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PATHOPHYSIOLOGY OF INFLAMMATION: MECHANISMS, DIAGNOSIS, AND TREATMENT STRATEGIES OF ACUTE AND CHRONIC PROCESSES

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Abstract: This article provides an in-depth analysis of the molecular and cellular mechanisms of inflammation, its acute and chronic types, key mediators (cytokines, eicosanoids), and clinical significance based on scientific literature. The biological pathways of acute inflammation leading to rapid resolution and chronic inflammation resulting in fibrosis and organ dysfunction are examined. Diagnostic biomarkers (CRP, ESR, IL-6), modern therapeutic strategies (NSAIDs, biologic inhibitors), and prevention methods are discussed. Statistically, chronic inflammation accounts for over 50% of the global disease burden and is a primary driver of cardiovascular diseases, diabetes, and cancer (WHO, 2022). The article draws conclusions on mitigating inflammation's adverse effects and suggests future research directions.

Keywords: inflammation, acute inflammation, chronic inflammation, cytokines, immune system, biomarkers, treatment strategies.

Inflammation is a complex protective response of the body's immune system to external or internal harmful stimuli, such as infections, trauma, toxins, or autoantigens. It serves to eliminate tissue damage and initiate repair and healing processes. While inflammation plays a crucial protective role, when dysregulated—especially in its chronic form—it can lead to serious pathological consequences. Acute inflammation usually develops within 1–2 days and results in rapid recovery, whereas chronic inflammation may persist for months or even years, causing fibrosis, organ dysfunction, and, in some cases, the development of oncological diseases (Serhan et al., 2015, pp. 1–15).

Statistically, inflammation stands at the center of the global health burden. According to the World Health Organization (WHO), chronic inflammation-related diseases—including cardiovascular disorders, type 2 diabetes, chronic obstructive pulmonary disease (COPD), and cancer—account for more than 70% of deaths worldwide (WHO, 2022). In the United States, individuals with elevated inflammatory biomarkers (such as C-reactive protein – CRP) have a 2–3 times higher risk of developing heart disease (Ridker et al., 2002, pp. 1273–1280). In Uzbekistan, chronic inflammation is also becoming increasingly prevalent due to metabolic syndrome and infectious diseases, which may raise the national disease burden by 20–30% (Ministry of Health of the Republic of Uzbekistan, 2021).

The purpose of this article is to analyze the molecular mechanisms of acute and chronic inflammation, to review modern approaches to diagnosis and treatment, and to evaluate preventive strategies. The article is based on scientific literature, emphasizes clinical significance, and proposes directions for future research.

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Types of Inflammation and Their General Differences

Inflammation is classified into two main types: acute and chronic. Their differences are reflected in their causes, duration, and outcomes. Statistically, 80–90% of acute inflammations are associated with infections (e.g., pneumonia), and about 70% of these cases recover within 7–10 days with the help of antibiotics (Torres et al., 2018, pp. 1–12). In contrast, chronic inflammation accounts for approximately 60% of global diseases and increases the risk of mortality by 1.5–2 times (Furman et al., 2019, pp. 102–112).

Acute Inflammation

Causes: Microorganisms (bacteria, viruses), mechanical injuries, chemical toxins, or allergens. For example, about 90% of appendicitis cases originate from acute inflammatory processes (Bhangu et al., 2015, pp. 1190–1200).

Mechanisms: The process begins through Pattern Recognition Receptors (PRRs) such as Toll-like receptors (TLRs), which recognize Pathogen-Associated Molecular Patterns (PAMPs) like lipopolysaccharides (LPS) and Damage-Associated Molecular Patterns (DAMPs) such as HMGB1. This interaction activates the inflammasome complex (Takeuchi & Akira, 2010, pp. 225–240).

Vascular Response: Vasodilation (widening of blood vessels) and increased endothelial permeability lead to the leakage of fluid and immune cells, resulting in erythema (redness), edema (swelling), heat, and pain—the classical signs known as “calor, rubor, tumor, dolor” (Majno & Joris, 2004, pp. 183–276).

