SA, JA, ethylene, and disease resistance in plants

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Exciting advances have been made during the past year: isolating mutants affecting plant disease resistance, cloning genes involved in the regulation of various defense responses, and characterizing novel defense signaling pathways. Recent studies have demonstrated that jasmonic acid and ethylene are important for the induction of nonspecific disease resistance through signaling pathways that are distinct from the classic systemic acquired resistance response pathway regulated by salicylic acid.

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Abbreviations
Avr avirulence
BTH benzo(1,2,3)thiadiazole-7-carbothioic acid S-methyl ester
eds enhanced disease susceptibility
HR hypersensitive response
INA 2,6-dichloroisonicotinic acid
ISR induced systemic resistance
JA jasmonic acid
MAP mitogen-activated protein
ndr non-race-specific disease resistance
pad phytoalexin deficient
PR pathogenesis-related
R resistance
SA salicylic acid
SAR systemic acquired resistance

Introduction
The battle between a plant and an invading pathogen is often dependent on speed. The winner is determined by how quickly the pathogen can proliferate and exert damage, compared to how fast the plant can respond with the necessary levels of defense. If a pathogen is immediately recognized by a plant, for example when a plant has a specific resistance (R) gene that interacts with the corresponding avirulence (Avr) gene from the pathogen, a rapid defense mechanism known as the hypersensitive response (HR) occurs to prevent infection [1-6]. A lack of rapid pathogen recognition often leads to a successful infection. In addition to this immediate, one-on-one contest, plants also employ general resistance mechanisms induced after an HR or during a successful infection to combat secondary infections from a broad spectrum of pathogens or to prevent an existing infection from spreading further. One such general defense mechanism is known as systemic acquired resistance (SAR) [7-9]. SAR induction requires the signal molecule salicylic acid (SA), which accumulates in plants prior to the onset of SAR. Removal of SA in transgenic plants expressing salicylate hydroxylase (encoded by the bacterial nahG gene) prevents the establishment of SAR [10]. In some plants such as tobacco, cucumber, and Arabidopsis, SA is not only necessary but also sufficient for the induction of SAR. Treatment of these plants with SA or its functional analogs such as 2,6-dichloroisonicotinic acid (INA) and benzo(1,2,3)thiadiazole-7-carbothioic acid S-methyl ester (BTH) induces SAR [11,12]. SAR is believed to be a result of the concerted activation of pathogenesis related (PR) genes [13-16]; overexpression of a single PR gene only provides limited protection to the plant [17-21]. Even though their roles in disease resistance have yet to be clearly demonstrated, expression levels of PR genes serve as convenient markers for monitoring SAR.

Characterization of SAR in a variety of plant species has suggested the existence of a complex signaling network that involves many factors affecting various aspects of general disease resistance. It has become evident that plants utilize multiple pathways to transduce pathogenic signals to activate HR, SAR, and other resistance responses, and that SA-mediated SAR is not the only pathway that can lead to broad-spectrum disease resistance. Evidence is emerging that strongly suggests the importance of jasmonic acid (JA) and ethylene as alternative signals in the induction of resistance against microbial pathogens, in addition to their well-characterized roles in the wounding response in plants. In this review, I focus on the genetic and molecular experiments carried out in the past year that define the signaling components in both SA-dependent and SA-independent acquired resistance.

