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Induced Plant Defense: Evolution of Induction and Adaptive Phenotypic Plasticity

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Abstract

Induced defenses may evolve in natural plant populations where heritable variation in inducibility affects plant fitness. Although this has not been documented for any plant – parasite system, genetic variation in induction has been recently reported for several plant-parasite systems. Correlations between induction and fitness in variable parasite environments suggest that inducibility may indeed be subject to selection, however, these studies focussed on phenotypic (not genetic) correlations. Our current state of knowledge suggests that induced defenses may be an example of adaptive plasticity, where defenses enhance plant fitness when parasites are present, and reduce fitness when parasites are absent. Future work that focuses on consequences of genetic variation in induction and macro-evolutionary trends in inducible versus constitutive defense will help advance our understanding of the evolutionary biology of induced plant defense.

Introduction

Induced plant "responses" to attack consist of any active or passive change in the plant following herbivory or infection. Induced plant "resistance" refers to reduced preference, performance, or pathogenicity of the attacker on induced plants compared to controls. Note that this definition of induced resistance, originally advocated by Karban and Myers (1989), is characterized by the behavior or fitness of the plant attacker, not by attributes of the plant itself. The focus of this chapter is induced plant "defense" rather than resistance. Defense is a term reserved for situations where plants having some resistance character have higher fitness than plants lacking that character. Measuring characteristics that affect plant performance are central to the goals of applied entomology and plant pathology, specifically reducing pest levels below some

threshold above which pests cause economic losses. In addition, understanding the factors that influence plant fitness is central to understanding basic ecological and evolutionary principles of plant-parasite interactions. This chapter will focus on two aspects of induced responses and their fitness consequences for plants: 1) Are induced defenses subject to natural selection? and 2) Are their phenotypic benefits and costs associated with induced defenses, indicating that they are an example of adaptive plasticity?

Microevolution of Induced Defenses

Variation in induced resistance (within plants and across populations of plants) can be caused by many biotic and abiotic factors of the environment (Agrawal and Karban 1998). However, induced plant resistance can evolve by natural selection only if there is heritable variation for induction that affects plant fitness. Such genetic variability is detected through a statistical interaction between genetically related families (or clones) of plants and their response to herbivory or pathogen attack. In this review, induced resistance is considered a single trait, in most cases determined by a negative effect on the herbivore or pathogen. Clearly, many genes and pathways are involved in most plant responses to attack. For simplicity, and for the purposes of defining heritability for a suite of traits that may be subject to natural selection, induced resistance is considered a single trait.

In wild plants, 14 plant-herbivore systems have been examined for genetic variation in inducibility (Table 1). Varietal variation in induced resistance against herbivores and pathogens of crop plants has also been reported for several systems. To my knowledge only one wild plant - pathogen system has been examined for genetic variation in induced resistance (Dirzo and Harper 1982). In this case, clover plants exhibited genetic variation in the pre-formed cyanogenic glucoside response to leaf damage.

No study to date has documented fitness consequences for the plant involving genetic variation in induced resistance. Circumstantial evidence that induced resistance may be evolving in response to herbivore attack was provided by Dirzo and Harper (1982), Raffa (1991) and Zangerl and colleagues (Zangerl and Berenbaum 1990, Zangerl and Rutledge 1996, Zangerl, this volume). Although it is likely that induced responses are, indeed, subject to natural selection, we can only infer this from the evidence of current genetic variation in the trait.

Why is there genetic variation in induced plant resistance present in natural populations? The least interesting answer to this question is that genetic variation in induced resistance does not directly affect plant fitness, and induced resistance is a trait that is genetically correlated with other traits that do affect plant fitness. For example, *Brassica rapa* plants with genetically divergent levels of myrosinase (an enzyme involved in hydrolyzing defense products in mustards) showed variation in fitness in a field experiment (Siemens and Mitchell-Olds 1998). However, this study showed that induced levels

Table 1. Systems for which genetic variation in induced resistance has been reported.

