

REVIEW

Priming: Getting Ready for Battle

Prime-A-Plant Group: Uwe Conrath,¹ Gerold J. M. Beckers,¹ Victor Flors,² Pilar García-Agustín,² Gábor Jakab,³ Felix Mauch,⁴ Mari-Anne Newman,⁵ Corné M. J. Pieterse,⁶ Benoit Poinssot,⁷ María J. Pozo,⁸ Alain Pugin,⁷ Ulrich Schaffrath,¹ Jurriaan Ton,⁶ David Wendehenne,⁷ Laurent Zimmerli,⁹ and Brigitte Mauch-Mani⁹

¹Department of Plant Physiology, RWTH Aachen University, 52056 Aachen, Germany; ²Área de Fisiología Vegetal, Departamento de Ciencias Experimentales, ESTCE, Universitat Jaume I, Campus Riu Sec, 12071 Castellón, Spain; ³Institute of Biology, Faculty of Sciences, University of Pécs, H 7601 Pécs Pf.: 266. Pécs, Hungary; ⁴Department of Biology-Plant Biology, University of Fribourg, 3 Rue Albert Gockel, Fribourg, CH-1700, Switzerland; ⁵Section for Plant Pathology, Royal Veterinary and Agricultural University (KVL), Thorvaldsensvej 40, 1871 Frederiksberg, Denmark; ⁶Section of Phytopathology, Institute of Environmental Biology, Faculty of Science, Utrecht University, PO Box 800.84, 3508 TB Utrecht, The Netherlands; ⁷UMR Plante-Microbe-Environnement INRA 1088/CNRS 5184/ Université de Bourgogne, 17 rue Sully, BP 86510, 21065 Dijon Cédex, France; ⁸Departamento de Microbiología del Suelo y Sistemas Simbióticos, Estación Experimental del Zaidín, CSIC, Profesor Albareda 1, 18008 Granada, Spain; and ⁹Institute of Botany, University of Neuchâtel, Rue Emile Argand 11, PO Box 158, CH-2009 Neuchâtel, Switzerland

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Infection of plants by necrotizing pathogens or colonization of plant roots with certain beneficial microbes causes the induction of a unique physiological state called “priming.” The primed state can also be induced by treatment of plants with various natural and synthetic compounds. Primed plants display either faster, stronger, or both activation of the various cellular defense responses that are induced following attack by either pathogens or insects or in response to abiotic stress. Although the phenomenon has been known for decades, most progress in our understanding of priming has been made over the past few years. Here, we summarize the current knowledge of priming in various induced-resistance phenomena in plants.

Additional keywords: β -aminobutyric acid, bacterial lipopolysaccharides, ethylene, jasmonic acid, mycorrhizal fungi, salicylic acid.

Over the past decades, there has been increasing evidence demonstrating that plants can be primed for more efficient activation of cellular defense responses. The priming-caused potentiation of plant defense responses frequently has been associated with enhanced resistance to various biotic or abiotic stresses. For example, cucumber plants that had been attacked by the fungus *Colletotrichum lagenarium* combated secondary penetration attempts by rapidly depositing effective papillae at the points of attempted pathogen ingress (Kuć 1982). The hypersensitive response (HR) has also been shown to be induced with greater efficacy in plants that had experienced pathogen attack before, resulting in the formation of smaller necrotic HR lesions (Kuć 1995; Ross 1961). The physiological condition in which plants are able to better or more rapidly mount defense responses, or both, to biotic or abiotic stress is called the “primed state” of the plant.

Priming is not exclusive to plants but has also been described in animals. A classic example comes from the enhanced response of mammalian monocytes and macrophages to bacterial lipopolysaccharides (LPS). Upon recognizing bacterial LPS, monocytes and macrophages produce various cytokines with important roles in the defense against viruses, bacteria, parasites, or tumor cells (Chen et al. 1992; Raetz et al. 1991). Since the late 1980s, it has been known that the endogenous protein interferon- γ plays a role in signaling between mammalian cells attacked by pathogens, induces a primed state that enhances the capacity of monocytes and macrophages to respond to lower doses of LPS in inducing cytokine biosynthesis (Gifford und Lohman-Matthes 1987; Hayes et al. 1991; 1995a; Koerner et al. 1987). Although a few studies have suggested that priming and the subsequent potentiation of cellular defense responses in monocytes and macrophages might be mediated partly at the level of transcription (Hayes et al. 1995b), the molecular mechanisms that regulate priming in monocytes and macrophages remain largely unclear.

Initial investigations on priming and the resulting augmentation of cellular defense responses in plants were performed using parsley cell cultures and a pathogen-associated molecular pattern (PAMP; elicitor) from the cell wall of *Phytophthora sojae*. Treatment of parsley cell suspensions with either low doses of the *Phytophthora sojae* elicitor or salicylic acid (SA) did not significantly induce the expression of the defense-related phenylalanine ammonia-lyase (*PAL*) gene (Kauss et al. 1992; Thulke and Conrath 1998). However, when parsley cells had been pretreated with SA and then challenged with a low dose of the *Phytophthora sojae* elicitor, *PAL* mRNA accumulated to extremely high levels (Fig. 1A), indicating that SA had primed the parsley cells for potentiated expression of elicitor-inducible genes, such as *PAL*. The synthetic SA analogs, 2,6-dichloroisonicotinic acid (INA) and benzo (1,2,3) thiadiazole-7-carbothioic acid (BTH) were shown to also prime parsley cells for augmented *PAL* gene expression and enhanced activity of other cellular defense responses. The latter included alterations in ion transport across the plasma membrane (Katz et

