JOURNAL OF INTERNATIONAL SCIENTIFIC RESEARCH
Volume 4, Issue 2, April 2026 Online ISSN: 3030-3508
https://spaceknowladge.com

# THE DIFFERENCE BETWEEN ACUTE AND CHRONIC INFLAMMATION

**Abdiraimova Vazira Shokir qizi**
*Scientific supervisor:* **Asatullayev Rustamjon Bakhtiyarovich**

**ARTICLE INFORMATION**

**ARTICLE HISTORY:**

*Received:12.04.2026*
*Revised: 13.04.2026*
*Accepted:14.04.2026*

**KEYWORDS:**

*Acute inflammation, chronic inflammation, neutrophils, macrophages, cytokines, resolution of inflammation, specialized pro-resolving mediators, fibrosis, systemic effects, metaflammation.*

**ABSTRACT:**

*Inflammation is the body's fundamental protective response to harmful stimuli such as pathogens, damaged cells, or irritants. It plays a vital role in eliminating the cause of injury and initiating the healing process. However, inflammation manifests in two distinct forms: acute and chronic. Acute inflammation is a rapid, short-term response that is usually beneficial and self-limiting, whereas chronic inflammation is a prolonged, low-grade process that can lead to tissue destruction, fibrosis, and contributes to the development of many serious diseases. This article provides a comprehensive overview of the differences between acute and chronic inflammation, including their causes, underlying mechanisms, clinical manifestations, diagnostic approaches, and management strategies. Understanding these distinctions is essential for accurate diagnosis, effective treatment, and prevention of long-term complications in clinical practice.*

Inflammation is the body's dynamic defense mechanism aimed at restoring homeostasis after injury or infection. It has been classically described by the four cardinal signs: redness (rubor), heat (calor), swelling (tumor), and pain (dolor). While acute inflammation serves as a rapid and protective response that helps contain damage and promote repair, chronic inflammation often becomes maladaptive. It is now widely recognized as a central underlying

factor in numerous non-communicable diseases, including cardiovascular disorders, type 2 diabetes, cancer, autoimmune conditions, and neurodegenerative diseases.

Acute inflammation develops suddenly and lasts for a short period, usually from minutes to hours up to a maximum of two to three weeks. It occurs in response to various triggers such as bacterial or viral infections, trauma, burns, chemical irritants, or allergic reactions. The process begins with rapid vascular changes, including vasodilation that increases blood flow and causes redness and heat, followed by increased vascular permeability that leads to fluid leakage and swelling. Neutrophils are the predominant immune cells involved in the early phase. They migrate to the site of injury, perform phagocytosis, release reactive oxygen species and enzymes, and help eliminate the harmful agent. Molecular mediators such as histamine, prostaglandins, leukotrienes, and pro-inflammatory cytokines (TNF-α, IL-1, IL-6) amplify the response. Importantly, acute inflammation usually resolves completely through an active process mediated by specialized pro-resolving mediators (SPMs) like resolvins, lipoxins, and protectins. These mediators limit further neutrophil recruitment, promote clearance of dead cells by macrophages, and stimulate tissue repair, allowing the affected area to return to normal function without significant scarring.

In contrast, chronic inflammation has a gradual onset and persists for months, years, or even a lifetime. It arises when the acute inflammatory response fails to eliminate the inciting agent, when the stimulus continues to be present, or when the immune system mistakenly attacks the body's own tissues. Common causes include persistent infections such as tuberculosis or viral hepatitis, autoimmune disorders like rheumatoid arthritis and systemic lupus erythematosus, prolonged exposure to irritants like cigarette smoke or industrial dust, and metabolic conditions associated with obesity. In chronic inflammation, the cellular infiltrate consists mainly of mononuclear cells, including macrophages, lymphocytes, and plasma cells rather than neutrophils. Tissue destruction occurs alongside attempts at repair, often leading to angiogenesis and progressive fibrosis or scarring. The process is usually less intense locally but has prominent systemic effects such as fatigue, low-grade fever, weight loss, and anemia of chronic disease. Molecularly, it involves sustained activation of signaling pathways like NF-κB and the NLRP3 inflammasome, resulting in continuous production of cytokines and growth factors. Oxidative stress and changes in synaptic plasticity can further perpetuate the inflammatory cycle.

