

Effects of growth hormone and insulin-like growth factor I on T- and B-lymphocytes and immune function.

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The concept of a neuro-endocrine-immune axis was proposed more than 50 years ago. Growth hormone (GH), a central component of this axis has many functions at both a molecular and cellular level, including thymocyte proliferation, stimulation of the cytotoxic activity of natural killer cells and induction of lymphocyte proliferation. Binding of GH to its receptors on lymphocytes stimulates the production of insulin-like growth factor I (IGF-I), which mediates the effects of GH on cell proliferation. Other effects of GH on the immune system appear to be direct, such as priming monocytes for enhanced production of hydrogen peroxide in response to phorbol esters, and stimulating neutrophils to secrete superoxide anions associated with enhanced phagocytic activity. Many of the effects of GH are shared by IGF-I. Despite these observations, and the fact that GH is produced and secreted in immunological tissues such as the thymus and spleen, immune deficiency is not characteristic of GH deficiency in humans. The question remains as to whether GH and IGF-I could be used as immunotherapy. Currently, both agents have been used in adults to diminish wasting due to acquired immunodeficiency syndrome, and GH has been shown to stimulate CD8⁺ cell counts. However, they had little impact on CD4⁺ cell counts, which may be due to IGF-I and GH resistance in these individuals. The use of GH and IGF-I as immunotherapies merits further study.

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