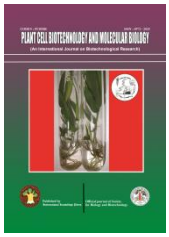

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A Review on Plant-microbe Interactions and its Defence Mechanism

Sarmistha Sahoo [a++], Saswati Sibani Panda [b†], Sanjay Kumar [c*], Rashmi Nigam [d++], Satyabrata Sarangi [e#], Mahima Choudhary [c#], R. Yuvarani [f++] and Shivam [g†]

[a] Department of Plant Pathology, School of Agriculture Giet University, Gunupur, Rayagada, Odisha. Pin – 765022, India.
[b] Department of Plant Pathology, Siksha 'O' Anusandhan University, Bhubaneswar, Pin – 751003, India.
[c] Department of Plant Pathology, Rajmata Vijayaraje Scindia Krishi Vishwavidyalaya, Gwalior, Madhya Pradesh - 474002, India.
[d] Department of Plant Pathology, J.V. College Baraut (Baghpat), C.C.S. University Meerut Uttar Pradesh, 250611, India.
[e] Department of Entomology, College of Agriculture, Odisha University of Agriculture and Technology, Bhubaneswar, 751003, India.
[f] Department of Plant Pathology, Adhiparasakthi Agricultural College, Kalavai, Ranipet, Tamil Nadu, 632506, India.
[g] Department of Plant Pathology, Chandra Shekhar Azad University of Agriculture and Technology, Kanpur, 208002, Uttar Pradesh, India.

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[++] Assistant Professor;
[#] Ph.D. Scholar;
[†] M.Sc.;
**Corresponding author: E-mail: sparihar734.sp@gmail.com;*

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Review Article

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ABSTRACT

Plant-microbe interactions are fundamental to plant health, growth, and defense, influencing agricultural productivity and ecosystem dynamics. These interactions range from symbiotic relationships, such as those with mycorrhizal fungi and nitrogen-fixing bacteria, to pathogenic encounters that trigger complex plant immune responses. Beneficial microbes, including rhizobacteria, mycorrhizae, and endophytes, play a crucial role in enhancing plant defenses through mechanisms like induced systemic resistance (ISR) and production of antimicrobial compounds. Advances in our understanding of these interactions have enabled the development of innovative strategies for crop protection, such as the use of biocontrol agents and microbial inoculants. Additionally, plant breeding and genetic engineering have been employed to introduce resistance genes and modify plant immune responses, resulting in disease-resistant varieties. However, harnessing plant-microbe interactions for sustainable agriculture faces challenges due to the complexity of these interactions in natural environments and the influence of abiotic factors. Limitations in research methodologies, such as difficulties in isolating and studying unculturable microbes, further complicate the translation of findings into practical applications. To overcome these barriers, future research should focus on integrating multi-omics approaches, employing synthetic microbial communities (SynComs), and leveraging CRISPR/Cas technologies for precise manipulation of plant and microbial genes. Microbiome engineering holds promise for promoting beneficial microbial communities, improving plant resilience, and reducing chemical inputs in agriculture. Addressing these challenges will be critical for realizing the full potential of plant-microbe interactions, ultimately contributing to sustainable crop production, improved food security, and ecosystem health. This review highlights current advances, applications, and future directions in the study of plant-microbe interactions, emphasizing their significance for modern agriculture.

Keywords: Biocontrol; microbiome engineering; disease resistance; rhizosphere; mycorrhizae.

1. INTRODUCTION

1.1 Plant-microbe Interactions

Plant-microbe interactions represent a complex and dynamic relationship that can significantly influence plant health, development, and productivity. These interactions can be broadly categorized into beneficial, neutral, and harmful, depending on the type of microorganism involved [1]. Beneficial microbes, such as symbiotic mycorrhizal fungi and nitrogen-fixing bacteria, play a crucial role in plant growth by enhancing nutrient uptake and providing resistance against biotic and abiotic stresses. In contrast, pathogenic microbes such as fungi, bacteria, and viruses cause a range of plant diseases, triggering intricate defense responses in host plants. Plant-microbe interactions are primarily governed by the molecular communication between the host plant and the invading or symbiotic microbe. These interactions can occur above and below the soil surface, encompassing various ecological niches, including the rhizosphere, phyllosphere, and endosphere. Recent advances in molecular biology and 'omics' technologies have provided insights into the complexity of these interactions and how they shape plant immunity and fitness [2].

2. IMPORTANCE OF PLANT DEFENSE MECHANISMS

Plant defense mechanisms are pivotal in determining the outcome of plant-microbe interactions. Plants possess an intricate immune system comprising both pre-formed barriers and inducible responses that are activated upon recognition of pathogens. The plant immune system operates through a two-tiered mechanism: pattern-triggered immunity (PTI) and effector-triggered immunity (ETI). PTI is initiated by the recognition of conserved microbial molecules, known as pathogen-associated molecular patterns (PAMPs), by pattern recognition receptors (PRRs) on the plant cell

surface [3]. In contrast, ETI is a more robust response activated by the detection of pathogen-derived effector proteins inside the plant cell, typically mediated by resistance (R) proteins. Studying these defense mechanisms is crucial for several reasons. Firstly, it provides insights into the evolutionary arms race between plants and pathogens, where pathogens continually evolve strategies to suppress or evade plant immunity, and plants, in turn, evolve new resistance mechanisms [4].

3. PURPOSE AND OBJECTIVES OF THE REVIEW

The purpose of this review is to provide a overview of plant-microbe interactions, with a specific focus on how these interactions function as a defense mechanism. Given the complexity of these interactions and the diversity of microbes involved, the review will cover both beneficial and pathogenic relationships, highlighting the molecular, biochemical, and ecological aspects that govern these interactions. The review aims to integrate current knowledge on the mechanisms by which plants detect and respond to microbial signals and how microbes, in turn, influence plant immunity and health [5-7].

