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Phenotypic Plasticity in the Interactions and Evolution of Species

Anurag A. Agrawal

When individuals of two species interact, they can adjust their phenotypes in response to their respective partner, be they antagonists or mutualists. The reciprocal phenotypic change between individuals of interacting species can reflect an evolutionary response to spatial and temporal variation in species interactions and ecologically result in the structuring of food chains. The evolution of adaptive phenotypic plasticity has led to the success of organisms in novel habitats, and potentially contributes to genetic differentiation and speciation. Taken together, phenotypic responses in species interactions represent modifications that can lead to reciprocal change in ecological time, altered community patterns, and expanded evolutionary potential of species.

Phenotypic plasticity is the ability of an organism to express different phenotypes depending on the biotic or abiotic environment (Fig. 1). Single genotypes can change their chemistry, physiology, development, morphology, or behavior in response to environmental cues. R. A. Fisher and other 20th century evolutionary biologists lacked explanations for phenotypic plasticity (1). Evolutionary biologists have been interested in studying the genetic basis of phenotypes, and early work was focused on traits presumed to be unaffected by the environment. Environmentally affected phenotypes were considered of lesser importance because of their apparent lack of a genetic basis. The modern view of phenotypic plasticity rejects this notion because phenotypic plasticity often has a genetic basis. Now, many ecologists and evolutionary biologists have embraced the idea that under many circumstances such phenotypic plasticity can be adaptive (2). This hypothesis parallels Lamarck's First Law proposed in *Philosophie zoologique* in 1809 (3), which stated that organisms acclimate to their environment to improve performance. Of course, Lamarck is largely discredited by his Second Law, which suggested that these adjustments to the environment were heritable. The modern view of plasticity can be generalized to the statement that phenotypic plasticity evolves to maximize fitness in variable environments (the adaptive plasticity hypothesis) (2). Here, I take the adaptive plasticity hypothesis as a starting point for evaluating ongoing and future research directions in the ecology and evolution of species interactions.

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Reciprocal phenotypic change in species interactions. The intersection between species interactions and phenotypic plasticity has generated considerable interest among evolutionary ecologists (Table 1). The study of phenotypic responses of one organism to another is by definition an investigation of a species interaction. Yet, biologists have almost entirely focused on plasticity in species interactions as one-sided events: What is the effect of variation in species *X* on the phenotype of species *Y*? In nature, however, it is quite likely that interacting individuals are continually responding to their interaction partners in a reciprocal fashion over ecological time (Fig. 2). Reciprocal interaction simply implies a back-and-forth response in terms of phenotypic change between individuals, and does not imply symmetry in the strength of responses or effects of one partner on the other. Many antagonistic or mutualistic interactions, including those that are not behavioral, may involve reciprocal phenotypic changes in ecological time. As an analog, studies of coevolution have long attempted to study the reciprocal evolutionary change in interacting species.

Thompson (4) proposed a similar examination of phenotypic plasticity in species interactions termed an interaction norm. An interaction norm is expressed as a genotype-by-genotype-by-environment interaction. In other words, the phenotype of an individual or the sign and strength of an interaction between species is determined by the genotypes of interacting individuals and the environmental conditions in which they occur. I propose a refinement of the interaction norm concept that distinguishes environmental effects that are generated by spatial versus temporal variation. Reciprocal phenotypic change between individuals of interacting

species represents an interaction norm where the response of one species to the other creates the environment to which the other species may then respond (Fig. 3). The current sign, strength, and variability in the species interaction then depends on the past reciprocal responses between the individuals. In this simplified view, spatial aspects of the biotic and abiotic environment are assumed to be constant. The decomposition of the environmental component of the interaction norm into temporal (Fig. 3) and spatial aspects allows for a more detailed analysis of variation in species interactions.

