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1: [Neuroimmunomodulation](#). 1999 Jan-Apr;6(1-2):56-68.

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The immune-endocrine loop during aging: role of growth hormone and insulin-like growth factor-I.

[Burgess W](#), [Liu Q](#), [Zhou J](#), [Tang Q](#), [Ozawa A](#), [VanHoy R](#), [Arkins S](#), [Dantzer R](#), [Kelley KW](#).

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Why a primary lymphoid organ such as the thymus involutes during aging remains a fundamental question in immunology. Aging is associated with a decrease in plasma growth hormone (somatotropin) and IGF-I, and this somatopause of aging suggests a connection between the neuroendocrine and immune systems. Several investigators have demonstrated that treatment with either growth hormone or IGF-I restores architecture of the involuted thymus gland by reversing the loss of immature cortical thymocytes and preventing the decline in thymulin synthesis that occurs in old or GH-deficient animals and humans. The proliferation, differentiation and functions of other components of the immune system, including T and B cells, macrophages and neutrophils, also demonstrate age-associated decrements that can be restored by IGF-I. Knowledge of the mechanism by which cytokines and hormones influence hematopoietic cells is critical to improving the health of aged individuals. Our laboratory has recently demonstrated that IGF-I prevents apoptosis in promyeloid cells, which subsequently permits these cells to differentiate into neutrophils. We also demonstrated that IL-4 acts much like IGF-I to promote survival of promyeloid cells and to activate the enzyme phosphatidylinositol 3'-kinase (PI 3-kinase). However, the receptors for IGF-I and IL-4 are completely different, with the intracellular beta chains of the IGF receptor possessing intrinsic tyrosine kinase activity and the alpha and gamma subunit of the heterodimeric IL-4 receptor utilizing the Janus kinase family of nonreceptor protein kinases to tyrosine phosphorylate downstream targets. Both receptors share many of the components of the PI 3-kinase signal transduction pathway, converging at the level of insulin receptor substrate-1 or insulin receptor substrate-2 (formally known as 4PS, or IL-4 Phosphorylated Substrate). Our investigations with IGF-I and IL-4 suggest that PI 3-kinase inhibits apoptosis by maintaining high levels of the anti-apoptotic protein Bcl-2. The sharing of common activation molecules, despite vastly different protein structures of their receptors, forms a molecular explanation for the possibility of cross talk between IL-4 and IGF-I in regulating many of the events associated with hematopoietic differentiation, proliferation and survival.

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