Plant	Latin name	Type	Mechanism	Parasite affected	Latin name	Reference
Goldenrod	*Solidago altissima*	Wild	Hypersensitivity	Fly eggs	*Eurosta solidaginis*	Abrahamson and Weis 1997
Wild parsnip	*Pastinaca sativa*	Wild	Furanocoumarins	Moth larvae	*Trichoplusia ni*	Zangerl and Berenbaum 1990
Black mustard	*Brassica nigra*	Wild	Hypersensitivity	Butterfly eggs	*Pieris spp.*	Shapiro and DeVay 1987
Rapid cycling mustard	*Brassica rapa*	Wild	Glucosinolates	Moth larvae	*Plutella xylostella*	Siemens and Mitchell-Olds 1998
Wild radish	*Raphanus sativus*	Wild	?	Aphids	*Myzus persicae*	Agrawal 1998, 1999a
Birch	*Betula pubesens*	Wild	?	Moth larvae	*Epirrita autumnata*	Haukioja and Hanhimaki 1985
Sunflower	*Helianthus annuus*	Wild	Coumarins	Beetles	*Zygogramma exclamationis*	Roseland and Grosz 1997
Quaking aspen	*Populus tremuloides*	Wild	?[1]	Moth larvae	*Lymantria dispar*	Osier and Lindroth, pers. comm.
Narrow leaf plantain	*Plantago lanceolata*	Wild	Iridoid glycosides	N/t	N/t	Bowers and Stamp 1993
Ponderosa pine	*Pinus ponderosa*	Wild	Monoterpenes/ necrosis	Beetles – fungus complex	*Scolytus ventralis, Trichosporium sybioticum*	Raffa 1991
Hounds tongue	*Cynoglossum officinale*	Wild	Pyrrolizidine alkaloids	N/t	N/t	van Dam and Vrieling 1994
Willow	*Salix myrsinifolia*	Wild	Salicortin	N/t	N/t	Julkunen-Tiitto et al. 1995
Turnera	*Turnera ulmifoli*	Wild	Cyanogenesis	N/t	N/t	Schappert and Shore 1995
White clover	*Trifolium repens*	Wild	Cyanogenesis	Several herbivores		Dirzo and Harper 1982
				Rust fungus	*Uromyces trifolii*	
Cotton	*Gossypium hirsutum*	Crop	?	Mites	*Tetranychus urticae*	Brody and Karban 1992
Soybean	*Glycine max*	Crop	?	Beetles	*Epilachna varivestis*	Underwood 1998, unpublished
Grape	*Vitis vinifera*	Crop	?	Mites	*Tetranychus spp.*	English-loeb et al. 1998
Cucumber	*Cucumis sativus*	Crop	Cucurbitacin C	Mites	*Tetranychus urticae*	Agrawal et al. 1999
Wheat	*Triticum aestivum*	Crop	Several enzymes	N/t	N/t	Rybka et al. 1998
			Hydroxamic acids	N/t	N/t	Gianoli et al. 1997

Radish	*Raphanus sativus*	Crop	Necrosis	Leaf spot	*Xanthomonas campestris*	Kamoun et al. 1993
Tomato	*Lycopersicon esculentum*	Crop	PR proteins	Fungal pathogen	*Alternaria solani*	Lawrence et al. 1996
Triticale	*Triticum X Secale*	Crop	Several enzymes	N/t	N/t	Rybka et al. 1998
Cacao	*Theobroma cacao*	Crop	?	Black pod disease	*Phytophthora spp.*	Pires et al. 1997
Pearl millet	*Pennisetum glaucum*	Crop	Hypersensitivity	Downy mildew	*Sclerospora graminicola*	Geetha et al. 1996
Chinese cabbage	*Brassica campestris*	Crop	Glucosinolates	Clubroot disease	*Plasmodiophora brassicae*	Ludwig-Mueller et al. 1997
Tobacco	*Nicotiana tabacum*	Crop	Necrosis	Fungal pathogen	*Phytophthora parasitica*	Bonnet et al. 1996
Barley	*Hordeum vulgare*	Crop	?	Powdery mildew	*Erysiphe graminis*	Newton and Dashwood 1998
Parsnip	*Pastinaca sativa*	Crop	Furanocoumarins	N/t	N/t	Cerkauskas and Chiba 1990

? = unknown
N/t = not tested
[1] not phenolic glycosides, tannins, nitrogen, or water content

of glucosinolates were positively genetically correlated with constitutive levels of glucosinolates. Thus, it is unknown whether induced defenses themselves were under selection, or simply carried along as a correlated trait.

