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MOLECULAR MECHANISMS OF INFLAMMATION

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Abstract

The inflammatory process is a complex, multi-stage protective biological response of the body to infectious agents, mechanical injuries, chemicals and immunological disorders. This process is aimed at limiting tissue damage, eliminating pathogenic factors and restoring homeostasis, and is carried out through mechanisms that are precisely controlled at the molecular level. In recent years, the development of molecular biology, immunology and genetic research has allowed us to better understand the pathogenesis of inflammation.

This article systematically analyzes the main molecular mechanisms involved in the initiation, development and resolution stages of the inflammatory process. In particular, the detection of PAMP and DAMP molecules released from pathogens and damaged cells by Toll-like receptors and other pattern-recognition receptors, the activation of intracellular signaling cascades (NF-κB, MAP kinases, JAK/STAT) and the increase in the synthesis of inflammatory mediators are highlighted.

The biological significance of cytokines, chemokines, prostaglandins, leukotrienes, and nitric oxide in the inflammatory process, their effects on vascular permeability, leukocyte migration, and tissue damage are also reviewed in detail. Along with the protective function of oxidative stress and reactive oxygen species in the destruction of pathogens, the role of their excess in causing damage to cells and tissues is also analyzed.

The article also discusses the genetic and epigenetic regulation of the inflammatory process, the influence of microRNAs on cytokine expression, and the importance of resolution mediators that ensure the physiological termination of inflammation. These data are of significant scientific and practical importance for a deep understanding of the mechanisms of inflammation, the prevention of diseases associated with chronic inflammation, and the development of modern anti-inflammatory therapeutic strategies.

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Keywords
Inflammation, cytokines, mediators, NF-κB, prostaglandins, immune response,

Relevance of the topic: Currently, it has been scientifically proven that the inflammatory process is closely related not only to classical infectious diseases, but also to many chronic and non-infectious pathologies. The inflammatory process plays an important pathogenetic role in the development of cardiovascular diseases, diabetes, rheumatoid arthritis, allergic diseases, autoimmune conditions, and oncological diseases. Therefore, in-depth study of the mechanisms of inflammation is one of the most relevant scientific directions for modern medicine and biological sciences.

In recent years, advances in molecular biology, cell biology, and immunology have made it possible to study the inflammatory process at the molecular level. In particular, the identification of Toll-like receptors located on the cell membrane, cytoplasmic signaling systems (NF-κB, MAP-kinases, JAK/STAT), and mechanisms controlling gene expression have become the basis for a new interpretation of the pathogenesis of inflammation. By studying these mechanisms, it is becoming possible to precisely control the initiation, development and termination of inflammation.

Identifying the molecular mechanisms of action of inflammatory mediators - cytokines, chemokines, prostaglandins, leukotrienes and reactive oxygen species - is of great importance in the development of anti-inflammatory drugs. Currently, widely used non-steroidal anti-inflammatory drugs and glucocorticosteroids have many side effects, and there is a need to replace them with more effective and targeted molecular therapies. This requires in-depth study of the molecular basis of inflammation.

Also, the association of chronic inflammation with oxidative stress, genetic and epigenetic changes has a long-term negative impact on human health. The identification of specific mediators that provide the resolution phase of the inflammatory process and the study of their biological significance expands the possibilities of physiologically controlling inflammation in the body. This is of significant scientific and practical importance for the fields of regenerative medicine and prevention.

Taking into account the above factors, the study of the molecular mechanisms of the inflammatory process is one of the current issues of modern biomedical science, and research in this area serves to develop methods for early diagnosis of diseases, effective treatment and individual therapy.

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Purpose of the topic: The main goal of this research work is to comprehensively and systematically study the mechanisms of the inflammatory process at the molecular level, to scientifically analyze the interrelationships of cellular signaling pathways, biologically active mediators and genetic and epigenetic regulation mechanisms involved in this process. It is intended to gain a deep understanding of its pathogenesis by elucidating the stages of the initiation, development and resolution of the inflammatory process from a molecular point of view.

One of the important goals of the study is to study the mechanisms of recognition of pathogenic and damaging factors by cell receptors, in particular, the activation of PAMP and DAMP molecules through Toll-like receptors and other pattern-recognition receptors. At the same time, one of the main directions of the research is to determine the effect of intracellular signaling cascades - NF-κB, MAP-kinases and JAK/STAT systems on the synthesis of inflammatory mediators.

Another important goal of this work is to assess the biological role of cytokines, chemokines, prostaglandins, leukotrienes and reactive oxygen species involved in the inflammatory process, to scientifically analyze their effect on vascular permeability, leukocyte migration and tissue damage. Also, one of the important goals of this research is to determine the role of gene expression, microRNAs and epigenetic mechanisms in the transition of inflammation to chronic forms.

