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Traffic Control: Subversion of Plant Membrane Trafficking by Pathogens

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INTRODUCTION

Plants engage in countless microscopic battles with a multitude of diverse pathogens both aboveand belowground. To detect and eliminate intruders, plants employ a multilayered innate immune system that fundamentally relies on membrane trafficking to provide effective resistance. Specifically, the endomembrane transport system coordinates the activities of membrane-bound cellular organelles to ensure the precise and timely deposition of immune components in the correct location and quantity. Consistent with this notion, a growing number of studies have revealed pathogen manipulation of pl ant intracellular transport systems as a crucial infection strategy.

PLANT MEMBRANE TRAFFICKING PATHWAYS

The membrane trafficking pathways cooperate to keep cells healthy and operational by ensuring the proper functioning of vital cellular processes, such as cell metabolism, immune response, and cell differentiation. Plants transport cargoes—such as extracellular, membrane, and lysosomal proteins—via membrane-bound vesicles. These trafficking pathways are tightly regulated by a series of vesicle transport regulators and vesicle fusion proteins such as small GTPases and tethering factors. Although trafficking mainly takes place through the default secretory and endocytic pathways (Abdul malik *et al.,* 2020)

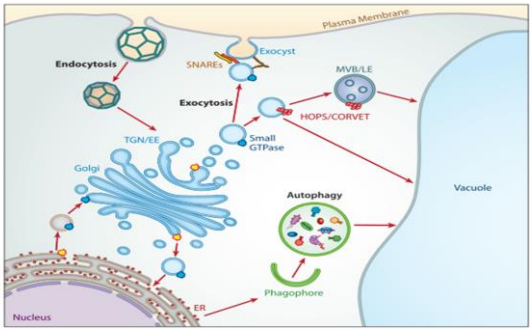

Figure 1- Overview of membrane trafficking pathways in plant

Endocytic System: Recycling or Destruction of Membrane Proteins

Much like other eukaryotes, plants recycle and remove membrane proteins from the cell surface through the endocytic system. The endocytic system consists of a network of membrane-bound organelles—including early endosomes (EEs), recycling endosomes, late endosomes (LEs), and the vacuole—that coordinate intracellular trafficking and protein homeostasis.

Positioning Pattern Recognition Receptors and Induced Defense Components by the Default Secretory Pathway

The plant innate immune system relies on timely recognition of pathogens/pests through surfacelocalized pattern recognition receptors (PRRs) and intracellular nucleotide-binding leucine-rich repeat immune receptors (NLRs). PRRs are transmembrane immune receptors that sense extracellular modified-self cues or nonself molecules (Baena *et al.*, 2022) such as pathogen-associated molecular patterns (PAMPs).

Focal Immunity: Diversion of Plant Defenses to the Sites of Pathogen Invasion

Pathogens and pests intimately interact with the host cells by forming specialized infection structures to deliver effectors and uptake nutrients. These structures vary from species to species: Bacteria form the type 3 (T3) injection apparatus to penetrate host cells; nematodes and insects deploy stylets; and most fungal and oomycete pathogens (herein referred to as filamentous pathogens) form specialized hyphae that invade the plant cell.

Reprogramming of membrane trafficking by pathogen effectors

Advances in genome sequencing have helped identify a plethora of effectors from various plant parasites. This paved the way for effector biology, which improved our understanding of the plant immune system by revealing major host defense components and susceptibility factors. Bacterial and oomycete effectors have been of particular interest because their unique amino acid sequence signatures make them relatively easy to identify through genomics-based approaches. Although host-translocated bacterial effectors carry an N-terminal T3 secretion signal, most oomycete effectors that are delivered inside the plant cells carry a conserved RXLR motif downstream of the N-terminal secretion signal (Derevnina *et al*., 2021).

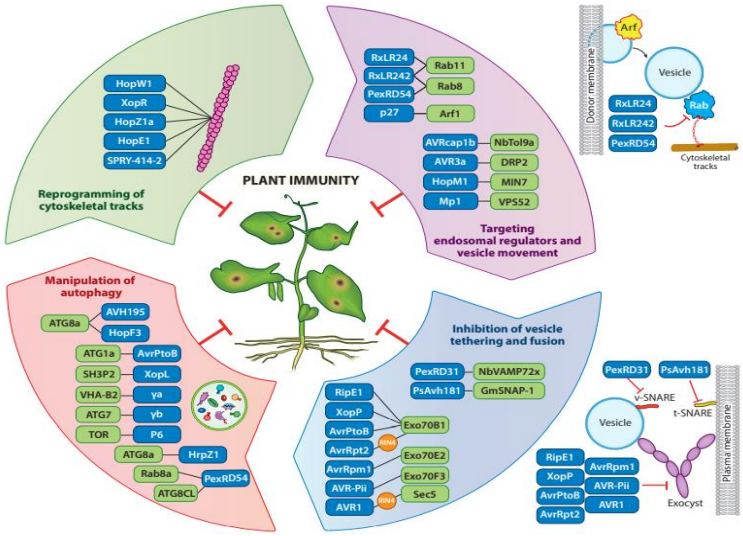

Figure 2- An overview of pathogen effectors and their plant targets implicated in host membrane trafficking.

Subversion of Plant Autophagy Machinery by Pathogen Effectors

Autophagy is an intracellular degradation pathway that heavily relies on membrane trafficking. Each step of autophagy is executed through the coordinated functions of core ATG proteins. Vesicle transport systems channel proteins and lipids required for autophagosome formation and facilitate the subsequent delivery of autophagosomes to their target compartments. A growing number of studies show that selective forms of autophagy contribute to plant immunity.

Bacterial tricks to subvert autophagy

Most bacterial pathogens employ T3Es to modulate host autophagy in various ways. *P. syringae* relies on a functional T3 secretion system that dampens basal plant resistance to promote autophagic acativity. The previously mentioned *P. syringae* effector HopM1 appears to have autophagy-inducing features during bacterial infection.

HopM1 promotes the removal of proteasomes through a selective autophagy process called proteaphagy, which in turn promotes bacterial virulence. As HopM1 was originally discovered to deplete MIN7 through the plant proteasome, its function of inducing proteaphagy directly challenges the notion of utilizing proteasomes (Ibrahim *et al.*, 2022).

Remodeling of Actin and Microtubule Dynamics by Pathogen Effectors

- HopZ1a is an acetyltransferase that can acetylate tubulin and lead to deconstruction of the host microtubule network. On the other hand, HopE1 purges the host microtubule-associated protein 65 (MAP65) from microtubules.
- Both effectors can inhibit protein secretion and compromise cell wall–based defenses (47, 65). In addition, nematode effectors also target plant microtubules to promote infection.
- An effector called SPRY-414-2 from the white potato cyst nematode *Globodera pallida* was identified to target a potato microtubule associated protein named cytoplasmic linker protein-associated protein (CLASP) that is involved in microtubule stability and growth

CONCLUSION

It is often difficult to determine the specific immune-related roles of trafficking components via standard genetic approaches, as many transport regulators show redundancy and are implicated in multiple trafficking routes. Another issue is that secretory systems required for growth are often co-opted by immune responses. That said, effectors present an alternative tool to study the cell biology of plant–pathogen interactions by guiding us toward the most critical components of defense-related trafficking in plants. A deeper understanding of these processes is essential to fully dissect the mechanisms employed by pathogens to subvert the plant immune system.

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