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Evidence for exercise therapy in the treatment of depression and anxiety

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Abstract

There has been over 25 years of systematic investigation examining the relationship between physical activity, especially aerobic exercise and weight training, and depression and anxiety. In this chapter, we first present the findings of the two most recent meta-analyses in the area of physical activity and depression and anxiety. Potential mechanisms by which exercise may reduce depression and anxiety are described. Depression and anxiety are associated with a high incidence of comorbid somatic illnesses, especially cardiovascular diseases and type 2 diabetes. Positive effects of regular exercise on these diseases in individuals with depression and anxiety disorders are discussed. Finally, we offer evidence based recommendations for good clinical practice.

Introduction

Depression

Depression is a common mental disorder that presents with depressed mood, loss of interest or pleasure, feelings of guilt or low self-worth, disturbed sleep and appetite, low energy, and poor concentration. The diagnostic criteria for major depressive disorder following the American Psychiatric Association (2004) are presented in Box I.

Five (or more) of the following symptoms have been present during the same 2-week period and represent a change from previous functioning; at least one the symptoms is either (1) depressed mood or (2) loss of interest or pleasure.

(1) Depressed mood, nearly every day during most of the day
(2) Marked diminished interest or pleasure in almost all activities
(3) Significant weight loss (when not dieting), weight gain, or a change in appetite
(4) Insomnia or hypersomnia (excess sleep)
(5) Psychomotor agitation or psychomotor retardation
(6) Fatigue or loss of energy
(7) Feelings of worthlessness or inappropriate guilt
(8) Impaired ability to concentrate or indecisiveness
(9) Recurrent thoughts of death, recurrent suicidal ideation

Depression is a widespread and often chronic condition. Lifetime prevalence estimates for major depressive disorder are approximately 15% to 20%; 1-year prevalence estimates are 5% to 10%. Moreover, depression is characterized by high rates of relapse: 22% to 50% of patients suffer recurrent episodes within 6 months after recovery (World Health Organization, 2004).

In 2000, depression was the leading cause of disability as measured by Years Lived with Disability and the 4th leading contributor to the global burden of disease. The World Health Organization expects that depression by the year 2020 depression will reach the second place on the ranking of Disability Adjusted Life Years calculated for all ages, including both sexes (World Health Organization, 2004).

The American Psychiatric Association (2004) describes several other mood disorders from which major depressive disorder should be distinguished. Dysthymic disorder is a mild, but chronic depression, lasting at least two years but usually longer. People with dysthymia may subsequently develop an episode of major depressive disorder. Bipolar disorder (manic depression) is characterized by a chronic dysregulation of mood, with fluctuations between low mood (i.e. depression) and elevated mood (i.e. mania or hypomania). The American Psychiatric Association (2004) distinguishes two forms: bipolar type 1 with episodes of mania and type 2 with hypomanic episodes. Manic episodes are typically associated with behavioural activation (e.g. increased goal-directed activity, decreased need for sleep) and altered cognitive functioning (e.g. racing thoughts, distractibility, grandiose delusions). The symptoms of hypomania are less severe.

Exercise as intervention for depression and depressive symptoms: findings of the most recent meta-analysis

There is growing evidence that exercise may be an effective therapy for mild to moderate depression and a valuable complementary therapy to the traditional treatments for severe depression. Several meta-analyses examined the effect of exercise on exercise, however, some meta-analyses have been criticized for including studies of poor methodological integrity and lacking analysis of moderating variables. The most recent meta-analysis of Rethorst, Wipfli, and Landers (2009) addresses this criticism by including only randomized control trials and analyzing moderating variables (i.e. population characteristics and exercise program characteristics). This meta-analysis examines the effects of exercise on depression/depressive symptoms in 58 randomized controlled trials (n = 2982). An overall effect size of -0.80 (large effect) indicates that participants in the exercise treatment had significantly lower depression scores than those receiving the control treatment or no-treatment.

