FOUR

Phenotypic Plasticity

KAILEN A. MOONEY AND ANURAG A. AGRAWAL

Phenotypic plasticity is the ability of an organism, a single genotype, to exhibit different phenotypes in different environments (Fig. 4.1A). Such plasticity is nearly ubiquitous in nature and occurs in various animal and plant phenotypes, including behavior, physiology, and morphology. Phenotypic plasticity may be observed as both adaptive and nonadaptive responses to the biotic or abiotic environment, though we focus on adaptive responses in interacting species.

Phenotypic plasticity plays an important role in the interactions between plants and herbivorous insects (Tables 4.1 and 4.2). In particular, plants and herbivores have traits that are expressed in response to their interactions with each other (e.g., defense and offense, respectively), and the induction of these traits may subsequently alter the dynamics of the plant-herbivore interaction. In addition, plants and herbivores also have traits that are induced by their interactions with third parties including predators, parasites, competitors, and mutualists. In the context of plant-herbivore interactions, plasticity in response to interactions with third parties is relevant in that the induced phenotypes may in turn have effects within the pairwise plant-herbivore interaction.

Below we take both an evolutionary and ecological approach to understanding the causes and consequences of phenotypic plasticity in plant-herbivore interactions. In particular, there has been a growing interest in understanding plasticity in two contexts: first, the various types of plasticity and their adaptive nature, and second, the ecological consequences of plastic phenotypes in food webs. Phenotypic plasticity has been discussed and studied extensively in both contexts, but work in these areas has mostly proceeded independently and without a significant synthesis of terminology and concepts. We thus make an effort to discuss this work in a common framework and to explicitly illustrate connections.

Adaptive Value and Costs of Phenotypic Plasticity

A comprehensive investigation of adaptive plasticity might proceed in the following manner. The first step is to document whether traits of plants or herbivores are induced by their interactions with each other. Having identified an inducible trait, the second and third steps are to determine whether the inducible trait is adaptive in two environments. Plasticity has the potential to be adaptive only when there are fitness benefits of an inducible trait in one environment (compared to organisms not expressing that phenotype), as well as costs associated with the same trait in an alternate environment (Fig. 4.1B). Consequently, establishing the benefits of plasticity means documenting that the inducible trait provides fitness benefits in the environment in which it is induced (step 2) and fitness costs in the environment in which it is not induced (step 3). Satisfying these two criteria (step 2 and step 3) substantiate the ecological conditions needed for plasticity to function adaptively but do not demonstrate that this is necessarily the case; there may also be costs associated with plasticity per se, and the adaptive value of plasticity comes when the fitness benefits of tailoring trait expression to the environment outweigh any costs associated with any being plastic. Thus, the last (fourth) step is to determine whether plasticity for that trait (i.e., the ability to change phenotypes) is, itself, adaptive. This is accomplished by showing that plastic genotypes have higher fitness than nonplastic genotypes under a set of variable environmental conditions.

In this section we summarize the work on plant and herbivore plasticity with respect to each of these steps. We document the wide range of plant and herbivore traits that have been shown to be inducible and to increase plant or herbivore fitness (step 1 and step 2). We then summarize the evidence that inducible traits carry fitness costs in some

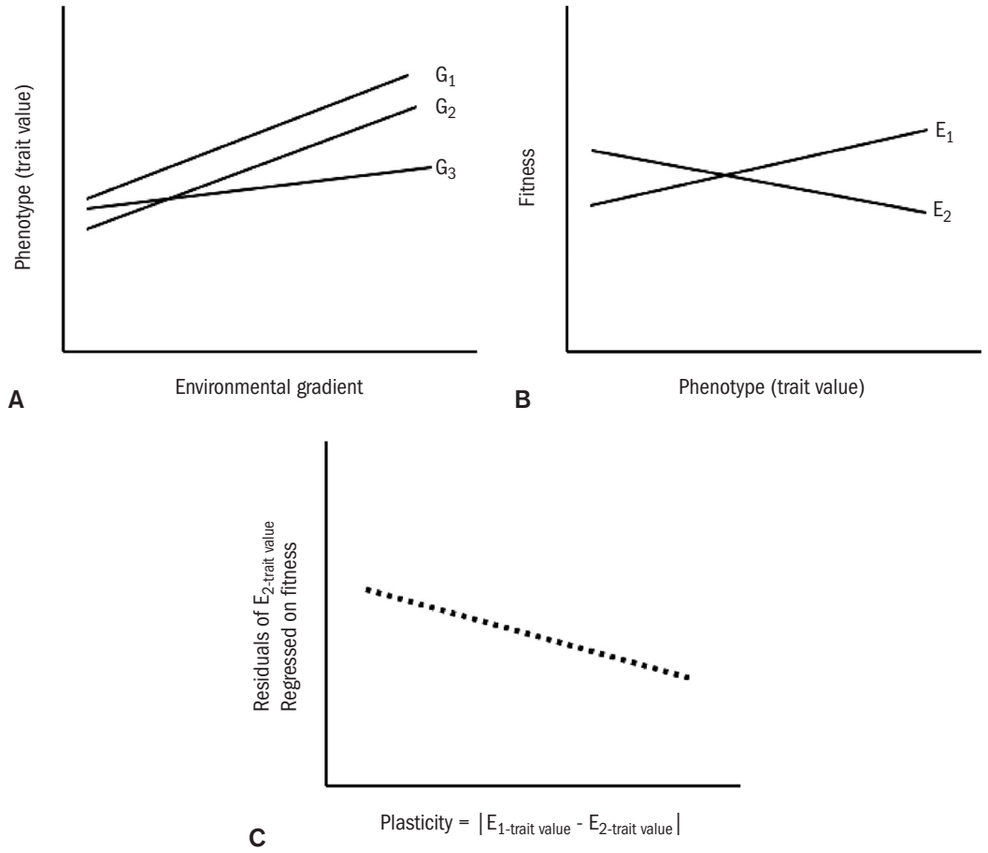

FIGURE 4.1. Graphical depictions of phenotypic plasticity (A), its adaptive value (B), and its costs (C). A. The environment-dependent or plastic phenotype of three genotypes of a single species (G_1–G_3). G_1 and G_2 show different mean trait values, but an equivalent response to the environmental gradient (equal slopes); this illustrates that plasticity does not require a genotype by environment interaction. Comparing G_3 to G_1 and G_2 illustrates differential responses to the environmental gradient, namely, a genotype by environment interaction. B. The relationship between the value of a trait and fitness for a single genotype in two environments (E_1 and E_2). Expression of the trait increases fitness in E_1 but reduces fitness in E_2. If the trait is expressed at high levels in E_1 but low levels in E_2, this plasticity has the potential to be adaptive by reducing the fitness costs of expressing the trait in E_2. Whether plasticity is in fact adaptive depends on whether the benefits of reduced trait expression in E_2 outweigh any costs plasticity. C. Each dot represents a genotype that exhibits a particular level of plasticity for a given trait, defined as the absolute value of the change in trait value across environments E_1 and E_2. The cost of plasticity is illustrated by a negative relationship between the plasticity of a genotype and the residuals of fitness regressed on uninduced trait values (i.e., trait values in E_2). Using these residuals corrects for any fitness effects associated with the trait and thus isolates the costs of plasticity per se.

environments, and thus that plasticity might function adaptively by reducing such costs (step 3) ("Plasticity and Its Adaptive Nature in Plant-Herbivore Interactions"). While we find that there are no studies directly demonstrating the adaptive value of plasticity (step 4), we review what is known about the costs of plasticity ("Costs of Plasticity"). Finally, we present a verbal model for the relative costs and benefits of different forms of plasticity ("Relative Costs and Benefits of Behavioral, Physiological, and Morphological Plasticity").

Plasticity and Its Adaptive Nature

PLANTS IN RESPONSE TO HERBIVORES

Though there were several early reports of plant phenotypic plasticity in response to insect attack, the study of induced plant responses to herbivory became of general interest in plant-herbivore interactions in the 1970s (Karban and Baldwin 1997). In particular, studies of tomato plants' chemical responses to beetle grazing (Green and Ryan 1972) and the

TABLE 4.1
Plasticity in Plants Induced by Interactions with Herbivores

Stimulus	*Notes*	*References*
Oviposition (egg deposition in or on the plant)	Remarkable plant responses, at least sometimes mediated by the plant horomone jasmonic acid, and shown to increase levels of egg parasitism.	Meiners and Hilker 2000, Hilker and Meiners 2002
Walking on the leaf surface	Unlikely to be adaptive.	Bown et al. 2002
Folivory (caterpillars, beetles, etc.)	The best studied category; often mediated by jasmonic acid, shown to be adaptive.	Karban and Baldwin 1997, Agrawal 1998
Sap sucking (e.g., aphids)	Well-studied responses, chemically distinct from folivory and often similar to plant responses to disease; mixed results for the impact on subsequent herbivores.	Stout et al. 1998, Walling 2000
Cell-content feeding (e.g., spider mites)	Tends to cause plant responses with attributes similar to both folivory and piercing sucking damage.	Ozawa et al. 2000
Leaf mining	Not well studied.	Faeth 1991, Karban 1993
Galling insects	Responses appear to be more adaptive for galler than for plants.	Larson and Whitham 1991, Stone and Schonrogge 2003

reduced growth of caterpillars on damaged compared to undamaged birch trees (Haukioja and Hakala 1975) were responsible for popularizing the study of induced responses. The study of plant morphological responses to herbivory has a more recent history, with studies demonstrating induced plant production of thorns (Young 1987; Young and Okello 1998) and trichomes (Baur et al. 1991; Agrawal 1999). There are now several hundred examples of traits in plants that are induced by herbivore feeding (see examples of these in Table 4.1). For the best-studied systems, those where insects chew the leaves of plants, there is a high level of evolutionary conservation in the hormonal regulation of induced plant responses. For example, responses including plant toxins (Bodnaryk 1994; Baldwin 1996), trichomes (Traw and Bergelson 2003), volatiles (Boland et al. 1999; Thaler 1999), and extrafloral nectar (Heil et al. 2001) all appear to be, at least in part, regulated by jasmonic acid.

