REVIEW & INTERPRETATION

Systemic Acquired Resistance and Induced Systemic Resistance in Conventional Agriculture

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ABSTRACT

Plants possess a range of defenses that can be actively expressed in response to pathogens and parasites of various scales, ranging from microscopic viruses to insect herbivores. The timing of these defense responses is critical and can be the difference between being able to cope or succumbing to the challenge of a pathogen or parasite. Systemic acquired resistance (SAR) and induced systemic resistance (ISR) are two forms of induced resistance; in both SAR and ISR, plant defenses are preconditioned by prior infection or treatment that results in resistance (or tolerance) against subsequent challenge by a pathogen or parasite. Great strides have been made over the past 20 yr in understanding the physiological and biochemical basis of SAR and ISR. Much of this knowledge is due to the identification of a number of chemical and biological elicitors, some of which are commercially available for use in conventional agriculture. However, the effectiveness of these elicitors to induce SAR and ISR as a practical means to control various plant diseases is just being realized. In this review, we first briefly summarize the fundamentals of ISR and SAR, for which a number of critical reviews already exist. We then examine the efficacy of SAR and ISR in published field-based studies. We place special emphasis on the benefits, drawbacks, and future considerations for the improved use of chemical and biological elicitors of induced resistance in conventional agriculture; this includes the potential to exploit genetic variability within populations of crop species to improve the utility of SAR and ISR in the field.

Plant researchers have known for over 100 yr that plants can be preconditioned against diseases caused by a variety of parasites. Initially, the debate was over the serological basis of what was then referred to as acquired physiological immunity (reviewed in Chester, 1933a, 1933b). It would take another 60 yr before Ross (1961a, 1961b), from results of experiments using *Tobacco mosaic virus* to sensitize tobacco (*Nicotiana tabacum* L.) against subsequent "challenge" inoculations of *Tobacco mosaic virus* on infected leaves or on distal uninfected leaves, would clearly articulate the concepts of localized acquired resistance and SAR, respectively. Cruikshank and Mandryk (1960) extended this concept to a nonviral pathogen in field-grown tobacco plants using stem injections of *Peronospora tabacina* (D.B. Adam, also referred to as *P. hyoscyami* de Bary f. sp. *tabacina*) to trigger SAR against subsequent inoculations of the same pathogen, the causative agent of blue mold of tobacco. These landmark studies led to the development of the classic SAR models during the 1980s in other plants, such as cucumber (*Cucumis sativus* L.), common bean (*Phaseolus vulgaris* L.), rice (*Oryza sativa* L.), and *Arabidopsis thaliana* (L.) Heynh., demonstrating that SAR was conserved across diverse plant families and was effective against a broad range of viral, bacterial, and fungal pathogens (reviewed in Sticher et al., 1997).

Additional interests in the biological control of soilborne diseases of plants led to the serendipitous discovery of another form of induced resistance associated with the colonization of plant roots by certain plant growth promoting rhizobacteria (PGPR), referred to as induced systemic resistance (ISR) (reviewed in van Loon et al., 1998). ISR is distinct from SAR in several key physiological and biochemical phenotypes that are best defined in *A. thaliana* (Knoester et al., 1999; Pieterse et al., 1996, 1998; Ton et al., 1999, 2001; van Wees et al., 1997). Results of lab and field studies show that, like SAR, ISR is effective against a broad range of diseases caused by viruses, bacteria, and fungi (Murphy et al., 2000; Nandakumar et al., 2001; Niranjan Raj et al., 2003; Raupach and Kloepper, 1998, 2000; van Loon et al., 1998; Wei et al., 1996; Zehnder et al., 2001).

Over the last 20 yr, research on SAR and ISR using model systems has furthered our understanding of the molecular basis of induced resistance and promoted the development of synthetic elicitors and PGPR formulations for use in conventional agriculture (see reviews by Benhamou and Nicole, 1999; Hammerschmidt and Kuc, 1995; Kessler and Baldwin, 2002; Kessmann et al., 1994; Leroux, 1996; Lucas, 1999; Lyon et al., 1995; Sticher et al., 1997; van Loon et al., 1998; Walling, 2000). The future use of SAR and ISR to control crop pests in conventional agriculture seems promising. Since synthetic elicitors and PGPR strains, in general, do not exhibit any direct antimicrobial activity, unlike traditional pesticides, they provide a way to control disease without asserting direct selective pressure on pathogen populations. In addition, the use of synthetic elicitors and PGPR strains seems to be environmentally benign relative to current pesticides. These characteristics make SAR and ISR, and other forms of induced resistance, an attractive approach for managing crop pests in a sustainable manner within the scope of a conventional agriculture system.

Systemic Acquired Resistance and Induced Systemic Resistance

Induced resistance is a physiological "state of enhanced defensive capacity" elicited by specific environmental stimuli, whereby the plant's innate defenses are potentiated against subsequent biotic challenges (van Loon et al., 1998). This enhanced state of resistance is

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Abbreviations: ISR, induced systemic resistance; PGPR, plant growth-promoting rhizobacteria; SAR, systemic acquired resistance.

effective against a broad range of pathogens and parasites, including fungi, bacteria, viruses, nematodes, parasitic plants, and even insect herbivores (Benhamou and Nicole, 1999; Hammerschmidt and Kuc, 1995; Kessler and Baldwin, 2002; McDowell and Dangl, 2000; Sticher et al., 1997; van Loon et al., 1998; Walling, 2000). The two most clearly defined forms of induced resistance are SAR and ISR (Fig. 1), which can be differentiated on the basis of the nature of the elicitor and the regulatory pathways involved, as demonstrated in model plant systems (Knoester et al., 1999; Maleck et al., 2000; Pieterse et al., 1996, 1998; Schenk et al., 2000; Uknes et al., 1992; van Wees et al., 2000; Ward et al., 1991, Yan et al., 2002).

The classic form of SAR can be triggered by exposing the plant to virulent, avirulent, and nonpathogenic microbes, or artificially with chemicals such as salicylic acid, 2,6-dichloro-isonicotinic acid (INA) or benzo (1,2,3) thiadiazole-7-carbothioic acid *S*-methyl ester (BTH) (reviewed in Sticher et al., 1997). Depending on the plant and elicitor, a set period of time is required for the establishment of SAR that corresponds to the time required for the coordinated accumulation of pathogenesis-related proteins (and transcripts) and salicylic acid throughout the plant (Cameron et al., 1994; Uknes et al., 1992; Ward et al., 1991). Any disruption in the plant's ability to accumulate salicylic acid results in the loss of pathogenesis-related gene expression and attenuation of the SAR response, when pathogens are used for induction (Gaffney et al., 1993; Lawton et al., 1995; Vernooij et al., 1994). BTH and INA, however, were still able to elicit SAR and pathogenesis-related gene expression in *A. thaliana* and tobacco plants defective in salicylic acid accumulation (Friedrich et al., 1996; Lawton et al., 1996).

ISR is potentiated by plant growth-promoting rhizobacteria (PGPR), of which the best characterized are strains within several species of *Pseudomonas* that cause no visible damage to the plant's root system (reviewed in van Loon et al., 1998). Unlike SAR, ISR does not involve the accumulation of pathogenesis-related proteins or salicylic acid (Pieterse et al., 1996), but instead, relies on pathways regulated by jasmonate and ethylene (Knoester et al., 1999; Pieterse et al., 1998; Yan et al., 2002). However, these molecular characterizations are based on a limited number of ISR systems. Other examples of ISR are linked to the production of siderophores or salicylic acid by PGPR strains and, therefore, have more in common with SAR (De Meyer and Höfte, 1997; Leeman et al., 1995a, 1996; Maurhofer et al., 1994). Neither the nature of the eliciting agent nor the site of elicitor action on the plant is as critical in the classification of induced resistance phenomena as the biochemical responses incited within the plant. Finally, SAR is effective across a wide array of plant species, whereas there is demonstrated specificity in the ability of PGPR strains to elicit ISR on certain plant species and genotypes (van Wees et al., 1997; Yan et al., 2002).

