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Apoptosis: Programmed Cell Death

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Editorial

Although many components of the apoptosis idea had been clearly defined many years before, the name apoptosis (a-po-toe-sis) was originally used in a now-classic work by Kerr, Wyllie, and Currie in 1972 to characterise a physically different form of cell death. In multicellular organisms, apoptosis is a type of planned cell death. Characteristic cell changes (morphology) and death are the result of biochemical processes. Blebbing, cell shrinkage, nuclear fragmentation, chromatin condensation, chromosomal DNA fragmentation, and global mRNA degradation are all examples of these alterations. Apoptosis kills between 50 and 70 billion cells every day in the ordinary adult person.

Unlike necrosis, which is a type of traumatic cell death caused by acute cellular damage; apoptosis is a carefully regulated and controlled process that provides benefits to an organism throughout its life cycle. Unlike necrosis, apoptosis results in the formation of apoptotic bodies, which phagocytic cells may engulf and remove before the contents of the cell flow out and cause harm to neighbouring cells.

Apoptosis is a highly controlled process since it cannot be stopped once it has started. Apoptosis can be triggered by one of two mechanisms. The intrinsic pathway involves a cell killing itself in response to cell stress, whereas the extrinsic pathway involves a cell killing itself in response to signals from other cells. Weak external signals can potentially trigger apoptosis via the intrinsic route. Both routes cause cell death by inducing the activation of caspases, which are proteases, or protein-degrading enzymes. Both routes activate initiator caspases, which in turn activate executioner caspases, which destroy the cell by indiscriminately degrading proteins.

Defective apoptotic processes have been linked to a range of illnesses, in addition to their relevance as a biological phenomena. A high level of apoptosis promotes atrophy, whereas a low level produces uncontrolled cell growth, which can lead to cancer. Some variables, such as Fas receptors and caspases, induce apoptosis, whereas others, such as members of the Bcl-2 protein family, prevent it.

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