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Educational Corner

Mitochondrial signals and energy requirement in cell death

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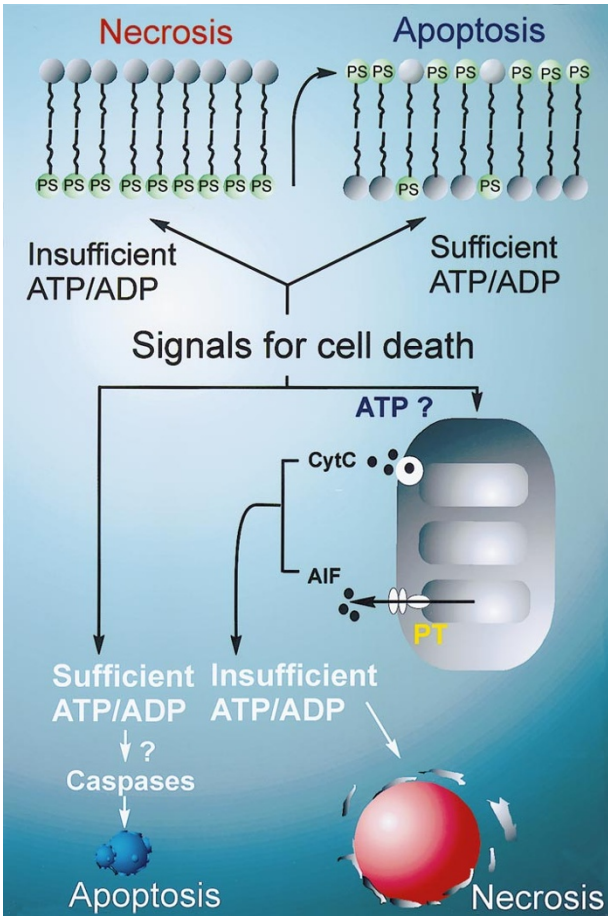

Although apoptosis and necrosis are considered as morphologically and conceptually distinct forms of cell death there is increasing evidence that some early events may be common in both. Downstream effectors may then determine the mode of demise. Recent work suggests that mitochondria can release molecules that contribute to the execution of cell death. An apoptosis inducing factor, AIF is released by mitochondria undergoing permeability transition (PT), whereas holocytochrome C (CytC) seems to be released by energised mitochondria. Notably, mitochondria and cell energy charge seem to be irreversibly compromised in necrosis, but not in apoptosis of neurons. Also, the type of demise caused by classic apoptotic triggers can change from apoptosis to necrosis when cells are preemptied of ATP. At least two distinct steps, the typical apoptotic nuclear degradation and the expression on the cell surface of annexin V-recognisable phosphatidylserines (PS) seem to require energy for execution. This suggests that while upstream signals may be common to both types of cell demise the residual intracellular energy may decide the shape of death and the implications for the neighbouring tissue.

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Further Reading

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