Elements of signal transduction leading to activation of plant defense

Jacek Hennig
Institute of Biochemistry and Biophysics
Polish Academy of Sciences
Warsaw

1. Introduction

When a plant is infected with a pathogen, the outcome primarily depends on whether the host plant can prevent growth or spread of that pathogen. Plants which are unable to prevent pathogen ingress are referred to as susceptible, and they frequently become systematically infected. This susceptibility may be caused by the inability of the plant either to recognize the pathogen or to mount an effective defense response. In contrast, plants capable of restricting pathogen replication and movement to the initial site of infection are termed resistant. Resistance may be passive, when there is no appropriate environment for pathogen growth; this form of resistance is often called non-host resistance.

Alternatively, resistance may be an active response involving the induction of a wide variety of defense responses that prevent pathogen colonization (42). Animals possess an immune system which allows them to develop systemic, long-lasting and highly specific resistance to invading pathogens. Following infection with a pathogen, plants can also establish a long-distance, systemic, although non-specific resistance to pathogens which is called systemic acquired resistance (SAR). Many different processes are associated with an active resistance response, such as the strengthening of cell walls through increased synthesis and deposition of hydroxyproline-rich glycoproteins, callose, lignin and other phenolic compounds. Increased peroxidase activity which is required for lignification and possibly for cell wall protein cross-linking is also observed.

Additionally, phytoalexins, which are low molecular weight compounds with anti-microbial activity, and the phenylpropanoid pathway enzymes involved in phytoalexin synthesis are rapidly induced. There also occurs a dramatic increase in the level of reactive oxygen species (ROS), known as an

oxidative burst. All these responses occur very rapidly (within minutes to a few hours) after infection. On the other hand, several other defense responses are induced many hours or even days after infection. These latter responses may include the induction of a variety of proteins, commonly referred to as pathogenesis-related (PR) proteins. Some of these proteins are hydrolytic enzymes (chitinases and ß-1,3-glucanases), proteinase inhibitors and, as yet poorly characterized, anti-viral activities. In addition, a more visible manifestation of the resistance response, called the hypersensitive response (HR), appears. The HR is characterized by the formation of necrotic lesions on the infected tissue, leading to the restriction of pathogen to the cells within or immediately surrounding these lesions (for reviews see (13, 36)).

2. Genetic analysis of disease resistance

Several plant resistance genes have recently been isolated and further characterization of the encoded proteins should help elucidate the early signaling events that activate the defense responses. The *Hm1* resistance gene from maize encodes a NADPH-dependent HC-toxin reductase, which inactivates the HC-toxin produced by virulent races of the fungal pathogen *Cochliobolus carbonum* (28). The *Pto* gene from tomato, which confers resistance to the bacterial pathogen *Pseudomonas syringae* pv. tomato encodes a serine/threonine kinase (41). While these two resistance genes are distinct, other recently isolated resistance genes exhibit one or more common features. Both the tobacco *N* gene (confers TMV resistance) and the *Arabidopsis thaliana Rps2* gene (determines resistance to *Pseudomonas syringae* strains containing the *avrRpt2* gene) encode a P-loop which binds GTP or ATP and leucine rich repeats motif (LRR) which may be involved in protein-protein interaction. In addition, these genes exhibit approximately 24% sequence identity and 50% similarity (3,44,55). The discovery that the viral and bacterial resistance genes are so highly conserved suggests that resistance to diverse pathogens may involve similar signaling mechanisms. This observation is supported by the discovery that the *Cf9* gene of tomato, which specifies resistance to strains of *Cladosporium fulvum* carrying the *avr9* gene, encodes LRR (29), while the L^6 gene of flax, which confers resistance to *Melampsona lini*, encodes both P-loop and LRR (33).

The isolation and characterization of mutant plants expressing aberrant defense responses will also help to identify and order the components of the signal transduction pathway(s). *A. thaliana* mutants that fail to accumulate PRs proteins after chemical induction or pathogen treatment have been generated (7). Analysis of one of these mutants (*npr1*) indicates that it fails to develop SAR after infection by an avirulent strain of *P. syringae* (7). *Arabidopsis* mutants that constitutively express PR genes in the absence of pathogen or chemical inducers have also been identified (5). The *cpr1* mutant, which constitutively expresses PR genes, contains a recessive mutation that causes elevated SA level and enhanced resistance to several pathogens. These

properties contradiet the hypothesis that the *cpr1* gene product acts as a negative regulator in the defense signaling pathway (5).

