Mécanismes de défense des plantes


Microorganisms use siderophores to obtain iron from the environment. In pathogenic interactions, siderophores are involved in iron acquisition from the host and are sometimes necessary for the expression of full virulence. This review summarizes the main data describing the role of these iron scavengers in animal and plant defence systems. To protect themselves against iron theft, mammalian hosts have developed a hypoferremia strategy that includes siderophore-binding molecules called siderocalins. In addition to microbial ferri-siderophore sequestration, siderocalins are involved in triggering immunity. In plants, no similar mechanisms have been described and many fewer data are available, although recent advances have shed light on the role of siderophores in plant–pathogen interactions. Siderophores can trigger immunity in plants in several contexts. The most frequently described situation involving siderophores is induced systemic resistance (ISR) triggered by plant-growth-promoting rhizobacteria. Although ISR responses have been observed after treating roots with certain siderophores, the underlying mechanisms are poorly understood. Immunity can also be triggered by siderophores in leaves. Siderophore perception in plants appears to be different from the well-known perception mechanisms of other microbial compounds, known as microbe-associated molecular patterns. Scavenging iron per se appears to be a novel mechanism of immunity activation, involving complex disturbance of metal homeostasis. Receptor-specific recognition of siderophores has been described in animals, but not in plants. The review closes with an overview of the possible mechanisms of defence activation, via iron scavenging by siderophores or specific siderophore recognition by the plant host.


Microbial consortia may provide protection against pathogenic ingress via enhancing plant defense responses. Pseudomonas aeruginosa PJHU15, Trichoderma harzianum TNHU27 and Bacillus subtilis BHHU100 were used either singly or in consortia in the pea rhizosphere to observe proteome level changes upon Sclerotinia sclerotiorum challenge. Thirty proteins were found to increase or decrease differentially in 2-DE gels of pea leaves, out of which 25 were identified by MALDI-TOF MS or MS/MS. These proteins were classified into several functional categories including photosynthesis, respiration, phenylpropanoid metabolism, protein synthesis, stress regulation, carbohydrate and nitrogen metabolism and disease/defense-related processes. The respective homologue of each protein identified was trapped in Pisum sativum and a phylogenetic tree was constructed to check the ancestry. The proteomic view of the defense response to S. sclerotiorum in pea, in the presence of beneficial microbes, highlights the enhanced protection that can be provided by these microbes in challenged plants.
Plant growth promoting rhizobacteria (PGPR) are known to confer disease resistance to plants. Bacillus sp. JS demonstrated antifungal activities against five fungal pathogens in vitro assays. To verify whether the volatiles of Bacillus sp. JS confer disease resistance, tobacco leaves pre-treated with the volatiles were damaged by the fungal pathogen, Rhizoctonia solani and oomycete Phytophthora nicotianae. Pre-treated tobacco leaves had smaller lesion than the control plant leaves. In pathogenesis-related (PR) gene expression analysis, volatiles of Bacillus sp. JS caused the up-regulation of PR-2 encoding β-1,3-glucanase and acidic PR-3 encoding chitinase. Expression of acidic PR-4 encoding chitinase and acidic PR-9 encoding peroxidase increased gradually after exposure of the volatiles to Bacillus sp. JS. Basic PR-14 encoding lipid transfer protein was also increased. However, PR-1 genes, as markers of salicylic acid (SA) induced resistance, were not expressed. These results suggested that the volatiles of Bacillus sp. JS confer disease resistance against fungal and oomycete pathogens through PR genes expression.

Enterobacter asburiae BQ9, a plant growth-promoting rhizobacterium, was shown to promote tomato plant growth and induce resistance to tomato yellow leaf curl virus (TYLCV) under greenhouse conditions. Compared with mock-treated tomato plants, plants that were pre-treated with BQ9 had increased the fresh mass, significantly reduced the disease severity and achieved 52% biocontrol efficacy 30 days after inoculation. The expression of the defense-related genes PR1a and PR1b and the initiation of H2O2 burst were quickly induced in BQ9 pre-treated plants. Further anti-oxidase activity analysis showed that the activities of phenylalanine ammonialyase (PAL), peroxidase (POD), catalase (CAT) and superoxide dismutase (SOD) were significantly increased in BQ9 pre-treated plants. Our results suggest that the plant growth-promoting rhizobacterium BQ9 induced a priming of the plant defense responses to TYLCV by increasing the expression of defense response genes, and the induced resistance was mechanistically connected to the expression of antioxidant enzymes and the production of H2O2.

Crown gall disease caused by Agrobacterium tumefaciens affects a wide range of horticultural plants, and has no effective treatment. During the evaluation of crown gall resistance of peach germplasm resources, we observed enhanced resistance to subsequent invasion that was activated by virulence of A. tumefaciens in two peach cultivars. To further verify the phenotype observed in field experiments, systemic acquired resistance (SAR)-related salicylic acid (SA) and PR1 genes were investigated. The levels of SA were elevated in two cultivars, and these high levels were maintained for 35 days postinoculation. Compared with mock-inoculated controls, eight of the 22 candidate PpPR1 genes in A. tumefaciens-inoculated samples were significantly upregulated and three were downregulated in response to inoculation with A. tumefaciens. These data suggested that SA-induced SAR was activated in two peach cultivars by virulent A. tumefaciens infection. In addition, the eight induced PpPR1 genes can be used as molecular markers in defense studies in peach.

In this study, a necrosis-inducing protein was purified from the culture filtrate of the necrotrophic fungus Botrytis cinerea BC-98 strain. Secreted proteins were collected and fractionated by liquid chromatography. The fraction with the highest necrosis-inducing activity was further purified. A glycoprotein named BcGs1 was identified by 2D electrophoresis and mass spectrometry. The BcGs1 protein consisted of 672 amino acids with a theoretical molecular weight of 70.487 kDa. Functional domain analysis indicated that BcGs1 was a glucan 1,4-alpha-glucosidase, a cell wall-degrading enzyme, with a Glyco_hydro_15 domain and a CBM20_glucoamylase domain. The BcGs1 protein caused necrotic lesions that mimicked a typical hypersensitive response and H2O2 production in tomato and tobacco leaves. BcGs1-treated plants exhibited resistance to B. cinerea, Pseudomonas syringae pv. tomato DC3000 and tobacco mosaic virus in systemic leaves. In addition, BcGs1 triggered elevation of the transcript levels of the defence-related genes PR-1a, TPK1b and Prosystemin. This is the first report of a Botrytis glucan 1,4-alpha-glucosidase triggering host plant immunity as an elicitor. These results lay a foundation for further study of the comprehensive interaction between plants and necrotrophic fungi.


