

Crosstalk Between Systemic Acquired Resistance and Induced Systemic Resistance: Implications for Holistic Plant Immunity

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Abstract

Plants, as sessile life forms, are constantly exposed to a wide range of pathogenic threats, including bacteria, fungi, viruses, and nematodes. To survive in such a hostile environment, they developed a sophisticated and multilayered immune system. Systemic Acquired Resistance (SAR) and Induced Systemic Resistance (ISR) are two of the most extensively studied types of long-distance immunity in plants. While SAR is generally activated by localised biotrophic pathogen infection and is characterised by salicylic acid (SA) signalling and the activation of pathogenesis-related (PR) genes, ISR is typically triggered by beneficial root-associated microbes and is mediated by jasmonic acid (JA) and ethylene (ET) signalling pathways without the accumulation of PR proteins. This review provides a comprehensive overview of these two pivotal systemic immune responses, their signalling mechanisms, and their roles in the establishment of holistic plant immunity.

Key words: SAR, ISR, Jasmonic acid, Necrotrophic pathogens, Plant immunity.

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I. Introduction

Systemic acquired resistance (SAR) and induced systemic resistance (ISR) are two essential components of the plant's systemic immune system that serve as complementary defence strategies against a wide range of pathogens. They are the two arms of plant immunity. SAR is typically activated in response to localised infection by biotrophic pathogens and is linked to salicylic acid accumulation. This hormonal signal induces the expression of pathogenesis-related (PR) proteins, which increase resistance throughout the plant. One of SAR's distinguishing features is its ability to confer long-term immune memory, allowing the plant to respond more effectively to future attacks. In contrast, beneficial rhizosphere microbes, such as plant growth-promoting rhizobacteria, cause ISR by activating signalling pathways involving jasmonic acid (JA) and ethylene (ET). Unlike SAR, ISR does not involve the accumulation of PR proteins; rather, it prepares the plant to be on high alert. This primed state allows the plant to activate defence responses more quickly and robustly when faced with a pathogen challenge. Thus, while SAR relies on a constitutive defence mechanism after activation, ISR is a cost-effective strategy for increasing readiness without requiring ongoing energy investment in defence. Together, these systems enable plants to adapt their immune responses to various pathogens and environmental conditions.

Plants face continuous threats from diverse pathogens, and in response, they have developed multiple layers of defense. Two key long-distance immune responses are SAR and ISR. SAR is activated upon localized infection by biotrophic pathogens and leads to enhanced resistance throughout the plant, primarily via SA accumulation and expression of pathogenesis-related (PR) proteins (Durrant & Dong, 2004). ISR, on the other hand, is typically induced by non-pathogenic rhizobacteria and relies on JA and ET Signalling without the direct involvement of PR proteins (Pieterse *et al.*, 2014).

Every plant has an innate immune system that allows it to detect and respond to pathogen attacks. This basal immunity is the first line of defence for plants and has been conserved throughout evolution. It detects pathogen-associated molecular patterns (PAMPs) such as flagellin or chitin, which are recognised by pattern recognition receptors (PRRs) found on plant cell surfaces. This recognition triggers PAMP-triggered immunity (PTI), which initiates a series of defence responses such as reactive oxygen species (ROS) production, cell wall fortification, phytoalexin synthesis, and the expression of defense-related genes.

However, the magnitude and efficiency of the immune response vary among plant varieties. Resistant cultivars can mount a rapid and robust defense, effectively halting pathogen progression. In contrast, susceptible varieties may exhibit weaker or delayed responses, allowing the pathogen to colonize the tissue and cause disease. This variability is often governed by genetic differences in immune Signalling pathways, receptor abundance, or expression levels of defense genes.

Moreover, successful pathogens, particularly adapted or specialized ones, have evolved sophisticated mechanisms to overcome plant immunity. These pathogens may evade detection by either masking or modifying their PAMPs, thus escaping recognition by the plant's PRRs. For example, some bacteria can modify flagellin epitopes to avoid triggering PTI. Additionally, many pathogens produce effector proteins that are delivered into host cells via specialized secretion systems (e.g., Type III secretion system in bacteria). These effectors suppress host defense responses, such as inhibiting Signalling components of PTI or interfering with hormone Signalling pathways like salicylic acid (SA), jasmonic acid (JA), or ethylene (ET), which are critical for defense.