Different signaling pathways leading to the induction of resistance
Genetic screens performed in Arabidopsis have generated several classes of mutants which define potential signaling components downstream of the avr-R recognition event. One group of mutants, represented by eds1 (for enhanced disease susceptibility) [22] and ndr1 (for non-race-specific disease resistance) [23], exhibit altered responses to multiple avr signals. The recessive eds1 mutation lacks the capability for signal transduction from a subset of R genes resulting in susceptibility to strains of Peronospora parasitica and Pseudomonas syringae carrying the corresponding avr genes. The ndr1 mutation appears to influence the function of a different subset of R genes causing a loss of resistance to Pseudomonas syringae pv. tomato DC3000 carrying the bacterial avirulence genes avrRpt2, avrB, or avrPph3. The requirement for EDS1 or NDR1 by different sets of R genes seems to be mutually exclusive (IE Parker, personal communication). It has been proposed, therefore, that separate signaling components are used in different avr-R interactions. The
selective use of a signaling component may be based on the structure of an individual \( R \) gene, with each \( R \) gene within a class sharing a particular component. Connections between these early signaling components and the downstream SA or JA and ethylene signaling pathways have yet to be established. The recent success in cloning the \( EDS1 \) gene which encodes a lipase (JE Parker, personal communication) and the \( NDR1 \) gene which encodes a protein with two putative transmembrane domains [24\*\*] will certainly lead to new information on the molecular functions of these genes.

A new class of mutants, designated \( dnd \) (for defense, no death), do not show HR after inoculation with \( P. syringae \) expressing \( avrRpt2 \) or \( avrRpm1 \) but maintain resistance to this pathogen (AF Dent, personal communication). The identification of \( dnd \) mutants may imply that cell death is not essential for \( avr-R \) specific resistance. Another plausible explanation is that the resistance observed in \( dnd \) mutants is a result of the downstream activation of the SAR response, signified by the constitutively elevated levels of SA and \( PR \) gene expression in these mutant plants.

Analysis of mutants that form HR-like lesions in the absence of pathogen infection and display enhanced nonspecific pathogen resistance has yielded additional insights into regulation of resistance responses. The \( Arabidopsis \) \( LSD1 \) gene encodes a novel zinc finger protein which may respond to superoxide accumulated during the HR to restrict the spreading of cell death [25\*\*]. The maize \( Lks1 \) gene has been found to encode a protein with two conserved substrate binding motifs of aromatic ring-hydroxylation dioxygenases and probably functions to degrade a phenolic mediator of cell death [26\*\*]. The \( Mlo \) gene of barley is predicted to be a membrane bound protein with at least six membrane-spanning helices [27\*\*]. Further characterization of these genes, in addition to the genes defined by the HR-compromised mutants, may provide clues as to how signals are produced during an HR leading to the induction of systemic resistance. Epistasis analyses between the HR-compromised mutants and the lesion mimic mutants may reveal the hierarchy of the signaling events.

A recent report has shown convincingly that reactive oxygen intermediates generated during an HR may serve as signals mediating the establishment of systemic resistance [28\*\*]. Alvarez et al. demonstrated that inoculation of \( Arabidopsis \) with an avirulent pathogen triggers not only an HR at the site of infection but also induces secondary oxidative bursts in discrete clusters of cells in distant, uninfected tissues, which ultimately lead to the formation of microscopic HR lesions. Both the primary oxidative burst and the reiterative secondary oxidative bursts in the systemic tissues have been shown to be required for the induction of SAR.

Virulent pathogens have also been used to identify components of nonspecific resistance responses. Characterization of these mutants has shown that there is a perception/signaling pathway for virulent pathogens leading to SA accumulation and resistance that is distinct from that triggered by avirulent pathogens. The \( eds \) mutants were isolated for their enhanced disease susceptibility after infection by the virulent pathogen \( P. syringae \)pv. \( maculicola \) ES4326 [29,30\*\*]. Inoculation of these mutant plants with an avirulent pathogen still results in an HR and an SAR response even though there is an upward shift in growth of the challenging pathogen in both control plants, pre-treated with 10 mM magnesium chloride, and induced plants, pre-inoculated with an avirulent pathogen. In \( eds5-1 \), \( PR-1 \) gene induction by the virulent pathogen \( Psm \) ES4326 is reduced to 10% of the wild-type levels but is fully restored by SA treatment. The \( pad4 \) mutants (for phytoalexin-deficient) are deficient in accumulating camalexin, an indolic phytoalexin, after infection by the virulent pathogen \( Psm \) ES4326 [31] and \( pad4 \), specifically, appears to affect a regulatory component in camalexin biosynthesis. The \( pad4 \) mutant displays enhanced susceptibility to \( Psm \) ES4326 and a number of isolates of \( P. parasitica \) [32\*]. In addition, it exhibits pleiotropic defects in \( Psm \) ES4326-induced SA accumulation and \( PR \) gene expression that can be partially reversed by the addition of SA [33\*\*]. The pleiotropic effects of \( pad4 \) are specific to the virulent pathogen \( Psm \) ES4326 (and also \( Pst \) DC3000) because the synthesis of camalexin, the accumulation of SA, and the expression of \( PR-1 \) gene induced by \( Psm \) ES4326 carrying \( avrRpt2 \) are all similar to wild-type in \( pad4 \).

