OPTIMIZING INDUCED RESISTANCE (IR)

Jacob E. Sirois

Thesis submitted to the faculty of the Virginia Polytechnic Institute and State University
in partial fulfillment of the requirements for the degree of

Master of Science
In
Agricultural and Life Sciences

Dr. Laurie J. Fox
Dr. Roger Harris
Dr. Joshua Freeman

Spring 2011
Blacksburg, VA

Keywords: SAR, ISR, Induced Resistance, Priming

OPTIMIZING INDUCED RESISTANCE (IR)

Jacob E. Sirois

ABSTRACT

The use of induced systemic resistance and systemic acquired resistance as a strategy for pest management is becoming more common and commercial products are increasingly available to the producer. Despite tremendous advances in the body of knowledge surrounding this method of crop protection, a complete picture of plant immunity is elusive. Despite the missing edges of the map, practical lessons can be drawn from the existing body of work to create a tentative model for optimizing the performance of elicitors of Induced Resistance (IR). The goal of this work is to develop a usable framework that will help local producers and extension agents alike to use the emerging IR products with optimal results, and provide a starting point for on-farm screening. First, a map of induction logic, Figure 1, will guide the user to the likely induction pathway depending on the nature of the stressor. Then, Table 1 and Table 2 should be helpful in verifying that the induction pathway chosen can be used in the context of the specific plant-pathosystem of interest or at least with another pathogen that employs a similar strategy. Tables 1 and 2 will also be helpful in determining the appropriate dosages, active ingredients to look for, and the expected efficacy if available. Finally, the general considerations and Figure 1 should be useful in integrating IR into IPM by pointing out potential negative/positive interactions, costs, tradeoffs and contraindications. The complex nature of these processes necessitates careful research in each product-plant pathosystem system—evolutionary divergence tends to create some surprising outcomes. Also, researchers and producers should be wary of treating IR activators like conventional products. Important differences like yield costs and a lack of direct antimicrobial action creates unique challenges. Yield costs can be minimized by combining the following approaches: using agents or concentrations that prime IR rather than activate direct defenses, using IR when pest pressures are relatively high or at least forecasted to be, activating IR during high light conditions, and carefully keeping abiotic stresses to a minimum. Furthermore, variability can be reduced by using multiple strains of Plant Growth Promoting Rhizobacteria (PGPR) and Arbuscular Mycorrhizal Fungi (AMF), avoiding frequent foliar sprays without an antimicrobial agent in the mix, and tailoring the treatment prescription to the specific plant-pathosystem (i.e. evolutionary divergence).

HOW TO USE THIS DOCUMENT

I. Use the Induction Logic Map (Figure 1.) to determine which induction pathway is appropriate for your crop protection issue.

II. Verify that the selected pathway can be used successfully for control in the specific plant-pathosystem of interest by using Table 1. If the exact plant/pathogen or plant/herbivore combination is not available in the table, select a pathogen or herbivore with a similar life cycle or feeding strategy or conduct a review of the literature.

III. Use Table 1 and 2 to select the appropriate active ingredients, dosages and application method.

IV. Finally, review Figure 1 and the general considerations to weigh the possible benefits against possible costs, tradeoffs and interactions.

TABLE OF CONTENTS

GENERAL CONSIDERATIONS FOR THE IMPLEMENTATION OF INDUCED RESISTANCE

I. Induced Resistance (IR) can be over-simplified to contain two general pathways: Salicylic acid (SA)-mediated Systemic Acquired Resistance (SAR) and Jasmonic acid/Ethylene (JA/ET)-mediated Induced Systemic Resistance (ISR). SAR can be very generally thought of as being effective against biotrophic pathogens (those that can invade living plant cells), whereas ISR can be thought of as being effective against necrotrophic pathogens (those that kill cells to derive their nutrition) and insects. Both ISR and SAR can generally provide control for hemibiotrophic pathogens (those that have both a biotrophic and necrotrophic phase). There is also some negative cross-talk: simultaneous activation of both pathways will result in inhibition of ISR in favor of SAR, but the plant can change its mind if later on (>2 days) an ISR elicitor is introduced. 'Activators' or 'elicitors' of IR may be specific like acibenzolar-s-methyl (BTH) and activate only one pathway, SAR in this case, or an activator may be more of a general and prime both SAR and ISR as in the case of Plant Growth Promoting Rhizobacteria (PGPR). Selection of products should be based upon the pathway activated and the lifestyle of the pathogen (i.e. biotrophic, necrotrophic, or hemibiotrophic). (See page 40 for more information)

II. Although the "limiting effect of resistance on yield" has not been rigorously studied, losses of up to7% have been reported due to increased respiration during successful resistance expression (Walters and Heil 2007). Thus, the producers should be reasonably sure that the pest left uncontrolled will cause at least 10% yield loss by harvest before induction of SAR is considered to be an economically viable option for disease management. (See page 25 for more information)

III. It is important to note that induction of Plant Growth Promoting Rhizobacteria (PGPR) or Arbuscular Mycorrhizal Fungi (AMF) mediated by Induced Systemic Resistance (ISR), Beta-aminobutyric acid induced resistance (BABA-IR), Vitamin B1 (Thiamine) priming of Systemic Acquired Resistance (SAR), and Hexanoic acid induced resistance (Hx-IR) only result in priming of defenses, so they do not incur as great a yield cost because direct defenses are only initiated when the primed plants are challenged by a pest (Flors et al. 2008; Vicedo et al. 2009). It has been shown that priming for defense provides enhanced resistance while it does not significantly reduce plant growth or seed set, and thus it can be used prophylactic manner (Van Hulten et al. 2006). (See page 27 for more information). On the other hand, acibenzolar-s-methyl (BTH, Actigard, Bion) concentrations greater than 210 mg/L can induce direct defenses without pathogen attack, and consequently causes large yield reductions when pest pressures are low.

IV. Furthermore, it is possible to minimize yield lowering costs somewhat with the correct management. Plants that are abiotically stressed are likely to suffer greater growth and yield consequences from induction or to express impaired resistance when induced. Thus, poor plant nutrition or water stress should be considered a contraindication for the use of induction for disease management—correct these abiotic issues prior to attempting induced resistance for optimal results.

V. It is well known that Arbuscular Mycorrhizal Fungi (AMF) symbiosis can substantially enhance water and nutrient uptake in plants under stressful conditions. Interestingly, this

phenomenon seems to be particularly relevant when host resistance is induced, where the effectiveness depends on the ability of the plant to balance the energy costs related to defense. Thus, it is recommended that plants be inoculated with AMF and the agroecosystem should be manipulated to encourage symbiosis with AMF (Kempel et al. 2010). This might mean considering the following: reduced tillage systems, addition of organic soil amendments, avoidance of conventional soil applied chemicals, avoiding non-hosts in the families Brassicaceae and Chenopodiaceae and avoiding excessive fertilization (Brady 2008).

VI. Next, when a severe chewing insect infestation and disease epidemic are present simultaneously, one should consider using IPM tactics to reduce the insect pest prior to or concomitantly with activation of IR. In fact, with a severe chewing insect infestation, it is likely that plants are already induced with broad-spectrum cross-resistance, so IR activators might not be the best option until insect levels are brought to lower levels (Baldwin 1998). Using high concentrations of BABA or BTH, which both induce Salicylic acid (SA)-mediated Systemic Acquired Resistance (SAR) ,would cause enhance susceptibility to the insect pest, so it is critical to use IPM tactics in conjunction with IR agents. Similarly, inducing Salicylic acid (SA)-mediated Systemic Acquired Resistance (SAR) will generally enhance susceptibility to necrotrophic pathogens. Therefore, it is important to use IPM tactics to control necrotroph levels while inducing SAR. The optimal results will be achieved when the chewing infestation is somewhat intermediate. In this case, priming or direct induction of defenses are acceptable. On the other hand, if the population of chewing insects is rather low, induced resistance will be more costly than it is worth in protection to the majority of plants. Biological control is optimal at these population sizes, so other IPM methods should be used to keep the chewing herbivore populations as low as possible. Avoid applying SAR inducing agents as this could cause enhanced susceptibility to the herbivore. In addition, GA should be avoided but Auxins or cytokinins might be beneficial

VII. On the other hand, if the insect infestation is of stealthy feeders that do not cause a large amount of wounding like aphids, then the situation becomes a bit more complex. Insects that utilize stylets to feed directly from phloem do not result in detectable increases in the levels of SA, JA or ET, which means induction of resistance does not occur. However, prior infection with an avirulent host did cause reductions in aphid feeding and reproduction, and there is evidence that JA can also achieve control (De Vos et al. 2007). Thus, if the insect infestation is of Phloem Feeders or Cell-Content Feeders (mites, aphids, etc.), then induction of either the Jasmonic acid (JA)-mediated Induced Systemic Resistance (ISR) or the Salicylic acid (SA)-mediated Systemic Acquired Resistance (SAR) should be effective for control, with some variability. Some phloem feeding insects like the Silverleaf Whitefly may actually use the plants defenses against it by purposely causing SA accumulation in order to inhibit JA production. Therefore, if the insect infestation is of *Bemisia tabaci*, then induction of SAR might not be effective, but induction of ISR by potentiating the JA/ET pathway will provide adequate control (Valenzuela-Soto et al. 2010). (See page 24 for more information)

VIII. Where Tissue Chewing insects are the concern, note that tissue maceration is generally capable of generating resistance to further feeding in addition to broad-spectrum resistance to pathogens, although there is some lag time involved between the onset of feeding and induction

of resistance. Thus, use IPM tactics other than IR for severe infestations, and prime the JA-mediated ISR pathway when insect levels are low or intermediate. (See Figure 1 on page 20 for more information)

IX. By extension, pruning is not contraindicated before, during or after induction of resistance; in fact, it can be used to temporarily induce resistance (Francia et al. 2007). This is an interesting finding in terms of integrated pest management because repeated reduction of inoculum by pruning and sanitation can not only delay an epidemic but decrease the per capita growth rate, slowing the spread of the disease (Baudoin 2010).

X. Also, SAR induction does not seem to be as effective against root-knot nematodes of the genus *Meloidogyne* root-lesion nematodes of the genus *Pratylenchus*. In two independent studies, induction was only able to affect a reduction of 23-25% (San Alfarez et al. 2008; Collins et al. 2006). These pests appear to alter the SA pathway, silencing some pathogenesis-related genes (Sanz-Alferez et al. 2008). Thus, if the pathogen is found to be a plant pathogenic nematode, then the producer should use the JA pathway, which has been shown to be much more effective (Elsen, 2008; Siddiqui and Shaukat 2003; Siddiqui et al. 2009). Note that Rhizobacteria and Arbuscular Mycorrhizal Fungi (AMF) are excellent for this purpose, and can be applied as seed treatments, soil amendments, soil drenches, transplant root dips, or foliar sprays.

XI. If one wishes to benefit from the growth promotion from Plant Growth Promoting Rhizobacter (PGPR), root colonization is required. The same is true to the benefits associated with Arbuscular Mycorrhizal Fungi (AMF). For this reason, conventional chemical soil drenches are not compatible with products, and should be avoid if either PGPR or AMF are employed.

XII. The Gibberellin (GA) phytohormones can actually enhance the SA pathway, while negatively impacting the JA pathway. Therefore, the induction of SAR can be integrated with GA application, whereas GA should be avoided when inducing ISR. Furthermore, the auxins and cytokinins have been shown to have the opposite effect of GA: auxin application enhances the JA pathway, while inhibiting the SA pathway. Therefore, auxins and cytokinins can be applied as a part of integrated pest management with ISR induction, but should be avoid when inducing SAR. It should be noted that both GA and Auxins have been approved for use in organic systems by OMRI. (See page 26 for more information)

XIII. Several studies have shown that some of the variability experienced with protection ISR through inoculation with Plant Growth Promoting Rhizobacteria (PGPR) can be overcome by using multiple strains (Choudhary and Johri 2009). Therefore, it is recommended that multiple strains be mixed together if at all possible when attempting to induce ISR via PGPR inoculation.

XV. If at all possible, attempt to induce direct defenses on the morning of sunny days, as ROS generated by light driven pathways is less energetically expensive than ROS generated in the dark (Bolton 2009). Predictions of extended cloud cover might factor into the producers cost-benefit analysis when deciding on a treatment option.

XVI. Also, consider that high humidity can, to a limited extent, slow the induction of resistance. The ideal environment for induction is a bright, low-humidity environment—avoid sprinkler irrigation following induction and avoid induction on high humidity days, wherever possible.

XVII. Due to the fact that most IR activators do not have direct antimicrobial properties, the action of repeated foliar sprays using water as a solvent can actually cause increased disease pressure. Therefore, where possible, mix IR activators with an antimicrobial agent like an OMRI approved Neem oil, Sesame oil, Tee Tree oil or a copper compound. Another option is to use a drench wherever possible as opposed to foliar sprays.

XIV. Finally, due to the inherent variability in post-penetration resistance signaling, consult Table 1 or review the literature for the specific plant pathosystem involved in order to find the appropriate products, dosages and application method. If the specific pathogen is not found in the table, select one with a similar life strategy. Furthermore, it is imperative that producers select the products based upon the mode of action of the active ingredient. For assistance with this, consult Table 2.

TABLE 1. INTERACTIONS BY SPECIFIC PLANT-PATHOSYSTEM

Host	Pest	Methods	Active Ingredient	Comments

Mouse-Ear Cress *Arabidopsis thaliana* **Brassicaceae**	Aphid *Myzus persica* **Stealthy Herbivore**; Cabbage White Butterfly *Pieris rapae* **Chewing Herbivore**	Inoculation with avirulent pathogen, feeding by herbiv-orous insects both stealthy and chewing	Pseudomonas fluorescens WCS417	First, it is reported that tissue maceration by the chewing insect Cabbage White Butterfly results increased resistance to future feeding and in significant cross protection from Turnip Crinkle Virus (TCV), *Pseudomonas syringae* pv. tomato, and *Xanthomonas campestris*. Next, it was shown that 3 days of stylet feeding by 40 stealthy insects like aphids does not significantly increase SA, JA or ET, despite major gene reprogramming. Aphid feeding does not result in resistance to future feeding in Arabidosis (Brassicaceae) or Potato (Solanaceae). Furthermore, *Pseudomonas fluorescens* WCS417, an ISR priming PGPR, was not effective against Aphids. On the other hand, SAR triggered by an avirulent pathogen was effective in increasing aphid resistance. Thus, the plant cannot effectively recognize stealthy feeders, so only induction of direct defenses will result in resistance—priming ISR is only effective when the plant can subsequently recognize an attacker. (DeVos et al. 2007)
	P. syringae **Hemi-biotroph**	Exogenous elicitor, foliar spray	Vitamin B1 (thiamine)	A small dosage of between 5 and 50mM thiamine has a priming effect that gives protection from bacterial infection (Ahn et al. 2007)

	Pieris rapae; *Spodoptera exigua* **Tissue Chewing Insects**	PGPR inoculation or avirulent pathogen inoculation	PGPR or avirulent pathogen	PGPR interaction primed defenses and effectively controlled the generalist S. exigua, but not the specialist *P. rapae* (Van Oosten et al. 2008)
	Gray Mold *Botrytis cinerea* **Necrotroph**; Bacterial Speck *Pseudo-monas syringae* **Biotrophic Hemi-biotroph**	Foliar spray with yeast suspension to run off with between 4 $\times 10^9$ and 9 $\times 10^9$ cells/mL.	Yeast suspension	Yeast was shown to provide control of P. syringae via SA-mediated responses (SAR), and was shown to generate enhance production of camalexin (CA). Strangely, protection from *Botrytis cinerea* was also provided, but by an unknown mechanism. Bakers Yeast contains about 20 billion yeast cells per gram, which would mean that protection could be achieved here at 250 mg/mL (Raacke et al. 2006)
	White Mold *Sclerotinia sclerotiorum* **Necrotroph (?)**	Foliar Spray with 300 mg/L BTH (Actigard)	BTH	*S. sclerotiorum* produces oxalate, which is essential to its virulence because it reduces the production of reactive oxygen species (ROS). In other words, the wild type fungus is able to avoid activating effective SA-mediated defenses by countering the 'oxidative burst' that initiates HR. Fortunately, activating direct defense (SAR) with 300 mg/L BTH gives protection (Guo, 2007). Although generalizations are common concerning the life strategies of pathogens, in reality there are very few true necrotrophs. In fact, the true nectrotrophs are probably confined to the genera *Penicilium, Sclerotinia*, and *Sclerotium* (Parbery 1996). It is possible that S. sclerotiorum is not a

				true necrotrophs, but it is also possible that this is just another example of divergence in a complex immunity network.
	Gray Mold *Botrytis cinerea* **Necrotroph**; Early Blight A*lternaria alternate* **Necrotroph**		GLV	GLV's induce a wide range of defenses (Kishimoto et al. 2005)
Banana *Musa spp.* **Musaceae**	Burrowing Nematode *Radopholus similis* **Biotroph**; Root-Lesion Nematode *Pratylenchus coffeae* **Biotroph**	300 grams of myco-rrhizal inoculum added to soil mix	Arbuscular Mycorrhizal Fungi (AMF) *Glomus intraradices*	The observed frequency of colonization was 100% and the intensity of colonization was 13-24%. AMF colonization was not affected by infection by nematodes. *Radopholus similis* populations were reduced by 72% and Pratylenchus coffeae populations were reduced by 84%. AMF colonization did cause a modest reduction in growth, but this could have been due to high P levels, which cause *G. intraradices* C-demand to increase. Split-root set-up establishes mechanism as induced systemic resistance. (Elsen et al. 2008)
Barley	Powdery Mildew *Blumeria graminis* **Biotroph**	Exogenous elicitor	BTH, CHT	BHT more effective than CHT. 76% reduction of severity (Faoro et al. 2008)
Bean *Phaseolus vulgaris* **Fabaceae**	Bean Rust *Uromyces appendicul-atus* **Biotroph**	Seed treatment with live rhizo-bacteria.	*Pseudomonas fluorescens* WM35, *Pseudomonas aureofaciens*	In the greenhouse, soil application of SA or seed treatment with *Pseudomonas aeruginosa* 7NSK2 and SA44 significantly reduced disease.

