



REVIEW ARTICLE

Host-pathogen interaction in bacterial leaf blight of rice: Mechanisms of infection, colonisation and symptom development

Syed Mohamed S¹, Rajinimala N^{1*}, Theradimani M¹, Jeberlin Prabina B², Kavitha Pushpam A³, Sheela J¹ & Varshini M¹

¹Department of Plant Pathology, V. O. Chidambaranar Agricultural College and Research Institute, Tamil Nadu Agricultural University, Vallanad 628 252, Tamil Nadu, India

²Department of Soil Science and Agricultural Chemistry, V. O. Chidambaranar Agricultural College and Research Institute, Tamil Nadu Agricultural University, Vallanad 628 252, Tamil Nadu, India

³Department of Crop Physiology and Biochemistry, V. O. Chidambaranar Agricultural College and Research Institute, Tamil Nadu Agricultural University, Vallanad 628 252, Tamil Nadu, India

*Correspondence email - rajinimala@tnau.ac.in

Received: 02 February 2026; Accepted: 22 April 2026; Available online: Version 1.0: 04 August 2026

Cite this article: Syed MS, Rajinimala N, Theradimani M, Jeberlin PB, Kavitha PA, Sheela J, Varshini M. Host-pathogen interaction in bacterial leaf blight of rice: Mechanisms of infection, colonisation and symptom development. Plant Science Today. 2026; 13(sp5): 1-11. <https://doi.org/10.14719/pst.13948>

Abstract

Bacterial leaf blight (BLB) is a major constraint to rice production worldwide due to its rapid spread and the high genetic variability of its causal agent, *Xanthomonas oryzae* pv. *oryzae*. The pathogen primarily enters rice plants through natural openings such as hydathodes or through wounds, subsequently colonising the xylem vessels and spreading systemically within the vascular system, leading to characteristic disease symptoms. The ability of the bacterium to infect the host depends on several virulence factors, including biofilm formation, exopolysaccharide production, quorum sensing and the secretion of effector proteins into the host. These proteins, known as transcription activator-like (TAL) effectors, play a crucial role in pathogenesis by modulating host gene expression to facilitate infection. Rice plants counter BLB through multiple defence mechanisms, including the formation of structural barriers, production of antimicrobial compounds and modulation of gene expression. Some rice cultivars exhibit greater resistance to BLB than others, which is largely determined by genetic factors. This review examines the defence strategies of rice and provides key insights into host-pathogen interactions that inform the development of durable resistance and improved disease management strategies. Understanding these interactions provides a foundation for integrating molecular breeding with sustainable agricultural practices to mitigate yield losses caused by BLB.

Keywords: bacterial leaf blight; disease resistance; host-pathogen interactions; *Xanthomonas oryzae* pv. *oryzae*

Introduction

Rice (*Oryza sativa* L.) is a staple food for more than half of the world's population. A significant portion of the global population, particularly in Asia and Africa, is dependent on rice, making the maintenance of optimal rice yields essential for global food security. Bacterial leaf blight (BLB), caused by *Xanthomonas oryzae* pv. *oryzae* (Xoo), is recognised as one of the most destructive diseases affecting rice. Under favourable conditions, this disease can dramatically reduce yields (1, 2). The BLB spreads rapidly, especially in regions where rice is cultivated intensively and irrigation is widely used (3). Despite the use of resistant rice varieties and improved agricultural practices, BLB remains problematic because the pathogen continues to adapt and evolve (4). The disease process involves a complex interaction between the rice plant and the pathogen. It begins when Xoo enters the host plant, primarily through hydathodes or mechanical wounds at the leaf tips. Once inside, the bacteria colonise the xylem, obstructing water transport as they proliferate and move throughout the plant (5). During infection, Xoo

employs various virulence factors, including the production of extracellular polysaccharides, biofilm formation and the secretion of effector proteins that enable it to evade plant defences and establish itself within the host (6). These mechanisms are closely tied to the rice plant's physiological and biochemical responses, influencing the speed and severity of disease development (7). From the plant's perspective, rice responds to Xoo through multiple strategies. It strengthens structural barriers, increases the activity of defence enzymes and alters the expression of immune-related genes (8). The outcome, whether resistance or susceptibility, largely depends on host genes and their interactions with pathogen effectors (9). Additionally, environmental factors such as temperature, humidity and crop growth further influence the course of infection in the field (10). A thorough understanding of how Xoo infects, spreads and induces symptoms is crucial. This knowledge is fundamental for breeding rice varieties with durable resistance and for sustainably managing BLB. This review compiles current knowledge on host-pathogen interactions in BLB, with emphasis on the biological mechanisms of disease progression and their relevance to

sustainable rice production.

Pathogen and host overview

Xanthomonas oryzae pv. *oryzae* (Xoo) causes BLB in rice—a disease that severely affects rice production and economic losses for farmers worldwide. The pathogen Xoo is classified within the Lysobacteraceae family. It is a gram-negative, rod-shaped, aerobic bacterium that colonises the vascular tissues deep within rice plants (1). It is highly adapted to rice and its genetic and pathogenic variability means the disease manifests differently depending on the region. The severity varies based on location, making it a challenging threat in various agro-ecological zones (3). The strong evolutionary link between Xoo and rice has established this pathosystem as a recognised model for examining host-pathogen interactions in cereal crops (11). Rice is the only known natural host of Xoo, but its response to BLB depends greatly on its genotype, growth stage and environmental conditions. The disease specifically targets the leaves. The pathogen Xoo enters the xylem, multiplies and disrupts water transport, which can severely damage tissues (2). Young seedlings and plants at the peak tillering stage are most vulnerable, especially when conditions favour the pathogen. In such cases, yields can drop significantly (12). The plant's own physiology—such as leaf age or nutrient balance—also influences how the disease manifests and how rapidly the pathogen spreads (13). The pathogen persists between growing seasons in infected crop debris, on volunteer rice plants and in irrigation water, enabling it to survive and spread wherever rice is cultivated. Rain splash, irrigation water and mechanical movement help disperse the pathogen, allowing it to reach leaf surfaces and enter through natural openings like hydathodes (1). Once established, the pathogen swiftly reproduces in the vascular system, supported by its capacity to evade or inhibit host defence mechanisms (3). Population genomic research has additionally demonstrated that Xoo shows significant regional structuring, indicating long-term adaptation to rice varieties cultivated locally (4). From the viewpoint of the host, rice has developed a variety of resistance mechanisms to combat BLB, spanning from basic defence responses to race-specific resistance facilitated by Xa genes. Nevertheless, the ongoing development of Xoo populations frequently results in the failure of resistance, emphasising the ever-changing dynamics of the host-pathogen interaction (2). Variations in host anatomy, especially the configuration and function of vascular tissues, significantly influence the degree of pathogen infiltration and the severity of disease (14). These host characteristics closely interact with pathogen virulence factors, influencing the result of the infection. Recent progress in genomics and molecular biology has greatly enhanced comprehension of both pathogen biology and host responses within the rice-Xoo system. Comparative genomics reveals striking differences in virulence genes among Xoo strains and these differences shape how the bacteria interact with rice (15). Concurrently, transcriptomic and physiological studies in rice elucidate on how the plant's metabolism and defence systems kick into gear during infection (11). Taken together, the evidence shows that BLB emerges from a finely tuned battle between a specialised pathogen and a genetically diverse host. To fully comprehend this pathosystem, it is necessary to conduct depth analyses into both sides of the equation.