Corresponding author: B. Mauch-Mani; E-mail: brigitte.mauch@unine.ch

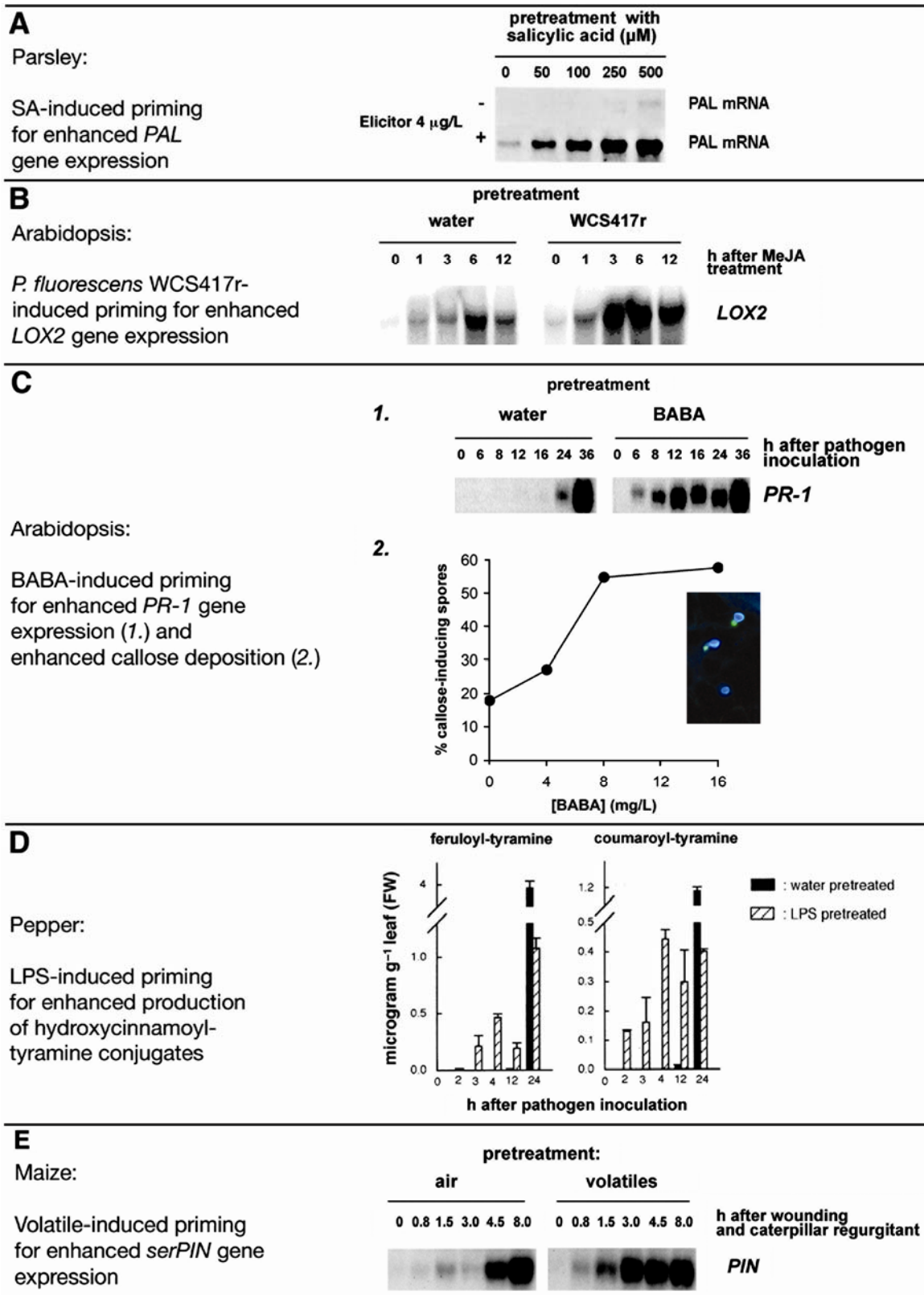


Fig. 1. Priming for enhancement of defense responses in various plant species. **A**, Salicylic acid (SA)-induced priming for augmented phenylalanine ammonia-lyase (*PAL*) gene expression in parsley cell suspensions. Pretreatment with SA results in enhanced *PAL* activation induced by subsequent treatment with an oomycete cell-wall elicitor (Thulke and Conrath 1998). **B**, *Pseudomonas fluorescens* WCS417r-induced priming in *Arabidopsis* for enhanced induction of the *LOX2* gene upon treatment with methyl jasmonate (MeJA). **C**, β -aminobutyric acid (BABA)-induced priming in *Arabidopsis* for earlier and stronger *PR-1* gene expression upon infection by *Pseudomonas syringae* pv. *tomato* DC3000 (1.) and BABA-induced priming for enhanced formation of papillae at two days after infection with spores of *Hyaloperonospora parasitica* WACO9 (2.). Inset shows a representative example of germinating *H. parasitica* spores triggering callose depositions in epidermal cells. (J. Ton, unpublished results). **D**, Lipopolysaccharide (LPS)-induced priming for faster production of the phenolic conjugates coumaroyl tyramine (CT) and feruloyl tyramine (FT) in pepper upon infection with *Xanthomonas campestris* pv. *campestris* (Newman et al. 2002). **E**, Volatile-induced priming for potentiated expression of the jasmonic acid-inducible *PIN* gene in maize upon treatment by wounding and caterpillar regurgitant (J. Ton and T. C. J. Turlings, unpublished results).

al. 2002), the activation of various defense genes (Thulke and Conrath 1998), and the synthesis and secretion of phytoalexins (Katz et al. 1998). Besides pathogens, SA, and its analogs, several other biological and chemical agents emerged as potent inducers of priming, which often has been associated with enhanced resistance to a broad spectrum of biotic or abiotic stresses or both.

Priming in systemic acquired resistance.

