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# Induced Systemic Resistance in Plant

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## Abstract:

Plants respond to localized attacks by herbivores or various pathogens by producing compounds that reduce or prevent further damage by these threats and the spread of infection. These responses occur both at the site of the initial attack (local response) and in distant, unaffected parts of the plant (systemic response). Systemic resistance is classified into two types: Induced Systemic Resistance (ISR), which is activated in plants through inoculation with beneficial soil-dwelling microorganisms or their plant growth-regulating metabolites, including Plant Growth-Promoting Bacteria (PGPB) and Plant Growth-Promoting Fungi (PGPF), in addition to certain synthetic or biogenic chemical compounds. This type of resistance relies on the jasmonic acid (JA) and ethylene (ET) signaling pathways and typically leads to the production of non-specific defense proteins.

The other type is Systemic Acquired Resistance (SAR), which is triggered following pathogen infection. SAR provides protection not only against the current infection but also against future attacks. It is mediated through the salicylic acid (SA) signaling pathway and results in the accumulation of specialized proteins that act against a broad spectrum of pathogens.

This study aims to elucidate the different types of systemic resistance and their critical role in protecting plants from various diseases, with particular emphasis on the importance of hormone-mediated signaling pathways.

**Keywords:** *Induced Systemic Resistance, Systemic Acquired Resistance, PGPR, PGPF.*

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## 1- Introduction

Over a century ago, researchers discovered that plants surviving an initial infection often develop resistance to subsequent attacks. By 1933, the concept of acquired immunity in animals—either partial or complete—was already documented following recovery from primary infections. That same year, after reviewing around 200 scientific publications, Chester (1933) concluded that plants also develop increased resistance to later infections following an initial one. He proposed that this acquired immunity likely plays an important role in limiting disease outbreaks in nature, and referred to the phenomenon as physiological acquired immunity (Chester, 1933).

Nearly three decades later, Ross (1961) further investigated this phenomenon in tobacco plants (Nicotiana tabacum), specifically the Samsun NN cultivar. Due to the presence of the N gene—introgressed from Nicotiana glutinosa—these plants responded to Tobacco Mosaic Virus (TMV) infection by forming necrotic lesions resulting from rapid localized cell death, a response known as the hypersensitive response (HR). This mechanism restricted viral spread to other parts of the plant. Ross demonstrated that when Samsun NN plants were inoculated with TMV on three lower leaves and then challenged again seven days later on two upper, uninoculated leaves, the resulting necrotic lesions on the upper leaves were significantly smaller than those from the initial inoculation—indicating the development of systemic resistance.

Disease resistance refers to a plant's ability to limit the growth and spread of pathogens. This resistance can vary in degree—ranging from highly resistant to moderately resistant to susceptible—depending on how quickly and effectively the plant responds to a pathogen's invasion. Plants possess both preformed structural and enzymatic defense mechanisms as well as inducible defenses that are activated upon exposure to either pathogenic or non-pathogenic microorganisms, their extracts, or certain chemical compounds. These defenses include pre-established barriers and constitutively present antimicrobial compounds, providing protection against a wide spectrum of pathogens (Jung *et al*., 2024).

Today, several beneficial microorganisms—such as Bacillus, Pseudomonas, and Trichoderma—are widely employed as biological control agents to manage various plant diseases or to induce systemic resistance in plants (Yu *et al*., 2022).

Furthermore, numerous chemical agents have been proven to enhance disease resistance in certain plants. Among them are salicylic acid (SA) (White, 1979), isonicotinic acid (INA) (Ward *et al*., 1991), jasmonic acid (JA) (Cohen *et al*., 1993), and acibenzolar-S-methyl (BTH, also known as Bion®) (Friedrich *et al*., 1996), as well as probenazole and the non-protein amino acid β-aminobutyric acid (BABA) (Walters *et al*., 2005).

