Controlling crop diseases using induced resistance: challenges for the future

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Abstract

A number of different types of induced resistance have been defined based on differences in signalling pathways and spectra of effectiveness, including systemic acquired resistance and induced systemic resistance. Such resistance can be induced in plants by application of a variety of biotic and abiotic agents. The resulting resistance tends to be broad-spectrum and can be long-lasting, but is rarely complete, with most inducing agents reducing disease by between 20 and 85%. Since induced resistance is a host response, its expression under field conditions is likely to be influenced by a number of factors, including the environment, genotype, crop nutrition and the extent to which plants are already induced. Although research in this area has increased over the last few years, our understanding of the impact of these influences on the expression of induced resistance is still poor. There have also been a number of studies in recent years aimed at understanding of how best to use induced resistance in practical crop protection. However, such studies are relatively rare and further research geared towards incorporating induced resistance into disease management programmes, if appropriate, is required.

Key words: crop protection, disease control, induced resistance, priming, resistance elicitors, systemic acquired resistance.

Introduction

Treatment of plants with various agents, including cell wall fragments, plant extracts and synthetic chemicals, can induce resistance to subsequent pathogen attack both locally and systemically (Walters and Fountaine, 2009). Such induced resistance rarely leads to complete pathogen control, resulting instead to a reduction in lesion size and/or number (Kuc, 1982). In broad terms, induced resistance can be split into two main types: systemic acquired resistance (SAR) and induced systemic resistance (ISR). SAR can be induced by treatment with a variety of agents, including necrotizing pathogens and certain chemicals (e.g. acibenzolar-S-methyl, ASM), and is mediated by a salicylic acid (SA)-dependent process (Spoel and Dong, 2012). ISR develops as a result of colonization of plant roots by certain strains of plant growth-promoting rhizobacteria (PGPR) and is mediated by a jasmonate (JA)- and ethylene (ET)-sensitive pathway (Spoel and Dong, 2012).

Induction of resistance can lead to the direct activation of defences, but can also lead to the priming of cells, resulting in stronger elicitation of those defences, or other defences, following pathogen attack (Goellner and Conrath, 2008). It seems likely that most induced resistance phenomena are based on a combination of direct induction and priming (Ahmad et al., 2010).

Agents that induce resistance

Resistance to pathogen infection can be induced in plants by a wide range of biotic and abiotic agents (da Rocha and Hammerschmidt, 2005; Lyon, 2007). The first chemical resistance activator, Probenazole, was registered in Japan as Oryzemate in 1975, and since then many other chemical...
and biological activators have been developed, including: ASM, registered as Bion and Actigard (Syngenta), Milsana (Reynoutria sacalinensis extract; KHH BioScience), Elexa (chitosan; SafeScience) and Messenger (harpin protein; Plant Health Care).

In the sections below we will provide selected recent examples of agents that have been demonstrated to induce resistance to pathogens in various crops. Research in this area is thriving, producing an ever-expanding literature. Table 1 provides a list of many of the papers published on the efficacy of resistance activators since 2010.

**Chemical activators**

**ASM**

ASM is widely reported to induce resistance against a broad spectrum of pathogens in many plant species. For example, ASM was shown to induce SAR in rust (*Uromyces viciae-fabae*) and ascochyta blight (*Ascochyta fabae*) on faba bean in both the glasshouse and under field conditions (Siller et al., 2012). ASM-induced protection was obtained in both a susceptible and an incompletely resistant accession of faba bean, and protection was still observed several weeks following application of the activator (Siller et al., 2012). The level of control achieved was not complete for either pathogen, with, for example, ASM reducing the severity of ascochyta blight by between 45 and 66% in field experiments. Interestingly, Siller et al. (2012) found that ASM applied to shoots also provided protection against the root-infecting parasitic plant *Orobanche crenata* on faba bean, confirming previous work, which demonstrated ASM-induced protection against *O. crenata* in pea plants (Pérez-de-Luque et al., 2004). ASM was also reported to control rust infection on pea plants, caused by *Uromyces pisi*, although again control was not complete (Barilli et al., 2010). Here, ASM-induced resistance was associated with increased activities of defence-related enzymes and phenolic content, and indeed evidence was provided for priming of defence enzymes by ASM treatment of both susceptible and resistant genotypes (Barilli et al., 2010).

A number of studies have documented the effectiveness of ASM in inducing resistance against a range of post-harvest diseases when applied before or after harvest (see Table 1). Thus, Lin et al. (2011) found that ASM applied to mango fruits reduced infection by the anthracnose pathogen *Colletotrichum gloeosporioides*. This ASM-induced resistance was accompanied by increased gene expression and activities of peroxidase and polyphenol oxidase, and the effectiveness of ASM was dependent on variety (Lin et al., 2011). Multiple pre-harvest applications of ASM were found to reduce latent infections caused by *Alternaria alternata* and *Fusarium* spp. on muskmelons and here the enhanced resistance was associated with elevated activities of defence-related enzymes and accumulation of phenolic compounds, lignin and flavonoids (Zhang et al., 2011b). ASM was also shown to reduce infection of muskmelon fruits by *Trichothecium roseum* and to reduce disease incidence on fruits during storage at room temperature (Ren et al., 2012).

However, ASM does not always provide protection against pathogens. Thus, field trials on barley failed to show any effect of ASM against barley yellow dwarf virus (Huth and Balke, 2002), while ASM failed to provide significant control of the leaf pathogen *Xanthomonas axonopodis* pvs *citriunelo* and *citris* on sweet oranges (Graham and Leite, 2004). ASM was even found to increase infection of peanut by the late leaf spot pathogen, *Cercosporidium personatatum* (Zhang et al., 2001).

A study of the structure-activity relationships in the benzothiadiazoles, of which ASM is a member, found that the benzothiadiazole skeleton with a 7-carboxylate group is essential for good activity. This study also showed that induced resistance activity is generally determined by the ester group and the greater the molecular weight of the carboxylate derivative the lower the activity (Kunz et al., 1997). In some recent work Du et al. (2011) synthesized a series of benzo-1,2,3-thiadiazole-7-carboxylate derivatives and found that two fluoro-containing derivatives displayed excellent SAR-inducing activity against *Erysiphe cichoracearum* and *Colletotrichum lagenarium* on cucumber. Not only were these derivatives more effective SAR inducers than ASM, but one of them was also cheaper to produce.

