yeung, R. R. (1996). The acute effects of exercise on mood state. *Journal of Psychosomatic Research, 40*(2), 123-141. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1016/0022-3999(95)00554-4

Youngstedt, S. D., O'Connor, P. J., & Dishman, R. K. (1997). The effects of acute exercise on sleep: A quantitative synthesis. *Sleep: Journal of Sleep Research, & Sleep Medicine, 20*(3), 203-214.

Zajonc, R. B., & McIntosh, D. N. (1992). Emotions research: Some promising questions and some questionable promises. *Psychological Science, 3*(1), 70-74. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1111/j.1467-9280.1992.tb00261.x

Zarrindast, M., Lahmi, A., & Ahamadi, S. (2008). Possible involvement of μ-opioid receptors in effect of lithium on inhibitory avoidance response in mice. *Journal of Psychopharmacology, 22*(8), 865-871. doi:http://dx.doi.org.ezproxy.lakeheadu.ca/10.1177/0269881107083848

Appendix A Anxiety

Sensitivity Index

Reiss-Epstein-Gursky A.S.I.

Name _____Today's Date _____

Circle the one phrase that best represents the extent to which you agree with the item. If any of the items concern something that is not part of your experience (e.g., "It scares me when I feel shaky" for someone who has never trembled or had the "shakes"), answer on the basis of how you think you might feel *if you had* such an experience. Otherwise, answer all items on the basis of your own experience.

1. It is important to me not to appear nervous.

Very Little A Little Some Much Very Much

2. When I cannot keep my mind on a task, I worry that I might be going crazy.

Very Little A Little Some Much Very Much

3. It scares me when I feel "shaky" (trembling).

Very Little A Little Some Much Very Much

4. It scares me when I feel faint.

Very Little A Little Some Much Very Much

5. It is important to me to stay in control of my emotions.

Very Little A Little Some Much Very Much

6. It scares me when my heart beats rapidly.

Very Little A Little Some Much Very Much

7. It embarrasses me when my stomach growls.

Very Little A Little Some Much Very Much

8. It scares me when I am nauseous.

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

11. When my stomach is upset, I worry that I might be seriously ill.

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

14. Unusual body sensations scare me.

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

16. It scares me when I am nervous.

Very Little A Little Some Much Very Much

Appendix B

Credibility and Expectancy Questionnaire

We would like you to indicate below how much you believe, *right now*, that the group condition you are assigned to will help to impact your general mood. Belief usually has two aspects to it: (1) what one *thinks* will happen and (2) what one *feels* will happen. Sometimes these are similar; sometimes they are different. Please answer the questions below. In the first set, answer in terms of what you *think*. In the second set answer in terms of what you really and truly *feel*.

Set I

1. At this point, how logical does the intervention offered to you seem?

1 2 3 4 5 6 7 8 9

not at all logical somewhat logical very logical

2. At this point, how useful do you think this intervention will be in affecting your mood?

1 2 3 4 5 6 7 8 9

not at all useful somewhat useful very useful

3. How confident would you be in recommending this intervention to a friend to impact their mood?

1 2 3 4 5 6 7 8 9

not at all confident somewhat confident very confident

4. By the end of the intervention period, how much impact on your mood do you think will occur?

0% 10% 20% 30% 40% 50% 60% 70% 80% 90% 100%

Set II

For this set, close your eyes for a few moments and try to identify what you really *feel* about the therapy and its likely success. Then answer the following questions.

1. At this point, how much do you really *feel* that this intervention will help you to improve your mood?

1 2 3 4 5 6 7 8 9
not at all somewhat very much

2. By the end of the intervention period, how much improvement in mood do you really *feel* will occur?

0% 10% 20% 30% 40% 50% 60% 70% 80% 90% 100%

Appendix C

Borg Rating of Perceived Effort Scale

While doing physical activity, we want you to rate your perception of exertion. This feeling should reflect how heavy and strenuous the exercise feels to you, combining all sensations and feelings of physical stress, effort and fatigue. Do not concern yourself with any one factor such as leg pain or shortness of breath, but try to focus on your total feeling of exertion.

