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Host-Pathogen Crosstalk: Implications in Cellular Processes by Intracellular Bacteria.

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Introduction

The dynamic interplay between host cells and intracellular bacterial pathogens commonly referred to as host-pathogen crosstalk is a cornerstone of infectious disease biology. Intracellular bacteria such as *Mycobacterium tuberculosis*, *Salmonella enterica*, *Listeria monocytogenes*, and *Rickettsia* species have evolved sophisticated mechanisms to invade, survive, and replicate within host cells. This interaction not only determines the outcome of infection but also profoundly influences host cellular processes, including immune signaling, metabolism, and cell death pathways [1, 2].

Unlike extracellular pathogens, intracellular bacteria must navigate the hostile environment of the host cell. To do so, they deploy a range of virulence factors—often delivered via specialized secretion systems that manipulate host cell biology [3].

Type III and IV secretion systems: Used by *Salmonella* and *Legionella* to inject effector proteins. Employed by *Listeria* and *Rickettsia* to access the cytosol. These strategies allow bacteria to create replication niches, evade immune detection, and hijack host resources [4, 5].

Host cells detect intracellular pathogens through pattern recognition receptors (PRRs) such as Toll-like receptors (TLRs), NOD-like receptors (NLRs), and RIG-I-like helicases. Intracellular bacteria often manipulate these pathways [6, 7].

For example, *Salmonella* initially activates the NLRC4 inflamma some to promote dissemination, then suppresses it to evade detection. Pathogens rewire host metabolism to support their survival. *Mycobacterium tuberculosis* induces lipid droplet formation in macrophages, creating nutrient-rich niches. *Rickettsia* scavenges host metabolites due to its reduced genome, which lacks biosynthetic pathways [8, 9].

Cell-autonomous immunity refers to the ability of individual cells to defend against pathogens without relying on specialized immune cells. Mechanisms include: Intracellular bacteria counteract these defenses by secreting effector proteins that inhibit autophagy, neutralize reactive species, or block ISG expression [10].

Conclusion

Recent studies highlight the role of host microRNAs (miRNAs) in regulating immune responses during infection. Intracellular pathogens can manipulate host miRNA profiles to suppress immunity and promote persistence. For instance:*Mycobacterium tuberculosis* alters miRNA expression to inhibit macrophage activation. *Listeria monocytogenes* modulates miRNAs involved in apoptosis and cytokine productionThis layer of regulation adds complexity to host-pathogen crosstalk and offers potential therapeutic targets. *Rickettsia parkeri*, an obligate intracellular pathogen, replicates in the host cytosol a compartment rich in immune defenses. It secretes membranolytic proteins to escape the phagosome and uses actin polymerization for motility and cell-to-cell spread. Its reduced genome makes it an ideal model for studying minimal virulence factor repertoires.Understanding how *Rickettsia* manipulates host cytosolic defenses can illuminate broader principles of intracellular survival and immune evasion.strategies.

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