When Is Correlation Coevolution?

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ABSTRACT: Studying the correlation between traits of interacting species has long been a popular approach for identifying putative cases of coevolution. More recently, such approaches have been used as a means to evaluate support for the geographic mosaic theory of coevolution. Here we examine the utility of these approaches, using mathematical and computational models to predict the correlation that evolves between traits of interacting species for a broad range of interaction types. Our results reveal that coevolution is neither a necessary nor a sufficient condition for the evolution of spatially correlated traits between two species. Specifically, our results show that coevolutionary selection fails to consistently generate statistically significant correlations and, conversely, that non-coevolutionary processes can readily cause statistically significant correlations to evolve. In addition, our results demonstrate that studies of trait correlations per se cannot be used as evidence either for or against a geographic mosaic process. Taken together, our results suggest that understanding the coevolutionary process in natural populations will require detailed mechanistic studies conducted in multiple populations or the use of more sophisticated statistical approaches that better use information contained in existing data sets.

Keywords: geographic mosaic, trait matching, species interactions, local adaptation, character displacement.

> Thus I can understand how a flower and a bee might slowly become, either simultaneously or one after the other, modified and adapted to each other in the most perfect manner, by the continued preservation of all the individuals which presented slight deviations of structure mutually favourable to each other. (Darwin 1909, p. 109)

Introduction

Since Darwin sketched this outline for a process of evolution between a plant and its pollinator, numerous studies have focused on identifying examples of such coevolution, now formally defined as the reciprocal evolution of interacting species (Janzen 1980). Examples include studies of phenotype matching between pollinator and plant floral morphologies (e.g., Steiner and Whitehead 1991; Anderson and Johnson 2008) and studies of character displacement in competitors (e.g., Grant and Grant 2006; Albert et al. 2007; Carlson et al. 2009; Moen and Wiens 2009). In these examples, positive correlations between pollinator and floral morphology across locations or negative correlations between trait values of competitors across locations are commonly taken as partial evidence for coevolution. Similar approaches have been applied to interactions between hosts and parasites or predators and prey as a method to evaluate support for a coevolutionary hypothesis (e.g., Berenbaum and Zangerl 1998; Benkman 1999; Brodie et al. 2002; Zangerl and Berenbaum 2003; Toju and Sota 2006; Nash et al. 2008).

Although studies of correlations between traits of interacting species are intuitively appealing, it has been argued that such studies cannot provide unequivocal evidence for coevolution. The argument against using correlated trait values as evidence for coevolution was made most forcefully by Janzen (1980) in his paper entitled "When is it coevolution?" He argued that well-matched or strongly correlated traits could evolve between interacting species through processes other than coevolution (Janzen 1980). Similar arguments have been made against using character displacement as evidence for competitive coevolution (Strong et al. 1979). At least three non-coevolutionary mechanisms could explain correlations between the traits of interacting species across sites. First, as Janzen argued, positive correlations will arise if, for instance, long-tongued pollinator individuals congregate in regions where plants tend to have, on average, long corollas but short-tongued pollinator individuals tend to congregate in regions where plants have, on average, short corollas. Second, traits may become correlated any time one species evolves to match the phenotype of an interacting species that fails to evolve in response either because it experiences only weak selection from the interaction or because it lacks heritable variation (evolutionary commensalism). Third, correlated traits could evolve if the

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abiotic environment favors similar traits in both of the interacting species.

Although these verbal arguments provide compelling reasons to avoid using correlations between traits as evidence for coevolution, they do not address whether a failure to identify correlated traits indicates an absence of coevolution. Nevertheless, following the publication of Janzen's (1980) arguments, studies appeared suggesting that a lack of well-matched or significantly correlated phenotypes demonstrates a lack of coevolution (see review in Thompson 1994). It was partially in response to this argument that Thompson (1994, 2005) developed his geographic mosaic theory of coevolution. A central theme of this theory is that reciprocal selection need not lead to well-matched or significantly correlated traits in all cases. Instead, the geographic mosaic theory predicts that—for a variety of reasons including drift, gene flow, and time lags in the coevolutionary process itself—reciprocal selection should lead to significantly correlated traits in only a subset of coevolutionary interactions, whereas others will show loose matching or even "trait mismatching" (Thompson 1994, 2005). Although a number of models now support the basic predictions of the geographic mosaic theory (Nuismer et al. 1999, 2000; Gomulkiewicz et al. 2000; Nuismer 2006), none of these models predicts the distribution of correlations expected to evolve as a result of coevolutionary and non-coevolutionary processes. Thus, we lack a quantitative framework within which to interpret the results of existing empirical studies and to evaluate the support they provide for coevolutionary and non-coevolutionary hypotheses.

In summary, compelling verbal arguments suggest that correlations between traits of interacting species observed across populations are not sufficient evidence for inferring a coevolutionary process (Janzen 1980). Equally compelling verbal arguments (Thompson 1994, 2005) supported by population and quantitative genetic theory (Nuismer et al. 1999, 2000; Gomulkiewicz et al. 2000; Nuismer 2006; Ridenhour and Nuismer 2007) suggest that a failure to demonstrate correlated traits is not evidence for an absence of coevolution. Despite these arguments, studies of trait correlations across populations continue to be used as partial evidence either for or against a coevolutionary hypothesis (e.g., Zangerl and Berenbaum 2003; Toju and Sota 2006; Anderson and Johnson 2008). There appear to be three primary reasons that studies of trait correlations persist. First, correlations between traits of interacting species are relatively easy to estimate, requiring only the measurement of population mean trait values for the interacting species at multiple locations. Second, the geographic mosaic theory predicts that traits will be well-matched in some locations but mismatched in others, a prediction that appears to be testable using information on population mean trait values and their correlations (Brodie et al. 2002; Zangerl and Berenbaum 2003; Toju and Sota 2006; Anderson and Johnson 2008). Finally, we lack quantitative predictions for the distribution of correlation coefficients expected to evolve under coevolutionary and non-coevolutionary scenarios, leaving the interpretation of measured correlations open to creative interpretation.

Here, we address this gap in existing theory by analyzing mathematical models that predict the distribution of correlation coefficients that evolves across a broad range of scenarios, ranging from an absence of coevolutionary selection to very intense coevolutionary selection. Our results provide a quantitative framework within which the support for coevolutionary or non-coevolutionary hypotheses can be evaluated on the basis of estimated values of interspecific correlations. Our results allow us to answer three specific questions. (1) When, if ever, can correlations be used to infer a coevolutionary process? (2) Does an absence of correlations preclude a coevolutionary process? (3) Do correlations provide information that can be used to evaluate support for the geographic mosaic theory?

Model Development and Analysis

The General Model

We model coevolution between a pair of species whose interactions with each other and the abiotic environment are mediated by a single quantitative trait. Our primary goal is to understand how the bivariate distribution of species' mean phenotypes is shaped by biotic and abiotic selection, random genetic drift, and migration. To that end, we model two species that are distributed in finite populations across a large number of ecologically variable locations. Gene flow is assumed to occur at rate m_i in species i and to follow an island model (Wright 1931).