It is likely that induced defense is a trait subject to natural selection in natural populations. However, evolution may very well be acting on induced and constitutive resistance as correlated traits. Theory predicts that there should be a negative genetic correlation between constitutive resistance and inducibility of plants (Brody and Karban 1992, Herms and Mattson 1992). In two studies, levels of constitutive phytochemicals were positively correlated to inducibility of phytochemicals (Zangerl and Berenbaum 1990, Siemens and Mitchell-Olds 1997), while in a third study there was no relationship (Brody and Karban 1992). It is currently unknown whether or not constitutive and induced resistance can be genetically uncoupled. However, many traits with strong phenotypic correlations can be uncoupled with only a few generations of strong selection (e.g., Stanton and Young 1994).

The experimental uncoupling of constitutive and induced resistance will allow us to understand several basic and applied aspects of plant defense. Experimental tests of the benefits of induced resistance over constitutive resistance (and vice versa) in different environments will require this uncoupling. Several non-mutually exclusive alternative benefits of induction have been proposed and are reviewed elsewhere (Karban and Baldwin 1997, Karban et al. 1997, Agrawal and Karban 1998). In addition, understanding how or why induced resistance may be a useful strategy in agriculture will require it to be tested within different backgrounds of constitutive resistance.

As noted above, no studies have documented the fitness consequences for plants with genetically variable induced resistance. However, a few studies have examined the fitness consequences of plants expressing induced versus uninduced phenotypes. In other words, studies have asked if induced plants have higher fitness than uninduced plants when growing in an environment with herbivores; and, if induced plants have lower fitness than uninduced plants when growing in an environment without herbivores. These studies have linked the plant phenotype to plant fitness. This is important for several basic and applied reasons (discussed in the following sections), however, these phenotypic correlations do not directly address the consequences of genetic variation and the evolution of induction. Some authors, have argued and provided evidence that phenotypic correlations are good estimates of genetic correlation (e.g., Cheverud 1988), although this remains controversial.

The Cost-Benefit Framework and Genotype-by-Environment Interaction

The cost-benefit framework is a long standing construct of evolutionary biology that attempts to address why genetic variation in traits that affect fitness persist in natural populations. These ideas were verbally applied to plant defense theory 20 years ago (reviewed by Rhoades 1979). Simms and Rausher quantitatively formalized these arguments for plant defense with theory and

experiments in the late 1980's (Simms and Rausher 1987, 1989, Rausher and Simms 1989, Simms 1992a). The basic arguments have stemmed from the observation that plant defenses are beneficial to plants, and yet plant populations are not fixed at maximal levels of defense. Why are not all plants maximally defended? The proposed answer is that although defense is beneficial in some environments, it is also costly. These costs could reduce plant fitness in environments without herbivores, where the benefits of defense cannot be realized (Parker 1992, Simms 1992b, Mole 1994, Agrawal and Karban 1998). This is a classic example of genotype-by-environment interaction, which is thought to be a crucial component in the maintenance of variation in traits affecting fitness (Gillespie and Turelli 1989).

The application of the cost-benefit framework to evolutionary issues of induced defense has been somewhat muddy, in part because variation in defense can exist within a genetic individual (i.e., a single plant can be induced or not). Initial arguments suggested that plants should express high levels of defense when herbivores are present, but not all of the time because of costs. Although this makes perfect sense for an individual plant, as noted above, phenotypic benefits and costs do not necessarily reflect genetic benefits and costs. When under genetic control, such traits may have epistatic, linked, or pleiotropic effects which minimize or exacerbate the phenotypic benefit or cost. Adaptive evolution can only follow from genetic benefits, albeit constrained by genetic costs. However, phenotypic benefits and costs provide valuable information about the adaptive value of phenotypic variation in plant defense.

