

## Editorial / Editöryal Yorum

### Depression is a systemic disease

#### Depresyon sistemik bir hastalıktır

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**S**tress, a state of perturbed homeostasis, can be induced by internal or external stressors. In response to this perturbation, the body begins a series of physical and behavioral responses that constitute the adaptive stress response. The acute stress syndrome leads to a cluster of transient physical and behavioral changes. These changes include increased attention, cardiac output, and respiratory rate. The brain focuses on the stressor as the blood flow is redirected to the brain, heart, and muscles. During acute stress, these reactions, aimed to improve survival, are necessary and short-lived.

Chronic stress is a pathological state of prolonged imbalance of homeostasis caused by excessive, persistent, and frequently repeated stressors. This may lead to irreversible tissue and organ damage, as well as induce a wide range of diseases and syndromes. Chronic stress can play an important role in metabolic syndrome, obesity, chronic obstructive pulmonary disease, diabetes mellitus, atherosclerosis, and depression—especially melancholic depression.<sup>[1]</sup>

Mental stress can change the physiology and chemistry of the central and peripheral nervous systems. Therefore, it is important to consider depression not only as a psychological disorder but also as a systemic disease that adversely affects the entire body.<sup>[2]</sup> Effects of mental stress include increased risk of cardiovascular disease, cerebrovascular disease, hor-

monal disease, and cancer.

Heart failure (HF) is not a disease limited to the heart tissue itself; it is a complex

disease that affects other tissues such as the lung, kidneys, liver, and brain. Both depression and HF have high comorbidities, which are identical: diabetes mellitus, chronic pain, arrhythmias, hypertension, stroke, and metabolic syndrome.

Biologically, it has been demonstrated that HF and depression share common pathologies. These are hyperactivity of the sympathoadrenomedullary system, and hyper-stimulation of the hypothalamo-pituitary-adrenal axis.<sup>[2,3]</sup>

Hyperactivation of the sympathetic nervous system and decreased vagal tone increase plasma catecholamines, stimulate vasoconstriction, elevate heart rate, and activate platelets. Hypercortisolemia is responsible for hypertriglycemia, hypertension, and induction of atherosclerosis; additionally, it accelerates injury to vascular endothelial cells. Endothelial damage and diminished vagal tone lead to the release of proinflammatory cytokines, including interleukin 6, interleukin-1 beta, and tumor necrosis factor. Inflammation is closely associated with endothelial dysfunction, which is associated with onset of atherosclerosis and atherothrombosis.<sup>[2,4]</sup>

#### Abbreviations:

HF	Heart failure
SCI	Silent cerebral infarct

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Proinflammatory cytokines, factors in thrombosis, and vascular occlusion affect neurotransmitter regulation, especially serotonergic transmission. Serotonin mediates positive effects and mood in the healthy brain; thus, the balance of serotonin metabolism may play a central role in the pathogenesis of depression. More than 99% of serotonin in the body is stored in the dense granules of platelets. Platelet abnormalities are observed in depressed patients, and depression is also associated with increased platelet reactivation. Antidepressants may protect the vascular system by preventing blood clots.

Kozdag et al.<sup>[5]</sup> suggest that patients with silent cerebral infarct (SCI) have higher prevalence of depression compared with non-SCI patients. Although both depression and HF are complex systemic diseases, they share pathophysiological mechanisms. These mechanisms may also be shared by other diseases such as chronic obstructive pulmonary disease, diabetes, hypertension, and stroke.<sup>[6]</sup> In such cases, chronic stress—along with age, gender, genetic predisposition, and environmental exposure—may determine the outcome.

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