Seventeen studies examined the effect of exercise on clinically depressed individuals (n = 547). Clinically depressed participants in the exercise treatment had significantly lower depression scores than those in no-treatment or wait-list control condition (effect size = -1.03, large effect). Four studies compared exercise treatment with psychotherapy in clinically depressed people, resulting in an overall effect size of -0.26, indicating that exercise resulted in larger antidepressant effects than psychotherapy. However, this difference was not significant. Three studies compared exercise versus antidepressant medication and found an overall equal effect. Nine studies investigated the clinical significance improvement of exercise groups; six of the 9 groups were classified as 'recovered' at post-treatment and two groups as 'improved' and only one group as 'unchanged'. Forty one studies investigated effects of exercise intervention in non-clinically depressed samples, participants in the exercise condition had significantly lower depression score than those in non-treatment control conditions (effect size = -0.59, medium effect). The overall effect size of -0.80 (large effect) of both clinically and non-clinically depressed samples provides a strong evidence for the effects of exercise upon depression. To evaluate the efficacy of exercise in the treatment of major depression and depressive symptoms, it is useful to examine the criteria for grading treatment recommendations that have been refined by the American College of Chest Physicians Task Force (Guyatt et al., 2006). Based upon these criteria, a treatment that achieves Level 1, Grade A status receives the highest level of recommendation. In order for evidence to reach Level 1, Grade A status, the following criteria must be met: the evidence must come from randomized controlled trials with a large sample size; the results must be clear consistently; and there must be a high benefit-to-risk ratio. By including only randomized controlled trials with a cumulative sample of nearly 3000 participants, the results of this meta-analysis provide Level 1, Grade A of recommendation. In an earlier meta-analysis (Wipfli, Rehorst, & Landers 2009), the same authors also analysed the moderating variables of exercise programs (i.e. duration, exercise type, frequency and intensity). Within the overall population, exercise interventions lasting 4-9 weeks resulted in greater improvements in depressive symptoms than interventions with shorter or longer duration. However, within the clinically depressed population, programs lasting 10 – 16 weeks resulted in larger effects than shorter interventions. The exercise programs that combined aerobic and resistance exercise resulted in larger effects than aerobic or resistance training alone. In the overall population, exercise with a frequency 3 or 4 times per week resulted in a significantly larger effect than programs with 2 or 5 exercise sessions per week. Within the overall population, exercise bouts of 20 – 29 minutes resulted in larger effects than bouts of ≥ 45 minutes, while within the clinically depressed population, exercise bouts of 45 – 59 minutes resulted in larger effects than bouts of 30 – 44 minutes and of ≥ 60 minutes.

The aetiology of bipolar disorder is considered to be to primarily biological in nature (Barbour, Edenfield, & Blumenthal, 2007). Therefore, psychopharmacologic intervention is the golden standard treatment, paired with psychotherapeutic interventions (e.g. cognitive behavioural therapy). However, adjunctive therapies that target symptoms of the disorder and relapse prevention are also beneficial. The potential impact of exercise on mood fluctuation and stress adaptation responses in bipolar disorder has not been widely studied. Only two studies investigated the effects of walking; in the walking program of Edenfield (2007), 30 minutes 4 times per week for 4 weeks, resulted an increase in the use of adaptive coping strategies to stressful life events in bipolar patients. In the non-random controlled trial of Ng, Dodd, and Berk (2007), the walking group showed significantly lower scores than the non-walking group for the self-reported 21-item Depression Anxiety Stress Scales and all its subscales (i.e. Depression, Anxiety, Stress).

Summary and conclusions for clinical practice

The strong evidence for exercise upon depression supports the use exercise in the treatment of major depression.

Clinically depressed individuals show greater improvements than non-clinically depressed people.

Within the clinically depressed population exercise treatment seems to be at least equally effective than antidepressant medication and psychotherapy.

Within the clinically depressed population, interventions lasting 10 – 16 weeks result in larger effects than interventions lasting 4 – 9 weeks; and exercise bouts of 45 – 59 minutes produce larger effects than bouts of 30 – 44 minutes and of ≥ 60 minutes.

Within the overall population (i.e. both clinically depressed and non-clinically depressed people) exercise programs that combined aerobic and resistance exercise resulted in larger effects than aerobic or resistance training alone; and programs with a frequency 3 or 4 times per week in a significantly larger effect than programs with 2 or 5 exercise sessions per week.

The impact of exercise in bipolar disorder has not been widely studied. Two studies report beneficial effects of walking programs on adaptive coping strategies to stressful life events, and symptoms of depression, anxiety and stress.

Anxiety

Anxiety disorder is a blanket term covering several different forms of abnormal and pathological fear and anxiety. The anxiety response consists of several clusters of symptoms, including cognitive (worry, apprehension, fear of failure and future consequences), emotional (negative affect), behavioural (nervousness, exaggerated mannerisms, tics), and physiological (increases in heart rate, blood pressure, muscle tension, perspiration, stress hormone levels) symptoms. The American Psychiatric Association (2004) recognizes a wide variety of anxiety disorders. Generalized anxiety disorder, one of the most common anxiety disorders, is characterised by experience of excessive worry in a number of life domains (e.g. family, work) which are difficult to control. The worry is associated with insomnia, tension, and restlessness. People with panic disorder experience sudden, unexpected periods of extreme fear known as panic attacks. Some symptoms of panic attack include sweating, heart palpitations, feeling chocking, dizziness, and fear to dying. Obsessive compulsive disorder is typified by the presence of disturbing and uncontrollable thoughts (obsessions). To elevate these thoughts, individuals use repetitive behaviours (compulsions) in an effort to prevent or reduce distress or prevent some dreaded event or situation (e.g. excessive hand washing to prevent contamination). Post traumatic stress disorder arises in response to experiencing or witnessing a traumatic event. Although many people exposed to trauma temporarily experience stress-related symptoms, those with post traumatic stress disorder continue to struggle with intrusive thoughts and nightmares, as well as increased arousal (e.g. anger) and avoidance of reminders of the trauma. Social phobia is characterised by an intense fear to make mistakes or looking foolish in public. This fear often leads to avoidance to certain people, places, or social events.

