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SOME ASPECTS OF APOPTOSIS AND INFECTIOUS DISEASES

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Abstract: Apoptosis, as an immunomodulatory form of cell death, plays a stabilizing role in maintaining the optimal number of cells in the body. The biological essence of cell death in infectious pathology is not sufficiently illuminated, and the concept of the negative and positive significance of apoptosis in the pathogenesis of diseases is presented ambiguously. The review focuses on the classical caspase-dependent apoptosis of innate and adaptive immune cells, the reactivity of which is associated with the initiation and outcome of infectious processes. A number of pathogens of bacterial and viral infections are capable of being triggers or inhibitors of apoptosis in the eukaryotic host cell, avoiding the action of immune system factors.
Keywords: Apoptosis, neutrophils, immune system, infectious diseases, macrophages, lymphocytes.

INTRODUCTION

Apoptosis, an immunomodulatory form of cell death, is a genetically determined cell suicide process that is activated in response to cellular stress or damage, as well as in response to evolutionary signals [1]. It has been proven that apoptosis plays an important stabilizing role in maintaining the optimal number of cells in a multicellular organism [2]. Despite the 20-year period of intensive study of apoptosis [4], the biological essence of cell death during infectious pathology has not been sufficiently elucidated to date.

MATERIALS AND METHODS

Apoptosis remodels tissue during normal growth and development and regulates labile cell populations such as epithelial cells, lymphocytes, dendritic cells, neutrophils and monocytes/macrophages. Since its description, cell death has been identified as the main fundamental biological phenomenon that is important for the regulation of tissue homeostasis, changes in which contribute to the development of pathology [2]. This regulated, energy-expensive process can be initiated through two different mechanisms, each of which is based on the successful activation of pre-existing quiescent cysteine-aspartate proteases, or caspases.

RESULTS AND DISCUSSION

As suggested [4], the intrinsic mitochondrial pathway of apoptosis begins in the cell when toxic damage, including oxidative stress, causes a decrease in the mitochondrial membrane potential, leading to the opening of the pores of the mitochondrial membrane and the release of cytochrome C and other substances into the cytoplasm. In contrast, the extrinsic pathway is initiated by extracellular events through cell surface binding receptors to the tumor necrosis factor superfamily of death ligands, including TNFα and Fas ligands. Although the intrinsic apoptotic pathway involves early activation of caspase-9 and the extrinsic pathway is mediated through caspase-8, both lead to activation “executive” caspase-3, various proteases and endonucleases [4]. Apoptosis can be characterized as the orderly disassembly of the cell from the inside, as a result of which chromosomal DNA is split into oligonucleosomal segments, the nucleus is divided into discrete subunits, and the cell itself is broken into multiple membrane-surrounded fragments, the outer surface of which is marked with a large number of phosphatidylserine molecules, which leads to their rapid absorption by phagocytes.

The initiation of infectious and inflammatory processes is directly related to the reactivity of innate immune cells (neutrophils, monocytes/macrophages, dendritic cells), which constitute the first line of defense against pathogens and provide the basic level of immunity in response to infection of the body [5]. Some pathogens are capable of being triggers or inhibitors of apoptosis in the host eukaryotic cell, avoiding the action of immune system factors. On the other hand, the host may use apoptosis in an attempt to protect itself from pathogens.

In addition to the innate immune cells involved in the initiation of the infectious process and determining the body's defense reactions, pathogen-associated damage to target organ cells is important in the development of diseases. Therefore, apoptosis plays a significant role in cellular host-pathogen relationships, which depends on the nature of the pathogen, cell type and intensity of infection.

To date, a number of bacteria and viruses have been identified as mediators of apoptosis in vitro. Programmed cell death in the parasite-host system includes at least three pathogenetic strategies [2]: 1) apoptosis as a mechanism for the destruction of host cells; 2) apoptosis as a trigger for inflammation; 3) apoptosis as a host defense mechanism.

Despite the fact that programmed cell death has been historically studied in eukaryotes, it is assumed that it also occurs in prokaryotes, both during the life cycle of the development of certain bacteria and for the removal of defective cells from the bacterial population in response to a wide variety of stresses [3]. Although beyond the scope of this review, it is worth noting the physiological role of apoptosis in bacteria, which may influence the development of infectious diseases. For example, autolysis, the most common type of apoptosis in bacterial cells, is used during sporulation of Bacillis subtilis, the cell wall of which can be an obstacle to spore germination. Bacterial apoptosis maintains biofilm homeostasis by balancing the number of dead and viable cells. Some apoptotic molecules can be detected in bacteria, such as common structural components - lipopolysaccharides in gram-negative bacteria [2], lipoteichoic acid in the cell wall of gram-positive bacteria and lipoarabinomannan in mycobacteria, as well as virulence factors such as such as exotoxins, cytolysins and hemolysins. Through these different molecules, bacteria are capable of triggering both intrinsic and extrinsic apoptosis pathways in different types of intracellular and extracellular bacteria.

CONCLUSION

Further research into identifying the molecular elements by which pathogenic agents interfere with cell death pathways will enable the development of new therapeutic approaches, including inhibition of apoptosis, that can be used to prevent the progression of severe infectious diseases.

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