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REVIEW

Practical application of induced resistance to plant diseases: an appraisal of effectiveness under field conditions

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SUMMARY

Plants resist pathogen attack through a combination of constitutive and inducible defences. Different types of induced resistance have been defined based on differences in signalling pathways and spectra of effectiveness. Systemic acquired resistance (SAR) occurs in distal plant parts following localized infection by a necrotizing pathogen. It is controlled by a signalling pathway that depends upon the accumulation of salicylic acid (SA) and the regulatory protein NPR1. In contrast, induced systemic resistance (ISR) is promoted by selected strains of non-pathogenic plant growth-promoting rhizobacteria (PGPR). ISR functions independently of SA, but requires NPR1 and is regulated by jasmonic acid (JA) and ethylene (ET).

Resistance can be induced by treatment with a variety of biotic and abiotic inducers. The resistance induced is broad spectrum and can be long-lasting, but is rarely complete, with most inducing agents providing between 0·20 and 0·85 disease control. In the field, expression of induced resistance is likely to be influenced by the environment, genotype, crop nutrition and the extent to which plants are already induced. Unfortunately, understanding of the impact of these influences on the expression of induced resistance is rudimentary. So too is understanding of how best to use induced resistance in practical crop protection. This situation will need to change if induced resistance is to fulfil its potential in crop protection.

INTRODUCTION

Induced resistance

Plants protect themselves from attack by pathogens using a complex array of mechanisms involving recognition, attack and defence. In the early stages of the interaction between the plant and the pathogen, elicitor molecules are released. These elicitors can be of plant or pathogen origin and include carbohydrate polymers, peptides, lipids and glycopeptides (Walters *et al*. 2005). Plant perception of these elicitor molecules leads to activation of a signalling pathway and ultimately to the production of plant defences. These defences include production of reactive oxygen species (ROS), phytoalexin biosynthesis, accumulation of pathogenesis-related (PR) proteins and cell wall reinforcement (Hammerschmidt 1999). In race-specific resistance to a pathogen, the major gene controlling this resistance (*R* gene) codes for a product that recognizes the product of a matching avirulence (*Avr*) gene in the pathogen. In this situation, the plant quickly recognizes the pathogen and there is rapid activation of defences, e.g. a hypersensitive response (HR). In contrast, if the pathogen does not possess an *Avr* gene that is recognized by the host plant, HR is not activated and the pathogen is kept in check by a range of non-specific defences. This is known as polygenic or basal resistance.

It is well established that, following infection by a microbial pathogen, susceptible plants can develop an enhanced resistance to further infection (Kuć 1982; Hammerschmidt 2007). This is known as induced resistance and can be split broadly into two

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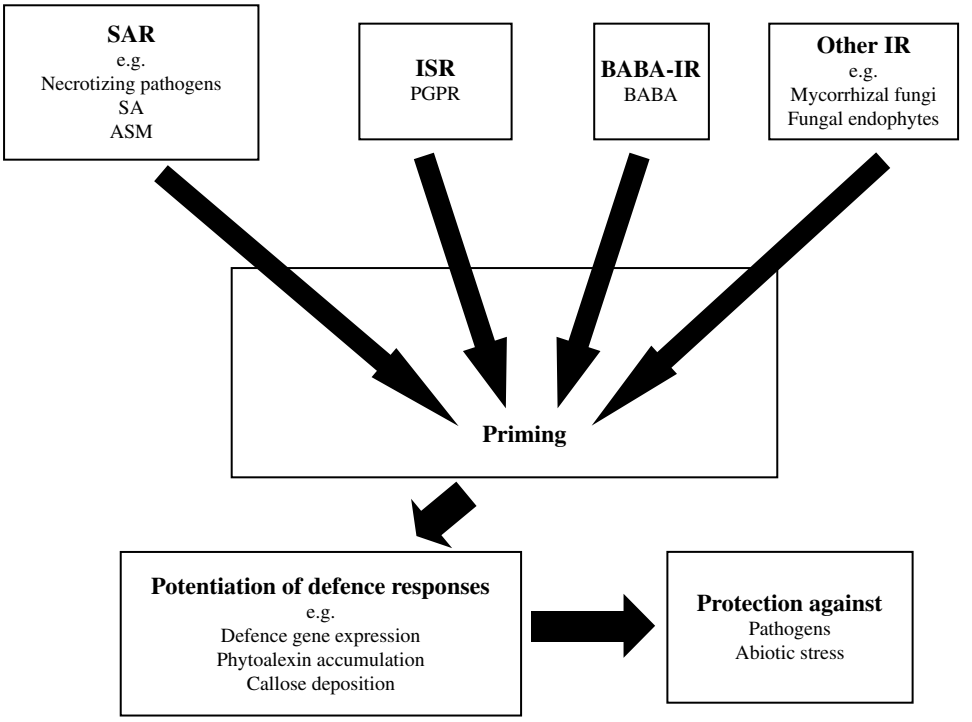

Fig. 1. Events associated with induced resistance phenomena in plants (adapted from Goellner & Conrath (2008), with kind permission from Springer Science + Business Media).

types: systemic acquired resistance (SAR) and induced systemic resistance (ISR).

