



REVIEW

# Integrative Perspectives on Multi-Level Mechanisms in Plant-Pathogen Interactions: From Molecular Defense to Ecological Resilience

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Received: 15 May 2025; Accepted: 20 June 2025; Published: 31 July 2025

**ABSTRACT:** Plant-pathogen interactions involve complex biological processes that operate across molecular, cellular, microbiome, and ecological levels, significantly influencing plant health and agricultural productivity. In response to pathogenic threats, plants have developed sophisticated defense mechanisms, such as pattern-triggered immunity (PTI) and effector-triggered immunity (ETI), which rely on specialized recognition systems such as pattern recognition receptors (PRRs) and nucleotide-binding leucine-rich repeat (NLR) proteins. These immune responses activate intricate signaling pathways involving mitogen-activated protein kinase cascades, calcium fluxes, reactive oxygen species production, and hormonal cross-talk among salicylic acid, jasmonic acid, and ethylene. Furthermore, structural barriers such as callose deposition and lignification, along with the synthesis of secondary metabolites and antimicrobial enzymes, play crucial roles in inhibiting pathogen invasion and proliferation. The plant microbiome further enhances host immunity through beneficial associations with plant growth-promoting rhizobacteria (PGPR) and mycorrhizal fungi, which facilitate induced systemic resistance (ISR) and improve nutrient acquisition. As climate change exacerbates the impact of pathogens, these molecular and microbiome-driven defenses influence disease distribution and plant resilience, highlighting the importance of integrating ecological insights for sustainable disease management. Advancements in microbiome engineering, including the application of synthetic microbial communities and commercial bio-inoculants, offer promising strategies for sustainable disease management. However, the impacts of climate change on pathogen virulence, host susceptibility, and disease distribution complicate these interactions, emphasizing the need for resilient and adaptive agricultural practices. This review highlights the necessity of a holistic, interdisciplinary approach that integrates multi-omics technologies, microbiome research, and ecological insights to develop effective and sustainable solutions for managing plant diseases and ensuring global food security.

**KEYWORDS:** Plant immunity; plant microbiome; induced systemic resistance; multi-omics integration; climate change; plant-pathogen co-evolution; sustainable agriculture

## 1 Introduction

Plant-pathogen interactions represent a complex and dynamic battlefield in which plants, as sessile organisms, have evolved intricate defense systems to mitigate the continuous threat posed by a diverse range of pathogens, such as bacteria, fungi, viruses, and nematodes [1,2]. These interactions occur across multiple biological levels, from molecular signaling networks to ecological adaptations, influencing plant health and, consequently, the stability of agricultural ecosystems [3–5]. Plants rely on a sophisticated immune system that includes both constitutive defenses, such as structural barriers and antimicrobial compounds, and inducible responses triggered upon pathogen attack [6,7]. Early detection of pathogens through conserved microbial patterns initiates a cascade of molecular events, enabling plants to mount timely and effective



defense responses [8]. However, pathogens have co-evolved sophisticated mechanisms to evade or suppress plant defenses, leading to a continual arms race that drives the evolutionary trajectories of both hosts and pathogens [9,10].

At the molecular level, plants use pattern recognition receptors (PRRs) to detect pathogen-associated molecular patterns (PAMPs), thereby triggering a basal defense response known as pattern-triggered immunity (PTI) [11,12]. In response, many pathogens deploy effector proteins that disrupt host immune responses, prompting the activation of a more potent defense mechanism known as effector-triggered immunity (ETI) [13–15]. These layered immune responses involve complex signal transduction pathways, including the activation of mitogen-activated protein kinases (MAPKs), calcium signaling, and the production of reactive oxygen species (ROS) [9]. Hormonal signaling networks further coordinate immune responses, with salicylic acid, jasmonic acid, and ethylene playing pivotal roles in modulating resistance against various pathogen types. The intricate cross-talk between these hormonal pathways determines the specificity and intensity of the plant's immune response, underscoring the importance of understanding these molecular mechanisms to develop effective strategies for disease management [9,16,17].

Beyond molecular and biochemical defenses, plants exhibit several physiological and structural adaptations that contribute to pathogen resistance. These include the rapid deposition of callose, reinforcement of cell walls through lignification, and initiation of localized cell death via the hypersensitive response, which serve as critical barriers to pathogen proliferation [18,19]. Secondary metabolites, such as phenolics, flavonoids, alkaloids, and phytoalexins, play a critical role in plant chemical defense by either directly suppressing pathogen proliferation or by modulating host immune signaling pathways [20–22]. Additionally, antimicrobial enzymes such as chitinases and glucanases degrade pathogen cell walls, further enhancing resistance [9,19]. These physiological responses are intricately linked to the plant's metabolic state and environmental conditions, highlighting the need for a comprehensive understanding of plant defense strategies that integrates both cellular and physiological perspectives [23].

The plant-associated microbiome is a critical component of plant defense, influencing immunity through complex interactions with both beneficial and pathogenic microorganisms [24]. Plant growth-promoting rhizobacteria (PGPR) and mycorrhizal fungi play essential roles in enhancing host immunity by inducing systemic resistance and improving nutrient acquisition [25,26]. Advances in microbiome engineering and the development of synthetic microbial communities offer promising avenues for developing sustainable disease management strategies. Understanding the interplay between host plants and their microbiomes is particularly important in the context of changing environmental conditions, where the stability and functionality of microbial communities can significantly influence plant health and productivity [27,28].

Ecological factors, such as climate variability and human activities, complicate plant-pathogen dynamics by affecting pathogen virulence, host susceptibility, and disease distribution [29]. Rising temperatures, shifting precipitation patterns, and increased atmospheric CO<sub>2</sub> levels affect both pathogen life cycles and the efficacy of plant defense mechanisms [30]. Co-evolutionary interactions between plants and pathogens, influenced by these environmental pressures, drive the emergence of novel virulence factors and resistance genes [31]. These evolutionary dynamics highlight the need for interdisciplinary approaches that combine genetic, ecological, and environmental factors to effectively reduce the impact of plant diseases on global food security.

In light of these considerations, this review provides a comprehensive examination of the multi-level mechanisms involved in plant-pathogen interactions, focusing on molecular recognition systems, intracellular signaling, physiological defenses, and the role of the plant microbiome. It also addresses ecological factors and environmental influences on these interactions. It further examines the ecological and environmental contexts that influence these interactions and highlights emerging strategies to enhance plant immunity

through microbiome management and sustainable agricultural practices. This review integrates insights from molecular, physiological, microbiome, and ecological research, aiming to advance crop protection and sustainable farming practices.

## 2 Molecular Mechanisms of Plant Defense

The molecular mechanisms underlying plant defense provide a crucial framework for understanding how plants detect invading pathogens and initiate highly specialized immune responses. These mechanisms involve a sophisticated surveillance system capable of detecting both general pathogen-associated signals and highly specific virulence factors. Upon detection, plants activate an intricate network of signal transduction pathways that coordinate both localized and systemic defense responses. This multilayered defense strategy halts the progression of pathogen invasion and primes the plant for future encounters, contributing to both immediate survival and long-term resilience.

