

DEPRESSION AND EXERCISE

Mechanisms: How Physical Activity Helps Psychological Health

Cognitive-Behavioral Hypothesis

Individuals who master something that they perceive as difficult (exercising regularly) may experience a positive change in their psychological health manifested by increased self-confidence, improved self-efficacy (an “I can do it” attitude), ability to cope with personal problems, uplifted vigor and general well-being, and lessening of anxiety and depression. On the other hand, some people may simply report feeling better following exercise because they expect such a change (14). In the best-designed studies, however, researchers have tried to control for this by using random assignment, the use of mild calisthenic control groups, and concealing the intention of the study from the subjects (5).

Social Interaction Hypothesis

Exercise is often performed with others, leading to improved opportunities for social interaction, pleasure, and personal attention. It has been hypothesized that this could account for the antidepressant and mood elevation effects of exercise. However, in one meta-analysis, exercise was shown to be a significantly better antidepressant than enjoyable group activities alone (13). In studies that have attempted to control for social interaction, most have concluded that favorable psychological responses to aerobic training were not due to this factor (5).

Time-Out/Distraction Hypothesis

This hypothesis maintains that being distracted from stressful stimuli, or taking a “time-out” from the daily routine, is responsible for the mood elevation seen with exercise (13, 14). However, the evidence suggests that the mood elevation experienced after exercise is due to more than simply taking time out from one’s daily routine. For example, exercise has been found to reduce depression and anxiety more than relaxation (time-out) or enjoyable activities (distraction) (13). Thus, regular exercise may be a more effective long-term mood elevator than habitual relaxation.

Cardiovascular Fitness Hypothesis

According to this theory, mood elevation and a reduction in anxiety and depression are directly related to the level of aerobic fitness. However, several studies have reported that the psychological improvements seen with exercise take place within the first few weeks of treatment before significant increases in aerobic fitness have occurred (13, 5). Also, in some studies, depending on the psychological variable tested, improvement in anaerobic fitness from weight training was just as effective as gains in aerobic fitness (13). There is some thinking that aerobic exercise may increase oxygen transport to the brain and elevate deep body temperature, inducing an elevation in mood state (7). However, the research linking these changes to improved mental health is tenuous at best, and further research is needed before conclusions can be made.

Monoamine Neurotransmitter Hypothesis

Disturbances in the brain secretions of three monoamine neurotransmitters- serotonin, dopamine, and nor-epinephrine- have been implicated in depression and other psychological disorders (8). There is evidence that depressed individuals have decreased secretions of these neurotransmitters, and various medications are used to increase their transmission.

In animal studies, an acute bout of exercise increases both dopamine and nor-epinephrine synthesis and metabolism in various parts of the brain, including the midbrain, cortex, and hypothalamus (11). Exercise could play a role in the treatment and prevention of depression and other mental disorders by promoting optimal neurotransmitter secretions, but this remains uncertain on the basis of unavailable data (13).

Endogenous Opioid Hypothesis

Opiates have been used for centuries to relieve pain and induce euphoria. In 1975, researchers were successful in isolating chemicals from the body that were found to have morphine-like qualities. Since then, many more endogenous opioids have been identified and can roughly be divided into three groups: endorphins, enkephalins, and dynorphins. These endogenous opioids are widely distributed throughout areas of the central nervous system and influence many important systems of the body, including the cardiovascular, respiratory, and immune systems, and metabolism of fuels(17, 15, 6). Of special interest has been the B-endorphin system, which contributes to the regulation of blood pressure, pain perception, and the control of body temperature. B-endorphin has receptors in the hypothalamus and limbic systems of the brain, areas associated with emotion and behavior.

During vigorous exercise, the pituitary increases its production of B-endorphin, leading to an increase in its concentration in the blood. B-endorphin is a late-acting hormone, rising sharply only during and immediately following intense exercise. In a study, the increases in B-endorphin did not differ significantly between athletes and nonathletes when compared on a percent maximum basis. B-endorphin concentrations peaked during recovery at 3-3.5 times resting levels and fell to near-resting levels after 45 minutes of recovery.

Most researchers have found that B-endorphin does not increase unless the exercise intensity exceeds 75% maximum or the duration exceeds 1 hour and the exercise is performed at a steady state between lactate production and elimination (15).