## 2- Types of Systemic Resistance

To activate plant resistance, an initial elicitor is required, which can be biotic (pathogenic or non-pathogenic microbes), chemical, or physical in nature (Jung, 2020). Fungal parasites are classified into two major groups according to their nutritional strategies: necrotrophs, which kill host cells prior to or during colonization and extract nutrients from dead tissue, and biotrophs, which require living plant tissue for nourishment (Meena *et al*., 2022).

Once a plant's defense mechanisms are initiated, it develops a form of systemic resistance. This involves a complex signaling network that integrates structural defenses with biochemical responses and is classified into Induced Systemic Resistance (ISR) and Systemic Acquired Resistance (SAR). Both ISR and SAR rely on distinct molecular signals and hormonal pathways—primarily the salicylic acid (SA) pathway and the jasmonic acid/ethylene (JA/ET) pathway (Choudhary *et al*., 2007). The nature of the elicitor, whether biotic or abiotic, significantly influences which systemic resistance pathway is activated.

### 2.1Induced Systemic Resistance (ISR)

Induced Systemic Resistance (ISR) emerged as a critical defense mechanism driven by certain Plant Growth-Promoting Rhizobacteria (PGPR) and fungi (PGPF), including species of Bacillus, Pseudomonas, Trichoderma, and mycorrhizal fungi. These microorganisms enhance the plant's systemic defenses against a wide range of pathogens and herbivores (Pieterse *et al*., 2014).

Recent research indicates that beneficial microbes stimulate early immune responses in plants by upregulating disease-related defense genes and enhancing key defensive enzymes—such as phenylalanine ammonia-lyase, polyphenol oxidase, peroxidase, β-1,3-glucanase, chitinase—and by accumulating reactive oxygen species (Guo *et al*., 2019; Wang *et al*., 2020).

ISR is defined as the activation of defense mechanisms in plants against multiple pathogens through the application of plant‑growth‑promoting microorganisms in the soil or foliar zone (Hossain *et al*., 2008; Maksimov *et al*., 2011). Common ISR-inducing microbes include Bacillus spp., Paenibacillus alvei, Pseudomonas aeruginosa, Pseudomonas fluorescens, Streptomyces lydicus, Streptomyces pactum, Acrophialophora jodhpurensis, Trichoderma spp., and Serendipita vermifera (Siddiqui *et al*., 2023).

## 2.2. Systemic Acquired Resistance (SAR)

The concept of SAR—and its counterpart, Locally Acquired Resistance (LAR)—was first described by Ross in 1961, following TMV infection in tobacco (Nicotiana tabacum). He observed that upon a second infection of upper leaves two days after initial viral inoculation, the resulting necrotic lesions were smaller and appeared more swiftly than initial lesions. This indicated enhanced resistance—likely due to restricted virus replication or movement—occurring even in distant, uninoculated leaves. Importantly, SAR proved to be a long-lasting defense, observed up to 20 days post-infection (Klessig *et al*., 2018).

Ross (1961) also noted that SAR could only be triggered by infection with virulent pathogens—not by chemical injury, mechanical damage, or inoculation with UV-inactivated TMV or non-pathogenic viruses. He emphasized environmental conditions as modulators of resistance efficacy.

Subsequent studies revealed an accumulation of specific proteins in both proximal and distal leaves of infected plants, absent in healthy or susceptible controls. These proteins—termed Pathogenesis-Related (PR) proteins—serve as molecular markers of both local and systemic resistance (Van Loon & Van Kammen, 1970; Gianinazzi & Kassanis, 1974; Van Loon, 1975).

Many PR proteins exert direct antimicrobial activity by inhibiting pathogen growth or spore germination, functioning as degrading enzymes or protease inhibitors. Indirectly, they stimulate structural defenses, including lignin and callose deposition, phytohormone accumulation, and hypersensitive cell death to limit pathogen spread (Mhlongo *et al*., 2018; Finkina *et al*., 2017). Crucial PR proteins include chitinases, β-1,3‑glucanases, peroxidases, and ribonucleases encoded by PR genes activated during SAR (Dos Santos & Franco, 2023).

