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PATHOGENS ADAPT TO THE XYLEM ENVIRONMENT TO SURVIVE AND FACILITATE SYSTEMIC SPREAD

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Abstract

The xylem, vital vascular tissue in higher plants, serves a dual role in transporting water and minerals, while also providing mechanical support. However, its strategic importance also makes it a primary target for pathogenic invasion. Several classes of pathogens—bacteria, fungi, and viruses—have evolved specialised mechanisms to colonise and persist within this nutrient-limited and high-pressure environment. The unique structural features of xylem, such as lignified cell walls and negative pressure-driven flow, present formidable barriers that only highly adapted pathogens can overcome. These invaders employ multifaceted survival strategies, including biofilm formation, secretion of cell wall–degrading enzymes, production of virulence factors, and modulation of host defence signalling pathways. Understanding the molecular interplay between pathogens and host xylem tissues provides insight into disease progression and vascular wilt syndromes that threaten global agriculture. This highlights the intricate biological and biochemical mechanisms governing xylem colonisation and the dynamic responses of host plants during vascular pathogenesis.

Introduction

Numerous pathogens, fungi, bacteria, viruses, nematodes and oomycetes are constantly attacking plants. Some invade roots and tubers, while others infect aerial parts. Some specifically target the vascular system, including phloem, which carries sugar and xylem, which carries water and minerals. Most pathogens colonize the nutrient-poor xylem because of hollow cells are easier to spread. Devastating vascular wilt diseases that can wipe out entire crops are caused by xylem-invading pathogens, which include bacteria, fungi and oomycetes. Xylem, derived from the Greek word xylon, meaning "wood," its responsible for water transport through the entire plant. It primarily consists of tracheary elements, tracheids and vessel elements, along with fibres and parenchyma cells, which provide the physical support. Since the term was first introduced by Carl Nageli in 1858, these tissues have not only physiological importance but also a vital role site of pathogen interaction. Although prominent pathogens such as Ralstonia solanacearum, *Fusarium oxysporum* and *Xylella fastidiosa* cause devastating vascular wilt diseases in a wide range of crops. These pathogens use various strategies for colonization and virulence, including as biofilm formation, secretion of hydrolytic enzymes, quorum sensing, and manipulation of host immune. These adaptation mechanisms are crucial because they provide insights into the molecular dialogue between host plant and pathogen, the evolution of virulence, and plant defense responses. Subsequently, studies on xylem–pathogen interactions serve as a crucial foundation for developing innovative approaches to manage vascular diseases that continue to

threaten global food production and agricultural sustainability.

Environment of xylem

Xylem, an essential plant vascular tissue, transfers water and minerals from the roots to the shoots and leaves. Parallel tubes with end-to-end holes and lateral pit membranes act as selective filters (<20 nm) to eliminate germs. It develops as primary xylem from the procambium, which contains protoxylem and metaxylem, and as secondary xylem from the cambium of the vascular system during secondary growth, especially in conifers and angiosperms. The xylem consists of four cells: tracheids, which consist of long cells with bordered pits for conduction of water and plant support; vessel elements, which are wide axial tubes in angiosperms that enable efficient water transport; xylem parenchyma, which consists of living cells that are arranged radially or axially for storage and downstream transport; and xylem fiber, which is a lignified dead cell its provide the mechanical strength for plants. While in microscopic view, the xylem cells are shown to have a star-like shape. The Xylem sap transports water and soluble nutrients through a passive process, such as transpiration pull, root pressure, and pressure flow interaction with phloem. While the osmotic gradients provide the transpirational pull from evaporation at the mesophyll cell, which generates the negative pressure, it may indirectly influence the xylem sap flow speed, approximately 0.1 to 0.4 mm/s . According to the cohesion theory. The transpirational pull is primarily liable for the upward (acropetal) movement of the water. However, the positive pressure may occur on a particular day or season. In recent research, xylem sap, vessel wall, and pit membranes consist of insoluble surfactants that coat the hydrophobic cover with nanobubbles. Thereby decreases the surface tension and avoids the formation of embolism. The result ensures the stability of continuously moving water columns, which play a vital role in plant growth and survival. Furthermore, research explores pathogens like Fungi, Oomycetes, and bacteria that use the xylem as a pathway to colonize for their survival. Over 400 million years of xylem evolution allowed plants to effectively supply water to photosynthetic tissues, promoting growth and survival