SAR

SAR describes the phenomenon whereby plants develop a broad-spectrum systemic resistance to pathogen infection following a localized infection by a necrotizing pathogen or treatment with various agents e.g. acibenzolar-*S*-methyl (ASM) or Probenazole (Oryzemate®). SAR is associated with increased levels of salicylic acid (SA) both locally and systemically and with the coordinate expression of a specific set of genes encoding PR proteins (Pieterse & Van Loon 2007; Fig. 1). Moreover, application of SA or one of its functional analogues, such as ASM, induces SAR and activates the same set of *PR* genes (Ryals *et al.* 1996). Transgenic plants that are unable to accumulate SA, and mutants compromised in pathogen-induced SA accumulation, cannot develop SAR (Gaffney *et al.* 1993; Lawton *et al.* 1995). Triggering of *PR* gene expression and development of SAR requires transduction of the SA signal and this, in turn, is dependent on the regulatory protein NPR1 (Shah *et al.* 1997; Fig. 1). Expression of a set of *PR* genes, and *PR-1* in particular, is used as a marker for SAR induction. It is important to note, however, that the function of some *PR* genes in SAR is unclear and the induction of resistance is not always accompanied by *PR-1* expression (Durrant & Dong 2004; Sparla *et al.* 2004).

ISR

ISR develops as a result of colonization of plant roots by certain strains of plant growth-promoting rhizobacteria (PGPR) and is mediated by a jasmonic acid (JA)- and ethylene (ET)-sensitive pathway (Pieterse & Van Loon 2007; Fig. 1). Phenotypically, ISR is similar to SAR in that it acts unspecifically against taxonomically different pathogens (Zehnder *et al.* 2001; Pieterse & Van Loon 2007). Further, like SAR, ISR also requires a functioning NPR1 and can be associated with an accumulation of PR-proteins and phytoalexins, and alterations in cell wall composition (Ramamoorthy *et al.* 2001).

Priming

The systemic resistance responses outlined above can be associated with direct activation of defences. However, such responses can also be associated with an ability to 'recall' previous infection, root colonization or chemical treatment. This phenomenon is known as

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priming and results in plants responding more rapidly and effectively when exposed to subsequent pathogen attack (Goellner & Conrath 2008; Fig. 1). Usually, no changes in gene expression or in the levels of resistance traits are detectable in response to the priming agent alone, which might be a chemical elicitor such as ASM or a challenging pathogen. Interestingly, priming of resistance is usually caused by agents that fully induce resistance when applied at higher doses (Kohler *et al*. 2002; van Hulten *et al*. 2006) and suggests that direct resistance induction and priming might differ from one another quantitatively rather than qualitatively. However, other work has shown that different genes are involved in both phenomena. Thus, in parsley cell cultures, while some genes responded directly to low concentrations of SA, ASM or Probenazole, a different set of genes was only slightly responsive to these inducers, but following pre-treatment with them, responded much more strongly to a pathogen-derived elicitor (Katz *et al*. 1998).

Other forms of induced resistance

A wide range of microbes and chemicals are known to induce resistance. Although in many cases, the signalling and defence mechanisms involved are not yet known, it seems likely that other forms of induced resistance exist. For example, it is well known that the non-protein amino acid, β-aminobutyric acid (BABA), can induce resistance in a variety of crop plants (Jakab *et al*. 2001). BABA-induced resistance (BABA-IR) has been used as a model for the study of priming and in *Arabidopsis* it is based on various mechanisms. Thus, BABA-IR against *Pseudomonas syringae* and *Botrytis cinerea* functions via priming for SA-inducible defences, while against a different set of pathogens (*Hyaloperonospora parasitica*, *Plectosphaerella cucumerina* and *Alternaria brassicicola*), it is based on priming for resistance through the formation of callose-rich papillae (Zimmerli *et al*. 2000, 2001; Ton & Mauch-Mani 2004). Interestingly, BABA-induced priming of callose deposition involves proteins that play a role in biosynthesis or perception of abscisic acid (ABA), and disruption of the ABA signalling pathway results in loss of BABA-induced priming for formation of callose-rich papillae (Ton & Mauch-Mani 2004; Ton *et al*. 2005).

