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Priming: it's all the world to induced disease resistance

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Abstract After infection by a necrotising pathogen, colonisation of the roots with certain beneficial microbes, or after treatment with various chemicals, many plants establish a unique physiological situation that is called the 'primed' state of the plant. In the primed condition, plants are able to 'recall' the previous infection, root colonisation or chemical treatment. As a consequence, primed plants respond more rapidly and/or effectively when re-exposed to biotic or abiotic stress, a feature that is frequently associated with enhanced disease resistance. Though priming has been known as a component of induced resistance for a long time, most progress in the understanding of the phenomenon has been made over the past few years. Here we summarize the current knowledge of priming and its relevance for plant protection in the field.

Keywords Benzothiadiazole · 2,6-dichloroisonicotinic acid · Potentiation of defence responses · Salicylic acid · Sensitisation · Stress resistance

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Abbreviations

BABA	β-aminobutyric acid
IR	induced resistance
ISR	induced systemic resistance
MAMP	microbe-associated molecular pattern
SA	salicylic acid
SAR	systemic acquired resistance

Introduction

When a plant becomes infected by a necrotising pathogen it often develops an enhanced resistance to a broad and distinctive spectrum of pathogens in organs distant from the site of infection. This type of induced resistance (IR) is called systemic acquired resistance (SAR; for reviews see Conrath 2006; Durrant and Dong 2004). The identity of the long-distance signal (s) that moves from the primary infection site to the remote organs to boost the plant's disease resistance is still unknown (Conrath 2006; Durrant and Dong 2004). In the 1990s, however, studies with transgenic tobacco and *Arabidopsis* plants constitutively expressing a bacterial salicylic acid (SA) hydroxylase (Delaney et al. 1994; Gaffney et al. 1993), and more recent work with *Arabidopsis* mutants affected in either SA production or SA signalling (reviewed by Dong 2001), clearly demonstrated an essential role for SA in the establishment of SAR in remote tissue.

Colonisation of plant roots with selected strains of non-pathogenic growth-promoting rhizosphere bacteria can also provoke broad-spectrum disease resistance in plants. This type of IR is called induced systemic resistance (ISR; reviewed by Van Loon et al. 1998). In contrast to SAR, the ISR response does not require SA. It depends rather on responsiveness to jasmonic acid and ethylene (Pieterse et al. 1998). In addition to ISR, other interactions between plants and beneficial micro-organisms can also elicit systemic broad-spectrum disease resistance in plants. For example, during its symbiosis with barley roots, the endophytic basidio-mycete *Piriformospora indica* confers systemic resis-tance to fungal diseases and salt stress (Waller et al. 2005). Furthermore, enhanced pathogen resistance was reported from plants whose roots have been colonized by mycorrhizal symbionts (Pozo et al. 2005).

Induction of IR by chemicals

Many different organic and inorganic compounds have been shown to activate IR in plants (Kuć 2001). When SA was identified as an essential endogenous signal for the SAR response (see the above text), an intensive search was initiated in order to identify synthetic chemicals able to mimic SA in SAR induction. 2,6-dichloroisonicotinic acid and its methyl ester (both are named INA) were the first synthetic compounds reported to activate the *bona fide* SAR response in plants (Kessmann et al. 1994). Later, benzo(1,2,3) thiadiazole-7-carbothioic acid *S*-methyl ester (BTH) became an attractive synthetic SAR activator (Friedrich et al. 1996; Görlach et al. 1996; Lawton et al. 1996). SA, INA and BTH are assumed to activate SAR via the same signalling pathway (Ryals et al. 1996).

