NOVATEUR PUBLICATIONS
JournalNX- A Multidisciplinary Peer Reviewed Journal
ISSN No: 2581 - 4230
VOLUME 8, ISSUE 3, Mar. -2022

PROGRAMMED CELL DEATH THE ROLE OF MITOCHONDRIA IN THE OCCASION OF APOPTOSIS

Ikromova Muxlisa Akram qizi
Karshi State University Biology Student, Uzbekistan
Ikrom_ch@rambler.ru

Hamzayeva Nargiza Rajabboyevna
Karshi State University Biology Student,
Uzbekistan

Sattorova Iroda Yangiboyevna
Senior Lecturer, Karshi State University, b.f.n.,
Uzbekistan

Cho'liyev Ikrom Ne'matullayevich
2.Senior Lecturer, Karshi State University, b.f.n.,
Uzbekistan

ANNOTATION:

This article is devoted to programmed cell death - apoptosis. The death of a cell after it has performed all its vital functions is apoptotic. This is a natural process. In doing so, the molecules that make up the cell are gradually broken down, allowing other cells in the same organism to use them. Apoptosis cannot be equated with necrosis. In apoptosis, the cells that kill the cells are genes. Apoptosis is a programmed death of a cell that is controlled by suicidal genes. The process of apoptosis is not rapid and it is different from necrosis. First the mitochondria, then the nucleus, and finally the cytoplasm breaks down into fragments, that is, the cell divides and forms apoptotic bodies, which are phagocytosed or dissolved by macrophages. Apoptosis, as an immunomodulatory form of cell death, plays a stabilizing role in maintaining the number of cells in the body. Although much is known and proven about apoptosis, the role of mitochondria in their origin is insufficient. Therefore, this article provides information on the effect of mitochondrial dysfunction on the development of programmed cell death - apoptosis.

Keywords: cell, mitochondria, nucleus, cyclosporine A, sensitive bribe, embryogenesis, apoptosis, necrosis.

INTRODUCTION:

A number of scientific studies have shown that mitochondria play an important role in the regulation of cellular processes among cell organelles. The process of cell apoptosis occurs in cells that have completed their activity. In recent years, it has been reported that mitochondria play a key role in apoptosis.

In 1972, the first apoptosis was performed. These authors appeared in a classic science fiction article written by Kerr, Wiley Kerry. Kerr and others describe the morphological shape of the cell death itself.

Although these features have already been described in more detail, these authors were the first to name the event.

Apoptosis is a type of programmed cell death. As a result of apoptosis, the cell divides into apoptotic cells. In apoptotic lesions (average 90 minutes), macrophages or adjacent cells prevent inflammation.

The term "apoptosis" in greek "apoptosis" in Uzbek means "leaf breakage." This means programmed cell death. The death of a cell after it has performed all its vital functions is apoptotic. This is a natural process. In doing so, the molecules that make up the cell are gradually broken down, allowing other cells in the same organism to use them. Apoptosis cannot be equated with necrosis. Necrosis is an unplanned destruction of a cell that results not only in the cell itself but also in other adjacent cells. The process opposite to apoptosis is not controlled by the necrosis cell control system, resulting in chaotic metabolic processes in the cell, the hydrolytic activity of lipolytic and proteolytic enzymes maximizing the hydrolysis of proteins and lipids.

LITERATURE REVIEW:

In apoptosis, the cells that kill the cells are genes. Apoptosis is a programmed death of a cell that is controlled by suicidal genes. The process of apoptosis is not rapid and it is different from necrosis. First the mitochondria, then the nucleus, and finally the cytoplasm breaks down into fragments, that is, the cell divides and forms apoptotic bodies, which are phagocytosed or dissolved by macrophages. Apoptosis plays an important role in embryogenesis (Elmore, 2007). In this process, a violation of apoptosis leads to various cases of stenosis, atresia (narrowing or closure of the openings of the intestine, esophagus). There is a normal balance between mitosis and apoptosis. When apoptosis accelerates or slows down, this balance is disturbed and can lead to disease. At present, various tumors are explained by apoptosis.

RESULTS:

There is a wide range of information about apoptosis and there are many theories about apoptosis.

Mechanism of apoptosis. In a multicellular organism, apoptosis can occur in two years:

1) Along external signals;
2) Mitochondrial signaling.