4. PLANT IMMUNE SYSTEM

4.1 Innate Immune Responses in Plants

Plants, unlike animals, lack an adaptive immune system, but they have evolved a sophisticated and multi-layered innate immune system to recognize and respond to microbial threats [8]. Plant immunity is predominantly characterized by the ability to detect conserved microbial signatures and respond through a cascade of defense responses. The two main branches of the plant innate immune system are pattern-triggered immunity (PTI) and effector-triggered immunity (ETI). PTI is the first line of defense, activated upon recognition of microbe-associated molecular patterns (MAMPs) or pathogen-associated molecular patterns (PAMPs), such as bacterial flagellin or fungal chitin, by cell-surface pattern recognition receptors (PRRs). PRR activation triggers a broad-spectrum response that includes production of reactive oxygen species (ROS), mitogen-activated protein kinase (MAPK) cascades, and transcriptional reprogramming, leading to the expression of defense-related genes [9]. When pathogens deploy effector proteins to suppress PTI and promote virulence, plants counteract with a second tier of defense known as ETI. ETI is mediated by intracellular resistance (R) proteins, which can specifically recognize these effectors, leading to a robust immune response, often associated with localized cell death known as the hypersensitive response (HR). While PTI is often described as a broad, basal response, ETI is highly specific and results in a more potent but localized immune reaction.

4.2 Components and Types of Plant Defense Mechanisms

Plant defense mechanisms can be categorized into preformed defenses and inducible defenses, depending on whether they are constitutively expressed or activated in response to pathogen attack [10]. Preformed defenses include physical barriers such as the cuticle, cell walls, and stomatal closure, which act as the first point of contact preventing pathogen entry. Additionally, plants produce antimicrobial compounds, such as saponins and phenolics, as a constitutive defense. Inducible defenses are triggered upon pathogen recognition and include both local responses at the infection site and systemic defenses throughout the plant. At the local level, the hypersensitive response (HR) results in programmed cell death around the infection site, effectively restricting pathogen spread. Furthermore, inducible defenses include the activation of pathogenesis-related (PR) proteins, accumulation of phytoalexins (antimicrobial secondary metabolites), and cell wall reinforcement through callose deposition [11]. The molecular components of the plant immune system include a variety of receptor-like kinases (RLKs) and receptor-like proteins (RLPs), which detect MAMPs/PAMPs and initiate signaling cascades. A key feature of plant immunity is the guard model, where R proteins act as "guards" monitoring specific host proteins targeted by pathogen effectors. This system ensures that plants can mount a specific response even against rapidly evolving pathogens.

4.3 Mechanisms of Local and Systemic Defense Responses

Plant immune responses can be broadly divided into local and systemic mechanisms, both of which contribute to the overall resistance strategy against pathogens [12]. Local defenses are immediate responses that occur at the site of infection, including the oxidative burst, cell wall fortification, and localized cell death (hypersensitive response), which collectively

create a hostile environment for pathogens. One of the hallmark features of local immune responses is the rapid generation of reactive oxygen species (ROS) and the activation of calcium-dependent signaling pathways. The oxidative burst not only serves to directly inhibit pathogen growth but also acts as a signaling molecule, triggering downstream defense responses. In addition to ROS, the accumulation of antimicrobial compounds, such as phytoalexins and PR proteins, is a critical component of local defense. Systemic responses, on the other hand, are designed to protect uninfected tissues from subsequent attacks. One of the best-studied systemic responses is systemic acquired resistance (SAR), which provides long-lasting resistance throughout the plant after a localized infection. SAR is primarily mediated by the plant hormone salicylic acid (SA) and involves the systemic expression of PR genes, enhancing the plant's readiness to respond to future infections [13]. In SAR, plants can also activate induced systemic resistance (ISR), which is often associated with beneficial rhizobacteria. ISR is regulated by jasmonic acid (JA) and ethylene (ET) signaling pathways, rather than SA, and primes the plant to respond more robustly to pathogen attack. Unlike SAR, which is generally effective against biotrophic pathogens, ISR is more effective against necrotrophic pathogens and insect pests. The interplay between local and systemic defenses is a key feature of plant immunity, allowing plants to fine-tune their responses based on the type of pathogen and the environmental context. Cross-talk between signaling pathways, such as SA-JA-ET interactions, helps integrate various defense signals, ensuring that the plant mounts an appropriate and coordinated response to complex pathogen pressures [14].

5. PLANT-MICROBE INTERACTIONS: TYPES AND MECHANISMS

5.1 Symbiotic Interactions and their Significance

Symbiotic interactions between plants and microbes encompass a wide array of relationships that range from mutualism to commensalism, where both or at least one partner benefits without causing harm to the other (Table 1). A classic example of mutualistic symbiosis is the relationship between leguminous plants and nitrogen-fixing rhizobia bacteria. In this interaction, rhizobia colonize the root nodules of legumes and convert atmospheric nitrogen (N2) into ammonia (NH3), a form of nitrogen that the plant can readily assimilate. This symbiosis is pivotal for plant growth in nitrogen-poor soils, enhancing agricultural productivity and reducing the need for synthetic nitrogen fertilizers [15]. Mycorrhizal fungi form another key group of symbiotic partners, colonizing the roots of over 80% of terrestrial plant species. Arbuscular mycorrhizal fungi (AMF), for example, form intricate networks of hyphae that extend beyond the root zone, significantly improving the uptake of nutrients such as phosphorus and enhancing plant water absorption. In return, plants supply the fungi with carbohydrates derived from photosynthesis, highlighting the reciprocal nature of the symbiosis. The mycorrhizal symbiosis also increases plant tolerance to biotic and abiotic stresses, such as drought, heavy metal toxicity, and pathogen infection [16].