INDUCED RESISTANCE UNDER FIELD CONDITIONS

A large number of biotic and abiotic agents are now known to induce resistance to pathogen infection in plants (da Rocha & Hammerschmidt 2005; Lyon 2007). Since the introduction of the first chemical resistance activator, Probenazole (registered in Japan as Oryzemate®, Meiji Seika Kaisha Ltd), in 1975, many other chemical and microbial activators have been developed. These include: ASM, registered as Bion® and Actigard®, Syngenta, Milsana® (*Reynoutria sachalinensis* extract, KHH BioScience Inc., USA), Elexa® (chitosan, SafeScience, USA), and Messenger® (harpin protein, Eden Bioscience, USA).

Although high levels of disease control can be achieved with plant activators in controlled environments, their performance under field conditions has been less impressive. Indeed, the moderate levels of disease control and high levels of variability exhibited by plant activators in the field have been instrumental in the very slow uptake of induced resistance in crop production systems. In the next section, the performance of plant activators under field conditions are examined, followed by the possible reasons for the often lacklustre performance of induced resistance.

Probenazole

Probenazole (3-allyloxy-1,2-benziothiazole-1,1-oxide) was developed by Meiji Seika Kaisha Ltd. This resistance inducer was first introduced in 1975 for the control of rice blast disease (*Pyricularia oryzae*) and bacterial blight (*Xanthomonas oryzae*). Probenazole is widely used in Asia, where it is applied as a granular treatment either to paddy fields or as a seedling box treatment. After its application, the compound is absorbed by the roots, then systemically transferred to the rest of the plant and can control rice blast disease for 40–70 days post application (Iwata *et al*. 2001). However, despite continuous use since its introduction, there have been no reports of pathogen insensitivity to probenazole and indeed, it still accounted for 0·53 of the chemicals used for seedling box treatments on rice in 2005 (Ishii 2008). It is believed this is because the compound is only weakly toxic to fungi and activates disease defence systems in rice (Watanabe 1977; Watanabe *et al*. 1977; Iwata *et al*. 1980). These are thought to be the result of the activation of a signal transduction pathway, which in turn alters the balance of the plant–pathogen relationship in favour of the plant.

ASM

Since the introduction of ASM more than 10 years ago, a sizeable body of data has accumulated on its efficacy against a range of diseases under field conditions (Vallad & Goodman 2004; Walters *et al*. 2005). Most studies report disease control, although the level of control ranges from 0·04 to 0·99. Particularly high levels of disease control were achieved on tobacco, where infection by *P*. syringae pv. *tabaci*, *Cercospora nicotianae* and *Alternaria alternata* was reduced by 99, 91 and 89 %, respectively (Cole 1999; Perez *et al*. 2003). In wheat, the crop that ASM was originally aimed at, disease control was not so impressive,

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ranging from 0·35 for *Puccinia recondita* and *Septoria* spp. to 0·77 for *Blumeria graminis* f. sp. *hordei* (Gorlach *et al*. 1996; Stadnik & Buchenauer 1999). ASM even increased disease levels in peanut, where infection by *Cercosporidium personatum* was greater than untreated controls by 52% (Zhang *et al*. 2001). Working on oilseed rape, Liu *et al*. (2006) found that pre-treatment with ASM in October/November decreased the number of leaf lesions caused by the Phoma stem canker pathogen *Leptosphaeria maculans* in the autumn/winter, as well as the severity of stem canker in the subsequent spring/summer. Liu *et al*. (2006) found that reductions in numbers of leaves with lesions were between 25 and 55%. In some more recent work, ASM was shown to reduce infection of barley by the leaf scald pathogen *Rhynchosporium secalis* by 45% (Paterson *et al*. 2008).

Chitosan/Elexa®

Chitosan is a de-acetylated form of *N*-acetylchitooligosaccharide containing poly-D-glucosamine and is a common polymer in shells of crustaceans, exoskeletons of insects and cell walls of fungi (Hadwiger 1999). A commercial formulation of chitosan, Elexa®, contains 0·04 chitosan as its active ingredient and has been shown to protect a range of crops against pathogens. For example, when used as a seed treatment, it reduced downy mildew severity on pearl millet by 58% and when used as a foliar spray, it reduced infection by 75% (Sharathchandra *et al*. 2004). When used on grapevines, eight applications of Elexa® applied over the season, reduced the incidence of downy mildew by 50% and powdery mildew by 75% compared with untreated controls (Schilder *et al*. 2002).

