The Genetics of Phenotypic Plasticity. XIV. Coevolution

Samuel M. Scheiner,[1,*] Richard Gomulkiewicz,[2] and Robert D. Holt[3]

1. Division of Environmental Biology, National Science Foundation, Arlington, Virginia 22230; 2. School of Biological Sciences, Washington State University, Pullman, Washington 99164; 3. Department of Biology, University of Florida, Gainesville, Florida 32611

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abstract: Plastic changes in organisms' phenotypes can result from either abiotic or biotic effectors. Biotic effectors create the potential for a coevolutionary dynamic. Through the use of individual-based simulations, we examined the coevolutionary dynamic of two species that are phenotypically plastic. We explored two modes of biotic and abiotic interactions: ecological interactions that determine the form of natural selection and developmental interactions that determine phenotypes. Overall, coevolution had a larger effect on the evolution of phenotypic plasticity than plasticity had on the outcome of coevolution. Effects on the evolution of plasticity were greater when the fitness-maximizing coevolutionary outcomes were antagonistic between the species pair (predator-prey interactions) than when those outcomes were augmenting (competitive or mutualistic). Overall, evolution in the context of biotic interactions reduced selection for plasticity even when trait development was responding to just the abiotic environment. Thus, the evolution of phenotypic plasticity must always be interpreted in the full context of a species' ecology. Our results show how the merging of two theory domains—coevolution and phenotypic plasticity—can deepen our understanding of both and point to new empirical research.

Keywords: coevolution, competition, mutualism, phenotypic plasticity, predator-prey.

Introduction

Adaptation by natural selection often occurs in response to effects of the environment on fitness. When those environmental effects are caused by interactions with other species, there is the potential for the species to coevolve, and an extensive literature has explored this dynamic (Thompson 2005). The environment can play a second role in the evolutionary dynamic beyond the determination of fitness; it can shape the phenotype of the organism through the process of phenotypic plasticity. Again, there is an extensive literature about this process (DeWitt and Scheiner 2004). However, in all of the theoretical explorations of the evolution of phenotypic plasticity, none has examined the possibility that the environmental factor responsible for that plasticity is itself an evolving entity. In this article, we ask two complementary questions: How does adaptation by plasticity versus genetic differentiation change when the environment includes another, coevolving species? How is the process of coevolution altered by the presence of phenotypic plasticity? We will address these questions by exploring two modes of biotic and abiotic interactions: ecological interactions that determine the form of natural selection and developmental interactions that determine phenotypes.

Coevolving systems that involve plasticity are ubiquitous. For example, claw size in some crabs changes in response to prey hardness (e.g., Smith and Palmer 1994), and some marine snails will alter shell morphology in response to the presence of crabs (e.g., Appleton and Palmer 1988). When some plants experience herbivory, they alter the process of leaf development to produce tougher leaves (e.g., Walker et al. 1999) or other plant traits (Ohgushi 2012), and some insects developmentally alter their mouthparts in response to the toughness of their food (e.g., Thompson 1992). Plants can respond to changes in light quality, an indicator of the presence of other plants competing for light, by plastically altering growth form and height (e.g., Dudley and Schmitt 1996). Tadpoles can respond to decreases in food availability caused by heterospecific competition (e.g., Pfennig 1992). Plants that form mutualistic associations with root nodule–inhabiting bacteria can alter the amount of resources directed to those bacteria, depending on the level of benefit being received (e.g., Simms et al. 2006). Indeed, phenotypic plasticity underlies many examples of trait-mediated community interactions (Aschehoug and Callaway 2012; Schoener and Spiller 2012). For all of these examples, the plasticity of only one of the two species in a given interaction was measured in a particular study. Adding to this complexity, one or both species may also have a plastic response to an abiotic variable as well as or instead of its coevolving partner. As far as we are aware, no one has empirically examined the evolution of plasticity in any coevolutionary system (Robinson and Pfennig 2013), and we currently do not know what evolutionary outcomes to expect. Here we take a theoretical approach to

* Corresponding author; e-mail: sscheine@nsf.gov.

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generate expectations for a pair of interacting species distributed along an environmental gradient. In doing so, we identify which empirical systems might be most fruitful for such studies and what patterns are likely to be found.

Modeling Plasticity

In our model of developmental plasticity in response to another species, the phenotype of one species is determined first; that phenotype may be fixed or respond plastically to just the abiotic environment. Then the phenotype of the second species is determined in response to that of the first species alone or also in response to the abiotic environment. The phenotype of the first species may evolve in response to the phenotype of the second species, while the phenotype of the second species can both evolve and have a plastic phenotypic response to the first. Such plasticity could directly influence the traits observed in the second species but also indirectly modulate the outcome of evolution in the first species.

This asymmetric scenario for phenotypic determination is captured by the top half of the conceptual model shown in figure 1. This causal network encompasses the four possible ways that the phenotypes of species 1 and species 2 were determined in our model. In all cases, the phenotypes of all individuals of species 1 are determined before those of species 2. Species 1 is either nonplastic or plastic in response to the abiotic environment; species 2 is always plastic in response to the biotic environment (the mean phenotype of species 1) and sometimes plastic in response to the abiotic environment.

In all cases, an individual's fitness is determined by its phenotype, the abiotic environment, and the mean phenotype of the other species. In our model, the effects of the biotic environment—the biotic plastic cue for species 2 and the fitness effects for both species—are determined by the mean phenotype of the other species experienced in a local community. That is, we assume that a given individual interacts with the aggregate effect of all individuals of the other species within the local deme rather than as a pairwise encounter between individuals (a mean field assumption). Examples of such diffuse interactions include plants and their pollinators, herbivory by wide-ranging herbivores, and exploitative competition for pools of shared resources (e.g., phytoplankton competing for nutrients in a well-mixed water body).

An alternative approach to modeling plasticity would be to have each species respond in a continuing plastic fashion to the other species. We decided against using that approach for this study because it can lead to an infinite regress of individuals of each species altering their phenotypes in response to changes by the other species. Such a reciprocal response pattern could be modeled if development were treated as a multistage process with some stopping rule based on age or stage. Such an approach might be especially appropriate if interactions among species emerge from repeated encounters between the same individuals rather than on the basis of population means. It might also be appropriate if trait plasticity was reversible so that the phenotype of the individual changed as it interacted with different individuals (as would be appropriate for labile behavioral responses by individuals to other individuals).

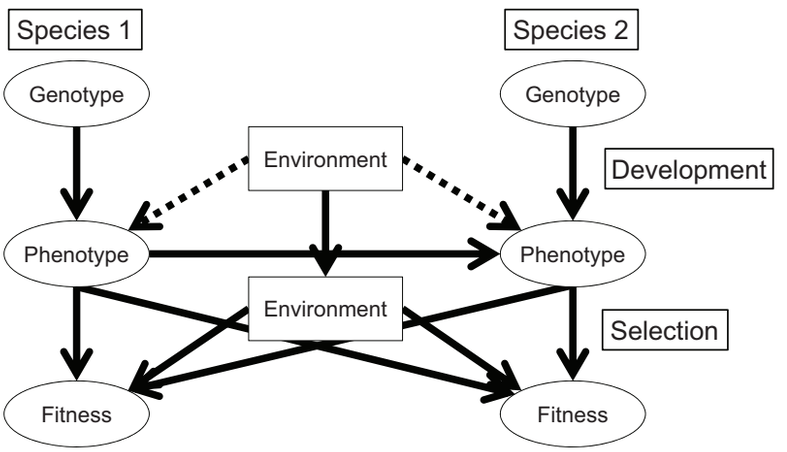

Figure 1: Relationships between genotype, phenotype, and fitness in our two-species community. The fitness of each species is a function of the abiotic environment and the phenotypes of both species. The trait value of each individual is determined by its genotype and possibly by its plastic response to the abiotic or biotic environment. The phenotype of species 2 is always dependent to some degree on the phenotype of species 1 (horizontal solid arrow). It may also depend on the abiotic environment (right dashed arrow). The phenotype of species 1 may either be nonplastic or depend on the abiotic environment (left dashed arrow).

Lande (2014) presents a single-species model for plasticity evolution of a continually changing trait that might provide a template for developing such a model that fully encapsulates symmetric and time-varying plastic responses in a coevolutionary context.

Fitness and Evolutionary Outcomes

For the ecological interactions that determine the form of natural selection (bottom half of fig. 1), we assume that the fitness of a given phenotype at a given location along an environmental gradient may be influenced both by the local abiotic environment and by local interactions with the partner species. We model the effect of the abiotic environment on fitness by a Gaussian function of the phenotype with an optimum that can vary from deme to deme (see below for details). We assume that the biotic component of an individual's fitness depends on both its own phenotype and the mean phenotype of the other species at a particular location. We further assume that the traits in each species that influence the interspecific interaction are on a common scale if similar in type (e.g., body size) or can be relativized to a common scale if different in type (e.g., levels of a toxin in a prey species and the ability to detoxify that chemical in a predator species).