MAIN CONCLUSION: Systemic acquired resistance elicitors, BTH and BABA, reduce rust penetration in pea through phytoalexins pathway but differing in their mode of action. It has been previously shown that rust (Uromyces pisi) infection can be reduced in pea (Pisum sativum) by exogenous applications of systemic acquired resistance elicitors such as BTH and BABA. This protection is known to be related with the induction of the phenolic pathway but the particular metabolites involved have not been determined yet. In this work, we tackled the changes induced in phytoalexin content by BTH and BABA treatments in the context of the resistance responses to pea rust. Detailed analysis through high-performance liquid chromatography (HPLC) showed qualitative and quantitative differences in the content, as well as in the distribution of phytoalexins. Thus, following BTH treatment, we observed an increase in scopoletin, pisatin and medicarpin contents in all, excreted, soluble and cell wall-bound fraction. This suggests fungal growth impairment by both direct toxic effect as well as plant cell wall reinforcement. The response mediated by BTH was genotype-dependent, since coumarin accumulation was observed only in the resistant genotype whereas treatment by BABA primed phytoalexin accumulation in both genotypes equally. Exogenous application to the leaves of scopoletin, medicarpin and pisatin lead to a reduction of the different fungal growth stages, confirming a role for these phytoalexins in BTH- and BABA-induced resistance against U. pisi hampering pre- and postpenetration fungal stages.

Plants can be primed to respond faster and more strongly to stress and multiple pathways, specific for the encountered challenge, are involved in priming. This adaptability of priming makes it difficult to pinpoint an exact mechanism: the same phenotypic observation might be the consequence of unrelated underlying events. Recently, details of the molecular aspects of establishing a primed state and its transfer to offspring have come to light. Advances in techniques for detection and quantification of elements spanning the fields of transcriptomics, proteomics, and metabolomics, together with adequate bioinformatics tools, will soon allow us to take a holistic approach to plant defence. This review highlights the state of the art of new strategies to study defence priming in plants and provides perspectives towards 'prime-omics'.


Glucosinolates are nitrogen and sulfur containing secondary metabolites found mainly in the Brassicaceae. They function as plant defense compounds against a broad spectrum of pathogens and pests. Since these molecules form part of the plant defense mechanism, glucosinolate biosynthesis may be modulated by environmental signals leading to activation of a biological stress response. In the current study, we have mimicked such conditions by exogenously applying biotic elicitors such as methyl jasmonate, salicylic acid, glucose and mechanical injury in Brassica juncea seedling over a time course experiment. We found that total glucosinolates over-accumulated under these stress conditions with maximum accumulation observed 24h post treatment. Indole glucosinolates like 1-methoxy-indol-3-ylmethyl and its precursor indol-3-methyl glucosinolates showed a more significant induction compared to aliphatic glucosinolates thereby suggesting a prominent role of indole glucosinolates during plant defense response in B. juncea seedlings. In contrast, the higher amounts of aliphatic glucosinolates were less regulated by the tested biotic elicitors in B. juncea. Expression profiling of multiple homologs of key transcriptional regulators of glucosinolate biosynthesis further showed that a complex interplay of these regulators exists in polyploid B. juncea where they exert co-ordinated and overlapping effects toward altering glucosinolate accumulation. This study has a significant role toward understanding and augmenting plant defense mechanisms in B. juncea, a globally important oilseed crop of genus Brassica.


Effect of sublethal heavy metal stress as plant biotic elicitor for triggering innate immunity in tomato plant was investigated. Copper in in vivo condition induced accumulation of defense enzymes like peroxidase (PO), polyphenol oxidase (PPO), phenylalanine ammonia-lyase (PAL), and β-1,3 glucanase along with higher accumulation of total phenol, antioxidative enzymes (catalase and ascorbate peroxidase), and total chlorophyll content. Furthermore, the treatment also induced nitric oxide (NO) production which was confirmed by realtime visualization of NO burst using a fluorescent probe 4,5-diaminofluorescein diacetate (DAF-2DA) and spectrophotometric analysis. The result suggested that the sublethal dose of heavy metal can induce an array of plant defense responses that lead to the improvement of innate immunity in plants.

Downy mildew is one of the severe diseases of pearl millet, globally affecting its commercial production. Priming of seeds of a susceptible cultivar of pearl millet with β-aminobutyric acid (BABA) and Pseudomonas fluorescens has reduced the downy mildew disease incidence level under field studies. In the current study, proteomic approach was used to elucidate the poorly studied resistance mechanism in these elicitor primed pearl millet seeds in response to Sclerospora graminicola infection. 2DE-MS/MS based proteomic approach revealed that majority of the 63 differentially accumulated (p≤0.05) proteins associated with energy and metabolism followed by stress and defense category. Multivariate statistics disclosed that infection caused by the pathogen rather than elicitor treatment had a major influence on the dynamics of protein abundance. Mechanism of priming mediated by BABA and P. fluorescens were different from each other as evident by the protein abundance profile of hierarchical clustering analysis. Over-representation of proteins pertaining to glucose metabolism suggests that seed priming ensures plant protection against disease without compromising its normal growth and development. In addition the study forms a basis for future investigation by functional analysis of these differentially accumulated proteins to further unravel the resistance mechanism of elicitor primed plant against the S. graminicola.

BIOLOGICAL SIGNIFICANCE: The study is based on the comparative proteomic analysis between BABA and P. fluorescens mediated resistance in pearl millet, in response to downy mildew causing biotroph - S. graminicola. To our knowledge, this article is the first to report on seedling proteome of pearl millet whose genome is not yet sequenced. In addition, the study also provides clue for the plausible antagonistic cross-talk that might exist between jasmonic acid signaling and salicylic acid signaling in SAR and ISR mediated resistance by BABA and P. fluorescens against the downy mildew pathogen. Furthermore, pearl millet seedling proteome being perturbed by pathogen inoculation was more apparent than that caused by elicitor treatment, as revealed by multivariate statistics like PCA. Analysis by gene enrichment tools further revealed that the glucose metabolism pathway was majorly being affected in our study. This could be attributed to the essential balance that is being maintained in energy diversion towards stress and normal physiological process due to the priming effect of the elicitors against biotic stress.