The study of the plants which spontaneously form lesions in the absence of pathogen should also help to understand how the HR and SAR are triggered. The spontaneous lesion forming phenotype was first observed in maize (19) and since then it has been documented in tomato (32), barley (57) and *Arabidopsis* (16,25). Detailed analysis of several A*rabidopsis* mutants has demonstrated that plants developing spontaneous lesions exhibit many characteristics associated with the HR, including increased defense gene expression and phytoalexin synthesis. The appearance of lesions is also correlated with the increase in salicylic acid (SA) level and defense gene expression in non-lesion bearing leaves (25). In addition, plants actively forming lesions exhibit SAR in response to bacterial and fungal infection (16,25). The discovery that mutations in specific genetic loci cause the induction of several HR-associated responses suggests that HR could be the result of genetically programmed cell death. In mammalian systems, programmed cell death, or apoptosis, is carefully regulated and occurs in specific cells during development or after pathogen infection (for review, see (51)).

Recent studies have suggested a link between ROS and mammalian apoptosis. Since the HR in plants is also associated with increases in ROS and lipid peroxidation, it is possible that related mechanisms lead to apoptosis in mammals and the HR in plants. Further analysis of the *Arabidopsis* mutants should help to determine whether a homologous component in the pathway leading to programmed cell death and SAR activation in plants exists.

3. Nature of SAR

Within hours to a few days after the appearance of the HR, a long-distance, broad-based resistance to attack by a variety of pathogens is usually developed in both the infected leaf (local acquired resistance) and throughout the uninfected portions of the plant (systemic acquired resistance) (12). SAR has been demonstrated in a wide variety of plant species in response to infection with bacterial, fungal and other viral pathogens. While SAR is usually associated with an active resistance response, certain compatible plant-pathogen interactions (in which the plant is susceptible to the pathogen) can also lead to SAR (31).

Thus, it is so far unclear which components of the interaction between plant and pathogen are necessary to trigger SAR. The efforts to elucidate the biochemical basis for resistance, including SAR, have led to the identification of a variety of proteins belonging to the PR family, which are induced after pathogen attack. In tobacco the coordinated induction of five or more unrelated classes of PR proteins is associated with the development of the HR after TMV infection (for reviews, see (13,36)).

A subset of genes encoding these PR proteins, designated SAR genes, is

also induced in the upper, uninfected leaves at the time of SAR development (53). PR proteins have since been identified and their genes isolated from a large number of plant species. In general, their induction is correlated with local and systemic resistance to fungal, bacterial and viral pathogens. Recent evidence has demonstrated that PR proteins are not only convenient markers for SAR but also effective anti-microbial agents. Some PR-3 proteins exhibit lysozymal as well as chitinase activities, suggesting an anti-bacterial or anti-fungal function (4). In addition, PRs 2, 3, 4 and 5 inhibit fungal growth *in vitro* (43,46,56). The over expression of PRs 1, 2, 3 and 5 in transgenic plants also enhances resistance to several fungal pathogens (1,6,37,61). Thus, the induction of PR proteins may directly contribute to SAR development and maintenance, as well as reduction of pathogen growth and spread during the initial infection. However, no SAR-associated anti-viral activities have been identified which indicates that many of the components involved in SAR are yet to be discovered. The ability of a plant to develop SAR after the infection of a single leaf indicates a requirement for a signal that is mobile over long distances (31). Over the years, several long distance signals have been identified, such as ethylene, jasmonates, systemin. Below I will discuss the role of salicylic acid in plant pathogen interactions in a greater detail.

4. SA is required for SAR development

The first hint that SA was involved in SAR induction came from studies by White and coworkers (54). They observed that tobacco leaves treated with SA or acetylsalicylic acid (aspirin) exhibited both enhanced resistance to TMV infection and increased PR protein accumulation (2,54). SA treatment has since been shown to enhance resistance to bacterial, fungal, and viral pathogens in many plants. In addition, SA is able to induce PR protein accumulation in a wide variety of both monocotyledonous and dicotyledonous plants (for reviews, see (30)). Furthermore, it has been demonstrated that SA treatment of tobacco induces the same set of genes as those activated during SAR development following TMV infection (53). More recently, the hybrid *Nicotiana glutinosa* x *N. debneyi* has been shown to accumulate high levels of SA and to constitutively synthesize PR proteins. Following TMV infection, the lesions produced by these plants were approximately several times smaller than those observed on plants from the TMV-resistant tobacco cultivar Xanthi-nc, indicating that viral localization was extremely rapid and effective (60).

