SIII-3-2

Differential regulation of TNF- or TRAF2-mediated NF- κ B activation by cellular inhibitor of apoptosis protein c-IAP2

Tae Ho Lee Department of Biology, Yonsei University

Cellular inhibitor of apoptosis protein c-IAP2, which interacts with TNF receptor associated factor TRAF2, has been implicated in the regulation of NF- κ B activation and anti-apoptotic signals elicited by tumor necrosis factor α (TNF). To establish a functional interplay between c-IAP2 and TRAF2, we generated several truncated forms of c-IAP2 and demonstrated that functionally distinct domains of c-IAP2 differentially modulate TNF- and TRAF2-mediated NF- κ B activation. In addition, we identified a novel variant form of c-IAP2, which may indicate the presence of multiple mechanisms for regulating NF- κ B activation or anti-apoptotic signals through c-IAP2.

Down-regulation of Tcf-1 and Surface TCR/CD3 Complex during Apoptosis of T-cell

Sunjoo Jeong*¹, Sung H. Jeon and Rho H. Seong Institute for Molecular Biology and Genetics, Seoul National University, ;¹Department of Molecular Biology, Dankook University, Seoul 140-714,

Tcf-1 is a transcription factor expressed mainly in developing thymocytes and mature T cells. When an apoptotic cell death was induced in T-cell hybridoma and mature T-cells, the expression of Tcf-1 mRNA was greatly reduced. However, cells did not display such a change when a necrosis-like cell death was induced. In addition, a mutant line of T-cell hybridoma, which produces IL-2 but is resistant to apoptosis upon activation did not show any reduction of the Tcf-1 expression. Therefore, the reduced expression of Tcf-1 is specific for the apoptotic process of T cells. Apoptotic T-cell hybridoma also expressed $CD3-\varepsilon$ mRNA and cell surface TCR/CD3 complex at a reduced level, all of which seemed to be due to the reduced expression of Tcf-1. Furthermore, cells with overexpressed Tcf-1 did not show any reduction in TCR proteins, suggesting that Tcf-1 is responsible for down-regulation of cell surface TCR/CD3 complex during activation-induced apoptosis of T-cells.