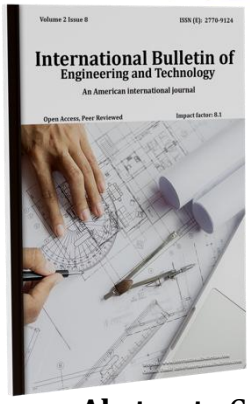

THE ROLE OF CYTOKINES PRODUCED BY LEUKOCYTES IN CARDIOVASCULAR DISEASES

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Abstract: Cardiovascular diseases (CVD) remain one of the leading causes of death worldwide, and inflammatory processes play a key role in their pathogenesis. Cytokines produced by leukocytes are key mediators of inflammation, which play a crucial role in various pathological conditions of the cardiovascular system, including atherosclerosis, myocardial infarction, heart failure, and arterial hypertension. This review article comprehensively reviews the recent scientific evidence on the complex roles of leukocyte-derived cytokines in the pathogenesis of CVD, their types, signaling pathways, and potential therapeutic targets. Pro-inflammatory cytokines produced by macrophages, such as IL-1β, IL-6, TNF-α, and IFN-γ, play a key role in the development of atherosclerosis by promoting endothelial dysfunction, monocyte adhesion and migration, and their differentiation into foam cells. In contrast, anti-inflammatory cytokines such as IL-10 and TGF-β produced by lymphocytes act as a protective factor and limit inflammation. After myocardial ischemia and reperfusion injury, neutrophils and monocytes are rapidly recruited to the myocardium and release a series of cytokines that induce local inflammation, leading to initial myocardial salvage and subsequent coronary reperfusion. However, excessive or prolonged inflammatory responses are detrimental, leading to remodeling and heart failure. In heart failure, pro-inflammatory cytokines exacerbate myocardial dysfunction, hypertrophy, and fibrosis, worsening clinical outcomes. This article also discusses current clinical trials of cytokine-targeted therapies for HF that have shown clinical impact, such as IL-1β inhibitors (canakinumab) and broad-spectrum anti-inflammatory agents such as colchicine. Targeting inflammatory pathways in the treatment of HF offers new strategies. In conclusion, a deeper understanding of leukocyte-derived cytokines offers the potential to shed light on the pathophysiology of CVD and to identify new prognostic markers and therapeutic targets for these serious diseases.

Keywords: leukocytes, cytokines, cardiovascular disease, inflammation, atherosclerosis, myocardial infarction, heart failure, interleukin-6, tumor necrosis factor-alpha, anticytokine therapy

Research Objectives

The aim of this article is to systematically review and comment on the complex role of leukocyte-produced cytokines in the pathogenesis of cardiovascular disease based on scientific articles published between 2015 and 2025. The study aims to determine how different cytokines (pro- and anti-inflammatory) contribute to the initiation and progression of major CVDs such as atherosclerosis, myocardial infarction, heart failure, and arterial hypertension. It also explores the molecular mechanisms and signaling pathways of these cytokines, their clinical relevance, and their potential as novel therapeutic approaches in the treatment of CVD.

Research Methods

To generate this comprehensive review, a search was conducted for articles published between 2015 and 2025 in scientific databases such as PubMed, Scopus, Web of Science, and Google Scholar. The search used key word combinations such as "leukocyte-derived cytokines", "inflammation in cardiovascular disease", "cytokines and atherosclerosis", "interleukin-6 and heart failure", "TNF-alpha in myocardial infarction", "anti-cytokine therapy in CVD", and "immunity in cardiovascular pathology". The search results were limited to English-language articles that primarily included clinical trials, animal models, molecular and cellular studies, as well as systematic reviews and meta-analyses. The bibliographies of the selected articles were reviewed to assess the quality of the data and to identify additional relevant sources. The data obtained were grouped according to the pathophysiological roles of the main cytokines produced by different leukocytes (monocytes/macrophages, neutrophils, lymphocytes) in different types of SLE.

