



Apoptosis-inducing peptides as novel therapeutics for cancer treatment (*Jianjie Ma, RWJ 05-08, 05-169*) *Oncology*

ATAPs (Amphipathic Tail-Anchor Peptides)

The new cancer therapeutic peptides, ATAPs induce apoptotic cell death of diverse cancer cells. ATAPs contain a single amphipathic transmembrane segment and specific targeting property to mitochondrial membrane. By these two linked properties (the amphipathicity and targeting signal peptide), ATAPs specifically accumulate at the mitochondrial membrane and then disrupt mitochondrial membrane potential, causing acute apoptosis of cancer cells. Apoptosis induced by ATAPs is not blocked by anti-apoptotic Bcl-xL and Bfl-1 proteins that are involved in resistance of cancer cells against various cancer drugs. Therefore, these ATAPs have high potential as a new cancer drug.

PLP (Presenilin-2 Loop Peptide)

Presenilin-2 is a critical component of γ -secretase which is related to Alzheimer's disease. It is a membrane protein with eight predicted transmembrane (TM) domains and a hydrophilic loop of approximately 120 amino acids between the sixth and seventh TM domains. Presenilin-2 loop region could be cleaved by presenilinase and caspase -3 into a small, 22 amino acid peptide, presenilin-2 loop peptide (PLP). Through our combination of biochemistry, confocal microscopy, and cell culture method, we found that PLP when coupled with a membrane penetrating peptide (TAT) can readily enter the cell, and cause perturbation of intracellular Ca homeostasis. Cells treated with TAT-PLP (~1 μ M) quickly undergo apoptosis within 6 hours after treatment. As control, a scrambled peptide, TAT-PSP, is not effective in triggering apoptosis. TAT-PLP interacts with the IP3 receptor Ca release channel located on the endoplasmic reticulum, and triggers release of cytochrome c release from mitochondria. These results demonstrate that PLP can be used as a potential therapeutic agent for the treatment of cancer cells.

Patent Status

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