Evolution, 60(11), 2006, pp. 2207–2217

COEVOLUTIONARY ALTERNATION IN ANTAGONISTIC INTERACTIONS

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Abstract.—Coevolution between parasites and hosts or predators and prey often involves multiple species with similar kinds of defenses and counter-defenses. Classic examples include the interactions between phytophagous insects and their host plants, thick-shelled invertebrates and their shell-crushing predators, and ungulates and their predators. There are three major hypotheses for the nonequilibrium coevolutionary dynamics of these multispecific trophic interactions: escalation in traits, cycles in traits leading to fluctuating polymorphisms, and coevolutionary alternation. The conditions under which cycles and escalation are likely to occur have been well developed theoretically. In contrast, the conditions favoring coevolutionary alternation—evolutionary fluctuations in predator or prey preference driven by evolutionary shifts in relative levels of prey defense and vice versa—have yet to be identified. Using a set of quantitative coevolutionary models, we demonstrate that coevolutionary alternation can occur across a wide range of biologically plausible conditions. The result is often repeated, and potentially rapid, evolutionary shifts in patterns of specialization within networks of interacting species.

Key words.—Coevolution, community genetics, host switching, multispecific coevolution, specialization.

Received February 22, 2006. Accepted August 10, 2006.

Coevolutionary biology is increasingly confronting the problem of how networks, rather than simply pairs, of species coevolve within communities (Steinberg et al. 1995; Matsuda et al. 1996; Juenger and Bergelson 1998; Agrawal 2003; Gomulkiewicz et al. 2003; Losos et al. 2003; Neuhauser et al. 2003; Wade 2003; Nuismer and Doebeli 2004; Strauss et al. 2005; Thompson 2005; Bascompte et al. 2006). There are now multiple hypotheses for the coevolutionary dynamics that characterize groups of competitors and mutualists (see review in Thompson 2005), but coevolution within antagonistic food webs has been difficult to evaluate, because current coevolutionary hypotheses predict very different dynamics between even just pairs of interacting species. Hypotheses of parasite-host or predator-prey coevolution often assume that these interactions will escalate in defenses and counter-defenses; for example, selection imposed by predators for faster prey may in turn favor selection for faster or stealthier predators (Dawkins and Krebs 1979; but see Abrams 1986). Alternatively, some hypotheses suggest that frequency-dependent selection may lead to cycles in defenses and counter-defenses by favoring rare genotypes in host populations (Seger 1988; Dieckmann et al. 1995; Abrams and Matsuda 1997; Gavrilets 1997; Thrall and Burdon 2002; Borghans et al. 2004; Nuismer et al. 2005), as has been observed in coevolving snails and trematodes (Dybdahl and Lively 1998; Lively and Dybdahl 2000).

The addition of more species to the interaction creates yet other coevolutionary possibilities, one of which is a process first suggested by Davies and Brooke (1989) and now called coevolutionary alternation (Thompson 1994, 2005; Soler et al. 2003). In this form of multispecific coevolution, natural selection favors individual parasites or predators that prefer the currently least defended host or prey species. That, in turn, imposes selection on the preferred host or prey species for increased levels of defense, while favoring decreased defenses in less frequently attacked host or prey species. Coevolutionary alternation proceeds through repeated cycles of evolutionary change in parasite or predator preference and relative levels of host or prey defense.

Coevolutionary escalation, coevolutionary cycles leading to fluctuating polymorphisms, and coevolutionary alternation are alternative hypotheses for the nonequilibrium coevolutionary dynamics of traits within networks of interacting parasites and hosts or predators and prey that share similar defenses and counter-defenses, and all have some empirical support (Thompson 2005). Although a substantial body of mathematical theory has been developed to identify the conditions that lead to escalation or cycles (Abrams 1986; Dieckmann et al. 1995; Abrams and Matsuda 1997; Gavrilets 1997; Hochberg and van Baalen 1998; Sasaki 2000; Nuismer and Kirkpatrick 2003), little theoretical work has been devoted to elucidating the conditions that favor coevolutionary alternation. Kawecki's (1998) analysis of a genetic model for how a population of generalist parasites may evolve to be more specialized is perhaps the closest to the model evaluated here, because coevolutionary alternation will necessarily involve time periods in which specialist genotypes are favored over generalist genotypes. That analysis, however, did not attempt to quantify when coevolutionary alternation is possible, but instead focused on the conditions that lead to specialization on a single host species.

