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Systemic acquired resistance and salicylic acid: current state of knowledge

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Abstract

Plants can induce defense reactions to a broad range of pathogens as a result of prior exposure to pathogens, various chemicals or physical stress. Induced resistance is expressed locally, at the site of the infection or systemically, at sites remotely located from the initial infection. The reactions occurring locally in the inducer leaf, the systemic signal and reactions in the upper leaf will be briefly reviewed here, with a special emphasis on the role played by salicylic acid in this process.

Introduction

Plants are defended against pathogens by constitutive and inducible barriers. Induced resistance may be expressed locally at the site of infection as well as systemically. The potential of plants to react to an invader by triggering local and systemic responses was first described by phytopathologists such as Carbone and Arnaudi (1930), Chester (1933) and Gäumann (1946). It was explained by the production of a signal released from the infected leaf and translocated to other parts of the plant where it induces defense reactions. This form of induced resistance was called systemic acquired resistance (SAR). It emphasizes the power of plants to acquire a state of general resistance after an initial infection. The term was first coined by Ross to describe induced resistance in the upper leaves of tobacco plants which had developed necrotic lesions on the lower leaves after inoculation with tobacco mosaic virus (TMV) (Ross, 1966). Non-pathogenic root-colonizing bacteria have also been found to induce resistance in leaves (Pieterse and Van Loon, 1999). This phenomenon was described as systemic induced resistance to differentiate it from resistance induced after inoculation with leaf pathogens. To avoid confusions with terminologies, it was agreed by the participants of this meeting that the terms SAR and ISR are synonymous, and can be used by an appropriate prefix such as 'rhizobacteria-induced-' or 'pathogen-induced-' to adequately describe the phenomenon.

The work of Ross was taken up and many other groups observed pathogen-induced-SAR in a diversity of plants belonging to all classes of higher plants (Sticher et al., 1997). Kuć and his coworkers have extensively described SAR in cucumber and documented the broad spectrum of SAR. These studies made it clear that SAR was independent of the nature of the initial inoculant (Madamanchi and Kuć, 1991). The biochemical nature of the changes induced in infected plants became an important target of research for several groups. This led to the discovery of a number of proteins termed PR- or pathogenesis-related proteins (Van Loon, 1997; Van Loon and Van Strien, 1999). The simple phenolic metabolite salicylic acid (SA) was found to induce PRs in tobacco and treatments of tobacco with SA can protect the plant against TMV. Later, SA was shown to be produced by plants locally, at the site of infection, but was also found in the phloem sap and in uninfected systemic leaves, making SA a possible signal for SAR (Sticher et al., 1997). These observations unleashed impressive amounts of studies to understand the role of SA in SAR.

Reactions in the inducer leaf

Generally, the success of the induced defense mechanisms depends on the outcome of the race between the invading pathogen and the reactions of the plant. In compatible interactions, the virulent pathogen is often recognized too late and the plant will be infected. In the case of avirulent pathogens, plants rapidly recognize the microbe and induce resistance mechanisms which act very efficiently against the invader. Induced mechanisms include modifications of the cell wall (Hammerschmidt, 1999a), production of phytoalexins (Hammerschmidt, 1999b), synthesis of PR proteins (Hunt and Ryals, 1996; Van Loon, 1997; Van Loon and Van Strien, 1999), or activation of programmed cell death, also called the hypersensitive reaction (HR), (Gilchrist, 1998; Grant and Mansfield, 1999; Lamb and Dixon, 1997; Morel and Dangl, 1997). HR is mostly associated with specific recognition of an avirulent pathogen by the host during a so-called gene-for-gene interaction (Bonas and Van den Ackerveken, 1999; Ellis et al., 2000; Hammond-Kosack and Jones, 1997).

The sequence of reactions taking place in a leaf undergoing an initial attack by a pathogen has been extensively studied using mutants of Arabidopsis. A summarized tentative and personal representation of some of the results using this approach is shown in Figure 1. After initial recognition of the pathogen by the plant, a cascade of early events, that include ion fluxes, phosphorylation events, generation of nitric oxide and active oxygen species, is induced. SA acts as a secondary signal molecule, the level of which increases during this process. It is likely, but has not been formally demonstrated, that this increase results from an increased expression of SA biosynthetic enzymes. Increased expression of such enzymes might not be induced by SA but by another earlier signal. SA in turn is required for the increased expression of various defense-related proteins such as the PRs. On the left side of Figure 1 it can be seen that the signal transduction pathway takes a different course depending on the nature of the interaction (virulent versus avirulent pathogen, rhizobacteria). A further level of complexity exists among the incompatible interactions

