CONTROL OF CELL DEATH PROCESS

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Abstract: *This in the article cell death process biological the essence of it management mechanisms and organism life in the activity importance illuminated. Cell death main types — apoptosis, necrosis and autophagy processes scientific sources based on analysis. Also, the cell death in management participation provider genes, signaling molecules and enzymes functional tasks seeing published. In the article cell death process immune system, tissues update and various diseases, including oncological and degenerative diseases in development place about information given. Research results cell death mechanisms deep study modern medicine and biology sciences in development important importance has that shows.*

Keywords: *Apoptosis, plasma membrane, cell, DNA, cytochrome C, necropathological, receptor, T-lymphocyte.*

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One of the most important physiological and biochemical processes in living organisms is **apoptosis**. Apoptosis (*from the Greek for "leaf shedding* ") is programmed cell death, the term being introduced into science by Kerr in 1972. Characteristic signs of the process: release of phosphatidylserine from the inner layer of the cytoplasmic membrane to the outer (monolayer) layer, release of cytochrome C into the cytoplasm through mitochondrial intermembrane transport, activation of cysteine proteinases (caspases), production of active forms of oxygen, formation of bulges in the cytoplasmic membrane, reduction in cell size, breakage of DNA strands in the nucleus between nucleosome sites, condensation of chromatin, separation of the

nucleus into parts, fragmentation of the cell into vesicles containing apoptotic bodies.

Apoptosis is triggered by receptors on the plasma membrane. This type of cell death occurs under the influence of the destroyer receptors, which belong to the group of receptors for tumor necrosis factor - Fas -SDS or ARO-1. Fas-protein is located in the plasma membrane, it is found in various tissues of organs: thymus, liver, heart, kidneys. Its ligand FasL is co-expressed by cytotoxic T-lymphocytes (T-killers) and natural killer (NK) cells.

These two cell types are different. T cells undergo training before destroying an infected cell, while natural killer cells do not.