It is important to realize that SAR and ISR, as just defined, are probably only two outcomes out of an array of possibilities. It is likely that other forms of induced resistance exist that vary in their reliance on salicylic acid, ethylene, and jasmonate and other as yet discovered plant regulators. However, it is the availability of chemical inducers of SAR, such as BTH, and the characterization of numerous PGPR strains, that makes the applied use of induced resistance in conventional agriculture a reality. The next two sections will review published studies assessing SAR and ISR under field conditions. These studies on SAR and ISR are also examined in further detail in Tables 1 and 2, respectively. The SAR

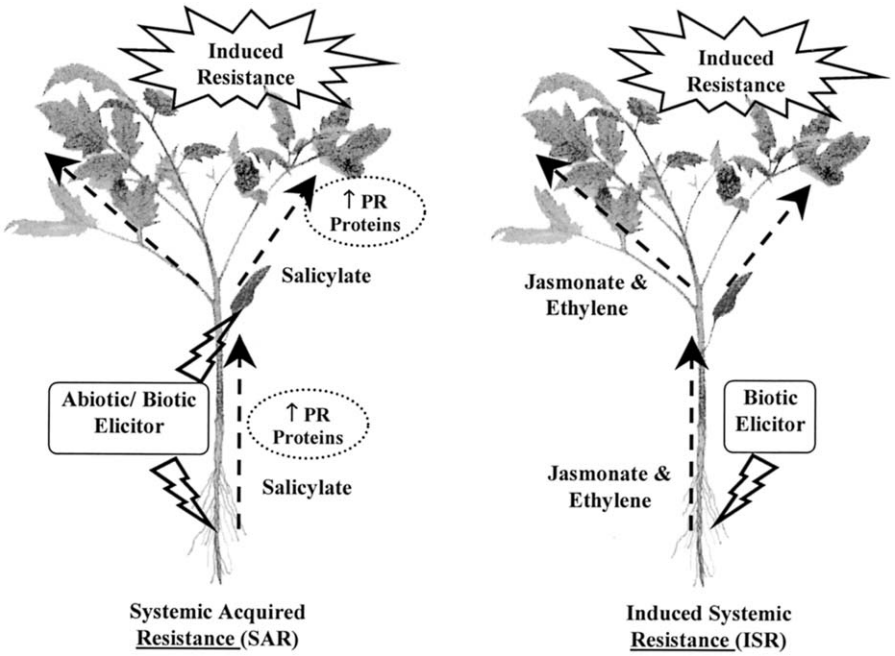

Fig. 1. A pictorial comparison of the two best characterized forms of induced resistance in plants, both which lead to similar phenotypic responses. Systemic acquired resistance, induced by the exposure of root or foliar tissues to abiotic or biotic elicitors, is dependent of the phytohormone salicylate (salicylic acid), and associated with the accumulation of pathogenesis-related (PR) proteins. Induced systemic resistance, induced by the exposure of roots to specific strains of plant growth-promoting rhizobacteria, is dependent of the phytohormones ethylene and jasmonate (jasmonic acid), independent of salicylate, and is not associated with the accumulation of PR proteins (or transcripts). However, both responses are intertwined molecularly, as demonstrated by their reliance on a functional version of the gene *NPR1* in *Arabidopsis thaliana*.

Table 1. The average percentage difference in disease and yield relative to a nontreated control in field experiments using elicitors of systemic acquired resistance and pesticides to control crop diseases.

Crop	Pathogen/disease	SAR elicitor†	Disease (±%)‡		Yield (±%)‡		Comments§	Source
			SAR elicitor	Pesticide standard	SAR elicitor	Pesticide standard		
			%					
Monocots								
Maize	*Peronoscleropora sorghi*/downy mildew	BTH	−35	−31	NA	NA	percent symptomatic plants	Morris et al., 1998
Wheat	*Blumeria graminis* f. sp. *tritici*/powdery mildew	BTH	−64	−55	+3	+28	percent infected leaf area	Stadnik and Buchenauer, 1999a
	Blumeria graminis f. sp. *tritici*/powdery mildew	BTH	−77	NA	0	NA	percent infected leaf area	Stadnik and Buchenauer, 1999b
	Septoria spp./Septoria leaf spot	BTH	−35	NA	+18	+17	values reported in text	Görlach et al., 1996
	Septoria tritici/Septoria leaf blotch	BTH	−52	−45	+3	+31	percent infected leaf area	Stadnik and Buchenauer, 1999a
	Septoria tritici/Septoria leaf blotch	BTH	−46	NA	0	NA	percent infected leaf area	Stadnik and Buchenauer, 1999b
	Puccinia recondita/leaf rust	BTH	−35	NA	+18	+17	values reported in text	Görlach et al., 1996
Dicots								
Solanaceous								
Tobacco	*Pseudomonas syringae* pv. *tabaci* (tox+)/bacterial wildfire	BTH	−99	−32	NA	NA	foliar symptoms rated as lesions/plant	Cole, 1999
	Perenospora hyoscyami f. sp. *tabacina*/blue mold	BTH	−76	−42	NA	NA	foliar symptoms	Perez et al., 2003
	Cercospora nicotiana/eyespot	BTH	−91	−85	NA	NA	foliar symptoms rated as lesions/leaf	Perez et al., 2003
	Alternaria alternata/brown spot	BTH	−89	−61	NA	NA	foliar symptoms rated as lesions/leaf	Perez et al., 2003
	Thanetephorus cucumeris/Rhizoctonia leaf spot	BTH	−71	−10	NA	NA	foliar symptoms rated as lesions/plant	Cole, 1999
	Tomato spotted wilt virus	BTH	−61	−38	NA	NA	percent symptomatic plants	Csinos et al., 2001
Tomato	*Pseudomonas syringae* pv. *tomato*/bacterial speck	BTH	−47	−27	−1	NA	foliar symptoms	Louws et al., 2001
	Pseudomonas syringae pv. *tomato*/bacterial speck	BTH	−42	NA	NA	NA	foliar symptoms rated as lesions/cm^2	Thaler et al., 1999
	Xanthomonas axonopodis pv. *vesicatoria*/bacterial spot	BTH	−50	−40	0	+13	foliar symptoms; total yield	Louws et al., 2001
	Xanthomonas axonopodis pv. *vesicatoria*/bacterial spot and *Colletotrichum coccodes*/Anthracnose	BTH	−51	NA	+26	NA	disease assessed by AUDPC; marketable yield	Abbasi et al., 2002
	Xanthomonas campestris pv. *vesicatoria*/bacterial spot	BTH	−47	NA	NA	NA	foliar symptoms	Inbar et al., 1998
	Alternari solani/early blight	BTH	−33	NA	NA	NA	foliar symptoms	Inbar et al., 1998
	Fulvia fulva/leaf mold	BTH	−52	NA	NA	NA	foliar symptoms	Inbar et al., 1998
	Alternaria solani/early blight, *Phtophthora infestans*/late blight, and *X. axonopodis* pv. *vesicatoria*/bacterial spot	BTH	−4	−94	−27	NA	disease measured in terms of defoliation; marketable yield	Louws et al., 2001
Pepper	*Xanthomonas campestris* pv. *vesicatoria*/bacterial spot	BTH	−64	−30	NA	NA	percent infected leaf area	Buonaurio et al., 2002
	Xanthomonas campestris pv. *vesicatoria*/bacterial spot	BTH	−32	−43	0	+50	disease assessed by AUDPC	Romero et al., 2001
Leguminous								
Bean	*Uromyces appendiculatus*/rust	INA	−70	−24¶	NA	NA	foliar symptoms rated as uridinia/leaf	Dann and Deverall, 1996
Soybean	*Sclerotinia sclerotiorum*/white mold	INA	−46	NA	+9	NA	foliar symptoms	Dann et al., 1998
	Sclerotinia sclerotiorum/white mold	BTH	−59	NA	NA	NA	foliar symptoms; BTH was ineffective in second field trial	Dann et al., 1998
Miscellaneous								
Cotton	*Xanthomonas campestris* pv. *malvacearum*/bacterial blight	BTH	−42	NA	NA	NA	percent infected leaf area	Colson-Hanks et al., 2000
	Alternaria macrospora/Alternaria leaf spot	INA	−32	NA	NA	NA		
	Alternaria macrospora/Alternaria leaf spot	BTH	−45	NA	NA	NA		
	Verticillium dahliae	INA	−38	NA	NA	NA	foliar symptoms	
	Verticillium dahliae	BTH	−37	NA	NA	NA		

Continued next page.