### 2.1 Pathogen Recognition Systems

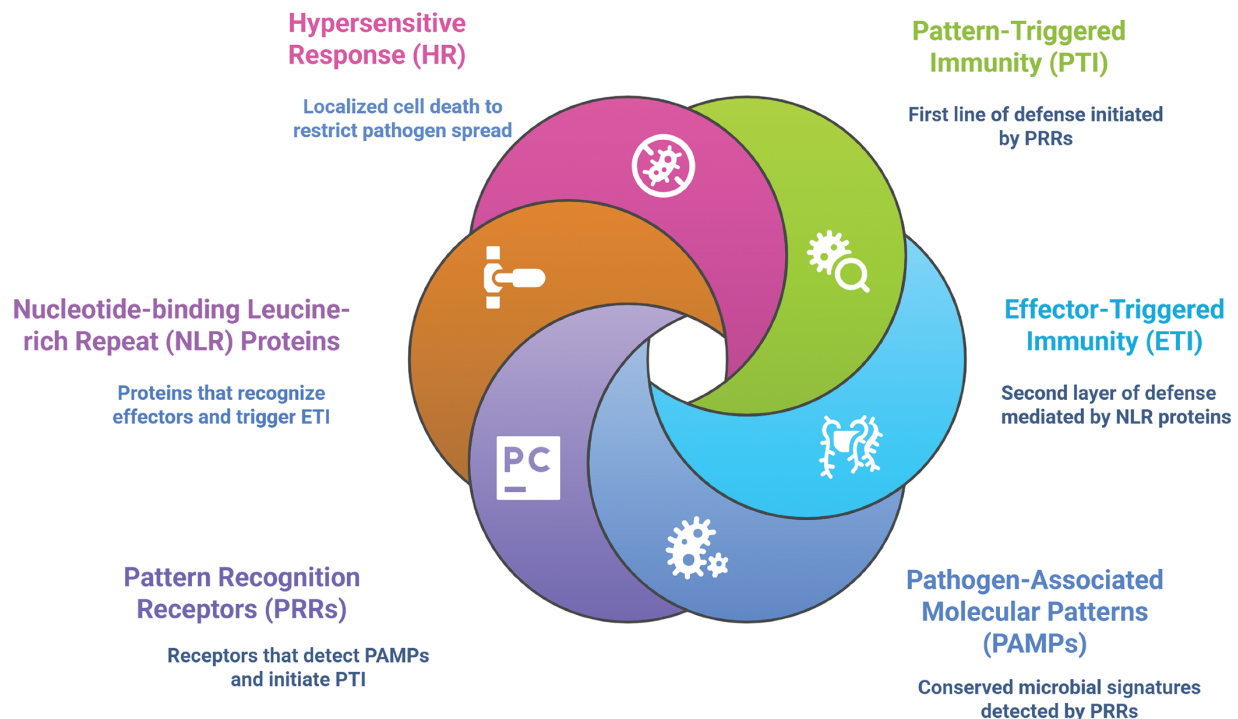
Plants employ two primary immune recognition strategies: pattern-triggered immunity (PTI) and effector-triggered immunity (ETI) [25]. PTI, which is the first line of defense, is initiated when plant cells recognize conserved molecular patterns present on pathogens, known as Pathogen-Associated Molecular Patterns (PAMPs), via cell surface-localized Pattern Recognition Receptors (PRRs). These receptors are able to detect broad microbial signatures, such as bacterial flagellin and fungal chitin, and initiate an immune response that serves as the plant's initial defense [32,33]. Key PRRs include FLS2, which recognizes bacterial flagellin; EFR, which detects elongation factor Tu (EF-Tu); and CERK1, which identifies fungal chitin fragments [34]. Upon activation, these receptors initiate a cascade of defense responses aimed at restricting pathogen growth and invasion. However, despite the robustness of PTI, many pathogens secrete specialized effector proteins that suppress these defenses, thereby facilitating successful colonization. In response to these pathogen effectors, plants activate ETI, which is a more specific and often stronger immune response triggered by the detection of pathogen-derived effector proteins. ETI typically involves a rapid, localized cell death response, known as the hypersensitive response (HR), which limits pathogen spread and reinforces plant immunity [35,36].

To counteract this suppression, plants have evolved a second layer of immunity—effector-triggered immunity (ETI)—which is mediated by intracellular nucleotide-binding leucine-rich repeat (NLR) proteins [33,37]. These NLRs recognize pathogen effectors either directly or indirectly, triggering a rapid and often more robust immune response compared to PTI. ETI is frequently associated with the hypersensitive response (HR), a localized form of programmed cell death at the site of infection that restricts pathogen spread [38]. The coordinated activation of PTI and ETI forms a dynamic and highly effective defense system that is essential for plant survival under pathogen pressure [39,40]. The overall signaling architecture and interaction between PTI and ETI pathways are summarized in Fig. 1, illustrating the sequential and overlapping nature of these critical immune responses.

### 2.2 Signal Transduction Pathways

Following pathogen recognition, plants rapidly convert extracellular signals into intracellular responses through complex signaling cascades. Mitogen-activated protein kinase (MAPK) cascades are central to this process. They mediate the phosphorylation of key transcription factors, leading to the activation of defense-related gene expression [41,42]. These cascades play a crucial role in regulating the production of antimicrobial compounds, reinforcing physical barriers, and initiating localized defense responses [43]. Once the MAPK cascade is activated, it triggers a series of downstream signaling events, including the activation

of calcium signaling, which is a critical component of plant immune responses [44]. Calcium signaling is initiated by a rapid influx of  $\text{Ca}^{2+}$  ions into the cytosol, which in turn activates Calcium-Dependent Protein Kinases (CDPKs) [45]. These kinases modulate gene expression, enzyme activation, and stomatal closure, all of which help limit pathogen entry points into the plant [46]. This calcium-induced signaling cascade also interacts with the production of Reactive Oxygen Species (ROS), which further enhances the plant's defense mechanisms [47].



**Figure 1:** Schematic diagram of plant immune responses including HR, PTI, ETI, PAMPs, PRRs, and NLR pathways (Created with [app.napkin.ai](#))

Another critical component of the plant defense signaling network is the production of ROS, generated through the activation of NADPH oxidases [48]. ROS act as signaling molecules as well as direct antimicrobial agents. They contribute to the reinforcement of the cell wall through lignification and trigger localized programmed cell death, thereby limiting pathogen proliferation [48,49]. Collectively, these signaling events form a highly interconnected network that coordinates effective defense responses at the site of infection and systemically throughout the plant.