Infection process and entry mechanisms

Bacterial leaf blight (BLB) in rice begins when Xoo reaches susceptible parts of the plant under favourable environmental

conditions. Rain splash, irrigation water and fragments of infected plant debris mainly facilitate the spread, transporting the bacteria to the above-ground tissues of the rice plant as it grows (2, 12). Elevated temperatures and increased relative humidity greatly improve bacterial persistence on leaf surfaces and facilitate initial infection occurrences, especially in densely planted and nitrogen-abundant cropping systems (10). Hydathodes are located right at the edges of leaves and serve as the primary entry point for Xoo, especially in the early morning when guttation fluid seeps out (5). These structures open directly into the xylem, allowing the pathogen to bypass the usual epidermal barriers and enter straight into the plant's vascular system. This is how Xoo establishes itself and initiates infection (1). Researchers have employed transcriptomics and microscopy to demonstrate that Xoo colonises hydathodal tissue prior to systemic dissemination. This makes hydathodes a key entry route in BLB pathogenesis (16, 17). Of course, hydathodes are not the only entry points—mechanical damage from wind, insects, or agricultural practices can also allow bacteria to enter leaf tissue (18). While wound-mediated entry is deemed secondary, it gains epidemiological significance during extreme weather conditions or in intensively managed agricultural fields (12). Once inside, Xoo attaches itself to the host's cell walls and rapidly multiplies in the spaces between the cells. It does not remain there for long. Soon, it moves into the vascular system, where it finds abundant nutrients and continues to grow (19). During the early stages, the host's defence response is minimal especially in susceptible cultivars, so the bacteria become established with little resistance. In this stage, Xoo inhibits basal defence systems and alters host cellular activities to enhance colonisation (20). When this pathogen escapes early detection, it rapidly accumulates in the xylem. This is how it moves throughout the plant and symptoms begin to appear (21). The entry and establishment of Xoo is not random—it follows a process influenced by the host's structure, environmental conditions and the pathogen's own adaptability. Gaining insight into what occurs during those initial moments is crucial. It is essential for developing targeted disease management strategies, particularly those that prevent early colonisation and stop the pathogen from spreading in rice crops (3, 11). Although hydathodes are widely acknowledged as the principal entry point for Xoo, the relative relevance of other entry pathways varies depending on the environment. While some studies focus on hydathode-mediated infection as the primary channel (5, 16), others highlight the epidemiological significance of wound-based entry under field stress settings (12, 18). This shows that infection is not controlled by a single fixed mechanism, but rather by environmental and host-related factors. Collectively, these findings suggest that early infection success is dependent on the interplay of entry route, host vulnerability and environmental factors rather than pathogen presence alone (Fig. 1).

Host-pathogen recognition events

The early stage of BLB progression in rice is regulated by intricate host-pathogen recognition interactions that decide if infection continues or is halted. When Xoo arrives on a rice leaf, it encounters both physical and biochemical barriers that influence subsequent interaction events (1). Rice plants detect Xoo by sensing conserved microbial markers, such as flagellin and lipopolysaccharides. Pattern recognition receptors (PRRs) located on the surface of rice cells identify these signals and initiate the plant's primary immune response (20). Notably, while some studies report rapid activation of basal immunity following pathogen detection (3), others indicate

Disease cycle of bacterial leaf blight

(*Xanthomonas oryzae* pv. *oryzae*)

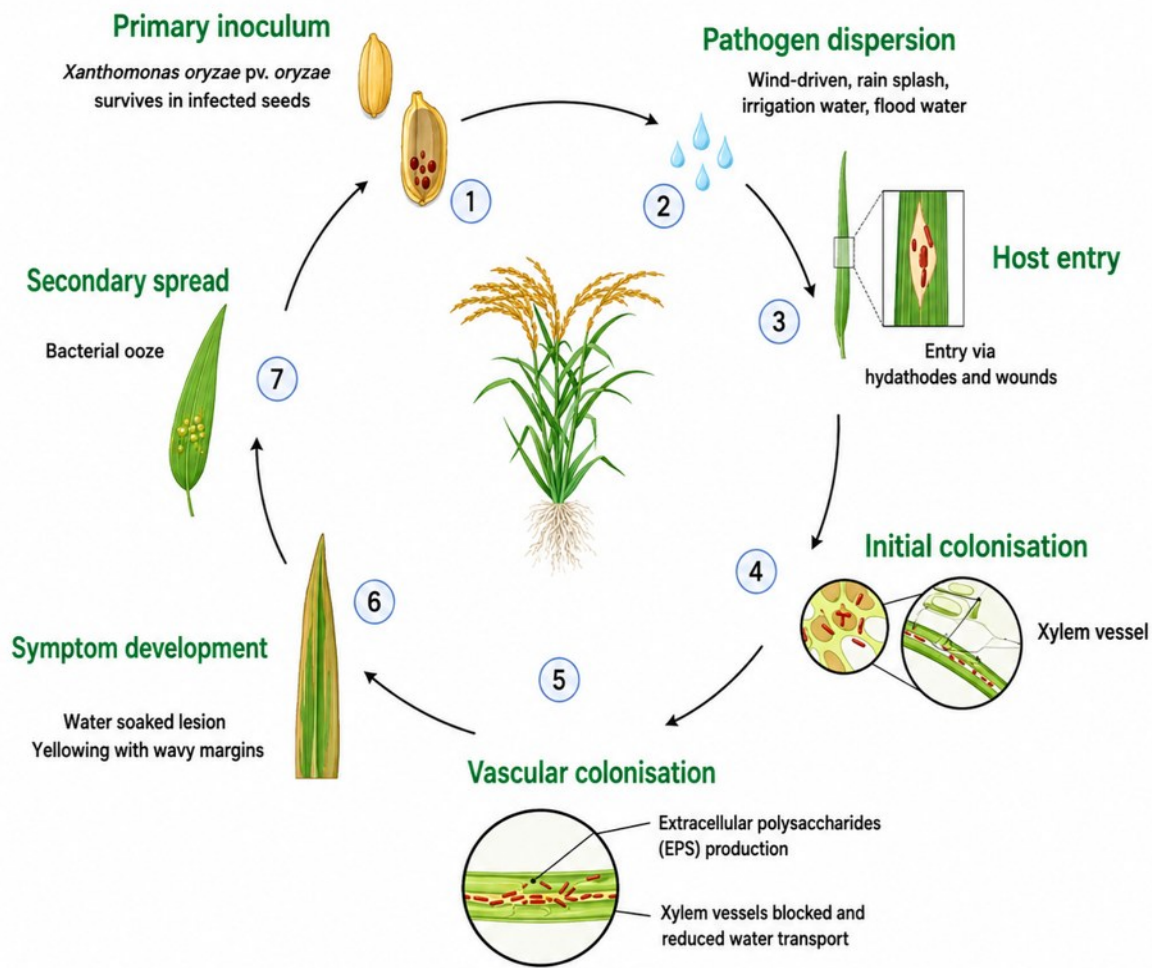


Fig. 1. Schematic representation of the disease cycle of bacterial leaf blight in rice, illustrating initial colonisation, vascular colonisation, extracellular polysaccharides (EPS) production and symptom development.

that this response is delayed or insufficient in susceptible cultivars (21), suggesting that timing and intensity of immune activation are critical determinants of disease outcome. Upon detection, the plant initiates an immediate response involving altered ion fluxes, increases the production of reactive oxygen species (ROS) and activates numerous defence-related genes. These responses hinder the pathogen's ability to establish itself (22). However, not all rice plants react identically. The efficiency of this recognition is determined by the rice genotype and specific characteristics of the pathogen. Ultimately, the outcome whether the rice becomes diseased or remains healthy, depends on the interplay between the plant's initial immune response and the pathogen's capacity to overcome it (11). After the first detection, Xoo quickly implements molecular tactics to disrupt host identification processes. The pathogen employs its type III secretion system to inject effector proteins into host cells, several of which attack essential elements of immune signalling pathways (23). These effectors adjust the transcriptional machinery of the host, allowing for the repression of defence-related genes soon after infection (24). Transcriptomic analyses indicate that susceptible rice varieties initiate their defence gene response later or less robustly than resistant types at this stage (25). Phytohormones including salicylic acid, jasmonic acid and

ethylene modulate the recognition process. In susceptible plants, these hormonal interactions often end up facilitating the pathogen's establishment (26). This early molecular-level disruption underscores how adept Xoo is at evading the plant's defences (27). Host recognition events are influenced by the spatial context of infection, especially when pathogens enter through hydathodes and injuries. Cells that border this access points show unique immune functionality and transcriptomic reactions in contrast to mesophyll tissues (16). Research has shown that rice hydathodes have immune responses that can be triggered, but these defences frequently fall short of completely controlling vascular pathogens such as Xoo (17). Visualisation of initial infection phases has shown localised inhibition of defence signalling near colonised vascular tissues, implying specific pathogen disruption (19). Tissue-specific differential recognition leads to varied defence activation throughout the leaf (28). This spatial variation in recognition responses is crucial for influencing the effectiveness of systemic infection (18). Host genetic elements additionally affect the effectiveness of pathogen detection. Multiple rice resistance (*Xa*) genes code for receptor-like kinases or executor proteins that either directly or indirectly identify pathogen effectors (9). In resistant varieties, the detection of TAL effectors

triggers swift initiation of defence signalling pathways and localised cell death, effectively restricting bacterial dissemination (29). On the other hand, in vulnerable genotypes, TAL effectors activate host susceptibility genes such as SWEET sugar transporter genes, which facilitate nutrient supply to the pathogen (30). Analyses at the population level suggest that ongoing pathogen evolution modifies effector repertoires, facilitating the avoidance of host recognition systems as time progresses (15). This dynamic co-evolution highlights the volatility of resistance founded on recognition (4). Together, host-pathogen recognition interactions signify a critical stage in BLB progression, encompassing molecular detection, immune signalling, tissue specificity and genetic compatibility. Effective disease onset relies on the pathogen's capacity to hinder or alter initial recognition responses prior to strong defences being activated (2). Progress in genomics and functional research has greatly enhanced comprehension of these initial occurrences, uncovering possible targets for sustainable resistance breeding (31). Nonetheless, the swift evolution of Xoo populations persists in posing difficulties for defence strategies reliant on host recognition (4). An in-depth comprehension of recognition mechanisms will be crucial for creating sustainable strategies for managing BLB (14). Taken together, these results indicate that host-pathogen identification is a dynamic relationship influenced by timing, tissue specificity and genetic compatibility. While resistant genotypes immediately convert identification into effective defence, susceptible contacts appear to include partial or delayed immune activation, which is then inhibited by pathogen effector proteins. This suggests that disease establishment is caused by the pathogen's ability to alter early immune signals rather than a lack of recognition (11, 14).