Table 1. Continued.

Crop	Pathogen/disease	SAR elicitor†	Disease (±%)‡		Yield (±%)‡		Comments§	Source
			SAR elicitor	Pesticide standard	SAR elicitor	Pesticide standard		
			%					
Peanut	*Cercosporidium personatum*/late leaf spot	INA	+52	NA	NA	NA	disease assessed by AUDPC	Zhang et al., 2001
	Cercosporidium personatum/late leaf spot	BTH	+35	NA	NA	NA		
Spinach	*Albugo occidentalis*/white rust	BTH	−39	−40	+50	+72	percent infected leaf area	Leskovar and Kolenda, 2002
Apple	*Erwinia amylovora*/fire blight	BTH	−73	−38	NA	NA	percent infected shoots/tree	Maxson-Stein et al., 2002
Pear	*Gymnosporangium asiaticum*/rust	BTH	−63	−96	NA	NA	foliar symptoms rated as lesions/leaf	Ishii et al., 1999
	Venturia nashicola/scab	BTH	−50	−55	NA	NA	percent leaves with symptoms	Ishii et al., 1999

† BTH, benzo (1,2,3) thiadiazole-7-carbothioic acid *S*-methyl ester (also known as acibenzolar-*S*-methyl); INA, 2,6-dichloro-isonicotinic acid.
‡ NA, not available. In those papers where data for multiple pesticide treatments are presented, calculations are based on the pesticide treatments that were most efficacious or most representative.
§ Refer to how disease or yield was measured; AUDPC, area under the disease progress curve.
¶ Plants treated with wetable powder and other inert ingredients from INA formulation (INA not included).

studies examined in this review cover an assortment of crop-pest interactions, but are centered on the use of BTH and INA, where the mechanism of resistance has been well established in laboratory studies. On the other hand, for ISR, 21 different strains of bacteria were investigated in field studies, of which only two of the strains (*Bacillus pumilus* SE34 and *Pseudomonas fluorescens* 89B-61) were further studied to substantiate ISR as the mechanism (Yan et al., 2002). Other up-and-coming "technologies" exist for eliciting induced resistance, such as Messenger (Eden Bioscience Corp., Bothell, WA, USA) and Oxycom (Redox Chemicals, ID, USA; see Kim et al., 2001) not covered here because of the lack, or small number, of published field studies.

Field Studies on Systemic Acquired Resistance

BTH and INA are by far the best studied chemical elicitors available; both are considered functional analogs of salicylic acid, and elicit a systemic form of induced resistance across a broad range of plant–pathogen interactions (Friedrich et al., 1996; Lawton et al., 1996; Maleck et al., 2000; Uknes et al., 1992; Ward et al., 1991). In general, neither chemical elicitor exhibits any direct antimicrobial activity; however, some examples of antimicrobial activity have been documented in association with high elicitor concentrations (Ishii et al., 1999; Rohilla et al., 2002; Tosi and Zazzerini, 2000). Plants exposed to high concentrations of BTH or INA may also exhibit signs of phytotoxicity, but this effect seems to be independent of the induced resistance response (discussed in Louws et al., 2001; Sticher et al., 1997).

Out of the 37 crop disease examples examined in the following SAR section (summarized in Table 1), 32 examples used BTH covering 12 diverse crops, while the remaining five examples used INA. BTH [also known as acibenzolar-*S*-methyl] is distributed by Syngenta Crop Protection (Raleigh, NC, USA; formerly Novartis Crop Protection) as Actigard in the USA and as Bion in Europe. BTH was originally marketed as a means to control powdery mildew of wheat (*Triticum aestivum* L.) and barley (*Hordeum vulgare* L.) in Europe (Görlach et al., 1996).

Efficacy among the Monocots

Görlach et al. (1996) reported successful control of powdery mildew, Septoria leaf spot (caused by *Septoria* spp.), and leaf rust [caused by *P. recondita* Roberge ex Desmaz. f. sp. *tritici* (Eriks. & E. Henn.) D.M. Henderson] of wheat in field trials. BTH was also effective against downy mildew of maize [caused by *Peronosclerospora sorghi* (W. Weston & Uppal) C.G. Shaw] in the field when applied as a seed treatment (Morris et al., 1998). Stadnik and Buchenauer (1999a, 1999b) reported success in field experiments with single applications of BTH for controlling powdery mildew of wheat, but had mixed results against Septoria leaf blotch (caused by *Septoria tritici* Roberge in Desmaz.). However, several fungicides were superior to BTH at controlling powdery mildew and Septoria leaf blotch in the field, and improving overall wheat yields. There was no improvement in the control of powdery mildew or Septoria leaf blotch of wheat with additional applications of BTH over single applications, or with combined applications of BTH with fungicides over lone fungicide applications, and no improvements in yield were associated with the use of BTH over nontreated controls in field trials (Stadnik and Buchenauer, 1999a, 1999b).

Efficacy among Solanaceous Crops

Ample evidence exists supporting the effectiveness of BTH against a variety of fungal, bacterial, and viral diseases of solanaceous plants in experiments under field conditions (Abbasi et al., 2002; Buonaurio et al., 2002; Csinos et al., 2001; Louws et al., 2001; Matheron and Porchas, 2002; Inbar et al., 1998; Perez et al., 2003; Romero et al., 2001; Thaler et al., 1999). The eliciting activity of BTH was quite effective against foliar diseases caused by bacterial pathogens. In a series of field experiments across the eastern USA, BTH applied at a rate of 35 g a.i./ha every 7 to 10 d was as effective

Table 2. The average percentage difference in disease and yield relative to a nontreated control in field experiments using strains of plant growth-promoting rhizobacteria (PGPR) and pesticides to control crop diseases.