			WM09, *Pseudomonas putida* WM06, *Pseudomonas aeruginosa* 7NSK2 and SA44.	In the field, WM35, WM09 and WM06 races all gave excellent control in the field when applied as seed treatments. ISR induction lasted greater than 30 days. The physical separation between the pathogen and the rhizobacteria means this phenomenon can only be attributed to ISR (Abeysinghe 2009).
Canola, *Brassica napus* **Brassicaceae**	Branched Broom Rape, *Orobanche ramose* **Biotroph**	Exogenous elicitor: foliar spray or soil drench	ASM, Potassium Phosphonate	ASM reduced severity by 70% and prevented crop losses, but Potassium Phosphonate had no effect (Veronesi et al. 2009)
Cantaloupe *Cucumis melo* **Cucurbit-aceae**	*Fusarium spp.* **Necro-trophic Hemi-biotroph;** *Alternaria spp.* **Necrotroph**; *Rhizopus spp.* **Necrotroph**	Exogenous elicitor	BTH, INA, BABA	All treatments reduced fruit rot and did not reduce yields. A single spray 2 weeks before harvest gave same control as multiple sprays (Bokshi et al. 2006).
Cotton *Gossypium hirsutum* **Malvaceae**	Tobacco Budworm *Heliothis virescens,* **Chewing Insect**	Exogenous elicitor	BTH	Induction of SAR did not affect health nor that of a natural enemy (Plymale et al. 2007)
Cucumber *Cucumis sativus* **Cucurbit-aceae**	Phytophthora Root and Crown Rot *Phytoph-thora capsici* **Biotrophic Hemibio-troph**	Inoculation with beneficial fungus, compost amend-ments and exogenous elicitors	*Trichoderma hamatum* 382, Cow manure compost, BTH, mefenoxam	Compost provided good control not significantly different than BTH drench. Compost inoculated with *Trichoderma hamatum* also gave significant control of Phytophthora leaf blight. This result again was similar to that of a BTH drench (Khan et al. 2004).

	Two-Spotted Spider Mite *Tetranychus urticae* **Cell-Content Feeder**	Foliar Spray of 1% Milsana by volume or seed inoculation with *P. fluorescens* at $3x10^9$ bacterial cells/mL	Extract of *Reynoutria sachalinensis* (1% Milsana) or *Pseudomonas fluorescens*	Application of either Milsana or bacterialization of seeds reduced mite densities 2-fold, but mite densities were a bit higher in Milsana + bacteria plants. Mites laid 35% fewer eggs on Milsana treated plants, 26% fewer eggs on Milsana + bacteria plants, and 17.5% fewer eggs on bacterialized plants. This might be a demonstration of negative cross-talk between SAR and ISR inducers. (Tomczyk 2006)
Grape *Vitis vinifera* **Vitaceae**	Gray Mold *Botrytis cinerea* **Necrotroph**	Inoculation with live or crude extract of non-pathogenic rhizo-bacteria	*Pseudomonas fluorescens* CHA0, *P. aeruginosa* 7NSK2, *P. fluorescens* WCS417 (Pch-deficient), *P. putida* WCS358 (Pch-and SA-deficient) and *P. fluorescens* Q2-87 (a DAPG producer)	All of the Pseudomonas strains shown to the left are capable of inducing resistance to equal extent in grape. Also, induction was equivalent for live and crude extracts of the bacteria, illustrating the involvement of elicitors in the induction of ISR (Verhagen et al. 2010).
Lettuce *Lactuca sativa* **Asteraceae**	*Downy Mildew* **Biotroph**	Exogenous elicitor: spray	Phytogard (Active ingredient is phosphoric acid, K2HPO3) and BABA (beta-amino butyric acid)	It was found that BABA (beta-amino butyric acid) in a 10mM solution used as a foliar spray gives complete systemic protection and has curative effects for up to 15 days. Also, 40.6 ppm of phosphoric acid K2HPO3 gave complete protection for 15 days. No products are registered from BABA, but it is a compound that can be created naturally. Phosphoric acid is a synthetic product and

				cannot be used in organic production. (Pajot et al. 2001)
Lima Bean	Spider Mite *Tetranychus urticae* **Cell-Content Feeder**	Exposure to volatiles	Volatiles	Mite infested plant produce more volatiles. There is a time lag in plant defense (>50 female mites), but the time to defense induction can be reduced by pre-treatment with related volatiles, which probably prime the JA-pathway (Choh et al. 2004)
Peach *Prunus persica* **Rosaceae**	Blue Mold *Penicillium expansum*, **Necrotroph**	Exogenous elicitor: fruit dip post-harvest	BTH	Post-harvest fruit dipped for 5 min in 200mg/dm3 BTH yielded significant control of Blue Mold without altering fruit quality (Liu et al. 2005).
Pea *Pisum sativum* **Fabaceae**	Bean Broomrape *Orobanche crenata* **Biotroph**	Exogenous elicitor:	BTH	0.6-1.0mM BTH foliar applied gives control of this parasite (Perez-de-Luque et al. 2004).
Pepper *Capsicum annum* **Solanaceae**	Anthracnose *Colleto-trichum coccodes* **Hemi-biotroph**	Foliar Spray or soil drench. 1mg/mL BABA, 1 mg/ml AABA, and 1 mg/ml GABA	BABA, AABA, and GABA	1 mg/ml BABA gives 85%-100% control. Protection remained for over 15 days. 1 g/ml of AABA gave 70% protection, but AABA was shown to have some direct antimicrobial properties at this concentration (29%). GABA provides some protection (35%), which is evidence of its ability to protect against pathogens with necrotrophic phases, either due to its ability to induce ET biosynthesis or some other mechanism. (Hong et al. 1999)
	Verticillium Wilt *Verticillium* **Biotrophic Hemi-biotroph**	Incorporation of *Asco-phyllum* seaweed extract as soil amend-ment	Seaweed extract (*Ascophyllum spp.*)	The incidence and severity of Verticillium wilt was reduced by incorporating seaweed extract into the soil. Although, it was subsequently shown that foliar spray is more effective. (Garcia-Mina et al. 2004)

Potato *Solanum tuberosum* **Solanaceae**	Root Lesion Nematode *Pratylenchus spp.* **Biotroph**; Root Knot Nematode *Meloidogyne spp* **Biotroph**	Exogenous elicitor	BTH, Harpin	Both Harpin and BTH reduced nematode infection by 23%, but reduced number of culled potatoes by 26% (Collins et al. 2006)
Strawberry *Fragaria chiloensis* **Rosaceae**	White Mold *Botrytis cinerea* **Necrotroph**	Exogenous elicitor	ASM (Bion, Novartis Crop Protection)	A single spray at anthesis resulted in 15-20% increase in storage life, which was equivalent to weekly sprays from 4 weeks after planting to 9 weeks through flowering. Also, the degree of suppression was similar for concentrations from 0.25mg/ml through 2.0mg/ml. No costs or phytoxicity were observed relative to control (Terry and Joyce 2000)
Sugarcane *Saccharum spp.* **Poaceae**	Red Rot *Colleto-trichum falcatum* **Hemi-biotroph**	Exogenous elicitor	BTH	Significantly reduced disease in field conditions (Sundar et al. 2009)
Sunflower *Helianthus annuus* **Asteraceae**	Rust *Puccinia helianthi* **Biotroph**	Exogenous elicitor: foliar spray and root dip	BABA, NaSA, BTH, INA	BABA most effective and NaSA least effective. BABA at 25 mg/ml applied before or up to 48h after inoculation provide control for 8 days (Amzalek and Cohen 2007)
	Downy Mildew *Plasmopara halstedii* **Biotroph**	Exogenous elicitor: seedling dip	BTH	Seedlings immersed in 200mg/dm^3 of BTH for 12 h in the dark were protected from infection (Serrano et al. 2007).
	Rust *Puccinia helianthi* **Biotroph**	Foliar Spray. Optimal control at 0.025 mg/mL	AABA, GABA, BABA, INA, BTH, NaSA	GABA was found to be ineffective against rust, and AABA was only partially effective. BABA, INA, BTH, and NaSA gave significant control of this biotroph.

		BABA, 0.100 mg/mL BTH, 0.100 mg/ml INA, 0.200 mg/mL NaSA		(Amzalek and Cohen 2007)
Tobacco *Nicotiana spp*. **Solanaceae**	Tobacco Blue Mold *Peronospora hyoscyami*; Frog Eye Leaf Spot *Cercospora nicotianae*; *Alternaria alternata*	Exogenous elicitor: foliar spray	ASM	25-37.5 gallons per hectare or Bion 50 WG at 10 day intervals gave significantly reduced incidence and severity of these three diseases. 17 gallons per hectare did not give sufficient control for the purpose of wrap leaf cigar manufacture. General leaf quality was unaffected (Perez 405).
	Botrytis cinerea and Oidium neo-lycopersici	Foliar Spray	BTH	In this experiment, we see that chemical induction with SAR provides protection from the biotrophs *Oidium*, but has no effect on *Botrytis*. In this same experiment, tomato was protected in the opposite manner, demonstrating the variability among different plant species. (Achuo et al. 2004)
	Branched Broomrape *Orobanche ramose* **Biotroph**	Soil drench with either 2.5% *Asco-phyllum nodosum* algae extract, 1 mg/ml BTH, or 2 mg/ml *Pseudo-monas spp.*	*Pseudomonas spp.,* BTH and seaweed extract	Pseudomonas spp. Rhizobacteria colonized the roots, priming ISR and SAR concomitantly, providing 80% control. The seaweed extract reduced Broomrape by 84%. BTH was most effective at 92% compared with control. (Gonsior et al. 2004)
Tomato *Lycopersicon esculentum*	Root Knot Nematode (RKN)	Exogenous elicitor	BTH, INA	Induction of direct defenses using the SA-pathway (SAR) gave low levels of control

Solanaceae	*Meloidogyne javanica* **Biotroph**			against nematodes in this study. Reduced galls by 25% (Sanz-Alferez et al. 2008)
	Root Knot Nematode (RKN) *Meloidogyne javanica* **Biotroph**	25 mL soil drench of cell suspension per plant containing 10^9 cfu/mL	*Pseudomonas fluorescens* strains CHAO and CHA89	Strain CHAO reduced galls by 57%. Strain CHA89 was less effective. (Siddiqui and Shaukat 2003)
	Southern Root Knot Nematode *Meloidogyne incognita* **Biotroph**	Root dip/Soil Drench: 10 ml per plant of 1.5 x 10^7 cells/mL	*Pseudomonas putida*, *Bacillus subtilis*, and *Azotobacter chroococcum*	Induction of indirect defenses (priming) of JA-pathway (ISR) by Plant Growth Promoting Rhizobacteria (PGPR) gave significant control of RKN. Without manure, *P. putida* reduced the number of galls by about 56% and reduced the number of nematodes by about 50%. With manure addition, *P. putida* gave about 75% reduction in gall number and about 71% reduction in number of nematodes. This is a great example of the critical need for proper nutrition while mounting a defense against a pathogen. (Siddiqui et al. 2009)
	Bacterial Speck *Pseudo-monas syringae* **Biotrophic Hemi-biotroph**	Exogenous elicitor	BTH, PGPR	BTH reduced incidence and severity, while the action of growth stimulating rhizobacteria was more variable and less effective (Herman et al. 2004).
	Tomato Wilt *Fusarium oxysporum* **Necro-trophic Hemi-biotroph**; Late Blight	Exogenous elicitor: foliar spray.	validamycin A (VMA) or validoxyl-amine A (VAA)	At a concentration of 100 g/ml, the induction of SAR lasted approximately 64 days. Excellent control of all three tomato diseases was attained (Ishikawa et al. 2005)

	Phytoph-thora infestans **Biotrophic Hemi-biotroph**; Powdery Mildew *Oidium spp.* **Biotroph**			
	Sweet potato Whitefly *Bemisia tabaci* **Phloem Feeder**	Roots of week old transplants briefly dipped in overnight culture with 10^7cfu/ml. After transplant, the solution was diluted and added to soil as a drench.	*Bacillus subtilis* strain BEB-DN	Analysis showed 100% of plants treated were successfully colonized by *B. subtilis*. All inoculated plants showed significantly lower survival for whitefly nymphs and adults (Valenzuela-Soto et al. 2010).
	Silverleaf Whitefly, *Bemisia argentifolii* **Phloem Feeder;** Tomato Mottle Virus	Seed treatment with10^7cfu per gram of seed or as soil amendment at 10^7cfu per gram planting medium	*Bacillus amyloliquefa-ciens* 937a, *Bacillus subtilis* 937b, *Bacillus pumilus* SE34	In all cases, severity and incidence of Tomato Mottle Virus were significantly reduced. In addition, treated plants were found to have significantly fewer whitefly crawlers, nymphs, pupae (Murphy et al. 2000).
	Potato Aphid *Macro-siphum euphorbiae* **Phloem Feeder**	Foliar Spray	BTH dissolved in acetone at a rate of 28 g/l and dispersed in water to achieve a 1.2mM solution; JA	In this study, it was shown that both JA and SA pathways can be induced for control of stylet-feeding insects like Aphids. (Cooper et al. 2004)

			dissolved in acetone at 1g/l and dispersed in water to achieve a 1.5mM solution	
	Gray Mold *Botrytis cinerea* **Necrotroph**	Root dip for 48 hours	*Hexanoic Acid*	After treatment of roots with a 0.6 mM solution of hexanoic acid, plants showed significant reduction in lesion size 72 hours after inoculation with this nectrotrophic pathogen. Result was comparable to treatment with 0.5 mM BABA, whereas 0.5mM SA treatment had minimal effect. Protection from this necrotrophs was provided through ABA mediated callose formation. Plants that were not challenged by the pathogen did not develop calloses, demonstrating that Hx-IR works through priming defenses. Induction of Hx-IR primes both ABA and JA responsive genes, providing broad spectrum control (Vicedo et al. 2009)
	Gray Mold *Botrytis cinerea* **Necrotroph**		*Hexanoic Acid*	Induction of Hx-IR gives protection from Botrytis (Leyva et al., 2008).
	Gray Mold *Botrytis cinerea* **Necrotroph;** Powdery Mildew *Oidium neo-lycopersici* **Biotroph**		*BTH*	BTH has been shown to induce SAR, which is generally most effective against biotrophic pathogens. This is a great example of the divergence of these mechanisms within different species. Here, we see that induction of SAR gives resistance to Botrytis (a necrotroph) but not to Oidium