Phenotypic plasticity occurs not only as a relatively immediate response to an environmental change, but also across growing seasons and across generations. For example, many woody plant species, when defoliated, show an increase in the resistance of foliage in the following growing season (Haukioja 1991). The adaptive nature of this response is believed to be based on the correlations between herbivore densities across years. Maternal effects occur when the environment of an organism in one generation affects the phenotype expressed by its offspring (Rossiter 1996). In order to be adaptive, the environment in one generation must predict the environmental conditions experienced by offspring. We have found that damage by *Pieris rapae* caterpillars to *Raphanus raphanistrum* plants, even before they are reproductive, induces a response that makes progeny (seedlings) more resistant to caterpillars than progeny from undamaged plants (Agrawal et al. 1999b). Applying jasmonic acid to plants can induce this same response (Agrawal 2002). Finally, there is genetic variation for this maternal effect, and some evidence for selection acting on maternal effects from a field experiment (Agrawal 2001a).

Many of the traits induced by herbivores have been shown to reduce herbivory, but these studies do not document the adaptive value of plasticity in these traits. For plasticity to be adaptive, these same traits must carry a fitness cost in the absence of herbivores (Fig. 4.1B), because it is the avoidance of these costs that makes plasticity adaptive. Only two systems have been studied to determine whether the ecological conditions favor plasticity, and for both systems this was the case. Wild tobacco plants that were induced to produce alkaloids with jasmonates enjoyed benefits of this phenotype in the presence of herbivores and costs in the absence of herbivores (Baldwin 1998). Inducing plants with herbivores as well as jasmonates, similar benefits and costs were demonstrated for wild radish plants (Agrawal 1998, 1999; Agrawal et al. 1999a).

The source of trait costs can be a matter of trade-offs due to resources allocated to those traits. At the same time, trait costs may also be more ecologically complex (Agrawal and Karban 1999). For example, with the induced responses of plants to herbivore damage, a given "defensive" trait may have a negative effect on most herbivores, but also a beneficial effect on some herbivores (i.e., in host location) (Giamoustaris and Mithen 1995; Agrawal and Sherriffs 2001). Consequently, inducible expression may protect the plant

TABLE 4.2
Plasticity in Herbivores Induced by Interactions with Plants

Stimulus	*Notes*	*References*
Plant part consumed/ chemical defense	Mimicry of plant part (catkin vs. twig)	Greene 1989, 1996
Physical plant defense	Behavioral deactivation	Dussourd and Eisner 1987; Malcolm 1995
Antidigestive plant defense	Induced alternate digestive enzymes	Broadway 1997; Cloutier et al. 2000
Plant toxins	Enzymatic detoxification	Krieger et al. 1971; Feyereisen 1999

against being a continuously attractive target to adapted insect species (Agrawal and Karban 1999).

Studies by Baldwin (1998) and Agrawal and colleagues (1998, 1999; Agrawal et al. 1999a) measured the benefits and costs of the full repertoires of multifaceted, coordinated induced responses. In contrast, we know relatively little about the adaptive value or costs of the distinct components of such induced responses that involve multiple traits. In wild tobacco, trypsin proteinase inhibitors function as a defense that directly deters herbivory. The costs and benefits of this single induced defense have been studied, but this has been restricted to laboratory environments (Zavala et al. 2004a, 2004b). For responses that involve damaged plants attracting predators or parasitoids of herbivores (i.e., induced indirect defenses), a single study has estimated benefits to plants (Kessler and Baldwin 2001). No studies have been successful in measuring the costs of induced predator attractants, though some calculations have been attempted (Dicke and Sabelis 1992). There is as yet little evidence that induction of extrafloral nectar to attract ants is costly in the absence of herbivores (Heil et al. 2001). Consequently, while predator recruitment itself has proven fitness benefits, there is little evidence to suggest that plasticity in predator recruitment is adaptive.

Attempts to study individual components of herbivore-induced responses are made difficult by the fact that the trait of interest cannot necessarily be separated from other components of the plant response. For instance, most studies of costs or benefits of induced predator-attracting volatiles confound the induction of direct foliar defenses and indirect volatile defenses (Heil 2004; Hoballah et al. 2004). Similarly, benefits of induced extrafloral nectar production have not been separated from other induced effects (Heil et al. 2001).

Although it is part of our reductionist nature as scientists to want to decompose each of the induced response systems of a plant into adaptive plasticity (or not), perhaps this is not the most fruitful path. The very fact that multiple plant responses in a single individual are induced by herbivory and co-regulated by the same plant hormones indicates that "plastic responses" are correlated in nature and may not decompose into adaptive parts. Indeed, the summed effects of multiple plant responses may be greater (or less) than additive in their effects on herbivores. As an example, consider the fact that the benefits of a "direct" induced plant response (i.e., slowed herbivore growth) may depend on the "indirect" attraction of natural enemies of herbivores (i.e., the slow growth–high mortality hypothesis) (Clancy and Price 1987; Benrey and Denno 1997). When suites of plant traits function synergistically with respect to herbivore resistance, we predict that selection should favor their co-occurrence and that such "defense syndromes" should evolve repeatedly and independently in a diversity of plant taxa (Kursar and Coley 2003; Agrawal and Fishbein 2006). Thus, care needs to be taken when trying to decompose the independent adaptive value of coregulated traits.

HERBIVORES IN RESPONSE TO PLANTS

Herbivorous insects, like plants, exhibit phenotypic plasticity in morphology and physiology, but they also have plastic behaviors. Because herbivores are caught in the trophic sandwich between plant defenses and predators and parasitoids, and because there are so many varied lifestyles among herbivorous insects, herbivores face a greater diversity of challenges. Perhaps for this reason, they employ a greater diversity of plastic phenotypes than plants (Table 4.2).

While it was recognized in the 1960s that cytochrome P-450 enzymes in insects detoxified pesticides and plant secondary compounds (Brattsten et al. 1977; Estabrook 1996), it was not until the 1970s that it was determined they were inducible. Brattsen et al. (1977) determined that their production in *Spodoptera eridania* was induced by exposure to a variety of plant secondary compounds. It is now known that the production of proteases that are insensitive to plant-produced protease inhibitors are inducible (Broadway 1997). With respect to induced changes in herbivore morphology, Bernays (1986) showed that caterpillar head size responded plastically to the toughness of foods, and this apparently provides the appropriate musculature for individuals feeding on hard versus soft leaves. Similarly, Thompson (1992) demonstrated that plant-induced changes in mandible morphology increased feeding efficiency.

Herbivores also respond to plant defense behaviorally. Insect behaviors to circumvent plant defenses had been noted for decades, but the first work to rigorously document

the benefits of such behaviors came in the 1980s (Carroll and Hoffman 1980; Dussourd and Eisner 1987). These studies showed how in two systems leaf trenching locally reduced plant defense and increased herbivore feeding. Previous work on bark beetles (Scolytidae) documented how aggregated attack of conifer hosts, behaviorally mediated via pheromones, overwhelms plant defenses and allows for successful host colonization (Mitton and Sturgeon 1982).

As with plants, herbivore traits can be induced by the maternal environment adaptively when the maternal environment is predictive of the conditions experienced by offspring. Where the maternal environment is the host plant on which an herbivore is laying its eggs, the maternal environment can be a perfect predictor of the offspring's environment. Indeed, in cases such as for ovipositing seed beetles, there is strong evidence for adaptive maternal effects (Fox et al. 1997). Other work on herbivores also suggests a role for maternal effects in the plant-herbivore interaction (Gould 1988; Futuyma et al. 1993; Rossiter 1996). As discussed above, maternal effects have been strongly implicated in the interaction between the cabbage white butterfly, *P. rapae*, and its host plant, wild radish, *R. raphanistrum*. From the herbivore's perspective, the protein content of the diets of *P. rapae* caterpillars was correlated negatively with the size of eggs produced by adults (Rotem et al. 2003). Moreover, mothers reared in extreme conditions (high and low protein) produced progeny that grew best under those conditions. These potentially adaptive maternal effects in both the plant and the insect were detected early in progeny growth but not later in their development (Agrawal 2002; Rotem et al. 2003).

Quite surprisingly, for only a few induced traits in herbivores has it been demonstrated that their expression can be costly in some environments, and thus that plasticity might function adaptively by reducing these costs. A few studies come close. For example, the two-spotted spider mite *(Tetranychus urticae)* is one of the most polyphagous herbivorous arthropods, and one mechanism it uses for coping with the defenses of diverse plants is the P-450 detoxification system (Mullin and Croft 1983). Spider mites with induced detoxification enzymes had 60% higher fecundity when feeding on toxic host plants compared to uninduced mites (i.e., a fitness benefit) (Agrawal et al. 2002a). Blockage of the detoxification system reduced fecundity on the toxic host. Furthermore, there was an apparent 30% cost of induction, as evidenced by lower fecundity of induced mites on nontoxic host plants (Agrawal et al. 2002a). A few other studies have attempted to study the costs and benefits of detoxification systems in herbivores (Neal 1987; Appel and Martin 1992; Berenbaum and Zangerl 1992; Berenbaum and Zangerl 1994).

There is little other direct evidence of both costs and benefits of herbivore phenotypes induced by plants, but there are many suggestive examples where this is likely to be the case. Herbivore trenching behaviors are time-consuming and likely increase predation risk by increasing time spent on exposed leaves (Dussourd 1993; Bernays 1997), as well as by slowing herbivore development time generally (Clancy and Price 1987). Strategies of aggregated feeding such as those employed by bark beetles likely increase risk of predation when enemies function in a density-dependent fashion, as well as increase competition. Plant-induced changes in herbivore mouthparts to cope with harder foods might reduce feeding efficiency if those same individuals later moved to soft foods. While such fitness costs seem plausible, they are putative. In order to document the fitness benefits of plasticity, such costs must be documented and compared to the benefits these traits have been shown to confer.