Characteristics of all these mutants suggest the existence of separate components involved in transducing diverse signals generated by different pathogens. \( PAD4 \) and \( EDSS \) may be regulators controlled by signals produced during a virulent infection while \( NDR1 \), \( EDS1 \), \( DND \) and \( LSD1 \) may represent components involved in the \( avr \)-induced HR, with \( NDR1 \) and \( EDS1 \) responding to distinct \( avr-R \) interactions. Without further knowledge of the signal molecules, however, classification of these signal transducing components can only be done arbitrarily. A component identified in an avirulent pathogen-induced response, such as \( eds1 \), may also affect the response to a virulent pathogen. Nevertheless, all these components seem to be involved in early signaling events. Whether they play a direct role in the induction of SAR or other general resistance responses has yet to be determined.

**SA-dependent signaling pathway**

In tobacco, \( Arabidopsis \), and cucumber, an increase in SA concentration leads to the onset of SAR [34,36], but in other plants such as potato and rice, high levels of SA are detected even under noninducing conditions [37,38,39]. It has been questioned whether SA is a signal required for SAR in potato and rice, and if it is, how is
the resistance response is activated in these plants with constitutively high levels of SA. Using transgenic potato plants expressing bacterial salicylate hydroxylase (which metabolizes SA), it has recently been demonstrated that SA is required for arachidonic acid-induced resistance to Phytophthora infestans in potato. The resistance, however, is induced as a result of an increase in sensitivity to SA rather than an increase in SA synthesis [40**]. The mechanism of this induction is unknown; it could occur by increasing the accessibility of SA to a receptor, or by inducing synthesis or activity of an SA receptor.

Biochemical approaches used to identify components involved in the transduction of the SA signal have produced promising candidates. An SA binding protein has been identified [41*] which has a 150-fold higher affinity for SA than catalase: catalase being the first SA binding protein isolated using 14C-labeled SA. Protein phosphorylation and dephosphorylation have been indicated in various defense responses including SA-regulated PR gene expression [42]. Consistent with these results, a mitogen-activated protein (MAP) kinase has been shown to be activated in an SA-induced tobacco suspension cell extract [43**]. The sequence of this p48SIP kinase (SA-induced protein kinase) is distinct from other plant MAP kinases previously implicated in stress responses.

In addition to the biochemical approaches, genetic screens performed in Arabidopsis have uncovered one locus, NPR1 (for nonexpresser of PR genes; also called NIM1 or SAII), that clearly functions downstream of SA [29,44–46]. NPR1 has been cloned independently by two research groups [47**,48**]. Sequence analysis of the gene and its twelve mutant alleles has shown that NPR1 contains a functionally important ankyrin-repeat domain which may be involved in protein–protein interaction. The carboxyl end of NPR1, where a nuclear localization signal (NLS) resides, has also been shown to be essential for protein function. The importance of this NLS has been demonstrated by the strict nuclear localization of a biologically active NPR1-GFP fusion protein after SAR induction (M Kinkema, X Dong, unpublished data). SAR induction not only modestly increases NPR1 levels, but also is required for activation of the NPR1 protein. Overexpression of NPR1 in Arabidopsis confers significant resistance to strains of P. syringae and P. parasitica that are normally virulent on wild-type Arabidopsis [49**]. By measuring the induction kinetics of the PR genes during the infection, it has been demonstrated that PR genes are not constitutively expressed in the NPR1-overexpressing lines and that pathogen infections cause a stronger, but not faster, expression of these genes compared to wild-type. These characteristics make NPR1 a favorable target for genetic engineering of disease resistance in crops, because constitutive activation of SAR would be likely to have detrimental effects on plant growth and also might result in high selection pressure for more virulent pathogens. There are still many questions that remain to be answered with regard to the molecular function of NPR1. For example, it is unknown if NPR1 regulates PR gene expression through activation of a positive transcription factor, or inactivation of a negative transcription factor. To completely understand NPR1 function, the NPR1-interacting component(s) will have to be identified and characterized.