The efficiency of plant defense is further regulated by a complex hormonal signaling network involving salicylic acid (SA), jasmonic acid (JA), and ethylene (ET) [50]. These hormones regulate immune responses based on the lifestyle of the invading pathogen [51]. SA signaling predominantly mediates resistance against biotrophic pathogens by activating systemic acquired resistance (SAR), a long-lasting defense mechanism [52]. In contrast, the JA and ET signaling pathways are activated in response to necrotrophic pathogens and herbivorous insects, promoting the synthesis of defense-related compounds toxic to these organisms [53,54]. The cross-regulation between SA and JA/ET pathways ensures that plants mount appropriate immune responses while maintaining growth and metabolic homeostasis [50]. Together, these signaling events create a highly interconnected network that coordinates effective defense responses both

locally at the site of infection and systemically throughout the plant, ensuring a robust and coordinated immune response. A detailed summary of these signaling molecules, their primary defense roles, and the types of pathogens they target is presented in [Table 1](#), providing a comprehensive reference for understanding the hormonal coordination underlying plant immunity ([Fig. 2](#)).

**Table 1:** Major signaling molecules and their roles in plant immunity

| Signal Molecule/<br>Pathway | Primary Defense Roles                                                                                                                                                                                                                                                                 | Target Pathogens/<br>Threats                                           | Key Components/<br>Effectors                                         | Cross-Talk and<br>Interaction                                                                                      | References           |
|-----------------------------|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|------------------------------------------------------------------------|----------------------------------------------------------------------|--------------------------------------------------------------------------------------------------------------------|----------------------|
| MAPK cascades               | Phosphorylation of transcription factors (e.g., WRKY, ERF families) Activation of pathogenesis-related (PR) genes Induces production of antimicrobial compounds (phytoalexins) Strengthens cell walls via callose deposition and lignification Initiates hypersensitive response (HR) | Broad-spectrum defense against fungi, bacteria, and viruses            | MAPKKKs, MAPKKs, MAPKs Transcription factors: WRKY, ERF              | Activated downstream of pattern recognition receptors (PRRs) Interacts with ROS and hormone signaling (SA, JA, ET) | <a href="#">[55]</a> |
| Calcium signaling and CDPKs | Rapid influx of Ca <sup>2+</sup> upon pathogen attack Activation of CDPKs that regulate defense gene transcription Modulates stomatal closure to prevent pathogen entry Controls production of defense enzymes and metabolites                                                        | Broad defense against bacterial and fungal pathogens; stress signaling | CDPK family kinases Calmodulin-binding proteins Ion channels (CNGCs) | Cross-talk with ROS and MAPK cascades Activates downstream hormone pathways (JA, SA)                               | <a href="#">[56]</a> |

(Continued)

Table 1 (continued)

| Signal Molecule/ Pathway      | Primary Defense Roles                                                                                                                                                                                                 | Target Pathogens/ Threats                                                           | Key Components/ Effectors                                  | Cross-Talk and Interaction                                                     | References |
|-------------------------------|-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|-------------------------------------------------------------------------------------|------------------------------------------------------------|--------------------------------------------------------------------------------|------------|
| Reactive Oxygen Species (ROS) | Produced by NADPH oxidases (RBOH family) Acts as a signaling molecule for defense gene activation<br>Directly toxic to pathogens Triggers localized cell death (HR) Reinforces cell walls via oxidative cross-linking | Broad-spectrum defense; crucial in early defense stages                             | NADPH oxidases (RBOHD/F) Peroxidases Superoxide dismutases | Induces MAPK cascades and calcium influx Interacts with SA and JA/ET signaling | [57]       |
| Salicylic Acid (SA)           | Activates systemic acquired resistance (SAR)<br>Upregulates PR genes (PR1, PR2, PR5) Induces defense metabolite production Essential for long-term immunity                                                           | Biotrophic pathogens (fungi, bacteria, viruses)                                     | NPRI, TGA transcription factors PR proteins                | Antagonistic to JA/ET pathways Promotes ROS burst                              | [58]       |
| Jasmonic Acid (JA)            | Induces production of protease inhibitors and secondary metabolites toxic to herbivores and necrotrophs<br>Activates induced systemic resistance (ISR) Regulates defense-related transcription factors (MYC2)         | Necrotrophic pathogens ( <i>Botrytis</i> , <i>Alternaria</i> ), herbivorous insects | COI1 receptor JAZ repressors MYC transcription factors     | Synergistic with ET signaling Antagonistic to SA pathway                       | [59]       |

(Continued)

**Table 1 (continued)**

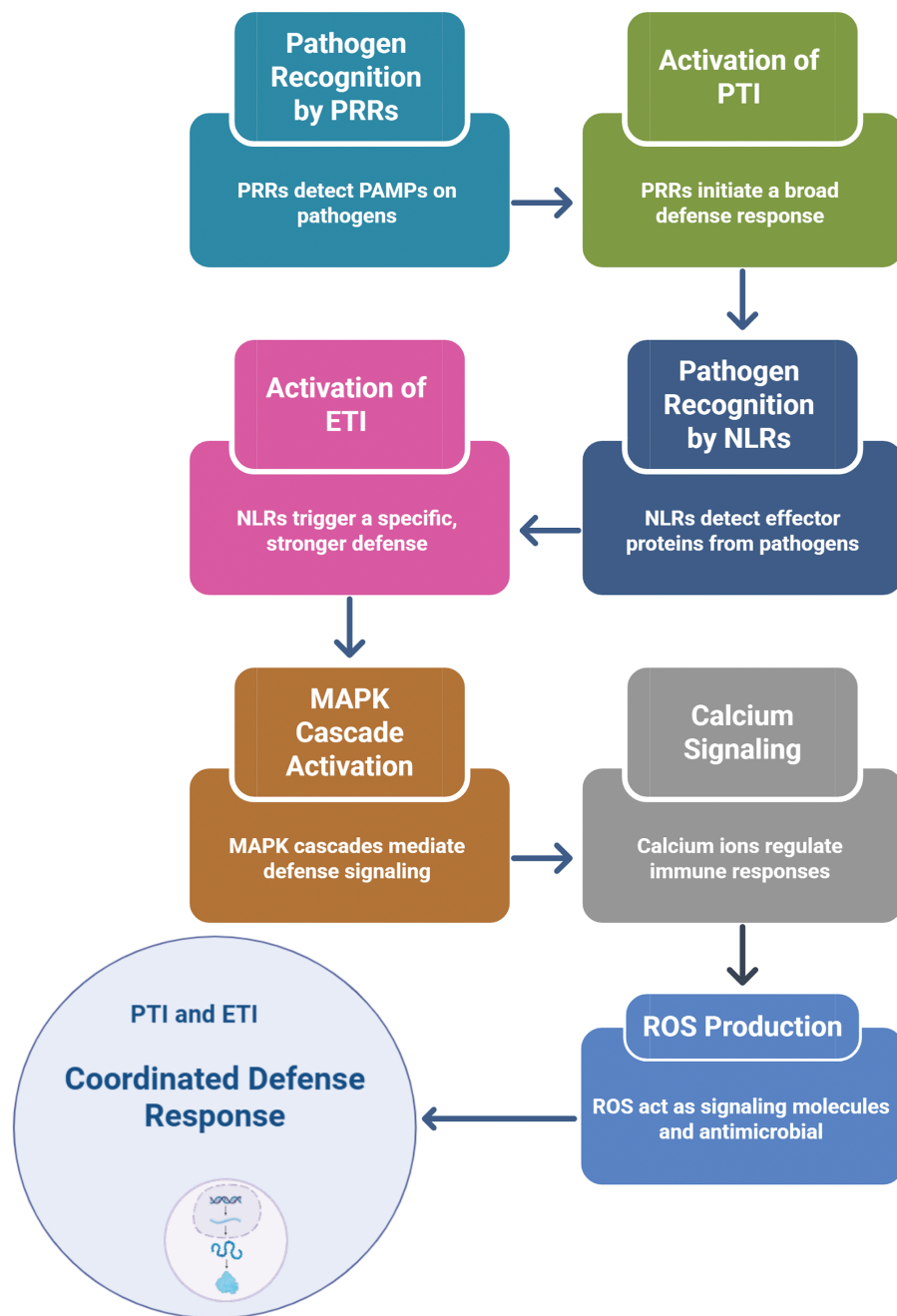
| <b>Signal Molecule/ Pathway</b> | <b>Primary Defense Roles</b>                                                                                                                               | <b>Target Pathogens/ Threats</b>                         | <b>Key Components/ Effectors</b>                                     | <b>Cross-Talk and Interaction</b>                                                                                   | <b>References</b> |
|---------------------------------|------------------------------------------------------------------------------------------------------------------------------------------------------------|----------------------------------------------------------|----------------------------------------------------------------------|---------------------------------------------------------------------------------------------------------------------|-------------------|
| Ethylene (ET)                   | Regulates expression of defense genes (PDF1.2) Enhances cell wall remodeling and lignification Facilitates ISR in combination with JA                      | Necrotrophic pathogens and insect herbivores             | ETR1 receptor EIN2/3 transcription factors ERF1 transcription factor | Works synergistically with JA Modulates ROS responses                                                               | [60]              |
| SA-JA/ET Cross-Talk             | Fine-tunes defense responses to balance growth and immunity Ensures energy-efficient defense deployment Determines pathogen-specific resistance mechanisms | Biotrophic and necrotrophic pathogens; insect herbivores | NPR1 (central regulator) WRKY transcription factors                  | Antagonism: SA suppresses JA/ET-mediated responses Balance prevents overactivation of defense at the cost of growth | [61]              |