Pathogen entry into the xylem

Plant pathogen entry through natural openings like stomata, wounds triggered by environmental damage, insects, nematodes and natural new emerging root sites, which are more susceptible to pathogen entry (Table 1).

Impact of wilt pathogens on xylem structure and function

Wilt diseases, xylem-invading pathogens, seriously impair plant water transport and subsequently result in death. Bacteria like *Xylella fastidiosa* and fungi like Fusarium oxysporum and *Verticillium longisporum* are major wilt-inducing pathogens. These pathogens are usually introduced directly by insect vectors (leafhopper, aphids) or through roots, which move from the epidermis to the cortex and endodermis before colonising the xylem. Rapid xylem-based responses, such as the deposition of lignin, suberin, and gels, and also the formation of tyloses to defend against the pathogen spread, are essential. For instance, plants create tyloses and gels to restrict bacterial movement in *X. fastidiosa* infections, but cultivars with larger vessels and a higher risk of embolism are more vulnerable. Additionally, pathogens can rewire host xylem. When *Arabidopsis* is infected with *V. longisporum*, bundle sheath cells undergo VND7-mediated trans differentiation into new xylem elements, which enhances water transport and partially drought tolerance. Similar to this, *Xanthomonas oryzae pv. oryzae* infection in rice

causes quick pit narrowing and secondary wall thickening, which limit bacterial spread and are responses associated with peroxidase accumulation.

Entry Point	Host	Pathogen
Stomata	*Clavibacter michiganensis* subsp. *michiganensis*	Tomato
	Ralstonia solanacearum	Tomato, Banana
	Pseudomonas syringae pv. *syringae*	Bean, Cherry
	Ralstonia syzygii subsp. *indonesiensis*	Clove, Banana
	Xanthomonas campestris pv. *malvacearum*	Cotton
	Puccinia	Cereals
Hydathodes	*Stenotrophomonas xanthophila*	Various
	Xanthomonas campestris pv. *campestris*	Cabbage
	Xanthomonas oryzae pv. *oryzae*	Rice
Root	*Fusarium oxysporum*	Banana, Tomato, Cotton
	Verticillium dahliae	Cotton, Olive, Potato
	Verticillium klebahnii	Tomato, Eggplant
	Ralstonia solanacearum	Tomato, Banana, Potato
	Ralstonia syzygii subsp. indonesiensis	Clove, Banana

Pathogen Adaptation

A) Cell wall degradation

The plant pathogen, Fungi and bacteria secrete the cell wall-degrading enzymes to break the structural barrier of the plant cell wall, which is made up of cellulose, hemicellulose, pectin, lignin, and proteins, all of which together provide the strength. To successfully invade, the pathogen secretes a high amount of cell wall-degrading enzymes. It is particularly abundant in fungal pathogens compared to symbiotic microbes. Cellulose is a major component in the cell wall; it is targeted by cellulases such as endoglucanases, cellobiohydrolases, and β-glucosidases, which are produced by pathogens. However, cellulase is a key role in infection. *Magnaporthe grisea* (rice blast fungus). Cellulases help penetration and *Clavibacter michiganensis* (bacterium): cellulases are key for tomato wilt disease and spreading inside tissues. However, infection starts with pectin degradation. Because pectin present in the middle lamella acts as a glue that holds plant cells together. Pathogens degrade the cell wall adherence and develop the entry site by releasing enzymes such as polygalacturonases (PGs), pectin lyases (PLs), and pectin methylesterases (PMEs). These initial infection influences the breakdown of hemicelluloses such as xylans, mannans, and xyloglucans, which are disintegrated by xylanases, mannanases, and other hemicellulose. The enzymes help to enable the infection and colonize the pathogen. which have been proven in some research. The full virulence of *Alternaria citri* on citrus depends on the activity of the single polygalacturonases (PGs). While *Botrytis cinerea* secretes massive amounts of pectinases and xylanases to macerate tissues and spread throughout the tissues and leading to necrosis symptoms in tomato and apple.