Reciprocal phenotypic change in ecological time may be (i) a primary determinant of an organism's phenotype in nature; (ii) the result of long-term evolution where the environment (i.e., the species interaction) has been variable; and (iii) a stabilizing factor in mutualistic interactions. A signature of reciprocal phenotypic change is the escalation of phenotypes between individuals of two species over an extended bout of interactions. This can be a directional change in the phenotype of partners, where exposure to certain cues activates genes in a dose-dependent manner (Fig. 3). For example, in a mutualistic interaction, individuals may increase rewards in response to increased services from a partner, and this back-and-forth changing of phenotypes can be a continuous or iterative process. However, reciprocal phenotypic change does not have to be directional (5–7). Here again, there is an analogy to coevolutionary dynamics, where two possible outcomes are escalating (directional) arms races or polymorphisms that are stable or fluctuating in space or time. The latter case predicts high levels of genetic and phenotypic variation between populations of the interacting species because of variation in the cost-benefit ratio of a particular adaptation to the partner (8). Nondirectional phenotypic changes in ecological time may similarly result in variable phenotypes between different pairs of interacting species. Recent advances in the understanding of transposable elements suggest that stress-induced retrotransposons may be a mechanism for nondirectional change. Defensive responses in plants and animals result in increased transpositional activity that may result in immunity to parasites (6, 7).

Although studies have not been conducted

to test the specific hypothesis of reciprocal phenotypic change in ecological time, several examples suggest that it is common. Interactions between probable mutualists such as leguminous plants (Fabaceae) and nitrogen-fixing bacteria (rhizobia) demonstrate reciprocal phenotypic change (9). Bacteria near roots start by producing lipo-oligosaccharides (termed Nod factors). Plants then distort root hairs that curl around the bacteria and subse-

quently produce tubules. After additional reciprocal signaling, rhizobia differentiate into bacteroids that fix atmospheric nitrogen. Subsequent reciprocity dictates the level to which legumes and rhizobia cooperate and exchange resources (10).

Reciprocal phenotypic change has also been indicated in largely antagonistic interactions. Smith and Palmer (11) elegantly demonstrated plasticity in morphology and claw

strength of predaceous crabs: crabs eating mussels without shells grew smaller and weaker claws than crabs eating intact mussels with shells. Conversely, mussels respond to cues from predators, including crabs, by inducing increased shell thickness, abductor muscle strength, and byssal threads (12, 13). In the relationship between plants and herbivores, plants may induce defenses that are dependent on the density of attackers, and herbivores may induce counterdefenses that are dependent on the concentrations of plant defenses consumed (14–17) (Fig. 4). The continuous range of phenotypes induced by each partner exemplifies a hallmark requirement of the ecological arms race hypothesis because it allows for escalating phenotypic change. If these responses are not continuous, then “arms races” in ecological time are less likely. If reciprocal phenotypic change is the result of adaptive plasticity in both partners, then it is predicted that coevolution may result in phenotypic plasticity, as opposed to, or in addition to fixed adaptations.

Phenotypic changes in species interactions typically revert back to the original phenotypic state after each extended bout of interactions (e.g., each year). However, four possibilities may delay this resetting of the interaction: (i) Maternal environment effects may persist across generations and years (18, 19); (ii) some phenotypic modifications, such as tree responses to defoliation, occur across years (20); (iii) some phenotypic responses, especially morphological responses, are canalized and cannot revert to the original state even if the interaction is over (21); and (iv) some environments have reduced seasonality, which may prolong the duration of the interaction. Alternatively, reciprocal phenotypic responses, especially in behavioral interactions, may begin and end in a matter of seconds. Thus, the time scale of reciprocal phenotypic responses between species varies from seconds to years, and it may depend on whether the responses are chemical, physiological, morphological, or behavioral.

Phenotypic plasticity as a determinant of food chain structure. The distribution and abundance of organisms in a multitrophic community context can also be influenced by phenotypic plasticity. For example, there are manifold plastic responses of prey to predators, some of which may affect other species in an ecological community (Table 1). The consumption of prey by predators can obviously have strong ramifications for the community; however, nonconsumptive effects of predators on the phenotype of prey and lower trophic levels may be important, but they have only recently been examined. One common response of prey to the scent or visual presence of predators is to hide and/or to reduce feeding (22, 23). These behavioral responses have the potential to affect not only

Fig. 1. Two individuals of a single clone of the Asian and African water flea, *Daphnia lumholzi*. The individual on the left was exposed to chemical cues from predaceous fish (induced); the individual on the right was not (control). The sharp helmet and extended tail spine of the induced morph protect *D. lumholzi* from fish predators. The uninduced form was formerly described as a different species (*D. monacha* Brehm 1912). Green (83), in an accurate and prophetic study, related the occurrence of both morphs to differences in fish predation. The induction of this morphological defense has now been implicated as a key factor in the success of *D. lumholzi* invading North America (84).



