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The dual nature of inflammation: From essential host defense to driver of chronic disease: A review

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Abstract

Inflammation is a fundamental biological process with a dual nature, serving as an essential protective mechanism while also acting as a central driver of chronic disease when it becomes dysregulated. This review explores the critical distinction between acute, self-limiting inflammation and a persistent, damaging chronic state that underpins the pathogenesis of a wide range of pathologies, including cardiovascular disease, cancer, and autoimmune and metabolic disorders. The report examines the fundamental biology of the inflammatory response, detailing the roles of key cellular components and molecular mediators, such as the pro-inflammatory cytokines Interleukin-1β, Interleukin-6, and Tumor Necrosis Factor-alpha. It further highlights how the same inflammatory pathways can drive seemingly disparate diseases and discusses the profound role of chronic inflammation in post-COVID-19 conditions. Finally, the review outlines recent advancements in diagnostics, including the use of both established and novel biomarkers for personalized medicine, and highlights a new era of targeted therapeutic strategies, from specific cytokine inhibitors to innovative nanomedicine approaches, aimed at precisely managing the detrimental effects of inflammation.

Keywords: Inflammation; Acute; Chronic; Infection; Immune Response

1. Introduction: The Dual Nature of Inflammation

Inflammation is a complex and highly coordinated protective response involving a sophisticated interplay of immune cells, blood vessels, and a cascade of molecular mediators [1]. Its fundamental purpose is to eliminate the initial cause of cellular injury, clear away damaged tissues, and initiate the necessary repair processes [1]. In its acute, temporary form, inflammation is a normal and vital part of the body's healing system [2]. This beneficial process is exemplified by the physiological response to a simple injury, an allergic reaction, or a common infection like a cold [3]. This response is typically self-limiting and resolves once the inciting stimulus, such as trauma or infection, has been neutralized [4]. However, this finely-tuned mechanism can become dysregulated, leading to a pathological shift from acute to chronic

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inflammation [5]. Chronic inflammation is a more severe, prolonged state that can persist for months or even years [3]. In this condition, the body's immune system overcompensates and continually responds to an injury or illness that has not resolved, gradually damaging healthy cells and tissues [3]. This chronic systemic inflammation is a central driver in the pathogenesis of a wide array of devastating diseases [6]. These conditions include cardiovascular disease, cancer, diabetes, and various autoimmune disorders [4]. The modern understanding of inflammation, therefore, necessitates a nuanced view, recognizing its essential role in maintaining health as well as its profound contribution to disease when it becomes persistent [1]. Recent research, particularly in the context of post-COVID-19 syndromes, has further underscored the critical importance of understanding and modulating this process [9].

2. The Fundamental Biology of the Inflammatory Response

2.1. Cellular and Vascular Dynamics: The Mobilization of the Immune System

The inflammatory response is fundamentally a mobilization of the innate and adaptive immune systems to the site of injury or infection. This process is governed by a complex interplay of cellular and vascular events. The cellular component of inflammation is dominated by leukocytes, a class of white blood cells that normally reside in the bloodstream [1]. A critical step in the inflammatory cascade is the movement of these leukocytes from the blood into the inflamed tissue, a process known as extravasation [1]. This directed migration is a series of biochemical events that allows immune cells to reach the site of injury and combat harmful stimuli [1]. In cases of acute inflammation, this cellular component is characterized by a rapid and increased movement of granulocytes, while chronic inflammation involves a progressive shift to other types of cells, such as mononuclear cells [1]. Once at the site of inflammation, some leukocytes, acting as phagocytes, engulf and destroy invading pathogens, foreign particles, and cellular debris [1]. This process of phagocytosis is essential for eliminating the cause of the injury and clearing out damaged cells and tissues, preparing the area for subsequent repair [1].

