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The Integration of Mental Health and Immunity: A Review of Human Immune Function in Stressful Conditions

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Abstract: **Physiologic processes and outcomes sometimes are divorced from mental health status. However, scientific research has proven this to be incorrect and reckless. There is a close relationship between mental health and human immune functioning. The incidences of infectious and autoimmune disorders link them with stressful conditions. The objective of this review paper was to explore the functioning of the human immune system during stressful conditions.**

Keywords: **Immunity, central nervous system, psychoneuroimmunology, stress, infections, antigens, antibodies, catecholamine.**

1. INTRODUCTION

Science has firmly established the interconnectedness of the systems of the body and their effectiveness in conjunction with each other. The relationship between the immune system and mental health in the development of infectious diseases and autoimmune disorders, and recovery from them is an interesting and exciting area of study called psychoneuroimmunology. Once thought to be independent of each other, the field of psychoneuroimmunology has established that the immune system is highly integrated with the nervous system, allowing it to react to changes in the psychosocial environment. This review paper examines the interactions between the brain and immune system in stressful conditions.

2. ROLE OF THE IMMUNE SYSTEM

The immune system comprises cells and organs that function to: - recognize non-self-entities such as pathogens including viruses, bacteria, fungi, prions and parasites in the body; non-pathogenic substances such as pollen; and tumour cells; and 2 - destroy / neutralize them[1,2]. These are accomplished via a complex controlling system of organs, cells, cell receptors, and proteins.

The immune system functions in two lines of defense, innate and adaptive immunity. Innate immunity, the first line of defense, comprises cells such as neutrophils and macrophages. These respond to nonspecific indicators of attack like infection and trauma, with inflammation as the correspondingly nonspecific defense[1,2]. Inflammation is modulated by proteins called cytokines, which are secreted by these and other cells. Cytokines such as tumor necrosis factor-α, interleukin (IL)-1, and IL-6, facilitate communication between cells. They promote local responses such as vasodilation and infiltration of circulating immune cells into the affected tissue, as well as general responses such as fever. Although the inflammatory response is important for the localization of early responses to infection, it is not effective enough to clear them. Consequently, acquired or adaptive immunity, the second line of defense, is activated.

The adaptive immune system comprises groups of cells that respond to specific antigenic stimulation. Antigens are particular molecules that are displayed or produced by pathogens, marking them for recognition by the immune cells as foreign or non-self. The antigen-specific blood cells that function in adaptive immunity are lymphocytes. These include helper T lymphocytes, which release cytokines such as IL-2, IL-4, IL-5, and IL-10 to activate and direct other immune cells; cytotoxic T lymphocytes, which are capable of killing compromised cells such as an epithelial cell infected by a virus; and B cells, which produce antibodies. Antibodies are proteins produced in response to a specific antigen on a pathogen, and attach to it to either inactivate it or mark the pathogen for destruction by other immune cells[1,2].

Though the immune system is quite effective in carrying out its functions, there is a relationship between stress and changes in the immune system, whether the stressors are physical or psychological in nature[3]. However, the ability to adjust or habituate to repeated stress is greatly determined by the way a person perceives a situation[4].Thus, the immune consequences are also determined by a person's psychological state.

The mind affects immunity:

The concept that psychological states can affect the outcome of human disease is an old one. Galen, Roman surgeon and philosopher, who lived until AD200, wrote that melancholic women were more susceptible to "swellings" of the breasts than were sanguine women[4,5]. It was apparent from those early days of physiology and psychology that a person's mental functioning plays an important role in disease processes. The temperament of a person, to a great extent determines how he or she will adapt to stressful situations. Furthermore, the nature of the stress experienced can influence the effects on the immune system.

3. INTERACTION BETWEEN THE CNS AND THE ENDOCRINE AND IMMUNE SYSTEMS

Various aspects of the immune response can be down-regulated by stress. Communication between the central nervous system (CNS) and the immune system occurs through chemical messengers secreted by nerve cells, endocrine organs, or immune cells, and psychological stressors can disrupt these networks.