Crop	Pathogen/ disease	PGPR strain	Disease (±%)†		Yield (±%)†		Comments‡	Source
			ISR elicitor	Pesticide standard	ISR elicitor	Pesticide standard		
			%					
Monocots								
Rice	*Rhizoctonia solani*/sheath blight	*Pseudomonas fluorescens* PF1	−51	−54	+25	+14	foliar symptoms	Nandakumar et al., 2001
		Pseudomonas fluorescens FP7	−57	−53	+23	+12		
Pearl Millet	*Sclerospora glaucum*/ downy mildew	*Bacillus pumilus* T4	−52	−87	+16	NA	disease incidence from a single field trial	Niranjan Raj et al., 2003
		Bacillus pumilus INR7	−63	−87	+40	NA		
		Bacillus amyloliquefaciens IN937a	−44	−87	+17	NA		
		Bacillus subtilis IN937b	−46	−87	+33	NA		
		Bacillus pumilus SE34	−55	−87	+37	NA		
		Brevibacillus brevis IPC11	−51	−87	+24	NA		
		Bacillus subtilis GB03	−53	−87	+27	NA		
Dicots								
Tomato	*Cucumber mosaic virus*	*Bacillus pumilus* SE34	−45	NA	+43	NA	viral titer	Zehnder et al., 2001
		Kluyvera cryocrescens IN114	−30	NA	+14	NA		
		Bacillus amyloliquefaciens IN937a	−53	NA	+41	NA		
Tomato	*Cucumber mosaic virus*	*Bacillus subtilus* IN937b	−54	NA	+27	NA	viral titer	Zehnder et al., 2001
	Tomato mottle virus	*Bacillus pumilus* SE34	−30	NA	+5	NA	foliar symptoms from a single field trial	
		Bacillus amyloliquefaciens IN937a	−58	NA	+19	NA		
		Bacillus subtilus IN937b	−29	NA	+42	NA	foliar symptoms	
Cucumber	*Erwinia tracheiphila*/ bacterial wilt	*Bacillus pumilis* INR-7	−86	−53	+27	+5	% vines wilted from a single field trial; insecticide used for pesticide standarad	Zehnder et al., 2001
		Serratia marcesens 90-166	−89	−53	+35	+5		
	Pseudomonas syringae pv. *lachrymans*/ angular leaf spot	*Pseudomonas putida* 89B-61	−34	−38¶	+27	−2¶	foliar symptoms	Wei et al., 1996
		Serratia marcescens 90-166	−20	−38	+27	−2		
		Flavomonas oryzihabitans INR-5	−32	−38	+16	−2		
		Bacillus pumilus INR-7	−42	−38	+28	−2		
		Bacillus pumilis INR-7	−69	NA	+16	NA	disease incidence	Raupach and Kloepper, 1998
		Curtobacterium flaccumfaciens ME1	−16	NA	+20	NA		
		Bacillus pumilus GB03	−52	NA	+19	NA		
		INR7 + ME1	−80	NA	+8	NA		
		INR7 + GB03	−67	NA	+3	NA		
Cucumber	*Pseudomonas syringae* pv. *lachrymans*/ angular leaf spot	ME1 + GB03	−50	NA	+27	NA	disease incidence	Raupach and Kloepper, 1998
		INR7 + ME1 + GB3	−86	NA	+27	NA		
		Bacillus pumilus INR-7	−57	NA	+57#	NA	disease severity and yield averaged across methyl bromide treated and nontreated field trials	Raupach and Kloepper, 2000
		Curtobacterium flaccumfaciens ME1	−38	NA	+32#	NA		
		Bacillus pumilus GB03	−38	NA	+22#	NA		

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at reducing the severity of bacterial spot [caused by *Xanthomonas axonopodis* pv. *vesicatoria* Vauterin et al. or *X. campestris* pv. *vesicatoria* (Doidge) Dye] and bacterial speck [caused by *Pseudomonas syringae* pv. *tomato* (Okabe) Young et al.] on foliage and fruit of tomato (*Lycopersicon esculentum* Mill.) as standard bacterization treatments that always included copper hydroxide (Louws et al., 2001). BTH was especially effective at controlling field epidemics of bacterial speck and spot when copper resistant strains of *P. syringae* pv. *tomato* and *X. axonopodis* pv. *vesicatoria* predominated (Louws et al., 2001). Similar control was obtained with

Table 2. Continued.

Crop	Pathogen/ disease	PGPR strain	Disease (±%)†		Yield (±%)†		Comments‡	Source
			ISR elicitor	Pesticide standard	ISR elicitor	Pesticide standard		
			%					
		INR7 + ME1	−54	NA	+44#	NA		
		INR7 + GB03	−57	NA	+41#	NA		
		ME1 + GB03	−42	NA	+36#	NA		
		INR7 + ME1 + GB03	−57	NA	+35#	NA		
	Pseudomonas syringae pv. *lachrymans*/ angular leaf spot; *Colletotrichum orbiculare*/ anthracnose	*Bacillus pumilus* INR7	−48	−62††	+59#	NA	foliar symptoms of angular leaf spot and anthracnose; pesticide standard used plants treated with BTH	Raupach and Kloepper, 1998
		Burkholderia gladioli IN26	−26	−62††	+43#	NA		
		INR7 + IN26	−45	−62††	+66#	NA		
		INR7 + ME1 + GB03	−55	−62††	+61#	NA		
		Bacillus pumilus INR-7	−36	NA	+29#	NA	disease severity and yield averaged across methyl bromide treated and nontreated field trials	Raupach and Kloepper, 2000
		Curtobacterium flaccumfaciens ME1	−24	NA	+21#	NA		
		Bacillus pumilus GB03	−29	NA	+25#	NA		
		INR7 + ME1	−54	NA	+23#	NA		
Tomato	*Pseudomonas syringae* pv. *lachrymans*/ angular leaf spot; *Colletotrichum orbiculare*/ anthracnose	INR7 + GB03	−44	NA	+27#	NA	disease severity; averaged across methyl bromide treated and nontreated field trials	Raupach and Kloepper, 2000
		ME1 + GB03	−47	NA	+29#	NA		
		INR7 + ME1 + GB03	−56	NA	+34#	NA		
	Colletotrichum orbiculare/ anthracnose	*Pseudomonas putida* 89B-61	−54	+153¶	+27	−2	foliar symptoms; used plants preinoculated with *C. orbiculare* to elicit SAR for pesticide standard	Wei et al., 1996
		Serratia marcescens 90-166	−37	+153¶	+27	−2		
		Flavomonas oryzihabitans INR-5	−51	+153¶	+16	−2		
		Bacillus pumilus INR-7	−56	+153¶	+28	−2		
Pepper	*Meloidogyne incognita*/root-knot nematode	GB03 + IN937a	+12	NA	−5	NA	disease severity	Kokalis-Burelle et al., 2002
		GB03 + SE34	−15	NA	−15	NA		
		GB03 + IN937b	−14	NA	−17	NA		
		GB03 + INR7	−6	NA	−15	NA		
		GB03 + *Bacillus cereus* C4	−32	NA	−2	NA		
Peanut	*Cercosporidium personatum*/ late leaf spot	*Bacillus cereus* C1	−33‡‡	−37‡‡	NA	NA	disease severity based on defoliation at 90 d after planting;	Zhang et al., 2001
		Bacillus cereus C3	+2‡‡	−37‡‡	NA	NA		
		Bacillus cereus C5	−30‡‡	−37‡‡	NA	NA		
		Bacillus pumilus T4	−11‡‡	−37‡‡	NA	NA		

† NA, not available. In those papers where data for multiple pesticide treatments are presented, calculations are based on the pesticide treatments that were most efficacious or most representative.
‡ Refer to how disease or yield was measured; AUDPC, area under the disease progress curve.
¶ Plants preinoculated with *C. orbiculare* to elicit systemic acquired resistance.
# Yield assessed by main runner length.
†† Plants treated with BTH, benzo (1,2,3) thiadiazole-7-carbothioic acid *S*-methyl ester (also known as acibenzolar-*S*-methyl).
‡‡ Disease severity across pesticide and PGPR treatments averaged +3% relative to nontreated control at 105 d after planting.

repeated applications of BTH in pepper (*Capsicum* spp.) against bacterial spot (caused by *X. campestris* pv. *vesicatoria*) and in tobacco against wildfire [caused by *P. syringae* pv. *tabaci* (Wolf & Foster) Young et al.] (Buonaurio et al., 2002; Cole, 1999). In the field, tomato plants treated repeatedly with BTH carried reduced population densities of *X. axonopodis* pv. *vesicatoria* and *P. syringae* pv. *tomato* on leaves until 1 mo and 2 mo, respectively, when there were no differences in bacterial leaf populations relative to the controls, suggesting a resistance mechanism based on tolerance to the pathogen rather than exclusion or inhibition of bacterial growth (Louws et al., 2001). In field-grown tomatoes, BTH was found to increase the expression level of the pathogenesis-related gene, *P4* (equivalent to *PR-1* of tobacco and *A. thaliana*), extending findings from lab-based experiments to the field (Thaler et al., 1999).