**β-Aminobutyric acid**

β-Aminobutyric acid (BABA) is a non-protein amino acid known to induce resistance against many plant pathogens on a range of crop plants. Working on lettuce, Cohen et al. (2010) demonstrated that application of BABA prior to inoculation with the oomycete pathogen *Bremia lactucae* prevented pathogen development. They also showed that SA, JA and abscisic acid played no role in the development of this BABA-induced resistance. Subsequent work showed that BABA was even effective if applied to shoots or roots after infection with *B. lactucae* (Cohen et al., 2011). In fact, if BABA was applied 1 day after inoculation, it led to an induced hypersensitive response in penetrated epidermal cells, whereas when applied 2 days after inoculation it caused massive encasement of the primary haustoria of the pathogen with callose. BABA also provided significant control of the late blight pathogen *Phytophthora infestans* on tomato (Sharma et al., 2012), and reduced severity of *Plasmopara viticola* on grapevine by 62% in field experiments (Tamm et al., 2011). Control of post-harvest blue mould of apple fruit, caused by *Penicillium expansum*, was also provided by BABA, and here, although the BABA-induced protection was associated with increased activities of defence-related enzymes, there was also evidence of a direct fungitoxic effect (Zhang et al., 2011b). Interestingly, BABA-induced protection of *Brassica napus* against the fungal pathogen *Leptosphaeria maculans* was also associated with a combination of modes of action, since it induced SA synthesis and expression of *PR-1*, but was also found to exert a direct fungitoxic effect against the pathogen (Sašek et al., 2012).

**Probenazole**

This activator has been widely used against rice blast, caused by *Magnaporthe grisea*, and bacterial leaf blight, caused...
Table 1. Examples of resistance elicitors and resistance-inducing agents reported to provide plant disease control (from 2010 to present).

<table>
<thead>
<tr>
<th>Type of elicitor/resistance inducer</th>
<th>Protected plant</th>
<th>Targeted pathogen</th>
<th>Reference</th>
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<tbody>
<tr>
<td>Chemical and non-biological inducers</td>
<td>Cucumber</td>
<td>Tricostachium roseum (post-harvest)</td>
<td>Ren et al. (2012)</td>
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<td>Colletotrichum lagenarium</td>
<td>Du et al. (2011)</td>
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<td></td>
<td>Faba bean</td>
<td>Uromyces viciae-fabae</td>
<td>Sillero et al. (2012)</td>
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<td>Orobanche crenata (parasitic plant)</td>
<td>Sillero et al. (2012)</td>
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<td></td>
<td>Cabbage</td>
<td>Peronospora parasitica</td>
<td>van der Wolf et al. (2012)</td>
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<td>Tomato</td>
<td>Colletotrichum gloeosporioides</td>
<td>Lin et al. (2011)</td>
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<td>Alternaria alternata and Fusarium spp.</td>
<td>Zhang et al. (2011)</td>
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<td>Xanthomonas oryzae</td>
<td>Du et al. (2011)</td>
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<td>Maize</td>
<td>Bipolaris maydis</td>
<td>Du et al. (2011)</td>
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<td>Apple</td>
<td>Penicillium expansum</td>
<td>Quaglia et al. (2011)</td>
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<td>Apple</td>
<td>Penicillium expansum (post-harvest)</td>
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<td>Pea</td>
<td>Uromyces pisi</td>
<td>Barilli et al. (2010)</td>
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<td>Peronospora parasitica</td>
<td>van der Wolf et al. (2012)</td>
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<td>Tomato</td>
<td>Phytophthora infestans</td>
<td>Sharma et al. (2012)</td>
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<td>Lettuce</td>
<td>Bremia lactucae</td>
<td>Cohen et al. (2011)</td>
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<td></td>
<td>Oilseed rape</td>
<td>Leptosphaeria macularis</td>
<td>Šašek et al. (2012)</td>
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<tr>
<td></td>
<td>Probenazole</td>
<td>Maize</td>
<td>Bipolaris maydis</td>
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<td>Saccharin</td>
<td>Soybean</td>
<td>Phakopsora pachyrhizi</td>
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<td>Potassium phosphate</td>
<td>Grapevine</td>
<td>Plasmopara viticola</td>
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<td>Arabidopsis</td>
<td>Arabidopsis</td>
<td>Phytophthora cinnamomi</td>
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<td>Thiamine</td>
<td>Pearl millet</td>
<td>Sclerospora graminicola</td>
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<td>Silicon</td>
<td>Rose</td>
<td>Podosphaera pannosa</td>
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<td>Biochar</td>
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<td>Leveillula taurica</td>
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<td>Tomato</td>
<td>Botrytis cinerea</td>
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<td>Strawberry</td>
<td>Colletotrichum acutatum</td>
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<td>Podosphaera parviflora</td>
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<td>Asparagus</td>
<td>Fusarium oxysporum/F. proliferatum</td>
<td>Elmer and Pignatello (2011)</td>
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<tr>
<td>Biological inducers</td>
<td>Plant growth-promoting rhizobacteria</td>
<td>Pepper</td>
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<td>Ochrobactrum lupinum</td>
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<td>Novosphingobium pentaromativorans</td>
<td>Strawberry</td>
<td>Sphaerotheca macularis</td>
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<td>Azospirillum brasilense REC3</td>
<td>Strawberry</td>
<td>Pseudomonas syringae pv. tomato</td>
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<td></td>
<td>Bacillus subtilis FZB24</td>
<td>Arabidopsis</td>
<td>Pseudomonas syringae pv. tomato</td>
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<td>Pseudomonas fluorescens WCS417r</td>
<td>Plant growth-promoting fungi</td>
<td>Colletotrichum orbiculare</td>
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<td>Fusarium equiseti</td>
<td>Cucumber</td>
<td>Rhizoctonia solani</td>
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<td>Biocatalysed fungi</td>
<td>Trichoderma asperellum SKT-1</td>
<td>Arabidopsis</td>
<td>Pseudomonas syringae pv. tomato</td>
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<td>T. harzianum T39</td>
<td>Grapevine</td>
<td>Plasmopara viticola</td>
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<td>T. harzianum/T. atroviride</td>
<td>Tomato</td>
<td>Botrytis cinerea</td>
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<td>T. atroviride</td>
<td>Pine</td>
<td>Diplodia pinae</td>
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<td>Arbuscular mycorrhizal fungi</td>
<td>Glomus intraradices</td>
<td>Rice</td>
<td>Magnaporthe oryzae</td>
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<td></td>
<td>Glomus mosseae</td>
<td>Maize</td>
<td>Rhizoctonia solani</td>
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<tr>
<td>Endophytes</td>
<td>Pseudomonas indica</td>
<td>Barley</td>
<td>Blumeria graminis f.sp. hordei</td>
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</table>
by Xanthomonas oryzae pv. oryzae, for more than 30 years (Iwata, 2001). Despite its extensive use over this period, development of resistance in the target pathogens has not been observed (Iwata, 2001). Probenazole and its active metabolite 1,2-benzisothiazole-1,1-dioxide induce SAR in Arabidopsis by stimulating a site upstream of the point of accumulation of SA in the SAR-signalling pathway (Yoshioka et al., 2001). At present use of probenazole appears restricted to rice, although this might change in the future. Recent studies demonstrated effective control of southern corn leaf blight on maize, caused by Cochliobolus heterostrophus, under glasshouse and field conditions, without detrimental effects on plant growth (Yang et al., 2011b). Since southern corn leaf blight is currently controlled using multiple applications of the fungicide maneby, the ready availability and low toxicity of probenazole could prove useful as an alternative control for this important disease of maize (Yang et al., 2011b).