To combat this, some plants have developed a second layer of immunity known as effector-triggered immunity (ETI), which is mediated by resistance (R) proteins that recognise specific pathogen effectors directly or indirectly. ETI is typically more robust and frequently associated with a localised hypersensitive response (HR), a type of programmed cell death that limits pathogen spread.

Regardless of these defence layers, pathogens that can effectively suppress or bypass both PTI and ETI can cause infections and disease symptoms. Thus, the outcome of plant-pathogen interaction is determined by a complex interplay between the plant's ability to recognise and respond to invaders and the pathogen's ability to avoid or suppress these defences. Understanding these interactions is critical for creating disease-resistant crop varieties using molecular breeding or biotechnological approaches.

Plants lack an adaptive immune system similar to that found in animals, yet they have evolved a complex innate immune system comprising both local and systemic defense mechanisms. Upon pathogen recognition, local defenses such as the hypersensitive response (HR) are activated. However, plants also possess the remarkable ability to initiate systemic signals that travel from the site of infection to distant uninfected tissues, priming them for enhanced resistance. This long-distance Signalling manifests primarily through two distinct yet sometimes overlapping pathways: Systemic Acquired Resistance (SAR) and Induced Systemic Resistance (ISR) (Jones & Dangl, 2006).

Plants rely on an intricate immune system comprising physical, biochemical, and molecular defense mechanisms to perceive and counteract pathogenic threats. As illustrated in Figure 1, the immune response is initiated when plant cells recognize conserved pathogen-derived molecules known as pathogen-associated molecular patterns (PAMPs). These molecules, such as flagellin or chitin, are detected by pattern recognition receptors (PRRs) embedded in the plasma membrane, triggering a PAMP-triggered immunity (PTI) cascade (Boller & Felix, 2009).