Within each location, our model assumes that individual fitness is determined by biotic interactions and the abiotic environment. Specifically, the fitness of an individual of species i and phenotype z_i, given an encounter with an individual of species j and phenotype z_j, is

$$W(z_i, z_j) = \exp[-\gamma_i(z_i - \theta_i)^2]\omega_i(z_i, z_j). \quad (1)$$

The exponential on the right side of equation (1) models stabilizing selection imposed by the abiotic environment that occurs any time a trait involved in species interactions also contributes to performance in the physical environment (e.g., Fellowes et al. 1998; Brodie and Brodie 1999; Webster and Woolhouse 1999; Bergelson et al. 2001; Lahti 2005). The parameter θ_i is the optimum phenotype with respect to the abiotic environment, and the parameter γ_i determines how rapidly fitness declines with distance from

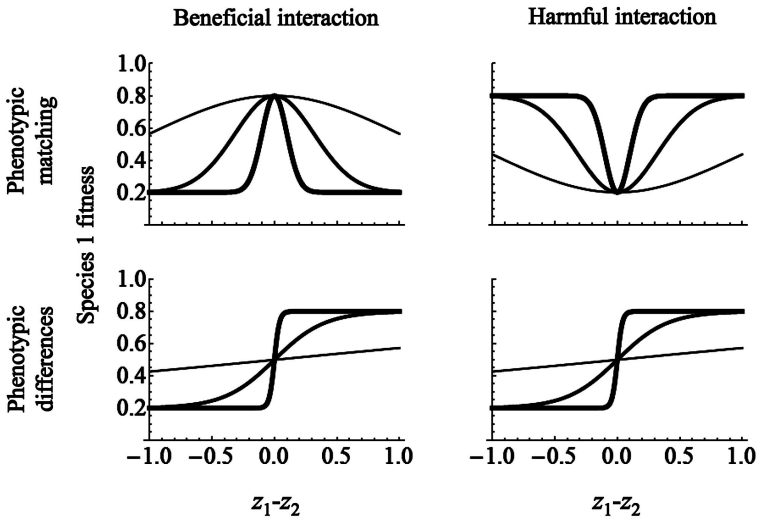

Figure 1: Fitness of an individual of species 1 in an interaction with an individual of species 2 as a function of the difference between their phenotypes ($z_1 - z_2$). The three lines on each plot correspond to three values of the parameter α {0.5, 5.0, 50.0}. The left-hand column shows the case where species 1 is benefitted by the interaction, whereas the right-hand column shows the case where species 1 is harmed by the interaction. The first row represents interactions mediated by phenotypic matching, whereas the second row represents interactions mediated by phenotypic differences. The parameter K was set to 0.2 in all plots where the species benefitted through interacting and to 0.8 in all plots where the species was harmed by interacting. The parameter ξ was set to 0.6 in all cases. Note that the species identities can be interchanged.

this optimum. We model spatial variation in the abiotic environment by drawing a pair of optima (one for each species) for each location at random from a bivariate normal distribution with means $\bar{\theta}_i$ and $\bar{\theta}_j$, variances $\sigma^2_{\theta_i}$ and $\sigma^2_{\theta_j}$, and covariance $\sigma_{\theta_i\theta_j}$. The factor $\omega_i(z_i, z_j)$ in equation (1) measures the fitness consequences of interspecific interaction for species i and depends on the trait values of the interacting individuals (z_1, z_2) and on whether the interaction is mediated by phenotypic matching (Gavrilets 1997; Nuismer et al. 2005; Kopp and Gavrilets 2006) or phenotypic differences (Nuismer et al. 2007).

For interactions mediated by phenotypic matching, fitness outcomes depend on the degree of similarity $|z_1 - z_2|$ of an interacting pair. This corresponds to Abrams's (2000) bidirectional axis of vulnerability. Examples include the egg morphology of cuckoos and their host birds (Soler et al. 2003), beak morphology in competing bird species (Grant and Grant 2006), and nectar spur length in plants and pollinator proboscis length in pollinators, where both plant and pollinator benefit by tighter phenotypic matching (Steiner and Whitehead 1991). Alternatively, interactions mediated by phenotypic differences depend on the signed pairwise difference, $z_1 - z_2$. This corresponds to a unidirectional axis of vulnerability (Abrams 2000). Examples include pericarp thickness in camellias and rostrum length in seed-predatory camellia weevils (Toju and Sota 2006), nectar spur length in plants and pollinator proboscis length in pollinators, where both plant and pollinator benefit by having larger trait values than the individuals they interact with (e.g., "Darwin's race"; Anderson and Johnson 2008; Muchhala and Thomson 2009), and body size in competing species of fig wasps (Moore et al. 2008).

To model phenotypic matching, we assume that the effect on fitness of an individual of species i in an encounter with an individual of species j, $i \neq j$, is

$$\omega_{\text{match},\,i}(z_i, z_j) = K_i + \xi_i \exp\left[-\alpha(z_i - z_j)^2\right], \qquad (2a)$$

where $\{|\xi_i| \leq K_i \leq 1;\ -1 \leq \xi_i \leq 0\}$ if the interaction reduces the fitness of species i and $\{0 \leq K_i \leq 1 - \xi_i;\ 0 \leq \xi_i \leq 1\}$ if its fitness is increased. The parameter α determines the impact of phenotypic matching (fig. 1, *top*). In the phenotypic difference case, we model the fitness outcome for an individual of species i in an encounter with an individual of species j, $i \neq j$, as

$$\omega_{\text{diffs},\,i}(z_i, z_j) = K_i + \frac{\xi_i}{1 + \exp\left[-\alpha(z_i - z_j)\right]}, \qquad (2b)$$

where $\{0 \leq K_i \leq 1 - \xi_i;\ 0 \leq \xi_i \leq 1\}$ in all cases (fig. 1, *bottom*).

Finally, we assume that random genetic drift occurs at the end of each generation. This is modeled, following

Lande (1976), by randomly sampling n_i individuals independently at each location of species i to form the next generation.

Quantitative Genetic Approximation

The model just described is mathematically complex and difficult to analyze except numerically. Here we derive a more tractable approximation by making several additional assumptions. First, we assume $\alpha \sim O(\varepsilon)$ and $\gamma_i \sim O(\varepsilon)$, where $\varepsilon \ll 1$ such that the fitness function (eq. [1]) can be well approximated by a first-order Taylor series in ε (Nuismer et al. 2005, 2007; Ridenhour and Nuismer 2007). This implies that fitness is not too sensitive to the distance between the phenotypes of the interacting individuals or to an individual's deviation from the optimum phenotype favored by abiotic selection. Next, we assume that phenotypes are normally distributed and that additive genetic variances of the interacting species' traits (G_i, G_j) are constant. These assumptions will be relaxed later through the use of individual-based simulations.

With these assumptions, the change in mean phenotype of species i at a given location is

$$\Delta\bar{z}_i = G_i[2\gamma_i(\theta_i - \tilde{\bar{z}}_i) + 2s_{M_i}(\tilde{\bar{z}}_j - \tilde{\bar{z}}_i) + s_{D_i}] + (\tilde{\bar{z}}_i - \bar{z}_i) + \zeta_i + O(\varepsilon^2), \quad (3)$$

where $i \neq j$; $\tilde{\bar{z}}_i = (1 - m_i)\bar{z}_i + m_i\mu_i$ is the mean phenotype within the location immediately after juvenile migration; and ζ_i, the change in mean phenotype caused by drift, is a random variable with a mean of 0 and a variance of G_i/n_i (see appendix in the online edition of the *American Naturalist*). For the case of phenotype matching, $s_{D_i} = 0$ and $s_{M_i} = \alpha_i\xi_i/(K_i + \xi_i)$, whereas for the case of phenotype differences, $s_{M_i} = 0$ and $s_{D_i} = \alpha_i\xi_i/(4K_i + 2\xi_i)$. We use equation (3) along with standard properties of the expectation and covariance to project changes in first and second central moments of the joint distribution of mean phenotypes over locations (appendix). The result is a system of nine recursions that track evolution of each species' expected mean phenotype across all populations (μ_1, μ_2), the spatial variances of each species' mean phenotype ($\sigma^2_{\bar{z}_1}$, $\sigma^2_{\bar{z}_2}$), the covariance between trait means ($\sigma_{\bar{z}_1\bar{z}_2}$), and the covariances between each trait mean and each local abiotic optimum ($\sigma_{\bar{z}_1\theta_1}$, $\sigma_{\bar{z}_2\theta_2}$, $\sigma_{\bar{z}_1\theta_2}$, $\sigma_{\bar{z}_2\theta_1}$).