We explored selection in a metacommunity of demes arrayed linearly along an environmental gradient. Selection acted via survival, which was determined by both the abiotic and biotic environments and an individual's phenotypic state. Making survival a function of the abiotic environment provides a stabilizing force on the coevolutionary process. It prevents runaway selection when coevolution favors differences in trait values between species and minimizes drift when it favors matching trait values.

Making survival a function of both the abiotic and biotic environments creates the potential for either augmenting or conflicting selection. Augmentation would be expected to occur, for example, when the interaction is mutualistic and determined by phenotype matching. In this case, selection drives both species toward matching optimal trait values based on the abiotic environment. For example, in Müllerian mimicry, two poisonous species that share a predator will converge on the same warning signal, and that warning signal may differ among locations with different visual environments. Conversely, competition based on phenotype matching results in conflict, if individuals with similar phenotypes compete more strongly; selection toward the shared abiotic optimum is opposed by competitive interactions driving phenotypes of the competing species apart.

For predator-prey interactions, we assume the predator maximizes its fitness when it can match both the abiotic optimum and the phenotype of the prey species, whereas the prey species maximizes its fitness by matching the abiotic optimum while being as different as possible from the predator's phenotype. For example, selection might be expected to act on the times of peak activity during the day for both a predator and its prey. Abiotic factors (e.g., ambient temperature) could influence the fitness consequences of activity time. If the time that is best for avoiding thermal stress is also when the prey is active, selection on the predator is augmentative (but not for the prey). This conflict can be reversed if the optimal phenotypes due to the abiotic environment differ for the predator and its prey. For example, at one end of the abiotic gradient, the prey species may be selected to be large and the predator to be small because of selection from the abiotic environment, with the opposite at the other end of the gradient, even though fitness of the predator is maximized when it is large wherever the prey is large.

This interplay between augmentation and conflict also plays out in selection on phenotypic plasticity, especially when plasticity is in response to both the biotic and the abiotic environments. Plasticity is favored when the environment at the time of development provides a reliable cue about the environment at the time of selection (the theory of the evolution of phenotypic plasticity proposition 4; see appendix in Scheiner 2013). In the case of mutualism, the abiotic and biotic signals augment each other because the fitness of both species is maximized when their phenotypes match each other and both match the optimal phenotype determined by the abiotic environment. In contrast, consider the case of competitive species where the phenotype of the nonplastic species matches the optimal phenotype determined by the abiotic environment. For the plastic species, selection by the abiotic environment would favor plasticity that matches the abiotic optimal phenotype, whereas selection by the biotic environment (the phenotype of the other species) would favor plasticity that moves the phenotype away from the abiotic optimum.

Our article investigates how these potential patterns of augmentation and conflict play out in a two-species coevolving community distributed along an environmental gradient. First, we consider all three types of species ecological interactions—competition, mutualism, and predator-prey—for the case of a single type of phenotypic determination: species 1 nonplastic and species 2 plastic only in response to the biotic environment. Second, we consider all types of phenotypic determination for a single type of ecological interaction—predator-prey—because those revealed the most disparate outcomes across the interaction types. A full exposition of all possible ecological interactions crossed with all types of phenotypic determination are beyond the scale of what can be presented here.

Adaptive evolution of plasticity requires a variable environment. In this article, we examine an abiotic environ-

ment that varies over space but not through time. We focused on spatial abiotic variation for two reasons. First, with regard to coevolution, we wanted to compare our results with those of Nuismer et al. (2010) concerning patterns of phenotypic correlation between interacting species distributed across communities in a landscape. More broadly, we are interested in how plasticity might modulate coevolution in a landscape mosaic (Thompson 2005). Second, with regard to phenotypic plasticity, Scheiner (2013) showed that the combination of spatial and temporal variation can sometimes lead to highly complex patterns of plasticity evolution. That article also compared the effects of pure spatial variation with those of pure temporal variation and showed that they can have very different propensities to select for plasticity (for a detailed discussion, see Scheiner 2013). Even in a constant environment, species interactions on their own can lead to fluctuating strengths of selection as a result of unstable population dynamics (e.g., predator-prey cycles). Because this article is an exploratory study, such added complexities are best left to a future manuscript.

Dispersal is critical to the process of adaptation to a spatially variable environment for several reasons. First, dispersal tends to increase the local heritable genetic variation of quantitative traits (Barton 1999), thereby enabling relatively larger evolutionary responses to local selection. Second, the local fitness of a dispersing individual is inversely related to the distance between the optimal phenotype favored in its environment of origination and development and that favored in its new location. (In our model, species disperse along an environmental gradient so that dispersal distance is related to the distance between these optima; the further individuals disperse, the more maladapted they are likely to be.) In addition, immigrants impose a migrational load by mating with residents. The net effect of movement is thus to depress mean local fitness and to push local populations away from their optimum; this increases the intensity of local directional selection (e.g., Polechová et al. 2009). In general, given density dependence, dispersal can also indirectly influence fitness and selection by perturbing local population size (Gomulkiewicz et al. 1999); in the models we use, however, we assume a form of density dependence where this does not happen. All of these effects of dispersal on adaptive evolution apply to any trait—including phenotype plasticity per se—and to patterns of spatial variation in fitness determined by abiotic or biotic causes.

Beyond these effects, dispersal plays an additional role specifically with regard to the evolution of plasticity. In our mode, we assume that movement occurs before selection. Dispersal then creates intergenerational variation experienced by a given lineage, because some descendants will be in a range of environments broader than that experienced by their ancestor. When selection acts along an abiotic gradient with a spatially varying phenotypic optima, higher dispersal rates increasingly favor plasticity (Scheiner and Holt 2012; Scheiner 2013). In the context of this study, that effect might differ, however, because both species are moving, possibly changing how that variation is perceived by each species. It is the combination of variation among generations and certainty within a generation that favors plasticity (Scheiner and Holt 2012). In our simulations, varying dispersal rate—and thus how differences in environmental variation are experienced over many generations—provides a useful tool for revealing how different types of ecological and developmental interactions respond to spatial variation.

It is far from straightforward to intuit how dispersal might affect coevolution in response to spatial variability in both the biotic and the abiotic environments, given phenotypic plasticity. Previous studies of interacting species distributed over space show that in the absence of phenotypic plasticity, dispersal can have important influences on coevolution in some cases (e.g., Gandon and Nuismer 2009) but not others (e.g., Nuismer et al. 2010). As we show here, the effects of dispersal and, indeed, the outcomes of evolution (including plasticity) are especially complex when abiotic and biotic sources of environmental variability generate conflicting patterns of selection across space.

The Model

Model Structure

Our model is an individual-based simulation of quantitative trait evolution for two interacting species with discrete, nonoverlapping generations distributed along a single environmental gradient. The model was implemented in Fortran 77 (see supplementary material, available online). A summary of parameters is given in table 1.

The genotype of an individual consisted of two types of loci: (1) genes with deterministic expression (i.e., no developmental noise) that was independent of the environment (nonplastic loci) and (2) genes with deterministic expression that was dependent on the abiotic and/or biotic environment (plastic loci). As limiting cases, spatial variation in adaptation can occur by two routes: genetic differentiation in which the allelic values of the plastic loci go to 0 (i.e., are not expressed) or phenotypic plasticity in which the allelic values of the nonplastic loci go to 0 (i.e., are not expressed). Intermediate outcomes are possible in which individuals express the optimal phenotype in a particular environment through nonzero values of both the plastic and the nonplastic loci.

The metacommunity consisted of a linear array of 50 demes (indexed by i from 1 to 50). The length of the gradient was chosen to minimize edge effects and allow comparisons with previous studies (Scheiner and Holt 2012;

Table 1: Summary of the model parameters

Parameters	Values
Parameters explored:	
Source of plasticity cue	Biotic versus abiotic
Type of species interaction	Mutualism, competition, predator-prey
Dispersal rate	5%–84%
Direction of selection gradient for each species	Same versus opposite
Fixed parameters:	
No. nonplastic and plastic loci	5 each
Steepness of gradient (change in optimum in adjacent demes)	.1 units
Length of environmental gradient	50 demes
Strength of selection within demes (σ)	2 units
Strength of biotic interaction (α)	.1
Life-history pattern	Selection before dispersal
Population size	100 individuals/deme/species
Mutation rate	10%/allele/generation
Mutational effect (standard deviation)	.1 units
No. generations	10,000
No. runs per parameter combination	20

Scheiner 2013). (Our conclusions would not change if the demes at or near the ends of the gradient were not included in any calculations.) An abiotic environmental gradient was created by varying the optimal value (θ_i) of a single trait (phenotype) in a linear increasing fashion along the array from -2.45 arbitrary units at one end of the gradient to $+2.45$ arbitrary units at the other; that is, the optimal abiotic phenotypes in adjacent demes differed by 0.1 units. The community consisted of two interacting species. Either the phenotype of species 1 was not plastic or it responded in a plastic manner to the abiotic environment only. The phenotype of species 2 was always plastic, with the plastic response due to the biotic environment only or to both the biotic and abiotic environment. For comparative purposes, we also modeled cases where the community consisted of just a single species that was plastic in response to the abiotic environment only and cases where the community consisted of two species, neither of which was plastic.