The 1-year and life-time prevalence times for all anxiety disorders are 10.6% and 16.6%, respectively (Somers, Goldner, Waraich, & Hsu, 2006). In addition, anxiety disorders are often accompanied by co-morbid mental disease, including depression (at rate of 75 %) and substance abuse (Wipfli, Rethorst, & Landers, 2008).

Exercise as intervention for anxiety disorders and anxiety symptoms: findings of the most recent meta-analysis

Wipfli, Rethorst, and Landers (2008) analysed the results from 49 randomized controlled trials in the area 'exercise and anxiety' (n = 3566). Forty six studies used subjects with anxiety symptoms (non-clinical population), 3 studies involved patients with anxiety disorders (clinical population). The overall effect size of both clinical and non-clinical population was -0.48 (medium effect), indicating larger reductions in anxiety among exercise groups than no-exercise control groups. The clinical population effect size (-0.52) was greater than the non-clinical population effect size (-0.40). Twenty seven randomized controlled trials (n = 1924) compared exercise treatment to other therapy forms. The overall effect size of -0.19 was significantly different from zero, indicating that exercise is slightly better at reducing anxiety than other therapy forms. Exercise was more effective than stress management education, slightly more effective than group therapy, stretching and yoga, relaxation and meditation, and as effective as cognitive behavioural therapy. Only psychopharmacother-

apy produced a very small greater anxiety reducing effect than exercise. Because only randomized controlled trials were examined, the results provided Level 1, Grade A evidence for using exercise in the treatment of anxiety. The authors also analysed the moderating variables of exercise programs (e.g. frequency, type exercise, acute versus chronic exercise). Exercise programs with a frequency of 3 or 4 times per week resulted in a significantly higher effect sizes than programs with 1 – 2 or 5 exercise sessions per week. Compared to aerobic training, weight training and combination of aerobic and weight training, provided greater effects, however effect sizes of weight training and combined training were based upon only three trials. The American Psychiatric Association (2004) makes a distinction between anxiety as a state and anxiety as a trait. State anxiety is the acute (or short-term) emotional response that follows the appraisal of threat. Trait anxiety is the predisposition to interpret a variety of situations as threatening and to respond to them with increases in state anxiety. The assessment of anxiety in intervention studies usually follows this important theoretical distinction. Thus, studies of the effects of 'acute exercise' (i.e. a single bout of exercise) typically focus on changes in state anxiety, whereas studies of the effects of 'chronic exercise' (i.e. a program of exercise lasting for several weeks or months) typically focus on changes in trait anxiety. By analysing the role of duration of exercise intervention as a moderating variable, Wipfli, Rethorst and Landers (2008) found significantly effect sizes for both acute bouts and exercise interventions lasting 4 to 15 weeks (effect sizes varying from -0.39 to -0.59). The limitation of this meta-analysis is that majority of the studies used non-clinical anxiety individuals, only three studies employed patients with anxiety disorders (clinical population). However, the authors only included studies that were published before January 2006. We found four studies using patients with anxiety disorders published after January 2006.

Some of these studies have focused on individuals diagnosed with panic disorder. Such individuals experience recurrent, unexpected panic attacks. In addition, this disorder is accompanied by anticipatory anxiety regarding the occurrence of episodes of panic, worry regarding the possible implications of panic attacks, and associated behavioural change such as avoidance of anxiety-provoking stimuli. Before 2006, Broocks et al. (1998) compared aerobic exercise with pharmacotherapy (clomipramine) and placebo in 46 patients with moderate to severe panic disorder. At the end of the 10-week treatment period, aerobic exercise and medication significantly reduced panic symptoms relative to placebo; though clomipramine was superior to exercise. Recently, Ströhle et al. (2009) showed that also acute bouts of aerobic exercise at mild to moderate intensity yield an antipanic and anxiolytic response in patients with panic disorder. Two studies investigated the potential impact of aerobic exercise in patients with obsessive compulsive disorder. Abrantes et al. (2009) examined acute benefits of 12-week aerobic training program on depression, anxiety, obsessions and compulsions among patients with obsessive compulsive disorder. After each exercise session, patients reported reductions in depression, anxiety and obsessive and compulsive symptoms. At 6 months follow-up, improvements in obsessive and compulsive symptom severity appeared to persist (Brown et al., 2007). In a randomized controlled trial, Merom et al. (2008) compared effects of home-based walking program added to standard cognitive behavioural therapy with educational sessions added to cognitive behavioural therapy within patients with panic disorders, generalized anxiety disorder and social phobia. After 10-weeks intervention, participants in the walking condition showed greater improvements in depression, anxiety and stress than those in the educational control condition.