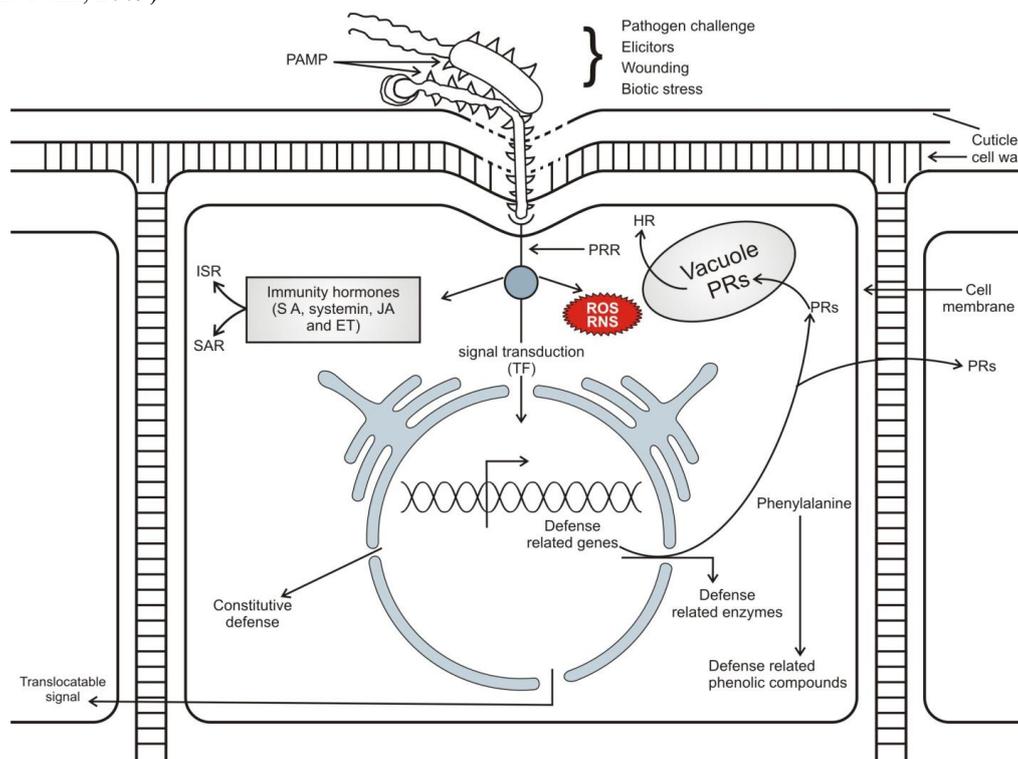


Figure-1: Initiation of immune response in plant cell

1.1 Physical Defense Mechanisms

The first line of defense involves physical barriers, such as the cuticle and cell wall, which prevent initial pathogen entry. Upon pathogen recognition, structural reinforcements are rapidly induced, including lignin deposition and cell wall remodeling, which act to limit pathogen progression and colonization (Underwood, 2012). These modifications are especially critical in halting necrotrophic pathogens and restricting vascular access.

1.2 Biochemical Defense Mechanisms

Biochemical responses are activated soon after PAMP perception. These include the generation of reactive oxygen species (ROS) and reactive nitrogen species (RNS)—Signalling molecules that serve both antimicrobial functions and as secondary messengers to amplify defense Signalling (Mur et al., 2013). Additionally, the phenylpropanoid pathway is activated, resulting in the production of phenolic compounds that strengthen cell walls and have direct antimicrobial activity (Lattanzio *et al.*, 2006).

Along with these, defense-related enzymes like chitinases, glucanases, and peroxidases are produced, which either degrade pathogen cell walls or help to fortify them. This response also promotes the accumulation of defence phytoalexins, a type of antimicrobial secondary metabolite that inhibits microbial growth.

1.3 Hormonal Signalling and Systemic Responses

Plant immunity is distinguished by its ability to send signals beyond the infection site via systemic responses, most notably Systemic Acquired Resistance (SAR) and Induced Systemic Resistance (ISR). These processes are regulated by distinct but interconnected hormonal pathways:

SAR is linked to the salicylic acid (SA) pathway and is frequently activated following a biotrophic pathogen attack. It increases the expression of pathogenesis-related (PR) proteins, which are stored in vacuoles and secreted into the apoplast for defence (Durrant & Dong, 2004).

ISR, which is activated by beneficial microbes such as *Pseudomonas fluorescens*, involves jasmonic acid (JA) and ethylene (ET) signalling. It does not typically induce PR proteins directly, but rather "primes" the plant for a faster and stronger response to pathogen attack (Pieterse et al., 2014).

The synergistic action of SA, JA, ET, and other immunity-related molecules like systemin, enhances the efficiency and breadth of the defense response (Wasternack & Hause, 2013).

1.4 Molecular Defense Regulation

At the molecular level, transcriptional reprogramming controls the immune response. The PAMP-PRR interaction starts a signal transduction cascade, which activates transcription factors (TFs) like WRKYs and TGAs. These transcription factors promote the expression of defense-related genes, such as those encoding PR proteins, biosynthetic enzymes, and secondary metabolite pathway components (Pandey & Somssich, 2009). Furthermore, systemic signalling employs mobile signals such as methyl salicylate, azelaic acid, and pipecolic acid, which facilitate communication between infected and uninfected tissues, priming distal cells for defence (Hartmann *et al.*, 2018).

1.5 Integrated Defense Execution

The coordinated interaction of all these mechanisms results in a robust immune response. For instance, following PRR activation and TF-mediated Signalling, PR proteins are synthesized in the endoplasmic reticulum, trafficked to vacuoles, and eventually secreted into the apoplast. Simultaneously, translocated systemic signals initiate SAR/ISR in distant tissues, conferring broad-spectrum resistance (Fu & Dong, 2013).

II. Mechanisms of Systemic Acquired Resistance (SAR)

Systemic acquired resistance (SAR) is an important component of the plant immune system, providing broad-spectrum and long-lasting resistance to a variety of pathogens after a localised infection. Unlike localised defence responses, which are limited to the site of infection, SAR increases resistance throughout the plant, including tissues that have not yet been exposed to the pathogen (Durrant & Dong, 2004). This systemic response is especially effective against biotrophic and hemibiotrophic pathogens and is a critical component of the plant's long-term immune memory.