Saccharin
Saccharin is a metabolite of probenazole (Uchiyama et al., 1973) and has been shown to induce SAR in rice against M. grisea and X. oryzae (Siegrist et al., 1997; Oostendorp et al., 2001). It has also been shown to induce resistance against rust on broad bean (Boyle and Walters, 2005), and powdery mildew and Rhynchosporium commune on barley (Boyle and Walters, 2006; Walters et al., 2009). Saccharin was also shown to protect soybeans against rust (Phakospora pachyrhizi), with root-drench treatment proving more effective than foliar application (Srivastava et al., 2011). In this work, systemic protection was still apparent 15 days following application of saccharin as a root drench.

Phosphite
Phosphite has been used for more than 30 years in the management of Phytophthora diseases in a range of plant species (Hardy et al., 2001). It exhibits a complex mode of action, including direct inhibition of pathogen growth, and direct stimulation of host defences (e.g. Daniel and Guest, 2006). Recent work by Eshraghi et al. (2011) showed that phosphite primes Arabidopsis for a rapid and intense response to infection by Phytophthora cinnamomi, involving heightened activation of a range of defences. Defences genes from both the SA and JA/ET pathways were activated, suggesting the recruitment of a broad array of host defences.

Biochar
Biochar is one of the products of pyrolysis: the direct thermal decomposition of biomass in the absence of oxygen (Laird, 2008). Amendment of soil with biochar has been reported to improve crop performance by, for example, increasing nutrient retention (Chan et al., 2007), promoting mycorrhizal fungi in soil (Warnock et al., 2007) and altering soil microbial populations and functions (Steiner et al., 2008). Soil-applied biochar was also demonstrated to induce systemic resistance to grey mould (Botrytis cinerea) on pepper, powdery mildew (Leveillula taurica) on tomato, and the broad mite pest (Polyphagotarsonemus latus) on pepper (Elad et al., 2010). Similar broad-spectrum control was achieved with strawberry plants grown in biochar-amended medium, where protection was induced against three pathogens with different infection strategies, B. cinerea, Colletotrichum acutatum and Podosphaera aphanis (Harel et al., 2012). In the latter work, soil amendment with biochar primed strawberry plants for enhanced expression of several defence-related genes.

Biological activators

Mycorrhizal fungi
One of the benefits conferred on plants by the arbuscular mycorrhizal (AM) symbiosis is an improved level of resistance to root-infected pathogens (Cordier et al., 1998; Pozo and Azcón-Aguilar, 2007). Effects of the AM symbiosis can also be observed with foliar pathogens, although enhanced resistance and susceptibility have been reported (Shaull et al., 1999; Fritz et al., 2006; Liu et al., 2007). Whether mycorrhizal plants are protected against pathogens seems to depend on the lifestyle of the pathogen. For example, mycorrhizal tomato plants were protected against the necrotrophic fungus Alternaria solani (Fritz et al., 2006; de la Noval et al., 2007), whereas pathogens with a biotrophic lifestyle such as powdery mildews appear to perform better in mycorrhizal plants (Pozo and Azcón-Aguilar, 2007).

In rice, root colonization by the AM fungus Glomus intraradices was accompanied by the systemic induction of genes involved in regulating host defences (such as OsNPR1 and OsAAP2), as well as genes involved in signal transduction and calcium-mediated signalling (e.g. OsDUF26 and OsCaM, respectively) (Campos-Soriano et al., 2012). Mycorrhizal rice plants also exhibited a stronger induction of PR genes in response to infection by M. grisea, suggesting that the protective effect of the AM symbiosis in rice is dependent on both systemic activation of defence regulatory genes and priming for enhanced expression of PR genes (Campos-Soriano et al., 2012).