Although SA can act directly as toxin against some pathogens, such as *Colletotrichum falcatum*, *Fusarium oxysporum*, and *Agrobacterium tumefaciens*, it is not toxic to TMV or *C. lagenarium* (30). Thus, the enhanced resistance observed in these hybrid plants is most likely due to constitutive expression of the defense responses. Studies of tobacco and cucumber have provided more direct evidence that SA might be a signal for plant defense responses. By direct measuring of endogenous SA levels, it was determined that SA

concentrations increased 20- to 50-fold in the infected leaves of tobacco resisting TMV infection. As much as a 10-fold increase in SA was also observed in the upper uninfected leaves of these plants. In both cases, the SA increases were parallel to or preceded PR-1 gene activation. In contrast, when TMV-susceptible tobacco was infected, no increase in SA or PR genes expression was observed in either the infected or uninfected leaves (38). Cucumber cultivars resisting *C. lagenarium*, tobacco necrosis virus (TNV) or *P. syringae* infection also exhibited an increase in SA levels.

Increased SA level has also been shown to correlate with the response in *A. thaliana* after turnip crinkle virus (TCV) or *P. syringae* infection (30), and in tobacco following *Erwinia carotovora* infection (45). A 10- to 50-fold increase in SA was also observed in the *Solanum tuberosum* infected with potato virus Y (PVY) (27). The most direct evidence that SA is required for SAR comes from the analysis of genetically engineered tobacco and *Arabidopsis* plants which are unable to accumulate SA. The *nahG* gene from *Pseudomonas putida* encodes salicylate hydroxylase (SH), an enzyme that converts SA to catechol. Since catechol does not induce PR gene expression or enhance resistance, any alteration in the resistance levels of these plants after pathogen infection should theoretically be caused by the lack of SA. When tobacco synthesizing high levels of SH was infected with TMV, only a two- to three-fold increase in the level of SA was observed in the infected leaves compared to over 150-fold increase in untransformed control plants (24). The *nahG* tobacco was observed to accumulate PR-1 mRNA in its infected leaves, they failed to express PR genes in its uninfected leaves (52). In addition, neither the *nahG* transgenic tobacco nor *Arabidopsis* plants developed SAR after pathogen treatment (14,24). These transgenic plants also exhibited enhanced susceptibility to infection with pathogens that normally induced a resistance response (14).

These experiments strongly support the hypothesis that SA is a signal for SAR induction. SAR development must involve a long distance signal since the entire plant becomes resistant even when only a small portion has been infected. SA levels were shown to increase prior to PR-1 gene activation in the uninfected leaves of TMV-resistant tobacco (38). Based on early observations, SA was proposed to be this mobile signal. However, more recent results from other experiments have strongly suggested that SA is not the translocated SAR signal. Studies with the *nahG* transgenic tobacco have also confirmed that the translocated signal is not SA. Either wild type (wt) or *nahG* transgenic tobacco shoots (scions) were grafted onto wt or *nahG* rootstocks, producing four chimeric sets of plants. After infection of rootstock leaves, the chimeras were assayed for induction of SAR and PR-1 gene expression in the upper uninfected scion leaves. Regardless of rootstock origin, SAR was observed in all plants containing wt scions. Thus, even though *nahG* expressing rootstocks were unable to accumulate high levels of SA, they were as capable to generate the mobile signal for SAR as wt rootstocks. In contrast, all of the plants bearing *nahG* scions failed to exhibit SAR or

PR genes induction (52).

Taken together, these results suggest that SA is probably not the mobile signal for SAR. However, SA does appear to be required downstream of this signal for proper SAR development in the uninfected leaves.

