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Systemic Acquired Resistance (SAR) and it's Application in Crop Plants Improvement to Biotic Stresses: Review

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ABSTRACT

Plants are challenged by a variety of biotic stresses like fungal, bacterial, or viral infections. Host plants can be protected against further pathogen attack if they have survived earlier infection by phytopathogenic viruses, bacteria, or fungi. Following the activation of HR, uninfected distal parts of the plant may develop resistance to further infection, by a phenomenon known as systemic acquired resistance (SAR), which is effective against diverse pathogens, including viruses, bacteria, and fungi. Systemic acquired resistance (SAR) is classically described as a plant defense response that provides long-lasting, broad-spectrum pathogen resistance to uninfected systemic leaves following an initial localized infection. The onset of SAR in non-infected plant organs is triggered by the phloem mobile signal which is released following pathogen infection. The signal for SAR may be generated within 4–6 hours from inoculation. SA could be detected in the phloem by 8 hours after inoculation, and increases in SA occurred in the phloem of the leaf above the inoculated one within 12 hours from inoculation of the lower leaf. The study results showed that the different treatment of (SA) were effective in reducing the disease infection and it could advice to utilize the Salicylic Acid as stimulating agent to decrease the degree of infection by Fusarium diseases by immersing the seeds of Chickpea in the mentioned concentration for 24 hours before the planting.

Keywords: *Systemic, Acquired, Resistance, Salicylic acid, SAR*

INTRODUCTION

Plants are challenged by a variety of biotic stresses like fungal, bacterial, or viral infections. This lead to a great loss to plant yield. There are various options available for the farmers to protect their crop from the disease. Some options include development of resistant cultivars, biological control, crop rotation, tillage, and chemical pesticides (Meenakshi and Baldev, 2013). Plants can activate separate defense pathways depending on the type of pathogen encountered. Jasmonic acid (JA) and ethylene dependent responses seem to be initiated by necrotrophs, whereas salicylic acid (SA) dependent response is activated by biotrophic pathogens. The mechanisms responsible for this differential recognition and response may involve crosstalk among these three different signal transduction pathways: JA, ethylene, and SA. The better understanding of plant signaling pathways has led to the discovery of natural and synthetic compounds called elicitors that induce similar defense responses in plants as induced by the pathogen infection (Meenakshi and Baldev, 2013).

Different types of elicitors have been characterized, including carbohydrate polymers, lipids, glycopeptides, and glycol-proteins. In plants, a complex array of defense response is induced after detection of microorganism via recognition of elicitor molecules released during plant-pathogen interaction. Following elicitor perception, the activation of signal transduction pathways generally lead to the production of active oxygen species (AOS), phytoalexin biosynthesis, reinforcement of plant cell wall associated with phenyl propanoid compounds, deposition of callose, synthesis of defense enzymes, and the accumulation of pathogenesis-related (PR) proteins, some of which possess antimicrobial properties. AOS lead to hypersensitive response (HR) in plants which is a localized or rapid death of one or few cells at the infection site to delimit the pathogen growth. Following the activation of HR, uninfected distal parts of the plant may develop resistance to further infection, by a phenomenon known as systemic acquired resistance (SAR), which is effective against diverse pathogens, including viruses, bacteria, and fungi (Meenakshi and Baldev, 2013).

Host plants can be protected against further pathogen attack if they have survived earlier infection by phytopathogenic viruses, bacteria, or fungi. It appears that the first infecting pathogen immunizes the plant against further infections by homologous pathogens, even though the plant may not carry gene determining cultivar-specific resistance. The readiness of the plant to repel subsequent pathogen attacks spread throughout the whole plant. An avirulent pathogen causing local programmed cell death can induce SAR through generation of mobile signals, accumulation of the defense hormone salicylic acid, and secretion of the antimicrobial PR (pathogenesis-related) proteins (Fu and Dong, 2013). SAR can even be passed on to progeny through epigenetic regulation. The *Arabidopsis* NPR1 (non-expresser of *PR* genes 1) protein is a master regulator of SAR (Fu and Dong, 2013). SAR signaling downstream of SA is controlled by the redox-regulated protein Non-expressor of *PR* Genes1 (NPR1), which upon activation by SA acts as a transcriptional co-activator of a large set of *PR* genes (Mukhtar *et al*.,2013, Pieterse *et al*., 2012, Pieterse *et al*.,2014 and Spoel and Dong , 2012).