Pre-inoculation of susceptible maize with the AM fungus Glomus mosseae led to significant reductions in the incidence

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Table 1. Continued

<table>
<thead>
<tr>
<th>Type of elicitor/resistance inducer</th>
<th>Protected plant</th>
<th>Targeted pathogen</th>
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<tbody>
<tr>
<td>Algal extracts</td>
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<tr>
<td>Ulva armoricana</td>
<td>Bean/grapevine/</td>
<td>Powdery mildew (Erysiphe polygoni,</td>
<td>Jaulneau et al. (2011)</td>
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<td></td>
<td>cucumber</td>
<td>Erysiphe necator, Sphaerotheca fulgiginea)</td>
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<tr>
<td>Ulva lactuca</td>
<td>Tomato</td>
<td>Fusarium oxysporum f.sp. lycopersici</td>
<td>El Modafar et al. (2012)</td>
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The authors apologise to researchers whose work we could not include in this review because of space limitations.
and severity of sheath blight, caused by Rhizoctonia solani (Song et al., 2011). This protective effect was accompanied by increased concentrations of DIMBOA (2,4-dihydroxy-7-methoxy-2-1,4-benzoxazin-3(4 H)-one) and enhanced expression of defence-related genes. Indeed, it was suggested that priming was an important mechanism in the AM-induced resistance observed (Song et al., 2011). Mycorrhiza-induced protection has also been reported against endoparasitic nematodes such as Pratylenchus and Meloidogyne (e.g. De la Peña et al., 2006; Vos et al., 2011) and ectoparasitic nematodes (Hao et al., 2012), and even against parasitic angiosperms such as Orobanche and Striga (López-Ráez et al., 2011).

**Plant growth-promoting rhizobacteria and plant growth-promoting fungi**

PGPR are free-living or root-associated bacteria in the rhizosphere of many plant species that increase plant growth and suppress plant disease (Ryu et al., 2006). In addition to suppressing plant pathogens by secretion of antibiotics, some PGPR can also elicit ISR against a broad range of pathogens, nematodes and insects (Jetiyanon and Kloepper, 2002; Ryu et al., 2003, 2007). A particularly well-studied PGPR strain is Pseudomonas fluorescens WCS417r. Recent work by Weller et al. (2012) demonstrated that the antibiotic 2,4-diacylphloroglucinol (2,4-DAPG) is a major determinant of ISR in 2,4-DAPG-producing P. fluorescens, that the genotype of the strain does not affect its ability to induce ISR and that the activity induced by these bacteria operates through the ET- and JA-dependent signal transduction pathway. In other recent research, Hahn et al. (2012) demonstrated effective protection of pepper against bacterial spot disease by mixtures of PGPR under glasshouse and field conditions, while the PGPR Azospirillum brasilense was shown to induce resistance in strawberry plants against anthracnose, caused by Colletotrichum acutatum (Tortora et al., 2012). In the latter work, the protective effect was associated with induction of defence-related genes, increases in phenolic compounds, and deposition of callose. *Penicillium simplicissimum* is a plant growth-promoting fungus isolated from the rhizosphere of zoysia grass (Zoysia tenuifolia) and which has been shown to induce ISR responses in cucumber (Koike et al., 2001) and Arabidopsis thaliana (Hossain et al., 2007). In recent work, P. simplicissimum GP17-2 was found to enhance growth of *A. thaliana* and tobacco, and to induce ISR against cucumber mosaic virus (Elsharkawy et al., 2012). The authors observed that multiple defence pathways were involved in both plants in the GP17-2-mediated resistance to cucumber mosaic virus.

**Biocontrol fungi and endophytes**

A range of biopesticides is now available commercially, most of which are based on the fungal genus Trichoderma (Woo et al., 2006). Some strains of Trichoderma have been reported to elicit ISR and, moreover, colonized roots appear to be primed for an intense defence response to subsequent pathogen attack (Hanson and Howell, 2004; Segarra et al., 2007; Reglinski et al., 2012; Tucci et al., 2011). Thus, Trichoderma harzianum T39 induced resistance against downy mildew in grapevine by priming (Perazzolli et al., 2011), while Trichoderma asperellum SKT-1 induced resistance in Arabidopsis against the bacterial pathogen Pseudomonas syringae pv. tomato DC3000 using a SA-based signalling pathway (Yoshioka et al., 2011). Interestingly, in the work by Yoshioka et al. (2011) a culture filtrate of *T. asperellum* SKT-1 also induced resistance in *Arabidopsis*, but in this case signalling involved a combination of SA as well as JA/ET pathways.

The ISR-inducing effect of *Trichoderma* appears to be genotype-dependent. Thus, in tomato, genetic variability among cultivated and wild tomato lines affected the outcome of the interaction with strains of *T. harzianum* and *Trichoderma atroviridae*, with ISR to *B. cinerea* being observed in some, but not all, tomato lines examined (Tucci et al., 2011). A priming mechanism appeared to be activated, with *B. cinerea* infection of plants pre-treated with *Trichoderma* leading to enhanced activation of JA-responsive genes, boosting systemic resistance in a plant genotype-dependent manner (Tucci et al., 2011).

**Root colonization by the endophytic basidiomycete fungus Piriformospora indica** protects various plant species against abiotic and biotic stresses (Waller et al., 2005; Stein et al., 2008). The protection conferred against pathogens by *P. indica* colonization of roots is classified as ISR, since protection is compromised in *Arabidopsis* defective in the JA pathway (Stein et al., 2008). Recent work on barley found that *P. indica* has only subtle systemic effects on gene expression and host metabolism, in line with ISR (Molitor et al., 2011). However, following inoculation of *P. indica*-colonized plants with powdery mildew, there is a priming of defence-associated genes at an early stage of the interaction. Some of the genes expressed code for antifungal proteins, thereby representing one of the mechanisms responsible for the observed reduction in haustorium formation in leaves of *P. indica*-colonized plants (Molitor et al., 2011).

**Algal extracts and elicitors from algae**

Marine algae provide a source of numerous elicitors, including β-1,3-glucans (laminarin), β-1,3-sulphated fucans, carrageenans and ulvans (e.g. Cluzet et al., 2004; Jaulneau et al., 2010). Extracts of the green alga *Ulva* were shown to contain elicitor-active compounds called ulvans, complex sulphated heteropolysaccharides. Crude *Ulva* extracts were shown to protect Medicago truncatula plants against attack by Colletotrichum trifolii (Cluzet et al., 2004), and to protect Phaseolus vulgaris against infection by Colletotrichum lindemuthianum (Pauliert et al., 2009). More recently, a crude extract prepared from the green macroalga *Ulva armoricana* was found to protect bean, grapevine and cucumber against powdery mildew (Jaulneau et al., 2011). In this work reductions in disease severity of up to 90% were obtained using the algal extract at a concentration of 6 g.l⁻¹ of dry matter. The authors conclude that extracts of this common macroalga could provide an inexpensive source of bioactive compounds for use in crop protection (Jaulneau et al., 2011).

