Proliferative Effect of sTRAIL on Mouse Pancreatic Beta Cells

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Beta cell loss/impairment of function appears as a significant problem in both type 1 and type 2 diabetes. TRAIL (TNF-related apoptosis-inducing ligand) was recently correlated with both types of diabetes with a proposed protective effect. TRAIL was also shown to promote survival and proliferation in different cells such as vascular smooth muscle cells and human vascular endothelial cells. Recently, TRAIL was claimed to protect pancreatic beta cells against cytokine-related harm. We hypothesized a proliferative effect for TRAIL on beta cells, and used Min6 mouse pancreatic beta cell line to test our hypothesis. Min6 cells were treated with various doses of (0, 0.1, 1, 10, 100 ng/mL) soluble TRAIL molecule (sTRAIL) for 24, 48, and 72 hours. Survival and proliferation tests (WST-1 and Ki67, respectively) were performed. Phosphorylation levels of intracellular ERK, p38, and Akt molecules were studied by western blotting. We found that sTRAIL did not lead to apoptosis in Min6 cells, but increased survival and induced proliferation at 10 ng/mL dose. ERK and p38 phosphorylation was induced substantially, and Akt was phosphorylated at a lower degree in these cells. Our results suggest that sTRAIL increases cell survival and proliferation in Min6 mouse pancreatic beta cell line. These findings, while requiring further investigation, support a possible therapeutic role for TRAIL in diabetes.

Key words Cell Line, Tumor; Cell Proliferation; Insulin-Secreting Cells; Mice; TNF-Related Apoptosis-Inducing Ligand