2.2. Molecular Mediators: The Cytokine Network

Table 1 Key Pro-inflammatory Cytokines and Their Functions

Cytokine	Primary Cellular Source	Key Functions
IL-1β	Monocytes, Macrophages, Fibroblasts, Endothelial cells	Induces fever (pyrogen), promotes hyperalgesia, increases production of pain mediators like [10]
IL-6	T helper cells, Macrophages, various other cells	Induces fever (pyrogen), enhances synthesis of secondary mediators, plays a central role in neuronal reaction to nerve injury [10]
TNF-α	T helper cells, Macrophages	Induces fever (pyrogen), regulates apoptotic pathways, activates stress-activated protein kinases (SAPKs), plays a key role in inflammatory and neuropathic hyperalgesia [10]
IFNγ	T helper cells	Plays a role in host defense against pathogens, affects renal ion channels [6]
GM-CSF	T helper cells, Macrophages	Involved in the innate immune response, acts as a growth factor [11]

The inflammatory cascade is orchestrated and modulated by a network of molecular mediators, particularly a class of signaling molecules known as cytokines. These molecules are secreted from various cell types, including immune cells like T helper cells and macrophages, as well as non-immune cells such as fibroblasts and endothelial cells [10]. Cytokines are instrumental in propagating and maturing the inflammatory response. Key pro-inflammatory cytokines that drive early acute responses include Interleukin-1β (IL-1β), Interleukin-6 (IL-6), and Tumor Necrosis Factor-alpha (TNF-α) [10]. These molecules serve multiple, integrated functions. For instance, IL-1, IL-6, and TNF-α can act as endogenous pyrogens, leading to the systemic symptom of fever [10]. They also enhance the synthesis of other inflammatory mediators and stimulate the production of acute phase proteins. A critical function is their ability to chemoattract inflammatory cells to the site of the stimulus, further amplifying the local response [10].

A deeper examination of cytokine function reveals a direct and specific link between the inflammatory response and the clinical manifestation of pain. Pain is one of the cardinal signs of inflammation [3], and this sensation is not merely a consequence of physical tissue damage but is actively mediated at the molecular level. Research has demonstrated that IL-1β, IL-6, and TNF-α can trigger pathological pain, a phenomenon known as hyperalgesia [10]. This is made possible by the presence of receptors for these cytokines on both neurons and their supporting glial cells [10]. For

example, IL-1β is released by monocytes and macrophages during injury, and its expression is upregulated in nociceptive dorsal root ganglion (DRG) neurons following nerve trauma [10]. Studies have shown that the administration of IL-1β can directly induce hyperalgesia and increase the production of pain mediators such as substance P and prostaglandin E2 (PGE2) [10]. This establishes a clear cause-and-effect relationship: the same molecules that signal an immune response also directly sensitize the nervous system, thereby translating the biological state of inflammation into the subjective experience of pain.

3. Acute Versus Chronic Inflammation: A Pathophysiological Divide

The distinction between acute and chronic inflammation is fundamental to understanding their respective roles in health and disease.

3.1. Acute Inflammation: A Rapid and Self-Limiting Response

Acute inflammation is defined by its sudden onset and temporary nature, typically subsiding within a few hours to a few days [3]. It is the body's immediate, self-healing response to tissue damage, injury, or infection. The classic, easily recognizable signs of this response are pain, redness, immobility, swelling, and heat [3]. In this state, the immune system kicks in to fix the injury by rapidly mobilizing immune cells to the affected site [3]. A more severe subset, known as subacute inflammation, can persist for a longer period, ranging from two to six weeks, and is generally associated with more severe injuries or illnesses [3].

3.2. Chronic Inflammation: A Sustained and Damaging Condition

In contrast, chronic inflammation is a more severe, prolonged condition that can last for months or even years [3]. In this state, the immune system's response becomes overcompensating and persistent, continually responding to a perceived threat, even in the absence of a clear injury or illness [3]. This sustained immune activity gradually damages healthy tissue, organs, and cells over time, leading to long-term health complications [3]. Chronic inflammation can be triggered by a number of factors, including the incomplete resolution of an acute inflammatory response, exposure to environmental agents, or internal factors such as genetic variations [6]. It is also a defining feature of many autoimmune diseases, where the immune system mistakenly attacks its own healthy tissues, as seen in conditions like rheumatoid arthritis, psoriasis, and lupus [3].