Look at the rating scale below while you are engaging in an activity; it ranges from 6 to 20, where 6 means "no exertion at all" and 20 means "maximal exertion." Choose the number from below that best describes your level of exertion. This will give you a good idea of the intensity level of your activity and you can use this information to speed up or slow down your movements to reach your desired range.

Try to appraise your feeling of exertion as honestly as possible, without thinking about what the actual physical load is. Your own feeling of effort and exertion is important, not how it compares to other people's. Look at the scales and the expressions and then give a number.

6 No exertion at all
7
Extremely light (7.5)
8
9 Very light
10
11 Light
12
13 Somewhat hard
14
15 Hard (heavy)
16
17 Very hard
18
19 Extremely hard
20 Maximal exertion

9 corresponds to "very light" exercise. For a healthy person, it is like walking slowly at his or her own pace for some minutes. 13 on the scale is "somewhat hard" exercise, but it still feels OK to continue. 17 "very hard" is very strenuous. A healthy person can still go on, but he or she really has to push him- or herself. It feels very heavy and the person is very tired. 19 on the scale is an extremely strenuous exercise level. For most people this is the most strenuous exercise they have ever experienced.

Borg RPE scale

Appendix D

Physical Activity Readiness Questionnaire

Phy>ical ActMiy Reodiness
stioMairo- R-Q
(ro,.st<l2002)

PAR-Q & YOU

(A Questionnaire for People Aged 15 to 69)

flegular physicalactivity ıs fun and heaHhy,and increasingmore people are starting to become more active evef'j clay; Betng more actiVe is very safe for most people.However,some people should che<k With thei'dodor before they start becoming rooch more physicalactive.

If yoo are planntng to become much more physJC.a!actiVe than yoo are now.start by answeong the seven questions in the box below. If yoo are between the ages of 15 and 69,the PAA-Q will telyoo d yoo should check Wllh yoor doctor before you start K yoo are CNer 69 years of age,and you are not used to betng very actrve.check with your doctor.

Common sense ıs your best guide when you answer these questions. Please read the questiOns carefuland answer each one honest: check YES or NO.

YES	NO	
O	O	1. Has your doctor ewer said that you hue a he.>rt co11ditfo11 lrulthat you should only do physical activity recomme11ded by a doctor?
O	O	Z. Do you feel pal• ie your chest whee you do physical actlvlty?
O	O	3. le the past •oeth, laawe yo• load cltest pal•whea you were aot dolag pltysical activity?
O	O	4. Do you lose your balaace becaue of dlzdaess or do you ner lose consciousness?
O	O	5. Do you have a bone or joint probleoa (for euoaple, back, ka"or hlp) that could be made worse by a change in your physical activity?
O	O	6. Is your doctor currently prescribing drugs (for eumple, water pills) for your blood pressure or heart con·dillon?
O	O	7. Do you know of anr other reasol! why you should not do physical activity?

If you answered

YES to one or more questions

Td with you doctor by phone *or* in personBEFORf ycu start beccmng much more physically aciM *or* BEFORE ycu have a fitness apprasal Tol your doctor about the PAR-Q and whichquesliorts ycu answered YES.

- You may be able to do any actlriy ycu want as long as ycu slart slcM4y and bUid up gradually. Or,ycu may need to r nct your actMties to tt-ose ...,ich *are* sale for l"""·Talk With)OUr doctor abOU the kinds ol actovitoes)'OU wish to participate in and folow hos/her advice.
- Find out whichcommlllity programs *are* safe and htlpful for you.

NO to all questions

ij ycu answered NO honesdy to ill PAR-Q questions, ycu can be reasonably swe that ycu can:

start becoming much more physocaly active – begin slcM4y and bUid l4l graduatj. This is the sal and easoest way to go.

Idke part in a fitnoss apprasal thos is an exc way to determone ycur basic fitness so that ycu canplan the best way for ycu to *live* ıs also hoghfy <Kommended that ycu have ycur blood presstre evaluat- If your reading is <Ner 1 4/94, talk wih ycur doctor before ycu start becoming much more physically active.