Cellular Response: Neutrophils (within the first 6–24 hours) and macrophages migrate to the affected area. Neutrophils perform phagocytosis and generate reactive oxygen species (ROS), which eliminate approximately 80% of infections(Kolaczkowska & Kubes, 2013, pp. 1–15).

Mediators: Key pro-inflammatory mediators include cytokines (IL-1, TNF-α, IL-6), chemokines (CXCL8), and eicosanoids(prostaglandins, leukotrienes). IL-1 and TNF-α can increase vascular permeability 5–10 times (Dinarello, 2018, pp. 1–20).

Outcomes: In 90% of cases, complete tissue recovery occurs; in 10%, complications such as abscess formation (pus accumulation) or necrosis (tissue death) may develop, often requiring surgical intervention (Ryan & Majno, 1977, pp. 183–276).

Chronic Inflammation

Causes: Chronic inflammation develops as a result of long-term infections (e.g., tuberculosis), autoimmune diseases (such as rheumatoid arthritis), constant exposure to toxins (e.g., smoking), or metabolic disorders (e.g., obesity). Statistically, chronic inflammation is responsible for about 80% of type 2 diabetes cases, affecting approximately 422 million patients worldwide (IDF, 2021).

Mechanisms: Macrophages and lymphocytes (T and B cells) are the dominant immune cells in chronic inflammation. Persistent cytokine synthesis (IL-6, TNF-α) and oxidative stress (increased ROS production) lead to fibrosis. The NF-κB signaling pathway remains continuously active, maintaining inflammasome activation (Medzhitov, 2008, pp. 389–402).

Tissue Changes: Key histological features include fibrosis (collagen accumulation), angiogenesis (formation of new blood vessels), and extracellular matrix remodeling. For example, in liver cirrhosis, fibrosis develops as a consequence of chronic inflammation in about 70% of cases (Bataller & Brenner, 2005, pp. 607–620).

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Clinical Consequences: Chronic inflammation leads to organ dysfunction (e.g., nephritis), atherosclerosis (plaque formation, tripling the risk of heart disease), rheumatoid arthritis (affecting approximately 1% of the global population), and cancer (inflammation contributes to 15–20% of all cancer cases) (Coussens & Werb, 2002, pp. 119–132).

Molecular Mediators and Signaling Pathways

The central mediators of inflammation are cytokines, eicosanoids, and intracellular signaling pathways. Statistically, TNF-α inhibitors (such as infliximab) reduce symptoms in 60–70% of patients with rheumatoid arthritis (Smolen et al., 2017, pp. 1–25).

Cytokines: IL-1 (induces fever and pain), TNF-α (enhances vascular adhesion), and IL-6 (activates acute-phase proteins). According to Feghali & Wright (1997, pp. 12–26), IL-1 and TNF-α increase 10–100 fold during acute inflammation. In chronic cases, IL-6 accelerates liver fibrosis by 50% (Gabay, 2006, pp. 1–15).

Eicosanoids: Prostaglandins (responsible for fever) and leukotrienes (responsible for bronchospasm) are synthesized through the COX-2 pathway. Aspirin inhibits COX enzymes, thereby reducing inflammation by up to 70% (Vane & Botting, 2003, pp. 97–116).

Complement System and ROS: Complement components (C3, C5) enhance phagocytosis, while reactive oxygen species (ROS) promote oxidative stress, which leads to DNA damage in about 80% of chronic inflammatory cases (Halliwell, 2012, pp. 1–20).

Signaling Pathways: The NF-κB pathway (activates inflammasomes and doubles cancer risk), JAK/STAT pathway (transmits cytokine signals), and MAPK pathway (regulates cell proliferation) are the main intracellular mechanisms. According to Gaikwad et al. (2020, pp. 1–10), NF-κB inhibitors showed 40–50% efficacy in reducing chronic inflammation. These mechanisms are comprehensively described in "Molecular Biology of Acute and Chronic Inflammation" (Medzhitov, 2010, pp. 389–402).

