

GENETIC CORRELATIONS AND THE COEVOLUTIONARY DYNAMICS OF THREE-SPECIES SYSTEMS

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Abstract.—The majority of species interact with at least several others. We develop simple genetic models of coevolution between three species where interactions are mediated by quantitative traits. We assume that one of the species has two quantitative traits, each of which governs its interaction with one of the other two species. We use this model to explore how genetic correlations between the two traits in the multivariate species shape the evolutionary dynamics and outcomes of three species interactions. Our results suggest that genetic correlations are most important when at least one of the interactions is between a predator and prey or parasite and host. In these cases, genetic correlations between traits lead to a wide variety of novel coevolutionary outcomes and dynamics. In particular, genetic correlations can affect the existence and stability of coevolutionary equilibrium points, and they can lead to recurrent or permanent maladaptation. When the three species interact only as competitors or mutualists, however, genetic correlations have no effect on the outcome of coevolution. In all cases, our results reveal the surprising conclusion that both positive and negative genetic correlations between traits have qualitatively identical effects on coevolutionary dynamics.

Key words.—Coevolution, genetic correlation, multispecific coevolution, pleiotropy, specialization.

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The vast majority of species interact with a multitude of others, whether as predators, prey, competitors, or mutualists. To take a familiar example, the human population interacts with hundreds of different species of parasites alone (Kiple 1993). Plants, too, face an incredible diversity of interactions. Tetraploid populations of the perennial plant *Heuchera grossulariifolia*, for instance, interact mutualistically with several pollinator species (Segraves and Thompson 1999), are parasitized by four species of phytophagous insects (Thompson et al. 1997; Nuismer and Thompson 2001), are regularly infected by a rust fungus, and face chronic competition from their diploid progenitors. As Darwin emphasized in his metaphor of the tangled bank, these diverse interactions are not only a source of natural selection, but also an inevitable consequence of its action (Darwin 1859; see also Thompson 1994).

Despite the abundance of empirical evidence suggesting that most species interact and coevolve with at least several others, the majority of coevolutionary models have focused only on pairwise interactions involving a single trait in each of the two interacting species. In fact, we know of only one model that has explicitly analyzed the dynamics of multispecific coevolution, demonstrating several novel and unexpected dynamical consequences of adding a third species (Gomulkiewicz et al. 2003). As is the case for many models of coevolution, however, this model assumed that interactions between species are mediated by only a single trait or genetic locus (e.g., Seger 1988; Gavrillets and Hastings 1998; Nuismer et al. 1999; Case and Taper 2000). In reality, though, most species engage in multiple interactions, each of which may be mediated by different traits or sets of loci (Rausher 1996; Brodie and Brodie 1999; Benkman et al. 2001; Dupas et al. 2003). More generally, coevolution between multiple species is likely to be mediated by suites of genetically correlated characters. These empirical observations suggest that multispecific coevolution may frequently be influenced by

the correlation structure of traits involved in different interactions.

Although no general theory has been developed for the effects of genetic correlations on coevolutionary dynamics, a well-developed body of theory exists for single species (e.g., Lande 1979, 1982; Lande and Arnold 1983; Slatkin 1984; Charlesworth 1993). This work has shown that if the adaptive landscape is smooth, genetic correlations between traits do not prevent a population from evolving to the global fitness optimum (Lande 1979). Genetic correlations can, however, slow the approach to this optimum by altering the trajectory of evolution (Lande 1979). Thus, in simple single-species models, the influence of genetic correlations is generally quantitative rather than qualitative. In contrast, more complicated models that incorporate frequency dependence (e.g., sexual selection), demonstrate that genetic correlations can lead to qualitative shifts in evolutionary dynamics (e.g., Lande 1981). It is this latter result that suggests genetic correlations may play a central role in shaping the dynamics and outcome of coevolution.

Here we make a first attempt to address these issues by developing models of multispecific coevolution that explicitly incorporate genetic correlations. Our general approach is to analyze two different types of models, each based upon quite different biological assumptions. The first approach we take is to extend single species models for the evolution of correlated traits (Lande 1979; Charlesworth 1993) to coevolution between multiple species. The second uses individual-based numerical simulations that explicitly incorporate multilocus genetics (e.g., Dieckmann and Doebeli 1999; Doebeli and Dieckmann 2000). The former approach uses deterministic equations with fixed genetic variances and covariances, whereas the latter approach uses stochastic simulations in which genetic variances and covariances can evolve. However, the stochastic models should not be viewed as a test of the deterministic models (or vice versa). Rather, the two ap-

proaches represent different biological assumptions and should be viewed as complementary. It is our hope that this two pronged modeling approach will yield results of greater generality than could be achieved by either approach in isolation.

We use these models to investigate the coevolutionary dynamics of a breadth of ecological interactions; in fact we consider simple models of all possible combinations of competitive, mutualistic, and antagonistic interactions between three species. The basic structure of our models is that there is one species—the multivariate or focal species—in which two traits are evolving that each determine coevolutionary interactions with a single trait in another species. Thus, there are two pairs of coevolving traits, and the evolutionary dynamics of these two trait pairs may be linked by a genetic correlation between the two traits in the focal species. The interaction in each of the two trait pairs may be competitive, mutualistic, or antagonistic, and for all the resulting ecological scenarios we test the effects of genetic correlation against the baseline case in which the two traits pairs coevolve independently of each other. Our overarching goal is to identify ecological constellations for which varying the degree of genetic correlations between the two traits in the focal species leads to qualitative changes in the coevolutionary dynamics of the three species system. More specifically, we address the following questions: What qualitative changes in the coevolutionary dynamics of multispecies interactions can be generated by genetic correlations? What are the ecological scenarios in which genetic correlations have the greatest effects? Can genetic correlations generate constraints that lead to maladaptation in one or both of the species, thus potentially fostering the evolution of ecological specialization?

THE GENERAL MODEL

We assume a single, closed community composed of three interacting species labeled X, Y, and Z. The interactions between these species are mediated by quantitative traits. We further assume that species X (the focal or multivariate species) interacts with both species Y and Z, while species Y and species Z interact only with species X and not each other. Thus, our model is capable of describing all simple ecological interactions between three species. Our model is not, however, capable of considering more complex interactions such as two parasites that coevolve with a shared host and with each other due to resource competition. Given the exploratory nature of our models, this would not seem to be a serious restriction, and the exploration of more complicated scenarios is left for future work.

Interactions between species are assumed to be mediated by the degree of phenotypic matching between quantitative traits (Dieckmann et al. 1995; Doebeli 1997; Gavrillets 1997). Specifically, we assume that the interaction between species X and Y depends upon the phenotypic distance between quantitative traits x_1 and y . Similarly, the interaction between species X and Z is assumed to depend upon the phenotypic distance between quantitative traits x_2 and z . Assuming a simple exponential relationship between fitness and phenotypic distance, and that the fitness of species X is determined multiplicatively by its interactions with Y and Z, the fitness

of individuals with the phenotypes (x_1, x_2) , y , and z , respectively can be written as:

$$W(x_1, x_2) = \left\{ \int \exp[-\alpha_{xy}(x_1 - y)^2] \phi_y dy \right\} \times \left\{ \int \exp[-\alpha_{xz}(x_2 - z)^2] \phi_z dz \right\}, \quad (1a)$$

$$W(y) = \int \exp[-\alpha_{yx}(x_1 - y)^2] \phi_{x_1} dx_1, \quad \text{and} \quad (1b)$$

$$W(z) = \int \exp[-\alpha_{zx}(x_2 - z)^2] \phi_{x_2} dx_2, \quad (1c)$$

where ϕ_i is the frequency distribution of trait i , and α_{ij} determines how the fitness of species i is effected by interactions with species j . If α_{ij} is positive, individuals of species i have increased fitness when encountering species j individuals of similar phenotype. In contrast, if α_{ij} is negative, individuals of species i benefit from interacting with species j individuals of dissimilar phenotypes. Any pairwise interaction between two traits is qualitatively described by the signs of the two corresponding α -values. When describing the various possible types of sign combinations, we adhere to the following terminology. When both α -values in an interaction pair are negative, we call it a competitive interaction; in this case, the traits in the given pair have a tendency to diverge. When both α -values in an interaction pair are positive, we call it a mutualistic interaction; in this case, the traits in the given pair have a tendency to match. Finally, when one α -value is positive and the other is negative, we call this interaction antagonistic; in this case, the species with the positive α -value, the predator (or parasite), tries to match the species with the negative α -value, the prey (host), which in turn tries to escape.

We take two different approaches to modeling this general setup. In the first, which we term the “fixed genetic variance” model, we make several assumptions that make the model analytically tractable. Specifically, we assume that additive genetic variances and covariances are constant and that phenotypes follow normal or multivariate normal distributions. These assumptions imply that selection must be relatively weak. In the second approach, which we term the “explicit multilocus” model, we use numerical simulations to analyze an individual-based and genetically explicit model incorporating the fitness assumptions (1a–c). In this model, trait values can only vary within a finite trait interval, and genetic variances and covariances can evolve. As a consequence, the individual-based model can generate types of evolutionary dynamics that cannot be observed in the fixed genetic variance model. It is our hope that this two-pronged approach to analysis brackets the range of possibilities found in natural systems. In addition, we anticipate that this approach will highlight differences in coevolutionary dynamics generated by systems adhering more to one or the other set of assumptions. In the sections that follow, we describe the detailed assumptions and results from each of these approaches.