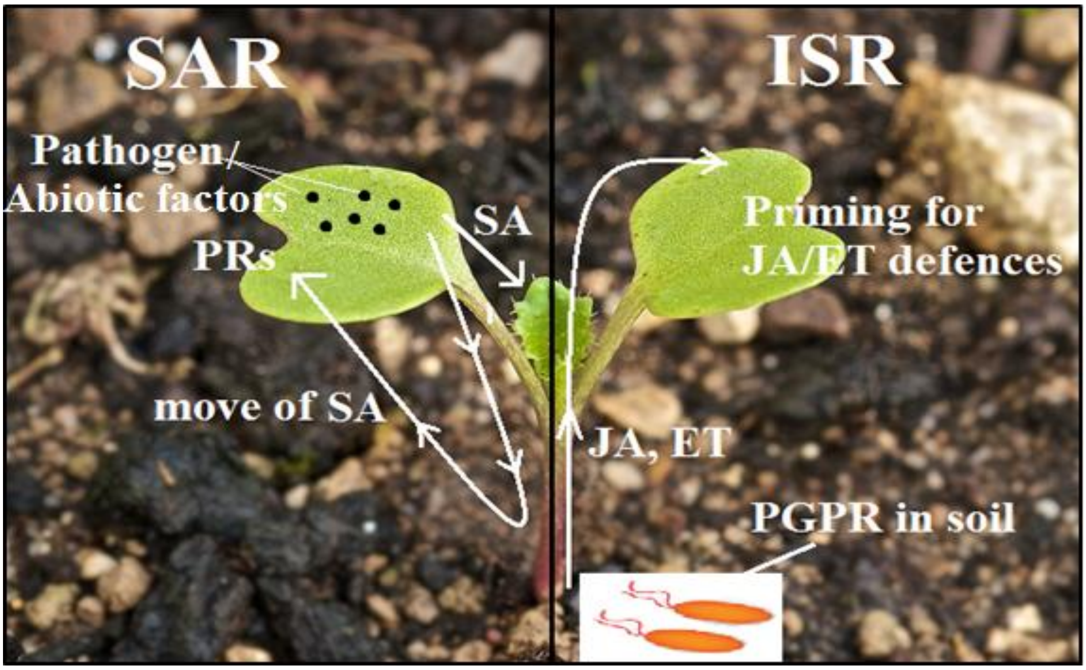

**Figure 1. Induced Systemic Resistance (ISR) and Systemic Acquired Resistance (SAR) Pathways**

## 2.3Chemical Elicitors of Systemic Resistance

Biotic and abiotic elicitors—natural or synthetic—have emerged as alternatives to traditional fungicides and insecticides. These compounds trigger local or systemic resistance in susceptible hosts, often conferring extended disease protection and can be applied alone or alongside fungicides (Thakur & Sohal, 2013).

According to Kessmann *et al*. (1994), three criteria must be met before a chemical can be classified as an abiotic elicitor in SAR:

1- It must not exhibit direct antimicrobial activity or be metabolized by the plant into antibiotic compounds.
2- Treatment must confer resistance to pathogens in a manner similar to biotic elicitors.
3- It must stimulate the same biochemical pathways as those activated by biotic elicitors.

Key chemical elicitors include salicylic acid (SA), quinolizidine alkaloids, gossypol, non-protein amino acids, glucosinolates, furanocoumarins, and β-aminobutyric acid (Heil & Bostock, 2002)

## 2.4. Hormonal Pathways in ISR and SAR

Plants produce a suite of hormones contributing to defense against pathogens and herbivores, notably jasmonic acid (JA), ethylene (ET), and salicylic acid (SA) (Kliebenstein, 2012). The interplay between regulatory and defensive hormones governs the timing and nature of resistance response (Yu *et al*, 2022).

In SAR, pathogen-triggered induction leads to structural increases in SA and its translocation through the plant, priming SA biosynthesis in distal tissues. This systemic SA accumulation is both necessary and sufficient to establish SAR (Durrant & Dong, 2004). ISR, in contrast, depends on the combined signaling of JA and ET (Verhagen *et al*., 2004).