### 3 Cellular and Physiological Responses to Pathogen Attack

In addition to molecular recognition and signaling, plants activate a range of cellular and physiological defense responses to prevent pathogen entry and limit disease progression [1]. These responses act as immediate, localized barriers and are often triggered within minutes to hours following pathogen detection [8]. Structural defenses reinforce the physical integrity of plant tissues, whereas metabolic and enzymatic responses create a hostile environment for pathogen survival. Collectively, these mechanisms constitute a robust first line of defense, operating in coordination with molecular signaling pathways to restrict pathogen proliferation and minimize tissue damage.

#### 3.1 Structural Barriers and Cellular Responses

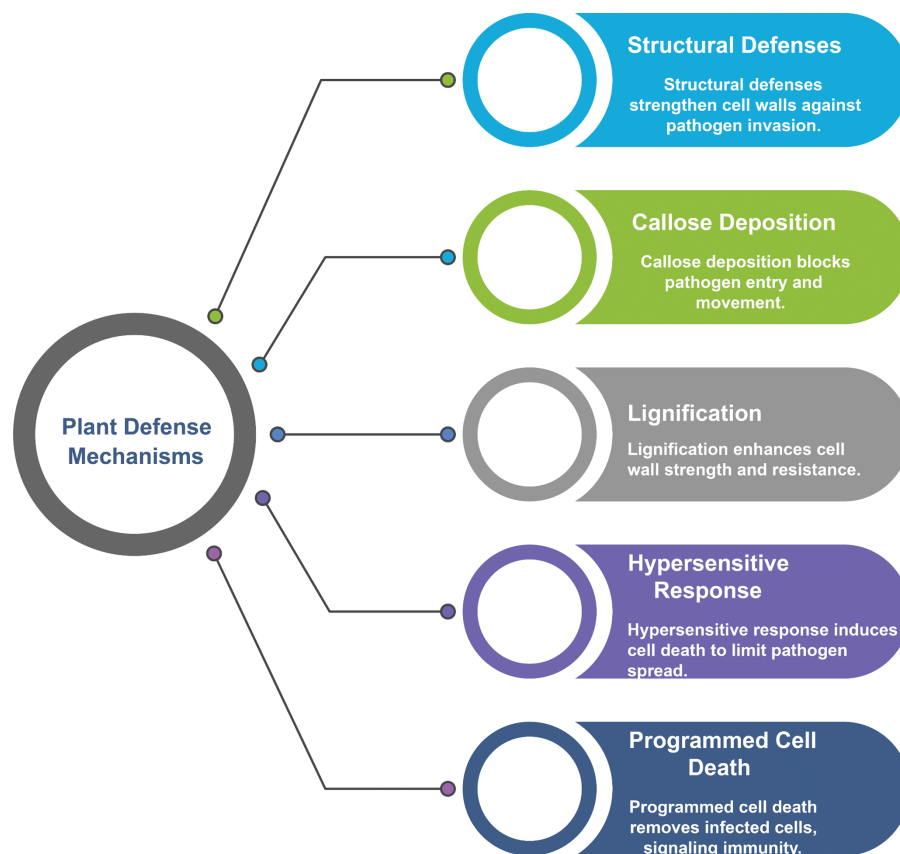
The reinforcement of structural barriers is a critical component of the plant's initial defense strategy. One of the earliest cellular responses is the deposition of callose, a  $\beta$ -1,3-glucan polymer, at the sites of pathogen invasion [62,63]. Callose deposition strengthens the cell wall and serves as a physical barrier, blocking pathogen entry and movement through plasmodesmata [64]. Concurrently, plants initiate lignification, the deposition of lignin polymers, which further fortifies cell walls, enhancing mechanical strength and providing resistance against enzymatic degradation by pathogens [65,66]. Lignification becomes particularly important during the later stages of infection, as many pathogens attempt to breach plant tissues through enzymatic activity.



**Figure 2:** Summary of pathogen recognition by PRRs and NLRs, signaling activation, and the initiation of PTI and ETI (Created with [app.napkin.ai](#) and [BioRender.com](#))

When structural defenses are insufficient to contain pathogen invasion, plants initiate a localized form of programmed cell death known as the hypersensitive response (HR) [67]. HR involves the rapid death of cells at the site of infection, effectively cutting off nutrient supply to the pathogen and limiting its spread [68]. This response is closely associated with the generation of ROS and the activation of defense-related genes that contribute to cell wall strengthening and the synthesis of antimicrobial compounds [69]. Programmed cell death (PCD) also plays a broader role in tissue development and systemic immunity by eliminating infected

or damaged cells and signaling neighboring tissues to initiate defense measures [70]. The coordinated interaction between these structural and cellular defense mechanisms is visually summarized in Fig. 3.