Colonisation and systemic movement

Upon successful invasion of the host, Xoo settles within the rice leaf by occupying intercellular spaces prior to infiltrating the vascular system. Initial research showed that bacterial growth first takes place in the apoplast around hydathodes before spreading systemically (32). Colonisation patterns specific to tissues indicate that virulent strains quickly adapt to the xylem environment, whereas avirulent strains remain localised (28). Microscopic examinations revealed that bacterial cells congregate along vascular bundles, suggesting guided movement within host tissues (19). Quantitative analyses verified that the size of pathogen populations grows exponentially in the initial stages of colonisation (33). These results collectively indicate that effective colonisation is necessary for the progression of systemic disease (12). The pathogen primarily spreads systemically via the xylem vessels, allowing it to travel long distances from the infection site to distant leaf areas. Fluorescent labelling studies offered direct proof of bacterial movement via xylem elements, linking it to the manifestation of distinct blight symptoms (34). *Xanthomonas oryzae* pv. *oryzae* survives effectively in xylem sap, which enables it to persist and spread within its host (35). When researchers compared resistant and susceptible rice genotypes, they observed that resistant plants restricted the movement of the bacteria, demonstrating how the host actively regulates the pathogen's capacity to colonise the plant (36). Analyses of population dynamics revealed that systemic spread correlates with heightened bacterial density instead of just physical movement (21). These findings highlight the significance of vascular adjustment in disease advancement. The efficiency of colonisation is significantly impacted by bacterial movement and chemotactic reactions in host

tissues. Mutants lacking motility showed a diminished capacity to navigate the leaf vasculature, leading to less severe disease symptoms (37). Research on tissue invasion indicated that synchronised movement through xylem vessels promotes swift colonisation in advantageous environmental conditions (32). Host anatomical traits, including vessel diameter and pit membrane configuration, likewise influence the efficiency of bacterial movement (28). Additionally, studies on the time course of infections showed that systemic colonisation occurs alongside expanded lesions at the edges of leaves (38). These results emphasise the relationship between bacterial characteristics and host architecture in influencing colonisation achievement. The degree of systemic movement is directly related to the severity of the disease and the expression of symptoms. Quantitative evaluations indicated that increased bacterial levels in the vascular system lead to significant leaf blight and wilting (36). Physiological research associated systemic colonisation with disrupted water movement, resulting in chlorosis and necrosis (39). The spread of pathogens within xylem tissues has been linked to changes in host metabolism and nutrient reallocation (40). Field investigations demonstrated that cultivars with restricted systemic movement show slower symptom progression (12). Consequently, systemic colonisation serves as a key factor influencing disease severity. Host defence mechanisms significantly contribute to limiting the colonisation and systemic dissemination of the pathogen. Resistant rice lines have been found to inhibit bacterial growth in vascular tissues by triggering early defence mechanisms (36). Genetic studies showed that resistance genes affect the geographic distribution of bacterial populations inside the leaf (31). Histological analyses additionally revealed structural obstacles that hinder xylem colonisation in resistant genotypes (19). Decreased systemic movement in resistant plants ultimately leads to reduction in disease occurrence and severity (35). These shows significance of colonisation and movement of BLB. Although individual studies describe bacterial movement, population dynamics and host anatomical influences separately, their integration reveals that systemic colonisation is a coordinated process rather than a purely passive spread. While high bacterial density is strongly associated with symptom development (21, 36), host structural barriers and early defence responses can significantly restrict this movement (31). This suggests that disease progression depends on the balance between bacterial proliferation and host-imposed spatial limitations, highlighting colonisation as a regulated interaction rather than uncontrolled expansion.

Biofilm formation, extracellular polysaccharides and vascular occlusion in the pathogenesis of bacterial leaf blight

The formation of biofilm is an essential virulence tactic used by Xoo that aids in bacterial survival and persistence in the vascular system of the rice host (41). The establishment and persistence of biofilms are closely governed by cell-cell communication systems, especially quorum sensing, which synchronises behaviour at the population level during infection (42). Genetic research has shown that alterations in regulatory genes governing signalling pathways greatly hinder biofilm formation and diminish pathogenicity in rice (6). Comparative studies among *Xanthomonas* species also suggest that biofilm production is a preserved characteristic linked to host colonisation and the development of disease (43). Taken together, these results demonstrate that biofilm formation is crucial for the virulence of Xoo during BLB (41). Extracellular polysaccharides, particularly xanthan, maintain the integrity of the biofilm and

stabilise it within plant tissues (44). When Xoo synthesises more exopolysaccharides (EPS), the bacteria adhere more firmly to the xylem vessels and shield themselves from the host's antimicrobial compounds (45). Even though EPS isn't always crucial for the initial infection, it plays a major role in the epiphytic survival and the lasting presence of *Xanthomonas* populations on plant surfaces (46). Disruption of EPS biosynthesis pathways has been demonstrated to impair biofilm structure and reduce virulence in rice (44). These findings highlight the significance of EPS facilitated biofilm strengthening in effective vascular colonisation by Xoo (45). Interestingly, while some studies suggest that EPS is not essential for initial infection (46), others demonstrate its critical role in maintaining long-term colonisation and virulence (44, 45), indicating that EPS functions are stage-specific during disease development. Biofilm accumulates inside rice xylem vessels and obstructs the plant's vascular system. Consequently, the transport of water and nutrients is impaired, causing the plant to display obvious symptoms (41). Under the microscope, dense clusters of bacteria embedded in viscous EPS matrices are observed occluding the xylem lumens, particularly in the later stages of infection (43). As the vascular system deteriorates, leaves wilt, turn yellow and the characteristic blight symptoms appear, especially in rice varieties that are more susceptible (6). For the pathogen to spread throughout the plant, it must regulate when biofilm cells detach and disperse. There is a balance between adhering to the vessel walls and releasing cells to colonise new areas (41). When biofilms block the xylem, it is not merely a physical barrier, but it is a direct indicator of how this disease develops and damages rice. Chemotaxis and flagella-driven movement enable Xoo cells to navigate host tissues, allowing them to settle in locations within the vascular system where they can form new biofilms and perpetuate the infection. Mutants lacking motility show decreased biofilm formation and a lower capacity to initiate systemic infections in rice plants (37). The shift from a mobile to a stationary way of life is governed by quorum-sensing networks that manage EPS production and surface adhesion (42). Disruption of these regulatory pathways experimentally changes biofilm structure and undermines bacterial fitness in the host (6). These results highlight the interconnected functions of motility, signalling and biofilm formation in Xoo pathogenicity (43). Focusing on biofilm formation has arisen as a promising approach for decreasing Xoo virulence and controlling BLB. The use of chemical inhibitors to disrupt biofilm formation has been demonstrated to greatly reduce bacterial colonisation and disease severity in experimental settings (47). Genetic alteration of genes related to biofilm also leads to reduced virulence and restricted vascular obstruction in rice plants. These investigations show that the integrity of biofilm is crucial for the complete pathogenic expression of Xoo during an infection (48). As a result, interventions aimed at biofilms provide significant information for creating sustainable disease management approaches for BLB in rice (47). Collectively, these findings suggest that biofilm formation is not merely a structural adaptation but a dynamic strategy that integrates bacterial communication, protection and dispersal to sustain infection within the host vascular system.