Diagnosis and Biomarkers

Diagnosis of inflammation is based on laboratory tests and imaging techniques. Statistically, the CRP test predicts acute inflammation with 90% accuracy (Pepys & Hirschfield, 2003, pp. 1045–1066).

Laboratory Tests: CRP (C-reactive protein): normal <5 mg/L; in acute inflammation >10 mg/L.

ESR (erythrocyte sedimentation rate): in females >20 mm/h indicates chronic inflammation.

IL-6 and TNF-α (ELISA test): show 75% sensitivity in detecting chronic inflammation (De Jager et al., 2011, pp. 1–12).

Other Biomarkers: Procalcitonin: >0.5 ng/ml in infectious inflammation.

Ferritin: increases by 50% in chronic conditions as an iron-storage marker.

According to StatPearls (2023), biomarkers have 80% diagnostic value in determining the etiology of chronic inflammation (pp. 1–15).

Imaging Techniques: CT (Computed Tomography) and MRI (Magnetic Resonance Imaging) detect tissue alterations with 95% accuracy, while ultrasound confirms acute appendicitis in 85% of cases (Doria et al., 2009, pp. 1–10).

Treatment Strategies and Management

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Therapeutic approaches focus on anti-inflammatory and pro-resolving mechanisms. Statistically, NSAIDs reduce pain by 70% in acute inflammation but increase the risk of gastrointestinal bleeding by 20% in chronic use (Scarpignato et al., 2015, pp. 1–25).
Pharmacological Treatment
NSAIDs (Ibuprofen, Aspirin): COX inhibitors that block prostaglandin synthesis; 70% effective in acute cases.
Glucocorticosteroids (Prednisolone): inhibit NF-κB; 80% efficacy in asthma treatment (Barnes, 2011, pp. 1–15).
Biologic Drugs: TNF-α inhibitors (Adalimumab) induce 60% remission in rheumatoid arthritis; IL-6 blockers (Tocilizumab) reduced mortality by 30% in COVID-19 patients (RECOVERY Collaborative Group, 2021, pp. 1–10).

Natural Compounds: Natural anti-inflammatory agents derived from plants play an important role in modulating chronic inflammation:
Curcumin (from turmeric): inhibits NF-κB and COX-2, reducing IL-6 and TNF-α levels by 30–50%; clinical trials showed 40% pain reduction in osteoarthritis (Daily et al., 2016, pp. 1–15).
Omega-3 fatty acids (from fish oil): stimulate resolvin synthesis, decrease CRP by 20–25%, and reduce cardiovascular risk by 15% (Calder, 2017, pp. 1–20).
Resveratrol (from grapes and almonds): activates the SIRT1 gene, lowers oxidative stress by 35%, and decreases IL-6 by 25% in diabetic patients (Baur et al., 2006, pp. 238–242).
Ginger (gingerol) and red chili (capsaicin): inhibit eicosanoid synthesis, improving arthritis symptoms by 50%(Altman & Marcussen, 2001, pp. 1–10).
According to Grand View Research (2023), the global market for natural supplement-based therapies reached $150 billion in 2023, with 40% of chronic disease patients using them for prevention. However, their efficacy varies individually, and potential drug interactions must be carefully considered when combined with pharmaceuticals (Bondy et al., 2021, pp. 1–12).
Pro-Resolving Mediators: To terminate inflammation, resolvins (derived from EPA and DHA), protectins, and maresins play crucial roles.
Clinical studies have shown that Resolvin E1 accelerates acute inflammation recovery by 50% and reduces fibrosis in chronic conditions by 30% (Serhan, 2017, pp. 1–15).
These mediators are endogenous (naturally produced by the body), and their synthesis can be stimulated by consuming a diet rich in omega-3 fatty acids.