Systemic acquired resistance (SAR) is a plant defense state that is induced upon local infection with necrotizing pathogens and which confers resistance to a broad spectrum of attackers (Ryals et al. 1996; Sticher et al. 1997). Induction of SAR requires accumulation of the endogenous signaling molecule SA, which mediates activation of a large set of pathogenesis-related (*PR*) genes (Durrant and Dong 2004). In *Arabidopsis*, biological induction of SAR by previous infection with avirulent *Pseudomonas syringae* pv. *tomato* DC3000 is not only associated with direct activation of *PR* genes, it also primes systemic tissues for potentiated activation of defense-related *PAL*, *PR-1*, *PR-2*, and *PR-5* genes. The potentiated induction of defense responses only becomes apparent after further pathogen challenge (Cameron et al. 1999; Kohler et al. 2002; Van Wees et al. 1999) and, therefore, has often been overseen when studying SAR. In *Arabidopsis*, priming for potentiated *PAL* expression could be mimicked by pretreatment with low doses of the synthetic SAR inducer BTH, which resulted in augmented accumulation of *PAL* mRNA after infection with virulent *Pseudomonas syringae* pv. *tomato* DC3000 (Kohler et al. 2002). Similarly, SA-primed transgenic tobacco plants carrying chimeric *Asparagus officinalis PR-1::uidA* and *PAL-3::uidA* reporter genes displayed potentiated *PR-1* and *PAL* activation after pathogen attack (Mur et al. 1996). These studies indicated a dual role of SA in SAR signaling; either SA directly activates *PR* gene expression or, alternatively, low doses of SA that do not activate defense genes directly prime the tissue for potentiated defense-gene expression upon subsequent pathogen infection.

In *Arabidopsis*, several mutants have been identified with an altered priming phenotype. For instance, mutant *edr1-1*, which had been isolated in a screen for enhanced resistance to *Pseudomonas syringae* pv. *tomato* DC3000, also displays increased resistance to a variety of other pathogens, with no constitutive expression of defense-related *PR-1* and β -1,3 *GLUCANASE* (*BGL2*) genes (Frye and Innes 1998; Van Hulst et al. 2006). However, upon pathogen infection, expression of both genes was augmented in comparison with the expression in infected wild-type plants. In addition, *edr1* plants showed stronger induction of other defense responses, such as the HR and callose deposition, indicating that *edr1* plants are constitutively primed for enhanced activation of various defense mechanisms. *EDR1* encodes a putative mitogen-activated protein kinase (MAPK) kinase kinase (Frye et al. 2001), and the observation that the *edr1* mutant is constitutively primed suggests that *EDR1* functions as a negative regulator of priming.

Another *Arabidopsis* gene implicated in priming is *NPR1* (also known as *NIM1* or *SAI1*). In response to treatment with avirulent pathogens *npr1* mutant plants accumulate wild-type levels of SA, but they are unable to express *PR* genes or SAR (Cao et al. 1994; Delaney et al. 1995; Shah et al. 1997). Not only is the direct activation of defense genes blocked in *npr1*, but also, priming for potentiation of *Pseudomonas syringae* pv. *tomato* DC3000-induced *PAL* gene expression by low doses of BTH is absent in *npr1* (Conrath et al. 2002; Kohler et al. 2002). Thus, *NPR1* apparently plays a role in SA-mediated priming for enhanced defense-gene expression.

In the *cpr1* and *cpr5* mutants of *Arabidopsis*, which both constitutively express *PR* genes and SAR (Bowling et al. 1994;

1997), there was constitutive priming for augmented *PAL* activation upon *Pseudomonas syringae* pv. *tomato* DC3000 infection (Conrath et al. 2002; Kohler et al. 2002). The permanently primed state in *cpr1* and *cpr5* is likely to be caused by the enhanced levels of SA in these plants (Boch et al. 1998; Clarke et al. 2001). In this context, it is noteworthy that the constitutively enhanced pathogen resistance in *cpr5-2* has been ascribed to the potentiated induction of the *PR-1* gene (Boch et al. 1998).

Priming in beneficial plant-microbe interactions.

Mutually beneficial interactions between plants and microbes are frequent in nature and either improve plant nutrition, help the plant to overcome biotic or abiotic stresses, or both. In many cases, plant-microbe associations enhance the defense capacity of the plant and effectively ward off a broad spectrum of pathogens (Pozo et al. 2005). For example, colonization of tomato roots by the mycorrhizal fungus *Glomus mossae* systemically protects the plant against infection by *Phytophthora parasitica* (Cordier et al. 1998; Pozo et al. 2002). Similarly, selected strains of nonpathogenic plant growth-promoting rhizobacteria (PGPR) can reduce disease in above-ground plant parts through the induction of a defense state that is commonly referred to as rhizobacteria-induced systemic resistance (ISR; Van Loon et al. 1998). In general, systemic resistance responses induced by beneficial microorganisms are not associated with major changes in the expression of defense genes (Pieterse et al. 2002), probably because this would lead to heavy investments in resources and reduced fitness of the host (Heil 2002; Van Hulst et al. 2006).

A common feature of the resistance responses induced by beneficial microorganisms is priming. For example, colonization of tomato roots by mycorrhizal fungi systemically protects the plant against *Phytophthora parasitica* infection without direct accumulation of *PR* proteins. However, upon pathogen attack, mycorrhizal plants accumulate significantly more *PR-1a* and basic BGL proteins than nonmycorrhizal plants. Ultrastructure studies revealed that the former displayed pectin- and callose-containing host cell wall depositions at the sites of subsequent pathogen infection, whereas nonmycorrhizal plants did not (Cordier et al. 1998; Pozo et al. 1999, 2002). Similarly, certain plant growth-promoting fungi (PGPF) were reported to induce priming in plants; challenge infection with the leaf pathogen *Pseudomonas syringae* pv. *lachrymans* of cucumber plants that had been preinoculated with the PGPF *Trichoderma asperellum* T203 leads to potentiated *PR* gene expression (Shoresh et al. 2005).

