EDITORIAL

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Neuroimmunomodulatory responses in brain inflammation

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The human body is protected against different infections by identifying and killing the pathogens and tumor cells which attacks and alters the functions of the healthy cells and organs; this is referred to as the human body's immune system. This system can be described as a homeostatic mechanism as this maintains the constancy and integrity of body cells and tissues under physiological conditions [1]. Research established a connection in between the central nervous system (CNS) and the immune system where the former is known to interact with the later via a complex set of network that includes different neural, hormonal and paracrine actions which can emit and receive bi-directional signals. Stimulation of the immune system by any foreign pathogens produces a series of responses like alteration in the activity of neuroendocrine axis, fever, anorexia, inactivity and changes in the sleep-awake cycle in the CNS. These responses are mediated by the hypothalamus; this is referred to as Neuroimmunomodulation [2].

Studies showed that any sort of brain injury and trauma can initiate an inflammatory response in that injured portion. This inflammatory response consists of mediators (cytokines, complement activation, chemokines and adhesion molecules) [3]. Cytokines, which are produced by white blood corpuscles (WBC) of the immune system, mediate the activity of CNS such as interleukin-1 beta (IL-Iβ) activate the Hypothalamus- Pituitary – Adrenal axis (HPA axis). This in turn causes increased secretion of glucocorticoids, which suppress the immune response via negative feedback mechanism. Inflammatory stress leads to increased generation of reactive oxygen species (ROS) and high plasma cytokine levels. The susceptibility to infection, cancer and autoimmune diseases increases with the impairment of the physiological systems including the nervous, endocrine and immune systems; this is correlated with the Neuroimmunomodulation [4]. Evidences showed that, in order to maintain the defense mechanism of the human body a connection need to be set up in-between the neuroendocrine and immune system. A line of investigation proved that stress has negative effects on the immune system. In addition, corticotrophin releasing hormone (CRH) is found to have a series of responses during the stress reaction. Scientific investigations confirmed that CRH, the

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major stress-integrating peptide, modulates the immune system directly. Scientific investigations confirmed that CRH, the major stress-integrating peptide, modulates the immune system directly and this hormone is also known to stimulate ACTH secretion. CRH is found to act as an immunosuppressant agent free of circulating glucocorticoids. The stimulation of sympathetic outflow is partly responsible for this effect. Oxidative stress is also associated with increased secretion of CRH and thus related to inactivation of macrophages. This increased secretion of CRH stimulates anterior pituitary, which in turn alters the production of IL-1, the later mediates stress-induced immunosuppression [2]. Brain injuries like head trauma, stroke, seizure, infection may lead to brain inflammation [5]. Some molecular and structural changes have been observed to occur during and after the seizure like activities. These are mediated by the associated inflammatory reactions occurring in the brain which finally activates the innate immune system. The release of pro-inflammatory mediators like interleukin (IL)-1, tumor necrosis factor (TNF), vasoactive mediators, adhesion molecules and reactive oxygen species indicates the commencement of inflammation [6]. The activated macrophages release pro-inflammatory cytokines which plays a crucial role in triggering the local inflammatory response [7]. TNF and IL-1β are studied to function as the signaling molecules for activation of neuroendocrine immunomodulatory responses. The inflammation reaction is managed as an anti-inflammatory balancing mechanism by the sympathetic division of the autonomic nervous system (SNS) and the HPA axis [8].

Finally, from the different research works it is evident that in response to any sort of pathogen attack the immune cells produce pro-inflammatory cytokines which crosses the blood brain barrier and alters the brain's behavior. Thus, in order to fight against the inflammation reactions of the host's body it need to activate both the immunomodulatory resources of the nervous and endocrine systems.

Keywords:

Glucocorticoid, hypothalamus- pituitary – adrenal axis, immunosuppression, inflammation, neuroimmunomodulation, reactive oxygen species

Abbreviations

central nervous system (CNS), corticotrophin releasing hormone (CRH), hypothalamus-pituitary-adrenal axis (HPA axis), IL-Iβ (Interleukin-1 beta), reactive oxygen species (ROS), sympathetic division of the autonomic nervous system (SNS), tumor necrosis factor (TNF), white blood corpuscles (WBC).

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