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Immunity in Plants and Applications

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Abstract

Systemic acquired resistance (SAR) or generated systemic resistance is researched via local plant–microbe interactions (ISR). Pathogens on leaves induce SAR, whereas plant-helping microbes on roots create ISR. SAR includes salicylic acid (SA), but other signals enhance the immune system. SAR and maybe ISR-related signalling networks govern the immune system via these signals. N-hydroxy-pipecolic acid (pipecolic acid) drives non-SA SAR. When plants are stimulated by SAR, they release volatile organic chemicals that other plants use as defensive signals to control the spread of defences between plants. SAR and ISR affect how phytohormones work together to make plants more resistant to pathogens and change the way their microbiomes are made up. Plant defence, interactions between plants and microorganisms, and interactions between plants may change. So, interactions between organisms can be used together to protect plants in a very effective way.

Keywords: Systematic immunity; Phytochrome; Signaling networks; Plant interaction;

Introduction

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Most of these responses terminate on phytohormone pathways that interact with one other and presumably fine-tune the plant's response for optimal health. Plants combat pathogens via phytohormones like SA, JA, or ethylene (ET) [1]. SA-dependent immune responses fight biotrophic and hemibiotrophic pathogens. Necrophilic diseases and insects, along with ET, help JA protect itself. Systemic signals from the part of the plant that is sick spread to the rest of the plant, preparing systemic tissues that are not sick to handle more stress. "Priming" strengthens stress responses and induced or systemic resistance. (Half-)biotrophic pathogens activate SA-dependent defence. Pattern recognition receptors in plant cells recognise pathogens' P/MAMPs. PAMP-triggered immunity (PTI) prevents pathogen development[2].

Pathogen effectors block PTI in the host cell's cytoplasm, helping the pathogen flourish. ETI destroys infected tissue and surrounding tissue if pathogen effectors activate host RESISTANCE (R) protein-dependent responses. Stops pathogen spread. SA causes SAR in PTI and ETI (SAR). SAR long-term protects against several (hemi-) biotrophic infections. SAR may endure from 3 to 10 days in the lab, depending on the plant and pathogen, but trans-generational SAR, when induced plants pass on the SAR state to their offspring, can last much longer [3].

Systemic resistance gained over time (SAR)

SAR is commonly investigated as a leaf-to-leaf interaction that relies on two parallel and interrelated pathways: one that depends on SA and one on the non-proteinogenic amino acid pipecolic acid (Pip or its supposed bio-active derivative Nhydroxy-Pip) (NHP) [4].

SA-dependent SAR

Early investigations demonstrated that the bacterial SA hydroxylase NahG damaged local immunity and SAR. In Arabidopsis thaliana, long-distance apoplastic route SA does not generate SAR. In grafting tests, NahG-expressing tobacco rootstocks send systemic signals to wild-type scions, causing SAR. SAR requires SA accumulation in systemic tissues, since NahG-expressing scions cannot react to long-distance signals from wild-type rootstocks (Fig. 1). Infected petiole exudates of cuticle-defective Arabidopsis mutants, which have poor apoplastic SA production and transport, obtain SAR signals. Infected wild-type plants don't (Fig. 1). NahG tobacco and cuticledefective Arabidopsis petiole exudates have reduced

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apoplastic SA. So, SA and other signalling molecules may help with defensive signalling over long distances. Systemic SAR signal reception or spread is dominated by SA buildup. The SAR signal could be spread out and made stronger by SA partitioning between the apoplast and the cytoplasm. However, this process is slowed down in mutants with bad cuticles. This is supported by the fact that SAR-related resistance inducers like Pip have no effect on mutant cuticles [5].

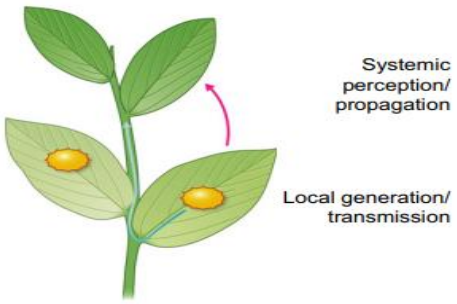

Fig. 1: Local and systemic SAR signal creation, transmission, perception, and propagation