Priming in beneficial plant-microbe associations has most extensively been studied in the interaction of plants with ISR-inducing PGPR. The first evidence that potentiation of plant defense responses is involved in PGPR-mediated ISR came from experiments with ISR-expressing carnation (*Dianthus caryophyllus*) in which inoculation with *Fusarium oxysporum* f. sp. *dianthi* caused a faster rise in phytoalexin levels than in uninoculated control plants (Van Peer et al. 1991). In bean, *Bacillus pumilus* SE34 induced ISR against the root-rot fungus *F. oxysporum* f. sp. *pisi* (Benhamou et al. 1996). Upon challenge infection with the fungus, the root cell walls were rapidly strengthened at the sites of attempted fungal penetration through appositions that contained callose as well as phenolic material and fungal ingress was effectively prevented (Benhamou et al. 1996). In *Arabidopsis*, ISR triggered by *Pseudomonas fluorescens* WCS417r is effective against different types of pathogens, but it is not associated with the activation of *PR* genes (Pieterse et al. 1996). Mutant analysis showed that WCS417r-mediated ISR requires an intact response to jasmonic acid (JA) and ethylene (ET) (Pieterse et al. 1998). Analysis of the *Arabidopsis* transcriptome revealed that, locally in the roots, ISR-inducing

WCS417r bacteria elicited a substantial change in the expression of almost 100 genes (Léon-Kloosterziel et al. 2005; Verhagen et al. 2004). However, in systemic leaves, no consistent alteration in gene expression was observed. Thus, the onset of ISR in leaves is not associated with obvious changes in gene expression (Verhagen et al. 2004). In addition, no alterations in the production of either JA or ET could be detected in plants with ISR, suggesting that the induced resistance state is based on an enhanced sensitivity to these plant hormones rather than on an increase in their production (Pieterse et al. 2000). Analysis of the transcriptome of *Arabidopsis* leaves during ISR to *Pseudomonas syringae* pv. *tomato* DC3000 revealed 81 genes with augmented expression, indicating that the plants were primed to respond either faster, more strongly, or both to pathogen attack (Verhagen et al. 2004). An example for such an augmented expression is shown in Figure 1B. The majority of genes with potentiated expression were predicted to be regulated by either JA, ET, or both, thus confirming earlier findings that colonization of the roots by WCS417r-primed *Arabidopsis* plants for augmented expression of JA- or ET-responsive, or both, genes *AtVSP2*, *PDF1.2*, and *HEL* (Hase et al. 2003; Van Wees et al. 1999).

Other ISR-inducing PGPR have also been demonstrated to enhance the plant's defense capacity by priming for potentiated expression of defense genes (Ahn et al. 2002; De Meyer et al. 1999; Kim et al. 2004; Tjamos et al. 2005), strongly suggesting that priming is a common feature of PGPR-mediated ISR. Ryu and associates (2004) demonstrated that some PGPR can induce priming by the release of volatiles. For instance, *Bacillus subtilis* GB03 induces a signaling pathway that is independent of SA, JA, and the *NPR1* gene, yet it requires ET (Ryu et al. 2004). The GB03 strain produces the C4 carbon compounds 3-hydroxy-2-butanone and (2R,3R)-(-)-2,3 butanediol, which can prime plants for augmented defense responses to attack by herbivores or pathogens (Paré et al. 2005).

Priming by chemicals for potentiated responses to biotic stress.

Priming in BABA-induced resistance to biotic stress. The nonprotein amino acid β -aminobutyric acid (BABA) is known as a potent inducer of resistance in plants against microbial pathogens (Cohen 2002; Jakab et al. 2001), nematodes (Oka et al. 1999), insects (Hodge et al. 2005), and abiotic stresses (Jakab et al. 2005). Research on the mechanisms of BABA-induced resistance (BABA-IR) in *Arabidopsis* has shown that this form of induced resistance, like SAR, is mostly based on priming for different pathogen-inducible defense mechanisms. However, the signaling pathway controlling BABA-IR in *Arabidopsis* is partially different from that of SAR. The pathway that controls BABA-IR against the bacterial pathogen *Pseudomonas syringae* pv. *tomato* DC3000 and the fungal pathogen *Botrytis cinerea* resembles SAR in that it requires endogenous accumulation of SA and an intact NPR1 protein (Zimmerli et al. 2000; 2001). However, in contrast to SAR, a clear priming effect was observed concerning the induction of *PRI* transcripts in BABA-treated plants (Fig. 1C). Yet, BABA-IR against the oomycete pathogen *Hyaloperonospora parasitica* was expressed in *Arabidopsis* genotypes impaired in either the SA, ET, or JA signaling pathway (Zimmerli et al. 2000). Hence, BABA protected *Arabidopsis* independently of these defense signaling cascades, suggesting an additional mechanism of protection. Interestingly, the expression of BABA-IR against *H. parasitica* coincided with a rapid and enhanced deposition of massive callose-containing papillae (Fig. 1C; Zimmerli et al. 2000). The priming for improved papillae formation provided a plausible explanation for the observed protection against *H. parasitica*, since the resulting increase in resistance could make downstream defense

mechanisms, such as PR proteins or camalexin accumulation, no longer critical for resistance.