				(a biotrophs). (Achuo et al. 2004)
	Bacterial Speck *Pseudo-monas syringae* **Biotrophic Hemi-biotroph**	Root dip for 48 hours at 0.6 mM	*Hexanoic acid*	Hx-IR gave a significant reduction in symptoms 72 hours after inoculation with P. syringae. Note that *P. syringae* is considered by some to be a hemibiotroph because it has both biotrophic and necrotrophic phases within the life cycle of the disease. (Vicedo et al. 2009)
	Herbivorous insects	Wounding.	JA	In this study, wounding induced ET biosynthesis, which led to enhanced endogenous levels of JA. Also, exogenous applied JA increased ET production, which again increases JA production. ET blockers inhibited the wound response pathway, leading to the conclusion that JA and ET are required simultaneously for wound responsive gene induction in tomato. It is important to note that this is yet another case of divergence: it has been widely shown in many other plant pathosystems that ET blocks the wound responsive JA regulated genes. In fact, it seems that the same is true for other members of the family Solanaceae (O'Donnell et al. 1995; Adie et al. 2007)
	Crown Rot *Phytoph-thora capsici* **Necrotroph**; Gray Mold *Botrytis cinerea* **Necrotroph**; *Pseudomona*	Wounding of root system on both ET perceptive and never ripe tomato mutants	Not applicable.	Although ET is not essential, it improves resistance to necrotrophs while lowering resistance to biotroph/hemibiotrophs. This is due to the ability of only ET to induce specific wound-responsive genes. Regardless of ET, wounding induced

	s syringae pv. tomato **Hemi-biotroph**			broad spectrum resistance to all the pathogens studied. (Francia et al. 2007)
Sesame *Sesamum indicum* **Pedaliaceae**	Cyst Nematode *Heterodera cajani* **Biotroph**	Seeds coating: Sterile seed treated with slurry containing cell suspension at $1x10^8$ cells/ml mixed with 1% carboxy-methyl-cellulose (CMC).	*Pseudomonas aeruginosa* LPT3 and LPT5	The Plant Growth Promoting Rhizobacteria (PGPR) *P. aeruginosa* strain LPT5 was shown to reduce cysts and juveniles of *H. cajani* by about 60% and LPT3 was also effective with a 49% reduction. Interestingly, it was shown that a reduction in chemical fertilizers increased yields due to PGPR interaction. (Kumar et al. 2009)
Snap Bean *Phaseolus vulgaris* **Fabaceae**	White Mold *Sclerotinia sclerotiorum* **Necrotroph (?);** Gray Mold *Botrytis cinerea* **Necrotroph**	Foliar spray at 82 psi in 74 gallons of water with 3 D2-35 nozzles. Bakers Yeast applied at a rate of 6.2 lbs./Acre	Bakers Yeast 22 mg/L	A foliar spray trial at the University of Cornell demonstrates that Bakers Yeast can give significant control of White Mold but not Gray Mold. Yeast has generally been shown to activate the SA pathway, and SAR has been previously reported to give resistance to *S. sclerotiorum* (Guo 2007). Thus, despite close relationship between *Botrytis* and *Sclerotinia*, different control tactics must be employed. This is either an example of divergence in the resistance network or it is possible that White Mold is not purely necrotrophic. (Dillard et al. 1998)
Squash (*Cucurbita pepo*)	*Phytoph-thora capsici*	Exogenous elicitor: foliar spray	INA, BABA, ASM	All products were provided significant control of phytophthora blight in squash.

				Control was achieve when chemicals were foliar applied at 25-50 g/ml. (Kone et al. 2009)
Urad *Vigna mungo* **Fabaceae**	Urdbean Leaf Crinkle Virus	Inoculation with live bacteria: soil drench and foliar spray	*Pseudomonas fluorescens* Pf1 and *P. fluoresens* CHAO	Both of these *Pseudomonas fluorescens* races were able to significantly reduce this disease via induced systemic resistance (Karthikeyan et al. 2009)
Watermelon *Citrullus lanatus* **Cucurbit-aceae**	Gummy Stem Blight *Didymella bryoniae* **Hemi-biotroph**	Seeds soaked in bacterial suspension at 10^8 CFU/ml for 45 minutes at room temp.	*Pseudomonas aeruginosa* (strain isolated from roots of watermelon in the field)	*P. aeruginosa* has previously been found to prime the SAR pathway (Audenaert et al. 2002). This experiment, seed treatment with a *P. aeruginosa* strain isolated from the roots of watermelon in the field provided 44-62% control against the hemibiotroph *Didymella bryoniae*. This pathogen has been ambiguously referred to as a necrotroph, this study documents a biotrophic phase. (Nga et al. 2010)

BTH = acibenzolar-S-methyl ester, acts downstream of SA
CHT = Chitosan, a deacetylated chitin derivative
INA = 2,6 dichloroisonicotinic acid
ASM = Acibenzolar (S-methyl benzo[1,2,3]thiadiazole-7-carbothioate), a benzothiadiazole (Bion, Actigard, Boost 500SC)
Saccharin = 1,1-Dioxo-1,2-benzothiazol-3-one, a benzoic sulfimide
NaSA = Sodium Salicylate
ASA = Acetyl Salicylic Acid
BABA = β-aminobutyric acid
Harpin = Messenger
BIT = 1,2-benzisothiazol-3(2H)-one1,1-dioxide, SAR inducer that acts upstream of SA
PGPR = Growth Stimulating Rhizobacteria
Mefenoxam = Subdue MAXX
Candida oleophila = Yeast, Aspire©
*Green color indicates that the product has the potential to be used in organic systems
*Yellow color indicates that the method gave low levels of control
Red Color indicates that the method of control was ineffective

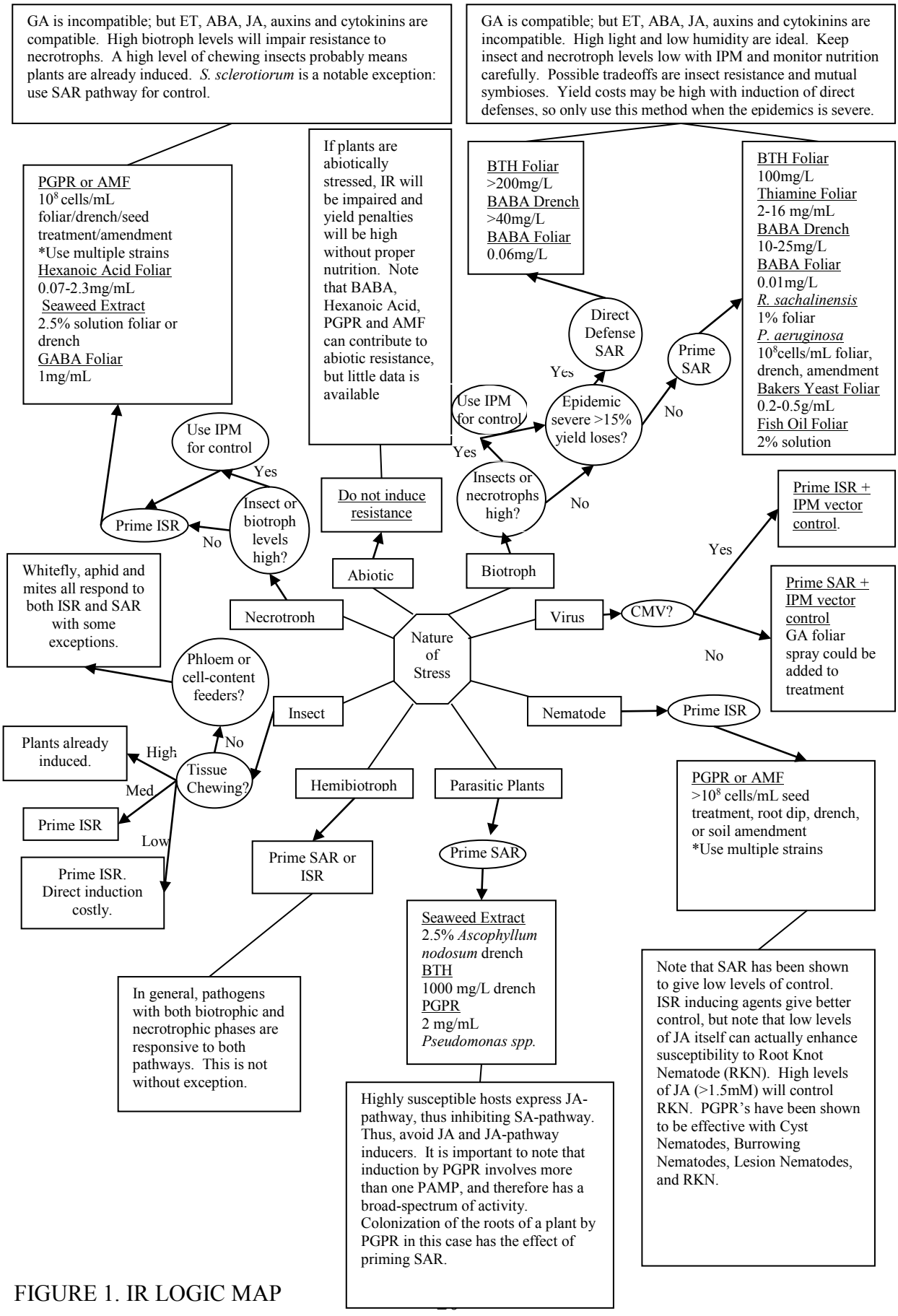

FIGURE 1. IR LOGIC MAP

TABLE 2. MODE OF ACTION BY ACTIVE INGREDIENT

Active Ingredient	Mode of Action	Description
AABA (L-2-amino-n-butanoic acid)	SAR	Chemically related to BABA, but much lower activity. Shown to be partially effective against biotrophs.
ACC (1-amino-cyclopropane-1-carboxylic acid)	ISR	Precursor to Ethylene (ET) biosynthesis, which can induce ISR when applied exogenously. (Bostock 2005)
Arbuscular Mycorrhizal Fungi (AMF)	Root colonization acts as a general elicitor, priming broad spectrum defenses	Mycorrhizal associations improve abiotic stress resistance through improved access to water and nutrients. In addition, the root colonization acts to prime general defenses, and AMF has been shown to defend against nematodes. (Elsen et al. 2008)
Ascophyllum nodosum	Mechanism uncertain.	A temperate, aquatic plant found in the Atlantic and Arctic seas. Aqueous extracts of this seaweed have been shown to provide significant control of *Botrytis cinerea* and *Alternaria radicina*. (Jayaraj et al. 2008)
Aspirin (ASA)	SAR	ASA has been shown to block JA-regulated wound response genes in tomato. Its close chemical relationship with SA allows it to induce SAR and therefore inhibit JA pathways. (Doares et al. 1995)
Aerated Compost Tea (ACT)	Variable combination of competition or production of various MAMP's that prime ISR priming, prime SAR priming, or cause induction of direct defenses.	Aerated and Nonaerated Compost Teas are essentially extracts of various composts that are either aerated or fermented. This 'home-remedy' for plants is widely used and can be thought of as a way of propagating various potential plant immunity inducers or biological control agents on the farm. NTC made from vermicompost have been shown to be a bit more predictable in their action, but overall these products are extremely variable in activity. Mode of action therefore cannot be predicted on a batch to batch basis, making them a bit risky to use. Nonetheless, they are cheap and easy to create while being acceptable in organic systems. The anaerobic process might be more consistent due to the presence of proven inducers like BABA and Vitamin B1 (yeast contain high concentrations of thiamine, which is water soluble).
BABA (Beta-amino butyric acid or Beta-aminobutanoic acid)	BABA-IR.	This inducer primes both SA and ABA related defenses. No products are currently registered. This chemical is present in butter parmesan cheese, vomit and is a product of anaerobic fermentation. When butter goes rancid, butyric acid is liberated from the glyceride by hydrolysis. Industrially, it is prepared by the fermentation of sugar or starch brought about by the addition of putrefying cheese. CaCO3 can be

		added to neutralize the acids formed. The butyric fermentation of starch can be aided through addition of *Bacillus subtilis*. Phytotoxicity has been observed in cauliflower at 20mM and above (Pajot et al. 2007)
Bacillus	ISR inducing Rhizobacteria	Many Bacillus species and specific strains have been shown to elicit ISR: *B. pumilus*, *B. amyloliquefaciens, B. subtilis, B. pasteurii, B. cereus, B. mycoides,* and *B. sphaericus* (Choudhary and Johri 2009)
BTH		
(2R, 3R) butanediol	Induces ISR with PGPR-like effect	This is an example of a Volatile Organic Compound (VOC) produced by bacteria that stimulates ISR(Cortes-Barco et al. 2010)
Chitin	Microbe Associated Molecular Pattern acts as a general elicitor, priming SAR	Nonsynthetic chitin is OMRI approved for organic use and is available from many sources. It is important to note that a threshold exists specific to each pathosystem for the activation of the Hypersensitivity Response (HR), which is the dividing line between priming and induction of direct defenses (Iriti and Faoro 2009). Priming is a more effective defense strategy.
Chitosan	Combines active antimicrobial properties with priming of SAR	Despite the chemical relationship with chitin, this product is synthetic and has not been approved for use by OMRI.
Cell Wall Derived Oligogalacturonides (OGA's)	Local Wound Response	Locally inhibitory to JA, which is simultaneously stimulated systemically and acts distal to the wounded area. These are considered PAMP's or Pathogen Associated Molecular Patterns that are recognized by plant receptors and initiate phytohormone de novo synthesis.
3,5-dichlorosalicylic acid (DC-SA)	Priming SAR (SAR')	One of many Salicylic acid derivatives used to induce SAR.
Ethylene (ET)	ISR, blocks Wound Induced Resistance (WIR) in most crops, but is essential for WIR in Solanaceous crops.	Products like ethephon. Synthetic ethylene gas is allowed with restrictions by OMRI. Also, ET is naturally produced by senescing climacteric fruits. Unfortunately, it does not make a good activator due to its other effects on crop physiology.
Fatty Acids	SAR	Free fatty acids are PAMP's that are recognized by plants and stimulate the SA-pathway. Oleic acid, linoleic acid, linolenic acid, arachidonic acid and eicosapentaenoic acid have all been shown to induce resistance.

