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AN OVERVIEW OF SYSTEMIC ACQUIRED RESISTANCE (SAR), A SIGNAL TRANSDUCTION PATHWAY OF PLANT DEFENSE

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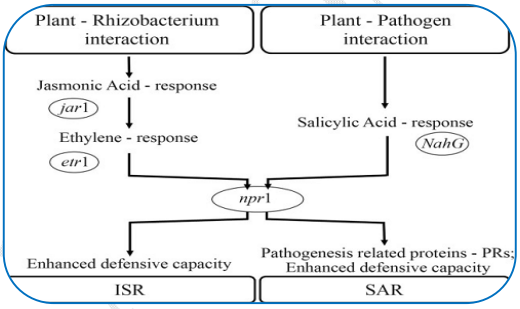
ABSTRACT :

Plants are exposed to various pest and pathogen (viz. fungi, bacteria, virus, insect, nematodes) which may cause physiological and morphological disorder or diseases in plant. To overcome these incidence plants have some signal transduction pathways of defense mechanisms such as Salicylic acid (SA) dependant- necrotizing pathogen mediated systemic acquired resistance, so called SAR, and plant growth promoting rhizobacteria (PGPR) mediated induced systemic resistance (ISR). SAR is responsible for expression of pathogenesis related proteins (PR-proteins) which play direct defensive role against various pests and pathogens regulated by NPR1 and gene NahG. In this present work SA-dependent SAR is focused for protection of crops.



KEYWORDS : *Systemic Acquired Resistance (SAR), Elicitor, Inducer, Salicylic acid (SA),*

INTRODUCTION:

Higher plants are able to trigger various defense machineries when they are exposed to various pathogenic agents such as fungi, bacteria, virus, insect, etc. These machineries do not allow pathogen for infection, if the reaction occurs in a timely manner. However, if the reactions occur at too late or suppressed, infection of pathogen proceed successfully which lead to disease in plant (Somssich and Hahlbrock, 1998). There are two different signal transduction pathways in plant: systemic acquired resistance (SAR) and induced systemic resistance (ISR). SAR is designated as salicylic acid (SA) dependant- necrotizing pathogen mediated resistance for expression of pathogenesis related proteins (PR-proteins) which plays a direct defensive role against pathogenic agents. SAR differs from ISR for many reasons, although there are some similarities (**Table-1 and Figure-1**). Management of plant disease is possible through proper investigation of signal molecules and elicitor molecules for immunization of plants in order to induction of SAR for better protection against pathogenic infection.

SYSTEMIC ACQUIRED RESISTANCE (SAR)

During the last few decade extensive research works has been performed for development of systemic acquired resistance (SAR). SAR development switch on after necrosis by necrotizing pathogen, either as a part of hypersensitive reaction (HR) or as a part of the symptom of infection, which is mediated by a mobile endogenous signal molecule, salicylic acid (SA) and transported through phloem from the sites of its origin to distal part of plant (Rasmussen e*t al.,* 1991; Smith-Beeker e*t al.,* 1998). Leaves inoculated with narcotizing pathogen exhibits high level of endogenous SA accumulation (Malamy e*t al.,* 1990) at the concentration of 1mM which is involved for the accumulation of novel pathogenesis–related (PR) proteins (Meena e*t al.,* 2001). SAR is a broad spectrum resistance

mechanism that can be triggered in plants through artificial inoculation with a necrotizing pathogen or treatment with a variety of biotic and abiotic elicitors/inducers/ (Mauch-Mani and Metraux, 1998; Sticher e*t al.,* 1997; Meena e*t al.,* 2001; Ryals e*t al.,* 1996).

