## SIII-3-2

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

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Cellular inhibitor of apoptosis protein c-IAP2, which interacts with TNF receptor associated factor TRAF2, has been implicated in the regulation of NF-κB 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-κB activation. In addition, we identified a novel variant form of c-IAP2, which may indicate the presence of multiple mechanisms for regulating NF-κB activation or anti-apoptotic signals through c-IAP2.

## SIII-3-3

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

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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-ε* 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.