SAR activation begins with a localised pathogen attack, which typically results in a hypersensitive response (HR) and localised cell death at the infection site. This local reaction is accompanied by the generation of mobile signalling molecules that travel through the plant's vascular system, activating defence pathways in uninfected tissues (Vlot, Dempsey, & Klessig, 2009). One of the most well-studied of these mobile signals is salicylic acid (SA), a phenolic phytohormone that accumulates both locally and systemically in response to infection.

Stemic Acquired Resistance (SAR) is an important aspect of plant immunity that provides long-term protection against a variety of pathogens after a primary infection. Bhooshan and Kumar, D. (2022) found that the

widespread presence of *Alternaria brassicae* in cauliflower fields in Agra highlighted the importance of improving plant defence mechanisms such as SAR. Their findings highlight the potential role of SAR in managing recurring infections, as understanding the pathogen's cultural characteristics is critical for eliciting appropriate resistance responses in host plants.

SA is important in SAR because it functions as both a local and systemic signal. Glazebrook (2005) found that activating pathogenesis-related (PR) genes, including PR-1, PR-2 (β -1,3-glucanase), and PR-5 (thaumatin-like protein), enhances plant resistance to infections. The protein NPR1 (Nonexpressor of PR genes 1), a transcriptional co-regulator that normally remains in the cytoplasm as an oligomer, is central to the SA signalling pathway. When a pathogen accumulates SA, NPR1 undergoes redox-mediated monomerization, allowing it to translocate to the nucleus and interact with TGA transcription factors to activate PR gene expression (Dong, 2004).

Beyond SA, recent studies have uncovered additional mobile signals that contribute to SAR Signalling. In this regard, two molecules stand out: azelaic acid (AzA) and pipelicolic acid (Pip). Azelaic acid, a nine-carbon dicarboxylic acid obtained through lipid peroxidation, has been identified as a priming agent that improves the plant's ability to respond to future infections. It acts upstream of SA accumulation and works together with SA to enhance SAR (Jung et al., 2009). Similarly, pipelicolic acid, a lysine-derived non-protein amino acid, has emerged as a key player in SAR signalling. It not only accumulates in distal tissues after local infection, but it also increases the SA response and PR gene expression (Hartmann et al., 2018). AzA and Pip work together to create a "primed state" in uninfected tissues, allowing defence mechanisms to be activated more quickly and effectively when a secondary pathogen is introduced.

Further research has shown that a volatile derivative of SA such as methyl salicylate (MeSA) may act as a long-distance airborne or phloem-mobile signal, which is converted back to active form of SA in systemic tissues by the esterase SABP2 (Salicylic acid-binding protein 2) (Park et al., 2007). This adds to the complexity and redundancy of the SAR signalling network, which uses multiple overlapping mechanisms to propagate and maintain systemic immunity. To summarise, SAR is a sophisticated defence strategy that includes local perception of pathogen invasion, generation of mobile signals, systemic signalling, and transcriptional reprogramming of defence genes in distal tissues (Bhooshan, B., & Kumar, D., 2023). SA signalling and NPR1-mediated activation of PR genes play central roles in this response, which is aided by additional priming molecules such as azelaic acid and pipelicolic acid. These findings not only improve our understanding of plant immune responses, but also provide valuable targets for breeding or engineering crops with increased pathogen resistance.

III. Mechanisms of Induced Systemic Resistance (ISR) in Plants: Microbial Induction and Hormonal Signalling

Induced Systemic Resistance (ISR) is an important type of plant immunity that is distinct from but complements Systemic Acquired Resistance (SAR). SAR is typically initiated by pathogenic infection and is dependent on salicylic acid (SA) signalling and pathogenesis-related (PR) gene expression, whereas ISR is usually activated by non-pathogenic, beneficial microbes in the rhizosphere. These include different strains of *Pseudomonas fluorescens*, *Bacillus subtilis*, and *Trichoderma* species that stimulate plant immune responses without causing disease symptoms (Van Wees *et al.*, 2008).