Elicitors

Many elicitor molecules were reported by several workers. All signalling molecules that are perceived by plant and trigger induction of defense machineries are considered as elicitors. Elicitors are categorized in two classes: general (non-specific) elicitors which are similar in their effect on population of plant species and involved in general resistance mechanism. On the other hand specific elicitors are formed in response of specialized pathogen races or strains which are formed by specialized pathogen races or strains and function only in plant cultivars carrying the corresponding disease resistance gene (Montesano *et al.,* 2003). A variety of biotic and abiotic inducers/elicitors were reported for the establishment of induced systemic resistance (ISR) and systemic acquired resistance (SAR) in different crops. The abiotic elicitors were reported for induction of defense resistance in different crops as salicylic acid (Meena e*t al.*, 2001), active oxygen radicals (Higa e*t al.*, 2001), SA and 4–hydroxybenzoic acid (Smith-Beaker e*t al.,* 1998), β-aminobutyric acid (Kaur and Kolte, 2001), UV-light (Brederode e*t al.*, 1991), ozone (Ernst e*t al.*,1992), nitric oxide (Klessig e*t al.*, 2000) and N-acetylchitooligosaccharide (Kaku e*t al.*,1997). Biotic elicitors were reported to enhance in plant defense reactions as leaf extract of *Azadirachta indica* in barley (Paul and Sharma, 2002), *Acalypha indica* in ginger (Ghosh and Purkayastha, 2003), *Reynoutria sachaliensis* in cucumber (Daayf e*t al.,* 1995). Thus it is important for plants for early detection of pathogen and early delivering elicitors/induces (intracellularly/ intercellularly) to plant for activation of defense machinary (Shibuya and Minami, 2001) for management of crop diseases.

Pathogenesis–related proteins (PR-proteins)

Pathogenesis–related proteins (PR-proteins) may be considered as stress proteins which are produced in response of narcotizing pathogens (such as viruses, viroids, fungi and bacteria) with endogenous accumulation of signalling molecules, salicylic acid (SA) and thought to be defensive against further pathogenic infection (Van Loon, 1999). However, in contrast to most of the other types of stress proteins, they accumulate in plant tissues in a certain level that can be easily detectable on gels by general protein staining. The pathogenesis related proteins (PR-proteins) are grouped into families by amino acid homology, serological relationship and biochemical properties (Van loon *et al*.,1999). For example, PR-5 family exhibits the degree of homology of amino acid sequences to the sweet tasting thaumatins from *Thaumatococcus daniellii* (Dudler *et al*., 1994). Total 14 families of PR-proteins with its type member and function are reported by Van Loon and Van Strien (1999) such as PR-1 (Tobacco PR-1a; unknown function), PR-2 (Tobacco PR-2; β-1,3-glucanase), PR-3 (Tobacco P, Q; chitinase type I, II, IV, V, VI, VII), PR-4 (Tobacco 'R'; chitinase type I, II), PR-5 (Tobacco S; thaumatin-like), PR-6 (Tomato Inhibitor I; proteinase-inhibitor), PR-7 (Tomato P_{69}; endoproteinase), PR-8 (Cucumber chitinase; chitinase type III), PR-9 (Tobacco 'lignin-forming peroxidase'; peroxidise), PR-10 (Parsley 'PR1'; 'ribonuclease-like'), PR-11 (Tobacco class V chitinase; chitinase, type I), PR-12 (Radish Rs-AFP3; defensin), PR-13 (Arabidopsis THI2.1; thionin) and PR-14 (Barley LTP4; lipid-transfer protein). Among PR-proteins, two plant hydrolases, β-1,3 glucanase (PR-2) and chitinase (PR-3, PR-4, PR-8 and PR-11) are given special importance for their potential role to degrade fungal cell wall components because many pathogenic fungi contain β-1,3 glucans and chitin as major structural component of cell wall (Wessels and Sietsma, 1981; Bishop e*t al.*, 2000; Arlorio e*t al.*, 1992; Mauch e*t al.*, 1988) resulting in growth inhibition of fungi. Chitin is a linear polymer of β-1,4-glycosidic linked N-acetylglucosamine residues that is the predominant constituent of fungal cell walls, nematode eggs, and mid gut layers of insects. Chitinase was constitutively expressed at low level in plants but were dramatically enhanced by abiotic stresses such as ethylene, salicylic acid, salt solution, ozone and uv light (Brederode *et al*.,1991; Collinge *et al*.,1993; Ernst *et al*.,1992; Mauch F *et al*.,1989) and by biotic factors such as fungi, bacteria, viruses, viroids, fugal cell wall components and oligosaccharides (Rederode *et al*.,1991). The lytic