Although the resulting dynamical system is linear, the recursions are still too complex to allow biological insight. For this reason, we further assume that the rate of gene flow is small ($m_i \sim O(\varepsilon)$) and that abiotic optima vary only weakly across space ($\sigma^2_{\theta_i} \sim O(\varepsilon)$). The latter implies $\sigma_{\theta_1\theta_2} \sim O(\varepsilon)$ as well. These assumptions will also be relaxed using the individual-based simulations described below.

Analysis of this simpler set of recursions reveals that the system evolves to an equilibrium or undergoes a runaway process in which at least some of the statistical moments evolve without bound (analyses performed in Mathematica 7.0; notebook available on request). Which outcome occurs depends on the type of interaction (mutualism, exploiter-victim, competition, or commensalism), whether the interaction is mediated by phenotypic matching or differences, and the relative strengths of abiotic and biotic selection. Although we do not explicitly model changes in population sizes over time, when a runaway process occurs, the population mean fitness of at least one of the species decreases over time, suggesting that the likely outcome is extinction of one or both species.

For interactions mediated by phenotypic differences, the covariance between the trait means of the interacting species always evolves to a stable equilibrium:

$$\hat{\sigma}_{\bar{z}_1\bar{z}_2} = 0 + O(\varepsilon^2). \quad (4)$$

Thus, interactions mediated by phenotypic differences and weak coevolutionary selection do not generate significant spatial associations between the mean phenotypes of two interacting species.

Unlike phenotypic differences, interactions mediated by phenotypic matching do not necessarily lead to an evolutionary equilibrium. Indeed, the statistical moments equilibrate only when

$$\frac{2R_{M_1}}{m_1 + 2R_{A_1}} + \frac{2R_{M_2}}{m_2 + 2R_{A_2}} > -1, \quad (5)$$

where $R_{M_i} = G_i s_{M_i}$ and $R_{A_i} = G_i\gamma_i$ (appendix). Biologically, inequality (5) shows that a stable equilibrium exists when gene flow is (relatively) high, abiotic selection is strong, and biotic interactions have intense effects on species that benefit (mutualist, parasite, predator) but weak effects on species that are harmed (competitors, hosts, prey). Consequently, a stable equilibrium will evolve for all mutualisms, for a subset of exploiter-victim interactions, and for a comparatively narrow range of competitive interactions.

When inequality (5) holds, the covariance between trait means equilibrates at

$$\hat{\sigma}_{\bar{z}_1\bar{z}_2} = \frac{2(G_1 s_{M_1}\hat{\sigma}^2_{\bar{z}_2} + G_2 s_{M_2}\hat{\sigma}^2_{\bar{z}_1})}{m_1 + m_2 + 2[G_1(s_{M_1} + \gamma_1) + G_2(s_{M_2} + \gamma_2)]} + O(\varepsilon^2), \quad (6)$$

where $\hat{\sigma}^2_{\bar{z}_i}$ is the equilibrium spatial variance of the trait mean in species i that depends on the population sizes of the interacting species n_i (appendix). Thus, interactions

mediated by phenotype matching, unlike those mediated by phenotypic differences, generate nonzero associations between the trait means of interacting species.

Numerical evaluations of equation (6), using equilibrium expressions for the variances (appendix), shed light on factors determining the sign and magnitude of the correlation. For mutualistic interactions, the correlation is always positive; its magnitude increases with the strength of mutualistic selection but decreases with that of abiotic selection (fig. 2, *upper right*). In contrast, competitive interactions lead to negative correlations whose magnitudes increase with the strength of competitive selection but decrease with abiotic selection (fig. 2, *lower left*). Finally, equilibrium correlations generated by antagonistic interactions at equilibrium vary in sign and size, depending on the relative strengths of biotic and abiotic selection (fig. 2, *upper left*, *lower right*). In all cases, parameter combinations exist where coevolutionary selection fails to generate statistically significant correlations (areas between red lines in fig. 2).

Although these results clearly demonstrate that coevolutionary selection can produce significant correlations between the traits of interacting species in some cases, they also reveal that coevolutionary selection per se is not required for substantial correlations to evolve. Specifically, the equilibrium (eq. [A6] in the appendix) shows that traits become correlated even if the fitness of only one species is affected by interactions (evolutionary commensalism). For instance, a host-parasite interaction in which all host phenotypes are equally likely to be infected can evolve

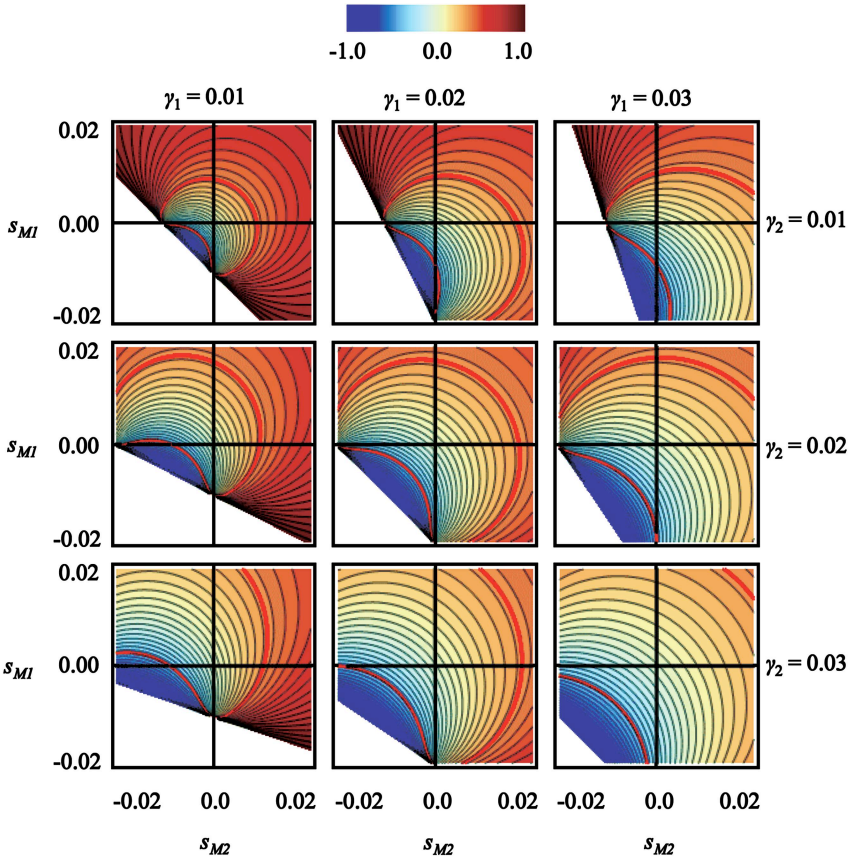

Figure 2: Contour plots of the equilibrium correlation coefficient shown for various strengths of abiotic selection as a function of the strength of biotic selection and $m_1 = m_2 = 0.001$ and $n_1 = n_2 = 1{,}000$ in all panels. Each panel shows the correlation as a function of the strength of biotic selection in species 1 and 2. The upper right-hand quadrant of each plot represents a mutualistic interaction, the lower left-hand quadrant a competitive interaction, and the remaining two quadrants antagonistic interactions, with species 1 the exploiter in the upper left-hand quadrant and species 2 the exploiter in the lower right-hand quadrant. Each of the nine plots corresponds to different strengths of stabilizing selection acting on the two species. White areas within the plots are areas where the equilibrium is locally unstable and a runaway process ensues. Red lines indicate the threshold magnitude of the correlation that must be exceeded for statistical significance in an empirical study of 30 populations. Thus, only those correlations that lie outside of the area defined by the red lines would be deemed statistically significant.

strong correlations as long as parasite phenotypes differ in their infection success and are heritable (e.g., figs. 2–4, cases where $s_{M_1} = 0$ and $s_{M_2} > 0$). In addition, it is straightforward to show analytically that correlated abiotic environments, acting in the absence of biotic interactions, lead to equilibria where the traits of the interacting species are correlated (results not shown). Consequently, as Janzen (1980) argued, a correlation need not imply a reciprocal coevolutionary process.