FIXED GENETIC VARIANCE MODEL

In this section we make several assumptions that facilitate a mathematical analysis of the model. Specifically, we as-

sume that traits y and z are normally distributed, with fixed additive genetic variances G_y and G_z . Similarly, the joint distribution of x_1 and x_2 is assumed to be multivariate normal with fixed additive genetic variances G_{x_1} and G_{x_2} and covariance G_{Cov} . All traits are assumed to be perfectly heritable.

For the following, we make the basic assumption that selection is weak. More precisely, we assume that the fitness functions (1a–c) are broad relative to the width of the phenotypic distributions, and that the differences between mean phenotypes of the interacting species are small. Under these assumptions, we can approximate the exponential expression in the fitness functions by a quadratic term of the form $1 - \alpha(\mu - \nu)^2$, where α stands for the various interaction coefficients, and (μ, ν) stand for the possible interacting trait pairs. Integrating these quadratic terms over the relevant phenotypic distributions, ϕ , yields expressions involving only the mean and variance of these distributions as follows:

$$W(x_1, x_2) \approx 1 - \alpha_{xy}[(x_1 - \bar{y})^2 + G_y] - \alpha_{xz}[(x_2 - \bar{z})^2 + G_z], \quad (2a)$$

$$W(y) \approx 1 - \alpha_{yx}[(\bar{x}_1 - y)^2 + G_{x_1}], \quad \text{and} \quad (2b)$$

$$W(z) \approx 1 - \alpha_{zx}[(\bar{x}_2 - z)^2 + G_{x_2}]. \quad (2c)$$

(Note that we have neglected fourth-order and higher terms in eq. 2a).

Selection gradients for the various traits can be obtained by taking partial derivatives of the logarithm of mean fitness with respect to the trait means (Lande 1979). The mean fitness is obtained by integrating expressions (2a–c) over the relevant phenotypic distributions. Noting that

$$\beta_\mu = \frac{\partial \ln \bar{W}}{\partial \bar{\mu}} = \frac{1}{\bar{W}} \frac{\partial \bar{W}}{\partial \bar{\mu}}$$

for any trait mean $\bar{\mu}$, and that $1/\bar{W} \approx 1$ because of our assumption of weak selection, we obtain the following equations for the change in population mean phenotypes over one generation:

$$\Delta \bar{x}_1 = 2G_{x_1} \alpha_{xy}(\bar{y} - \bar{x}_1) + 2G_{Cov} \alpha_{xz}(\bar{z} - \bar{x}_2), \quad (3a)$$

$$\Delta \bar{x}_2 = 2G_{x_2} \alpha_{xz}(\bar{z} - \bar{x}_2) + 2G_{Cov} \alpha_{xy}(\bar{y} - \bar{x}_1), \quad (3b)$$

$$\Delta \bar{y} = 2G_y \alpha_{yx}(\bar{y} - \bar{x}_1), \quad \text{and} \quad (3c)$$

$$\Delta \bar{z} = 2G_z \alpha_{zx}(\bar{z} - \bar{x}_2). \quad (3d)$$

Similar derivations for two coevolving species and for the evolution of correlated traits in single species can be found in Gavrillets (1997) and Lande (1979), respectively.

Analysis of equations (3a–d) can be simplified in two ways. First, introducing the new variables $D_{xy} = \bar{x}_1 - \bar{y}$ and $D_{xz} = \bar{x}_2 - \bar{z}$ allows equations (3a–d) to be reduced to two equations describing the phenotypic distance between the mean phenotypes of the interacting species. In addition to simplifying the analysis, this change of variables focuses attention on the biologically critical difference between mean trait values. It is these differences in mean trait values that largely determine fitness, and hence coevolutionary dynamics. Second, if the rate of evolution is relatively slow, the difference equations (3a–d) can be approximated by their analogous differential

equations. Because we have already assumed weak selection, this does not require any additional assumptions. The dynamics of the coevolutionary system are then described by the following differential equations:

$$\frac{dD_{xy}}{dt} = -2[\alpha_{xz} G_{Cov} D_{xz} + (\alpha_{xy} G_{x_1} + \alpha_{yx} G_y) D_{xy}] \quad \text{and} \quad (4a)$$

$$\frac{dD_{xz}}{dt} = -2[\alpha_{xy} G_{Cov} D_{xy} + (\alpha_{xz} G_{x_2} + \alpha_{zx} G_z) D_{xz}]. \quad (4b)$$

In the absence of covariance between the two traits x_1 and x_2 in species X ($G_{Cov} = 0$), this model reduces to the case of pairwise coevolution considered by Gavrillets (1997). This confirms that pairwise models of coevolution can be directly extended to multispecific coevolution whenever traits are uncorrelated and fitnesses are multiplicative (Hougenieztman and Rausher 1994; Rausher 1996).

Analysis of the coevolutionary model described by (4a,b) shows that genetic covariance does not affect the equilibrium solution. The model has a single biologically relevant equilibrium that occurs when the phenotypic distance between the trait means \bar{x}_1 and \bar{y} and \bar{x}_2 and \bar{z} is zero ($D_{xy} = 0$, $D_{xz} = 0$), irrespective of the value of G_{Cov} . Biologically, this equilibrium corresponds to a case where interacting species match phenotypes, resulting in maximal fitness for species benefiting from phenotypic similarity (parasite, predator, mutualist) but minimal fitness for species harmed by phenotypic similarity (host, prey, competitor). This equilibrium is simply the three species equivalent of that found in the model of pairwise coevolution analyzed by Gavrillets (1997). As a consequence, it seems that genetic covariance plays no role in shaping the equilibrium outcome of coevolution between multiple species. This result closely mirrors the classical result from single-species theory: genetic correlations generally change the trajectory of approach to equilibrium, but not the equilibrium value itself (Lande 1979). However, we will see that this particular result is model dependent and does not hold for the individual-based models introduced later.

To further explore how genetic correlations shape the coevolutionary dynamics of multispecific interactions, we analyzed the stability of the matching equilibrium ($D_{xy} = 0$, $D_{xz} = 0$). This is done following the usual procedure of generating the Jacobian matrix of partial derivatives of the right sides of equations (4a,b) with respect to D_{xy} and D_{xz} , and then evaluating this Jacobian matrix at the equilibrium. The matching equilibrium is stable if both eigenvalues of the Jacobian have negative real parts (e.g., Bulmer 1994, pp. 307–312). In contrast to the equilibrium analysis, this analysis shows that the stability of the coevolutionary system can be altered by the genetic correlation. Specifically, stability analysis shows that the matching equilibrium will be stable whenever the eigenvalue

$$E_L = \frac{1}{2}[-R_{x_1} - R_{x_2} - R_y - R_z + \sqrt{4\rho^2 R_{x_1} R_{x_2} + (R_{x_1} - R_{x_2} + R_y - R_z)^2}] \quad (5)$$

has real part less than zero, where ρ is the genetic correlation between traits x_1 and x_2 , and $R_{x_1} = \alpha_{xy} G_{x_1}$, $R_{x_2} = \alpha_{xz} G_{x_2}$, $R_y = \alpha_{yx} G_y$, and $R_z = \alpha_{zx} G_z$. These R -values measure the speed

of evolutionary change, with positive R -values indicating selection for matching (i.e., mutualism, parasite) and negative R -values indicating selection for mismatching (i.e., competition, host). Whenever the real part of the eigenvalue (5) is positive, the matching equilibrium is unstable, and hence the phenotypic distance between trait means in at least one of the trait pairs increases without bound; for predator-prey interactions, this would indicate that the prey species is winning the coevolutionary race.

Insight into the factors determining the coevolutionary stability of three species systems can be gained by further examining expression (5), yielding four general results. First, since the genetic correlation, ρ , appears only as a square, both positive and negative correlations have identical effects on the stability properties of the matching equilibrium. Second, for the genetic correlation between traits x_1 and x_2 to affect stability, the following conditions must hold:

$$R_{x_1} + R_{x_2} + R_y + R_z > 0 \quad \text{and} \quad (6a)$$

$$\left| \frac{\sqrt{(R_{x_1} + R_y)(R_{x_2} + R_z)}}{\sqrt{R_{x_1}}\sqrt{R_{x_2}}} \right| \leq 1 \quad (6b)$$

Condition (6a) is needed to ensure that expression (5) can in principle be negative, and condition (6b) is needed to ensure that the left side of (5) changes sign as ρ^2 is varied between zero and one. Third, if conditions (6a,b) are satisfied and the genetic correlation, ρ , affects stability, the effect will be to increase stability if $R_{x_1}R_{x_2} < 0$ but to decrease stability if $R_{x_1}R_{x_2} > 0$. Thus, the qualitative effect of increasing ρ^2 on the stability of the matching equilibrium depends on whether selection has the same direction (either for matching or for mismatching) in the two traits of the multivariate species. Fourth, if $R_{x_1}R_{x_2} < 0$, then increasing the genetic correlation between traits x_1 and x_2 can cause the matching equilibrium to be a focus (as the square root in expression 5 becomes imaginary), so that away from the equilibrium the system will exhibit coevolutionary cycles (of either decreasing or increasing magnitude, depending on whether the focus is stable). Note that cyclic dynamics are not possible in an identical two species model (Gavrilets 1997). The results from the stability analysis of model (4a,b) are summarized schematically in Figure 1.

