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Commentary

Mechanisms Involving in Cell Apoptosis

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DESCRIPTION

The initiation of apoptosis is tightly controlled by activation mechanisms, because apoptosis inevitably leads to cell death. The intrinsic pathway (also known as the mitochondrial pathway) and the extrinsic pathway are the two most well-studied activation mechanisms. The intrinsic pathway is activated by intracellular signals generated when cells are stressed and is dependent on the release of proteins from the intramembranous space of mitochondria. The extrinsic pathway is activated by extracellular ligands binding to cell-surface death receptors, resulting in the formation of the Death-Inducing Signalling Complex (DISC).

In response to stress, a cell initiates intracellular apoptotic signaling, which may result in cell suicide. Nuclear receptor binding by glucocorticoids, heat, radiation, nutrient deprivation, viral infection, hypoxia, increased intracellular concentration of free fatty acids, and increased intracellular calcium concentration (for example, due to membrane damage), can all cause a damaged cell to release intracellular apoptotic signals. A number of cellular components, including poly Adenosine Di Phosphate (ADP) ribose polymerase, may also aid in the regulation of apoptosis. Single cell fluctuations have been observed in stress-induced apoptosis experiments.

Apoptotic signals must cause regulatory proteins to initiate the apoptosis pathway before the actual process of cell death is precipitated by enzymes. This step allows those signals to cause cell death or to halt the process if the cell no longer needs to die. Several proteins are involved, but two main modes of regulation have been identified: targeting mitochondrial functionality or directly transducing the signal to apoptotic mechanisms *via* adaptor proteins. An increase in calcium concentration within a cell caused by drug activity, which can also cause apoptosis *via* a calcium binding protease chaplain, has been identified as an extrinsic pathway for initiation in several toxin studies.

Intrinsic pathway

The mitochondrial pathway is another name for the intrinsic pathway. Mitochondria are required for multicellular life. Without mitochindria, a cell's aerobic respiration stops and dies quickly. Hence, mitochondria are called as “Power house of the cell”. This fact serves as the foundation for some apoptotic pathways. Apoptotic proteins that target mitochondria have varying effects on them. They may cause mitochondrial swelling by causing membrane pores to form, or they may increase the permeability of the mitochondrial membrane, allowing apoptotic effectors to leak out. They are very closely related to the intrinsic pathway, and tumours arise more frequently through the intrinsic pathway than through the extrinsic pathway due to sensitivity.

Extrinsic pathway

The extrinsic pathway is usually the first pathway that is to be activated during the coagulation process, and it is triggered by a protein called tissue factor, which is produced by cells found outside of blood arteries. The extrinsic pathway that initiates apoptosis is triggered by a death ligand binding to a death receptor, such as TNF-α to TNFR1. TNF receptor-1 for example, interacts with TNF to induce the recruitment of adaptor proteins such as Fas-associated protein with death domain and Tumor necrosis factor receptor type 1-associated death domain protein which recruits a series of downstream factors, including Caspase-8, a critical mediator of the extrinsic pathway, ultimately leading to cell apoptosis.

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