Previous theoretical studies have found that gene flow and local population size can have important effects on coevolution under some circumstances (Gandon et al. 1996; Gandon and Michalakis 2002; Gandon and Nuismer 2009) but not others (Ridenhour and Nuismer 2007; Gavrilets and Michalakis 2008). We investigated how rates of gene flow and local population sizes influence the correlation between traits, using our equilibrium equations. For the scenarios considered here, gene flow has only a relatively weak impact on the trait correlation between species (fig. 3). However, asymmetry in local population sizes n_i can have conspicuous effects on the magnitude of the correlation in mutualistic and competitive interactions and can even alter the sign of the correlation in antagonistic interactions (fig. 4). The strong effect of relative population sizes arises in antagonistic interactions because when the exploiter has a larger population size than the victim, selection is relatively more effective in the exploiter than in the victim (all else being equal). Consequently, the victim cannot evolve away from the exploiter as rapidly as the exploiter can respond. Consequently, exploiters closely track the evolution of their victims, generating a significant correlation.

Individual-Based Simulations

Our quantitative genetic approximation provides insight into the conditions where correlations evolve but makes four important assumptions. First, the approximation assumes fixed additive genetic variances, an assumption unlikely to hold in most natural systems. This assumption is

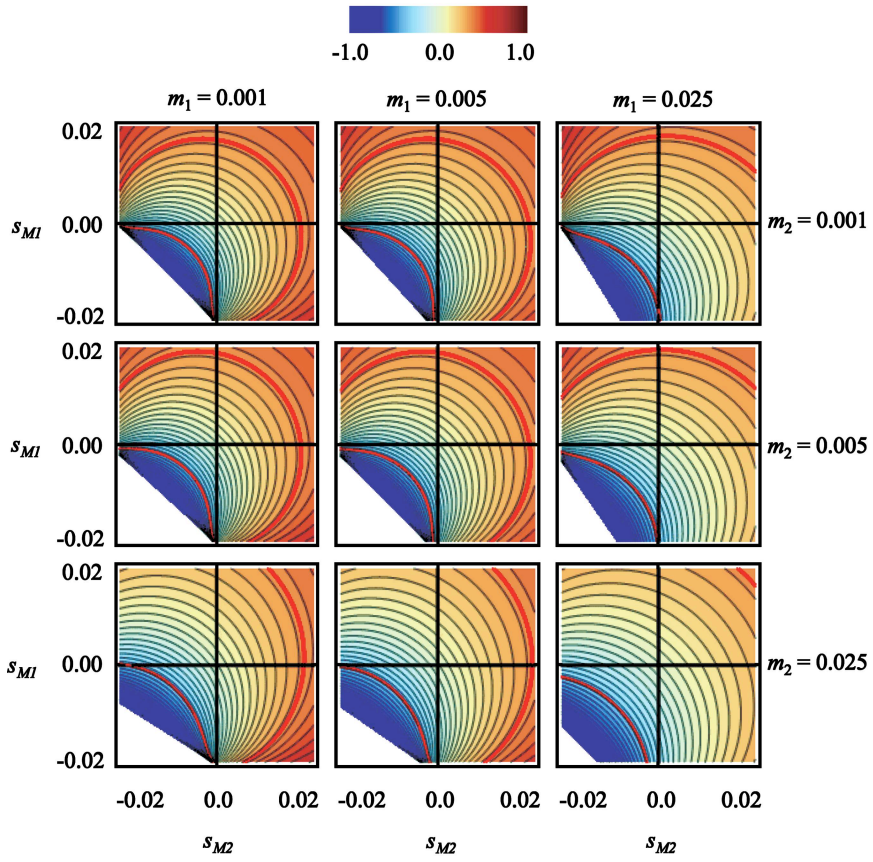

Figure 3: Contour plots of the equilibrium correlation coefficient shown for various rates of gene flow as a function of the strength of biotic selection and $\gamma_1 = \gamma_2 = 0.02$ and $n_1 = n_2 = 1{,}000$. Additional details as in figure 2.

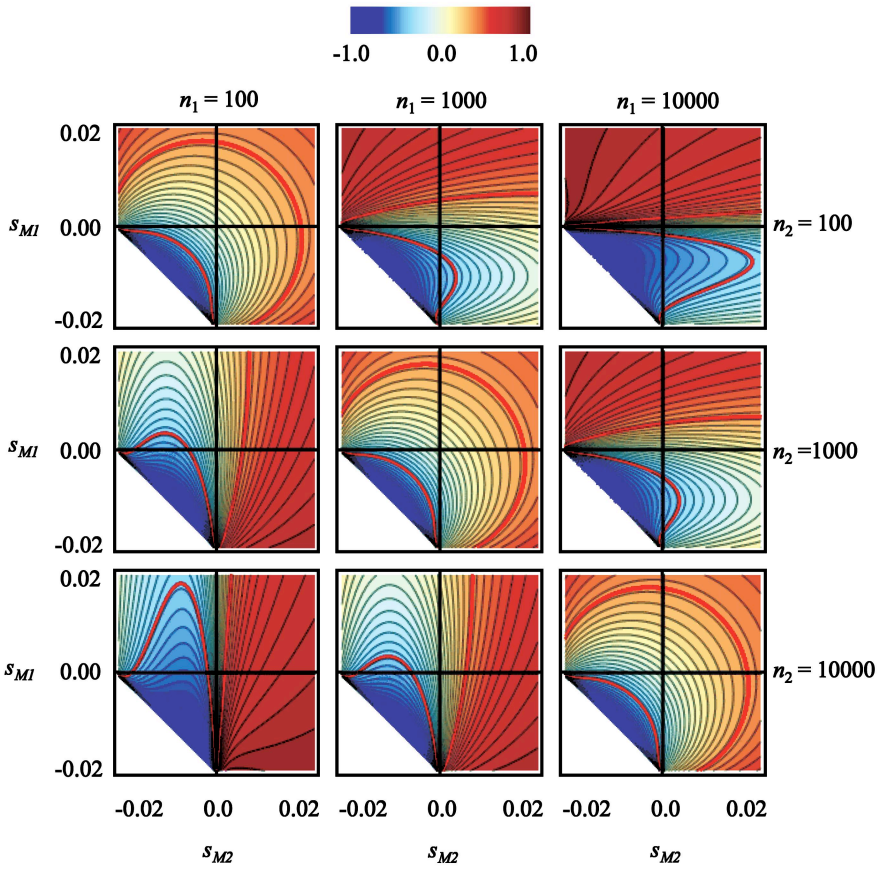

Figure 4: Contour plots of the equilibrium correlation coefficient shown for various local population sizes as a function of the strength of biotic selection and $\gamma_1 = \gamma_2 = 0.02$ and $m_1 = m_2 = 0.001$. Additional details as in figure 2.

particularly relevant in the context of coevolution because previous work has demonstrated that allowing additive genetic variance to evolve changes the dynamics of coevolution (Nuismer et al. 2005; Kopp and Gavrilets 2006). Second, the approximation assumes weak selection, meaning that the phenotypes of the interacting species have only small effects on individual fitness. This assumption may also be unlikely to hold in all cases and can have important consequences for the model of phenotypic differences considered here (Nuismer et al. 2007). Third, the approximation assumes that the optimal phenotypes favored by stabilizing selection are weakly variable across space. Finally, our approximation assumes that the rate of gene flow among populations is low. To evaluate whether our analytical predictions are qualitatively robust to violations of these assumptions, we developed and analyzed individual-based simulations.