Using the results summarized in the preceding paragraph, it is possible to categorize various ecological interactions according to the influence of genetic correlation on the coevolutionary outcome. These results are summarized in Figure 2 and reveal a general conclusion: when interactions are either all mutualistic or all competitive, genetic correlations in the focal species have no qualitative effect on the coevolutionary dynamics. Irrespective of genetic correlations these interactions eventually coevolve to the state that is predicted by coevolution in the component interactions. Thus, in three-way mutualisms, both trait pairs will always match up, and in three-way competitive interactions both trait pairs will always maximally mismatch, irrespective of the amount of genetic correlation between traits in the focal species. This result is similar to the finding for the single-species models of Lande (1979), in which the evolutionary equilibrium does

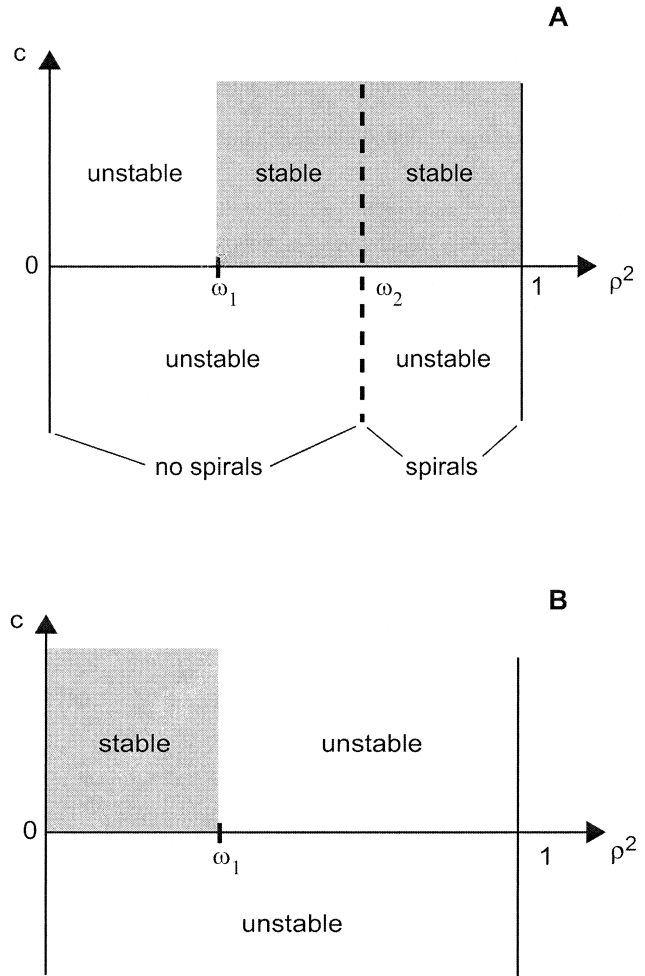


FIG. 1. Schematic illustration of the effect of the genetic correlation on the stability of the matching equilibrium $D_{xy} = D_{xz} = 0$ in model (4). The panels indicate stability depending on the two parameters $c = R_{x_1} + R_{x_2} + R_y + R_z$ and ρ^2 in the two cases $R_{x_1}R_{x_2} < 0$ (A) and $R_{x_1}R_{x_2} > 0$ (B). In both cases, the matching equilibrium is unstable if $c < 0$. If $c > 0$ in A, the value of ρ^2 at which the matching equilibrium becomes unstable is given by $\omega_1 = [(R_{x_1} + R_{x_2} + R_y + R_z)^2 - (R_{x_1} - R_{x_2} + R_y - R_z)^2] / (4R_{x_1}R_{x_2})$ (note that this value may not lie in the interval $[0, 1]$, in which case genetic correlation cannot affect the stability of the matching equilibrium). In addition, if ρ^2 is increased above $\omega_2 = [-(R_{x_1} - R_{x_2} + R_y - R_z)^2] / (4R_{x_1}R_{x_2}) > \omega_1$ the analytical model exhibits oscillatory dynamics (independent of whether the matching equilibrium is stable). If $c > 0$ in B, the value of ρ^2 at which the matching equilibrium becomes stable is again given by ω_1 . The fact that stability only depends on ρ^2 implies that stability does not depend on the sign of the genetic correlation in model (4).

not depend on the amount of genetic correlation between the evolving traits.

However, due to the dynamic nature of coevolution, the ultimate outcome of other three-way interactions may be profoundly altered by correlations between the two traits in the focal species. Because the number of ecological interactions contained in Figure 2 is so large, an exhaustive summary of each type of interaction is impractical. Instead, we present a few salient examples from the subset of interactions offering the richest spectrum of biologically important results.

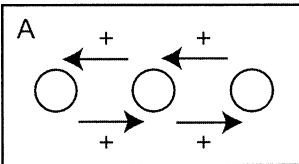
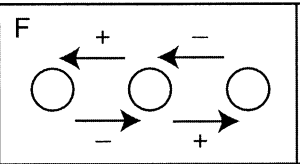
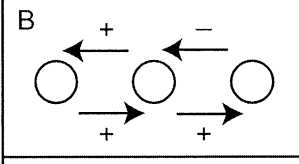
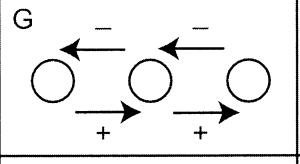
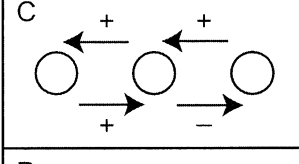
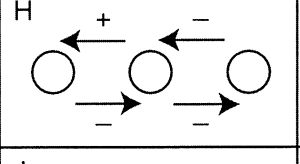
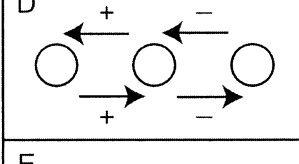
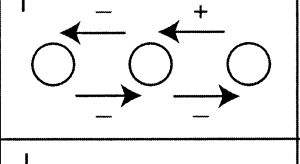
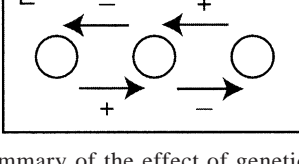
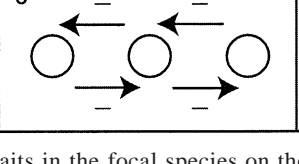
Interaction	Effect of correlation	Interaction	Effect of correlation
A 	ρ^2 has no effect	F 	Increasing ρ^2 decreases stability
B 	Increasing ρ^2 increases stability	G 	increasing ρ^2 increases stability
C 	Increasing ρ^2 decreases stability	H 	ρ^2 has no effect
D 	ρ^2 has no effect	I 	Increasing ρ^2 increases stability
E 	Increasing ρ^2 decreases stability	J 	ρ^2 has no effect

FIG. 2. A summary of the effect of genetic correlations between traits in the focal species on the stability of the matching equilibrium in the deterministic model. Genetic correlations alter the stability of the matching equilibrium if conditions (6) of the text are satisfied. If $R_{x_1}R_{x_2} < 0$ genetic correlations increase stability, whereas if $R_{x_1}R_{x_2} > 0$, genetic correlations decrease stability. The three species are represented by circles, with the focal species in the center. Arrows connecting species labeled with a plus sign indicate that the species toward which the arrow points benefits from having matching trait values. Arrows labeled with a minus sign indicate that the species toward which the arrow points is harmed by matching trait values.

Case 1: mutualism-antagonism

We first consider the two interactions that couple parasitism and mutualism (Fig. 2B,C). Take as the first example the case where the focal species X acts as an antagonist to species Z, but as a mutualist with species Y. Biologically, this case would correspond to an insect that pollinates species Y but simply robs nectar from species Z. In the absence of genetic correlation between traits x_1 and x_2 , species Y and X would coevolve matching phenotypes and be maximally coadapted ($D_{xy} = 0$). In contrast, the outcome of the interaction between X and Z is not so clear cut. If $-R_z > R_{x_2}$, species Z wins the coevolutionary race and escapes from parasitism by X. But if $-R_z < R_{x_2}$, species X wins the coevolutionary race and evolves to maximally parasitize Z. Thus, the species with the more rapid response to selection will win the coevolutionary race (Gavrilets 1997).

If a genetic correlation exists between traits x_1 and x_2 , however, species X is faced with a trade-off: either pursue matching with species Y or matching with species Z. Stability condition (5) shows that the greater the genetic correlation, either positive or negative, the more likely it is that the matching equilibrium is unstable. This could correspond to two

different biological scenarios. In one case, the mutualism between X and Y could dissolve as phenotypic matching between these species is disrupted by species X tracking the rapidly changing phenotype of its host species Y. In the other case, species Z could escape parasitism by species X as correlated selection for matching the mutualist species Y prevents species X from successfully tracking the phenotype of its host. Either way, species X is effectively forced into specializing as a parasite of species Z or as a mutualist of species Y. If the traits in species X are only weakly correlated, however, species X could remain a generalist and capitalize on resources provided by both species Y and Z.

Now consider the similar case where multivariate species X acts as a mutualist of species Y, but instead of parasitizing species Z, species X is now parasitized by Z. This is much like the interactions between *Lithophragma parviflorum* and its associated floral pollinators and parasites (Thompson and Pellmyr 1992). In contrast to the previous case, stability condition (5) shows that increasing genetic correlations now increase the stability of the matching equilibrium. Once again the reason is that genetic correlation poses a trade-off for species X: either escape from parasite Z or capitalize on interactions with mutualist Y.