Summary and conclusions for clinical practice

There exists evidence for the impact of exercise upon both state and trait anxiety.

Patients with anxiety disorders show greater improvements than individuals with only anxiety symptoms.

Recent studies confirm beneficial effects of both acute bouts of exercise and exercise programs lasting 10 – 12 weeks in patients with panic disorder, obsessive compulsive disorder, generalized anxiety disorder and social phobia.

Exercise is more effective than stress management education, slightly more effective than group therapy, stretching and yoga, relaxation and meditation, and as effective as cognitive behavioural therapy. Only psychopharmacotherapy produces a very small greater anxiety reducing effect than exercise.

Single exercise sessions at moderate intensity result in reductions in state anxiety; however, acute bouts of exercise at high intensity (i.e. above 80% of VO2max) produce significant elevations in state anxiety (Knapen et al., 2009).

Exercise programs with a frequency 3 or 4 times per week result in significantly higher effect sizes than programs with 1 – 2 or 5 exercise sessions per week.

Potential mechanisms of action

Several hypotheses that involve biochemical, physiological, and psychosocial mechanisms have been offered to account for the effect of exercise on depression and anxiety. Despite continuing research that demonstrates the positive effects of exercise on depression and anxiety, no single theory adequately explains how exercise leads to a reduction in depressive and anxiety symptoms. A plethora of physiological and psychological mechanisms have been proposed to explain the interaction between exercise and negative affect. The physiological mechanisms hypothesized include the central monoamines theory (i.e., exercise corrects dysregulation of the central monoamines and serotonin believed to lead to depression and anxiety), as well as the role of the hypothalamic-pituitary-adrenal (HPA) axis (Wipfli, Rethorst, & Landers, 2008; Rethorst, Wipfli, & Landers, 2009). In response to psychological stress experiences, depressed and anxious individuals exhibit an increased secretion of stress hormones from the HPA axis. Regular exercise leads to decreases in the amount of these stress hormones, resulting in lower levels of depression and anxiety. The central premise of these biochemical theories is that exercise acts on the same pathway that antidepressant medications target in the treatment of clinical depression and anxiety. Animal studies have observed increases in neuromodulators, like serotonin and norepinephrine, when the animals exercised. The increased production of neuromodulators caused by exercise is consistent with the target of antidepressant pharmacotherapy. In fact, exercise produces the same neuro-chemical changes that are often targeted by pharmacotherapy (Otto et al., 2007).

In addition to the physiological models, several psychological theories have been proposed to explain the effect that exercise has on depression and anxiety. A first theory, the Exercise and Self-Esteem Model of Sonstroem and Morgan (1989), focuses on the established association between depression and anxiety and negative self-evaluations, including lowered self-esteem and self-efficacy (Fox, 2000). In fact, prospective data suggest that negative self-evaluations may play a causal role in major depression and anxiety disorders. It has therefore been hypothesised that effective depression and anxiety therapies work by improving self-evaluations. Indeed, different studies demonstrated that exercise training resulted in enhanced self-esteem, which was attributed to improved physical self-evaluations (such as exercise self-efficacy and body image). This enhanced self-esteem was accompanied by a decrease in depressive and anxiety symptoms, suggesting that that an enhancement of self-esteem may be responsible for the alleviation of depressive and anxiety symptoms (Blumenthal et al., 1999; Knapen et al., 2005). Also several other psychosocial mechanism have been linked to depression and anxiety (Barbour, Edenfield, & Blumenthal, 2007). For example, a response style that favours distraction from negative emotions (as opposed to rumination or repetitive analytic focus on one's negative feelings) is associated with a more favourable prognosis for depression; exercise may create a break from these unproductive negative thoughts and may be a means of distraction. In addition, exercise may be a form of behavioural activation, which is an important component of cognitive behavioural therapy for depression and anxiety disorders. Other psychological theories focus on different constructs such as intrinsic motivation, self-determination, perception of control over physical and mental health, and exposure to anxiety-related physical sensations in case of panic disorder or high anxiety sensitivity (Brosse, Sheets, Lett, & Blumenthal, 2002; Otto et al., 2007). In general terms, these models focus on a variety of mechanisms that partially explain the link between exercise and reduced depression and anxiety, such as improved accomplishments and self-confidence,

improved self-esteem, positive distraction, social reinforcement, increasing positive coping skills available for use during stressful situations, and exposure in vivo. All these mechanisms seem to be very usefully in clinical practice.