Our simulations tracked the evolution of interacting populations composed of hermaphroditic individuals of each species distributed across 30 habitats. Individuals were completely characterized by their phenotype, z. Each simulation began by creating n_i individuals of each species within each habitat i, where the phenotypes of these individuals were drawn at random from a uniform distribution on $\{\theta_i \pm 0.1\}$. Drawing initial phenotypes in this way assumes that populations are initially at least moderately well adapted to their abiotic environment. After initialization, simulations tracked the fate of individuals over a life cycle in which individuals (1) moved among habitats, (2) mated and reproduced, (3) experienced abiotic selection, and (4) experienced biotic selection. At the end of each generation, the phenotypic means for each population were recorded.

Movement among habitats occurred following an island model where a fixed proportion of individuals of species i, m_i, entered a global migrant pool in each generation. Individuals from this global migrant pool were then assigned to the different habitats at random until the population of species i in each habitat reached its size before creation of the migrant pool. Consequently, movement of

individuals had the desired effect of altering gene frequencies but not local population sizes.

After migration, parental pairs were chosen randomly (with replacement) from the local population to found a new population of size n_i. Each breeding pair produced a single offspring whose phenotype was generated by adding a zero-mean, normally distributed random variable with segregational variance σ^2_{seg} to the average phenotype of the two parents; σ^2_{seg} is assumed to be constant across generations. This approach allows additive genetic variance to evolve (because the genetic variance of the parental population changes in response to selection, drift, and gene flow) and accommodates arbitrary phenotype distributions while saving large amounts of computational time by ignoring explicit multilocus genetics. Nevertheless, this approach does assume that the segregational variance remains constant, an assumption likely to hold over long periods of evolutionary time only when selection is sufficiently weak for mutation to replenish additive genetic variance eroded by the action of selection. We set $\sigma^2_{seg} = 0.1$ in all simulations.

Abiotic selection occurred after mating, with each individual surviving with a probability determined by the abiotic portion of equation (1). The parameter γ_i, which measures the strength of stabilizing selection acting on species i in all populations, was drawn from a uniform distribution on {0.005, 1.0}. The strength of stabilizing selection was thus temporally and spatially homogenous. In contrast, pairs of abiotic phenotypic optima were drawn for each habitat from a bivariate normal distribution and were thus spatially variable. The two means of this bivariate normal distribution were drawn independently at the start of each simulation from a uniform distribution on {0.4, 0.6}, the two variances were drawn from a uniform distribution on {0.0, 0.1}, and the correlation was drawn from a uniform distribution on {−0.1, 0.1}.

Finally, biotic selection was implemented by drawing a pair of individuals, one from each species, at random without replacement; the chosen individuals survived the encounter with probabilities given by equations (2). This was repeated until all individuals of the species with the smaller population size had interacted; individuals of the species with the larger population size who did not interact survived if this species was a competitor, victim, or host of a commensal but died if this species was an exploiter, mutualist, or commensal. If species interactions were mediated by phenotype matching, the parameter ξ_i was drawn from a uniform distribution on {0, 0.5} if a species benefitted from interacting; however, ξ_i was drawn from a uniform distribution on {−0.5, 0} if a species was harmed by interacting. For the case of interactions mediated by phenotype differences, the parameter ξ_i was drawn from a uniform distribution on {0, 0.5} for both beneficial and harmful interactions. In all cases, the parameter ξ_i was spatially and temporally fixed. The entire life cycle was repeated for 1,000 generations, at which point the correlation between the trait means of the interacting species was calculated and recorded.

We ran 1,000 simulations for antagonistic, mutualistic, competitive, and commensalistic interactions mediated by either phenotypic matching or phenotypic differences. In addition, we ran 1,000 simulations for interactions lacking biotic selection to generate a null distribution for the correlation expected in the absence of reciprocal interactions between species. Thus, in total, we ran 9,000 simulations. At the beginning of every simulation run, the local population size was drawn at random from a uniform distribution on {300, 2,000} for each species, migration rates were drawn from a uniform distribution on {0, 0.1}, and the parameter K_i was fixed at 1/2. These parameters were identical across all habitats. Source C code and simulation data are available on request.

We compared simulated distributions of correlation coefficients that evolved under different ecological scenarios and mechanisms of interaction (fig. 5). This comparison revealed several important results. First, contrary to our analytical predictions, interactions mediated by phenotypic differences can yield substantial correlations when reciprocal selection is strong (fig. 5). Nevertheless, interactions mediated by phenotypic differences tended to support weaker correlations than phenotypic matching (fig. 5), in accord with our analytic results. In fact, the average magnitude ($\pm$SE) of the correlation for phenotypic matching is 0.563 ± 0.005 but only 0.316 ± 0.005 for phenotypic differences. More important, from an empirical standpoint, only 32.1% of the correlation coefficients observed for phenotypic differences reach statistical significance at the $P = .05$ level (based on sampling 30 sites), whereas 69.7% would be statistically significant for phenotypic matching. Second, trait matching tends, as predicted, to generate positive correlations in mutualisms and commensalisms, negative correlations in competitive interactions, and both positive and negative correlations in antagonistic interactions (fig. 5).

Our results provide a sobering view of what can be inferred from even precise estimates of trait correlations. Two findings are of particular relevance. First, the simulations demonstrate that correlations between species readily evolve in the absence of reciprocal biotic selection. This can occur if interactions cause selection on only one of the species (fig. 5, *bottom*) or if the trait optima favored by stabilizing selection on the two species are strongly correlated (results not shown). Second, the simulations show that reciprocal selection often fails to generate statistically significant correlations between trait means even when it occurs. For phenotypic differences, 67.9% of sim-

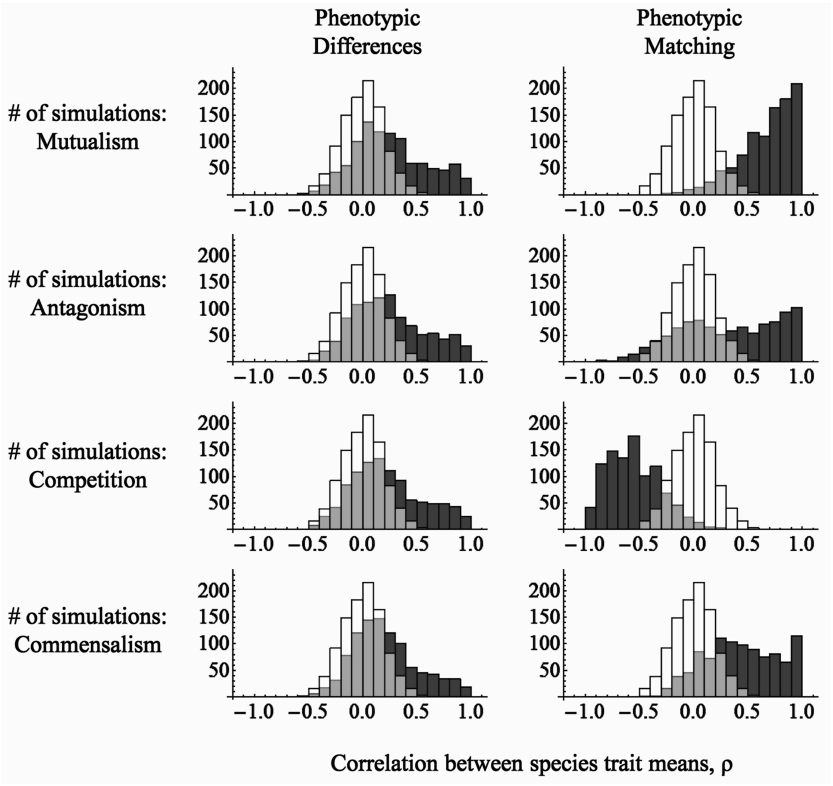

Figure 5: Distribution of correlation coefficients generated by biotic interactions (*filled bars*) compared to the distribution of correlation coefficients generated by drift, abiotic selection, and gene flow alone (*open bars*) for four forms of ecological interaction. *Left*, distribution for interactions mediated by phenotypic differences; *right*, distribution for interactions mediated by phenotypic matching.

ulations failed to yield statistically significant correlations even though the species were experiencing coevolution in every simulation run. Thus, as Thompson (1994, 2005) has pointed out, an absence of trait matching is not evidence for the absence of coevolutionary selection.