5. SA Metabolism and Mechanism of Action

In plants, the precursor for SA is phenylalanine. Phenylalanine is converted to *trans*-cinnamic acid by phenylalanine ammonia-lyase (PAL), the first enzyme in the phenylpropanoid pathway. This pathway is rapidly induced after pathogen infection and is responsible for the synthesis of several de-

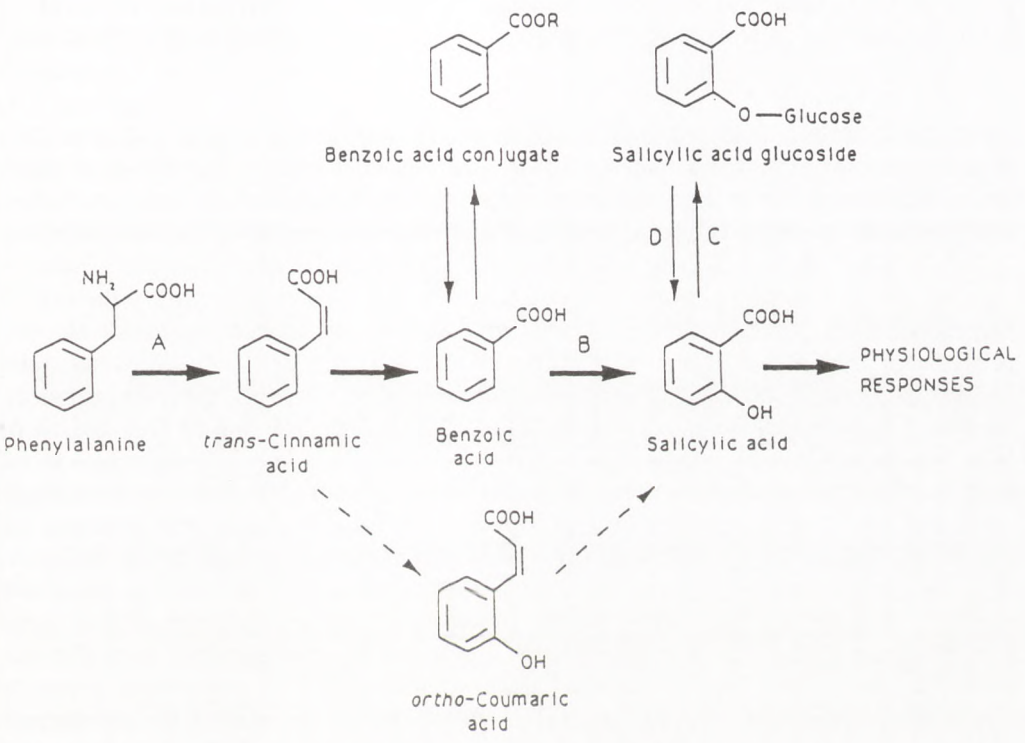

Fig. 1. Pathways of SA synthesis.

SA can be synthesized from phenylalanine through *ortho*-coumaric acid or benzoic acid (BA). In TMV-infected tobacco the induction of the BA intermediate (bold arrows) was observed. Both BA and SA can be converted to conjugated forms, which may serve as rapidly releasable sources of these compounds. The structure of the BA conjugate is unclear; an ester conjugate is shown as an example. After infection, SA level rises and induces a variety of physiological responses, including activation of PR gene expression and the development of SAR. The enzymatic steps A through D are catalyzed by phenylalanine ammonia-lyase, BA-2-hydroxylase, UDP-glucose:SA glucosyltransferase and SA ß-glucosidase, respectively.