The correlation between BABA-induced protection and augmented papillae formation, was further investigated in the interaction between *Arabidopsis* and two necrotrophic fungi, *Alternaria brassicicola* and *Plectosphaerella cucumerina*. BABA-IR against *A. brassicicola* was not affected in the camalexin-deficient mutant *pad3-1* or the JA-insensitive mutant *coi1-1*. Furthermore, *Arabidopsis* mutants impaired in SA, ET, or JA signaling or in camalexin biosynthesis were all capable of expressing BABA-IR against *Plectosphaerella cucumerina* (Ton and Mauch-Mani 2004). Hence, camalexin accumulation, as well as SA-, JA-, and ET-dependent defense mechanisms could be excluded as potential pathways of BABA-IR against the two fungi. As observed upon infection by *H. parasitica*, cytological observations at the sites of infection by *A. brassicicola* and *Plectosphaerella cucumerina* revealed that papillae formation was increased in the epidermal cell layer in BABA-treated plants (Ton and Mauch-Mani 2004). Since callose forms an important structural matrix of papillae, 2-deoxy-D-glucose, an inhibitor of callose formation (Jaffe and Leopold 1984), was used to determine the contribution of callose-rich papillae to the protection. The inhibitor decreased the effectiveness of BABA-IR against *A. brassicicola*, suggesting a critical role for callose in BABA-induced protection against this necrotrophic pathogen (Ton and Mauch-Mani 2004). In addition, the callose-deficient mutant *pmr4-1* was completely blocked in expressing BABA-IR against *Plectosphaerella cucumerina*, demonstrating an equally important role for callose in the protection against this pathogen. Interestingly, the priming for enhanced papillae formation at the onset of fungal infection was absent in the abscisic acid (ABA)-signaling mutants *abi4-1* and *aba1-5* (Ton and Mauch-Mani 2004). In addition, exogenous application of ABA mimicked the effect of BABA on increased formation of callose-rich papillae and resistance against fungal infection. Importantly, both *abi4-1* and *aba1-5* mutants retained their capacity to accumulate callose upon infection, but the BABA-mediated priming of papillae formation was lost. These findings indicate that ABA signaling is involved in the regulation of BABA-induced priming for enhanced papillae formation. Additional support for this conclusion came from the characterization of mutants impaired in BABA-induced sterility (*ibs*; Ton et al. 2005). *Arabidopsis* becomes female-sterile when treated with high doses of BABA (Jakab et al. 2001). This fact was used to screen for T-DNA insertion mutants with an altered responsiveness to BABA (Ton et al. 2005). The *ibs3* mutant carries a mutation at the 3' end of the *ABA1* gene that encodes the ABA biosynthetic enzyme zeaxanthin epoxidase. *Ibs3* was impaired in BABA-induced priming for enhanced papillae formation upon infection of BABA-treated plants with *H. parasitica* (Ton et al. 2005).

The molecular mechanisms governing ABA-dependent priming of callose deposition are not clear. However, a possible role for ABA in callose deposition might be deduced from research on abiotic stress, in which different plant SNARE proteins have been implicated in the mediation of ABA-dependent responses (Leymann et al. 1999; Zhu et al. 2002). SNARE proteins have also been linked to disease-resistance mechanisms in the plant cell wall. Mutations in two SNARE-encoding genes, *PEN1* and *ROR2* (Collins et al. 2003; Lipka et al. 2005), lead to a partial loss of resistance at the level of the plant cell wall, which manifests as an enhanced penetration by nonhost fungal pathogens. *PEN1* and *ROR2* are both thought to be involved in cellular targeting of vesicles transporting phytoalexins and callose synthase proteins to sites of fungal penetration. This is supported by the high degree of homology between the above-mentioned SNARE genes and tobacco

NtSyp121, an ABA-inducible SNARE gene (Collins et al. 2003; Leymann et al. 1999). The results point to a prominent role for ABA in the accelerated formation of callose-rich papillae through enhanced, ABA-dependent transcription or activity of SNARE genes upon pathogen attack.

Priming by other chemicals for potentiated responses to biotic stress. Many chemicals with the capacity to induce stress resistance have been described in plants (Sticher et al. 1997). While most reports described chemicals as elicitors that directly induce defense responses in the absence of pathogens, we focus here on chemicals with the potential to function as inducers of priming.

Biosynthesis of the phytoalexin scoparone has been associated with postharvest resistance to pathogens in *Citrus* fruits (Ortuño et al. 1997). Brotomax, a commercial product containing aluminum lignosulphonate, induces resistance against *Phytophthora parasitica* in *Citrus* fruits by priming for enhanced production of scoparone upon infection by *Phytophthora parasitica parasitica* (Ortuño et al. 1997). Similar results have been found for the accumulation of flavanone in *Citrus* fruits after pathogen infection of Brotomax-pretreated plants (Fuster et al. 1995), indicating that aluminum lignosulphonate can prime *Citrus* fruit for augmented phytoalexin production.

The strobilurin fungicide Pyraclostrobin enhances resistance of the tobacco cultivar Xanthi nc against infection by *Tobacco mosaic virus* (TMV) and the wild-fire pathogen *Pseudomonas syringae* pv. *tabaci*. Pyraclostrobin also increases TMV resistance in *NahG*-transgenic tobacco plants (Herms et al. 2002), suggesting that this fungicide enhances TMV resistance in tobacco either by acting downstream of SA in the SA signaling cascade or by functioning independently of SA. Pyraclostrobin did not cause the accumulation of SA-inducible PR-1 defense proteins in infiltrated leaves. However, accumulation of PR-1 was induced more rapidly in Pyraclostrobin-pretreated plants than in water-pretreated controls following TMV infection of tobacco leaves (Herms et al. 2002). These results suggest that Pyraclostrobin, in addition to exerting direct antifungal activity, may also protect plants by priming them for potentiation of subsequent pathogen-induced cellular defense responses. This conclusion is consistent with an earlier report demonstrating that another commercial fungicide, Oryzemat, enhanced the resistance to a bacterial and an oomycete pathogen in *Arabidopsis* (Yoshioka et al. 2001) and to TMV, *Pseudomonas syringae* pv. *tabaci*, and *Oidium* sp. in tobacco (Nakashita et al. 2002). In this context, it is interesting to note that the activity of the fungicides Metalaxyl, Cu(OH)₂, and Fosethyl was impaired in *Arabidopsis* disease-resistance signal transduction mutants (Molina et al. 1998), while their effectiveness was enhanced in *Arabidopsis* plants overexpressing *NPR1* (Friedrich et al. 2001).