Fig. 2. In the mutualism between many herbivorous insects and ants, herbivores produce a sugar- and amino acid-rich nectar as food for ants, and ants protect the herbivores from predators and parasitoids. Here an ant is drinking from the nectar gland of a scale insect on an aspen tree. Because the herbivores can alter the level of food rewards offered depending on the biotic and abiotic environment and the ants can similarly alter their protective services, reciprocal phenotypic change determines the ecological outcome of the species interaction. [Photograph by A. A. Agrawal]



the predator and prey but also the prey's food. Indeed, in both aquatic and terrestrial systems, it has now been shown that nonconsumptive effects of predators on prey behavior can cascade through the trophic web to have effects on standing plant biomass (22, 23). In other words, the mere threat of predation can result in decreased feeding to the point where plant biomass increases. For example, predator-induced morphological structures in an intertidal barnacle (*Chthamalus anisopoma*) resulted in complex interactions whereby the abundance of mussels was reduced and algal cover was increased (24). Because mussels and algae compete for settlement space, and the plastic response of the barnacles reduced settlement space for mussels, algae indirectly benefited from higher trophic interactions.

Phenotypic plasticity in plants can affect the abundance and distribution of herbivores and the predators and parasitoids of herbivores. In 1975, Haukioja (25) proposed that the well-documented dramatic multiyear cycles of herbivores could, in part, be mediated by plant responses that depended on the density of herbivores. In his model, herbivore populations rise until inducing a plant defense strong enough to precipitate a crash in the herbivore population. Although density-dependent induction of defenses has been documented in some systems (Fig. 4) and theoretical arguments suggest that plasticity in plant defense could cause cycles, its true

regulatory role is still unknown (15).

Plants may also affect herbivores through phenotypic plasticity in indirect defense. Herbivore-damaged plants emit volatile compounds that attract natural enemies of herbivores (26, 27). The production of plant volatiles elicits responses from natural enemies of herbivores in two ways. Predators and parasitoids are innately attracted to some plant volatiles and learn to associate other volatiles with the presence of prey (28, 29). Hence, the reciprocal plastic responses (i.e., volatile production and associative learning) can be important for the ultimate benefit to the plants. Inappropriate volatile signals or repeated exposure to volatiles without prey can backfire for the plant resulting in natural enemies of herbivores that learn to avoid plants (29). Thus, reciprocal phenotypic responses may mediate the abundance and distribution of trophic levels in food chains.

In species interactions, especially mutualisms, there is often extensive signaling between partners that may cause reciprocal phenotypic change (Fig. 2). This reciprocal change may ameliorate the conflict of interest that often ensues between the partners in mutualism: The increase in fitness of one species that comes at the expense of the other (30). For example, the interaction between lycaenid caterpillars and ants has been well studied: Caterpillars use vibrations and chemical signals to attract ants and then feed them sweet nectar; consequently, tending ants de-

fend caterpillars against predators and parasitoids. Various environmental factors including caterpillar group size and threat of predation influence the cost-benefit ratio of providing rewards to ants and, thus, dictate a caterpillar's signaling and subsequent rewards (31, 32). The level of the caterpillars' signaling and rewards reciprocally influences the number and attentiveness of mutualistic ants. Here, the multilevel responses of both partners are essential for maintaining stability in the interaction. The presence of rewards affects the abundance and distribution of ants; however, ineffective defense by ants may result in reduction in the rewards offered by caterpillars. Plants display similar flexibility in interactions with pollinating mutualists, with the ability to decrease (30) or increase (33) rewards based on previous pollination success. Thus, reciprocal phenotypic change may be an answer to the often-discussed evolutionary instability of mutualisms. Reciprocal phenotypic change may promote fidelity among mutualists, because it allows for partner choice, retaliation, or a general adjustment of rewards or services (34, 35). Genotypes that cheat may be cut off by their partner and thus disfavored by natural selection (10, 30).

