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Perspective

Role of Apoptosis in Cancer Treatment

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DESCRIPTION

Apoptosis is programmed cell death that maintains a healthy balance between survival and death in metazoan cells. Apoptotic defects can cause cancer and autoimmunity, and enhanced apoptosis can cause degenerative diseases. Apoptotic signals helps to maintain genomic integrity, but apoptotic defects can promote carcinogenesis. Apoptosis signals are complex and regulated at multiple levels. Carcinogenic signals regulate key checkpoints in apoptotic signalling pathways such as Inhibitor of Ant proliferative Protein (IAP) and FLICE Inhibitory Protein (cFLIP). Tumor cells can use any of several molecular mechanisms to suppress apoptosis and acquire resistance to apoptotic agents. It contributes to the elimination of unwanted cells in order to maintain a healthy balance between cell survival and cell death in multicellular organisms. For animals, especially long-lived mammals, it is important that multiple physiological and pathological death signals must be integrated. Although there is evidence that inadequate apoptosis can manifest itself as cancer or autoimmunity accelerated cell death is evident in acute and chronic degenerative diseases, immunodeficiency, and infertility. Under many stress conditions, such as precancerous lesions, activation of DNA damage checkpoint signalling helps to eliminate potentially harmful DNA-damaged cells through induction of apoptosis and prevent carcinogenesis. Cancer cells carry changes that impair apoptotic signalling and promote tumor development and metastasis. This section outlines the mechanisms by which major regulatory molecules regulate apoptosis in normal cells and describes a model of apoptosis deregulation based on altered functions that promote the avoidance of apoptosis in cancer cells. It also briefly describes the development of some promising cancer treatment strategies based on targeting apoptosis inhibitors and stimulating apoptosis signals.

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. Intracellular signals include DNA damage, growth factor depletion, and cytokine depletion, but the most common extracellular signals are cytotoxic to the immune system in response to damaged or infected cells. It is a death-causing signal produced by T cells. The path converges on the execution caspase. Cancer features are present in all cancer cells, regardless of cause or type. These include uncontrolled growth, angiogenesis, and avoidance of apoptosis. Cancer prevention is one of the main functions of apoptosis. Normally, cancer inhibits the endogenous pathway, but there are various drugs to inhibit apoptosis. Due to the loss of apoptotic control, cancer cells survive longer, increase the on-going invasiveness of the tumor, stimulate angiogenesis, deregulate cell proliferation, and accumulate mutations that can interfere with differentiation. You can give a lot of time. Upregulation of the anti-apoptotic BCL2 protein and loss of BAX and/or BAK are the primary work arounds. BCL2 is not considered an oncogene, but mutations in BCL2 increase the incidence of tumors. Overexpression of the BCL2 protein is present in more than half of all cancers, regardless of type.

Cancer cells avoid apoptosis by a variety of mechanisms. De*via*tions from normal signalling pathways can result in either survival-promoting or apoptosis-promoting regulation. Although not so classified, survival-promoting genes are potentially carcinogenic and may carry mutations that increase their expression. In this sense, pro-apoptotic genes can function as tumor suppressors. All inhibitors and activators were found outside the normal range of expression in cancer cell lines. For example, almost half of all human cancers show elevated BCL2 expression. There are numerous inhibitors of both apoptotic pathways that are overexpressed in tumors. Increased expression of anti-apoptotic proteins such as BCL2 and down regulation of pro-apoptotic proteins such as BAX are two methods that cells can use to resist apoptosis. Apoptotic defects allow tumor cells to resist traditional treatments such as chemotherapy and radiation therapy. This is done by raising the threshold required for cell death.

Aplasia 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. A limited number of FDA-approved anticancer drugs directly target the apoptotic pathway. These small molecules are designed to inhibit the anti-apoptotic members of the BCL2 family.

Other promising therapeutic strategies that activate cancer cell apoptosis include agents that induce exogenous apoptosis pathways, agents that target tumor suppressor 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 signalling pathway. Both cell-mediated immunotherapy and immune checkpoint inhibition induce apoptosis of cancer cells *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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