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Regulation and Characterization of Ice/Ced-3 (Caspase)-Mediated Apoptosis

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The interleukin-1β converting enzyme (ICE) family of proteins (caspsases), homologs of the C. elegans cell death gene product CED-3, play important roles in controlling vertebrate programed cell death. Because inhibition of apoptosis is an essential step in tumorigenesis, the interaction of the simian virus 40 large T antigen (Tag) with the ICE family was examined. Expression of T ag alone significantly prevents the ICE-induced apoptosis. p53, but not pBb, or p107, antagonizes the effect of T ag on the sippression of ICE-induced cell death, but not ICH-1L-mediated cell death. Thus wild type p53 may potentiate ICE-induced apoptosis. By using a temperature sensitive mutant p53 Val135 in COS-1 cell, induction of bax, p21, or cyclin D1 gene expression was observed in the cells and molulation of the cell's susceptibility to death will be discussed.

Regulation of ICE-mediated apoptosis by growth factor was wxamined in Rat-1 cells expressing ICE-lacZ, mutant ICE-lacZ, and Bcl-2. SErum-deprivation induced cell death was suppressed in the cell expressing mutant ICE. Activation of ICE observed in the cells underging of Bcl-2 family. These results shows the possibe link between ICE family and growth factor for regulation of apoptosis. In additon, death sustrates of ICE/CED-3 family were identified and characterized.