B) Manipulation of host stomal responses

Stomata are small pores on the plant surface that regulate gas exchange, transpiration, and water balance, but also act as a natural site for pathogen invasion. Plants can rapidly close their stomata in response to pathogen-associated molecular patterns (PAMPs), thereby preventing the pathogen from entering. It has been determined that this PAMP-triggered closure in stomata is a common and significant protective mechanism. The pathogen has some complex functions to control the plant's function. For instance, bacteria such as *Xanthomonas, Erwinia amylovora,* and *Pseudomonas syringae* release effector proteins such as XopD and AvrE, which trigger the ABA signaling in plants and this leads to closure of stomatal pores. The plant is not able to be involved in the transpiration process and store the water, which is a suitable environment for plant growth. Likewise, *Xanthomonas campestris* pv. *Campestris* (Xcc) causes the disease in *Brassicaceae* plants through hydathodes and stomata by secreting molecules regulated by diffusible signal factor (DSF) that also help the bacteria adhere to the plant by using quorum sensing, which also governs biofilm formation and expresses the virulence factor.Toxins produced by certain pathogens can affect stomatal opening and closure either directly or indirectly. For example, *Alternaria tenuis* produces Ten toxin, which causes stomatal closure, while *Drechslera maydis* race T produces T-toxin and *Fusicoccum amygdali* produces fusicoccin, which encourage stomatal opening. In order to promote pathogen infection and colonization, these toxins are essential for modifying host physiological processes These findings suggest that stomatal immunity is a crucial component of plant defense mechanisms, but the pathogen manipulates stomatal closure, enabling successful adaptation and colonization.

C) Vascular plugging theory and Phytotoxin

Some fungal pathogens cause wilt diseases that penetrate the plant through the growth of mycelium and spore production within xylem vessels. These spores are carried by the xylem sap stream upward in the plant and germinate the spore forming the new mycelium and continuing the colonization. It had two mechanisms for wilt development: Vascular plugging and toxin production. In vascular plugging, the fungal hyphae, spores accumulate in the xylem vessel (e.g., Verticillium, Fusarium), while the host defences respond to produce tylose formation, callose and gum, which lead to inadequate water movement and reduce the hydraulic conductivity in xylem. In the toxin production, some pathogens secrete the phytotoxin that affects water movement in xylem and damages the living cell and causing the wilt symptoms without any physical blockage. For instance, Experimental studies prove that *Fusarium oxysporum* survives in soil or debris as a chlamydospore and penetrates the host root, spreads through the cortex and proliferates in the xylem. Inside the vessels, microconidia and macroconidia are systematically spread to the entire plant via the sap stream and causing clogging and wilt symptoms. Likewise, *Fusarium*. Sp produces toxins such as Fusaric acid and T-2 toxin, which degrade the protein and cause the wilt symptoms. The Tri5 gene plays a significant role in the biosynthesis of T-2 toxin; it is enabled by a transcription factor (Yap1) under stress conditions. Pathogens are using a potential target for manipulating the strategies to survive the pathogen within the xylem.