Costs of Plasticity

Phenotypic plasticity can be adaptive only when a trait increases fitness in some environments but reduces fitness in others due to the trait's production costs or ecological costs (Fig. 4.1B). When these criteria are met, plasticity carries a fitness benefit. Yet at the same time there may be costs of plasticity per se (i.e., costs not associated with the inducible traits themselves). These costs include the energy needed to construct and maintain the physiological machinery to sense the environment and to subsequently alter phenotypes. Costs of plasticity may also be based in pleiotropy, linkage disequilibrium, or epistasis, influencing other fitness-enhancing traits. Ecological costs of plasticity (also called limits) are defined by a reduction in fitness of plastic organisms compared with less plastic organisms, where the fitness difference is not inherent, but rather is only realized in some environments (DeWitt et al. 1998; Agrawal 2001b). An imperfect match between a phenotype and the environment that results in relatively low fitness exemplifies an ecological cost. Because the success of plasticity is dependent on the predictability of the environment, lags in the response to environmental stimuli and an unpredictable environment can result in a significant ecological cost to plasticity compared with that experienced by an organism with a fixed strategy.

The adaptive value of plasticity is thus a function of both benefits and costs. The evolution of plasticity in a trait may thus be constrained by costs, in addition to other constraints including phylogeny and genetic variation. In other words, some organisms may have less plasticity than others (even though plasticity is beneficial) simply because there are trade-offs associated with high levels of plasticity. The definitive test for adaptive plasticity shows that plastic genotypes have higher lifetime fitness than nonplastic genotypes when sequentially exposed to environments in which the inducible traits carry fitness costs and benefits. This approach would thus measure natural selection on plasticity under conditions that integrate both the benefits and costs of plasticity.

In fact, no such studies have been conducted to date in plant-herbivore interactions. However, there is work demonstrating a cost of plasticity. The signature of this cost is lower

fitness of a plastic organism than a less-plastic conspecific genotype in a single environment (Fig. 4.1C). Costs of plasticity have been examined in a few plant species (for plastic response to light, competition, and herbivory) (Dorn et al. 2000; van Kleunen et al. 2000; Agrawal et al. 2002b; Steinger et al. 2003), in a few animal species (Krebs and Feder 1997; DeWitt 1998; Scheiner and Berrigan 1998; Relyea 2002), and bacteria (Nguyen et al. 1989). Much to our surprise, though phenotypic plasticity and genetic variation in plasticity has been studied extensively in insects, only a single study of thermal tolerance induction in *Drosophila* estimated fitness costs of plasticity (Krebs and Feder 1997).

Only a single study has examined the costs of plasticity in the realm of plant-herbivore interactions, and that work investigated the induced responses of wild radish plants to damage by *P. rapae* (Agrawal et al. 2002b). Plasticity was defined as the absolute value of the difference in glucosinolate levels between undamaged and herbivore-damaged plants. Indeed, there was additive genetic variation for this plasticity in plant chemistry. To estimate costs, paternal family breeding values ($N = 28$) were regressed against the residuals from the relationship between constitutive levels of glucosinolates and lifetime fruit mass. Using these residuals corrects for the relationship between constitutive levels of glucosinolates and fitness in the undamaged environment (Van Tienderen 1991; DeWitt et al. 1998). More-plastic families of wild radish had lower fitness than less-plastic families in the absence of herbivory, but this relationship was only marginally significant by a two-tailed test ($P = 0.10$) (Agrawal et al. 2002b). Such marginally significant results in experimental tests for plasticity costs have been the typical finding in this literature. Indeed, sample sizes for such analyses are the number of genetic families, and statistical power is often a limiting factor.

Agrawal (2001b) has argued that knowing the mechanism of phenotypic plasticity will be critical in detecting plasticity costs. In using quantitative genetic approaches with more- and less-plastic families of organisms, less-responsive families may lack the receptors and the physiological machinery needed to respond to the environment. Alternatively, less-responsive families may have all of the receptors and machinery necessary to respond, but may simply be defective in some final step of the response pathway. These two extremes of the continuum illustrate a potential pitfall to detecting costs of plasticity. Nonetheless, ecological, rather than genetic, costs of plasticity may be readily determined using such an approach. As with the attempt to detect costs or benefits of a trait, the results may be context dependent.

Relative Costs and Benefits

The relative fitness value of plasticity in physiology, morphology, and behavior likely differs as a function of production costs, the time frames in which the trait changes operate, and the extent to which they are reversible. For example, the success of phenotypic plasticity as an adaptive strategy depends on the current environment predicting the future environment (Karban et al. 1999). The ability to predict the future environment from the current environment declines with the time separating the two, and this leads to the prediction that rapid phenotypic changes should have greater benefits than slower responses. The time frame of induction generally increases from behavioral responses to physiological responses to morphological changes. An herbivore's decision to remove leaf trichomes before feeding occurs in a mater of seconds or minutes. Plant and herbivore induction of secondary compounds and detoxification enzymes, respectively, occurs over minutes to days. Morphological change occurs over days to months; changes in plant morphology only occur in new growth, and changes in herbivore morphology occur between molts.

Production costs of plastic phenotypes are also likely to be loosely proportional to these time frames of operation, with expense increasing from behavior to physiological response to morphological response. The reversibility of induced responses relates to the importance of predicting future environments from current ones. Where behaviors and physiological traits can respond dynamically to changing environments (if at different time scales), morphological changes in both plants and herbivores are frequently irreversible.

Induced responses can thus be arranged in a continuum. Behavioral changes occur in response to the most reliable information, are the least costly, are fastest acting, and are the most easily reversed. Morphological changes are based on inherently less certain information, are the most costly, are the slowest acting, and are largely irreversible. Physiological responses are intermediate in these respects. This verbal model thus predicts that plasticity should be most common in behavioral responses, less common with respect to physiology, and least common in morphological responses. For insect herbivores, where plasticity occurs at all three levels, we would argue the predictions of this model are generally met.

The Consequences of Phenotypic Plasticity

The adaptive function of phenotypic plasticity occurs by altering ecological interactions (through changes in traits) that have implications for the fitness of the plastic organism. In this section we discuss more broadly the evolutionary and ecological consequences of phenotypic plasticity. In some cases, the effects of an induced trait may include a positive outcome for the plastic organism with respect to fitness (or its correlates). Alternatively, phenotypic plasticity in one organism can have effects on other organisms that do not relate to the adaptive value of plasticity, but rather exemplify the importance of species' traits in the larger community context in which the organisms live. Below, we describe the consequences of plasticity beyond those dynamics related to the adaptive value of plasticity itself (see the section "Ecological Consequences of Plasticity"), compare plasticity-based interactions that relate to the adaptive value of plasticity to those interactions that are

simply the consequence of plasticity ("Unifying Ecological and Evolutionary Concepts"), and evaluate the evidence for the ecological and evolutionary importance of phenotypic plasticity ("Is Phenotypic Plasticity Important in General?").

Ecological Consequences

Prior to the mid-1990s, both population and community ecology were based on models in which species interactions occurred via changes in population densities. In this type of interaction the net result is a change in the density of at least one species. The theoretical work of Abrams (1995) suggested that phenotypic plasticity allows organisms to interact not only via changes in density, but also by altering each other's phenotypes (Fig. 4.2). Abrams called these "density-mediated interactions" (DMIs) and "trait-mediated interactions" (TMIs), respectively. Schmitz et al. (1998) provided one of the first empirical tests of these ideas with the tritrophic interactions between spiders, grasshoppers, and old-field plants. Surprisingly, predatory spiders that did not feed upon grasshoppers (i.e., no DMI) nevertheless had a TMI when their presence induced predator-avoidance behaviors.

Abrams (1995) went on to argue that the principal significance of phenotypic plasticity occurs when a change in a trait has subsequent consequences for the same individual that induced the phenotypic change, or for other individuals of the same or a different species (Fig. 4.2). He termed such events "trait-mediated indirect interactions" (TMIIs) (Abrams 1995), as they are the indirect consequence of one species inducing trait changes in another. While the importance of indirect density effects (so-called density-mediated indirect interactions [DMIIs]) had been recognized for decades in plant-herbivore interactions (i.e., trophic cascades) (Hairston et al. 1960), the importance TMIIs first became apparent through these theoretical arguments. The study by Schmitz et al. (1998) provided empirical evidence for the importance of these dynamics: the indirect effects of spiders on plants via induction of predator-avoidance behaviors was equal in magnitude to their effects as consumers of spiders (i.e., TMIIs and DMIIs were equal in magnitude). Subsequent work has documented the importance of TMIIs in numerous contexts.

There are ecological implications for TMIIs strictly within the context of plant-herbivore interactions. For instance, Van Zandt and Agrawal (2004a, 2004b) documented that feeding by different species of herbivores on milkweed *(Asclepias syriaca)* induced distinct responses in plants that influenced the composition of the herbivore community later in the growing season. Cyclical population dynamics in some herbivores may be attributable to induced plant resistance, where feeding by one herbivore generation reduces the growth rates of subsequent generations (Haukioja and Hakala 1975; Haukioja 1980; Klemola et al. 2004). These examples demonstrate how phenotypic plasticity provides a mode of interaction among herbivores—both within and across species—that use the same plant,

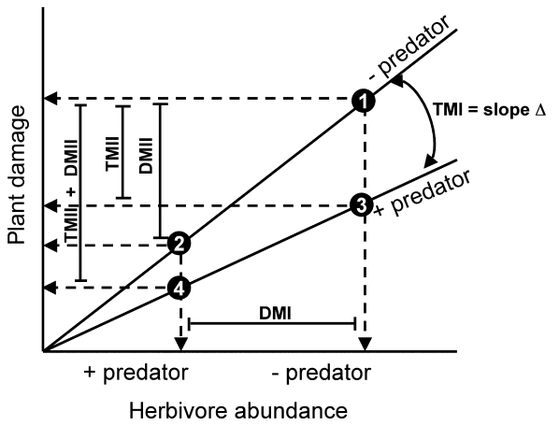

FIGURE 4.2. Graphical depiction of trait-mediated and density-mediated direct interactions (TMI and DMI) and indirect interactions (TMII and DMII) using the tri-trophic interactions among predators, herbivores, and plants as an example. The DMI between predators and herbivores is the difference in herbivore abundance as a function of predator presence versus absence (change on *x* axis from point 1 to point 2). The TMI between predators and herbivores is the change in per capita effects of herbivores on plants, namely, the change in the slope of plant damage regressed on herbivore abundance as a function of predator presence versus absence. The DMII between predators and plants is the difference in plant damage as a function of predator presence versus absence when controlling for the per capita effects of herbivores on plants (change on *y* axis from point 1 to point 2). The TMII between predators and plants is the change in plant damage due to changes in the per capita effects of herbivores on plants as a function of predator presence versus absence, when controlling for herbivore abundance (change in *y* axis from point 1 to point 3). The total effect of predators on plants is the difference in plant damage as a function of predator presence versus absence due both to changes in herbivore abundance and to changes in the per capita effects of herbivores on plants, namely, the sum of the TMII and the DMII (change on *y* axis from point 1 to point 4).

but not necessarily at the same time. Though these interactions are often competitive (Denno et al. 1995), facilitative effects are also common (Carroll and Hoffman 1980; Haukioja et al. 1990; Karban and Baldwin 1997).