Priming is a part of IR in plants

In addition to the direct activation of some anti-microbial defence reactions, systemic resistance responses in plants are frequently associated with a primed state in which the plants are able to 'recall' previous infection, root colonisation or chemical treatment (Fig. 1). In consequence, primed plants respond more rapidly and/or effectively when re-exposed to biotic or abiotic stress (reviewed by Conrath et al. 2002, 2006). Although priming has been known to represent part of the defence arsenal of plants for decades (reviewed by Kuć 1987), the phenomenon did not attract much attention until the early 1990s. In a thorough analysis of the priming phenomenon, Conrath and associates employed a parsley cell culture and a microbe-associated molecular pattern (MAMP) from the cell wall of *Phytophthora sojae* to elucidate molecular aspects of priming and the associated amplification of MAMP-induced defence responses (for reviews see Conrath et al. 2002, 2006). By doing so, it was demonstrated that pre-treatment with SA, INA or BTH in a time-dependent manner primed the parsley cells for stronger activation by low MAMP doses of various cellular defence responses. These included the so-called early oxidative burst, rapidly induced ion transport changes at the plasma membrane, the synthesis and secretion of phytoalexins (coumarin derivatives), cell wall phenolics and a lignin-like polymer, as well as the accumulation of transcripts for various defence- associated genes (Katz et al. 1998; Kauss and Jeblick 1995; Kauss et al. 1992; 1993; Thulke and Conrath 1998). In ensuing inves-tigations with the parsley cells, it was demonstrated that the effect of SAR inducers on defence gene activation depended on both the gene whose expres-sion was being assayed and the dose of the inducer to be applied (Katz et al. 1998; Thulke and Conrath 1998). Whereas certain defence genes were found to be directly responsive to moderate concentrations of SA or BTH, other defence genes were not expressed. However, this second set of defence genes exhibited a very intense activation after pre-treatment with moder-ate concentrations of SA or BTH followed by treatment of the cells with a very low, sub-optimal MAMP dose (Katz et al. 1998; Thulke and Conrath 1998). These results documented a dual role for SAR inducers in the activation of plant defence responses: on the one hand, moderate doses of the SAR activators directly induced certain defence-associated genes, while on the other they primed cells for boosted expression of some other defence genes induced by a challenge treatment.

Mur et al. (1996) provided the first in-depth analysis of the priming phenomenon in intact plants. The authors reported that soil drench pre-application of SA to transgenic tobacco plants expressing chimeric *PR-1::GUS* or *PAL-3::GUS* defence genes did not cause significant gene activation. However, upon infection with *Pseudomonas syringae* (*Ps*) pv. *syringae* or after wounding, activation of the reporter genes was much stronger in the SA pre-treated plants