Induced Plant Defense as Adaptive Plasticity

Plasticity in plant defense reflects biotic or abiotic environmental conditions that affect the expression of resistance characters that can affect plant fitness (Agrawal and Karban 1998). A focus of this chapter is whether or not these induced responses enhance the fitness of plants compared to plants not having the induced responses.

Phenotypic plasticity is thought to evolve as a mechanism for organisms to express adaptive phenotypes in variable environments (Via and Lande 1985, Thompson 1991). Thus, a fundamental prediction of the evolution of adaptive plasticity is that organisms expressing particular phenotypes in particular environments should have higher relative fitness than conspecifics expressing alternative phenotypes (Thomson 1991, Dudley and Schmitt 1996, Kingsolver and Huey 1998). In other words, if an organism changes its phenotype in response to the environment, this is expected to increase its relative fitness compared to organisms that do not alter their phenotypes. An excellent example of adaptive plasticity that has received attention is the stem elongation response of plants growing in crowded conditions. Dudley and Schmitt (1995, 1996) conducted experiments using phenotypic manipulations of plant morphology (elongated or not) by exposing young plants to different red to far red light ratios. Plants of both phenotypes were then placed in both

environments (high and low crowding) to evaluate the consequences for relative fitness. Dudley and Schmitt showed that elongated plants in competitive environments have higher relative fitness than non-elongated plants, whereas in non-competitive environments, non-elongated plants have higher relative fitness than elongated plants. It is of value to be tall and spindly if there are many competitors for limiting light; these same traits can be detrimental in the absence of competition.

Inducible plant defenses provide another ideal system to test the adaptive plasticity hypothesis. Do induced plants have higher relative fitness than uninduced plants in environments with plant parasites, and do uninduced control plants have higher relative fitness than induced plants in environments lacking plant parasites? The answer to these questions are not only relevant to understanding whether this type of phenotypic plasticity is adaptive, but also to understanding the potential yield consequences of manipulating induced resistance in agriculture. Is induced defense a viable strategy that will enhance crop yields where pests are present? Under low pest pressure, are induced defenses wasteful and will their costs outweigh their benefits (reducing yield)? Note that viewing induced defenses as adaptive plasticity fits well within the cost-benefit framework outlined above, however, the adaptive plasticity framework is concerned specifically with phenotypic costs and benefits, not genetic costs and benefits (which are potentially responsible for the maintenance of genetic variation).

Recent studies of wild radish plants (reviewed in the next section, Agrawal 1998, 1999a, Agrawal et al. 1999), pepper weed (Agrawal 1999b), and wild tobacco plants (Baldwin 1998) have found support for induced defenses against herbivores as examples of adaptive plasticity by demonstrating fitness benefits and costs of induced resistance. Examples of adaptive plasticity in response to pathogens are fewer. Classic work by Kuc and colleagues in several cucurbit plant – pathogen systems has shown that induced resistance can increase survival and reproduction of challenged plants in the field compared to uninduced control plants (Kuc 1982, 1987, Caruso and Kuc 1977). Similarly, these workers and others have found circumstantial evidence that induction may reduce plant fitness in the absence of pathogens (Kuc 1987, Rasmussen et al. 1991, Hoffland et al.1998). Studies of barley (Smedagaard-Peterson and Stølen 1981) and tobacco (Lagrimini et al. 1997) show that there may be phenotypic costs of induced defense against pathogens, although benefits have not been demonstrated clearly in these systems. Experimental induction of tobacco did protect field-grown plants against blue mold and increased plant growth and yield (Tuzun et al. 1992).

Recent experiments with tomato plants have shown that infection by non-pathogenic *Meloidogyne* spp. nematodes can protect plants against a serious pest nematode, *Meloidogyne hapla* (Ogallo and McClure 1995). In field plots, plants with the initial inducing inoculum were stunted 30% less compared to controls, when both treatments were challenged. A cost assessment has not been made in this system.