The potential biopesticide activity of polysaccharides from *Ulva lactuca* has also been examined. Ulvans and oligoulvans isolated from the alga were shown to stimulate
defence responses and activate SAR in tomato against the vascular wilt pathogen *Fusarium oxysporum* f.sp. *lycopersici* (El Modafar et al., 2012). The high elicitor activity of these polysaccharides appeared to be related to the presence of the sulphate group and rhamnose residues.

**Factors affecting the performance of induced resistance in the field**

**Host genotype**

Host genotype is known to affect the expression of induced resistance (Martenelli et al., 1993; Hijwegen and Verhaar, 1994; Dann et al., 1998; Resende et al., 2002; Tucci et al., 2011). For example, Walters et al. (2011a) examined the influence of host genotype on the expression of induced resistance in barley to foliar pathogens, including the leaf scald fungus *R. commune*. Using a combination of resistance elicitors (ASM, BABA and cis-jasmone) they found that expression of induced resistance varied greatly across a range of spring barley varieties. Some varieties did not express induced resistance at all and, interestingly, there was no apparent association between the resistance rating of the variety to *R. commune* and its ability to express induced resistance (Walters et al., 2011a). In a similar vein, tomato genotypes varied significantly in their expression of BABA-induced resistance against *Phytophthora infestans* (Sharma et al., 2010). Here, the level of induction was not always related to the resistance rating of the tomato accession, and was significantly influenced by the pathogen isolate. Sharma et al. (2010) also observed that the degree of resistance induction tended to decrease with increasing leaf age, possibly reflecting an acropetal systemic effect of BABA (Cohen and Gisi, 1994). In subsequent work, Sharma et al. (2012) found that BABA performed well when single pathogen isolates were used for inoculation, but performed less well when two or three pathogen isolates were mixed. As these authors point out, studies using a single pathogen isolate or a single host genotype might lead to the wrong conclusions being drawn concerning the effectiveness of resistance elicitors in practice.

In some interesting recent work, basal and induced resistance in *P. vulgaris* was shown to be influenced by domestication. Córdova-Campos et al. (2012) found that basal resistance to the bacterial pathogen *P. syringae* pv. *syringae* was significantly greater in wild accessions of bean than in modern cultivars. Moreover, ASM treatment elevated resistance to pathogen infection in a wild accession and a landrace, but not in the modern cultivars. It appears, therefore, that the yield-improved modern cultivars of *P. vulgaris* have lost a considerable part of the basal and induced broad-spectrum resistance that characterizes their wild relatives and landraces (Córdova-Campos et al., 2012).

**Are plants in the field already induced: possible implications of prior induction?**

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 and 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 and McClure, 1996; Kosaka et al., 2001). In addition, insect attack can also induce resistance (Bostock, 2005).

It is likely, therefore, that in the field plants will be at least partly induced through interaction with both the biotic and abiotic environment. In an interesting study, 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. Subsequently, 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). Interestingly, a range of wild (non-cultivated) plants was found to exhibit markedly high constitutive activities of a number of defence-related enzymes (Heil and Ploss, 2006). The authors suggested that these high constitutive enzyme activities might be the result of prior, natural infections.

An obvious question to ask at this point is, if plants in the field are already induced, does this compromise the ability of plants to induce resistance further? Heil and Ploss (2006), working 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 which flowered in late spring or summer were able to induce resistance to much higher levels following ASM treatment (Heil and Ploss, 2006). Further, Herman et al. (2007) found that while ASM induced defence gene expression in tomato 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. However, more recent work suggests that not all host–pathogen interactions respond in the same way (Walters et al., 2011b). In an attempt to create a state of ‘prior induction’, these authors inoculated leaves one and two of barley with *R. commune*, prior to treating leaves three and four with an elicitor combination, followed by inoculation of these leaves with *R. commune*. In plants where the first two leaves were not inoculated, application of elicitor to leaves three and four induced resistance against *R. commune* and increased expression of the SAR marker gene *PR1b*. In contrast, in plants where the first two leaves were inoculated elicitor applied to leaves three and four did not induce resistance to *R. commune* and did not...
increase expression of PR1b (Walters et al., 2011b). Although it seemed unlikely that the prior inoculation of leaves one and two led to induced resistance, the results show clearly that prior infection of young barley plants with R. commune compromises their ability to respond effectively to elicitors. These results might help to explain the relatively poor performance of induced resistance in the field, particularly in cereals, compared to plants grown under controlled conditions.

Our 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, such as genotype (Herman et al., 2007), but information in this area remains woefully inadequate.

Other considerations: costs and trade-offs associated with induced resistance

Plant defence is costly, requiring energy and resources that would otherwise be used for growth and development. Constitutive resistance would, therefore, appear to be a costly option for a plant. In contrast, induced resistance, where defences are only activated following pathogen attack, is thought to represent a selective advantage over constitutive resistance (Walters and Heil, 2007). This selective advantage might be related to fitness costs, where resistant plants would have decreased reproductive success compared to non-resistant plants under pathogen-free conditions (Heil and Baldwin, 2002). The costs of plant defence include not just allocation costs, which result from the diversion of metabolites and energy from fitness-relevant processes such as growth and reproduction, towards defence, but also 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, for example mycorrhizal associations, and genetic or pleiotropy costs that arise when resistance genes negatively affect fitness-relevant traits (Heil and Baldwin, 2002).