At a minimum, coevolutionary alternation requires genetic variation within and among host or prey species for defenses and genetic variation in parasites or predators in their preferences among host or prey species. Substantial variation in defense against enemies has been demonstrated repeatedly in plants and animals (Davies and Brooke 1989; Linhart and Grant 1996; Brodie et al. 2002; Decaestecker et al. 2003; Kliebenstein et al. 2005; Toju and Sota 2006). Moreover, selection experiments have shown that defenses within populations can evolve quickly in the presence or absence of selective predation or parasitism (e.g., Reznick et al. 1997; Fellowes et al. 1999). Additional correlative evidence of rapid evolution of defenses has been found in introduced species,

TABLE 1. Description of model variables and parameters.

Variable/Parameter	Description
z_P	preference phenotype of a parasite individual
z_A	attack phenotype of a parasite individual
$z_{R,i}$	resistance phenotype of a host individual of species i
ω	sensitivity of parasite host preference to parasite preference phenotype
α_i	sensitivity of the probability of successful parasite attack to parasite attack phenotype and the host resistance phenotype of host species i
$\gamma_{H,i}$	strength of stabilizing selection acting on host species i
γ_P	strength of stabilizing selection acting on the parasite attack trait
$\theta_{H,i}$	phenotypic optima favored by stabilizing selection acting on host species i
θ_P	phenotypic optima favored by stabilizing selection acting on the parasite attack trait
f_i	frequency of host species i
κ_i	fitness increment gained by the parasite for successfully attacking host species i
π_i	probability of using host species i; equal to P_P for $i = 1$ and $(1 - P_P)$ for $i = 2$
$\phi_{z_{R,i}}$	frequency of host resistance phenotype $z_{R,i}$ in species i
$\phi_{z_A z_P}$	frequency of parasite phenotype vector $\{z_A, z_P\}$
$N_{R,i}$	set of all resistance phenotypes in host species i
N_A	set of all attack phenotypes in the parasite
N_P	set of all preference phenotypes in the parasite
r_P	recombination rate in the parasite
$r_{R,i}$	recombination rate in host i
n_i	number of loci that determine the phenotype of parasite trait i
$n_{R,i}$	number of loci that determine the phenotype of host i
b_i	phenotypic effect of locus i
X_i	indicator variable that takes value zero if an individual carries a 0 allele at locus i or value one if an individual carries a 1 allele at locus i

in which some populations lacking their former enemies have been shown to have lower defenses than their ancestral populations in the native range of the species (e.g., Lahti 2006) and some populations reassociated with a former enemy exhibit subsequent rapid increases in levels of defense (Zangerl and Berenbaum 2005). These results imply that there is a cost of defense, at least under some environmental conditions (Siemens et al. 2002; Marak et al. 2003; Tian et al. 2003), that favors individuals whose level of defense best matches the overall current risk of attack.

Some of the genetic conditions required for coevolutionary alternation have also been found in predators and parasites that actively search for their victims. Multiple studies have demonstrated genetic variation in innate preference hierarchies among prey or hosts within and among natural populations. Examples include species as different as garter snakes that differ among populations in their tendency to avoid slugs (Arnold 1981) and insects that differ within and among populations in their relative preferences for host-plant species (Thompson 1988; Janz 2003; Singer 2003; Nylin et al. 2005). In addition to these genetically determined preference hierarchies, natural selection could also favor individuals that optimally switch among prey or choice of hosts during their lifetimes as the victim species vary locally in abundance or availability, with selection favoring different decision rules for switching as prey species evolve defenses (Matsuda 1985; Abrams and Matsuda 1993; Abrams 1999; Yamauchi and Yamamura 2005).

