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## Review Article

### The Effects of Exercise on the Mental Health

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#### ABSTRACT

Anxiety and depression are the most frequently diagnosed psychological diseases showing a high co-morbidity. They have a severe impact on the lives of the persons concerned. The physical, mental and social benefits of exercise have been well-documented. In Iran, mental disorders are one of the largest sources of disease burden. The aim of this paper is to critically review the currently available literature with respect to the association of physical activity, exercise and the prevalence and incidence of depression and anxiety disorders

### 1. Introduction

The physical benefits of exercise are numerous, and affect multiple body systems. The most obvious benefit is the decreased body fat and increased lean body tissue. However, physical exercise has benefits far beyond body composition; it has been shown that there is a direct relationship between physical inactivity and cardiovascular mortality, and that physical inactivity in itself is an independent risk factor for the development of coronary artery disease (Powell, et al., 1987). Aside from the physical benefits of exercise, there are also numerous mental benefits. Individuals who exercise regularly tend to be better adjusted and perform better on tests of cognitive functioning (Eysenck, et al., 1982).

Mental disorders constitute a huge social and economic burden for health care systems worldwide (Wittchen, 2011). The population impact of mental disorders is principally on disability rather than on mortality. On a neurochemical and physiological level, a number of acute changes occur during and following bouts of exercise. Different mechanisms of action for Physical activity and exercise have been discussed. For instance, EX has been found to normalize reduced levels of brain-derived

neurotrophic factor (BDNF) and therefore has neuroprotective or even neurotrophic effects (Seifert et al., 2010; Fumoto et al., 2010; Sylvia et al., 2010). Animal studies found EX-induced changes in different neurotransmitters such as serotonin and endorphins (Meeusen et al., 2011). Potential psychological mechanisms of action include learning and extinction, changes in body scheme and health attitudes/ behaviors, social reinforcement, experience of mastery, shift of external to more internal locus of control, improved coping strategies, or simple distraction (Read and Brown, 2003).

In Iran, mental disorders are one of the largest sources of disease burden (Ellis, 2004). For psychotic disorders, antipsychotic medication is a key component of treatment, but needs to be accompanied by psychosocial intervention and practical support (Rhodes, 2011). The present review looks at the evidence for exercise as a treatment option, for some mental disorders.

#### The Effects of Exercise on the Brain

One of the most exciting changes that exercise causes is neurogenesis, or the creation of new neurons. The new

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neurons are created in the hippocampus, the center of learning and memory in the brain (Modie, 2003).

At a cellular level, it is possible that the mild stress generated by exercise stimulates an influx of calcium, which activates transcription factors in existing hippocampus neurons. The transcription factors initiate the expression of the BDNF (Brain-Derived Neurotrophic Factor) gene, creating BDNF proteins that act to promote neurogenesis (Mattson *et al.*, 2004).

### **Relationship Between Exercise and Endorphin Release**

Endorphins are endogenous opioids released from the pituitary gland that are believed to mediate analgesia, induce euphoria, and play a role in the reward system in the brain (Oswald & Wand, 2004). These hormones act similar to morphine (a natural opiate) within the natural opioid system (McKim, 2003). Researchers have found a correlation between vigorous exercise and elevated endorphin levels in blood plasma (Pierce *et al.*, 1993). Evidence showing that endorphins can interfere with the release of other neurotransmitters, including norepinephrine, dopamine, and acetylcholine, have led to a belief that they work by modulating the presynaptic membranes of synapses other than their own (McKim, 2003). Many studies have examined the relationship between exercise and endorphin release, studying the role of these peptides in exercise-induced euphoria as well as the reduction of pain (Goldfarb *et al.*, 1998). Analyses of these data indicated a trend of elevated endorphin levels after exercise in all studies, although not all were significant. Endorphins are known to play a role in depersonalization disorder. The opioid antagonist's naloxone and naltrexone have both been proven to be successful in treating depersonalization. Stress and pain are the two most common factors leading to the release of endorphins. Endorphins interact with the opiate receptors in the brain to reduce our perception of pain and act similarly to drugs such as morphine and codeine. In contrast to the opiate drugs, however, activation of the opiate receptors by the body's endorphins does not lead to addiction or dependence. The release of beta-endorphin into the blood in horses is particularly evident during the course of stress reactions. A study on 42 healthy pure blood stallions was aimed at determining the impact of transport stress on the levels of beta-endorphin, cortisol and ACTH (Fazio *et al.*, 2008). The concentration of beta-endorphin was raised when compared to the basic level only after the distance of 100 kilometers.

