The Role of Salicylic Acid and Jasmonic Acid in Pathogen Defence

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Abstract: **Phytohormones are not only instrumental in regulating developmental processes in plants but also play important roles for the plant's responses to biotic and abiotic stresses. In particular, abscisic acid, ethylene, jasmonic acid, and salicylic acid have been shown to possess crucial functions in mediating or orchestrating stress responses in plants. Here, we review the role of salicylic acid and jasmonic acid in pathogen defence responses with special emphasis on their function in the solanaceous plant potato.**

Key words: **Plant-pathogen interaction, pathogen-associated molecular patterns, systemic acquired resistance, *Phytophthora infestans, Solanum tuberosum*.**

Pathogen Defence

Plants are able to successfully defend themselves against most pathogens by either constitutive barriers or by the activation of multicomponent defence responses. The ability of all members of a given plant species to prevent propagation of any member of a given pathogen species is described as species resistance or nonhost resistance. According to recent models, this durable form of resistance was overcome during evolution by races of a pathogen which had acquired virulence factors enabling them to evade or suppress defence of a particular plant species (Nürnberger and Lipka, 2005). Consequently, this plant species became a host for this particular pathogen. The susceptibility of a plant species to thus "adapted" pathogens is called basic compatibility. In turn, plant resistance (R) genes evolved which allowed the recognition of strain- or race-specific factors of the adapted pathogen. These resistance genes therefore confer race-cultivar-specific resistance which is best described by the gene-for-gene hypothesis (Flor, 1971).

Upon recognition of pathogens by a plant, signal transduction cascades are initiated that comprise changes in ion fluxes, generation of reactive oxygen species, activation of mitogen-activated protein kinase cascades, defence gene expression, and accumulation of antimicrobial compounds (Nürnberger and Scheel, 2001). Qualitatively, defence responses initiated during nonhost-pathogen interactions are similar to those induced by R proteins, although the latter have been shown to be stronger and activated more rapidly (Navarro et al., 2004).

In addition to local reactions, plants often activate systemic defence responses in uninfected parts of the plant, which are characterized by the systemic activation of pathogenesis-related (*PR*) genes and enhanced resistance to subsequent infections. This systemic acquired resistance is long-lasting and active against a broad range of pathogens, including viruses, bacteria, fungi, and oomycetes (Durrant and Dong, 2004).

Signals from the Pathogen

Recognition in nonhost resistance is mediated by pathogen-associated molecular patterns (PAMPs) which are evolutionarily conserved, not present in the host and of importance for the viability of the pathogen (Gomez-Gomez and Boller, 2002; Nürnberger and Brunner, 2002). PAMP recognition initiates signal transduction chains that lead to the activation of defence responses. Importantly, these PAMP-mediated defence reactions can contribute to resistance (Zipfel et al., 2004). In susceptible plants, PAMP-induced defences occur, but are not sufficient to arrest pathogen growth, possibly because pathogen effectors are able to suppress PAMP-mediated defence (Abramovitch and Martin, 2004; Nomura et al., 2005; Kim et al., 2005).

The interaction of potato with *Phytophthora infestans,* the causal agent of late blight disease, represents one of the most important pathosystems for solanaceous plants. *P. infestans* belongs to the oomycetes which comprise the most devastating plant pathogens including *Phytophthora sojae* and *Phytophthora ramorum*. *Phytophthora* is one of the plant pathogen species for which a PAMP has been identified. An extracellular 42-kD glycoprotein with transglutaminase activity, initially isolated from *P. sojae,* but present in at least 10 different *Phytophthora* species, acts as an elicitor of defence responses in parsley (Brunner et al., 2002). 13 amino acids from this transglutaminase are both necessary and sufficient for the elicitor activity of the 42-kD glycoprotein (Sacks et al., 1995). The oligopeptide elicitor Pep-13 initiates a receptor-mediated signal transduction chain leading to the activation of defence responses in parsley (Nürnberger et al., 1994). Interestingly, sub-

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stitution of amino acids which are crucial for elicitor activity also compromises transglutaminase enzyme activity (Brunner et al., 2002). Consequently, the oligopeptide elicitor Pep-13 fulfills criteria required for PAMPs, since the surface-exposed Pep-13 motif in the transglutaminase is highly conserved among different *Phytophthora* species, of importance for transglutaminase enzyme activity and not present in plant proteins (Brunner et al., 2002).