Summary and conclusions for clinical practice

The scientifically evidence supporting the proposed physiological as well as the psychological mechanisms is very different. There are probably several different mechanisms mediating the positive effects of exercise on depression and anxiety. Different mechanisms may work for different people and several mechanisms may operate interactively (Otto et al., 2007; Rethorst, Wipfli, & Landers, 2009). Thus, it is difficult to separate physiological mechanisms from psychological explanations. In an analysis of the possible mechanisms and their interactions, Biddle and Mutrie (2001) propose that for people just starting exercise programs, greater emphasis should be placed on the psychological mechanisms since the exerciser had not adapted, physiologically, to the exercise stimulus. In the maintenance phase, Biddle and Mutrie (2001) suggest that both psychological and physiological mechanisms are likely to be important, and in the final habituation phase, they suggest that emphasis should be placed on the physiological mechanisms and the influence of behavioural conditioning.

Psychological models/mechanisms, such as the Exercise and Self-Esteem Model, distraction from negative emotions, behavioural aviation, intrinsic motivation, self-determination, perception of control over physical and mental health, and exposure to anxiety-related bodily sensations, provide therapists very useful therapeutic strategies for clinical practice.

Depression, anxiety disorders and physical health

Depression and anxiety disorders are associated with a high incidence of co-morbid somatic illness. Individuals suffering from major depression and/or anxiety disorders run a higher relative risk of coronary heart disease and type 2 diabetes compared with the general population (Huang, Su, Chen, Chou, & Bai, 2009). In general, depressed and anxious individuals exhibit a less active life-style and have a reduced cardio-respiratory fitness in comparison with the general population (Zoeller, 2007). Strong evidence demonstrates that lack of physical activity is associated with an unhealthier body mass and composition, and a biomarker risk profile for cardiovascular disease and type 2 diabetes. In addition to this less active life-style, people with depression and/or anxiety disorders are more likely to smoke and have a higher risk for development of overweight and obesity (Zoeller, 2007). However, the association between cardiovascular disease, diabetes and depression and anxiety disorders is bilateral, i.e. patients with anxiety and depression experience cardiovascular events more frequently, and the patients with type 2 diabetes and cardiovascular diseases suffer more frequently from anxious-depressive disorder.

Summary and conclusion for clinical practice

Depression and anxiety disorders are independent risk factors for cardiovascular disease and type 2 diabetes. Individuals suffering from both major depression and generalized anxiety disorder show the highest risk for cardiovascular disease.

Exercise is extremely powerful in preventing cardiovascular disease and type 2 diabetes.

Exercise is an outstanding opportunity in treatment of patients who have a mix of mental and physical health problems and; exercise it is a holistic care option (Zoeller, 2007).

Depression, anxiety disorders and cognitive functioning

Depression and anxiety disorders are associated with declined cognitive functioning. Being depressed and anxious is accompanied by slower information processing, psychomotor retardation, and poor memory functioning. There is growing evidence that exercise has a beneficial impact on cognitive function in older adults (Dishman et al., 2006). Positive effects of regular aerobic and resistance training on cognition have been observed in studies among subjects with and without cognitive decline (Erickson & Kramer, 2009; Liu-Ambrose & Donaldson, 2009). The positive effects on cognition occur generally, with the largest effects on cognitive speed, auditory and visual attention. Chronic exercise increases the expression of brain growth factors and may have neurogenerative and neuroprotective influences on the brain by stimulating the growth and development of new cells (Cotman, Berchtold, & Christie, 2007).

Exercise training not only improves negative affect and physical health in depressed and anxious individuals, but also produces 'positive side effects' on depression and anxiety associated cognitive decline.

Evidence based recommendations for clinical practice

In this last section, we offer some recommendations for physical fitness assessment and exercise prescription, and strategies for improving patient's motivation and adherence to exercise.

Physical fitness assessment and exercise prescription

Developing an exercise prescription for people with depression and anxiety disorder differs from the prescription used for healthy individuals. Therapists should be aware, that several characteristics of depression and anxiety disorders (i.e. loss of interest, motivation and energy, generalised fatigue, a low self-worth and self-confidence, fear to move, and social fear) and physical health problems interfere with participation in exercise.

Designing well-considered exercise programs for these patients requires (1) assessment of physical fitness and the perceived exertion during exercise, (2) a risk stratification for patients with comorbid somatic disease, (3) an inventory of the perceived barriers and benefits towards exercise participation.

Assessment of physical fitness and the perceived exertion during exercise

Direct measurement of maximal oxygen intake by way of a maximal exercise test is the most accurate indicator of cardio-respiratory fitness (American College of Sports Medicine, 2009). Maximal tests, however, have the disadvantage of requiring the subject's optimal motivation to work to 'near exhaustion', and require the supervision of a physician and the use of expensive equipment. For depressed and anxious patients, however, submaximal measures are highly recommended for the reasons that many patients have poor physical health, low levels of fitness and physical self-worth, few experience with aerobic training, and less energy and motivation for heavy physical effort (Knapen, Van de Vliet, Van Coppenolle, Peuskens, & Pieters, 2003). Salmon pointed out that, especially in this population, physiological measurements studied in a laboratory could be influenced due to pre-test anxiety (Salmon, 2001). Patients with an increased trait/state anxiety, for example, might fear that maximal aerobic effort will provoke physiological reactions such as hyperventilation, tachycardia, dizziness or sweating, which they associate with symptoms of panic attacks (Ströhle et al., 2009). These clinical considerations usually lead to the application of submaximal exercise tests in psychiatric settings. At the University Psychiatric Centre KULeuven, Campus Sint-Jozef Kortenberg, the six-minute walk test (Vancampfort et al., in press) and the Franz ergocycle test (Knapen, Van de Vliet, Van Coppenolle, Peuskens, & Pieters, 2003) are most commonly used.

