ABSTRACT

THESIS: The Influence of Endogenous Expression of Tal-1 on Apoptotic Gene Expression.

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Tal-1 is a transcription factor that is frequently ectopically expressed in the majority of cases of T-cell acute lymphoblastic leukemia (T-ALL). The ectopic expression of Tal-1 in patients with ALL has been found to decrease susceptibility to chemotherapeutic drugs and apoptosis. Thus, this study focuses on the effects of endogenously expressed Tal-1 in the Jurkat cell line on three Bcl-2 family members (Bcl-2, Bcl-xL, and Bid) and the inhibition of apoptosis and cell viability when exposed to apoptosis inducing drugs such as etoposide. The data obtained indicate that when treated with etoposide for 12 h Jurkat cells endogenously expressing Tal-1 have an 81% higher
level of anti-apoptotic \textit{Bcl-2} expression, an 18\% lower level of anti-apoptotic \textit{Bcl-xL} expression, and a 31\% lower level of pro-apoptotic \textit{Bid} expression compared to Jurkat cells lacking \textit{Tal-1} expression.

The data also demonstrates that Jurkat cells endogenously expressing \textit{Tal-1} have a 15.94\% lower amount of cell death after treatment with etoposide for 12 h and a 20.34\% lower amount of cell death after treatment with etoposide for 24 h when compared to Jurkat cells that lack \textit{Tal-1} expression. Thus, the endogenous expression of \textit{Tal-1} increases the amount of the anti-apoptotic \textit{Bcl-2} expression and decreases the amount of the pro-apoptotic \textit{Bid} creating an overall anti-apoptotic signal within the cell.