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Commentary

Regulation of Cell Survival by Apoptosis

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DESCRIPTION

The precisely regulated, energy-dependent process of cell death known as apoptosis is also referred to as "planned cell death." A series of distinctive biochemical processes known as apoptosis induction cause changes in cellular shape and cell death. Blebbing, cell shrinkage, nuclear fragmentation, and DNA fragmentation are all signs that a cell is going through apoptosis. Unlike necrosis, which results in the release of cellular contents, apoptotic cells generate apoptotic bodies that are phagocytized by nearby cells. Apoptosis can be induced by a variety of factors, including ischemia, hypoxia, exposure to specific medications and chemicals, immunological reactions, infectious agents, extreme temperatures, radiation, and other disease states. It also plays significant roles in physiology and pathology.

A cell typically undergoes apoptosis when its nucleus and cytoplasm condense, its DNA is broken down into numerous small (200 bp-250 kbp) fragments, the plasma membrane blebs noticeably, and, in the final stages, the cell breaks down into numerous intact fragments that are eaten by phagocytes before the cellular contents have a chance to escape. The significant condensation and breakdown of the nucleus into many, highly stained spheres distributed throughout the cytoplasm is likely the most notable morphologic hallmark of apoptosis. This stands in stark contrast to the process of necrosis, where the nucleus typically swells to fill the majority of the cytoplasm and the staining intensity of the chromatin decreases rather than increases. Other incredibly distinctive characteristics of apoptosis are the blebbing of the plasma membrane and the fragmentation of the cell into many apoptotic entities. The death of the cell is likely removed by surrounding phagocytes through phagocytosis, which is facilitated by the collapse of the cell into apoptotic bodies, which maintain membrane integrity for several hours. Apoptosis essentially stands for a clear and effective method of eliminating undesired or unhealthy cells while reducing an inflammatory reaction.

Pituitary adenoma cells that are going through apoptosis show a typical shared route of alterations. Based on the unique pattern of nucleosomal fragmentation, apoptosis can also be identified using DNA and biochemical studies.

The major target for apoptosis detection is provided by intranucleosomal DNA breakage, which creates lengths of about 180 base pairs with several additional 3OH ends. The TUNEL method relies on terminal deoxynucleotidyl transferase's inclusion of labelled deoxyribonucleotide triphosphates (TdT). An alternative test is *in situ* end-labeling, which involves tailing of the 3′ -OH ends by Klenow DNA polymerase.

Apoptosis has been demonstrated to occur with low frequency in a subgroup of pituitary adenomas, despite the fact that the data are not consistent. Alternative methods based on caspase activity modifying the cytoskeleton can be used to detect apoptosis in its early phases. Apoptosis must be taken into account in any study of nervous system development since it is crucial to both the healthy development of the mammalian nervous system and the harm caused by a variety of toxins. We apologies for not being able to mention all pertinent publications given the size of recent research articles in this topic. A sophisticated network of protease pathways called apoptosis is used to selectively kill off cells. Simple linear definitions of apoptosis are possible, however the activity of each route can be precisely controlled at various stages. The results of a main apoptotic signal can be affected by other types of cell survival regulation, such as autophagy. Alternately, autophagy may serve as the major signal that triggers apoptosis, which kills cells. As a result, after many years of research, there are still numerous unsolved concerns regarding how apoptosis controls cell life.

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