JA- and ethylene-dependent signaling pathway

An exciting new development in the past year is the discovery of SA-independent pathway(s) that also lead to broad-spectrum, systemic resistance. Both JA and ethylene have been shown to be important for the induction of these alternative responses. Fortunately, there already exists a wealth of information about the role of JA in plants response to wounding and insect attack [50]. The wound-induced octadecanoid pathway results in the synthesis of the signal molecule JA and subsequent activation of proteinase inhibitor genes. In addition to JA, ethylene has recently been shown to be a required, though not sufficient, signal that acts with JA to induce the wound response in tomato [51]. Ethylene is produced rapidly and transiently in leaves after injury or upon induction by JA or cell wall oligosaccharide fragments. Inhibition of ethylene production or sensitivity through mutation, antisense technology, or the use of chemical inhibitors all negatively affect induction of the JA pathway.

The JA regulated wound- and insect-induced pathway has long been thought to be involved in conferring resistance to microbial pathogens because it is also induced by oligosaccharide fragments released by the action of lytic enzymes produced by an invading pathogen; however, the role of ethylene and JA in plant disease resistance seemed at times to be ambiguous. For example, in the Arabidopsis ethylene-insensitive mutant ein2, disease symptoms caused by Pst DC3000, Psm 4326, and Xanthomonas campestris pv. campestris were shown to be reduced while growth of these pathogens remained unaffected [52,53]. In the same ethylene-insensitive mutant, the involvement of ethylene in SA-mediated SAR was ruled out because SA- and INA-induced PR-1 gene expression and resistance to P. parasitica were shown to be unaffected by the mutation [54]. In fact, the basal levels of PR-1 mRNA seemed to be higher in the ein2 mutant than in the wild-type. Similar results have recently been obtained using the tomato Never ripe mutant impaired in ethylene perception [55*]. In another example, the Arabidopsis coil mutants identified for their insensitivity to coronatine and methyl JA (a volatile form of JA) were shown to be resistant to a bacterial pathogen producing the phytotoxin coronatine [56]; however, this result is inconsistent with the hypothesis that methyl JA induces disease resistance. These results can be explained if the ethylene/JA pathway negatively affects the SA pathway. When the ethylene/JA pathway is blocked in the ein2 and coil mutants, the SA pathway may be slightly upregulated, leading to elevated
levels of PR gene expression and increased tolerance to bacterial pathogens.

A more clear-cut connection between systemic disease resistance, JA and ethylene signaling molecules has recently been established in Arabidopsis by the characterization of the systemically inducible antimicrobial peptides, thionin Th12.1 and defensin PDF1.2 [57, 58]. The expression of Th12.1 is induced by methyl JA, silver nitrate, and Fusarium oxysporum f. sp. matthioliæ, but not by SA [57]. The antimicrobial activity of thionin has been demonstrated in Arabidopsis by the enhanced resistance to F. oxysporum sp matthioliæ observed in transgenic plants overexpressing the Th12.1 gene [59*]. The PDF1.2 gene is induced by JA, ethylene, rose bengal, and the non-host pathogen Alternaria brassicicola [58]. The induction of PDF1.2 by A. brassicicola is inhibited in the JA-insensitive mutant coil and in the ethylene-insensitive mutant ein2, indicating that JA and ethylene are required for the induction. PDF1.2 expression, however, is unaffected in auxG transgenic plants which are unable to accumulate SA, in the npr1 mutant which is insensitive to SA induction, or in the crp1 mutant which has elevated levels of SA and constitutive SAR. It is evident that PDF1.2 and Th12.1 represent end products of a systemically expressed resistance pathway(s) that is distinct from the SA-dependent SAR and is potentially regulated by both JA and ethylene.