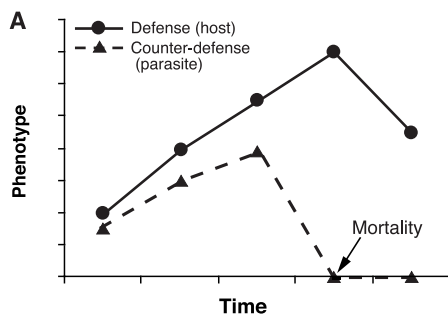


Fig. 3. (A) One possible course of reciprocal phenotypic change between individuals in an antagonistic species interaction. Key: Circles, levels of defense in a host organism; triangles, levels of counterdefense in the parasite. The dynamic nature of such increase in defense following attack and decrease following removal of parasites has been recently demonstrated for spines on *Acacia drepanolobium* that are induced by vertebrate herbivores (75). Ecological reciprocity may take place in all interactions, irrespective of the sign of effect on an individual species. However, phenotypic change in response to a species interaction need not be directional (5). (B) Colorado potato beetle (*Leptinotarsa decemlineata*) with eggs. Although damage to potato leaves by this herbivore causes an induction of plant defenses (proteinase inhibitors), following induction beetles cleverly adjust their arsenal of digestive proteases to be insensitive to plant defense (14). [Photograph by Jack Kelly Clark, ANR, University of California at Davis, ©The Regents, University of California]

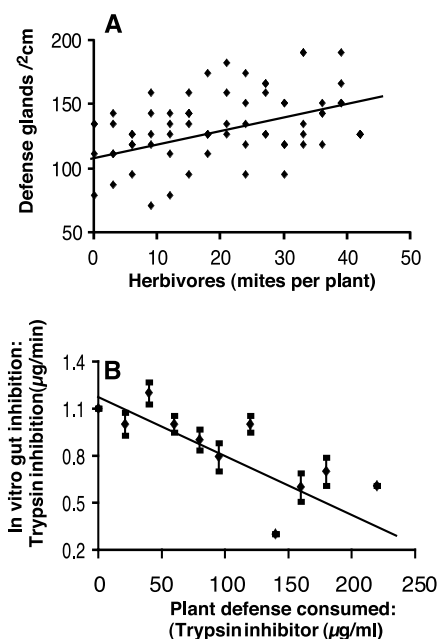


Fig. 4. Potential for an ecological arms race between plants and herbivores. (A) Phenotypic escalation in a plant defense (pigmented glands containing hemigossypolone in cotton) that is dependent on the number of attacking mite herbivores [redrawn from (16) with permission from Kluwer Academic Publishers]; and (B) phenotypic escalation in a herbivore's (*Trichoplusia ni*) counterdefense (production of inhibitor insensitive proteases) dependent on the concentration of plant defense (proteinase inhibitors) consumed. [Redrawn from (17) with permission from Elsevier Science]

Ecological and evolutionary consequences of phenotypic plasticity in novel habitats.

How does phenotypic plasticity affect ecological success and evolutionary divergence in new or novel habitats, where "habitat" refers to a biotic or abiotic neighborhood? As an example consider host-use in parasitoids, insects with a well-developed ability for associative learning. Learning can be a general form of phenotypic plasticity that organisms can apply to different environmental stimuli. Can the ability to learn, and therefore associate particular environmental cues with particular hosts, influence the potential to use new hosts and potentially the ability for a race of parasitoids to specialize on that new host? Such questions about the ecological and evolutionary consequences of plasticity have been raised in the past, as far back as Baldwin in 1896 (1, 36). Yet, empirically these questions remain completely unanswered. For example, are invasive species more phenotypically plastic than unsuccessful

invaders? Two studies highlight the fact that organisms rely on plasticity in novel habitats: (i) Lizards introduced to islands with different vegetation types and (ii) snails in habitats invaded by exotic predators were thought to have genetically adapted to these new environments (37, 38). However, in both of these cases, the respective phenotypes in the new habitats (long hind limbs and increased shell thickness) were ultimately explained by phenotypic plasticity, not genetic change (39, 40). One reason why plasticity was not strongly considered in the original studies is that the respective native habitats were apparently not particularly variable in vegetation structure and predation risk (37, 38). In another study, Fox has discovered that the host-range of a seed beetle has expanded to a new tree species and that maternal environmental effects (i.e., phenotypic plasticity) in the maternal generation and expressed in progeny) on progeny size and composition are facilitating

this shift (41). By definition, organisms in novel habitats will experience new challenges that may cause the expression of potentially beneficial traits through plasticity.