D) Quorum Sensing

Quorum sensing is an initial regulatory mechanism for bacterial communication and biofilm development. This mechanism is mediated by the two-component signal transduction system (TCSTS), which consists of a membrane-bound histidine kinase sensor, signaling molecule and an Intercellular response regulator known as the autoinducer (AI). When the AI component reaches threshold concentration, it activates the transcriptional regulation of genes, which involves expressing the virulence's and forming the biofilm. Gram-negative bacteria commonly use the LuxI/LuxR-type QS system via acyl homoserine lactones (AHLs), whereas gram-positive bacteria depend on a peptide-based two-component system. Furthermore, a ubiquitous system, mediated by LuxS-produced autoinducer-2 (AI-2) is found in both negative and positive bacteria, there is considered as a common QS pathway. It has been developing the biofilm growth, maturation and modulating the virulence factor. Research found, *Streptococcus pneumoniae* utilizes luxS to stimulate the virulence factors like ply and lytA, other than *Staphylococcus epidermidis* uses a similar pathway, but the secondary gene regulator (agr) system also promotes the early formation of biofilm by releasing the extracellular DNA via Autolysin E (AtlE) enzymes. It leads the facilitated surface adhesion and supports biofilm formation.

Strategies for Nutrient Acquisition:

The xylem had a poor and low oxygen environment, so the microorganisms are adapted to survive on scarce resources. The metabolite concentration in the xylem is 100 times less than in the apoplast or phloem. Xylem had some organic compounds such as amino acids (glutamine, asparagine), carbohydrates (glucose, fructose, sucrose, maltose, raffinose, trehalose, ribose), organic acids (malate, citrate), and protein, whose concentration differs from plant species. But Pathogens have developed numerous strategies to acquire nutrients, such as responding to metabolites as signals, enzymatically digesting cell walls and invading neighbouring tissues. In research, *Ralstonia solanacearum*–tomato, changes the quantity of some nutrients during the infection, which may act as the metabolic resources and signaling molecules that promote the growth of the pathogen. When compared to sap from healthy plants and xylem sap extracted from diseased plants consistently promotes bacterial growth(Lowe-Power et al., 2018). Some pathogens manipulate the pH level of the xylem sap its create the favorable condition for their growth. Normally, the xylem under slightly acidic in nature, but during the infection, it shifts to an alkaline state. For instance, *Fusarium oxysporum* not only produces the ammonium to increase the pH level, but also secretes the alkalinization peptide that can raise the pH level; these also help to modify the nutrients and enable the metal element within xylem, altering the physiological pressure on the host. For instance, mutagenesis studies revealed that sucrose levels accumulate in the xylem sap of plants infected with sucrose utilization mutants (scrA), whereas no such increase occurs in wild-type infections, indicating that wild-type *R. solanacearum* consumes available sucrose. In addition to sucrose, alanine, trehalose-6-phosphate, GABA, and putrescine also accumulate in the xylem following infection. A similar enrichment of putrescine has been observed in the xylem sap of tomato infected by Verticillium dahliae. Such nutrient enrichment is thought to be mediated in part by bacterial effectors, analogous to the TAL effectors of Xanthomonas that activate host SWEET genes, thereby enhancing sucrose transport to the apoplast. While it is sometimes unclear whether

these compounds act primarily as nutrients or as signaling molecules, they most likely serve both roles.

Host defence mechanisms:

Higher plants' ability to withstand stress is vital to their existence. As a result, plants have been forced to evolve to adapt to stress through modifications to their normal patterns of defense mechanisms. In response to a pathogen attack, every plant has a wide range of defense mechanisms. All plants have the capacity to activate these defenses. However, if they are activated too little, too late, or in the wrong place, they will fail to restrict the pathogen, and the plant will be susceptible. These induced defense responses are controlled by defense-related genes, which is a required trade-off in terms of plant fitness.

A) Abscission layer

An abscission layer is made up between the gap of healthy and infected tissues. Abscission occurs at a specialized layer of cells called the abscission zone (AZ). The extracellular matrix of AZ is degraded by polygalacturonases to cell separation. Pectin depolymerases, known as polygalacturonases, hydrolyze alpha-1,4 glycosidic linkages that bind the galacturonic acid residues of pectin. The production of the abscission layer is also induced by salicylic acid and hormones like as ethylene, auxin and jasmonic acid. They help in the production of placygalacturonase, which plays a role in the creation of the abscission layer. E.g. *Closterosporium carpophylumon* on peach leaves.