Introduction

Cardiovascular diseases (CVD), including ischemic heart disease, cerebrovascular disease, and peripheral arterial disease, are the leading causes of morbidity and mortality worldwide [1]. While traditional risk factors, such as hypertension, dyslipidemia, diabetes, and tobacco smoking, contribute significantly to the development of CVD, extensive research in recent decades has reinforced the central role of inflammation in the pathogenesis of these diseases and highlighted the important role of the immune system, in particular the innate and adaptive immune systems [2]. As the main effectors of inflammation, leukocytes—neutrophils, monocytes, lymphocytes, and others—are activated at various stages of cardiovascular pathology and secrete a wide range of signaling molecules, including cytokines, chemokines, and growth factors [3]. Cytokines are a broad spectrum of small molecular weight proteins produced by immune cells; they control cell-cell communication, regulate immune and inflammatory responses, and are crucial for maintaining homeostasis [4]. However, their aberrant production or regulation can lead to chronic inflammation, which is a key feature of IBD. Leukocytes, particularly monocytes/macrophages and T lymphocytes, are the main source of many pro- and anti-inflammatory cytokines associated with IBD [5]. These cytokines profoundly affect not only the local immune response but also endothelial function, vascular wall stability, myocardial contractility, and repair processes. Atherosclerosis, the underlying pathological basis of IBD, is a complex process characterized by endothelial dysfunction, lipid uptake, immune cell activation, and chronic inflammation [6]. In the early stages, under atherogenic stimuli, endothelial cells express adhesion molecules, such as vascular cell adhesion molecule-1 (VCAM-1), which induce the adhesion and entry of monocytes and T lymphocytes into the vessel wall [7]. Once in the intima, monocytes differentiate into macrophages and phagocytose substances such as oxidized low-density lipoproteins (oxLDL), transforming them into foam cells—a characteristic feature of atherosclerotic plaques. Activated immune cells, particularly macrophages and T lymphocytes, secrete a variety of cytokines, such as interferon (IFN)-γ, interleukin-1 (IL-1), IL-6, tumor necrosis factor-alpha (TNF-α), and chemokines, which promote inflammation and lesion growth [8].

Acute myocardial infarction (MI) is characterized by the sudden cessation of blood flow to a portion of the myocardium and subsequent cardiomyocyte necrosis. This event triggers a strong inflammatory response in response to myocardial injury [9]. Necrotic cardiomyocytes release “danger signaling” molecules that activate innate immune receptors and lead to the rapid recruitment of neutrophils and then monocytes/macrophages to the injured area [10].

Pro-inflammatory cytokines and chemokines released by neutrophils and monocytes perpetuate inflammation, which, although necessary for initial myocardial salvage and clearance of necrotic tissue, can exacerbate myocardial injury and lead to remodeling [11]. Subsequently, anti-inflammatory signals, such as IL-10 and transforming growth factor-beta (TGF-β), are released, which resolve inflammation and promote vascular repair [12].

Heart failure (HF) is often associated with a chronic inflammatory state, in which elevated levels of circulating cytokines, particularly IL-6, TNF-α, and IL-1β, are observed [13]. These pro-inflammatory mediators induce myocardial dysfunction, promote cardiomyocyte apoptosis, increase hypertrophy and fibrosis, and ultimately lead to deterioration of cardiac output [14]. Although the exact sources of inflammation in HF are not fully understood, various provocations, such as myocardial injury, hypertension, and endothelial dysfunction, can activate local and systemic immune responses. In addition, conditions outside the cardiovascular diagnosis, such as pulmonary congestion and renal dysfunction, can contribute to immune activation [15]. In recent years, large clinical trials such as CANTOS and COLCOT have highlighted the importance of anticytokine therapy in the treatment of HF [16, 17]. Treatment with canakinumab (an IL-1β inhibitor) or colchicine (a broad-spectrum anti-inflammatory drug) significantly reduced the rate of cardiovascular events in patients with CKD, confirming inflammation and its cytokine mediators as viable targets in the pathogenesis of CKD.