Harpin/Messenger®

Several commercially available products are based on microbial proteins. One of these is Messenger®, which is based on the protein harpin obtained from *Erwinia amylovora* (Wei *et al*. 1992). Used as a crop protectant, Messenger® has had mixed success. For example, although it possessed good efficacy against blue mould in apples (de Capdeville *et al*. 2003), its efficacy against grey mould in strawberry (Meszka & Bielenin 2004) and target spot of tomato (Pernezny *et al*. 2002) was poor. In some interesting recent work, Chen *et al*. (2008*a*) generated specific fragments of $HpaG_{Xooc}$, a harpin from *X. oryzae* pv. *oryzicola*, and found that one of these fragments, $HpaG_{10–42}$, stimulated growth of rice plants and provided enhanced resistance to *X. oryzae* pv. *oryzae* and *Magnaporthe grisea*. $HpaG_{10–42}$ was also shown to control bacterial blight, rice blast and sheath blight, and to increase grain yields, under field conditions (Chen *et al*. 2008*b*). Here the level of disease control depended on the cultivar, with greater control obtained with *indica* compared with *japonica* cultivars.

Milsana®

Milsana® is an ethanolic extract of giant knotweed (*R. sachalinensis*) and is registered as a plant activator for use on glasshouse-grown ornamental plants in the USA. It has been shown to control fungal pathogens on various crops, including strawberry (Carlen *et al*. 2004) and cucumber (Daayf *et al*. 1995; Fofana *et al*. 2002). Used on grapes, Milsana® applied every 7–10 days provided similar levels of control of powdery mildew and bunch rot on grape berries to those obtained using a commercial fungicide (Schmitt *et al*. 2002). More recently, Milsana® was found to reduce powdery mildew (*Leveillula taurica*) infection of tomato by 42–64%, with efficacy depending on application rate and disease pressure (Konstantinidou-Doltsinis *et al*. 2006). Milsana® also controlled powdery mildew (*Uncinula necator*) on grape under field conditions (Konstantinidou-Doltsinis *et al*. 2007).

BABA

The non-protein amino acid BABA has been shown to induce resistance in a range of crops (Jakab *et al*. 2001). In field trials with grapevines, BABA reduced infection by the downy mildew fungus *Plasmopara viticola* by 57% on cv. Chardonnay and by 98% on cv. Cabernet Sauvignon (Reuveni *et al*. 2001). BABA has also been shown to provide protection against the late blight pathogen *Phytophthora infestans* on potato. For example, BABA-protected potato plants against *P. infestans*, especially when applied early in crop development and also provided some protection in tubers against late blight (Andreu *et al*. 2006). Similar results were obtained by Altamiranda *et al*. (2008), who obtained 0·20–0·60 protection of potato plants against late blight, with efficacy dependent upon cultivar and time of application.

ISR

PGPR-mediated ISR was first shown to be effective under field conditions in the mid-1990s. Thus, application of PGPR as a seed treatment followed by soil drench application led to a reduction in severity of bacterial wilt (Wei *et al*. 1995), and control of bacterial angular leaf spot and anthracnose (Wei *et al*. 1996). Later work by Raupach & Kloepper (2000) demonstrated that treatment of cucumber seed with PGPR led to increased plant growth and control of angular leaf spot and anthracnose. Field experiments in Thailand in 2001 and 2002 studied the effects of PGPR, used alone or as mixtures, on control of southern blight of tomato caused by *Sclerotium*