Virulence factors and effector-mediated interactions

Xanthomonas oryzae pv. *oryzae* relies heavily on a wide range of factors that disrupt host cell processes to achieve its virulence. Transcription activator-like (TAL) effectors are particularly significant because these proteins translocate into rice cells. This direct

manipulation makes TAL effectors especially critical for Xoo's pathogenic success (49). This indicates that TAL effectors function not only as virulence determinants but also as precise regulators of host cellular processes, enabling the pathogen to reprogram host physiology in a highly targeted manner. The transport of these effectors relies on an operational type III secretion system, serving as a molecular syringe that allows Xoo to insert virulence proteins into host tissues during the infection process (50). Comparative studies have shown that the makeup of effector repertoires differs considerably across Xoo populations, indicating adaptation to various host genotypes (51). Genomic research additionally suggests that the development of virulence characteristics is influenced by selection pressures from the implementation of host resistance (15). Together, these elements highlight the dynamic characteristics of effector-driven pathogenicity in BLB. A key characteristic of TAL effectors is their capacity to attach to DNA sequences in the rice genome and promote the transcription of host susceptibility genes that aid in bacterial colonisation (49). Comprehensive molecular investigations have shown that Xoo utilises this mechanism to modify host transcriptional networks to facilitate disease progression (52). Specific TAL effectors aim at SWEET sugar transporter genes, thus boosting nutrient accessibility for bacterial growth in vascular tissues (30). The accurate identification of host promoters by TAL effectors accounts for the pronounced genotype-specific interactions noted between rice cultivars and Xoo strains (53). These results underscore the complexity of transcriptional manipulation techniques used by Xoo during infection. In addition to manipulating hosts, Xoo has developed strategies to mitigate plant immune responses by diversifying its effectors. Certain TAL effectors have demonstrated the ability to disrupt the recognition mediated by resistance genes, successfully inhibiting the activation of host defences (29). This type of interference leads to the breakdown of resistance in cultivars that were once deemed durable (54). Studies at the population level demonstrate a strong correlation between effector diversity and both pathogenic fitness and host adaptation in various geographic areas (51). Genomic data indicates that horizontal gene transfer and recombination processes enhance the development of new virulence traits (15). This evolutionary adaptability allows Xoo to swiftly react to shifting host resistance environments. The delivery and expression of effectors are closely regulated in sync with the infection process. Functional analyses of the type III secretion system reveal its essential role in facilitating effective host-pathogen interactions (50). Interference with this secretion system markedly diminishes bacterial virulence, validating its crucial role in disease progression (49). Transcriptomic analyses show that the expression of effector genes is dynamically controlled throughout various stages of infection to enhance manipulation of the host (53). Additionally, host-driven selection pressures aid in the fixation of highly efficient effector combinations within Xoo populations (51). These regulatory processes guarantee the accurate timing of virulence factor release during infection. Recent genomic studies have offered enhanced understanding of the long-term development of effector-driven virulence in Xoo. Comparative genome studies indicate that pandemic lineages have larger effector inventories linked to heightened aggressiveness (15). The preservation of crucial TAL effector functions among various strains highlights their vital importance in pathogenicity (49). Simultaneously, changes in effector content allow Xoo to bypass newly introduced resistance genes in rice breeding initiatives (54). Grasping these evolutionary processes is essential for forecasting

resistance longevity (30). As a result, resistance strategies informed by effectors are increasingly recognised as vital for the sustainable management of BLB. The body of evidence suggests that the coordinated activity of several effectors that operate in a time regulated way, rather than individual variables, is what drives virulence in Xoo. Some studies highlight the conservation of important effector activities across strains (49), while others stress effector variety as a strategy for overcoming host resistance (51). This seeming paradox implies that to sustain pathogenic success, Xoo strikes a balance between evolutionary novelty and functional stability. In the end, it is important to see virulence as a dynamic and adaptable system that is moulded by ongoing co-evolution between the host and the pathogen (11, 14) (Table 1).

Host defence responses (structural, biochemical and molecular)

Rice plants exhibit a proactive defence response upon invasion by *X. oryzae* pv. *oryzae*. They react fast, switching on several structural defences to block the pathogen from getting in and spreading. An initial defence mechanism involves the reinforcement of host cell walls. This happens through more lignification and callose piling up, which makes it harder for the bacteria to move through the plant's vascular tissues (50). The thickening of xylem vessel walls has been identified as a major obstacle to systemic colonisation in resistant genotypes (8). The buildup of phenolic compounds at infection locations also aids in mechanical fortification and antimicrobial protection (55). Resistant varieties respond more quickly and intensely than susceptible ones, as seen in how their structures change to slow lesion development (56). These early physical modifications create an initial barrier, helping to stop the disease from the outset. However, rice plants go further. They also activate complex biochemical defences that make survival difficult for pathogens. When Xoo infects the plant, rice cells promptly release a surge of ROS. These molecules not only attack the bacteria directly but also signal the rest of the plant to boost its defences (57). During this oxidative burst, antioxidant enzymes such as peroxidase, catalase and superoxide dismutase are produced in greater quantities to regulate ROS levels (58). Simultaneously, the plant increases the synthesis of defensive compounds like phenolics and phytoalexins, which help restrict bacterial growth (55). These biochemical responses are stronger in resistant plants, highlighting their role in disease control (56). Properly balancing ROS production and detoxification is critical-if either is out of balance, the plant's defence is compromised. At the molecular level, rice's battle against BLB involves significant changes in gene expression. Genome-wide studies indicate that after Xoo infection, rice activates a range of pathogenesis-related (PR) genes, enzymes involved in secondary metabolism and genes responsible for strengthening cell walls (59).

Receptor-mediated signalling pathways are triggered, activating defence-related transcription factors that orchestrate these responses. Together, these systems provide a rapid and effective defence (60). Distinct gene expression profiles between resistant and susceptible cultivars emphasise the significance of early and ongoing activation of defence genes (56). Molecular reactions also involve the inhibition of susceptibility-associated genes that otherwise aid in pathogen nourishment (61). All these changes in gene expression influence how the host and pathogen interact. Hormonal signals are crucial in helping plants organise their defences-structural, biochemical and molecular-against bacterial blight. Pathways driven by salicylic acid are central to resistance, particularly in restricting bacterial proliferation. However, salicylic acid does not act alone (60). It interacts with hormones such as jasmonic acid and ethylene, which modulate both the intensity and duration of defence responses. Hormones also affect the production of ROS and the plant's antioxidant mechanisms, integrating various signalling pathways with the plant's biochemical defences (57). Variations in hormone levels are directly linked to disease severity in different rice genotypes (56). All of this highlights the complexity and precision of plant defence regulation. In response to BLB, plants depend on a sophisticated and coordinated defence system-physical barriers to block the pathogen, biochemical mechanisms to inhibit its growth and gene regulation that swiftly activates resistance pathways (8). Effective resistance relies more on the velocity, power and cohesion of these reactions than on any one defensive element (59). Comprehending the interactions among these defence layers offers important knowledge for developing and engineering lasting resistance (60). Ongoing investigation of host defence systems is crucial for the sustainable control of BLB in rice. From an integrative perspective, rice defence against Xoo cannot be attributed to a single mechanism but rather to the coordinated activation of structural, biochemical and molecular responses. The effectiveness of resistance depends not only on the presence of these defences but on their timing, intensity and integration, which collectively determine whether pathogen spread is restricted or allowed to progress.

Symptom development and disease progression

Symptoms of BLB manifest rapidly. The disease starts with water-soaked spots at the leaf margins, clear signs that Xoo is entering through the hydathodes (62). As the bacteria establish themselves, these lesions extend along the veins, marking the bacteria's progression deeper into the xylem vessels (38). The initial manifestation of symptoms is closely linked to the density of the pathogen population and the physiological condition of the host (35). Susceptible varieties frequently exhibit swift lesion growth

Table 1. Major virulence factors of *Xanthomonas oryzae* pv. *oryzae* and their functional roles in host infection and disease progression