Flg22	PAMP inducing	This is a peptide 22 amino acids long created from a conserved region of microbe DNA. This peptide is recognized by plant pattern recognition receptors (PRR's) and initiates induced resistance.
K2HPO3 (dipotassium phosphate or phosphoric acid)	Not fully understood. It is likely a combination of antimicrobial and systemic acquired resistance.	Phytogard is one product registered for use in crop protection, which has been shown to give systemic protection for at least 15 days at 40.6 ppm concentration. Phytogard is not OMRI approved; however, the use of phosphoric acid as a fertilizer is approved, so it is possible that an organic source of phosphoric acid could be used in organic production with the certifiers approval. Aliette is a similar compound. Chipco Signature was the original phosphonate generating phoshpite fungicide. Since the patent has expired, many similar products are now available: Magellan, Resyst, Alude, Prodigy Signature, Vital, K-phite, PK-Plus, Stress Phiter.
(GABA) Gamma-aminobutyric acid	Exogenous GABA functions as N source, insecticide (inhibitory neurotransmitter) and inducer of ET biosynthesis	GABA has been shown to be ineffective in inducing SAR. In fact, pathogens have been shown to hijack defenses in order to cause the accumulation of GABA to prove Nitrogen nutrition. Furthermore, GABA is a scavenger of reactive oxygen species, and thus may act to inhibit pathogen-related defenses. On the other hand, as a well-known invertebrate inhibitory neurotransmitter it can be used to effectively control herbivory by arthropods. Exogenous GABA application causes biosysnthesis of ET (Kathiresan et al., 1997). 10mM GABA exogenously applied inhibits hydrogen peroxide production, whereas 0.25mM concentration actually increases ROS production, while other N sources do not. ET biosynthesis can stimulate the JA-pathway.
L-glutamic acid, L-Glutamine		Recently found to inhibit BABA-IR. Precursor to GABA, food addative (MSG). Other active ingredient in Auxigro. (Wu et al. 2010)
Harpin	SAR	A protein product of genetically modified E. coli. This protein acts as an elicitor of the SA-pathway. Since the origin of this product is synthetic in nature, there are no products approved for use by OMRI.
Hexanoic acid (Caproic Acid)	Hx-IR and direct antimicrobial activity.	This novel inducer primes both JA and ABA defenses. Found naturally in cow butterfat, goat butterfat, coconut oil at about 0.6%. , so organic sources of coconut oil might present an opportunity for organic systems. (Vicedo et al., 2009)
Laminarin	Primes SAR	A beta 1, 3 glucan (a polysaccharide of glucose) derived from brown algae (*Laminaria digitata*). Like

		other beta 1,3 glucans, laminarin is a MAMP and induces resistance in plants (Trouvelot et al. 2008)
PC1 (isoparaffin-based mixture)	Induced ISR with a PGPR-like effect	This is a mixture of synthetic isoparafins that activates ISR. (Cortes-Barco et al. 2010)
Pseudomonas aeruginosa 7NSK2	Primes *SAR*	This is an example of the so-called 'SA-ISR," where a PGPR causes SAR induction rather than ISR (Audenaert et al. 2002)
Pseudomonas fluorescens	ISR inducing PGPR	Strain CHAO has been shown to elicit ISR via 2,4 DAPG or siderophores. Similar elicitors of ISR are proposed for stains GRP, WCS 417, WCS 374, and Q2-87.
Reynoutria sachalinensis	SAR	Extracts of Giant Knotweed (*Reynoutria sachalinensis*) have been shown to induce resistance in plants and has been marketed recently as Milsana.
Streptomyces spp.	JA/ET	Endophytic bacteria like members of *Streptomyces* have been shown to induce systemic resistance in crops. This is in contradiction to traditionally held mode of action as antibiosis. This is an example of the complex modes of actions that these sorts of organisms have when used in crop protection. The commercial product Actinovate probably has IR as a part of its mechanism of action. (Shimizu et al. 2006)
Sulfated Fucans	Primes SAR	Sulfated fucans are oligosaccharides found in the cell walls of marine brown algae. These MAMP's have been shown to prime SAR and give protection against TMV. (Klarzynski et al. 2003)
Vitamin B1 (Thiamine)	Primes SAR	Thiamine at 10mM concentrations mixed with Tween 20 primes SAR when applied as a foliar spray. (Ahn et al. 2007)
Yeast or Yeast Extract	Primes SAR	Various MAMP's contained in the extract of yeast (like Vit B1 and BABA) are primers of SAR.
Yucca Extract	Directly Antimicrobial and activates SA-pathway	Yucca extract has been demonstrated to be both directly antimicrobial and an inducer of resistance, probably the SA-pathway. (Bengtsson et al. 2009)

EXTENSION DOCUMENT: INDUCED RESISTANCE (IR) FOR ORGANIC TOMATO PRODUCTION

INTRODUCTION

Induced Resistance (IR) is a process that activates a plants own defenses for protection from a wide variety of pests. This process is very complex. Despite a limited amount of trials with an even more limited array of products, there have still been notable successes, especially for organic disease management. Products like Yucca Ag-Aide (yucca extract), Regalia (Giant Knotweed extract), Serenade (*B. subtilis*), Sonata (*B. pumilis*), Ballad (*B. pumilis*), Actinovate (*Streptomyces lydicus*), Mycostop (*S. griseoviridis*), Omegagrow Plus (fish oil), Seacide (seaweed extract), Stimplex (seaweed extract) are products that have been shown to induce resistance and are approved by the Organic Materials Review Institute for use in organic systems. In a very general sense, two major pathways are involved:

- Salicylic acid-mediated Systemic Acquired Resistance (SAR)
    - Useful for biotrophs (pathogens that invade living cells) and hemibiotrophs (pathogens with a biotrophic and necrotrophic phase)
- Jasmonic acid/Ethylene-mediated Induced Systemic Resistance (ISR)
    - Useful for insects, necrotrophs (pathogens that kills cells for nutrition), and hemibiotrophs

The pathway selected should be based upon the lifestyle of the pathogen (biotroph, hemibiotroph or necrotroph). Responses to induced resistance can vary by crop species, so crop-specific recommendations below would not necessarily be effective in another crop. Additional variation in success rates with IR can arise from: seasonal variations in light intensity, humidity, disease pressure, and the presence of multiple pests.

Activators of IR are not all equal: some activate only SAR like Regalia, while others are more general like Plant Growth Promoting Rhizobacteria (PGPR). Unfortunately, the mode of action of many activators has not been determined at this point. This publication reviews some of the considerations that are important for using IR activators optimally, and specific treatment options that have achieved at least low levels of disease reduction in field trials. Absence of a product from these recommendations is no indication of potential effectiveness.

GENERAL CONSIDERATIONS

I. Inducing resistance can have yield costs.

II. Plant Growth Promoting Rhizobacteria (PGPR) or Arbuscular Mycorrhizal Fungi (AMF) prime defenses, and are not generally associated with yield losses.

III. It is possible to minimize yield lowering costs somewhat with the correct management. Plants that are under environmental stress are likely to suffer greater growth and yield consequences from induction or to express impaired resistance when induced.

IV. A severe generalist chewing insect is likely to induce broad-spectrum cross-resistance.

V. By extension, pruning is compatible with induction of resistance; in fact, it can be used to temporarily induce resistance.

VI. If Plant Growth Promoting Rhizobacteria (PGPR) or Arbuscular Mycorrhizal Fungi (AMF) are used to colonize roots, avoid chemical drenches.

VII. The Gibberellins (GA) can enhance the SA pathway, while it negatively impacts the JA pathway. Auxins and Cytokinins have been shown to have the opposite effect of GA.
VIII. Results can be improved by mixing multiple strains of Plant Growth Promoting Rhizobacteria together.
IX. Attempt to induce resistance on the morning of sunny days.
X. Use Integrated Pest Management. When inducing SAR it is important to attempt to keep insects and necrotroph levels low with multiple tactics. Similarly, when inducing ISR, it is important to attempt to keep biotroph levels low with multiple tactics.
XI. Due to the fact that most IR activators do not have direct antimicrobial effects, the action of repeated foliar sprays using water as a solvent can actually cause increased disease pressure. Therefore, IR activators for foliar application should be used in a mixture with an antimicrobial agent like an Organic Materials Review Institute (OMRI) approved Neem oil, Sesame oil, Tea Tree oil or a copper compound. Another option is to use a drench as opposed to foliar sprays.

COMMON NAME	SCIENTIFIC NAME	LIFESTYLE	TREATMENT EXAMPLES	COMMENTS
Anthracnose	*Colletotrichum coccodes*	Hemibiotroph	2lbs/Acre *Bacillus subtilis* + 2lbs/Acre Copper Hydroxide (Champ, Nu Cop, etc.)	foliar spray, weekly or biweekly
			2lbs/Acre *Bacillus subtilis*	foliar spray, weekly or biweekly
Bacterial Canker	*Clavibacter michiganensis*	Hemibiotroph	3.5oz/100 gal Serenade (*Bacillus subtilis*)	foliar spray, weekly
Bacterial Leaf Spot	*Xanthomonas campetris*	Biotrophic Hemibiotroph	2% Omegagrow Plus (fish oil)	foliar spray, weekly
			1% Seacide (Seaweed extract)	
			1lbs/Acre Serenade (*B. subtilis*) + 2lbs/Acre Copper Hydroxide (Champ, Nu Cop, etc.)	
Bacterial Speck	*Pseudomonas syringae*	Biotrophic Hemibiotroph	1lbs/Acre Serenade (*Bacillus subtilis*) + 2lbs/Acre Copper Hydroxide (Champ, Nu Cop, etc.)	foliar spray, weekly
			4-9 x 10^9 cells/mL Yeast	
Early Blight	*Alternaria solani*	Necrotroph	200:1 Drench + 4 foliar sprays on 7-day interval Seaweed extract.	Avoid blocking ethylene perception. Gibberellins (GA), ethylene (ET), abscisic acid (ABA), cytokinins or auxins are compatible. Keep biotroph and hemibiotroph levels low with IPM tactics.
			4lbs/Acre *Bacillus subtilis* + 2lbs/Acre Copper Hydroxide (Champ, Nu Cop, etc.)	
			Mycostop (*Streptomyces griseoviridis*), use as directed	
Gray Mold	*Botrytis cinerea*	Necrotroph	2.5% Serenade (*Bacillus subtilis*), weekly foliar spray	Avoid blocking ethylene perception. Gibberellins. ethylene, abscisic acid, cytokinins or auxins are compatible. Keep biotroph and hemibiotroph levels low with IPM tactics.

Gummy Stem Blight	*Didymella lycopersici*	Hemibiotroph	1.6 pt/Acre *Reynoutria sachalinensis* extract (Regalia), weekly foliar spray	Use IPM to maintain low insect and necrotroph levels. GA is compatible, but avoid ET, ABA, Auxins and cytokinins.
Late Blight	*Phytophthora infestans*	Biotrophic Hemibiotroph	2pt/Acre Yeast extract, weekly or biweekly foliar spray	Use IPM to maintain low insect and necrotroph levels. GA is compatible, but avoid ET, ABA, Auxins and cytokinins.
Mites and Thrips	*Tetranychus spp*., *Thrips spp*., etc.	Cell-Content Feeders	1% *Reynoutria sachalinensis* extract (Regalia) foliar spray	ISR or SAR pathways for control of mites and thrips.
			Pseudomonas fluorescens or other Plant Growth Promoting Rhizobacteria as seed treatment, soil drench, root dip or foliar spray	
Nematodes	*Meloidogyne spp., Pratylenchus spp., Rodopholus spp. Heterodera spp*, etc.	Biotroph	*Pseudomonas putida*, *Bacillus subtilis*, *Azobacter chroococum* or other Plant Growth Promoting Rhizobacteria (PGPR), 10^8 cells/mL	PGPR can be applied as a seed treatment, transplant root dip, drench, or soil amendment. Also, note that SAR pathway does provide some limited control.
			Arbuscular Mycorrhizal Fungi (AMF) such as *Glomus intraradices,* 10^8 cells/mL	
Parasitic Plants	*Orobanche spp., Striga spp*., etc.	Biotroph	2.5% Seaweed extract (*Ascophyllum nodosum*), Soil Drench	SAR pathway can be very effective.
Phloem Feeders: Aphids, Whitefly, etc.	*Bemisia spp., Myzus spp.*, etc.	Phloem Feeders	*Bacillus amyloliquifaciens*, *Bacillus subtilis* or *Bacillus pumilis* as seed treatment, soil amendment, drench or transplant root dip.	Generally, activation of either ISR of SAR will reduce phloem feeders.
Powdery Mildew	*Oidium lycopersicum*	Biotroph	1% *Reynoutria sachalinensis* extract (Regalia), weekly foliar spray	Plant Growth Promoting Rhizobacteria like *Streptomyces spp.* are capable of priming defenses for a wide variety of pests.
			12oz/Acre *Streptomyces lydicus* (Actinovate) + 4pt/100gal BioLink (surfactant), weekly foliar spray	

Pythium Root Rot	*Pythium myriotylum*	Necrotroph	0.2% *Streptomyces griseoviridis* (Mycostop), 2 drench applications	Avoid ethylene blockers and Gibberellins. Ethylene, abscisic acid, cytokinins or auxins are compatible. Keep biotroph and hemibiotroph levels low with IPM tactics.
Septoria Leaf Spot	*Septoria lycopersici*	Necrotrophic Hemibiotroph	2oz/gal Seaweed extract, biweekly foliar spray	Often, pathogens that have both a biotrophic phase and a nectrotrophic phase can be controlled with either ISR or SAR pathways.
			1% *R. sachalinensis* extract alt. 2lbs./Acre Copper Hydroxide (Champ, Nu Cop, etc.), weekly foliar spray	
			6lbs/Acre *Bacillus pumilis* (Sonata, YieldShield), weekly foliar spray	
			12oz/Acre Actinovate (*Streptomyces lydicus*) + Biolink (surfactant), biweekly foliar spray	
Soil Rot	*Rhizoctonia solani*	Nectrotroph	8pt/Acre *Bacillus subtilis*, drench	Avoid ethylene blockers and Gibberellins. Ethylene, abscisic acid, cytokinins or auxins are compatible Keep biotroph and hemibiotroph levels low with IPM tactics.
Tissue-Chewing Insects	Various	Macerate Plant Tissues	Plant Growth Promoting Rhizobacteria (PGPR)	Avoid ethylene blockers and Gibberellins. Ethylene, abscisic acid, cytokinins or auxins are compatible. Keep biotroph and hemibiotroph levels low with IPM tactics. Use multiple tactics for severe infestations.
White Mold	*Sclerotinia sclerotiorum*	Nectroph	6.2lbs./Acre Yeast, weekly foliar spray	This is an exception to the rule: use SAR for this necrotroph.

SUPPLEMENTAL READING

Host Resistance:

Among the different mechanisms that plants use for defense against pests, the single gene type, what Vanderplank calls Vertical or race-specific, has proven to be very useful. Not only is it easier for breeders to manipulate than quantitative forms of resistance, what Vanderplank calls Horizontal or Non-race specific, but it often provides unqualified immunity (Baudoin 2010). Although quantitative resistance is much more durable, meaning that it won't breakdown as easily, protection is often partial. The problem with the single gene approach is that it is sometimes rather easy to overcome, making breeding improvements for disease control a temporary and costly fix. Fortunately, there may be a way around costly conventional and transgenic breeding programs. The systemic process that is initiated when a host recognizes a pathogen attacker can be hijacked and controlled. Simply by applying chemicals or other substances to the plant's tissue, we can simulate an attack and induce a systemic type of resistance. This paper will provide a detailed description of the (i) processes involved, (ii) helpful guidelines for induction of host resistance, (iii) specifics on incorporating inducted resistance into an integrated pest management plan, (iv) and an in depth review of products that can be used for induction of resistance.

There are many forms of resistance: "host-resistance" can result from escape, klendusity, tolerance, non-host interaction, monogenic resistance or quantitative resistance. When a plant is not infected because it is a non-host, the interaction is not referred to as host-resistance. Escape refers broadly to a susceptible plant that does not become infected by a virulent pathogen for some reason—whether it is an effect of microclimate or a physiological or morphological characteristic. More specifically, klendusity refers to the morphological form of host resistance, which might include cleistogamy, trichomes, etc. The mechanism of tolerance is not fully understood; it might be a hardy structure or exceptional vigor or even related to horizontal resistance. Essentially, tolerance is when a susceptible plant has the ability to produce a good crop despite infection by a virulent pathogen (Baudoin 2010). In part, the process we are interested in for inducing host resistance is related to monogenic (Vertical or race-specific) resistance.

So, what traits make a pathogen virulent? Virulence genes are those that create products that enhance pathogenicity; for example, pectinases and cutinases that help degrade structural obstacles for penetration (Agrios 2005). There is a tremendous amount of redundancy among virulence genes, and the degree of redundancy is correlated with the severity of many diseases (Agrios 2005). Additionally, the pathogen needs to attack without being recognized by the plant cell. If it can achieve penetration without setting off the alarms, serious damage to the crop can result.