Genetic studies have shown that JA and ethylene are essential signals in another resistance response known as induced systemic resistance (ISR) that is established after treatment with various strains of root-colonizing biocontrol bacteria Pseudomonas fluorescens (CMJ Pieterse, personal communication). ISR is effective against the virulent Pst DC3000 and F. oxysporum f. sp. raphani and functions independently of SA and PR gene expression [60, 61]. Intriguingly, during ISR, PDF1.2 is not induced, suggesting that JA and ethylene may regulate ISR through a pathway different from that which includes PDF1.2 and Th12.1. ISR, therefore, appears to be a novel systemic resistance response in plants that can be triggered in roots by rhizobacteria and is regulated by JA and ethylene.

Interactions between different resistant pathways

The antagonistic relationship between the JA and SA pathways has been well documented in the studies of wound responses in plants [50, 62]. The inhibitory effect of SA on the proteinase inhibitor genes is partially overcome by pretreatment of plants with both JA and ethylene, but not by either signal alone [51]. This further supports the notion that both ethylene and JA are required signals for wound responses and implies that SA inhibits not only the synthesis of JA and ethylene but also a signaling step downstream of JA and ethylene.

For example, in tobacco, treatments with SA and the pathogenic Erwinia carotovora or Erwinia-derived elicitors seem to induce two distinct pathways leading to the expression of separate sets of genes [63*]. Erwinia-derived elicitors antagonize the SA-mediated induction of PR genes while SA inhibits the induction of Erwinia-activated genes. Such antagonism suggests the presence of common regulatory components for both pathways, and possibly signifies the evolution of a mechanism designated to prioritize different resistance responses.

Common regulatory components have been found in separate signaling pathways. In the root-colonizing bacterium-induced ISR, which is SA-independent but JA- and ethylene-dependent, the function of NPR1 has been shown to be essential (CMJ Pieterse, personal communication). This implies that NPR1 not only plays a key regulatory role in SAR but also participates in the JA- and ethylene-regulated, SA-independent ISR. This is consistent with the detection of high levels of NPR1 in roots (T Liao, M Aitken, X Dong, unpublished data) where ISR is induced and where PR gene expression and SAR are absent [64].

Other components potentially shared by the SA-induced SAR and the JA- and ethylene-induced disease resistance pathways have been identified through epistasis analyses between Arabidopsis mutants with enhanced resistance and mutants with compromised defense responses. The crp5 mutant expresses both the PR genes and the PDF1.2, and Th12.1 genes, and is resistant to Pst ES4326 and P. parasitica Noco2. In the crp5:npr1 double mutant, the expression of PR genes and resistance to Pst ES4326 are abolished while the expression of PDF1.2 and resistance to P. parasitica remain unaffected [65*]. These results suggest that a mutation in a single gene can upregulate an NPR1-dependent pathway and an NPR1-independent pathway, both of which can provide resistance to P. parasitica. This conclusion is supported by the epistasis analysis performed in the crp1:npr1 double mutant. In crp1, where only the NPR1-dependent pathway is activated, the enhanced resistance to P. parasitica is completely abolished by npr1 in the crp1:npr1 double mutant (SA Bowling, X Dong, unpublished data).