Allocation costs

In some early work in this area, Smedegaard-Petersen and Stolen (1981) showed 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. However, 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 (Csinos et al., 2001; Latunde-Dada and Lucas, 2001; Ziadi et al., 2001; Prats et al., 2002), suggesting 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 (Hermes and Mattson, 1992). But not all work on ASM yielded the same results. For example, in work on bean treated with ASM, no evidence could be found for the existence of allocation costs (Iriti and Faoro, 2003), while the work of Oerke et al. (1989) and Reglinski et al. (1994) showed either no effect on yield or even yield increases in induced plants. These data suggest either that the plants possessed sufficient resources to finance both growth and defence, or they compensated for the resources diverted from growth to defence (e.g. Murray and Walters, 1992). Nevertheless, 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 (e.g. Heil et al., 2000; Dietrich et al., 2005).

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. This was borne out by the work of van Hulten et al. (2006), who found that priming involved fewer costs than direct induction of defences and indeed, was beneficial in terms of plant growth rate and fitness under disease pressure. This was confirmed in subsequent work on spring barley by Walters et al. (2009), who also showed that whether yield benefits were obtained depended on disease pressure, with benefits obtained only under high disease pressure. Priming appears therefore to have clear ecological benefits and would also represent a promising approach for crop protection.

Trade-offs with insects and pathogens with different lifestyles

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 (Thaler et al., 2012). Thus, 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). There are fewer reports of negative crosstalk in the opposite direction. Nevertheless, JA has been shown to suppress SA-induced responses (Nike et al., 1998; Glazebrook et al., 2003). But not all interactions between pathogen and insect resistance are negative: some workers could find no effect (Ajlan and Potter, 1992; Inbar et al., 1998), while others reported a positive effect (Stout et al., 1999; Hatcher and Paul, 2000; Walters et al., 2006). In fact, recent work demonstrated that foliar attack of pepper plants by the sap-sucking whitefly, Bemisia tabaci, elicited resistance against the leaf pathogen X. axonopodis pv. vesicatoria (Xav) (Yang et al., 2011a). Moreover, whitefly attack altered the composition of the rhizosphere microflora, and enhanced resistance to the soil-borne pathogen, Ralstonia solanacearum. Similar results were obtained in subsequent work using pepper plants attacked by the aphid, Myzus persicae (Lee et al., 2012). Aphid attack
elicited resistance against Xav, and primed plants for up-regulation of several defence-related genes following pathogen challenge. These results highlight an important issue and demonstrate why caution should be exercised in studies of prior induction or potential trade-offs, because although prior insect or pathogen attack might well compromise the ability of the plant to induce resistance further, if that prior attack results in priming, it will undoubtedly enhance the plant’s ability to deal with subsequent attacks.

SA is thought to induce resistance against biotrophic pathogens and some phloem-feeding insects, while JA induces resistance against necrotroph pathogens, some phloem-feeding insects and chewing herbivores (Thaler et al., 2012). Working with Arabidopsis, Spoel et al. (2007) found that infection with the biotrophic pathogen P. syringae pv. tomato, which induces SA-mediated defence, made plants more susceptible to the necrotrophic pathogen, Alternaria brassicicola, by suppressing the JA signalling pathway. Recent work under field conditions found an association between resistance induced against a biotroph, Blumeria graminis f.sp. hordei, and a hemibiotroph, R. commune, and increased susceptibility to Ramularia collo-cygni (Walters et al., 2011c). These data hint at the possibility that prior infection with a pathogen with one lifestyle might well compromise the ability of the plant to defend itself against pathogens with another lifestyle. It also suggests the possibility that resistance elicitors which activate SA-mediated defence might compromise defence against pathogens mediated by JA/ET. At present this is speculative but research is required to examine these possibilities.

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).

**Trade-offs with mutualistic symbioses**

Induced resistance is a broad-spectrum resistance against microorganisms and therefore it is not unreasonable to suggest that it might interfere with plant-microbe mutualisms. Several studies have shown that application of SA to the rooting substrate had a negative effect on nodule formation and/or function (Martinez-Abarca et al., 1998; Ramanujam et al., 1998; Lian et al., 2000), and, in ASM-induced SAR in broad bean, treated plants were found to develop fewer and smaller nodules than untreated controls (Heil, 2001). When considering the impact of induced resistance on mycorrhizal infection, colonization and establishment, three apparently different responses have been observed following the induction of resistance: (1) inhibition of colonization of roots by AM fungi (Salazar Costa et al., 2000; Faessel et al., 2010), (2) a transitory decrease in AM colonization (Tosi and Zazzerini, 2000) and (3) no effect on AM colonization (Sonnemann et al., 2002). However, inhibitory effects on AM colonization might be expected to occur if high concentrations of elicitor are used or repeated application of elicitor leads to its accumulation to high concentrations in and around plant roots (1), or the time lag between elicitor application and examination of AM colonization is too long, allowing any transient effects to have disappeared (3). Interestingly, recent work by de Román et al. (2011) found that foliar application of ASM to soybean led to a transient reduction in AM colonization of roots. The negative effects of the resistance induction appeared to be linked directly to the defences activated and not to any possible shifts in allocation of resources from growth to defence. The authors speculate that the ability of the AM fungus to modulate plant defences might be responsible for the transitory nature of the observed reduction in AM colonization (de Román et al., 2011).

**Interactions with the abiotic environment**

As noted above, plant defence is costly, requiring energy and resources, thereby creating a dilemma for the plant: to grow or defend (Herms and Mattson, 1992). It seems reasonable to assume, therefore, that if resources are limiting the ability of a plant to mount an effective defence response is likely to be affected. Indeed, application of ASM to wheat incurred greater allocation costs under nitrogen-limiting conditions than under nitrogen-sufficient conditions (Heil et al., 2000). Subsequent work by Dietrich et al. (2005) found that whether allocation costs were incurred in Arabidopsis treated with ASM was determined by environmental conditions, such as nitrogen supply, water stress and competition with other plants. They also showed that the ASM-treated Arabidopsis plants exhibited a reduction in growth in the week following induction, supporting the observation that genes involved in primary metabolism are down-regulated following activation of resistance (Somsich and Hahlbrock, 1998). However, induced plants seem able to compensate for diversion of energy and resources towards defence by increasing photosynthetic rates (Murray and Walters, 1992) and growth rates (Dietrich et al., 2005).

