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Effect of Etoposide-Induced JNK Activation upon the Anti-Apoptotic Action of Bcl-xL

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The bcl-x gene, a member of the bcl-2 family, regulates apoptosis. The alternative mRNA splicing of the gene gives rise to two proteins, Bcl-xL, a dominant inhibitor of apoptotic cell death and Bcl-xS, an apoptosis-promoting protein. Like Bcl-2, Bcl-xL is an intracellular membrane-associated protein containing all the BH4, BH3, BH1, BH2 and TM domains. Here, we showed that expression of Bcl-xL suppressed the c-Jun N-terminal kinase (JNK) activation, nuclear fragmentation and cell death caused by etoposide treatment in N18TG neuroglioma cells. In this study, stable expression of JNK1 at least partly antagonized the anti-apoptotic environment attained by overexpression of Bcl-xl, which implies that the down-regulation of JNK signaling pathway may be involved in the anti-apoptotic effect of Bcl-xL. The possibility that the Bcl-xl or Bcl-2 induces its anti-apoptotic effect in a slightly different mechanism was discussed.

Key words) *anti-apoptotic action/Bcl-xL/etoposide/JNK/*