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incl. Portable Version.Apoptosis is a program of
controlled cell suicide that plays a central role in tissue
development and self-renewal and that goes awry in
disease and under some forms of physiological stress.
Although apoptosis is commonly triggered via extrinsic
cues provided by the immune system, a wide variety
of intrinsic signals have also been linked to
programmed cell death. These include the p53 and
p73 tumor suppressor proteins, which exert their
effects via common target genes, and members of the
recently defined BCL-2 family of proteins. The BCL-2
family can be divided into 2 groups based on the type
of cell death they induce. The "classical" BCL-2
proteins (BCL-2, BCL-XL, and MCL-1) promote survival
and prevent apoptosis in most cells. By contrast, the
BH3-only proteins, which include BID, BAD, BIK, NOXA,
PUMA, and HRK, all members of the BCL-2 family
implicated in apoptosis, lack the BCL-2 homology
region (BH) and, thus, cannot directly bind to classical
BCL-2 family members. Rather, they may function by
forming heterodimers with the former proteins,
thereby freeing the classical BCL-2 family members
from their anti-apoptotic interactions. Each BH3-only
protein is multifunctional and exerts a context-
dependent cellular outcome, presumably by
associating with different combinations of BCL-2 family

members in different cell types. Although BH3-only proteins and classical BCL-2 family proteins regulate apoptosis in opposing ways, they often have complementary functions. The Bcl-2-interacting killer (BIK) gene was discovered in a search for novel apoptosis regulators (Agarwal et al., 1995 6d1f23a050

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