IL-5 Inhibits Apoptosis by Upregulation of c-myc Expression in Human Hematopoietic Cells

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摘要

Abstract

Interleukin 5 (IL-5) inhibition of apoptosis is required throughout many hematopoietic lineages to regulate proliferation and differentiation. It is not clear how IL-5 mediates the intracellular molecular events regulating the anti-apoptotic effect. The cell lines TF-1 and JYTF-1 expressed different amounts of the IL-5 receptor alpha (IL-5R alpha) subunit, which caused contrasting effects in response to IL-5. IL-5 supported the survival but not the anti-apoptotic activities of TF-1 cells, which have a low expression of IL-5R alpha. In contrast, IL-5 supported both the survival and the anti-apoptotic activities of JYTF-1 cells, which overexpressed IL-5R alpha compared to TF-1 cells. In this study, IL-5 stimulation increased Annexin V binding (indicative of apoptosis) in TF-1 cells and decreased apoptosis in JYTF-1 cells. The proto-oncogenes c-fos, fosB, and c-jun were not detected, whereas junB was induced by IL-5 stimulation in both types of cells. Most importantly, IL-5 significantly induced c-myc expression in JYTF-1 cells, but did not in TF-1 cells. These results are consistent with the possibility that IL-5 inhibits apoptosis in JYTF-1 cells via the upregulation of c-myc expression.