



REVIEW ARTICLE

The role of the plant microbiome in modulating plant immune responses in disease management: A review

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Abstract

The plant microbiome, which encompasses diverse communities of bacteria, fungi, archaea, viruses and oomycetes that colonise plant surfaces and internal tissues, is a critical determinant of plant health, resilience and productivity. This review systematically examines how plant-associated microbial communities modulate plant immune signalling pathways, with particular emphasis on pattern-triggered immunity (PTI), effector-triggered immunity (ETI), systemic acquired resistance (SAR) and induced systemic resistance (ISR). Critical evaluation of the molecular mechanisms through which beneficial microorganisms prime host defences, including microbe-associated molecular pattern (MAMP) perception, salicylate and jasmonate signalling cascades, reactive oxygen species (ROS) dynamics and transcriptional reprogramming of immune networks. Additionally, we address pathogen-mediated immune suppression strategies, including effector-driven interference, hormonal hijacking and pathobiome-associated dysbiosis, drawing explicit connections to the limitations of classical Koch's postulates in the context of community-level disease causation. This review further explores how environmental factors, including climate change, drought and agricultural intensification, reshape the microbiome composition and immune competence. Translational applications, including synthetic microbial communities (SynComs), microbiome-assisted breeding and microbiome restoration strategies, have been evaluated for field efficacy, scalability and regulatory feasibility. Current methodological limitations and future research priorities are discussed to guide the development of microbiome-informed, climate-resilient and sustainable disease management frameworks. This review highlights the transformative potential of harnessing plant microbiomes for next-generation agricultural protection strategies.

Keywords: dysbiosis; induced systemic resistance; microbiome; pattern-triggered immunity; salicylic acid

Introduction

The plant as a holobiont: Conceptual framework

Plants do not exist as isolated biological entities; rather, they function as complex ecological units known as holobionts, a term that describes the host organism together with all its associated microbial symbionts, which collectively contribute to host fitness, development and adaptive responses to environmental stress (1, 2). The holobiont concept has fundamentally transformed our understanding of plant biology, shifting the paradigm from a plant-centric to a systems-level perspective, in which host genetics, microbial ecology and environmental conditions interact dynamically to determine plant health outcomes (3).

The plant microbiome encompasses all microbial communities, including bacteria, fungi, archaea, viruses, oomycetes and protists, that colonise the rhizosphere (root-surrounding soil zone), phyllosphere (aerial plant surfaces), endosphere (internal plant tissues) and spermosphere (seed-associated zone) (4, 5). Each of these compartment's harbours

compositionally distinct microbial assemblages shaped by plant exudates, tissue chemistry, oxygen availability and host immune signalling (6). The rhizosphere, in particular, represents one of the most microbially dense and metabolically active ecosystems on Earth, where plant roots deposit up to 40 % of photosynthetically fixed carbon as root exudates, thereby selectively recruiting and sustaining microbial consortia that profoundly influence plant nutrient acquisition, stress tolerance and immune priming (7, 8).

Key concepts and terminology

To ensure conceptual clarity throughout this review, the following terms are explicitly defined:

Holobiont: A composite biological unit comprising the plant host and its associated microbiome, whose collective genomic capacity (hologenome) determines adaptive fitness (1).

Dysbiosis: A disruption in the structural and functional balance of the microbiome, often triggered by pathogen invasion, abiotic stress, or agricultural perturbation, leading to impaired host immune competence and increased disease susceptibility (9).

Pathobiome: A complex ecological network of microbial communities, including pathogens, opportunists and dysbiosis-associated taxa, that collectively drive disease development, extending beyond the single-pathogen paradigm (10, 11).

Pattern-triggered immunity (PTI): The first layer of plant innate immunity, activated when plant pattern recognition receptors (PRRs) detect conserved microbial signatures known as microbe-associated molecular patterns (MAMPs) or damage-associated molecular patterns (DAMPs), triggering basal defence responses (12).

Effector-triggered immunity (ETI): A more robust immune response activated when plant intracellular nucleotide-binding leucine-rich repeat (NLR) receptors recognise pathogen-secreted effector proteins, frequently culminating in the hypersensitive response (HR) (13).

Microbe-associated molecular patterns (MAMPs): Conserved molecular signatures of microbial origin, including flagellin, chitin, lipopolysaccharides and peptidoglycans, recognised by plant PRRs to initiate immune signalling (14).

Effectoromics: A systems-level approach to comprehensively identify, characterise and functionally annotate pathogen effector protein repertoires and their corresponding host immune targets, enabling the design of durable resistance strategies (15).

NLR engineering: The rational or computationally guided modification of plant NLR immune receptor genes to broaden pathogen recognition specificity, enhance ETI activation, or stack multiple resistance specificities within a single plant genome (16).

Synthetic microbial communities (SynComs): Defined, reproducible consortia of selected microbial strains assembled under controlled conditions to recapitulate specific microbiome functions, including immune priming, pathogen suppression and nutrient cycling, for field application (17).

Review rationale, novelty and knowledge gaps

Despite remarkable advances in plant microbiome research over the past decade, critical knowledge gaps persist at the intersection of microbiome ecology and immunology. While substantial literature documents the association between specific microbial taxa and improved plant health outcomes, the mechanistic understanding of how microbial community structure translates into quantifiable immune outputs remains incomplete (18, 19). Furthermore, most existing reviews treat microbiome ecology and plant immunity as parallel disciplines rather than as integrated, bidirectionally interacting systems (20).

This review addresses this gap by synthesising current knowledge across four interconnected dimensions: (i) the molecular mechanisms through which plant-associated microbiomes modulate pattern-triggered immunity (PTI), effector-triggered immunity (ETI), systemic acquired resistance (SAR) and induced systemic resistance (ISR); (ii) pathogen strategies for immune evasion, effector deployment and microbiome manipulation; (iii) environmental drivers of microbiome composition and immune competence; and (iv) translational applications of microbiome-based technologies for sustainable disease management.

A particular novelty of this review is the explicit integration of the pathobiome concept with a critical reassessment of Koch's postulates, the examination of salicylic acid-jasmonic acid (SA-JA)

signalling crosstalk controversies and the rigorous evaluation of SynCom technologies from laboratory proof-of-concept to field-scale implementation. Understanding how the plant microbiome shapes immune responses is not merely of academic interest; it offers transformative prospects for developing biologically based, environmentally sustainable and climate-resilient alternatives to chemical pesticides (21, 22).

The plant microbiome: Composition, assembly and functional ecology

Rhizosphere, phyllosphere and endosphere communities

The compositional architecture of the plant microbiome is organised into spatially distinct compartments, each with characteristic microbial assemblages. The rhizosphere microbiome, dominated by Proteobacteria, Actinobacteria, Firmicutes and Bacteroidetes, is primarily assembled through the selective enrichment of soil microbial pools by root exudate chemistry, including organic acids, sugars, amino acids and secondary metabolites (23, 24). Beneficial rhizobacteria from genera including *Pseudomonas*, *Bacillus*, *Streptomyces*, *Trichoderma* and *Rhizobium* are functionally critical members of plant-protective microbiome consortia (25).

The phyllosphere, comprising leaf surfaces, stems and flowers, supports a distinct and comparatively lower-biomass microbial community dominated by Proteobacteria, Firmicutes and Actinobacteria, with fungal communities including *Cladosporium*, *Alternaria* and *Epicoccum* species (26). The endosphere harbours a more phylogenetically restricted microbial community that has successfully breached plant surface barriers, primarily comprising Proteobacteria and Actinobacteria capable of endophytic colonisation without eliciting detectable immune activation (5).

Factors governing microbiome assembly

Microbiome assembly is governed by a hierarchical combination of factors, including (i) soil type and physicochemical properties, (ii) plant genotype and developmental stage, (iii) root exudate composition, (iv) climatic conditions, (v) agricultural management practices and (vi) existing microbial community dynamics (27, 28). Plant immune signalling plays an underappreciated role in microbiome assembly, as mutants deficient in SA or JA signalling display altered rhizosphere microbiome composition, demonstrating that host immunity and microbiome structure are bidirectionally coupled (29).