On tobacco, BTH offered effective control against blue mold and against several fungal diseases such as frogeye leaf spot (caused by *Cercospora nicotianae* El-

lis & Everh.), brown spot [caused by *Alternaria alternata* (Fr.:Fr.) Keissl.] and Rhizoctonia leaf spot (caused by *Rhizoctonia solani* Kühn) (Csinos et al., 2001; Perez et al., 2003). Repeated applications of BTH also controlled early blight of tomato (caused by *Alternaria solani* Sorauer), while others found that BTH offered little protection against the combined effects of late blight [caused by *Phytophthora infestans* (Mont.) de Bary] and early blight of tomato, or against blackshank of tobacco [caused by *Phytophthora parasitica* Dastur var. *nicotianae* (Breda de Haan) Tucker] (Csinos et al., 2001; Inbar et al., 1998; Louws et al., 2001). Finally, BTH was also found to protect tobacco plants against the effects of the thrip-vectored *Tomato spotted wilt virus* in field-grown tobacco, as assessed by a reduction in the number of symptomatic plants (Csinos et al., 2001).

Efficacy among Leguminous Crops

Limited field experiments involving the effects of INA and BTH on diseases of legumes have been reported (Dann and Deverall, 1996; Dann et al., 1998). Reduced densities of uredinia of the rust fungus, *Uromyces appendiculatus* Unger, on trifoliolates of common bean were obtained when INA was applied at least 7 d before inoculation, but not at 2 h before inoculation (Dann and Deverall, 1996). An additional application of INA during pod-set did not improve resistance of common bean plants to *U. appendiculatus*, as opposed to a single application to the first trifoliolate (Dann and Deverall, 1996).

Repeated applications of INA to field-grown soybean [*Glycine max* (L.) Merr.] partially reduced symptoms of white mold [caused by *Sclerotinia sclerotiorum* (Lib.) deBary] in two out of three field trials. INA was most efficacious in suppressing white mold on the susceptible cultivars Elgin 87 and Williams 82 but not on partially resistant cultivars Corsoy 79 or NKS19-90 (Dann et al., 1998). However, the reductions in white mold observed on cultivars Elgin 87 and Williams 82 varied with field location and year. The first of two field trials that included BTH found it most effective against white mold when applied to soybean cultivar Elgin 87, but was ineffective in a second field trial where a 10-fold higher dose of BTH was applied (Dann et al., 1998).

Efficacy on Fruit Trees, Cotton, and Spinach

Of particular interest is the reported control of several diseases of apple (*Malus domestica* Borkh.) and Japanese pear (*Pyrus pyrifolia* Nakai) with BTH in field trials (Ishii et al., 1999; Maxson-Stein et al., 2002). When applied to field grown Japanese pear trees, BTH controlled scab (caused by *Venturia nashicola* Tanaka & Yamamoto) and rust (caused by *Gymnosporangium asiaticum* Mayabe ex. Yamada) with multiple applications. Control of scab with BTH was equivalent to that observed with the fungicide, polycarbamate; however, this was not the case with rust where the control exhibited by BTH was not as effective as that with polycarbamate (Ishii et al., 1999). Weekly applications of BTH as a foliar spray effectively reduced the incidence and severity of fire blight [caused by *Erwinia amylovora* (Burrill 1882) Winslow et al., 1920] comparable to foliar applications of the antibiotic streptomycin (Maxson-Stein et al., 2002). The ability of BTH to inhibit the extension of fire blight cankers was directly proportional to the application rate. BTH application induced the expression of several pathogenesis-related genes (*PR-1*, *PR-2*, and *PR-8*) in greenhouse grown apple seedlings (Maxson-Stein et al., 2002). Other greenhouse studies have found promise for the use of BTH in citrus against scab (caused by *Elsinoë fawcettii* Bitancourt & Jenk.), melanose (caused by *Diaporthe citri* F.A. Wolf), and Alternaria brown spot [caused by *Alternaria alternata* (Fr.:Fr) Keissl.] (Agostini et al., 2003).

Colson-Hanks and Deverall (2000) reported successful control of Alternaria leaf spot (caused by *Alternaria macrospora* A. Zimmerm.), bacterial blight [caused by *Xanthomonas campestris* pv. *malvacearum* (Smith) Dye], and Verticillium wilt (caused by *Verticillium dahliae* Kleb.) of cotton [*Gossypium hirsutum* L. or *G. barbadense* L. (Pima cotton)] in a series of field experiments with the use of BTH and INA. Single applications of either BTH or INA reduced the symptoms of both Alternaria leaf spot and bacterial blight. Multiple applications of BTH or a single application of INA reduced the severity of Verticillium wilt of cotton (Colson-Hanks et al., 2000).

In field trials, BTH use was as effective at controlling white rust (caused by *Albugo occidentalis* G.W. Wils.) of spinach (*Spinacea oleracea* L.) as a standard fungicide cocktail of mefenoxam [methyl *N*-(methoxyacetyl)-*N*-(2,6-xylyl)-D-alaninate] and copper hydroxide, relative to the nontreated control (Leskovar and Kolenda, 2002). The use of BTH to control white rust was not as effective as the fungicide trifloxistrobin or azoxystrobilurin, strobilurin derivatives that inhibit mitochondrial respiration in fungi. While the additional application of BTH with either of the strobilurin fungicides, improved the control of white rust relative to lone applications of either strobilurin fungicide (Leskovar and Kolenda, 2002).

Field Studies on Induced Systemic Resistance

A number of plant growth-promoting rhizobacteria (PGPR) have been identified as potential ISR elicitors, for their ability to control systemically various diseases when localized to plant roots, as a soil drench, transplant mix, root dip, or seed treatment (reviewed in van Loon et al., 1998). In field trials, the use of several PGPR strains was effective at controlling several diseases of cucumber, including anthracnose [caused by *Colletotrichum lagenarium* (Pass.) Ellis & Halst, also referred to as *C. orbiculare* (Berk. & Mont.) Arx], angular leaf spot [caused by *Pseudomonas syringae* pv. *lachrymans* (Smith and Bryan) Young et al.], and bacterial wilt [caused by *Erwinia tracheiphila* (Smith) Bergey et al.] (Raupach and Kloepper, 1998, 2000; Wei et al., 1996; Zehnder et al., 2001). While the protection afforded by many of these PGPR strains varied under field conditions, a few PGPR strains have shown remarkable efficacy. *Bacillus pumilus* INR-7 was an exemplary example of a PGPR

strain that effectively protected cucumber plants against angular leaf spot and anthracnose in several field trials in three different years (Raupach and Kloepper 1998, 2000; Wei et al., 1996). Cucumber plants seed-treated with either *Bacillus pumilus* INR-7 or *Serratia marcesens* 90-166, in comparison to nontreated plants, exhibited a lower incidence of bacterial wilt and harbored fewer cucumber beetles (*Diabrotica undecimpuctata* Howardi Barber and *Acalymma vittata* Fabricius) in two independent field trials (Zehnder et al., 2001). Further experiments in a controlled environment attributed the protection against bacterial wilt to ISR and a corresponding reduction in the beetle feeding stimulant cucurbitacin in PGPR treated cucumber plants (Zehnder et al., 2001). In greenhouse and field experiments, *Bacillus pumilus* INR-7 was also among several PGPR strains that protected pearl millet [*Pennisetum glaucum* (L.) R. Br.] against downy mildew [caused by *Sclerospora graminicola* (Sacc.) J. Schröt] but not to the same extent as the systemic fungicide metalaxyl [*N*-(2,6-dimethylphenyl)-*N*-(methoxyacetyl)alanine methyl ester] (Niranjan Raj et al., 2003).

The use of PGPR strains to control viral diseases of tomato has shown limited success in the field (Murphy et al., 2000; Zehnder et al., 2001). While several PGPR strains were identified in greenhouse trials to induce resistance against *Cucumber mosaic virus*, a cucumovirus, on tomato, the effectiveness of these strains varied under field conditions when under intense disease pressure (Raupach et al., 1996; Zehnder et al., 2001). Similar results were found with *Tomato mottle virus*, a geminivirus, on tomato; none of the PGPR strains tested were able consistently to control disease severity in three field trials, regardless of application method (Murphy et al., 2000; Zehnder et al., 2001). The inconsistency of PGPR-mediated ISR against *Tomato mottle virus* in field trials was attributed to an increase in disease pressure associated with the increased density of the whitefly vector of the virus (Murphy et al., 2000).