Phenotypic plasticity can also be relevant to plant-herbivore interactions when a third party (predator, mutualist, or competitor) induces changes in a plant or herbivore phenotype that then indirectly affects herbivores or plants, respectively. This is what Schmitz et al. (1998) documented when they showed that a predator caused a change in herbivore behavior that resulted in reduced damage to plants. Rudgers et al. (2003) showed not only that ants *(Forelius pruinosus)* reduced caterpillar *(Bucculatrix thuberiella)* feeding on wild cotton *(Gossypium therberi)*—a result parallel to that of Schmitz et al. (1998)—but also that ants altered the spatial distribution of both caterpillars and plant damage; ants caused caterpillars to spend relatively less time on the undersurfaces of leaves and caused feeding damage to be closer to the leaf margin. Thus, in addition to the tritrophic impact of predator-induced reduction in herbivore feeding rates, if patterns of damage are important for plant fitness

(Marquis 1992) this TMII may also have evolutionary implications for herbivore selection on plant traits.

Predators may also indirectly affect plants by altering the dynamics between herbivores and their mutualists. Mooney investigated the effects of insectivorous birds on the mutualism between *Cinara* spp. aphids and the ant *Formica podzolica* (Mooney 2006), and the consequent indirect effects of birds on ponderosa pine *(Pinus ponderosa)* growth and secondary chemistry (Mooney 2007). These studies suggested that the negative effect of birds on aphids was largely due to bird-induced changes in aphid and ant behavior that caused the mutualism to break down, and that birds increased pine growth and the concentration of several phloem monoterpenes by disrupting the ant-aphid mutualism.

Another third-party effect can be seen when one plant alters another's interaction with herbivores via competition-induced plasticity. Agrawal and Van Zandt (2003) demonstrated that milkweeds *(A. syriaca)* grown in competition with grass were taller and thinner than plants grown in the absence of competition. As a result of these induced morphological changes, plants received less herbivory by the specialist weevil *Rhyssomatus lineaticollis* that preferentially oviposited on thicker stems. Perhaps more common is the situation where competition induces changes that increase plant susceptibility to herbivores. For instance, where competition reduces resources available to plants, induced plant responses to herbivores may be subsequently lowered, indirectly leading to higher levels of herbivory in the field (Karban et al. 1989; Karban 1993; Cipollini and Bergelson 2001, 2002).

Mutualists of plants and herbivores also induce traits that affect plant-herbivore interactions. Gange and West (1994) showed that *Plantago lanceolata* grown with mutualist arbuscular mycorrhizal fungi responded to caterpillar feeding damage with increased production of secondary compounds (iridoid glycosides) compared to fungicide-treated plants, and that mutualist fungi thus indirectly reduced herbivore feeding. A similar pattern has been reported for several grass-endophyte-herbivore interactions (Eichenseer et al. 1991; Bultman and Ganey 1995; Bultman et al. 2004). Mutualist *Lasius niger* ants directly affected the aphid *Metopeurum fuscoviride* (i.e., in the absence of predators) by increasing aphid life span, development, and reproduction (Flatt and Weisser 2000), possibly because of increased feeding rate by ant-attended individuals (Herzig 1937). While the indirect effects of ant attendance on plant growth were not shown, it is likely that under some circumstances such effects occur.

Unifying Ecological and Evolutionary Concepts

The terminology and conceptual model first proposed by Abrams (1995) are now thoroughly integrated into studies of community-level interactions, and there are hundreds of studies that have identified nonconsumptive, trait-mediated interactions in a wide variety of systems (Werner and Peacor 2003; Preisser et al. 2005). These plasticity-mediated interactions have typically been interpreted in an ecological context, but of course ecological interactions frequently have important evolutionary implications. In most cases the TMIIs are *linear* indirect effects in that one organism indirectly affects another by inducing trait changes in an intermediate species. For example, in Schmitz (1998) spiders indirectly affected old-field grasses by inducing predator-avoidance behaviors in grasshoppers that reduced feeding. Just as TMIIs provide a mode of indirect interaction between two species, TMIIs likewise represent a mode for indirect selection. In the work by Schmitz (1998), one could imagine that grass fitness is increased by the TMII between spiders and grasshoppers; this TMII could reasonably be expected to lead to selection for grass traits that increased the ability of spiders to intimidate grasshoppers. Predators can also induce traits that alter the patterns of selection herbivores impose on plants. For instance, as was discussed above, Rudgers et al. (2003) showed that ants changed not only the amount of caterpillar feeding on wild cotton, but also the location of feeding. Such changes in herbivore feeding location could alter selection on plants if, for instance, certain traits that provide resistance to herbivores are present in one feeding location but not on another. Parallel scenarios can be envisioned for TMIIs in other contexts as well (Miller and Travis 1996).

Evolutionary ecologists have typically considered plasticity with respect to its adaptive value and not within the theoretical constructs of Abrams (1995) that are central to modern community ecology. Yet adaptive plasticity also functions by TMII. Instead of these indirect interactions being linear (see above), these indirect effects *loop* such that the end consequence of an induced effect is to alter the fitness of the plastic organism. For example, consider the interaction dynamics of induced defense by plants to herbivores using the ecological lexicon. Induced defense occurs when herbivore damage of plants induces plant phenotypes that reduce future herbivory. This is a TMII, where one herbivore indirectly affects another by induced changes in a plant. This TMII in itself does not affect plant fitness. The putative adaptive explanation of the induced plant phenotype only occurs when this TMII continues to propagate as an additional indirect effect to increase plant fitness, thus forming a complete interaction loop. Induced defense is thus a three-step indirect interaction (herbivore → plant → herbivore → plant) initiated and propagated by TMIIs. Indirect induced defense occurs when plants respond to herbivores by attracting herbivore enemies, increasing predation or parasitism of herbivores, which in turn reduces plant damage. This interaction is thus a four-step indirect interaction (herbivore → plant → predator → herbivore → plant), again initiated and propagated by TMIIs. Plant induction (herbivore plasticity similarly can be considered in this light: plant secondary compounds induce changes in herbivores (such as the production of P-450 detoxification enzymes) that subsequently allow for increased herbivore

feeding (plant → herbivore → plant). Looping and linear plasticity-based interactions (TMIIs) are thus analogous with respect to the interaction mechanism and only differ in that the former, but not the latter, provides putative explanations for the evolution of that plasticity.

The ecological and evolutionary consequences of phenotypic plasticity are also united through the parallel issues of ecological contingency and diffuse selection. Ecological contingency comes from the fact that the structure of any given pairwise interaction may not be fixed but rather may depend on the context in which the interaction takes place. Such "interaction modification" (Wootton 1994) is essentially a result of phenotypic plasticity, where the pairwise interaction between two species is altered because the phenotype of one or both interactors is modified by third parties. It is also now recognized that selection imposed by one species on another may be dependent on the ecological context, or "diffuse" (Iwao and Rausher 1997; Linhart et al. 2005; Strauss et al. 2005). While diffuse selection can be a result of genetic correlations, another principal cause is the change in effect of one species on another due to phenotypic plasticity. In other words, diffuse selection occurs when ecological interactions that affect fitness are context dependent (Inouye and Stinchcombe 2001). Consequently, phenotypic plasticity is one of the root causes for diffuse selection and ecological contingency.

Is Plasticity Important?

There are strong arguments and some good empirical evidence that phenotypic plasticity is an adaptive strategy in many traits of taxonomically disparate organisms with varied life histories. Consequently, opportunities exist for interactions to be trait mediated in every ecological and evolutionary context. There is a growing appreciation for the ecological and evolutionary consequences of organisms being plastic. One indication comes from the explicit comparison of TMIIs and DMIIs when they act on the same dependent variable. Many studies have compared the effects of predators on plants, when predators function as consumers of herbivores versus their effects on herbivore behavior. Preisser et al. (2005) synthesized the results of many such studies. Their meta-analysis showed that the TMIIs between predators and plants were as strong or stronger than the DMIIs. In this scenario, trait-mediated effects provided interaction pathways within food webs that supplemented the consumption-based pathways upon which most thought and theory on community ecology has historically been based. In other scenarios TMIIs may have effects of opposite sign from DMIIs. But whether TMIIs and DMIIs work in unison or against each other, an understanding is emerging that plasticity-based interactions play a central role in community structure and function and in trait evolution in species.

Evidence for the strength of TMIIs also comes from the commonness of induced resistance in plants. Induced resistance (direct and indirect) is adaptive only because of remarkably long chains of TMIIs (see above). These chains are as long or longer than those typically reported from the ecological literature. While there are arguments for why DMIIs are likely to attenuate, this may not be the case for TMIIs (Preisser et al. 2005). The fact that three-step (direct induced resistance) and four-step (indirect induced resistance) interactions are sufficiently strong and consistent to shape the evolution of plant traits is circumstantial evidence for both the ecological and evolutionary importance of interactions based on organism plasticity.