Instead of directly inducing defence responses, ISR activation is characterised by immune priming. Plants in this primed state do not accumulate defense-related transcripts or proteins on a continuous basis, but can mount faster and stronger responses to pathogen attack. This feature reduces the fitness costs associated with constitutive defence activation and is considered a sustainable strategy in agriculture (Pieterse *et al.*, 2014).

ISR is largely independent of SA signalling, relying instead on the jasmonic acid (JA) and ethylene (ET) pathways. Root cells recognise microbe-associated molecular patterns (MAMPs) or microbial elicitors, which initiate signalling cascades that activate downstream transcription factors and defense-related metabolites in distant, uninfected tissues. This systemic effect does not always involve the accumulation of PR proteins seen in SAR, but it does result in increased defence readiness (Zamioudis & Pieterse, 2012).

MYC2, a transcription factor that regulates JA-responsive genes, is an important component of ISR signalling. MYC2 and its homologs control a variety of JA-induced responses, including the expression of defense-related genes and the modulation of hormonal crosstalk between JA and other signalling pathways (Dombrecht *et al.*, 2007). MYC2 also coordinates ISR activation by controlling the expression of defence genes, which contribute to increased resistance to necrotrophic pathogens and herbivorous insects.

The activation of ISR by rhizobacteria does not involve a direct pathogenic interaction but rather relies on microbial metabolites, such as siderophores, lipopeptides, and volatile organic compounds. These microbial products act as elicitors that modulate host plant Signalling networks. For instance, *Pseudomonas fluorescens* WCS417 has been shown to elicit ISR through the perception of lipopolysaccharides and flagellin, which, while not leading to visible HR or SA accumulation, result in systemic immune sensitization (Van der Ent *et al.*, 2009).

Ethylene Signalling also plays an important modulatory role in ISR. Mutant plants defective in ethylene perception (e.g., *ein2*) fail to develop ISR despite successful colonization by beneficial microbes, underscoring the essential nature of ET in this response (Pieterse et al., 2000). JA and ET pathways often act synergistically in ISR to regulate a distinct set of defense genes, which are largely non-overlapping with the SA-regulated PR genes active in SAR.

To summarise, ISR is a strong and environmentally sustainable form of disease resistance mediated by beneficial microbes that prepare the plant for increased defence. It uses JA and ET signalling and transcriptional regulators like MYC2 to activate a variety of defences. Understanding the molecular mechanisms and key regulators of ISR opens up possibilities for developing biocontrol-based crop protection strategies that reduce reliance on chemical pesticides while improving plant health and productivity.

IV. Crosstalk Between SAR and ISR Pathways in Plant Immunity

Plants are continuously exposed to a wide array of biotic stresses, including pathogenic microbes and herbivores. To survive such environmental challenges, they have developed sophisticated immune systems that include both local and systemic responses. Systemic Acquired Resistance (SAR) and Induced Systemic Resistance (ISR) have historically been studied separately as systemic defence pathways. SAR is classically activated by biotrophic pathogens and is associated with the accumulation of salicylic acid (SA) and the expression of pathogenesis-related (PR) genes, whereas ISR is triggered by beneficial rhizobacteria and is primarily dependent on jasmonic acid (JA) and ethylene (ET) signalling, functioning without the direct involvement of PR proteins (Pieterse et al., 2014). However, emerging evidence suggests that the SAR and ISR pathways are not mutually exclusive, and that their signalling networks frequently converge or interact in complex ways—a phenomenon known as hormonal crosstalk.

One of the central players in this interaction is the Nonexpressor of PR Genes 1 (NPR1), which was first identified as a master regulator of SAR. NPR1 is activated when SA accumulates in the nucleus, where it promotes the transcription of PR genes. Surprisingly, NPR1 plays an important role in ISR, despite its independence from SA. ISR induced by non-pathogenic rhizobacteria is abolished in *npr1* mutant plants, implying that NPR1 functions as a common integrator of both SAR and ISR signalling pathways (Pieterse et al., 2009). This dual functionality of NPR1 emphasises its importance as a key convergence node in plant immune signalling.