In the case of monogenic resistance, what makes a host susceptible to a pathogen? It is not known exactly what makes a plant a non-host to most pathogens and host to a small number of pathogens on average (Agrios 2005). What is known is that within a host species, some individuals may coevolve with pathogens or be artificially selected to recognize the gene product of a specific pathogenicity trait like those mentioned above. When this occurs, the pathogenicity gene product is thought of as the "elicitor" because it is recognized by the resistance (R) gene

product of the host, which is involved in the transduction of a signal that triggers a resistance response; the specific pathogenicity gene that codes for the elicitor is thought of as the avirulence (avr) gene (Agrios 2005).

In this case, inheritance of resistance to a disease as well as pathogen virulence and avirulence is controlled by single genes: "*R* genes in the host are specific for *avr* genes in the pathogen" (Agrios 2005). This arms-race has developed through coevolution and is referred to as the gene-for-gene concept (Agrios 2005). A hypersensitivity reaction occurs as the direct result of the interaction of *avr* and R gene products, which maintains the plants resistance in part by sacrificing cells local to the attack (Agrios 2005). This mode of resistance has been called by many names: known as *R*-gene-mediated resistance, also referred to as gene-for-gene resistance, or effector-triggered immunity (ETI) (van der Ent 2007). If the resistance is highly efficient, the hypersensitivity reaction may only involve a single cell or just the few cells that came into direct contact with the pathogen; in this case, the attempted attack on the plant would go unnoticed by the unaided eye. It should also be noted that resistance comes in many degrees, and even if a pathogen sneaks into the host unrecognized, it is unusual for a disease to advance to kill the entire plant unchecked (Agrios 2005). As mentioned above, the inheritance of these genotypes constitutes an evolutionary arms race. Mutation of an existing avirulence gene in a haploid pathogen like deuteromycetes, ascomycetes, viruses, or bacteria could lead to a very rapid "breakdown" of host resistance, and the turnover involved with producing resistant cultivars of important crops can be relatively rapid—this limits the effectiveness of using resistant cultivars as a disease management strategy (Baudoin 2010)).

As aforesaid, it is the induction of the hypersensitivity reaction that enables the R-gene expressing host to remain uninfected. How is this accomplished? The answer to this question is complex and not entirely elucidated at this point. However, we do know that the elicitor binds the R gene encoded cell receptor either at the membrane or in the cytosol, and a signal is transduced culminating in the attachment of a nuclear binding domain protein to the cell's DNA (Agrios 2005). This binding alters transcription, up-regulating a myriad of disease fighting genes. The "transcriptional reprogramming" that takes place causes extracellular alkalinization and Programmed Cell Death (PCD) mediated by the production of reactive oxygen species like superoxide (Agrios 2005). In addition, before its death, the infected cell also causes the production and release of antimicrobial compounds and signaling molecules. Extracellular alkalinization and phytoalexins (antimicrobial plant products) kill the pathogen and PCD destroys the breached cell, which can all be thought of as the localized response (Agrios 2005). The signaling molecules like Salicylic Acid (SA) produced during this period of alter DNA transcription activate Systemic Acquired Resistance (SAR) (Agrios 2005). PCD and the accumulation of signaling molecules are necessary because plants do not produce antibodies nor do they have scavenger cells like our macrophages that clean out dead and infected cells. Within 4-6 hours of inoculation, the signal for SAR will be generated and systemic expression will occur within 24 hours. When inoculation occurs, Nitric Oxide (NO) synthase activity is drastically increased and NO acts through induction of SA (Agrios 2005). At this point, all plant tissues contain greatly increases levels of SA. In cucumber, a hypersensitivity reaction produced when an incompatible pathogen is purposely introduced leads to resistance to 13 diseases for a period of 4-6 weeks, and a booster inoculation administered at 2-3 weeks after the initial inoculation can provide season long resistance to these many pathogens (Agrios 2005).

Interestingly, exogenous application of SA or a functional analog like acibenzolar-S-methyl (BTH) activates SAR as well as a few of the defense genes that it regulates (Walters and

Heil 2007). In addition, a network of interconnected pathways unrelated to ETI can be used to activate induced resistance (IR) through the recognition of Pathogen Associated Molecular Patterns (PAMP's); PAMP's include flagellin, chitin, glycoproteins and other elicitors associated with potentially pathogenic microbes (van der Ent 2007). This network of resistance-related pathways is called PAMP-Triggered Immunity (PTI); and although avr- and R-gene products are not involved, this interaction can induce the Salicylic acid (SA)-mediated Systemic Acquired Resistance (SAR), Wound Induced Resistance (WIR), Induced Systemic Resistance (ISR) or pre-penetration PTI. Similar to ETI, PTI uses a pathogen-related substance as the impetus for the transduction of a systemic signal. PTI that occurs pre-penetration is mediated by ABA, which causes alkalinization, stomatal closure and callose formation to prevent penetration (van der Ent 2007). PTI that occurs post-penetration might involve the Jasmonic acid (JA)/Ethylene (ET)-mediated Induced Systemic Resistance (ISR) pathway, which can be generally thought of as useful against necrotrophs (pathogens that kill cells for nutrition) (Walters and Heil 2007). Otherwise, PTI might involve SAR, which can be generalized as effective against biotrophs (pathogens that can invade living cells), while both SAR and ISR can be effective against hemibiotrophs (pathogens that have a biotrophic and necrotrophic phase) (Thaler et al. 2004). In most species except for plants in the family Solanaceae, ET production guides the plant away from herbivore resistance provided by WIR and toward ISR; however, in *Lycopersicon esculentum* ET is actually required for the generation of WIR (O'Donnell et al. 1995; Adie et al. 2007). This is an example of evolutionary divergence within the plant immunity network, and tends to be a rather common theme within the body of literature surrounding these phenomena. The pathway selected depends on the PAMP recognized (i.e. the nature of the pest) and the specific crop (i.e. divergent evolution), and the network of pathways provides redundancy in case a pathogen has developed a mechanism for suppressing a particular pathway (Robert-Seilaniantz et al. 2007). Some elicitors are very specific and activate only one pathway like acibenzolar-s-methyl ester (BTH), while others are more general in action because they contain multiple PAMP's like Plant Growth Promoting Rhizobacteria (PGPR). Unfortunately, the variation in elicitor mode of action adds an additional layer of complexity to the practical use of IR for pest management.

Herbivore Resistance and ISR:

Induced Systemic Resistance (ISR) is considered a separate mechanism from SAR and confers resistance to different types of pathogens (Da Rocha and Hammerschmidt 2005). Whereas SAR is induced by pathogens that set off the hypersensitivity response, ISR is induced by growth promoting rhizobacteria that colonize the root surfaces in the rhizosphere, and induce resistance in the leaves and stem without causing any of their own symptoms (Da Rocha and Hammmerschmidt 2005). In fact, about 25% of the bacteria that generally inhabit the rhizosphere are considered beneficial in this way, including several fluorescent *Pseudomonas* species and many strains of *Bacillus* (Walters and Heil 2007). It is now accepted that these organisms interact with the host plant through perception of volatile organic compounds (Watson 6). SAR and ISR also differ in that the SAR pathway is mediated by Salicylic Acid, whereas the signaling pathway inducing ISR is mediated by Jasmonic Acid (JA) and Ethylene (ET) (Da Rocha and Hammerschmidt 2005). The difference between these pathways that is of great significance for us to consider is that each pathway leads to protection from a different spectrum of pathogens (Da Rocha and Hammerschmidt 2005). Despite these differences, they both yield

broad spectrum resistance to pathogens and can sometimes be activated simultaneously within the same host plant, providing an even broader spectrum of resistance (Hammerschmidt 520). In fact, a study in 2008 showed that application SAR and ISR inducers in combination were not antagonistic but additive in the degree of resistance expressed (Herman 1226). Furthermore, van Wees *et al.* (2000) showed that simultaneous stimulation of ISR by growth promoting rhizobacteria and SAR by inoculation with an avirulent pathogen had an additive effect on resistance from *Pseudomas syringae* pv. *tomato* in *Arabidopsis*. Despite these findings, the JA/ET pathway and the SA pathway can generally be thought of as opposing forces: the JA/ET pathway gives necrotroph and insect resistance at the detriment of biotroph resistance, while the SA pathway gives biotrophs and hemibiotroph resistance at the detriment of necrotroph and insect resistance (Robert-Seilaniantz et al. 2007). The reason for the variability in the literature is that each plant pathosystem responds in a unique way to a unique set of phytohormones (van der Ent 2007).

Wounding caused by chewing insects causes the accumulation of Jasmonate (JA), which sets off transcriptional changes that enhance the plants ability to repel these attackers (Walters and Heil 2007). Coevolution with insect vectors has led to an additional consequence of insect feeding—JA has the ability to trigger SAR downstream of SA (Walters and Heil 2007). The consequence is that with insect infestation comes not only enhanced resistance to that pest but to pathogens as well. However, if both and disease epidemic and an insect infestation are occurring at the same time, then the plant will shift resource allocation almost entirely to SAR. SA accumulates when the hypersensitivity reaction occurs and the plant accomplishes this shunting of resources through negative feedback of SA on the JA pathway and herbivore resistance (Walters and Heil 2007). In the event of a particularly damaging insect infestation and a severe disease epidemic occur at the same time, it would be unwise to use to SAR inducing agents due to the negative feedback on the herbivore resistance pathway. However, if the agent works downstream of SA like derivatives of isonicotinic acid, this might allow for enhanced SAR without negative impact on herbivore resistance. At the same time, heavy insect feeding may also cause some induced resistance to pathogens. The perception of saprophytes through volatile organic compounds is interpreted in the same way as wounding by herbivores or necrotrophs: "thus, in general, biotrophs appear to be more sensitive to SA-dependent responses, while necrotrophs and herbivorous insects are sensitive to JA/ET-dependent defenses" (Walters and Heil 2007). In fact, in the same way that plants perceive the growth promoting rhizobacteria through volatile organic compounds, herbivore resistance can be induced by the volatile organics emitted from a nearby plant that is under attack by insects (Walters and Heil 2007). More stealthy feeders like aphids that do not cause much wounding are able to avoid setting off the Jasmonate pathway to herbivore resistance and thus avoid setting off SAR as well (De Vos 527). Aphids and other sneaky feeders thus avoid tripping the security system, making them more efficient herbivore and vectors of disease (De Vos 527).

Antagonism of SAR with Abscisic acid (ABA) Mediated Abiotic Stress Response:

In the same way that Ethylene, Jasmonic Acid and Salicylic acid hormonally modulate responses to biotic stressors, Abscisic acid helps the plant respond to environmental abiotic stress like "drought, salinity and low temperatures" (Yasuda 1678). In this capacity, it actively inhibits the expression of resistance, shunting the allocation of plant resources towards surviving the harsh environmental conditions. In fact, exogenous application of ABA causes enhanced

susceptibility to pathogens (Yasuda 1679). Wounding of the plant causes recruitment of "wound-inducible genes" that activate herbivore resistance, and ABA acts upstream of JA in this pathway (Yasuda 1682). Thus, exogenous application of ABA can also be used to induce herbivore resistance (Yasuda et al. 2008). In summary, ABA signaling allows the plant to prioritize abiotic stresses over biotic stresses (Robert-Seilaniantz et al., 2007).

Costs, Trade-offs and Problems:

One potential problem for commercial application is that induction of SAR takes time; there is lag phase between application and the systemic expression of disease resistance (Da Rocha and Hammerschmidt 2005). Additionally, variation has been observed in the efficacy of induction. This variation might cause differences in the effectiveness of induction due to genetic differences between species and even cultivars. With ISR induced by beneficial organisms, different bacteria-host complexes interact in different ways and the resulting degree of resistance and efficacy depends on the exact pathogen (Walters and Heil 2007). Further complication arises from the fact that induction in a specific species induces resistance to a specific suite of pathogens, and the determination of whether to induce herbivore resistance, ISR, SAR or none of the above might be dependent on a comprehensive risk assessment based upon relative pest pressures and expected economic losses (Da Rocha and Hammerschmidt 2005).

Finally and perhaps most importantly, certain studies have shown specific inducers to affect the growth and/or yield of the host plant (Da Rocha and Hammerschmidt 2005). In essence, there is a very good reason that plants have evolved a mechanism for turning on defenses only when they are needed—induction of defenses is energetically expensive. "In fact, negative correlations between growth rate and resistance to insects or disease represent a long-known phenomenon" (Walters and Heil 2007). This price of mounting a defense is called the allocation cost, but there are additional costs that might not be as obvious at first glance. For example, expressing resistance to a pathogen might inhibit mycorrhizal associations with the root and thus inhibit water and nutrient uptake by the host plant, which would constitute an "Ecological Cost" (Walters and Heil 2007). Again, tests in Arabidopsis tell us that SAR in the absence of disease pressure is expensive, as mutants that over-express SAR are lower yielding and stunted (Walters and Heil 2007). Plants exposed to exogenous JA have reduced seed yield, leaf growth, and delayed flowering in the absence of insect herbivores (Walters and Heil 2007). However, some studies have shown no effect on growth even in a pathogen free environment. These curious findings at odds with our intuition and the growth differentiation balance hypothesis could be explained by the increased photosynthesis that some plants express when attacked by insects or pathogens (Walters and Heil 2007). These reports indicate that a plant can switch into overdrive in order to avoid allocation deficits for growth and development while expressing resistance, but plants growing under conditions that are water or nutrient limiting cannot compensate adequately (Walters and Heil 2007). Thus, plants that are not grown under optimal conditions are likely to suffer the most from the costs associated with induction or weakly express resistance (Walters and Heil 2007). In fact, the variation of some of these studies from the expected outcome can be explained by the dependence of costs on nutrient availability (Walters and Heil 2007). Stunting and yield costs are a major reason why transgenic crops over-expressing IR genes are not a good idea. A transgenic mutant that over-expresses NPR1, the gene found to mediate both ISR and SAR pathways, is subject to the allocation costs, ecological costs and genetic costs associated with defense season long, even in a pest free environment; so

these mutants are invariably stunted and produce lower yields (Walters and Heil 2007). Furthermore, like all plant systems, generation of resistance is energy dependent, and one can therefore expect that the costs associated with induced resistance will go up (and the efficacy may go down due to impaired expression) during periods of low-light (i.e. extended periods with extensive cloud cover). Since extended periods of overcast skies can be predicted, this might be an additional consideration for the use of direct defenses versus another treatment option. Furthermore, Reactive Oxygen Species (ROS) generation is promoted by stomatal closure, which occurs as a result of abiotic or biotic stresses. High humidity can slow stomatal closure and thus inhibit resistance (Bolton 2009). Although the latter finding may not be a limiting factor to the induction of effective resistance, it is significant and may at the least further explain some of the variability among field experiments.

Other Phytohormone Signals:

Post-penetration resistance is regulated by phytohormones, with SA and JA/ET being the major players. However, other phytohormones like ABA, Auxin, Cytokinin, Gibberellic acid, and Brassinosteroids also modulate the signaling pathways. It was once accepted that SA induced defenses were most effective against biotrophs and that JA/ET pathways functioned against necrotrophs and insects, but this view is now known to be far too simplistic. In fact, it turns out that a highly specialized transduction pathway with a unique set of signals is used for each plant pathosystem, called the "signal signature (van der Ent 2007). Due to this inherent variability in the system, plant pathosystem screening is critical for this disease management strategy—it is not the broad spectrum silver bullet that it was once thought to be. For the purpose of selecting appropriate products, dosages and application methods for the specific plant pathosystem involved, producers should consult Table 1 or review the literature.

