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The immuno-endocrine component in the pathogenesis of tuberculosis.

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Source

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Abstract

Tuberculosis (TB) may be regarded as a disease in which the immune response to Mycobacterium tuberculosis, its etiologic agent, is engaged both in protection and pathology. Different T-lymphocyte subsets are involved in the immune response against M. tuberculosis, but production of interferon-gamma (IFN-gamma) by T cells seems to be fundamental for disease control. Th1-type cytokine responses predominate in patients with mild or moderate forms of pulmonary TB, whereas the production of Th2-type cytokines prevails in the severe disease. Since the immune response fails to definitely eradicate the pathogen, a chronic infection is established, and it is likely that a broad range of regulatory mechanisms operate in this situation. Cytokines released during the course of an immune response activate the hypothalamus-pituitary-adrenal axis leading to the production of glucocorticoids and dehydroepiandrosterone (DHEA), with known immunomodulatory effects. TB patients exhibit increased concentrations of interleukin-6 and cortisol in plasma, reduced DHEA and testosterone levels, together with remarkably increased growth hormone concentrations that were not accompanied by an expected raise in insulin-like growth factor-1. Significant increases in estradiol, prolactin, and thyroid hormone concentrations were also detected in patients. Cortisol inhibits the mycobacterial antigen-driven proliferation and IFN-gamma production, whereas DHEA suppresses transforming growth factor beta production by lymphoid cells from TB patients with advanced disease. Furthermore, supernatants from cultures of M. tuberculosis-stimulated mononuclear cells of TB patients inhibit DHEA secretion by a human adrenal cell line. This type of immuno-endocrine interactions may affect the control of tissue damage and the development of protective immune responses, partly accounting for disease aggravation.

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