Plant innate immunity is composed of two layers - a basal immunity, and a specific effector-triggered immunity, which is often accompanied by hypersensitive cell death. Initiation of cell death depends on a complex network of signalling pathways. The phytohormone auxin as central regulator of plant growth and development represents an important component for the modulation of plant defence. In our previous work, we showed that cell death is heralded by detachment of actin from the membrane. Both, actin response and cell death, are triggered by the bacterial elicitor harpin in grapevine cells. In this study we investigated, whether harpin-triggered actin bundling is necessary for harpin-triggered cell death. Since actin organisation is dependent upon auxin, we used different auxins to suppress actin bundling. Extracellular alkalisation and transcription of defence genes as the basal immunity were examined as well as cell death. Furthermore, organisation of actin was observed in response to pharmacological manipulation of reactive oxygen species and phospholipase D. We find that induction of defence genes is independent of auxin. However, auxin can suppress harpin-induced cell death and also counteract actin bundling. We integrate our findings into a model, where harpin interferes with an auxin dependent pathway that sustains dynamic cortical actin through the activity of phospholipase D. The
Antagonism between growth and defence is explained by mutual competition for signal molecules such as superoxide and phosphatidic acid. Perturbations of the auxin-actin pathway might be used to detect disturbed integrity of the plasma membrane and channel defence signalling towards programmed cell death.


The involvement of glutathione (GSH) in plant defense against pathogen invasion is an established fact. However, the molecular mechanism conferring this tolerance remains to be explored. Here, proteomic analysis of pad2-1, an Arabidopsis thaliana GSH-depleted mutant, in response to Pseudomonas syringae infection has been performed to explore the intricate position of GSH in defense against biotrophic pathogens. The pad2-1 mutant displayed severe susceptibility to P. syringae infection compared to the wild-type (Col-0) thus re-establishing a fundamental role of GSH in defense. Apart from general up-accumulation of energy metabolism-related protein-species in both infected Col-0 and pad2-1, several crucial defense-related protein-species were identified to be differentially accumulated. Leucine-rich repeat-receptor kinase (LRR-RK) and nucleotide-binding site-leucine-rich repeat resistance protein (NBS-LRR), known to play a pioneering role against pathogen attack, were only weakly up-accumulated in pad2-1 after infection. Transcriptional and post-transcriptional regulators like MYB-P1 and glycine-rich repeat RNA-binding protein (GRP) and several other stress-related protein-species like heat shock protein 17 (HSP17) and glutathione-S-transferase (GST) were also identified to be differentially regulated in pad2-1 and Col-0 in response to infection. Together, the present investigation reveals that the optimum GSH-level is essential for the efficient activation of plant defense signaling cascades thus conferring resistance to pathogen invasion.


Terpenoid phytoalexins function as defense compound against a broad spectrum of pathogens and pests in the plant kingdom. However, the role of phytoalexin in antiviral defense is still elusive. In this study, we identified the biosynthesis pathway of a sesquiterpenoid phytoalexin, capsidiol 3-acetate as an antiviral response against RNA virus Potato Virus X (PVX) in Nicotiana benthamiana. NbTPS1 and NbEAH genes were found strongly induced by PVX-infection. Enzymatic activity and genetic evidence indicated that both genes were involved in the PVX-induced biosynthesis of capsidiol 3-acetate. NbTPS1- or NbEAH-silenced plant was more susceptible to PVX. The accumulation of capsidiol 3-acetate in PVX-infected plant was partially regulated by jasmonic acid signaling receptor COI1. These findings provide an insight into a novel mechanism of how plant uses the basal arsenal machinery to mount a fight against virus attack even in susceptible species.

Plants are protected from microbial infection by a robust immune system. Two of the earliest responses mediated by surface-localized immune receptors include an increase in cytosolic calcium (Ca(2+)) and a burst of apoplastic reactive oxygen species (ROS). The Arabidopsis plasma membrane-associated cytoplasmic kinase BIK1 is an immediate convergent substrate of multiple surface-localized immune receptors that is genetically required for the PAMP-induced Ca(2+) burst and directly regulates ROS production catalyzed by the NADPH oxidase RBOHD. We recently demonstrated that Arabidopsis plants maintain an optimal level of BIK1 through a process of continuous degradation regulated by the Ca(2+)-dependent protein kinase CPK28. cpk28 mutants accumulate more BIK1 protein and display enhanced immune signaling, while plants over-expressing CPK28 accumulate less BIK1 protein and display impaired immune signaling. Here, we show that CPK28 additionally contributes to the PAMP-induced Ca(2+) burst, supporting its role as a negative regulator of BIK1.


In our study, we report the identification, characterization, and gene cloning of a novel protein elicitor (PeBL1) secreted from Brevibacillus laterosporus strain A60. Through a purification process consisting of ion exchange chromatography and HPLC, we isolated a protein that was identified by ESI-QTOF-MS/MS. The 351-bp PeBL1 gene produces a 12833 Da protein with 116 amino acids that contains a 30-residue signal peptide. The PeBL1 protein was expressed in Escherichia coli. The recombinant protein can induce a typical hypersensitive response (HR) and systemic resistance in Nicotiana benthamiana as the endogenous protein. PeBL1-treated N. benthamiana exhibited a strong resistance to the infection of TMV-GFP and P. syringae pv. tabaci compared to control N. benthamiana. In addition, PeBL1 triggered a cascade of events that resulted in defense responses in plants, including reactive oxygen species (ROS) production, extracellular medium alkalization, phenolic compounds deposition and several defense-related genes. Real-time quantitative PCR analysis indicated that the known defense-related genes PR-1, PR-5, PDF1.2, NPR1 and PAL were up-regulated to varying degrees by PeBL1. This research not only provides insights into the mechanism by which beneficial bacteria activate plant systemic resistance but also sheds new light on a novel strategy for biocontrol using strain A60.


