ISSN: 2155-9597
Journal of Bacteriology & Parasitology

OPEN ACCESS Freely available online

Perspective

Cellular and Molecular Mechanisms of Host-Pathogen Interactions in Neglected Tropical Diseases

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DESCRIPTION

Neglected Tropical Diseases (NTDs) affect millions in resource-limited settings, with host-pathogen interactions playing a major role in pathogenesis and disease progression. Understanding these interactions at cellular and molecular levels is essential for developing targeted therapies and control strategies. The key mechanisms by which pathogens invade, survive and evade host defenses in NTDs such as Leishmaniasis, Chagas disease and Schistosomiasis. We discuss recent insights into host immune responses, pathogen evasion strategies and cellular pathways exploited by pathogens. By illuminating these molecular dialogues, we can highlight potential targets for novel therapeutic interventions.

NTDs are a diverse group of communicable diseases prevalent in low-income populations, affecting more than a billion people globally. Pathogens causing NTDs employ unique strategies to evade immune defenses, adapt to host environments and persist within the human body. Elucidating the cellular and molecular mechanisms of these host-pathogen interactions provides insight into disease pathogenesis and highlights targets for potential therapeutic development.

NTD-causing pathogens such as protozoa, helminths and bacteria use specialized surface proteins to recognize, attach to and enter host cells. For instance, *Trypanosoma cruzi*, the causative agent of chagas disease, exploits a surface protein known as trans-sialidase to facilitate its adhesion to host cell receptors, initiating entry into target cells such as cardiomyocytes and macrophages. This interaction triggers calcium signaling within the host cell, which rearranges the cytoskeleton to permit pathogen entry. Similarly, *Leishmania* species utilize lipophosphoglycan, a surface glycolipid, to interact with host macrophages, enhancing their uptake and promoting intracellular survival.

Once inside the host, pathogens face the immune system's robust response. However, many NTD pathogens have evolved evasion tactics to subvert this response. *Trypanosoma brucei*, responsible for African sleeping sickness, changes its surface glycoproteins periodically to avoid detection by antibodies.

In Leishmaniasis, *Leishmania* parasites manipulate macrophages, the primary cells responsible for their uptake, by inhibiting reactive oxygen and nitrogen intermediates, essential components of pathogen killing. Intracellular pathogens like *Mycobacterium leprae* (causing leprosy) survive within macrophages by preventing phagosome-lysosome fusion, thereby avoiding degradation. Many NTD pathogens manipulate host signaling pathways to create a favorable environment for survival and replication.

Several pathogens, including *Leishmania* and *Schistosoma*, suppress NF-κB signaling, which is vital for initiating an effective immune response. By inhibiting NF-κB, these pathogens reduce the expression of pro-inflammatory cytokines, decreasing the immune system's capacity to respond effectively.

Certain pathogens, like *Toxoplasma gondii*, modulate autophagy pathways to gain a survival advantage within host cells. By regulating autophagic flux, these pathogens prevent cellular degradation processes that could target them. Host-pathogen interactions in NTDs often lead to significant tissue damage, contributing to the morbidity associated with these diseases. Chronic infection with pathogens such as *Schistosoma mansoni* results in granuloma formation due to continuous immune stimulation by parasite eggs. These granulomas can lead to liver fibrosis, a severe complication in chronic Schistosomiasis. Additionally, persistent infection with *T. cruzi* leads to an inflammatory response that contributes to cardiac tissue damage, resulting in Chagas cardiomyopathy, a major cause of heart failure in endemic regions.

Understanding host-pathogen interactions at the cellular and molecular level has identified several promising targets for

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Received: 29-Oct-2024, Manuscript No. JBP-24-27530; **Editor assigned:** 31-Oct-2024, PreQC No. JBP-24-27530 (PQ); **Reviewed:** 14-Nov-2024, QC No. JBP-24-27530; **Revised:** 21-Nov-2024, Manuscript No. JBP-24-27530 (R); **Published:** 28-Nov-2024, DOI: 10.35248/2155-9597.24. S28.112

Citation: Parveena C (2024). Cellular and Molecular Mechanisms of Host-Pathogen Interactions in Neglected Tropical Diseases. J Bacteriol Parasitol. S28:112.

therapeutic intervention. For example, targeting parasite enzymes involved in nutrient acquisition or immune modulation, such as Leishmania's lipophosphoglycan, could impair pathogen survival. Immunomodulatory therapies, such as drugs that enhance host autophagy pathways or prevent NF-κB suppression, may also restore immune functionality against these pathogens.

However, challenges remain in translating these insights into effective treatments, particularly given the complexity of immune responses and the potential for unintended off-target effects in humans. Furthermore, the socioeconomic and logistical challenges of delivering these treatments to resource-poor regions where NTDs are most prevalent pose additional barriers.

CONCLUSION

The complex interaction between host and pathogen in NTDs reveals both vulnerabilities and adaptive strategies that pathogens employ to survive within their hosts. Further research into the molecular basis of these interactions will not only advance our understanding of NTD pathology but also open avenues for novel treatments. Bridging the gap between molecular research and clinical application could significantly reduce the burden of NTDs, particularly in vulnerable populations with limited access to healthcare.