Case 2: antagonism-antagonism

The second class of interactions we consider in detail occurs when two host-parasite interactions are coupled (Fig. 2E,F). We first consider a scenario where the focal species X parasitizes or preys upon both species Y and Z. Under this ecological scenario condition (5) shows that increasing the magnitude of the genetic correlation between traits x_1 and x_2 destabilizes the matching equilibrium. Once again the reason is that species X is confronted by the familiar trade-off: either pursue species Y or species Z. For sufficiently large genetic correlations, either species Y or Z escapes evolutionarily and X is forced into specializing on a single prey or host.

The second scenario we consider occurs when the focal species X is parasitized or preyed upon by both species Y and Z. Once again, condition (5) shows that an increasing magnitude of genetic correlation destabilizes the matching equilibrium. In this case, however, the reason has nothing to do with a trade-off. Instead, the counterintuitive result is that both positive and negative genetic correlations facilitate the evolutionary escape of species X from species Y and Z by effectively magnifying the strength of selection on species X. Instead of leading to a trade-off, the genetic correlation actually magnifies the strength of selection on species X, and hence the incentive for species X to escape. This prevents both species Y and Z from evolving to be effective parasites or predators, hence the counterintuitive result that both positive and negative genetic correlations facilitate the evolutionary escape of species X from species Y and Z.

Case 3: competition-antagonism

The final scenario that we consider in detail occurs when the focal species X is a competitor in one interaction and a predator in the other (Figure 2I). In this case, condition (5) predicts that increasing the genetic correlation between traits in the focal species should increase the stability of the matching equilibrium of the whole system if the antagonistic interaction has a stable equilibrium in isolation (i.e., if the predator can evolve faster than the prey). Biologically, this means that those traits mediating the competitive interaction can, under some conditions, be prevented from diverging by the genetic correlation between traits. The consequence is that substantial levels of maladaptation are maintained as both species are forced to compete intensely in perpetuity. This is again a counterintuitive result, for one would expect that the competitive traits should always be able to diverge, irrespective of the correlation to the antagonistic trait pair. After all, for the matching antagonistic pair it does not matter where in the trait interval the traits match up. As a consequence, the predator trait should be able to both match the prey trait and diverge from the competitor simultaneously. Nevertheless, the analytical result that strong antagonistic interactions can induce matching in the competitive traits holds true. We will provide an intuitive explanation for this result in the next section, in which we use individual-based multilocus models to investigate coevolutionary dynamics in more detail.

EXPLICIT MULTILOCUS MODEL

Because we are not only interested in the local stability properties of matching equilibria and in how correlation

might affect such properties, but more generally in the unfolding of the whole coevolutionary dynamics away from equilibrium, we now turn to genetically explicit individual-based simulations. These simulations make assumptions that are generally compatible with those of the analytical model. For instance, we assume free recombination, additive polygenic traits, random mating, fixed population sizes, and coevolutionary selection governed by equations (1a–c). However, these simulations differ in that they explicitly allow genetic variances and covariances to evolve, as would be the case under strong selection. Because selection may be particularly strong in species interactions (e.g., Thompson 1998, 1999), this represents an important perspective.

Genetic correlations are incorporated into the simulations by assuming that a subset of the loci in the focal species X act in a pleiotropic fashion (Fig. 3). As a consequence, genetic correlations between traits can be maintained in the absence of disequilibria. Because the fitness of the focal species X is determined multiplicatively, the degree of pleiotropy between traits largely determines the genetic correlation. This allows us to couch the results of what follows in terms of the genetic correlation rather than in the more strictly accurate terms of pleiotropy. We feel that this use of shorthand is justified in this case because it greatly facilitates comparison with results from the analytical model.

We assume that the phenotypes of the two single-trait species Y and Z are determined by the additive action of n_y and n_z diploid diallelic loci, respectively. In contrast, the phenotypes of the traits x_1 and x_2 in the focal species X are determined by the additive action of three sets of diallelic diploid loci. Specifically, the value of trait x_1 is determined by n_{x_1} x_1 -loci that affect only trait x_1 in addition to n_p pleiotropic loci that effect both x_1 and x_2 . The value of trait x_2 depends upon n_{x_2} x_2 -loci unique to that trait as well as the n_p pleiotropic loci. With positive pleiotropy the n_p shared loci increase the phenotypic value of both trait x_1 and x_2 , creating a positive genetic correlation between these traits. With negative pleiotropy, the n_p shared loci increase the phenotypic value of trait x_1 but decrease the value of x_2 , creating a negative genetic correlation between these traits. For the simulation results reported below the number of loci affecting each of the four traits was set to 10. For the multivariate species, this means that $n_p + n_{x_1} = n_p + n_{x_2} = 10$ in all simulations. To quantify the amount of pleiotropy, we define a pleiotropy index $P = \pm n_p / (n_p + n_{x_1}) = \pm n_p / (n_p + n_{x_2})$, with the sign depending on whether alleles at the shared loci have the same or opposite effects on the two traits. For example, $P = -0.5$ means that $n_p = 5$, and that alleles at the shared loci have opposite effects on the two traits. Highly positive or negative pleiotropy indices generally cause strong positive or negative genetic correlations between the two traits in the multivariate species. A more detailed description of the genetic architecture of the three interacting species and additional simulation details are explained in Figure 3.

To facilitate comparison of the evolutionary dynamics of the various traits and to level the playing field for the interacting species, the phenotypic values of all traits were scaled to lie within the interval [0, 1]. This prevents the interacting species with the greatest number of loci from winning the coevolutionary race simply by having a greater range of pos-

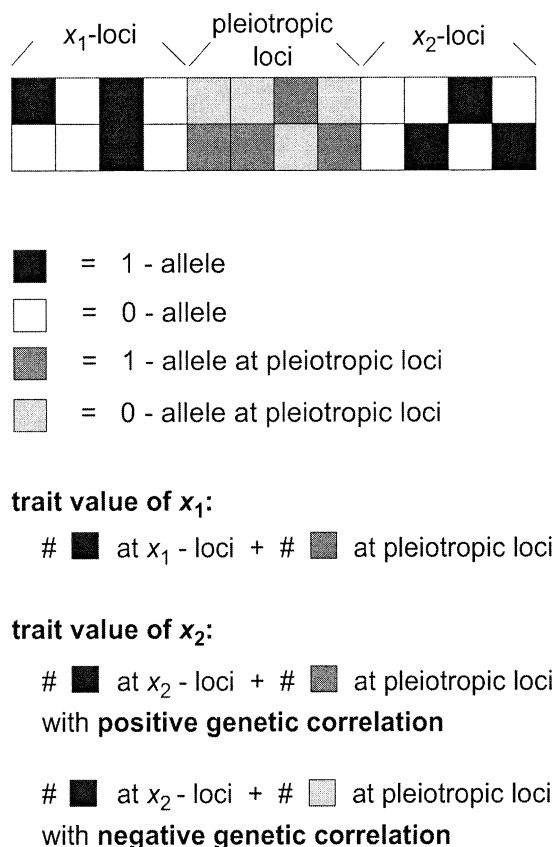


FIG. 3. Model description. The figure is a schematic representation of the genetic architecture in the multivariate species. The genetic architecture in the single-trait species is analogous to that for a single trait in the multivariate species. Further model specifications are as follows. Population size is finite and constant and was set to 1000 in all simulation results reported below. The population is initialized with individuals whose alleles are chosen uniformly randomly (so that the mean phenotypes are close to 0.5 for all traits). In each generation, the relative fitness of each individual is calculated according to the fitness functions (1a–c), with the integrals replaced by appropriate sums over all individuals in a population. Pairs of individuals are then drawn within each species with probabilities according to the relative fitnesses of individuals. Each mating pair produces one diploid offspring individual using Mendelian segregation and free recombination between all loci. In addition, we assume that reversible mutations occur in the offspring at a rate of 10^{-4} per locus and generation. Choosing different values for this mutation rate does not lead to qualitative differences from the results we report. Mating is repeated until the fixed population size is reached, thus completing one generation.

sible phenotypes. A consequence of this scaling is that increasing the number of loci controlling a specific trait decreases the effect of each locus, leading to a decrease in the additive genetic variance. This effect is not specific to our chosen scaling of $[0, 1]$, but rather is an inevitable consequence of imposing any finite trait interval.

While the ultimate goal of the individual-based model is to investigate the full coevolutionary dynamics of three species systems, it is worth first considering how the evolution of genetic variance changes our expectations for a single pair of interacting species. With only two interacting species with a single trait in each, there are three possible cases: $+/+$ (mutualism), $-/-$ (competition), and $+/-$ (antagonism). The

individual-based model does not yield any new insights for the first two cases: mutualistic interactions simply coevolve matching phenotypes generally centered in the middle of the trait intervals (due to mutation), while competitive interactions lead to the coevolution of maximally mismatched phenotypes $[0, 1]$. The antagonistic case can, however, yield results that are fundamentally different from those obtained with the analytical model with fixed genetic variances.