Classic work by Raffa and Berryman (1982, reviewed by Raffa 1991) integrates the responses of grand fir trees to insect and pathogen enemies with effects on plant fitness. Fir trees that are attacked by bark beetles are also subject to a fungus which the beetles introduce. The plants respond with localized necrosis and systemic monoterpene emissions. These defensive responses can protect plants against subsequent infection and infestation of trees; inoculated (induced) trees were more likely to survive than uninoculated trees during an outbreak of the pests and pathogens (Raffa and Berryman 1982). Again, a cost assessment of the induced defense has not been made in this system.

Studies of benefits and costs of induced defense have employed various phenotypic manipulations of induced resistance, including: 1) the use of real plant parasites, 2) avirulent plant parasites, and 3) natural and artificial chemical elicitors of induced resistance. In addition, genetic mutants or engineered plants, over- and under-expressing particular inducible gene products can be used to test the adaptive plasticity hypothesis (for theory and a stem elongation example see Schmitt et al. 1995). Plant mutants over- and under-expressing induced defense against herbivores (McConn et al. 1997, Eichenseer et al. 1998) and pathogens (Bowling et al. 1994, Cao et al. 1994, Lagrimini et al. 1997) have also been reported. The technique of using mutants to assess phenotypic benefits and costs is useful because the phenotype does not have to be manipulated and the phenotypes will not change or adjust in different environments through time. However, it is essential to have replicated bred or engineered isogenic lines of over- and under-expressers to properly account for other changes introduced to the plant via the mutagenesis process. Finally, although using engineered plants and mutants are powerful tools for understanding benefits and costs, it is imperative to know where in the biochemical pathway of induction the mutations lie. For example, an under-expresser of induced resistance may have a completely functional pathway, except for one final product. In this case, the under-expresser may not show "cost savings" because most of the expense of the induced defense is already invested.

Case Study: Wild Radish and its Herbivores

In the wild radish system (*Raphanus raphanistrum* and *R. sativus*), induced responses to herbivory include elevated concentrations of indole glucosinolates and increased densities and total numbers of setose trichomes on newly formed leaves of previously damaged plants compared to undamaged controls (Agrawal 1999a, Agrawal et al. 1999). A broad array of herbivores feed on wild radish plants in nature; many of these herbivores are negatively affected by induced resistance, including earwigs, grasshoppers, aphids, flea beetles, and several species of lepidopteran larvae (Agrawal 1998, 1999a, c, unpublished data). Induced resistance in wild radish can be elicited by herbivory by both specialist and generalist herbivores (Agrawal 1999c) and natural elicitors of induced resistance such as jasmonic acid (Agrawal 1999a), but not by some types of mechanical damage (Agrawal 1998, 1999a).

In an experiment to test the first half of the adaptive plasticity hypothesis, that induced plants should have higher relative fitness in the presence of plant parasites than uninduced plants, nearly 500 plants were grown in the field. The plants were randomly distributed among three treatments: unmanipulated control plants, induced plants (plants treated with caterpillar herbivory), and leaf-damage control plants. Leaf-damaged control plants were treated such that an amount of leaf area was removed with a pair of scissors to equal that removed by the caterpillars in the induced resistance treatment. Leaf-damage control plants did not exhibit induced resistance, presumably because there were fewer plant cells damaged by the quick scissors clip and herbivore saliva was lacking (see Felton and Eichenseer, this volume). Thus, leaf damaged control plants assayed for the fitness consequences of losing leaf tissue without the effects of induced resistance.

Herbivores were hand-picked off plants twice daily when the treatments were being imposed. This is important because the experimental procedure did not impose induced resistance on a haphazard set of plants and

