Editorial

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Cytokines in Inflammation: Roles of TNF-α, IL-6, IL-17, and IL-23 in Autoimmune Diseases

Hana Al-Masri*

Department of Immunology and Rheumatology, Desert Valley Medical University, Amman, Jordan

***Author for Correspondence:**
hana.almasri@dvmu.edu.jo

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Introduction

Cytokines are small signaling proteins that play a pivotal role in immune regulation and inflammation. Among them, tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), interleukin-17 (IL-17), and interleukin-23 (IL-23) are central mediators in the pathogenesis of autoimmune and inflammatory diseases. Dysregulation of these cytokines contributes to chronic inflammation, tissue damage, and systemic manifestations observed in conditions such as rheumatoid arthritis, psoriasis, and inflammatory bowel disease.

Key Cytokines and Their Roles

TNF-α is a pro-inflammatory cytokine produced primarily by macrophages. It promotes leukocyte recruitment, activates endothelial cells, and induces the expression of other inflammatory mediators. TNF-α is critical in driving synovial inflammation and joint destruction in rheumatoid arthritis.

IL-6 is produced by T cells, macrophages, and fibroblasts and has both pro- and anti-inflammatory roles. It stimulates acute-phase protein synthesis, B cell differentiation, and T cell activation. Elevated IL-6 levels are associated with systemic inflammation and complications such as anemia and fatigue in autoimmune disorders.

IL-17, primarily secreted by Th17 cells, recruits neutrophils and stimulates epithelial and stromal cells to produce inflammatory mediators. IL-17 contributes to tissue damage in autoimmune diseases and plays a key role in skin and joint pathology in psoriasis and psoriatic arthritis.

IL-23 is produced by dendritic cells and macrophages and is essential for the differentiation and maintenance of Th17 cells. It amplifies IL-17–mediated inflammation and sustains chronic immune responses, making it a critical upstream target in the IL-23/IL-17 axis.

Clinical Implications

Targeting these cytokines has revolutionized treatment strategies in autoimmune and inflammatory diseases. Biologic therapies, including TNF inhibitors, IL-6 receptor blockers, IL-17 inhibitors, and IL-23 inhibitors, provide precise modulation of immune pathways, reduce inflammation, and improve clinical outcomes. Understanding cytokine networks allows for personalized therapy selection and optimization of treatment response.

Conclusion

TNF-α, IL-6, IL-17, and IL-23 are central drivers of inflammation in autoimmune and chronic inflammatory diseases. Dysregulation of these cytokines contributes to tissue damage and systemic symptoms, while targeted inhibition has transformed patient care. Advances in cytokine biology continue to enhance precision medicine approaches, offering more effective and tailored treatments for immune-mediated disorders.