5.2 Pathogenic Interactions and Defense Responses

In contrast to symbiotic interactions, pathogenic interactions involve microbes that cause harm to the plant, leading to diseases that can significantly reduce crop yield and quality. Plant pathogens include a wide variety of organisms such as bacteria (e.g., *Xanthomonas* spp.), fungi (e.g., *Botrytis cinerea*), oomycetes (e.g., *Phytophthora infestans*), and viruses, each employing unique strategies to invade and exploit plant tissues [17]. The infection process typically involves the recognition of the host by pathogen-derived signals, followed by attachment, colonization, and tissue invasion. Plants have evolved a robust immune system to detect and counteract pathogenic attack through two primary layers of defense: pattern-triggered immunity (PTI) and effector-triggered immunity (ETI). PTI is initiated when cell surface receptors recognize conserved pathogen molecules called pathogen-associated molecular patterns (PAMPs). For example, the recognition of bacterial flagellin by the plant receptor kinase FLS2 (Flagellin-Sensing 2) triggers downstream signaling events that result in the production of reactive oxygen species (ROS), cell wall reinforcement, and expression of defense-related genes. To overcome PTI, pathogens secrete effector proteins into plant cells to manipulate host cellular processes and suppress immunity. In response, plants have evolved a second line of defense, ETI, which is activated upon the intracellular detection of these effectors by

specific resistance (R) proteins [18]. ETI is often associated with a more localized and robust response, such as the hypersensitive response (HR), which results in programmed cell death at the infection site to limit pathogen spread. This interaction between pathogen effectors and plant R proteins is described by the "gene-for-gene" model, where each R gene in the plant corresponds to a specific avirulence (Avr) gene in the pathogen. Some pathogens, such as necrotrophic fungi (*Botrytis cinerea*), use toxins to kill host tissue and feed on the dead material, circumventing ETI, which is more effective against biotrophic pathogens that rely on living host tissue. Plants, in turn, employ defense mechanisms that are specifically tailored to necrotrophic pathogens, such as the activation of jasmonic acid (JA)- and ethylene (ET)-mediated signaling pathways. Thus, the outcome of pathogenic interactions is determined by a dynamic interplay between microbial virulence strategies and plant immune responses, influencing disease development and progression [19].

5.3 Role of Beneficial Microbes in Enhancing Plant Defenses

Beneficial microbes, such as plant growth-promoting rhizobacteria (PGPR) and mycorrhizal fungi, play a significant role in enhancing plant immunity through a phenomenon known as induced systemic resistance (ISR). Unlike systemic acquired resistance (SAR), which is triggered by pathogen infection and relies on salicylic acid (SA) signaling, ISR is primarily mediated by jasmonic acid (JA) and ethylene (ET) pathways. Beneficial microbes such as *Pseudomonas fluorescens* and *Bacillus subtilis* colonize plant roots and stimulate ISR, resulting in the systemic activation of defense responses without causing any damage to the host. ISR enhances the plant's ability to defend against a broad spectrum of pathogens, including bacteria, fungi, and nematodes, by priming the plant to respond more rapidly and robustly to subsequent pathogen attacks [20]. This "priming" effect is characterized by a heightened state of alert in the plant's immune system, allowing it to mount a stronger defense upon pathogen challenge. The primed state is achieved through modifications in defensesignaling pathways, chromatin structure, and epigenetic changes, which collectively contribute to a faster and more efficient immune response. Mycorrhizal fungi also play a critical role in enhancing plant defenses. For instance, arbuscular mycorrhizal fungi (AMF) not only improve nutrient uptake but also induce changes in root architecture and defense gene expression, leading to increased resistance against root and foliar pathogen. The presence of AMF has been shown to alter the expression of genes involved in secondary metabolism, cell wall reinforcement, and hormone signaling, thereby providing a multifaceted enhancement of plant immunity [21]. The impact of beneficial microbes on plant defense extends beyond direct interactions with pathogens. They can also influence the composition and functionality of the root microbiome, promoting the establishment of beneficial microbial communities that outcompete potential pathogens. This concept of "microbiome-mediated defense" suggests that plants can actively recruit and maintain beneficial microbes to enhance their own resistance, highlighting the intricate and co-evolved relationships between plants and their associated microbiota.

6. MOLECULAR BASIS OF PLANT DEFENSE AGAINST MICROBES

6.1 Mechanisms of Microbial Recognition

The ability of plants to detect and respond to microbial invaders is pivotal for activating their defense responses. Recognition of pathogens is primarily mediated by two main classes of receptors: pattern recognition receptors (PRRs) located on the cell surface and nucleotide-binding leucine-rich repeat (NLR) receptors present within the cytoplasm [22]. PRRs are involved in the detection of conserved microbial molecules known as microbe-associated molecular patterns (MAMPs) or pathogen-associated molecular patterns (PAMPs), which include bacterial flagellin, fungal chitin, and lipopolysaccharides. For example, the well-studied receptor kinase FLS2 (Flagellin Sensing 2) in *Arabidopsis thaliana* recognizes the bacterial flagellin-derived peptide flg22, initiating downstream defensesignaling. Another key PRR is CERK1 (Chitin Elicitor Receptor Kinase 1), which binds to fungal chitin, a major component of fungal cell walls, thereby triggering a cascade of defense responses. These receptors often function in conjunction with co-receptors like BAK1 (BRI1-Associated Kinase 1), which form complexes with PRRs to enhance the sensitivity and specificity of pathogen detection [23]. Intracellular NLR receptors, on the other hand, recognize pathogen effectors—virulence proteins secreted by pathogens to manipulate host cellular processes and suppress immunity.

These receptors typically contain a nucleotide-binding (NB) domain and leucine-rich repeat (LRR) motifs, which facilitate specific interactions with pathogen effectors or modified host proteins. The recognition of effectors by NLRs triggers a robust immune response known as effector-triggered immunity (ETI), often accompanied by localized cell death called the hypersensitive response (HR). The guard model and decoy model are two prominent theories explaining NLR function. In the guard model, NLRs monitor specific host proteins targeted by pathogen effectors and activate defense upon detecting modifications in these "guarded" proteins [24]. In contrast, the decoy model posits that some host proteins act as decoys to mimic effector targets, diverting effectors away from their true host targets, thereby allowing NLRs to detect effector activities.