Here we explore the conditions favoring coevolutionary alternation within local populations. We focus on predators or parasites that exhibit genetically based preference hierarchies for prey or host species. We model coevolution of a predator or parasite species and two prey or host species, because this is most basic module of a multispecific network. Our model structure is similar to that developed by Kawecki (1998) but focuses on host or prey resistance determined by functional relationships between quantitative traits rather than by explicit gene-for-gene or matching alleles mechanisms. We analyze this genetic model using a combination of analytical and numerical techniques.

THE GENERAL MODEL

We modeled coevolution between a parasite species and two host species or between a predator species and two prey species. For simplicity, we use the words ''parasite'' and ''host'' throughout our description, but the same model structure could apply to coevolution between actively searching predators and prey. Each species is assumed to mate at random, encounter individuals of interacting species at random, and have a population size sufficiently large for the effects of genetic drift to be negligible. In addition, we assume that the population sizes of the interacting species are fixed. The biological interpretation of model parameters and variables is summarized in Table 1.

Our modeling framework allows the parasite to evolve to use less well defended host species rather than remain perpetually locked in a coevolutionary race with a single host species. Specifically, the models assume that parasites are genetically variable for a trait, z_P, that affects host preference, such that individual parasites with a trait value greater than 1/2 prefer host species 1, whereas individual parasites with a trait value less than 1/2 prefer host species 2 (Fig. 1A). We modeled this scenario by assuming that a parasite uses host species 1 with probability:

$$P_P = 1/\{1 + \exp[\omega(1/2 - z_P)]\}, \quad (1)$$

where ω measures the sensitivity of parasite preference to

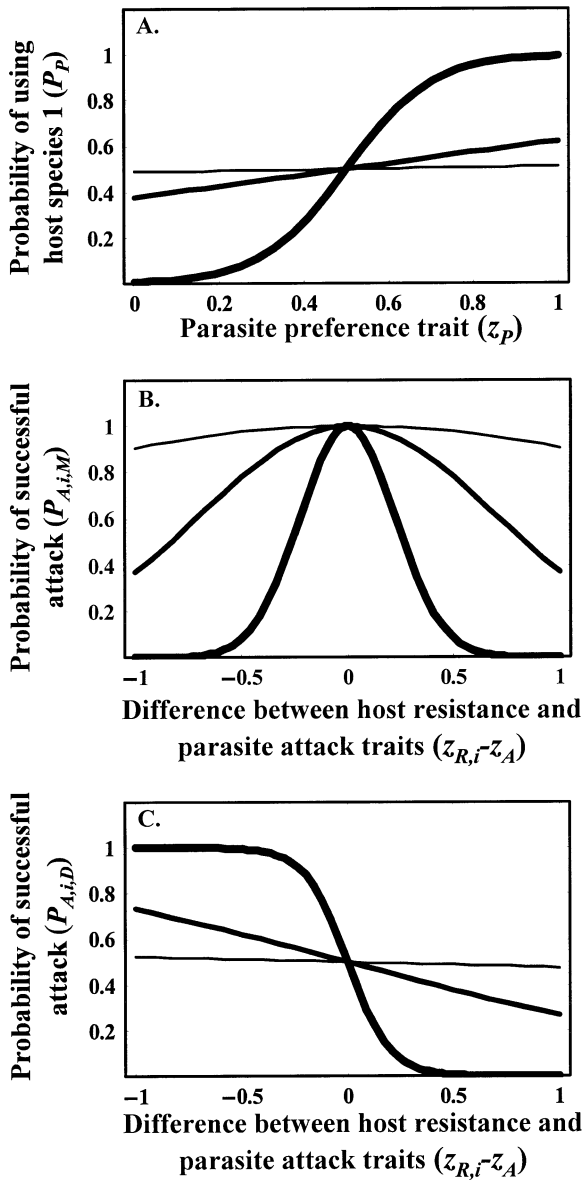

FIG. 1. The functional relationship between (A) parasite choice phenotype z_P and the strength of parasite preference P_P for three different values of ω (thickest line, $\omega = 10$; mid-thickness line, $\omega = 1$; thinnest line, $\omega = 0.1$); (B) parasite attack phenotype z_A, host resistance phenotype $z_{R,i}$, and the probability of successful parasite attack $P_{A,i,M}$ for the model of phenotype matching and three difference values of α (thickest line, $\alpha = 10$; mid-thickness line, $\alpha = 1$; thinnest line, $\alpha = 0.1$); (C) parasite attack phenotype z_A, host resistance phenotype $z_{R,i}$, and the probability of successful parasite attack $P_{A,i,D}$ for the model of phenotype distance and three difference values of α (thickest line, $\alpha = 10$; mid-thickness line, $\alpha = 1$; thinnest line, $\alpha = 0.1$).

changes in the parasite preference trait z_P. When a random encounter occurs between a parasite and a host species that it prefers, the outcome depends on the resistance phenotype of the host species and an additional parasite attack trait.