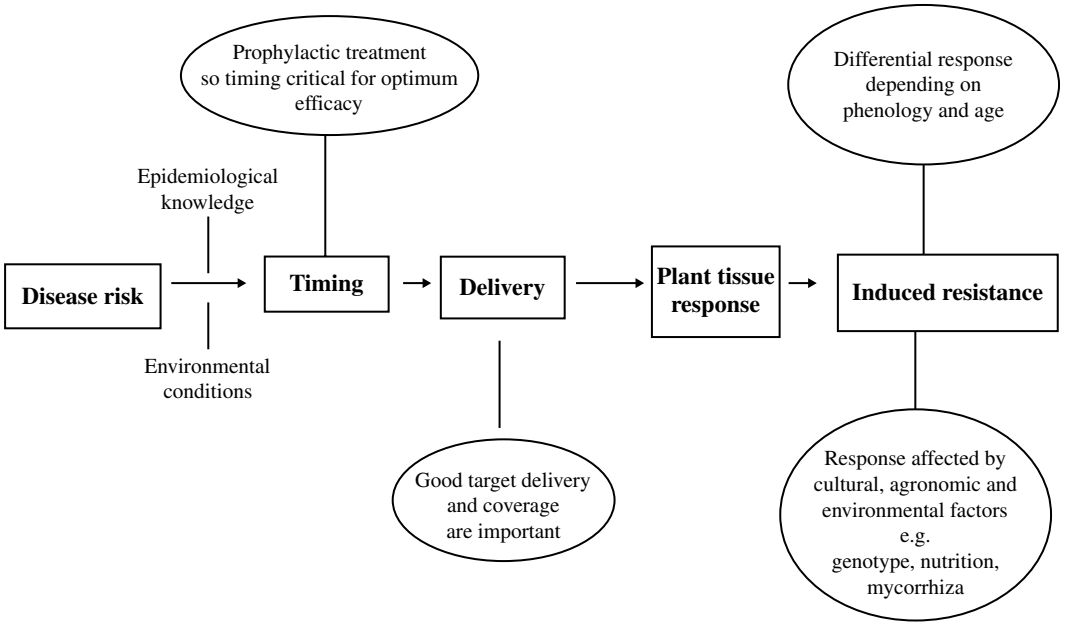

Fig. 2. Factors influencing the efficacy of induced resistance (adapted from Reglinski *et al*. (2007), with kind permission from Wiley-Blackwell).

rolfsii, anthracnose of long cayenne pepper caused by *Colletotrichum gloeosporoides* and mosaic disease of cucumber caused by cucumber mosaic virus (CMV) (Jetiyanon *et al*. 2003). Mixtures of PGPR (all *Bacillus* spp.) were found to suppress disease more consistently than the PGPR strain *Bacillus pumilus* IN937b, used alone. In more recent work, Harish *et al*. (2008) found that certain strains of PGPR and bacterial endophytes induced ISR in banana against banana bunchy top virus, while the PGPR strain *Pseudomonas aeruginosa* LY-11, applied as an alginate seed coating, reduced infection of lettuce by *Rhizoctonia solani* by 70–85% (Heo *et al*. 2008).

EFFECTIVENESS OF INDUCED RESISTANCE UNDER FIELD CONDITIONS: WHAT AFFECTS ITS EXPRESSION?

A survey of the literature on the use of induced resistance under field conditions reveals a lack of consistency and an efficacy that is usually less than that achieved with fungicides. In some cases, induced resistance has failed to provide any disease control. For example, field trials on barley failed to show any effect of ASM against barley yellow dwarf virus (Huth & Balke 2002), while ASM and Messenger® failed to provide significant control of *Xanthomonas axonopodis* pvs *citrumelo* and *citris* on sweet oranges (Graham & Leite 2004). ASM was even found to increase infection of peanut by the late leaf spot pathogen, *C. personatum* (Zhang *et al*. 2001), and probenazole, although highly effective when used for disease control in rice, is not effective in any other plant, to our knowledge (Siegrist *et al*. 1998). This lack of consistency and incomplete disease control should not be a surprise, since induced resistance is a host response and as such, will be affected by many factors, including the abiotic environment, host genotype and the extent to which plants in the field are already induced (Fig. 2).

Host genotype and the efficacy of induced resistance

It has been known for some time that the expression of induced resistance can be influenced by host genotype (Fig. 2). Twenty years ago, Steiner *et al*. (1988) found that control of powdery mildew on barley using a *Bacillus subtilis* culture filtrate was dependent on the cultivar used, while later work by Martenelli *et al*. (1993) showed that induction of resistance in barley by prior inoculation with an avirulent isolate of *B. graminis* f. sp. *hordei*, differed in lines carrying different race-specific resistance genes. Clearly therefore, expression of induced resistance is cultivar-dependent, although the influence of the resistance rating of the cultivar on induced resistance is less clear. For example, resistance in cucumber to the powdery mildew pathogen, *Sphaerotheca fuliginea*, induced by treatment with 2,6-dichloroisonicotinic acid (INA), was cultivar-dependent, with highest

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levels of resistance expressed in a partially resistant cultivar and lower levels of resistance expressed in susceptible cultivars (Hijwegen & Verhaar 1994). In contrast, resistance in soybean to *Sclerotinia sclerotiorum*, induced with INA or ASM, was greatest in susceptible cultivars (Dann *et al*. 1998). In some interesting work, Resende *et al*. (2002) found that ASM provided 55 and 85% control of *Verticillium dahliae* and *Crinipellis perniciosa*, respectively, on cocoa seedlings, although the defences activated differed depending on the host cultivar used.