Upon recognition of microbe-associated molecular patterns (MAMPs) such as the bacterial flagellin (or the derived peptide flg22) by pattern-recognition receptors (PRRs) such as the FLAGELLIN SENSING2 (FLS2), plants activate the pattern-triggered immunity (PTI) response. The L-type lectin receptor kinase VI.2 (LecRK-VI.2) is a positive regulator of Arabidopsis thaliana PTI. Cysteine-rich receptor-like kinases (CRKs) possess two copies of the C-X8-C-X2-C (DUF26) motif in their extracellular domains and are thought to be involved in plant stress resistance, but data about CRK functions are scarce. Here, we show that Arabidopsis overexpressing the LecRK-VI.2-responsive CRK4, CRK6, and CRK36 demonstrated an enhanced PTI response and were resistant to virulent bacteria Pseudomonas syringae pv. tomato DC3000. Notably, the flg22-triggered oxidative burst was primed in CRK4, CRK6, and CRK36 transgenics and up-regulation of the PTI-responsive gene FLG22-INDUCED RECEPTOR-LIKE 1 (FRK1) was potentiated upon flg22 treatment in CRK4
and CRK6 overexpression lines or constitutively increased by CRK36 overexpression. PTI-mediated callose deposition was not affected by overexpression of CRK4 and CRK6, while CRK36 overexpression lines demonstrated constitutive accumulation of callose. In addition, Pst DC3000-mediated stomatal reopening was blocked in CRK4 and CRK36 overexpression lines, while overexpression of CRK6 induced constitutive stomatal closure suggesting a strengthening of stomatal immunity. Finally, bimolecular fluorescence complementation and co-immunoprecipitation analyses in Arabidopsis protoplasts suggested that the plasma membrane localized CRK4, CRK6, and CRK36 associate with the PRR FLS2. Association with FLS2 and the observation that overexpression of CRK4, CRK6, and CRK36 boosts specific PTI outputs and resistance to bacteria suggest a role for these CRKs in Arabidopsis innate immunity.


In our experimental approach we investigated how post-infection nitric oxide-dependent signaling activated in potato leaves was related to defense against avirulent (avr) and virulent (vr) races of Phytophthora infestans. Results revealed that only in an incompatible response, early NO and superoxide (O2 •−) generation led to peroxynitrite (ONOO−) formation and together with hydrogen peroxide (H2O2) production synchronized with SOD activity induced effective defense against avr pathogen. Early oxidative and nitrosative bursts triggered an imbalance in redox homeostasis in inoculated tissue. To counteract that effect, a total antioxidative capacity, ascorbate and sulfhydryl (−SH) group compounds increased both synergistically and markedly, confirming the precise mechanism of redox re-adjustment in avr oomycete -potato interaction. Moreover, the NO-coded message was stored and converted into an enhanced total SNO pool and particular S-nitrosylation of targeted proteins. Overall, we identified 104 proteins typed for S-nitrosylation in mock- or P. infestans-inoculated potato leaves. The S-nitrosoproteome structure comprised a wide repertoire of proteins, i.e. defense- and redox-related. Finally, only in the incompatible interaction, NO-based signal was re-written on the rapid PR-1 gene and PR-2 protein activation and was tuned with a limitation of late blight disease symptoms.


Induced resistance to the necrotrophic pathogen Botrytis cinerea depends on jasmonate metabolism and signalling in Arabidopsis. We have presented here extensive jasmonate profiling in this pathosystem and investigated the impact of the recently reported jasmonoyl-isoleucine (JA-Ile) catabolic pathway mediated by cytochrome P450 (CYP94) enzymes. Using a series of mutant and overexpressing (OE) plant lines, we showed that CYP94B3 and CYP94C1 are integral components of the fungus-induced jasmonate metabolic pathway and control the abundance of oxidized conjugated but also some unconjugated derivatives, such as sulfated 12-HSO4-JA. Despite causing JA-Ile overaccumulation due to impaired oxidation, CYP94 deficiency had negligible impacts on resistance, associated with enhanced JAZ repressor transcript levels. In contrast, plants overexpressing (OE) CYP94B3 or CYP94C1 were enriched in 12-OH-JA-Ile or 12-COOH-JA-Ile respectively. This shift towards oxidized JA-Ile derivatives was concomitant with strongly impaired defence gene induction and reduced disease resistance. CYP94B3-OE, but unexpectedly not CYP94C1-OE, plants displayed reduced JA-Ile levels compared with the wild type, suggesting that increased susceptibility in CYP94C1-OE plants may result from changes in the hormone oxidation ratio rather than absolute changes in JA-Ile levels. Consistently, while feeding JA-Ile to seedlings triggered strong induction of JA pathway genes, induction was largely
reduced or abolished after feeding with the CYP94 products 12-OH-JA-Ile and 12-COOH-JA-Ile, respectively. This trend paralleled in vitro pull-down assays where 12-COOH-JA-Ile was unable to promote COI1–JAZ9 co-receptor assembly. Our results highlight the dual function of CYP94B3/C1 in antimicrobial defence: by controlling hormone oxidation status for signal attenuation, these enzymes also define JA-Ile as a metabolic hub directing jasmonate profile complexity.


MicroRNAs (miRNAs) are small non-coding RNAs that have important regulatory functions in plant growth, development, and response to abiotic stress. Increasing evidence also supports that plant miRNAs contribute to immune responses to pathogens. Here, we used deep sequencing of small RNA libraries for global identification of rice miRNAs that are regulated by fungal elicitors. We also describe 9 previously uncharacterized miRNAs in rice. Combined small RNA and degradome analyses revealed regulatory networks enriched in elicitor-regulated miRNAs supported by the identification of their corresponding target genes. Specifically, we identified an important number of miRNA/target gene pairs involved in small RNA pathways, including miRNA, heterochromatic and trans-acting siRNA pathways. We present evidence for miRNA/target gene pairs implicated in hormone signaling and cross-talk among hormone pathways having great potential in regulating rice immunity. Furthermore, we describe miRNA-mediated regulation of Conserved-Peptide upstream Open Reading Frame (CPuORF)-containing genes in rice, which suggests the existence of a novel regulatory network that integrates miRNA and CPuORF functions in plants. The knowledge gained in this study will help in understanding the underlying regulatory mechanisms of miRNAs in rice immunity and develop appropriate strategies for rice protection.


KEY MESSAGE: The present work demonstrates that induction of defense-related genes in tomato by neem extract was mediated by protein-protein and DNA-protein interactions. The induction of elicitor-mediated defense responses in plants is known, but the molecular mechanisms underlying its induction are not well studied. In the present study, third node leaf from the base of aseptically raised tomato plants was treated with aqueous fruit extracts of Azadirachta indica A. Juss. (neem). Samples were collected from the treated node at 24-h intervals for up to 96 h and analyzed for the gene expression of phenylalanine ammonia lyase (PAL), Peroxidase (POX) and Polyphenol Oxidase (PPO), β-actin (standard). Samples were collected from elicitor-induced node at 5-min interval up to 70 min for analysis of protein-protein and DNA-protein interactions. The results demonstrated the induction of expression of PAL, POX and PPO due to the treatment whereas no change was observed in the expression of β-actin. There was disappearance of lower molecular weight proteins which cross-linked with other proteins to form complexes. MALDI-TOF MS analysis revealed the interaction of mitogen-activated protein kinases (MAPK). The analysis of proteins interacted with DNA after induction by neem extract indicated the involvement of WRKY transcriptional factors. Neem-elicited defense responses could possibly due to interaction of proteins with other proteins and transcription factors with DNA which might be crucial in enhancing the expression of defense-related genes (PAL, POX and PPO).