**Figure 3:** Schematic representation of various modes of plant defense mechanisms (Created with [app.napkin.ai](#))

### 3.2 Secondary Metabolite Production and Antimicrobial Compounds

In addition to physical barriers, plants deploy a wide range of chemical defenses, including secondary metabolites and antimicrobial enzymes [20,71]. These compounds play essential roles in directly inhibiting pathogen growth, disrupting pathogen metabolism, and modulating the plant's internal defense signaling pathways. Phenolic compounds, such as tannins and lignans, contribute to cell wall rigidity and possess antimicrobial properties that inhibit bacterial and fungal growth [72]. Flavonoids, another diverse class of phenolic compounds, function only as antioxidants as well as antimicrobial agents, targeting a wide range of pathogens [73]. Additionally, plants synthesize phytoalexins—low molecular weight antimicrobial compounds rapidly produced in response to pathogen attack. Examples include resveratrol in grapevines and camalexin in *Arabidopsis*, both of which demonstrate potent antifungal and antibacterial activity [74,75].

Alkaloids, a diverse class of nitrogen-containing secondary metabolites, exhibit potent antimicrobial activity by disrupting critical pathogen cellular processes such as DNA replication and protein synthesis [76,77]. These compounds often accumulate at sites of infection, thereby contributing to localized defense responses. In addition to chemical defenses, plants produce a range of antimicrobial enzymes, such as chitinases and glucanases, that degrade the structural components of fungal and bacterial cell walls [78]. Chitinases specifically target chitin in fungal cell walls, whereas  $\beta$ -1,3-glucanases target glucan polymers, resulting in the structural collapse of pathogen cell walls and ultimately resulting in cell lysis [79,80]. These

enzymatic defenses work synergistically with secondary metabolites to inhibit pathogen growth and limit their spread [78]. The diversity and functionality of secondary metabolites and antimicrobial enzymes are summarized in Table 2, which outlines their classes, representative compounds, and pathogen targets.

**Table 2:** Key secondary metabolites and their antimicrobial activities

| Class          | Example compounds                                 | Targeted pathogens                                                        | Mechanism of action                                                                           | References |
|----------------|---------------------------------------------------|---------------------------------------------------------------------------|-----------------------------------------------------------------------------------------------|------------|
| Phenolics      | Tannins, Lignans, Quercetin, Gallic Acid.         | Bacteria (e.g., <i>E. coli</i> ), fungi ( <i>Candida</i> spp.).           | Precipitate proteins, disrupt cell walls, inhibit microbial enzymes, act as antioxidants.     | [81,82]    |
| Flavonoids     | Resveratrol, Camalexin, Luteolin, Kaempferol.     | Bacteria ( <i>Staphylococcus aureus</i> ), fungi ( <i>Fusarium</i> spp.). | Inhibit DNA gyrase and topoisomerase, disrupt energy metabolism, scavenge free radicals.      | [83,84]    |
| Alkaloids      | Berberine, Sanguinarine, Quinine.                 | Bacteria, viruses (HSV), fungi.                                           | Interfere with nucleic acid synthesis, disrupt protein biosynthesis, alter cell permeability. | [85,86]    |
| Terpenoids     | Eugenol, Limonene, Menthol.                       | Gram-positive and Gram-negative bacteria, fungi.                          | Disrupt microbial membranes, impair respiration, inhibit biofilm formation.                   | [87]       |
| Phytoalexins   | Resveratrol (Grapevine), Camalexin (Arabidopsis). | Fungi ( <i>Botrytis cinerea</i> ), bacteria ( <i>Pseudomonas</i> spp.).   | Induced rapidly after infection, disrupt pathogen metabolism and cell integrity.              | [88]       |
| Saponins       | Glycyrrhizin, Aescin.                             | Bacteria, fungi, protozoa                                                 | Create pores in microbial membranes, leading to cell lysis.                                   | [83]       |
| Glucosinolates | Sinigrin, Glucoraphanin.                          | Fungi ( <i>Alternaria</i> spp.), bacteria.                                | Break down into isothiocyanates, inhibit microbial enzymes.                                   | [86]       |
| Enzymes        | Chitinases, $\beta$ -1,3-Glucanases.              | Fungi ( <i>Aspergillus</i> spp.), bacteria.                               | Degrade fungal cell walls, cause osmotic imbalance and cell lysis.                            | [89]       |

### 3.3 Stomatal Closure and Pathogen Entry

Upon pathogen attack, stomatal closure serves as a critical early defense response to limit pathogen entry [90]. Stomata, the pores on plant leaves, are the primary points of pathogen ingress, especially for biotrophic pathogens that require living host cells. Closure is often triggered by reactive oxygen species (ROS) and calcium signaling, which induce signaling pathways that reinforce defense responses [91]. Calcium-dependent protein kinases (CDPKs) activate additional signaling, further promoting stomatal closure to prevent the pathogen from entering the plant. This rapid response is crucial in the early stages of infection to limit pathogen spread [92].

### **3.4 Callose Deposition and Strengthening of Physical Barriers**

In response to pathogen attack, callose deposition forms a physical barrier at the site of infection, limiting pathogen movement [93]. Callose, a  $\beta$ -1,3-glucan, is synthesized and deposited around plant cells, particularly in response to PAMP recognition by Pattern Recognition Receptors (PRRs) [94]. This action is a key component of the localized defense response, which also includes lignification of cell walls, making them more resistant to pathogen invasion. Together, these structural changes serve to isolate the pathogen and prevent its spread within the plant.