fense-related compounds, such as phytoalexins and lignin, as well as SA. SA is subsequently synthesized from *trans*-cinnamic acid through one of two possible intermediates: *ortho*-coumaric acid (*o*) or benzoic acid (BA) (Fig.1). Different plants use either or both of these intermediates for SA synthesis. *Primula acaulis* utilize the *o*-coumaric acid intermediate (18). Tomato seedlings utilize both intermediates, with healthy seedlings synthesizing SA from BA and *Agrobacterium*-infected plants utilizing *o*-coumaric acid (8). In contrast, Yalpani et al. have demonstrated that healthy and TMV-infected tobacco primarily synthesize SA through BA (58). The enzyme activity responsible for converting BA to SA, BA-2 hydroxylase (BA-2H), is induced by increases in BA or TMV infection. This induction can be blocked by the addition of cycloheximide (34). Since both BA-2H activity and the resulting SA levels are directly proportional to BA levels, the rate limiting step for SA synthesis must occur further upstream, possibly at the level of BA formation. Analysis of SA levels in TMV-infected tobacco has indicated that a substantial amount of SA is converted into an acid hydrolyzable conjugate. Treatment of this conjugate with ß-glucosidase released free SA, demonstrating that the conjugate is SA ß-glucoside (SAG) (39). The enzyme responsible for conjugating SA, UDP-glucose:SA glucosyltransferase, has been characterized in tobacco (20) and several other plant species (59). In uninfected tobacco, the levels of both SA and SAG are very low. However, their levels substantially increase after TMV infection, with SAG becoming the predominant form in the infected leaves (21,39). SAG accumulation has also been documented in the uninfected leaves of resistant tobacco, cucumber, *Arabidopsis* (30) and potato (27) after pathogen infection. The role of SAG in the resistance response is currently unclear. After injection of chemically synthesized SAG into tobacco leaves, Hennig et al. (26) observed rapid induction of PR-1 gene expression. However, this expression was most likely due to the release of free SA from SAG by a non-specific cell wall-associated ß-glucosidase (9). While SAG itself may not be biologically active, it could serve a valuable role as a storage form for SA. Conjugation to sugars appears to be a common mechanism for plants to safely store large amounts of otherwise toxic or highly active compounds, including hormones (30). In addition, this way of storage does not need to be irreversible. Through de-conjugation, an active form of the compound can rapidly be mobilized. Recent reports have suggested that the activity of giberellin (49), cytokinin (22), and auxin (23) is regulated through such mechanism. A conjugate of BA which is accumulated to significant levels in uninfected tobacco has been identified. Upon TMV infection, the level of this conjugate decreases and the levels of BA and SA concurrently rise, suggesting that it serves as a readily hydrolyzable storage form of BA (58). A similar storage role has been postulated for SAG (30).

After the primary infection, SAR is established and SAG is accumulated throughout the plant. Following the secondary attack, damage of infected cells could release cytosolic SAG into the extracellular spaces where the ß-glucosidase would hydrolyze it to free SA. This rapid release of SA might

superinduce defense responses at the infection site. In combination with the generalized increases in PR gene expression and enhanced resistance associated with SAR, this response suggests a possible mechanism for the extremely efficient restriction of pathogen that is the hallmark of SAR and local acquired resistance.

The isolation and characterization of a SA binding protein (SABP) has led to an understanding of how SA induces disease resistance. The SABP is a 240-280 kDa protein complex, containing at least one 57 kDa subunit. It binds only SA and those SA analogs which are capable of inducing resistance and PR gene expression. SABP's binding affinity for SA is 14 μM, consistent with the endogenous SA levels measured during the resistance response in TMV-infected tobacco leaves (10). Sequence analysis of a cDNA clone encoding the 57 kDa subunit has indicated that SABP is highly homologous to catalase. Final confirmation that SABP is a catalase came from the demonstration that highly purified SABP is able to convert H_2O_2 to H_2O and O_2 *in vitro*. Interestingly, SA was shown to inhibit the catalase activity of SABP *in vitro*. Biologically active SA analogs were also shown to inhibit catalase activity, whereas the inactive analogs did not (11). Treatment of tobacco leaves with SA caused significant increases in H_2O_2 level, similarly to those observed after addition of the catalase inhibitor 3-amino-1,2,4-triazole (3AT). Moreover, injection of H_2O_2 or 3AT into tobacco leaves activated PR-1 gene expression (11). Thus, the SA signal appears to be transduced through catalase via the inhibition of its enzymatic activity and the subsequent increase in H_2O_2 or other ROS derived from it. Increased levels of ROS might then serve as second messengers which induce PR gene expression and disease resistance. H_2O_2 has been proposed to be a second messenger for gene activation in mammalian systems as well as in plants, although its mechanism of action is currently unclear. In plants, increased H_2O_2 levels induce expression not only of defense genes, but also chilling tolerance genes (47). In mammals, elevated H_2O_2 concentrations activate the transcription factor NF-κB, which regulates the genes involved in immune and allergic responses (50).