In potato, treatment with Pep-13 results in typical defence responses, such as the oxidative burst, activation of defence gene expression, and the accumulation of the signalling compounds salicylic acid and jasmonic acid (Halim et al., 2004). However, despite the presence of the Pep-13-containing glycoprotein on the mycelial surface of *P. infestans,* no or only slow defence responses are activated upon infection of susceptible potato plants. It might therefore be concluded that *P. infestans* is able to suppress plant defence responses or that it possesses virulence factors that overcome basal defence.

The Role of Salicylic Acid in Pathogen Defence

Salicylic acid was identified as a crucial signaling molecule required for the expression of plant defence responses. Transgenic *Arabidopsis* and tobacco plants that cannot accumulate salicylic acid due to the expression of the bacterial *NahG* gene encoding a salicylate hydroxylase, are hyper-susceptible to virulent pathogens, unable to mount systemic acquired resistance, and impaired, although not completely compromised, in race-cultivar-specific resistance (Delaney et al., 1994; Rairdan and Delaney, 2002). Salicylic acid is also required for the hypersensitive cell death response, which is usually associated with resistance (Alvarez, 2000).

Salicylic acid signalling proceeds *via* the ankyrin repeat-containing protein NPR1, encoded by the *NON EXPRESSOR OF PR1* gene, which was identified in mutant screens of *Arabidopsis* (Cao et al., 1994; Delaney et al., 1995; Shah et al., 1997). NPR1 is present in the cytoplasm, presumably as an oligomer, and responds to pathogen-induced alterations in the cellular redox state by translocating to the nucleus in its monomeric form (Fobert and Despres, 2005; Dong, 2004). Activation of defence gene expression is mediated by interaction of NPR1 with distinct members of the TGA/OBF class of basic domain/Leu zipper transcription factors in the nucleus (Zhang et al., 1999; Despres et al., 2000; Zhou et al., 2000; Niggeweg et al., 2000, Subramaniam et al., 2001), as well as with presumably negative regulatory proteins (Weigel et al., 2005).

In *Arabidopsis,* pathogen-induced salicylic acid accumulation requires *de novo* biosynthesis *via* the isochorismate pathway, since mutants compromised in salicylic acid accumulation carry a defective gene for isochorismate synthase (Wildermuth et al., 2001). On the other hand, labelling studies in tobacco revealed that salicylic acid is also produced *via* the phenylpropanoid pathway from phenylalanine, cinnamic acid, and benzoic acid, or conjugates thereof (Ribnicky et al., 1998). In potato, these compounds were also shown to be precursors for salicylic acid synthesis in untreated plants; however, after elicitor treatment, salicylic acid accumulated that was not derived from phenylalanine (Coquoz et al., 1998), suggesting that here, as in *Arabidopsis,* pathogen or elicitor-induced accumulation of salicylic acid involves synthesis *via* an alternative, possibly the isochorismate, pathway.

The role of salicylic acid for pathogen defence in potato has been controversial. Since a number of potato cultivars already display high background levels of salicylic acid, its function might indeed be different from that in *Arabidopsis* or tobacco. A correlation between the levels of salicylic acid and the degree of resistance against *P. infestans* was postulated, based on the observation that young potato leaves with enhanced levels of salicylic acid are less susceptible to *P. infestans* than older leaves. Moreover, potato varieties with race-nonspecific resistance contain higher levels of salicylic acid than susceptible ones (Coquoz et al., 1995). In addition, the correlation of high basal *PR* gene expression in potato cultivars with higher nonspecific resistance against *P. infestans* suggests a role of salicylic acid-dependent responses for resistance (Vleeshouwers et al., 2000).

Concerning salicylic acid inducibility of pathogen defence responses in potato, contradictory results have been reported. Thus, exogenous application of salicylic acid led to increased *PR* gene expression, but not to enhanced resistance (Coquoz et al., 1995). Yu and co-workers (Yu et al., 1997) reported induction of resistance by exogenous application of salicylic acid only for resistant potato cultivars. In susceptible cultivars, treatment with salicylic acid even increased disease symptoms. More recent reports indicate that benzothiadiazole, which induces systemic acquired resistance in *Arabidopsis,* and, to a lesser extent, acetyl salicylic acid are able to induce resistance against *Alternaria solani* and *Erysiphe cichoracearum* in potato (Bokshi et al., 2003). Expression of bacteriopsin, a proton pump, in transgenic potato plants resulted in a lesion-mimic phenotype with increased pathogen defence responses, including higher salicylic acid levels. However, enhanced resistance was only observed for a specific isolate of *P. infestans*. Other isolates were even more virulent on bacteriopsin-expressing plants, as were other pathogens (Abad et al., 1997). Thus, the contribution of salicylic acid to resistance responses in potato is far from clear.