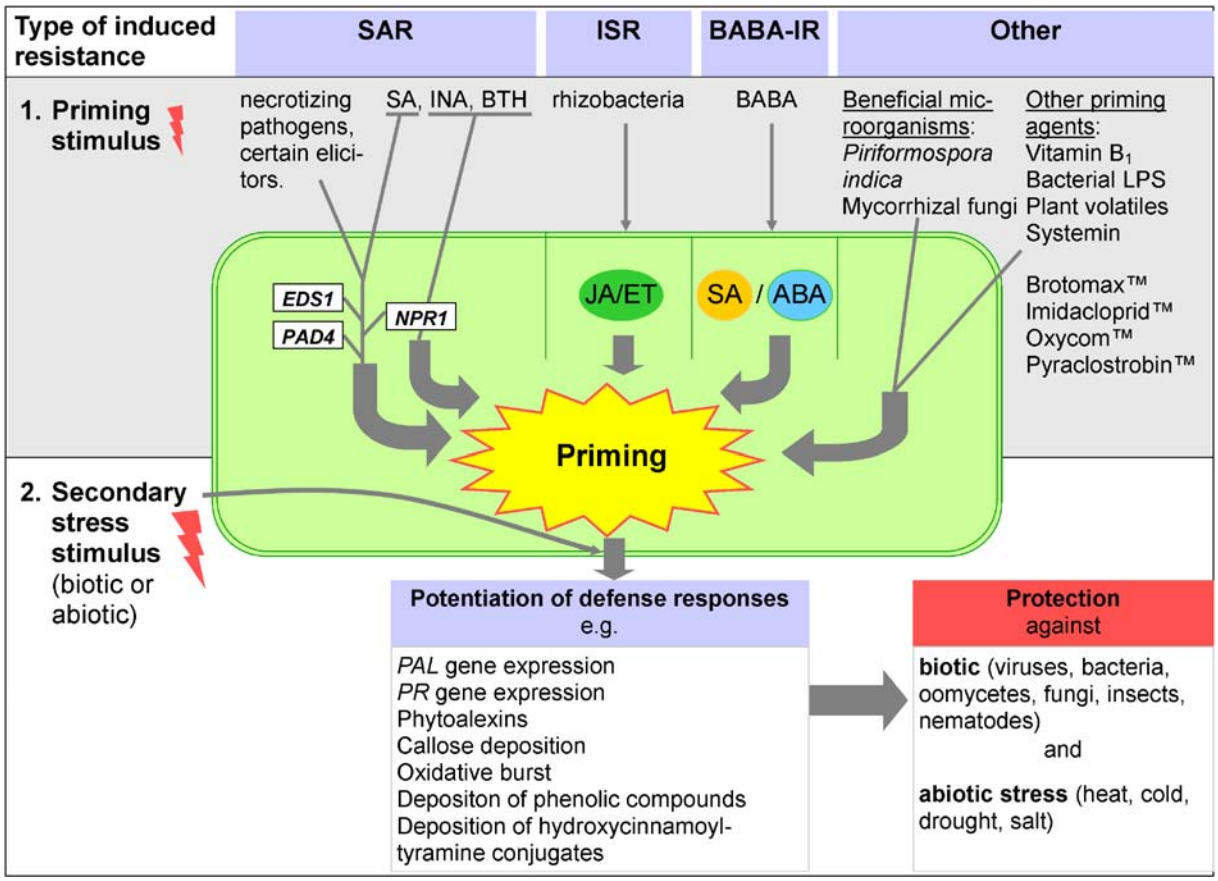

Fig. 1 Events associated with induced resistance phenomena in plants

than in the plants that had not been pre-treated with SA (Mur et al. 1996). Similarly, pre-treatment with BTH was shown to prime *Arabidopsis* for expression of *PAL* by *Ps* pv. *tomato* DC3000 that was much stronger than in a control plant not primed with BTH and then exposed to *Ps* pv. *tomato* DC3000 challenge (Kohler et al. 2002). BTH-induced priming also boosted *PAL* gene activation and callose deposition when these responses were induced either by mechanically wounding the leaves with forceps or by infiltration with water (Kohler et al. 2002). The observations made with *Arabidopsis* suggested that priming might be a common component that mediates cross-talk between pathogen defence reactions on one hand, and wound and osmotic stress responses on the other (Kohler et al. 2002).

The *Arabidopsis edr* (*enhanced disease resistance*) *1* mutant has constitutively enhanced resistance to the bacterium *Ps* pv. *tomato* DC3000 and to the fungus *Erisyphe cichoracearum*. Interestingly, *edr1* differs from other mutants with enhanced disease resistance in that it displays no constitutive activation of the defence genes *PR-1* and *BGL2*, though transcripts for both these genes accumulate after pathogen infection (Frye and Innes 1998). This fact, and the finding that *edr1* displays more intense induction of defence responses after infection than the wild type, strongly suggest an engagement of EDR1 in priming.