Two types of model were developed to contrast different ways in which a host resistance trait and parasite attack trait could possibly interact: phenotype matching and phenotype difference. In the model of phenotype matching, the probability of successful parasite attack increases with the degree to which phenotypes match between the parasite attack trait (z_A) and the resistance traits ($z_{R,1}$, $z_{R,2}$) of the two host species (Fig. 1B). We modeled this scenario by assuming that the probability of successful parasite attack is:

$$P_{A,i,M} = \exp[-\alpha_i(z_{R,i} - z_A)^2], \tag{2}$$

where α_i measures how sensitive the probability of successful parasite attack is to parasite and host phenotypes and the subscript i indicates host species 1 or 2. Matching has been shown to be important in coevolving interactions among brood parasites and their hosts (Soler et al. 2003), and it readily leads to coevolutionary cycles and fluctuating polymorphisms (Gavrilets 1997; Nuismer et al. 2005). In contrast, for the model of phenotype difference, the probability of successful parasite attack increases as parasite attack phenotype (z_A) increases relative to the resistance traits ($z_{R,1}$, $z_{R,2}$) of the two host species (Fig. 1C). We modeled this scenario by assuming that the probability of successful parasite attack is:

$$P_{A,i,D} = 1/\{1 + \exp[\alpha_i(z_{R,i} - z_A)]\}, \tag{3}$$

where α_i measures how sensitive the probability of successful parasite attack is to parasite and host phenotypes and the subscript i indicates host species 1 or 2. Success driven by phenotype differences in one or a few traits has been shown to be important in interactions between parasites and toxic host plants and between seed parasites and host plants (Bergelson et al. 2001; Toju and Sota 2006) and can lead to coevolutionary escalation.

Because traits that mediate species interactions are also likely to be subject to constraints imposed by physiology or the abiotic environment, we allow stabilizing selection to act on the resistance/attack traits ($z_{R,1}$, $z_{R,2}$, z_A) of the interacting species. Specifically, we assume that the fitness of an individual is determined by the product of its fitness with respect to the abiotic environment and its fitness with respect to interspecific interactions. Together, these assumptions allow us to calculate the expected fitness of each species. For host species i individuals with phenotype $z_{R,i}$ the expected fitness is:

$$E[W_{H,i}(z_{R,i})] = \exp[-\gamma_{H,i}(z_{R,i} - \theta_{H,i})^2] \times \left(1 - \sum_{z_A \in N_A} \sum_{z_P \in N_P} \pi_i P_{A,i,j} \phi_{z_A z_P}\right), \tag{4}$$

where π_i is equal to P_P when $i = 1$ and $(1 - P_P)$ when $i = 2$, the subscript j indicates whether the probability of successful parasite attack is mediated by the model of phenotype matching (M) or the model of phenotype difference (D). The first term in equation (4) accounts for stabilizing selection of strength $\gamma_{H,i}$ acting on the host resistance trait in species i, such that individuals with resistance phenotypes further from the static optimum value, $\theta_{H,i}$, suffer reduced fitness. The second term in equation (4) accounts for the fitness consequences of interactions with the parasite and is composed of two summations. The first sum is carried over the set of all attack phenotypes in the parasite population (N_A) and incorporates the probability ($P_{A,i,j}$) that an individual parasite

with attack phenotype (z_A) successfully attacks a host individual of species i with resistance phenotype ($z_{R,i}$). The second sum is carried over the set of all preference phenotypes in the parasite population (N_P), and incorporates the probability (π_i) that an individual parasite with preference phenotype (z_P) attempts to attack a host individual of species i. Each element of this double summation is weighted by the frequency ($\phi_{z_A z_P}$) of the parasite phenotype vector $\{z_A, z_P\}$.