6.2 Signal Transduction Pathways in Defense Activation

Upon pathogen recognition, plants activate a complex network of signaling pathways that transduce extracellular signals to intracellular responses, ultimately leading to defense activation. Central to this process are the mitogen-activated protein kinase (MAPK) cascades, calcium-dependent protein kinases (CDPKs), and the production of reactive oxygen species (ROS), which act as second messengers in defensesignaling [25]. MAPK cascades consist of three types of kinases: MAP kinase kinasekinase (MAPKKK), MAP kinase kinase (MAPKK), and MAP kinase (MAPK), which sequentially phosphorylate each other in response to pathogen perception. A classic example is the activation of MAPKs MPK3 and MPK6 upon recognition of flg22 by FLS2, leading to the phosphorylation of downstream transcription factors and the activation of defense genes. Similarly, CDPKs are activated by calcium influxes that occur upon pathogen detection, and these kinases regulate various defense responses, including the production of ROS and expression of pathogenesis-related (PR) genes. Hormonal signaling pathways also play a crucial role in orchestrating defense responses. The salicylic acid (SA), jasmonic acid (JA), and ethylene (ET) pathways are the primary hormonal routes involved in defense regulation, often exhibiting complex cross-talk that fine-tunes the plant's response based on the type of pathogen encountered [26]. SA signaling is typically associated with defense against biotrophic pathogens, which require living host tissue, and is essential for systemic acquired resistance (SAR). In contrast, JA and ET signaling are more effective against necrotrophic pathogens and herbivorous insects, which kill host tissue for nutrients. The NPR1 (Nonexpressor of Pathogenesis-Related Genes 1) protein is a central regulator of SA-mediated defense, acting as a transcriptional coactivator that modulates the expression of PR genes during SAR. JA signaling is regulated by the COI1 (Coronatine Insensitive 1) receptor, which forms a complex with JAZ repressors to regulate JA-responsive gene expression. The interplay between these pathways allows plants to prioritize and coordinate their defenses depending on the nature of the microbial threat.

6.3 Defense-related Gene Expression and Regulatory Networks

The activation of plant defense responses involves extensive transcriptional reprogramming, leading to the expression of hundreds of defense-related genes. Transcription factors such as WRKY, NAC, and MYB play crucial roles in regulating these responses [27]. For instance, the WRKY family of transcription factors is extensively involved in modulating both PTI and ETI responses, binding to W-box elements in the promoters of defense genes. In *Arabidopsis*, WRKY33 is a key regulator of responses to *Botrytis cinerea*, controlling the expression of genes involved in JA and ET signaling. Regulatory networks in plant defense are also influenced by noncoding RNAs, chromatin modifications, and protein degradation pathways. MicroRNAs (miRNAs) such as miR393 and miR398 modulate the expression of target genes involved in defense, adding an additional layer of control to gene regulation. Chromatin modifications, such as histone acetylation and methylation, play a role in priming the plant immune system, where previous exposure to pathogens leads to faster and stronger activation of defense genes upon subsequent attacks. Protein degradation via the ubiquitin-proteasome system (UPS) is another critical regulatory mechanism in plant immunity. E3 ubiquitin ligases such as PUB22 and PUB13 regulate the stability of immune receptors and signaling proteins, ensuring that defense responses are tightly controlled to avoid unnecessary energy expenditure [28]. Moreover, the interaction between ubiquitination and SUMOylation (small ubiquitin-like modifier) pathways adds complexity to the post-translational regulation of immune signaling.

Table 1. Role of beneficial microbes in enhancing plant defenses

Microbial Group	Microbial Species/Strains	Plant Host	Defense Mechanism	Plant Defense Pathway Activated
Rhizobacteria	*Bacillus subtilis*, *Pseudomonas spp.*	Tomato (*Solanum lycopersicum*)	Induction of systemic resistance (ISR), production of antibiotics and volatile organic compounds (VOCs)	ISR via jasmonic acid (JA) and ethylene (ET) signaling
	Azospirillum brasilense	Wheat (*Triticum aestivum*)	Root colonization, increased nutrient uptake, modulation of hormone signaling	Priming of JA and salicylic acid (SA) pathways
Mycorrhizal Fungi	*Rhizophagus irregularis*	Maize (*Zea mays*)	Improved nutrient acquisition, altered root exudates, enhanced root architecture	Priming of JA and SA pathways; reduced ROS accumulation
	Glomus spp.	Tomato (*Solanum lycopersicum*)	Increased phosphorus uptake, induction of defense genes	ISR via JA and SA pathways, enhanced lignin production
Endophytic Fungi	*Trichoderma harzianum*	Cucumber (*Cucumis sativus*)	Induced systemic resistance, production of secondary metabolites	JA/ET-dependent ISR, increased callose deposition
Endophytic Bacteria	*Bacillus amyloliquefaciens*	Tomato (*Solanum lycopersicum*)	Modulation of phytohormones, increased antioxidant activity	Priming of SA and JA pathways, increased reactive oxygen species (ROS) scavenging
Nitrogen-fixing Bacteria	*Rhizobium leguminosarum*	Pea (*Pisum sativum*)	Symbiotic nitrogen fixation, production of nodulation factors	Priming of defense genes, increased phytoalexin production
Actinomycetes	*Streptomyces spp.*	Tomato (*Solanum lycopersicum*)	Production of antimicrobial compounds, promotion of plant growth	Priming of SA pathway, induction of pathogenesis-related (PR) proteins
Yeasts	*Saccharomyces cerevisiae*	Grapevine (*Vitis vinifera*)	Induction of systemic acquired resistance (SAR), secretion of defense elicitors	Activation of SA-dependent SAR, increased chitinase activity
Arbuscular Mycorrhizal Fungi (AMF)	*Funneliformis mosseae*	Wheat (*Triticum aestivum*)	Enhanced nutrient uptake, modulation of stress response	JA/SA priming, increased antioxidant enzyme activity
Phosphate-solubilizing Bacteria	*Pseudomonas fluorescens*	Arabidopsis (*Arabidopsis thaliana*)	Phosphate solubilization, production of siderophores and phytohormones	JA-dependent ISR, enhanced resistance to root pathogens