SA-Pip SAR signals connected SAR's SA and Pip/NHP pathways collaborate, according to new study. Seven transcription factors regulate Pip, NHP, and ISOCHORISMATE SYNTHASE 1 genes (ICS1, also known as SID2). Arabidopsis's CAMTA1-3 mutation confers SA and Pip/NHP-dependent disease resistance. SAR-DEFICIENT 1 (SARD1) and CALMODULIN BINDING PROTEIN 60g (CBP60g), another set of common CAMTA-regulated transcription factors, influence biosynthetic enzyme expression to generate more of both substances (Fig. 2). TGACG-binding factor 1 and 4 govern transcription factors well. Sid2 mutant plants have little SAR- and Pip- or NHP-induced resistance, unlike Pip-deficient mutants. SA builds up and sends signals to resist external Pip or NHP. Pip also stabilises cytosolic NONEXPRESSOR OF PR GENES 1 (NPR1). Three SA receptors, like NPR1, activate SA-mediated nucleus defences [6]. NPR1 protein controls Pip, SA, SARD1, and CBP60g gene expression. SA activates Pip and NHP genes and instructs the same genes when SA levels are low.

Pip-dependent SAR

AGD2-like Defense Response Protein 1 (ALD1) and SAR-DEFICIENT 4 synthesise Pip from L-lysine to fight infections (SARD4). FMO1 converts Pip outside plastids into bioactive

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NHP. Gene changes eliminate SAR. The transcription factor-mediated loop and three more positive feedback loops create Pip/NHP and SA [7]. MAP kinases 3 and 6 activate WRKY33, which binds to ALD1's promoter and produces more Pip. ALD1, FMO1, and NPR activating MAP kinases 3 and 6 improves systemic immunity. Local SAR signal creation doesn't need ALD1 or Pip from scratch (Fig. 1). Instead, systemic organs make PIPs using ALD1 to deliver or receive SAR signals (Figs 1, 2).

SAR signal perception lectins, SAR-related events that transmit SAR signals from locally infected tissues, and systemic mechanisms that increase resistance are well recognised. Systemic leaves' mobile SAR recognition is unknown. LEGUME LECTIN-LIKE PROTEIN 1 helps systemic leaves recognise SAR signals (LLP1). Legumes have lectin-legB glycoprotein LLP1. SAR required apoplastic plasma membrane protein. EDS1 builds SA and LLP1 proteins in the apoplast before they do [8]. LLP1 doesn't influence local PTI, ETI, or SA-induced resistance. LLP1, FMO1, AzA, and monoterpenes form a defence mechanism that requires EDS1 but not SA. LLP1 may temporarily attenuate ETI. LLP1 modulates SAR and defensive responses within and across plants through a positive feedback loop upstream of Pip, G3P, and monoterpenes (Fig.2). EDS1 precedes the SA and SAR Pip, NO-ROS, AzA, and G3P pathways. The SA and Pip/NHP SAR pathways communicate, and EDS1 may regulate both upstream [9].

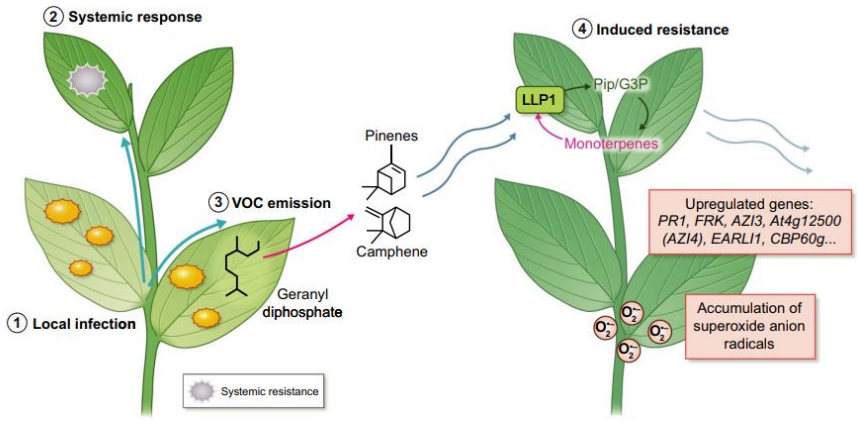

Fig:2 Plant-to-plant propagation of systemic acquired resistance (SAR). After a pathogen is put on a leaf, the plant goes through a series of reactions that cause a systemic response (2) in distant tissues (1). Geranyl diphosphate precursors are used by infected plants to make pinenes and camphene (3).