PGPR-mediated ISR has also been shown to be influenced by genotypic effects. The PGPR strain *Pseudomonas fluorescens* WCS417r elicited ISR in all ecotypes of *Arabidopsis thaliana* examined, apart from the ecotypes RLD and Wassilewskija (Van Wees *et al*. 1997; Ton *et al*. 1999). Further work revealed the presence of a locus (*ISR1*) involved in the ET signalling pathway, and it appeared therefore that the ecotypes RLD and Wassilewskija carried a recessive trait that affected ISR by perturbing ET signalling, although the plants could still express SAR (Ton *et al*. 2001). This is clear evidence that in ecotypes of *A. thaliana*, allelic variability exists in genes that exert an influence on ISR pathways. Unfortunately, it is not known whether allelic variability exists for regulatory genes of SAR.

Costs and trade-offs associated with induced resistance

Plants are a source of food for a great many pathogens and herbivores. Survival in an environment teeming with consumers requires good defences. Plants possess a remarkable array of defence mechanisms to protect themselves from pathogens and pests and it would seem sensible for such defences to be continually present, i.e. to be constitutively expressed. However, plant defence is a costly business, requiring energy and resources that would otherwise be used for growth and development. In this context, constitutive resistance would appear to be a costly option. Indeed, it is argued that induced resistance, where defences are only activated following pathogen attack, represents a selective advantage over constitutive resistance (Walters & Heil 2007). One explanation for this selective advantage lies with fitness costs, where resistant plants would have decreased reproductive success than non-resistant plants under pathogen free conditions (Heil & Baldwin 2002). These costs include: (i) allocation costs arising from the diversion of metabolites and energy away from fitness-relevant processes such as growth and reproduction towards defence, (ii) ecological costs that result when the expression of a defence trait negatively interacts with one of the other ecological interactions that the plant has with the environment, e.g. mycorrhizal associations and (iii) genetic or pleiotropic costs that arise when resistance genes negatively affect fitness-relevant traits.

The study of costs of induced resistance to insect herbivory is well established and there is now much evidence to support the existence of such costs (e.g. Zavala *et al*. 2004). In contrast, costs and trade-offs in relation to induced resistance to pathogens have received considerably less attention. In the early 1980s, it was shown that the expression of resistance in barley to powdery mildew was associated with a 7% reduction in grain yield and a 4% reduction in grain size and protein content (Smedegaard-Petersen & Stolen 1981). This pioneering work received little attention at the time and in contrast, later work found either no effects of induced resistance on yield or increased yield associated with induced resistance in barley (Oerke *et al*. 1989; Reglinski *et al*. 1994). Subsequent studies examined the effects of chemically induced resistance on costs in the absence of pathogen pressure. For example, Heil *et al*. (2000) applied ASM to wheat in the absence of pathogens and found that treated plants had reduced biomass and reduced numbers of ears and grains, with most marked effects under nitrogen-limiting conditions. Work on other systems produced similar results. Thus, ASM reduced shoot fresh weight in sunflower (Prats *et al*. 2002), suppressed growth of tobacco and cauliflower (Csinos *et al*. 2001; Ziadi *et al*. 2001) and reduced shoot growth and leaf enlargement in cowpea (Latunde-Dada & Lucas 2001).

These data suggest that use of ASM incurs allocations costs and supports the 'growth-differentiation balance' hypothesis, which assumes a metabolic competition between processes involved in plant growth and those necessary for plant differentiation, such as the synthesis of chemicals for plant defence (Herms & Mattson 1992). Interestingly however, not all work on ASM yielded the same results. Thus, in the work on bean treated with ASM, no evidence could be found for the existence of allocation costs (Iriti & Faoro 2003). That work, together with that of Oerke *et al*. (1989) and Reglinski *et al*. (1994), showing either no effect on yield or even yield increases in induced plants suggests either that the plants possessed sufficient resources to finance both growth and defence, or they compensated for the resources diverted from growth to defence. Compensation might take the form of increased photosynthetic rates and, interestingly, Murray & Walters (1992), working on broad bean, found that in plants induced by prior inoculation with rust, systemically protected leaves also exhibited increased rates of photosynthesis. Unfortunately, little information exists on rates of photosynthesis in plants expressing SAR or ISR.

From the above discussion, it seems reasonable to assume that plants growing under resource-limiting conditions will experience greater costs associated with resistance expression or that their ability to

express resistance should be compromised. Indeed, competition has been shown to reduce peroxidize activity in SA-treated *Arabidopsis* plants and reduced seed set compared with non-competing or untreated controls (Cipollini 2002), a dependency of activities of two defence-related enzymes on CO_2 concentration was observed by Plessl *et al.* (2005) and mycorrhizal colonization and establishment might be necessary for successful resistance induction by ASM (Sonnemann *et al.* 2005). Further, whether costs were incurred as a result of resistance induction in wheat and *Arabidopsis* was dependent on nitrogen supply (Heil *et al.* 2000; Dietrich *et al.* 2005).