In response to infection with avirulent pathogens, the *Arabidopsis non-expresser of PR genes* (*npr*) *1* mutant (also named *nim1* or *sai1*) accumulates SA levels comparable to those of the wild-type. However, *npr1* is unable to express biologically or chemically induced SAR (Cao et al. 1994; Delaney et al. 1995). Intriguingly, the higher activation by BTH-mediated priming of *Ps* pv. *tomato*-induced, wound-elicited and water infiltration-activated defence response are absent in *npr1* (Kohler et al. 2002). This result indicates that a functional *NPR1* gene is required for priming. Conversely, the *constitutive expresser of PR genes*

(*cpr*) *1* and *cpr5* mutants of *Arabidopsis,* which both express SAR in the absence of pre-treatment with SAR activators (Bowling et al. 1994, 1997), are in a state of 'enhanced defence readiness' that resembles the primed state. In contrast to the wild-type, these plants are already sensitised in the absence of BTH pre-treatment, for higher *PAL* activation by *Ps* pv. *tomato* DC3000 infection, and for enhanced *PAL* gene expression and callose deposition upon wounding or infiltrating the leaves with water (Kohler et al. 2002). Thus, it is likely that, due to the enhanced SA levels in *cpr1* and *cpr5* (Bowling et al. 1994, 1997), these plants are permanently in a sensitised state. Because of the permanent presence of the primed condition, *cpr1* and *cpr5* might be able to induce various cellular defence reactions rapidly and strongly when attacked by pathogens, wounded or infiltrated on the leaves with water. In this context, it is noteworthy that the constitutively enhanced pathogen resistance of another *Arabidopsis* mutant referred to as *cpr5-2* has been ascribed to the boosted expression of the *PR-1* defence gene (Boch et al. 1998).

The close correlation between the SAR state and presence of the primed condition supported the assumption that priming is a key mechanism in the *bona fide* SAR response in plants. The assumption was further supported by the close correlation between the capability of various chemicals to activate SAR against *Tobacco mosaic virus* in tobacco (Conrath et al. 1995) and their capacity to prime for enhanced *PAL* expression induced by MAMP treatment in parsley cells (Katz et al. 1998; Thulke and Conrath 1998) or *Ps* pv. *tomato* DC3000 infection, wounding or water infiltration in intact *Arabidopsis* plants (Kohler et al. 2002). Moreover, an attenuation of priming and the associated loss of the enhanced activation of the oxidative burst have been associated with a lack of resistance to avirulent bacterial pathogens in tobacco (Mur et al. 2000). Finally, over-expression of the disease resistance-associated gene *PTI5* in tomato boosts pathogen-induced defence gene expression and enhances the resistance to *Ps* pv. *tomato* (He et al. 2001).

Observations similar to those made with parsley cell cultures, and tobacco, tomato, and *Arabidopsis* plants have been reported from SA-treated soybean cell cultures infected with *Ps* pv. *glycinea* (Shirasu et al. 1997), from BTH-primed and elicited *Agastache rugosa* suspension cells (Kim et al. 2001) and from SAR-induced and subsequently infected sunflower (Prats et al. 2002), cucumber (Cools and Ishii 2002), asparagus (He and Wolyn 2005; He et al. 2002), and cowpea plants (Latunde-Dada and Lucas 2001). Taken together, these studies suggested that priming is a major mechanism in the SAR response in various plant species.

Putative molecular mechanisms of priming

The molecular mechanisms behind priming are largely unclear. It has been proposed that sensitisation was associated with biosynthesis and pre-infectional accumulation or post-translational modification of cellular components with important roles in signal transduction and/or amplification. Accumulation or modification of the components per se would not activate the majority of the plant's defence responses. Yet, due to their enhanced level in primed cells, there is increased activation of the signal transmission components and thus, potentiated activation of downstream defence responses, but only after subsequent exposure to biotic or abiotic stress (Conrath et al. 2006). Recently, mitogen-activated protein kinases have been identified that accumulate after priming in *Arabidopsis* plants without displaying enzyme activity (G. J. M. Beckers, Y. Liu, S. Zhang and U. Conrath, unpubl.). Probably due to the enhanced level of the inactive mitogen-activated protein kinases in primed plants, there is increased MAP kinase activity after stimulation by biotic or abiotic stress associated with boosted induction of defence responses (G. J. M. Beckers, Y. Liu, S. Zhang and U. Conrath, unpubl.). Thus, certain mitogen-activated protein kinases are likely potential candidates for cellular components that mediate priming. Future genetic analysis will probably yield more molecular markers of priming for enhanced stress resistance.