Source: [17-19]

Table 2. Microbial Strategies to Overcome Plant Defenses

Microbial Group	Microbial Species/Strain	Plant Host	Defense Evasion/Suppression Strategy	Molecular Mechanism
Bacteria	*Pseudomonas syringae*	Arabidopsis (*Arabidopsis thaliana*)	Suppression of host immune responses through effector proteins	Type III secretion system (T3SS); Effectors like AvrPto block PTI and ETI
	Xanthomonas campestris	Tomato (*Solanum lycopersicum*)	Inhibition of stomatal closure, suppression of SA signaling	T3SS effector proteins (e.g., AvrBs3) target host transcription factors
Fungi	*Magnaporthe oryzae*	Rice (*Oryza sativa*)	Secretion of effectors to interfere with host immune receptors	Secreted effector AVR-Pii inhibits Pii-mediated immune response
	Fusarium oxysporum	Tomato (*Solanum lycopersicum*)	Production of secondary metabolites that inhibit host defense	Secretion of fusaric acid to inhibit JA/ET-mediated defenses
Oomycetes	*Phytophthora infestans*	Potato (*Solanum tuberosum*)	Inhibition of cell wall reinforcement, suppression of hypersensitive response (HR)	RXLR effectors (e.g., Avr3a) block callose deposition and HR response
	Hyaloperonospora arabidopsidis	Arabidopsis (*Arabidopsis thaliana*)	Interference with SA signaling, host cell death suppression	RXLR effector HaRxL44 suppresses SA-mediated immunity
Viruses	*Tomato spotted wilt virus* (TSWV)	Tomato (*Solanum lycopersicum*)	Suppression of RNA silencing, inhibition of SA signaling	NSs protein acts as a suppressor of RNA silencing
	Cucumber mosaic virus (CMV)	Tobacco (*Nicotiana tabacum*)	Viral proteins interfere with JA signaling	2b protein suppresses JA-mediated antiviral defense
Nematodes	*Meloidogyne incognita*	Tomato (*Solanum lycopersicum*)	Alteration of plant cell morphology, suppression of defense-related gene expression	Secretion of effectors to alter cell wall dynamics and hormonal pathways
	Heterodera schachtii	Arabidopsis (*Arabidopsis thaliana*)	Modulation of host hormonal balance to promote susceptibility	CLE peptides mimic plant peptides, disrupt SA/JA signaling
Fungal-like Protists	*Plasmodiophora brassicae*	Arabidopsis (*Arabidopsis thaliana*)	Induction of hypertrophy and suppression of host immune responses	Auxin and cytokinin biosynthesis manipulation

Microbial Group	Microbial Species/Strain	Plant Host	Defense Evasion/Suppression Strategy	Molecular Mechanism
Insect Vectors	*Myzus persicae* (Green peach aphid)	Arabidopsis (*Arabidopsis thaliana*)	Saliva-based effector secretion, suppression of host defense	Aphid effector proteins modulate JA/SA crosstalk to facilitate feeding
	Bemisia tabaci (Whitefly)	Tomato (*Solanum lycopersicum*)	Evasion of host immune recognition, suppression of defenses	Effectors suppress SA signaling and ROS production

Source: [29], [30], [32]

7. MICROBIAL STRATEGIES TO OVERCOME PLANT DEFENSES

7.1 Effector Molecules and their Impact on Host Defenses

Effector molecules are specialized proteins secreted by pathogens to facilitate infection by manipulating host cellular processes and suppressing plant immune responses (Table 2). These effectors can be secreted into the extracellular space (apoplastic effectors) or delivered directly into host cells (cytoplasmic effectors) via sophisticated secretion systems, such as the type III secretion system (T3SS) in bacteria. For instance, the bacterial pathogen *Pseudomonas syringae* delivers a suite of effectors through its T3SS to target multiple components of the plant immune system, ultimately dampening host defenses and promoting pathogen proliferation [29]. One well-studied effector is *AvrPtoB* from *Pseudomonas syringae*, which targets the plant receptor kinase FLS2, a key component of pattern-triggered immunity (PTI), and promotes its degradation, thereby preventing the activation of downstream defensesignaling. Similarly, the effector *AvrPphB* cleaves the plant kinase PBS1, disrupting the immune signaling pathway and facilitating bacterial infection. Pathogenic fungi, such as *Magnaporthe oryzae*, also employ a variety of effectors, like *AvrPiz-t*, which targets the rice R protein Piz-t, interfering with immune receptor function and suppressing cell death. Effector proteins not only suppress PTI but also interfere with effector-triggered immunity (ETI). ETI is typically activated by the recognition of specific effectors by intracellular resistance (R) proteins. To counteract ETI, pathogens continuously evolve new effectors or modify existing ones to evade detection by host R proteins, leading to a dynamic arms race between pathogen virulence and plant resistance [30]. For instance, the oomycete *Phytophthora infestans*, which causes late blight in potato, produces the effector *Avr3a* that interacts with and stabilizes the plant ubiquitin E3 ligase CMPG1, thereby suppressing host cell death and immunity.