The mechanistic basis for this crosstalk is rooted in the modulation of hormonal Signalling. Although SAR and ISR utilize distinct Signalling molecules, their responses are modulated by each other. For example, the presence of SA can antagonize JA Signalling in certain contexts, a phenomenon known as SA–JA antagonism. However, under ISR, the interplay becomes more nuanced, often resulting in synergistic rather than antagonistic effects, especially when both pathways are activated in a sequential or compatible manner (Van Wees et al., 2000).

In addition, transcription factors like WRKY70 and MYC2 contribute to regulatory overlap. WRKY70 is SA-inducible, promoting SAR while suppressing JA-dependent genes, whereas MYC2 is required for JA signalling and the expression of ISR-associated genes. Their balance determines whether SAR or ISR responses are dominant, depending on the stimulus (Verhagen et al., 2004).

The crosstalk is further complicated by the actions of pathogens and beneficial microbes that modulate host hormonal pathways. Pathogens may produce effectors that manipulate the SA-JA/ET balance to suppress host defense. For instance, *Pseudomonas syringae* effectors can activate JA Signalling to repress SA-mediated defenses, thereby enhancing susceptibility to biotrophic pathogens. Conversely, beneficial microbes such as *Pseudomonas fluorescens* may prime JA/ET Signalling to promote ISR while indirectly enhancing SAR through yet undefined mechanisms (De Vleeschauwer et al., 2013).

Recent research suggests that SAR and ISR may act synergistically under certain environmental conditions or microbial consortia, rather than simply additive. This opens up new possibilities for developing integrated disease management strategies that take advantage of both types of resistance for improved and sustainable crop protection. The interaction of SAR and ISR forms a dynamic and integrated network of hormonal signalling in plant immunity. NPR1 emerges as a critical point of convergence between these two pathways, where transcription factors and hormone signalling molecules influence the balance of defence responses. Understanding this crosstalk provides critical insights into the fine-tuning of plant immune responses, as well as promising opportunities for the development of biocontrol agents and resistant crop varieties via molecular breeding or biotechnological approaches.

In recent research, the interplay between Systemic Acquired Resistance (SAR) and Induced Systemic Resistance (ISR) has gained significant attention for its role in shaping comprehensive plant defense strategies. SAR is typically triggered by pathogenic attacks and is characterized by the accumulation of salicylic acid (SA) and the activation of pathogenesis-related (PR) proteins, while ISR is commonly initiated by beneficial microbes and involves jasmonic acid (JA) and ethylene (ET) signaling pathways. Bhooshan and Kumar (2024) demonstrated that both biotic and abiotic inducers, such as microbial extracts and chemical elicitors, can effectively stimulate defense responses in cauliflower against *Alternaria brassicae*. Their findings underscore the

potential synergy between SAR and ISR pathways, as both mechanisms contributed to enhanced resistance, suggesting that coordinated activation of these responses can provide robust and sustainable protection. This crosstalk not only broadens the spectrum of resistance but also reflects the complexity and adaptability of plant immune systems in managing diverse environmental challenges.

V. Molecular Mediators of Crosstalk

NPR1, WRKY transcription factors, and mitogen-activated protein kinases are key molecules involved in SAR-ISR crosstalk. Crosstalk enables the plant to prioritise responses based on the nature of the attack. Certain WRKY proteins, for example, have been shown to enhance or suppress SA and JA signalling depending on the context (Birkenbihl *et al.*, 2017). Such dynamic regulation ensures optimal defense while minimizing energy costs. WRKY transcription factors represent another class of molecular players that contribute significantly to the integration of SAR and ISR signals. The WRKY family is one of the largest transcription factor families in plants, and its members play a variety of roles in regulating defence gene expression, which are often context dependent. Some WRKY proteins, such as WRKY70, positively regulate SA-mediated defence while repressing JA-responsive genes, reinforcing SAR at the expense of ISR (Li *et al.*, 2004). Conversely, other WRKYs, like WRKY33, have been shown to activate JA- and ET-dependent genes and suppress SA Signalling, thereby favoring ISR (Birkenbihl *et al.*, 2017). This differential regulation reflects the flexibility of the plant immune system to modulate its response depending on the nature of the invading pathogen.