LITERATURE REVIEW

Systemic Acquired Resistance (SAR)

Systemic acquired resistance (SAR) is classically described as a plant defense response that provides long-lasting, broad-spectrum pathogen resistance to uninfected systemic leaves following an initial localized infection (Carella et al., 2016). Systemic acquired resistance (SAR) is an induced immune mechanism in plants and unlike vertebrate adaptive immunity; SAR is broad spectrum, with no specificity to the initial infection (Fu and Dong, 2013).

In the 1960s, Ross coined the term SAR for the phenomenon in which uninfected systemic plant parts become more resistant in response to a localized infection elsewhere in the plant (Pieterse *et al*., 2014). In the current concept of the plant immune system, the onset of pathogen-induced SAR is triggered upon local activation of a PTI or ETI response (Pieterse *et al*., 2014). In systemic tissues, SAR is characterized by increased levels of the hormone salicylic acid (SA). Early genetic studies in tobacco demonstrated that SA accumulation and signaling are essential for the establishment of SAR. In addition, SAR is accompanied by the coordinate activation of Pathogenesis-Related (PR) genes, many of which encode PR proteins with antimicrobial activity (Pieterse *et al*., 2014). Among the best-characterized *PR* genes is *PR-1*, which is often used as a marker for SAR (Pieterse *et al*., 2014).

Activation of HR at the local level establishes the second whole plant immune response, systemic acquired resistance (SAR). Chemical receptors in the sprayed leaves' pathogenesis-related genes trigger a response in the plant's DNA, which induces the SAR response throughout the entire plant. Signals travel quickly through one of three chemical transduction pathways to all parts of the plant, providing long-lasting protection against a broad spectrum of pathogens. This process is much like a vaccination that protects people from a single strain of influenza (Titus, 2012).

Plants can also use these defense mechanisms to resist invading pests. Thorns on roses evolved over millennia as a defense mechanism against foraging animals. Research conducted at Washington State University discovered the first molecular peptide signal, systemin, which activates the entire plant to produce inhibitors of the gut enzymes insects need to digest proteins from the plant sap after just one feeding. Plant-produced protease inhibitors cause digestive upset so insects stop feeding and die from starvation (Titus, 2012).

Beneficial Microbes Triggering the Systemic Acquired Resistance Pathway

Although many rhizobacteria have the capacity to produce SA, it is usually not the causal agent of the observed systemic resistance (Djavaheri et al., 2012). This is likely caused by the fact that rhizobacteria-produced SA is often not released in the rhizosphere but becomes incorporated into SA moiety-containing siderophores that are produced under iron-limiting conditions to improve uptake of ferric iron (Fe3+), which makes SA unavailable for triggering the SAR pathway (Bakker et al., 2014). In the cases that beneficial microbes trigger SA-dependent SAR, reactive oxygen species that accumulate at the site of tissue colonization seem to be important elicitors (Pieterse et al., 2014). Because SA-dependent signaling triggered by beneficial microbes is likely to follow the SAR signaling pathway (Pieterse et al., 2014).

NPR1: A Common Regulator of Systemic Acquired Resistance and Induced Systemic Resistance

In SAR, NPR1 functions as a transcriptional co-activator of SA-responsive *PR* genes; rhizobacteria-mediated ISR typically functions without *PR* gene activation (Pieterse *et al*., 2014). Hence, the role of NPR1 in ISR seems to be different from that in SAR. In SA signaling, NPR1 is clearly connected to a function in the nucleus (Pieterse *et al*., 2014). In contrast, evidence is accumulating for a cytosolic function of NPR1 in JA/ET signaling and ISR (Pieterse *et al*., 2014). Interestingly, simultaneous activation of SAR and ISR leads to an additively enhanced defensive capacity. It is, however, interesting to note that the *NPR1*, *NPR3*, and *NPR4* genes are highly expressed in *Arabidopsis* roots, suggesting a role in the regulation of root-associated immune responses (Pieterse *et al*., 2014).