Microbiome-mediated modulation of plant immune responses

Priming of pattern-triggered immunity

The capacity of plant-associated microbiomes to modulate host immunity operates through multiple, partially overlapping mechanisms. Beneficial rhizobacteria and mycorrhizal fungi prime plant PTI by exposing PRRs to sub-threshold concentrations of MAMPs during non-pathogenic colonisation, establishing a sensitised immune state that enables faster and stronger responses upon subsequent pathogen encounters, a phenomenon termed immune priming (30, 31).

For example, flagellin from *Pseudomonas fluorescens* WCS417r is recognised by the FLS2-BAK1 receptor complex to activate PTI-associated signalling cascades; however, colonisation-competent strains have evolved mechanisms to limit full PTI

activation, enabling rhizosphere persistence while maintaining priming competence (32). Similarly, chitin fragments released from mycorrhizal fungal cell walls activate chitin elicitor receptor kinase 1 (CERK1)-mediated immune signalling at concentrations sufficient to prime immunity without triggering growth-inhibitory defence responses (33).

Microbiome-derived volatile organic compounds and immune activation

In addition to MAMP-mediated priming, plant-associated microorganisms produce a diverse repertoire of volatile organic compounds (VOCs), including 2,3-butanediol, acetoin, dimethyl disulfide and geosmin, which activate plant immune signalling through receptor-mediated and epigenetic mechanisms (34, 35). *Bacillus subtilis* GB03-derived 2,3-butanediol activates ISR in *Arabidopsis thaliana* through an SA-independent, NPR3/NPR4-independent pathway, demonstrating that VOC-mediated immune activation operates through distinct molecular circuits from classical SAR (36).

Recent metabolomic and transcriptomic analyses have revealed that VOC-exposed plants display broad transcriptional reprogramming of defence gene networks, including the upregulation of pathogenesis-related (PR) proteins, WRKY transcription factors and JA-biosynthetic genes, collectively establishing a systemic primed state (37, 38).

Microbiome effects on effector-triggered immunity (ETI) and nucleotide-binding leucine-rich repeat (NLR) activation

Emerging evidence indicates that the microbiome also modulates ETI, although the mechanistic basis remains less well characterised than that of PTI. Certain endophytic bacteria have been shown to potentiate NLR-mediated resistance against *Pseudomonas syringae* in *A. thaliana*, with transcriptomic analyses revealing the upregulation of *TIR-NLR* gene clusters and associated helper NLRs (*ADR1* and *NRG1*) in microbiome-colonised plants (39). This suggests that the microbiome may modulate the threshold sensitivity of ETI activation, potentially through epigenetic modification of NLR gene expression or its effects on the proteostasis machinery governing NLR protein accumulation (40).

Pathogen-mediated immune suppression

Effector-mediated suppression

Successful plant pathogens have co-evolved sophisticated molecular arsenals to suppress, subvert and circumvent plant immune responses (41). Pathogen-secreted effector proteins, delivered into host cells via type III secretion systems (bacteria) or haustoria (fungi and oomycetes), collectively constitute the effectorome, whose primary function is to suppress PTI and ETI to facilitate pathogen colonisation (42).

Bacterial effectors, such as AvrPto and AvrPtoB from *P. syringae*, directly inhibit flagellin-sensing 2 (FLS2)–BRI1-associated receptor kinase 1 (BAK1) complex assembly and PRR kinase activity, thereby blocking MAMP-triggered signalling at the receptor level (43). HopA1 irreversibly inactivates MAP kinases *MPK3*, *MPK4* and *MPK6* through phosphothreonine lyase activity, disrupting the MAPK cascade that is central to PTI signal transduction (44). Oomycete RXLR effectors, exemplified by AVR3a from *Phytophthora infestans*, stabilise the U-box E3 ubiquitin ligase CMPG1 to suppress the hypersensitive response while simultaneously interfering with PAMP-triggered reactive oxygen species (ROS) bursts (45).

The concept of effectormics, which involves the systematic genome-wide identification and functional characterisation of pathogen effector repertoires, has transformed resistance breeding and molecular plant pathology (15, 46). By combining bioinformatics-based effector prediction with yeast two-hybrid, co-immunoprecipitation and structural proteomics approaches, researchers can now map effector-host target interaction networks at a near-proteome scale, enabling the rational design of engineered NLR receptors (NLR engineering) capable of recognising novel effector variants (16, 47).

Hormonal manipulation by pathogens

In addition to direct effector-mediated PTI suppression, many pathogens manipulate plant hormone signalling to create physiological environments conducive to colonisation (48). *Pseudomonas syringae* produces coronatine, a structural and functional mimic of jasmonoyl-isoleucine (JA-Ile), which activates the JA signalling pathway and simultaneously suppresses SA-mediated defence through COI1-JAZ-MYC2 complex-mediated transcriptional reprogramming, effectively exploiting the endogenous SA-JA antagonism to disarm SA-dependent immunity (49, 50).

Gibberellin-producing fungal pathogens, including *Fusarium fujikuroi*, manipulate host developmental programs and suppress JA-mediated defences through DELLA protein degradation, whereas cytokinin-secreting pathogens delay senescence to maintain green island nutritional zones (51). Manipulation of ethylene signalling by *Botrytis cinerea* exploits host ET signalling to accelerate lesion development and facilitate necrotrophic colonisation (52). These hormone mimicry and manipulation strategies underscore the sophisticated co-evolutionary arms race between pathogens and plant immune networks (53).

Microbiome restructuring and dysbiosis

Beyond direct immune suppression, pathogens strategically reshape the composition of host-associated microbial communities, inducing dysbiosis to create microbiome states that favour pathogen proliferation and suppress the recruitment of beneficial microorganisms (9, 54). This pathobiome-associated restructuring represents a critical but historically underappreciated dimension of plant diseases (10).

For example, *Fusarium oxysporum* infection significantly depletes *Pseudomonas* and *Bacillus* populations from the tomato rhizosphere while enriching opportunistic taxa from the Oxalobacteraceae family, effectively dismantling the microbiome-based suppressive capacity of the rhizosphere (55). Similarly, *Phytophthora parasitica* infection alters the avocado rhizosphere communities by reducing the population of Actinobacteria, which are well-recognised producers of antifungal secondary metabolites, thereby compromising the biological buffering capacity of the soil microbiome (56).

Dysbiosis-driven microbiome restructuring amplifies pathogen fitness by simultaneously suppressing beneficial microbial competitors and dismantling the collective contribution of the microbiome to plant immune priming (57) (Fig. 1).

