Basic fibroblast growth factor antagonizes activin A-mediated growth inhibition and hemoglobin synthesis in K562 cells by activating ERK1/2 and deactivating p38 MAP kinase.

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Abstract

Activin A can induce erythroid differentiation, whereas basic fibroblast growth factor (bFGF) can maintain the undifferentiated status of erythroid progenitors. How these two factors together can affect the regulation of erythroid differentiation in hematopoietic cells has not been elucidated. This study demonstrates that bFGF antagonizes activin A-mediated growth inhibition and hemoglobin (Hb) synthesis in K562 cells. Analyses of mitogen-activated protein kinases revealed that activin A-induced p38 phosphorylation and inhibited ERK1/2 phosphorylation. In contrast, bFGF worked antagonistically to induce ERK1/2 phosphorylation and inhibited p38 phosphorylation in K562 cells. Furthermore, co-treatment of cells with activin A and bFGF decreased p38 phosphorylation and increased ERK1/2 phosphorylation. SB203580 inhibition of p38 activity eliminated activin A-mediated growth inhibition and Hb synthesis, whereas U0126 inhibition of ERK1/2 activity augmented the effects of activin A on K562 cells. These results suggest that bFGF can negatively modulate p38 and positively modulate ERK1/2 to antagonize activin A-mediated growth inhibition and Hb synthesis in K562 cells.