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Neutrophils in Immunity and Inflammation: From Pathogen Clearance to Tissue Repair and Autoimmunity

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Abstract

Neutrophils, the most abundant circulating white blood cells, play a central role in both innate immunity and inflammation. These highly versatile cells act as the first line of defense against microbial pathogens, orchestrating pathogen clearance through phagocytosis, degranulation, and the release of neutrophil extracellular traps (NETs). However, beyond their antimicrobial functions, neutrophils have emerged as key players in the regulation of inflammation, tissue repair, and even the pathogenesis of autoimmune diseases. This article reviews the multifaceted role of neutrophils in immunity and inflammation, highlighting their protective functions as well as their involvement in tissue damage, chronic inflammation, and autoimmunity. We explore the mechanisms by which neutrophils contribute to both protective and pathogenic responses, focusing on their involvement in inflammatory diseases such as rheumatoid arthritis, lupus, and inflammatory bowel disease. Understanding the dual nature of neutrophil responses in health and disease is crucial for developing targeted therapies that modulate neutrophil function for therapeutic benefit.

Keywords: Neutrophils; Immunity; Inflammation; Pathogen clearance; Tissue repair; Autoimmunity; Neutrophil extracellular traps; Rheumatoid arthritis; Lupus; Inflammatory bowel disease; Chronic inflammation

Introduction

Neutrophils; or polymorphonuclear leukocytes; are the most abundant type of white blood cells in the peripheral circulation. They are part of the innate immune system and serve as the first responders to infections [1]. Traditionally neutrophils have been recognized for their ability to rapidly eliminate invading pathogens primarily bacteria; through mechanisms such as phagocytosis; degranulation; and the formation of neutrophil extracellular traps (NETs) [2]. Their quick mobilization and potent antimicrobial functions make them crucial in the defense against infections. In addition to pathogen clearance; neutrophils have a critical role in regulating inflammation and facilitating tissue repair. However; their ability to induce inflammation and cause tissue damage has also been linked to the pathogenesis of various chronic inflammatory diseases and autoimmune conditions. The ability of neutrophils to participate in both protective and pathogenic responses underscores their versatility and complexity [3]. This review aims to explore the dual role of neutrophils in immunity and inflammation; with a particular focus on their involvement in tissue repair processes and the development of autoimmune diseases. We will discuss recent advances in understanding neutrophil biology; the molecular pathways governing their activation; and their contribution to both beneficial and harmful inflammation.

Results

Neutrophil-mediated pathogen clearance: Neutrophils are key players in the early stages of immune response to infection. Upon encountering pathogens; neutrophils undergo rapid activation; resulting in the release of reactive oxygen species (ROS); antimicrobial peptides; and enzymes from their granules. These substances contribute to the direct killing of microorganisms. Neutrophils can also form NETs—web-like structures composed of DNA and antimicrobial proteins—that trap and neutralize pathogens [4]. The formation of NETs has been demonstrated to be an important defense mechanism against a variety of pathogens; including bacteria; fungi; and viruses.

Neutrophil involvement in inflammation: Neutrophils contribute to both the initiation and resolution of inflammation. Upon activation; they release a wide array of pro-inflammatory cytokines; chemokines; and lipid mediators that amplify the inflammatory response. These mediators recruit additional immune cells to the site of infection or injury; which facilitates pathogen elimination and tissue repair. However; prolonged activation of neutrophils can lead to tissue damage and the development of chronic inflammation [5]. Inflammatory mediators released by neutrophils; such as matrix metalloproteinases (MMPs); can degrade tissue components; contributing to the pathology of inflammatory diseases.

Neutrophils in tissue repair and resolution of inflammation: Beyond their role in inflammation; neutrophils are involved in the resolution phase of tissue injury. They help clear apoptotic cells and release factors that promote tissue repair; such as transforming growth factor-beta (TGF-β) and vascular endothelial growth factor (VEGF). Additionally; neutrophils are implicated in modulating the activity of other immune cells involved in tissue regeneration [6]. The switch from pro-inflammatory to pro-resolution functions of neutrophils is essential for maintaining tissue homeostasis and preventing chronic inflammation.

Neutrophils in autoimmune disease: Neutrophils have also been implicated in the pathogenesis of several autoimmune diseases; including rheumatoid arthritis (RA); systemic lupus erythematosus (SLE); and inflammatory bowel disease (IBD). In these conditions; dysregulated neutrophil activity leads to excessive inflammation and tissue damage. In RA; neutrophils contribute to joint destruction

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through the release of proteolytic enzymes and ROS. In SLE; neutrophils are involved in the formation of autoantibodies and the exacerbation of systemic inflammation [7]. In IBD; neutrophil-driven inflammation in the gut mucosa leads to tissue damage and disease progression.

Discussion

Neutrophils and the balance between protection and pathology

Neutrophils are a double-edged sword in immune responses. Their ability to rapidly respond to infection and initiate the clearance of pathogens is indispensable for host defense. However; the same effector functions that protect against pathogens can also contribute to tissue damage when not properly regulated. The persistent activation of neutrophils can lead to chronic inflammation; which is a hallmark of several inflammatory diseases. Therefore; the challenge lies in understanding the mechanisms that govern neutrophil activation and resolution of inflammation. A critical aspect of neutrophil biology is their ability to adapt their response depending on the microenvironment. For example; in acute infections; neutrophils are activated to engage in pathogen clearance. In contrast; during tissue repair; neutrophils transition from a pro-inflammatory state to one that promotes resolution and healing. This plasticity is mediated by a complex network of signaling pathways; including those triggered by cytokines; lipid mediators; and immune cell interactions [8]. The production of NETs represents a fascinating aspect of neutrophil function. While NETs are highly effective at capturing and neutralizing pathogens; their excessive or inappropriate formation has been implicated in tissue damage and the development of autoimmune diseases. In diseases such as lupus; NETs can serve as a source of autoantigens; further fueling the autoimmune response. The therapeutic targeting of NET formation or the neutralization of NET-associated molecules could offer new avenues for treating autoimmune diseases.

Conclusion

Neutrophils are essential components of the immune system; playing a critical role in both pathogen clearance and the regulation of inflammation. Their ability to transition between protective and pathogenic functions highlights their versatility and the complexity of their role in health and disease. While neutrophils are indispensable for fighting infections and promoting tissue repair; dysregulated neutrophil responses can contribute to chronic inflammation and the development of autoimmune diseases. Understanding the molecular mechanisms that govern neutrophil activation; their contribution to tissue damage and repair; and their role in autoimmunity is key to developing novel therapeutic strategies aimed at modulating neutrophil function. Future research should continue to explore the fine balance between neutrophil-driven immunity and inflammation; with the goal of harnessing their beneficial effects while minimizing their pathogenic potential.

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