The potato cultivar Désirée, which does not contain known R genes, but has good nonspecific resistance against *P. infestans,* was transformed with the *NahG* gene from *Pseudomonas putida*. Transgenic plants were deficient in their ability to accumulate salicylic acid in response to pathogen infection or treatment with the oligopeptide elicitor Pep-13 (Halim et al., 2004). Notably, in response to infiltration with Pep-13, *NahG* plants failed to mount an oxidative burst, cell death, accumulation of jasmonic acid, and activation of a subset of defence genes, indicating that salicylic acid is required for elicitor activity of Pep-13 in potato. However, since some *PR* genes were still activated in *NahG* plants in response to Pep-13 infiltration, there are apparently also salicylic acid-independent reactions (Halim et al., 2004). Interestingly, despite reports that *P. infestans* infection is not significantly altered in *NahG* plants compared to wild-type plants (Yu et al., 1997), we found drastically increased growth of the oomycete on *NahG* plants (unpublished). This difference is possibly due to the different methods applied to determine the degree of disease caused by *P. infestans*. Thus, in accordance with Yu et al., (1997), *P. infestans*-induced lesion size is indeed not significantly different on wild-type and *NahG* potato plants (unpublished). Measurement of

pathogen biomass by real time PCR using primers directed against a repetitive sequence of the *P. infestans* genome (Judelson and Tooley, 2000), however, revealed at least ten-fold higher levels of *P. infestans* DNA in samples taken from *NahG* plants, thus suggesting that, as in *Arabidopsis,* tobacco, and other plants, potato also requires salicylic acid for basal defence (unpublished).

Systemic resistance in potato can be induced not only by pathogen infection (Kombrink et al., 1996) but also by treatment with elicitors such as arachidonic acid and other polyunsaturated fatty acids (Coquoz et al., 1995; Cohen et al., 1991), and by Pep-13 (unpublished). In both cases, local, but not systemic, accumulation of salicylic acid was observed (Coquoz et al., 1995; unpublished). However, Yu et al. (1997) showed that arachidonic acid-induced systemic resistance was compromised in *NahG* plants. Considering our observation that *P. infestans* can grow better on *NahG* plants, this loss of arachidonic acid-induced systemic resistance might reflect the better ability of the pathogen to grow in the absence of salicylic acid accumulation. Similar to the loss of arachidonic acid-induced systemic acquired resistance, Pep-13 also did not induce systemic resistance in *NahG* potato plants (unpublished).

The Role of Jasmonic Acid in Pathogen Defence

Based on studies in *Arabidopsis,* it is generally assumed that the life-style of a pathogen determines which signal transduction pathway becomes activated, with biotrophic pathogens inducing defence responses *via* the salicylic acid signalling pathway (McDowell and Dangl, 2000). Jasmonic acid-dependent defence responses, on the other hand, are considered to be activated in *Arabidopsis* in response to infection with necrotrophic pathogens which require host cell death to obtain nutrients (Glazebrook, 2005).

Jasmonic acid is an important signalling molecule for the activation of defence in response to wounding, herbivores and pathogen attack (Rosahl and Feussner, 2004). It is synthesized from α-linolenic acid by enzymes of the lipoxygenase pathway (Feussner and Wasternack, 2002). Linolenic acid liberated from chloroplast membranes, putatively by phospho-/galactolipases, is oxygenated by plastid-localized 13-lipoxygenases to 13-hydroperoxyoctadecatrienoic acid, which serves as a substrate for a number of cytochrome P450 enzymes. One of these, allene oxide synthase (AOS), catalyzes the conversion of 13-hydroperoxyoctadecatrienoic acid to an unstable epoxide, allene oxide, which is subsequently cyclized by allene oxide cyclase (AOC) to *cis*(+)-12-oxophytodienoic acid (OPDA). OPDA is also found in a membrane-bound form as sn1-O-(12-oxophytodienoyl)-sn2-O-(hexadecatrienoyl)-monogalactosyldiglyceride (Stelmach et al., 2001). Further enzymatic conversions take place in the peroxisome, to which OPDA is translocated. OPDA reductase (OPR3) reduces the double bond of the cyclopentenone, leading to the formation of 12-oxophytoenoic acid. In the peroxisome, three rounds of beta-oxidation are required to synthesize jasmonic acid. A new type of peroxisomal acyl-coenzyme A synthetase was identified in *A. thaliana* which is capable of catalyzing the synthesis of the CoA ester of OPDA, as well as of 3-oxopentenyl-cyclopentane-hexanoic acid (Schneider et al., 2005). The wound-inducible acyl-CoA oxidase gene *ACX1* from *Arabidopsis* was shown to be required for jasmonic acid-dependent responses (Cruz Castillo et al., 2004). Recombinant ACX1A, encoded by a gene identified from a jasmonic acid-deficient tomato mutant, is indeed able to catalyze the first step in the β-oxidative chain shortening of 12-oxophytoenoic acid (Li et al., 2005). Multifunctional proteins which possess 2-trans-enoyl-CoA hydratase and 3-hydroxyacyl-CoA dehydrogenase activities, as well as a 3-ketoacyl-CoA thiolase are responsible for the β-oxidation steps (Cruz Castillo et al., 2004; Afitlhile et al., 2005). Finally, a thioesterase is presumably involved in the release of jasmonic acid from its CoA ester (Li et al., 2005).