This article aims to provide an in-depth analysis of the complex role of cytokines produced by leukocytes in IBD, summarizing the latest scientific data published between 2015 and 2025. This review examines the pathophysiological effects of different cytokines in different forms of IBD, their molecular mechanisms, clinical significance, and potential as therapeutic targets. A better understanding of the relationship between inflammation and IBD will help develop new diagnostic, prognostic, and therapeutic approaches for these serious diseases.

Results

1. Leukocyte and cytokine system

Leukocytes, or white blood cells, are an integral part of the immune system and are divided into several types with different functions: neutrophils, monocytes, lymphocytes, eosinophils, and basophils. Each of these cells produces a specific cytokine profile that plays an important role in the regulation of inflammation and immunity [18]. Cytokines are widespread, small molecular weight proteins and glycoproteins that function as signaling molecules between cells. They can have autocrine, paracrine, or endocrine effects. In the context of IBD, cytokines produced by monocytes/macrophages, T lymphocytes, and to a lesser extent neutrophils are of particular importance [19].

2. Cytokines in atherosclerosis

Atherosclerosis is a chronic inflammatory disease in which immune cells and their cytokine mediators play a crucial role.

IL-1 family: IL-1β, produced primarily by activated macrophages, is a potent pro-inflammatory cytokine that promotes atherosclerosis by inducing IL-6 production and other inflammatory pathways [20]. IL-1α also belongs to this family and has similar effects. The CANTOS study showed that blocking IL-1β (with canakinumab) significantly reduced the incidence of recurrent cardiovascular events in patients with prior myocardial infarction, confirming its clinical relevance in atherosclerosis [16].

IL-6: IL-6 is a key component of atherosclerosis. It is produced primarily by macrophages and endothelial cells and induces inflammation, increased protein synthesis, and activation of immune cells [21]. High circulating levels of IL-6 are associated with an increased risk of CVD. IL-6 signaling is mediated through the JAK/STAT pathway, and although its systemic effects can cause side effects such as thyroid disease and anemia, targeted therapies are the subject of active research [22].

TNF-α: TNF-α is produced primarily by macrophages and T lymphocytes and increases endothelial dysfunction, expression of adhesion molecules, disrupts vascular smooth muscle stability, and promotes apoptotic signaling [23]. In animal models, blocking TNF-α results in a reduction in atherosclerotic lesions. However, although anti-TNF-α therapy is effective in inflammatory diseases such as rheumatoid arthritis, its benefit in SLE is unclear and may increase cardiovascular risk, suggesting a complex role [24]. IFN-γ: IFN-γ is produced primarily by Th1 lymphocytes and natural killer (NK) cells and regulates cellular immunity. In atherosclerosis, IFN-γ promotes the activation and differentiation of macrophages into foam cells, and also inhibits the migration and proliferation of squamous cells, which may reduce plaque stability [25]. Blockade of IFN-γ reduces atherosclerosis in animals, but is difficult to target as a therapeutic in humans due to immunosuppression. Anti-inflammatory cytokines: IL-10 and TGF-β are cytokines with potent anti-inflammatory effects. IL-10 is produced primarily by regulatory T lymphocytes (Tregs) and macrophages and blocks pro-inflammatory cytokines, as well as inhibiting the differentiation of macrophages into foam cells [26]. TGF-β inhibits the migration and proliferation of squamous cells and promotes extracellular matrix deposition, but it also has opposing roles, as it can also promote fibrosis [27]. Deficiency of these cytokines leads to increased atherosclerosis in animal models.

3. Cytokines in acute coronary syndromes and myocardial infarction

Acute myocardial infarction (MI) induces a strong inflammatory response, in which a variety of leukocytes and their cytokines are involved in a temporal sequence.