DELAT BE<OMIIIG MUCH MOllE ACnYE:

- if ycu are ncl lE•eling wei because ol a temporary ii1ess such as a cold or a fel'l!f wait l.f1d ycu better:*or*
- if)'OU are *or* may be prognant taft< toyotr doctor before ycu start becoming more acto...,_

PI.£ASE IIOTE: If)'OUr htaJth changesso that)'OU then answer YES to arry olthe aboYe questions, t•l)'OUr fitnoss or htalth professoonal. Asl<ycu shoUd change ycur physical acriYity plan.

krtormod lbc o!thePltf!.O: The C..adion SodctytorExmise P\oy>ioloCJl' tloalthc.n..t.,nt their agents.,...,.no W.lly!or ponom who me physical octi.,d i1doxbt alttl'COfi1'iotng this quostioonolro,consUl *your* doctor prior to physkol octivity

No cha"9es perlllittecl. You are •-raged to photocopy tile l'llR·Q but oniJ if J041 use the entire form.

NOTE: I 1110 *PAR-Q* is bMg ,_to•penon before be or she es-,-•*physiuloc!M"f* -or•lr>Hs opprtial.this soction ,---,ust<l lor legal or "'*"mtriiNO pwposos

'Ihave read,understood and completed this eslionnaire. Ant ques11011s I had were answered to my full sahslaclton.'

ruRE---------- M+ ----------

EOF ---- ---------- MmB !

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Ilole: This physical activity dearaooao Is nlid to. •IIIHI-•of 1Z –Its fro•the date It Is cooapleted becomes innlld if your condition changesso dl•t you –•ld ••–• YES to any of the seven questions.

Appendix E

Participant Information Form/Consent Form

Cover Letter (to be printed on LU letterhead)

Dear Potential Participant:

Thank you for taking part in this research study investigating the effect of exercise on general mood states. This study is being conducted by Thomas Newman, a Ph.D Psychology graduate student at Lakehead University and is supervised by Dr. Gordon Hayman, PhD. from the department of psychology at Lakehead University.

The primary purpose of this study is to examine the effect of various levels of exercise on general mood states of the exerciser. All participants will be randomly assigned to one of three possible groups including a moderate exercise group (70% of maximum heart rate), a mild exercise group (35% of maximum heart rate) or a quiet rest group. As an example, moderate exercise is seen as exercise that is able to raise heart rate, yet allow the exerciser to carry on a conversation while engaging in physical activity. For most individuals this would be similar too walking at a brisk pace or a slow jog. Mild exercise would be at an even lower level.

Participants in all groups will be asked to attend six 20 minute sessions over a two week period (i.e., three sessions per week). Participants will be asked to engage in their level of assigned activity during these sessions and complete short questionnaires. A wide variety of times will be available to accommodate each participant's schedule. You are asked to wear appropriate footwear for exercising (i.e., cross trainers) and comfortable clothing. Participants will also be asked to complete a questionnaire one week after completing their sessions. This last questionnaire will take only 2-5 minutes to complete.

To compensate participants for their time, all participants will be entered in a draw to win a $250.00 prepaid Visa™ gift card.

Although minimal in nature, there are some risks associated with this study. Some participants may not feel comfortable exercising on a treadmill for 20 minutes. In addition, participants will be asked to wear a heart rate monitor for the duration of their session which although unlikely, may cause minor discomfort.

This research project has been approved by the Lakehead University Senate Research Ethics Board. Only Dr. Hayman and I will have access to the information you provide. Any information that is obtained in connection with this study and that can be identified with you, will remain confidential and will only be published as composite data. Your responses to questionnaires will not be identified by your name. The data you supply will only be identified by number. When the study is completed, all data will be securely stored for five years. A summary of findings will be available to those interested via e-mail.

Participation in this research study is voluntary. If for any reason you wish to withdraw from the study, you may do so at any time without penalty. In addition, you are not obligated to answer any question you do not wish to answer. Please do not hesitate to contact me at tcnewman@lakeheadu.ca or Dr. Hayman (343-8480) should you have any questions or concerns about this study. You may also contact the Lakehead University Research Ethics Board at 343-8283.