Priming by LPS. Innate immune defenses or the nonspecific defense mechanisms that a host uses immediately or within several hours after exposure to nonself molecules can be triggered in eukaryotes by the perception of PAMP, which include LPS and lipooligosaccharides (LOS). LPS are ubiquitous, indispensable components of the cell surface of gram-negative bacteria that have a number of effects in plants, including the ability to prevent the HR induced by avirulent bacteria, priming for more effective induction of plant defense responses such as the oxidative burst, the synthesis of nitric oxide (NO), and the phosphorylation of MAPK (Erbs and Newman 2003). In addition to direct effects on plant tissue, treatment with LPS affect the pattern of accumulation of gene expression and accumulation of certain phenolics in plants in response to subsequent inoculation with virulent or avirulent bacteria. LPS pretreatment of pepper leaves altered patterns of gene expression induced by subsequent challenge with bacteria. Genes encod-

ing the PR proteins P6, acidic BGL, and basic BGL were not induced by *Salmonella minnesota* LPS, while *Xanthomonas campestris* pv. *campestris* LPS gave weak, transient expression (Newman et al. 2000). However, pretreatment of pepper leaves with LPS affects the pattern of expression and accumulation of the above-mentioned genes following subsequent challenge with *X. campestris* pv. *campestris* (avirulent) and *X. campestris* pv. *vesicatoria* (virulent) (Newman et al. 2000).

Newman and associates (2002) also examined the effects of LPS pretreatment on the accumulation of SA and the synthesis of the phenolic conjugates coumaroyl tyramine (CT) and feruloyl tyramine (FT). The HR response in pepper is associated with increased levels of SA (Newman et al. 2001). CT and FT are suggested to have two possible roles in plant defense, as direct antimicrobial agents and in cell-wall reinforcement (Keller et al. 1996; Newman et al. 2001). LPS had apparently little effect on the timing of accumulation of SA, while the timing of accumulation of FT and CT was considerably altered. LPS pretreatment caused these two compounds to accumulate much more rapidly upon inoculation with *X. campestris* pv. *campestris*. Yet LPS alone did not induce CT and FT synthesis (Fig. 1D).

Little is known about the mechanisms of perception of LPS and LOS by plants and about the associated signal transduction pathways that trigger LPS-induced plant disease resistance.

Pretreatment of leaves of *Arabidopsis* with purified *X. campestris* pv. *campestris* LOS prevented the HR caused by subsequently inoculated avirulent strains of *Pseudomonas syringae* pv. *tomato* DC3000 carrying different avirulence genes. The one or more mechanisms by which LOS prevents the HR are as yet unknown, but this phenomenon is associated with an enhanced resistance of the plant tissue to bacteria, which is presumed to occur through LOS-dependent induction or priming for enhanced plant defense responses (Newman et al. 2002).

There is increasing evidence that the activation of the plant innate immunity response upon recognition of PAMP partly resembles the mechanisms of activation of the innate immunity response in mammalian and insect systems. However, it is not clear whether mechanistic parallels for LPS perception by plants, animals, and insects exist, as has been shown for flagellin perception (Zipfel et al. 2004). An understanding of the effects that LPS have on plants and how these are triggered has implications both for the control of bacterial diseases through general nonspecific mechanisms and for biocontrol of bacterial and fungal diseases by beneficial bacteria, which in some cases is believed to occur as a consequence of LPS recognition (Leeman et al. 1995). The challenge ahead is to identify the plant components involved in LPS recognition and subsequent signal transduction. The appreciation that structurally distinct components within LPS may trigger the same plant responses (Silipo et al. 2005) (possibly via different receptors) has substantial implications on the design of genetic or biochemical screens to identify such receptors.

Priming by alterations in primary metabolism.

Tubers of transgenic potato plants with decreased activity of the plastid ATP/ADP transporter *StAATP1* display reduced levels of starch, modified tuber morphology, and altered concentrations of primary metabolites (Tjaden et al. 1998). The spontaneous production of hydrogen peroxide, the endogenous content of SA, and the levels of transcripts of various defense-related genes are similar in tuber disks of wild-type and *StAATP1* antisense potato plants (Linke et al. 2002). However, upon challenging the tissue with various fungal elicitors or culture supernatants of the bacterial pathogen *Erwinia carotovora* subsp. *atroseptica*, the *StAATP1*-silenced tubers exhibited

highly potentiated activation of defense. This augmented defense included enhanced expression of defense-related genes and augmented incidence of early bursts of reactive oxygen species (ROS) (Linke et al. 2002). The potentiated activation of cellular defense responses in *StAATP1* plants was not preceded by an increase in endogenous SA levels but, nevertheless, coincided with a strongly enhanced resistance against *E. carotovora* (Linke et al. 2002) and *Alternaria solani* (Conrath et al. 2003) in the tubers or against *Phytophthora infestans* in the leaves (Conrath et al. 2003). Thus, inhibition of steps in primary metabolism can lead to the induction of a primed state that sensitizes the tissue for improved elicitation of various cellular defense responses.

Priming for enhanced resistance against abiotic stress.

Priming by BABA for enhanced responses to abiotic stress. A large number of genes are believed to be involved in plant responses to abiotic stress. By analyzing the expression pattern of genes induced by drought, cold, or high salinity, the existence of distinct mechanisms involved in the regulation of stress-responsive genes has been suggested (Bray 1997; Fowler and Thomashow 2002; Hasegawa et al. 2000; Ingram and Bartels 1996; Pastori and Foyer 2002; Seki et al. 2002a and b; Thomashow 1999). Some of the responses require ABA signaling, while others are ABA-independent (Bray 1997; Shinozaki and Yamaguchi-Shinozaki 1996, 1997, 2000; Thomashow 1999). Other molecules and hormones, such as calcium, JA, and SA have also been suggested to modulate the abiotic stress response of plants (Janda et al. 1999; Kang and Saltveit 2002; Knight et al. 1998).