Activation of the transcription factor AP-1 after UV irradiation of mammalian cells also requires increased H_2O_2 level (15). Thus, stress from a variety of sources appears to induce plant and mammalian gene expression via ROS. However, it seems that only plants can use SA to generate a H_2O_2 signal since SA-inhibitable catalases have been identified in several plant species but not yet in animals (11,48).

6. Signaling pathways leading to defense response

It has been known that treatment of a plant with certain chemicals can induce resistance. These chemicals include: L-α-amino butyric acid (α-AB), polyacrylic acid (PAA), thiamine-HCl, barium chloride ($BaCl_2$), 2,6-dichloroisonicotinic acid (INA) and SA. By analyzing the effect of these compounds on

PR expression and SA/SAG accumulation, Malamy et al. (40) have begun to dissect the defense signaling pathway. Except for INA, all of the tested compounds induce PRs accumulation via SA synthesis, which suggests that they enter the pathway upstream of SA. More precise studies have demonstrated that thiamine-HCl and PAA do not enter the pathway at the same point. Thiamine-HCl clearly requires SA for PR gene induction. PAA treatment causes SAG accumulation, several experiments suggest that PAA also enters the pathway in at least two points (downstream and upstream of SA) or induces PR expression through SA-independent and SA-dependent pathways. INA has recently been shown to directly bind and inhibit catalase, which explains its ability to induce resistance without stimulating SA/SAG accumulation. Comparison of the structures of biologically active SA and INA analogs has revealed several common features. SA and INA thus appear to share a similar mechanism for inducing PR expression and resistance through their ability to interact with catalase (11).

Taken together, these results demarcate 3 different points in the defense signaling pathway. Additional analysis with other chemical inducers should provide a more detailed picture of this pathway, and may help identify the step at which the mobile signal for SAR development is generated.

7. Final remarks and future prospects

Recent reports correlating the activation of defense responses with a variety of processes known to be involved in signal transduction in other systems are beginning to shed light on the mechanisms through which resistance is induced. Using a variety of plant-pathogen systems, researchers have been investigating the role of protein phosphorylation in activating the defense responses. In soybean suspension culture cells, Levine et al. (35) demonstrated that protein kinase inhibitors block the ability of elicitor treatment to induce both the oxidative burst and the H_2O_2-mediated activation of defense genes. In other experiments, it was shown that the phosphatase inhibitor okadaic acid inhibited lesion formation in tobacco after TMV infection and blocked the SA-mediated induction of PR-1 gene expression (17). Thus, changes in protein phosphorylation appear to be involved in transducing the resistance signal in several plant species. However, further research will be required both to resolve the discrepancies in the currently available data and to define the step(s) at which protein phosphorylation/dephosphorylation occurs. All together, the available results strongly support the idea that SA plays an important role as signal for disease resistance and SAR development. Furthermore, SA appears to transduce this signal, at least in part, by inhibiting catalase activity and generating increased level of ROS. These findings represent a significant advancement in our understanding of the signaling events leading to the disease resistance but the mechanisms through which the defense response signals are transduced and SAR is ini-

tiated need further analysis. In the next years we should expect exciting discoveries in basic and applied sciences leading us to understanding of how plants resist the pathogen attack.

I would like to thank the researchers who provided unpublished data for this review. This review was partially supported by KBN grant 6P20302006 to Jacek Hennig.

