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Pathogen Tactics: Hacking Plant Trafficking for Invasion and Immune Evasion

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In the intricate play between plants and pathogens, membrane trafficking emerges as a crucial player in plant immunity. Coordinated by the endomembrane transport system, this defense mechanism ensures precise deployment of immune components. However, pathogens have evolved to exploit this system, manipulating intracellular transport to establish infection. By secreting virulence effectors, pathogens disrupt host trafficking pathways, subverting cellular control. The membrane trafficking control by pathogens highlights the arms race between plants and pathogens, revealing the intricate strategies employed to reprogram plant vesicle trafficking and evade immunity.

Membrane trafficking pathways play a vital role in maintaining cellular health and functionality, orchestrating essential processes such as cell metabolism, immune response, and cell differentiation. Plant cells transport various cargoes-extracellular, membrane, and lysosomal proteins—via membrane-bound vesicles, tightly regulated by a network of vesicle transport regulators and fusion proteins. While conventional trafficking primarily occurs through the default secretory and endocytic pathways, alternative routes also exist. The default secretory pathway initiates with the co-translational transport of secretory proteins from the endoplasmic reticulum (ER) to the Golgi apparatus, followed by further modification and packaging into secretory vesicles. Small GTPases and tethering complexes regulate vesicle budding, transport, and fusion, with Rabs and Arf1/Sar1 acting as key regulators. The subsequent fusion process involves SNARE proteins and tethering factors, ultimately directing vesicle contents to their intended destinations, such as the plasma membrane or vacuole. Meanwhile, the endocytic system governs the recycling or degradation of membrane proteins through a series of membrane-bound organelles, including early endosomes, recycling endosomes, late endosomes, and the vacuole.

In addition to conventional pathways, plants exhibit alternative secretion mechanisms, collectively termed unconventional protein secretion (UPS), which bypass the need for signal peptide sequences. UPS pathways involve transmembrane transport, multivesicular bodies (MVBs)/late endosomes, and autophagy, allowing for the secretion or degradation of specific cargoes. Autophagy, a conserved eukaryotic pathway, facilitates the degradation of cellular components through the formation and transport of autophagosomes to the vacuole. While selective forms of autophagy have been identified in various species, the existence of secretory autophagy in plants remains uncertain. Nonetheless, the intricate interplay between these trafficking pathways underscores their significance in cellular homeostasis and plant physiology.

Membrane trafficking pathways serve as integral players in plant innate immunity, directing immune receptors, signalling molecules, antimicrobial compounds, and other immune components to combat infectious diseases. Through the default secretory pathway, immune receptors like pattern recognition receptors (PRRs) are positioned on the cell surface to sense pathogen-associated molecular patterns (PAMPs) and initiate defense responses. Disruptions in membrane trafficking can lead to the depletion of immune components at the cell surface, increasing plant susceptibility to pathogens. Furthermore, constitutive endocytic recycling of PRRs preserves their presence on the cell surface, ensuring the integrity of the pathogen surveillance system. Ligand-induced endocytosis of PRRs and their signaling partners enhances immune signal propagation, highlighting the intricate role of endomembrane trafficking in orchestrating plant immune responses.

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Advancements in genome sequencing have led to the identification of numerous effectors from various plant parasites, enhancing our understanding of plant immune responses. Effector biology has uncovered key defense components and susceptibility factors, particularly in bacterial and oomycete pathogens. Effectoromics studies reveal a common trend wherein effectors from a given pathogen target specific host pathways redundantly or converge on shared host targets despite diverse origins. Notably, pathogens manipulate major host transport pathways using effectors to enhance virulence. Functional screens have identified effectors capable of modifying defense-related secretion, targeting various stages of membrane trafficking. For instance, some effectors interact with small GTPases like Rab8 and Rab11, crucial for immune component positioning and secretion regulation. RXLR effectors, such as RXLR24 from Phytophthora species, inhibit PR1 secretion by interacting with Rab11, underscoring the intricate manipulation of host trafficking by pathogens (Li et al., 2022). Efforts to validate these interactions highlight the complexity of host-pathogen interactions and the importance of understanding effector-mediated reprogramming of membrane trafficking in plant immunity.
Pathogen effectors intricately manipulate vesicle trafficking pathways in plants, impacting crucial aspects of immunity. The exocyst complex, vital for vesicle tethering to the plasma membrane, is targeted by various effectors, disrupting immune functions. Effectors also disrupt SNARE complexes crucial for defense-related secretion and NLR-mediated immunity. PexRD12/31 from Phytophthora infestans associates with host SNAREs, likely preventing vesicle fusion (Petre et al. 2021). In addition, effectors target the endosomal sorting pathway, disrupting immune receptor endocytosis. AVR3a from P. infestans impedes FLS2 internalization, suppressing FLS2-mediated immune signaling by associating with DRP2a, a GTPase involved in endocytosis (Chaparro-Garcia et al., 2015). Pathogen effectors adeptly reprogram the trans-Golgi network/early endosomes (TGN/EE), impacting both secretion and the endocytic pathway. Effectors like HopM1 from Pseudomonas syringae target MIN7, a TGN/EE-localized protein crucial for immunity, by promoting its degradation through the host proteasome, thus dampening immune responses (Nomura et al., 2011).