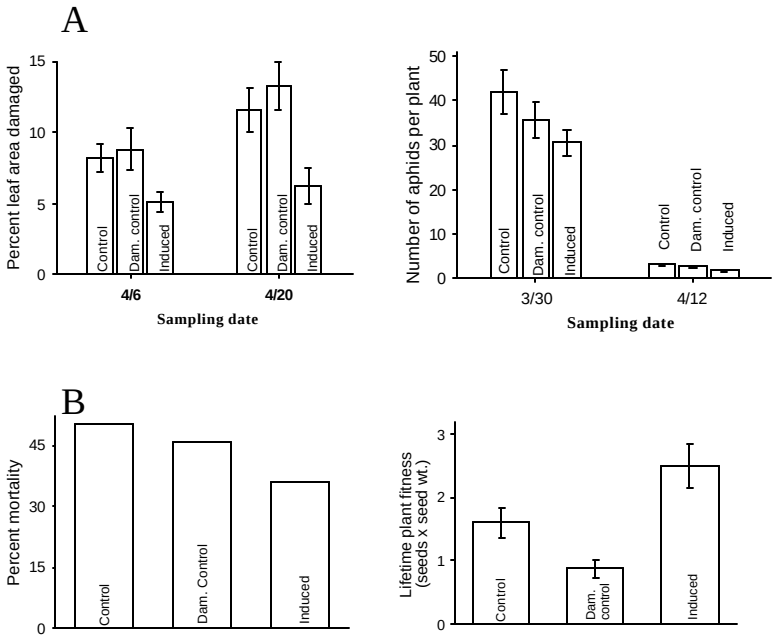

Figure 1: Consequences of induced responses to herbivory in wild radish plants for A) subsequent herbivory by leaf chewers and aphids, and B) components of plant fitness including herbivore imposed mortality and seed production. Dam. control plants received equal leaf area loss to that in the induced treatment but did not have the induced resistance. Error bars represent standard errors. Redrawn from Agrawal (1998).

determine the consequences. Rather, early season induction of resistance was denied in a set of plants (unmanipulated controls, and leaf damage controls) and uniformly imposed on another set of plants (that would have received natural herbivory and induced resistance, if all of the herbivores were not picked off).

The results indicate that plants expressing induced resistance were protected against several herbivores, including aphids, earwigs, and flea beetles (Agrawal 1998, 1999a, Fig 1a). Mortality of plants, due in large part to flea beetles, was differentially seen in the three treatments (Fig. 1b). Finally, induced plants had 60% higher fitness than control plants, calculated per plant as the number of seeds multiplied by mean seed weight (Fig. 1b). Because wild radishes are weedy annual plants, survival and seed production are good estimates of lifetime fitness. Leaf damage controls had the lowest fitness, and thus there was an even greater gap in fitness between leaf damage controls and induced plants. This result demonstrates that leaf loss itself is costly to the plant. However, when plants have induced resistance (coupled with leaf loss) the costs of leaf loss and other costs associated with induced resistance are far outweighed by the benefits (Agrawal 1998, 1999a). The benefits of induction were consistently demonstrated with two species of wild radish (*R. sativus*, and *R. raphanistrum*), conducted over two years and at two field sites (Agrawal 1998, 1999a).

The other half of the adaptive plasticity hypothesis is that plants not expressing defenses should have relatively higher fitness than plants expressing defenses in the absence of plant parasites. In other words, are there phenotypic costs of expressing induced defenses? Costs of induced plant resistance have received relatively more attention than benefits (Brown 1988, Karban 1993, Gianoli and Niemeyer 1997, Zangerl et al. 1997, Baldwin 1998). Among the many studies of phenotypic costs of induced resistance, there have been mixed results, with some studies finding costs (i.e., reduced growth or seed production), while other studies not finding costs. In the studies of costs of induced defenses in wild radish, a broad measure of costs was employed to include male fitness components in addition to traditional female fitness components (i.e., seed set). The number of seeds sired through pollen (male plant fitness) represent half of the genes contributed to the next generation in outcrossing plants.