Generally, necrotrophic pathogens activate the JA/ET pathway, whereas biotrophs and hemi-biotrophs trigger the SA pathway. JA/ET signaling enhances defense against necrotrophs, while SA signaling controls expression of SAR-related PR genes such as PR1, chitinases, PR2, and PR5 (Spoel *et al*., 2003; Mur *et al*., 2006). Non-pathogenic rhizosphere microbes also induce ISR through JA/ET signaling (Van Loon *et al*., 1998).

Key defense genes marking pathway activation include *PR1*, *PR2*, and *PR5* for SA-dependent SAR, and *PDF1.2*, *PR3*, and *PR4* for JA-dependent responses. Transcription factors such as *TGA5* and *TGA6* indicate SA pathway activation, while *AOC3* and *ERF2* are markers of ET signaling (Yang *et al*, 2010., Singh *et al*, 2014., Alkooranee *et al*, 2017).

Despite early assumptions that SAR is pathogenesis-specific, recent studies have demonstrated its induction via non-pathogenic microbes and chemical elicitors, involving both SA and JA/ET signaling (Yu *et al*., 2022). Pathogen lifestyle also plays a role: necrotrophs typically induce

ISR, while biotrophs and hemi-biotrophs tend to trigger SAR, as observed in mustard crops affected by powdery mildew and sclerotinia stalk rot (Alkooranee *et al*., 2017).

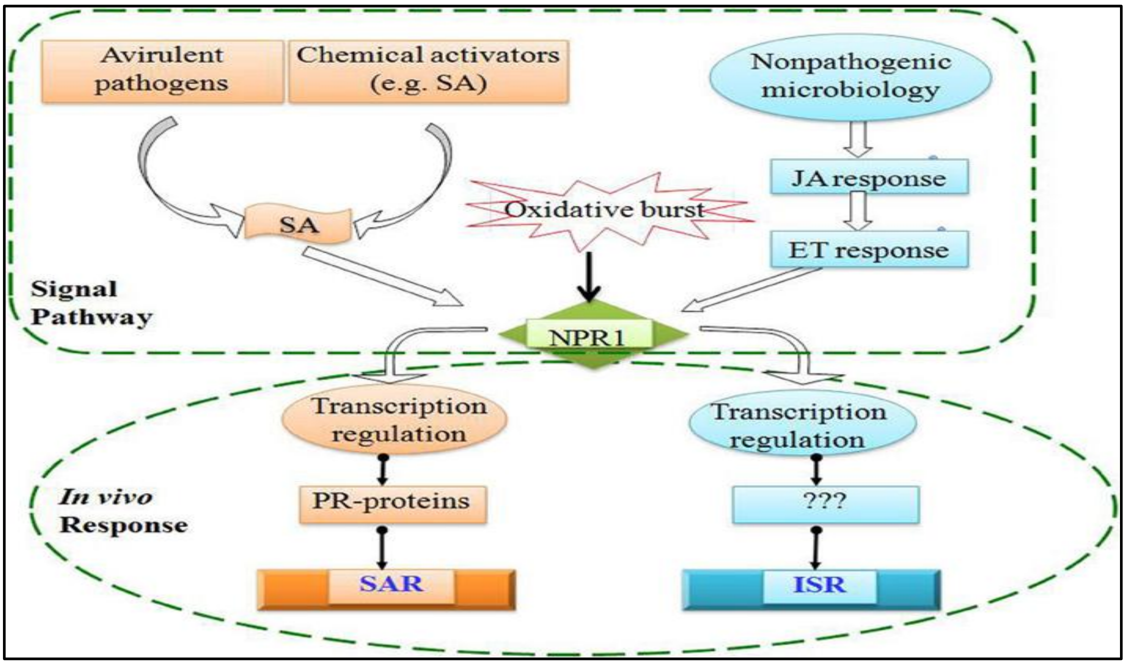

**Figure 2. Signal transduction pathways for induced systemic resistance (ISR) and systemic acquired resistance (SAR) (Chan, 2013)**

Many previous studies (Table 1) referred to biotic and abiotic elicitors in stimulating the systemic resistance in different plants against various pathogens and each elicitor capable of taking a hormone pathway to transfer the resistance signal from treated part of plant to other parts non-treated with elicitor.

