Systemic Induced Resistance: A Strategy in Target of Achieving Sustainable Agriculture Goals through Plant Disease Management

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When just these chemical treatments were used, resistance developed, pests returned, residues accumulated, and the environment became contaminated. Plants have developed numerous defence mechanisms to stave off predators such as insects and diseases. Systemic acquired resistance (SAR) is a form of induced resistance that can develop in plants after they are exposed to elicitors from pathogenic, avirulent, or nonpathogenic microbes or synthetic chemical stimuli like chitosan or salicylic acid (SA). According to (Gozzo and Faoro, 2013).

Systemic acquired resistance (SAR) and induced systemic resistance (ISR) are two categories of induced resistance. Phenotypically, ISR is similar to pathogen-SAR because it increases resistance to pathogens such fungi, bacteria, viruses, and nematodes (Ran et al., 2005). Localised acquired resistance (LAR) and systemic acquired resistance (ISR, SAR) were initially defined by Ross (1961).

Diseases reduce crop yields and quality, and the harvest may contain poisons from microbes. In the past, plant diseases have triggered severe economic and food shortages, and they still play a major role in today's declining worldwide crop supply. Multiple phytochemical pesticides are being employed extensively to guarantee an adequate production and harvest quality. However, pesticides have harmful effects on food production, ecosystems, and human health.

Most current methods of protecting plants against disease and pests involve the use of harmful chemicals that are bad for the environment. Induced resistance, which makes use of plants' natural defence systems, is an alternative, unorthodox, and environmentally responsible form of plant protection.

Incorporating SAR into farming practises has the potential to lessen reliance on chemical pesticides, which is a win for eco-friendly farming. Plants can develop induced resistance when the expression of their innate defence systems against pathogens is stimulated. Pathogens that trigger hypersensitive necrotic reactions, avirulent or attenuated pathogenic strains, elicitors of pathogenic strains (proteins, glucans, lipids), and abiotic elicitors such as 2,6-dichloroisonicotinic acid (INA), benzathiadiazole (BTH), and -aminobutyric acid (BABA) are all potential triggers.

However, SAR may take several days to develop across the entire host plant (Kuc, 1982). Induced buildup of reactive oxygen species (ROS) has been postulated as an integrator between SA and ABA signals in the regulation of stomatal closure (Miura et al., 2013). Due to their roles in stomatal closure, SA and ABA have been hypothesised to mediate drought resistance.

Exogenous induction of SAR can be achieved by administering either the natural defence hormone SA or its synthetic equivalents 2,6-dichloroisonicotinic acid (INA) or benzothiadiazole S-methyl ester (BTH). SAR provides protection from a wide variety of pathogens, including bacteria, viruses, fungi, and oomycetes. The immunological "memory" that SAR imparts to plants may last for several weeks, months, or even the entire growth season. SAR, in contrast to ETI, encourages cell survival and is unrelated to programmed cell death.

SAR is predominantly mediated by and dependent on SA, since it was eliminated in the npr1, ics1, and other SA-accumulating mutants (Gozzo and Faoro, 2013; Kachroo and Robin). Many man-made chemicals, including salicylic acid, BTH (an SA analogue), and probenazole, have been linked to SAR. They play a significant role in the field of crop protection as chemical plant immunity activators. According to Kachroo and Robin (2013), many inducers of SAR signalling prime for SA accumulation in systemic tissues via a network of shared nodes.

Biotrophic infections were formerly assumed to be the primary focus of SA-dependent immune responses, as stated by Glazebrook (2005). Gozzo and

Faoro (2013) state that SAR is successful in combating several pathogens, nematodes, and parasitic plants. SAR is associated with a plethora of mobile transmissions. The most extensively researched mobile signal is MeSA, which is produced as a volatile emission during infection and is required for SAR (Park et al., 2007).

Instead of changing the genome (via mutations or the introduction of foreign genetic material), induced resistance relies on the manifestation of dormant genetic information within the plant, making it more physiologically safe. Topics covered include receptor-elicitor interactions, signal transduction pathways, and SAR gene expression as they pertain to the molecular underpinnings of induced resistance.

Understanding the SAR procedure has made great strides recently. The Arabidopsis model plant was used to determine that the "isochorismate (ICS) pathway" is the primary source of SA during SAR. In response to SA, the nucleus-localized positive regulator protein NPR1 interacts with TGA transcription factors (TFs) to activate SAR and induce the expression of defense genes. In their 2007 paper, Vasyukova and Ozeretskovskaya summarised what is known about SA's role in plant resistance.

The SA-dependent pathway relies on SA as a signalling molecule. The ability of SA to inhibit enzymes in the plant's antioxidant system is crucial to comprehending how SA builds resistance in plants by causing an accumulation of active oxygen species and the activation of defence genes.

As a result of redox changes induced by SA, NPR1 is transformed from inactive, disulfide-bound oligomers into functional monomers in the cytoplasm. NPR1 monomers localise to the nucleus and interact with the TGA family of basic leucine zipper TFs, resulting in the expression of several SA-dependent genes. WRKY TFs appear to play an important role in SA defensive responses as activators and repressors of SA transcription, either in parallel with or downstream of NPR1 (Wang et al., 2006). Although it is now clear that effectors can suppress SA defence, it is not yet clear how the various effectors link up with and alter SA signals (Loake and Grant, 2007).