The life-history pattern was as follows: birth, followed by development (i.e., the phase in the life cycle when the phenotype is determined), then selection, dispersal, and reproduction. Rates of dispersal and reproduction were assumed to be invariant across the gradient. This is the "select first" life-history pattern of our previous models (Scheiner and Holt 2012; Scheiner 2013). For this life-history pattern, the environment of development provides a very reliable cue to the environment of selection. Our previous modeling for this life history showed that in response to just an abiotic gradient, plasticity is strongly favored at intermediate to high dispersal rates (Scheiner and Holt 2012).

For development of species 1, an individual's phenotype (trait value) was determined by 10 unlinked diploid loci: five nonplastic loci and five plastic loci. The loci contributed additively to the trait. Allelic values at the plastic loci were multiplied by an environment-dependent quantity before being summed over all allelic values. The effect of the environment (E_i for deme i) on the phenotypic contribution of each unit of plastic allelic value varied in a linear fashion with position along the array, with a slope of 0.01 units; specifically, we used in our simulations $E_i = 0.01(i - 25.5)$ (changing these particular parameters values in effect just rescales dispersal relative to the slope of the gradient and does not alter our qualitative conclusions). For species 1, the phenotype of each individual was determined at the time of development as

$$T_{1ij} = \sum_{k=1,10} N_{1ijk} + E_i \sum_{k=1,10} P_{1ijk}, \quad (1)$$

where T_{1ij} is the phenotype of the jth individual that develops in the ith deme, N_{1ijk} is the allelic value of the kth nonplastic allele of that individual, and P_{1ijk} is the allelic value of the kth plastic allele. For a given genotype, the quantity ΣN_{1ijk} can also be thought of as the intercept of its reaction norm at the point along the gradient where $E_i = 0$ or the phenotype of the individual in the absence of plasticity, and the slope of $(E_i)\Sigma P_{1ijk}$ calculated across demes can be thought of as the slope of its reaction norm. Thus, both the optimal phenotype due to the abiotic environment and the reaction norm due to plasticity were linear across environments. If species 1 was not plastic, the environmental effect was set to 0.

When new offspring were generated, each allele at each locus mutated with a probability of 10%. (Lower mutation rates changes the timescale over which evolution happens

rather than the eventual outcome for the models considered here [Scheiner and Holt 2012].) When a mutation occurred, the allelic value was changed by adding a Gaussian deviate (mean of 0 and a standard deviation of 0.1 units) to the previous allelic value (i.e., this is a continuum of alleles model; Kimura 1965). Allelic values were unconstrained. Both the plastic and the nonplastic loci—and the subsequent phenotypes—could take any value from $-\infty$ to ∞, and those phenotypes could be the result of just the plastic loci, just the nonplastic loci, or some combination of both.

For development of species 2, the phenotype was again determined by five nonplastic and five plastic loci, but in contrast to species 1, the phenotype of this species could respond plastically to the mean phenotype of species 1 and, in some cases, the abiotic environment as a linear function of these plastic loci:

$$T_{2ij} = \sum_{k=1,10} N_{2ijk} + (E_i + z_{1i}) \sum_{k=1,10} P_{2ijk}, \tag{2}$$

where z_{1i} is the mean phenotype of species 1 in the ith deme, and the other quantities are as defined above for species 1. If species 2 was plastic only in response to its biotic environment, the abiotic environmental effect (E_i) was set to 0. In this case, evolution in species 1 that changes its phenotype leads to corresponding variation in the realized phenotypes of individuals in species 2, responding developmentally to trait values of species 1.

Selection was based on survival, with the probability of surviving being a Gaussian function of the difference between an individual's phenotype and both the locally optimal phenotype and the mean phenotype of the other species. Fitness (a term that we use as shorthand for one component of fitness: the probability of surviving after birth before dispersal and reproduction) was determined as the product of the effects of the fitness effects of the abiotic and biotic environments: $W_{ij} = Wa_{ij} \times Wb_{ij}$, where

$$Wa_{ij} = \exp\left[-\frac{1}{2}\left(T_{ij} - \frac{\theta_i}{\sigma}\right)^2\right], \tag{3a}$$

$$Wb_{ij} = \frac{1 + I\exp\left[-\alpha(T_{ij} - z_{si})^2/(1 + 2\alpha z_{si})\right]}{2\sqrt{1 + 2\alpha z_{si}}}, \tag{3b}$$

where W_{ij} is the fitness of the jth individual in the ith deme, T_{ij} is the phenotype of that individual, $\theta_i = 0.1(i - 25.5)$ is the abiotic optimal phenotype in that deme, σ is the strength of selection from abiotic factors (selection weakens as σ increases), z_{si} is the mean phenotype of the other species in the ith deme, and α is the strength of the biotic interaction. The biotic effect is maximized when an individual's phenotype matches the other species' mean phenotype; I is -1 for negative biotic effects (competition and prey species) or 1 for positive biotic effects (mutualism and predator species). (Eq. [3b] is the phenotypic-matching fitness function of Nuismer et al. [2010]. For a visualization of its form, see fig. 1 in that article.) For all simulations, we set $\sigma = 2$; the length of the spatial gradient across all demes was then approximately 1.25 times the width (2σ) of the within-deme selection function, that is, weak abiotic selection. For the parameters used here, the reaction norm that maximized fitness with respect to the abiotic gradient had a slope of 0.1.

After selection, surviving individuals could disperse. Dispersal occurred in a single bout of movement per generation. The dispersal probability and the distance moved were determined using a zero-mean Gaussian random number (with the truncated magnitude determining the distance, and the sign the direction), so that the probability of moving and the average distance moved were correlated (see fig. 1 of Scheiner and Holt 2012). Increasing the dispersal probability (also called the dispersal rate) was done by increasing the variance of the Gaussian, so that at a higher dispersal rate, more individuals were likely to move, and they were also likely to move farther. Individuals that would otherwise disperse beyond the end of the gradient moved to the terminal demes and stayed there. Dispersal per se had no cost; namely, survival during dispersal was 100%. Unless otherwise noted, dispersal rates were the same for both species.

Sexual reproduction was accomplished by assembling pairs of individuals within a deme at random with replacement (allowing for self-fertilization), with each parent producing a haploid gamete of 10 unlinked alleles. Each pair then produced one offspring, repeating until the carrying capacity of that deme (100 for all demes) was reached. This procedure assumes soft selection, in that local population size (after reproduction) was determined independently of the outcome of selection. This implies that individuals in effect compete to produce successful offspring. This protocol also implicitly assumes that no local populations become extinct; with the strengths of selection used here, extinctions never occurred. The model assumes that the spatial scale of reproduction and mating matches that of density dependence and the grain of the selective environment. Maintaining constant deme size also means that species interactions did not explicitly depend on density but only on mean local trait values. Introducing such density dependence would be an interesting extension for future work.

Each simulation was initialized with 100 individuals of each species being born in each deme. For each individual in the initial generation, allelic values for both plastic and nonplastic loci were chosen independently from the values -2, -1, 0, 1, and 2, with each value being equally likely. Even though the alleles are integer-valued initially, their values could assume any real number in subsequent generations due to mutation. All simulations were run for 10,000 generations to ensure that equilibrium (the point after which all calculated quantities showed no further ob-

vious directional trend) was reached. Each parameter combination was replicated 20 times, and the results shown are the means of those replicates. Coefficients of variation of reported parameters were generally low (1%–5%).

Response Variables

We examined one measure of phenotypic plasticity and two measures of coevolution. For phenotypic plasticity, the reaction norm describes the phenotypes that are actually or potentially expressed by a given genotype in all environments. For a reaction norm that is linear over an environmental gradient, its plasticity can be well characterized by its slope over this gradient. In our model, the slope of the reaction norm for an individual j of species x ($=1, 2$) in deme i is the coefficient ΣP_{1ijk} in the right-hand products of equation (1) and (2) when normalized against the rate of environmental change along the gradient.