In the fixed genetic variance model the matching equilibrium is predicted to be stable if the predator/parasite has a greater response to selection than the prey/host (Gavrilets 1997). In the individual-based model, however, the evolution of additive genetic variance can often preclude the long-term stability of this equilibrium. The mechanism behind this novel phenomenon is as follows. At the matching equilibrium the parasite/predator species experiences stabilizing selection due to the increased fitness of individuals with phenotypes closer to that of the majority of prey/host individuals. In contrast, the prey/host species experiences disruptive selection. If selection on the parasite is quite strong, stabilizing selection erodes parasite genetic variance faster than it can be replenished by mutation. At the same time, the host experiences a modest increase in its genetic variance due to disruptive selection. The net result is a decrease in the response to selection of the predator/parasite relative to that in the prey/host. As a consequence, the prey can escape to one side of the predator, which destabilizes the matching equilibrium and initializes a coevolutionary race exhibiting permanent cyclic dynamics (Fig. 4). It is important to note that this dynamical regime does not occur in the fixed variance model of the previous section. As we will see, the possibility of cyclic coevolutionary dynamics can have important consequences for the multivariate case, which we consider next.

Just as the analytical model, the individual-based model can be used to study all the cases in Figure 2. However, we will again restrict ourselves to a subset of salient examples. To develop results parallel to the previous section, we consider the same subset of three-way interactions detailed for the model with fixed genetic variance. The results of simulations show that the effect of covariance is often in accordance with what is predicted by the analytical model for the stability of matching equilibria. Even in these cases, though, the simulations enrich our understanding of the coevolutionary process by revealing novel dynamics and outcomes. In particular, the individual-based model can have stable evolutionary equilibria that are different from the matching equilibrium, leading to permanent maladaptation.

Case 1: mutualism-antagonism

In this case, the prediction made by the analytical model for the effect of increasing the genetic correlation between traits depends on whether the multivariate species is the prey or the predator in the antagonistic interaction. If the multivariate species is the prey, then increasing the covariance should stabilize the matching equilibrium. This is indeed generally the case in the individual-based model, as is illustrated in Figure 5. Figure 5A shows the dynamics in the presence of a low level of pleiotropy between the traits affecting the mutualistic and antagonistic interactions. The mutualistic in-

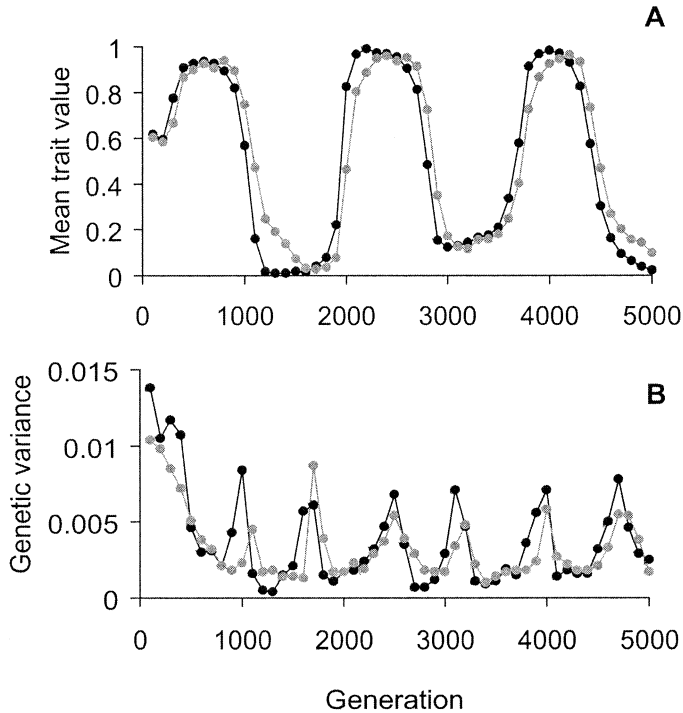


FIG. 4. Cyclic arms race generated by a predator-prey interaction in the multilocus model. Mean trait values of the prey (black dots, A) and predator (gray dots, A) match near the boundaries of the trait interval, where the predator can catch up with the prey. Escape of the prey from the matching state is precipitated by a drop in the genetic variance in the predator (gray dots, B) and leads to a phase of directional selection in both prey and predator, during which genetic variances increase. Directional phases and quasi-stable matching alternate, leading to cyclic coevolutionary dynamics. $\alpha_{xy} = -1.5$, $\alpha_{yx} = 1.9$.

teraction stabilizes at the matching equilibrium, while the antagonistic interaction leads to a cyclic arms race (due to evolving genetic variances, see above). However, the genetic correlation already induces lower amplitudes in the arms race cycles. Figure 5B shows that an increase in the level of pleiotropy can lead to stabilization of the matching equilibrium for both interactions if the mutualistic interaction is stronger than the antagonistic interaction (i.e., if the interaction coefficients α in expressions 1a–c are larger in magnitude for the mutualistic interaction), as we assumed in Figure 5. This is because via the genetic correlation generated by pleiotropy, the mutualistic trait in the multivariate species keeps the prey trait in check so that the latter can no longer escape from the predator. Such stabilization is prevented if the antagonistic interaction becomes too strong.

Again in accordance with the analytical prediction, the matching equilibrium is not stabilized by increased covariance when the multivariate species is the predator. However, if the mutualistic interaction is strong enough, a new type of dynamics is seen in the individual-based model: coevolution stabilizes at an equilibrium that is different from the matching equilibrium (Fig. 5). Figure 5C again shows the dynamics with only a weak genetic correlation, which is essentially the same as in Figure 5A. In Figure 5D, the dynamics stabilize at an equilibrium at which the prey species has permanently

escaped the multivariate predator. Thus, in this case the genetic correlation between the mutualistic and the predator traits can again prevent the cyclic arms race, but at a permanent cost to the predator. Note that the dynamics shown in Figure 5D are quite different from those shown in Figure 5B, despite the fact that both cases depend on the effect of covariance between a mutualistic and an antagonistic trait pair. The difference is that in one case the genetic correlation acts on the prey trait and in the other on the predator trait.

Case 2: antagonism-antagonism

We first consider the case in which the multivariate species preys upon both single-trait species (Fig. 2E). As is expected from the results for a single antagonistic trait pair (Fig. 4), the coevolutionary dynamics results in two cyclic arms races if there is a low level of pleiotropy between the two predator traits (Fig. 6A). The prediction from the analytical model is that increasing the covariance between these traits should decrease the stability of the matching equilibrium. In the present situation, however, this is an irrelevant result because the evolution of genetic variance renders matching unstable even in the absence of genetic correlation. Figure 6B shows an example of coevolution with a high negative correlation: the system is very far from matching and instead stabilizes at an equilibrium at which both prey species have escaped their predator. The two predator traits are held in the middle of the trait interval by the genetic correlation, which allows the prey to escape and causes a substantial degree of maladaptation in the predator. This result is in good qualitative agreement with the prediction of the analytical model that increasing genetic correlations favor the escape of the prey species. What we see again, however, is that the evolution of genetic variances and covariance leads to the existence of a novel equilibrium state not possible with fixed genetic variances and covariances. We note that a stable equilibrium representing permanent maladaptation in the predator also emerges with large positive correlations, in agreement with the prediction from the deterministic model of the previous section that the sign of the correlation should not matter for the qualitative features of the coevolutionary dynamics.

A very different picture emerges when the multivariate species is the prey of both single-trait species (Fig. 6C,D). In this case, the coevolutionary dynamics never stabilize and instead remain cyclic, independent of the amount of genetic correlation (Fig. 6D). Thus, even though the analytical prediction for the effect of increased covariance is the same as before, the actual dynamics play out very differently in the two cases. We note that the dynamics of the case in which the multivariate species is both predator and prey are very similar to the case just shown in Figures 6C,D, even though in this case the analytical prediction is that increased covariance should have a stabilizing effect.

Case 3: competition-antagonism

We now give an example of how genetic correlations can induce maladaptation in competitive interactions. We consider a case where the focal species is a competitor in one interaction and a predator in the other interaction, and we assume that the antagonistic interaction is much stronger than