7.2 Microbial Suppression and Evasion Strategies

Pathogens employ multiple strategies to suppress host immune responses and evade detection. One common strategy is effector-mediated suppression, where effectors inhibit specific components of the plant immune system [31]. For instance, the *Pseudomonas syringae* effector *HopAI1* inactivates MAP kinases (MPK3 and MPK6) by removing a critical phosphate group, effectively shutting down MAPK-mediated defensesignaling. Similarly, the *Ralstonia solanacearum* effector *PopP2* acetylates WRKY transcription factors, impairing their ability to bind DNA and activate defense genes. Another evasion strategy involves masking or modifying PAMPs to prevent their recognition by host PRRs. Some bacteria alter their flagellin structure to avoid detection by FLS2, a key PRR in plants. Similarly, *Agrobacterium tumefaciens* modifies its lipopolysaccharides (LPS) to evade detection by plant LPS receptors. These modifications enable pathogens to avoid triggering PTI, allowing them to establish infection without being detected. Fungi and oomycetes also employ apoplastic effectors that neutralize host-derived antimicrobial compounds. For example, the fungal pathogen *Botrytis cinerea* secretes enzymes such as BcPG1, a polygalacturonase, which degrades plant cell wall polysaccharides and releases oligogalacturonides (OGs) that act as damage-associated molecular patterns (DAMPs) [32]. To counteract OG-induced defenses, *B. cinerea* produces the effector BcPG2, which suppresses the immune response triggered by OGs, allowing the pathogen to colonize plant tissues. In suppressing immunity, pathogens can also manipulate host hormone signaling to create conditions favorable for infection. The bacterium *Pseudomonas syringae* produces coronatine, a mimic of the plant hormone jasmonic acid (JA), which interferes with the salicylic acid (SA)-mediated defense pathway. By activating JA signaling, *P. syringae* suppresses SA-dependent defenses that are critical for resistance against biotrophic pathogens, thereby enhancing its virulence. Pathogens can also evade host detection through epigenetic modifications that alter the expression of genes involved in immune responses. Recent studies have shown that certain effectors from *Ustilago maydis* and *Phytophthora infestans* target host chromatin regulators, modifying histone marks to suppress the expression of defense-related genes [33]. Such strategies underscore the complexity of microbial tactics to avoid host defenses and establish successful infections.

7.3 Co-evolutionary Dynamics between Plants and Microbes

The ongoing arms race between plants and their pathogens has led to co-evolutionary dynamics, where each party continuously evolves new

strategies to outcompete the other. This evolutionary interplay is driven by reciprocal selection pressures: plants evolve new resistance mechanisms to detect and neutralize pathogen effectors, while pathogens evolve to evade or counteract these defenses. One of the best-known models of this dynamic is the gene-for-gene interaction described by Flor (1971), in which plant resistance (R) genes correspond to specific avirulence (Avr) genes in pathogens. When an R protein recognizes an Avr effector, it triggers a robust immune response, often culminating in the hypersensitive response (HR) [34]. However, to overcome this, pathogens often lose or alter these Avr genes, thereby evading recognition. In response, plants must evolve new R genes to detect the modified effectors, perpetuating a cycle of adaptation known as the Red Queen hypothesis. A more recent perspective on co-evolution involves the zig-zag model of plant immunity, which postulates that plant-pathogen interactions involve alternating phases of pathogen recognition and suppression. Initially, plants recognize PAMPs to activate PTI, but pathogens counteract this with effectors, leading to ETI. If the pathogen can evolve new effectors that escape ETI, it temporarily regains virulence, forcing the plant to evolve new R proteins to restore immunity. This cyclical co-evolutionary process results in a highly dynamic interaction, driving diversification in both pathogen effectors and plant immune receptors [35]. The impact of co-evolution is evident in the genetic diversity observed in plant R genes and pathogen effector repertoires. For example, the R gene *RPS2* in *Arabidopsis thaliana* has evolved to recognize the bacterial effector *AvrRpt2*, which in turn has diversified across different *Pseudomonas* strains to evade detection. This evolutionary interplay is also reflected in the effector specialization observed in pathogens, where different strains within the same species can harbor distinct sets of effectors, allowing them to adapt to specific plant hosts [36].

8. BENEFICIAL MICROBES: ENHANCING PLANT DEFENSE AND GROWTH

8.1 Role of Rhizobacteria, Mycorrhizae, and Endophytes

Beneficial microbes, including rhizobacteria, mycorrhizal fungi, and endophytes, play a crucial role in enhancing plant defense and promoting growth by forming complex associations with their host plants. Plant growth-promoting rhizobacteria (PGPR), such as species of *Pseudomonas*, *Bacillus*, and *Azospirillum*, colonize the rhizosphere-the soil region around plant roots-and are known to promote plant health through several mechanisms. These bacteria can enhance nutrient availability, produce phytohormones like indole-3-acetic acid (IAA), and suppress soilborne pathogens through the production of antimicrobial compounds such as siderophores and antibiotics [37]. Mycorrhizal fungi, particularly arbuscular mycorrhizal fungi (AMF), form symbiotic relationships with over 80% of terrestrial plant species. AMF penetrate the root cortical cells and develop highly branched structures called arbuscules, which facilitate nutrient exchange between the host and the fungus. In this symbiosis, the plant provides carbohydrates to the fungi, while the fungi enhance the plant's uptake of phosphorus and other immobile nutrients. In addition to improving nutrient acquisition, mycorrhizal fungi can modulate plant immune responses, enhancing the plant's tolerance to both abiotic and biotic stresses. The presence of AMF has been shown to alter root architecture and increase resistance to root pathogens, such as *Fusarium oxysporum* [38]. Endophytic microbes, including bacteria and fungi, reside within plant tissues without causing apparent harm to the host. These microbes can inhabit roots, stems, and leaves, and are known to enhance plant fitness by improving nutrient acquisition, producing growth-promoting hormones, and activating plant defense pathways. For example, the endophytic bacterium *Bacillus amyloliquefaciens* promotes plant growth and provides resistance against pathogens like *Botrytis cinerea* through the production of volatile organic compounds and the induction of systemic resistance. Similarly, fungal endophytes such as *Piriformospora indica* can colonize plant roots and confer resistance to multiple pathogens by priming the host's immune system. The combined effects of rhizobacteria, mycorrhizae, and endophytes result in improved plant growth, enhanced nutrient uptake, and increased tolerance to environmental stresses [39]. These beneficial microbes not only promote plant health directly but also play a significant role in shaping the plant's associated microbiome, creating a more resilient and diverse microbial community that supports plant development and defense.