The ERF family is composed of transcription factors (TFs) that are critical for appropriate Arabidopsis thaliana responses to biotic and abiotic stresses. Here we identified and characterized a member of the ERF TFs group IX, namely ERF96, that when over-expressed enhances Arabidopsis resistance to necrotrophic pathogens such as the fungus Botrytis cinerea and the bacterium Pectobacterium carotovorum. ERF96 is jasmonate (JA)- and ethylene (ET)-responsive and ERF96 transcripts accumulation was abolished in JA-insensitive coi1-16 and in ET-insensitive ein2-1 mutants. Protoplast transactivation and electrophoresis mobility shift analyses revealed that ERF96 is an activator of transcription that binds to GCC elements. In addition, ERF96 mainly localized to the nucleus. Microarray analysis coupled to chromatin immunoprecipitation-PCR of Arabidopsis over-expressing ERF96 revealed that ERF96 enhances the expression of the JA/ET defence genes PDF1.2a, PR-3 and PR-4 as well as the TF ORA59 by direct binding to GCC elements present in their promoters. While ERF96-RNAi plants demonstrated wild-type resistance to necrotrophic pathogens, basal PDF1.2 expression levels were reduced in ERF96 silenced plants. This work revealed ERF96 as a key player of the ERF network that positively regulates the Arabidopsis resistance response to necrotrophic pathogens.


Mitogen-activated protein kinase (MAPK) cascades play central roles in innate immune signalling networks in plants and animals. In plants, however, the molecular mechanisms of how signal perception is transduced to MAPK activation remain elusive. Here we report that pathogen-secreted proteases activate a previously unknown signalling pathway in Arabidopsis thaliana involving the Ga, Gβ, and Gγ subunits of heterotrimeric G-protein complexes, which function upstream of an MAPK cascade. In this pathway, receptor for activated C kinase 1 (RACK1) functions as a novel scaffold that binds to the Gβ subunit as well as to all three tiers of the MAPK cascade, thereby linking upstream G-protein signalling to downstream activation of an MAPK cascade. The protease–G-protein–RACK1–MAPK cascade modules identified in these studies are distinct from previously described plant immune signalling pathways such as that elicited by bacterial flagellin, in which G proteins function downstream of or in parallel to an MAPK cascade without the involvement of the RACK1 scaffolding protein. The discovery of the new protease-mediated immune signalling pathway described here was facilitated by the use of the broad host range, opportunistic bacterial pathogen Pseudomonas aeruginosa. The ability of P. aeruginosa to infect both plants and animals makes it an excellent model to identify novel immunoregulatory strategies that account for its niche adaptation to diverse host tissues and immune systems.
Plants face several challenges by bacterial, fungal, oomycete and viral pathogens during their life cycle. In order to defend against these biotic stresses, plants possess a dynamic, innate, natural immune system that efficiently detects potential pathogens and initiates a resistance response in the form of basal resistance and/or resistance (R)-gene-mediated defense, which is often associated with a hypersensitive response. Depending upon the nature of plant-pathogen interactions, plants generally have two main defense mechanisms, host resistance and nonhost resistance. Host resistance is generally controlled by single R genes and less durable compared to nonhost resistance. In contrast, nonhost resistance is believed to be a multi-gene trait and more durable. In this review, we describe the mechanisms of host and nonhost resistance against fungal and bacterial plant pathogens. In addition, we also attempt to compare host and nonhost resistance responses to identify similarities and differences, and their practical applications in crop improvement.


BACKGROUND: Leucine-rich repeat receptor-like kinases (LRR-RLKs) represent a large class of proteins in regulating plant development and immunity. The LRR-RLK XA21 confers resistance to the bacterial disease caused by the pathogen of Xanthomonas oryzae pv. oryzae (Xoo). Several XA21 binding proteins have been characterized, however the early events governing XA21 signaling have not been fully elucidated.

RESULTS: Here we report the identification of one LRR-RLK gene (XIK1) whose expression is induced rapidly upon the infection with the pathogen of Xoo. Expression pattern analysis reveals that XIK1 is preferentially expressed in reproductive leaves and panicles, and that expression is associated with plant development. By using RNA interference (RNAi), we silenced the expression of XIK1 in rice with Xa21 and found that reduced expression of XIK1 compromised disease resistance mediated by XA21. In addition, we found that the expression of the downstream marker genes of pathogen associated molecular pattern (PAMP) triggered immunity (PTI) in rice was compromised in Xa21 plants silenced for XIK1.

CONCLUSION: Our study reveals that the LRR-RLK gene XIK1 is Xoo-responsive and positively regulates Xa21-mediated disease resistance.


Pathogen recognition induces the production of reactive oxygen species (ROS) by NADPH oxidases in both plants and animals. ROS has direct anti-microbial properties, but also serve as signaling molecules to activate further immune outputs. However, ROS production has to be tightly controlled to avoid detrimental effects on host cells, but yet must be produced in the right amount, at the right place and at the right time upon pathogen perception. Plant NADPH oxidases belong to the respiratory burst oxidase homolog (RBOH) family, which contains 10 members in the model plant Arabidopsis thaliana. The perception of pathogen-associated molecular patterns (PAMPs) by pattern recognition receptors (PRRs) leads to a rapid, specific and strong production of ROS, which is dependent on RBOHD. RBOHD is mainly controlled by Ca2+ via direct binding to EF-hand motifs and phosphorylation by Ca2+-dependent protein kinases. Recent studies have, however, revealed a critical role for a Ca2+-independent regulation of RBOHD. The plasma membrane-associated cytoplasmic kinase BIK1, which is a direct substrate of the PRR
complex, directly interacts with and phosphorylates RBOHD upon PAMP perception. Impairment of these phosphorylation events completely abolishes the function of RBOHD in immunity. These results suggest that RBOHD activity is tightly controlled by multilayered regulations. In this review, we summarize recent advances in our understanding of the regulatory mechanisms controlling RBOHD activation.


