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The Role of CARD9 in Cell and Organ-specific Immune Responses in Various Infections

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Abstract

Caspase recruitment domain-containing protein 9 is a crucial component of innate immune responses, particularly in orchestrating inflammatory reactions and shaping adaptive immunity. Its significance spans across various infections, where it modulates cell- and organ-specific immune responses. This article explores the multifaceted role of CARD9 in different infections, elucidating its involvement in pathogen recognition, activation of immune cells, and regulation of inflammatory pathways. Additionally, it delves into how CARD9 influences the immune response in specific cell types and organs, shedding light on its potential as a therapeutic target for infectious diseases. CARD9, a key adaptor protein, is involved in various cellular processes, including inflammation, autophagy, and cytokine production, making it a crucial player in the host defense against infections. In this review, we delve into the multifaceted roles of CARD9 in cell and organ-specific immune responses across different infections.

Keywords: Pathogen • Immune • Infections

Introduction

The immune system relies on intricate signaling networks to mount effective responses against pathogens. CARD9, a signaling adaptor protein, plays a pivotal role in coordinating these responses by integrating signals from various pattern recognition receptors and facilitating downstream immune activation. Its involvement in diverse infections highlights its significance in host defense mechanisms. CARD9 is predominantly expressed in myeloid cells, including macrophages, dendritic cells, and neutrophils, where it plays a crucial role in mediating immune responses. Upon recognition of pathogens by Pattern Recognition Receptors (PRRs) such as Toll-Like Receptors (TLRs) and C-type Lectin Receptors (CLRs), CARD9 is recruited to the receptor complex through its Caspase Recruitment Domain (CARD), initiating downstream signaling cascades. This leads to the activation of various transcription factors, including nuclear factor-kappa B (NF-κB) and Mitogen-Activated Protein Kinases (MAPKs), culminating in the production of pro-inflammatory cytokines and antimicrobial molecules [1].

Literature Review

It acts as an adaptor molecule, linking PRRs to downstream signaling pathways, particularly those involving NF-ĸB and MAP kinases. Through its interactions with other proteins, CARD9 regulates cytokine production, phagocytosis, and antimicrobial activity in immune cells. CARD9 mediates the recognition of fungal, bacterial, and viral pathogens by various PRRs, including Dectin-1, Dectin-2, and Mincle. Upon pathogen recognition, CARD9 forms complexes with these receptors, initiating signaling cascades that lead to immune cell activation and cytokine secretion. Macrophages, dendritic cells and neutrophils are key immune cells that express CARD9 and depend on its signaling for effective antimicrobial responses. CARD9-deficient mice exhibit impaired activation of these cells, resulting in compromised clearance of pathogens and increased susceptibility to infections crucial for fungal clearance. Genetic variations in CARD9 have been associated with susceptibility to fungal infections in humans [2,3].

Discussion

CARD9 contributes to host defense against bacterial pathogens such as Listeria monocytogenes and Mycobacterium tuberculosis. It regulates the production of antimicrobial peptides and the recruitment of immune cells to sites of infection, promoting bacterial clearance. While less studied, CARD9 has been implicated in antiviral immunity against certain viruses. It modulates inflammatory responses during viral infections, influencing disease severity and immune-mediated tissue damage. Targeting CARD9 signaling pathways holds promise for the development of novel immunotherapies for infectious diseases. Small molecule inhibitors or immunomodulatory agents that modulate CARD9 activity could be explored as adjunctive treatments to enhance host defense mechanisms [4]. Although initially characterized for its role in fungal and bacterial immunity, emerging evidence suggests that CARD9 may also play a role in antiviral defense. Studies have shown that CARD9-deficient mice exhibit impaired immune responses to certain viral infections, such as influenza A virus and Herpes Simplex Virus type 1 (HSV-1). Notably, CARD9 has been implicated in the regulation of type Interferon (IFN) responses, which are essential for limiting viral replication and spread. However, the specific mechanisms by which CARD9 modulates antiviral immunity require elucidation [5].

Beyond its role in systemic immunity, CARD9 also influences organ-specific immune responses. For instance, in the gut, CARD9 signaling regulates the balance between protective immunity and Inflammatory Bowel Diseases (IBD), such as Crohn's disease and ulcerative colitis. Loss of CARD9 function has been associated with dysregulated intestinal inflammation and altered gut microbiota composition, highlighting its importance in maintaining gut homeostasis. Similarly, in the skin, CARD9 has been implicated in the pathogenesis of psoriasis, a chronic inflammatory skin disorder characterized by dysregulated immune responses. CARD9 signaling in skin-resident immune cells regulates the production of pro-inflammatory cytokines and the recruitment of inflammatory cells, contributing to disease progression [6].

Conclusion

CARD9 plays a critical role in orchestrating cell- and organ-specific

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immune responses to various infections. CARD9 exerts differential effects on immune cells and organs depending on the nature of the infection. In the gut, CARD9 signaling in intestinal epithelial cells and DCs contributes to the maintenance of gut homeostasis and defense against enteric pathogens. In the lung, CARD9 regulates immune responses to respiratory infections, including fungal and bacterial pathogens, influencing disease outcomes. Its involvement in pathogen recognition, immune cell activation, and regulation of inflammatory pathways highlights its significance in host defense mechanisms. Further understanding of CARD9-mediated immune responses could pave the way for innovative therapeutic strategies to combat infectious diseases.

Acknowledgement

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Conflict of Interest

None.

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