Neutrophils and early cytokines: In the first hours and days after MI, neutrophils are recruited to the injured myocardium. They secrete pro-inflammatory cytokines such as IL-1β, TNF-α, and IL-6, as well as neutrophil-recruiting chemokines such as IL-8 (CXCL8) [28]. These mediators increase inflammation and promote subsequent recruitment of monocytes. Neutrophils also release reactive oxygen species (ROS) and proteases that can exacerbate myocardial injury.

Monocytes/macrophages and the continuation of inflammation: Neutrophils are followed by monocytes, which differentiate into macrophages at the site of injury. Initially, they are predominantly pro-inflammatory M1 macrophages, secreting IL-1β, TNF-α, IL-6, and chemokines such as monocyte chemoattractant protein-1 (MCP-1/CCL2), which recruit more immune cells [29]. These pro-inflammatory macrophages are important in clearing necrotic tissue by phagocytosis. However, their excessive activity can promote cardiomyocyte apoptosis and extracellular matrix degradation, which increases the risk of cardiac wall rupture.

Inflammation resolution and repair: In the next stage, macrophages switch their phenotype from pro-inflammatory to an anti-inflammatory and pro-fibrotic M2 phenotype. M2 macrophages secrete factors such as IL-10, TGF-β, and ornithine decarboxylase (Arg-1), which suppress inflammation, promote angiogenesis, and initiate fibrosis, which are essential for myocardial repair [30]. IL-10 has been shown to limit inflammation and improve cardiac

function after myocardial injury. Similarly, TGF-β strengthens the heart through fibrosis, but excessive activation can lead to pathological fibrosis and stiffening of the heart.

4. Cytokines in heart failure

Chronic heart failure (HF) is often associated with persistently elevated levels of circulating pro-inflammatory mediators, often referred to as "inflammatory cytokines."

IL-6 family: IL-6 and its receptors have been extensively studied in HF. Elevated levels of IL-6 activate signaling through the JAK/STAT pathway in cardiomyocytes, leading to myocardial hypertrophy, fibrosis, and dysfunction [31]. Blocking IL-6 improves cardiac function in animal models. High IL-6 levels in patients with YY are associated with a poor prognosis.

TNF-α: TNF-α causes a negative inotropic effect on cardiomyocytes, reducing their contractile force. It also promotes cardiomyocyte apoptosis and extracellular matrix remodeling [32]. However, anti-TNF-α therapies (e.g., infliximab and etanercept) have been ineffective or harmful in clinical trials in YY, highlighting the complexity of inflammation in YY and the known physiological roles of TNF-α [33].

IL-1 family: IL-1β is also involved in YY, where it promotes myocardial hypertrophy and fibrosis. Data from CANTOS have shown that blocking IL-1β helps reduce YY [34]. Blocking IL-1β may reduce inflammation and ultimately improve cardiac function.

IL-18: IL-18 is a member of the IL-1 family that enhances IFN-γ production and has pro-inflammatory effects. High IL-18 levels are associated with poor outcomes in YY patients [35]. IL-10: Anti-inflammatory cytokines such as IL-10 may have a protective role in YY. High IL-10 levels are associated with better cardiac function and survival [36].

5. Cytokines in arterial hypertension

Arterial hypertension (AH) is a major risk factor for many CVDs, and inflammation and immunity are implicated in its pathogenesis.

T lymphocytes and cytokines: T lymphocytes, particularly Th1 cells (producing IFN-γ) and cytotoxic T lymphocytes, are involved in AG. They contribute to vascular dysfunction, oxidative stress, and vascular remodeling, leading to increased vascular resistance [37]. T lymphocyte depletion or Treg depletion reduces hypertension in animal models.

IL-17: IL-17, produced by Th17 lymphocytes, has a novel role in AG. IL-17 induces endothelial dysfunction, increases oxidative stress, and may increase vascular tone [38]. Anti-IL-17 therapy reduces blood pressure in animal models.

TNF-α and IL-6: These cytokines contribute to hypertension by inducing endothelial dysfunction and increasing vascular resistance. Elevated levels of TNF-α and IL-6 have been observed in patients with AG [39].