For these simulations, as the slope of E_i was constant, the final outcome was measured as the average across all demes of the sum of the values of the plasticity alleles for each individual born in the final generation. That is, $\overline{P}_i = (1/R)\sum_{n=1,R}[(1/N)\sum_{j=1,N} P_{ijn}]$, where $\overline{P}_i$ is the mean plasticity of the ith deme over all R runs (20), $N = 100$ is the number of individuals per deme, and P_{ijn} is the sum of the values of the plasticity alleles of the jth individual developing in the ith deme in the nth run. The overall mean plasticity $\overline{P}$ is the average of $\overline{P}_i$ across deme, and is given by $\overline{P} = (1/D)\sum_{i=1,D} P_i$, where D is the number of demes (50). (The order of averaging—over runs within demes first or over demes within runs first—does not affect the final average, because the number of demes was the same for all runs.) This average plasticity was standardized relative to the slope of the local abiotic optima θ_i so that a reaction norm with a relative plasticity of 1 has the same slope as the θ_i; a pure differentiation outcome (where all phenotypic differentiation across space reflects genetic differentiation) would have a relative plasticity of 0. A reaction norm with relative plasticity equal to 1 is not necessarily an evolutionary optimum (Lande 2014); in fact, our results include outcomes with relative plasticities above and below 1 and even negative values (slopes in a direction opposite of that of the θ_i).

Coevolution was assessed by two measures of the extent to which the phenotypes of the two species matched each other or were otherwise affected by the presence of the other species. First, we measured the slope of the expressed phenotype of each species (slope of the mean T_{ij}), standardized against the slope of the local abiotic optima θ_i in the absence of the other species. The mean phenotype in each deme for each species was calculated as $\overline{T}_i = (1/R) \times \sum_{n=1,R}[(1/N)\sum_{j=1,N} T_{ijn}]$, where $\overline{T}_i$ is the mean phenotype of the ith deme over all R runs, N is the number of individuals per deme, and T_{ijn} is the phenotype of the jth individual born in the final generation in the ith deme in the nth run. This standardization is similar to that of relative plasticity so that a perfect match of mean phenotypes to the local abiotic optima equals 1. Second, we measured the cross-deme correlation coefficient of those phenotypes between the two species, $\rho = \text{correlation}(\overline{T}_{1i}\overline{T}_{2i})$, where $\overline{T}_{1i}$ and $\overline{T}_{2i}$ are the means of species 1 and 2 in the ith deme.

Results

Comparisons among Types of Ecological Interactions

Equal Dispersal Rates between Species. We first focus on comparisons among different types of ecological interac-

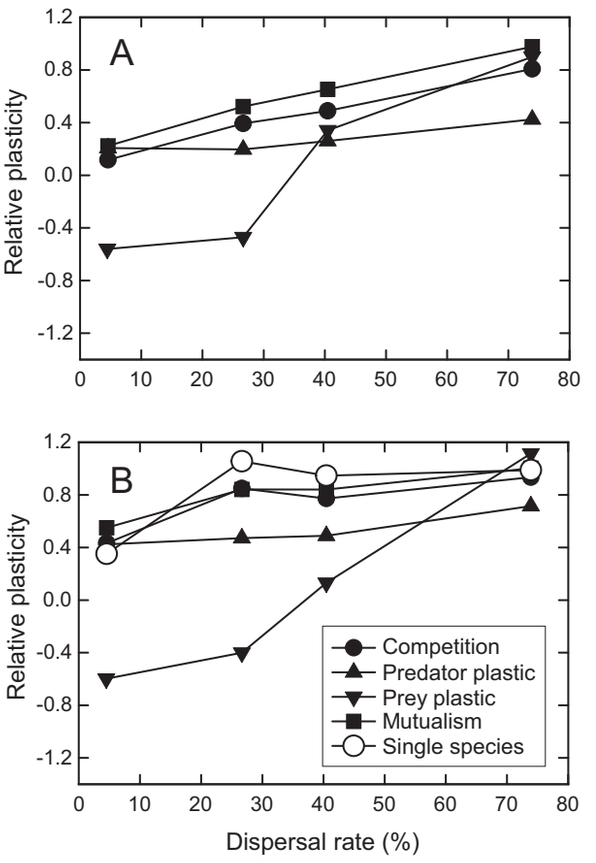

Figure 2: Effect of dispersal rate (probability) and type of species interaction on selection for phenotypic plasticity (mean P_{ij}) of species 2. Plasticity is relative to the optimal reaction norm for the abiotic gradient. Species 1 was not phenotypically plastic. *A*, Phenotypic plasticity due to the biotic environment only (pattern 1). *B*, Phenotypic plasticity due to the biotic and abiotic environments (pattern 2). Open circles show the effect of dispersal rate on selection for phenotypic plasticity when there is only one species and plasticity is in response to the abiotic environment only.

tions that determine the form of natural selection (bottom half of fig. 1), comparing competition, mutualism, and predator-prey interactions. We consider these interactions with regard to two forms of interaction that determine the phenotype: trait development depending on just the phenotype of the other species or also jointly depending on the abiotic environment. When only one species (species 2) was plastic, the extent to which spatial variation in a trait evolves through plasticity rather than genetic differentiation depends on the type of species interaction, the determinants of trait development, and the rate of dispersal (fig. 2).

For competitive and mutualistic interactions, increasing dispersal rates resulted in greater trait plasticity of species 2 (mean P_{ij}). Comparing plasticity due to the biotic environment only (fig. 2*A*) with plasticity due to both the biotic and abiotic environments (fig. 2*B*), the latter resulted in greater plasticity at low dispersal rates. At high dispersal rates, the amount of plasticity was similar to the single-species, abiotic-only response. That plasticity due to just the biotic environment is favored most at high dispersal rates implies, for example, that for tadpoles competing for food or for plant-bacterial mutualistic associations, plasticity would be favored by decreased philopatry of the tadpoles or by high dispersal rates of the bacteria.

For predator-prey interactions, the effect of dispersal rate on the amount of plasticity was greatest when the prey species was plastic, and least when the predator was plastic. For the prey species, at low dispersal rates, its relative plasticity was negative, indicating that the reaction norm sloped in the opposite direction from what would be expected from the optimum of the abiotic gradient. Because the abiotic gradient selects for the same phenotype in the predator and the prey species, at low dispersal rates, the fitness of the prey species is maximized whenever its phenotype is as different from the predator as possible. As with the other types of interactions, plasticity was greater in magnitude when it was due to both the abiotic and the biotic environments. These results imply, for example, that for traits involving antagonistic plant-herbivore interactions, the plant traits are more likely to be plastic than are the herbivore traits.

In contrast to the effect of coevolution on the evolution of trait plasticity, trait plasticity had only modest effects on coevolution, as measured by the among-deme correlations between the two species (fig. 3). As expected, at low dispersal rates, the correlation was near unity for mutualistic interactions, zero to negative for competitive interactions, and intermediate for predator-prey interactions. All correlations tended to increase with dispersal rate and tended to be similar whether trait plasticity was in response to just the other species or to both the other species and the abiotic environment, or there was no plasticity.

Different Dispersal Rates. In the previous comparison, both species dispersed at the same rate. We next compared the effect of different dispersal rates between the species responding in a plastic fashion to the other species but

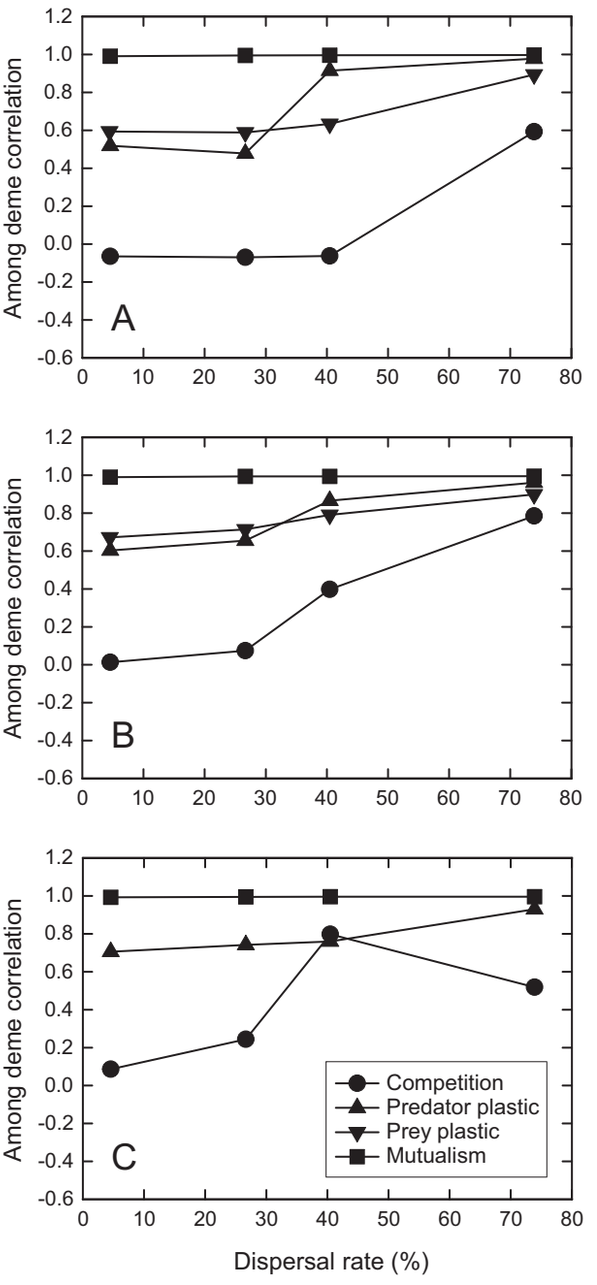

Figure 3: Effect of dispersal rate and type of species interaction on the phenotypic correlation between the two species among demes. Species 1 was not phenotypically plastic. *A*, Phenotypic plasticity due to the biotic environment only (pattern 1). *B*, Phenotypic plasticity due to both the biotic and the abiotic environment (pattern 2). *C*, No phenotypic plasticity.

not the abiotic environment (species 2 in fig. 1, which responded plastically only to species 1) and a nonplastic species (species 1 in fig. 1). Such asymmetric dispersal rates are quite likely; for example, plants may disperse less readily than do their herbivores or bacterial mutualists, while among competing marine invertebrates, some have long-lived, highly dispersing pelagic larvae and others have larvae that settle quickly.