Similarly, the expected fitness of a parasite individual with phenotype vector $\{z_A, z_P\}$ is given by:

$$E[W_P(z_A, z_P)] = \exp[-\gamma_P(z_A - \theta_P)^2] \times \sum_{i=1}^{2}\left(f_i\kappa_i\pi_i \sum_{z_{R,i}\in N_{R,i}} P_{A,i,j}\phi_{z_{R,i}}\right), \quad (5)$$

where subscript j indicates whether coevolution is mediated by the model of phenotype matching (M) or the model of phenotype difference (D). The first term in equation (5) accounts for stabilizing selection of strength γ_P acting on the parasite attack trait, such that individuals with attack phenotypes that lie further from the static optimum value, θ_P, suffer reduced fitness. The first sum in equation (5) is taken over the two potential host species $\{i = 1, 2\}$ and accounts for the frequency of host species i (f_i), the fitness benefit received by the parasite for successfully attacking host species i (κ_i), and the probability that the parasite attempts to attack an individual of host species i (π_i). The second sum in equation (5) is taken over the set of all resistance phenotypes in host species i ($N_{R,i}$), and incorporates the probability that the parasite is successful in attacking an individual of host species i with resistance phenotype $z_{R,i}$ ($P_{A,i,j}$), and the frequency of host phenotype $z_{R,i}$ in the population of host species i($\phi_{z_{R,i}}$).

Analytical Approximation

We developed mathematically tractable approximations to coevolutionary dynamics by initially assuming that the phenotype of each trait was determined by a single diallelic haploid locus. In addition, we assumed that the probability of successful parasite attack varied only weakly with parasite and host phenotype, stabilizing selection was weak, and recombination was frequent in the parasite. These assumptions allowed us to use a quasi-linkage equilibrium approximation (e.g., Barton and Turelli 1991; Nagylaki 1993; Kirkpatrick et al. 2002; Otto and Nuismer 2004), which identified conditions under which coevolutionary alternation—characterized by evolutionary fluctuations in parasite preference—is possible (see Supplementary Material, available online at: http://dx.doi.org/10.1554/06-111.1.s1). A Mathematica notebook that derives the quasi-linkage equilibrium approximation is also available upon request.

The key condition that must be met for coevolutionary alternation to occur is that the effect of host resistance phenotypes on the probability of successful parasite attack must be strong relative to differences in host species frequencies and qualities as food items. Specifically, results derived in the supplementary online material show that for the model of phenotype matching a necessary but not sufficient condition for coevolutionary alternation to occur is:

$$|f_1\kappa_1 - f_2\kappa_2| < \bar{\alpha}/2 \quad (6a)$$

and for the model of phenotype differences the same condition is:

$$|f_1\kappa_1 - f_2\kappa_2| < \bar{\alpha}/4, \quad (6b)$$

where $\bar{\alpha} = (\alpha_1 + \alpha_2)/2$ measures how sensitive parasite attack success is, on average, to host resistance traits (see Supplementary Material eqs. A17 and A19, available online). Intuitively, this result is obtained because if both host species are equally resistant, those parasite individuals that prefer the more common or better quality host species will be favored, leading to the evolution of specialization on a single host. Thus, only when it is possible for the two host species to evolve levels of parasite resistance that are sufficiently different to outweigh differences in host frequency and host quality can coevolutionary alternation possibly occur. Results (6a,b) also demonstrate that coevolutionary alternation is possible across a wider range of host species frequencies and qualities in the phenotype matching model than in the phenotype difference model.

In addition to identifying conditions under which coevolutionary alternation can occur, our analysis demonstrates important differences between the phenotype matching and phenotype difference models in the magnitude of the genetic correlation between parasite preference and attack traits maintained by coevolution. To the order of our approximation, the phenotype difference model showed an absence of a genetic correlation between parasite preference and attack traits, whereas the matching model produced a genetic correlation that could fluctuate in sign (see Supplementary Material, available online). The genetic correlation that emerges in the parasite for the matching model is driven by differences in the resistance levels of the two host species (δ_R) and is equal to:

$$\rho = \frac{\bar{\alpha}\omega\delta_R\sqrt{p_P(1 - p_P)}\sqrt{p_A(1 - p_A)}(1 - r_P)}{2r_P}, \quad (7)$$

where r_P is the rate of recombination between parasite preference and attack loci, and p_P and p_A are the allele frequencies at the parasite preference and attack loci respectively.