In the cost studies (Agrawal et al. 1999), we found that induced wild radish plants had reduced fitness compared to uninduced plants in an environment without herbivores. This experiment also controlled for leaf area removal using a factorial design employing plants with and without leaf tissue removal, and plants with and without induced resistance (Fig. 2). This approach is powerful for several reasons, including the fact that the effects of induced defenses and leaf tissue loss can be uncoupled. From a statistical standpoint, the factorial analysis has superior power than non-crossed designs, and has the ability to detect interactions between leaf area removal and induction. Finally, although elicitors of induced plant defenses (and other techniques such as mutagenesis) provide an exciting opportunity to answer ecological and

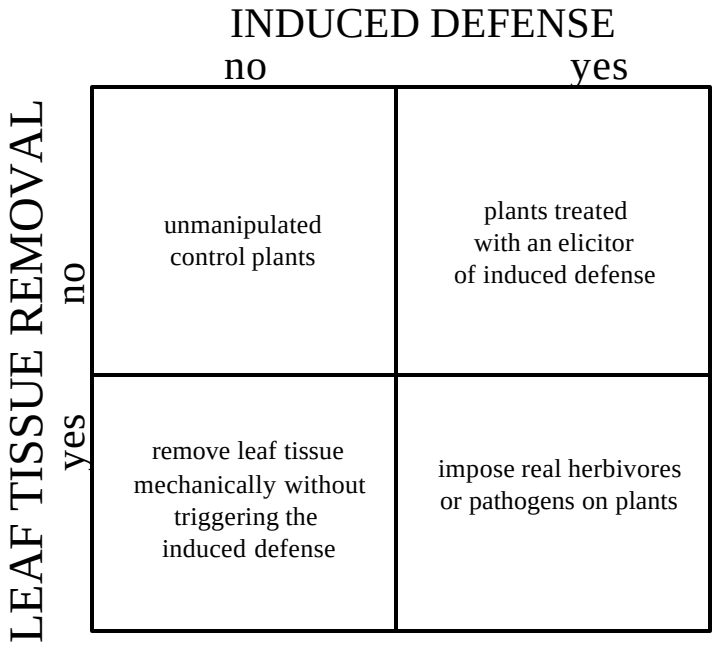

Figure 2: Experimental design recommendation for testing for the phenotypic benefits and costs of induced plant defenses. The factorial design is statistically powerful and can detect an interaction, which is important for calibration of real and artificial eliciting agents.

evolutionary questions, it is important to include real herbivory treatments when using elicitors, so as to calibrate your system and be sure that real herbivory and the elicitors are having the same effect.

The cost of induced defense detected in wild radish was only detected in components of male fitness, pollen grain production and size. These traits have been linked to seed siring ability in wild radish and other systems. Surprisingly, seed number and seed weight were not reduced on induced plants compared to controls. These results point to the need for consideration of other fitness components than seed production when studying fitness in sexually reproducing plants. Physiological studies in plants have directly linked jasmonates to viable pollen production (see Staswick and Leman, this volume), suggesting a mechanism for the cost of induced resistance in terms of male fitness characters.

In summary, induced defenses in wild radish plants are an example of adaptive plasticity because of the phenotypic benefits of induction in the presence of herbivores, and the phenotypic costs of induction in the absence of

herbivores. Costs may be manifested in ways other than reduced seed production, including costs in terms of ecological interactions (reviewed by Agrawal and Karban 1998). The key result, however, is that the benefits far outweighed the costs under field conditions.

Predictability: a Requirement for Adaptive Plasticity

In order for a plant parasite to induce an adaptive defense in the plant, the initial attack must provide reliable information, and predict future attack (Karban and Adler 1996, Karban et al. 1999). For example, if plants infected with a pathogen induce production of phytochemicals that protect the plants against subsequent attack, but there is no subsequent attack, then investment in the induced products is presumably wasted (and not adaptive). In many systems, heavy parasite loads early in the season may predict heavy parasite loads later in the season, thereby making induced resistance a good strategy. Likewise, many plant parasites predict future attack by themselves. For example, pathogens that can systemically infect plants may be controlled by induction at the time of initial infection. Herbivores that are relatively immobile, such as leaf miners, small caterpillars, and spider mites all may provide reliable information that predict their own continued presence at the initial time of attack. This predictability is an essential component of most models of induced plant defense (See Table 1 in Karban and Adler 1996). Given that many plant parasites are highly mobile, or seemingly have unpredictable infection dynamics, an important and missing component of evolutionary studies of plasticity in defense is that the parasites are predictable.