In our model, if the interaction was mutualistic or the predator species was plastic, the dispersal rate of the nonplastic species had no effect on the evolution of trait plasticity in the other species (fig. 4*B*, 4*C*). By contrast, when the interaction was competitive or the prey species was plastic, trait plasticity increased with the dispersal rate of the nonplastic species (fig. 4*A*, 4*D*). That is, the dispersal rate of the nonplastic species mattered for the evolution of plasticity only when the abiotic selective optimum conflicted with the biotic selective optimum. This effect was greatest for low dispersal rates of the plastic species, especially for predator-prey interactions. In all cases, plasticity tended to

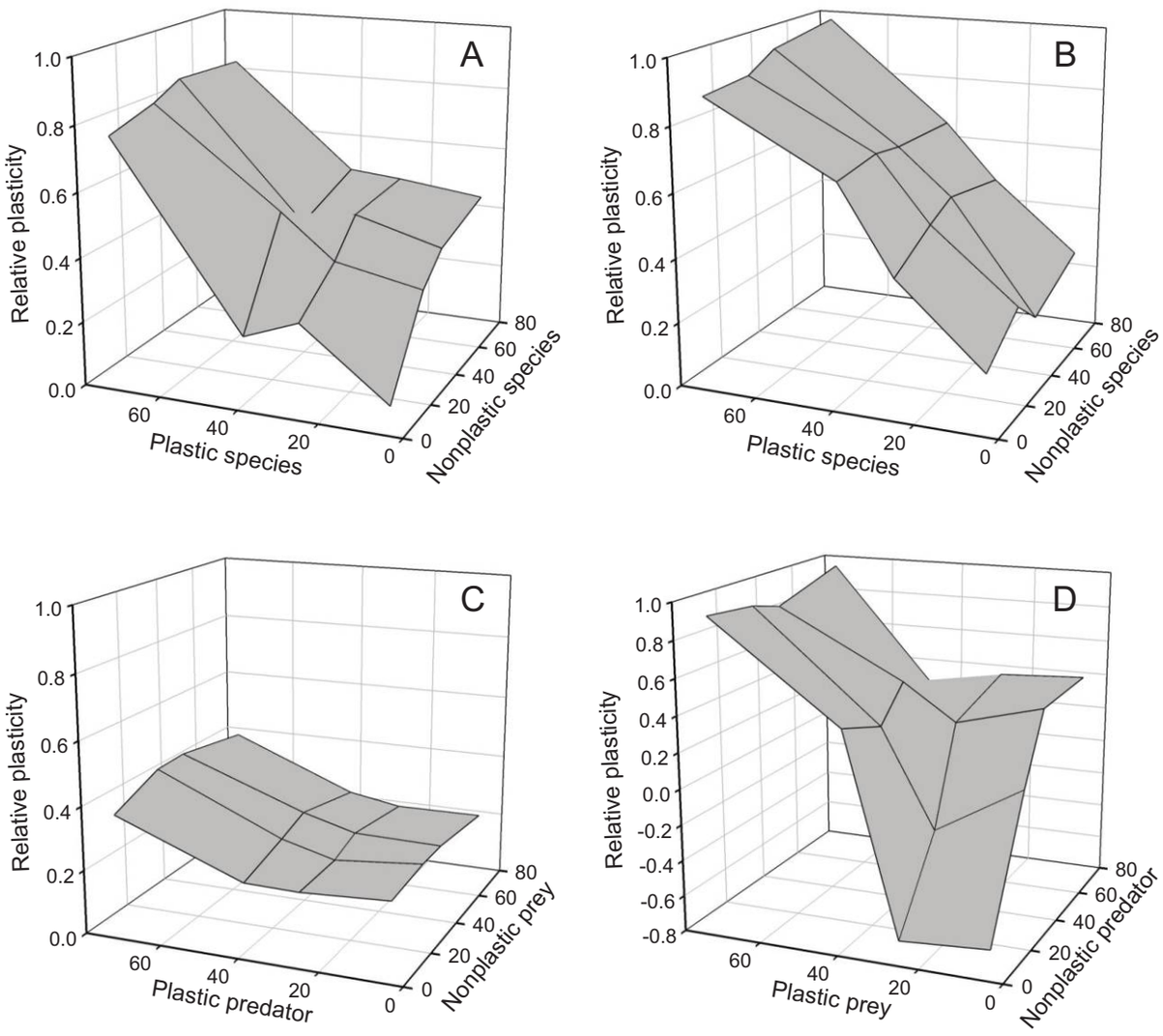

Figure 4: Effect of dispersal rates of the interacting species (axes on bottom plane) on selection for phenotypic plasticity (mean P_{ij}) of species 2. Plasticity is relative to the optimal reaction norm for the abiotic gradient. Species 1 was not phenotypically plastic, and phenotypic plasticity was due to the biotic environment only (pattern 1). *A*, Competition. *B*, Mutualism. *C*, Predator plastic. *D*, Prey plastic.

increase with increasing dispersal rate of the plastic species, although this effect was weak when the predator was plastic. These results imply, for example, that for traits involving plant-herbivore interactions, the traits of the plant are more likely to be plastic than are those of the herbivore.

Comparisons among Types of Phenotypic Determinations

Abiotic and Biotic Selection in Agreement. Next we focus on comparisons among different types of interactions that determine the phenotype (top half of fig. 1). We compared all four possible patterns: (1) species 1 nonplastic and species 2 plastic to just its biotic environment; (2) species 1 nonplastic and species 2 responding to both its biotic and abiotic environment; (3) species 1 plastic to the abiotic environment and species 2 responding to just its biotic environment; and (4) species 1 responding to the abiotic environment and species 2 responding to both its biotic and abiotic environment. The first two patterns are those explored above; in these comparisons, species 1 can now also be plastic. For these comparisons, we focused on predator-prey interactions, since they were found to be the most disparate in the previous comparisons. Examples of prey traits that might depend on both biotic and abiotic conditions include nitrogen-based chemical defenses of plants and calcium-based shell hardness of mollusks. In both cases, selection on the prey could be influenced by environmental availability of these limiting resources, and plasticity might jointly depend on both the abiotic and the biotic environment. If predator-prey interactions depend on the timing of events, then both species might use cues—such as day length or total heat days—to determine life-history events. Again, fitness might be influenced by both species' interactions and abiotic conditions.