A variety of application methods has also been evaluated in the field that should improve the integration of PGPR-mediated ISR into conventional agriculture, and in some cases with improved efficacy. For example, Raupach and Kloepper (1998) found that repeated foliar spray applications of *Bacillus pumilus* INR-7 or *Flavomonas oryzihabitans* INR-5 were as effective as seed-treatments at controlling a mixed infection of angular leaf spot and anthracnose on cucumber. Similar results were obtained when comparing seed applications of a talc-based formulation versus a fresh suspension of PGPR strains for protection against downy mildew on pearl millet (Niranjan Raj et al., 2003). Talc-based formulations would allow for easier storage and application of PGPR strains, rather than dealing with liquid bacterial suspensions. Nandakumar et al. (2001) applied PGPR strains to rice plants as a single seed, root, soil, or foliar application or in combinations of two, three and four application methods, and found that combinations of three or four application methods were more effective than single applications at controlling sheath blight (caused by *Rhizoctonia solani*) of rice in two field trials. Finally, Raupach and Kloepper (1998, 2000) found that applying several PGPR strains, rather than a single strain, improved the control of anthracnose and angular leaf spot in several field and greenhouse experiments, as effectively as the use of BTH.

ISR and SAR within the Context of Organic Soil Amendments

Organic soil amendments have traditionally been associated with the suppression of diseases caused by soilborne pathogens in greenhouse (Boehm and Hoitink, 1992; Zhang et al., 1996, 1998) and field environments (Drinkwater et al., 1995; Workneh et al., 1993; Workneh and van Bruggen, 1994), but there are also examples of foliar disease suppression in both environments as well. Zhang et al. (1996, 1998) demonstrated that cucumbers and *A. thaliana* plants grown in composted pine bark potting mixes exhibited resistance to foliar diseases caused by *Colletotrichum lindemuthianum* (Sacc. & Magnus) Lams.-Scrib. and *Pseudomonas syringae* pv. *maculicola* (McCulloch) Young et al., respectively, relative to plants grown in a peat mix conducive to disease. This compost-mediated plant resistance corresponded to an increase in the activities of several defense- and pathogenesis-related proteins following inoculation of the pathogen, implicating the involvement of induced plant defenses (Zhang et al., 1996, 1998).

Abbasi et al. (2002) evaluated the effects of soil amendments derived from composted yard wastes and composted cannery waste on tomato production in field trials over two growing seasons. While each amendment offered some protection against anthracnose [caused by *Colletotrichum coccodes* (Wallr.) S.J. Hughes] and bacterial spot (caused by *Xanthomonas axonopodis* pv. *vesicatoria* or *X. campestris* pv. *vesicatoria*) of tomato, neither was consistent over the two year duration of the field trials. Tomato plants grown in compost-amended soils consistently produced higher marketable yields. However, the addition of composted amendments also increased the progress of foliar diseases, as measured over time, relative to tomato plants grown in nonamended plots, while the application of BTH impeded disease progress by 55% to 60% regardless of soil treatment (Abbasi et al., 2002).

Field experiments focused on assessing the effects of soil amendments derived from paper mill residuals on plant health, found that soils amended with batches of composted paper mill residuals suppressed the severity of foliar brown spot (caused by *Pseudomonas syringae* pv. *syringae* van Hall) of bean and the incidence of angular leaf spot of cucumber compared to that on plants grown in nonamended soils or soils amended with batches of paper mill residuals that were not composted (Stone et al., 2003). The same field soils also conferred resistance on tomato and *A. thaliana* plants against bacterial speck (caused by *P. syringae* pv. *tomato*) in growth chamber experiments, as demonstrated by a 35 to 65% reduction of disease symptoms on plants grown in soils amended with batches of composted paper mill residuals relative to plants grown in nonamended soil or soil

amended with batches of a noncomposted paper mill residuals (Vallad et al., 2000, 2003). The suppression of bacterial speck associated with plants grown in batches of composted paper mill residuals, in several experiments, was equivalent to that induced by treating plants with BTH.

Growth chamber experiments with *A. thaliana* also revealed that the resistance induced by composted paper mill residuals was associated with increases in the expression of plant pathogenesis-related genes before pathogen inoculation, and was disrupted in a *npr1* defense mutant and a *NahG* transgenic line, which are molecular features characteristic of SAR (Vallad et al., 2000, 2003). Others have also documented beneficial cytological changes in tomato plants grown in batches of paper mill residuals suppressive to crown and root rot, as a result of the formation of physical barriers at sites of attempted penetration and reduced colonization of plant roots by *Fusarium oxysporum* (Schlechtend.:Fr.) f. sp. *radicis-lycopersici* (W.R. Jarvis & Shoemaker), the causal fungal pathogen (Pharand et al., 2002). These studies stress the importance of the soil environment, beyond crop growth, to improve the defensive potential of the crop, and the potential to use organic amendments to modify the soil environment accordingly.

Considerations for the Use of Induced Resistance

The previous sections examined how chemical and biological elicitors can be practically applied to control pathogen and parasite problems encountered in an assortment of conventional agricultural settings, in lieu of pesticides in many cases. However, like all technologies, there are benefits and drawbacks that need to be considered. There are also biological limitations that may hinder the practical use of chemical and biological elicitors without further research and development. The next section will address these topics.

Physiological Costs of Induced Resistance

The phenomenological characterization of induced plant resistance in a number of controlled and uncontrolled environments has given us an idea about the breadth of these defenses and the costs of fitness that plants incur when deploying these defenses, as measured in terms of vegetative and reproductive growth (summarized as yield in Tables 1 and 2). Most experiments evaluated a range of BTH application rates, and reported a tradeoff between effective disease control and either phytotoxic effects or reduced plant productivity (Abbasi et al., 2002; Cole, 1999; Perez et al., 2003). A consistent theme from several of the field experiments using BTH or INA was the reduction of crop yield (Louws et al., 2001; Romero et al., 2001). Often these reductions were statistically insignificant. For example, when Louws et al. (2001) summarized yield data across 22 field experiments with tomato, plots treated with BTH yielded 11% less, on average, than plots treated with a standardized bacterization treatment (that always included copper hydroxide) and 2.1% less than nontreated control plots. However, only in 1 out of the 22 field experiments was the affect of BTH on yield statistically less than obtained using a standardized bacterization treatment (an 18% reduction in yield); coincidently, only in 1 out of the 22 field experiments did the standardized bacterization treatment statistically yield more than the nontreated control treatment. They also observed that tomato seedlings treated with BTH were smaller than nontreated plants in greenhouse experiments (Louws et al., 2001).

Romero et al. (2001) found that the growth of pepper plants was also greatly influenced by the use of BTH, manifested as a reduction in yield or a delay in plant maturity. These effects on plant growth were apparent across several pepper cultivars when compared to plants treated with copper hydroxide, but only when plants were infected with *Xanthomonas campestris* pv. *vesicatoria* (causal agent of bacterial spot). In experiments that carefully excluded *Xanthomonas campestris* pv. *vesicatoria*, there were no significant yield differences between BTH treated and copper hydroxide treated plants (Romero et al., 2001). In field trials, PGPR strains generally improved plant growth and yields over nontreated controls; but improved plant growth was not associated with ISR efficacy (Murphy et al., 2000; Niranjan Raj et al., 2003; Nandakumar et al., 2001; Raupach and Kloepper, 1998, 2000; Wei et al., 1996; Yan et al., 2002).

An inherent difference between monocots and dicots exists in terms of the longevity of the induced resistance elicited by BTH. While single applications of BTH were generally sufficient at inducing resistance over the life span of a monocot crop, such as wheat, dicot crops required repeated applications of BTH to extend protection over time (Cole, 1999; Dann and Deverall, 1995; Görlach et al., 1996; Louws et al., 2001; Morris et al., 1998; Romero et al., 2001). Regardless, there is evidence that there are physiological costs associated with the induction of resistance in monocots as well.