Analyses of the dominant, gain-of-resistance Arabidopsis mutant crp6 define another possible point of interaction between the SA-regulated and the JA- and ethylene-regulated pathways [66**]. Similar to crp5, the crp6 mutant displays enhanced levels of PR and PDF1.2 gene expression. In the crp6:npr1 double mutant, however, neither PR nor PDF1.2 gene expression is significantly affected by npr1. Even though it is difficult to determine the precise position of crp6 in the disease resistance signaling network, the data suggest a dual role for crp6 in regulating both the PR and PDF1.2 genes. Perhaps the wild-type CPR6 works with NPR1 to induce the expression of PR genes, and the dominant crp6 mutation renders
the protein NPR1-independent. The interplay between CPR6, NPR1 and the two distinct signaling pathways has been further suggested by the complete suppression of PDF1.2 expression by INA treatment observed in cpr6, in comparison to only a partial suppression of PDF1.2 expression in the cpr6:npr1 double mutant. A functional NPR1 protein seems to be required for INA to sequester all the mutant cpr6 protein thereby preventing activation of PDF1.2.

The relationship between the SA-regulated SAR and the JA- and ethylene-regulated disease resistance needs to be explored further using mutants of both pathways. For example, epistasis analysis between the cpr5 or cpr6 mutant and the ethylene-insensitive mutant, ein2, or the jasmonic acid-non-responsive mutant, jar1 [67], will be carried out and the results will be compared with those of cpr5:npr1 or cpr6:npr1. Preliminary analysis of the cpr5:ein2 and cpr6:ein2 double mutants have indicated that a full

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**Figure 1**

A working model of pathways leading to broad-spectrum disease resistance in plants. The Arabidopsis genes defined by the mutants described in this review are presented. After plant–pathogen recognition, multiple signaling components (CPR5, NDR1, EDS1, DNDs, LSD1) are employed that lead to the formation of reactive oxygen species (ROS) and HR, and the synthesis of SA. NDR1 and EDS1 are involved in transducing signals from separate subsets of avr-R interactions, the relative positions of other genes have yet to be determined. It is also unknown whether these genes (except CPR5) are involved in the JA- and ethylene-mediated resistant responses. (This ambiguity is represented by a dashed line.) Virulent pathogens can also induce SA synthesis and local resistance, probably through separate regulatory components such as PAD4 and EDSs. Further downstream, the CPR1-SA-NPR1 linear pathway has been demonstrated by various genetic analyses. The pathway of JA and ethylene is proposed on the basis of the wound response in tomato [61], showing that JA and ethylene induce each other's synthesis and that JA and ethylene are both required for activating the wound response, PDF1.2 and Th2.1 gene expression, resistance to P. parasitica, and ISR. It remains to be determined whether these responses (shown in rectangular boxes) share regulatory components downstream of JA and ethylene. The SA pathway and the JA/ethylene pathway may interact antagonistically, with SA inhibiting both the synthesis and the signal transduction of JA and ethylene (illustrated with blocked lines). The possible involvement of NPR1 in the SA-independent but JA- and ethylene-dependent ISR is intriguing. Further characterization of NPR1 and another Arabidopsis component CPR6, which is involved in regulating the expression of both PR and PDF1.2 genes, should provide new insight into the relationship of these pathways.
induction of PDF1.2 in cpr5 and cpro requires the function of EIN2 and probably ethylene (JD Clarke, X Dong, unpublished data).