Host Pathogen Interaction

The biochemical changes that occur during infection are very similar in host and non-host resistant plants (Bakker *et al*., 2013). Disease spreads only in susceptible plants (compatible interactions) which are unable to recognize the pathogen or respond too slowly (Pieterse *et al*., 2014). The hypersensitive response is triggered by the plant when it recognizes a pathogen. The identification of a pathogen typically occurs when avirulence (Avr) gene products, secreted by pathogen, bind to or indirectly interact with the product of a plant resistance (R) gene (gene for gene model). When both the R gene and corresponding Avr genes are present, recognition occur, which lead to active resistance of the plant and avirulence of the pathogen. If either Avr gene in the pathogen or R gene in the host is absent or is mutated, no recognition will occur and outcome will be a compatible reaction and disease (Bakker *et al*., 2014).

The hormone of Salicylic Acid was used to stimulate the Systematic Acquired Resistance (SAR) against the fungus pathogen *Fusarium roseum* in the Chickpea plants. Results of researches on other substances showed the presence of stimulating effects in the Systematic Acquired Resistance in the Chickpea plants which can decrease the intensity of disease by other fungus and it could advice to utilize the Salicylic Acid as stimulating agent to decrease the degree of infection by Fusarium diseases by immersing the seeds of Chickpea in the mentioned concentration for 24 hours before the planting. Also, it could sprinkle the plantlets before a sufficient period of the infection by pathogens. This can significantly contribute in limiting the appearance and development of diseases (Bassa, 2016).

In 1961, Ross found that the zone surrounding TMV-induced local lesions on some tobacco species was completely resistant to subsequent TMV infection, as well as to unrelated viruses, including Tobacco necrosis virus and Tobacco ring spot virus (Ross, 1961a, 1961b).However, in beans (Phaseolus vulgaris), the zone around the TMV-induced lesions protected the plant only from subsequent challenges by TMV, not against infection by heterologous viruses such as Tobacco necrosis virus or Alfalfa mosaic virus. From these results, Ross suggested that the differences in these "local acquired resistance" responses were indicative of differential host responses to virus infections (Mandadi and Scholthof, 2013).

Hypersensitive Response (HR) and Systemic Acquired Resistance (SAR) Association

Similar to HR, SAR is triggered during an incompatible interaction involving Avr and R proteins in the primary infected cells. However, the resistance is transduced to the non-infected distant tissues. Although the exact mechanisms of SAR are not defined, it is initiated through a local interaction among Avr and R proteins and results in accumulation of phytohormones such as SA and JA in the distant tissues. Unlike HR, SAR is a long-lasting immune response primed to provide distant tissue resistance against subsequent infections. In the case of TMV-triggered SAR, the response persists up to 3 weeks (Ross, 1961b). However, epigenetic modifications, such as DNA methylation and chromatin remodeling, may be critical to maintain a stable SAR signal (Spoel and Dong, 2012). Recent studies of *Arabidopsis* infected with PstDC3000 demonstrated that SAR can be stably inherited to the next generation, even when the progeny was not exposed to the pathogen possibly via PstDC3000-triggered hypomethylation of host chromatin (Luna *et al*., 2012). Interestingly, the trans-generational stability of SAR requires NPR1, as progeny of the SA-insensitive *npr1-1* mutant plants failed to possess SAR in the next generation (Luna *et al*., 2012). This induced resistance phenomena is also triggered in the progeny of plants exposed

to caterpillar herbivory (Rasmann *et al.*, 2012). In this case, the stable resistance response is dependent on intact JA signaling and requires the biogenesis of short interfering RNA that could mediate the epigenetic chromatin modifications (Rasmann *et al.*, 2012). Nevertheless, SAR is yet another conserved plant defense response triggered against diverse pathogenic bacteria, fungi, and viruses. Moreover, in contrast with the HR, SAR renders a broader and long-lasting resistance to diverse pathogen types simultaneously (Mandadi and Scholthof, 2013).