### **The Effects of Exercise on Psychological Disorders**

Patients with mental disorders display a high comorbidity of physical conditions such as respiratory, metabolic, cardio-vascular and neurologic diseases (Lin *et al.*, 2011, Scott *et al.*, 2008). Many of the conditions named above are linked to overweight, smoking, and unhealthy lifestyle (Scott *et al.*, 2011); therefore lifestyle

interventions based on nutrition and EX are promising approaches for reducing physical comorbidity (Chacón *et al.*, 2011). Furthermore, psychiatric patients who regularly exercised reported higher health-related quality of life in a cross-sectional study.

### **Anxiety Disorder**

Anxiety disorders are a category of mental disorders characterized by feelings of anxiety and fear. Low levels of GABA, a neurotransmitter that reduces activity in the central nervous system, contribute to anxiety. A number of anxiolytics achieve their effect by modulating the GABA receptors (Lydiard, 2003). Selective serotonin reuptake inhibitors, the drugs most commonly used to treat depression, are frequently considered a first line treatment for anxiety disorders (Dunlop, 2008). Compared to the wide range of research on the positive effects of exercise in major depression, anxiety disorders have been less frequently studied.

However, there is no doubt on the possible anxiolytic effects of aerobic exercise training on healthy volunteers (Long and Satvel 1995). In addition, studies on healthy subjects and two case reports (Orwin 1974; Muller and Armstrong 1975) suggest that an acute bout of exercise is anxiolytic as well. In contrast, exercise may induce acute panic attacks (Broocks *et al.* 1998; Barlow and Craske 1994) or increase subjective anxiety in patients with panic disorder more than in other people.

In patients with high trait anxiety or generalized anxiety disorder, aerobic exercise training was superior to strength and mobility exercises (Steptoe *et al.* 1989) or no treatment and comparable effective as cognitive behavior therapy (McEntee and Halgin 1999).

Case reports (Dractu 2001) and two published clinical studies suggest that exercise training may be used therapeutically in patients with anxiety neurosis (Sexton *et al.* 1989) and panic disorder (Broocks *et al.* 1998). Posttraumatic stress disorder may also respond to exercise training (Manger 2000). Exercise session durations greater than 30 minutes showed larger effects than durations of 10 to 30 minute and similar effects have been found in studies of exercise training effects on cognitive function in older adults (Herring *et al.*, 2010).

It has been shown that stress can reduce the expression of several neurotrophic growth factors in the hippocampus, whereas chronic treatment with antidepressants acts in an opposite manner (Duman and Monteggia, 2006). Protective effects of physical activity from stress have also shown exercise-induced neurogenesis and growth factor expression (Castren *et al.*, 2007) in the hippocampus. Different stressful stimuli as acute immobilization stress (Hölzel *et al.*, 2001) or the administration of corticosterone (Barbany *et al.*, 1992) reduces the expression of neurotrophic factors in the hippocampus. Human studies have demonstrated that impaired BDNF availability is associated with the vulnerability to depressive disorders (Neves-Pereira *et al.*, 2002) and are enhanced after 30

minutes of exercise in panic disorder patients but not in healthy controls (Ströhle *et al.*, 2012). Enhanced expression of cerebral growth factor such as brain derived neurotrophic factor (BDNF), nerve growth factor (NGF), insulin-like growth factor (IGF-1), and vascular endothelial growth factor (VEGF) are most reproducible effects of exercise in the rodent brain inducing exercise-related effects in brain plasticity, function, and health (Bhui *et al.*, 2000). Physical activity therefore counteracts atrophic changes in the hippocampus by its impact on the expression of neurotrophic growth factors acting as an antidepressant and anxiolytic agent similar to that hypothesized for antidepressant drug treatment (Carro *et al.*, 2000). Exercising in a group setting, and in fitness centers, may be effective because it provides social interaction and because participants can continue the pattern of exercise behavior established during the intervention by continuing to work out at similar fitness centres (Conn, 2010). Research on physical activity and anxiety found that interventions were most effective when they were supervised and recommended fitness-center-based activity following the intervention (Conn, 2010).

