

The Mind – Body Connection
Depression and Heart Disease: Deadly Relationship
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Introduction

Increased awareness of the relationship between depression and heart disease promises to provide more successful treatment and better quality of life for patients suffering from both illnesses. It has now been shown that depression has profound implications for vascular health, and treatment protocols should not ignore relevant causal factors.

Recent research indicates that depression should be treated independently of heart disease when both conditions present in the same patient. This is a departure from conventional approaches by the medical community, which has interpreted depressive symptoms as an inevitable side effect of heart disease and has overlooked the importance of treating the depression.

The link between these two diseases goes far beyond the simple notion that if you have suffered a heart attack you are entitled to be a bit depressed. While people with heart disease are more likely to suffer from depression, there is evidence that people with depression are at greater risk of developing heart disease.¹ In middle age depression is one of the strongest independent risk factors for cardiac disease.² Minor depression increases the risk of cardiac death in half. Major depression triples or quadruples the risk. Controlling for other risk factors (smoking, high BP, diabetes) does not change the fact that if you have depression you are five times as likely to die of coronary artery disease than if you don't have depression.³

The research into the precise etiologic biological pathways explaining the co-morbidity of these two common chronic illnesses is not conclusive at this time. What is known, however, is that depression and anxiety disorders may affect heart rhythms, increase blood pressure, and alter blood clotting. They can also lead to elevated insulin and cholesterol levels.⁴ While depression's impact on vascular health is a new finding, it has long been recognized that depression prohibits effective behavioral coping responses. This leads to poor compliance with treatment plans and in turn, poor outcome. Depressives are less able to summon the energy for life-style change and other self-care regimens and consequently are more likely to fail in their cardiac rehabilitation efforts.

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² Jaing, W., et al., Patients with CHF and Depression have the greater risk of mortality and morbidity than patients without Depression. Amer College of Cardiology. 39(2002): 919-21.

³ Wilson, L & Singal, B. Do Depressive symptoms increase the risk for the onset of coronary Disease? A systematic quantitative review. Psychosomatic Med. 65 (2003): 201-10.

⁴ Sapolsky, R. Stress, The Aging Brain, The Mechanism of Neuron Death, 1992.

Given the high incidence of both these medical conditions and the risk of not treating them concurrently, it is critical for the physician to identify patients with heart disease who are showing symptoms of depression and treat them with a multi-disciplined approach.⁵

Against Depression

In his recent book, *Against Depression*, Peter Kramer postulates that depression is the most costly of chronic diseases and describes depression as a “derailing of functions” (p.184) where the regulation of heart rhythms is a bit off, platelets are out of control, and as a result, strokes and heart attacks become more likely.⁶ He identifies depression as a multi-system disease:

Depression is not a brain disease merely; it is a neurological, hematological, and cardiovascular disease. Over-activation of stress pathways causes a liability to clots and arrhythmias, and alone or together, these predispose to heart attacks, silent strokes, disturbed mood, and sudden death (p.182).

There are biochemical and behavioral explanations for the relationship between depression and cardiovascular disorders. The two disorders appear to work synergistically with grim consequences. Depression has biochemical, vascular, and behavioral effects and if you have heart disease, you are likely to be depressed. The interaction between the two separate but correlated entities and been investigated in two ways. One, by studying how stress is managed by depressives and two, by focusing on biologic changes present in depressives that are relevant for vascular health.

Depressives are vulnerable to stress and less able to cope with potentially difficult events. They have a lowered threshold for threat perception and cognitively distort situations as being highly threatening. This distorted perception continually triggers the fight/flight response, whereby they either mobilize to meet the threat or retreat and flee from it. Stress hormones are secreted in response to a cognitive appraisal of perceived threat and a secondary appraisal of poor resources for dealing with the threat. These stress hormones liberate energy stores in the form of serum cholesterol, which is elevated to dangerous levels and ultimately finds its way to the lining of the arteries.⁷

In *Against Depression* Kramer explores the link between depression, stress and cardiovascular health and presents two intriguing biological hypotheses. The first concerns blood clots and serotonin and the second pertains to stress related proteins in the blood.