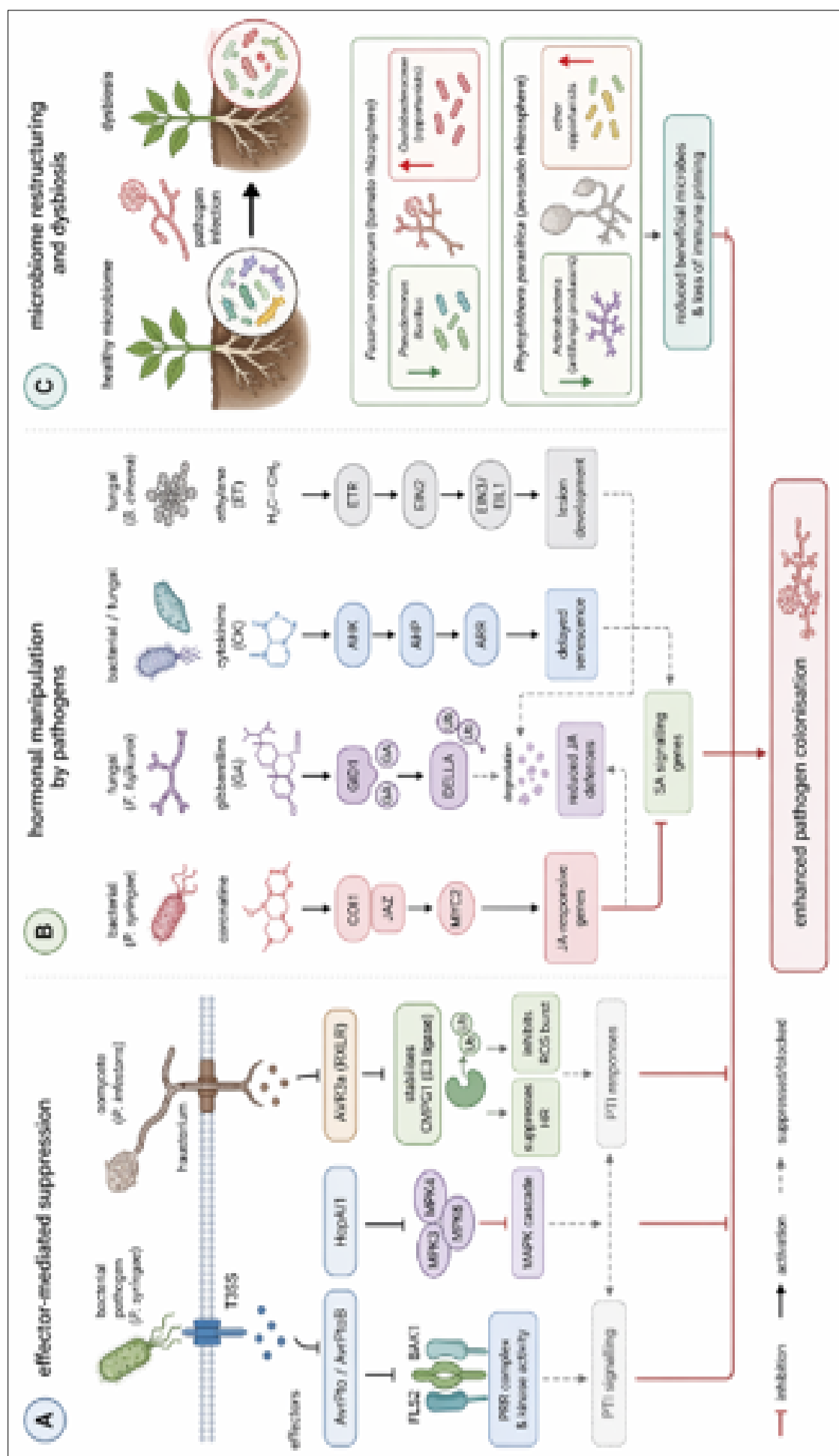


Fig. 1. Mechanisms of pathogen-mediated immune suppression and microbiome dysbiosis. (A) Effector-mediated suppression: Bacterial and oomycete pathogens deliver effectors (e.g., AvrPto, HopA1, AVR3a) via Type III Secretion Systems (T3SS) or haustoria to inhibit PRR complexes, MAPK cascades and ROS bursts, thereby blocking pattern-triggered immunity (PTI); (B) Hormonal manipulation: Pathogens secrete hormone mimics (e.g., coronatine, gibberellins) or modulate endogenous phytohormone pathways (JA, GA, CK, ET) to suppress salicylic acid (SA)-dependent defences and promote susceptibility; (C) Microbiome restructuring and dysbiosis: Pathogen infection shifts the rhizosphere from a healthy microbiome to a dysbiotic state, characterised by the depletion of beneficial taxa (e.g., *Pseudomonas*, *Bacillus*, *Actinobacteria*) and the enrichment of opportunistic pathogens, leading to a loss of immune priming. Collectively, these strategies converge to enhance pathogen colonisation. Symbols: Red bars, inhibition; solid black arrows, activation; dashed arrows, suppressed/blocked pathways.

Immune signalling networks

The salicylic acid pathway and systemic acquired resistance

Salicylic acid (SA) is the central signalling molecule mediating SAR, a durable, broad-spectrum resistance state induced systemically throughout the plant following localised pathogen infection (58, 59). Salicylic acid biosynthesis proceeds primarily through the isochorismate synthase (ICS) pathway in chloroplasts, which is catalysed by *ICS1* (*SID2* in *Arabidopsis*), which converts chorismate to isochorismate, which is subsequently converted to SA by isochorismate pyruvate lyase (IPL) (60).

The master regulator of SA signalling is NPR1 (NONEXPRESSER OF PR GENES 1), which functions as a transcriptional coactivator and SA receptor (61). At basal SA concentrations, NPR1 predominantly exists as an oligomeric, disulfide-bonded, inactive complex that is sequestered in the cytoplasm. Upon pathogen infection and SA accumulation, cellular redox changes mediated by thioredoxins (TRX-h5 and TRX-h3) reduce the intermolecular disulfide bonds, releasing NPR1 monomers that translocate to the nucleus (62). Nuclear NPR1 interacts with TGA transcription factors to activate PR gene expression, including PR-1, PR-2 and PR-5 and simultaneously promotes the expression of SAR-associated genes in systemic tissues (63).

The mobile SAR signal, which has long been debated, is now understood to involve multiple chemical messengers acting in concert, including glycerol-3-phosphate (G3P), azelaic acid (AzA), dehydroabietinal (DA), pipecolic acid (Pip) and N-hydroxypipecolic acid (NHP) (64, 65). N-hydroxypipecolic acid, biosynthesised through *FMO1*-catalysed N-hydroxylation of Pip, has emerged as a particularly potent and evolutionarily conserved SAR signal and its exogenous application is sufficient to induce broad-spectrum resistance, independent of SA (66). The SAR establishment requires a functional Pip/NHP amplification loop, phloem translocation of mobile signals and NPR1-dependent transcriptional reprogramming in the systemic tissues (67).

Salicylic acid-mediated SAR provides durable resistance against biotrophic pathogens, including *Hyalospora arabidopsidis*, *Blumeria graminis* and *P. syringae*, which depend on living host tissue for their nutritional requirements and are therefore particularly vulnerable to SA-induced defence activation (68) (Fig. 2; Table 1).

Jasmonic acid and ethylene pathways in induced systemic resistance (ISR)

Induced systemic resistance (ISR), which is primed by beneficial rhizosphere microorganisms, primarily operates through JA and ethylene (ET) signalling, largely independently of SA accumulation and PR gene induction (69, 70). The ISR was first characterised in *A. thaliana* colonised by *P. fluorescens* WCS417r, which conferred systemic resistance against *P. syringae* pv. *tomato* DC3000 through a mechanism requiring functional JA and ET signalling components, but not *NahG*-suppressed SA accumulation (71) (Table 1).

The molecular mechanism of ISR involves (i) root perception of MAMP signals from beneficial rhizobacteria, triggering localised PTI-like responses that are rapidly dampened to permit colonisation; (ii) generation of a systemic signal, putatively involving JA, ACC (1-aminocyclopropane-1-carboxylate) and microbial VOCs transmitted through the vascular system; (iii) NPR1-dependent but SA-independent sensitisation of JA/ET-responsive gene expression in shoot tissues; (iv) enhanced responsiveness of MYC2 and ERF transcription factors to subsequent pathogen challenge; and (v) primed activation of JAZ-regulated defence genes, Plant Defensin 1.2 (PDF1.2) and Vegetative storage protein (VSP2) upon pathogen encounter (72, 73).

Microbial VOC perception represents an important but mechanistically under-characterised dimension of ISR. 2,3-butanediol from *Bacillus* spp. is perceived by plants through a mechanism potentially involving cytoplasmic receptor proteins or membrane-associated receptor-like kinases, activating downstream MKK9-MPK3/6 MAPK cascades and subsequently, JA-dependent gene expression (74). For example, dimethyl disulfide (DMDS) from *Bacillus cereus* C1L activates ISR in tomatoes against *Botrytis cinerea* through ET-dependent signalling, whereas *Trichoderma virens* Sm1 protein activates ISR through an SA-dependent pathway in maize, demonstrating considerable mechanistic diversity across plant-microbe systems (75, 76) (Fig. 3; Table 2).

Salicylic acid-jasmonic acid (SA-JA) crosstalk: Antagonism, synergism and current controversies

The interaction between the SA and JA signalling pathways constitutes one of the most extensively studied and contextually complex regulatory relationships in plant immunity (82).