Next, we used our simulation results to graphically explore how model parameters influence the sign and magnitude of interspecific correlations between the trait means of the interacting species. For this, we used least squares to fit a quadratic model for the correlation. Specifically, we plotted the least squares regression fit for a quadratic model of the general form $\rho = b + c_1X_1 + c_2X_2 + c_3X_1X_2 + c_4X_1^2 + c_5X_2^2$ to the simulated data, where the variables X_i represent the fitness consequences of biotic interactions (ξ_i), the strengths of abiotic selection (γ_i), population sizes (n_i), or rates of gene flow (m_i).

The least squares fits suggest that local population size and gene flow had only very weak effects on the magnitude of the correlation. In contrast, our least squares fits showed that abiotic and biotic selection have very strong effects on the correlation. For interactions mediated by phenotypic differences, increasing the influence of biotic interactions on fitness increases correlations, whereas strengthening abiotic selection decreases correlations (figs. A1, A2, *left*, in the appendix). For phenotypic matching, the correlation is increased by weakening abiotic selection but increasing the strength of biotic selection acting on the mutualist or enemy species. In contrast, the correlation is decreased by reducing the strength of abiotic selection and strengthening biotic selection acting on the competitor or victim species (figs. A1, A2, *right*).

Reconciling Analytical and Simulation Results

The results of our analytical approximation and individual-based simulations differ in two important respects. First, our analytical model predicts that coevolutionary interactions mediated by phenotypic differences do not generate correlations, whereas our simulations suggest that under some circumstances, they can. This discrepancy arises primarily because our analytical approximations assume that the fitness consequences of species interactions depend only weakly on the phenotypes of the interacting individuals. When this assumption is violated—as it is in

the simulations—our Taylor series approximation for the phenotypic differences model breaks down because the changes in mean phenotypes due to species interactions are no longer independent of the means themselves (for additional discussion, see Nuismer et al. 2007). Thus, whether interactions mediated by phenotypic differences cause interspecific correlations to evolve depends on the extent to which the relationship between the traits of interacting individuals and fitness is linear.

The second important difference between our analytical and simulation results is the predicted impact of local population sizes on the correlation between traits of the interacting species. Specifically, our analytical model predicts that population sizes have a strong effect on the correlation, whereas our individual-based simulations suggest only a weak effect. As with the first discrepancy, this appears to be due to the assumption of weak selection made by our analytical model. Because our analytical model assumes that selection is weak, an interesting interplay between selection and drift develops for the population sizes that we have considered here. However, because our simulations consider much stronger selection than our analytical model but study an equivalent range of local population sizes, this interplay between drift and selection is lost. Additional simulations run with very small population sizes or very weak selection support this explanation (results not shown). Consequently, whether local population sizes influence the correlation observed in natural interactions depends on local population sizes and the strength of selection generated by species interactions. If local population sizes are small and/or selection is weak, population size should matter, as predicted by our analytical model; if instead local population sizes are very large and/or selection is very strong, local population sizes should be unimportant, as predicted by our simulations.

Discussion

Our analyses allow us to specify the conditions that must be met for coevolutionary selection to lead to significantly correlated traits in interacting species. Specifically, our results show that for correlations to be detected as statistically significant in studies of modest size (e.g., 30 populations), several conditions must be met. First, for interactions mediated by a mechanism of phenotype matching (e.g., similarity in color pattern of cuckoo and host eggs), reciprocal selection must be strong relative to stabilizing selection imposed by the abiotic environment. The condition for interactions mediated by phenotypic differences (e.g., concentration of toxic defensive compound in prey and detoxification ability in predator) is even more stringent, requiring that reciprocal selection be strong not only relative to abiotic selection but also in an absolute sense. Thus, as Thompson (1994) has argued, an absence of correlated traits is not evidence for an absence of coevolution.

Results presented here also provide a formal justification for Janzen's (1980) verbal argument that matching traits between interacting species do not provide conclusive evidence for a coevolutionary process. Indeed, biotic selection that affects only one of the interacting species can itself cause trait matching. This can occur if interactions have potent fitness consequences for only one of the species (ecological commensalism) or if the outcome of interactions depends on the phenotype of only one of the species (evolutionary commensalism). For instance, studies of the interaction between two species of ducks and their egg-mimicking brood parasite have shown that parasitism has little fitness cost to the host but egg rejection imposes large costs on the parasite (Lyon and Eadie 2004). Our results show that these one-way interactions generate distributions of correlation coefficients virtually indistinguishable from those that evolve due to a coevolutionary process.

Another non-coevolutionary process that our analyses show readily leads to correlated traits between interacting species is stabilizing selection with correlated optimal phenotypes. Such a scenario is likely to arise if traits involved in interaction are influenced by similar abiotic variables. For instance, if a host plant requires a minimum threshold temperature to initiate flowering and a phytophagous insect requires a minimum threshold temperature to emerge, the optimal abiotic phenotypes of the two species are likely to be positively correlated across elevational or latitudinal gradients. The substantial environmental gradients observed in some studies that document large-magnitude correlations between traits of interacting species (e.g., Toju 2008) suggest that evidence for a reciprocal coevolutionary process is tentative in these systems.

In addition to confirming Janzen's (1980) verbal argument, our results provide new insight into the conditions that promote local adaptation for different types of interactions. Because local adaptation—as generally studied using fully reciprocal transplant or common-garden designs—measures the covariance between genotypes of interacting species (Nuismer and Gandon 2008), it should be possible to use our results to predict local adaptation. Specifically, positive correlations should indicate local adaptation for species that benefit through interacting (mutualists, parasites, etc.) but local maladaptation for species that are harmed by interacting (competitors, hosts, etc.). Because our results show that the sign of the correlation (and thus which species is locally adapted) does not change as a function of the relative rates of gene flow in the interacting species, our results support previous analyses of host-pathogen interactions mediated by quantitative traits (Ridenhour and Nuismer 2007) and previous pop-

ulation genetic models that have assumed infinite population sizes (Nuismer 2006; Gavrilets and Michalakis 2008) but conflict with those of some models where interactions are mediated by specific genetic mechanisms of infection/resistance (Gandon et al. 1996; Gandon 2002; Gandon and Michalakis 2002; Gandon and Nuismer 2009). This discrepancy may arise because genetic variation underlying infection/resistance is eroded less rapidly (or not at all) in quantitative genetic models and in population genetic models that assume infinite population sizes than in population genetic models with finite populations, creating less opportunity for gene flow to introduce novel favorable genotypes (Gandon and Nuismer 2009).

In contrast to the limited impact of relative rates of gene flow, our analytical results demonstrate that relative population sizes of host and pathogen populations can play a role in determining the identity of the locally adapted species (fig. 4). This result, which is also observed in some population genetic models (Gandon and Michalakis 2002; Gandon and Nuismer 2009), suggests that contrary to conventional wisdom, this effect is not driven by the impact of drift on levels of standing genetic variation because additive genetic variance is fixed in our analytic model. Instead, the effect appears to be driven by drift impeding adaptation in one of the interacting species more than the other. For instance, if parasite population size is very small relative to host population size such that adaptive evolution is countered by drift more in the parasite than in the host, the parasite cannot effectively evolve to match the phenotype of the host (fig. 4; *off-diagonal*). This effect operates even if standing genetic variation is constant and abundant.

