Proteolysis of β-catenin in Apoptotic Leukemia Cell Induced by Several Apoptosis Stimuli

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**Background** β-catenin, a known regulator of strong cell-cell adhesion and transcriptional regulation, is proteolytically processed in various adherent cell types after induction of apoptosis. Although β-catenin has been implicated in the apoptosis of adherent cell population, the role of β-catenin and its regulation in non-adherent cells have not been examined.

**Methods** Since β-catenin reported to be present in Jurkat T-acute lymphoblastic and U937 acute myeloblastic leukemia cells, we examined the role and fate of β-catenin during hematopoietic cell apoptosis.

**Results** The data presented here demonstrate that treatment of the Jurkat cells with the apoptosis inducers anti-Fas, TRAIL, staurosporin, and etoposide induced proteolytic fragments of β-catenin, as did TRAIL and staurosporin in U937 cells. In Jurkat cells, β-catenin was cleaved at both the N- and C-terminus after anti-Fas. Densitometric analysis demonstrated that the loss of intact β-catenin was more rapid in the cell nucleus (β-catenin T1/2 of 1.5 h in cytoplasm and 0.5 h in nucleus). Although β-catenin has been shown to be a co-activator of the T cell transcription factors Lef and Lc, these studies have been restricted to normal immature T cell. Down-regulation of β-catenin-associated transcription was also an early event in response to anti-Fas.

**Conclusion** The data suggest that β-catenin, which is not expressed in normal mature T cells, may play a role in promoting Jurkat survival.