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# STRESS AND PATHOPHYSIOLOGIC EFFECTS OF STRESS INDUCED CHRONIC CORTISOL

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#### **Annotation**

Some people refer to cortisol as the stress hormone. The body produces cortisol to prepare the body to respond to a perceived danger or stressful situation. When facing sudden stress, the body enters an alarm reaction stage called the fight-or-flight response. This triggers a cascade of events to enable a person to act quickly. Stress raises the cortisol level in the body, triggering bodily changes that enable a person to react to the stressor.

**Keywords:** hormone, cortisol, individual, metabolic syndrome, hypertension, diabetes, atherosclerosis.

Stress is broadly defined as a perceived or anticipated threat that disrupts a person's wellbeing, or homeostasis, and exceeds the individual's capacity to meet the demands. The demands can be physical in nature (e.g., exposure cold temperatures, moving heavy equipment) or psychological (e.g., rush to meet exam deadlines, attempting to complete multiple job assignments). In everyday life, the term stress is commonly used to describe negative thoughts and uncontrolled feelings. Experiences that cannot be easily reconciled and threaten one's sense of security are considered stressful, and the random and constant external and internal challenges are called stressors. Stressful events perceived as especially threatening, chaotic, and chronic are personal, such as loss of a family member, loss of job security, cancer diagnosis, physical abuse, social neglect, or feelings of social discrimination. The National Institute of Mental Health further defines stress in terms of how the brain responds to a demand by activating the neuroendocrine system (NES), the autonomic nervous system (ANS), and the immune system (IS). Importantly, chronic stress induced activation of these physiologic systems has the potential to compromise recovery and predispose the individual to engage in a wide





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range of unhealthy coping strategies, such as foregoing sleep, eating high-calorie comfort foods, and withdrawing from physical activity. Continued engagement in these behavioral activities is linked to a number of serious illnesses, such as hypertension, depression, diabetes, and obesity. This chapter discusses the role played by the body and brain in relation to psychological and emotional stressors that promote the onset and progression of human diseases.

The perception or anticipation of a threat activates three major physiologic stress systems: the hypothalamic–pituitary–adrenal (HPA) axis; the sympathetic nervous system (SNS); and the immune system (IS). Acute activation of these stress-related systems modulates a broad range of mediators on the body and brain to protect and meet the physiologic and behavioral demands of the stressor to facilitate recovery. For example, stress induced activation of the SNS and the HPA axis triggers the release of hormones (e.g., epinephrine, cortisol), which rapidly mobilizes resources necessary to prepare the body and brain for "fight or flight" responses to threat. In addition, both stress systems send signals to the immune system to release proinflammatory cytokines, such as interleukin-1 (IL-1), IL-6, and tumor necrosis factor- $\alpha$  (TNF- $\alpha$ ). The release of these sympathetic, neuroendocrine, and immune factors has profound effects on immunity, behavior, and physiology.

Chronic dysregulation of the HPA axis, especially abnormal elevation of cortisol, is linked to a wide variety of disorders, including obesity, sleep deprivation, lipid abnormalities, hypertension, diabetes, atherosclerosis, and loss of bone density. 4 In the brain, chronic GC secretion may reduce hippocampal volume, enlarge the ventricles, and modulate reversible cortical atrophy. 4 These CNS changes may contribute to cognitive impairments and emotional disorders. For example, chronic depression is accompanied with shrinkage of the hippocampus and the prefrontal cortex. In the periphery, heightened stress-induced cortisol levels promote gastric secretion in the stomach and intestines, potentially causing gastric ulcers, which may account for the gastrointestinal ulceration observed by Selye. Furthermore, GCs contribute to the development of metabolic syndrome and the pathogenesis of obesity by directly causing insulin resistance and influencing genetic variations that predispose to obesity.





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In conclusion, when a person faces an unusual or unexpected stressor, the body's sympathetic nervous system activates. This leads to a cascade of hormonal and other responses to help the body react to the stressor.

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