Beyond the simple binary of acute and chronic, the systemic nature of persistent inflammation reveals a more complex, age-related process. This cumulative burden, termed “inflammaging” describes the increase in systemic chronic inflammation that occurs with age due to unresolved inflammatory events and an individual's unique exposome [6]. This perspective suggests that chronic inflammation is not always a direct, linear outcome of a single failed resolution, but can also be the result of a lifetime of low-grade inflammatory triggers. The presence of this age-related systemic chronic inflammation is associated with specific cytokines, such as CXCL9 and interferon-gamma, and a proposed measurement, iAge, has been shown to correlate with immunosenescence and predict the risk for cardiovascular disease and frailty [6]. This reframing of inflammation as a cumulative, age-dependent process, rather than just a binary condition, highlights a broader understanding of its role in long-term health and age-related disease.

Table 2 Differentiating Acute and Chronic Inflammation

Characteristic	Acute Inflammation	Chronic Inflammation
Duration	Sudden and temporary (hours to a few days)	Prolonged (months to years) [3]
Severity	Subacute subset can last 2-6 weeks	More severe, with long-term damage [3]
Primary Cause	Tissue damage, injury, infection, allergic reaction	Persistent overcompensation to illness or injury, autoimmune attack of self-tissue [3]
Cellular Infiltration	Predominantly granulocytes (e.g., neutrophils)	Progressive shift to mononuclear cells [1]
Common Examples	Bruise, cold, allergic reaction, tendonitis, tonsillitis	Rheumatoid arthritis, psoriasis, lupus, cardiovascular disease [3]

4. Chronic Inflammation as a Driver of Major Diseases

The transition from a beneficial acute response to a persistent chronic state transforms inflammation into a potent driver of various major diseases, including cancer and cardiovascular disease.

4.1. Shared Pathogenesis in Cardiovascular Disease and Cancer

The historical separation of cancer and cardiovascular disease into distinct disciplines is being challenged by a growing body of evidence highlighting their profound pathophysiological commonalities [8]. The link between inflammation and these two major scourges of humanity is not merely coincidental; inflammation contributes to the initiation, progression, and complication of both malignant tumors and atherosclerotic plaques [8]. The convergence of cytokine biology provides a key mechanistic link, with research demonstrating that an intervention targeting a single inflammatory pathway can yield benefits for both conditions. Specifically, the blockade of the pro-inflammatory cytokine Interleukin-1β (IL-1β) has been shown to reduce both cardiovascular events and cancer incidence and mortality [8].

This evidence suggests that the same inflammatory pathways can drive both diseases. The shared pathology is manifested in multiple ways, including the defective resolution of inflammation, extensive extracellular matrix (ECM) remodeling, and the decisive involvement of leukocytes in the progression of both tumor growth and atherosclerotic lesions [8]. This recognition of shared mechanisms has led to the emergence of a new paradigm in medicine that moves beyond traditional disciplinary boundaries. This approach, often associated with the expanded concept of cardio-oncology, suggests that a deep understanding of these shared inflammatory pathways could lead to the development of novel therapeutic strategies that have a dual-purpose role in both the prevention and treatment of heart disease and cancer.

4.2. Inflammation in Metabolic, Autoimmune, and Neurodegenerative Conditions

The damaging effects of chronic systemic inflammation extend beyond cancer and cardiovascular disease to encompass a wide range of other pathologies. The persistent release of pro-inflammatory cytokines from immune cells contributes to the development or progression of autoimmune disorders, such as lupus and rheumatoid arthritis [3], as well as metabolic conditions like diabetes and non-alcoholic fatty liver disease [6]. Neurodegenerative disorders are also intimately linked to inflammation, with persistent inflammatory activity in the central nervous system (CNS) being a central feature of nearly all neurological conditions [12].

A specific example of this systemic connection is observed in Polycystic Ovary Syndrome (PCOS), a condition typically viewed through an endocrine lens. PCOS is associated with a state of low-grade chronic inflammation, which is clinically evidenced by elevated levels of inflammatory markers like C-reactive protein (CRP) and interleukins [13]. This inflammatory state then acts as an intermediary, accelerating the development of atherosclerosis and contributing to endothelial dysfunction, a condition characterized by reduced nitric oxide availability and impaired vasodilation [13]. These vascular changes are the direct precursors to increased cardiovascular risk, such as heart attacks and strokes. The observation that chronic inflammation is the key link between an endocrine disorder and life-threatening cardiovascular pathology, even in women of healthy weight, provides a powerful demonstration of how a disease traditionally defined by one system can be fundamentally driven by systemic inflammation.