Costs of Plasticity Verus Costs of Induced Defenses

For organisms which exhibit phenotypic plasticity, there may be several types of evolutionary constraints that limit expression of maximally beneficial plasticity. Above, phenotypic costs associated with producing defenses were considered. In addition, organisms may be constrained by costs associated with the ability to express alternate phenotypes (plasticity genes, sensory and regulatory mechanisms, etc). Costs of plasticity and their distinction from phenotypic costs of producing an inducible response have been recently reviewed by DeWitt et al. (1998). If quantitative genetic variation in inducibility exists (Table 1), these may be ideal systems to test for costs of plasticity by assessing the relationship between inducibility *per se* and fitness in various environments (DeWitt 1998, Scheiner and Berrigan 1998).

Macroevolution of Induced Plant Defenses

Little is currently known about the macroevolution of induced plant defenses. A phylogenetic perspective could address whether induced defenses

are ancestral or derived. Most theoretical models predict that induced defenses are evolutionarily derived from constitutive strategies as a cost saving mechanism (Karban and Adler 1996, Karban and Baldwin 1997, Thaler and Karban 1997). The scenario that has been envisioned is that constitutive defenses were ancestral and that high costs of constitutive defenses were saved as plasticity in defense was favored (Karban and Baldwin 1997). Here costs of plasticity are assumed to be low because plasticity was presumably favored over a fixed defense (i.e., costs of plasticity were smaller than costs of constitutive defense) (Cipollini 1998). An alternative scenario proposed by Karban and Baldwin (1997) and briefly discussed by Cipollini (1998) is that the ancestral state was undefended, not constitutively defended. Here, costs of plasticity are predicted to be higher, and perhaps more easily detectable, because plastic defense was favored as an initial defensive strategy, not as a cheaper alternative to constitutive defense.

Only one study to date has attempted to examine the phylogenetics of induced plant defenses (Thaler and Karban 1997). In this study, the authors examined constitutive and induced resistance of 21 *Gossypium* species to herbivory by spider mites. *Gossypium* spp. are distributed worldwide, and are of economic importance as several species are cultivated for the cotton lint. Constitutive and induced resistance were found to be derived traits in *Gossypium*, suggesting that the ancestral state may have been undefended. Further studies of the macroevolution of induced defenses will be important in understanding large scale patterns in plant defense and constraints on their evolution.

Synthesis

Induced responses against herbivores and pathogens can provide a fitness benefit to plants. Phenotypic benefits and costs of induced resistance have been demonstrated in a few studies; more are needed however, especially those experiments employing a combination of approaches to phenotypic manipulations (i.e., using combinations of real plant parasites, avirulent parasites, elicitors, mutants, etc.). If induced defenses are an example of adaptive plasticity, the next goal should be to understand how they evolve. Although a handful of studies document quantitative genetic variation in inducibility, these studies were largely conducted for other purposes. The evolution of induced defenses and other phenotypically plastic traits is a young field of investigation, and will benefit from field experiments employing phenotypic and genetic manipulations.

Induced defenses add a novel level of complexity to the study of coevolution between plants and their parasites. Specificity in induced resistance (Stout and Bostock, this volume) may promote pairwise coevolution by allowing plants and their enemies to interact on a one-to-one level within the background of other interactions. Inducibilty per se may have been favored to maximize

defenses against multiple enemies—allowing plants to fine-tune their defenses upon recognition of their attackers.

Future Directions

Understanding the evolution of induced defense will be advanced by applying our knowledge of quantitative genetic variation in induced resistance to fitness consequences for the plant. Such experiments should be conducted in environments with and without plant attackers. For plant-pathogen interactions, researchers will need to first quantify such genetic variation. It will be useful to impose artificial selection on constitutive and induced defenses, to ask if they can be uncoupled, and to ask how selection might act on them independently and in concert. It is still not clear what biotic and abiotic conditions favor induced plant defenses. Addressing the macroevolution of inducible and constitutive defenses may give us insight into how these traits evolve, and how costs of plasticity may constrain the evolution of plant defense.

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