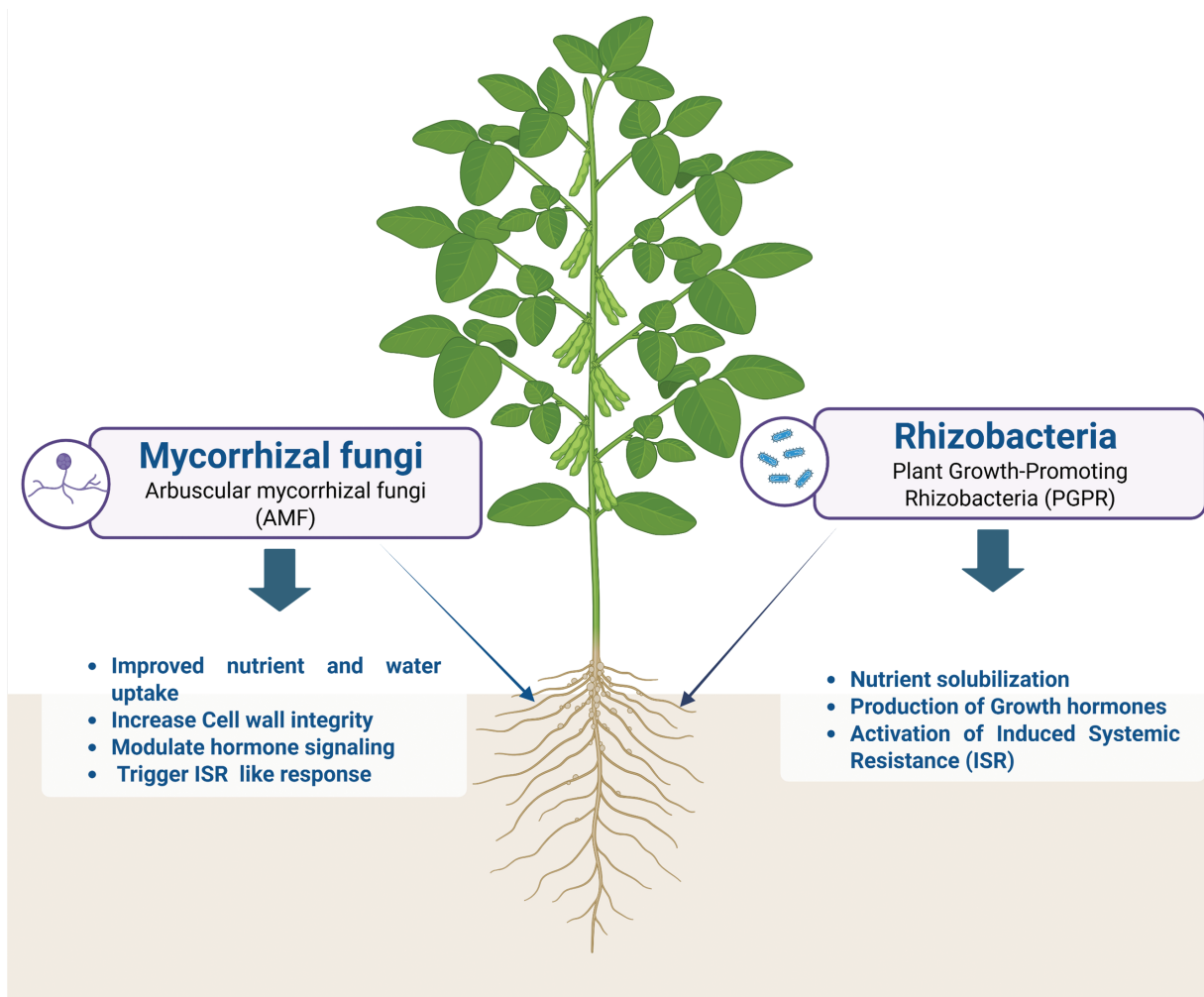
## **4 Microbiome and Plant Immunity Interactions**

The plant microbiome has emerged as a critical component in modulating plant immunity, offering an additional layer of defense that complements the plant's intrinsic molecular, cellular, and physiological responses [95]. The rhizosphere, phyllosphere, and endosphere host diverse microbial communities that significantly influence plant health, nutrient acquisition, and resistance to pathogens [96]. These microbial assemblages engage in complex chemical and molecular interactions with the host plant, enhancing immune responses and improving resilience to both biotic and abiotic stresses [97]. As our understanding of plant-microbiome interactions deepens, the potential to harness beneficial microbes as sustainable alternatives to chemical pesticides and fertilizers becomes increasingly evident, offering an environmentally friendly approach to enhance crop productivity and disease resistance.

### **4.1 Beneficial Microbes in Enhancing Plant Immunity**

Among beneficial microbes, PGPR and mycorrhizal fungi play particularly important roles in enhancing plant immunity [98]. PGPR colonize plant roots and exert their beneficial effects through various mechanisms, including nutrient solubilization, production of growth hormones, and most notably, the activation of induced systemic resistance (ISR) [99,100]. ISR is a defense mechanism triggered by beneficial microbes that primes the plant for enhanced resistance against a wide range of pathogens without the direct involvement of pathogen recognition [101]. Unlike systemic acquired resistance (SAR), which is primarily mediated by salicylic acid, ISR is regulated mainly through jasmonic acid and ethylene signaling pathways [102,103]. This allows plants to mount more effective responses against necrotrophic pathogens and herbivorous insects.

Mycorrhizal fungi, particularly arbuscular mycorrhizal fungi (AMF), form symbiotic associations with plant roots, enhancing nutrient and water uptake [104]. In addition to their nutritional benefits, mycorrhizal fungi contribute to plant immunity by enhancing cell wall integrity, modulating hormone signaling pathways, and triggering ISR-like defense responses [105,106]. Specific microbial interactions also play a crucial role in enhancing plant resistance to particular pathogens. PGPR and mycorrhizal fungi, for instance, can directly or indirectly ISR by stimulating plant defense pathways. These microbes interact with the plant's immune system, often priming it to respond more effectively to specific pathogen threats. Additionally, these interactions can influence the plant's metabolic processes, leading to the production of secondary metabolites and enzymes that inhibit pathogen growth. These beneficial interactions improve the plant's resilience to soil-borne pathogens and contribute to long-term soil health by fostering a stable and diverse microbial ecosystem. The complex interactions between plant roots and their associated microbiota, and the pathways through which these microbes enhance plant immunity, are illustrated in [Fig. 4](#), providing a comprehensive overview of microbial contributions to plant defense.



**Figure 4:** Interaction between plant roots and beneficial microbiome (Created with [BioRender.com](https://www.biorender.com))

#### 4.2 Microbiome Engineering for Disease Resistance

Recent advances in microbiome research have paved the way for microbiome engineering approaches aimed at enhancing plant health and disease resistance. One such strategy involves the development of synthetic microbial communities (SynComs), which are carefully designed consortia of beneficial microbes selected for their synergistic interactions and ability to promote plant growth and immunity [107,108]. SynComs are engineered to deliver targeted benefits, such as improved nutrient acquisition, suppression of soil-borne pathogens, and activation of plant defense pathways. These communities can be tailored to specific crops and environmental conditions, offering a highly customizable solution for sustainable agriculture [109,110].

In addition to SynComs, the use of commercial bio-inoculants has gained significant attention as a practical strategy for enhancing plant immunity in agricultural systems. These bio-inoculants consist of beneficial microbial strains known to promote plant growth and suppress pathogens through various mechanisms, including competitive exclusion, production of antimicrobial metabolites, and induction of host defense responses [111,112]. Bio-inoculants offer an environmentally sustainable alternative to synthetic agrochemical inputs, reducing reliance on targetischemical pesticides and fertilizers while improving soil

health and crop resilience. A summary of widely used commercial bio-inoculants, including their microbial compositions and pathogens they target is provided in Table 3, highlighting their practical applications in modern agricultural practices.