Based on the scientific conclusions obtained as a result of the research, it is planned to create a theoretical basis for the development of modern molecular therapeutic approaches aimed at controlling the inflammatory process, including targeted drugs and individual therapy strategies. At the same time, one of the ultimate goals of this work is to reveal the possibilities of natural control of inflammation in the body by highlighting the importance of resolution mediators that ensure the physiological termination of the inflammatory process.

Main part: The inflammatory process is a complex, multi-stage set of molecular and cellular reactions that begin under the influence of a factor that damages the body. At the heart of this process are components of the innate and adaptive immune system, intracellular signaling pathways, and biologically active mediators.

Initiation of inflammation: pathogen recognition the inflammatory process is initiated primarily by pattern-recognition receptors (PRRs) located in damaged tissues. These receptors detect molecular structures specific to pathogens — PAMPs (pathogen-associated molecular patterns) and DAMPs (damage-associated

molecular patterns) released from damaged cells. The most important group of PRRs includes Toll-like receptors (TLRs).

When TLRs are activated, a signal is transmitted inside the cell through adapter proteins such as MyD88 and TRIF, and the expression of inflammatory genes begins. As a result, the synthesis of inflammatory mediators increases dramatically.

Cytokines and their molecular role

Cytokines are the main controlling molecules in the inflammatory process. Proinflammatory cytokines such as TNF-α, IL-1β, and IL-6:

- ✓ increase vascular permeability;
- ✓ activate adhesion molecules (ICAM-1, VCAM-1) in the endothelium;
- ✓ enhance the migration of leukocytes to the site of inflammation.

IL-6, on the other hand, stimulates the synthesis of acute phase proteins (CRP, fibrinogen) in hepatocytes, causing a systemic inflammatory response.

Chemokines and cell migration

Chemokines provide a directing mechanism for the inflammatory process. CXCL8 (IL-8) attracts neutrophils and CCL2 attracts monocytes to the site of inflammation. This process occurs through chemotaxis, which allows leukocytes to cross the blood vessel wall and enter the tissue. Leukocyte migration is mediated by the interaction of selectins, integrins, and adhesion molecules. This step constitutes the cellular component of inflammation.

Phagocytosis and oxidative stress

Neurophils and macrophages that reach the site of inflammation phagocytose pathogens. This process produces reactive oxygen species (ROS) and nitric oxide (NO). Although these molecules are important in killing microorganisms, excessive production can damage healthy tissues.

Oxidative stress damages cell membranes, proteins, and DNA, further aggravating inflammation.

NF-κB and intracellular signaling pathways

The transcription factor NF-κB plays a central role in the molecular control of inflammation. When this factor is activated, the expression of the following genes increases:

- ✓ proinflammatory cytokines;
- ✓ COX-2 and iNOS enzymes;
- ✓ adhesion molecules.

Prolonged NF-κB activity leads to the development of chronic inflammation and autoimmune processes.

Lipid mediators and inflammatory markers

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Prostaglandins and leukotrienes, which are formed from arachidonic acid, play an important role in the development of the clinical signs of inflammation - pain, fever, and edema. Prostaglandin E2 (PGE2) acts on the thermoregulatory center to cause fever, while bradykinin increases pain sensitivity.

Termination of inflammation and resolution

Under healthy conditions, the inflammatory process ends with a resolution phase. At this stage, anti-inflammatory cytokines such as IL-10 and TGF-β are released, macrophages destroy apoptotic cells, and tissue regeneration begins.

If this mechanism is disrupted, inflammation becomes chronic, leading to fibrosis and organ failure.

Conclusion: In conclusion, the inflammatory process is a complex, multi-level protective reaction of the body to external and internal harmful factors, based on which molecular and cellular mechanisms operate in harmony with each other. Although this process is aimed at identifying pathogens, eliminating them and restoring damaged tissues, in uncontrolled or prolonged cases it can cause the development of various pathological processes.

Studies show that Toll-like receptors, cytokines, chemokines and intracellular signaling pathways, in particular the NF-κB system, constitute the molecular basis of the inflammatory process. Through these mechanisms, the production of inflammatory mediators is enhanced, the migration of leukocytes to the site of inflammation is ensured and the immune response is formed. At the same time, excessive production of reactive oxygen species and nitric oxide can increase oxidative stress and damage healthy tissues.

The physiological end of inflammation — the resolution phase — is important for restoring homeostasis in the body. At this stage, anti-inflammatory cytokines are activated, ensuring a balance between cell death and tissue regeneration. If this mechanism is disrupted, inflammation becomes chronic, creating the basis for the development of cardiovascular, autoimmune, metabolic and neurodegenerative diseases. Therefore, a deep study of the molecular mechanisms of the inflammatory process is important not only for understanding pathogenesis, but also for developing modern treatment strategies. Today, the use of biological drugs, cytokine antagonists and drugs directed at signaling pathways allows for effective control of inflammatory diseases. In general, a deep understanding of the molecular basis of inflammation is of great scientific and practical importance in the formation of an individual approach in medical practice, early prevention of diseases and increasing the effectiveness of treatment.

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