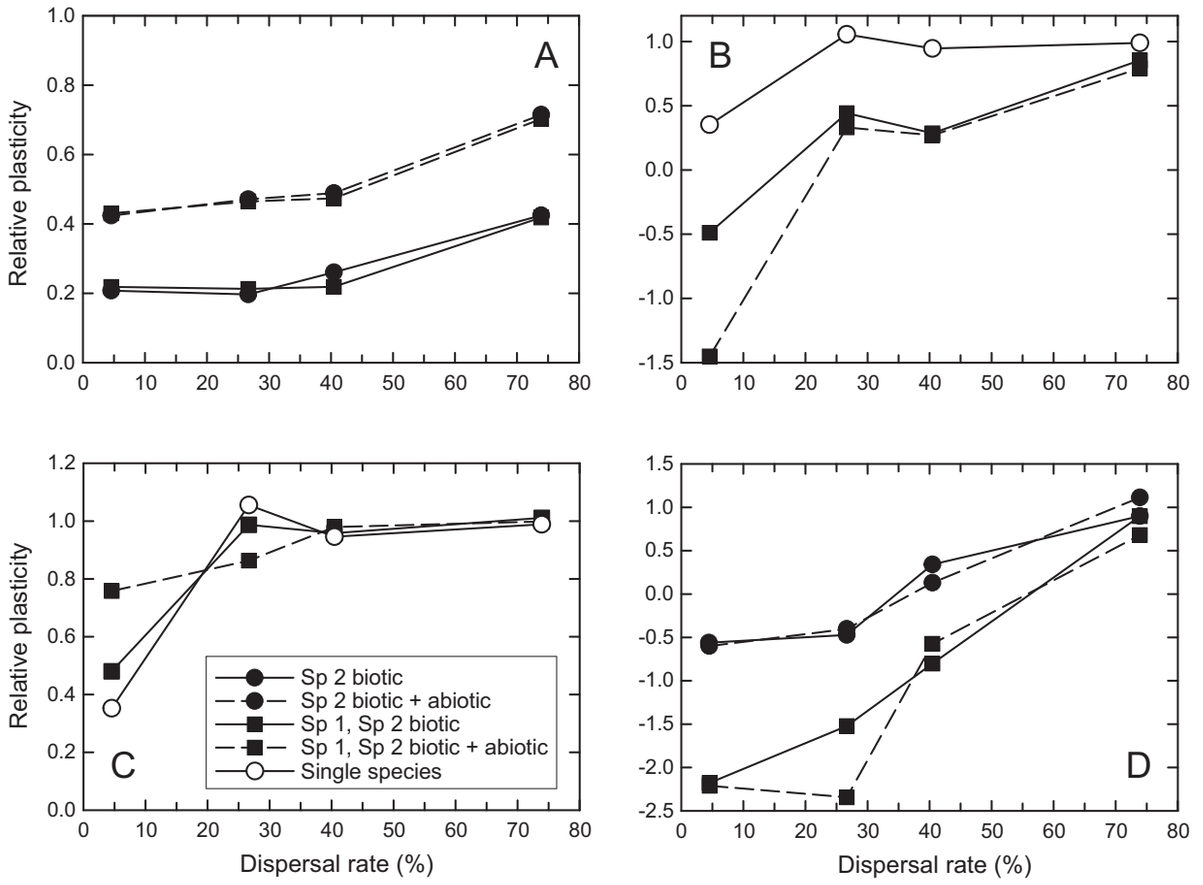

Figure 5: Effect of dispersal rate and pattern of phenotypic determination on trait plasticity (mean P_{ij}). Plasticity is relative to the optimal reaction norm for the abiotic gradient. *A*, *B*, Species 2 = predator. *C*, *D*, Species 2 = prey. *A*, *C*, Plasticity of the predator. *B*, *D*, Plasticity of the prey. Open circles show the effect of dispersal rate on selection for phenotypic plasticity when there is only one species and plasticity is in response to the abiotic environment only.

When the predator was species 2 and responded in a plastic manner to the prey species (with or without abiotic plasticity), the evolution of predator plasticity was independent of the plasticity of the prey species (fig. 5*A*). As before, the predator tended to evolve low levels of phenotypic plasticity, especially when it was responding to just its biotic environment; moreover, dispersal rate had only a modest effect on plasticity. In these scenarios, the prey species (when plastic) responded directly in a plastic manner only to the abiotic environment. At low to intermediate dispersal rates, its plasticity depended on the type of environmental response of the predator species (fig. 5*B*). When the predator species was more plastic (i.e., in response to both the biotic and the abiotic environment), the prey species had a steeply negative reaction norm. Thus, plasticity in a given species can indirectly reflect plasticity in a coevolving species, even if there is no direct effect of the latter on the plastic response of the other.

When the prey was species 2 and responded in a plastic manner to traits of the predator species, a plastic predator increased the plasticity of the prey species (fig. 5*D*), in contrast to the previous scenario. When dispersal rates were low, the prey species had a very steep reaction norm in the direction opposite of that favored by the abiotic gradient. Thus, coevolution combined with plasticity can lead to what looks like countergradient selection. The plasticity of the predator, which depended on just the abiotic environment, was very similar to that of a single species community with no coevolution (fig. 5*C*). Again, the type of plasticity of the prey species (biotic only or biotic and abiotic) had little to no effect of the plasticity of the predator species.

These asymmetric differences in plasticity by trophic level resulted in differences in the expressed phenotypes of the predator and prey species. In figure 6, the slope of the expressed phenotype (slope of the mean T_{ij}) is shown relative to the slope that would match the locally optimal

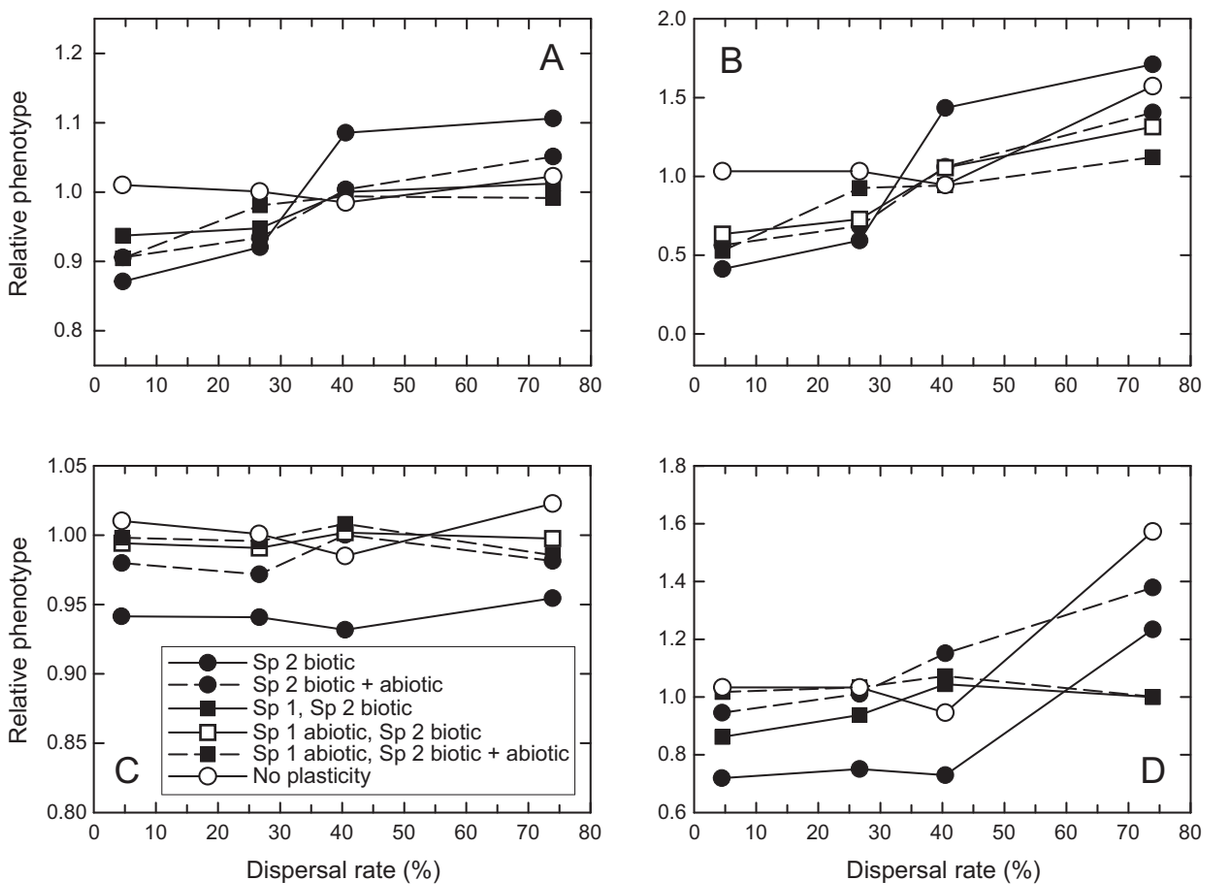

Figure 6: Effect of dispersal rate and pattern of phenotypic determination on the slope of the expressed phenotype (slope of the mean T_{ij} divided by slope of θ_i). *A*, *B*, Species 2 = predator. *C*, *D*, Species 2 = prey. *A*, *C*, Phenotype of the predator. *B*, *D*, Phenotype of the prey. Open circles show the effect of dispersal rate on the expressed phenotype when there is no trait plasticity.

abiotic phenotype in all demes (the value 1 means that the two slopes are the same). When the predator's phenotype depended developmentally on that of the prey, its phenotypes closely tracked the abiotic optima, similar to when plasticity was absent (fig. 6*A*). The slope was shallower than for the local abiotic optimum at low dispersal rates and very close to or steeper than the abiotic optimum at high dispersal rates. The phenotype of the prey species showed an effect of dispersal rate similar to that of the predator species, although the effect magnitude was greater (fig. 6*B*; note difference in scale with fig. 6*A*). These results imply, for example, that sedentary prey species are likely to show phenotypes that appear to be nonadaptive with respect to the abiotic environment when predator traits are plastic and the prey are coevolving with the predator.

When the prey's phenotype depended on that of the predator (i.e., the prey is species 2 in fig. 1), its phenotype had a slope that again was shallower than the optimum at low dispersal rates and steeper than the optimum at high dispersal rates if the predator was not plastic (fig. 6*D*; similar to the previous pattern). The phenotype of the prey species was closest to the abiotic optimum when the predator was also phenotypically plastic. In contrast, for the predator, there was little effect of dispersal rate (fig. 6*C*). The slope was very close to that of the optimum, similar to when plasticity was absent. There thus appears to be an emergent asymmetry by trophic level of the effect of dispersal on plasticity.