B) Cork layer formation

Plants are infected by fungi, nematodes. Bacteria and certain viruses lead to the formation of cork cells beyond the cell of infection. The cork cell stops the pathogen from penetrating, further lesions and also blocks the spread of the pathogen and its toxin, which stops the flow of nutrients and water from healthy to infected area and deprives the pathogen of nourishment. These secondary meristems that secreted cork toward the outside. The quantity of Suberin in cork cells' cell walls is primarily responsible for their protective role. Periderm is the aggregate term for phellogen and phelloderm collectively is known as Periderm. E.g. Potato tuber disease caused by *Rhizoctonia* sp., Scab of potato caused by *Streptomyces scabies*.

C) Gel deposition

The plant produces various types of gum around the lesion after infection. Although gums are secreted by various plants, stone fruit trees are the most frequent. When a plant is under stress, it usually exudes gum. Gums have a protective role because they are rapidly deposited in the cells and intercellular spaces surrounding the site of infection, creating an impenetrable barrier that entirely encloses the pathogen. After that, the pathogen isolates itself, starves, and eventually perishes.

D) Tyloses formation

Tyloses are outgrowths of xylem parenchyma cells that extend into xylem vessels, forming cellulosic walls that can completely block the vessel lumen. Their formation is often regulated by plant hormones such as auxin, ethylene, and jasmonate, and they serve as a defense mechanism by acting as antimicrobial barriers, preventing the spread of pathogens through the vascular system.

Pathogen genomic features in connection with xylem

Because of few species that are exclusively xylem-resident, it is difficult to comprehend xylem specialisation in pathogens. *Xylella fastidiosa* dominates the genomic data in bacteria with over 150 genomes. Important elements have been brought to light by comparative genomics, such as the hydrolase

enzyme CbsA in *Xanthomonadaceae*, which is necessary for vascular colonization. In X. fastidiosa, xylem-limited bacteria not have type III secretion systems (T3SS), which is consistent with little interaction with living cells. To suppress plant defenses and manipulate neighbouring parenchyma cells, facultative xylem pathogens such *as Ralstonia solanacearum* and *Erwinia tracheiphila* express T3SS effectors, suggesting a variety of host interaction strategies. Adaptation to the nutrient-poor xylem is frequently reflected in genome reduction and pseudogene accumulation. While more recent colonists like *Leifsonia xyli* subsp. xyli and R. solanacearum have a higher pseudogene content, X. fastidiosa exhibits a streamlined genome, perhaps as a result of ancient adaptation (>700 Mya). In fungi, genomic characteristics are also connected to host adaptation. In *Fusarium oxysporum* and *Verticillium dahliae*, virulence genes, including all known small cysteine-rich Secreted in Xylem (Six) effectors, are carried by supernumerary chromosomes, some of which contribute to pathogenicity. Host-specific virulence factor pseudogenes, like FoPDA1, are a reflection of evolutionary divergence and host-specific adaptation. All things considered, xylem-adapted pathogens employ a mix of effector deployment, genome reduction, and specialized chromosomes to endure in an environment deficient in nutrients. It is still unclear how many pseudogenes and effectors function during infection.

Conclusion:

To infect and colonise the vascular system, pathogens that live in the xylem must get past formidable physical and metabolic obstacles. Effective entrance mechanisms, cell wall disintegration, host response manipulation, and sophisticated communication systems like quorum sensing are all examples of their adaptations. By adopting these tactics, viruses can withstand nutrient shortages and circumvent host defenses, which eventually results in water stress, wilting, and other disease signs. A thorough comprehension of these adaptive processes' safeguards plant health and productivity by shedding light on plant vascular diseases and

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