Table 1. Comparison of induced systemic resistance (ISR) and systemic acquired resistance (SAR)

Parameter	ISR	SAR
Trigger	Beneficial rhizobacteria and mycorrhizal fungi; VOCs; MAMPs	Pathogen infection; necrotising agents; chemical inducers (BTH)
Primary signal molecule	JA, ET, ACC	Salicylic acid (SA)
Key regulators	NPR1, MYC2, ERF1, ORA59, JAZ	NPR1, TGA factors, WRKY18/40/60
Mobile signals	JA, ACC, VOCs (putative)	Pip, NHP, G3P, AzA, DA
Defence gene markers	PDF1.2, VSP2, LOX2	PR-1, PR-2, PR-5
Effective against	Necrotrophic fungi, bacteria, herbivores	Biotrophic/hemibiotrophic pathogens, bacteria, viruses
SA dependency	SA-independent (NPR1-dependent)	SA-dependent
PR gene induction	Not directly induced (primed)	Directly induced
Associated epigenetic changes	H3K4me3, H3K9ac at JA gene loci	H3K4me3, H3K27me3 dynamics at PR loci
Advantages	No growth penalty; triggered by non-pathogens; compatible with beneficial microbiome	Broad-spectrum; durable; well-characterised
Limitations	Context-dependent efficacy; JA-SA antagonism constrains biotrophic resistance	SA-JA antagonism compromises necrotrophic resistance; growth-defence trade-offs

ISR: induced systemic resistance; SAR: systemic acquired resistance; VSR: virus-induced systemic resistance; MAMP: microbe-associated molecular pattern; JA: jasmonic acid; ACC: 1-aminocyclopropane-1-carboxylic acid; VOC: volatile organic compounds; SA: salicylic acid; BTH: benzothiadiazole; PR: pathogenesis-related.

Table 2. Key microbiome taxa and their immune-modulating functions

Taxon	Compartment	Mechanism of immune modulation	Activated pathway	Target pathogens	Key references
<i>Rhizophagus irregularis</i>	Endosphere (AM)	Myc-factor priming, ETI modulation	JA, SA	<i>Phytophthora</i> spp.	(33)
<i>Bacillus subtilis</i> GB03	Rhizosphere	2,3-Butanediol VOC, ISR	JA/ET-independent	Multiple pathogens	(36)
<i>Pseudomonas fluorescens</i> WCS417r	Rhizosphere	MAMP priming, ISR induction	JA/ET (NPR1-dep.)	<i>P. syringae</i> , <i>F. oxysporum</i>	(71)
<i>Trichoderma harzianum</i>	Rhizosphere/endosphere	Enzyme-mediated lysis, ISR	SA + JA	<i>Rhizoctonia</i> , <i>Sclerotinia</i>	(75)
<i>Trichoderma virens</i> Sm1	Rhizosphere	Sm1 protein-mediated ISR	SA-dependent (maize)	<i>Pythium</i> spp.	(76)
<i>Serratia plymuthica</i> HRO-C48	Rhizosphere	Chitinase, pyrrolnitrin	Direct antibiosis	<i>Sclerotinia sclerotiorum</i>	(77)
<i>Streptomyces</i> spp.	Rhizosphere	Antibiosis (antifungals), ISR	SA/JA	Soil-borne fungi	(78)
<i>Bacillus subtilis</i> QST713	Rhizosphere/phylosphere	Lipopeptide antibiosis + ISR	JA/ET	<i>Botrytis</i> , <i>Fusarium</i>	(79)
<i>Paenibacillus polymyxa</i>	Rhizosphere	Paenibactin siderophore, ISR	JA/ET	<i>Fusarium</i> , bacteria	(80)
<i>Sebacinales</i> fungi	Endosphere	Broad-spectrum ISR	SA-independent	Multiple pathogens	(81)

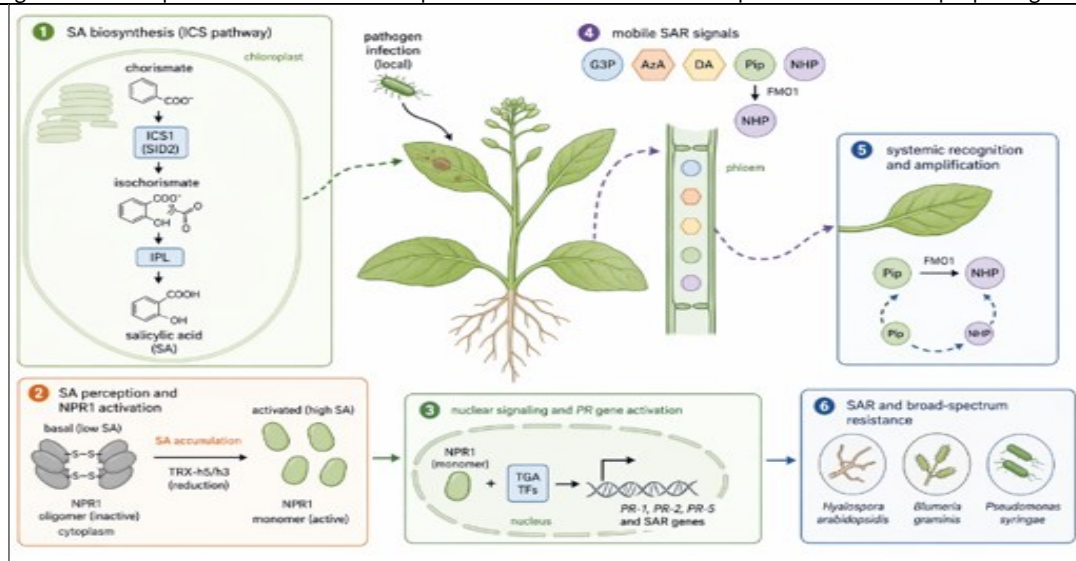


Fig. 2. Salicylic acid (SA) biosynthesis and systemic acquired resistance (SAR) signalling. (1–3) Local pathogen infection triggers chloroplastic SA biosynthesis via ICS1. Accumulated SA promotes TRX-mediated reduction of cytosolic NPR1 oligomers into active monomers, which translocate to the nucleus to activate PR genes via TGA transcription factors; (4–5) Mobile signals (G3P, AzA, DA, Pip) travel via the phloem, where Pip is converted to NHP by FMO1 to amplify the systemic signal; (6) This cascade establishes broad-spectrum resistance against diverse biotrophic and hemibiotrophic pathogens.

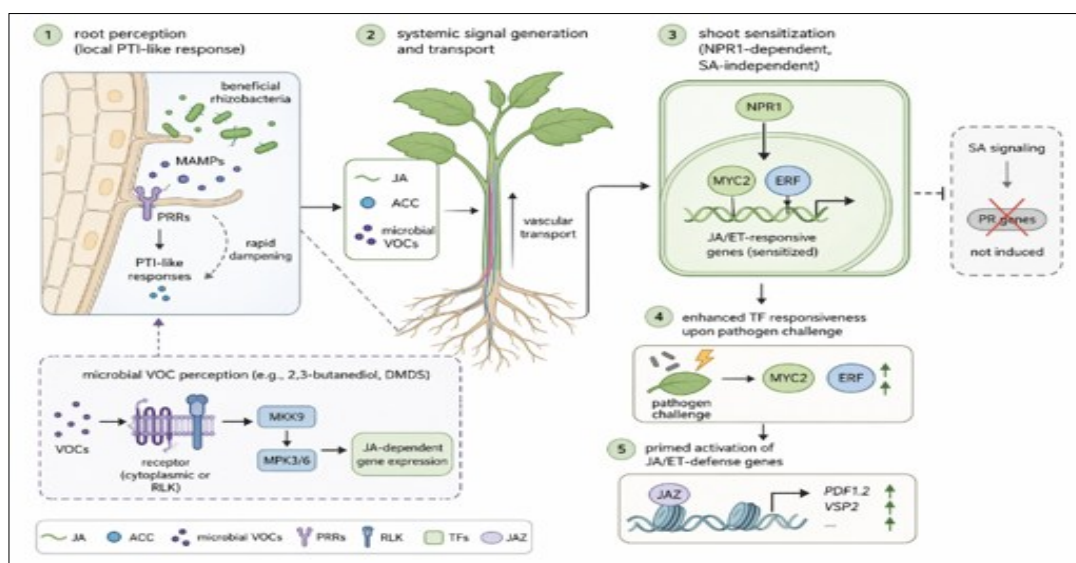


Fig. 3. Salicylic acid-jasmonic acid (SA-JA) crosstalk network regulating defence specificity. (1) SA–JA antagonism: SA suppresses JA-responsive genes via NPR1/WRKY62/70 induction and JAZ stabilisation, while JA reciprocally suppresses SA biosynthesis via MYC2-mediated repression of ICS1; (2) SA–JA synergism: At low SA concentrations or in specific tissue contexts, SA potentiates rather than suppresses JA-responsive gene expression; (3) JA–ET interaction: ET-dependent activation of the ERF branch drives necrotrophic resistance (e.g., PDF1.2), whereas ET-independent MYC2 signalling mediates ISR and herbivore defence.

