



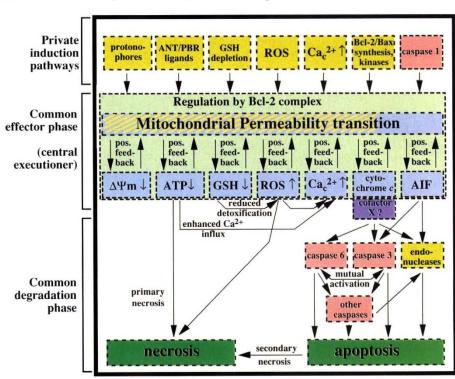
The proto-oncogene Bcl-2 and its role in regulating apoptosis

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Nature Medicine 3, 614-620 (1997)

Owing to a production error, Figure 3 was presented incorrectly. The correct presentation is provided below.

Fig. 3 The apoptotic process and its regulation by Bcl-2. Different apoptosis-triggering pathways employ distinct ("private") signal transduction pathways that will culminate in the induction of permeability transition (PT) and/or associated phenomena. Bcl-2 inhibits the induction of PT in response to most of these inducers (shaded area), with the notable exception of posttranslational modifications of the Bcl-2 protein itself (proteolysis by HIV protease and hyperphosphorylation in response to cytoskeleton-targeted chemotherapeutic drugs) and ICE-like caspases. The exact mechanisms by which Bcl-2 inhibits PT is unknown. Several of the consequences of PT $(\Delta \Psi_m$ disruption, AIF and cytochrome c release, changes in redox potentials and so on) favor PT, thereby creating positive feedback loops. Bcl-2 could either act to inhibit opening of the PT pore or disrupt one or several of the self-amplifying feedback loops (for example, by influencing the release of cytochrome c). Apoptogenic factors are released from the mitochondrial intermembrane space and leak into the cytosol. In addition, PT causes major changes in cellular redox potentials (depletion of nonoxidized glutathione and hyperproduction of superoxide anion), energy metabolism (depletion of NAD(P)H2 and ATP) and ion compartmentalization.





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