These patterns of phenotypic expression affected the coevolutionary pattern as measured by the among-deme phenotypic correlation between the two species (fig. 7). Again, as expected, the correlation was close to 1.0 when the dispersal rate was high and generally declined to moderate values as the dispersal rate decreased. In general, however, this pattern did not depend on the pattern of plasticity. These results imply that in general, assessments of coevolution based on among-deme correlations will not be affected by trait plasticity.

Abiotic and Biotic Selection Opposite. For predator-prey interactions, we also examined the case where augmentation and conflict between abiotic and biotic selection were reversed. For these simulations, both the environmental effector (E_i) and selection (θ_i) were reversed for one species relative to the other species, leading to complex and interestingly different patterns.

In this instance, when the predator species responded in a plastic manner to the prey species (fig. 8*A*), the evolution of its plasticity was independent of the plasticity of the prey species, as with the previous scenario (fig. 5*A*). As before, the predator tended to have low levels of phenotypic plasticity, although now the reaction norms tended to be in the opposite direction of that favored by the

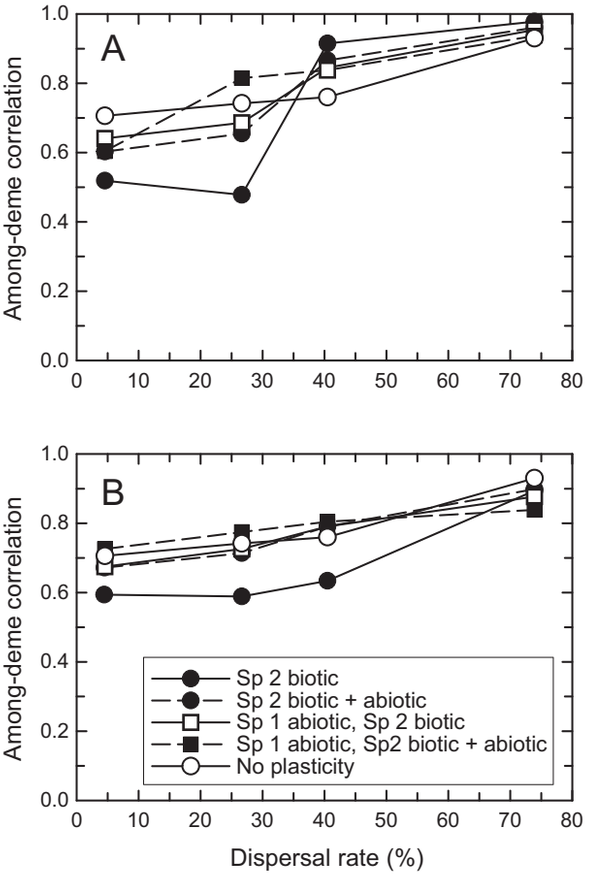

Figure 7: Effect of dispersal rate and pattern of phenotypic determination on the phenotypic correlation between the two species among demes when they interact as predator and prey. *A*, Species 2 = predator. *B*, Species 2 = prey. Open circles show the effect of dispersal rate on the among-deme correlation when there is no trait plasticity.

abiotic environment, especially when the predator was responding to just its biotic environment, or at low dispersal when also responding to the abiotic environment. However, the prey species responded very differently than in the previous scenario (compare fig. 8*B* and fig. 5*B*), with intermediate levels of plasticity at low dispersal rates and low levels of plasticity at high dispersal rates, especially when the predator was responding to just the biotic environment.

When the prey species responded in a plastic manner to the predator species (fig. 8*D*), the plasticity of the predator had little effect on the plasticity of the prey species, in contrast to the previous scenario (fig. 5*D*). When dispersal rates were low, the prey species had an intermediate level of plasticity that usually declined as dispersal rates increased, especially when the prey species was responding to just the biotic environment. The exception to these patterns was when the prey species' phenotype was determined by a

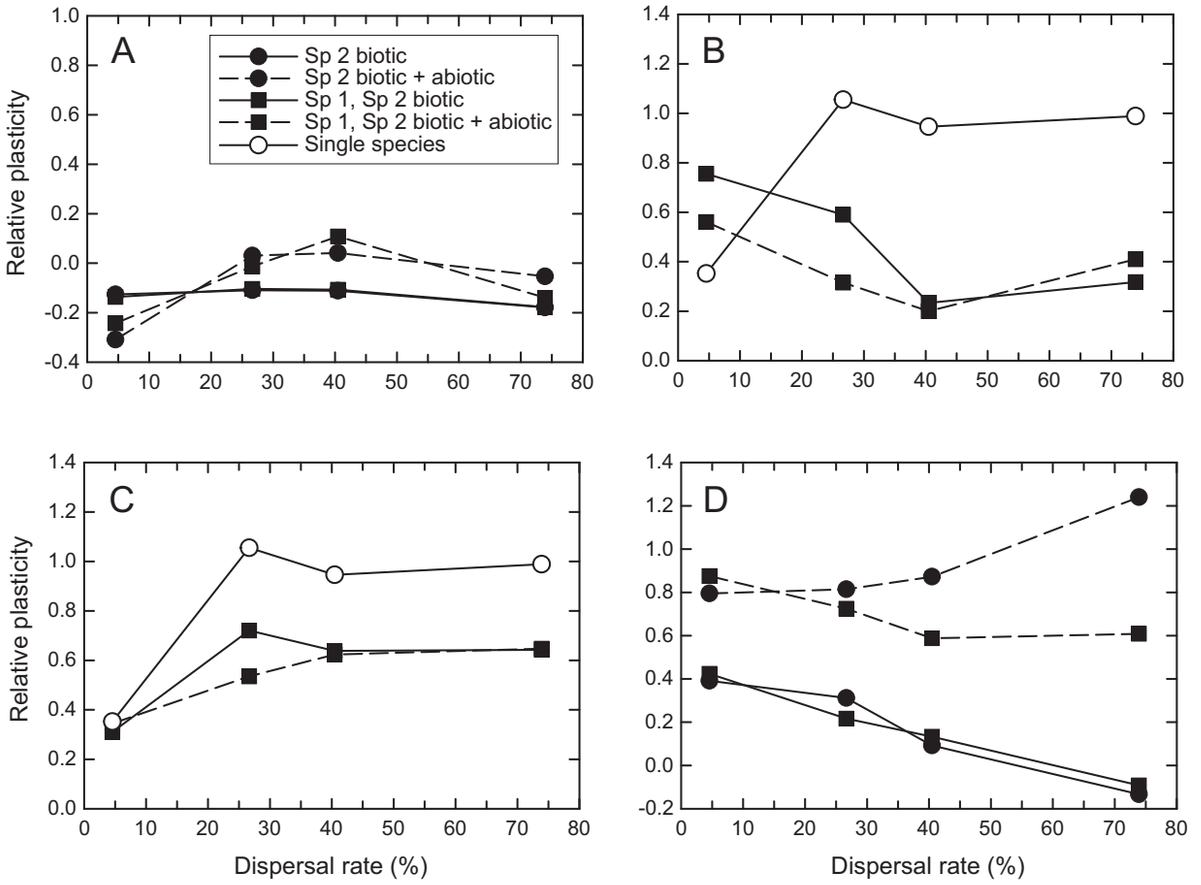

Figure 8: Effect of dispersal rate and pattern of phenotypic determination on trait plasticity (mean P_{ij}) when the abiotic optima have opposite slopes. Plasticity is relative to the optimal reaction norm for the abiotic gradient. *A*, *B*, Species 2 = predator. *C*, *D*, Species 2 = prey. *A*, *C*, Plasticity of the predator. *B*, *D*, Plasticity of the prey. Open circles show the effect of dispersal rate on selection for phenotypic plasticity when there is only one species and plasticity is in response to the abiotic environment only.

nonplastic predator and the abiotic environment; in this case, the prey species' plasticity increased as the dispersal rate increased. The plasticity of the predator (fig. 8*C*), which was a function of just the abiotic environment, was lower than that of a single species in the absence of coevolution, and there was little to no effect of the type of plasticity of the prey species.

The expressed phenotypes of both the predator and prey species were quite different from the previous scenario. For the predator, the slope of the expressed phenotype was approximately 0.7, while that of the prey was approximately 1.6. These values did not depend on the type of plasticity of either species or the dispersal rate. The among-deme correlation ranged from approximately −0.75 to −0.85, increasing in magnitude with the dispersal rate. Again, the correlation did not depend on the type of plasticity of either species. All of these values were identical to those that arose when plasticity was absent. Thus, assessments of coevolution are not affected by discordant biotic and abiotic selection. However, assessment of phenotypes relative to just the abiotic environment will appear to imply that spatial variation in traits are nonadaptive. This suggests that ignoring coevolution can lead to misleading interpretations of how adaptation is playing out across environmental gradients.