Equation (7) shows that if the relative resistance of the two host species (δ_R) fluctuates over time, so too will the sign of the genetic correlation. For instance, if during some periods of time host species 1 is characterized by a larger phenotypic value for the resistance trait than host species 2, the genetic correlation in the parasite will be positive. This positive genetic correlation is driven by epistatic selection for individual parasites that prefer host species 1 and have large values for the attack trait and for other individual parasites that prefer host species 2 and have small values of the attack trait. If during other periods of time, host species 2 had the greater value of the resistance trait, the sign of epistasis is reversed and the genetic correlation in the parasite would be negative.

Together, our analytical results suggest that coevolutionary alternation is possible in both the matching phenotype and phenotype difference models as long as differences in host species frequency and quality are small relative to the sensitivity of parasite attack success to host resistance traits. Our analytical results, however, cannot fully evaluate the con-

ditions under which coevolutionary alternation will occur, nor do they apply to cases of strong selection, infrequent recombination, or polygenic inheritance. We addressed these issues by analyzing deterministic simulations that incorporate multiple loci, mutation, strong selection, arbitrary rates of parasite recombination, and varying degrees of pleiotropy between parasite preference and attack traits.

Multilocus Simulations

Simulations assumed that the resistance phenotype of each host species i was determined by $n_{R,i}$ haploid diallelic loci that recombine at rate $r_{R,i}$ and experience reversible mutation at a rate of 1×10^{-6} per locus. The phenotype of host species i was assumed to be determined by the additive action of the $n_{R,i}$ loci such that:

$$z_{R,i} = \sum_{j=1}^{n_{R,i}} b_{i,j} X_{i,j}, \tag{8}$$

where $b_{i,j}$ is the phenotypic effect of locus j, and $X_{i,j}$ is an indicator variable that is equal to one when locus j in host species i carries a 1 allele and equal to zero when locus j in host species i carries a 0 allele. To constrain the two host species to an equal phenotypic range $\{0 < z_{R,i} < 1\}$, the $b_{i,j}$ are assumed to equal $1/n_{R,i}$ (Nuismer and Doebeli 2004; Nuismer et al. 2005).

Because we track two discrete phenotypic traits in the parasite (choice and attack), we must explicitly address the potential for pleiotropic effects of loci that contribute to the phenotype of both traits. Such a scenario might arise if, for instance, individual parasites that carry an allele for increased preference for host species 1 must have a decreased attack phenotype due to physiological or anatomical trade-offs. We incorporated such pleiotropic effects using a modified version of the methodology used by Nuismer and Doebeli (2004). Specifically, we assume that the parasite has a total of n_{tot} loci in its genome, with the first n_P loci determining the choice trait and the last n_A loci determining the attack trait. This genetic architecture allows loci in the middle of the parasite genome to have pleiotropic effects.

We assume that the pleiotropic loci always contribute to the choice trait in a positive fashion so that the choice phenotype of the parasite is given by:

$$z_P = \sum_{j=1}^{n_P} b_{P,j} X_j \tag{9}$$

where $b_{P,j}$ is the effect of choice locus j on the parasite choice phenotype and X_j is again an indicator variable taking values zero or one. We assume that $b_{P,j} = 1/n_P$ so that the choice phenotype is constrained to lie on the interval $\{0 < z_P < 1\}$. In contrast, we assume that the shared loci can either increase (positive pleiotropy) or decrease (negative pleiotropy) the attack phenotype of the parasite. With positive pleiotropy the attack phenotype of the parasite is given by:

$$z_A = \sum_{j=n_{tot}-n_A+1}^{n_{tot}} b_{A,j} X_j, \tag{10}$$

where $b_{A,j}$ is the effect of locus j on the parasite attack phenotype, X_j is again an indicator variable taking values zero or one, and $b_{A,j}$ is set equal to $1/n_A$ to ensure a phenotypic range confined to $\{0 < z_A < 1\}$. With negative pleiotropy, the attack phenotype of the parasite is given by:

$$z_A = \sum_{j=n_{tot}-n_A+1}^{n_P} b_{A,j}(1 - X_j) + \sum_{j=n_P+1}^{n_{tot}} b_{A,j} X_j, \tag{11}$$

where $b_{A,j}$ is the effect of locus j on the parasite attack phenotype, X_j is again an indicator variable taking values zero or one, and $b_{A,j}$ is set equal to $1/n_A$ to ensure a phenotypic range confined to $\{0 < z_A < 1\}$. All parasite loci were assumed to recombine at rate r_P, and undergo reversible mutation at a rate of 1×10^{-6} per locus.

Simulations were used to explore the likelihood of coevolutionary alternation for both the phenotype matching and phenotype difference models as well as three different genetic architectures: case 1: host resistance determined by three freely recombining loci ($n_{R,1} = n_{R,2} = 3$) and parasite preference and attack traits determined by three independent loci ($n_A = 3$, $n_P = 3$, $n_{tot} = 6$); case 2: host resistance determined by four freely recombining loci ($n_{R,1} = n_{R,2} = 4$) and parasite preference and attack traits each determined by four loci, with two shared loci exhibiting positive pleiotropy ($n_A = 4$, $n_P = 4$, $n_{tot} = 6$); case 3: host resistance determined by four freely recombining loci ($n_{R,1} = n_{R,2} = 4$) and parasite preference and attack traits each determined by four loci, with two shared loci exhibiting negative pleiotropy ($n_A = 4$, $n_P = 4$, $n_{tot} = 6$).

For each of the genetic architectures described in the three cases above, we ran simulations that explored a range of parasite recombination rates $\{r_P = 0.005, 0.05, 0.5\}$, sensitivities of the probability of successful parasite attack to parasite attack phenotype and host resistance phenotype $\{\alpha_2 = 0.1, 1, 10, \alpha_1 < \alpha_2\}$, sensitivities of parasite host preference to parasite preference phenotype $\{\omega = 0.1, 1, 10\}$, strengths of stabilizing selection acting on host species i $\{\gamma_i = 0.05\ \alpha_i, 0.25\ \alpha_i\}$, strengths of stabilizing selection acting on the parasite attack trait $\{\gamma_P = 0.05\ (\alpha_1 + \alpha_2)/2, 0.25\ (\alpha_1 + \alpha_2)/2\}$, and differences in the overall quality of the two host species $\{f_1\kappa_1 - f_2\kappa_2 = -0.398, 0.002, 0.402\}$. In all cases the optimum phenotype favored by stabilizing selection (θ) was set to zero in both host and parasite. In addition to covering a wide range of parameter combinations, we ran three replicate simulations for each parameter combination, each initiated from random genotype frequencies. In total, we ran 23,328 simulations.

An individual simulation run was considered an example of coevolutionary alternation if the evolutionary trajectory of the parasite preference trait changed direction at least twice between generations 1000 and 3000. Although this criterion is somewhat arbitrary, it was chosen because tests of individual cases suggested that it was sufficient to detect the majority of cases of biologically relevant coevolutionary alternation. Based on this criterion, we calculated the percentage of simulation runs in which coevolutionary alternation occurred for each combination of parameters (Appendix Tables A1–A6). In addition to calculating the frequency of coevolutionary alternation, we quantified its strength in those cases where it occurred by calculating its average amplitude. The amplitude of coevolutionary alternation was cal-