Virulence factor	Function	Effect on host	Contribution to disease	References
Extracellular enzymes	Cell wall degradation	Tissue maceration	Facilitates invasion	(6)
Effector diversity	Host adaptation	Overcomes resistance	Emergence of virulent strains	(15)
SWEET gene activation	Sugar efflux	Nutrient acquisition	Pathogen multiplication	(30)
Chemotaxis	Directed movement	Efficient host colonisation	Early infection success	(37)
Motility	Surface and vascular movement	Enhanced spread	Colonisation efficiency	(37, 39)
Xanthan gum (EPS)	Biofilm formation	Vascular blockage	Restricts water movement	(41)
Biofilms	Bacterial aggregation	Protection from host defences	Enhances survival	(41, 44)
Quorum sensing	Population coordination	Regulates virulence genes	Disease progression	(42, 48)
TAL effectors	Host gene activation	Induces susceptibility genes	Disease development	(49, 52)
Type III secretion system	Effector delivery	Immune suppression	Essential for virulence	(50)

because of constrained limitation on bacterial spread (12). In comparison, resistant genotypes show a slower appearance of symptoms, signifying successful control of the pathogen (36). As the infection progresses, lesions change from water-soaked streaks to yellowish and eventually necrotic blight symptoms resulting from tissue collapse and chlorophyll breakdown (39). When leaves gradually turn yellow, it indicates problems in photosynthesis and obstructed nutrient transport as described by previous studies (13). Bacterial exopolysaccharides accumulate in the xylem, blocking vessels and exacerbating symptoms (35). Infected tissues experience increased oxidative stress, which intensifies cell damage and accelerates lesion formation (58). When leaves gradually turn yellow, it indicates problems in photosynthesis and obstructed nutrient transport (63). Bacterial exopolysaccharides accumulate in the xylem, blocking vessels and exacerbating symptoms (35). Infected tissues experience increased oxidative stress, which intensifies cell damage and accelerates lesion formation (58). The severity of these symptoms fluctuates based on environmental factors and the vulnerability of the host (12). The progression of disease is intricately connected to the spatial and temporal patterns of bacterial colonisation. Elevated pathogen levels in the vascular system are linked to quick lesion growth and the emergence of systemic symptoms (36). Therefore, symptoms appear as a delayed reaction to bacteria that have already established themselves within the plant (35). The extent to which the bacteria penetrate the vascular tissue determines whether symptoms are confined to a single area or spread throughout the entire leaf blade (38). The host genotype significantly influences the restriction of bacterial dispersion and the intensity of symptoms (62). These findings emphasise the significance of internal infection mechanisms in influencing apparent disease results (12). Physiological changes in infected rice plants greatly affect the manifestation of symptoms. A decline in chlorophyll levels and photosynthetic efficiency is frequently seen in the later stages of disease (39). Metabolic reprogramming in infected tissues results in the redirection of resources toward protective mechanisms, frequently at the expense of growth and yield (63). Increased generation of ROS aids in both defence signalling and cellular harm (58). In vulnerable plants, unmanaged oxidative stress speeds up tissue decay and wilting (35). These biological alterations together influence the intensity and duration of disease symptoms (36). Environmental factors additionally influence symptom emergence and disease advancement in field situations. Elevated humidity and warm conditions promote quick lesion growth and intense blight symptoms (12). Water supply and nutrient status are also crucial factors. When crops receive sufficient water and nutrients, they are more resilient and display fewer symptoms and when these are lacking, the situation deteriorates (39). Environmental stress and pathogen pressure do not always act predictably. Their interaction often results in disease outcomes that surprise us. For accurate disease assessment and effective management, it is essential to understand how these factors interact (36). Collectively, these observations suggest that symptom development is a downstream consequence of successful vascular colonisation rather than an immediate response to infection. This highlights the importance of early-stage host-pathogen interactions in determining later disease severity and visible outcomes.

Resistance vs susceptibility outcomes in bacterial leaf blight of rice

The molecular result of interactions between host resistance genes

and pathogen virulence factors mainly dictates resistance or vulnerability to BLB in rice. Initial genetic research showed that resistant rice varieties limit bacterial growth in vascular tissues, while susceptible varieties allow considerable systemic colonisation (9). The *Xa* gene family serves as a significant source of resistance, operating through various mechanisms such as receptor mediated detection and transcriptional control (64). If you compare resistant and susceptible genotypes, you observe significant differences in the behaviour of bacterial populations and the development of lesions (31, 56). These differing results highlight the intricacy of expressing resistance in field settings (14). A key aspect that differentiates resistance from susceptibility is the capacity of the host to detect pathogen effectors and trigger robust immune responses. Resistant varieties possessing active *Xa* genes quickly trigger defence signalling pathways that restrict pathogen dissemination (9). In non-resistant plants, some strains of *X. oryzae* strains use TAL effectors to sidestep the plant's defences and drive disease forward (27). When TAL effectors interact with the host's transcription machinery, this exchange plays a key role in shaping how the disease unfolds (30). Functional analyses indicate that effector-triggered susceptibility frequently takes precedence over basal defence responses (65). Consequently, resistance or vulnerability indicates the equilibrium between host immune response and pathogen effector function (31). Host vulnerability is often associated with the utilisation of plant genes that support the survival and spread of pathogens. The SWEET sugar transporter genes serve as notable instances of susceptibility factors that are transcriptionally induced during compatible interactions (30). Transcription activator-like effector-driven activation of these genes boosts sugar transport into the xylem, creating a nutrient-abundant setting for bacterial proliferation (27). In vulnerable rice varieties, this mechanism results in rapid bacterial growth and significant symptom expression (36). Genome-wide research has uncovered more host genes linked to susceptibility, emphasising the polygenic character of disease results (65). These results highlight that susceptibility is a dynamic, genetically controlled mechanism instead of a mere breakdown of defence (14). Progress in functional genomics has allowed precise alteration of susceptibility genes to enhance the longevity of resistance. Genome editing techniques, especially those using Clustered Regularly Interspaced Short Palindromic Repeats/CRISPR-associated proteins (CRISPR/Cas) to modify SWEET gene promoters, have effectively diminished BLB severity while maintaining agronomic performance (66). These strategies show that resistance can be attained by interfering with pathogen exploitation methods instead of depending only on traditional resistance genes (31). Field assessments show that modified lines display consistent resistance in various environments (56). Combining genetic resistance with thorough monitoring of pathogen populations offers the best chance of sustainably managing BLB (15). A plant's ability to resist or fall victim to BLB depends on a dynamic interplay among host genetics, the pathogen's suite of effectors and environmental factors (14). Resistance genes are effective, but their durability hinges on the speed at which the pathogen adapts (3). When plants become susceptible, it highlights the need to understand which host factors facilitate disease progression (65). By integrating molecular resistance, wisely managing susceptibility genes and considering ecological aspects, we establish a comprehensive approach to disease control (31). Delving further into host-pathogen interactions will be essential for breeding rice cultivars that can reliably resist BLB (11). These insights provide a foundation for future resistance

breeding and precision disease management strategies (14). Resistance and susceptibility seem to occur along a continuum shaped by the balance between pathogen effector activity and host defence activation rather than being strictly antagonistic states. Although resistance genes can successfully stop the spread of pathogens, *Xoo* populations' ability to adapt limits how long they can last (15). On the other hand, susceptibility is frequently caused by pathogen effectors actively taking advantage of host processes rather than being a passive state. This viewpoint emphasises the necessity of switching from single-gene resistance tactics to integrated techniques that target both susceptibility and resistance pathways (Fig. 2).

Environmental modulation of host–pathogen interaction

Environmental factors profoundly influence the outcomes of rice-*X. oryzae* interaction, they affect everything from the pathogen's survival to the plant's response and the way the disease manifests in the field. Temperature is particularly important. As it increases, the bacteria reproduce more rapidly and the disease spreads faster through the crop (10). Relative humidity also plays a role. High humidity promotes the formation of guttation fluid at hydathodes, which serve as key infection points for the bacteria (12). Rainfall is another factor. It physically spreads bacteria across leaves and between plants, increasing the likelihood of major outbreaks, especially in large fields (67). The growth stage of the crop is significant as well. During the tillering to booting phases, rice plants are more severely affected and display more pronounced symptoms (10). All these factors interact, shifting the balance between the rice and the pathogen. Temperature not only accelerates bacterial growth but also influences the plant's defence mechanisms, thereby

altering the progression of the disease. Increased temperatures have been found to weaken resistance expression in specific rice genotypes, resulting in greater vulnerability (39). In contrast, moderate temperatures can boost basal defence responses, such as oxidative burst and the activity of defence-related enzymes (68). Disease severity shifts depending on the environment, thanks in part to the way temperature changes influence bacterial virulence gene expression. All of this points to a tangled mix of thermal factors, host defences and pathogen virulence driving disease outcomes (10, 12). Moisture is a critical factor in the epidemiology of bacterial leaf blight. High humidity keeps leaf surfaces wet longer, letting bacteria survive and infect more easily (67). When fields stay soaked, bacteria move through the plant's vascular tissues more freely, causing larger lesions and cutting into yields (39). On the flip side, dry spells can slow down bacterial growth, but they also stress the plants, which weakens their defences (68). Dew that forms in the early morning, especially in tropical regions often signals a spike in new disease cases. So, moisture levels directly shape both how well the pathogen thrives and how vulnerable the plants become (61). Climatic shifts, both seasonal and geographic, lead to big differences in how common and severe diseases get. Long-term studies show warm, humid areas tend to see more cases of BLB (67). As climate change brings more extreme precipitation and temperature swings, the pathogen's range is set to expand (12). Seasonal changes in environmental factors similarly impact pathogen population behaviours and the stability of their virulence (10). Understanding local conditions is important if we want to anticipate disease outbreaks. The environment and agricultural practices interact, shaping the relationships between hosts and pathogens (39). Environmental elements also combine with agricultural methods to