In the studies on costs of induced resistance described above, it is likely that defences were directly activated by the inducing agent. Direct induction of defences is likely to be wasteful in the absence of disease, in contrast to priming, where defences are activated upon pathogen challenge. Work carried out by van Hulten *et al.* (2006) found that priming involved fewer costs than direct induction of defences and, indeed, was beneficial in terms of the plant growth rate and fitness under disease pressure. Priming appears therefore to have clear ecological benefits and would also represent a promising approach for crop protection.

Induced resistance can also lead to trade-offs with other defence responses, e.g. with defence against insect pests. JA is known to be important in regulating induced resistance to insect attack (Bostock 2005) and there are several reports of negative crosstalk between the JA pathway for defence against insects and the SA pathway for defence against pathogens. For example, activation of SA-dependent SAR has been shown to suppress JA signalling, thereby compromising induced defence responses to insect attack (Bostock 2005; Stout *et al.* 1999; Thaler *et al.* 1999, 2002). Although there are fewer reports of negative crosstalk in the opposite direction, JA has been shown to suppress SA-induced responses (Niki *et al.* 1998; Glazebrook *et al.* 2003). It should be noted, however, that not all interactions between pathogen and insect resistance are negative, with some workers finding no effect (Ajlan & Potter 1992; Inbar *et al.* 1998) and others reporting a positive effect (Stout *et al.* 1999; Hatcher & Paul 2000; Walters *et al.* 2006). These examples of negative and positive crosstalk between the JA and SA signalling pathways highlight the complexity of signalling in pest and disease resistance. Interestingly, it seems that although JA, SA and ET play a primary role in orchestrating plant defence, the final defence response is shaped by other regulatory mechanisms, such as crosstalk between different signalling pathways and other attacker-induced signals (De Vos *et al.* 2005).

Since induced resistance is a broad-spectrum resistance against micro-organisms, it is not unreasonable to suggest that it might interfere with plant-microbe mutualisms. Unfortunately, however, little work has been carried out in this area. Nevertheless, several studies have shown that application of SA to the rooting substrate had a negative effect on nodule formation and/or function (Martínez-Abarca *et al.* 1998; Ramanujam *et al.* 1998; Lian *et al.* 2000), while in ASM-induced SAR in broad bean, treated plants were found to develop fewer and smaller nodules than untreated controls (Heil 2001). Even less effort has been spent on the impact of induced resistance on mycorrhizal infection, colonization and establishment, although a field study by Sonnemann *et al.* (2002) found no effect of ASM on mycorrhizal infection of barley roots.

Influence of the abiotic environment on induced resistance

As indicated in the previous section, allocation costs can be incurred if plants expressing induced resistance are grown under resource-limiting conditions. Indeed, nitrogen supply was shown to exert a marked effect on the expression of both constitutive and induced resistance (Dietrich *et al.* 2004). Here, constitutive levels of defence-related enzymes, as well as levels in plants induced by ASM treatment, were significantly lower under limiting nitrogen supply. Dietrich *et al.* (2004) also found significantly reduced levels of total soluble protein during the first 12 h following ASM treatment. This observation agrees with the other work demonstrating reductions in expression of genes relating to primary metabolism following elicitation of resistance (Somssich & Hahlbrock 1998) and suggests that such a down-regulation of primary metabolism might be necessary in order to make available the substrates and metabolites required for the synthesis of defence compounds (Heil 2002). This concept is supported by work showing that plants treated with ASM exhibited a growth reduction in the week following treatment, but recovered thereafter (Dietrich *et al.* 2005). These workers also found that costs, no costs or even higher seed production could be obtained in ASM-induced plants depending on the combination of environmental factors to which the plants were exposed.

Expression of induced resistance can also be influenced by abiotic stress. For example, Wiese *et al.* (2004) showed that osmotic stress and proton stress led to the induction of active defences against powdery mildew in barley. This suggests that under field conditions, the effectiveness of induced resistance will be greatly influenced by both the biotic and abiotic environment.

Are plants in the field already induced?