Future Directions

Individual and Combined Effects of TMIIs and DMIIs

A major focus of empirical studies on TMIIs has been to compare the strength of TMIIs, the more recently recognized mode of interaction, with DMIIs, the mode by which most interactions were historically assumed to occur. In almost all studies to date, the experimenters have assumed, either explicitly or implicitly, that TMIIs and DMIIs function additively. In other words, the assumption is that the strength of TMIIs when they operate alone is neither stronger nor weaker than their effects when they operate simultaneously with DMIIs.

In fact, no a priori reason exists to assume that TMIIs and DMIIs are additive and do not interact with each other. Where they do not function additively, estimates of the relative strengths of TMIIs and DMIIs are inaccurate because of the experimental designs typically used. The standard design employs three treatments: a control treatment without any indirect interaction, a full-effect treatment where TMIIs and DMIIs operate simultaneously, and a treatment where TMIIs operate but DMIIs are prevented from occurring. For example, in studies investigating the TMIIs and DMIIs between predators, herbivores, and plants, these treatments have been herbivores and plants alone (no TMIIs or DMIIs occurring), herbivores and plants with predators capable of consuming herbivores (lethal predators with TMIIs and DMIIs), and predator cues in the absence of herbivore consumption (risk predators with TMIIs alone). The DMII has then been calculated—under this questionable assumption of additivity—by subtracting the TMII from the full predator effect. If TMIIs and DMIIs do not function additively, the strength of TMIIs (relative to DMIIs) will have been overestimated or underestimated based on whether the combined effects of the two are synergistic (i.e., superadditive) or antagonistic (i.e., subadditive), respectively.

Given the lack of additivity in the combined effects of many other types of interactions, this assumption should be explicitly tested. Two studies have done so and outline appropriate methodologies for disentangling the independent and combined effects of TMIIs and DMIIs (Peacor and Werner 2001; Griffin and Thaler 2006): in a fourth treatment, DMIIs are imposed in the absence of TMIIs. In the tritrophic example outlined above, herbivores can be hand-removed at a rate

that mimics that of the predator in the lethal predator treatment. This added treatment thus creates a fully crossed two-by-two factorial design where DMIIs and TMIIs operate alone and in combination, thus allowing for an explicit statistical test for the interaction between these two factors (simply, the interaction term in a two-way ANOVA).

Future studies should adopt this or similar approaches that explicitly test for additivity in TMIIs and DMIIs. Indeed, both studies report nonadditive effects (Peacor and Werner 2001; Griffin and Thaler 2006). Such studies will better inform us as to the relative strengths of TMIIs and DMIIs. Perhaps of greater importance, this approach will allow us to predict under in what circumstances TMIIs and DMIIs are likely to work synergistically and increase total interaction strength, versus antagonistically and decrease total interaction strength.

Expanding the Types of Interactions

The ecological literature on TMIIs has been dominated by studies where effects are transmitted by induced changes in herbivore or predator behavior (Werner and Peacor 2003; Preisser et al. 2005). While evolutionary studies have investigated the consequences of plasticity in plants, this work has often been conducted under relatively controlled conditions with the goal of providing proof of principle rather than testing for relative ecological importance. For example, while volatiles from one plant have been shown to induce defenses and reduce herbivory in neighboring plants, the broader importance of such dynamics in a field setting is still being evaluated (Karban et al. 2004). Future studies should investigate the broad significance of plasticity in plants, and compare the strength such effects with other forms of ecological interaction (Agrawal and Van Zandt 2003; Callaway et al. 2003).

Broad Patterns?

It is presently unclear whether the commonness, magnitude, and ecological importance of phenotypic plasticity show broad patterns in terms of habitats (terrestrial verus aquatic, tropical versus temperate), trophic roles (plants, herbivores, predators, parasites), or phylogeny (angiosperms versus nonangiosperm plants, vascular versus nonvascular plants, vertebrate versus nonvertebrate animals, etc.). When contrasting habitats and trophic roles, predictions for patterns should be based on differences in the expected benefits and costs. Inherent limitations to plasticity should also be considered with respect to phylogenetic patterns.

An example of this approach is the contrast between terrestrial and aquatic systems. Though herbivore morphological and behavioral plasticity in response to predators has been more widely documented in aquatic compared to terrestrial systems (Kats and Dill 1998), it is unclear whether this is simply an artifact of research traditions. If this distinction is based in a true difference between the levels of plasticity across habitats, it may be due to the fact that chemical cues from predators persist longer and are more easily detected in water than in air. Indeed, the meta-analysis of Preisser et al. (2005) found that TMIIs were stronger in aquatic than terrestrial systems. The total effect of predators on reducing herbivory via both DMIIs and TMIIs may thus be greater in wet than dry systems, perhaps explaining in part the greater strength of trophic cascades in aquatic communities (Strong 1992).

The relative importance of plasticity may also vary predictably within communities. For instance, herbivores may be expected to respond differently to predator cues based on the foraging strategy of the predator. Schmitz and Suttle (2001) compared the effects of three species of hunting spider on grasshoppers. Grasshopper behavior changed in response to the sit-and-wait and sit-and-pursue predators, but not to the species that employed an active hunting strategy. Such predator-specific responses by herbivores would be expected if cues from active (as compared to less mobile) hunters provide unreliable information about the likelihood of future predator encounters. Plants also demonstrate specificity in their responses to herbivores (Agrawal 2000; Van Zandt and Agrawal 2004a, 2004b). One factor leading to variable responses may be differences in the information contained in feeding by different herbivore species: plants would be expected to respond differently to herbivore damage when current feeding is indicative of future feeding compared to when the information content of current feeding is relatively low (Karban et al. 1999).

Phenotypic plasticity may also be a predictive axis to explain variation in the strategies that herbivores employ in consuming plants (Karban and Agrawal 2002). Herbivores' strategies vary from the relatively passive, such as host choice (choosing the best foods to eat), to more active strategies, such as detoxification of defense chemicals. Given that generalist herbivores are more likely to employ plastic strategies, presumably because they encounter more variable hosts than specialists, we predict that one benefit of specialization is that costs of plasticity may be avoided. Circumstantially it appears that specialists that do not invest in plastic choices benefit from this lack of plasticity, especially in complex environments (Bernays 1999, 2001).

While phenotypic plasticity is often assumed to be an evolutionarily derived trait evolved from fixed strategies, this has been little tested. The data we have for induced plant responses are ambiguous in terms of the ancestral state (Thaler and Karban 1997; Heil et al. 2004). Karban and Baldwin (1997) have further argued that there is no a priori reason why we should expect induciblity to be the derived state. Furthermore, plasticity may in turn shape the macroevolution of species. According to a somewhat controversial hypothesis, phenotypic plasticity may lead to ecological success in a novel habitat that ultimately leads to evolutionary divergence and speciation (West-Eberhard 1989; Robinson and Dukas 1999). Ironically enough, evolutionary biologists have historically viewed phenotypic

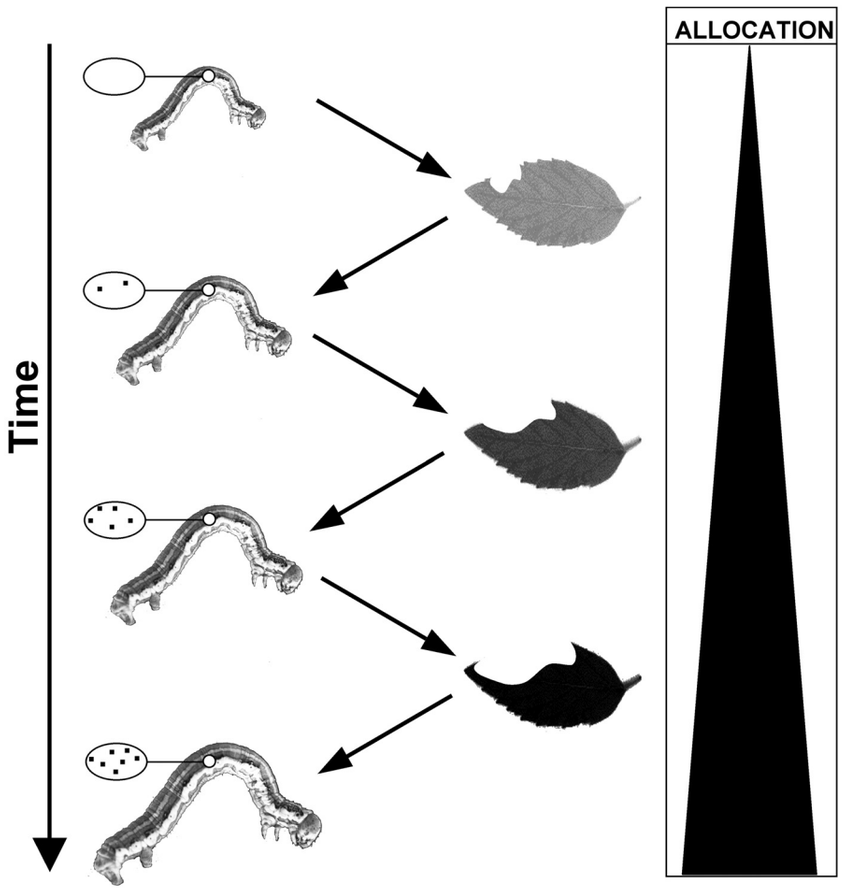

FIGURE 4.3. Graphical depiction of looping reciprocal plasticity. In this example the pairwise interaction between an herbivore and a plant escalates over time. As the herbivore feeds and grows, the phenotypes of both herbivore and plant gradually change: induced defenses increase in the plant (indicated by changes in leaf color) and induced offenses in the herbivore (indicated by dots, which represents concentration of detoxification enzymes). Allocation of resources by the plant and herbivore to the interaction increases over time. In addition to such quantitative escalation (current example), escalation could be qualitative, occurring via switches among types of defensive or offensive strategies. Looping reciprocal plasticity may occur only if both interacting parties induce dose-dependent changes in each other. See text for further discussion.

plasticity as a mechanism that reduces the response to natural selection (because the phenotype and genotype are somewhat uncoupled). However, if plasticity allows for the colonization and success in some novel habitat, and other forces (e.g., allopatry or induced preference for the novel habitat) cause restricted gene flow to organisms in the original habitat, genetic differentiation may ensue. Further studies on the macroevolutionary patterns of phenotypic plasticity are likely to be quite informative.