Salicylic acid-jasmonic acid (SA-JA) antagonism

Salicylic acid-jasmonic acid (SA-JA) antagonism, the reciprocal suppression of SA-mediated biotrophic resistance by JA signalling and of JA-mediated necrotrophic/herbivore resistance by SA signalling, is well established and serves as the primary mechanism exploited by pathogens to manipulate host immunity (83, 84). The molecular basis of SA-mediated JA suppression involves the following:

1. NPR1-dependent induction of WRKY62 and WRKY70, which act as transcriptional repressors of JA-responsive genes.
2. SA-induced stabilisation of the JAZ repressor proteins through interference with COI1-mediated JAZ ubiquitination and degradation.
3. GRX480/ROXY19-mediated suppression of TGA-activated ORA59 expression, disrupting ET/JA-dependent PDF1.2 activation (85, 86).

Reciprocally, JA-mediated suppression of SA signalling involves MYC2-dependent activation of JA carboxyl methyltransferase (JMT) and suppression of *ICS1* expression, effectively limiting SA biosynthetic flux (87).

Salicylic acid-jasmonic acid (SA-JA) synergism

While antagonism has dominated the conceptual framework, emerging evidence demonstrates that SA and JA signalling can act synergistically under specific conditions, particularly at low concentrations or in specific tissue contexts (88, 89). Low-level SA has been shown to potentiate JA-responsive gene expression

rather than suppress it, with the switch between synergism and antagonism determined by the relative concentrations, timing and spatial distribution of the two signals (90). This concentration dependency creates a regulatory landscape of considerable complexity, where the same hormonal signals can produce opposing outcomes depending on the plant's physiological state and pathogen challenge context (91).

Jasmonic acid- ethylene (JA-ET) interaction and defence specificity

The interaction between JA and ET signalling defines the specificity of ISR and necrotrophic pathogen resistance (92). The JA and ET act synergistically to activate the ethylene response factor (ERF) branch of JA signalling, particularly ERF1 and ORA59, which drive the expression of PDF1.2 and other antimicrobial defensins that are effective against necrotrophic pathogens, including *Botrytis cinerea* and *Alternaria brassicicola* (93). In contrast, the MYC2 branch of JA signalling, independent of ET, mediates resistance against herbivores and some biotrophic pathogens and is preferentially activated during ISR by rhizobacteria (94). The balance between the ERF and MYC2 branches determines whether JA signalling is channelled toward ISR or wound/herbivory responses, with ET serving as a critical branch-point regulator (95) (Fig. 4).

The pathobiome concept and Koch's postulates revisited

Classical Koch's postulates and their limitations

The germ theory of infectious disease, formalised through Koch's postulates in the 19th century, established the foundational paradigm of single-pathogen causation: a specific microorganism

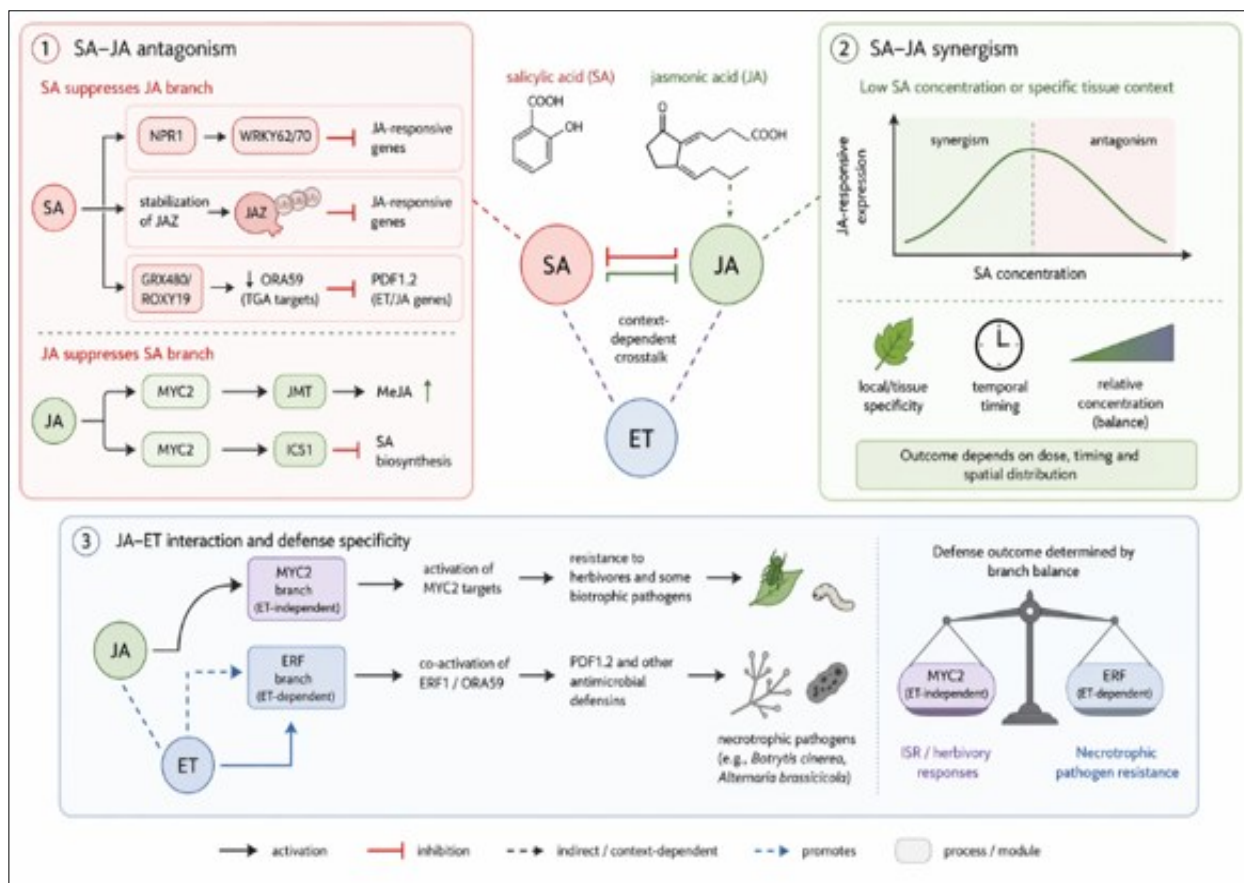


Fig. 4. Beneficial rhizobacteria-induced systemic resistance (ISR) signalling pathway. (1–2) Root colonisation by beneficial rhizobacteria triggers a dampened local PTI-like response and perception of microbial VOCs, generating systemic signals (JA, ACC, VOCs) that are transported to the shoots; (3–5) In leaves, NPR1 sensitises MYC2 and ERF transcription factors in an SA-independent manner. Upon pathogen challenge, these primed factors trigger rapid JAZ degradation and robust expression of JA/ET-responsive defence genes (e.g., *PDF1.2*, *VSP2*), conferring enhanced resistance without the metabolic cost of constitutive activation.

must be consistently associated with the disease, isolated in pure culture, capable of reproducing the disease upon inoculation of a healthy host and re-isolated from experimentally diseased tissue (96). This framework has been extraordinarily productive in identifying the causal agents of numerous infectious diseases and remains foundational to plant pathology diagnostic practices.

However, Koch's classical postulates have fundamental limitations when applied to complex microbiome-mediated disease syndromes (97, 98).

1. Many plant pathogens cannot be cultivated in pure culture (e.g., obligate biotrophs, phloem-limited bacteria and most plant viruses).
2. Disease severity frequently depends on the composition of the surrounding microbial community rather than on pathogen presence alone.
3. The same pathogen inoculated into hosts with different microbiome compositions produces markedly different disease outcomes.
4. Microbiome members that are individually non-pathogenic can collectively drive disease in a community context (synergistic pathogenesis).

Emerging infectious diseases increasingly involve polymicrobial consortia whose collective pathogenicity exceeds the sum of individual members (10, 99).