Sequence of Events Associated with the Establishment of SAR

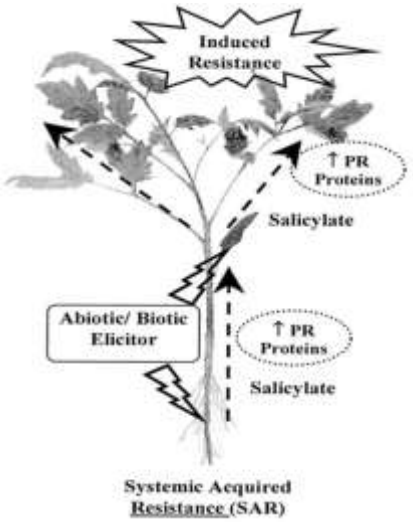
The onset of SAR in non-infected plant organs is triggered by the phloem mobile signal which is released following pathogen infection. The signal travels throughout the plant and transduced in target tissues. Following signal transduction, resistance is maintained for several days and weeks and this is likely due to de novo gene expression. The biochemical changes that occur during SAR can be divided into two phases, that is, initiation and maintenance. Physiological changes during initiation phase may be transient and short lived, but during maintenance a quasisteady state should exist (Meenakshi and Baldev ,2013).

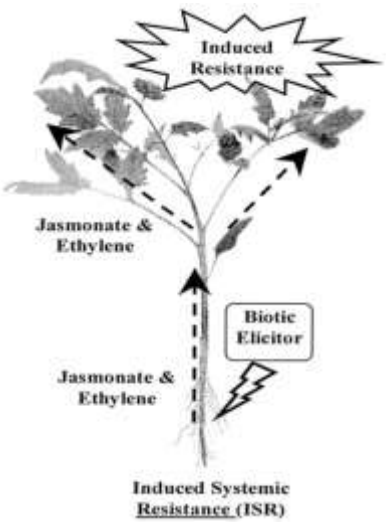
The signal for SAR may be generated within 4–6 hours from inoculation. SA could be detected in the phloem by 8 hours after inoculation, and increases in SA occurred in the phloem of the leaf above the inoculated one within 12 hours from inoculation of the lower leaf. Expression of SAR occurred within 24 hours from inoculation. By that time the entire plant contained greatly increased levels of SA, even when the inoculated leaf had been removed before any SA increase had been detected (Agrios, 2005).

The establishment of SAR follows production and accumulation of the systemic signal salicylic acid at the primary infection site, and in both local and systemic tissues. One of the first steps toward SAR is over expression of the NIM1/NPR1 gene, the protein of which is essential for transduction of the SA signal. This protein is translocated to the nucleus, where, in the presence of SA, nuclear localization of the genes results in regulated expression (Agrios, 2005).

Methods for Inducing SAR

Other materials we use in the nursery activate pathogenesis-related gene expression and induce SAR. Silicon induces the SAR response and enables suberization (cork development in cell walls). Since most soil less mixes do not contain silicon, we add wood ash from burning tree branches from around the nursery and the mineral olivine, which is magnesium/iron silicate. As a result, the stiffening of cell walls almost eliminates the need to use PGRs on most plants (Titus, 2012). There are several neonicotinoides on the market that contain imidacloprid, which is the active ingredient that induces the SAR response. At least one company has picked up on the SAR attributes by advertising the vigor response side effect when using this class of insecticide, which is easier than explaining systemic acquired resistance (Titus, 2012).