Gibberellic Acid (GA) has been used for some time in integrated pest management because application overcomes the symptoms of many virus and mollicute disease (Agrios, 2005). For example, GA application alleviates the symptoms of corn stunt spiroplasma, tobacco etch virus, and axillary bud suppression caused by Prunus Dwarf Virus (PDV) (Agrios 2005). Within IPM, there are many more possible uses of GA, such as loosening grape clusters for better disease control (*Botrytis,* etc.). Since GA is already used in Organic IPM, we need to know if inducing resistance is compatible with these applications. In fact, it turns out that GA can actually enhance the SA pathway, while it is negatively impacts the JA pathway (Robert-Seilaniantz et al. 2007). Therefore, the induction of SAR can be integrated with GA application, whereas induction of ISR should be considered a contraindication for the application of GA.

Resistance Suppression:

Xanthomonas campestris, *Ralstonia solanacearum* and *Pseudomonas syringae* are all biotrophic pathogens. The SA pathway leading to SAR would be the most effective means of establishing host resistance to these pathogens; however, all of these biotrophs have been shown to directly secrete Auxins or to upregulate Auxin production within the plant's cells. Auxins antagonize the JA pathway while inhibiting the SA pathway (Robert-Seilaniantz et al. 2007). Since SAR often gives resistance to biotrophs and hemibiotrophs, by modulating the phytohormone signaling in favor of ISR these pathogens have jumped the major hurtle to virulence. Further support for the hypothesis that pathogens are using phytohormone crosstalk to

their advantage is the fact that many biotrophic fungi such as *Cladosporium fulvum, Blumeria graminis, Pyrenopeziza brassicae* and *Venturia inaequalis* produce cytokinins; whereas no necrotrophic fungi are known to produce cytokinins (Robert-Seilaniantz et al. 2007). Cytokinins are another group of phytohormones that antagonize the JA pathway and inhibit the SA pathway, modulating the *in planta* signaling in favor of ISR over SAR. This group of biotrophs avoids the effective host resistance that SAR would provide by tricking the plant into using ISR. Similarly, *Botrytis, Ceratocystis, Fusarium and Rhizoctonia* secrete ABA in order to shunt resources away from pathogen stresses towards abiotic stress tolerance Robert-Seilaniantz et al. 2007). In order to counteract these resistance suppression mechanisms, select the appropriate pathway and product for the plant pathosystems involved based on Table 1 or a literature review. To emphasize the importance of selecting the appropriate pathway for the specific plant pathosystem involved, consider the following: the necrotrophic pathogen *Botrytis cinerea* is controlled by the ET pathway in Arabidopsis, whereas in tomato it is controlled by the SA pathway due to a brief biotrophic phase in its life cycle (Robert-Seilaniantz et al. 2007). This is significant because induction of ISR in tomato will not control Botrytis, instead it may actually enhance susceptibility by negative crosstalk between JA and SA pathways. In this case, *Botrytis* is producing ABA in order to inhibit SAR, so exogenous application of SA or other SAR inducer will reduce susceptibility to infection.

Insects like the Silverleaf whitefly (*Bemisia tabaci*) also have the ability to induce "ineffective plant signaling cascades as a decoy mechanism" (Van der Ent 2008). This particular whitefly has been credited with the ability to activate SA responsive genes and thereby inhibit the JA pathway that would effectively control the insect. In addition, large cabbage white (*Pieris brassicae*) and the small cabbage white (*Pieris rapae*) have been credited with the same ability. In this case, resistance can be achieved by applying MeJA or other JA/ET inducers (Zarate et al. 2007). By extension, it should be possible to turn the tide toward JA/ET pathway expression with Auxin and Cytokinins application.

Priming:

Our immune systems have long memories: once attacked, antibodies are generated to epitopes characteristic of the antigen and a subsequent assault is met with a coordinated counter offensive. The immune systems of plants are quite different, but they have a similar ability to 'remember' a previous attack that creates a faster and more powerful defense response should the assailant return (Walters and Heil 2007). Many agents can be used to 'prime' a plant for defense: "The priming agent might be a chemical elicitor such as ASM, saccharin or BABA, a challenging pathogen, or even just volatile compounds released by herbivore-damaged neighboring plants" (Walters and Heil 2007). The difference is quantitative in that these agents at higher dosages would induce full resistance expression, whereas low dosages cause a priming effect (Walters and Heil 2007). For example, between 5 and 50 mM concentrations of Vitamin B1 or thiamine has been shown to protect *Arabidopsis* from bacterial infection from *Pseudomonas syringae* pv. *Tomato* (Ahn et al. 2007)

The interesting thing to note with the priming effect is that the allocation costs seem to be minimal, but the disease control upon introduction of a pest is still substantial (Walters and Heil 2007). For a producer who sprays high concentrations of BABA for fully induce resistance in the entire orchard because of an epidemic, the producers will see significant yield reductions in all trees, even those that were never attacked by pathogen. On the other hand, a producer that

uses lower dosages to prime all the trees in his orchard would still get the benefit of disease control in those trees that are attacked with the benefit of lower growth and yield penalties for those trees that were not attacked (Walters and Heil 2007). This is very promising and exciting area of research in crop protection.

Aerated and Non-aerated Compost Teas:

As aforementioned, there are numerous examples of chemicals that were thought to be toxic to bacteria or fungi but turned out to be SAR or ISR inducing. In addition to these examples, there may be other long-practiced methods that derive their mysterious and highly variable results from these mechanisms; and as we have discussed, the distinction could not be more important to disease management. An example is the application of compost teas, which are essentially fermented leachates of either tradition compost or vermicompost. Not only can soil incorporation of compost and vermicompost improve soil quality, but 'Worm tea' or the unfermented leachate of mature vermicompost has been documented to be a high quality organic fertilizer in both Sorghum and Maize (Antonio et al. 2008; Carlos et al. 2008). Also, 24 hour aqueous extracts of vermicompost were shown to prevent attack by multiple insect and mite pests (Edwards et al. 2010). Vermicompost has been widely documented to cause disease suppression in forms ranging from potting mix to soil drench to foliar applied spray (Scheuerell and Mahafee 2004; Scheuerell and Mahafee 2005). The parameters concerning the production of non-aerated compost teas (NCT) have not been optimized, nor is there a consensus on the efficacy of these procedures (Scheuerell and Mahafee 2004). Vermicompost tea used as a soil drench or vermicompost used as a soil amendment have shown more consistent disease suppression (Scheuerell and Mahafee 2004). Along with this evidence, several sources have documented that any disease suppression exhibited by a NCT is negated if the liquid is heated or diluted extensively (Scheuerell and Mahafee 2004; Malandraki et al. 2008). Many have claimed a biocontrol mechanism for the disease suppression function of NCT, and yet many have been surprised by the fickle nature of its real world application. In fact, if NCT's are so inconsistent, perhaps that should also be evidence of a biocontrol mechanism, as the field application of mycoherbicides has been plagued by equally inconsistent results (Ghosheh et al. 2005). Further evidence of this exists in the tested use of adjuvants in NCT spray applications: Scheuerell and Mahaffee (2006) stated that the enhanced disease suppression in NCT's combined with adjuvants could be attributed to "increased attachment and subsequent survival of applied organisms (Scheuerell and Mahafee 2006).

The function of Aerated Compost Teas (ACT's) and NCT is more than likely the induction of SAR or ISR, which has been suggested by numerous authors on this subject (Siddiqui et al. 2009; Zhang et al. 1998). Saprophytes sprayed on the leaf surface might trigger an extremely efficient hypersensitivity reaction such that the local lesions produced are too small to be visible to the naked eye. There are a number of commercial products available for the production of ACT's and NCT's mostly targeted at the home gardener. The important consideration here is that consumers understand the potential costs of inducing these mechanisms in order that they might better weigh their options for disease management.

Compost amendments prime SAR, and have been shown to control some diseases (Zhang et al. 1998). Compost amendments essentially boil down to a natural means of inoculating your plants with SAR or ISR inducing strains, but there is obvious variability in the efficacy of these treatments due to the biological component of the system—keeping the right strains of bacteria

happy is not always easy. There is also evidence that a population threshold must be met, adding additional variability (Zhang et al. 1998). Despite their variability, this represents an organic means of disease control that can be produced on the farm, with little expense. Additionally, since compost amendments only prime SAR or ISR, no costs are associated with application in the event the plant is not challenged by a pathogen. Extracts of compost have been used for years as foliar sprays to control grey mold of strawberry, late blight of potato, downy mildew and powdery mildew of grape to name a few (Zhang et al. 1998). Interestingly, the condition of the soil seems to play a role in the efficacy of these extracts: if the soil is amended with compost, the plant in the field was already induced and so no further reduction in disease occurred, but when the spray is applied to a plant in a peat mix the disease control is achieved (Zhang et al, 1998). In fact, treatment of the plant in the compost amended soil with SA had no effect on disease severity, further demonstrating the SAR was already induced (Zhang et al. 1998). These water extracts were derived by adding tap water at a 1:1 dilution and allowing the mixture to ferment for 1 week, and this Non-aerated tea was as effective as SA against bacterial speck in Arabidopsis (Zhang et al. 1998).

Timing of Inoculation Affects SAR Response:

The timing of inoculation is an important factor controlling the rapidity of systemic expression and the degree of resistance conferred. The response to an inoculation that occurs during the morning and afternoon hours will be more acute than one occurring in the evening or at night (Griebel and Zeier 2008). The reason for this behavior seems to be that the process is actually phytochrome dependent, as mutants lacking phytochromeA-phytochromeB were SAR deficient (Griebel and Zeier 2008). Systemic alteration of gene expression is an energy-expensive operation, and thus phytochromA/B involvement is critical to producing a low-cost expression (Griebel and Zeier 2008).

Also, the antagonism or synergism existing between the SA and JA mediated resistance pathways seem to be based upon both dosage and timing. In fact, the reported inhibition of the JA pathway by SA is only consistent when both SA and JA pathways are simultaneously elicited (Thaler et al. 2002). A temporal separation of just 2 days negates the antagonism, and these responses are dose dependent—the higher the dosages the more consistent the predicted response (Thaler et al. 2002). Also, JA elicitation can exhibit a synergistic effect on the SA pathway—when the JA-pathway is elicited first, it can prime the SA pathway, allowing for increased resistance to a biotroph/hemibiotroph (Thaler et al. 2002). The inconsistency among reports concerning the JA-SA interaction is probably due to the dose and time dependent nature of the interaction. The significance of this finding is that so long as the induction is temporally separate, one can change the plant's mind as to which pathway to choose without interference from negative crosstalk (Valenzuela-Soto et al. 2010).

Physical Induction:

Low temperature storage, wounding, CO_2 treatment, heat treatment, ionizing irradiation, and UV C irradiation can each induce resistance (Terry and Joyce 2000). Wounding Induced Resistance (WIR) is well documented (van der Ent, 2007). Francia *et al* (2008) used *Botrytis cinerea, Fusarium oxysporum, Phytophthora capsici* and *Pseudomonas syringae* to challenge wounded or unwounded tomatoes (Francia et al. 2008). Wounding provided significant

protection from all of the pathogens used despite their different lifestyles. Protection peaks in plants wounded 3-7 days prior to challenge by a pathogen, acts both locally and systemically and ethylene affects basal resistance either positively (*Phytophthora capsici* and *Botrytis cinerea*) or negatively (*Pseudomonas syringae* and *Fusarium oxysporum*) (Francia et al. 2008). The significance of this finding is that the metabolic costs associated with wound repair do not lower the fitness of the plant if it is subsequently attacked by a pathogen. The finding that herbivory or wounding can stimulate induced resistance genes agrees with other earlier work. Thus, when plants are heavily infested with tissue-chewing insects, broad-spectrum defense priming is probably established, so activating IR will be wasteful. Also, this finding means that pruning is compatible with induced resistance as a pest management strategy.

The JA, ET, ABA, SA Signaling Network:

The function of ethylene (ET) is divergent in tomato, which has been used as a model system for induced resistance. In most systems, ET inhibits Wound Induced Resistance (WIR), except in tomatoes where both ET and JA are a requirement for the activation of wound responsive genes (Adie et al. 2007; O'Donnell et al. 1996). In most other systems, ET biosynthesis is stimulated by perception of PAMP's, and it shunts gene regulation toward protection from nectrotrophic pathogens (ISR). Thus, resources are allocated away from herbivore resistance and biotroph/hemi-biotroph resistance through inhibition by ET regulated transcription factors(Adie et al. 2007). Surprisingly, abscisic acid (ABA) blocks the modulating effect of ET on the JA pathway, leading to wound induced resistance for protection from herbivorous insects (Adie et al. 2007). ABA is involved in pre-penetration resistance form of Pathogen Associated Molecular Pattern (PAMP)-Triggered Immunity, and it inhibits both the JA/ET necrotrophic pathogen resistance pathway and the biosynthesis of SA in order to shunt resources specifically toward reinforcement of physical barriers prior to pathogen penetration—stomatal closure and callose formation are critical functions (van der Ent 2007). Interestingly, *de novo* SA biosynthesis is positively looped with ABA production, which creates a means of negatively feeding back on the SAR pathway in the latter stages of disease development (Adie et al. 2007). Thus, ABA not only provides a means of dividing resource allocation between environmental stress and disease, but also a means of shunting protection from pre-penetration defenses to post-penetration defenses, of drawing down protection when the threat is gone, and a means of shunting resources from necrotrophic pathogen defense toward wound resistance and healing (Adie et al. 2007). The exquisite regulation and interaction of these various phytohormones allows the plant to fine tune responses and allocate energy where it is needed most. Specifically, it seems most plants prioritize environmental stress over disease and biotrophs/hemibiotrophs over necrotrophs and insects.

There is evidence to suggest that the network is not strictly autocratic, meaning that the system is extensively interconnected to such an extent as to confound the results of any research that focuses on a single pathway or interactions between just two of the pathways (Tsuda et al. 2009). Overall, this redundancy allows the plant to back up its immunity against various counter attacks by pathogens and insects. Specifically, what may appear to the direct inhibition of JA on the SA pathway may actually be the result of a weaker but effective backup defense response (JA) as compared to the more efficient immunity given by the primary defense system (SA) (Tsuda et al. 2009). Much of the most recent research points to a plant immunity network of dazzling complexity, and a complete picture of the process is elusive to this day.

SA-ISR, Hx-IR, PS3-IR, BABA-IR and Cross-Resistance:

All elicitors of IR are not created equal; in fact, some have characteristic patterns of expression that are unique. For example, SA-ISR is the accumulation of SA biosynthesis and related defenses by specific strains of Plant Growth Promoting Rhizobacteria (PGPR) and it has been reported in many plant-pathosystems. A particularly well documented SA-ISR elicitor is *Pseudomonas aeruginosa*, a PGPR strain that causes the accumulation of SA (Choudhary et al. 2009). Interestingly, many Pathogen-Associated Molecular Patterns (PAMP or MAMP) do not simply trigger ISR or SAR, but instead provide variable levels of cross resistance through the simultaneous priming of some combination of ABA responsive genes, SA-responsive genes, JA-responsive genes or ET-responsive genes. Although this seems beneficial, it gives each individual activator-crop system a unique pattern of expression and thus an unpredictability that makes study and adoption more tedious (van der Ent 2007). When a PAMP triggers SAR, we can then get a general idea about which pathogens it is probably effective against, but when combinations of genes are activated, it becomes a guessing game that will have to be teased out by rigorous study. Beta-aminobutyric acid (BABA)- induced resistance or BABA-IR primes the SAR pathway as well as the ABA pathway. This combination provides some interesting cross-resistance, and it has been suggested that PS3, a sulfated beta 1,3 glucan, might share similar attributes (Trouvelot et al. 2008). Hexanoic acid induced resistance or Hx-IR primes the ABA pathway as well as the JA-pathway, providing protection from a unique set of pathogens.

A more predictable form of cross resistance occurs when herbivores that macerate plant tissues feed on crops. In this case, resistance against future herbivory is activated in the form of Wound-Inducible genes like PIN. Also, ISR and SAR are primed, which is evidenced by broad spectrum resistance to viruses, necrotrophs and biotrophs. Furthermore, cross-resistance is also a consistent consequence of prior infection—that is to say colonization of the roots by Arbuscular Mycorrhizal Fungi (AMF) or Plant Growth Promoting Rhizobacteria (PGPR). It has been shown that root colonization of the roots by AMF or PGPR, traditionally thought of as a way to stimulate ISR, has been shown to provide significant protection from Nematodes, which are biotrophic pathogens. The most tantalizing aspect of the above mentioned mechanisms of stimulating cross-protection is that these reactions involve priming, which has been shown has the ability to avoid most of the yield-costs associated with inducing direct defenses.