The above provides just a few examples of the scenarios where predictions can be made about the role of plasticity in ecological and evolutionary interactions. While these and other examples are suggestive, in no case has such a pattern been broadly demonstrated in a rigorous fashion. What these examples demonstrate is how theory can make concrete and testable predictions about broad patterns in the mechanisms by which species interact via phenotypic plasticity.

Looping with Reciprocal Plasticity

The adaptive value of plasticity often comes from the induced trait having an effect on another organism, and frequently this effect is on the same individual that caused the induction. Given that both organisms in an interacting pair are likely to demonstrate phenotypic plasticity, it is almost certain that loops of reciprocal plastic responses are common (Agrawal 2001b) (Fig. 4.3). In the context of plant-herbivore interactions, there is evidence for looping from laboratory experiments, although the structure of the interactions has not been characterized in this way. For example, a common experimental design to test for induced defense is to damage a plant with one individual herbivore (or not), and then to compare the performance and physiological adaptation of subsequent "challenge" herbivores on induced and uninduced plants. These challenge herbivores typically did not previously feed on induced plants, thus biasing results toward finding strong induced plant resistance. Indeed, given the physiological response of insects to plant responses (Broadway 1997), phenotypic looping is likely to be the rule.

While the above example discusses escalating defense and offense, similar looping may occur in the context of plant and herbivore mutualisms with third parties. For instance, plants may provide an initial reward to attract mutualist ants. This reward might increase if ants remove herbivores, thus recruiting more ants. The aphid *Tuberculatus quercicola* is tended by ants on oak trees (Yao et al. 2000; Yao and Akimoto 2001, 2002). Yao et al. have demonstrated that there

are costs and benefits of ant attendance to the aphid, and that the apparent plastic response in the aphid that regulates ant attendance is the adjustment of sugar and amino acid concentration of the honeydew secreted by aphids. Thus ants and aphids each dynamically adjust their investment in the mutualism, with aphids altering reward production and ants (at the colony level) adjust forager numbers. Similar dynamics have also been demonstrated between ants and myrmecophilous lycaenid caterpillars (Axen and Pierce 1998). Just as such looping could cause an escalation of benefit exchange, a disruption of a mutualism might similarly set off a spiral of negative consequences where pairs of mutualists reciprocally withhold benefits. Whether reciprocal plasticity occurs between antagonists or mutualists, we predict that the cycle should end when the costs of further induction outweigh the benefits for one member of the interaction, or at some physiological limit.

Several criteria define the circumstances under which we predict that reciprocal plasticity should occur. First, both interacting parties must be capable of inducing phenotypic changes in the other. These induced changes may be caused by direct interactions, but they can also be indirect as, for instance, in ant-plant mutualisms where ants indirectly benefit plants by removing herbivores. Second, phenotypic responses must be dose dependent and not binary in nature: only when plasticity is continuous is there the possibility of reciprocal escalation of responses. Third, both parties must be capable of responding dynamically to their effects on each other. In other words, the induced responses must not only be continuous, but the continuity of possible phenotypes must be driven (at least in part) by induction from the other party. For instance, in the case of plant-herbivore interactions, plants must be able to respond dynamically to levels of herbivore damage, while herbivores must respond dynamically to levels of plant defense.

One empirical approach to studying reciprocal plasticity would be to initiate a pairwise interaction and destructively subsample the replicates to generate a time series of character states for each interacting party. Some of the key questions that such a study could address are the following: (1) Is an equilibrium state (unchanging final traits) reached? (2) How different is equilibrium from the trait state following the initial interaction? Answering this question quantifies the relative importance of looping versus initial responses in a pairwise interaction. (3) Is escalation quantitative, with increases in the level of a phenotype, or qualitative, with the addition of or switching to different discrete phenotypes? In the case of qualitative changes in the phenotype over time, dose-dependent effects are still implied, but different traits may be induced only after a particular level in the interaction (i.e., thresholds of damage in the plant or poisoning in the herbivore). (4) Does history of interaction matter? If A and B are interacting over time, is the effect of A_X (i.e., A at time X) on B_X the same as the effect of A_X on B_{X-1}? In other words, if an ecologically naïve individual stepped into a reciprocal plasticity loop that had reached an escalated state, would this naïve individual be induced to a state comparable to that of individuals that had a longer history in the interaction? (5) To what extent does looping plasticity result in reciprocal shifts in interaction strength with consequences for mean fitness versus shifts in other aspects of the interaction that have consequences for selection? For example, to what extent do the induced responses of herbivores have consequences for rates of herbivore feeding (quantitative escalation which will affect plant fitness but not necessarily change the shape of selection) versus the locations on plants that herbivores feed (qualitative escalation which may alter the shape or target of selection imposed by herbivores on plants)?

A better understanding of reciprocal plasticity will be important in several contexts. First, it will provide a more detailed mechanistic understanding of the ways organisms interact. Perhaps the most important point in this regard will be to better understand whether interactions are discrete events, or whether they are dynamic process that occur over time. Second, if plastic responses result in changes in not only fitness, but also selection (item 4 above), then the point where the looping ceases or equilibrates will affect selection. More generally, it is yet to be widely recognized that the response to selection in many coevolving interactions may be phenotypically plastic. What this means is that selection in coevolving systems may leave behind the ecological signature of reciprocal plasticity.

Acknowledgments

During the preparation of this review we were supported by the Cornell Biogeochemistry and Biocomplexity Initiative and NSF DEB 0447550.

References Cited

Abrams, P. A. 1995. Implications of dynamically variable traits for identifying, classifying, and measuring direct and indirect effects in ecological communities. Am. Nat. 146: 112–134.

Agrawal, A. A. 1998. Induced responses to herbivory and increased plant performance. Science 279: 1201–1202.

Agrawal, A. A. 1999. Induced responses to herbivory in wild radish: effects on several herbivores and plant fitness. Ecology 80: 1713–1723.

Agrawal, A. A. 2000. Specificity of induced resistance in wild radish: causes and consequences for two specialist and two generalist caterpillars. Oikos 89: 493–500.

Agrawal, A. A. 2001a. Transgenerational consequences of plant responses to herbivory: an adaptive maternal effect? Am. Nat. 157: 555–569.

Agrawal, A. A. 2001b. Phenotypic plasticity in the interactions and evolution of species. Science 294: 321–326.

Agrawal, A. A. 2002. Herbivory and maternal effects: mechanisms and consequences of transgenerational induced plant resistance. Ecology 83: 3408–3415.

Agrawal, A. A., and M. Fishbein. 2006. Plant defense syndromes. Ecology 87: S132–S149.

Agrawal, A. A., and R. Karban. 1999. Why induced defenses may be favored over constitutive strategies in plants, pp. 45–61. In R. Tollrian and C. D. Harvell (eds.), The ecology and evolution of inducible defenses. Princeton University Press, Princeton, NJ.

Agrawal, A. A., and M. F. Sherriffs. 2001. Induced plant resistance and susceptibility to late-season herbivores of wild radish. Ann. Entomol. Soc. Am. 94: 71–75.

Agrawal, A. A., and P. A. Van Zandt. 2003. Ecological play in the coevolutionary theatre: genetic and environmental determinants of attack by a specialist weevil on milkweed. J. Ecol. 91: 1049–1059.

Agrawal, A. A., S. Y. Strauss, and M. J. Stout. 1999a. Costs of induced responses and tolerance to herbivory in male and female fitness components of wild radish. Evolution 53: 1093–1104.

Agrawal, A. A., C. Laforsch, and R. Tollrian. 1999b. Transgenerational induction of defences in animals and plants. Nature 401: 60–63.

Agrawal, A. A., F. Vala, and M. W. Sabelis. 2002a. Induction of preference and performance after acclimation to novel hosts in a phytophagous spider mite: adaptive plasticity? Am. Nat. 159: 553–565.

Agrawal, A. A., J. K. Conner, M. T. J. Johnson, and R. Wallsgrove. 2002b. Ecological genetics of an induced plant defense against herbivores: additive genetic variance and costs of phenotypic plasticity. Evolution 56: 2206–2213.

Appel, H. M., and M. M. Martin. 1992. Significance of metabolic load in the evolution of host specificity of *Manduca sexta*. Ecology 73: 216–228.

Axen, A. H., and N. E. Pierce. 1998. Aggregation as a cost-reducing strategy for lycaenid larvae. Behav. Ecol. 9: 109–115.

Baldwin, I. T. 1996. Methyl jasmonate–induced nicotine production in *Nicotiana attenuata*: inducing defenses in the field without wounding. Entomol. Exp. Appl. 80: 213–220.

Baldwin, I. T. 1998. Jasmonate-induced responses are costly but benefit plants under attack in native populations. Proc. Nat. Acad. Sci. USA 95: 8113–8118.

Baur, R., S. Binder, and G. Benz. 1991. Nonglandular leaf trichomes as short-term inducible defense of the gray alder, *Alnus incana* (L.), against the chrysomelid beetle, *Agelastica alni* L. Oecologia 87: 219–226.

Benrey, B., and R. F. Denno. 1997. The slow-growth-high-mortality hypothesis: a test using the cabbage butterfly. Ecology 78: 987–999.

Berenbaum, M. R., and A. R. Zangerl. 1992. Genetics of physiological and behavioral resistance to host furanocoumarins in the parsnip webworm. Evolution 46: 1373–1384.

Berenbaum, M. R., and A. R. Zangerl. 1994. Costs of inducible defense: Protein limitation, growth, and detoxification in parsnip webworms. Ecology 75: 2311–2317.

Bernays, E. A. 1986. Diet-induced head allometry among foilage-chewing insects and its importance for graminivores. Science 231:495–497.

Bernays, E. A. 1997. Feeding by lepidopteran larvae is dangerous. Ecol. Entomol. 22: 121–123.