Ongoing scientific research suggests that using more than one method of activating SAR may employ all three of the plants transduction pathways and amplify the plant's ability to resist

pests and accelerate growth with reduced fertilizer and pesticide inputs. With more than a decade of experience inducing systemic acquired resistance in the nursery and gaining the reputation for consistently having the best quality in the region, the advantage we enjoy is the plant health element. More robust plants with improved stress tolerance leads to a healthier, more marketable plant that is better looking and more likely to be purchased by the home gardener shopping at the independent garden centers (Titus, 2012).

A pictorial comparison of the two best characterized forms of induced resistance in plants, both which lead to similar phenotypic responses. Systemic acquired resistance, induced by the exposure of root or foliar tissues to abiotic or biotic elicitors, is dependent of the phytohormones salicylate (salicylic acid), and associated with the accumulation of pathogenesis-related (PR) proteins. Induced systemic resistance, induced by the exposure of roots to specific strains of plant growth-promoting rhizobacteria, is dependent of the phytohormones ethylene and jasmonate (jasmonic acid), independent of salicylate, and is not associated with the accumulation of PR proteins (or transcripts). However, both responses are intertwined molecularly, as demonstrated by their reliance on a functional version of the gene *NPR1* in *Arabidopsis thaliana*.

Application of Systemic Acquired Resistance in Some Crop Plants

Application of SAR on Fababean

Exogenous applications of salicylic acid (SA) and benzothiadiazole (BTH) solutions have been used in fababean to induce systemic acquired resistance (SAR) to rust (Uromyces viciae-fabae), ascochyta blight (Ascochyta fabae) and broomrape (Orobanche crenata). Both SA and BTH solutions were effective inducing SAR to U. viciae-fabae and A. fabae on susceptible accessions under controlled conditions, although SA was less effective than BTH for A. fabae. BTH treatments reduced the infection of all pathogens studied under field conditions in susceptible accessions, and rust infection was also reduced by SA applications. Moderately resistant accessions became immune to ascochyta blight with BTH treatment, and showed a lower degree of infection to rust after SA or BTH treatments. No effect was observed in the highly resistant accessions. Chemical induction of systemic resistance may provide an additional method for controlling fababean diseases to be considered in an integrated diseases management (*Sillero et al., 2012).*

Table3. *Effect of application of different concentration of benzothiadiazole (BTH) and salicylic acid (SA) on third leaf of fababean susceptible accession VF-172 in the resistance to A. fabae on upper untreated leave.*

Treatment	4th leaf		5th leaf		6th leaf	
	ITa	IFb	ITa	IFb	ITa	IFb
Control	4	0.2	4	0.3	5	1.2
BTH 0.05 mM	4	0.1*	4	0.3	5	0.6
BTH 0.5 mM	0	0	1	0.1*	3	0.1*
BTH2.5 mM	0	0	0	0	0	0
BTH5 mM	0	0	0	0	0	0
Control	4	0.3	5	1.3	5	1.8
SA0.1 mM	2	0.1	3	0.3*	5	1.3
SA 1 mM	2	0.1	3	0.4*	5	1.0
SA5 mM	2	0.1	4	0.7	5	0.9
SA10 mM	3	0.3	4	0.8	5	0.8

Source: *Sillero et al., 2012*

**Significantly different from control (P < 0.05, Duncan test). All data are the means of three repetitions.*

a IT = Infection type according to Rashid et al. (1991). b IF =Infection frequency (number of lesions/cm2).