Priming in IR to herbivores and between plant species in nature

In response to wounding or herbivore attack, plants often release extrafloral nectar or volatile organic compounds (VOCs). Whereas some of these traits serve to attract parasitic or predatory natural enemies of the herbivores (Paré and Tumlinson 1999), others have a role in enhancing the disease resistance in the wounded or herbivore-attacked plants themselves (Heil and Silva

Bueno 2007) or neighbouring, unharmed plants (Baldwin and Schultz 1983; Heil and Kost 2006). During past years, there is increasing evidence to suggest that the VOC-induced disease resistance is mediated by priming. In a pioneering paper, Engelberth et al. (2004) showed that maize seedlings, when exposed to certain volatiles from neighbouring plants and subsequently challenged by a combination of mechanical damage and exposure to caterpillar regurgitant, show a higher production of volatile sesquiterpenes and jasmonic acid than triggered plants not exposed to the volatiles previously. In a subsequent study, it was shown that VOC-induced priming for boosted activation of defence genes and potentiated emission of aromatic and terpenoid volatiles in maize correlates with reduced caterpillar feeding and improved attraction of the parasitoid *Cotesia marginiventris*, respectively (Ton et al. 2006). In field studies, Heil and Kost (2006) demonstrated that lima bean plants respond to leaf damage with the secretion of extrafloral nectar. This response was much higher in plants that had been previously primed by exposure to VOCs. Intriguingly, Kessler et al. (2006) also provided evidence that priming can even be the result of plant–plant communication in nature and that VOCs can even serve as priming-inducing signals between different plant species. The authors reported that VOCs from clipped sagebrush (*Artemesia tridentata*) prime nearby tobacco plants for accelerated production of trypsin proteinase inhibitors concomitant with lower total herbivore damage and a higher mortality rate of young *Manduca sexta* caterpillars (Kessler et al. 2006). Thus, plants can use chemical signals in their environment to assess the risk of herbivory and use this information to adjust their overall defence strategy.

Costs and benefits of priming

The activation of direct defence reactions by external application of high doses of SA, jasmonic acid or by the action of resistance (R)-genes was shown to reduce plant fitness traits such as growth and fruit or seed set under pathogen-free conditions (Agrawal et al. 1999; Baldwin 1998; Cipollini 2002; Heidel et al. 2004; Heil et al. 2000; Korves and Bergelson 2004; Tian et al. 2003; Van Dam and Baldwin 2001). Also, plants transformed with genes encoding SA biosynthesis enzymes (Mauch et al. 2001) or gain-of-resistance mutations in *Arabidopsis* such as *cpr1, cpr5* and *cpr6*, which all contain constitutively high levels of SA, permanent expression of defence-related *PR* genes and a dwarf phenotype, have been associated with reduced fitness (Bowling et al. 1994, 1997). These observations were also made in the field when Heidel et al. (2004) demonstrated that *Arabidopsis* mutants blocked in SA-inducible defence, as well as mutants showing constitutive expression of these defences, were affected in growth and seed set. The authors reasoned that optimal plant fitness is reached at a certain level of resistance that balances fitness and defence (Heidel et al. 2004). Similar conclusions were drawn from studies on the costs of jasmonic acid-inducible defences, which seem to be affordable only when the plant is actually exposed to herbivore attack (Agrawal et al. 1999; Baldwin 1998).