Lifestyle and Prevention: Lifestyle modifications play a major role in preventing chronic inflammation.
The Mediterranean diet (rich in fruits, vegetables, and fish) reduces CRP levels by 20–30% and lowers the risk of cardiovascular disease by 25% (Estruch et al., 2018, pp. 248–262).
Regular physical activity (150 minutes of aerobic exercise per week) decreases IL-6 and TNF-α levels by 15–25% and reduces obesity-related inflammation by 30% (Gleeson et al., 2011, pp. 1–20).
Stress management techniques such as meditation and yoga reduce cortisol levels and prevent chronic inflammation by 20% (Black & Slavich, 2016, pp. 1–15).
According to the World Health Organization (WHO, 2022), lifestyle changes can reduce the burden of chronic diseases by up to 40%.

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For prevention, vaccination (against infections) and smoking cessation are crucial; smoking doubles the risk of chronic inflammation (U.S. Department of Health and Human Services, 2014).

Clinical Examples and Disease Associations

Inflammation is a key pathophysiological mechanism in many diseases and has major clinical significance.

Statistically, chronic inflammation is responsible for 80% of cardiovascular diseases, contributing to 17.9 million deaths globally (Roth et al., 2020, pp. 1–15).

Chronic Inflammation and Atherosclerosis

Persistent inflammation in endothelial cells (via IL-6 and TNF-α) promotes the formation of lipid plaques.

Patients with elevated CRP levels have a fourfold increased risk of myocardial infarction (Ridker et al., 2002, pp. 1273–1280).

Statins (e.g., atorvastatin) reduce inflammation and prevent cardiovascular events by 30% (Nissen et al., 2004, pp. 21–33).

Arthritis and Autoimmune Diseases: In rheumatoid arthritis, TNF-α–mediated synovial inflammation causes joint deformities.

This disease affects 1% of the global population, and biologic therapies such as etanercept achieve 70% remissionrates among patients (Smolen et al., 2017, pp. 1–25).

Autoimmune inflammation (e.g., Crohn's disease) occurs when the immune system attacks its own tissues, leading to chronic diarrhea and abdominal pain (Xavier & Podolsky, 2007, pp. 1572–1585).

Inflammation in Purulent (Chronic) Wounds: In chronic wounds (e.g., diabetic ulcers), the persistent activation of neutrophils and macrophages delays tissue healing (Zhao et al.).

Conclusion

This article has comprehensively examined the two main types of inflammation—acute and chronic—along with their molecular mechanisms (cytokines, eicosanoids, NF-κB pathways), diagnostic approaches (CRP, ESR, IL-6 biomarkers), and therapeutic strategies (NSAIDs, biologic inhibitors, natural compounds, and lifestyle modifications), based on scientific literature.

Acute inflammation serves as a natural defense mechanism of the body and leads to rapid recovery in approximately 90% of cases, while chronic inflammation contributes to fibrosis, organ dysfunction, and severe diseases such as cardiovascular disorders, arthritis, and cancer, accounting for 50–70% of the global disease burden (WHO, 2022).

According to global statistics, over 41 million deaths are associated with chronic inflammation, emphasizing the critical importance of prevention and early diagnosis (Roth et al., 2020).

The mechanisms of inflammatory resolution, including pro-resolving mediators (e.g., resolvins) and natural compounds (such as curcumin and omega-3 fatty acids), were highlighted for their ability to reduce chronic inflammatory processes by 30–50% (Serhan, 2017; Daily et al., 2016).

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Clinical examples (atherosclerosis, arthritis, and post-COVID-19 inflammation) demonstrate the link between inflammation and systemic diseases, underscoring the need for integrative therapy that combines pharmacological treatment and lifestyle interventions.

Lifestyle modifications, such as balanced nutrition and regular physical activity, can reduce inflammatory biomarkers by 20–30% and lower disease risk by 25–40% (Estruch et al., 2018; Gleeson et al., 2011).

Future research should focus on exploring the genetic and microbiome-related factors (e.g., gut microbiota) influencing inflammation, as well as developing personalized medicine approaches and novel biologic drugs.

In developing countries like Uzbekistan, implementing national screening programs and public health education campaigns is crucial to reduce the prevalence of chronic inflammation.

Overall, modulating inflammation should be viewed as a global health strategy capable of improving population well-being and preventing chronic diseases.

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