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Opinion Article

Role of Apoptosis in Cancer Development and its Treatment

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DESCRIPTION

Apoptosis is a natural mechanism of cell death, a promising target for cancer treatment. In both the endogenous and extrinsic pathways, caspases are used to perform apoptosis by cleaving hundreds of proteins. In cancer, the apoptotic pathway is usually blocked by a variety of drugs, including overexpression of anti-apoptotic proteins and under expression of pro-apoptotic proteins. Many of these changes cause intrinsic resistance to the most common cancer treatment, chemotherapy. A promising new anti-cancer therapy is a plant-derived compound that exhibits anti-cancer activity by activating the apoptotic pathway.

Apoptosis is the natural mechanism of programmed cell death cells. It is especially important in long-lived mammals because it plays an important role in both development and homeostasis. It is a highly regulated process that helps to eliminate unwanted cells. There are various conditions that activate the apoptotic pathway, such as DNA damage and uncontrolled proliferation. Apoptotic pathways are activated by both intracellular and extracellular signals. There are two different pathways leading to apoptosis, intrinsic and extrinsic which correlate with the type of signal transduction. They are also called mitochondrial receptor pathways. Intracellular signals include DNA damage, growth factor deprivation, and cytokine deprivation, but the most common extracellular signals are by cytotoxic T cells of the immune system in response to damaged or infected cells, the signal that causes death of the unwanted cells. The path converges on the execution caspase. Programmed cell death or inactivation of apoptosis is centre to the development of cancer. This inactivation of the apoptotic response may contribute significantly to both treatment and the observation that apoptosis is not the primary mechanism of cancer cell death in response to common treatment regimens in many tumors.

Apoptosis can be induced in cancer cells *via* endogenous and extrinsic pathways that converge on the regulation of caspase-dependent proteolysis of thousands of cellular proteins, membrane vesicle formation, and cleavage by endonucleases of chromosomal DNA. The morphological changes in apoptotic cell death that affect both the nucleus and cytoplasm are markedly similar between cell types and species. It usually takes several hours from the onset of cell death to the final fragmentation of cells. However, the time required depends on the cell type, stimulation, and apoptotic pathway. Morphological features of nuclear apoptosis are chromatin condensation and nuclear fragmentation, which are associated with cell roundness, decreased cell volume, and pseudopodia contraction. Chromatin condensation begins around the nuclear envelope, forming a crescent-shaped or ring-shaped structure. Chromatin continues to condense until it divides within cells with an intact membrane, a feature called karyorrhexis. The plasma membrane is intact throughout the process. In the late stages of apoptosis, some morphological features include organelles, and hyper structural modification of cytoplasmic cancer is a series of genetic conversions of normal cells into malignant cells while avoiding cell death. It is one of the essential changes in one cell that causes this malignant change in organelles and the loss of membrane integrity, which can be considered as the result of changes.

A limited number of FDA-approved anticancer drugs directly target the apoptotic pathway. These small molecules were designed to inhibit the anti-apoptotic members of the BCL-2 family. Other promising therapeutic strategies for activating apoptosis in cancer cells include drugs that induce exogenous apoptosis pathways, drugs that target tumor suppression pathways or tumor microenvironments, and drug combination therapies. The antitumor effects of several FDA-approved drugs that target the cell survival and proliferation pathways of cancer cells also depend on their effects on the apoptotic signaling pathway. Both cell-mediated immunotherapy and immune checkpoint inhibition induce cancer cell apoptosis *via* extrinsic pathways. The potential for enhancing this effect with combination therapies (targeted therapy, cell proliferation inhibitors or radiation therapy) is currently being investigated.

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