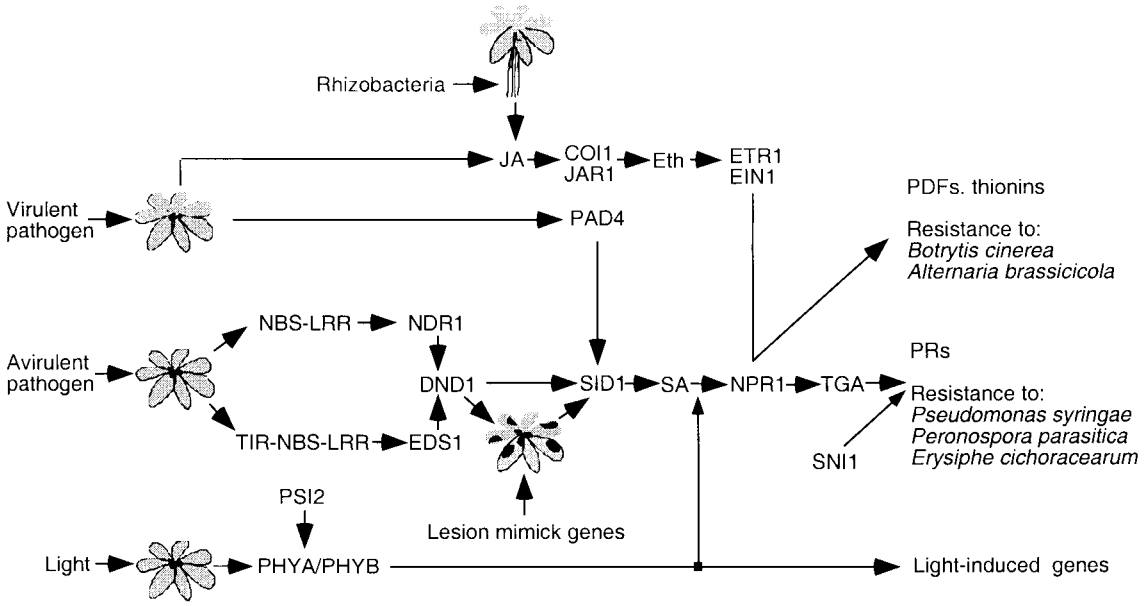

Figure 1. Schematic, tentative and simplified diagram of the signal transduction network operating in SAR. In this diagram, arrows represent the flow of the signals and proteins are ordered from the incoming signals (left side) to the responses (right side). Abbreviations: COI: coronatine insensitive; DND: defense no death; EDS: enhanced disease susceptibility; ETR: ethylene resistant; EIN; ethylene insensitive; JAR: jasmonate resistant; NBS-LRR: nucleotide-binding site leucine-rich-repeat protein; NPR: non-expresser of PR genes; NDR: non-race-specific disease resistance; PAD: phytoalexin deficient; PHY: phytochrome; PSI: phytochrome signalling; SID: salicylic acid induction deficient; SNI: suppressor of *NPR1*-1, inducible; TGA: basic leucine zipper (bZIP) transcription factor; TIR-NBS-LRR: Toll-interleukin-receptor nucleotide-binding-site leucine-rich-repeat protein.

where the pathway shows a dependency on either one of two classes of leucine-rich-repeats proteins (LRRs) (Young, 2000) (Figure 1). The DND (defense no death) protein was found to control the formation of HR cell death (Yu et al., 1998). Interestingly, the *dnd* mutants exhibit constitutive immunity with accumulation of high levels of SA and enhanced expression of *PR-1*. In *dnd* mutants, the defense response to pathogens can be disconnected from lesion formation (Figure 1). The *DND* gene encodes a cyclic nucleotide gated ion channel (Bent et al., 1999; Kohler et al., 1999). The importance of ion fluxes for plant defense response has already been recognized and it might well be possible that DND controls ion fluxes involved in HR formation.

Resistance against a given pathogen might also be activated via a different signal transduction pathway. For example, infection with leaf pathogens or with some rhizobacteria strains induces resistance to *P. syringae* via different pathways, the former involving SA, the latter involving ethylene and jasmonic acid (Pieterse et al., 1998). The complexity of these signalling pathways is further illustrated by the occurrence of crosstalk or interference between pathways (Genoud and Métraux, 1999). For example, both the induction of PR-1 and the resistance to *P. syringae* in Arabidopsis show a strong dependency on light. The *phyA* and *phyB* light receptor mutants or the double mutants *phyA/phyB* are strongly impaired in SA-induced PR-1 and resistance. This effect is not the result of a lack of a photosynthetic carbon source, since dark-grown plants fed with sugars show the same phenotype. Light seems to enhance the sensitivity of the tissue to its own SA rather than stimulate SA production (Genoud and Métraux, unpublished results) and the light pathway connects to the SA pathway after SA (Figure 1). Studies are under way to identify the elements integrating the signals from the light and the SA pathways. Another example of crosstalk is given by plants after rhizobacteria infection. In this case, the NPR1 protein is recruited, that otherwise is part of the SA pathway (Pieterse et al., 1998) (Figure 1).