References

1. Alexander D., Goodman R. M., Gut-Relia M., Glascock C., Weymann K., Friedrich L., Maddox D., Ahl-Goy P., Luntz T., Ward E., Ryals J., (1993), Proc. Nat. Acad. Sci. USA, 90, 7327-7331.
2. Antoniw J. F., White R. F., (1980), Phytopath. Z., 98, 331-341.
3. Bent A. F., Kunkel B. N., Dahlbeck D., Brown K. L., Schmidt R., Giraudat J., Leung J., Staskawicz B. J., (1994), Science, 265, 1856-1860.
4. Boller T., Gehri A., Mauch F., Vögeli U., (1983), Planta, 157, 22-31.
5. Bowling S. A., Guo A., Cao H., Gordon A. S., Klessig D. F., Dong X., (1994), Plant Cell, 6, 1845-1857.
6. Broglie K., Chet I., Holliday M., Cressman R., Biddle P., Knowiton S., Mauvais C. J., Brogiie R., (1991), Science, 254, 1194-1197.
7. Cao H., Bowling S. A., Gordon A. S., Dong X., (1994), Plant Cell, 6, 1583-1592.
8. Chadha K.C., Brown S. A., (1974), Can. J. Bot., 52, 2041-2046.
9. Chen Z., Malamy J., Hennig J., Conrath U., Sánchez-Casas P., Silva H., Ricigliano J., Klessig D. F., (1995), Proc. Nat. Acad. Sci. USA, 92, 4134-4137.
10. Chen Z., Ricigliano J., Klessig D. F., (1993), Proc. Nat. Acad. Sci. USA, 90, 9533-9537.
11. Chen Z., Silva H., Klessig D. F., (1993), Science, 262, 1883-1886.
12. Chester K. S., (1933), Quart. Rev. Biol., 8, 275-324.
13. Cutt J. R., Klessig D. F., (1992), in: *Plant Gene Research, Genes Involved in Plant Defense*, Eds. F. Meins, T. Boller, Springer-Verlag, New York, 209-243.
14. Delaney T., Uknes S., Vernooij B., Friedrich L., Weymann K., Negrotto D., Gaffney T., Gut-Rella M., Kessmann H., Ward E., Ryals J., (1994), Science, 266, 1247-1250.
15. Devary Y., Gottlieb R. A., Lau L. F., Karin M., (1991), Mol. Cell Biol., 11, 2804-2811.
16. Dietrich R. A., Delaney T. P., Uknes S. J., Ward E. R., Ryals J. A., Dangl J. L., (1994), Cell, 77, 565-577.
17. Dunigan D. D., Madiener J. C., (1995), in: *Keystone symposium on signal transduction in plants*, (Hilton Head, SC, USA).
18. El-Basyouni S. Z., Chen D., Ibrahim R. K., Neish A. C., Towers G. H. N., (1964), Phytochem., 3, 485-492.
19. Emerson R. A., (1923), *The inheritance of blotched leaf in maize*, Cornell Univ. Memoir, 70, 303-307.
20. Enyedi A. J., Raskin I., (1993), Plant Physiol., 101, 1375-1380.
21. Enyedi A. J., Yalpani N., Silverman P., Raskin I., (1992), Proc. Nat. Acad. Sci. USA, 89, 2480-2484.
22. Estruch J. J., Chriqui D., Grossmann K., Schell J., Spena A., (1991), EMBO J., 10, 2889-2895.
23. Estruch J. J., Schell J., Spena A., (1991), EMBO J., 10, 3125-3128.
24. Gaffney T., Friedrich L., Vernooij B., Negrotto D., Nye G., Uknes S., Ward E., Kessmann H., Ryals J., (1993), Science, 261, 754-756.
25. Greenberg J. T., Guo A., Klessig D. F., Ausubel F. M., (1994), Cell, 77, 551-563.
26. Hennig J., Malamy J., Grynkiewicz G., Indulski J., Klessig D. F., (1993), Plant J., 4, 593-600.