Thank you,

Thomas Newman
Ph.D. graduate student, Clinical Psychology
Department of Psychology, Lakehead University

Dr. Gord Hayman,
Ph.D.

Participant Consent Form

My signature on this form indicates that I agree to participate in the study investigating the effect of various exercise intensities on general mood state. This study is being conducted by Thomas Newman, in the Department of Psychology, for his Ph.D. dissertation under the supervision of Dr. Gordon Hayman (343-8480). I understand that my participation in this study is conditional on the following:

1. I have read the cover letter and I fully understand what I will be required to do as a participant in the study.
2. I agree to participate in the study.
3. I understand that the associated risks involved in the study are exercising on a treadmill at a mild or moderate pace for six 20 minute sessions.
4. I am a volunteer and may withdraw from the study or choose not to answer any question at any time without penalty.
5. My data will remain confidential and will be securely stored for a period of five years.
6. I will remain anonymous in any publication/public presentation of research findings.
7. I may receive a summary of the project, upon request, following the completion of the project.

_____ _____

Name of Participant (please print) Date

_____

Signature of Participant

_____

Email Address (please print clearly)

Appendix F

Screening/Demographic Questionnaire

Screening/Demographic Questionnaire

If you are interested in participating in this research study in the Department of Psychology, please answer the following questions. Participants will be selected based upon the answers they provide to this brief questionnaire.

Name: _____ Date of birth: _____

Email: _____ Phone number: _____

How do you prefer to be contacted? Email □ Phone □

What is your preferred time to be contacted? Day/time_____

Gender: Male_____ Female_____

Height: _____

Weight: _____

What is your ethnic background?

Caucasian □ South Asian □ Hispanic □ African-Canadian □ European □

Native-Canadian □ East Asian □ Other (please specify):_____

1.a) Are you currently taking any prescription medication for a mental health disorder.

YES or NO

1. b) if you answered yes to question 1a), have you had any changes made to your medication in the past three months?

YES or NO

2. Are you currently receiving psychotherapy for any mental health disorders?

YES or NO

3. How many times per week do you engage in regular aerobic exercise (i.e., 30 minutes or more of specific aerobic exercise)?

No times per week Once per week More than once per week

Appendix G

Debriefing Letter

Debriefing Letter

You are receiving this letter as a result of your participation in the exercise study I conducted. The purpose of this letter is to provide you with the results of the study as well as to reveal the true nature of the study.

Currently in the research literature there is much debate as to why exercise is able to decrease anxiety in both clinical and non-clinical populations. Two of the proposed mechanisms involved are the expectancy effect and perceived effort. The expectancy effect refers to the fact that when individuals are exercising they 'expect' that their mood will improve, and anxiety and depression will decrease, and thus it does decrease. Likewise, another hypothesis is that if participants feel they are working harder (i.e., perceived effort) they too will experience greater improvements in mood and anxiety.

The true purpose of this study was to determine if expectancy or perceived effort moderated the effects of exercise on anxiety sensitivity. In order to ensure the integrity of the study participants were informed that the purpose of the study was to assess the effects of exercise on general mood states. This was done so as not to prime participants about expectancy or perceived effort which may have confounded the results. I was also interested in the differences between low and moderate exercise. As a result participants who scored higher on a measure of anxiety sensitivity were recruited for this study, and three experimental groups were used. The two exercise groups exercised at a mild or moderate pace for 20 minutes during six scheduled e sessions, while the quiet rest group sat quietly for 20 minutes during six sessions. It was hypothesized that participants who had increased expectations about the effectiveness of the treatment group would experience greater reductions in anxiety sensitivity. In addition it was hypothesized that participants who perceived that they worked harder (as measured by perceived effort) would also experience greater reductions in anxiety sensitivity. Finally, it was expected that participants in the quiet rest group would experience no significant change in anxiety sensitivity. Data analysis revealed that ….

Should you have any questions or concerns please do not hesitate to contact me by e-mail at tcnewman@lakeheadu.ca, any feedback is appreciated. Once again, thank you for your time, and effort in this study. One name will be chosen at random to win the $250.00 Visa ™ gift card and that person will be notified in a separate e-mail to maintain confidentiality.

Sincerely

Tom Newman