Clinically, acute inflammation presents with obvious local signs such as redness, swelling, heat, and pain, as seen in a sprained ankle, sore throat during a cold, or acute appendicitis. These signs help clinicians quickly identify and address the problem. Chronic inflammation,

however, often manifests more subtly. It may present as persistent joint pain in rheumatoid arthritis, back pain in inflammatory bowel disease, or contribute silently to plaque formation in atherosclerosis. Many modern diseases are now linked to low-grade chronic inflammation, often called metaflammation in the context of obesity and metabolic syndrome.

The diagnostic process for both types requires a thorough approach. For acute inflammation, a detailed patient history combined with physical examination revealing classic signs is usually sufficient, supported by laboratory findings such as elevated white blood cell count with neutrophilia and a sharp rise in C-reactive protein (CRP). Imaging techniques like ultrasound or computed tomography may be used in specific cases. In chronic inflammation, diagnosis often relies on persistently elevated inflammatory markers such as high-sensitivity CRP, erythrocyte sedimentation rate (ESR), and specific cytokines. Autoantibody tests are important in autoimmune conditions, while biopsy may reveal mononuclear cell infiltrates and fibrosis. Advanced imaging helps assess organ damage or scarring.

Management strategies differ significantly between the two forms. In acute inflammation, treatment focuses on removing the cause and providing symptomatic relief. This includes rest, ice, compression, elevation, nonsteroidal anti-inflammatory drugs (NSAIDs), and antibiotics when infection is present. Most cases resolve spontaneously once the threat is eliminated. For chronic inflammation, the primary goal is to address the underlying cause, whether through antimicrobial therapy, immunosuppressive drugs, or lifestyle interventions. Long-term management often involves anti-inflammatory medications such as corticosteroids, disease-modifying antirheumatic drugs, or biologic agents targeting specific cytokines like TNF-α or IL-6. Emerging therapies aim to promote active resolution using analogs of specialized pro-resolving mediators. Lifestyle modifications play a crucial role and include adopting an anti-inflammatory diet rich in omega-3 fatty acids, regular physical activity, weight reduction, smoking cessation, and stress management. In some cases, multidisciplinary approaches incorporating cognitive-behavioral therapy are beneficial for patients with persistent symptoms.

Understanding the differences between acute and chronic inflammation has important implications in clinical practice. It improves diagnostic accuracy, prevents unnecessary delays in treatment, and reduces the risk of overlooking serious underlying conditions. Moreover, recognizing the role of chronic low-grade inflammation helps in the prevention of many age-related and lifestyle-related diseases.

In conclusion, acute and chronic inflammation represent two ends of the inflammatory spectrum. Acute inflammation is a short-term, protective mechanism that usually resolves

completely and supports healing, while chronic inflammation is a persistent process that can cause progressive tissue damage and drive the pathogenesis of numerous diseases. Accurate differentiation and timely intervention are essential for optimal patient outcomes. Continued research into the resolution pathways of inflammation and the mechanisms of chronic inflammatory states will further enhance our ability to manage these conditions effectively in modern medicine.

## References

1. Guyton and Hall Textbook of Medical Physiology
2. Robbins and Cotran Pathologic Basis of Disease
3. Gray's Anatomy
4. Serhan C.N. et al. Novel Pro-Resolving Lipid Mediators in Inflammation Resolution.
5. Furman D. et al. Chronic inflammation in the etiology of disease across the life span. Nature Medicine, 2019.
6. World Health Organization – Guidelines on non-communicable diseases and inflammation
7. International Association for the Study of Pain (IASP) and related immunology literature