Interactions can also occur between abiotic stress and resistance to pathogens (Ayres, 1984; Atkinson and Urwin, 2012). For example, water stress has been reported to enhance resistance to powdery mildew in barley grown in dry soil (Ayres and Woolacott, 1980) and, in other work, to increase susceptibility to powdery mildew in barley (Oerke et al., 1992). Also on barley, osmotic stress and proton stress were found to induce active defences against powdery mildew, with the magnitude of the response dependent on the intensity of the stress (Wiese et al., 2004). The ability of a plant to deal with cold temperatures is linked to pathogen resistance in some plants. For example, in winter wheat, resistance to snow mould pathogens is related to plant development and acclimation during the autumn and early winter (Gaudet and Chen, 1987). In more recent work cold hardening of winter wheat increased resistance to the snow mould pathogens Typhula incarnata and Microdochium nivale, as well as to powdery mildew and stripe rust (Gaudet et al., 2011). Under field conditions, plants are exposed to multiple environmental challenges simultaneously (Atkinson and Urwin, 2012). The challenge for plant biologists is to design experiments robust enough to allow such interactions to be revealed and more importantly, understood.
How might induced resistance be used in practice?

Induced resistance and fungicides

If induced resistance is to find its way into crop protection practice, it needs to fit into disease control and crop management programmes. This might be easier for crops where there is no adequate disease control, but what about crops where fungicides and resistance varieties still offer reasonably high levels of disease control? A good example is spring barley where, in the UK for example, good disease control can be obtained using fungicides and choosing appropriate varieties. Here, for elicitors to be taken seriously, they must either out-perform existing control methods or provide other significant benefits, such as reducing the amount of fungicide required, and/or using varieties with good agronomic characteristics but poor disease resistance. Work over several years using an elicitor combination showed that although the disease control provided by the elicitor was inferior to that obtained with fungicides, using the elicitor in conjunction with reduced rate fungicide could provide much higher levels of disease control (Walters et al., 2010). The idea here is that application of elicitor early in the season might reduce levels of pathogen inoculum in the crops, thereby allowing less fungicide to be used later in the season. Reducing the amount of fungicide required for disease control is particularly important for control of late blight on potato, where between 15 and 20 fungicide applications might be necessary in a season. In this regard, it is interesting that incorporation of BABA into the blight control programme allowed up to 25% less fungicide to be used to provide disease control (Liljeroth et al., 2010). Combined use of ASM and standard fungicides was found to reduce the severity of blight on squash, caused by Phytophthora capsici, under field conditions, and provided superior disease control compared to ASM and the fungicides applied singly (Ji et al., 2011).

Frequency of elicitor application

In terms of practical disease control, the frequency of application is a crucial consideration, and although it might be expected that induced resistance might provide long-lasting protection, thus requiring fewer applications of elicitor, this might not always be the case. For example, in field experiments examining the efficacy of ASM against bacterial spot on tomato, Huang et al. (2012) found that weekly applications provided considerably better disease control than applications every 2 weeks. The effectiveness of ASM-induced resistance has been reported to decline significantly 9–12 days following application (Cavalcanti et al., 2006a, 2006b), and although re-application of ASM can lead to the rapid (within 2 days) expression of PR-1, expression declined considerably thereafter (Herman et al., 2007). This information provides a possible reason for the poor control provided by ASM applied every 2 weeks compared to control achieved with weekly applications (Huang et al., 2012).

Control of post-harvest diseases

The possibility that induced resistance might be used to control post-harvest diseases has been the subject of a number of studies. For example, ASM applied during the winter growing period was shown to delay the development of grey mould on harvested strawberry fruit, although no control was obtained if ASM was applied in the summer (Terry and Joyce, 2000, 2004). Also, a single ASM spray was found to reduce the post-harvest severity of Alternaria spp. and Fusarium spp. on rock melons if applied prior to flowering (Huang et al. 2000). More recently, Zhang et al. (2011a) found that ASM application at four different growth stages (flowering, young fruit, fruit enlargement, netting) reduced latent infections by A. alternata and Fusarium spp. in muskmelon fruit, compared to untreated controls. The timings for ASM application were based on the fact that infection by A. alternata and Fusarium spp. occurs at flowering (Ge et al., 2005). The research also demonstrated that reductions in post-harvest disease control increased in proportion to the number of ASM applications (Zhang et al., 2011a).

Effect of species of crop plant

Another important consideration when using induced resistance is the crop plant itself. We have already seen that different varieties of a crop plant can exhibit marked differences in the expression of induced resistance. But what about different crop plants? In the previous sections we saw that the combination of Bion, BABA and cis-jasmoné provided relatively poor levels of disease control on spring barley. Interestingly, the same elicitor combination applied to oilseed rape (Brassica napus) provided control of light leaf spot (caused by Pyrenopeziza brassicae) that was often superior to that provided by fungicides (Fig. 1; Oxley and Walters, 2012). It is possible that induced resistance might not be appropriate for use in some crops, especially if research shows the crop to be poor at responding to elicitors.

Seed treatments

In most of the work described above elicitors were applied to plants as either foliar sprays or a root drench. A convenient means of applying crop protection treatments involves treating the seed. Seed treatments can be particularly useful, since they can provide protection to young plants during a vulnerable stage in their development. Recent work by Worrall et al. (2012) demonstrated that treating tomato seeds with JA and/or BABA primed plants for enhanced defence against pests and pathogens. Plants grown from JA-treated seed exhibited increased resistance against attack by spider mites, caterpillars and aphids, as well as against the necrotrophic fungal pathogen B. cinerea. In contrast, seed treatment with BABA primed plants for enhanced resistance against powdery mildew, caused by Oidium neolycopersici. What was particularly interesting about this work was the finding that protection was long-lasting, with enhanced resistance sustained for up to 8 weeks. Usefully, these priming seed treatments were not associated with reductions in growth (Worrall et al., 2012). Priming of induced resistance by treating seeds would be of great value in agriculture and horticulture, especially for crops which are likely to face pest and pathogen attack early in the growing season. If the priming effect is long-lasting, the need for further crop protection treatments (elicitors or pesticides) would be reduced.
Transgenerational effects