4.3. A Modern Perspective: The Role of Inflammation in Post-COVID-19 Syndromes

The COVID-19 pandemic has provided a dramatic, real-world context for understanding the destructive potential of dysregulated inflammation. In severe cases of the disease, a hyperinflammatory state known as a "cytokine storm" can occur, characterized by an uncontrolled overproduction of pro-inflammatory cytokines like IL-6, TNF-α, and IL-1β [6]. This excessive inflammation can lead to widespread tissue damage, particularly in respiratory tissues, and contributes to the severe manifestations of the disease, such as acute respiratory distress syndrome (ARDS) [9].

Crucially, this acute inflammatory response does not always resolve. Chronic inflammation following a COVID-19 infection is now recognized as a pivotal factor contributing to the prolonged and recurrent symptoms of post-COVID-19 conditions, or "long COVID" [9]. This persistent inflammatory state is marked by the continued activation of immune cells and the release of inflammatory mediators long after the acute infection has passed [9]. Studies indicate that this chronic inflammation can lead to scarring and long-term damage in vital organs, including the lungs, heart, and brain, which directly correlates with symptoms such as breathlessness, fatigue, and cognitive dysfunction observed in long COVID patients [9].

5. Recent Advances in Diagnostics and Therapeutics

The profound clinical implications of chronic inflammation have spurred a new era of research focused on advanced diagnostics and targeted therapeutic strategies.

5.1. Novel Biomarkers and Personalized Medicine

Recent advancements have highlighted the instrumental role of biomarkers in the clinical management of inflammatory conditions, particularly in the context of post-COVID-19 syndromes [9]. Biomarkers are moving beyond simple diagnostic tools to become essential guides for personalized therapeutic interventions. Established inflammatory markers, such as C-reactive protein (CRP) and Interleukin-6 (IL-6), continue to be crucial for monitoring persistent inflammatory activity and predicting disease severity and prognosis [9]. In the context of COVID-19, elevated levels of D-dimer, a marker of blood clot formation, are used to identify patients at high risk for thrombotic complications, guiding decisions on extended anticoagulation therapy [9].

Furthermore, recent research has led to the discovery of a host of novel biomarkers that offer deeper insights into specific pathophysiological processes. For example, the soluble urokinase plasminogen activator receptor (suPAR) has been linked to persistent fatigue and endothelial dysfunction, while markers such as KL-6 and SP-D are used to assess the risk of post-COVID-19 pulmonary fibrosis [9]. The clinical utility of these biomarkers is in their ability to provide a more detailed understanding of the specific inflammatory processes at play, allowing clinicians to tailor immunomodulatory treatments and other therapies to the individual patient, thereby reducing unnecessary interventions and optimizing outcomes [9].

Table 3 Established and Emerging Biomarkers in Post-COVID-19 Inflammation

Biomarker	Classification	Clinical Significance
C-reactive protein (CRP)	Established	Signals persistent inflammatory activity; correlates with disease severity and long-term complications [9]
Interleukin-6 (IL-6)	Established	Key driver of hyperinflammation; elevated levels predict progression to severe disease and the persistence of long COVID [9]
D-dimer	Established	Indicates blood clot formation; strong predictor of thrombotic complications and mortality [9]
Soluble urokinase plasminogen activator receptor (suPAR)	Emerging	Linked to persistent fatigue and endothelial dysfunction; may guide therapies for vascular inflammation [9]
Neutrophil extracellular traps (NETs)	Emerging	Offers insights into pathophysiological mechanisms driving long-term symptoms [9]
KL-6 and SP-D	Emerging	Used to assess the risk for post-COVID-19 pulmonary fibrosis [9]

5.2. Targeted Therapeutic Approaches

The identification of specific molecular pathways and mediators in inflammation is driving a fundamental shift in drug discovery, moving away from broad-spectrum anti-inflammatory agents toward highly targeted therapies. Traditional anti-inflammatory drugs often have a complex balance of risks and benefits, with potential for systemic side effects [8]. The new direction in the field is to develop therapies that act on specific molecules and pathways. This is exemplified by the use of cytokine inhibitors, such as monoclonal antibodies that target IL-1β, IL-4, or IL-13, which are now being used to treat a variety of inflammatory diseases, including asthma and atopic dermatitis [8].