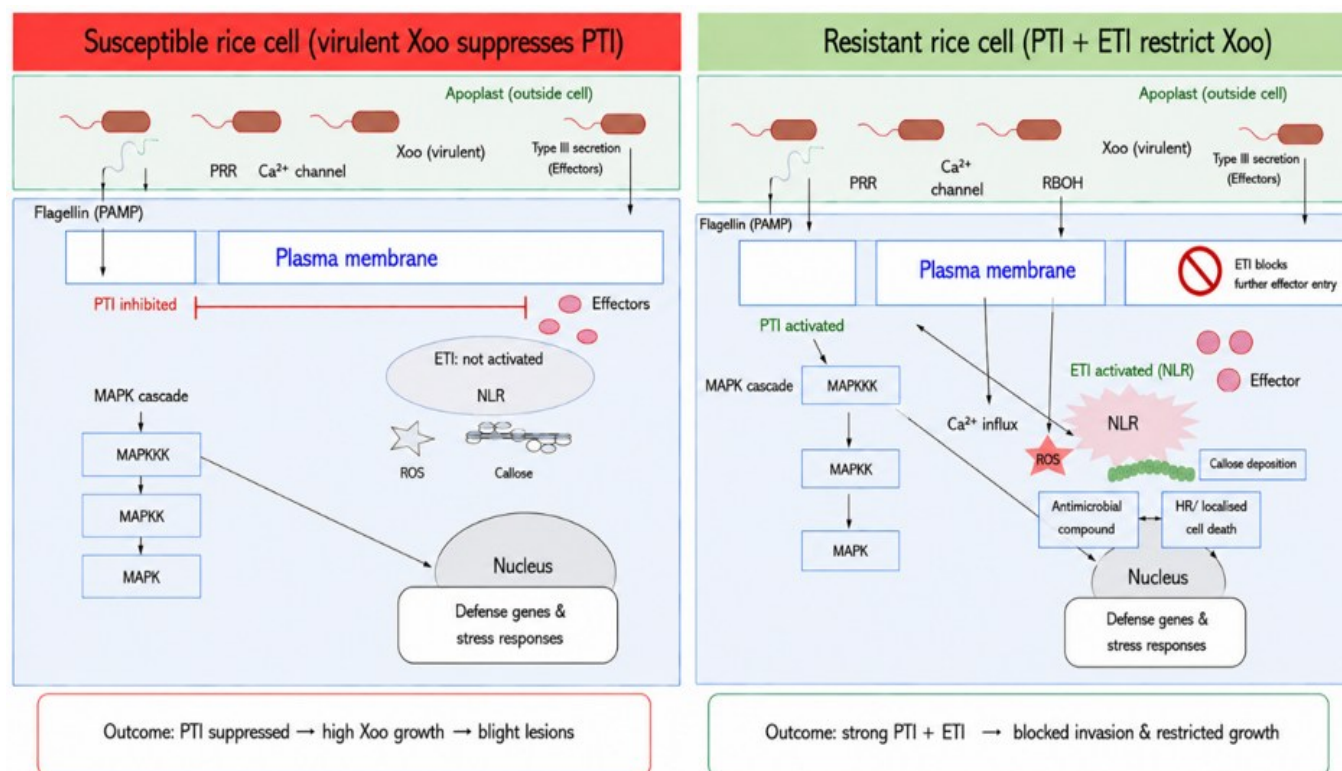


Fig. 2. Conceptual model of pattern-triggered immunity (PTI) and effector-triggered immunity (ETI) signalling pathways in susceptible and resistant rice cells during *Xanthomonas oryzae* pv. *oryzae* infection.

Xoo: *Xanthomonas oryzae* pv. *oryzae*; PTI: pattern-triggered immunity; ETI: effector-triggered immunity; PRR: pattern recognition receptor; PAMP: pathogen-associated molecular pattern; ROS: reactive oxygen species; NLR: nucleotide-binding leucine-rich repeat receptor; RBOH: respiratory burst oxidase homolog; MAPK: mitogen-activated protein kinase; MAPKK: mitogen-activated protein kinase kinase; MAPKKK: mitogen-activated protein kinase kinase kinase; HR: hypersensitive response; Ca²⁺: calcium ion.

influence host–pathogen relationships. When farmers plant crops too closely together and use excessive nitrogen, they create humid spaces where diseases can flourish. How they irrigate the crops also plays a role, especially with rice, since flooding can help pathogens spread and persist (67). Stressful situations caused by less than ideal environments can diminish host defence effectiveness, heightening susceptibility to infection (68). Incorporating environmental risk evaluation into disease prediction models has been suggested as a successful approach for controlling BLB (61). In general, environmental modulation continues to be a crucial factor in disease dynamics and the effectiveness of management strategies. Environmental elements influence illness outcomes through interactions with host and pathogen components rather than acting independently. While ideal circumstances promote the growth and spread of pathogens, they can also alter host defensive mechanisms, resulting in inconsistent disease manifestation. This suggests that rather than fixed biological pathways, BLB should be seen as a context-dependent disease system impacted by dynamic environmental interactions.

Conclusion

Rice bacterial leaf blight (BLB) stands out as one of the most intensely studied plant-bacterial pathosystems. Its economic impact keeps researchers coming back and the complex interaction between the rice and its pathogen remains as a subject of intensive study. Scientists have shown that *Xanthomonas oryzae* pv. *oryzae* is not just any vascular pathogen but it skillfully hijacks the plant's normal functions, setting off disease with impressive precision. The variety of pathogen populations and their swift evolutionary changes persist in posing challenges to effective long-term disease management strategies. Progress in molecular biology has greatly enhanced comprehension of infection, colonisation and symptom manifestation. However, on the ground disease management it continues to be uneven across agroecological regions. These findings emphasise the importance of combining molecular insights with effective resistance implementation methods. Research on host resistance has shown that the resistance to BLB is controlled by a complex interaction of dominant resistance genes and factors that contribute to susceptibility. The cloning and functional analysis of Xa resistance genes have elucidated how host recognition limits pathogen proliferation, although resistance breakdown continues to be a persistent problem. Studies on susceptibility genes, especially those impacted by transcription activator-like effectors, have transformed the comprehension of effector-triggered susceptibility in rice. The functional diversification of resistance genes underscores the significance of adopting pyramiding strategies instead of depending on single-gene resistance. Nonetheless, the durability of resistance is significantly affected by the structure of pathogen populations and the selection pressure exerted by host genotypes. Recent genomic and population-level research has yielded important information regarding the evolutionary processes that influence virulence variation in Xoo. Comparative genomics has uncovered lineage-specific adaptations that lead to regional disease outbreaks. The swift development of TAL effector collections makes resistance breeding initiatives more challenging by allowing pathogens to evade host immune responses. Sustained disease management necessitates ongoing observation of pathogen populations and changes in virulence. The integration of genomic monitoring with breeding programs facilitates a practical way to

spot resistance decline early, before major field losses hit. To move forward, researchers need to dig deeper into how hosts and pathogens interact at every level, from molecules to entire environments. As climate change shifts disease patterns, we need to rethink how the environment shapes both host defences and pathogen aggression. Turning breakthroughs from the lab into tough, field-ready cultivars is not easy but it takes real collaboration across disciplines. But when we combine these approaches, we get better at predicting and managing resistance in the unpredictable world with changing climatic circumstances. To summarise, effective management of BLB requires integrating molecular resistance, monitoring pathogen populations and adapting agroecologically. Progress in comprehending TAL effector biology and the susceptibility of hosts offers a robust basis for creating long-lasting resistance strategies. We need to continue investing in genomics-driven breeding and applying resistance genes to reduce yield losses from this disease. Strengthening the connection between fundamental research and practical plant pathology accelerates progress toward more resilient rice essential for securing sustainable food production in rice-cultivating areas globally.

Acknowledgements

The authors express gratitude to V.O. Chidambaranar Agricultural College and Research Institute, Tamil Nadu Agricultural University, Killikulam for the support provided.

Authors' contributions

SMS wrote the manuscript. RN contributed core ideas for the article. TM, JPB, KPA, SJ and VM carried out the corrections. All authors read and approved the final manuscript.

Compliance with ethical standards

Conflict of interest: Authors do not have any conflict of interests to declare.