For patients with depression and anxiety disorders who often suffer from fatigue and low motivation, the rate of perceived exertion during physical activity is an important parameter when designing an appropriate exercise program (Knapen, Van de Vliet, Van Coppenolle, Peuskens, & Pieters, 2003). The fatigue and recovery time after an effort are not only dependent upon physiological stressors (intensity, duration, and frequency of

the training stimulus) but also upon psychosocial factors. Psychological and social problems cause considerable stress. Generalised fatigue and lack of energy are typical symptoms of depressive syndrome. The exercise tolerance of anxious patients is reduced due to the fact that they are preoccupied with physiological reactions during effort such as palpitations, perspiration, and hyperventilation. These psychological factors cannot be ignored when developing a well-designed fitness program. The evaluation of degree of perceived exertion can be derived from the psychophysiological concept of Borg (Borg, 1998). The Borg 15 Graded Category Scale and the Borg Category Ratio 10 Scale quantify the sensations that the subject experiences during physical effort. The Borg 15 Graded Category Scale has a score range from 6 to 20 (15 grades), and the Borg Category Ratio 10 Scale from 0 to 10 (10 grades). Both scales show a linear relationship with heart rate during progressive incrementally exercise ($r = 0.94$ and $r = 0.88$, respectively). At the University Psychiatric Centre KULeuven, Campus Sint-Jozef Kortenberg, we use the Borg Category Ratio 10 Scale because the longer Borg 15 Graded Category Scale requires a greater differentiation capacity.

Risk stratification for patients with comorbid somatic disease

Before initial treatment, the clinician should identify high-risk individuals, such as patients with a history of cardiovascular disease or diabetes (American College of Sports Medicine, 2009). These patients should be medically cleared before beginning physical activity. For the vast majority of people, the risk of sudden cardiac events is minimal, as long as they start at a realistic pace. Low intensity physical activity is related to a low risk. For example, a walking program at light to moderate intensity is safe for most patients. Intensity can be increased over time, and the patient and therapist should pay attention to symptoms such as chest pain or shortness of breath. Besides, the moderate training stimulus should be adapted to the training status and side effects of psychotropic medication (such as constipation, dizziness, dry mouth, nausea, sweating and tremor).

Inventorying perceived barriers and benefits towards exercise participation

Depressed and anxious patients accumulate a lot of barriers for participation in exercise such as a low self-worth and self-confidence, loss of energy, interest and motivation, generalised fatigue, weak physical fitness and health condition, fear to move, social fear, overweight and a low feeling of personal control concerning own fitness and health, and helplessness and hopelessness. For that reason, it is highly recommended to have a conversation before starting an exercise program concerning barriers and possible strategies that assist a patient in overcoming these barriers (e.g. problem solving, planning activity, seeking social support). Furthermore, giving information regarding both mental and physical health benefits of regular physical activity and determining which benefits are most salient to each patient, is essential. For inventorying of perceived barriers and benefits therapists may use a decision balance that patients helps to reflect the relative weighing of the pros and cons of exercise participation (Marshall & Biddle, 2001).

Strategies for improving motivation and adherence to exercise

Strategies can be based on the ideas of Motivational Interviewing following Miller and Rollnick (2002), and the Transtheoretical Model of Behaviour Change (Prochaska & Velicer, 1997; Marshall & Biddle, 2001). This model postulates that exercise behaviour change involves progress through six stages of change: precontemplation, contemplation, preparation, action, maintenance, and termination.

Initial phase: starting with supervised exercise

- Create exercise programs based on the patient's current preferences and expectations, the initial physical fitness assessment and the measurement of perceived exertion during exercise.
- Draw up an individual plan with the patient taking into account emotional, cognitive and physiological components of depression and anxiety.

- Help the patient set realistic and achievable goals which lead to success experiences; this generally gives courage to persevere.

- Adapt the moderate exercise stimulus to the individual's physical abilities, training status, expectations and goals, side effects of psychotropic medication, exercise tolerance and perceived exertion.

- Follow the program with exercise cards and a logbook and provide regular progress feedback to the patients.

- Avoid between-patient comparisons.

- Emphasize the short-term benefits after single exercise sessions: improvements in mood and state anxiety, stress level, energy level, distraction of negative thoughts, the ability to concentrate and focus, and quality of sleep. Many patients are focused on the distant outcomes, such as weight loss and improved self-worth, so emphasizing short-term benefits can help patients adhere exercise participation.