Moreover, pathogen effectors interfere with plant autophagy machinery, crucial for intracellular degradation and immunity. Viral effectors like VPg from turnip mosaic virus (TuMV) hijack autophagy to evade host antiviral defences (Hafrén et al., 2018), while some effectors disrupt host RNA silencing machinery, aiding viral colonization. Additionally, pathogens remodel the host cytoskeleton to evade defenses and support virulence. Strategies like inhibiting actin-dependent endocytosis by T3E HopW1 from P. syringae or cross-linking F-actin by XopR from Xanthomonas campestris highlight the intricate interplay between pathogens and the host cytoskeleton to manipulate vesicle trafficking and immune responses(Sun et al., 2021).These findings highlight the sophisticated strategies employed by pathogens to subvert host vesicle trafficking and evade plant immunity.

In conclusion, the intricate dance between plant pathogens and their host's membrane trafficking and immune systems reveals a sophisticated battlefield where pathogens deploy an arsenal of effectors to subvert, manipulate, and exploit host cellular processes for their own benefit. From reprogramming the trans-Golgi network to hijacking autophagy machinery, these effectors orchestrate a complex interplay that undermines the plant's defense mechanisms, allowing pathogens to evade immune surveillance and colonize their host. However, as our understanding of these molecular interactions deepens, so does our potential to develop innovative strategies to bolster plant immunity and mitigate the devastating impact of plant diseases. By unraveling the secrets of pathogen effector-mediated reprogramming, researchers pave the way for new avenues in agriculture, offering hope for more resilient crops and sustainable food production systems in the face of evolving pathogenic threats.

References:

Chaparro-Garcia, A., Schwizer, S., Sklenar, J., Yoshida, K., Petre, B., Bos, J.I., Schornack, S., Jones, A.M.,

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Bozkurt, T.O. and Kamoun, S., 2015. Phytophthora infestans RXLR-WY effector AVR3a associates with dynamin-related protein 2 required for endocytosis of the plant pattern recognition receptor FLS2. PLoS One, 10(9), p.e0137071.

Hafrén, A., Üstün, S., Hochmuth, A., Svenning, S., Johansen, T. and Hofius, D., 2018. Turnip mosaic virus counteracts selective autophagy of the viral silencing suppressor HCpro. Plant physiology, 176(1), pp.649-662.

Li, T., Ai, G., Fu, X., Liu, J., Zhu, H., Zhai, Y., Pan, W., Shen, D., Jing, M., Xia, A. and Dou, D., 2022. A Phytophthora capsici RXLR effector manipulates plant immunity by targeting RAB proteins and disturbing the protein trafficking pathway. Molecular Plant Pathology, 23(12), p.1721.

Nomura, K., Mecey, C., Lee, Y.N., Imboden, L.A., Chang, J.H. and He, S.Y., 2011. Effector-triggered immunity blocks pathogen degradation of an immunity-associated vesicle traffic regulator in Arabidopsis. Proceedings of the National Academy of Sciences, 108(26), pp.10774-10779.

Petre, B., Contreras, M.P., Bozkurt, T.O., Schattat, M.H., Sklenar, J., Schornack, S., Abd-El-Haliem, A., Castells-Graells, R., Lozano-Durán, R., Dagdas, Y.F. and Menke, F.L., 2021. Host-interactor screens of Phytophthora infestans RXLR proteins reveal vesicle trafficking as a major effector-targeted process. The Plant Cell, 33(5), pp.1447-1471.

Sun, H., Zhu, X., Li, C., Ma, Z., Han, X., Luo, Y., Yang, L., Yu, J. and Miao, Y., 2021. Xanthomonas effector XopR hijacks host actin cytoskeleton via complex coacervation. Nature communications, 12(1), p.4064.