Bernays, E. A. 1999. Plasticity and the problem of choice in food selection. Ann. Entomol. Soc. Am. 92: 944–951.

Bernays, E. A. 2001. Neural limitations in phytophagous insects: implications for diet breadth and evolution of host affiliation. Annu. Rev. Entomol. 46: 703–727.

Bodnaryk, R. P. 1994. Potent effect of jasmonates on indole glucosinolates in oilseed rape and mustard. Phytochemistry 35: 301–305.

Boland, W., J. Engelberth, J. Piel, V. Jung, A. Jux, T. Koch, T. Krumm, and G. Schuler. 1999. Induced biosynthesis of terpenoid insect semiochemicals in plants. Chemicke Listy 93: S1–S69.

Bown, A. W., D. E. Hall, and K. B. MacGregor. 2002. Insect footsteps on leaves stimulate the accumulation of 4-aminobutyrate and can be visualized through increased chlorophyll fluorescence and superoxide production. Plant Physiol. 130: 514–514.

Brattsten, L. B., C. F. Wilkinson, and T. Eisner. 1977. Herbivore-plant interactions: mixed-function oxidases and secondary plant substances. Science 196: 1349–1352.

Broadway, R. M. 1997. Dietary regulation of serine proteinases that are resistant to serine proteinase inhibitors. J. Insect Physiol. 43: 855–874.

Bultman, T. L., and D. T. Ganey. 1995. Induced resistance to fall armyworm (Lepidoptera: Noctuidae) mediated by a fungal endophyte. Environ. Entomol. 24: 1196–1200.

Bultman, T. L., G. Bell, and W. D. Martin. 2004. A fungal endophyte mediates reversal of wound-induced resistance and constrains tolerance in a grass. Ecology 85: 679–685.

Callaway, R. M., S. C. Pennings, and C. L. Richards. 2003. Phenotypic plasticity and interactions among plants. Ecology 84: 1115–1128.

Carroll, C. R., and C. A. Hoffman. 1980. Chemical feeding deterrent mobilized in response to insect herbivory and counteradaptation by *Epilachna tredecimnotata*. Science 209: 414–416.

Cipollini, D. F., and J. Bergelson. 2001. Plant density and nutrient availability constrain constitutive and wound-induced expression of trypsin inhibitors in *Brassica napus*. J. Chem. Ecol. 27: 593–610.

Cipollini, D. F., and J. Bergelson. 2002. Interspecific competition affects growth and herbivore damage of *Brassica napus* in the field. Plant Ecol. 162: 227–231.

Clancy, K. M., and P. W. Price. 1987. Rapid herbivore growth enhances enemy attack—sublethal plant defenses remain a paradox. Ecology 68: 733–737.

Cloutier, C., C. Jean, M. Fournier, S. Yelle, and D. Michaud. 2000. Adult Colorado potato beetles, *Leptinotarsa decemlineata,* compensate for nutritional stress on oryzacystatin I–transgenic potato plants by hypertrophic behavior and over-production of insensitive proteases. Arch. Insect Biochem. Physiol. 44: 69–81.

Denno, R. F., M. S. McClure, and J. R. Ott. 1995. Interspecific interactions in phytophagous insects: competition reexamined and resurrected. Annu. Rev. Entomol. 40: 297–331.

DeWitt, T. J. 1998. Costs and limits of phenotypic plasticity: tests with predator-induced morphology and life history in a freshwater snail. J. Evol. Biol. 11: 465–480.

DeWitt, T. J., A. Sih, and D. S. Wilson. 1998. Costs and limits of phenotypic plasticity. Trends Ecol. Evol. 13: 77–81.

Dicke, M., and M. W. Sabelis. 1992. Cost and benefits of chemical information conveyance: proximate and ultimate factors, pp. 122–155. In B. D. Roitberg and M. B. Isman (eds.), Insect chemical ecology. Chapman and Hall, New York.

Dorn, L. A., E. H. Pyle, and J. Schmitt. 2000. Plasticity to light cues and resources in *Arabidopsis thaliana*: testing for adaptive value and costs. Evolution 54: 1982–1994.

Dussourd, D. E. 1993. Foraging with finesse caterpillar adaptations for circumventing plant defenses. In N. E. Stamp and T. M. Casey (eds.), Caterpillars: ecological and evolutionary constraints on foraging. Chapman and Hall, New York.

Dussourd, D. E., and T. Eisner. 1987. Vein-cutting behavior: insect counterploy to the latex defense of plants. Science 237: 898–900.

Eichenseer, H., D. L. Dahlman, and L. P. Bush. 1991. Influence of endophyte infection, plant age and harvest interval on *Rhopalosiphum*

padi survival and its relation to quantity of N-formyl and N-acetyl loline in tall fescue. Entomol. Exp. Appl. 60: 29–38.

Estabrook, R. W. 1996. The remarkable P4150s: a historical overview of these versatile hemeprotein catalysts. FASEB J. 10: 202–204.

Faeth, S. H. 1991. Variable induced responses: direct and indirect effects on oak folivores, pp. 293–323. In D. W. Tallamy and M. J. Raupp (eds.), Phytochemical induction by herbivores. Wiley, New York.

Feyereisen, R. 1999. Insect P450 enzymes. Annu. Rev. Entomol. 44: 507–533.

Flat, T., and W.W, Weisser. 2000. The effects of mutualistic ants on aphid life history traits. Ecology 81: 3522–3529.

Fox, C. W., M. S. Thakar, and T. A. Mousseau. 1997. Egg size plasticity in a seed beetle: an adaptive maternal effect. Am. Nat. 149: 149–163.

Futuyma, D. J., C. Herrmann, S. Milstein, and M. C. Keese. 1993. Apparent transgenerational effects of host plant in the leaf beetle *Ophraella notulata* (Coleoptera: Chrysomelidae). Oecologia 96: 365–372.

Gange, A. C., and H. M. West. 1994. Interactions between arbuscular mycorrhizal fungi and foliar-feeding insects in *Plantago lanceolata* L. New Phytol. 128: 79–87.

Giamoustaris, A., and R. Mithen. 1995. The effect of modifying the glucosinolate content of leaves of oilseed rape (*Brassica napus* ssp. oleifera) on its interaction with specialist and generalist pests. Ann. Appl. Biol. 126: 347–363.

Gould, F. 1988. Stress specificity of maternal effects in *Heliothis virescens* (Boddie) (Lepidoptera, Noctuidae) larvae. Mem. Entomol. Soc. Can. 191–197.

Green, T. R., and C. A. Ryan. 1972. Wound induced proteinase inhibitor in plant leaves: a possible defense mechanism against insects. Science 175: 776–777.

Greene, E. 1989. A diet-induced developmental polymorphism in a caterpillar. Science 243: 643–646.

Greene, E. 1996. Effect of light quality and larval diet on morph induction in the polymorphic caterpillar *Nemoria arizonaria* (Lepidoptera: Geometridae). Biol. J. Linn. Soc. 58: 277–285.

Griffin, C. A. M., and J. S. Thaler. 2006. Insect predators affect plant resistance via density- and trait- mediated indirect interactions. Ecol. Lett. 9: 338–346.

Hairston, N. G., F. E. Smith, and L. B. Slobodkin. 1960. Community structure, population control, and competition. Am. Nat. 94: 421–425.

Haukioja, E. 1980. On the role of plant defenses in the fluctuation of herbivore populations. Oikos 35: 202–213.

Haukioja, E. 1991. Induction of defenses in trees. Annu. Rev. Entomol. 36: 25–42.

Haukioja, E., and T. Hakala. 1975. Herbivore cycles and periodic outbreaks: formation of a general hypothesis. Rept. Kevo Subarctic Res. Statn. 12: 1–9.

Haukioja, E., K. Ruohomaki, J. Senn, J. Suomela, and M. Walls. 1990. Consequences of herbivory in the mountain birch (*Betula pubescens* ssp. tortuosa): importance of the functional organization of the tree. Oecologia 82: 238–247.

Heil, M. 2004. Induction of two indirect defences benefits lima bean (*Phaseolus lunatus*, Fabaceae) in nature. J. Ecol. 92: 527–536.

Heil, M., T. Koch, A. Hilpert, B. Fiala, W. Boland, and K. E. Linsenmair. 2001. Extrafloral nectar production of the ant-associated plant, *Macaranga tanarius*, is an induced, indirect, defensive response elicited by jasmonic acid. Proc. Nat. Acad. Sci. USA 98: 1083–1088.

Heil, M., S. Greiner, H. Meimberg, R. Kruger, J. L. Noyer, G. Heubl, K. E. Linsenmair, and W. Boland. 2004. Evolutionary change from induced to constitutive expression of an indirect plant resistance. Nature 430: 205–208.

Herzig, J., 1937. Ameisen und blattläuse. Zeitschrift für Angewandte Entomologie 24: 367–435.

Hilker, M., and T. Meiners. 2002. Induction of plant responses to oviposition and feeding by herbivorous arthropods: a comparison. Entomol. Exp. Appl. 104: 181–192.

Hoballah, M. E., T. G. Kollner, J. Degenhardt, and T. C. J. Turlings. 2004. Costs of induced volatile production in maize. Oikos 105: 168–180.

Inouye, B., and J. R. Stinchcombe. 2001. Relationships between ecological interaction modifications and diffuse coevolution: similarities, differences, and causal links. Oikos 95: 353–360.

Iwao, K., and M. D. Rausher. 1997. Evolution of plant resistance to multiple herbivores: quantifying diffuse coevolution. Am. Nat. 149: 316–335.

Karban, R. 1993. Induced resistance and plant-density of a native shrub, *Gossypium thurberi*, affect its herbivores. Ecology 74: 1–8.

Karban, R., and A. A. Agrawal. 2002. Herbivore offense. Annu. Rev. Ecol. System. 33: 641–664.

Karban, R., and I. T. Baldwin. 1997. Induced responses to herbivory. University of Chicago Press, Chicago.

Karban, R., A. K. Brody, and W. C. Schnathorst. 1989. Crowding and a plant's ability to defend itself against herbivors and diseases. Am. Nat. 134: 749–760.