Xanthomonas type III effector AvrBsT induces hypersensitive cell death and defence responses in pepper (Capsicum annuum) and Nicotiana benthamiana. Little is known about the host factors that interact with AvrBsT. Here, we identified pepper aldehyde dehydrogenase 1 (CaALDH1) as an AvrBsT-interacting protein. Bimolecular fluorescence complementation and co-immunoprecipitation assays confirmed the interaction between CaALDH1 and AvrBsT in planta. CaALDH1:smGFP fluorescence was detected in the cytoplasm. CaALDH1 expression in pepper was rapidly and strongly induced by avirulent Xanthomonas campestris pv. vesicatoria (Xcv) Ds1 (avrBsT) infection. Transient co-expression of CaALDH1 with avrBsT significantly enhanced avrBsT-triggered cell death in N. benthamiana leaves. Aldehyde dehydrogenase activity was higher in leaves transiently expressing CaALDH1, suggesting that CaALDH1 acts as a cell death enhancer, independently of AvrBsT. CaALDH1 silencing disrupted phenolic compound accumulation, H2O2 production, defence response gene expression, and cell death during avirulent Xcv Ds1 (avrBsT) infection. Transgenic Arabidopsis thaliana overexpressing CaALDH1 exhibited enhanced defence response to Pseudomonas syringae pv. tomato and Hyaloperonospora arabidopsis infection. These results indicate that cytoplasmic CaALDH1 interacts with AvrBsT and promotes plant cell death and defence responses.


The peptide elicitor PIP-1 can induce various immune responses in tobacco cells. Previously, we showed that types of responses induced by PIP-1 are different depending on its stimulation periods; short-term stimulation induces weak responses, whereas long-term stimulation leads to strong responses including production of the phytoalexin capsidiol. However, key components that directly regulate the initiation of capsidiol biosynthesis in response to continuous stimulation with PIP-1 remain unclear. In this study, we designed a photocleavable PIP-1 analog containing 3-amino-3-(2-nitrophenyl)propionic acid as a photocleavable residue. The activity of the analog can be switched-off using UV-irradiation without undesired side effects. This analog induced a significant level of capsidiol production unless UV-irradiated, whereas no capsidiol production was observed when tobacco cells were UV-irradiated 1 h after treatment. Using this analog, we found that the elicitor-inducible 3-hydroxy-3-methylglutaryl-CoA reductase activity is regulated based on the duration of the stimulation with PIP-1, which could be associated with the initiation of capsidiol biosynthesis.

Elicitins are elicitors that can trigger hypersensitive cell death in most Nicotiana spp., but their underlying molecular mechanism is not well understood. The gene Phytophthora capsici INF1 (PcINF1) coding for an elicitin from P. capsici was characterized in this study. Transient overexpression of PcINF1 triggered cell death in pepper (Capsicum annuum L.) and was accompanied by upregulation of the hypersensitive response marker, Hypersensitive Induced Reaction gene 1 (HIR1), and the pathogenesis-related genes SAR82, DEF1, BPR1, and PO2. A putative PcINF1-interacting protein, SRC2-1, was isolated from a pepper cDNA library by yeast two-hybrid screening and was observed to target the plasma membrane. The interaction between PcINF1 and SRC2-1 was confirmed by bimolecular fluorescence complementation and co-immunoprecipitation. Simultaneous transient overexpression of SRC2-1 and PcINF1 in pepper plants triggered intensive cell death, whereas silencing of SRC2-1 by virus-induced gene silencing blocked the cell death induction of PcINF1 and increased the susceptibility of pepper plants to P. capsici infection. Additionally, membrane targeting of the PcINF1–SRC2-1 complex was required for cell death induction. The C2 domain of SRC2-1 was crucial for SRC2-1 plasma membrane targeting and the PcINF1–SRC2-1 interaction. These results suggest that SRC2-1 interacts with PcINF1 and is required in PcINF1-induced pepper immunity.


Clavibacter michiganensis subsp. michiganensis (Cmm) and subsp. sepedonicus (Cms) cause diseases on solanaceous crops. The genomes of both subspecies encode members of the pat-1 family of putative serine proteases known to function in virulence on host plants and induction of hypersensitive responses (HR) on non-hosts. One gene of this family in Cms, chp-7, is required for triggering HR in Nicotiana tabacum. Here, further investigation revealed that mutation of the putative catalytic serine residue at position 232 to threonine abolished the HR induction activity of Chp-7, suggesting that enzymatic activity is required. Purified Chp-7 triggered an HR in N. tabacum leaves in the absence of the pathogen, indicating Chp-7 itself is the HR elicitor from Cms. Ectopic expression of chp-7 constructs in N. tabacum leaves revealed that Chp-7 targeted to the apoplast triggered an HR, while cytoplasmic Chp-7 did not, indicating that Chp-7 induces the HR in the apoplast of N. tabacum leaves. Chp-7 also induced HR in N. sylvestris, a progenitor of N. tabacum, but not in other Nicotiana species tested. ChpG, a related protein from Clavibacter michiganensis subsp. michiganensis, also triggered HR in N. tabacum and N. sylvestris. Unlike Chp-7, ChpG triggered HR in N. clevelandii and N. glutinosa.


Two studies published in Cell describe the presence of a DNA-binding domain in a plant immune receptor that mimics the host transcription factors targeted by pathogen virulence proteins. This binding domain enables the host to detect attempted manipulation of its transcriptional response to infection.

Plants deploy a finely tuned balance between growth and defence responses for better fitness. Crosstalk between defence signalling hormones such as salicylic acid (SA) and jasmonates (JAs) as well as growth regulators plays a significant role in mediating the trade-off between growth and defence in plants. Here, we specifically discuss how the mutual antagonism between the signalling of auxin and SA impacts on plant growth and defence. Furthermore, the synergism between auxin and JA benefits a class of plant pathogens. JA signalling also poses growth cuts through auxin. We discuss how the effect of cytokinins (CKs) is multifaceted and is effective against a broad range of pathogens in mediating immunity. The synergism between CKs and SA promotes defence against biotrophs. Reciprocally, SA inhibits CK-mediated growth responses. Recent reports show that CKs promote JA responses; however, in a feedback loop, JA suppresses CK responses. We also highlight crosstalk between auxin and CKs and discuss their antagonistic effects on plant immunity. Efforts to minimize the negative effects of auxin on immunity and a reduction in SA- and JA-mediated growth losses should lead to better sustainable plant protection strategies.


In this paper we describe PATERN-TRIGGERED IMMUNITY (PTI) COMPROMISED RECEPTOR-LIKE CYTOPLASMIC KINASE 1 (PCRK1) of Arabidopsis thaliana, an RLCK that is important for defense against the pathogen Pseudomonas syringae pv. maculicola ES4326 (Pma ES4326).