Immune function is affected by (1) neurotransmitters such as norepinephrine and serotonin; (2) neuropeptides such as vasoactive intestinal peptide and corticotrophin-releasing factor; (3) neurohormones such as growth hormone, and prolactin; and (4) adrenal hormones such as corticosteroids and epinephrine[4]. The receptors for these are present on lymphocytes and macrophages that secrete cytokines. The neuroendocrine and immune systems share common signal mediators and receptors. This suggests that the brain has an immunoregulatory role and the immune system a sensory function. The cytokines interleukin 1, tumour necrosis factor (TNF), interferon α and interferon γ secreted from activated immune cells can in turn change the function of the neuroendocrine system. Interleukin 1 mediates inflammation, promotes fever and sepsis. The interaction with the immune system involves most of the brain, where moderate to high concentrations of receptors for interleukin 1 have been detected.

Many of these mediators are produced in the brain by glial or neuronal cells and have functions similar to those of neurotransmitters. Interactions between emotions and immune functions might underlie the increased clinical susceptibility to infectious diseases.

For a clearer understanding of the remainder of this discussion, the definition of the term catecholamines is given. *Catecholamines* are hormones produced by the adrenal glands, which are found on top of the kidneys. They are released into the blood during times of physical or emotional stress. The major catecholamines are dopamine, norepinephrine, and epinephrine (which used to be called adrenalin) these are especially active in the fight or flight response[6].

Responses in acute stress:

Stress experiments suggest that the plasma concentration of the catecholamine epinephrine is related to specific immune functions of lymphocytes and macrophages. When there are sudden changes in the environment, causing acute stress, the plasma concentrations of epinephrine can increase more than a hundred fold[7]. Several experiments on rats showed that macrophage and T cytotoxic lymphocyte concentrations and activities increased shortly after the infusion of epinephrine returning to baseline levels after one hour[3,8,9]. This suggests that cells of innate immunity which clear pathogens like bacteria and fungi, and adaptive immunity via T cytotoxic lymphocytes that are able to directly destroy virus infected

cells, are immediately activated when acute stress is experienced. Innate immunity requires minimal time for changes of activity and has fewer restrictions to acting quickly[3] while adaptive immunity takes a longer time to be invoked. Remarkable evidence in these responses show that the immune system prepares for the invasion of pathogens which may enter the body due to injuries sustained during fight or flight; a typical response in acute stress.

Responses in chronic stress:

Studies also show that there is an inverse relationship between immune functioning involving macrophage and lymphocyte concentrations and activities and chronic stress[4]. Chronic stress suppresses immune functions, so these cells become impaired and are less effectual during these periods resulting in a greater vulnerability to infections and slower wound healing[10].

Studies revealing the relationship between the immune system and the central nervous system indicate that stress can alter the function of lymphocytes and macrophages. Persons experiencing chronic stress have a weaker adaptive immune response. This is mediated by the release of the steroid hormone cortisol, the production of which is regulated by the pituitary gland that is controlled by the hypothalamus. During chronic stress, cortisol is over produced, causing fewer receptors to be expressed on macrophages and other immune cells. This down-regulation, in turn, reduces the cells' capacity to respond to anti-inflammatory signals and allows cytokine-mediated inflammatory processes to flourish[3]. An exaggerated inflammatory response leads to tissue damage, down-regulation of the cells of the innate immune system recruited to the area, inflammation cannot be terminated, and wound healing is slow[10]. In addition, the proliferation of lymphocytes is slowed, resulting in increased vulnerability to diseases[11].

4. CONCLUSION

The interactions between the immune system and the CNS in the development of infectious diseases cannot be underplayed. Stressful conditions may have deleterious effects on immune functioning, down-regulating immune cells and leaving the body vulnerable to infections. During acute stress, stress hormones may boost immune functioning by informing the immune system about imminent encounters with stressors. Hence, the activities and numbers of lymphocyte and macrophage immune cells are generally increased in acute stress to prepare the body for short-term stressors. On the contrary, immunosuppression has been found as the result of chronic stress which has been shown to have an adverse effect on health, leading to exhaustion, distress, and disease. Mental and immune processes are interconnected.

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