Ethical issues: None

References

1. Niño-Liu DO, Ronald PC, Bogdanove AJ. *Xanthomonas oryzae* pathovars: Model pathogens of a model crop. *Mol Plant Pathol*. 2006;7(5):303–24. <https://doi.org/10.1111/j.1364-3703.2006.00344.x>
2. Sundaram RM. Update on bacterial blight of rice: Fourth international conference on bacterial blight. *Rice*. 2014;7(1):12. <https://doi.org/10.1186/s12284-014-0012-7>
3. Verdier V, Cruz CV, Leach JE. Controlling rice bacterial blight in Africa: Needs and prospects. *J Biotechnol*. 2012;159(4):320–8. <https://doi.org/10.1016/j.jbiotec.2011.09.020>
4. Midha S. Population genomic insights into variation and evolution of *Xanthomonas oryzae* pv. *oryzae*. *Sci Rep*. 2017;7(1):40694. <https://doi.org/10.1038/srep40694>
5. Ritbamrung O. Evaluating *Xanthomonas oryzae* pv. *oryzae* infection dynamics in rice for distribution routes and environmental reservoirs by molecular approaches. *Sci Rep*. 2025;15(1):1408. <https://doi.org/10.1038/s41598-025-85422-3>
6. Chatterjee S, Sonti RV. *prfF* mutants of *Xanthomonas oryzae* pv. *oryzae* are deficient for virulence and growth under low iron conditions. *Mol Plant Microbe Interact*. 2002;15(5):463–71. <https://doi.org/10.1094/MPMI.2002.15.5.463>

7. Sharma N, Kaur C, Singh J, Gupta AK. Biochemical responses associated with bacterial blight of rice (*Oryza sativa*). Arch Phytopathol Plant Prot. 2012;45(1):47–61. <https://doi.org/10.1080/03235400903395336>
8. Zhang H, Wang S. Rice versus *Xanthomonas oryzae* pv. *oryzae*: A unique pathosystem. Curr Opin Plant Biol. 2013;16(2):188–95. <https://doi.org/10.1016/j.pbi.2013.02.008>
9. Sun X. Xa26, a gene conferring resistance to *Xanthomonas oryzae* pv. *oryzae* in rice, encodes an LRR receptor kinase-like protein. Plant J. 2004;37(4):517–27. <https://doi.org/10.1046/j.1365-3113X.2003.01976.x>
10. Echeverri-Rico J. Understanding the complexity of disease-climate interactions for rice bacterial panicle blight under tropical conditions. PLoS One. 2021;16(5):e0252061. <https://doi.org/10.1371/journal.pone.0252061>
11. Qi Y, Rao Q, Lu C, Gong J, Hou Y. Recent progress in rice-*Xanthomonas oryzae* interactions. Biology. 2025;14(5):471. <https://doi.org/10.3390/biology14050471>
12. Aftab U. Epidemiological studies of bacterial leaf blight of rice and its management. Basrah J Agric Sci. 2022;35(1):106–19. <https://doi.org/10.37077/25200860.2022.35.1.09>
13. Kumar A. Deployment of genetic and genomic tools toward gaining a better understanding of rice-*Xanthomonas oryzae* pv. *oryzae* interactions for development of durable bacterial blight resistant rice. Front Plant Sci. 2020;11:1152. <https://doi.org/10.3389/fpls.2020.01152>
14. Timilsina S. *Xanthomonas* diversity, virulence and plant-pathogen interactions. Nat Rev Microbiol. 2020;18(8):415–27. <https://doi.org/10.1038/s41579-020-0361-8>
15. Agarwal V, Stubits R, Nassrullah Z, Dillon MM. Pangenome insights into the diversification and disease specificity of worldwide *Xanthomonas* outbreaks. Front Microbiol. 2023;14:1213261. <https://doi.org/10.3389/fmicb.2023.1213261>
16. Luneau JS. *Xanthomonas* transcriptome inside cauliflower hydathodes reveals bacterial virulence strategies and physiological adaptations at early infection stages. Mol Plant Pathol. 2022;23(2):159–74. <https://doi.org/10.1111/mpp.13117>
17. Paauw M. Hydathode immunity protects the *Arabidopsis* leaf vasculature against colonization by bacterial pathogens. Curr Biol. 2023;33(4):697–710. <https://doi.org/10.1016/j.cub.2023.01.013>
18. Pradhan SK. Genetics, molecular mechanisms and deployment of bacterial blight resistance genes in rice. Crit Rev Plant Sci. 2020;39(4):360–85. <https://doi.org/10.1080/07352689.2020.1801559>
19. Redzich L. *Xanthomonas oryzae* pv. *oryzae* differentiation *in vitro* and during rice leaf infection. 2025.
20. Yang F. Bacterial blight induced shifts in endophytic microbiome of rice leaves and the enrichment of specific bacterial strains with pathogen antagonism. Front Plant Sci. 2020;11:963. <https://doi.org/10.3389/fpls.2020.00963>
21. Song Z. Population genomics and pathotypic evaluation of the bacterial leaf blight pathogen of rice reveals rapid evolutionary dynamics of a plant pathogen. Front Cell Infect Microbiol. 2023;13:1183416. <https://doi.org/10.3389/fcimb.2023.1183416>
22. Mücke S. Transcriptional reprogramming of rice cells by *Xanthomonas oryzae* TALEs. Front Plant Sci. 2019;10:162. <https://doi.org/10.3389/fpls.2019.00162>
23. Char SN, Park S, Yang B. Interaction of rice and *Xanthomonas* TAL effectors. In: Rice genomics, genetics and breeding. 2018; p. 375–91. https://doi.org/10.1007/978-981-10-7461-5_19
24. Xu X. TALE-induced immunity against the bacterial blight pathogen *Xanthomonas oryzae* pv. *oryzae* in rice. Phytopathol Res. 2022;4(1):47. <https://doi.org/10.1186/s42483-022-00153-x>
25. Kanipriya R. Screening for disease resistance and profiling the expression of defense-related genes contributing to resistance against bacterial blight (*Xanthomonas oryzae* pv. *oryzae*) in rice genotypes. Physiol Mol Plant Pathol. 2024;131:102286. <https://doi.org/10.1016/j.pmpp.2024.102286>
26. Yang DL, Yang Y, He Z. Roles of plant hormones and their interplay in rice immunity. Mol Plant. 2013;6(3):675–85. <https://doi.org/10.1093/mp/sst056>
27. Younas MU. Molecular insights into rice immunity: Unveiling mechanisms and innovative approaches to combat major pathogens. Plants. 2025;14(11):1694. <https://doi.org/10.3390/plants14111694>
28. Cao J. Different cell wall-degradation ability leads to tissue-specificity between *Xanthomonas oryzae* pv. *oryzae* and *Xanthomonas oryzae* pv. *oryzicola*. Pathogens. 2020;9(3):187. <https://doi.org/10.3390/pathogens9030187>
29. Li T, Huang S, Zhou J, Yang B. Designer TAL effectors induce disease susceptibility and resistance to *Xanthomonas oryzae* pv. *oryzae* in rice. Mol Plant. 2013;6(3):781–9. <https://doi.org/10.1093/mp/sst034>
30. Gupta PK, Balyan HS, Gautam T. SWEET genes and TAL effectors for disease resistance in plants: Present status and future prospects. Mol Plant Pathol. 2021;22(8):1014–26. <https://doi.org/10.1111/mpp.13075>
31. Jiang N. Resistance genes and their interactions with bacterial blight/leaf streak pathogens (*Xanthomonas oryzae*) in rice (*Oryza sativa* L.)-an updated review. Rice. 2020;13(1):3. <https://doi.org/10.1186/s12284-019-0358-y>
32. Nguyen MH. Host-induced adaptation of *Xanthomonas oryzae* pv. *oryzae*. 2020.
33. Ji Z, Wang C, Zhao K. Rice routes of countering *Xanthomonas oryzae*. Int J Mol Sci. 2018;19(10):3008. <https://doi.org/10.3390/ijms19103008>
34. An SQ. Mechanistic insights into host adaptation, virulence and epidemiology of the phytopathogen *Xanthomonas*. FEMS Microbiol Rev. 2020;44(1):1–32. <https://doi.org/10.1093/femsre/fuz024>
35. Niones JT. Dynamics of bacterial blight disease in resistant and susceptible rice varieties. Eur J Plant Pathol. 2022;163(1):1–7. <https://doi.org/10.1007/s10658-021-02452-z>
36. Sarker S, Rahman MA, Anwar MP, Hossain MA. Varietal mixture effect on the disease incidence and severity of bacterial blight in rice. J Bangladesh Agric Univ. 2020;18(2):354–61. <https://doi.org/10.5455/JBAU.76259>
37. Kumar Verma R, Samal B, Chatterjee S. *Xanthomonas oryzae* pv. *oryzae* chemotaxis components and chemoreceptor Mcp2 are involved in sensing xylem sap constituents and contribute to virulence regulation. Mol Plant Pathol. 2018;19(11):2397–415. <https://doi.org/10.1111/mpp.12718>
38. Pradhan SK. Characterization of morpho-quality traits and validation of bacterial blight resistance in pyramided rice genotypes under various hotspots of India. Aust J Crop Sci. 2015;9(2):127–34.
39. Kaur A, Bhatia D, Bhardwaj SC. Advances in the *Xanthomonas oryzae* pv. *oryzae*-rice pathosystem interaction and its pathogenicity. J Biosci. 2020;45:85.
40. Joshi JB, Arul L, Ramalingam J, Uthandi S. Advances in the Xoo-rice pathosystem interaction and its exploitation in disease management. J Biosci. 2020;45(1). <https://doi.org/10.1007/s12038-020-00085-8>
41. Mina IR, Jara NP, Criollo JE, Castillo JA. The critical role of biofilms in bacterial vascular plant pathogenesis. Plant Pathol. 2019;68(8):1439–47. <https://doi.org/10.1111/ppa.13073>
42. Feng YM. Research on diffusible signal factor-mediated quorum sensing in *Xanthomonas*: A mini-review. Molecules. 2023;28(2):876. <https://doi.org/10.3390/molecules28020876>
43. Pandey SS, Chatterjee S. Insights into cell-to-cell signaling and iron homeostasis in *Xanthomonas* virulence and lifestyle. Phytopathology. 2022;112(2):209–18. <https://doi.org/10.1094/PHYTO-11-20-0513-RWW>