The pathobiome concept: Community-level disease causation

The pathobiome concept, emerging from metagenomic and network ecological analyses, reconceptualises plant disease as an ecological phenomenon driven by the entire microbial community network surrounding and within the host, rather than by a single causative agent (10, 11). The pathobiome encompasses (i) primary pathogens, microorganisms with established virulence mechanisms; (ii) opportunistic pathogens, normally non-pathogenic taxa that exploit host immune suppression; and (iii) microbiome amplifiers, taxa that promote pathogen fitness through resource liberation or toxin potentiation.

Dysbiosis-associated taxa: Microorganisms enriched during disease-associated microbiome restructuring (100). The pathobiome framework has been empirically validated in several plant disease models. Olive knot disease is primarily caused by *Pseudomonas savastanoi* pv. *savastanoi*, is significantly exacerbated by co-inhabiting *Erwinia toletana* and *Pantoea agglomerans*, which enhance bacterial colonisation, suppress host phenylpropanoid defences and promote biofilm formation, producing disease that cannot be replicated by single-species inoculation (101). Similarly, the replant disease complex in apple orchards involves synergistic interactions among *Pythium ultimum*, *Rhizoctonia solani* and dysbiosis-associated bacterial communities, which collectively overwhelm host immune responses (102).

Modern systems-based interpretation

A modern, systems-based reformulation of Koch's postulates, sometimes termed "ecological Koch's postulates" or the "microbiome causality framework," proposes that disease causation should be assessed at the community level (103). This framework requires (i) a consistent shift in microbiome composition associated with disease, (ii) transferability of disease susceptibility through microbiome transplantation into germ-free or microbiome-

depleted hosts, (iii) suppression of disease through restoration of healthy microbiome structure and (iv) identification of specific community-level properties (e.g., network topology, functional gene richness) that correlate with disease outcome (104). This community-level causality framework represents the conceptual foundation upon which pathobiome research and microbiome-based disease management strategies are increasingly being developed.

Molecular mechanisms of microbiome-mediated immune modulation

Immune recognition and pattern recognition receptors (PRR) signalling

Plant PRRs are transmembrane receptor proteins that survey the extracellular space for MAMPs. The two principal PRR classes are receptor-like kinases (RLKs), characterised by extracellular leucine-rich repeat (LRR) or lysin motif (LysM) domains coupled to intracellular kinase domains and receptor-like proteins (RLPs) that lack intrinsic kinase domains and require kinase-active co-receptors for signalling (105, 106).

The canonical FLS2-BAK1 complex mediates the perception of bacterial flagellin-derived flg22 peptide. Flagellin binding to FLS2 promotes rapid heterodimerisation with BAK1 (BRI1-Associated receptor kinase 1), enabling transphosphorylation of both receptors and activation of BIK1 (Botrytis-induced kinase 1) through phosphorylation (107). BIK1 subsequently phosphorylates NADPH oxidase RBOHD at Ser39 and Ser343, triggering an oxidative burst and phosphorylates SLAC1 anion channels, contributing to stomatal closure (108). Simultaneously, MAPK cascade activation through MKK4/5 and MPK3/6 drives transcriptional reprogramming via WRKY transcription factors, particularly WRKY22, WRKY29 and WRKY33 (109).

LysM-RLK CERK1 mediates chitin perception through an atypical receptor mechanism involving chitin-induced homodimerisation and transphosphorylation, activating overlapping but distinct downstream signalling components compared with FLS2 (110).

Metabolic competition and antibiosis

Microbiome-mediated pathogen suppression frequently operates through metabolic competition mechanisms that limit pathogen acquisition and growth (77). *Pseudomonas* spp. produces 2,4-diacetylphloroglucinol (2,4-DAPG), phenazines, pyoluteorin, pyrrolnitrin and siderophores (pyoverdines and pseudobactins) that inhibit soil-borne pathogens, including *Gaeumannomyces graminis* var. *tritici* (take-all disease of wheat) and *F. oxysporum*, through direct antibiotic mechanisms (78, 111). The suppression of take-all disease in long-term monoculture wheat soils, the phenomenon of take-all decline (TAD), is now understood to result from the natural enrichment of 2,4-DAPG-producing *P. fluorescens* through complex community-level succession dynamics triggered by disease-driven host metabolite release (112).

Bacillus spp. produces structurally diverse lipopeptides, including iturin A, surfactin, fengycin and bacillomycin, that disrupt fungal and oomycete membranes through detergent-like mechanisms while simultaneously activating plant ISR through MAMP and volatile organic compound (VOC) signalling (79) (Table 2). The dual functionality of *Bacillus* lipopeptides, direct pathogen suppression and host immune priming, makes them particularly attractive candidates for biocontrol formulations (80).

Transcriptional regulation of immune networks

Microbiome colonisation induces extensive transcriptional reprogramming of plant immune networks, characterised by epigenetic modifications that establish heritable, primed states (81). Beneficial rhizobacteria induce histone modifications, particularly H3K4me3 and H3K9ac marks at defence gene promoters, establishing transcriptionally permissive chromatin states that facilitate rapid defence gene activation upon pathogen challenge (113).

The WRKY transcription factors serve as central nodes that integrate microbiome-derived immune responses with pathogen recognition signals. WRKY18, WRKY40 and WRKY60 form a regulatory hub that modulates the balance between SA and JA signalling outputs, whereas WRKY33 mediates camalexin biosynthesis and necrotrophic resistance (114, 115). Microbiome-induced ethylene response factor (ERF) transcription factors regulate ISR-associated defensin gene expression and ethylene-dependent defence responses (116).

Reactive oxygen species (ROS) dynamics in microbiome-mediated immunity

Reactive oxygen species (ROS), particularly superoxide (O_2^-), hydrogen peroxide (H_2O_2) and hydroxyl radicals ($\bullet OH$), play multifunctional roles in plant immune signalling, serving simultaneously as direct antimicrobial agents, cell wall reinforcement mediators and systemic immune signal amplifiers (117).

The NADPH oxidase RBOHD generates a rapid apoplastic ROS burst during PTI, which is essential for stomatal closure, callose deposition and MAPK cascade amplification (118). Beneficial microorganisms induce moderate and sustained ROS production that differs kinetically and spatially from pathogen-induced oxidative bursts, potentially through differential regulation of the RBOHD phosphorylation state (119). Microbiome-produced antioxidant enzymes, such as catalases, peroxidases and superoxide dismutases, may also modulate the redox environment of the rhizosphere to influence plant immune thresholds (120).

The current understanding of ROS dynamics in microbiome-plant interactions is significantly constrained by the technical limitations of spatiotemporally resolved ROS measurements in intact root systems. Advances in genetically encoded H_2O_2 sensors (roGFP2-Orp1 and HyPer) and single-cell ROS imaging have begun to address these limitations (121).

Environmental factors shaping microbiome composition and immune function

Climate change and microbiome stability

Climate change, through concurrent shifts in temperature, precipitation patterns, atmospheric CO_2 concentrations and extreme weather event frequency, profoundly alters the composition, diversity and functional stability of plant microbiomes (122, 123). Elevated temperatures shift the rhizosphere bacterial community composition toward heat-tolerant taxa from Actinobacteria and Firmicutes, while reducing the populations of psychrotolerant *Pseudomonas* and *Flavobacterium* species with well-characterised biocontrol activities (124). This temperature-driven compositional shift may compromise the biological buffering capacity of agricultural soils against soil-borne pathogens under warming scenarios (125).

Elevated atmospheric CO_2 alters root exudate composition, particularly by increasing sucrose and decreasing organic acid secretion, with downstream consequences for rhizosphere microbial community structure and the representation of functional guilds involved in immune priming (126). Meta-analytic evidence indicates that elevated CO_2 conditions generally increase plant susceptibility to necrotrophic pathogens while reducing susceptibility to biotrophs, potentially through CO_2 -mediated modulation of the SA-JA signalling balance (127).

Drought stress and microbiome-mediated resilience

Drought is one of the most significant climate-related threats to global agricultural productivity and accumulating evidence demonstrates that the rhizosphere microbiome plays an active role in mediating plant drought tolerance through multiple mechanisms (128). Drought-associated soil drying selectively enriches taxa from Actinobacteria, particularly *Streptomyces* and *Arthrobacter* spp., which produce osmoprotectants, abscisic acid (ABA)-modulating compounds and auxin derivatives, contributing to root architecture remodelling and stomatal regulation (129).