BABA was found to increase drought and salt stress tolerance in *Arabidopsis* (Jakab et al. 2005). The protection correlated with an augmented expression of the SA-inducible *PR-1* and *PR-5* genes and the ABA-dependent *RAB-18* and *RD-29A* genes upon application of salt and drought stress (discussed above). Mutants impaired in ABA signaling could not be protected by BABA application, while SA-deficient plants showed a reaction that was similar to the one in wild-type plants. However, pretreatment with BABA did not induce ABA accumulation directly but accelerated ABA production following osmotic stress. The augmented ABA production resulted in augmented ABA-inducible gene expression and accelerated stomatal closure (Jakab et al. 2005). These findings demonstrate that BABA-induced tolerance to osmotic stress is based on priming for enhanced adaptation responses rather than on the direct activation of these responses, as is the case in acclimation treatment in which plants are gradually exposed to an increasing stress situation.

Priming in plant-insect interactions.

Plants release complex mixtures of chemical volatiles upon attack by herbivorous insects. These volatile organic compounds (VOC) are products of diverse metabolic pathways, including aromatic compounds, terpenoid compounds, and derivatives of the octadecanoid pathway that are often referred to as green leaf volatiles (GLV). VOC play an important role in attracting parasitic and predatory insects that are natural enemies of herbivores (Pare and Tumlinson 1999). In addition to this so-called indirect induced defense, there also is evidence that VOC can serve as airborne signals that induce resistance in neighboring, unharmed plants. Since the first evidence for VOC-induced resistance (Baldwin and Schultz 1983), a plethora of publications has supported this form of plant-plant communication (Baldwin et al. 2006; Dicke et al. 2003). In an attempt to identify key volatiles in VOC-induced resistance, experiments were done using purified synthetic volatiles. Apart from methyl jasmonate itself, particularly the C6 GLV were found

to trigger JA-dependent defense reactions (Bate and Rothstein 1998; Farag and Pare 2002; Ruther and Furstenuau 2005), although there are also studies reporting defense-eliciting activity by terpenoid compounds (Arimura et al. 2000, 2002). However, in all these studies, the VOC-induced defense activity remained rather moderate when compared with insect attack or elicitors from insect regurgitant. Furthermore, the concentrations of the applied volatiles were often unrealistically high and many of the purified volatiles were unable to induce the complete set of JA-responsive defense genes. Therefore, it remains doubtful whether direct defense induction is responsible for the phenomenon of VOC-induced resistance in lab experiments and field trials.

Recently, Engelberth and associates (2004) provided a more plausible mechanism of VOC-induced resistance. The authors discovered that the activity of three GLV, (Z)-3-hexenal, (Z)-3-hexen-1-ol, or (Z)-3-hexenyl acetate, is not so much reflected by their ability to activate defense directly but, rather, by a priming effect that leads to an augmented defense expression upon stress treatment. Maize plants pretreated with these C6 volatiles accumulated higher levels of endogenous JA upon elicitation and produced enhanced amounts of VOC upon treatment with caterpillar regurgitant, whereas other typical VOC from maize failed to trigger priming in maize. In support of this, Figure 1E shows that exposition to the total VOC blend from *Spodoptera littoralis*-infested corn does not directly induce a JA-inducible serine protease inhibitor (*serPIN*) gene but confers a faster and stronger transcriptional activation of this gene after subsequent defense elicitation by applying caterpillar regurgitant on wounded leaf areas (J. Ton and T. C. J. Turlings; *unpublished results*). Hence, VOC from insect-infested plants prime for JA-inducible defense mechanisms in corn. In contrast to these findings, Paschold and associates (2006) recently demonstrated that the VOC blend from *Manduca sexta*-infested native tobacco (*Nicotiana attenuate*) fails to prime neighboring tobacco plants. Surprisingly, this blend contained all compounds that had previously been shown to induce priming or resistance in other plant species (Paschold et al. 2006). Yet, another recently published study by Kessler and associates (2006) showed that VOC from clipped sagebrush (*Artemisia tridentate*) prime neighboring tobacco plants for augmented production of secondary metabolites and proteinase inhibitor activity. Together, the studies show that priming by VOC is a common defense strategy in plants, although the identity of VOC triggering the priming may differ among plant species.

The physiological and molecular mechanisms behind VOC-induced priming are largely unknown. Nevertheless, it seems evident that VOC-induced priming targets defense responses that are inducible by JA (Kessler et al. 2006; Paschold et al. 2006). Interestingly, VOC from *Spodoptera littoralis*-infested corn prime for enhanced expression of only a subset of JA-inducible defense genes (J. Ton and T. C. J. Turlings, *unpublished results*). This suggests that JA is not the only endogenous signal that controls the VOC-induced priming response. In this context, it will be of interest to determine mechanistic similarities between VOC-induced priming and PGPR-mediated ISR, which is also based on priming for JA-dependent defense responses (van Wees et al. 1999; Verhagen et al. 2004).

Priming in monocots.

In contrast to the large amount of information on inducible defense responses in dicotyledonous plants, the current knowledge of defense in monocots is limited. Both SAR (Kessmann 1994; Sticher 1997) and ISR (Krishnamurthy and Gnanamanickam 1997; 1998) have been described in monocots. Furthermore, master regulatory proteins such as NPR1 have been shown to function in monocots as well (Chern et al.