**Table 1: Biotic and Abiotic elicitors the systemic resistance in plants**

| Inducer | Plant | Pathogen | Hormonal pathway | Reference |
|---|---|---|---|---|
| Chitin | Rice | *Bipolaris oryzae* | SA | Takagi *et al*, 2022 |
| Benzothiadiazole | *Arabidopsis thaliana* | *Hyaloperonospora arabidopsidis* | SA | Schillheim *et al*., 2018 |
| Benzothiadiazole | Parsley | *H. arabidopsidis* | SA | Schillheim *et al*., 2018 |
| *P. fluorescens* WCS417r | *A. thaliana* | *H. parasitica* | JA-responsive | Pozo *et al*, 2008 |
| Salicylic acid | Tomato | tobacco mosaic virus (TMV) | SA | Mur *et al*, 1996 |
| N-hydroxypipecolic acid | *A. thaliana* | *P. syringae pv. maculicola* | SA | Chen *et al*, 2012 |

| | | | | |
|---|---|---|---|---|
| Pipecolic acid | Tabacco | *P. syringae pv. tabaci* | SA | Vogel-Adghough *et al*, 2013 |
| β-aminobutyric acid | *A. thaliana* | *P. syringae pv. tabaci* | SA | Ton *et al*, 2005 |
| β-aminobutyric acid | *A. thaliana* | *H. parasitica* | SA , ABA | Ton *et al*, 2005 |
| Methyl jasmonate | Tomato | *Rhizoctonia solani* | SA , JA | Koely *et al*, 2022 |
| Beta-aminobutyric acid | *Vitis vinifera* | *P. viticola* | SA, JA | Hamiduzzaman *et al*, 2005 |
| Gamma-aminobutyric acid | *Lactuca sativa var. Gisela* | *Botrytis cinerea* | Accumulation of ROS | Janse van and van den Ende, 2020 |
| Cytokinins | *A. thaliana* | *P. syringae pv. tabaci* | SA | Choi *et al*., 2010 |
| Hexanoic acid | Tomato | *B. cinerea* | ABA , JA | Vicedo *et al*, 2009 |
| 2,6-dichloroisonicotinic acid | *Cucumis sativus* | *P. sojae* | H2O2 Production | Fauth *et al*, 1996 |
| *Sclerotinia sclerotiorum* | *B. napus* | *S. sclerotiorum* | ETH | Yang *et al*, 2010 |
| *Trichoderma viride* | Rice | *Fusarium oxysporum* | | |
| Cell-free of *T. asperellum* STK-1 | *A. thaliana* | *P.syringae pv.Tomato* DC3000 | JA | Yoshioka *et al*, 2012 |
| *P. fluorescens* | Chickpea | *Fusarium oxysporum f.sp. ciceri* | SA | Saikia *et al*, 2003 |
| *P. fluorescens* WCS374r | *Oryza sativa* | *Magnaporthe oryzae* | SA | De Vleesschauwer *et al*, 2008 |
| *B. subtilis* UMAF6639 | Melon | *Podosphaera fusca* | JA | García-Gutiérrez *et al*, 2013 |

## 3-Conclusions

Plants possess a sophisticated immune system to resist biotic and environmental stresses. They have physical barriers, such as thick cuticles, wax layers, and specialized hairs, that prevent the entry of pathogens and insects. Additionally, plants produce various chemical compounds to defend themselves against herbivores and pathogens. This resistance can be long-lasting. The type and strength of resistance depend on several factors, including the nature of the elicitor (biotic or abiotic, pathogenic or non-pathogenic), the lifestyle of the invader (biotroph or necrotroph), the molecular signals involved, and the hormonal signaling pathways (JA/ET or SA). Accordingly, a plant may develop Induced Systemic Resistance (ISR) or Systemic Acquired Resistance (SAR).

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