The importance of jasmonic acid for wound signalling and its role in defence against insect attack was discovered in solanaceous plants. Thus, wound-induced expression of proteinase inhibitors was first described for tomato (Green and Ryan, 1972) and potato (Sanchez-Serrano et al., 1986), as well as the finding that expression of the corresponding genes is also inducible by exogenous application of jasmonic acid (Farmer et al., 1992).

In *Arabidopsis,* mutants were identified with either compromized jasmonic acid biosynthetic capacity *(fad3-2 fad7 fad8, dad1, opr3, dde1, dde2)* or with defects specifically in jasmonic acid perception or signal transduction *(jar1, coi1, jin1).* Thus, mutants containing only small amounts of linolenic acid due to three defective fatty acid desaturase genes (*fad3-2 fad7 fad8;* McConn and Browse, 1996) were not able to synthesize significant amounts of jasmonic acid. The mutant plants were severely compromized in their defence against insect attack and succumbed to infection by pathogenic soil fungi (Vijayan et al., 1998). Mutants defective specifically in the jasmonic acid biosynthetic pathway were reported for the *AOS* and *OPR3* genes of *Arabidopsis*. The *AOS* mutants *dde2* (*delayed-dehiscence;* von Malek et al., 2002) and *aos* (Park et al., 2002) were unable to accumulate jasmonic acid in response to wounding; however, no reports on *aos* mutants and their response to microbial pathogens are available so far. Mutations in the *OPR3* gene, on the other hand, were instrumental in demonstrating the role of OPDA for pathogen defence in *Arabidopsis*. *opr3* mutant plants, which do not accumulate jasmonic acid but still contain OPDA, are resistant both to infection with the necrotrophic fungus *Alternaria brassicicola* and to attack by the insect *Bradysia impatiens,* in contrast to the triple fad mutant *(fad3-2 fad7 fad8)* which was highly susceptible (Stintzi et al., 2001). These observations suggest that OPDA alone is sufficient for defence against herbivores and microbial pathogens.

As a crucial player in jasmonic acid signalling, the F-box protein COI1 was identified by isolation of the coronatine-insensitive mutant *coi1* (Feys et al., 1994). COI1 is part of the SCF complex involved in protein ubiquitination and degradation. Loss of *COI1* results in jasmonic acid insensitivity, indicating that removal of a negative factor is important for the activation of jasmonic acid-dependent responses. As a potential substrate for COI1-mediated ubiquitination, and thus a possible regulator of jasmonic acid responses, a histone deacetylase, which is assumed to act in repression of transcription, was identified as a COI1 interacting protein (Devoto et al., 2002). The *Arabidopsis* mutant *cos1,* which was isolated as a suppressor of the *coi1* phenotype, is defective in a gene encoding a lumazine synthase, which is involved in the biosynthesis of riboflavin (Xiao et al., 2004).

In contrast to resistant wild-type plants, *coi1* plants are highly susceptible to herbivore attack and to infection by *Alternaria brassicicola* (Stintzi et al., 2001). Defects in genes encoding other components or interacting proteins of SCF complexes also result in jasmonic acid insensitivity. Thus, *jasmonic-acid-insensitive 4 (jai4),* isolated in the ethylene-insensitive background *ein3,* is affected in *AtSGT1b,* a protein which has a role in regulation of the ubiquitin-proteasome pathway (Lorenzo and Solano, 2005). The auxin-resistant mutant, *axr1,* which is affected in a protein modulating SCF activity, was also isolated as a jasmonate-insensitive mutant (Tiryaki and Staswick, 2002). *axr1* itself was shown to be impaired in jasmonate responses (Xu et al., 2002), as are transgenic plants with reduced functions of the light-regulated COP9 signalosome (Xu et al., 2002).