Lifestyles and Susceptibility to IR:

It was McDowell and Dangle (2000) that described a model for pathogen susceptibility to IR. Their work suggested that it was the lifestyle of the pathogen that determined whether or not it could be control with the SA or JA pathway. The SA-pathway is associated with the Hypersensitivity Reaction (HR) and Programmed Cell Death (PCD), which would only serve to increase the numbers of necrotrophic pathogens. Thus, the JA-pathway is suggested to be an alternative to SA that can more effectively battle the true necrotrophs. As a generality, it holds true but with many exceptions and overlaps. Perhaps the best explanation for this is again a conceptual oversimplification, as suggested by Thaler *et al* (2004). Thaler *et al* suspect that there is a continuum of lifestyles from Biotrophic to Necrotrophic. For example, *Phytophthora infestans* is mainly biotrophic but also has a necrotrophic phase, and might be therefore referred to as a Biotrophic Hemibiotroph. Furthermore, *Septoria lycopersici* is mainly a necrotroph with a brief biotrophic phase, and might therefore be referred to as a Necrotrophic Hemibiotroph.

Within this system, True Biotrophs, True Hemibiotrophs, and True Necrotrophs might be said to exist, although the later has been shown to be quite rather rare (Parbery 1996). There have been widespread examples of what might be called 'lifestyle confusion,' which exemplifies our limited knowledge here. In the end, the application of IR is, exhaustingly, further limited by our knowledge concerning pathogen lifestyles.

With regard to arthropods, the situation appears to be a little bit clearer. In general, tissue-chewing insects activate the JA-pathway, and thus the JA pathway provides protection (Thaler et al. 2004). Next, Cell-Content Feeders like mites and thrips are susceptible to activation of the JA pathway, although there is evidence that mites can activate SA or JA and that either pathway leads to control (Tomczyk 2006). Finally, Phloem Feeders like aphids are susceptible to activation of the ISR pathway.

Trials and the Future:

Trials seem to be hindered by several confounding factors. First, it seems common to simultaneously inoculate plants with pathogens activating opposing pathways. Although this approach may be a time saver with conventional fungicides, it creates negative crosstalk between induced resistance pathways. Since the plant prioritizes biotrophic/hemibiotrophic pathogen attack over that of nectrotrophic pathogens, high disease pressure by a necrotrophs and biotrophs simultaneously creates that appearance that the product is only effective against the biotroph/hemibiotroph. For example, although it is well documented that *Bacillus spp*. is effective against *Alternaria spp*., trials that simultaneously inoculated with *Oidium* or other biotroph/hemibiotroph resulted in control of only the biotroph. In the future, trials should aim to select pathogens with similar lifestyles if multiple pathogens are to be used simultaneously in order to avoid confusing results. Furthermore, researchers should attempt to provide sufficient time (at least 3-7 days) for proper induction to occur before inoculating with high levels of disease—otherwise, the advantage of a product that primes defenses may be lost to many of the plants in the experiment.

Next, water sprays containing only inducers tends to increase the severity and incidence of foliar diseases. This is because current trials are usually applying inducers weekly, and these products generally have no direct antimicrobial activity, resulting in enhanced dispersal of inoculum and increased leaf wetness that enhances opportunity for penetration and sporulation. If the control is completely untreated, then the results essentially reflect higher disease pressures in the treatment areas, confounding the results. It has been demonstrated multiple times that utilizing a product with direct antimicrobial effects in combination with IR activators provides much greater control. Therefore, an alternative might be to either mix induced resistance activators with antimicrobial agents such as OMRI approved tea tree oil, neem oil, sesame oil, or copper products. Another option is to avoid wetting leaf surfaces at all by using the appropriate inducers as drenches, wherever possible. Also, it should be noted that weekly applications of inducers is most likely not required for optimal action. Biweekly applications are probably more than sufficient, and additional applications may keep relatively healthy plants from turning off defenses when they are not needed. The field trial data available is miniscule and biased toward conventional products. Despite all this, there have still been successes, especially for organic disease control.

It is reasonable to expect an additional time period of a decade or more before plant immunity is understood at a level that allows for the effective use of induced resistance as a

disease management strategy. Despite the vast body of work in existence, there are still very few cold, hard facts available to work with in this system, and it is unlikely that widespread adoption of this technique will be commonplace in the near future. Nonetheless, IR is a promising technology that will continue to be intensely studied, and may someday supplant breeding methods of producing resistance.

WORKS CITED

Adie, B., et al. "Modulation of Plant Defenses by Ethylene." *Journal of Plant Growth Regulation* 26.2 (2007), 160-177.

Abeysinghe, Saman. "Induced Systemic Resistance (ISR) in Bean (*Phaseolus vulgaris L*.) Mediated by Rhizobacteria Against Bean Rust Caused by *Uromyces appendiculatus* Under Greenhouse and Field Conditions." *Archives of Phytopathology and Plant Protection* 42.11 (2009), 1079-1087.

Achuo, E. A., et al. "The Salicylic Acid-Dependent Pathway is Effective Against Different Pathogens in Tomato and Tobacco." *Plant Pathology* 53.1 (2004), 65-72.

Agrios, George N. *Plant Pathology*. 5th ed. New York: Elsevier Academic Press, 2005.

Ahmed, A. S., Sanchez, C. P. & Candela, M. E. Evaluation of induction of systemic resistance in pepper plants (*Capsicum annuum*) to *Phytopthora capsici* using *Trichoderma harzianum* and its relation with capsidiol accumulation. *Eur. J. Plant Pathol.* 106, 817–824 (2000).

Ahn, I., *et al*. "Vitamin B1-Induced Priming Is Dependent on Hydrogen Peroxide and the NPR1 Gene in Arabidopsis1" *Plant Physiology* 143 (2007), 838–848.

Amruthesh, K. M., et al. "Unsaturated Fatty Acids from Zoospores of *Sclerospora graminicola* Induce Resistance in Pearl Millet." *European Journal of Plant Pathology* 111.2 (2005), 125-137.

Amzalek, E., and Cohen, Y. "Comparative Efficacy of Systemic Acquired Resistance-Inducing Compounds Against Rust Infection in Sunflower Plants." *Phytopathology* 97.2 (2007), 79-186.

Antonio, G. F., *et al*. "Formulation of a Liquid Fertilizer for Sorghum (*Sorghum bicolor (L.) Moench*) Using Vermicompost Leachate." *Bioresource Technology* 99.14 (2008), 6174-6180.

Audenaert, K., et al. "Induction of Systemic Resistance to *Botrytis cinerea* in Tomato by *Pseudomonas aeruginosa* 7NSK2: Role of Salicylic Acid, Pyochelin, and Pyocyanin." *Molecular Plant-Microbe Interactions* 15.11 (2002), 1147-1156.

Baldwin, I. T. "Jasmonate-Induced Responses Are Costly but Benefit Plants Under Attack in Native Populations." *Proceedings of the National Academy of Sciences of the United States of America* 95.14 (1998), 8113-8118.

Baudoin, Anton. *Plant Disease Management PPWS 5204*. Virginia Tech. Web, 2010

Baysal, O., et al. "Enhanced Tomato Resistance to Bacterial Canker by Application of Turtle Oil." *Journal of General Plant Pathology* 71.3 (2005), 204-210.

Bengtsson, M., et al. "Comparative Studies on the Effects of a Yucca Extract and Acibenzolar-S-methyl (ASM) on Inhibition of *Venturia inaequalis* in Apple Leaves." *European Journal of Plant Pathology* 124.2 (2009), 187-198.

Bigirimana, J. *et al.* Induction of systemic resistance on bean (*Phaseolus vulgaris*) by *Trichoderma harzianum*. *Med. Fac. Landbouww. Univ. Gent* 62 (1997), 1001–1007.

Bokshi, A. I., *et al*. "Pre-Harvest Application of 2,6-dichloroisonicotinic acid, (beta)-aminobutyric acid or Benzothiadiazole to Control Post-Harvest Storage Diseases of Melons by Inducing Systemic Acquired Resistance (SAR)." *Journal of Horticultural Science and Biotechnology* 81.4 (2006), 700-706.

Bolton, M. D. "Primary Metabolism and Plant Defense—Fuel for the Fire." *MPMI* 22.5 (2009), 487-497.

Bostock, R. M. "Signal Crosstalk and Induced Resistance: Straddling the Line Between Cost and Benefit." *Annual Review of Phytopathology* 43 (2005), 545-580.

Brady, N. C., and Weil, R. R. *The Nature and Properties of Soils*. 14th ed. Upper Saddle River: Prentice Hall, 2008. Print.

Carlos, G. R., et al. "Vermicomposting leachate (Worm Tea) as Liquid Fertilizer for Maize (Zea mays L.) Forage Production." Asian *Journal of Plant Sciences* 7.4 (2008), 360-367.

Choh, Y., et al. "Exposure of Lima Bean Leaves to Volatiles from Herbivore-Induced Conspecific Plants Results In Emission of Carnivore Attractants: Active or Passive Process?" *Journal of Chemical Ecology* 30.7 (2004), 1305-1317.

Choudhary, D. K., and Johri, B. N. "Interactions of *Bacillus spp.* and Plants - With special Reference to Induced Systemic Resistance (ISR)." *Microbiological Research* 164.5 (2009), 493-513.

Collins, H., *et al*. "Effect of Foliar Applied Plant Elicitors on Microbial and Nematode Populations in the Root Zone of Potato." *Communications in Soil Science and Plant Analysis*, 37.11-12 (2006), 1747-1759.

Conrath, U., et al. "Priming: Getting Ready for Battle." *Molecular Plant-Microbe Interactions* 19.10 (2006), 1062-1071.

Cooper, W. C., et al. "Acquired and r-gene-mediated resistance against the potato aphid in tomato." *Journal of Chemical Ecology* 30.12 (2004), 2527-2542.

Cortes-Barco, A. M., et al. "Induced Systemic Resistance Against Three Foliar Diseases of *Agrostis stolonifera* by (2R,3R)-Butanediol or an Isoparaffin Mixture." *Annals of Applied Biology* 157.2 (2010), 179-189.

Da Rocha, A. B., and Hammerschmidt, R. "History and Perspectives on the Use of Disease Resistance Inducers in Horticultural Crops." *Hort Technology* 15.3 (2005), 518-529.

De Meyer, G., Bigirimana, J., Elad, Y. & Hofte, M. Induced systemic resistance in *Trichoderma harzianum* T39 biocontrol of *Botrytis cinerea*. *Eur. J. Plant Pathol.* **104**, 279–286 (1998).

De Vos, M. *et al*. "Plants Under Attack: Multiple Interactions with Insects and Microbes." *Plant Signaling and Behavior* 2.6 (2007), 527-529.

Dillard, H., et al. "Evaluation of Foliar Sprays for Control of White and Gray Mold in Snap Beans, Field 48, 1998." *Plant Management Network F & N Tests* 55 (1998), 144.

Doares, S. H., et al. "Salicylic Acid Inhibits Synthesis of Proteinase Inhibitors in Tomato Leaves Induced by Systemin and Jasmonic Acid." *Plant Physiology* 108.4 (1995), 1741-1746.

Edwards, C. A., et al. "Suppression of Green Peach Aphid (*Myzus persicae) (Sulz*.), Citrus Mealybug (*Planococcus citri) (Risso*), and Two Spotted Spider Mite (*Tetranychus urticae*) (Koch.) Attacks on Tomatoes and Cucumbers by Aqueous Extracts from Vermicomposts." *Crop Protection* 29.1 (2010), 80-93.

Elsen, A., et al. "AMF-Induced Biocontrol Against Plant Parasitic Nematodes in Musa sp.: A Systemic Effect." *Mycorrhiza* 18.5 (2008), 251-256.

Faoro, F. *et al*. "Chemical-Induced Resistance Against Powdery Mildew in Barley: The Effects of Chitosan and Benzothiadiazole." *BioControl* 53.2 (2008), 387-401.

Felton, G. W. *et al*. "Inverse Relationship Between Systemic Resistance of Plants to Microorganisms and to Insect Herbivory." *Current Biology* 9.6 (1999), 317-320.

Flors, V., et al. "Interplay Between JA, SA and ABA Signalling During Basal and Induced Resistance Against Pseudomonas Syringae and *Alternaria brassicicola*." Plant Journal 54.1 (2008), 81-92.

Francia, D., et al. "Do Pathogen-Specific Defense Mechanisms Contribute to Wound-Induced Resistance in Tomato?" *Plant Signaling and Behavior* 3.5 (2008), 340-341.

Garcia-Mina, J.M., et al. "Alleviation of Verticillium Wilt in Pepper (*Capsicum annuum* L) by

Using the Organic Amendment COA H of Natural Origin." *Sci Hortic* 101 (2004), 23–37.
Ghosheh, H.Z., et al. "Constraints in Implementing *Biological Weed Control*: A Review." *Weed Biology and Management* 5.3 (2005), 83-92.
Goellner, K., and Conrath, U. "Priming: It's All the World to Induced Disease Resistance." *European Journal of Plant Pathology* 121.3 (2008), 233-242.
Gonsior, G., et al. "Induced Resistance - An Innovative Approach to Manage Branched Broomrape (*Orobanche ramosa*) in Hemp and Tobacco." *Weed Science* 52.6 (2004), 1050-1053.
Griebel, T., and Zeier, J. "Light Regulation and Daytime Dependency of Inducible Plant Defenses in Arabidopsis: Phytochrome Signaling Controls Systemic Acquired Resistance Rather Than Local Defense." *Plant Physiology* 147 (2008), 790–801.
Guo, X., and Stotz, H. U. "Defense Against *Sclerotinia sclerotiorum* in *Arabidopsis* is Dependent on Jasmonic Acid, Salicylic Acid, and Ethylene Signaling." *Molecular Plant-Microbe Interactions* 20.11 (2007), 1384-1395.
Gutjahr, C., and Paszkowski, U. "Weights in the Balance: Jasmonic Acid and Salicylic Acid Signaling in Root-Biotroph Interactions." *Molecular Plant-Microbe Interactions* 22.7 (2009), 763-772.
Harman, G. E., et al. "Trichoderma Species - Opportunistic, Avirulent Plant Symbionts." *Nature Reviews Microbiology* 2.1 (2004), 43-56.
---. Interactions between *Trichoderma harzianum* strain T22 and maize inbred line Mo17 and effects of this interaction on diseases caused by *Pythium ultimum* and *Colletotrichum graminicola*. *Phytopathology* (in the press).
Hashem, M., and Farrage, E. "Induction of Resistance in *Phaseolus vulgaris L*. cv. *Lima* Against Some Soil-Borne Fungal Pathogens by Pre-inoculation with Tobacco Necrosis Virus (TNV) Reacting Hypersensitivity." *Archives of Phytopathology & Plant Protection* 42.7 (2009), 676-687.
Hasky-Gunther, K., et al. "Resistance Against the Potato Cyst Nematode *Globodera pallida* Systemically Induced by the Rhizobacteria *Agrobacterium radiobacter* (G12) and *Bacillus sphaericus* (B43)." *Fundamental and Applied Nematology* 21.5 (1998), 511-517.
Herman, M., *et al*. "Induction of Plant Defense Gene Expression by Plant Activators and *Pseudomonas syringae pv. tomato* in Greenhouse-Grown Tomatoes." Phytopathology 98.11 (2008), 226-1232.
Hong, J. K., et al. "Induction of Local and Systemic Resistance to *Colletotrichum coccodes* in Pepper Plants by DL- beta -amino-n-butyric acid." *Journal of Phytopathology* 147.4 (1999), 193-198.
Howe, G. A., and Jander, G. "Plant Immunity to Insect Herbivores." *Annual Review of Plant Biology* 59 (2008), 41-66.
Howell, C. R., Hanson, L. E., Stipanovic, R. D. & Puckhaber, L. S. Induction of terpenoid synthesis in cotton roots and control of *Rhizoctonia solani* by seed treatment with *Trichoderma virens*. *Phytopathology* 90 (2000), 248–252.
Iriti, M., and Faoro, F. "Chitosan as a MAMP, Searching for a PRR." *Plant Signaling and Behavior* 4.1 (2009), 66-68.
Ishikawa, R., et al. "Foliar Spray of Validamycin A or Validoxylamine A Controls Tomato Fusarium Wilt." *Phytopathology* 95.10 (2005), 1209-1216.
Jayaraj, J., et al. "Seaweed Extract Reduces Foliar Fungal Diseases on Carrot." *Crop Protection*

