Article type: Editorial

Home Page URL: https://www.alliedacademies.org/immunology-case-reports/

Molecular pathways in host-parasite immune interaction.

Sarah Julie*

Department of Microbiology and Immunology, Cornell University, USA

Correspondence to: Nina Shah, Department of Microbiology and Immunology, Cornell University, USA, E-mail: julies@wright.edu

Received: *02-Jul-2024, Manuscript No. AAICR-25-169253;* ***Editor assigned:*** *03-Jul-2024, Pre QC No. AAICR-25-169253(PQ);* ***Reviewed:*** *18-Jul-2024, QC No. AAICR-25-169253;* ***Revised:*** *24-Jul-2024, Manuscript No. AAICR-25-169253(R);* ***Published:*** *31-Jul-2024, DOI: 10.35841/aaicr-7.2.177*

Introduction

Host-parasite interactions represent a dynamic biological battleground shaped by evolution. Parasites, ranging from protozoa to helminths, have evolved intricate mechanisms to evade or manipulate host immune responses, while hosts deploy a sophisticated array of immune defenses to detect and eliminate invading organisms. Understanding the molecular pathways involved in these interactions is key to developing novel therapeutics and vaccines against parasitic diseases that continue to burden global health [1, 2].

The first line of host defense involves recognition of parasite-derived molecules known as pathogen-associated molecular patterns (PAMPs) via pattern recognition receptors (PRRs). TLR2 and TLR4 play pivotal roles in recognizing helminth glycoproteins and protozoan lipophosphoglycan (LPG). These cytosolic sensors detect intracellular parasitic components such as *Toxoplasma gondii* profilin and trigger inflammasome activation. Critical in recognizing carbohydrate-rich helminth antigens, influencing dendritic cell polarization [3, 4].

Upon engagement, these receptors initiate signaling cascades involving adaptor proteins like MyD88 and TRIF, ultimately leading to nuclear translocation of transcription factors such as NF-κB and IRFs that regulate cytokine production. Cytokines orchestrate immune responses by directing the differentiation and function of immune cells. Parasites elicit distinct cytokine profiles depending on their biology and niche: Dominated by IFN-γ and IL-12, this pathway is crucial for intracellular protozoa like *Leishmania* and *Toxoplasma* [5, 6].

Characterized by IL-4, IL-5, and IL-13, this pathway defends against extracellular helminths, promoting eosinophil activation and IgE production. IL-17-producing Th17 cells have dual roles, sometimes contributing to pathology. Regulatory T cells (Tregs), often expanded by parasites, suppress inflammation and facilitate chronic infection. *Plasmodium falciparum* expresses variable surface antigens (VSA), including PfEMP1, allowing immune evasion. Helminths produce molecules resembling host cytokines to subvert immune responses. Parasites secrete enzymes, lipids, and glycans that interfere with host signaling. For instance, *Schistosoma mansoni* secretes omega-1, which skews dendritic cells toward a Th2-promoting phenotype [7, 8].

Activated via TLR engagement, this pathway regulates production of IL-1 and TNF-α. Involved in cell survival and anti-inflammatory signaling. Some parasites hijack this pathway to prevent apoptosis. Critical in cytokine signaling. STAT1 drives Th1 responses; STAT6 mediates Th2 responses. Parasites may manipulate STAT phosphorylation to suppress effective immunity [9, 10].

Conclusion

Host genetic background significantly influences susceptibility. Polymorphisms in TLRs, cytokine genes, and HLA loci alter the effectiveness of parasite recognition and clearance. For example, mutations in TLR9 have been associated with susceptibility to *Plasmodium vivax*. Understanding these pathways unlocks opportunities for intervention: Modulating pathways like JAK/STAT or boosting PRR responses may enhance host immunity.

References

1. Shadick NA, Liang MH, Partridge AJ, et al. The natural history of exercise-induced

anaphylaxis: survey results from a 10-year follow-up study. J allerg clinic immunol. 1999;104(1):123-7.
2. Maulitz RM, Pratt DS, Schocket AL. Exercise-induced anaphylactic reaction to shellfish. J Allerg Clinic Immunol. 1979;63(6):433-4.
3. Harada S, Horikawa T, Ashida M, et al. Aspirin enhances the induction of type I allergic symptoms when combined with food and exercise in patients with food?dependent exercise?induced anaphylaxis. Brit J Dermatol. 2001;145(2):336-9.Indexed at, Google Scholar, Cross Ref
4. Ansell GM, Tweedie MC, West CR, et al. The current status of reactions to intravenous contrast media. Invest radiol. 1980;15(6 Suppl):S32-9.
5. Palmer FJ. The RACR survey of intravenous contrast media reactions final report. Aus radiol. 1988;32(4):426-8.