The trade-off dilemma between disease resistance and costs of defence activation can probably be overcome by priming. During a comparative study by Van Hulten et al. (2006), the costs and benefits of priming in *Arabidopsis* were determined and compared to those of the direct induction of defence: application of low doses of the non-protein amino acid β-aminobutyric acid (BABA) induced a primed state, caused only minor reductions in growth and had no obvious effect on seed production. In contrast, direct induction of defence responses by high doses of either BABA or BTH strongly reduced both these fitness traits. The effects in primed plants, though being minor when compared to those of the direct defence activation, could be caused by an enhanced expression of genes encoding signalling compounds (see the above text) (Maleck et al. 2000; Van Hulten et al. 2006). As a consequence, it has been suggested that priming has a smaller effect on fitness than directly induced defence (Van Hulten et al. 2006). Intriguingly, when under attack by pathogens, primed plants displayed even higher fitness than non-primed ones. Thus, in environments of pathogen challenge, the costs of fitness of the primed state seem to be outweighed by its benefits (Van Hulten et al. 2006).

Employing priming in the greenhouse and field

Many natural and synthetic compounds are able to prime plants (summarized by Conrath et al. 2006). They include Brotomax®, a commercial product derived from urea, copper lignosulphonate, manganese lignosulphonate, and zinc lignosulphonate (Fuster et al.

1995; Ortuño et al. 1997), bacterial lipopolysaccharides (Newmann et al. 2007), vitamin B_1 (Ahn et al. 2005, 2007), systemin (Stennis et al. 1998), and BABA (Zimmerli et al. 2000). In laboratory trials, BABA-induced priming for enhanced stress responses was associated with augmented resistance not only to biotic but also to abiotic challenges such as drought and salt stress (Jakab et al. 2005). Moreover, BABA and some other priming-inducing compounds were also shown to be potent inducers of stress tolerance in the greenhouse and field (Cohen 2002). Foliar application of BABA protected field-grown grape against *Plasmopara viticola*. BABA also suppressed disease symptoms caused by *Phytophthora infestans* on potato and tomato plants in the field. BABA also shielded melon from the induction of sudden wilt disease by *Monosporascus cannonballus* (Cohen 2002). In addition, BABA decreased disease symptoms induced by *Cercosporidium personatum* on peanut in both greenhouse and field trials (Cohen 2002). As the compound also exhibited synergistic interaction with certain fungicides (Cohen 2002), BABA may successfully be integrated into plant disease management in the field.

INA was the first synthetic compound shown to induce both priming for improved activation of defence responses (Kauss et al. 1992) and resistance to fungal and bacterial pathogens on various crops, in the greenhouse as well as in the field (Kessmann et al. 1994). However both INA and SA were insufficiently tolerated by some crops to allow practical use as plant protection compounds (Ryals et al. 1996). BTH (synonym: acibenzolar-S-methyl) is another synthetic chemical and was synthesized some years after INA. BTH primed plant cell cultures and intact plants for better induction of defence responses (Katz et al. 1998; Kohler et al. 2002) and also protected various crops against many diseases in the field (Ryals et al. 1996). As BTH was sufficiently tolerated by most crop plants, the compound became attractive for practical agronomic use. In 1996, BTH was introduced as a 'plant activator' (Ruess et al. 1996) with the trade names Bion®, Actigard® or Boost®. However, the economic success of BTH was limited. BTH exerts protective rather than curative activity. Thus, to serve as a protectant the compound must be applied some time before a potential pathogen attack. Because of this strictly prophylactic activity, BTH was not sufficiently accepted by farmers who favoured the application of curative standard fungicides.

