

## Endocrine-Immune Cross Talk

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### Editorial

Research in recent years has indicated integrated role played by neuro-immuno-endocrine interactions in maintaining homeostasis and deranged cross talk in these intricately complicated systems in development of various complex chronic illnesses, autoimmune disorders. The most convincing evidence of importance of this interactions is the fact that immune cells express hormone receptors (which may be present under basal conditions like or may be expressed on stimulation), express cytokines which act on nervous system as well as on endocrine glands and in addition exert autocrine and paracrine effects. In turn endocrine glands express receptors for the cytokines released by immune cells. Thus there exists bidirectional communication between these systems [1].

### Hormonal Actions on Immune System

Lymphoid cells express receptors for various hormones like corticosteroids, PRL, insulin enkephalins and endorphins, growth hormone, adrenergic agents, acetylcholine, insulin, testosterone and estrogen in varying degree of density (e.g. B lymphocytes have more adrenergic receptors than T lymphocytes) also their number and activity changes as per changes in activity of the cell (insulin receptors appear on lymphocytes during mitogenic state or antigenic stimulation particularly allogenic antigens) [1,2].

Hypophysectomy leads to suppression of immunity. ACTH, glucocorticoids, androgens and progesterone suppress immune response while growth hormone, thyroxine, insulin and prolactin increase it. Vitamin D sufficiency is also required for normal immune function. More over local production of hormones by lymphoid organs can also influence the response of the immune system. Epithelial thymic cells can produce steroids. Selection of lymphocytes during ontogeny is also influenced by circulating glucocorticoids which stimulate apoptosis in immature lymphocytes which express high affinity for self-antigens. They also render antigen specificity to lymphocytes [1,3].

Hypothalamo-pituitary-adrenal axis is an important pathway for regulating immune response during stress. Many of its

actions are also mediated via systemic sympathetic system [1,4]. Proopiomelanocortin derivatives viz ACTH, alpha-melanocyte stimulating hormone(alpha-MSH), beta endorphins have antagonist action to PRL and growth hormone in their action on immune system. alpha-MSH directly inhibits NF-kappa B nuclear factor to exert its anti-inflammatory action whereas role of ACTH is immunomodulatory: anti-inflammatory via glucocorticoids and direct action on suppression of Th1 and increased cytokine production by Th2 [1,3,4]. Resting T and B lymphocytes are more sensitive to glucocorticoids than stimulated ones (hence administration after vaccination causes lesser immune suppression than before vaccination). Interestingly the antibody production of B lymphocyte is rather stimulated by glucocorticoids. There is evidence to believe that levels of glucocorticoids are increased late during immune response giving enough time to immune system while later as the immune response proceeds HPA axis causes immune suppression by inhibiting production of lymphokines and monokines. Thus HPA axis-immune system interaction protects body from excessive activation over- expansion of immunological cells.

Various adipokines are also known to have humoral effects on immune system some pro-inflammatory and others anti-inflammatory. Leptin which is most studied among them is pro-inflammatory and increases phagocytic activity, development, activation of Natural killer(NK) cells and increases cytokine secretion of TNF-alpha, IL-6 and IL-12. It promotes proliferation of T lymphocytes and switch over of memory T cells towards Helper T cells. Adiponectin is shown to have anti-inflammatory actions.

### Immune Actions on Endocrine System

Cytokines are chemical messengers that stimulate the HPA axis when the body is under stress or experiencing an infection important role being played by cytokines. In fact cytokines are important mediators in stimulating HPA during stress.

Pituitary corticotroph POMC gene expression is regulated by CRH and a vast group of around 130 cytokines acting as neuro-immuno-endocrine modulators. Cytokines (including IL-1, TNF, and members of the gp130 cytokine family) participate as

mediators of a complex HPA axis response to stress and inflammation [5,6].

Certain cytokines are released during immune activation also act on hypothalamo-gonadal axis resulting in inhibitory effects on reproductive functions. This has been demonstrated by reproductive abnormalities in mice with congenital absence of thymus and hence T lymphocytes i.e., Nude mice [7,8]. This can be viewed as role of hormone-immune interactions in natural selection process.

## Hormone Immune Interactions during Disease

Maintenance of homeostasis requires a harmonious bidirectional interaction between immune system and HPA axis. During periods of stress too his dual mode of communication is protective to the host. Disruption in this loop leads to the development of lympho proliferation as well as autoimmune diseases.

An active role is played by the adipokines (increased levels of leptin, reduction in adiponectin levels, released from visceral adipose tissue in development of pro-inflammatory state, insulin resistance, enhanced atherosclerosis and development of CAD which is an example of cross talk between hormone and immune systems.

## Sick Euthyroid Syndrome

Cytokines particularly interleukin (IL)-1, IL-6, tumor necrosis factor (TNF)-alpha, and interferon-beta paly role in Nonthyroidal illness. Cytokines are shown to affect the

hypothalamus, the pituitary, or other tissues, inhibiting production of TSH, thyroid-releasing hormone (TRH), thyroglobulin, T3, and thy-roid-binding globulins. Cytokines are also said to be responsible in decreasing the activity of type 1 deiodinase and to decrease the binding capacity of T3 nuclear receptors [1,9].

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