enzymes chitinase and β-1,3 glucanase play an important role in biological control by degrading chitin and β-glucan polymers from the cell walls of fungal pathogens (Sivam and Chet, 1989;). Several lines of evidence indicate that chitinases play a distinct role in plant defense by degrading chitin and β-1-3-glucanases by hydrolysing β-1, 3-glucans which form the matrix in which chitin is embedded (Lawrence, *et al*., 1996).

NahG (salicylate hydroxylase)

Transgenic *Arabidopsis* and Tobacco with the gene *NahG* (salicylate hydroxylase) from *Pseudomonas putida* which is responsible for degradation of salicylic acid (SA) to catechol resulting in the incapable of PR-proteins expressing in response to pathogen attack (**Figure-1)**. It indicated that SA acts as an endogenous signal for the induction of systemic acquired resistance (Gaffney *et al*., 1993). It is further reported that *NahG*-containing plants are more susceptible to a variety of fungal, bacterial and viral pathogens (Delaney *et al*., 1994). Thus, SA is required for the expression of acquired resistance in plant tissue in the form of SAR (Ryals *et al*., 1994, 1996). Salicylate is synthesized through phenylpropanoid dependent pathway from cinnamic acid (Yalpani *et al*., 1993). Accumulation of SA is required for the development of SAR and it is transported from infected leaves to distal part of plant (Shulaev *et al*., 1995; M¨olders *et al*., 1996).

NPR1

As the salicylic acid is an important signal molecule in plant defense mechanism and its signal is regulated by at least two mechanisms: one is NON-EXPRESSOR or PR1 (NPR1) dependent gene and a second that is independent of NPR1 (Shah, 2003). SAR induction results in the accumulation of the elicitor signal molecule salicylic acid (SA), which induces defense gene expression via activation of NPR1. In the nucleus, NPR1 regulates PR gene expression through physical interactions with transcription factors, TGA-binding promoter element, *as-1* required for SA-induced expression of PR-1. NPR1 itself contains no bona fide DNA-binding domains but rather protein–protein interaction domains (Lebel *et al*., 1998). NPR1 activation is regulated by its oligomeric structure through inter-molecular disulfide bonds. Upon SAR induction, a biphasic change in cellular reduction potential occurs which result in the reduction of NPR1 oligomeric structure to monomeric form in the nucleus which led to the expression of SAR genes and PR-proteins. A mutation of Cys82 or Cys216 in NPR1 leads to constitutive monomerization of the mutant NPR1 led to constitutive gene expression of SAR. In the systemic signalling transduction network, SNI1 (suppressor of NPR1) is a negative regulator of SAR (Lebel *et al*., 1998).

Mitogen Activated Protein Kinase 4 (MAPK4)

Another recently reported mutant, mpk4 shows elevated salicylic acid (SA) concentrations in the absence of spontaneous necrotizing lesions in Arabidopsis (Petersen *et al*., 2000). This recessive mutation in the MITOGEN ACTIVATED PROTEIN KINASE4 (MAPK4) gene results in the constitutive accumulation of SA which led to SAR and PR-proteins expression. Thus, the wild-type MAPK4 is characteristic to be a negative regulator of SAR gene expression and a positive regulator of ISR. A mutant,edr1 (enhanced disease resistance 1) has a defective activity of MAPK4 kinase that shows enhanced SA- and NPR1-dependent resistance to *P. syringae* and *Erysiphe cichoracearum* (Frye *et al*., 2001). Constitutive expression of active MAPK kinase (NtMEK2) in tobacco plant results in the activation of two MAPKs: salicylic-acid-induced protein kinase (SIPK) and wound-induced protein kinase (WIPK) that lead to the expression of phenylalanine ammonia lyase (PAL), the first enzyme in the phenylpropanoid pathway that ultimately cause cell death (Yang *et al*., 2001; Bent *et al*., 2001).