44. Carezzano ME. Biofilm-forming ability of phytopathogenic bacteria: A review of its involvement in plant stress. *Plants*. 2023;12(11):2207. <https://doi.org/10.3390/plants12112207>
45. Syaida AA, Raja Yahya MF. Biofilm formation by phytopathogens: A threat to crop health and agricultural sustainability. *Preprints*. 2025. <https://doi.org/10.20944/preprints202512.0700.v1>
46. Wintersteller P, Huber A, Koller M. A critical review of the microbial exopolysaccharide xanthan. *Chem Biochem Eng Q*. 2025;39(2):65–87. <https://doi.org/10.15255/CABEQ.2024.2348>
47. Sanya DR. A review of approaches to control bacterial leaf blight in rice. *World J Microbiol Biotechnol*. 2022;38(7):113. <https://doi.org/10.1007/s11274-022-03298-1>
48. Lami R, Urios L, Molmeret M, Grimaud R. Quorum sensing in biofilms: A key mechanism to target in ecotoxicological studies. *Crit Rev Microbiol*. 2023;49(6):786–804. <https://doi.org/10.1080/1040841X.2022.2142089>
49. Shamsbakhsh M, Falahi Charkhabi N, Rahimian H. Transcription activator-like effectors (TALEs) role in plant response to *Xanthomonas*. *Agric Biotechnol J*. 2018;10(3):87–110.
50. Schreiber KJ, Chau-Ly IJ, Lewis JD. What the wild things do: Mechanisms of plant host manipulation by bacterial type III-secreted effector proteins. *Microorganisms*. 2021;9(5):1029. <https://doi.org/10.3390/microorganisms9051029>
51. Jacques MA. Using ecology, physiology and genomics to understand host specificity in *Xanthomonas*. *Annu Rev Phytopathol*. 2016;54:163–87. <https://doi.org/10.1146/annurev-phyto-080615-100147>
52. Perez-Quintero AL, Szurek B. A decade decoded: Spies and hackers in the history of TAL effectors research. *Annu Rev Phytopathol*. 2019;57:459–81. <https://doi.org/10.1146/annurev-phyto-082718-100026>
53. Hutin M, Pérez-Quintero AL, Lopez C, Szurek B. MorTAL KomBAT: The story of defense against TAL effectors through loss-of-susceptibility. *Front Plant Sci*. 2015;6:535. <https://doi.org/10.3389/fpls.2015.00535>
54. Nowack MK, Holmes DR, Lahaye T. TALE-induced cell death executors: An origin outside immunity? *Trends Plant Sci*. 2022;27(6):536–48. <https://doi.org/10.1016/j.tplants.2021.11.003>
55. Vemanna RS. Cross-talk signaling in rice during combined drought and bacterial blight stress. *Front Plant Sci*. 2019;10:193. <https://doi.org/10.3389/fpls.2019.00193>
56. Gorshkov V, Tsers I. Plant susceptible responses: The underestimated side of plant-pathogen interactions. *Biol Rev*. 2022;97(1):45–66. <https://doi.org/10.1111/brev.12789>
57. Cardoso JL, Souza AA, Vieira ML. Molecular basis for host responses to *Xanthomonas* infection. *Planta*. 2022;256(4):84. <https://doi.org/10.1007/s00425-022-03994-0>
58. Pandey VP, Awasthi M, Singh S, Tiwari S, Dwivedi UN. A comprehensive review on function and application of plant peroxidases. *Biochem Anal Biochem*. 2017;6(1):308. <https://doi.org/10.4172/2161-1009.1000308>
59. Kumar J, Ramlal A, Kumar K, Rani A, Mishra V. Signaling pathways and downstream effectors of host innate immunity in plants. *Int J Mol Sci*. 2021;22(16):9022. <https://doi.org/10.3390/ijms22169022>
60. Yu X, Feng B, He P, Shan L. From chaos to harmony: Responses and signaling upon microbial pattern recognition. *Annu Rev Phytopathol*. 2017;55:109–37. <https://doi.org/10.1146/annurev-phyto-080516-035649>
61. White FF, Yang B. Host and pathogen factors controlling the rice-*Xanthomonas oryzae* interaction. *Plant Physiol*. 2009;150(4):1677–86. <https://doi.org/10.1104/pp.109.139360>
62. Shen Y, Ronald P. Molecular determinants of disease and resistance in interactions of *Xanthomonas oryzae* pv. *oryzae* and rice. *Microbes Infect*. 2002;4(13):1361–7. [https://doi.org/10.1016/S1286-4579\(02\)00004-7](https://doi.org/10.1016/S1286-4579(02)00004-7)
63. Shekhar S, Sinha D, Kumari A. An overview of bacterial leaf blight disease of rice and different strategies for its management. *Int J Curr Microbiol App Sci*. 2020;9(4):2250–65. <https://doi.org/10.20546/ijcmas.2020.904.270>
64. Yang Y. Research progress on cloning and function of Xa genes against rice bacterial blight. *Front Plant Sci*. 2022;13:847199. <https://doi.org/10.3389/fpls.2022.847199>
65. Huang Z. Exploiting susceptibility genes in rice: From molecular mechanism to application. *Phytopathol Res*. 2024;6:1–5. <https://doi.org/10.1186/s42483-024-00289-y>
66. Kayastha S, Behera A, Veda DJ, Rayanoothala PS. Precision genome editing for enhanced resistance against bacterial blight in rice: A comprehensive approach. *Cereal Res Commun*. 2025;53(3):1159–70. <https://doi.org/10.1007/s42976-024-00610-7>
67. Naqvi SA. Bacterial leaf blight of rice: An overview of epidemiology and management with special reference to Indian sub-continent. *Pak J Agric Res*. 2019;32(2):359–80. <https://doi.org/10.17582/journal.pjar/2019/32.2.359.380>
68. Yan J, Fang Y, Xue D. Advances in the genetic basis and molecular mechanism of lesion mimic formation in rice. *Plants*. 2022;11(16):2169. <https://doi.org/10.3390/plants11162169>

Additional information

Peer review: Publisher thanks Sectional Editor and the other anonymous reviewers for their contribution to the peer review of this work.

Reprints & permissions information is available at https://horizonpublishing.com/journals/index.php/PST/open_access_policy

Publisher's Note: Horizon e-Publishing Group remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

Indexing: Plant Science Today, published by Horizon e-Publishing Group, is covered by Scopus, Web of Science, BIOSIS Previews, Clarivate Analytics, NAAS, UGC Care, etc. See https://horizonpublishing.com/journals/index.php/PST/indexing_abstracting

Copyright: © The Author(s). This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution and reproduction in any medium, provided the original author and source are credited (<https://creativecommons.org/licenses/by/4.0/>)

Publishing information: Plant Science Today is published by HORIZON e-Publishing Group with support from Empirion Publishers Private Limited, Thiruvananthapuram, India.