Given the central role of SA in pathogen-induced signalling for induced resistance, studies have been directed at understanding the regulation of its production and its molecular mode of action. Many studies indicate that SA is produced from phenylalanine via coumaric and benzoic acid (reviewed in Sticher et al., 1997). The exact precursor of SA is still a matter of debate (Coquoz et al., 1998; Schalk et al., 1998) and the enzymes regulating SA biosynthesis have neither been identified nor isolated. Radioactive feeding experiments used to study the biosynthetic pathway for SA are often hampered by insufficient labelling of SA. Labelled precursors fed to plant tissues are either rapidly conjugated or, as in the case of phenylalanine, mostly directed to the lignin biosynthetic pathway. In tobacco, piperonylic acid, an inhibitor of coumarate 4-hydroxylase, was used to channel the label into the SA pathway (Schalk et al., 1998), but this inhibitor does not seem to operate efficiently in Arabidopsis (Nawrath and Métraux, unpublished results). More work is needed to understand the regulation of SA and its localisation after pathogen attack both locally and systemically. The search for mutants impaired in SA biosynthesis might be an alternative way to discover enzymes involved in SA biosynthesis. The *eds5/sid1* and *sid2* mutants impaired in SA accumulation after pathogen attack represent interesting candidates and the function of these genes is been actively sought (Nawrath and Métraux, 1999).

The mode of action of SA has been investigated by the search for a possible SA binding protein. SA was initially proposed to bind to catalase and ascorbate peroxidase (Chen et al., 1993; Durner and Klessig, 1995). The binding of SA to such enzymes might lead to the formation of a phenolic radical involved in lipid peroxidation. Lipid peroxidation products can activate defense gene expression (Farmer et al., 1998). It still remains to be shown whether sufficient lipid peroxides are formed by such phenolic radicals at the right time frame and right place for the defense response to take place. Other SA binding proteins (SAPs) were identified that show a higher affinity for SA and related functional analogues than catalase (Du and Klessig, 1997; Klessig, report at this meeting). While the relevance of these SAPs remains to be determined, they certainly offer exciting perspectives for understanding the mode of action of SA. Responses induced by SA include transcriptional activation of genes. For instance, a SA-inducible protein kinase (SIPK) belonging to the MAP kinase family was identified in tobacco (Zhang and Klessig, 1997). SIPK is also induced upon infection and is likely to be part of the chain of events taking place downstream of SA. A number of studies have concentrated on the upstream regulatory sequences of the *PR-1* gene, one of the culminating responses in SAR. One indispensable regulatory element for SA-induced *PR-1* expression is a consensus sequence (TGACG) for Arabidopsis, TGA proteins belonging to the plant bZIP transcription factors were shown to bind to the TGACG in the *PR-1* promoter. TGAs were also shown to interact physically with NPR1 providing a direct link between

NPR1 and SA-induced *PR-1* expression (Zhang et al., 1999; Despres et al., 2000; Zhou et al., 2000). A further level of complexity became apparent following the discovery of SNI1, a negative regulator of SAR. SNI1 represses *PR* gene expression, presumably by direct binding to a specific DNA sequence or via a transcription factor (Li et al., 1999). Other reports have identified a SA- and pathogen-inducible WRKY DNA-binding factor. This factor specifically recognizes the elicitor response element of the tobacco class I chitinase promoter. Protein phosphorylation is important for binding activity of WRKY DNA-binding factors emphasizing the role played by kinases in signalling (Yang et al., 1999).

An important event for the expression of SAR is the production of a systemic signal. Clearly, the systemic signal for SAR is the result of reactions taking place in the primary leaf after a first inoculation but its chemical nature remains uncertain. The possibility was raised that this signal is SA but the data supporting this are not completely convincing (see below). Therefore the localisation and the positioning of the primary systemic signal in the systemic pathway remains unknown.