We examined defense responses such as bacterial growth, production of reactive oxygen species (ROS) and callose deposition in pcrk1 mutant plants to determine the role of PCRK1 during pathogen infection.

Expression of PCRK1 was induced following pathogen infection. Pathogen growth was significantly higher in pcrk1 mutant lines than in wild-type Col-0. Mutant pcrk1 plants showed reduced pattern-triggered immunity (PTI) against Pma ES4326 after pretreatment with peptides derived from flagellin (flg22), elongation factor-Tu (elf18), or an endogenous protein (pep1). Deposition of callose was reduced in pcrk1 plants, indicating a role of PCRK1 in activation of early immune responses. A PCRK1 transgene containing a mutation in a conserved lysine residue important for phosphorylation activity of kinases (K118E) failed to complement a pcrk1 mutant for the Pma ES4326 growth phenotype.

Our study shows that PCRK1 plays an important role during PTI and that a conserved lysine residue in the putative kinase domain is important for PCRK1 function.

Plant nucleotide-binding, leucine-rich repeat (NB-LRR) proteins confer immunity to pathogens possessing the corresponding avirulence proteins. Activation of NB-LRR proteins is often associated with induction of the hypersensitive response (HR), a form of programmed cell death. NRC1 (NB-LRR Required for HR-Associated Cell Death-1) is a tomato (Solanum lycopersicum) NB-LRR protein that participates in the signalling cascade leading to resistance to the pathogens Cladosporium fulvum and Verticillium dahliae. To identify mutations in NRC1 that cause increased signalling activity, we generated a random library of NRC1 variants mutated in their nucleotide-binding domain and screened them for the ability to induce an elicitor-independent HR in Nicotiana tabacum. Screening of 1920 clones retrieved 11 gain-of-function mutants, with 10 of them caused by a single amino acid substitution. All substitutions are located in or very close to highly conserved motifs within the nucleotide-binding domain, suggesting modulation of the signalling activity of NRC1. Three-dimensional modelling of the nucleotide-binding domain of NRC1 revealed that the targeted residues are centred around the bound nucleotide. Our mutational approach has generated a wide set of novel gain-of-function mutations in NRC1 and provides insight into how the activity of this NB-LRR is regulated.


In response to herbivore attack, plants perceive herbivore associated elicitors (HAE) and rapidly accumulate jasmonic acid (JA) and other phytohormones, which interact in complex ways, such as the crosstalk between JA and salicylic acid (SA). Although recent studies have shown that HAE-induced individual phytohormones can be highly specific among closely related species, it remains unclear how conserved and specific the relationships among HAE-induced phytohormones are. Here we analyzed the correlations among four different phytohormones, JA, JA-isoleucine (JA-Ile), SA, and abscisic acid (ABA) in six closely related Nicotiana species that were induced by three different HAEs. Our results showed that while no clear association between ABA and other phytohormones were found, the positive association between JA and JA-Ile is mostly conserved among closely related Nicotiana species. Interestingly, the association between JA and SA are highly variable and can be regulated by different HAEs.


Plants and their biotic enemies, such as microbial pathogens and herbivorous insects, are engaged in a desperate battle which would determine their survival-death fate. Plants have evolved efficient and sophisticated systems to defend against such attackers. In recent years, significant progress has been made towards a comprehensive understanding of inducible defence system mediated by jasmonate (JA), a vital plant hormone essential for plant defence responses and developmental processes. This review presents an overview of JA action in plant defences and discusses how microbial pathogens evade plant defence system through hijacking the JA pathway.

Pathogen-associated molecular patterns (PAMPs) activate mitogen-activated protein kinases (MAPKs), essential components of plant defense signaling. Salicylic acid (SA) is also central to plant resistance responses, but its specific role in regulation of MAPK activation is not completely defined. We have investigated the role of SA in PAMP-triggered MAPKs pathways in Arabidopsis SA-related mutants, specifically in the flg22-triggered activation of MPK3 and MPK6. *cim6*, *sid2*, and *npr1* mutants exhibited wild-type-like flg22-triggered MAPKs activation, suggesting that impairment of SA signaling has no effect on the flg22-triggered MAPKs activation. Pretreatment with low concentrations of SA enhanced flg22-induced MPK3 and MPK6 activation in all seedlings except *npr1*, indicating that NPR1 is involved in SA-mediated priming that enhanced flg22-induced MAPKs activation.


Plants have evolved effective defense strategies to protect themselves from various pathogens. Salicylic acid (SA) is an essential signaling molecule that mediates pathogen-triggered signals perceived by different immune receptors to induce downstream defense responses. While many proteins play essential roles in regulating SA signaling, increasing evidence also supports important roles for signaling phospholipids in this process. In this review, we collate the experimental evidence in support of the regulatory roles of two phospholipids, phosphatidic acid (PA), and phosphatidylinositol 4-phosphate (PI4P), and their metabolizing enzymes in plant defense, and examine the possible mechanistic interaction between phospholipid signaling and SA-dependent immunity with a particular focus on the immunity-stimulated biphasic PA production that is reminiscent of and perhaps mechanistically connected to the biphasic reactive oxygen species (ROS) generation and SA accumulation during defense activation.


Mitogen-activated protein kinases (MAPKs) are major components of defense signaling pathways that transduce extracellular stimuli into intracellular responses in plants. Our previous study indicated that SIMPK1/2/3 were associated with nitric oxide-induced defense response in tomato fruit. In this study, we determine whether SIMPK1/2/3 influence tomato fruit’s innate immunity, and whether plant hormones and reactive oxygen species (ROS) are involved in SIMPK1/2/3 defense signaling pathways. Treatment with 10 μM U0126 significantly inhibited the relative expression of SIMPK1, SIMPK2, and SIMPK3 (P < 0.05). U0126-treated fruit showed higher concentrations of auxin indole acetic acid (IAA), abscisic acid (ABA), and gibberellic acid (GA), but a lower concentration of methyl jasmonate (MeJA). The activities of defense enzymes, including β-1, 3-glucanases (GLU), chitinase (CHI), phenylalanine ammonia-lyase (PAL), and polyphenol oxidase (PPO) decreased after U0126 treatment. Meanwhile, H2O2 content increased and catalase (CAT), ascorbate peroxid.
During plant-virus interactions, defence responses are linked to the accumulation of reactive oxygen species (ROS). Importantly, ROS play a dual role by (1) eliciting pathogen restriction and often localized death of host plant cells at infection sites and (2) as a diffusible signal that induces antioxidant and pathogenesis-related defence responses in adjacent plant cells. The outcome of these defences largely depends on the speed of host responses including early ROS accumulation at virus infection sites. Rapid host reactions may result in early virus elimination without any oxidative stress (i.e. a symptomless, extreme resistance). A slower host response allows a certain degree of virus replication and movement resulting in oxidative stress and programmed death of affected plant cells before conferring pathogen arrest (hypersensitive response, HR). On the other hand, delayed host attempts to elicit virus resistance result in an imbalance of antioxidative metabolism and massively stressed systemic plant tissues (e.g. systemic chlorotic or necrotic symptoms). The final consequence of these processes is a partial or almost complete loss of control over virus invasion (compatible infections).