Application of SAR on Tobacco (Nicotiana tabacum L)

Nicotiana tabacum L. cv. Xanthi nc plants were inoculated with tobacco mosaic virus (TMV) in order to develop a method for evaluation of lesion size and its distribution characteristics during the induction of systemic acquired resistance (SAR). All necrotic lesions were scored with image analysis software and subjected to statistical analysis. The diminished lesion size and its right-skewed, non-normal distribution seem to be an important feature of SAR response. The results showed that the degree of induced resistance differs according to

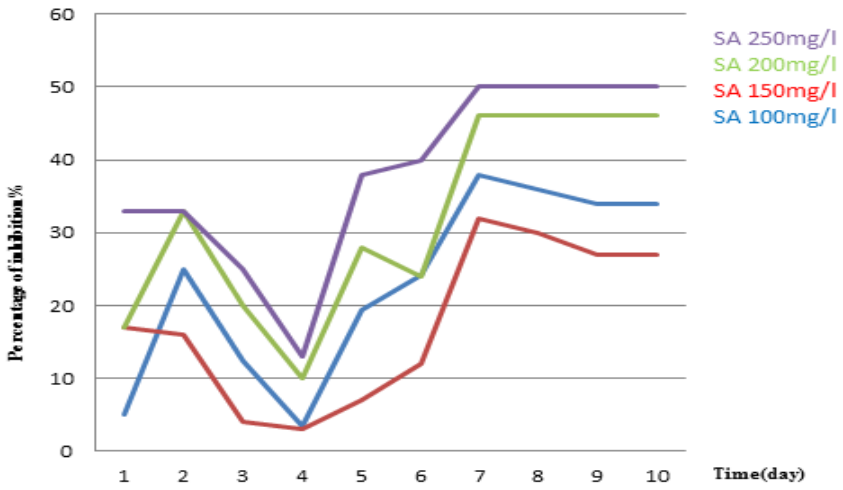
the position of the leaf on the plant's shoot. In order to detect the timing of signal transduction from TMV infected leaves to distant ones, the infected leaves were removed from the tobacco plants at different time intervals. When the infected leaves were removed after 4 days, the SAR was always induced on the distant leaves indicating complete signal transduction within 4 days. An easily applicable semi-automated method for the detection of the size of necrotic lesions and its distribution in tobacco leaves after TMV inoculation using appropriate statistical analysis was developed. Decreased lesion size diameter and its characteristic non normal, right-skewed distribution seem to be an accurate and important feature of the resistant response in distant leaves with SAR. Application of this method during SAR induction indicated that signal transduction is completed in distant leaves by the 4th day after inducing TMV inoculation. Further experiments are in progress to characterize the chemical nature on this signal (Nagy *et al.*, 2016).

Application of SAR on Chickpea (Cicer arietinum L.)

Fungal diseases are the most important biotic limiting the growth of Chickpea (*Cicer arietinum* L.). Salycilic acid application is known as a plant hormone that has the role of signal in responses of defense, whose the acquired systemic resistance. The study was aimed to evaluate the affectivity of some concentrations of Salicylic acid (SA) against the phytopathogenic fungus (*Fusarium roseum)* on two chickpea genotypes (ILC 3279 and FLIP 8555). Results showed that the inhibitory effect of (SA) on the development of *Fusarium roseum* increased linearly with increasing the concentration.

The colony diameter reduced significantly at 200, 250 mg/ l. Additionally, the results showed that the different treatment of (SA) were effective in reducing the disease infection and it could advice to utilize the Salicylic Acid as stimulating agent to decrease the degree of infection by Fusarium diseases by immersing the seeds of Chickpea in the mentioned concentration for 24 hours before the planting. Also, it could sprinkle the plantlets before a sufficient period of the infection by pathogens. This can significantly contribute in limiting the appearance and development of diseases (Noura et al., 2016).

Inhibition Percentage of *Fusarium Roseum at* Different Concentrations of Salicylic Acid



Source: *Noura et al., 2016*

CONCLUSION

Systemic acquired resistance (SAR) is classically described as a plant defense response that provides long-lasting, broad-spectrum pathogen resistance to uninfected systemic leaves following an initial localized infection and an induced immune mechanism in plants and unlike vertebrate adaptive immunity, SAR is broad spectrum, with no specificity to the initial infection.

Recent studies of *Arabidopsis* infected with PstDC3000 demonstrated that SAR can be stably inherited to the next generation, even when the progeny was not exposed to the

pathogen possibly via PstDC3000-triggered hypomethylation of host chromatin.

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