Our results also bear on the increasingly popular use of trait correlations as a means of testing the geographic mosaic theory of coevolution (GMTC; Berenbaum and Zangerl 1998; Brodie et al. 2002; Zangerl and Berenbaum 2003; Craig et al. 2007; Hanifin and Brodie 2008). The GMTC predicts that coevolutionary selection will not always lead to strong correlations between traits of interacting species. Instead, the theory argues that the coevolutionary process—due to the action of drift and gene flow (among other forces)—actually leads to considerable "trait mismatching" that we formally interpret as imperfect correlations between traits of interacting species. This rather loose prediction has spurred a proliferation of studies that estimate the population mean trait values of interacting species across broad spatial scales and interpret the results (typically an imperfect correlation) as support for the GMTC (e.g., Berenbaum and Zangerl 1998; Zangerl and Berenbaum 2003; Toju and Sota 2006; Anderson and Johnson 2008; Hanifin and Brodie 2008). Although these studies have provided valuable data and yielded interesting insights, reported correlations neither support nor refute the GMTC because there is no a priori expectation for the value of the correlation expected in the absence of a geographic mosaic process. In other words, our results demonstrate that the distribution of correlation coefficients that arises through a geographic mosaic process (some combination of spatially variable coevolutionary selection, gene flow, and finite populations) cannot be distinguished from a distribution resulting from a nongeographic mosaic process (i.e., no spatially variable coevolutionary selection).

In summary, we have used a quantitative genetic model of reciprocal selection between species to show that the correlation between traits of interacting species—when studied in isolation—reveals little about the presence or absence of coevolution and cannot be used to support or refute a geographic mosaic process. Instead, our results suggest that conclusive support for coevolutionary or geographic mosaic processes requires direct estimates of coevolutionary selection in natural populations (e.g., Benkman et al. 2003; Brodie and Ridenhour 2003; Ridenhour 2005; Gomulkiewicz et al. 2007); manipulative studies of local adaptation (Hoeksema and Thompson 2007; Nuismer and Gandon 2008; Gandon and Nuismer 2009); or pluralistic approaches using novel statistics that combine information from phylogeographic studies, estimates of spatial variability in traits, and a mechanistic understanding of the traits mediating interactions (Thompson 2005).

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Literature Cited

Abrams, P. A. 2000. The evolution of predator-prey interactions: theory and evidence. Annual Review of Ecology and Systematics 31:79–105.

Albert, A. Y. K., N. P. Millar, and D. Schluter. 2007. Character displacement of male nuptial colour in threespine sticklebacks (*Gasterosteus aculeatus*). Biological Journal of the Linnean Society 91: 37–48.

Anderson, B., and S. D. Johnson. 2008. The geographical mosaic of coevolution in a plant-pollinator mutualism. Evolution 62:220–225.

Benkman, C. W. 1999. The selection mosaic and diversifying coevolution between crossbills and lodgepole pine. American Naturalist 153(suppl.):S75–S91.

Benkman, C. W., T. L. Parchman, A. Favis, and A. M. Siepielski. 2003. Reciprocal selection causes a coevolutionary arms race between crossbills and lodgepole pine. American Naturalist 162:182–194.

Berenbaum, M. R., and A. R. Zangerl. 1998. Chemical phenotype

matching between a plant and its insect herbivore. Proceedings of the National Academy of Sciences of the USA 95:13743–13748.
Bergelson, J., G. Dwyer, and J. J. Emerson. 2001. Models and data on plant-enemy coevolution. Annual Review of Genetics 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.
Brodie, E. D., III, and B. J. Ridenhour. 2003. Reciprocal selection at the phenotypic interface of coevolution. Integrative and Comparative Biology 43:408–418.
Brodie, E. D., Jr., B. J. Ridenhour, and E. D. Brodie III. 2002. The evolutionary response of predators to dangerous prey: hotspots and coldspots in the geographic mosaic of coevolution between garter snakes and newts. Evolution 56:2067–2082.
Carlson, R. L., P. C. Wainwright, and T. J. Near. 2009. Relationship between species co-occurrence and rate of morphological change in *Percina* darters (*Percidae*: Etheostomatinae). Evolution 63:767–778.
Craig, T. P., J. K. Itami, and J. D. Horner. 2007. Geographic variation in the evolution and coevolution of a tritrophic interaction. Evolution 61:1137–1152.
Darwin, C. 1909. The origin of species. Collier, New York.
Fellowes, M. D. E., A. R. Kraaijeveld, and H. C. J. Godfray. 1998. Trade-off associated with selection for increased ability to resist parasitoid attack in *Drosophila melanogaster*. Proceedings of the Royal Society B: Biological Sciences 265:1553–1558.
Gandon, S. 2002. Local adaptation and the geometry of host-parasite coevolution. Ecology Letters 5:246–256.
Gandon, S., and Y. Michalakis. 2002. Local adaptation, evolutionary potential and host-parasite coevolution: interactions between migration, mutation, population size and generation time. Journal of Evolutionary Biology 15:451–462.
Gandon, S., and S. L. Nuismer. 2009. Interactions between genetic drift, gene flow, and selection mosaics drive parasite local adaptation. American Naturalist 173:212–224.
Gandon, S., Y. Capowiez, Y. Dubois, Y. Michalakis, and I. Olivieri. 1996. Local adaptation and gene-for-gene coevolution in a metapopulation model. Proceedings of the Royal Society B: Biological Sciences 263:1003–1009.
Gavrilets, S. 1997. Coevolutionary chase in exploiter-victim systems with polygenic characters. Journal of Theoretical Biology 186:527–534.
Gavrilets, S., and Y. Michalakis. 2008. Effects of environmental heterogeneity on victim-exploiter coevolution. Evolution 62:3100–3116.
Gomulkiewicz, R., J. N. Thompson, R. D. Holt, S. L. Nuismer, and M. E. Hochberg. 2000. Hot spots, cold spots, and the geographic mosaic theory of coevolution. American Naturalist 156:156–174.
Gomulkiewicz, R., D. M. Drown, M. F. Dybdahl, W. Godsoe, S. L. Nuismer, K. M. Pepin, B. J. Ridenhour, C. I. Smith, and J. B. Yoder. 2007. Dos and don'ts of testing the geographic mosaic theory of coevolution. Heredity 98:249–258.
Grant, P. R., and B. R. Grant. 2006. Evolution of character displacement in Darwin's finches. Science 313:224–226.
Hanifin, C. T., E. D. Brodie Jr., and E. D. Brodie III. 2008. Phenotypic mismatches reveal escape from arms-race coevolution. PLoS Biology 6:471–482.
Hoeksema, J. D., and J. N. Thompson. 2007. Geographic structure in a widespread plant-mycorrhizal interaction: pines and false truffles. Journal of Evolutionary Biology 20:1148–1163.
Janzen, D. H. 1980. When is it coevolution? Evolution 34:611–612.
Kopp, M., and S. Gavrilets. 2006. Multilocus genetics and the coevolution of quantitative traits. Evolution 60:1321–1336.
Lahti, D. C. 2005. Evolution of bird eggs in the absence of cuckoo parasitism. Proceedings of the National Academy of Sciences of the USA 102:18057–18062.
Lande, R. 1976. Natural selection and random genetic drift in phenotypic evolution. Evolution 30:314–334.
Lyon, B. E., and J. M. Eadie. 2004. An obligate brood parasite trapped in the intraspecific arms race of its hosts. Nature 432:390–393.
Moen, D. S., and J. J. Wiens. 2009. Phylogenetic evidence for competitively driven divergence: body-size evolution in Caribbean treefrogs (Hylidae: *Osteopilus*). Evolution 63:195–214.
Moore, J. C., D. J. Obbard, C. Reuter, S. A. West, and J. M. Cook. 2008. Fighting strategies in two species of fig wasp. Animal Behaviour 76:315–322.
Muchhala, N., and J. D. Thomson. 2009. Going to great lengths: selection for long corolla tubes in an extremely specialized batflower mutualism. Proceedings of the Royal Society B: Biological Sciences 276:2147–2152.
Nash, D. R., T. D. Als, R. Maile, G. R. Jones, and J. J. Boomsma. 2008. A mosaic of chemical coevolution in a large blue butterfly. Science 319:88–90.
Nuismer, S. L. 2006. Parasite local adaptation in a geographic mosaic. Evolution 60:83–88.
Nuismer, S. L., and S. Gandon. 2008. Moving beyond common-garden and transplant designs: insight into the causes of local adaptation in species interactions. American Naturalist 171:658–668.
Nuismer, S. L., J. N. Thompson, and R. Gomulkiewicz. 1999. Gene flow and geographically structured coevolution. Proceedings of the Royal Society B: Biological Sciences 266:605–609.
———. 2000. Coevolutionary clines across selection mosaics. Evolution 54:1102–1115.
Nuismer, S. L., M. Doebeli, and D. Browning. 2005. The coevolutionary dynamics of antagonistic interactions mediated by quantitative traits with evolving variances. Evolution 59:2073–2082.
Nuismer, S. L., B. J. Ridenhour, and B. P. Oswald. 2007. Antagonistic coevolution mediated by phenotypic differences between quantitative traits. Evolution 61:1823–1834.
Ridenhour, B. J. 2005. Identification of selective sources: partitioning selection based on interactions. American Naturalist 166:12–25.
Ridenhour, B. J., and S. L. Nuismer. 2007. Polygenic traits and parasite local adaptation. Evolution 61:368–376.
Soler, J. J., J. M. Aviles, M. Soler, and A. P. Møller. 2003. Evolution of host egg mimicry in a brood parasite, the great spotted cuckoo. Biological Journal of the Linnean Society 79:551–563.
Steiner, K. E., and V. B. Whitehead. 1991. Oil flowers and oil bees: further evidence for pollinator adaptation. Evolution 45:1493–1501.
Strong, D. R., L. A. Szyska, and D. S. Simberloff. 1979. Tests of community-wide character displacement against null hypotheses. Evolution 33:897–913.
Thompson, J. N. 1994. The coevolutionary process. University of Chicago Press, Chicago.
———. 2005. The geographic mosaic of coevolution. University of Chicago Press, Chicago.
Toju, H. 2008. Fine-scale local adaptation of weevil mouthpart length and camellia pericarp thickness: altitudinal gradient of a putative arms race. Evolution 62:1086–1102.
Toju, H., and T. Sota. 2006. Imbalance of predator and prey armament: geographic clines in phenotypic interface and natural selection. American Naturalist 167:105–117.