Mitogen-activated protein kinases (MAPKs) constitute another important Signalling component in plant immunity. These serine/threonine kinases transmit extracellular stimuli into intracellular responses via phosphorylation cascades and play essential roles in both SAR and ISR. MAPK cascades are involved in early Signalling events following pathogen recognition and influence downstream gene expression related to both SA and JA/ET pathways (Meng & Zhang, 2013). For instance, the activation of MPK3 and MPK6 in *Arabidopsis* has been associated with enhanced resistance responses mediated by both hormonal pathways. Thus, MAPKs act as early mediators that modulate transcriptional responses, contributing to the fine-tuning of immune crosstalk.

Crosstalk among these Signalling networks enables plants to prioritize or balance immune responses based on pathogen type, developmental stage, or environmental context. For example, antagonism between SA and JA Signalling helps the plant avoid unnecessary energy expenditure by preventing simultaneous activation of both pathways when only one is required. However, in certain cases, synergistic interactions may occur—particularly when plants face multiple stressors—allowing for a broad-spectrum and durable defense response (Kunkel & Brooks, 2002).

VI. Ecological and Agricultural Implications

The amalgamation of Systemic Acquired Resistance (SAR) and Induced Systemic Resistance (ISR) in plants has far-reaching ecological and agricultural implications, particularly in terms of sustainable crop protection and resource-efficient farming practices. SAR and ISR, despite being activated by different molecular mechanisms—salicylic acid (SA)-mediated for SAR and jasmonic acid (JA)/ethylene (ET)-mediated for ISR—can function synergistically when activated simultaneously. This dual activation enables plants to mount more robust and broad-spectrum defence responses against a wide variety of pathogens, including biotrophs, necrotrophs, and hemibiotrophs (Pieterse *et al.*, 2014). Knowing the integration and functional interplay of these two immune systems opens up possibilities for developing resilient crops that require fewer chemical inputs.

In conventional crop protection, reliance on synthetic pesticides has led to a number of ecological concerns including environmental contamination, development of resistant pathogen strains, and non-target effects on beneficial organisms. By contrast, activating endogenous plant defenses via SAR and ISR offers a more ecologically benign approach. However, inducing either SAR or ISR individually has limitations due to the specificity of their effectiveness; SAR is more effective against biotrophic pathogens, while ISR is typically more effective against necrotrophs and insect herbivores. Therefore, the simultaneous or sequential activation of both pathways may provide additive or synergistic protection across a wider range of pests and pathogens (Walters *et al.*, 2013).

One practical approach to dual activation is to combine SAR-inducing chemicals such as benzothiadiazole (BTH) or 2,6-dichloroisonicotinic acid (INA) with ISR-inducing biocontrol agents such as *Pseudomonas fluorescens* or *Trichoderma harzianum*. These treatments have been shown to induce both SA- and JA/ET-responsive genes, resulting in improved disease resistance in the field (Conrath *et al.*, 2006). Such integrative strategies have the added benefit of lowering pesticide use, improving soil health, and fostering beneficial microbial communities.

Furthermore, insights into SAR-ISR crosstalk have opened avenues in plant breeding. Selection for genotypes with enhanced responsiveness to both SAR and ISR inducers can lead to the development of cultivars with durable and broad-spectrum resistance. This is particularly valuable in integrated pest management (IPM) programs that aim to combine host resistance, biological control, and agronomic practices for sustainable

agriculture (Kumar et al., 2022). Marker-assisted selection (MAS) for key regulatory genes such as NPR1, WRKY70, and MYC2 that mediate SAR-ISR crosstalk may further facilitate the breeding of elite varieties with optimized immune responses (Fu & Dong, 2013).

From an ecological standpoint, the deployment of SAR-ISR-based immunity in crops minimizes negative impacts on beneficial insects, soil microflora, and surrounding ecosystems. Unlike conventional pesticides, SAR and ISR activation does not involve cytotoxic compounds, and therefore does not pose significant risk to non-target organisms. Moreover, resistance induced via SAR and ISR is systemic and long-lasting, often persisting throughout the life of the plant, thereby reducing the frequency of interventions needed during the cropping season (Walters *et al.*, 2013).

