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Mechanisms of Immune Evasion in Mycobacterium tuberculosis.

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Introduction

Mycobacterium tuberculosis (MTB), the causative agent of tuberculosis (TB), is one of the most successful human pathogens in history. Despite the presence of a robust immune system, MTB has evolved sophisticated strategies to evade host defenses, persist in tissues, and cause chronic infection. Understanding these immune evasion mechanisms is critical for developing effective vaccines, diagnostics, and therapies [1, 2].

TB remains a global health threat, with over 10 million new cases and 1.5 million deaths annually. Although the Bacille Calmette-Guérin (BCG) vaccine has been used for nearly a century, its efficacy is limited, especially in adults. MTB's ability to survive and replicate within host cells, particularly macrophages, underlies its resilience and pathogenicity.The innate immune system is the first line of defense against MTB. MTB inhibits the acidification and maturation of phagosomes, preventing their fusion with lysosomes. This allows the bacteria to survive within macrophages.MTB modulates calcium, iron, and hydrogen ion concentrations to create a favorable intracellular environment.MTB suppresses programmed cell death and autophagy in infected macrophages, which are essential for clearing intracellular pathogens2.MTB selectively engages or avoids PRRs like Toll-like receptors (TLRs) to dampen inflammatory signaling [3, 4].

These tactics allow MTB to persist in host cells and avoid early immune clearance.MTB disrupts the processing and presentation of antigens by major histocompatibility complex (MHC) molecules, impairing T-cell activation.While granulomas are intended to contain infection, MTB can manipulate their structure to create niches for persistence and latency.MTB induces Tregs that suppress effector T-cell responses, weakening immune control.MTB skews cytokine production, promoting anti-inflammatory signals (e.g., IL-10) and suppressing protective ones (e.g., IFN-γ).These mechanisms enable MTB to evade immune surveillance and establish long-term infection.These secreted proteins interfere with host cell signaling and promote phagosomal escape.These proteins modulate antigen presentation and immune recognition.A key cell wall component that inhibits macrophage activation and cytokine production.Suppresses host immune responses and is used as a diagnostic marker.These virulence factors are targets for vaccine development and therapeutic intervention [5, 6].

One of MTB's most remarkable features is its ability to enter a latent state, evading immune detection for years. During latency:MTB reduces its metabolic activity and antigen expression.Host immune responses are modulated to prevent bacterial clearance.Reactivation can occur when host immunity declines, leading to active TB.Understanding the molecular basis of latency is essential for controlling TB transmission and recurrence [7, 8].

Immune evasion by MTB presents major challenges for vaccine design. The failure of BCG to protect against pulmonary TB in adults is partly due to MTB's ability to suppress effective immune responses. New vaccine candidates aim to enhance T-cell immunity and overcome MTB's evasion tactics.Similarly, drug development must consider MTB's intracellular survival and immune

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modulation. Host-directed therapies that restore immune function—such as autophagy inducers or cytokine modulators—are being explored as adjuncts to antibiotic treatment [9, 10].

Conclusion

To combat MTB effectively, future research should focus on Identifying novel virulence factors involved in immune evasion.Developing biomarkers for early detection of latent and active TB.Designing vaccines that elicit robust and durable T-cell responses.Exploring host-directed therapies to enhance immune clearance.Multi-omics approaches, including proteomics, transcriptomics, and metabolomics, will be crucial for unraveling the complex host-pathogen interactions.

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