- Empathy, validation, praise and encouragement are necessary during all phases but especially when patients struggle with ambivalence and doubt their ability to accomplish the change.

Second phase: maintaining supervised exercise

- Focus on perceived fitness gains, achievement of personal goals, mastery experiences and sense of control over the body and its functioning.

- Use cognitive-behavioural strategies such as self-monitoring, stimulus cuing, goal-setting and contracting.

- Once patients begin to feel better as a result of exercise, they are eager to continue their exercise if the therapist can help them attribute their improved mood to the exercise regimen. Improved mood as a result of increased physical activity may be obvious to the therapist, but the connection is not always obvious to the patient. Exercise can give patients a sense of power over their recovery, which in itself counteracts the feelings of hopelessness often experienced in depression and anxiety.

- Reinforcing the connection between mood and anxiety change and exercise is particularly relevant for patients with panic disorder. The prescription for exercise for panic disorder requires patients to engage in progressive more intensive exercise in order to elevate their heart rates so that they can become accustomed to sensations that will help them overcome their fear of panic.

- Self-determined motivation towards exercise is very important and results in adaptive exercise-related behaviours, cognitions and physical self-evaluations. Therefore, it is important to make physical activity as self-determined as possible by focusing on the positive experiences of the activity itself, as well as helping to develop an identity of a physical active person.

Third phase: follow-up after supervised exercise

- Follow-up contact is very important: discuss problem-solving around barriers, reinforce all progress toward change (even if initially very small progress), and encourage modification of goals as needed.

- Seek support of others such as family and friends.
- Use relapse behaviours/strategies: it is important to explain to patients that relapses are part of the process of change, and that responding with guilt, frustration, and self-criticism may decrease their ability to maintain physical activity. Relapse prevention strategies such as realistic goals setting, planned activity, realistic expectations, identifying and modifying negative thinking, and focusing on benefits of single exercise sessions seem to be effective.

References

Abrantes, A., Strong, D., Cohn, A., Cameron, A., Greenberg, B., Mancebo, M., et al. (2009). Acute changes in obsessions and compulsions following moderate-intensity aerobic exercise among patients with obsessive-compulsive disorder. *Journal of Anxiety Disorders, 23*, 923–927.

American College of Sports Medicine. (2009). *A.C.S.M.'s guidelines for exercise testing and prescription* (8th ed.). Baltimore: Williams & Wilkins.

American Psychiatric Association. (2004). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: American Psychiatric Association.

Barbour, K., Edenfield, T., & Blumenthal, J. (2007). Exercise as a treatment for depression and other psychiatric disorders: a review. *Journal of Cardiopulmonary Rehabilitation and Prevention*, *27*, 359–367.

Biddle, S., & Mutrie, N. (2001). *Psychology of physical activity. Determinants, well-being and interventions*. London: Routledge.

Blumenthal, J., Babyak, M., Moore, K., Craighead, W., Herman, S., & Khatri, P., et al. (1999). Effects of exercise training on older patients with major depression. *Archives of Internal Medicine, 159*, 2349–2356.

Borg, G. (1998). External, physiological and psychological factors and perceived exertion. In G. Borg (Ed), *Borg's perceived exertion and pain scales* (pp. *68*–74). Champaign, IL: Human Kinetics.

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

Brosse, A., Sheets, E., Lett, H., & Blumenthal, J. (2000). Exercise and the treatment of clinical depression in adults: Recent findings and future directions. *Sports Medicine, 32*, 741–760.

Brown, R., Abrantes, A., Strong, D., Mancebo, M., Menard, J., Rasmussen, S., et al. (2007). A pilot study of moderate-intensity aerobic exercise for obsessive compulsive disorder. *Journal Nervous and Mental Disease, 195*, 514–520.

Cotman, C., Berchtold, N., Christie, L. (2007). Exercise builds brain health: key roles of growth factor cascades and inflammation. *Trends in Neurosciences*, *30*, 464–472.

Dishman, R., Berthoud, H., Booth, F., Cotman, C., Edgerton, V., Fleshner, M., et al. (2006). Neurobiology of exercise. *Obesity, 14*, 345–356.

Edenfield, T. (2007). Exercise and mood: Exploring the role of exercise in regulating stress reactivity in bipolar disorder. Unpublished doctoral dissertation. Orono: University of Maine.

Erickson, K., & Kramer, A. (2009). Aerobic exercise effects on cognitive and neural plasticity in older adults. *British Journal of Sports Medicine, 43*, 22–24.

Fox, K. (2000). Self-esteem, self-perceptions and exercise. *International Journal of Sport Psychology, 31*, 228–240.

Guyatt, G., Gutterman, D., Baumann, M., Addrizzo-Harris, D., Hylek, E., Phillips, B., et al. (2006). Grading strength of recommendations and quality of evidence in clinical guidelines: report from an American college of chest physicians task force. *Chest, 129*, 174–181.