Discussion

Overall, coevolution had a larger effect on the evolution of phenotypic plasticity than plasticity had on the outcome of coevolution. Effects on the evolution of plasticity were greater when the fitness-maximizing coevolutionary outcomes were antagonistic between the species pair (predator-prey interactions) than when those outcomes were augmenting (competitive or mutualistic interactions). Overall, biotic interactions reduced selection for plasticity. If this

result holds up in future studies, it may explain why plasticity is less ubiquitous than one might expect. Even when trait development was responding to just the abiotic environment, selection on plasticity, however, could be affected by biotic interactions (figs. 5, 8). In contrast, coevolution as expressed by the among-deme correlation was little affected by whether traits were plastic (figs. 3, 7).

Our most striking result may be the asymmetry in evolutionary responses of predators and prey (fig. 5), despite the fact that the fitness functions were identical except for a reversal of the sign of the interaction. This difference is due to an asymmetry in how fitness changes with phenotype. For the predator, fitness is maximized when its phenotype jointly matches the optimum for the abiotic environment and the phenotype of its prey. Selection is stabilizing for both optima, although those optima may differ. For the prey, again fitness is maximized when its phenotype matches the optimum for the abiotic environment but also when that phenotype is as different as possible from the phenotype of the predator. The latter selection is directional because fitness keeps increasing as the difference grows. The result is strong countergradient selection on plasticity of the prey species, that is, selection for a reaction norm that slopes in the opposite direction expected from the optima favored by the abiotic environment (fig. 5*C*, 5*D*). Because of this countergradient selection, if the plasticity of a wild-collected prey species were measured in a growth chamber, greenhouse, or transplant garden in the absence of the predator, the resulting reaction norm might be interpreted as maladaptive. We reinforce the message of Scheiner (2013) that phenotypic plasticity must always be interpreted in the full context of a species' ecology and selective milieu.

Related Models

The model in this article derives from those of Nuismer et al. (2010) and Scheiner and Holt (2012) but differs in key respects from both. The obvious difference with Scheiner and Holt (2012) is the addition of a biotic interaction for determining plasticity. In addition, in this article, the ecological gradient had smaller differences in the phenotypic optima between adjacent demes, weakening selection due to the abiotic environment. We made this change so that selection by biotic and abiotic factors would be comparable in magnitude. Despite these differences, the general patterns of plasticity evolution in the absence of biotic interactions (fig. 2*B*) were similar to those previously reported. We conclude that the effects on plasticity evolution seen here are specifically the result of our inclusion of biotic interactions.

Nuismer et al. (2010) focused on the question of whether coevolution would produce a signal of an among-deme correlation across species. We found similar among-deme correlations in our simulations (figs. 3, 7; see fig. 5 of Nuismer et al. 2010). The detailed differences with that model are twofold. First, in our model, fitness was determined by the mean phenotype of the other species in a given deme rather than by pairwise interactions of individuals. This difference alters which empirical examples are relevant for our results but does not seem to affect the overall pattern of coevolution. Second, in our model, dispersal was by a Gaussian dispersal kernel rather than by an island pattern. For such kernels, movement occurs among nearby demes, with the probability of movement decreasing with distance. For island migration, movement occurs among all demes with equal probability, a pattern of movement that will tend to erode among-deme correlations. The similarity of our results with those of Nuismer et al. (2010) is likely because the coevolutionary dynamic is driven by the phenotypes of each species rather than by the specific mechanistic bases of those phenotypes, that is, plasticity. We conclude, as did Nuismer et al. (2010), that the existence of an among-deme correlation is on its own weak evidence for the presence of a coevolutionary interaction. Our results suggest that whether such correlations occur may not be strongly influenced by the presence of plasticity in trait development.

Our simulations considered only the simplest case of spatial variation in the abiotic environment—a linear gradient—coupled with a Gaussian dispersal kernel. We did not consider the effects of temporal variation combined with spatial variation, alternative patterns of dispersal (e.g., island or non-Gaussian), or different life-history patterns (movement before selection). On the basis of Scheiner (2013), which considered both spatial and temporal variation for the single-species case, the resulting evolutionary outcomes could be quite complex. Although our equilibrium results were similar to those of Nuismer et al. (2010), we moreover did not examine the dynamics of coevolution with our model. On the basis of Nuismer et al. (1999, 2000), that dynamic can also be quite complex, especially if fitness interactions vary both spatially and temporally (Gomulkiewicz et al. 2000, 2003). This is likely if, for instance, instead of soft selection there is hard selection, with the population size of each species responding to the interaction with the other species. Exploration of these additional factors as well as temporal variation and alternative fitness functions (e.g., a phenotypic differences mechanism of species interactions; Nuismer et al. 2010) may reveal additional insights about plasticity evolution in a coevolutionary context and permit further scrutiny of our conclusion that plasticity may not play that large a role in coevolution. In this article, we explored only the effects of soft selection; local population size was determined independently of the outcome of selection. Hard selection—population size is density dependent—can have important effects on the transient dynamics of systems (e.g., fig. 6 of Gomulkiewicz and Kirkpatrick 1992), es-

pecially on the propensity for extinction. However, it has weaker effects on long-term evolutionary equilibria, unless there are genetic trade-offs (e.g., fig. 5 of Gomulkiewicz and Kirkpatrick 1992). We expect that the general patterns found here would be unchanged with hard selection; proving that assertion awaits future explorations.

Testing Our Model

Testing models of plasticity evolution empirically is difficult because measuring plasticity involves raising genetically related individuals in multiple environments; moreover, comparisons often require finding metapopulations or species with different patterns of environmental heterogeneity. One way around the second difficulty is to compare the plasticities of different traits of a single organism. Our model predicts overall greater plasticity in response to abiotic cues than to biotic cues. It also predicts different amounts of plasticity, depending on the type of species interaction. Nearly all species experience a variety of types of interspecific interactions. For example, an herbivore or predator can also be a prey species, or competing species can be prey for another species (as in keystone predation), or a mutualist can also be a predator. In the last case, for instance, we predict that traits that enhance the mutualistic interaction should be more plastic than traits that enhance the predatory interaction. This prediction holds whether the plasticity is in response to either the biotic or the abiotic environment, as long as the traits are not genetically or developmentally correlated. A full analysis of this prediction requires a model that explicitly incorporates such correlations. A comparison of trait plasticities within a single species based on responses to an abiotic environmental signal has the virtue of using a single environmental effector and traits that have been subject to a single evolutionary history. Comparisons can also be made using different biotic effectors, although it becomes necessary to standardize environmental differences in some fashion. As far as we know, no one has compared trait plasticities in this way; such data, however, may well exist.

Murren et al. (2014) examined the extent to which plasticity of a given trait varied among populations or closely related species, which provides a possible indication of selection on plasticity. Among types of environmental effectors, they found that biotic factors tended to result in greater plasticity differences than did abiotic factors. Most of those measures of biotic interactions were of traits of prey species (C. Murren, personal communication), consistent with our predictions that prey species will vary the most in their amount of plasticity (fig. 2).

We caution that our results provide only preliminary theoretical expectations about the interplay of interspecific interactions and plasticity and are not definitive. One challenge in developing tests of our theoretical predictions is that in natural communities, pairwise interactions are often embedded in richer communities, so a multispecies coevolutionary perspective may be needed (Ridenhour and Nuismer 2012). A given trait may influence how a species interacts with not just one but a whole suite of other species, and developmental cues might come from more than just one other species as well. Moreover, different functional forms for expressing the relationships among traits, interspecific interactions, and fitness than assumed above (eq. [3b]) might lead to different insights, as would our assumption about soft selection. These all would be important directions for future theoretical studies.

Conclusions

Both coevolution and plasticity evolution have extensive empirical and theoretical literatures. Despite this rich history, there been few attempts to directly test any of those theories and to assess how the two arenas affect each other. Our results provide encouragement for those attempting such tests. For tests of coevolution theory, our results suggest that researchers need not be unduly worried about the genetic bases of the traits involved in the species' interaction. Whether those traits are plastic or fixed, a similar evolutionary outcome is predicted. In addition, we found similar results to those of Nuismer and collaborators, despite major differences in model assumptions and structure, suggesting that these theoretical predictions are robust. Examining the evolution of trait plasticity may provide another—albeit indirect—indicator of coevolution, especially if such traits can be examined for both interacting species. For tests of plasticity evolution theory, our results point to a mode of testing by comparing different traits within the same individual. This across-trait mode of analysis may be more approachable than are comparisons among metapopulations or species. However, our results also lend caution to interpretations of plasticity patterns when those plasticity measurements are done outside the ecological context of selection. For both sets of theories, our results show how the merging of two theory domains can deepen our understanding of both. Empirical research is needed that will engage with plasticity evolution in a coevolutionary context.

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