### Depression

Depression has a huge impact on QoL and mental health, which is easy to understand. What is less well known is that aside from the mental and emotional effects, depressed patients also tend to have heightened somatic symptoms, such as headaches and abdominal pain (Dang, 2010).

A great number of studies suggest that exercise training may reduce depressive symptoms in nonclinical and clinical populations (DiLorenzo *et al.* 1999) and in patients with major depression (Blumenthal *et al.* 1999). However, some studies have reported that supervised exercise training results in larger improvements in functional capacity compared with home-based exercise (Ströhle, 2009). As a treatment for depression, exercise can be easily implemented and used in conjunction with other treatment plans, such as pharmacology or psychotherapy (Dang, 2010). Exercise can also have indirect psychological benefits by increasing self-efficacy and improving self-esteem (Dang, 2010). Although most research has emphasized endurance type exercise, recent findings suggest that including flexibility and resistance exercise may be important for improving depressive symptoms and that no difference between endurance and resistance exercise in major depression symptoms (Conn, 2010).

North *et al.* (1990) showed that exercise training reduced depression scores by approximately One half a standard deviation as compared to the comparison groups. Craft and Landers (1998) reported an effect size of -0.72, showing that only the length of the exercise program was a significant moderator of the clinical effects, with programs of at least 9 weeks being associated with larger reductions in depression. Patient characteristics (age,

gender, severity of depression) were not significant moderators and when compared with standard treatment of depression. A study of Dimeo *et al.* (2001) suggests that in treatment-resistant patients with major depression, 30 min of treadmill walking for ten consecutive days may be sufficient to produce a clinically relevant and statistically significant reduction in depression, as measured with the Hamilton Depression rating Scale. These findings are substantiated by a more recent study involving a placebo exercise group (low intensity stretching and relaxation exercises) in patients receiving a standard antidepressant treatment: the reduction of depression scores and the response rates were larger in the exercise training group Knubben *et al.* (2006).

While most studies employed walking or jogging programs, the efficacy of nonaerobic exercise has also been studied. In depressed elderly, a resistance training program was more effective than the control condition (Singh *et al.* 1997). Blumenthal *et al.* (1999) could show that 16 weeks of group exercise training in older patients with major depression was as effective as antidepressant treatment with sertraline. Additionally, it seems that exercise compares quite favorably with standard psychotherapy of major depression: in the few studies that have evaluated their relative efficacy, running was just as effective as psychotherapy (Greist *et al.* 1979), cognitive therapy or a combination of cognitive therapy and running (Fremont and Craighead 1987).

A dysregulation and an imbalance of monoamines in the central nervous system, especially serotonin, dopamine, and norepinephrine, have been implicated as a major cause of depressive disorders. Exercise has been shown to result in elevated levels of serotonin metabolites, suggesting increased serotonin metabolism from physical activity. Because most antidepressants increase levels of monoamines, it's been hypothesized that people with depression have an imbalance of these monoamines and that correcting this imbalance would improve mood. Many people with depression experience hyperactivity of the stress response, coupled with an overproduction of stress-related hormones such as cortisol. This hypersensitivity to stress has also been implicated as an important factor in depression. Adults who exercise have been found to have an attenuated physiological response to stress and a reduction in stress hormones (Pierce *et al.*, 1993).

Another well-known benefit of exercise is the surge of serum betaendorphins. Beta-endorphin is an endogenous opioid that not only provides an analgesic effect with rigorous exercise but elevates mood. However, studies on the long-term relationship of beta-endorphins to major depressive disorder have been inconclusive. One study revealed a positive correlation between beta-endorphins and depression, yet other studies found no relationship. Depression is a complex disorder that most likely involves multiple biochemical and psychosocial pathways (Pierce *et al.*, 1993).

## Conclusion

The physical, mental and social benefits of exercise have been well-documented. It has been found, however, that exercise training may help improve anxiety symptoms among patients as well as among healthy participants, and that anxiolytic effects may be more pronounced in clinical or subclinical populations. There are more traditional forms of anxiety management available, such as pharmacological and cognitive behavioural therapies, but there continues to be interest in alternative therapies such as relaxation and exercise.

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