Huang, K., Su, T., Chen, T., Chou, Y., & Bai, Y. (2009). Comorbidity of cardiovascular diseases with mood and anxiety disorder: A population based 4-year study. *Psychiatry and Clinical Neurosciences, 63*, 401–409.

Knapen, J., Sommerijns, E., Vancampfort, D., Sienaert, P., Pieters, G., Haake, P., et al. (2009). State anxiety and subjective well-being responses to acute bouts of aerobic exercise in patients with depressive and anxiety disorders. *British Journal of Sports Medicine, 43*, 756–759.

Knapen, J., Van de Vliet, P., Van Coppenolle, H., David, A., Peuskens, J., Pieters, G., et al. (2005). Comparison of changes in physical self-concept, global self-esteem, depression and anxiety following two different psychomotor therapy programs in non-psychotic psychiatric inpatients. *Psychotherapy and Psychosomatics, 74*, 353–361.

Knapen, J., Van de Vliet, P., Van Coppenolle, H., Peuskens, J., & Pieters, G. (2003). Evaluation of cardio-respiratory fitness and perceived exertion for patients with depressive and anxiety disorders: A study on reliability. *Disability and Rehabilitation, 25*, 1312–1315.

Knol, M., Twisk, J., Beekman, A., Heine, R., Snoek, F., & Pouwer, F. (2006). Depression as a risk factor for the onset of type 2 diabetes mellitus. A meta-analysis. *Diabetologia, 49*, 837–845.

Lett, H., Blumenthal, J., Babyak, M., Sherwood, A., Strauman, T., & Robins, C. (2004). Depression as a risk factor for coronary artery disease: evidence, mechanisms, and treatment. *Psychosomatic Medicine, 66*, 305–315.

Liu-Ambrose, T., & Donaldson, M. (2009). Exercise and cognition in older adults: is there a role for resistance training programmes? *British Journal of Sports Medicine, 43*, 25–27.

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

Merom, D., Phongsavan, P., Wagner, R., Chey, T., Marnane, C., Steel, Z., et al. (2008). Promoting walking as an adjunct intervention to group cognitive behavioral therapy for anxiety disorders: A pilot group randomized trial. *Journal Anxiety Disorders, 22*, 959–968.

Miller, W., & Rollnick, S. (2002). *Motivational interviewing: Preparing people for change*. New York: Guilford Press.

Ng, F., Dodd, S., & Berk, M. (2007). The effects of physical activity in the acute treatment of bipolar disorder: A pilot study. *Journal of Affective Disorders*, *101*, 259–262.

Otto, M., Church, T., Craft, L., Greer, T., Smits, J., & Trivedi, M. (2007). Exercise for mood and anxiety disorders. *Journal Clinical Psychiatry, 9*, 287–294.

Prochaska, J., & Velicer, W. (1997). The transtheoretical model of health behavior changes. *American Journal of Health Promotion, 12*, 38–45.

Rethorst, C., Wipfli, B., & Landers, D. (2009). The antidepressive effects of exercise: A meta-analysis of randomized trials. *Sports Medicine*, *39*, 491– 511.

Rugulies, R. (2002). Depression as a predictor for coronary heart disease. A review and meta-analysis. *American Journal of Preventive Medicine, 23*, 51–61.

Salmon, P. (2001). Effects of physical exercise on anxiety, depression, and sensitivity to to stress: A unifying theory. *Clinical Psychology Review, 21*, 33–61.

Somers, J., Goldner, E., Waraich, P., & Hsu, L. (2006). Prevalence and incidence studies of anxiety disorders: A systematic review of the literature. *Canadian Journal of Psychiatry*, *51*, 100–103.

Sonstroem, R., & Morgan, W. (1989). Exercise and self-esteem: Rationale and model. *Medicine in Science & Sports Exercise, 21*, 329–337.

Ströhle, A., Graetz, B., Scheel, M., Wittmann, A., Feller, C., Heinz, A., et al. (2009). The acute antipanic and anxiolytic activity of aerobic exercise in patients with panic disorder and healthy control subjects. *Journal of Psychiatric Research, 43*, 1013–1017.

Vancampfort, D., Sweers, K., De Hert, M., Probst, M., Knapen, J., van Winkel, R., et al. (in press). De test - hertest betrouwbaarheid van de 6-minuten wandeltest bij personen met schizofrenie (The test - retest reliability of the six-minute walk test in persons with schizophrenia). *Actuele themata uit de psychomotorische therapie*. Leuven: Acco.

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

World Health Organization. (2004). *Prevention of mental disorders: Effective interventions and policy option: Summary report*. Geneva: World Health Organization.

Zoeller, R. (2007). Physical activity: depression, anxiety, physical activity, and cardiovascular disease: What's the connection? *American Journal of Lifestyle Medicine, 1*, 175–180.

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