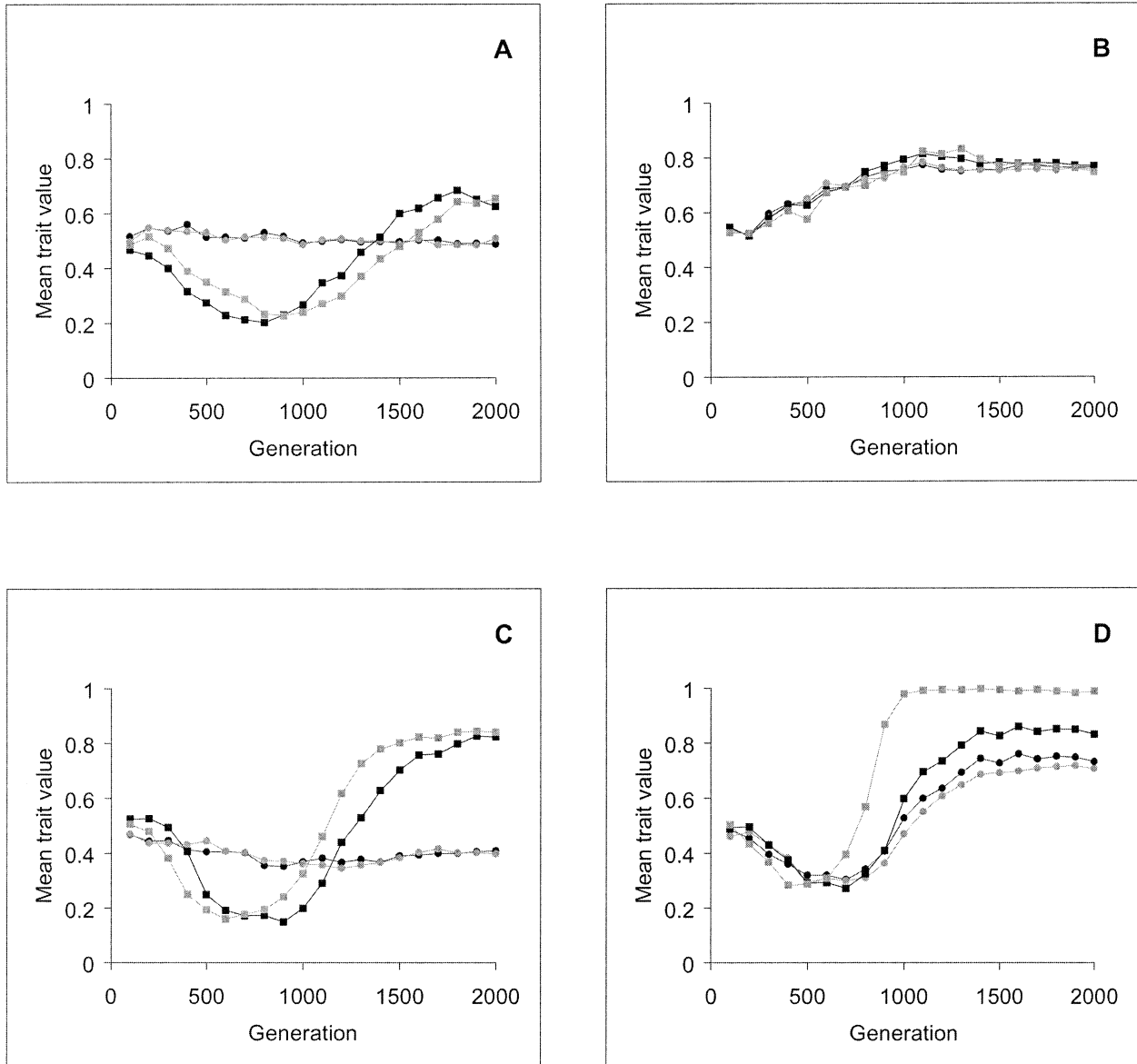


FIG. 5. Effects of genetic correlation in a multivariate species involved in a mutualistic and a predator-prey interaction. (A) The multivariate species is the prey in the antagonistic interaction, with a low correlation between the prey trait and the trait determining the mutualistic interaction (pleiotropy index $P = 0.1$). The mutualistic interaction (black dots: multivariate species; gray dots: single-trait mutualist) leads to a stable matching equilibrium, while the antagonistic interaction (black squares: multivariate prey species; gray squares: single-trait predator) exhibits cyclic dynamics. Note that the correlation induces damped oscillations in the cyclic arms race. (B) The genetic correlation is high (pleiotropy index $P = 0.9$) and the mutualistic interaction induces a stable matching equilibrium for all traits. (C) The same as panel A, but now the multivariate species is the predator (black squares). Again, for low correlation (pleiotropy index $P = 0.1$) there is cycling in the antagonistic interaction and stable matching in the mutualistic interaction. (D) A strong correlation exists (pleiotropy index $P = 0.9$) and the coevolutionary dynamics stabilize at an equilibrium at which the prey is permanently displaced from the predator. $\alpha_{x_1y} = 1.5$, $\alpha_{yx_1} = 1.7$, $\alpha_{xz_2} = -0.4$, $\alpha_{z_2x} = 0.5$ in A and B; $\alpha_{x_1y} = 1.5$, $\alpha_{yx_1} = 1.7$, $\alpha_{xz_2} = 0.4$, $\alpha_{z_2x} = -0.5$ in C and D.

the competitive interaction (Fig. 2I). If the correlation between the antagonistic trait and the competitive trait in the focal species is low, the antagonistic trait pair exhibits cycles, while the competitive trait pair diverges, as expected (Fig. 7A). However, if this correlation is high, the coevolutionary cycles in the antagonistic trait can drag the competitive trait along in such a way that the two competitors, rather than diverging to a permanent mismatch, undergo repeated temporary convergence (Fig. 7B). Thus, with a large genetic

correlation the cyclic arms race between predator and prey can lead to repeated crossings of the competitive traits, representing periods of maladaptation. It is worth noting that this is the effect that can lead to stabilization of the matching equilibrium in the analytical model, as described at the end of the previous section. If the antagonistic interaction has a stable matching equilibrium in the analytical model, then the analytical model exhibits decreasing cycles toward this matching equilibrium. If the antagonistic interaction is strong

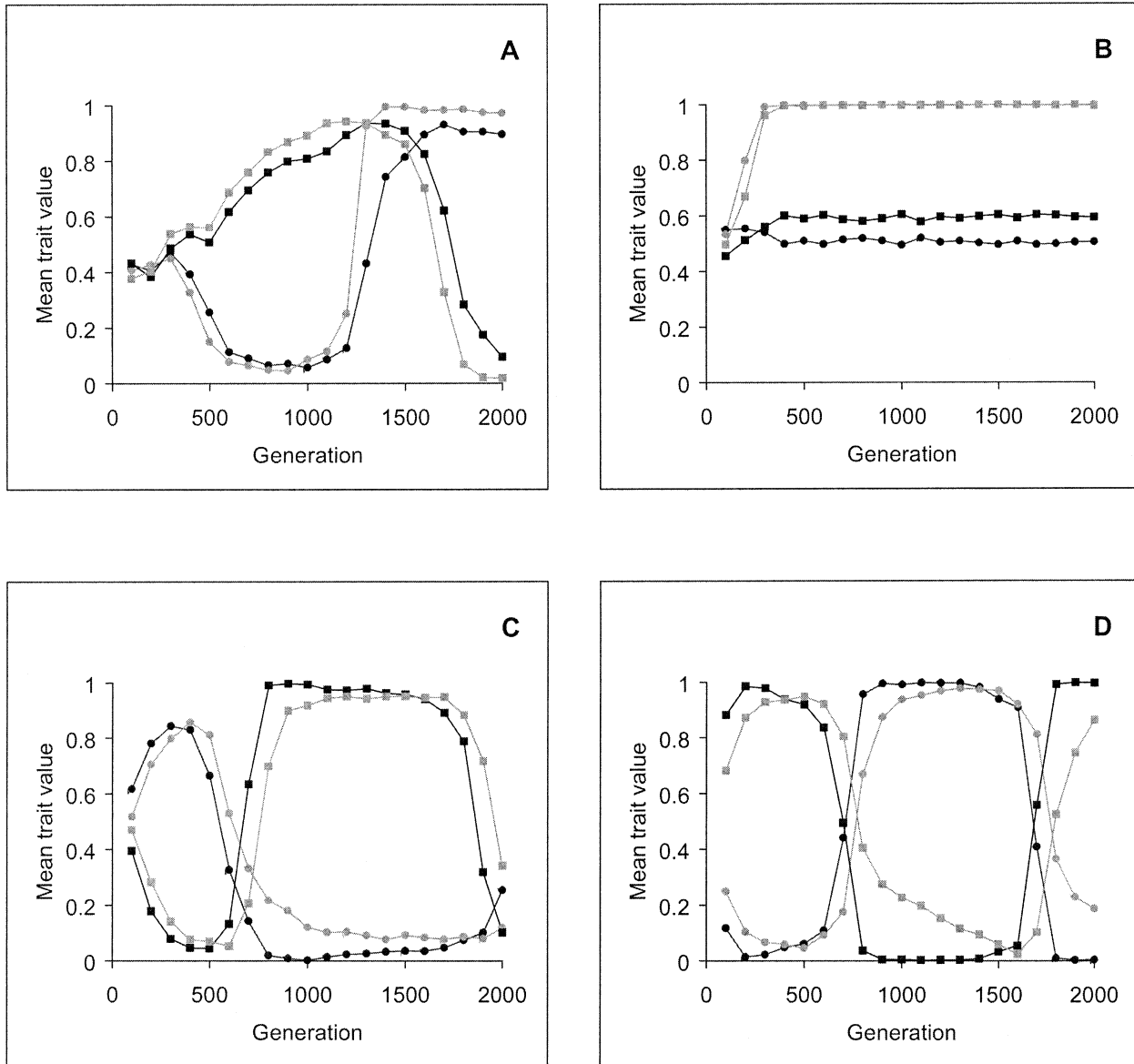


FIG. 6. Effects of genetic correlation in a multivariate species involved in two predator-prey interactions. (A) The multivariate species is the predator in both interactions (and hence feeds on two distinct prey species). With a low genetic correlation between the predator traits (pleiotropy index $P = 0.1$), both trait pairs exhibit cyclic dynamics (black squares and dots: multivariate species; gray squares and dots: corresponding prey species). (B) There is a large negative genetic correlation (pleiotropy index $P = -0.9$) that induces a new stable equilibrium at which both prey species have permanently escaped their predator. (C) The same as panel A, but now the multivariate species is the prey in both interactions (and hence is eaten by two different predators). With a low correlation (pleiotropy index $P = 0.1$), both trait pairs again exhibit cyclic dynamics. (D) A large negative correlation exists (pleiotropy index $P = -0.9$) and the dynamics remain essentially unchanged. $\alpha_{x_1y} = 1.0$, $\alpha_{yx_1} = -1.1$, $\alpha_{x_2z} = 1.2$, $\alpha_{zx_2} = -0.9$ in A and B; $\alpha_{x_1y} = -1.0$, $\alpha_{yx_1} = 1.1$, $\alpha_{x_2z} = -1.2$, $\alpha_{zx_2} = 0.9$ in C and D.

enough, these cycles induce the repeated crossing over of competitive traits described above, thus effectively trapping the competitive traits at the matching equilibrium, hence the analytical prediction that a high covariance can stabilize the matching equilibrium for the whole system.

