

Publication

Hematopoietic transcription factor GATA-2 promotes expression of alpha globin and cell death in FL5.12 cells

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Recently we showed that alpha globin is a novel pro-apoptotic factor in programmed cell death in the pro-B cell line, FL5.12. Alpha globin was also upregulated in various other cell lines after different apoptotic stimuli. Under withdrawal of IL-3, overexpression of alpha globin accelerated apoptosis in FL5.12. Here, we have studied how transcription of alpha globin is placed in the broader context of apoptosis. We used Affymetrix chip technology and RT QPCR to compare expression patterns of FL5.12 cells growing with or without IL-3 to search for transcription factors which were concomitantly upregulated with alpha globin. The erythroid-specific transcription factor GATA-2 was the earliest and most prominently upregulated candidate. GATA-1 was expressed at low levels and was weakly induced while GATA-3 was completely absent. To evaluate the influence of GATA-2 on alpha globin expression and cell viability we overexpressed GATA-2 in FL5.12 cells. Interestingly, high expression of GATA-2 resulted in cell death and elevated alpha globin levels in FL5.12 cells. Transduction of antisense GATA-2 prevented both increase of GATA-2 and alpha globin under apoptotic conditions and delayed cell death. We suggest a role of GATA-2 in apoptosis besides its function in maintenance and proliferation of immature hematopoietic progenitors.

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