VII. Future Directions

The advancement of SAR and ISR research presents significant opportunities for improving plant immunity and enhancing sustainable crop protection. Despite the progress made in understanding their molecular frameworks, further exploration is required to decode the intricate, context-specific interactions between these two long-distance defense responses. The dynamic interplay between SAR and ISR is influenced by numerous variables including pathogen lifestyle, host genotype, environmental conditions, and microbial community composition. Thus, future research must aim to dissect these layers of complexity using innovative and integrative scientific tools.

One promising avenue is the application of multi-omics approaches—particularly transcriptomics, proteomics, metabolomics, and epigenomics—to elucidate the regulatory networks governing SAR and ISR. These technologies allow for a system-level understanding of the gene expression changes, protein-protein interactions, secondary metabolite profiles, and epigenetic modifications that occur in response to SAR and ISR activation (Spoel et al., 2009). For instance, transcriptomic profiling can help identify unique and overlapping gene sets involved in SA- and JA/ET-mediated pathways, while metabolomics can reveal Signalling intermediates and defense-related compounds that serve as biochemical markers of resistance (Hartmann et al., 2018). The integration of these datasets through systems biology can offer new insights into how plants prioritize defense responses under multiple stress conditions.

Another critical direction is the functional characterization and genetic manipulation of key regulatory genes involved in SAR and ISR Signalling. Genes such as NPR1, MYC2, WRKY70, and TGA transcription factors act as central nodes in defense Signalling crosstalk (Pieterse et al., 2014). With the advent of genome editing technologies, especially CRISPR-Cas systems, it is now possible to precisely edit these genes to enhance plant immunity without introducing foreign DNA, making it suitable for both conventional and organic agriculture (Borrelli et al., 2018). Targeted modifications of regulatory elements or gene promoters could enable fine-tuning of immune responses, potentially leading to disease-resistant cultivars with minimal fitness trade-offs.

Moreover, synthetic biology approaches could be employed to engineer custom immune pathways that combine the best features of both SAR and ISR. This could involve the design of synthetic promoters responsive to specific immune signals or the assembly of synthetic gene circuits that activate defense in a programmable manner. These approaches, while still in early stages, hold the promise of delivering next-generation crops with enhanced resilience against emerging pathogens in the face of climate change.

VIII. Conclusion

The crosstalk between SAR and ISR represents a remarkable facet of plant immunity, reflecting its complexity, plasticity, and evolutionary refinement. While SAR is predominantly driven by salicylic acid (SA) and involves the activation of pathogenesis-related (PR) genes, ISR relies on jasmonic acid (JA) and ethylene (ET) Signalling and operates via a priming mechanism. Though once considered distinct, accumulating evidence reveals significant overlap between these defense pathways, especially through shared molecular regulators such as NPR1, WRKY transcription factors, and mitogen-activated protein kinases (MAPKs) (Pieterse *et al.*, 2009; Birkenbihl *et al.*, 2017). This convergence allows plants to fine-tune their responses according to the type of invading pathogen or environmental stressor, prioritizing energy-efficient immunity while minimizing growth-defense trade-offs.

The molecular mechanisms underlying SAR-ISR crosstalk holds great promise for developing integrated disease management strategies. Combining biological inducers like beneficial rhizobacteria (for ISR) and chemical elicitors like benzothiadiazole (for SAR) can synergistically improve plant resistance (Walters *et al.*, 2013). Such dual activation approaches have the potential to provide broad-spectrum and long-term protection against a variety of biotic challenges. Furthermore, breeding or engineering crops with increased ability to activate both SAR and ISR pathways can reduce reliance on synthetic pesticides, promoting more sustainable and environmentally friendly agriculture (Kumar, D., 2020). To summarise, harnessing the interplay between SAR and ISR has enormous potential for improving plant resilience. Future research should concentrate on decoding this interplay at the system level, combining omics-based tools with genome editing technologies. This knowledge

can be applied to practical crop improvement programs, thereby contributing to global food security and sustainable agricultural development.

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