2005; Dong 2004), suggesting that dicots and monocots possess similar defense signaling pathways. In fact, several examples of priming for enhanced defense responses have been described for monocots. As mentioned above, maize plants exposed to GLV from neighboring plants are primed to produce enhanced levels of JA and VOC upon subsequent insect attack (Engelberth 2004). Furthermore, treatment with thiamine (vitamin B₁) primes for stronger accumulation of rice *PR-1a*, *PR-10*, and *PR-11* transcripts after challenge with either *Xanthomonas oryzae* pv. *oryzae* or *Magnaporthe oryzae* (Ahn et al. 2005). In barley, preinoculation of the roots with the PGPF *Piriformospora indica* resulted in enhanced disease resistance and increased tolerance to salt stress without obvious changes in defense-related gene expression (Waller et al. 2005). Overexpression of the *Arabidopsis NPR1* gene primes wheat plants for faster and stronger activation of the *PR-1* gene after treatment with BTH or infection by *Fusarium graminearum* and the potentiation of *PR-1* expression was associated with an induced resistance to *Fusarium* head blight disease (Makandar et al. 2006). Taken together, these reports indicate that the priming phenomenon is conserved in monocots.

Does priming modulate early signal transduction pathways with a role in plant-pathogen interactions?

Pathogens are often recognized by the perception of pathogen-derived molecules (race-specific elicitors or PAMP, depending on the pathosystem), often through sensors or receptors that are localized in the plasma membrane or the cytosol (Bourque et al. 1999; Nürnberger et al. 2004). The molecular interaction initiates a signal transduction cascade and results in activation of various defense responses. Our current knowledge of pathogen-induced signal transduction in plants mainly originates from either the characterization, identification, or both of regulatory components that link receptors to target genes. Almost immediately after pathogen recognition, ion fluxes (Ca²⁺, NO₃⁻, Cl⁻, K⁺) and changes in the electrical potential differences across the plasma membrane can be detected in plant cells (Pugin et al. 1997; Scheel 1998; Wendehenne et al. 2002). In recent years, some of the kinases that are induced in plants upon contact with pathogens or pathogen-derived signals have been identified as MAPK and calcium-dependent protein kinases (CDPK; Peck 2003). Furthermore, similar to phosphorylation and dephosphorylation, S-nitrosylation (the reaction of NO with protein thiols), is emerging as an important post-translational modification of proteins (Lindermayr et al. 2005).

Using different plant and elicitor (PAMP) model systems (e.g., parsley and pep13; tobacco and elicitors; *Arabidopsis* and flg22), complex networks of signal transduction have emerged (Garcia-Brugger et al. 2006; Gómez-Gómez and Boller 2002; Lebrun-Garcia et al. 1999; Nürnberger et al. 1994). While some pathogen-derived molecules activate a broad array of early responses in certain plant species, they cannot elicit a detectable reaction in others. For example, although elicitor-specific binding sites exist in both tobacco and *Arabidopsis* (Bourque et al. 1999), elicitors trigger ion fluxes, ROS production, protein kinase activation, and other responses in tobacco but not in *Arabidopsis*. However, to our knowledge, nobody has checked if elicitors could induce resistance in *Arabidopsis* via the mechanism of priming. Preliminary data suggest that one molecule can act as a bona fide (direct) elicitor in one species, and as a priming agent in others (B. Poinssot, unpublished results).

So, how can a primed plant be distinguished from a non-primed one? By now, the only way to detect the primed state is to perform post-challenge defense response assays or even tedious resistance bioassays. Therefore, it becomes increasingly

important to identify reliable molecular markers for the primed state. These could be identified by integrating the data obtained from the different priming models.

Putative mechanisms of priming.

The molecular mechanisms underlying priming are not understood. Hypothetically, the primed state could be based on the accumulation or post-translational modification of one or more signaling proteins that, after being expressed or modified, still remain inactive. Upon subsequent perception of stress, a second signaling event could “hyperactivate” the signaling protein, triggering a potentiated signal transduction. For example, priming might first induce the phosphorylation of a CDPK, leading to conformational change, and then, stress could cause alterations in free Ca²⁺ levels, thus triggering kinase activity and leading to faster downstream signaling and enhanced transcription of target defense genes. A crucial role for a kinase in priming has been demonstrated in the *ibs1* mutant affected in a cyclin-dependent kinase-like gene. This mutant has lost the capacity to acquire BABA-induced priming for SA-dependent defenses (Ton et al. 2005).

Another hypothesis suggests that priming enables accelerated defense-gene expression by inducing the accumulation of crucial transcription factors. After stress recognition, signal transduction in primed cells then could directly induce an appropriate set of defense genes, thereby avoiding a preliminary step of transcription factor expression.

Conclusions.

Priming for enhanced resistance to biotic and abiotic stress obviously is operating via various pathways involved in different metabolic processes. In addition, priming in plants shows phenotypic similarity to potentiation phenomena seen in the defense response of animals and humans. This suggests that the mode of action of priming and the resulting potentiation of cellular defense responses rather than the direct upregulation of defense signaling cascades might be of great advantage for living organisms. Priming fits well in the ecological context of induced resistance. Various studies have demonstrated that activation of inducible defenses involves major costs that affect plant growth and reproduction (Heil 2002). In the case of JA-inducible defenses, the costs are only affordable when the plant is actually exposed to attack by herbivores (Agrawal 1998; Baldwin 1998). A recently published study on the costs and benefits of priming in *Arabidopsis* demonstrated that the fitness costs of priming are substantially lower than those of the directly induced defense against pathogens (Van Hulten et al. 2006). In addition, it was shown that the benefits of priming outweigh its costs when disease occurs. Consequently, plants in the primed state are efficiently protected against stress without major trade-off effects on commercially and ecologically important traits such as growth and seed set. Hence, from the economic point of view, priming is the plant's solution to the trade-off dilemma between disease protection and costs involved in defense activation. While the lymphocyte-based immune system constitutes an important adaptive defense strategy in humans and animals, priming, as described in this review, also may well represent an important adaptive defense strategy in plants. Apparently, adaptive immunity and induced resistance use priming as a common mechanism by which complex organisms acquire sustainable protection against environmental stress.

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