Transcriptional regulation of jasmonic acid-responsive genes is affected in the jasmonate-insensitive mutant *jin1* (Berger et al., 1996; Lorenzo et al., 2004). *JIN1* encodes a nuclear-localized basic helix-loop-helix leucine zipper transcription factor which, interestingly, appears to negatively regulate pathogen defence genes and to activate transcription of wound response genes (Lorenzo et al., 2004; Anderson et al., 2004). Consistent with this, *jin1* mutants were shown to be more resistant to pathogens (Nickstadt et al., 2004).

Another jasmonate-insensitive mutant, *jar1,* was isolated based on its resistance to root growth inhibition by the methyl ester of jasmonic acid (Staswick et al., 1992). The protein encoded by the affected gene is a jasmonic acid-amino acid synthetase that is required to activate jasmonic acid for optimal signalling in *Arabidopsis* (Staswick and Tiryaki, 2004). The ability of JAR1 to form jasmonic acid conjugates with the ethylene precursor 1-aminocyclopropane-1-carboxylic acid, moreover, suggests a role of *JAR1* in cross talk with ethylene signalling.

Mitogen-activated protein kinase cascades are activated in response to PAMP recognition (Ligterink et al., 1997; Asai et al., 2002) and in R gene-mediated resistance responses (Menke et al., 2004). Functional evidence for a role of mitogen-activated protein kinases in jasmonic acid-regulated signalling was based on the *Arabidopsis* mutant *mpk4,* which was initially isolated as a mutant exhibiting constitutive systemic acquired resistance with elevated levels of salicylic acid. The loss of *mitogen-activated protein kinase 4* expression also correlated with compromised jasmonic acid-responsive gene expression, which, however, was shown to be independent of the high SA levels in the *mpk4* mutant (Petersen et al., 2000).

Regarding the role of jasmonic acid for defence responses in solanaceous plants, analyses were carried out mostly with tomato mutants. Thus, *def1* tomato plants are defective in the wound-induced jasmonic acid biosynthesis and accumulate less jasmonic acid (Howe et al., 1996). Moreover, mutant plants show decreased resistance against attack by the tobacco hornworm *Manduca sexta,* indicating that octadecanoid metabolism also plays a crucial role for insect defence in tomato. The homologue of COI1 in tomato was identified in the mutant *jai1 (jasmonate insensitive1)* which did not respond to exogenous application of jasmonic acid and which was severely compromised in resistance to the two-spotted spider mite (Li et al., 2004).

In potato, transgenic approaches aiming at increased or reduced jasmonic acid biosynthetic capacity were carried out. Expression of antisense constructs of a wound- (Royo et al., 1996) and pathogen- (Göbel et al., 2002) inducible 13-lipoxygenase resulted in increased susceptibility to herbivore feeding (Royo et al., 1999). Proteinase inhibitor transcript levels were significantly reduced, and insects feeding on the transgenic plants gained more weight than those feeding on wild-type plants.

Indications for a role of jasmonic acid for pathogen defence in potato arose from reports that exogenous application of jasmonic acid leads to local and systemic protection against subsequent pathogen attack (Cohen et al., 1993). Moreover, in response to infection with the nonhost bacteria *Pseudomonas syringae* pv. *maculicola* or in response to treatment with the oligopeptide elicitor Pep-13, infiltrated potato leaves accumulate not only salicylic acid, but also jasmonic acid as well as OPDA (Landgraf et al., 2002; Halim et al., 2004). Jasmonic acid does not accumulate in susceptible potato cultivars after infection with *P. infestans* (Weber et al., 1999; Göbel et al., 2002), suggesting that jasmonic acid accumulation occurs only in the nonhost-pathogen interaction and in response to PAMP recognition. A specific role for OPDA in systemic acquired resistance might be hypothesized based on the observation that OPDA, but not salicylic acid or jasmonic acid, accumulates systemically in potato plants induced for systemic acquired resistance (Landgraf et al., 2002).

In order to specifically address the role of jasmonic acid biosynthesis for defence of potato against microbial pathogens, we generated RNA interference constructs derived from potato ESTs with sequence homology to either tomato or *Arabidopsis* genes encoding AOC and OPR3. Transgenic potato plants had highly reduced *AOC* or *OPR3* transcript levels after wounding, as well as highly reduced jasmonic acid levels (unpublished). *P. infestans* could grow to significantly higher levels on transgenic OPR3-RNAi plants than on wild-type plants as measured by real time PCR; however, pathogen growth was not as high as in *NahG* plants (unpublished). These data suggest that not only salicylic acid but also, albeit to a lower extent, jasmonic acid is of importance for basal defence against *P. infestans* in potato.

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