Stadnik and Buchenauer (1999a, 1999b) reported that the effect of BTH on disease and yield of wheat was influenced by the stage of plant growth. In field experiments, BTH treatments applied during the end of tillering (growth stage 28) controlled powdery mildew more effectively then when applied at the middle of tillering (growth stage 25), even though yields from plants treated at the later stages of tillering were numerically less (Stadnik and Buchenauer, 1999a). Several fungicide treatments also effectively controlled powdery mildew, although to a lesser extent than BTH, and improved wheat yields over a nontreated control. The combined use of BTH and fungicide treatments effectively controlled powdery mildew and Septoria leaf blotch of wheat better than either individual treatment and improved wheat yields over a nontreated control and BTH use alone (Stadnik and Buchenauer, 1999a). Foliar applications of urea with BTH also improved wheat yields without disrupting the effective control of BTH, regardless of the increased symptoms of powdery mildew associated with the increase in available nitrogen (Stadnik and Buchenauer, 1999b).

A number of hypotheses have been put forth to explain how plants reallocate resources during the induc-

tion of plant defenses and how induced resistance benefits the overall fitness of the plant relative to constitutive defense mechanisms (Coley et al., 1985; Hatcher and Paul, 2000; Heil, 1999; Heil et al., 2000; Herms and Mattson, 1992; Simms and Rausher, 1987; reviewed in Heil 2001). A few studies have tried to address these costs directly in various settings. In a series of greenhouse experiments, Stout et al. (1998) examined the expression of several constitutive and inducible defenses of tomato under different fertilization regiments that varied nitrogen availability. While nitrogen deficiency increased the level of total proteins and phenolics in tomato plants, it had little bearing on the activity levels of proteinase inhibitors or polyphenol oxidase in nontreated plants, or plants induced by a foliar application of jasmonic acid or by herbivore damage caused by *Helicoverpa zea* (Boddie). Increased nitrogen levels tended to decrease constitutive activity levels of polyphenol oxidase and proteinase inhibitors but did not interfere with the induction of these defenses (Stout et al., 1998). These results suggest that constitutive plant defenses are influenced more by nitrogen availability than inducible plant defenses (Stout et al., 1998).

In greenhouse and field experiments, radish (*Raphanus sativus* L.) plants induced by limited insect damage or a foliar application of jasmonic acid exhibited less tissue damage from subsequent insect herbivory, accumulated higher amounts of glucosinolates, and were reproductively more fit than mechanically damaged or nonmanipulated plants (Agrawal, 1998, 1999; Agrawal et al., 1999). However, in the absence of herbivore pressure, induced plants were reproductively less fit in terms of flower development and pollen production (Agrawal, 1999). Heil et al. (2000) observed effects on the vegetative growth and seed production of wheat plants induced with BTH in the absence of disease pressure, regardless of the growing conditions or fertilization regiment. However, they found that BTH impeded growth the most during the production of lateral shoots and under nitrogen-limiting conditions, demonstrating the importance of plant growth stage and nutritional status when assessing the physiological costs of induced resistance (Heil et al., 2000). Others have pointed out similar physiological costs associated with resistance induced by applications of jasmonic acid, and also the importance of induced resistance in competition among neighboring plants (Baldwin, 1998; van Dam and Baldwin, 1998). Interestingly, similar vegetative and reproductive costs have also been associated with *R*-gene mediated resistance (vertical resistance) (reviewed in Bergelson and Purrington, 1996; Bergelson, 1994; Tian et al., 2003).

Conflicts in Plant Signaling Pathways

In nature, plants respond to a multitude of parasites, often simultaneously, and must allocate resources to defenses and vegetative growth to ensure reproductive success. A number of laboratory studies assessing the role of defense responses dependent of salicylic acid, jasmonic acid, and ethylene have demonstrated conflictive and additive interactions among these pathways depending on the parasite(s) involved (see reviews of Bostock, 1999; Dempsey et al., 1999; Genoud and Métraux, 1999; Kessler and Baldwin, 2002; McDowell and Dangl, 2000; Walling, 2000). Interactions among defense pathways have also been studied in the field, primarily between foliar pathogens and herbivores.

Thaler et al. (1999) in a field experiment treated tomato plants with BTH, jasmonic acid, or both, in addition to nontreated controls, and then challenged plants with either a bacterial pathogen or an insect herbivore. Tomato plants treated with BTH exhibited reduced symptoms of foliar speck (caused by *Pseudomonas syringae* pv. *tomato*) compared to nontreated plants, but were also more susceptible to herbivore damage caused by larvae of the beet armyworm [*Spodoptera exigua* (Hübner)]. Conversely, tomato plants treated with jasmonic acid were more resistant to herbivore damage, but did not differ from the nontreated control in susceptibility to foliar speck. An analysis of several biochemical defenses found that the increased susceptibility to herbivory in BTH-treated plants was associated with the suppression of polyphenol oxidase activity and mRNA encoding the proeinase inhibitor PIN II, which are key antiherbivore responses. An increase in mRNA encoding the pathogenesis-related protein P4 was also observed in BTH-treated plants in association with suppression of symptoms of bacterial speck, but suppressed in plants treated with jasmonic acid. Interestingly, plants treated simultaneously with BTH and jasmonic acid exhibited symptoms of bacterial speck and herbivore damage intermediate of the plants treated individually with either BTH or jasmonic acid, and were associated with intermediate levels of polyphenol oxidase activity and P4 mRNA. Thaler (1999) further demonstrated that defenses induced by foliar applications of jasmonic acid on tomatoes effectively controlled insect herbivory in field trials over a 4 yr period.

Hatcher et al. (1994) and Hatcher and Paul (2000) found that undamaged leaves of the plant *Rumex obtusifolius* L. that received damage from the beetle *Gastrophysa viridula* (De Geer) were more resistant to several fungal pathogens than were plants undamaged by herbivory. Interestingly, induced resistance was not found to be a factor among the fungal pathogens studied, which included a biotrophic Basidiomycete [*Uromyces rumicis* (Schumach.) G. Winter], a hemibiotrophic Ascomycete [*Venturia rumicis* (Desm.) G. Winter], and a necrotrophic Ascomycete [*Ramularia rubella* (Bonord.) Nannf.] (Hatcher and Paul, 2000). It's quite possible that these fungal pathogens evolved strategies to avoid or neutralize the defenses of *R. obtusifolius* during pathogenesis, explaining why host defenses elicited by insect herbivory were effective against these fungi.

Genetic Variability in Plants

Results from the various field trials considered in this review demonstrate the potential of biological and chemical elicitors to control a wide variety of diseases. The crops utilized in these studies were modern cultivars selected in conventional agricultural settings, and they

generally exhibited only minor, often statistically insignificant, reductions in yield in association with chemical elicitors of SAR when compared with standard pesticide treatments. Since induced resistance is a biological response, it should be amenable to breeding strategies to minimize its negative impact on agronomic traits, and perhaps even improve its overall effectiveness. However, it will be necessary to determine the amount and types of variability that may exist among domesticated crops and their wild relatives.

Currently, it is unclear what variability exists within plant populations for the induction, maintenance, and overall robustness (in terms of the number and effectiveness of individual biochemical components involved) of induced resistance responses. A great deal is known from *A. thaliana* about the role of the phytohormones ethylene, jasmonic acid, and salicylic acid in induced resistance responses, mostly because of mutational screens that identified genes that when disrupted prevent the plant from producing or detecting such phytochemicals. However, little data are available assessing the allelic variability of these regulatory genes among the various ecotypes of *A. thaliana*, let alone what variability exists among the *A. thaliana* ecotypes for induced resistance responses, except in a limited case involving a single ISR-eliciting PGPR strain (van Wees et al., 1997).

