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Effects of STAT3 silencing on fate of chronic myelogenous leukemia K562 cells.

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Abstract

Signal transducer and activator of transcription 3 (STAT3), a transcription factor, is constitutively activated in various types of cancers. Previous investigations have demonstrated that this overexpression of STAT3 in human malignancies plays important roles in maintaining the characteristics of malignant tumors by having an effect on proliferation, differentiation, and/or immortalization. Thus, inhibition of STAT3 expression could be a potent therapeutic approach in cancer treatment. In this study, we introduced STAT3 siRNA into the human chronic myelogenous leukemia (CML) K562 cell line, which has constitutive activation of STAT3, to elucidate the role of STAT3 in CML. The cells were transducted with STAT3 siRNA using lentivirus. FACS, real-time PCR, and Western blot were used to study changes in STAT3 expression levels in transducted cells by comparing with negative control siRNA lentivirus transduction. Knockdown of STAT3 by STAT3 siRNA caused a decrease in STAT3 protein level, inhibition of growth and proliferation, cell cycle blockade, visible morphologic changes, and induction of apoptosis in K562 cells. These findings demonstrate that STAT3 does indeed play a critical role in the survival of K562 cells, which may have potential application in designing molecular therapies for CML treatment.

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MeSH Terms, Substances

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