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Moncot SAR

Monocotyledonous plants don't know SAR or signalling. Both monocots and dicots maintain SAR. NPR1, SA-associated transcription factors, and PR genes are SA signalling downstream players [10]. SA helps downstream signalling in monocots, although its physiological relevance during defensive reactions is unclear and tends to differ by species. Maize, barley, wheat, and banana show SAR-like systemic immunity. Colletotrichum graminicola and SA/ABA produce maize systemic immunity. *Fusarium oxysporum* causes banana systemic immunity and increased systemic SA levels. *Pseudomonas syringae* locally infects barley, making its tissues more resistant to diseases [11].

Mobile SAR signal

SAR signals—up? what's MeSA, G3P, DIR1/DIR1-like, AZI1/EARLI1, monoterpenes, and LLP1 promote systemic immunity, whereas SA and Pip influence local/basal and systemic defences. AZI1 and EARLI1 enhance SAR in AzA-dependent local, infectious, and systemic tissues. LLP1 likely perceives or distributes systemic phloem-mobile signals and airborne defence cues [12].ISR, unlike SAR, is made when good soil microbes interact with plant roots. ISR inducers are bacteria like Pseudomonas, Bacillus, and Streptomyces, as well as fungi like *Trichoderma* and *Serendipita* indica (formerly *Piriformospora indica*) [13]. Both monocotyledonous and dicotyledonous plants grow faster and are less likely to get sick because of PGPRs and PGPFs. Through induced resistance, (hemi-)biotrophic infections like SAR don't hurt the tissues in the air. ISR also kills microorganisms that feed on dead things.

ISR signaling

ISR depends on JA and ET, and MYC2 in the leaf tissue is very important.MYC2 may inhibit PTI so PGPR/F may root. ABA levels rise in the roots, but they rise higher in the leaves, closing stomata to keep foliar pathogens out [14]. ISR protects plants against (hemi-) biotrophic pathogens without signalling or boosting SA levels. SAR and ISR benefit from NPR1's non-SA function.

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Plants and their micro biomes interact

Many eukaryotes get food and protection from pathogens from their microbiome. Pathogens are killed by antimicrobials, competitive outgrowth, and lack of food. Beneficial microorganisms in a plant's microbiome may turn on the immune system, including ISR. The microbiota is affected by a plant's immunity [15]. In systemic interactions, good bacteria that live on leaves may trigger an immune response and make the body less likely to get sick. *Arabidopsis* leaves show changes in gene expression when *Sphingomonas melonis* Fr1 is present. This makes axenic plants more resistant to *Pseudomonas syringae.*

SAR and ISR hormones talk to each other

Plants seldom experience one stressor. Thus, SAR/ISR must interact with other signalling cascades such abiotic stress. When several environmental stressors occur, ABA and SAR/ISR may alter systemic defence. Exogenous ABA prevents SA-related reactions. ABA accelerates SA-receptor NPR1 proteasomal degradation [16]. SA defence suffers. SA functional analogues diminish ABA-responsive and ABA-biosynthesis genes after salt exposure. SA/SAR may also reduce ABA-induced stress responses. SA/SAR inducers disrupt tomato and Arabidopsis ABA signaling. SA does not influence several SAR induction pathway components in ABA-mediated abiotic stress responses [17].

Conclusions

About the ecology of induced resistance Synthetic agrochemicals are used to protect crops today. Most of the time, these insecticides make pests and diseases more resistant. This, along with the public's awareness of possible environmental and health risks, has made people want crop protection methods that are better for the environment [18]. Push-and-pull management of VOCs works well. The "push-and-pull" method makes it hard for pests to find, like, or use protected resources. This method uses companion plants that give off volatile organic compounds (VOCs) to attract or drive pests away from the main, economically valuable host plant. Pests in agriculture are lessened by this plan. SAR and ISR change how bacteria colonise leaves, roots, and soil, while the plant-associated microbiome changes how plants defend themselves. So, SAR inducers like Pip or intercropping may change the populations of microorganisms on plants to make them more resistant [19]. If ISR is linked to leaf VOC

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emissions, PGPR/Fs might turn on defence and cause more VOC emissions to send the defence signal to more plants. SAR inducers, intercropping, and PGPR/F may boost plant immunity. SAR and ISR's field performance is unknown [21]. The environmental impacts of PGPR/F inoculants are unclear. More study on economically significant agricultural plants including potato, barley, and wheat is required to understand species interactions. Field testing is required to see whether they can safely safeguard crops long-term [20].

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