Thousands of putative biosynthetic genes in Arabidopsis thaliana have no known function, which suggests that there are numerous molecules contributing to plant fitness that have not yet been discovered1, 2. Prime among these uncharacterized genes are cytochromes P450 upregulated in response to pathogens3, 4. Here we start with a single pathogen-induced P450 (ref. 5), CYP82C2, and use a combination of untargeted metabolomics and coexpression analysis to uncover the complete biosynthetic pathway to 4-hydroxyindole-3-carbonyl nitrile (4-OH-ICN), a previously unknown Arabidopsis metabolite. This metabolite harbours cyanogenic functionality that is unprecedented in plants and exceedingly rare in nature6, 7; furthermore, the aryl cyanohydrin intermediate in the 4-OH-ICN pathway reveals a latent capacity for cyanogenic glucoside biosynthesis8, 9 in Arabidopsis. By expressing 4-OH-ICN biosynthetic enzymes in Saccharomyces cerevisiae and Nicotiana benthamiana, we reconstitute the complete pathway in vitro and in vivo and validate the functions of its enzymes. Arabidopsis 4-OH-ICN pathway mutants show increased susceptibility to the bacterial pathogen Pseudomonas syringae, consistent with a role in inducible pathogen defence. Arabidopsis has been the pre-eminent model system10, 11 for studying the role of small molecules in plant innate immunity12; our results uncover a new branch of indole metabolism distinct from the canonical camalexin pathway, and support a role for this pathway in the Arabidopsis defence response13. These results establish a more complete framework for understanding how the model plant Arabidopsis uses small molecules in pathogen defence.

Disease resistance (R) genes have been isolated from many plant species. Most encode nucleotide binding leucine-rich repeat (NLR) proteins that trigger a rapid localized programmed cell death called the hypersensitive response (HR) upon pathogen recognition. Despite their structural similarities, different NLR are distributed in a range of subcellular locations, and analogous domains play diverse functional roles. The autoactive maize NLR gene Rp1-D21 derives from an intragenic recombination between two NLR genes, Rp1-D and Rp1-dp2, and confers a HR independent of the presence of a pathogen. Rp1-D21 and its N-terminal coiled coil (CC) domain (CCD21) confer autoactive HR when transiently expressed in Nicotiana benthamiana. Rp1-D21 was predominantly localized in cytoplasm with a small amount in the nucleus, while CCD21 was localized in both nucleus and cytoplasm. Targeting of Rp1-D21 or CCD21 predominantly to either the nucleus or the cytoplasm abolished HR-inducing activity. Coexpression of Rp1-D21 or CCD21 constructs confined, respectively, to the nucleus and cytoplasm did not rescue full activity, suggesting nucleocytoplasmic movement was important for HR induction. This work emphasizes the diverse structural and subcellular localization requirements for activity found among plant NLR R genes.


Upon pathogen infection, activation of immune response requires effective transcriptional reprogramming that regulates inducible expression of a large set of defense genes. A number of ethylene-responsive factor transcription factors have been shown to play critical roles in regulating immune responses in plants. In the present study, we explored the functions of Arabidopsis AtERF15 in immune responses against Pseudomonas syringae pv. tomato (Pst) DC3000, a (hemi)biotrophic bacterial pathogen, and Botrytis cinerea, a necrotrophic fungal pathogen. Expression of AtERF15 was induced by infection of Pst DC3000 and B. cinerea and by treatments with salicylic acid (SA) and methyl jasmonate. Biochemical assays demonstrated that AtERF15 is a nucleus-localized transcription activator. The AtERF15-overexpressing (AtERF15-OE) plants displayed enhanced resistance while the AtERF15-RNAi plants exhibited decreased resistance against Pst DC3000 and B. cinerea. Meanwhile, Pst DC3000- or B. cinerea-induced expression of defense genes was upregulated in AtERF15-OE plants but downregulated in AtERF15-RNAi plants, as compared to the expression in wild type plants. In response to infection with B. cinerea, the AtERF15-OE plants accumulated less reactive oxygen species (ROS) while the AtERF15-RNAi plants accumulated more ROS. The flg22- and chitin-induced oxidative burst was abolished and expression levels of the pattern-triggered immunity-responsive genes AtFRK1 and AtWRKY53 were suppressed in AtERF15-RNAi plants upon treatment with flg22 or chitin. Furthermore, SA-induced defense response was also partially impaired in the AtERF15-RNAi plants. These data demonstrate that AtERF15 is a positive regulator of multiple layers of the immune responses in Arabidopsis.
A variety of enzymatic responses of ginger plants to Pythium infection after induction of SAR (systemic acquired resistance) have been investigated. Results of pathogenicity test of *P. aphanidermatum* on a susceptible ginger cultivar showed that disease intensity increased with time up to 28 days but Polyphenol oxidase (PPO), Lipoxygenase (LOX) and Phenyl alanine ammonia lyase (PAL) activities increased up to 14 days following inoculation and then declined whereas Peroxidase (PO) activity reached their peaks on 21st day after inoculation and then decreased sharply. To induce SAR, rhizome seeds were soaked separately in salicylic acid (SA-5 mM) and Acalypha leaf extract (ALE – 10%) for 1 hour prior to sowing. Significant disease reduction was observed in both SA and ALE treated plants. SA and ALE treatment enhanced activities of all four defence related enzymes in ginger leaves but the rate of increase was higher in untreated inoculated and treated non-inoculated plants in relation to their respective controls. Treated inoculated plants exhibited maximum activity for all four enzymes. SA stimulated PO and PAL more than that of ALE. Results suggest that a correlation exists between reduction of disease intensity due to SAR induction and greater stimulation of specific enzymatic activities in ginger plants although not all four enzymes are equally responsive to a defence activator.