Two empirical examples demonstrate the possible role of phenotypic plasticity in evolutionary divergence. Phenotypic plasticity may have facilitated the host-shift of *Rhagoletis* flies from hawthorn to apple fruits (42, 43). Naïve adults emerging from apple hosts showed a strong preference for hawthorn in both choice and no-choice tests (42). However, those adults that experience apple in their ecological neighborhood after emerging from pupae overwhelmingly chose apple over hawthorn in subsequent oviposition. The contribution of this induction of host-fidelity to restricted gene flow and genetic substructuring has been demonstrated in nature for this system (43). Thus, phenotypic plasticity may have played a role in this sympatric speciation. Early abiotic experience in the form of light conditions of adult *Drosophila melanogaster* resulted in assortative mating in laboratory trials (44). Although the phenotype itself was unknown, the environmentally cued change resulted in organisms from similar environments preferentially mating with each other, a potential mechanism for facilitating evolutionary divergence. In both of these studies a plastic response led to assortative mating among individuals that experience the same environment.

The idea that plasticity may lead to ecological success in a novel habitat is perhaps intuitive, but the idea that plasticity may also lead to evolutionary divergence in novel habitats is less so. The hypothesis posits that plasticity may itself lead to genetic differentiation, a seeming contradiction. Here, the key is that plasticity allows for the colonization and success in a 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. If plastic organisms are restricted to or favor the novel habitat, a new host race may be formed. The eventual loss of plasticity may not be required in all cases, as it seems that *Rhagoletis* has retained its diet-induced phenotypic plasticity even in the new host race (42).

The ecological and evolutionary consequences of phenotypic plasticity depend, in part, on whether plasticity evolves in response to particular environmental variation (i.e., specialized sun versus shade leaves in plants) or evolves as an overall strategy of the organism (i.e., associative learning). For most plastic traits, the response is probably intermediate, in that the trait may be responsive to at least a few different environmental stimuli. Whether this range of response to stimuli is adaptive or not, I predict that the larger the range of stimuli to which a trait will respond, the more likely this plastic trait will affect the

Table 1. Apparently adaptive phenotypic responses of organisms in species interactions. In few of these interactions have reciprocal responses been investigated. Because of the variable nature of phenotypic plasticity, the sign of the interaction may change depending on the respective phenotypes of the interacting organisms.

Response	Reference
<i>Competition</i>	
Increased production of defensive caste in social insects	(55)
Production of defensive structures in aquatic invertebrates	(56, 57)
Production of dispersal morphs	(58)
Trophic specialization in tadpoles	(59)
Stem elongation in plants	(54)
Adjustment of progeny vigor in plants	(60)
<i>Mutualism</i>	
Rewards dependent on the presence of mutualists in plants and animals	(31, 61, 62)
Behavioral avoidance/attraction to partner depending of rewards in animals	(63, 64)
Plants punishing cheaters	(10, 30)
Adjustment of rewards according to need in plants and animals	(31–33, 65)
<i>Predation risk (animals)</i>	
Hiding, reduced activity and feeding	(22, 23)
Diet or habitat induced camouflage	(66, 67)
Induction of morphological defenses	(18, 21, 39, 68)
Production of dispersal morphs	(69)
Behavioral escape	(70, 71)
Transformation into a parasite of the predator	(72)
Adjustment of progeny defenses	(18, 73)
<i>Parasitism/herbivory</i>	
Increased immune function in animals	(68)
Induced chemical and/or morphological defense in plants	(15, 74, 75)
Increased heterophylly (in this case, producing leaves under water)	(76)
Behavioral shifts in the host (adaptive for host)	(77)
Behavioral shifts in the host (adaptive for parasite)	(78)
Gall formation on plants (adaptive for galler)	(15)
<i>Food quality (predators and herbivores)</i>	
Adjustment of gut enzymes	(14, 17, 79)
Adjustment of feeding structures	(11, 80)
Adjustment of progeny size and quality	(41, 81)
Adjustment of behavioral preferences	(82)

ecology and evolution of interactions in novel habitats. Learning and other general forms of phenotypic plasticity with responses to diverse stimuli have enormous potential as mechanisms to increase the success of organisms in novel habitats (45). If phenotypic plasticity is involved in the ecological and evolutionary success of organisms in novel habitats, then it might be expected that subdisciplines, from invasion biology to speciation, may benefit from considering the consequences of plasticity.