Although it is widely accepted by the exercising public that endorphins are responsible for exercise-induced euphoria, researchers disagree on the interpretation of the available data. At the center of the debate is whether blood concentrations of B-endorphin actually reflect what is happening in the brain's limbic system, where B-endorphin must activate the central nervous system to produce euphoria.

After the B-endorphin is secreted into the blood by the pituitary gland, it apparently is unable to be able to penetrate the blood-brain barrier to get into the brain. As a result, most researchers have been unable to correlate changes in blood B-endorphin concentrations with reduced tension and pain. Studies show, for example, that although subjects can tolerate more pain than normal during and for 15 minutes after intense exercise, plasma B-endorphin levels do not appear to be related (6).

However, there is some evidence from animal studies that brain concentrations of B-endorphin increase during exercise (17). Prolonged, submaximal exercise has been found to increase brain B-endorphin levels and to improve the pain tolerance of rats.

Other animal research suggests that prolonged rhythmic, large-muscle exercise can activate brain opioid systems by triggering certain sensory nerves that go from the muscle to the brain (17). It is uncertain, however, whether the brain is making its own B-endorphin or whether exercise-induced changes enable B-endorphin to cross the blood-brain barrier and pass into the brain (15). Further research is needed to resolve these issues, but there is evidence that intense exercise may activate brain opioid systems, increasing the pain threshold and improving mood state (17).

It is more than likely that both the physiological and the psychological mechanisms reviewed above play a role in explaining the improvements in psychological mood state seen after exercise.

References

1. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders (4th ed) Washington, DC: American Psychiatric Association, 1994.
2. Babyak M, Blumenthal JA et al. Psychosom Med 2000; 62: 633- 638.
3. Blumenthal JA, Babyak MA et al. Effects of exercise training on older patients with major depression. Arch Intern Med 1999; 159: 2349- 2356.
4. Chen MK. The epidemiology of self-perceived fatigue among adults. Prev Med 1986; 15: 74- 81.
5. Cramer SR, Nieman DC, Lee JW. The effects of moderate exercise training on psychological well-being and mood state in women. J Psychosom Res 1991; 35: 437- 449.
6. Droste C, Greenlee MW et al. Experimental pain thresholds and plasma beta-endorphin levels during exercise. Med Sci Sports Exerc 1991; 23: 334- 342.
7. Etnier JL, Landers DM. Brain function and exercise: current perspectives. Sports Med 1995; 19: 81- 85.
8. Forge RL. Exercise-associated mood alterations: a review of interactive neurobiologic mechanisms. Med Exerc Nutr Health 1995; 4: 17- 32.
9. Martinsen EW. Benefits of exercise for the treatment of depression. Sports Med 1990; 9: 380- 389.
10. Martinsen EW. Exercise and mental health in clinical populations. In Biddle SJH (ed). European Perspectives on Exercise and Sport Psychology. Champaign, IL: Human Kinetics, 1995.

11. Mazzeo RS. Catecholamine responses to acute and chronic exercise. *Med Sci Sports Exerc* 1991; 23: 839- 845.
12. McDonald DG, Hodgdon JA. *Psychological Effects of Aerobic Fitness Training*. New York: Springer- Verlag, 1991.
13. North TC, McCullagh P, Tran ZV. Effect of exercise on depression. *Exerc Sport Sci Review* 1990; 18: 379- 415.
14. Petruzzello SJ, Landers DM et al. A meta-analysis on the anxiety-reducing effects of acute and chronic exercise: outcomes and mechanisms. *Sports Med* 1991; 11: 143- 182.
15. Schwarz L, Kindermann W. Changes in beta-endorphin levels in response to aerobic and anaerobic exercise. *Sports Med* 1992; 13: 25- 36.
16. Stephens T. Physical activity and mental health in the United States and Canada: Evidence from four population surveys. *Prev Med* 1988; 17: 35- 47.
17. Thoren P, Floras IS et al. Endorphins and exercise: Physiological mechanisms and clinical implications. *Med Sci Sports Exerc* 1990; 22: 417- 428.
18. Weyerer S. Physical inactivity and depression in the community: evidence from the Upper Bavarian Field Study. *Int J Sports Med* 1992; 13: 492- 496.