The induction of ISR relies on specific plant-PGPR strain interactions (Leeman et al., 1995a, 1995b; Ton et al., 1999, 2001; van Wees et al., 1997). In *A. thaliana*, the PGPR strain *Pseudomonas fluorescens* WCS417r was capable of eliciting an ISR response on most ecotypes, but not on ecotypes RLD and Wassilewskija (van Wees et al., 1997; Ton et al., 1999). Subsequent genetic studies led to the identification of the *ISR1* locus that not only controls the ability to respond to *P. fluorescens* WCS417r, but also basal resistance to *P. syringae* pv. *tomato*. After further research, the *ISR1* locus was found to play a role in the ethylene signaling pathway of *A. thaliana*, therefore ecotypes RLD and Wassilewskija carried a recessive trait that affected ISR by disrupting ethylene signaling while leaving SAR intact (Ton et al., 2001). This demonstrates that among *A. thaliana* ecotypes, allelic variability exists in regulatory genes that influence ISR pathways.

What allelic variability exists for regulatory genes of SAR? Nothing has been found that compares with the previous example of the *ISR1* locus, since few studies have examined genotypic effects on SAR. Interestingly, some limited studies evaluating SAR among cultivars that differed in their level of resistance toward a particular pathogen found improved efficacy in partially resistant cultivars over susceptible cultivars, whereas others reported just the opposite or few differences among cultivars (Dann et al., 1998; Hijwegen and Verhaar, 1994; Romero et al., 2001; Stadnik and Buchenauer, 1999b). However, there is evidence from an ecological study using families of wild radish (*Raphanus raphanistrum* L.) derived from full- or half-sib matings that significant differences existed among families for fitness costs following induction, as measured by differences among various reproductive traits, suggesting that among the families there was either genetic variability for the induction of resistance or genetic variability for the physiological costs associated with the induced resistance response, which implies heritability (Agrawal et al., 1999).

Effects on Parasite Populations

Unlike race-specific (vertical) resistance or pesticides, induced resistance does not appear to apply selective pressure to pathogen or parasite populations on the basis of any single genetic determinant or specific mode of action, but rather is quantitative because of the cumulative effects of numerous plant defense mechanisms (Sticher et al., 1997; van Loon et al., 1998). However, because of its similarity to horizontal resistance, the effectiveness of induced resistance has the potential to "erode" over time as the pathogen or parasite population evolves (McDonald and Linde, 2002). There is a need for research evaluating the effects of SAR or ISR on the composition of pathogen or parasite populations.

In one such study, Bousset and Pons-Kühnemann (2003) constructed a population of *Blumeria graminis* (DC.) E. O. Speer = *Erysiphe graminis* DC. f. sp. *hordei* Em. Marchal (causes powdery mildew on barley) derived from crosses among 30 isolates collected from barley fields treated with either BTH or the fungicide ethirimol (5-butyl-2-ethylamino-6-methylpyrimidin-4-ol). The composition of the population, on the basis of the diversity of virulence pathotypes and sensitivity to the fungicide ethirimol and triadimenol [(1RS,2RS;1RS,2SR)-1-(4-chlorophenoxy)-3,3-dimethyl-1-(1*H*-1,2,4-triazol-1-yl)butan-2-ol], was monitored over several generations on barley plants treated with ethirimol, BTH or both, in addition to nontreated plants. On the basis of sensitivity to ethirimol and triadimenol, and the diversity of virulence pathotypes, the use of BTH alone did not affect the composition of the *B. graminis* population relative to the nontreated control beyond that attributable to genetic drift. As expected, the composition of the *B. graminis* population was influenced by the fungicide ethirimol, as detected by an increase in the number of isolates tolerant to the fungicide and a decrease in the diversity of virulence pathotypes. However, the combined use of ethirimol and BTH had an even larger affect on the composition of the pathogen population than ethirimol alone. This suggests that BTH alone may not have a direct effect on the genetic composition of a pathogen population, but may exert additional selective pressure either through SAR or synergy between BTH and ethirimol chemistries (Bousset and Pons-Kühnemann, 2003).

Future Directions

Elicitors of SAR and ISR could potentially revolutionize pest management in conventional agriculture. BTH, in particular, effectively and consistently reduced diseases caused by a broad spectrum of pathogens across a diverse range of crops and plant taxa. However, the efficacy of SAR induced by BTH depended on a number of variables, such as the dose and frequency of BTH application, host genotype and in one case the growth

stage of the plant. Data exist supporting heritability in the induction of plant defenses and physiological costs incurred from maintaining plant defenses, opening the possibility of breeding plants with improved inducible resistance responses or minimized fitness costs (Agrawal et al., 1999). Can the same types of genetic variability for induced resistance responses be found in domesticated plants, and applied during breeding?

The efficacy of SAR and ISR also depended on the pathogen, and some pathogens did not respond to elicitors of either. For example, SAR induced by BTH was ineffective against Fusarium wilt of cucumber (*Fusarium oxysporum* Schlechtend.:Fr. f. sp. *cucumerinum* J.H. Owen) (Ishii et al., 1999). Preinoculation of bean plants with *C. lindemuthianum* or foliar applications of INA was ineffective against two root pathogens, *Fusarium solan*i (Mart.) Sacc. f. sp. *phaseoli* (Burkholder) W. C. Snyder & H. N. Hans. or pathogenic *Rhizoctonia* spp. (Dann and Deverall, 1995). Several elicitors of SAR and ISR also failed to reduce symptoms of late leaf spot [caused by *Phaeoisariopsis personata* (Berk. & M.A. Curtis) Arx = *Cercosporidium personatum* (Berk. & M.A. Curtis) Deighton] of peanut (*Arachis hypogaea* L.) and in some cases exacerbated symptoms (Zhang et al., 2001). It is conceivable that in the previous examples disease was unaffected because the pathogen was able to thwart host defenses or because the plant lacked effective defenses or lacked the capacity to initiate defenses against the pathogen.

In many cases, the SAR activity induced by BTH or INA was as effective at controlling specific diseases as a standardized pesticide control, especially for diseases caused by phytopathogenic bacteria. Still, some researchers found it necessary either to alternate or to combine BTH with a pesticide(s) to reduce disease to a level comparable to the standardized pesticide control, or to control other diseases not influenced by SAR.

For example, while Louws et al. (2001) found that BTH alone sufficiently controlled bacterial spot and speck of tomato, in some trials it was still necessary to add specific fungicides to control late and early blight. While one would predict that the combined use of SAR elicitors with standard pesticides should extend the effectiveness of the pesticides, experiments conducted by Bousset and Pons-Kühnemann (2003) suggest just the opposite; SAR may actually intensify the selective pressure exerted by certain pesticides on pathogen and pest populations. Clearly this is a concern, especially when neither SAR nor ISR render the plant impervious to the pathogen, but rather seem to increase the plants overall tolerance.

Neither SAR nor ISR is a stand-alone method for pest control, but merely another tool that will need to be further integrated into pest management systems. Each crop and disease will be unique in some regard. The effective use of either SAR or ISR will also heavily depend on disease forecasting and the ability to activate plant defenses at critical developmental periods when the plant is most susceptible, since SAR and ISR are ineffective once the pathogen has established itself. There is also the risk of rendering the plant susceptible to other pathogens or insect herbivores, so it will be necessary to continuously monitor various pests in the field. On the other hand, there is evidence from the *A. thaliana* model system that the simultaneous induction of ISR and SAR may give additive protection against pathogens, but this approach has not been further investigated in crops or in the field (van Wees et al., 2000).

Newer and more effective elicitors of SAR and ISR will surely be developed, perhaps in part as the result of our growing understanding of the underlying mechanisms of these pathways within the plant. In the future, it may be possible to apply elicitor cocktails that induce a balance of defenses regulated by salicylic acid, jasmonic acid, ethylene, and other undefined regulators, against specific pests or complexes of threats. However, this future will require a shift in conventional agriculture away from the total reliance on pesticides to solve pest problems, and a concerted effort to manage pests as opposed to eliminating them.

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