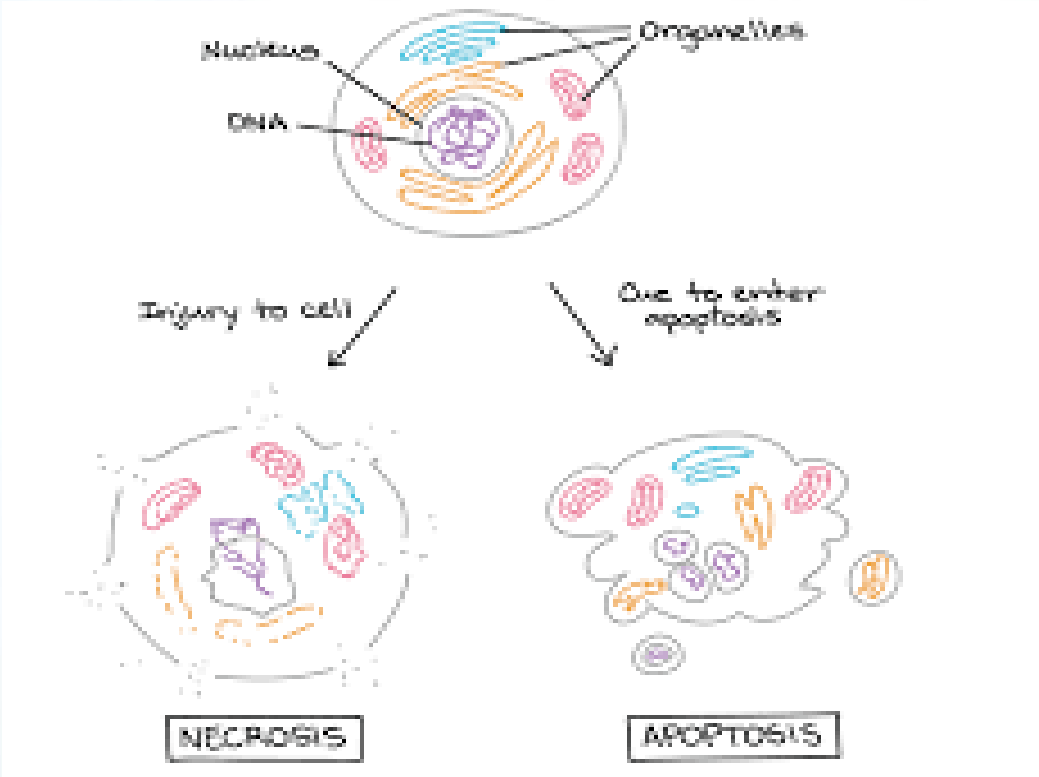


Figure 1. Apoptosis

In Fas-dependent apoptosis, cytotoxic T lymphocytes attack cells infected with viruses and bacteria, while killer T lymphocytes attack tumor cells. This apoptosis regulates lymphocyte homeostasis. In general, organs prone to inflammation are the eye and the male gonads. FasL is found mainly in the cornea, iris, ciliary body cells, and spermatozoa. If activated, diseased T lymphocytes enter these organs, FasL attacks them and the cells die.

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The body affect apoptosis. It is known that FasL binds to the apoptosis-inducing receptor (SD 95G' Aro-1) and plays an important role in immune homeostasis. Dysregulation of the Fas receptor leads to tumor metastasis and autoimmune lymphoproliferative syndrome (3). A decrease in glutathione levels in cells occurs during stress- or substance-induced cell death. When the relationship between glutathione depletion and apoptosis was studied, the idea that glutathione protects cells from apoptosis was suggested (8), but this mechanism has not been fully understood. To date, two families of transporters have been identified that are involved in the flow of GSH across the plasma membrane. These are the ABC gene-encoded (MRP) protein (ABC transporters) and the SLCO gene-encoded (OATP) organic anion transporter – polypeptide protein. During apoptosis, the depletion of GSH in the cell is accompanied by the activation of glutathione active transport, which contributes to the development of apoptosis [7,8,11,16].

Jurkat cells, it was not involved in glutathione release at all. Organic anion transporter (SLCOG'OATP) polypeptides are important in the transport of endogenous and exogenous organic components across the membrane. These transporters accept glutathione and convert it to organic anions. GSH release is enhanced by taurocholic acid, estran sulfate, which are substrates of SLCOG'OATP.

Therefore, SLCOG'OATP converts GSH to GSHGA'OA ion, and when its amount decreases, it leads to programmed cell death.

Glutathione depletion predicts early apoptosis. Subsequent studies have investigated the relationship between glutathione release, the formation of reactive oxygen species, and the development of apoptosis ( ). Depletion of glutathione in the cell leads to the production of hydrogen peroxide, superoxide anion, hydroxyl radical, and lipid peroxide, which are among the reactive oxygen species. At the same time, the antioxidant capacity against reactive oxygen species (ROS) is lost. Depletion of GSH leads to the production of

ROS. Interestingly, high intracellular thiols (GSH and N-acetylcysteine) prevent apoptosis. Apoptosis is a highly structured process characterized by a sequence of biochemical and morphological changes. The early phase of apoptosis is characterized by the formation of reactive oxygen species, which leads to changes in ionic homeostasis, cell shrinkage, and loss of the membrane lipid layer. The late phase is characterized by the activation of caspases and endonucleases, the formation of apoptotic bodies, and the fragmentation of the cell.

Apoptosis can be described as a change in the oxidative process in a cell, but its full mechanism is not clear.

Necrosis is the death of tissues and cells in pathological processes. There are several different causes of necrosis. They are five to the group to be possible.

1) Traumatic necrosis . Physics and chemical factors (mechanical , temperature) radiation acid and alkalis) of tissue and to cells directly impact as a result come comes out .

2) Toxic necrosis . Toxic insects , poisonous snakes and bacteria toxins to the tissues impact as a result come comes out .

3) Neurological necrosis central and pre-firic nerve system of tissues in diseases come comes out .

4) Allergic necro . immune reaction sensitivity increase or decrease as a result come comes out .

5) Blood veins necrosis blood rotation system , arterial and venous blood in the veins come comes out.

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