A unique form of phenotypic plasticity that may result in evolutionary change is mutation in response to stress. Transposable element activity increases in response to stress in both plants (6) and animals (7). Similarly, under stressful conditions organisms can produce DNA polymerases (mutases) that replicate faulty or mutated DNA, potentially introducing solutions in an environment where novelty is required (46). Such environmentally induced mutations have particularly potent evolutionary potential in clonal organisms such as microbes and plants. Even in nonclonal plants, stress-induced somatic mutations may be transferred to progeny because reproductive tissues may be differentiated from mutated somatic tissues. The adaptive benefits of high mutation rate in a novel interaction between species have now been demonstrated (47). Thus, if induced transposons and mutases have evolved by natural selection, then this form of phenotypic plasticity may be a new mechanism for generating variation subject to natural selection.

Evolution of phenotypic plasticity: Costs? Although phenotypic plasticity appears to be ubiquitous in species interactions, costs of plasticity must constrain its evolution. The following questions arise from this fundamental assumption: (i) Do costs of plasticity prevent the evolution of plasticity for particular traits; and (ii) given that plasticity has evolved in a trait, why does it not continue to evolve to the point where the species could be successful in all environments (48, 49)?

Ecological costs of plasticity are defined by a reduction in fitness of plastic organisms compared with less plastic organisms, where the fitness difference is only realized in a particular set of environments (i.e., with particular interaction partners present or during rapid environmental shifts). 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 a changing environment, lags in the response to environmental stimuli or changes in the environment that are not predictable can impose a significant ecological cost compared with that experienced by fixed

genotypes (48). These types of costs may result in selection disfavoring plasticity. For example, when transferred from antibiotic-free to antibiotic-containing media, inducible resistance to tetracycline in *Escherichia coli* is associated with a much longer lag phase in growth compared with that of bacteria constitutively expressing resistance (50).

The genetic costs of plasticity involve trade offs between the degree or pattern of plasticity and other traits that increase fitness. These costs include the maintenance of physiological machinery to sense or regulate phenotypic plasticity (i.e., an allocation cost), and are based in pleiotropy, linkage disequilibrium, or epistasis influencing other traits. The signature of genetic costs is lower fitness of plastic organisms compared with less plastic conspecific genotypes in a single environment. In the example of resistance to tetracycline in *E. coli*, possession of an inducible operon itself (in the absence of its expression or expressed and compared with a constitutively expressed genotype) has a very low fitness cost (51).

Both ecological and genetic costs of plasticity may impose an evolutionary constraint on responses to natural selection favoring plasticity, although little empirical progress has been made in this area. The most powerful approach to study the costs of phenotypic plasticity is to start with heritable variation for a plastic trait within a species. Traditionally, quantitative genetic variation within a population, selection experiments, or mutants that under- or overexpress a trait of interest have been employed. Hybridization of closely related species (52) and genetic engineering (53) has also been employed to manipulate the level of phenotypic responses in species interactions.

Knowing the mechanism of phenotypic plasticity will be of utmost importance in detecting genetic costs. Take for example, two individuals of a plant species, one that is plastic in its response to competitors and one that is not (54). The unresponsive individual may lack the receptors necessary to respond to competitors and also may lack all of the necessary physiological machinery to respond to the signal from the receptors; conversely, the unresponsive individual 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 represent potential pitfalls in detecting the genetic costs because the mechanistic basis for why some organisms are more plastic than others is not known. In the former case, genetic costs are likely detectable; in the latter case, genetic costs may be more difficult to detect. The ecological, rather than genetic, costs of plasticity may be readily determined using genetic (or phenotypic) ma-

nipulations where the mechanisms of plasticity are unknown. The ecological costs of phenotypic plasticity will be evident as long as the ecological arena for the experiment is representative of the typical (variable) habitats that organisms experience.

Conclusion. Understanding the interactions between species is an important goal of ecology and evolution. In addition to the perhaps obvious consumptive interactions between a consumer and a resource, or two exploitative competitors, we now have a firm grip on the sometimes more subtle interactions that organisms mediate through altered phenotypes when exposed to interaction partners. Because phenotypically plastic adaptations are more likely to evolve in variable environments than fixed adaptations, and species interactions are intrinsically variable in space and time, the (co)evolution of species interactions has certainly resulted in phenotypic plasticity. These phenotypic changes can result in the structuring of food chains, reciprocal ecological changes in antagonistic and mutualistic interactions, and the evolution of species. Although costs are likely to constrain the evolution of adaptive phenotypic plasticity, the ubiquity of plasticity in species interactions suggests that the benefits outweigh costs under a wide variety of conditions.

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