Furthermore, research is exploring even more granular therapeutic targets, including non-coding RNAs (ncRNAs) such as long non-coding RNAs (lncRNAs), which have been found to be dysregulated in inflammatory diseases [15]. For instance, lncRNA DLEU2 has been identified as an anti-inflammatory lncRNA that inhibits gut inflammation by negatively regulating the NF-κB signaling pathway [15]. Other novel targets include oncogenes like K-Ras, whose inhibition has been shown to promote inflammation via the RAS/ERK pathway, and transcription factors such as promyelocytic leukemia zinc finger protein (PLZF), which regulates hepatic lipid and glucose metabolism [15]. This intense focus on identifying specific genes, proteins, and signaling molecules represents a strategic shift toward

developing therapies that can address the root molecular cause of an inflammatory disease rather than simply managing its symptoms.

5.3. Nanomedicine and Theranostics

A significant challenge in anti-inflammatory therapy is the problem of systemic toxicity and non-specific targeting, where drugs affect healthy tissues in addition to the inflamed area [4]. Nanomedicine offers a promising solution to this problem through the use of theranostic nanoparticles [4]. Theranostics is an innovative approach that combines diagnostic biomarkers and therapeutic medicines into a single entity with a shared target [4].

These nanoparticles can be engineered to preferentially accumulate in inflamed tissues via a phenomenon known as the Enhanced Permeation and Retention (EPR) effect [4]. Due to the increased permeability of blood vessels in inflamed areas, nanoparticles can passively diffuse into the targeted tissue and accumulate, delivering their therapeutic payload directly to the site of action [4]. This targeted delivery mechanism allows for the use of more potent anti-inflammatory drugs at a therapeutic dose where they are most needed, while minimizing the collateral damage to healthy organs and reducing systemic side effects [4]. The development of nanomedicines represents a critical step toward overcoming the anatomical, physiological, and clinical obstacles that have long limited the efficacy and safety of conventional anti-inflammatory treatments.

6. Conclusion

Inflammation is a fundamental biological process with a dual nature, serving as an essential protective mechanism while also acting as a central driver of chronic disease when it becomes dysregulated. The report has detailed how the finely-tuned, self-limiting acute response can transition into a persistent, damaging chronic state, which in turn underpins the pathogenesis of a wide range of pathologies, from cardiovascular disease and cancer to autoimmune and neurodegenerative disorders. The profound connections between these seemingly disparate conditions, exemplified by shared cytokine pathways and the role of inflammation in conditions like PCOS and post-COVID-19 syndromes, underscore the need for a holistic and interdisciplinary approach to medicine.

The future of managing inflammatory diseases lies in the continued move toward personalized and targeted therapeutic strategies. The integration of advanced biomarkers, both established and emerging, is proving instrumental in identifying specific inflammatory processes, predicting disease progression, and guiding clinical decisions. This diagnostic precision is paralleled by the development of highly specific therapeutic interventions, including a new generation of drugs that target individual cytokines, non-coding RNAs, and transcription factors. Furthermore, innovative technologies such as nanomedicine and theranostics offer the potential to revolutionize drug delivery by ensuring anti-inflammatory agents are precisely targeted to inflamed tissues, thereby maximizing efficacy while minimizing systemic toxicity. Ongoing research in these areas will continue to yield novel lead structures and therapeutic targets, promising a future where the detrimental effects of chronic inflammation can be managed with greater precision and fewer side effects, ultimately improving patient outcomes across a wide spectrum of diseases.

Compliance with ethical standards

Disclosure of conflict of interest

The authors declare that there is no conflict of interests

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