In the work of Worrall et al. (2012) the authors speculate that the long-term effects of priming by seed treatment might be mediated by epigenetic modifications of JA- and BABA-responsive genes in embryonic tissues during imbibition. This speculation is interesting in view of the recent reports of the transgenerational effects of induced resistance in *A. thaliana*, tomato and barley (Luna et al., 2012; Rasmann et al., 2012; Slaughter et al., 2012; Walters and Paterson, 2012). Luna et al. (2012) found that progeny from *Arabidopsis* inoculated with PstDC3000 were primed to activate SA-inducible defence genes and were more resistant to both *Hyaloperonospora arabidopsidis* and PstDC3000. This transgenerational SAR effect was maintained over one pathogen-free generation, suggesting an epigenetic basis to the phenomenon. Similar results were obtained by Slaughter et al. (2012) who found that the descendants of *Arabidopsis* plants primed by treatment with BABA or inoculation with an avirulent isolate of *Pst* exhibited a more rapid and enhanced expression of defence-related genes, coupled with enhanced resistance to a virulent isolate of *Pst*, compared to the progeny of non-primed plants. These authors also found that when transgenerationally primed plants were subjected to an additional priming treatment their progeny displayed an even stronger primed response, suggesting that plants can inherit a sensitization of the priming phenomenon (Slaughter et al., 2012). These findings hint at the possibility of producing disease- and pest-resistant plants by exposing parent plants to a priming treatment.

Combining induced resistance and other biological approaches

As indicated, an important consideration when devising disease control programmes is whether different approaches are compatible. One example is the use of a combination of a chemical resistance elicitor and a biological control. Thus, a combination of ASM and *Pseudomonas fluorescens* Pf2 provided better control of *Ralstonia solanacearum* on tomato than either agent applied singly (Abo-Elyousr et al., 2012). Also on tomato, Myresiotis et al. (2012) found that four PGPR strains of *Bacillus* provided significantly greater control of *Fusarium* crown and root rot when combined with either ASM or a fungicide, compared to any treatment applied alone. In
a similar vein, co-inoculation of cucumber seedlings with the AM fungus *G. mosseae* and the plant growth-promoting fungus *Fusarium equiseti* led to an additive effect on the suppression of anthracnose (Saldajeno and Hyakumachi, 2011). These three examples demonstrate the effectiveness and compatibility of combinations of agents that induce resistance and other forms of biological control. However, caution should be exercised when considering such combinations, because what works in one system might not work in another.

**Putting it into practice**

In crop protection, as in life, one size does not fit all. If induced resistance is to be used in disease management then tailored programmes that use appropriate elicitors at the right time, at the right frequency, to responsive crop cultivars, along with other control methods, need to be devised (Fig. 2). For cereal crops, a suitable programme might involve seed treatment to protect young plants in the first few weeks after emergence. Whether or not foliar application to young plants early in the growing season is also required will depend upon the likelihood of pathogen attack, and will require access to good disease forecasting information. If these elicitor applications are successful in reducing pathogen inoculum, less fungicide might be required later in the season. Research aimed at determining whether, and how, induced resistance can be used in practice has been carried out (e.g. Hong et al., 2011; Tamm et al., 2011). Thus, a combination of thymol (a monoterpenic phenol from thyme) and ASM was found to provide significant control of bacterial wilt (*Ralstonia solanacearum*) on tomato under field conditions (Hong et al., 2011). In this work, thymol was applied as a soil fumigant prior to transplanting plants into the field, and ASM was applied as a foliar spray both prior to and after transplanting (Hong et al., 2011). Used on moderately resistant tomato cultivars, the combination of thymol and ASM not only controlled bacterial wilt but also protected marketable yield. Recently, Pinto et al. (2012) examined the use and economic viability of resistance elicitors (Agro-Mos and potassium phosphite) in the management of grapevine downy mildew, caused by *P. viticola*. They found that although both elicitors reduced infection, potassium phosphite, which induced resistance in this system, was an economically viable option for management of grapevine downy mildew, if used either on its own or in combination with a fungicide (Pinto et al., 2012). Such work is important because without it induced resistance is unlikely to make it into disease management programmes.

**Conclusions**

European Union policy is directed towards significant reductions in pesticide use in the short to medium term (Hillocks, 2012). There is, therefore, an urgent need for additional approaches to controlling plant disease, and induced resistance offers the prospect of durable, broad-spectrum disease control using the plant’s own resistance. However, induced resistance is plagued by inconsistency and relatively poor
disease control compared with pesticides, reflecting the fact that induced resistance is a host response and as such is greatly influenced by genotype and environment. Farmers and crop protectionists, who have grown accustomed to high levels, or even complete, disease and pest control, are unlikely to be enthusiastic about adopting a disease control method which is viewed as inferior to fungicides. Ultimately, for induced resistance to gain more widespread acceptance in global crop protection, there will need to be a lowering of expectation in terms of levels of disease and pest control. There is much to be done, therefore, to convince farmers and growers that induced resistance can work and could provide a useful addition to their disease management programmes. As important as this is, it will take more than getting farmers and growers on board to get induced resistance into practice. A serious obstacle to real progress in this area is getting induced resistance products to the marketplace (Walters et al., 2012). In particular, the high cost of registration, coupled with limited market size for some products, has been identified as a major barrier by Richardson (2005) and Kleebert (2007). However, this has been recognized as a problem by regulatory authorities and in the UK, for example, the Pesticides Safety Directorate (PSD) launched a pilot scheme for biopesticides in 2004. This allowed the requirements for registration to be tailored to the product type and, importantly, offered a significant reduction in the application fee (Richardson, 2005; Whittaker, 2007). This pilot scheme has since evolved into a permanent Biopesticides Scheme run by the PSD (now the Chemicals Regulation Directorate, CRD). This is progress, but clearly much still needs to be done before induced resistance becomes part of regular crop protection practice.

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