8.2 Mechanisms of Induced Systemic Resistance

One of the key contributions of beneficial microbes to plant defense is their ability to induce

induced systemic resistance (ISR), a plant-wide resistance state that primes the plant to respond more robustly to pathogen attack. Unlike systemic acquired resistance (SAR), which is activated by pathogenic microbes and relies on salicylic acid (SA) signaling, ISR is generally triggered by beneficial microbes and is mediated through the jasmonic acid (JA) and ethylene (ET) pathways [40]. ISR is typically initiated at the roots by PGPR, such as *Pseudomonas fluorescens* and *Bacillus subtilis*, which secrete elicitors like flagellin and lipopeptides (e.g., surfactin) that are perceived by plant receptors. These signals lead to the activation of JA and ET pathways, which in turn modulate the expression of a distinct set of defense-related genes. The priming effect of ISR results in the accumulation of inactive forms of defense-related proteins and metabolites, which can be rapidly activated upon pathogen attack. This primed state allows the plant to respond faster and more effectively to subsequent infections, providing enhanced resistance without the need for direct activation of defense responses, which can be metabolically costly [41]. ISR also involves extensive cross-talk between different hormonal pathways, enabling the plant to fine-tune its defense responses based on the specific environmental context. For example, the antagonistic interaction between the SA and JA pathways allows plants to prioritize defenses against different types of pathogens, with SA being more effective against biotrophs and JA against necrotrophs and herbivorous insects. This flexibility is crucial for optimizing defense responses and minimizing trade-offs between growth and immunity. In addition to priming, ISR is often associated with changes in root exudation, leading to the recruitment of beneficial microbes in the rhizosphere and the suppression of soilborne pathogens [42].

8.3 Applications in Promoting Plant Health and Disease Resistance

The ability of beneficial microbes to enhance plant defense and promote growth has significant implications for agriculture. The application of PGPR as bioinoculants is an environmentally friendly alternative to chemical fertilizers and pesticides. For instance, the inoculation of crop plants with *Azospirillumbrasilense* has been shown to enhance root growth, increase nutrient uptake, and improve yield in cereals like maize and wheat. Similarly, *Bacillus subtilis* has been used as a biocontrol agent to suppress soilborne pathogens like *Rhizoctonia solani*, reducing disease incidence in crops such as tomato and cucumber [43]. Mycorrhizal fungi are also widely used in agriculture to promote plant growth and health. Inoculation with AMF has been shown to enhance phosphorus uptake and improve drought tolerance in crops like soybean, maize, and tomato. The use of commercial AMF inoculants in sustainable agriculture can reduce the need for phosphate fertilizers, contributing to improved soil health and reduced environmental impact. Endophytes are emerging as a new frontier in plant-microbe interactions, with applications ranging from biocontrol to stress tolerance. For example, endophytic strains of *Trichoderma* have been used to enhance disease resistance in multiple crops by producing antimicrobial compounds and inducing systemic resistance. Similarly, bacterial endophytes such as *Enterobacter cloacae* have been shown to promote growth and reduce disease severity in rice, highlighting their potential as biocontrol agents [44]. The incorporation of beneficial microbes into integrated pest management (IPM) strategies represents a promising approach for reducing pesticide use and promoting sustainable agriculture. By enhancing plant resilience through microbial inoculants, farmers can achieve better control of pests and diseases while minimizing environmental damage. Moreover, the use of synthetic communities (SynComs), which involve defined consortia of beneficial microbes, is being explored as a means to optimize plant-microbe interactions and tailor microbial functions to specific crop needs.

9. APPLICATIONS AND IMPLICATIONS IN AGRICULTURE

9.1 Use of Plant-Microbe Interactions for Crop Protection

Harnessing plant-microbe interactions for crop protection offers a promising alternative to conventional chemical pesticides, supporting sustainable agriculture and reducing environmental impacts. Beneficial microorganisms, such as plant growth-promoting rhizobacteria (PGPR), mycorrhizal fungi, and biocontrol agents, can enhance plant immunity and suppress the growth of soilborne pathogens [45]. For instance, PGPR strains like *Pseudomonas fluorescens* and *Bacillus subtilis* promote root health and inhibit pathogens such as *Fusarium oxysporum* and *Rhizoctonia solani* by producing antibiotics, siderophores, and lytic enzymes. Biocontrol agents such as *Trichoderma* species are widely used to manage plant

diseases through various mechanisms, including competition for nutrients, mycoparasitism, and induction of plant defenses. These fungi can colonize root surfaces, outcompete pathogenic fungi for space, and release cell-wall-degrading enzymes that inhibit pathogen growth. Additionally, *Trichoderma* spp. can induce systemic resistance, making plants more resistant to subsequent pathogen attacks. Similarly, *Bacillus thuringiensis* is used as a biocontrol agent against insect pests due to its production of insecticidal crystal proteins [46]. Mycorrhizal fungi, particularly arbuscular mycorrhizal fungi (AMF), form symbiotic associations with plant roots, enhancing nutrient uptake and providing protection against pathogens. AMF can activate plant immune responses, producing systemic resistance against pathogens like *Phytophthora* and *Fusarium*. Recent studies suggest that AMF inoculation can be particularly effective in low-input and organic farming systems, where synthetic pesticides are limited.