Due to the general lack of consumer acceptance of BTH, it became opportune to identify plant-protecting compounds teaming both direct action on the pathogen and priming-inducing activity in the plant. Some strobilurin fungicides seem to combine both these activities (for a review on strobilurins, see Sauter 2007). In the laboratory, for example, the strobilurin fungicide Pyraclostrobin (trade names: Cabrio®, Headline®) primed the tobacco cv. Xanthi nc for more rapid accumulation of antimicrobial PR-1 defence proteins after infection with *Tobacco mosaic virus* and the wildfire pathogen *Ps* pv. *tabaci*. The Pyraclostrobin-induced priming for enhanced PR-1 accumulation in response to pathogen attack was associated with enhanced disease resistance (Herms et al. 2002). The enhanced resistance to pathogenic viruses and bacteria in Pyraclostrobin-treated plants was also seen on various crops and ornamental plants in both the greenhouse and field (Koehle et al. 2003, 2006). It is interesting that in the field, Pyraclostrobin-induced priming was associated with enhanced resistance also to abiotic stresses, including drought (www.agweb.com/aims/files/HeadlineAdvantage.pdf). In addition, treatment with Pyraclostrobin increased crop yield in the field (http://www.ag.iastate.edu/farms/05reports/n/SoybeanYield.pdf). Also, in various crops Pyraclostrobin and other strobilurin fungicides induce a 'greening effect.' The term refers to the phenomenon of delayed leaf senescence and an increased grain-filling period resulting in enhanced biomass and yield (Bartlett et al. 2002). Together, the findings made with Pyraclostrobin suggest that this chemistry, in addition to exerting direct antifungal activity, may also protect plants by priming them for a boosted activation of subsequently stress-induced defence responses. This conclusion is consistent with an earlier report demonstrating that another commercial fungicide, Oryzemate®, enhanced the resistance to *Tobacco mosaic virus* in tobacco (Koganezawa et al. 1998) and to a bacterial and an oomycete pathogen in *Arabidopsis* (Yoshioka et al. 2001). Oryzemate® contains Probenazole as the active ingredient which is metabolized to saccharin in treated plants (Koganezawa et al. 1998). The latter compound seems to elicit priming in Oryzemate®-treated plants (Siegrist et al. 1998).

Observations similar to those with Pyraclostrobin have recently been made in laboratory and field trials with the insecticide Imidacloprid (trade names: Admire®, Confidor®, Gaucho®, Merit®, Trimax™,

etc). One of its major degradation products, 6-chloronicotinic acid, has a structure very similar to INA and is suspected of causing the so-called 'stress shield effect' on crops by priming them for boosted expression of defence genes, enhancing their tolerance to biotic and abiotic stresses, and increasing plant growth and yield (Thielert 2006).

Various associations of plants with beneficial microbes in the soil have also been demonstrated to induce the primed state and to enhance the resistance of plants to above-ground pathogens and abiotic stresses in the greenhouse and field (Fig. 1). For example, growth-promoting *Pseudomonas fluorescens* not only induces priming in plants (Verhagen et al. 2004), but also suppresses Fusarium wilt disease and improves yield in radish in the greenhouse (Leeman et al. 1995). Furthermore, when growing various crops and ornamental plants after treatment of their seeds with a mixture of eight endo- and ectomycorrhizal fungi (available as MycoGrow™ Micronized Endo/Ecto Seed Mix), there is enhanced growth and augmented disease resistance in the greenhouse as well as under field conditions (www.fungi.com/index.html). Similarly, the root-endophyte *Piriformospora indica* systemically primes barley against biotic and abiotic challenges (Waller et al. 2005), and increases growth and yield of the medicinal plants *Spilanthes calva* and *Withania somnifera* in the field (Rai et al. 2001).

Conclusions

Over the past decade, it has become increasingly clear that priming is an important part of various induced stress resistance phenomena in plants (Fig. 1). In addition to being interesting for studying signal transduction and stress physiology, priming has the potential to emerge as a successful additional strategic tool for modern plant protection. Priming allows plants to activate defence responses more quickly and/or effectively when exposed to biotic or abiotic stress. Due to its advantageous economic features for the plant, priming also represents an ecologically important adaptation to withstand environmental challenges. The phenomenon can be interesting for the development of new concepts for disease control, since priming provides broad-spectrum disease resistance without significantly affecting growth and fruit or seed set. Together, priming offers a smart, effective and realistic option for effective plant protection, especially when combined with the performance of traditional pesticides. The utilization of the natural, broad-spectrum defence capacity of plants in the field will be facilitated by a better understanding of the molecular, physiological and ecological aspects of priming, which constitute an exciting challenge for future research.

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