DISCUSSION

Our analyses suggest that genetic correlations can play an important role in shaping the coevolutionary dynamics of three-species systems. In cases of weak selection, where ge-

netic variances and covariances may remain approximately constant, our analytical results show that genetic correlations can change the stability of coevolutionary equilibrium points, thus determining the outcome of coevolution. In cases of strong selection, where the genetic variances and covariances themselves evolve, results from simulations reveal that correlations can lead to novel equilibria and dynamic regimes. This is true, in particular, if at least one of the coevolving trait pairs is involved in an antagonistic interaction. In this case, genetic correlations can, for example, generate trade-

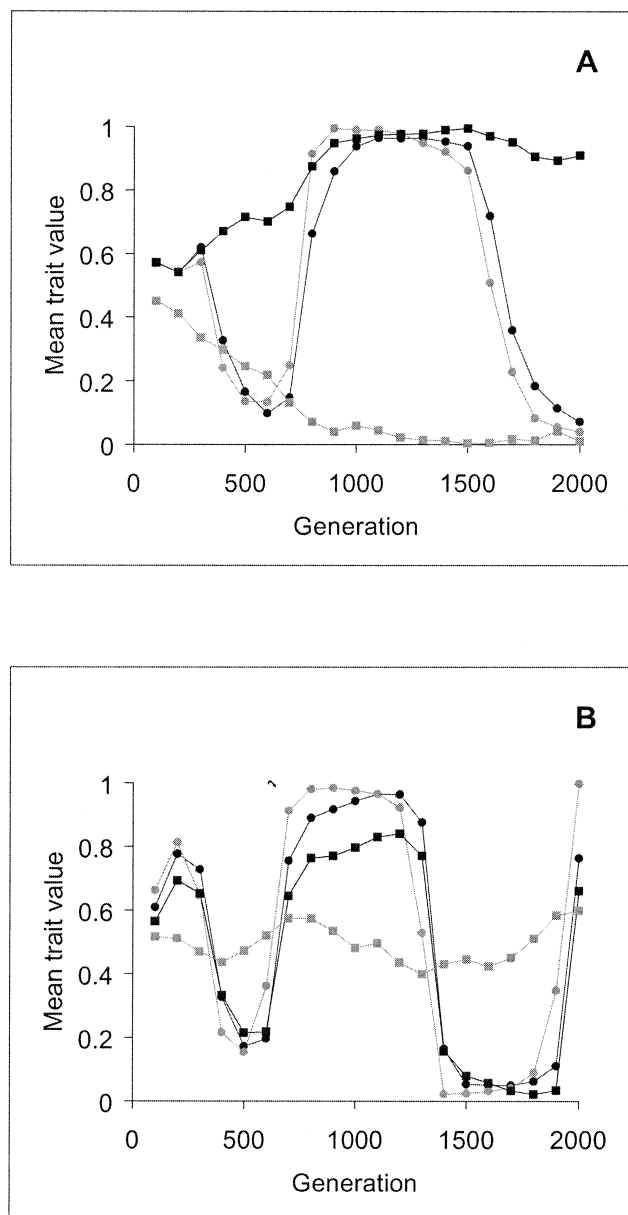


FIG. 7. Correlation between antagonistic and competitive traits can lead to maladaptive convergence of competitive trait pairs. The multivariate species is both a predator (black dots; gray dots represents the corresponding prey) and a competitor (black squares; gray squares is the corresponding competitor). With low genetic correlations (A, pleiotropy index $P = 0.1$) the competitive traits are expected to diverge. However, with a high correlation (B, pleiotropy index $P = 0.8$) the cycles in the predator trait (black dots) caused by the coevolutionary arms race drag along the competitive trait (black squares), leading to repeated crossings of the competitor traits. $\alpha_{x_1y} = 2.2$, $\alpha_{yx_1} = -0.05$, $\alpha_{x_2z} = -2.1$, $\alpha_{zx_2} = -0.04$.

offs that lead to cyclically recurring or permanent maladaptation in one of the coevolving species (see Figs. 5–7).

The importance of genetic correlations for the dynamics of three-species systems is not universal, however. Our analytical results allow us to categorize which interactions undergo qualitative changes in coevolutionary dynamics in response to changing strengths of genetic correlation. These

results are summarized in Figure 2 and generate two broad predictions. First, systems where a joint fitness maximum exists for all three species, such as three-way mutualistic or competitive interactions, reach the same equilibrium irrespective of genetic correlation. Second, whether correlations act to stabilize or destabilize the matching equilibrium is determined by the fitness effects of the interactions on the multivariate species. If the multivariate species is harmed or benefited by matching in both its interactions with the other two species, genetic correlations favor coevolutionary instability. In contrast, if the multivariate species benefits from matching in one of the interactions while being harmed by matching in the other, genetic correlations favor coevolutionary stability. These results are corroborated by the individual-based simulations. We have never seen cases in which increasing the genetic correlation resulted in more stability for the matching equilibrium in the individual-based model when a decrease in stability was predicted by the analytical model and vice versa.

These predictions have interesting biological implications for several specific three-way interactions. One of the most interesting is the role that genetic correlations may play in driving the evolution of specialization. For example, consider the interactions between a multivariate parasite and two host species. Here our prediction is that increasing genetic correlations should destabilize the matching equilibrium. As a consequence, increasing the genetic correlation between the parasite traits causes the parasite to become increasingly maladapted to both host species (Fig. 6B). As a consequence, given some sufficient level of genetic correlation, it becomes advantageous for the parasite to abandon attempts to use both host species and instead specialize on a single host. This suggests a readily testable prediction: generalist parasites should be characterized by a low genetic correlation between traits mediating interactions with different host species. A similar phenomenon occurs when the multivariate species interacts as a mutualist with one species, but as a parasite of the other. In this case, increasing the genetic correlation between traits tends to destabilize the matching equilibrium favoring the evolutionary escape of the prey or host species, effectively forcing the multivariate species into specializing as a mutualist rather than as a parasite (Fig. 5D).

Another interesting and counterintuitive result to emerge from our model is that positive and negative correlations should have similar effects. This is apparent in the stability analysis of the deterministic model and is supported by our extensive simulations of the individual-based model, in which we have never seen a qualitative difference in the coevolutionary dynamics for positive and negative genetic correlations. While it has often been assumed that negative genetic correlations should be of primary importance because they lead to trade-offs (e.g., Hougenitzman and Rausher 1994; Rausher 1996), our results show that positive correlations may be equally important. This result, however, is likely to depend on the mode of coevolution considered. Here we have considered a model of coevolutionary matching; models based on coevolutionary escalation often yield different results (e.g., Abrams and Matsuda 1997; Abrams 2000). This can be clearly seen by considering the case of a parasite that uses two host species. In our model of coevo-

lutionary matching, the parasite must match phenotypes with both host species. Hence, a positive genetic correlation may generate a trade-off in our model if, for instance, one host evolves a small phenotype while the other evolves a large phenotype. In a model of coevolutionary escalation, however, a large phenotype is always better, and negative genetic correlations are much more likely to generate a trade-off (e.g., Berenbaum et al. 1986; Bergelson et al. 2001)

To this point we have focused on the results that emerge from both the fixed genetic variance and explicit multilocus models. Not surprisingly, however, these models generate quite different results and predictions in some respects. Of primary importance is the qualitative difference in behavior observed when one of the interactions is antagonistic. While such interactions can be stable with fixed genetic variance (e.g., Gavrillets 1997), when the genetic variance evolves this may not be the case. Instead, coevolutionary cycles can emerge as parasite genetic variance is eroded by stabilizing selection (Fig. 4). A consequence of this discrepancy is that under a broad range of conditions three-way interactions containing an antagonistic component are predicted to behave quite differently by the two modeling frameworks. In particular, explicit multilocus genetics with fixed trait intervals generates more interesting dynamics than the simple analytical model, in which traits are often predicted to evolve to infinity. For example, the individual-based model can produce sustained and bounded oscillations. Perhaps more interestingly, however, high genetic correlations in the individual-based model can induce novel stable equilibrium states that are different from the matching equilibria found as the sole equilibrium states in the analytical model.

Rather than simply concluding that one of the models is wrong in these cases, it is worthwhile to consider the conditions under which the predictions of each model might be most profitably applied. For interactions characterized by weak selection, for instance, the fixed genetic variance model may offer accurate predictions. This view is largely supported by the maintenance of substantial and relatively constant levels of genetic variance in some long-term artificial selection experiments (e.g., Falconer and Mackay 1996). From a more practical perspective, the fixed genetic variance model has the added benefit of relying on parameters that can be measured in natural populations (e.g., Berenbaum et al. 1986; Berenbaum and Zangerl 1992). In contrast, the parameters required by the explicit multilocus model (e.g., the number of loci, effects of loci, degree of pleiotropy) are not currently available for most well-studied systems. Despite this practical limitation, the explicit multilocus model is likely to provide better predictions when the genetic variances or covariances of the interacting species themselves evolve. This may be particularly likely in interactions characterized by strong selection. It is encouraging, though, that for the wide spectrum of interesting biological scenarios we have considered, both approaches lead to qualitatively similar results and predictions.