27. Hennig J., Krzymowska M., (1995), in: *Keystone symposium on signal transduction in plants*, (Hilton Head, SC, USA).
28. Johal G., Briggs S., (1992), Science, 258, 985-987.
29. Jones D. A., Thomas C. M., Hammond-Kosack K. E., Balint-Kurti P. J., Jones J. D. G., (1994), Science, 266, 789-793.
30. Klessig D. F., Malamy J., (1994), Plant Molec. Biol., 26, 1439-1458.
31. Kuć J., (1982), Bioscience, 32, 854-860.
32. Langford A. N., (1948), Can. J. Res., 26, 35-64.
33. Lawrence G. J., Ellis J. G., Finnegan E. J., (1994), in: *Advances in Molecular Genetics of Plant-Microbe Interactions*, Eds. M.J. Daniels, J.A. Downie, A.E. Osbourn), Kluwer Academic Publishers, Dordrecht, Boston, London, 303-306.
34. León J., Yalpani N., Raskin I., Lawton M. A., (1993), Plant Physiol., 103, 323-328.
35. Levine A., Tenhaken R., Dixon R., Lamb C., (1994), Cell, 79, 583-593.
36. Linthorst H. J., (1991), Crit. Rev. Plant Sci., 10, 123-150.
37. Liu D., Raghothama K. G., Hasegawa P. M., Bressen R. A., (1994), Proc. Nat. Acad. Sci. USA, 91, 1888-1892.
38. Malamy J., Carr J. P., Klessig D., Raskin I., (1990), Science, 250, 1001-1004.
39. Malamy J., Hennig J., Klessig D. F., (1992), Plant Cell, 4, 359-366.
40. Malamy J., Sánchez-Casas P., Hennig J., Guo A., Klessig D. F., (1995), Mol. Plant-Microbe Interact, submitted.
41. Martin G. B., Brommonschenkei S. H., Chunwongse J., Frary S., Ganal M. W., Spivey R., Wu T., Earle E. D., Tanksley S. D., (1993), Science, 262, 1432-1436.
42. Matthews R. E. F., (1991), Plant Virology, Academic Press, San Diego.
43. Mauch F., Mauch-Mani B., Boller T., (1988), Plant Physiol., 88, 936-942.
44. Mindrinos M., Katagiri F., Yu G.-L., Ausubel F. M., (1994), Cell, 78, 1089-1099.
45. Palva T. K., Hurtig M., Saindrenan P., Palva E. T., (1994), Mol. Plant-Microbe Interact., 7, 356-363.
46. Ponstein A.S., Bres-Vloemans S.A., Sela-Buurlage M.B., van den Elzen P.J.M., Melchers L.S., Cornelissen B.J.C., (1994), Plant Physiol., 104, 109-118.
47. Prasad T. K., Anderson M. D., Martin B. A., Stewart C. R., (1994), Plant Cell, 6, 65-74.
48. Sánchez-Casas P., Klessig D. F., (1994), Plant Physiol., 106, 1675-1679.
49. Schneider G., Jensen E., Spray C., Phinney B. O., (1992), Proc. Nat. Acad. Sci. USA, 89, 8045-8048.
50. Schreck R., Baeuerle P. A., (1991), Trends Cell Biol., 1, 39-42.
51. Vaux D. L., Haecker G., Strasser A., (1994), Cell, 76, 777-779.
52. Vernooij B., Friedrich L., Morse A., Reist R., Kolditz-Jawhar R., Ward E. R., Uknes S., Kessmann H., Ryals J., (1994), Plant Cell, 6, 959-965.
53. Ward E. R., Uknes S. J., Williams S. C., Dincher S. S., Wiederhold D. L., Alexander D. C., Ahl-Goy P., Métraux J-P., Ryals J. A., (1991), Plant Cell, 3, 1085-1094.
54. White R. F., (1979), Virology, 99, 410-412.
55. Whitham S., Dinesh-Kumar S. P., Choi D., Hehi R., Corr C., Baker B., (1994), Cell, 78, 1101-1115.
56. Woloshuk C. P., Meulenhoff J. S., Sela-Buurlage M., van den Eizen P. J. M., Cornelissen B. J. C., (1991), Plant Cell, 3, 619-628.
57. Wolter M., Hollricher K., Salamini F., Schulze-Lefert P., (1993), Mol. Gen. Genet., 239, 122-128.
58. Yalpani N., León J., Lawton M.A., Raskin I., (1993), Plant Physiol., 103, 315-321 .
59. Yalpani N., Schulz M., Davis M.P., Balke N.E., (1992), Physiol., 100, 457-463.
60. Yalpani N., Shulaev V., Raskin I., (1993), Phytopathology, 83, 702-708.
61. Yoshikawa M., Tsuda M., Takeuchi Y., (1993), Naturwissenschaften, 80, 417-420.

Elements of signal transduction leading to activation of plant defense

Summary

Animals possess an inducible antibody immune system that acts as a defense against diseases. It is known that plants can also be actively immunized against disease-causing pathogens. This phenomenon is a result of the development of systemic acquired resistance (SAR). This article discusses recent studies on the role of salicylic acid (SA) in plants during the development of SAR. The understanding of molecular and physiological background SAR may be applied in modern agrobiotechnology and pharmaceutical industry.

Key words:
plant-microbe interaction, systemic acquired resistance, salicylic acid.

Address for correspondence:
Jacek Hennig, Institute of Biochemistry and Biophysics PAS, 5A Pawińskiego St., 02-106 Warsaw, Poland, fax: (48)39-121-623 or (48-2)658-46-36; e-mail: jacekh@ibbrain.ibb.waw.pl