Conclusions
Isolation of different classes of mutants affecting nonspecific disease resistance and characterization of various plant defense responses have revealed a complex and interesting network of pathways that function both separately and together to render broad-spectrum protection to plants. At the center of this network are the signal molecules SA, JA, and ethylene. A model, with the Arabidopsis genes described in this review highlighted, is represented here to stimulate more discussion (Figure 1). After plant–pathogen recognition, multiple signaling components (CPR5, NDR1, EDS1, DNDs, LSD1) are employed that lead to the formation of reactive oxygen species (ROS) and HR, and the synthesis of SA. While NDR1 and EDS1 are involved in transducing signals from separate subsets of avr-R interactions, the relative positions of other genes have yet to be determined. It is also unknown whether these genes (except CPR5) are involved in the JA- and ethylene-mediated resistant responses. Virulent pathogens can also induce SA synthesis and local resistance, probably through separate regulatory components such as PAD4 and EDSs. Further downstream, the CPR1-SA-NPR1 linear pathway has been demonstrated by various genetic analyses. The pathway of JA and ethylene is proposed on the basis of the wound response in tomato [51] showing that JA and ethylene induce each other’s synthesis and that JA and ethylene are both required for activating the wound response, PDF1.2 and Thg2.1 gene expression, resistance to P. parasitica, and ISR. It remains to be determined whether these responses share regulatory components downstream of JA and ethylene. The SA pathway and the JA/ethylene pathway may interact antagonistically, with SA inhibiting both the synthesis and the signal transduction of JA and ethylene. The possible involvement of NPR1 in the SA-independent but JA- and ethylene-dependent ISR is intriguing. Further characterization of NPR1 and another Arabidopsis component CPR6, which is involved in regulating the expression of both PR and PDF1.2 genes, should provide new insight into the relationship of these pathways.

A complete understanding of these signaling events calls for co-operation between laboratories working on different resistance responses. As more epistasis tests are carried out between different classes of mutants, new resistance-related phenotypes will be revealed and used to perform more in-depth analyses. Information from both genetic and molecular research will be combined to establish a more comprehensive outline of the disease resistance network in plants.

References and recommended reading
Papers of particular interest, published within the annual period of review have highlighted as:

• of special interest
•• of outstanding interest


The authors describe positional cloning of the *Arabidopsis* NDR1 gene which affects disease resistance against both bacterial and fungal pathogens mediated by several *R* gene products. The NDR1 protein contains two putative transmembrane domains.


The authors report positional cloning of the *Arabidopsis* LSD1 gene. The molecular function of LSD1 as a negative regulator of cell death in plants is suggested on the basis of amino acid sequence information which predicts the protein contains three zinc finger domains.


The maize Lis1 gene cloned by transposon-tagging encodes a protein with two aromatic ring-hydroxylating dioxygenase substrate binding motifs. It is suggested to play a role in controlling the spread of cell death in mature maize leaves by degrading a phenolic mediator of cell death.


The authors report positional cloning of the barley *Mlo* gene which, when mutated, causes leaf browning and broad spectrum resistance to *Erysiphe graminea* f. sp. *hordei*. The *Mlo* gene is shown to encode a protein with at least six membrane-spanning helices, and its dual function in down regulating cell death and resistance is discussed.


The authors show that inoculation of *Arabidopsis* with an avirulent pathogen not only leads to an immediate oxidative burst at the site of infection but also result in reiterative oxidative bursts in the unaffected systemic tissues, which eventually lead to formation of interspecific HR. Both the primary and the secondary oxidative bursts are required for the establishment of SAR.


An in-depth characterization of previously isolated *Arabidopsis* mutants ed5-1, ed6-1, ed7-1 and ed9-1 which have enhanced disease susceptibility to a virulent bacterial pathogen. Many of the mutants show a distinguishable phenotype in response to a panel of bacterial pathogens and with regard to *PR-1* gene inducibility. The data suggest the presence of an unknown resistance mechanism against virulent pathogens.


Among the first *pad* mutants (phytoalexin deficient) described in this paper, *pad1* is shown to affect the regulation of camalexin biosynthesis after infection by *Pseudomonas syringae*, but the mutant is still able to produce camalexin when *Cochliobolus sativus* is used in the infection. *pad4* not only shows enhanced disease susceptibility to *Pseudomonas syringae* but also is fully susceptible to four of the six incompatible strains of *Peronospora parasitica* tested.


An in-depth characterization of the previously isolated *pad4* mutant. *pad4* is shown to have pleiotropic defects on camalexin synthesis and *PR-1* gene expression after infection by the virulent pathogen *Pseudomonas syringae* pv. *maculicola* ES4326. SA synthesis induced by *Pad ES4326* is also reduced and delayed in *pad4*. Treatment of *pad4* with SA partially restores camalexin synthesis and *PR-1* gene expression. Therefore, *PAD4* is speculated to act upstream of SA accumulation in response to *Pad ES4326* infection. A model is proposed to illustrate the function of *PAD4*.


