

Stress and the immune response essay sample

[Health & Medicine](#), [Stress](#)



Cortisol is a steroid hormone synthesized in adrenal glands with a myriad of functions mainly in the endocrine and immune systems, and produced during stress circumstances. Under normal conditions, stimuli such as a pathogen release immune proteins called interleukins, which further activate T-lymphocyte cells. T-cells multiply to amplify the immune response and ultimately attack the pathogen (Abbas, 2014). Other immune responses are simultaneously activated, e. g. the complement system. The complement system exerts its functions through three pathways: classical, alternative or lectin pathways. The main activators of the classical pathway are antigen-antibody complexes, which trigger a series of events mainly mediated by complement proteins C1 complex, C2, C3, and C4. The alternative pathway triggers when the C3b protein binds pathogens directly, and its main effectors are proteins C3 and C5. The lectin pathway is similar to the classical one, but lacks participation of the C1 complex (Abbas, 2014).

Cortisol weakens immune response by interfering both with T-cell proliferation inhibiting the T-cell growth factor interleukin-2 (IL-2; Palacios & Sugawara, 1982; Randall, 2011), and with the complement system down regulating complement C9 and C4 protein concentrations (Teles, Boltaña, Reyes-López, Santos, Mackenzie & Tort, 2013). Cortisol helps to maintain a balance between triggering some immune pathways while inhibiting others, thus preventing an exaggerated response.

In a case-control study between health subjects and subjects suffering from post-traumatic stress disorder (PTSD), Hovhannisyan et al. (2010) found that -compared to controls- all PTSD subjects had higher white blood cell (WBC) counts, C-reactive protein concentrations, IL-1b, IL-6 and IL-6 receptor, while

exhibiting lower levels of IL-4 (anti-inflammatory protein). Plus, they also found that in subjects with PTSD, the classical pathway of the complement is hyperactivated, while the alternative pathway is hypoactivated and the terminal pathway is overactivated (Hovhannisyan et al., 2010), thus leading to dysfunction of the complement system and making subjects more vulnerable from the immunological point of view. This means that when exposed to a pathogen, these subjects might not be able to attack the microbe as quickly and efficient as they would under normal circumstances. While Hovhannisyan et al. (2010) explored the role of cortisol in the complement system, Randall (2011) focused on describing cortisol effects on T-cells.

Pro-inflammatory proteins such as interleukins also reach the brain during an immune response, connecting the immune system to the central nervous system (CNS), and affecting mood and behavior. Interleukin effects on the brain ultimately result in less activity, food intake, social interaction, sexual behavior, as well as acute pain, memory disruption and fever, all with the evolutionary purpose of forcing the individual to get some rest and not be exposed to potentially life-threatening situations while fighting against a disease or pathogen (Maier & Watkins, 2012). However, if the inflammatory response is prolonged or exaggerated -such in chronic disease like diabetes and cancer, or mental disorders like PTSD-, the mood and behavioral responses become established, thus exhibiting cognitive impairment, depression, fatigue and chronic pain (Maier & Watkins, 2012). As Maier and Watkins (2012) describe it, physiology becomes pathology.

In summary, stress exerts an influence on the immune system mediated by

cortisol and other hormones, and amplified by interleukins. T-cells and the complement system are the final effectors of the immune response. Cortisol balances immune activation both by stimulating and inhibiting different pathways, including T-cell proliferation and the complement system pathways. Chronic mental disorders such as PTSD dysregulate cortisol and immune functions, prolonging or exaggerating some immune responses, while downregulating others. Interleukins produced under these chronic states also affect CNS activities, leading to depression, fatigue and chronic pain.

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