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Membrane trafficking in host-pathogen interactions: A tug-of-war for cellular control.

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Introduction

Membrane trafficking is a fundamental cellular process that governs the movement of proteins, lipids, and other macromolecules within and between organelles. It ensures proper cellular organization, signaling, and homeostasis. However, during host-pathogen interactions, this tightly regulated system becomes a battleground. Pathogens—from viruses to bacteria and fungi—have evolved sophisticated strategies to hijack host membrane trafficking pathways, manipulating vesicle transport, organelle dynamics, and immune signaling to promote infection. This tug-of-war for cellular control has profound implications for immunity, pathogenesis, and therapeutic development [1].

Membrane trafficking encompasses two primary pathways: endocytosis and exocytosis. Endocytosis involves the internalization of extracellular materials and plasma membrane components into endosomes, while exocytosis exports cargo from the Golgi apparatus to the plasma membrane or extracellular space. These processes rely on transport vesicles coated with proteins such as clathrin, COPI, and COPII, which mediate vesicle budding, cargo selection, and fusion with target membranes [2].

In addition to these canonical pathways, cells utilize autophagy—a degradation and recycling process involving double-membrane autophagosomes—and phagocytosis, which engulfs large particles including pathogens. Together, these trafficking systems maintain cellular integrity and respond to environmental cues. Pathogens exploit membrane trafficking to gain entry, avoid degradation, and establish replicative niches. Their strategies include: Many pathogens secrete effectors that target host trafficking machinery. For example, bacterial type III secretion systems inject proteins that interfere with vesicle formation and fusion. Viruses like influenza and SARS-CoV-2 use clathrin-mediated endocytosis to enter host cells, while others exploit macropinocytosis or caveolae-dependent pathways [3].

Some pathogens, such as *Mycobacterium tuberculosis*, block autophagosome maturation to avoid degradation, while others redirect autophagy to support their replication. Intracellular pathogens like *Salmonella* and *Legionella* remodel host endosomes and Golgi-derived vesicles to form pathogen-containing vacuoles (PCVs), shielding themselves from immune detection. These receptors detect pathogen-associated molecular patterns (PAMPs) and are trafficked to the plasma membrane or endosomes to initiate signaling. Exocytosis delivers cytokines, chemokines, and antimicrobial peptides to infection sites [4].

The interplay between host and pathogen trafficking systems creates feedback loops that influence infection outcomes. For example: In plants, detection of effector-mediated trafficking disruption activates robust immune responses. Host cells may reprogram trafficking to isolate pathogens or repair damage, while pathogens counteract with further manipulation. Trafficking of immune receptors and signaling molecules amplifies defense responses, but pathogens may dampen these signals by rerouting vesicles. Autophagy targets intracellular pathogens for lysosomal degradation, a process known as xenophagy. Membrane trafficking contributes to

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the execution of apoptosis and pyroptosis, limiting pathogen spread. In plants, membrane trafficking is equally critical in host-pathogen interactions. Pathogens secrete effectors that target vesicle trafficking to suppress immunity. For instance, oomycete and fungal effectors interfere with exocyst complexes and Rab GTPases, disrupting the delivery of immune receptors and defense molecules. Plants respond by redirecting vesicle traffic to infection sites, enhancing secretion of cell wall reinforcements and antimicrobial compounds. Autophagy also plays a role in programmed cell death and immune signaling [5].

Conclusion

Membrane trafficking is a dynamic and multifaceted battleground in host-pathogen interactions. Pathogens deploy intricate strategies to hijack vesicle transport, while hosts mobilize trafficking pathways to detect, respond to, and eliminate invaders. This tug-of-war shapes infection outcomes and offers critical insights into cellular control mechanisms. As research advances, targeting trafficking pathways may yield novel strategies to combat infectious diseases and enhance immune resilience.

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