**Table 3:** Commercial bio-inoculants and their target pathogens

| Product name              | Microbial composition (Species)                                                                   | Target pathogens                                                          | References |
|---------------------------|---------------------------------------------------------------------------------------------------|---------------------------------------------------------------------------|------------|
| Bio-NPK                   | <i>Azotobacter chroococcum</i> ,<br><i>Azospirillum brasilense</i> , <i>Bacillus megaterium</i> . | Soil-borne pathogens, nutrient deficiencies.                              | [113]      |
| Bio-Fertilizer Consortium | <i>Rhizobium leguminosarum</i> ,<br><i>Bacillus subtilis</i> , <i>Trichoderma harzianum</i> .     | <i>Fusarium</i> spp., <i>Pythium</i> spp.,<br><i>Rhizoctonia solani</i> . | [114]      |
| Bio-Guard                 | <i>Pseudomonas fluorescens</i> , <i>Bacillus amyloliquefaciens</i> .                              | <i>Fusarium oxysporum</i> ,<br><i>Verticillium dahliae</i> .              | [115]      |
| Myco-Inoculant Plus       | <i>Glomus intraradices</i> , <i>Bacillus megaterium</i> .                                         | <i>Meloidogyne incognita</i> ,<br><i>Phytophthora capsici</i> .           | [116]      |
| Tricho-Mix                | <i>Trichoderma viride</i> , <i>Trichoderma harzianum</i> .                                        | Soil-borne fungal pathogens                                               | [117,118]  |
| EndoBoost                 | <i>Enterobacter cloacae</i> , <i>Pantoea agglomerans</i> , <i>Bacillus velezensis</i> .           | General pathogen suppression, growth promotion.                           |            |
| RhizoGuard                | <i>Rhizobium etli</i> , <i>Bacillus thuringiensis</i> .                                           | Root pathogens, nematodes.                                                | [119]      |
| EcoBioProtect             | <i>Pseudomonas putida</i> , <i>Bacillus cereus</i> .                                              | <i>Fusarium</i> spp., <i>Sclerotinia</i> spp., <i>Botrytis cinerea</i> .  | [120]      |
| MycoEndo Shield           | <i>Rhizophagus irregularis</i> ,<br><i>Piriformospora indica</i> , <i>Serendipita vermifera</i> . | Plant pathogenic fungi, stress tolerance.                                 | [112]      |
| PhosSol Enhancer          | <i>Bacillus polymyxa</i> , <i>Pseudomonas striata</i> .                                           | Nutrient deficiency correction                                            | [121]      |
| Yeast Shield              | <i>Saccharomyces cerevisiae</i> , <i>Pichia pastoris</i> .                                        | Postharvest fungal pathogens                                              | [122]      |
| AzotoPhos Max             | <i>Azospirillum lipoferum</i> , <i>Bacillus circulans</i> .                                       | Soil fertility improvement                                                | [123]      |
| Boni-Protect <sup>®</sup> | <i>Aureobasidium pullulans</i>                                                                    | Postharvest pathogens ( <i>Penicillium</i> , <i>Botrytis</i> )            | [124]      |
| Nexy <sup>®</sup>         | <i>Candida oleophila</i>                                                                          | Fruit storage pathogens                                                   | [124]      |
| Shemer <sup>®</sup>       | <i>Metschnikowia fructicola</i>                                                                   | Postharvest decay fungi                                                   | [124]      |
| Romeo <sup>®</sup>        | <i>Saccharomyces cerevisiae</i>                                                                   | Broad-spectrum pathogen protection                                        | [124]      |

## 5 Ecological and Environmental Perspectives

Plant-pathogen interactions are significantly influenced by ecological and environmental factors, which shape the prevalence, intensity, and outcomes of plant diseases at both local and global scales [125]. Changes

in environmental conditions directly impact the biology of pathogens as well as the physiological state and defense capabilities of host plants [126,127]. As ecosystems face increasing anthropogenic pressures, such as habitat fragmentation, intensive agricultural practices, and climate change, the dynamics of plant diseases become more complex and less predictable [126]. These evolving dynamics have significant implications for global food security, biodiversity conservation, and sustainable agriculture, highlighting the need for a deeper understanding of the influence of ecological and environmental factors on plant-pathogen interactions.

### 5.1 Impact of Climate Change on Plant-Pathogen Dynamics

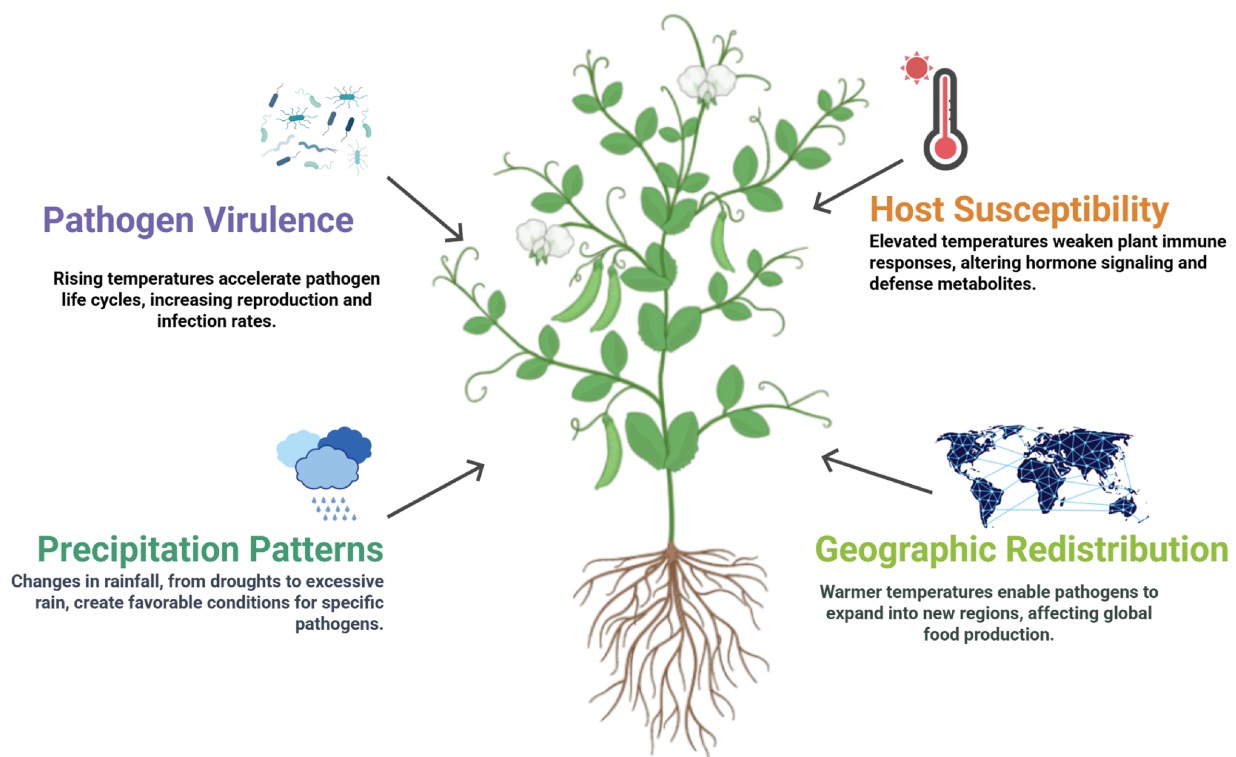
Climate change is a major driver of emerging and re-emerging plant diseases, influencing both pathogen virulence and host susceptibility. Rising global temperatures accelerate pathogen life cycles, resulting in increased reproduction rates and shortened infection cycles [128]. Elevated temperatures may also impair plant immune responses by disrupting hormone signaling pathways and suppressing the production of defense-related metabolites [129]. Furthermore, changes in precipitation patterns—ranging from prolonged droughts to excessive rainfall—create favorable conditions for specific pathogens [130]. High humidity and excessive rainfall typically promote the proliferation of fungal pathogens, whereas drought conditions can predispose plants to soil-borne diseases by weakening their structural integrity and physiological defenses [131,132].