The systemic signal

The importance of SA in SAR induction was first supported by correlations between the timing of SA accumulation and its endogenous concentration in relation to the appearance of resistance in the upper leaf. Experiments using temperature shifts also support this hypothesis (reviewed by Sticher et al., 1997). The importance of SA for SAR was also demonstrated using transgenic plants which overexpress a bacterial salicylate hydroxylase gene (the *NahG* gene) (Delaney et al., 1994; Gaffney et al., 1993). Studies using various Arabidopsis mutants also support the importance of SA for SAR (Glazebrook, 1999). The role of SA as a systemic signal was assessed using leaf detachment assays and various grafting experiments between NahG, PAL-suppressed, choleratoxin-producing plants and wild type plants (Sticher et al., 1997). These experiments all show that SA is necessary for the induction of SAR, but that a signal other than SA can be translocated to an upper leaves and induce resistance. However, several reports indicate that SA produced in a lower leaf during infection can be transported to the upper leaf before appearance of systemic resistance in that leaf (Mölders et al., 1996; Shulaev et al., 1995). Results presented at this meeting by Robert Darby and colleagues using transgenic tobacco expressing the SA hydroxylase gene targeted to the phloem showed strong inhibition of SAR. Thus, it appears that both SA as well as other systemic signals could be involved in SAR. More work is needed to elucidate this apparent redundancy. Mutants impaired in the systemic transmission of SAR might offer a good possibility to gain more insight in the process. Using a screen based on impaired SAR, two Arabidopsis mutants impaired in SAR have been identified. In uninduced plants, both mutants showed the same level of resistance as wild type plants, making it likely that the production, transport or perception of the systemic signal is affected (Mauch-Mani et al., unpublished results). During this meeting, Robin Cameron described a mutant defective in induced resistance (*dir1*) blocked in the ability to deploy SAR. The mutation occurred in a lipid transfer protein (LTP). Petiole exudates from wild type leaves collected 12 h after inoculation with an avirulent strain of *P. syringae* could induce resistance after introduction into leaves of both wild type and *dir1* mutants. Exudates from inoculated *dir1* mutants were unable to induce resistance in wild type plants. Thus, *dir1* perceived but did not transmit a systemic signal moving in leaf exudates. Possibly, LPT might be the systemic signal or might be closely associated with the formation of a systemic signal. These results show the power of the genetic approach and we are looking forward to hear more about the systemic signal.

Reactions in the systemic tissue

Systemic responses can be clearly separated from the reactions taking place in the infected parts of the pretreated plants. As shown by many studies, the upper leaves of plants inoculated locally have elevated levels in PRs. In this case, the systemic signal has triggered defense-related reactions before contact with the challenging pathogen. In contrast, other reactions such as changes in cell wall lignification were only detected after challenge infection of the upper leaf but with faster induction kinetics (reviewed in Sticher et al., 1997). In this case, the systemic signal has conditioned the tissue to a faster response. One line of evidence for conditioning has been provided using cultured cells pretreated with SA or functionally related inducers prior to elicitor exposure. Such pretreatments potentiate the elicitor-induced expression of defense-related phenylpropanoid genes such as *phenylalanine ammonia-lyse* (*PAL*) *or 4-coumarate:CoA ligase*. In the

same tissue, other genes not directly related to defense such as *mannitol dehydrogenase* or *anionic peroxidase* are induced directly by SA (Thulke and Conrath, 1998). SA as well as functional analogues such as 2,6-dichloroisonicotinic acid (INA) or benzothiadiazole (BTH; BION) appear to have a dual function by inducing directly or potentiating elicitor-induced genes (Thulke and Conrath, 1998; Katz et al., 1998). Observations were also made in cucumber hypocotyls, where pretreatments of the seedlings with INA or BTH lead to increased competence for elicitor-induced H_2O_2 generation. In this case, either gentle abrasion or germination of fungal spores on the epidermal surface was required for potentiation by INA or BTH (Kauss et al., 1999). Presumably, all these results are pertinent to the situation in a non-infected upper leaf of a plant infected on the lower leaf. Results on the conditioning of whole plants were presented at this meeting by Uwe Conrath. Pretreatment of Arabidopsis with SA or BTH (BION) increases the sensitivity to *Pseudomonas syringae*-induced activation of the *PAL* gene (Conrath et al., 2001). Future experiments should now be aimed at the action of the systemic signal in conditioning of defense in systemic leaves.

Conclusions

Much progress has been achieved in the study of SAR. An increasing number of new elements in the signal transduction pathway have been discovered and their number will undoubtedly increase with the advent of large-scale investigations of gene expression. Clearly, to understand how proteins encoded by the novel gene products operate and interact, the general interest in this field will swiftly move to the biochemical level to reach a functional understanding of SAR. Among the fascinating questions on SAR are those concerning the systemic signal of SAR and its regulation and mode of action. SA was implied in this process initially, and its role as a key signal in SAR process was later confirmed. Doubts were then cast on its function as a translocated systemic signal, but reports presented by the Darby and Draper group at this meeting invigorate the initial hypothesis. I hope that in the near future more can be learned about the regulation of SA synthesis and its mode of action. The signal transduction involved in the regulation of the SAR response turns out to be far from a linear chain of events. Several pathways interact, leading to sets of responses targeted at specific pathogens. Understanding and representing the structure of the SAR signalling network becomes a task that will require biological knowledge and a good deal of computational flair. We proposed the use of Boolean networks to apprehend the complexity of signalling systems (Genoud and Métraux, 1999). Further progress in this area undoubtedly will require collaborations between biologists and specialists in informatics.

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