In this light, it is worth considering the potential consequences of the assumptions shared by both models. Three assumptions in particular stand out as potentially important. First, we do not incorporate any form of stabilizing selection on the traits mediating the interactions. As pointed out by

Gavrillets (1997), this lack of stabilizing selection can generate biologically unreasonable results in the analytical framework by allowing trait values to become infinite. However, in our explicit multilocus models, trait values are constrained to be finite, so that stabilizing selection is not necessary to prevent infinite trait values or to generate coevolutionary cycles. Second, we have assumed that the fitness of the multivariate species is determined by the multiplicative action of its interactions with the other two species. While for some interactions this appears to be the case (Hougeneytman and Rausher 1994), there are clearly others where the fitness consequences of interacting with multiple other species are not independent (Agrawal 2000; Stinchcombe and Rausher 2001; Hufbauer and Root 2002). Third, we have assumed a very specific functional form between the phenotypes of the interacting species and fitness. We have assumed that fitness depends on the squared phenotypic distance. This is a reasonable assumption for many interactions, including those between mutualists, competitors, and predators and their prey (e.g., Steiner and Whitehead 1991; Benkman 1999; Schluter 2003). In other systems, however, the functional form of the relationship between fitness and the phenotypes of the interacting species may be quite different (e.g., Brodie and Brodie 1999; Bergelson et al. 2001). This can have important consequences for coevolutionary dynamics (e.g., Abrams 2000). Exploring these consequences is an important focus for future work.

We have analyzed a very simple model of coevolution between three interacting species. Our goal was not to completely describe or document the detailed dynamics of any particular interaction, but rather to provide a broad survey of the impact of genetic correlations on various three-species interactions. In light of this, we view this work as a roadmap to future research rather than as an endpoint in and of itself. Despite this simplicity, however, our model has demonstrated that genetic correlations can play a critical role in shaping the coevolutionary dynamics of three-species interactions. It is our hope that these results will stimulate further theoretical and empirical work on the coevolutionary dynamics of multispecies interactions.

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LITERATURE CITED

- Abrams, P. A. 2000. The evolution of predator-prey interactions: theory and evidence. *Annu. Rev. Ecol. Syst.* 31:79–105.
 Abrams, P. A., and H. Matsuda. 1997. Fitness minimization and dynamic instability as a consequence of predator-prey coevolution. *Evol. Ecol.* 11:1–20.
 Agrawal, A. A. 2000. Specificity of induced resistance in wild radish: causes and consequences for two specialist and two generalist caterpillars. *Oikos* 89:493–500.
 Benkman, C. W. 1999. The selection mosaic and diversifying coevolution between crossbills and lodgepole pine. *Am. Nat.* 153: S75–S91.

- Benkman, C. W., W. C. Holimon, and J. W. Smith. 2001. The influence of a competitor on the geographic mosaic of coevolution between crossbills and lodgepole pine. *Evolution* 55: 282–294.
- Berenbaum, M. R., and A. R. Zangerl. 1992. Genetics of physiological and behavioral resistance to host furanocoumarins in the parsnip webworm. *Evolution* 46:1373–1384.
- Berenbaum, M. R., A. R. Zangerl, and J. K. Nitao. 1986. Constraints on chemical coevolution: wild parsnips and the parsnip webworm. *Evolution* 40:1215–1228.
- Bergelson, J., G. Dwyer, and J. J. Emerson. 2001. Models and data on plant-enemy coevolution. *Annu. Rev. Genet.* 35:469–499.
- Brodie, E. D., III, and E. D. Brodie Jr. 1999. Costs of exploiting poisonous prey: evolutionary trade-offs in a predator-prey arms race. *Evolution* 53:626–631.
- Bulmer, M. 1994. *Theoretical evolutionary ecology*. Sinauer Associates, Sunderland, MA.
- Case, T. J., and M. L. Taper. 2000. Interspecific competition, environmental gradients, gene flow, and the coevolution of species' borders. *Am. Nat.* 155:583–605.
- Charlesworth, B. 1993. Natural selection on multivariate traits in age-structured populations. *Proc. R. Soc. Lond. B* 251:47–52.
- Darwin, C. 1859. *On the origin of species by means of natural selection, or preservation of favored races in the struggle for life*. John Murray, London.
- Dieckmann, U., and M. Doebeli. 1999. On the origin of species by sympatric speciation. *Nature* 400:354–357.
- Dieckmann, U., P. Marrow, and R. Law. 1995. Evolutionary cycling in predator-prey interactions: population dynamics and the red queen. *J. Theor. Biol.* 176:91–102.
- Doebeli, M. 1997. Genetic variation and the persistence of predator-prey interactions in the Nicholson-Bailey model. *J. Theor. Biol.* 188:109–120.
- Doebeli, M., and U. Dieckmann. 2000. Evolutionary branching and sympatric speciation caused by different types of ecological interactions. *Am. Nat.* 156:S77–S101.
- Dupas, S., Y. Carton, and M. Poirie. 2003. Genetic dimension of the coevolution of virulence-resistance in *Drosophila*-parasitoid wasp relationships. *Heredity* 90:84–89.
- Falconer, D. S., and T. F. C. Mackay. 1996. *Introduction to quantitative genetics*. Longman, Essex, England.
- Gavrilets, S. 1997. Coevolutionary chase in exploiter-victim systems with polygenic characters. *J. Theor. Biol.* 186:527–534.
- Gavrilets, S., and A. Hastings. 1998. Coevolutionary chase in two-species systems with applications to mimicry. *J. Theor. Biol.* 191:415–427.
- Gomulkiewicz, R., S. L. Nuismer, and J. N. Thompson. 2003. Coevolution when a mutualism is not always a mutualism. *Am. Nat.* 162:S80–S93.
- Hougeneytzman, D., and M. D. Rausher. 1994. Interactions between herbivorous insects and plant-insect coevolution. *Am. Nat.* 143: 677–697.
- Hufbauer, R. A., and R. B. Root. 2002. Interactive effects of different types of herbivore damage: *Trirhabda* beetle larvae and *Philaenus* spittlebugs on goldenrod (*Solidago altissima*). *Am. Midl. Nat.* 147:204–213.
- Kiple, K. F. 1993. *The Cambridge world history of human disease*. Cambridge Univ. Press, New York.
- Lande, R. 1979. Quantitative genetic analysis of multivariate evolution, applied to brain:body size allometry. *Evolution* 33: 402–416.
- . 1981. Models of speciation by sexual selection on polygenic traits. *Proc. Natl. Acad. Sci. USA* 78:3721–3725.
- . 1982. A quantitative genetic theory of life history evolution. *Ecology* 63:607–615.
- Lande, R., and S. J. Arnold. 1983. The measurement of selection on correlated characters. *Evolution* 37:1210–1226.
- Nuismer, S. L., and J. N. Thompson. 2001. Plant polyploidy and non-uniform effects on insect herbivores. *Proc. R. Soc. Lond. B* 268:1937–1940.
- Nuismer, S. L., J. N. Thompson, and R. Gomulkiewicz. 1999. Gene flow and geographically structured coevolution. *Proc. R. Soc. Lond. B* 266:605–609.
- Rausher, M. D. 1996. Genetic analysis of coevolution between plants and their natural enemies. *Trends Genet.* 12:212–217.
- Schluter, D. 2003. Frequency dependent natural selection during character displacement in sticklebacks. *Evolution* 57:1142–1150.
- Seeger, J. 1988. Dynamics of some simple host-parasite models with more than two genotypes in each species. *Philos. Trans. R. Soc. Lond. B* 319:541–555.
- Segraves, K. A., and J. N. Thompson. 1999. Plant polyploidy and pollination: floral traits and insect visits to diploid and tetraploid *Heuchera grossularifolia*. *Evolution* 53:1114–1127.
- Slatkin, M. 1984. Ecological causes of sexual dimorphism. *Evolution* 38:622–630.
- Steiner, K. E., and V. B. Whitehead. 1991. Oil flowers and oil bees: further evidence for pollinator adaptation. *Evolution* 45: 1493–1501.
- Stinchcombe, J. R., and M. D. Rausher. 2001. Diffuse selection on resistance to deer herbivory in the ivyleaf morning glory, *Ipomoea hederacea*. *Am. Nat.* 158:376–388.
- Thompson, J. N. 1994. *The coevolutionary process*. Univ. of Chicago Press, Chicago.
- . 1998. Rapid evolution as an ecological process. *Trends Ecol. Evol.* 13:329–332.
- . 1999. The evolution of species interactions. *Science* 284: 2116–2118.
- Thompson, J. N., and O. Pellmyr. 1992. Mutualism with pollinating seed parasites amid co-pollinators: constraints on specialization. *Ecology* 73:1780–1791.
- Thompson, J. N., B. C. Cunningham, K. A. Segraves, D. M. Althoff, and D. Wagner. 1997. Plant polyploidy and insect/plant interactions. *Am. Nat.* 150:730–743.

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