Climate change has also led to a shift in the geographic distribution of plant pathogens, facilitating their spread into new geographic regions [133,134]. Warmer temperatures and altered weather patterns enable pathogens to expand beyond their traditional habitats, leading to the emergence of diseases in regions where host plants may lack effective resistance mechanisms [135]. This geographic redistribution has been observed in several major crop systems and poses significant threats to global food production. The conceptual relationships between climate variables and disease dynamics are illustrated in Fig. 5, which provides an overview of how temperature, precipitation, and atmospheric variations influence pathogen virulence, host susceptibility, and disease spread.

### 5.2 Co-Evolutionary Dynamics between Plants and Pathogens

The evolutionary history of plants and their pathogens is characterized by a continual “arms race”, in which both parties evolve new strategies to outcompete one another. This dynamic is often explained through the “Red Queen Hypothesis”, which suggests that species must continuously adapt and evolve not only for reproductive success but also to survive against constantly evolving antagonists [136]. In the context of plant-pathogen interactions, plants develop new resistance (R) genes and defense mechanisms, where pathogens concurrently develop novel virulence factors and effectors to bypass these defenses [137]. This co-evolutionary struggle shapes both the genetic diversity of plant populations and the pathogenic potential of microbial species.

Several case studies highlight this ongoing battle between host plants and their pathogens. The rice-blast fungus (*Magnaporthe oryzae*), one of the most destructive pathogens of rice, continuously evolves new strains capable of overcoming resistance genes introduced through breeding programs [138–140]. Similarly, wheat rust diseases, particularly stem rust caused by *Puccinia graminis* f. sp. *tritici*, have resurged due to the emergence of highly virulent races such as the Ug99 lineage, which can overcome previously effective resistance genes in wheat [141,142]. These examples underscore the necessity of developing durable resistance strategies that account for the evolutionary potential of pathogens. Understanding these co-evolutionary dynamics is essential for designing breeding programs and disease management strategies that remain effective over time, even in the face of rapidly evolving pathogen populations.



**Figure 5:** Effects of climate change on plant-pathogen interactions (Created with [BioRender.com](#))

## 6 Challenges and Future Directions

Despite significant advancements in understanding the mechanisms of plant defense, several unresolved challenges continue to limit the development of effective and sustainable disease management strategies. One of the major challenges is the incomplete integration of multi-omics approaches, including genomics, transcriptomics, proteomics, and metabolomics, which are essential for comprehensively understanding the complex regulatory networks involved in plant-pathogen interactions [143,144]. For example, recent studies integrating transcriptomic and proteomic data have identified key immune-related genes and proteins that were previously undetected when analyzed separately, revealing new targets for breeding pathogen-resistant crops [145,146]. Similarly, the integration of metabolomic data with genomic information has provided insights into the metabolic shifts that occur during pathogen attack, helping to identify novel metabolites involved in plant defense [147]. Although these technologies have provided valuable insights individually, the lack of comprehensive data integration prevents the identification of key molecular targets that could be harnessed to improve disease resistance. Incorporating systems biology approaches, such as network-based integration of multi-omics data, has been instrumental in revealing cross-talk between various molecular pathways, metabolic processes, and phenotypic responses [148]. These integrated approaches are paving the way for a more holistic understanding of plant immunity. Advanced computational tools and systems biology frameworks are urgently needed to combine these datasets and reveal critical cross-talk among molecular pathways, metabolic processes, and phenotypic responses.

Translating laboratory discoveries into practical field-level solutions remains another significant challenge [149]. Most experimental studies are conducted under controlled conditions that fail to adequately capture the complexity of agricultural ecosystems, where plants are simultaneously exposed to multiple biotic and abiotic stresses [150,151]. Consequently, resistance traits and bio-inoculant applications that show

promise in laboratory settings often yield inconsistent results under variable field conditions. Bridging this gap requires greater emphasis on long-term field validation, the development of crop varieties suited to diverse agroecological zones, and the incorporation of real-world environmental variables into experimental designs [152].

Overcoming these challenges also requires interdisciplinary collaboration and the establishment of supportive policy frameworks. Integrating expertise from molecular biology, plant breeding, microbiome science, ecology, and climate science will be essential to develop resilient cropping systems. Policy initiatives should promote collaborative research, incentivize the adoption of sustainable agricultural technologies, and facilitate the responsible deployment of biotechnological innovations. Furthermore, investment in region-specific solutions and data-sharing platforms will enhance the scalability and impact of research efforts, ultimately contributing to improved global food security and agricultural sustainability.

## 7 Conclusions

Understanding plant–pathogen interactions requires a multidimensional perspective that incorporates molecular recognition systems, cellular defense mechanisms, microbiome contributions, and broader ecological and environmental contexts. Plants employ highly specialized immune strategies, including PTI and ETI, supported by complex intracellular signaling networks and structural barriers such as callose deposition and lignification. Secondary metabolites and antimicrobial enzymes further reinforce plant immunity by directly inhibiting pathogen growth and enhancing resistance responses.

In addition to these intrinsic mechanisms, beneficial interactions with microbial communities play a pivotal role in strengthening plant immunity. PGPR and mycorrhizal fungi contribute to ISR and improve plant resilience under various environmental stresses. At the ecosystem level, climate change continues to reshape plant–pathogen dynamics, leading to the emergence of new diseases and challenging existing resistance strategies. The ongoing evolutionary arms race between plants and pathogens highlights the need for continuous innovation in crop protection.

A holistic approach integrating advances in molecular research, microbial biotechnology, ecological understanding, and climate resilience is critical for the development of sustainable disease management strategies. Key findings from this review suggest that plant immunity is deeply interconnected with both intrinsic defense mechanisms and beneficial microbial interactions. Future research should focus on integrating multi-omics technologies to unlock novel molecular targets for resistance, with a particular emphasis on validating these findings under field conditions. Additionally, there is a growing need to explore microbiome engineering as a tool for enhancing plant defenses. The collaboration between bioinformatics and field-based research will be essential for translating lab-based discoveries into real-world agricultural applications. Policy support and international collaboration will be critical to ensure that these scientific advances translate into practical solutions that safeguard agricultural productivity and contribute to long-term global food security.

**Acknowledgement:** Not applicable.

**Funding Statement:** The authors received no specific funding for this study.

**Author Contributions:** Conceptualization, writing—original draft preparation, resources, software, validation, visualization, Adnan Amin and Wajid Zaman; writing—review and editing, supervision, Wajid Zaman. All authors reviewed the results and approved the final version of the manuscript.

**Availability of Data and Materials:** Data sharing not applicable to this article as no datasets were generated or analyzed during the current study.

**Ethics Approval:** Not applicable.

**Conflicts of Interest:** The authors declare no conflicts of interest to report regarding the present study.

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