⁵ Heart disease affects an estimated 12.2 million American women and men and is the leading cause of death in the U.S. While about 1 in 20 American adults experience major depression in a given year, the number goes to about 1 in 3 for people who have survived a heart attack. (See Morbidity and mortality: 2000 chart book on cardiovascular, lung, and blood diseases. National Heart, Lung, and Blood Institute, 2000.

⁶ Kramer, Peter *Against Depression*, Viking Press, 2005; p.184.

⁷ See Dougall, A. & Baum, A. *Stress, Health, and Illness* in Baum, A., et. Al., *Handbook of Health Psychology*, 2001.

Depressives have blood platelet abnormalities. Their platelets tend to be too sticky and more likely to become activated and to form clots along the walls of blood vessels.⁸ When stress triggers the fight/flight response, hormones prepare for possible blood loss by making platelets better able to clump.

Serotonin also plays a key role in the process. In depression, brain serotonin receptors and platelets in the blood stream show abnormalities in density. The clotting system, like other aspects of the stress response system, is chronically hyperactive. Depressives and highly anxious people cognitively distort events and have less confidence in their ability to summon the resources to cope with them. This results in frequent firing of the fight/flight response with subsequent maladaptive consequences for the vascular system.

Kramer's second hypothesis is consistent with the research of Edward Suarez at Duke University. Suarez found that men with low serotonin levels produced higher levels of two specific cytokines (proteins in the blood produced in response to stress), which are known to contribute to atherosclerosis, a build up of fatty plaques in the arteries.⁹ According to Suarez, stress activates the immune system, which in turn triggers white blood cells to attack bad cholesterol in the arteries. The consumption of the cholesterol leads to a hardening of the cells, resulting in the formation of deadly plaques.

The current theories relating to blood flow disorders triggered by depression compound the heart-mind behavior dynamic. However, the simplest explanation for a link between depression and heart disease, and for poor outcomes in depressed cardiac patients, is behavioral. Depressives are less able to make good lifestyle choices. Smoking, poor diet, lack of exercise, and drug and alcohol abuse often accompany depression. Non-compliance with prescribed medication regimens is common.

Interdisciplinary Treatment for the Heart Patient with Depression

In light of these findings, effective treatment for patients with heart disease and depression must take a multi-disciplined approach. Herb Benson of the Beth Israel-Deaconess Mind-Body Clinic has compared treatment in these cases to a three-legged stool, and he warns us that without all three legs in place, the stool will topple over.

The first leg is medical treatment of the depressive illness, which is typically through psychopharmacology and anti-depressive medications (SSRIs). While interaction effects need to be monitored, SSRIs are generally well tolerated and safe for people with heart disease.

The second leg involves psychotherapy. Preventive interventions based on cognitive-behavioral theories of depression have been effective. These advocate a problem-focused, skill-acquisition approach that addresses how the person perceives threat. Events previously appraised as catastrophic can be downgraded to simply annoying or

⁸ Kramer, P. *Against Depression*, 2005, p.181.

⁹ See Kramer, P. 2005, p319, FN 181 and 187 for a list of relevant articles.

challenging. Cognitive-behavioral therapy also promotes compliance with medical regimens and life-style changes that are crucial to the long-term outcome in heart disease patients.

The third leg involves regular exercise, which has proven to be effective in reducing both depression and risk of heart disease. In fact, a recent study found that participation in an exercise-training program was comparable to treatment with an anti-depressant medication for improving depressive symptoms in older adults.¹⁰

As health professionals dealing with patients with heart disease and depression, we are quite literally fighting for the hearts and minds of our patients, and we cannot ignore one in pursuit of the other. Chronic illness that cuts across the mind/body split demands intervention from a number of professional disciplines. To provide proper care, treatment for depression in the context of heart disease should be managed by physicians and mental health professionals in close consultation and cooperation with one another.

¹⁰ Blumenthal, J., Babyak, M., Moore, K., et.al. Effects of exercise training on older patients with Major depression. Arch of Int. Med. 1999. 159(19): 2349-56.