In this report, the authors argue that SA regulates a defense response in rice through differential accumulation of SA and tissue-specific expression of catalases with different levels of sensitivities to SA.


To investigate the role of SA in controlling resistance in potato plants where high basal levels of SA are detected, the bacterial salicylate hydroxylase (naH) gene was introduced into potato. The resulting transgenic plants show significant increase in disease severity when infected by *Phytophthora infestans*, while the arachidonic acid-induced systemic resistance to the same pathogen is abolished. These results indicate that SA is a necessary but not sufficient signal for arachidonic acid-induced resistance. In potato the development of SAR may involve an increase in sensitivity to SA.


The authors describe the identification of a new SA-binding, soluble protein with a low abundance in tobacco leaves and a 150-fold higher affinity for SA than that shown by catalase.


Using an in-gel kinase assay, a MAP kinase is shown to be activated rapidly and transiently in tobacco suspension cells after addition of SA. The protein has been purified, and the corresponding gene cloned and shown to be distinct from other previously identified stress-induced MAP kinases.


The authors describe positional cloning of the Arabidopsis NPR1 gene and analysis of four npr1 mutant alleles. The functional importance of the ankyrin-repeat domain found in NPR1 is speculated upon. Transforming the npr1 mutants with the wild-type NPR1 genomic clone not only complements the mutant phenotypes, but also results in enhanced resistance.


The authors describe positional cloning of the Arabidopsis NIM1 gene and characterization of five new nim1 mutant alleles. Sequence homology between NIM1 and the mammalian transcription factor inhibitor IB is outlined and the possible implication of this finding is discussed.


The authors present results which prove the concept that broad-spectrum disease resistance can be generated through manipulation of the NPR1 gene, a regulatory gene of SAR. NPR1 is shown to determine resistance in a dosage-dependent fashion. Overexpression of NPR1 leads to significant resistance in virulent strains of Pseudomonas syringae and Peronospora parasitica with little detrimental effect on the growth of the plants.


Using the tomato floury crip mutant impaired in ethylene perception, the authors show that development of disease symptoms caused by the virulent pathogen Xanthomonas campestris pv. vesicatoria, Pseudomonas syringae pv. tomato and Fusarium oxysporum f.sp. lycopersici requires ethylene. In the mutant, the disease symptoms are reduced compared to wild-type, but growth of the pathogen remains unaffected.


The authors demonstrate the antimicrobial activity of thionins by overexpressing an endogenous thionin gene in Arabidopsis. The transgenic line is shown to have enhanced resistance against Fusarium oxysporum.


The authors show that induced systemic resistance (ISR) triggered in Arabidopsis by the biocontrol bacteria involves expression of specific genes between the plant and the pathogen, which is reflected in the ectype- and strain-specifically observed in ISR. Mutant bacteria lacking the O antigens side chain of lipopolysaccharide (LPS) are still effective in inducing ISR, suggesting that in addition to LPS, other factors are also involved in triggering ISR in Arabidopsis.


Using Erwinia and SA as inducers, the authors describe two distinct pathways leading to expression of separate sets of genes. SA and Erwinia doped signals have antagonistic effects on each other.


The authors report the results of an epistasis analysis between the Arabidopsis cpr5 mutant (with enhanced resistance) and the npr1 mutant (with impaired SAR). An NPR1-independent pathway is uncovered in cpr5 which is shown to confer resistance to the virulent pathogen Peronospora parasitica. A model is presented to illustrate the NPR1-dependent and independent pathways.


Through genetic analysis of the dominant Arabidopsis cpr6 mutant which has constitutive expression of both the SA-regulated PR genes and the JA- and ethylene-regulated PDF1.2 gene, the authors show that CPR6 is involved in multiple resistance pathways, and that PR gene expression does not confer resistance to the bacterial pathogen Pseudomonas syringae observed in the cpr6 mutant.