27.10 (2008), 1360-1366.
Karthikeyan, G., et al. "*Pseudomonas fluorescens* Mediated Systemic Resistance Against Urdbean Leaf Crinkle Virus in Blackgram (*Vigna mungo*)." *Archives of Phytopathology and Plant Protection* 42.3 (2009), 201-212.
Kathiresan, A., et al. "Gamma-Aminobutyric Acid Stimulates Ethylene Biosynthesis in Sunflower ." *Plant Physiology* 115.1 (1997), 129-135.
Kempel, A., et al. "Support From the Underground: Induced Plant Resistance Depends on Arbuscular Mycorrhizal Fungi." *Functional Ecology* 24,2 (2010), 293-300.
Khan, J., *et al*. "Systemic Resistance Induced by *Trichoderma hamatum* 382 in Cucumber Against Phytophthora Crown Rot and Leaf Blight." *Plant Disease* 88.3 (2004) 280-286.
Kishimoto, K., et al. "Volatile C6-Aldehydes and Allo-ocimene Activate Defense Genes and Induce Resistance Against *Botrytis cinerea* in *Arabidopsis thaliana*. *Plant and Cell Physiology* 46.7 (2005), 1093-1102.
Klarzynski, O., et al. "Sulfated Fucan Oligosaccharides Elicit Defense Responses in Tobacco and Local and Systemic Resistance Against Tobacco Mosaic Virus." *Molecular Plant-Microbe Interactions* 16.2 (2003), 115-122.
Kloepper, J. W., et al. "Induced Systemic Resistance and Promotion of Plant Growth by *Bacillus spp*." *Phytopathology* 94.11 (2004), 1259-1266.
Ko, W. H., et al. "Effects of Sunflower Oil on Tomato Powdery Mildew Caused by *Oidium neolycopersici."* *Journal of Phytopathology*, 151.3 (2003), 144-148.
Koike, N. *et al.* Induction of systemic resistance in cucumber against several diseases by plant growth-promoting fungi: lignification and superoxide generation. *Eur. J. Plant Pathol.* 107, 523–533 (2001).
Kone, D., et al. "Evaluation of Systemic Acquired Resistance Inducers for Control of *Phytophthora capsici* on Squash." *Crop Protection* 28.6 (2009), 533-538.
Kumar, T., et al. "Rhizosphere Competent *Pseudomonas aeruginosa* in the Management of *Heterodera cajani* on Sesame." *World Journal of Microbiology and Biotechnology* 25.2 (2009), 277-285.
Leyva, M. O., et al. "Preventive and Post-Infection Control of *Botrytis cinerea* in Tomato Plants by Hexanoic Acid." *Plant Pathology* 57.6 (2008), 1038-1046.
Liu, H., *et al*. "Improving Disease Resistance in Peach Fruit During Storage Using Benzo-(1,2,3)-thiodiazole-7-carbothioic acid S-methyl ester (BTH)." *Journal of Horticultural Science and Biotechnology* 80.6 (2005), 736-740.
Lo, C.-T., Liao, T. F. & Deng, T. C. Induction of systemic resistance of cucumber to cucumber green mosaic virus by the root-colonizing *Trichoderma* spp. *Phytopathology* **90** (Suppl.), S47 (2000).
Malandraki, I., *et al*. "Thermal Inactivation of Compost Suppressiveness Implicates Possible Biological Factors in Disease Management." *Biological Control* 44 (2008), 180–187.
Mishina, T. E., and Zeier, J. "Pathogen-Associated Molecular Pattern Recognition Rather Than Development of Tissue Necrosis Contributes to Bacterial Induction of Systemic Acquired Resistance in Arabidopsis." *Plant Journal* 50.3 (2007), 500-513.
Murphy, J. F., et al. "Plant Growth-Promoting Rhizobacterial Mediated Protection in Tomato Against Tomato Mottle Virus." *Plant Disease* 84.7 (2000), 779-784.
Nair, Chitra B., et al. "Mitigation of Growth Retardation Effect of Plant Defense Activator, Acibenzolar- S -Methyl, in *Amaranthus* Plants by Plant Growth-Promoting ."

Nga, T., et al. "Rhizobacterially Induced Protection of Watermelon Against *Didymella bryoniae*." *Journal of Applied Microbiology* 109.2 (2010), 567-582.

O'Donnell, P. J., et al. "Ethylene as a Signal Mediating the Wound Response of Tomato Plants." Science 274.5294 (1996), 1914-1917.

Olson, H. A., and Benson, D. M. "Induced Systemic Resistance and the Role of Binucleate Rhizoctonia and *Trichoderma hamatum* 382 in Biocontrol of Botrytis blight in Geranium." Biological Control 42.2 (2007) 233-241.

Ozer, Nuray, and Koycu, N.. "The Ability of Plant Compost Leachates to Control Blackmold (*Aspergillus niger*) and to Induce the Accumulation of Antifungal Compounds in Onion Following Seed Treatment." *BioControl* 51 (2006), 229–243.

Pajot, E., et al. "Phytogard and DL-(Beta)-Amino Butyric Acid (BABA) Induce Resistance to Downy Mildew (*Bremia lactucae*) in Lettuce (*Lactuca sativa L*)." *European Journal of Plant Pathology* 107.9 (2001), 861-869.

Parbery, D. G. "Trophism and the Ecology of Fungi Associated with Plants." *Biological Reviews of the Cambridge Philosophical Society* 71.3 (1996), 473-527.

Perez, L., *et al.* "Efficacy of Acibenzolar-S-methyl, an Inducer of Systemic Acquired Resistance Against Tobacco Blue Mould Caused by *Peronospora hyoscyami f. sp. tabacina*." *Crop Protection* 22.2 (2003) 405-413.

Perez-de-Luque, A., *et al*. "Crenate Broomrape Control in Pea by Foliar Application of Benzothiadiazole (BTH)." *Phytoparasitica* 32.1(2004), 21-29.

Plymale, R. C., *et al*. "Induction of Systemic Acquired Resistance in Cotton Foliage Does Not Adversely Affect the Performance of an Entomopathogen." *Journal of Chemical Ecology* 33.8 (2007), 1570-1581.

Raccke, I. C., et al. "Yeast Increase Resistance in *Arabidopsis* against *Pseudomonas syringae* and Botrytis cinerea by Salicylic Acid-Dependent as Well as Independent Mechansims." *Mol Plant Microbe Interact* 19.10 (2006), 1138-46.

Reuveni, R., and Reuveni M. "Foliar-Fertilizer Therapy - A Concept in Integrated Pest Management." *Crop Protection* 17.2 (1998), 111-118.

Robert-Seilaniantz, A., et al. "Pathological Hormone Imbalances." *Current Opinion in Plant Biology* 10.4 (2007), 372-379.

Rohwer, C. L., and Erwin, J. E. "Horticultural Applications of Jasmonates: A Review." *Journal of Horticultural Science and Biotechnology* 83.3 (2008), 283-304.

Sanz-Alferez, S., *et al*. *"SAR* Induction in Tomato Plants is Not Effective Against Root-Knot Nematode Infection." *European Journal of Plant Pathology* 120.4 (2008), 417-425.

Scheuerell, S. J., and Mahaffee, W. F. "Compost Tea as a Container Medium Drench for Suppressing Seedling Damping-Off Caused by *Pythium ultimum*." *Phytopathology* 94.11 (2004), 1156- 1163.

---. "Suppression of Seedling Damping-Off Caused by *Pythium ultimum, P. irregulare*, and *Rhizoctonia solani* in Container Media Amended with a Diverse Range of Pacific Northwest compost." Phytopathology 95.3 (2005), 306-315.

---. "Variability Associated with Suppression of Gray Mold (*Botrytis cinerea)* on Geranium by Foliar Applications of Nonaerated and Aerated Compost Teas." Plant Disease 90.9 (2006), 1201-1208.

Seaman, A. *Efficacy of OMRI-approved products for tomato foliar disease control.* New York State Integrated Pest Management Program 129 (2003), 164–167.

Segarra, G., et al. "MYB72, a Node of Convergence in Induced Systemic Resistance Triggered

by a Fungal and a Bacterial Beneficial Microbe." *Plant Biology* 11.1 (2009), 90-96.
Serrano, A., *et al*. "Chitinase and Peroxidase Activities in Sunflower Hypocotyls: Effects of BTH and Inoculation with *Plasmopara halstedii*." *Biologia Plantarum* 51.1 (2007), 149-152.
Shi, S., et al. "Effects of Exogenous GABA on Gene Expression of *Caragana intermedia* Roots Under NaCl Stress: Regulatory Roles for H2O2 and Ethylene Production." *Plant, Cell and Environment* 33.2 (2010), 149-162.
Shimizu, M., et al. "Disease Resistance Induced by Nonantagonistic Endophytic *Streptomyces spp*. on Tissue-Cultured Seedlings of Rhododendron." *Journal of General Plant Pathology* 72.6 (2006), 351-354.
Shoresh, M., and Harman, G. E. "The Molecular Basis of Shoot Responses of Maize Seedlings to *Trichoderma harzianum* T22 Inoculation of the Root: A Proteomic Approach." *Plant Physiology* 147.4 (2008), 2147-2163.
Siddiqui, Y., *et al*. "Bio-Potential of Compost Tea from Agro-Waste to Suppress *Choanephora cucurbitarum L*. the Causal Pathogen of Wet Rot of Okra." *Biological Control* 49.1 (2009), 38-44.
Siddiqui, I. A., and Shaukat, S. S. "Suppression of Root-Knot Disease by *Pseudomonas fluorescens* CHA0 in Tomato: Importance of Bacterial Secondary Metabolite, 2,4-diacetylpholoroglucinol." *Soil Biology and Biochemistry* 35.12 (2003), 1615-1623.
Spoel, S. H., and Dong, X. "Making Sense of Hormone Crosstalk during Plant Immune Responses." *Cell Host and Microbe* 3.6 (2008), 348-351.
Spoel, S. H., et al. "Regulation of Tradeoffs Between Plant Defenses Against Pathogens with Different Lifestyles." *Proceedings of the National Academy of Sciences of the United States of America* 104.47 (2007), 18842-18847.
Sundar, A., *et al*. "Induction of Systemic Acquired Resistance (SAR) Using Synthetic Signal Molecules Against *Colletotrichum falcatum* in Sugarcane." *Sugar Tech* 11.3 (2009), 274-281.
Terrry, L. A., and Joyce, D. C. "Suppression of Grey Mould on Strawberry Fruit with the Chemical Plant Activator Acibenzolar." Pest Management Science 56.11 (2000), 989-992.
Thaler, J. S., et al. "Antagonism Between Jasmonate- and Salicylate-Mediated Induced Plant Resistance: Effects of Concentration and Timing of Elicitation on Defense-Related Proteins, Herbivore, and Pathogen Performance in Tomato." *Journal of Chemical Ecology* 28.6 (2002), 1131-1159.
---. "The Role of the Jasmonate Response in Plant Susceptibility to Diverse Pathogens with a Range of Lifestyles." Plant Physiology 135 (2004), 530-538.
Tomczyk, A. "Increasing Cucumber Resistance to Spider Mites by Biotic Plant Resistance Inducers." *Biological Lett*. 43.2 (2006), 381-387.
Trouvelot, S., et al. "A (beta)-1,3 Glucan Sulfate Induces Resistance in Grapevine Against *Plasmopara viticola* Through Priming of Defense Responses, Including HR-Like Cell Death." *Molecular Plant-Microbe Interactions* 21.2 (2008), 232-243.
Tsuda, K., et al. "Network Properties of Robust Immunity in Plants." *PLoS Genetics* 5.12 (2009).
Veronesi, C., *et al*. "Acibenzolar-S-methyl Induces Resistance in Oilseed Rape (*Brassica napus L.)* Against Branched Broomrape (*Orobanche ramosa L.)*." Crop Protection 28.1 (2009), 104-108.

Vavrina, C. S., et al. "Greenhouse Screening of Commercial Products Marketed as Systemic Resistance and Plant Growth Promotion Inducers." *HortScience* 39.2 (2004), 433-437.

Valenzuela-Soto, J. H., et al. "Inoculation of Tomato Plants (*Solanum lycopersicum*) with Growth-Promoting *Bacillus subtilis* Retards Whitefly *Bemisia tabaci* Development." *Planta* 231.2 (2010), 397-410.

Van der Ent, Sjoerd. *Transcriptional Regulators of Rhizobacteria-Induced Systemic Resistance.* Diss. Utrecht University, 2007. Print.

Van Hulten, M., et al. "Costs and benefits of priming for defense in Arabidopsis." *Proceedings of the National Academy of Sciences of the United States of America* 103.14 (2006), 5602-5607.

Van Oosten, V. R, et al. "Differential Effectiveness of Microbially Induced Resistance Against Herbivorous Insects in *Arabidopsis*." *Mol Plant Microbe Interact* 21.7 (2008), 919-30.

Van Wees, S. C., et al. "Enhancement of Induced Disease Resistance by Simultaneous Activation of Salicylate- and Jasmonate-Dependent Defense Pathways in *Arabidopsis thaliana*." *Proceedings of the National Academy of Sciences of the United States of America* 97.15 (2000), 8711-8716.

Verhagen, Bas W. M., et al. "Pseudomonas Spp.-Induced Systemic Resistance to *Botrytis cinerea* is Associated with Induction and Priming of Defense Responses in Grapevine." *Journal of Experimental Botany* 61.1 (2010), 249-260.

Vicedo, B., et al. "Hexanoic Acid-Induced Resistance Against *Botrytis cinerea* in Tomato Plants." *Molecular Plant-Microbe Interactions* 22.11 (2009), 1455-1465.

Walters, D. and Heil, M. "Costs and Trade-Offs Associated with Induced Resistance." *Physiological and Molecular Plant Pathology* 71.1-3 (2007), 3-17.

Wu, C., et al. "L-Glutamine Inhibits Beta-Aminobutyric Acid-Induced Stress Resistance and Priming in *Arabidopsis*." *Journal of Experimental Botany* 61.4 (2010), 995-1002.

Yasuda, M., *et al*. "Antagonistic Interaction between Systemic Acquired Resistance and the Abscisic Acid–Mediated Abiotic Stress Response in Arabidopsis." *The Plant Cell* 20 (2008), 1678–1692.

Yedidia, I. et al. Concomitant induction of systemic resistance to Pseudomonas syringae pv. lachrymans in cucumber by *Trichoderma asperellum* (T-203) and the accumulation of phytoalexins. *Appl. Environ. Microbiol*.

Zarate, S. I, et al. "Silverleaf Whitefly Induces Salicylic Acid Defenses and Suppresses Effectual Jasmonic Acid Defenses." *Plant Physiology* 143.2 (2007), 866-875.

Zhang, W., *et al*. "Compost and Compost Water Extract-Induced Systemic Acquired Resistance in Cucumber and Arabidopsis." *Phytopathology* 88.5 (1998) 450-455.

Zimmerli, L., et al. "Potentiation of Pathogen-Specific Defense Mechanisms in Arabidopsis by Beta -Aminobutyric Acid." *Proceedings of the National Academy of Sciences of the United States of America* 97.23 (2000), 12920-12925.