Webster, J. P., and M. E. J. Woolhouse. 1999. Cost of resistance: relationship between reduced fertility and increased resistance in a snail-schistosome host-parasite system. Proceedings of the Royal Society B: Biological Sciences 266:391–396.

Wright, S. 1931. Evolution in Mendelian populations. Genetics 16: 97–159.

Zangerl, A. R., and M. R. Berenbaum. 2003. Phenotype matching in wild parsnip and parsnip webworms: causes and consequences. Evolution 57:806–815.

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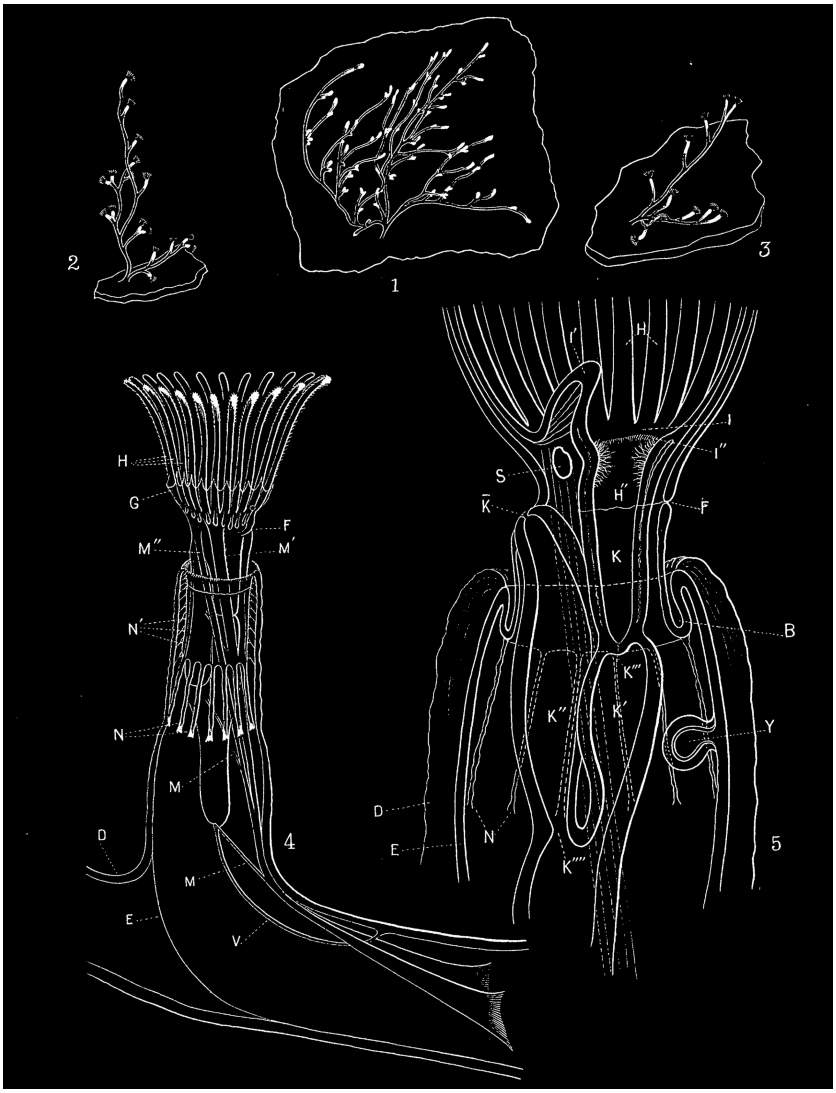

"*Fredericella regina* Leidy. Figs. 1, 2, 3. Colonies attached to pieces of bark. Fig. 4. Magnified view of one Polyzoön. D, brown envelope, the ectocyst; E, pellucid wall of the tube and cell, the endocyst; V, funiculus; M, M′, M″, upper branches of the muscles, the retractors; N, N′, muscles of the fold, the retentors; F, a small infolding of the endocyst, the brachial collar; G, the pointed ruffle, or calyx; H, the threads, or tentacles. Fig. 5. Outline of the interior part of a young specimen. Same letters as above, with the exception of B, invaginated fold of the tube; Y, a very young polyzoön, a bud; K, the throat or œsophagus; H″, cilia surrounding the mouth; K‴, the valve opening into the stomach, œsophageal valve; K′, stomach; K⁗, intestinal valve partly open; K″, intestine; $\overline{K}$, opening of intestine, the anus; I, disc, the lophomore; I′, the little flap, the epistome; I″, the mouth; S, nerve-mass." From "The Moss-Animals, or Fresh Water Polyzoa" by Alpheus Hyatt (*American Naturalist*, 1867, 1:57–64).