Current understanding of induced resistance is based on early research using pathogens to induce

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resistance. Thus, Cruickshank & Mandryk (1960) showed that resistance to infection by *Peronospora tabacina* in tobacco could be induced by prior injection of sporangia of the same pathogen into stems, while Ross (1961*a*, *b*) demonstrated local and systemic resistance in tobacco to infection by the tobacco mosaic virus (TMV). Since then, there have been many reports of resistance induction using prior inoculation with pathogens (see Hammerschmidt 2007). Resistance can also be induced by mycorrhizal infection and colonization and it appears to be effective against necrotrophic pathogens and generalist chewing insects, but not against biotrophic pathogens (Pozo & Azcón-Aguilar 2007). Fungal and bacterial endophytes have been shown to induce resistance (Waller *et al*. 2005; Kang *et al*. 2007), and resistance can also be induced by avirulent nematode species (Ogallo & McClure, 1996; Kosaka *et al*. 2001). In addition, and as already mentioned above, insect attack can also induce resistance (Bostock 2005).

It is clear, therefore, that in the field, plants will be at least partly induced through interaction with both the biotic and abiotic environment. Indeed, in a study of defence gene expression, Pasquer *et al*. (2005) found that when ASM was applied to wheat in the field, no differences in gene expression could be detected, because gene expression was already high in the untreated plants. In more recent work, Herman *et al*. (2007) examined defence gene expression in three tomato cultivars treated with ASM under field conditions. They found that some defence genes were already expressed prior to treatment, although gene expression was increased further following ASM treatment. Both the baseline levels of gene expression and the magnitude of the increase in gene expression following ASM treatment was cultivar dependent (Herman *et al*. 2007). The studies of both Pasquer *et al*. (2005) and Herman *et al*. (2007) examined gene expression, but work on spring barley has shown that activities of defence-related enzymes (peroxidise, cinnamyl alcohol dehydrogenase, chitinase and glucanase) are already induced in untreated plants under field conditions (Walters *et al*. unpublished results). Interestingly, Heil & Ploss (2006) found that a range of wild (non-cultivated) plants exhibited markedly high constitutive activities of a number of defence-related enzymes; they suggested that these high constitutive enzyme activities might be the result of prior, natural infections.

The studies outlined above suggest that plants in the field are already induced, but does this compromise the ability of plants to induce resistance further? The work of Heil & Ploss (2006) on wild plants showed that despite exhibiting high constitutive activities of defence enzymes, some species were clearly able to respond to ASM treatment by further induction of defence enzymes. However, the ability to induce resistance further was dependent on plant life history. Thus, species that flowered early in the spring exhibited low inducibility of resistance, while larger perennials that flowered in late spring or summer were able to induce resistance to much higher levels following ASM treatment (Heil & Ploss 2006). In their work on tomato, Herman *et al*. (2007) found that while ASM-induced defence gene expression following the first application, a much greater level of gene expression was observed following the second ASM application. This suggests that prior induction of resistance does not compromise the ability of the plant to respond to subsequent inductions. Understanding of this phenomenon is still rudimentary, with little information available on the factors controlling the ability of already induced plants to induce resistance further. For example, whether a plant that is already induced can be induced further will depend on other factors e.g. genotype (Walters *et al*. 2005; Herman *et al*. 2007), but information in this area remains inadequate.

CONCLUSIONS

Plant pathology faces great challenges in the years ahead. In addition to the continuing problems of fungicide insensitivity and breakdown of host resistance, there is the spectre of global climate change and the ever-increasing human population. Plant pathologists are charged with providing the means to protect crops from disease at a time when legislation is reducing the number of chemicals available for disease control. There is a clear and urgent need for additional approaches to controlling plant disease and induced resistance offers the prospect of durable, broad-spectrum disease control using the plants own resistance. However, induced resistance is plagued by inconsistency and relatively poor disease control compared with fungicides. These problems relate to the fact that induced resistance is a host response and as such is greatly influenced by genotype and environment. Unfortunately, our understanding of the impact of these influences on induced resistance is poor, as is our understanding of how best to use induced resistance in crop protection practice. There is a need not just for work on which crop varieties are appropriate to use for induced resistance, but also other areas, for example: (i) when should resistance-inducing agents be applied; early or late in the season? (ii) Is induced resistance effective against pathogens with long periods of asymptomatic growth in plant tissue, e.g. *R. secalis* on barley and (iii) can resistance inducers be used as a means of reducing fungicide applications to crops, e.g. can resistance inducers be applied early to reduce pathogen infection and colonization, thereby allowing less fungicide to be used? What is required is research related to specific crops aimed at trying to

determine how best to fit induced resistance into disease control programmes. Farmers and crop protectionists have grown accustomed to high levels, or even complete, disease control. Ultimately, for induced resistance to gain more widespread acceptance in crop protection, there will need to be a lowering of expectation in terms of levels of disease control.

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