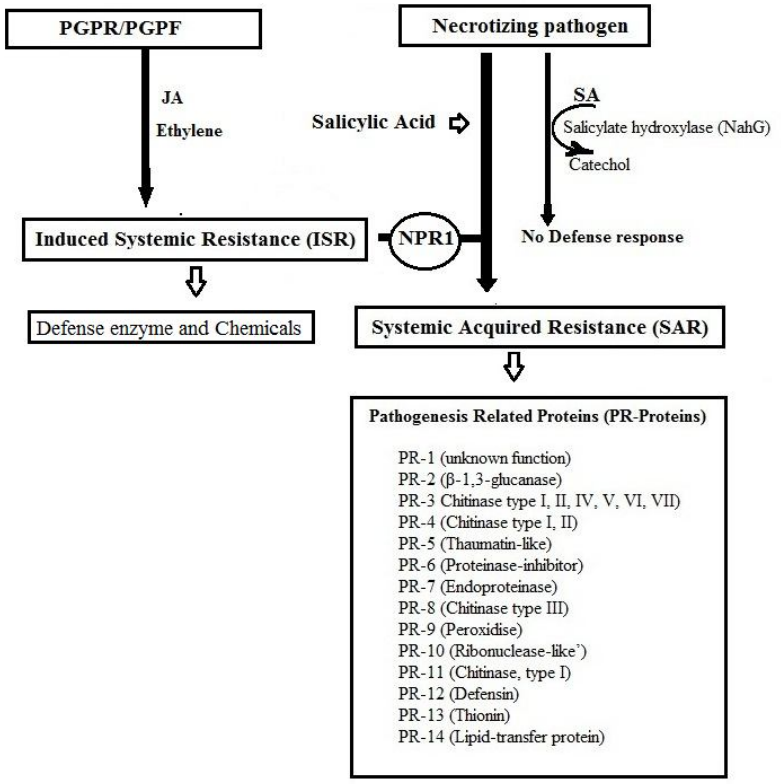


Figure-1: Molecular level plant defense related signal transduction pathway: Narcotizing pathogen mediated signalling pathway, Systemic Acquired Resistance (SAR) which require Salicylic Acid (SA) as endogenous elicitor signalling molecules and involved in the expression of Pathogenesis-related proteins (PR-proteins) is regulated by NahG and NPR1.

Table-1. Relationship between the mechanisms of Systemic acquired resistance (SAR) and Induced Systemic resistance (ISR)

Characteristics	SAR	ISR
Differences		
1. It is mediated by	Necrotizing pathogen	Plant growth promoting rhizobacteria (PGPR) or Plant growth promoting fungi (PGPF)
2. It requires signalling molecule	Salicylic acid (SA) response	Jasmonic acid (JA) and Ethylene (ET) response
3. Its expression lead to the production of	Pathogenesis related proteins (PR-proteins) only.	Diverse range of enzymes and defensive chemicals such as Phenylalanin ammonia lyase (PAL), Peroxidase (PO) and Polyphenol oxidase (PPO), Chalcone synthase phytoalexin, phenolic compound etc.)
4. Mechanism of defense	Direct inhibitory activity (such as lysis of fungal cell wall) against pathogenic agents	Indirect inhibitory activity (such as structural and chemical barrier) against pathogenic

		agents
5. NahG and NPR1	It is regulated by NahG and NPR1	Independent
6. It is used by the term	SAR	ISR
7. MAPK4 regulation	Negative regulator of SAR gene expression	Positive regulator of ISR gene expression
Similarities	Ability to repel subsequent pathogenic attacks. Induction of nonspecific basal resistance. Systemic and prolonged effect against pathogen. Activation is triggered by external application of inducers. The strength and stability of the resistance for several weeks may be influenced by various climatic and edaphic factors.	

CONCLUSION:

SAR is effective against a broad range of pest and pathogen such as fungi, bacteria, insect pest, virus and nematodes. It requires SA for its signal transduction. Expression of regulatory gene (NPR1) leads to the switch on the pathway of SAR and PR-proteins.

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