9.2 Strategies for Developing Disease-Resistant Varieties

Breeding disease-resistant varieties is a key strategy for reducing crop losses and minimizing pesticide use. Plant-microbe interactions offer valuable insights into the molecular mechanisms of resistance, enabling the development of varieties with enhanced immunity [47]. Traditional breeding methods often focus on introgression of resistance (R) genes from wild relatives into cultivated varieties. These R genes encode nucleotide-binding leucine-rich repeat (NLR) proteins that recognize specific pathogen effectors, triggering robust immune responses. For example, the *Xa21* gene from wild rice has been introduced into cultivated rice varieties, conferring resistance to *Xanthomonas oryzae*pv. *oryzae*, a major pathogen causing bacterial blight. To enhance resistance durability, pyramiding multiple R genes targeting different effectors is a common strategy. This approach has been used in wheat to develop varieties resistant to multiple rust pathogens, such as *Puccinia striiformis*, which causes stripe rust. Although effective, R gene-mediated resistance can be overcome by pathogen evolution, as pathogens lose or modify recognized effectors. Thus, integrating multiple resistance mechanisms and continuously monitoring pathogen populations is crucial for maintaining effective disease resistance [48]. Modern genetic engineering and genome-editing technologies, such as CRISPR/Cas9, provide new opportunities for developing disease-resistant crops by enabling precise modifications of plant immune genes. For example, CRISPR-mediated deletion of the susceptibility (S) gene *OsSWEET14* in rice has conferred resistance to *Xanthomonas oryzae* by preventing the pathogen from hijacking the gene for its own benefit. This strategy of editing S genes can be applied to a wide range of crops to develop durable resistance without the need for introducing foreign DNA. Synthetic biology offers another avenue for engineering disease resistance. Synthetic R genes that recognize conserved pathogen molecules can be designed to provide broad-spectrum resistance. For example, synthetic NLRs have been developed to detect a wider range of effector proteins, providing resistance to previously untargeted pathogen strains [49]. These approaches highlight the potential of advanced genetic tools to create crops with enhanced and durable resistance profiles.

9.3 Microbiome Engineering for Sustainable Agriculture

Microbiome engineering aims to manipulate the plant-associated microbiome to enhance plant health and productivity. Plants interact with a diverse array of microorganisms, including bacteria, fungi, and archaea, which collectively influence plant growth, disease resistance, and stress tolerance. By shaping the root and rhizosphere microbiome, it is possible to promote beneficial interactions and suppress pathogenic microbes, creating a healthier and more resilient crop system. One approach is the introduction of beneficial microbial consortia to the soil. Synthetic microbial communities, designed to provide complementary functions such as nitrogen fixation, phosphorus solubilization, and pathogen suppression, can improve plant health and yield [50]. For example, synthetic communities containing *Pseudomonas* and *Bacillus* species have been shown to enhance nutrient uptake and suppress soilborne pathogens in tomato and wheat. This strategy can be tailored to specific crops and environments, providing targeted solutions for enhancing crop resilience. Modifying root exudation patterns is another strategy for shaping the plant microbiome. Root exudates are a complex mixture of organic compounds that influence the composition and activity of the rhizosphere microbiome. Engineering plants to produce specific exudates can selectively recruit

beneficial microbes and suppress pathogens. For example, maize lines with altered benzoxazinoid production have been shown to recruit beneficial *Pseudomonas* strains that enhance resistance to root pathogens [51]. Microbiome transplantation, where healthy soil or rhizosphere microbiomes are introduced into crops, is another emerging approach. This strategy has been successful in improving growth and disease resistance in crops such as cucumber and tomato, by promoting beneficial microbial communities and reducing pathogen load. Microbiome transplantation, combined with precision agriculture technologies, offers a novel means of managing soil health and crop productivity.

10. CHALLENGES

Plant-microbe interactions are highly complex and context-dependent, influenced by a multitude of biotic and abiotic factors [52]. In natural environments, microbial communities are shaped by soil type, pH, nutrient availability, and climatic conditions, making it difficult to predict the outcomes of specific interactions. Additionally, plant genotypes can differentially shape their microbiomes, leading to variability in microbial community composition and function. This complexity poses a challenge for translating laboratory findings to field conditions, where the predictability and stability of microbial inoculants can be compromised. Current research approaches often rely on simplified model systems that do not capture the complexity of natural plant-microbe interactions [53]. Traditional culture-based methods are limited in their ability to isolate and study the full diversity of plant-associated microbes, many of which are unculturable. While high-throughput sequencing has provided insights into microbial diversity, linking microbial identity to function remains challenging. Moreover, the lack of standardization in experimental protocols makes it difficult to compare results across studies, hindering the development of consistent and reproducible strategies for microbiome manipulation. Future research should focus on integrating multi-omics approaches, including genomics, transcriptomics, proteomics, and metabolomics, to gain a holistic understanding of plant-microbe interactions. The use of synthetic microbial communities (SynComs), where defined microbial strains are assembled to mimic natural communities, can help unravel complex interactions and test hypotheses under controlled conditions [54]. Advances in gene-editing technologies such as CRISPR/Cas9 will enable precise manipulation of microbial genomes, allowing researchers to dissect the molecular mechanisms underlying beneficial interactions. Developing predictive models that account for environmental variability and microbial dynamics will be crucial for the successful implementation of microbiome-based solutions in agriculture. Additionally, research should prioritize understanding how climate change and other global stressors affect plant-microbe interactions, as these factors can alter microbial community composition and function, impacting plant health and productivity [55].

11. CONCLUSION

Plant-microbe interactions play a pivotal role in shaping plant health, growth, and resistance against pathogens. Beneficial microbes, including rhizobacteria, mycorrhizae, and endophytes, not only enhance nutrient uptake but also induce systemic resistance, promoting sustainable crop protection. Advances in understanding these interactions have led to innovative applications such as biocontrol agents, disease-resistant crop varieties, and microbiome engineering. However, the complexity of these interactions in natural environments and limitations in current research approaches pose challenges to translating lab-based findings into field conditions. Future research should focus on multi-omics technologies, synthetic communities, and predictive modeling to better harness these interactions. By integrating these strategies, plant-microbe research has the potential to revolutionize agriculture, supporting food security and environmental sustainability.

DISCLAIMER (ARTIFICIAL INTELLIGENCE)

Author(s) hereby declare that NO generative AI technologies such as Large Language Models (ChatGPT, COPILOT, etc.) and text-to-image generators have been used during the writing or editing of this manuscript.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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