In vertebrates, the programmed cell pathway through which the chondroendal signal is transmitted. The mitochondrial signaling pathway of apoptosis occurs as a result of the release of apoptogenic proteins into the cell cytoplasm. There are two reasons for this:

1. Rupture of mitochondrial membranes;
2. As a result of increased permeability of mitochondrial membranes.

Apoptotic Bcl-2-proteins (Bach and Bakh proteins) play an important role in increasing the permeability of mitochondrial membranes. Bcl-2-proteins sit on mitochondrial membranes and release into the cytoplasm the proteins involved in apoptosis - cytochrome C, procaspase and AIF-flavoproteins. The cytoplasm is involved in the formation of cytochrome c APAF-1 protein as apoptosomal organelle. APAF-1 is mediated by the addition of procaspase-9 protein to chromosome c to form capoptosomes. Caspase-9 proteins are found in the apoptosomas cytoplasm, which are combined with procaspase-3 proteins to form caspase-3 protein, which is involved in apoptosis (Salvesen2, 2010). AIF-flavoprotein caspase vapor caspase oxyls from alkaloids are involved in apoptosis. Caspase-9 forms a caspase caspase in the cytoplasm of the cell. The main function of caspases is to break down all the organelles in the cell. Caspases are involved in the destruction of nuclear membranes, cytoskeletal proteins, and intercellular connections.

Apoptosis genetic control is controlled by two genes. For example, if the proto oncogen BC2 inhibits the onset of apoptosis, the suppressor R53gen amplifies it. Increased levels of [Ca2 +] in the cell and activation of endogenous nuclease and stressor N5B70 genes are thought to be important in the mechanism of apoptosis.

Apoptosis involves many physiological processes, including:

1) Programmed destruction of cells during embryogenesis;

2) Hormone-related involution, such as endometrial, pancreatic involution;

3) Necessary for the emergence of processes such as the death of cells responsible for immunity after the release of quinine.

Not surprisingly, apoptosis is a mechanism that causes the death of auto reactive T cells in the developing thymus and leads to the phenomenon of negative selection. Apoptosis is also observed in pathological processes. For example, in toxic or viral hepatitis, the formation of acidophilic Kaunsilmen cells in the liver is one of them. It is noteworthy that this phenomenon is observed in tumors of various origins. However, apoptosis cannot be limited to the participation of the organism in certain stages of individual development. If a virus enters a cell, the cells that have become the nucleus of the electrolyte are destroyed by apoptosis. As a result, the virus is transmitted to healthy cells located near the cell, which can lead to infection.

Depolarization of the mitochondrial membrane is one of the first signs of apoptosis. Both depolarization and apoptosis are lost by inhibitors of the formation of "pores" (holes) in the inner membrane of the mitochondria. The above-mentioned "bribes" are very interesting in the mitochondria. For some reason, the inner membrane of the mitochondria does not pass hydrogen, potassium, sodium and chlorine, but through the formation of "bribes" begins to pass small molecules and non-ionized substances with a mass of less than 1.5 k Da. Cyclophilinkate is analyzed for the formation of pores, and cyclosporine A is used for its formation. When broken down, the cell is transformed from a "power plant" that stores useful energy into mitochondria, a "furnace" that oxygenates nutrients.

Kremer and his staff found that apoptotic changes in the structure of the nucleus accumbens, including the chromosome, are called apoptotic changes. Such changes are observed in the presence of organic hydroperoxides or carbon chloride-phenyl hydrazine, which are formed in the mitochondria "bribes", cyclosporine A and protein Vsl2, which are considered to be the causative agents of the formation of "bribes", have apoptotic effects.

The "apoptotic" effect has been shown to occur when the mitochondria are swollen in a hypotonic solution or when digotin is left under a detergent. In both cases there are ruptures in the outer membrane of the mitochondria. Based on these data, the authors hypothesized that there may be a factor between the outer and inner membranes of the mitochondria that attacks the nucleus and causes "apoptosis." Studies have shown that rupture of the outer membrane causes the factor to travel from there to the nucleus, injuring the nucleus and causing "apoptosis." Their guess was confirmed. The factor between the mitochondrial membranes was found to be a protein with a molecular weight of 50 kDa. When this protein is purified and added to the nucleus, a typical "apoptotic" effect is observed in the cell (Cohen, 1997).

Kumar S., Harvey N.L. and Thormberry N.A., Molineaux S.M. [1995] found the invention of a specific inhibitor of a new protein called apoptosis. It was found to be one of the protease inhibitors that convert N-benzyloxycarbonyl-val-ala-asp-ftormethyl-ketone (Z-VAD * fmk) to prointerleukin - 1 b to interleukin - 1 b.

Subsequent experiments have shown that when Z-VDD * fmk is added to a cell, it stops "apoptosis" and that this effect is specific not only to mammalian cells, but also to insect cells.

DISCUSSION"

Apoptosis is the programmed death of these cells. This process is important for maintaining a normal physiological state. The main goal of programmed cell death is to maintain the balance of cell proliferation. For example, erythrocytes or blood cells are destroyed in our body almost every day through the death of 5x 1011 cells 11. In addition, it allows the body to create a defense mechanism against cells that can be affected. In the case of cells that are victims of viral infection, they are usually killed by programmed cell death. Thus, the virus cannot continue to spread within the host.

In addition to apoptosis, there is another type of programmed death in cells called necrosis. This death is mainly due to the development of pathological conditions. Necrosis [wool. necrosis - death of a cell, tissue, whole organ or part of a living organism. Chemical or physical exposure (temperature, electricity, strong acids or alkalis, light energy, etc.), injury, toxins (bacterial infections). In most cases, local circulatory disorders (thrombus, embolism, heart attack, gangrene), as well as tissue innervation, create favorable conditions for necrosis.