Karban, R., A. A. Agrawal, J. S. Thaler, and L. S. Adler. 1999. Induced plant responses and information content about risk of herbivory. Trends Ecol. Evol. 14: 443–447.

Karban, R., M. Huntzinger, and A. C. McCall. 2004. The specificity of eavesdropping on sagebrush by other plants. Ecology 85: 1846–1852.

Kats, L. B., and L. M. Dill. 1998. The scent of death: chemosensory assessment of predation risk by prey animals. Ecoscience 5: 361–394.

Kessler, A., and I. T. Baldwin. 2001. Defensive function of herbivore-induced plant volatile emissions in nature. Science 291:2141–2144.

Klemola, T., K. Ruohomaki, T. Andersson, and S. Neuvonen. 2004. Reduction in size and fecundity of the autumnal moth, *Epirrita autumnata*, in the increase phase of a population cycle. Oecologia 141: 47–56.

Krebs, R. A., and M. E. Feder. 1997. Natural variation in the expression of the heat-shock protein Hsp70 in a population of *Drosophila melanogaster* and its correlation with tolerance of ecologically relevant thermal stress. Evolution 51: 173–179.

Krieger, R. I., P. P. Feeny, and C. F. Wilkinson. 1971. Detoxication enzymes in the guts of caterpillars: an evolutionary answer to plant defenses? Science 172: 579–580.

Kursar, T. A., and P. D. Coley. 2003. Convergence in defense syndromes of young leaves in tropical rainforests. Biochem. Syst. Ecol. 31: 929–949.

Larson, K. C., and T. G. Whitham. 1991. Manipulation of food resources by a gall-forming aphid: the physiology of sink-source interactions. Oecologia 88: 15–21.

Linhart, Y. B., K. Keefover-Ring, K. A. Mooney, B. Breland, and J. D. Thompson. 2005. A chemical polymorphism in a multi-trophic setting: thyme monoterpene composition and food web structure. Am. Nat. 166: 517–529.

Malcolm, S. B. 1995. Milkweeds, monarch butterflies and the ecological significance of cardenolides. Chemoecology 5/6: 101–117.

Marquis, R. J. 1992. A bite is a bite is a bite? Constraints on response to folivory in *Piper arieianum* (Piperaceae). Ecology 73: 143–152.

Meiners, T., and M. Hilker. 2000. Induction of plant synomones by oviposition of a phytophagous insect. J. Chem. Ecol. 26: 221–232.

Miller, T. E., and J. Travis. 1996. The evolutionary role of indirect effects in communities. Ecology 77: 1329–1335.

Mitton, J. B., and K. B. Sturgeon. 1982. Bark beetles in North American conifers: a system for the study of evolutionary biology. University of Texas Press, Austin.

Mooney, K. A. 2006. The effects of birds and ants on pine herbivore load are contingent on whether birds disrupt an ant-aphid mutualism. Ecology 87: 1805–1815.

Mooney, K. A. In press. Tritrophic effects of birds and ants on a canopy foodweb, tree growth and phytochemistry. Ecology 88(8): 2005–2014.

Mullin, C., and B. Croft. 1983. Host-related alterations of detoxification enzymes in *Tetranychus urticae* (Acari: Tetranychidae). Environ. Entomol. 12: 1278–1282.

Neal, J. J. 1987. Metabolic costs of mixed-function oxidase induction in *Heliothis zea*. Entomol. Exp. Appl. 43: 175–180.

Nguyen, T. N. M., Q. G. Phan, L. P. Duong, K. P. Bertrand, and R. E. Lenski. 1989. Effects of carriage and expression of the Tn10 tetracycline-resistance operon on the fitness of *Escherichia coli* K12. Mol. Biol. Evol. 6: 213–226.

Ozawa, R., G. Arimura, J. Takabayashi, T. Shimoda, and T. Nishioka. 2000. Involvement of jasmonate- and salicylate-related signaling pathways for the production of specific herbivore-induced volatiles in plants. Plant Cell Phys. 41: 391–398.

Peacor, S. D., and E. E. Werner. 2001. The contribution of trait-mediated indirect effects to the net effects of a predator. Proc. Nat. Acad. Sci. USA 98: 3904–3908.

Preisser, E. L., D. I. Bolnick, and M. F. Benard. 2005. Scared to death? The effects of intimidation and consumption in predator-prey interactions. Ecology 86: 501–509.

Relyea, R. A. 2002. Costs of phenotypic plasticity. Am. Nat. 159: 272–282.

Robinson, B. W., and R. Dukas. 1999. The influence of phenotypic modifications on evolution: the Baldwin effect and modern perspectives. Oikos 85: 582–589.

Rossiter, M. 1996. Incidence and consequences of inherited environmental effects. Annu. Rev. Ecol. Syst. 27: 451–476.

Rotem, K., A. A. Agrawal, and L. Kott. 2003. Parental effects in *Pieris rapae* in response to variation in food quality: adaptive plasticity across generations? Ecol. Entomol. 28: 211–218.

Rudgers, J. A., J. G. Hodgen, and J. W. White. 2003. Behavioral mechanisms underlie an ant-plant mutualism. Oecologia 135: 51–59.

Scheiner, S. M., and D. Berrigan. 1998. The genetics of phenotypic plasticity. VIII. The cost of plasticity in *Daphnia pulex*. Evolution 52: 368–378.

Schmitz, O. J. 1998. Direct and indirect effects of predation and predation risk in old-field interaction webs. Am. Nat. 151: 327–342.

Schmitz, O. J., and K. B. Suttle. 2001. Effects of top predator species on direct and indirect interactions in a food web. Ecology 82: 2072–2081.

Steinger, T., B. A. Roy, and M. L. Stanton. 2003. Evolution in stressful environments. II. Adaptive value and costs of plasticity in response to low light in *Sinapis arvensis*. J. Evol. Biol. 16: 313–323.

Stone, G. N., and K. Schonrogge. 2003. The adaptive significance of insect gall morphology. Trends Ecol. Evol. 18: 512–522.

Stout, M. J., K. V. Workman, R. M. Bostock, and S. S. Duffey. 1998. Specificity of induced resistance in the tomato, *Lycopersicon esculentum*. Oecologia 113: 74–81.

Strauss, S. Y., H. Sahli, and J. K. Conner. 2005. Toward a more trait-centered approach to diffuse (co)evolution. New Phytol. 165: 81–89.

Strong, D. R. 1992. Are trophic cascades all wet? Differentiation and donor-control in speciose ecosystems. Ecology 73: 747–754.

Thaler, J. S. 1999. Jasmonate-inducible plant defences cause increased parasitism of herbivores. Nature 399: 686–688.

Thaler, J. S., and R. Karban. 1997. A phylogenetic reconstruction of constitutive and induced resistance in *Gossypium*. Am. Nat. 149: 1139–1146.

Thompson, D. B. 1992. Consumption rates and the evolution of diet-induced plasticity in the head morphology of *Melanoplus femurrubrum* (Orthoptera, Acrididae). Oecologia 89: 204–213.

Traw, M. B., and J. Bergelson. 2003. Interactive effects of jasmonic acid, salicylic acid, and gibberellin on induction of trichomes in *Arabidopsis*. Plant Physiol. 133: 1367–1375.

van Kleunen, M., M. Fischer, and B. Schmid. 2000. Costs of plasticity in foraging characteristics of the clonal plant *Ranunculus reptans*. Evolution 54: 1947–1955.

Van Tienderen, P. H. 1991. Evolution of generalists and specialists in spatially heterogeneous environments. Evolution 45: 1317–1331.

Van Zandt, P. A., and A. A. Agrawal. 2004a. Specificity of induced plant responses to specialist herbivores of the common milkweed, *Asclepias syriaca*. Oikos 104: 401–409.

Van Zandt, P. A., and A. A. Agrawal. 2004b. Community-wide impacts of herbivore-induced plant responses in milkweed (*Asclepias syriaca*). Ecology 85: 2616–2629.

Walling, L. L. 2000. The myriad plant responses to herbivores. J. Plant Growth Reg. 19: 195–216.

Werner, E. E., and S. D. Peacor. 2003. A review of trait-mediated indirect interactions in ecological communities. Ecology 84: 1083–1100.

West-Eberhard, M. J. 1989. Phenotypic plasticity and the origins of diversity. Annu. Rev. Ecol. Syst. 20: 249–278.

Wootton, J. T. 1994. The nature and consequences of indirect effects in ecological communities. Annu. Rev. Ecol. Syst. 25: 443–466.

Yao, I., and S. Akimoto. 2001. Ant attendance changes the sugar composition of the honeydew of the drepanosiphid aphid *Tuberculatus quercicola*. Oecologia 128: 36–43.

Yao, I., and S. I. Akimoto. 2002. Flexibility in the composition and concentration of amino acids in honeydew of the drepanosiphid aphid *Tuberculatus quercicola*. Ecol. Entomol. 27: 745–752.

Yao, I., H. Shibao, and S. Akimato. 2000. Costs and benefits of ant attendance to the drepanosiphid aphid *Tuberculatus quercicola*. Oikos 89: 3–10.

Young, T. P. 1987. Increased thorn length in *Acacia drepanolobium*—an induced response to browsing. Oecologia 71: 436–438.

Young, T. P., and B. D. Okello. 1998. Relaxation of an induced defense after exclusion of herbivores: spines on *Acacia drepanolobium*. Oecologia 115: 508–513.

Zavala, J. A., A. G. Patankar, K. Gase, and I. T. Baldwin. 2004a. Constitutive and inducible trypsin proteinase inhibitor production incurs large fitness costs in *Nicotiana attenuata*. Proc. Nat. Acad. Sci. USA 101: 1607–1612.

Zavala, J. A., A. G. Patankar, K. Gase, D. Q. Hui, and I. T. Baldwin. 2004b. Manipulation of endogenous trypsin proteinase inhibitor production in *Nicotiana attenuata* demonstrates their function as antiherbivore defenses. Plant Physiol. 134: 1181–1190.