Drought-induced shifts in the microbiome can influence plant immune competence. Abscisic acid, a central drought stress hormone, activates stomatal closure through guard cell signalling but simultaneously suppresses SA-mediated SAR and PTI signalling through multiple molecular mechanisms, creating a potential trade-off between abiotic stress tolerance and biotic disease resistance (130, 131). Certain drought-associated microbiome members mitigate this trade-off by activating ABA-independent, JA-dependent drought tolerance mechanisms that do not compromise immune signalling capacity (132).

Soil properties and agricultural intensification

Soil physicochemical properties, including pH, organic matter content, texture, cation exchange capacity and nutrient availability, exert dominant control over rhizosphere microbiome composition and the functional representation of immune-modulating taxa (133, 134). Disease-suppressive soils, characterised by their natural ability to limit soil-borne pathogen proliferation and disease development, are consistently associated with high microbial diversity, enrichment of specific biocontrol taxa and elevated representation of genes encoding secondary metabolite biosynthesis, siderophore production and competitive saprophytic ability (135).

Agricultural intensification through tillage, synthetic fertiliser application, monoculture cropping and pesticide use disrupts soil microbiome diversity and erodes biological disease suppression capacity (136, 137). Fungicide applications targeting soil-borne pathogens frequently reduce non-target beneficial fungal populations, including mycorrhizal fungi and *Trichoderma* spp., disproportionately relative to target pathogens, potentially exacerbating long-term disease vulnerability (138). Organic amendments, cover cropping, reduced tillage and crop rotation are evidence-based management interventions that restore microbiome diversity and biological suppression functions (139, 140).

Microbiome resilience and recovery

Microbiome resilience, the capacity to resist compositional disturbance and recover functional diversity following perturbation, varies substantially across agricultural systems, soil types and management histories (141). Highly diverse microbiomes with high functional redundancy demonstrate

greater resilience to agricultural perturbations, whereas simplified, low-diversity communities, common in intensively managed monoculture systems, are more susceptible to permanent compositional shifts following disturbances (142). Understanding and managing microbiome resilience is an emerging priority for sustainable agriculture in climate change scenarios (143).

Translational applications of plant microbiome research

Synthetic microbial communities (SynComs)

SynComs, defined consortia of selected microbial strains assembled to recapitulate specific microbiome functions, represent the most actively pursued translational application of plant microbiome research (17, 144). Unlike single-strain biocontrol inoculants, SynComs are designed to replicate the functional redundancy, community stability and ecological interactions characteristic of naturally protective microbiome consortia (145).

Field case studies and efficacy data: Significant field-level evidence supports the efficacy of SynCom approaches in multiple pathosystems.

1. A 7-member bacterial SynCom comprising *Pseudomonas*, *Bacillus* and *Streptomyces* strains reduced *Fusarium* crown rot incidence in wheat by 52–68 % across three field seasons, outperforming single-strain *B. subtilis* QST713 applications (32 % reduction) and approaching the efficacy of tebuconazole fungicide treatment (71 % reduction) without adverse soil microbiome effects (146) (Table 3).
2. A *Trichoderma*-dominant SynCom reduced *Sclerotinia sclerotiorum* incidence in soybean by 44 % in field trials, with efficacy correlated with SynCom establishment density in the

rhizosphere (147).

3. An ISR-optimised bacterial SynCom-designed to maximise complementary MAMP and VOC signalling profiles reduced Tomato spotted wilt virus (TSWV) incidence in greenhouse tomato by 38 % through priming of SA-independent resistance mechanisms (148).

Multi-kingdom SynComs incorporating bacterial and mycorrhizal fungal members demonstrated superior colonisation stability and disease suppression durability compared to bacteria-only formulations across varying soil types (149) (Table 4).

Scalability, formulation challenges and biosafety: Despite promising efficacy data, SynCom translation faces significant challenges (150, 151).

1. Scalability: Multi-strain fermentation requiring individually optimised growth conditions creates significant manufacturing complexity and cost.
2. Formulation stability: Maintaining viability and community composition ratios during storage, transport and application represents a critical technical challenge.
3. Ecological interactions: Competitive exclusion among SynCom members following field inoculation can shift community composition away from the designed formulation.
4. Soil compatibility: SynCom establishment and persistence vary substantially across soil types, pH ranges and existing microbiome compositions.
5. Biosafety: The ecological consequences of introducing

Table 3. Translational microbiome applications: Field performance and implementation challenges

Application	Example/Case Study	Field efficacy	Key challenges	Regulatory status	Commercial readiness
SynCom inoculants	7-member <i>Pseudomonas/Bacillus/Streptomyces</i> SynCom (wheat)	52–68 % <i>Fusarium</i> crown rot reduction	Formulation stability; soil compatibility; colonisation persistence	Emerging EU/US frameworks	Pre-commercial (TRL 5–6)
Single-strain biocontrol	<i>Bacillus subtilis</i> QST713 (Serenade®)	30–40 % disease reduction	Efficacy consistency; spectrum limitations	Commercially registered	Market available
Mycorrhizal inoculants	<i>Rhizophagus irregularis</i> (mycorrhizal products)	20–35 % yield benefit; variable disease reduction	Soil phosphorus competition; formulation	Registered in most jurisdictions	Commercially available
Biofumigation	<i>Brassica</i> green manures (apple replant)	Soil-borne pathogen reduction; microbiome restoration	Timing; biomass requirements; crop rotation fit	No regulatory approval required	Farmer-adoptable
Compost amendment	Suppressive compost (strawberry <i>Fusarium</i>)	31–45 % disease reduction; positive standardisation; ROI	Quality variability	Organic certification compatible	Commercially available
Microbiome-assisted breeding	Maize genotype × microbiome QTL mapping	Proof-of-concept (genotype effect confirmed)	G×E×M interactions; long breeding timelines	No specific framework	Research stage (TRL 2–3)
Microbiome restoration	Cover crop diversification (European wheat take-all)	Improved biological suppression (2–3 rotations)	Adoption barriers; transition costs; knowledge gaps	Standard agronomic practice	Farmer-adoptable

TRL: Technology readiness level (EU definition).

Table 4. Comparison of single-strain inoculants, synthetic microbial communities (SynComs) and chemical control

Parameter	Single-strain inoculants	SynComs	Chemical control
Efficacy consistency	Low-moderate	Moderate-high	High
Spectrum of activity	Narrow	Broad	Variable
Resistance risk	Low	Very low	High
Environmental impact	Minimal	Minimal	Significant
Cost per application	Low	Moderate-high	Low-moderate
Regulatory complexity	Low	High	Moderate
Field persistence	Variable	Improved	N/A

defined microbial consortia into diverse agricultural environments require comprehensive risk assessment.

The regulatory frameworks for SynCom approval vary substantially across different jurisdictions. In the European Union, multi-strain microbial products require individual strain safety assessments under Regulation (EC) No. 1107/2009, significantly increasing the regulatory burden compared to single-strain registrations (152). In the United States, the EPA registration requirements under FIFRA present analogous challenges for multi-strain formulations (153). Harmonising international regulatory frameworks for SynCom products is a significant policy priority for accelerating commercialisation (154).

Microbiome-assisted breeding

Microbiome-assisted breeding, which involves the integration of microbiome interaction traits as selection criteria in plant breeding programs, represents an emerging strategy for developing cultivars with an enhanced capacity to recruit, support and respond to beneficial microbiome members (155, 156).

Practical examples and breeding targets

1. *Arabidopsis* natural accession screens have identified genotype-dependent variation in rhizosphere microbiome recruitment, with specific quantitative trait loci (QTL) associated with differential enrichment of ISR-inducing *Pseudomonas* taxa, demonstrating a genetic basis for microbiome recruitment that is accessible to selective breeding (157).
2. Maize inbred line comparisons have revealed significant genotypic variation in rhizosphere microbiome composition, with lines carrying specific root architecture and exudate chemistry alleles recruiting distinctly different microbial communities with varying disease suppression capacities (158).
3. Root exudate chemistry particularly benzoxazinoid glucoside composition in wheat and maize has been identified as a heritable trait regulating recruitment of *Pseudomonas* biocontrol strains, representing a targetable biochemical breeding trait (159).
4. Stomatal density and aperture regulation alleles influence phyllosphere microbiome composition and pathogen entry, representing additional breeding targets for microbiome-mediated disease resistance (160).