6. Molecular signaling pathways of cytokines

Cytokines mainly act through three major signaling pathways: the JAK-STAT pathway (activated by IL-6, IFNs), the NF-κB pathway (activated by TNF-α, IL-1β), and the MAPK pathway [40]. Activation of these pathways leads to transcription of inflammatory genes, cell growth, differentiation, and death. Targeted therapies for these pathways, such as JAK inhibitors or NF-κB inhibitors in CKD, are an active area of research [41].

7. Clinical therapeutics The CANTOS (Canakinumab) and COLCOT (Colxine) trials have demonstrated the practical value of anticytokine therapy in the treatment of NSCLC [16, 17]. In addition, conventional cardiovascular drugs such as statins have been shown to have anti-inflammatory effects in addition to their lipid-lowering effects [42]. However, the widespread

use of anticytokine therapy is limited by immunosuppression and side effects. Research is ongoing to identify targeted and safe anti-inflammatory therapies in NSCLC.

Discussion Although it is clear that cytokines produced by leukocytes play a central role in the pathogenesis of NSCLC, their precise functions and potential for therapeutic manipulation remain complex and multifaceted. Inflammation initially serves a protective function, but with chronicity or excessive severity it becomes a pathological process. In atherosclerosis, pro-inflammatory cytokines have been shown to promote the initiation and progression of lesions, while anti-inflammatory cytokines have been shown to play a protective role. The inflammatory process after MI resembles a delicate balance: the initial pro-inflammatory phase is necessary for tissue clearance, but the subsequent shift to an anti-inflammatory and pro-fibrotic phase is important for vascular repair and preservation of cardiac function. Disruption of this balance leads to poor outcomes. In HF, a chronic inflammatory state directly contributes to the decline in cardiac function.

Clinical trials such as CANTOS and COLCOT have validated the principle of targeting inflammation in the treatment of NSCLC. However, the failure of anti-TNF-α therapy suggests that not all cytokines are the same and their biological complexity makes targeted therapy difficult in some cases. Targeting key regulators of inflammation (e.g., IL-1β) may be more effective than targeting downstream mediators such as IL-6. In addition, drug side effects, such as increased risk of infections, are a continuing concern for broad immunotherapy. Future studies should focus on elucidating the precise role of individual cytokines in different stages of NSCLC and in different patients. More specific targets of inflammation, such as specific signaling pathways or intracellular components, may be more beneficial than broad immunotherapy. Personalized medicine approaches, such as patient stratification based on inflammatory biomarkers, may improve the efficacy of anticytokine therapy. In conclusion, a deeper understanding of cytokines produced by leukocytes opens a new era in the treatment of CHD.

Conclusion

In conclusion, cytokines produced by leukocytes play a crucial role in the pathogenesis of cardiovascular diseases. These small signaling molecules regulate a wide range of pathological processes, from the initiation and progression of atherosclerosis to inflammation and repair after myocardial infarction, as well as the chronic course of heart failure and arterial hypertension. Pro-inflammatory cytokines, such as IL-1β, IL-6, TNF-α, and IFN-γ, mainly exert deleterious effects, promoting inflammation, cell death, and tissue damage. In contrast, anti-inflammatory cytokines, such as IL-10 and TGF-β, play a protective role and limit pathological processes. Extensive research over the past decade, including large clinical trials, has confirmed inflammation as a major driver of CVD, reinforcing the clinical relevance of these mediators and the therapeutic potential of targeted therapies. However, the complexity of cytokine networks, the interactions between them, and the need to preserve the physiological functions of immunity are significant obstacles to practical therapy. Future research on the management of inflammation in CVD should focus on identifying the precise mechanisms of individual cytokines, their interactions with genetic and environmental factors, and on highly targeted, personalized therapeutic approaches. A fuller understanding of the relationship between inflammation and cardiovascular health will allow the development of new diagnostic, prognostic, and treatment strategies to reduce the burden of these serious diseases.

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