Enhanced resistance to nematodes, bacteria, viruses, and fungi is made possible by SAR. Exposure to elicitors such as pathogenic, nonpathogenic, or virulent microbes, or synthetic chemical stimuli like chitosan or salicylic acid (SA), can cause a plant to develop systemic acquired resistance, a type of induced resistance that is functional throughout the plant. However, it may take several days for SAR to travel throughout the host plant.

To begin SAR, mobile signals must be generated at the location of local infection within four to six hours of the onset of illness. After reaching the systemic tissues (perhaps via the phloem), the signals trigger the defensive response. Salicylic acid (SA) and its methylated equivalent, MeSA, Jasmonic acid, Auxin, Pipecolic acid, Pip, Dehdryoabietinal, DA, Azelaic acid, AzA, and BABA have all been found to cause SAR.

Regulation of cellular redox and NPR1 nuclear translocation in response to SA accumulation is mediated by thioredoxins (TRXs) and S-nitrosoglutathione (GSNO). NPR3 and NPR4 are SA receptor proteins that regulate the nuclear concentration of NPR1. To facilitate PCD and ETI, a high concentration of SA is required at the site of local infection, while a moderate concentration of SA in the surrounding cells restricts NPR1-NPR4 contact, leading to NPR1 accumulation.

Protein secretion, antimicrobial PR proteins including PR1, PR2, and PR5, and resistance to secondary infection are all aided by the ER genes that NPR1 activates through interactions with transcription factors (TFs). Markers of the SAR primed state include H3K9 acetylation (Ac) and H3K4 methylation (Me) at SAR-associated gene promoters.

The activation of plant defence genes and the maintenance of genome stability in the current generation and in future generations may involve DNA methylation, proteins that control chromatin structure like SNI1, DNA repair like RAD51 and BRCA2, and other biological processes. Salicylic acid (SA), methyl salicylic acid (MeSA), azelaic acid (AzA), glycerol-3-phosphate (G3P), and the abietane diterpenoid dehydroabietinal (DA) are only some of the signals that can be produced in response to an avirulent infection.

These signals cause the uninoculated distal tissue to produce antimicrobial PR (pathogenesis related) genes, protecting the rest of the plant against infection. A SAR describes this kind of situation. Reducing the need for pesticides and other plant protection products (PPP) through the phenomena of systemic acquired resistance (SAR) is encouraging. Plants use SAR, a form of innate immunity, to fight against pathogens. It strengthens the plant's immune system, making it more resistant to pathogens of all kinds. It's possible that the plant's SAR response will be long-lasting and can be passed on to other pathogens.

Prior infection or treatment with elicitors primes the plant's defences for enhanced resistance (or tolerance) to a pathogen in systemic acquired resistance (SAR) and induced systemic resistance (ISR) (Vallad and Goodman, 2004). In contrast to the broad-spectrum disease resistance (SAR) associated with induced systemic resistance (ISR), which is mediated by a jasmonate/ethylene-sensitive pathway, SAR is characterised by and mediated by a salicylic acid (SA) dependent process (Mauch-Mani & Metraux, 1998).

However, Metraux (2001) argues that SAR and ISR are synonymous. Inducing SAR in plants protects them from infections; benzothiadiazole (BTH) and -aminobutyric acid (BABA) are two examples (Zimmerli, 2001; Tonne, 2009; Slaughter, 2012). Exogenous application of specific inducers results in a comparable response in plants, as demonstrated by Harman (2004).

Plants undergo priming during the induction of SAR, a period during which the prolonged presence of the pathogen activates the plant's defences. Under heavy disease pressure, primed plants fare better than non-primed plants or plants that express defences. In response to initial infection, plants increase their defence capacity by upregulating the expression of defence genes and producing new antimicrobial compounds such PR proteins in previously uninfected tissue (Ramos Solano, 2008).

One approach to creating SAR involves modifying plants to express SAR genes in a constitutive manner. Recent years, however, have seen the development of new theories concerning the costs of the constitutive existence of protective qualities at fixed high levels (Heil, 2000). Plants that put more resources on defence are expected to be less successful in dangerous environments. Because defence expenditures are only undertaken under conditions where they are truly necessary, phenotypic plasticity, which results in SAR responses, may have evolved solely to minimise costs. This may greatly affect the development of plant defences.

In order to prevent harm to people, animals, or the environment from mulberry disease treatments, it is crucial that SAR chemicals be widely disseminated. A model to promote the use of these chemicals is required to increase the profitability of sericulture. This strategy for controlling pests and illnesses has positive ecological effects. Fungal infection and insect damage appear to activate chitinase genes in the mulberry plant, suggesting that these chitinases help the mulberry plant overcome these dangers (Wang et al., 2015).

Conclusion

Plants' induced resistance is currently little understood, but it provides fresh information on defence mechanisms and has potential as a technique for ecologically friendly disease control and sustainable agriculture. It remains challenging for both theoretical and applied study.

Systemic induced resistance is an important strategy for implementing IPM by bolstering plants' inherent defences. Synthetic chemicals have opened up useful avenues for developing systemic resistance in plants, which can be used for the treatment of plant diseases. When SAR inducers are widely used, they have fewer negative consequences on people and the planet. There isn't a single SAR drug that isn't at least as effective as a fungicide in protecting against diseases.

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