Implementation limitations: Microbiome-assisted breeding faces substantial implementation challenges, including (i) genotype × environment × microbiome interactions that create context dependency that complicates trait selection across diverse production environments; (ii) complex and quantitative microbiome traits that are influenced by numerous loci with small individual effects, making marker-assisted selection technically demanding; (iii) field microbiome variability that confounds microbiome trait heritability estimates; (iv) regulatory frameworks for cultivar registration that do not currently accommodate microbiome interaction traits as recognised selection criteria; and (v) long breeding timelines (8–12 years for major crops) that create challenges for incorporating rapidly evolving microbiome science (161, 162).

Microbiome restoration strategies

Microbiome restoration, the deliberate re-establishment of diverse, functionally complete microbial communities in degraded

agricultural soils, is a promising but operationally complex disease management strategy (163, 164).

Field examples and economic feasibility

1. Compost-mediated restoration of suppressive microbiome function in fumigated strawberry soils—a field demonstration in California production systems reduced *Fusarium* crown and root rot incidence by 31–45 % across two seasons, with positive net economic return relative to methyl bromide alternatives (165).
2. Biofumigation using *Brassica* species green manures restored Actinobacteria populations and disease suppression capacity in replant-diseased apple orchard soils within a single growing season in New Zealand trials (166).
3. Mycorrhizal network restoration through reduced fungicide application and cover crop inclusion improved biological disease suppression against *Phytophthora* in avocado production in South Africa (167).
4. Cover crop diversification in European grain production systems increased rhizosphere microbiome diversity and enhanced biological suppression of take-all disease within 2–3 rotation cycles (139).

Practical adoption barriers

1. Economic: Short-term yield costs during transition periods create adoption barriers in price-competitive commodity production systems.
2. Knowledge: Farmer awareness and technical capacity for microbiome-based management remain limited.
3. Infrastructure: Absence of on-farm microbiome diagnostic tools prevents adaptive management responses.
4. Policy: Agricultural subsidy structures frequently incentivise input-intensive practices incompatible with microbiome restoration goals.
5. Temporal: Microbiome restoration requires multi-year commitment before measurable disease suppression benefits emerge.

Current limitations and future challenges

Metagenomic and sequencing limitations

Current microbiome research is technically constrained by significant methodological limitations that affect the reliability, reproducibility and ecological relevance of the findings (168, 169). Amplicon sequencing of the 16S rRNA gene (bacteria) or ITS region (fungi), the most widely employed microbiome profiling approach introduces multiple biases: the choice of primer pairs systematically excludes or underrepresents specific taxonomic groups; polymerase chain reaction (PCR) amplification introduces chimeric sequences and amplification bias among templates with different GC content; and the inherent inability of amplicon approaches to resolve functional gene diversity limits ecological interpretation (170, 171). Variable region selection (V3–V4, V4 and V3–V5) significantly affects community composition profiles, complicating cross-study comparisons (172).

Shotgun metagenomic sequencing addresses functional gene coverage but introduces distinct limitations, including host genome contamination in plant-associated samples, assembly challenges in complex communities and substantial computational resource requirements (173). Metatranscriptomic approaches,

which capture expressed microbial functions, face additional challenges in terms of RNA extraction and stability in soil matrices (174).

Causality and correlation problems

A pervasive challenge in plant microbiome research is distinguishing causative microbiome-immunity relationships from correlative associations (175). Most published microbiome-disease association studies are observational and identify microbiome compositional correlates of disease states without establishing mechanistic causality (176). Germ-free and gnotobiotic plant model systems, which enable controlled microbiome reconstitution experiments, offer the most rigorous causal frameworks but are technically demanding and ecologically simplified compared to field conditions (177).

SynCom reproducibility and colonisation persistence

SynCom research faces reproducibility challenges arising from batch-to-batch variations in community composition ratios during fermentation, strain-specific growth dynamics that alter the designed community structures upon application and context-dependent colonisation outcomes across diverse soil types and host genotypes (178, 179). Maintaining the designed SynCom community structure following field inoculation represents a critical unresolved challenge, as competitive interactions with indigenous microbiome members frequently displace SynCom constituents within weeks of application (179).

Regulatory and commercialisation barriers

The regulatory landscape for microbiome-based agricultural products is fragmented, inconsistently applied across jurisdictions and frequently ill-adapted to the biological complexity of multi-strain products (180). Specific barriers include: (i) the absence of standardised efficacy testing protocols for SynCom products; (ii) strain-by-strain safety assessment requirements that scale multiplicatively with SynCom complexity; (iii) intellectual property fragmentation across multi-institution SynCom development consortia; (iv) the absence of internationally harmonised definitions distinguishing SynComs from conventional biocontrol products; and (v) the limited market size for specialty microbiome products limiting commercial investment in regulatory development (154, 22).

Microbiome stability under field variability

Field microbiome variability, driven by spatiotemporal heterogeneity in soil physicochemical properties, climatic fluctuations, cropping system diversity and stochastic community assembly dynamics, creates substantial challenges in designing reproducible and scalable microbiome-based interventions (181). The ecological concept of microbiome stability, which encompasses resistance to disturbance and resilience following perturbation, requires quantitative frameworks applicable to agricultural contexts for rational microbiome management (141).

Conclusion

The plant microbiome represents far more than a passive ecological backdrop to plant development; it constitutes an active, dynamic and functionally essential component of the plant immune system, whose disruption increases disease vulnerability and whose strategic manipulation offers transformative prospects for sustainable agriculture. This review synthesises evidence

across multiple dimensions of plant microbiome-immunity interactions and yields the following major insights:

1. Plant immunity and microbiome composition are bidirectionally coupled through pattern-triggered immunity (PTI), effector-triggered immunity (ETI), systemic acquired resistance (SAR) and induced systemic resistance (ISR) signalling networks, with microbiome-derived microbe-associated molecular pattern (MAMP), volatile organic compounds (VOCs) and secondary metabolites priming host defences through mechanisms that are mechanistically distinct from but functionally complementary to pathogen-activated immunity.
2. Pathogens are not passive targets of host immunity; they actively suppress PTI and ETI through effector deployment, exploit endogenous salicylic acid-jasmonic acid (SA-JA) antagonism through hormonal mimicry and reshape the microbiome through dysbiosis induction emphasising that disease outcomes are determined by tripartite plant-microbiome-pathogen interactions.
3. The pathobiome concept provides a necessary extension of classical Koch's postulates, reframing plant disease as a community-level ecological phenomenon requiring community-level management responses.
4. Environmental factors particularly climate change, drought and agricultural intensification reshape microbiome composition in ways that compromise immune competence, creating urgent imperatives for developing microbiome-resilient agricultural systems.
5. Translational applications, particularly SynComs, microbiome-assisted breeding and microbiome restoration strategies, demonstrate measurable field efficacy but face significant scalability, regulatory and economic challenges that must be systematically addressed for widespread agricultural adoption.

Future research priorities include: (i) the development of spatiotemporally resolved, single-cell resolution tools for characterising plant-microbiome immune interactions in intact tissues; (ii) mechanistic elucidation of VOC receptor biology and epigenetic priming mechanisms; (iii) standardisation of SynCom formulation, efficacy testing and regulatory assessment frameworks; (iv) integration of microbiome interaction traits into genomic selection pipelines for major crops; (v) longitudinal field studies characterising microbiome stability and disease suppression function under climate change scenarios; and (vi) development of microbiome diagnostic toolkits to enable adaptive on-farm management.

The convergence of microbiome science, molecular plant pathology and precision agriculture creates an unprecedented opportunity to move beyond prophylactic chemical disease management toward ecologically informed, microbiome-centred approaches that align productivity with environmental sustainability. Realising this potential requires sustained interdisciplinary collaboration among plant pathologists, microbial ecologists, plant breeders, agricultural engineers, regulatory scientists and policymakers, united by the recognition that the plant microbiome is not merely a research frontier but an

agricultural resource of fundamental and growing importance in the face of climate change, food security challenges and the imperative to reduce reliance on synthetic agrochemicals.

Authors' contributions

ATD contributed towards design and prepared the initial draft of the manuscript. YS contributed to supervision, reviewing and editing. VMG have worked on acquisition, collection, compilation of literature for the present review. ATD and RLC have revised the manuscript after review. All authors have read and approved the final manuscript.

Compliance with ethical standards

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