

Journal of Plant Physiology & Pathology

Opinion Article

A SCITECHNOL JOURNAL

Host Plant Resistance Mechanisms Against Citrus Canker Pathogen

Asma Saleh*

Department of Agriculture, Alexandria University, Alexandria City, Egypt 'Corresponding Author: Asma Saleh, Department of Agriculture, Alexandria University, Alexandria City, Egypt; E-mail: asma.saleh@gmail.com Received date: 23 February, 2024, Manuscript No. JPPP-24-131777; Editor assigned date: 26 February, 2024, Pre QC No. JPPP-24-131777 (PQ); Reviewed date: 12 March, 2024, QC No. JPPP-24-131777; Revised date: 20 March, 2024, Manuscript No. JPPP-24-131777 (R); Published date: 28 March, 2024, DOI: 10.4172/2329-955X.1000332

Description

Xanthomonas citri subsp. citri (Xcc) is the bacteria that causes citrus canker, which is a serious threat to citrus output globally. Scholars have been investigating several approaches to strengthen citrus's resilience against the infection in reaction to this terrible illness. *Citrus* sps. must be protected against citrus canker by use of host plant resistance mechanisms. Gaining an understanding of these mechanisms is essential to creating tactics that effectively lessen the disease's effects and guarantee the sustainability of citrus output in the long run.

Citrus sps. employ physical barriers to prevent the entry and spread of citrus canker pathogens. The cuticle, a waxy layer covering the surface of *citrus* sps. leaves and fruits, acts as the first line of defense by providing a waterproof barrier and hindering microbial penetration. Additionally, cell walls act as structural barriers, providing rigidity and strength to plant tissues. Thickening of the cuticle and reinforcement of cell walls through genetic manipulation or breeding programs can enhance citrus resistance to citrus canker. *Citrus* sps. produce a diverse array of secondary metabolites with antimicrobial properties, including flavonoids, terpenoids, and phenolic. These chemical compounds play an essential role in plant defense against pathogens by inhibiting pathogen growth and limiting disease development.

Manipulating the expression of genes involved in secondary metabolite biosynthesis pathways can enhance citrus canker resistance by increasing the production of antimicrobial compounds. Upon pathogen attack, *citrus* sps. activate defense mechanisms to reinforce cell walls, limiting pathogen invasion and colonization. The deposition of lignin, cellulose, and other structural components strengthens cell walls, impeding the spread of citrus canker pathogens. Activation of genes involved in cell wall synthesis and modification pathways can enhance citrus resistance to citrus canker by promoting cell wall reinforcement. *Citrus* sps. possess innate immune systems that recognize Pathogen-Associated Molecular Patterns (PAMPs) and

activate defense responses to combat citrus canker pathogens. Pattern Recognition Receptors (PRRs) located on the plant cell surface recognize conserved microbial molecules, triggering downstream signaling overflow that lead to the activation of defense genes.

Enhancing the expression of PRRs and downstream signaling components can enhance citrus resistance to citrus canker by improving the plant's ability to detect and respond to pathogen attack. Induced systemic resistance (ISR) is a plant defense mechanism in which local pathogen infection triggers systemic immune responses throughout the plant. Upon recognition of pathogen infection, citrus sps. produce signaling molecules such as Salicylic Acid (SA), Jasmonic Acid (JA), and Ethylene (ET), which activate defense pathways in distant tissues. This systemic immune response primes the plant for enhanced defense against subsequent pathogen attacks. Enhancing ISR through the application of beneficial microorganisms or defense-inducing compounds can boost citrus resistance to citrus canker. Plant hormones play an essential role in regulating plant immune responses to citrus canker pathogens. SA-mediated signaling pathways are associated with defense against bio trophic pathogens, while JA and ET pathways are involved in defense against necrotrophic pathogens.

Balancing hormonal signaling pathways and modulating hormone levels can enhance citrus resistance to citrus canker by promoting effective defense responses. Genetic resistance is one of the most effective and sustainable approaches for enhancing citrus resistance to citrus canker. Identifying and accessing resistance genes from wild citrus relatives into commercial cultivars through traditional breeding or genetic engineering can confer durable resistance to citrus canker. Resistance genes may encode proteins involved in pathogen recognition, signal transduction, or defense molecule synthesis, providing multiple layers of defense against citrus canker pathogens. Beneficial microorganisms such as endophytes, rhizobacteria, and mycorrhizae can enhance citrus resistance to citrus canker by promoting plant growth and inducing systemic resistance. These microorganisms colonize plant roots or aerial tissues, competing with pathogens for space and nutrients and activating defense responses in the host plant.

Exploiting the potential of beneficial microorganisms through inoculation or bio fertilization can improve citrus health and strength to citrus canker. Understanding and utilizing host plant resistance mechanisms are essential for developing effective strategies to enhance citrus resistance to citrus canker. By manipulating physical, chemical, and molecular defense mechanisms, researchers and growers can develop citrus cultivars with improved resistance to the disease. Incorporating genetic resistance, induced systemic resistance, and biological control into citrus breeding and management programs can help mitigate the impact of citrus canker and ensure the long-term sustainability of citrus production. Continued research and innovation in this field are vital for addressing the ongoing challenges posed by citrus canker and other citrus diseases.

Citation: Saleh A (2024) Host Plant Resistance Mechanisms Against Citrus Canker Pathogen. J Plant Physiol Pathol 12:2.



All articles published in Journal of Plant Physiology & Pathology are the property of SciTechnol and is protected by copyright laws. Copyright © 2024, SciTechnol, All Rights Reserved.