Specific changes in necrosis occur in cells (the nucleus and cytoplasm twist) and in the intercellular substance. The area around the necrosis separates (the cell nucleus and cytoplasm do not shrink) or p purulent; a scar appears in the place of the cell defect.

Necrosis can be dry or coagulation (dead cell dries), wet or collicative (dead cell decomposes). The presence of N. in the body of a organism indicates a general state of well-being(https://biosfera.uz/ensiklopediya/nekroz/).

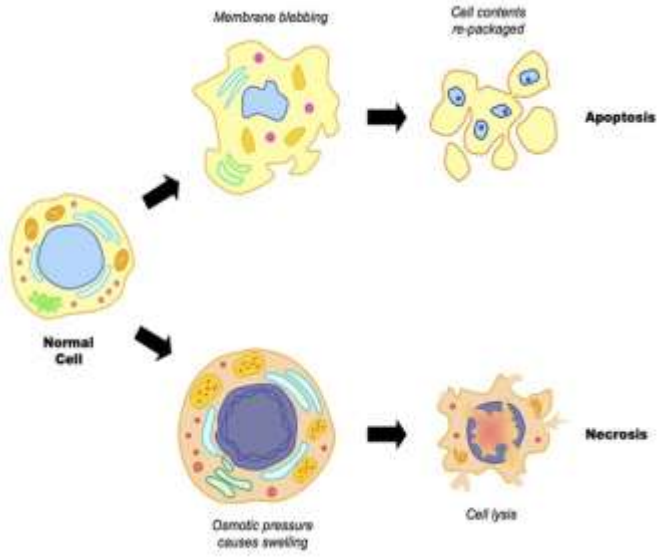

Figure 2. Cell apoptosis and necrosis.

Process Control:

A relatively high-level apoptosis-controlled active process is a necrosis-toxic process that is a passive victim of a cell-independent death regime. As we have said, the available evidence suggests that necrosis is not regulated.

Mortality:

Usually occurs in a single cell or in a small cell cluster, necrosis occurs in a permanent part of the cell.

Status of the Plasma Membrane:

At apoptosis the cell membrane remains intact and apoptotic bodies remain in the cytoplasm. The necrosis breaks down the plasma membrane and separates the cytoplasm.

Inflammatory Processes:

Inflammation is not observed in apoptosis; inflation is one of the most striking features of necrosis. The membrane transmits chemotactic signals that are absorbed by the airway due to the inflammatory process.

Using traditional histological methods, it is not possible to differentiate between apoptotic and gynecologic necrosis. The

morphological consequences of death, developed by necrotic and apoptotic pathways, differ in several respects, and in others they differ from each other.

Evidence suggests that apoptosis and necrosis represent a morphological expression of a general biochemical pathway called neoplastic necrosis. For example, the apoptotic pathway undergoes two changes in necrosis: a decrease in intracellular caspases and ATPs.

CONCLUSION:

Based on the above, we have come to the following conclusions:

Apoptosis is the death of well-preserved and highly controlled cells involved in the removal of unwanted, excess, aged, or damaged cells. Eventually, as a result of the regulation of apoptosis, mutated cells can emerge, which can lead to malformations, autoimmune diseases, and even cancer. Abnormal apoptosis can also lead to the destruction of healthy cells that occur in health problems such as infections, hypoxic-ischemic injury, neurodegenerative or neuromuscular diseases, and AIDS.

It differs from apoptotic necrotic cells. Necrosis of the cell leads to the formation of phagocytes, which can lead to a sharp inflammatory reaction due to the loss of extracellular tissue, damage to organs and the proliferation of cytoplasmic contents. In contrast, apoptosis is often thought of as cell suicide. Studies have shown that cells that have undergone apoptosis maintain the formation and function of membrane membranes and organelles in the event of puffing of the plasma membrane, a decrease in cytoplasmic volume, chromatin condensation, and nuclear fission.

In the final stages, the cells wrapped around the plasma membrane are converted into apoptotic bodies, which are phagocytosed by healthy cells. Removal of cell debris occurs even in the absence of an inflammatory reaction, which means that apoptotic cells can be rapidly and efficiently destroyed and that peptose can be found in the cells. However, 50% of cells in puberty can undergo apoptosis, and less than 10% of cells are simultaneously apoptotic.

REFERENCES

1) Cohen, G. M. (1997). Caspases: the executioners of apoptosis. Biochem J , 1-6.
2) Elmore, S. (2007). Apoptosis: A Review of Programmed Cell Death. Toxicol Pathol. , 35(4): 495–516.
3) Salvesen2, S. B. (2010). Regulation of the Apaf-1–caspase-9 apoptosome. J cell Sci , 123(19): 3209–3214.