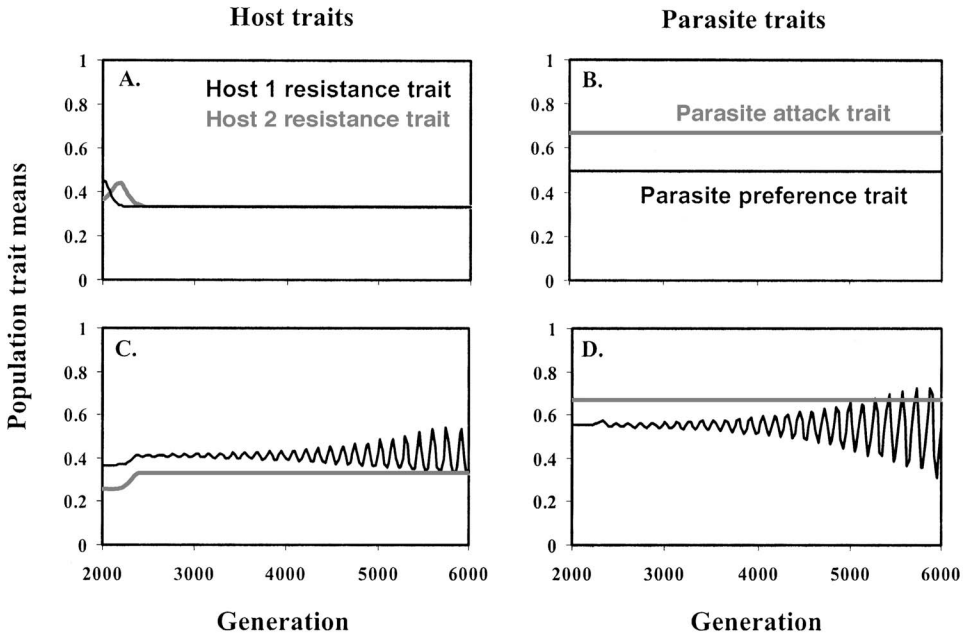

FIG. 2. Numerical simulations of coevolutionary dynamics for host resistance traits ((A, C) and the parasite attack and preference traits (B, D) for the phenotypic difference model. Panels (A) and (B) show a case where parasite genotypes do not differ in their preference for the two host species ($\omega = 0$), and hence parasite preference cannot evolve. Panels (C) and (D) are identical, except that parasite genotypes differ in their preference for the two host species ($\omega = 8$), allowing parasite preference to evolve. The first 2000 generations were truncated to improve the clarity of cyclical dynamics. Parameters used in the simulation were: $\alpha_1 = 2.3$, $\alpha_2 = 1.9$, $\gamma_P = 0.525$, $\gamma_{H,1} = 0.46$, $\gamma_{H,1} = 0.38$, $\theta_P = 0$, $\theta_{H,1} = 0$, $\theta_{H,2} = 0$, $f_1 = 0.505$, $\kappa_1 = \kappa_2 = 1$, $n_A = 3$, $n_P = 3$, $n_{tot} = 6$, $n_{R,1} = 3$, $n_{R,2} = 3$, and $r_P = 0.4$.

culated as the total change in the parasite preference trait, z_P, that took place between generations where the evolutionary trajectory of parasite preference switched directions. Thus, larger amplitudes indicate a greater change in parasite preference over the course of a single cycle of coevolutionary alternation. For each combination of parameter values, we calculated the average amplitude of coevolutionary alternation (Appendix Tables A1–A6).

The simulation results demonstrate that coevolutionary alternation readily arises in both the phenotype matching and phenotype difference models. In addition to demonstrating the feasibility of coevolutionary alternation, simulations support several of the key results derived from the single-locus analytical models. First, all else being equal, coevolutionary alternation is much more common in the matching model, with 46.61% of simulations showing coevolutionary alternation for the matching model but only 26.81% showing coevolutionary alternation for the model of phenotypic difference. Second, as differences in the overall quality ($f_1\kappa_1 - f_2\kappa_2$) of the two host species increased, the frequency of coevolutionary alternation decreased, whereas the frequency of coevolutionary alternation increased with increased average sensitivities of parasite attack to host and parasite phenotypes ($[\alpha_1 + \alpha_2]/2$) (see Supplementary Material, available online; Appendix Tables A1–A6). Third, genetic correlations between parasite preference and attack traits were indeed generally greater in magnitude for the matching model. Fourth, when coevolutionary alternation does not occur, parasite preference generally evolves toward maximum specialization on a single host (i.e., $z_P \rightarrow 0$ or 1).

In addition to these conclusions anticipated from the analytical model, simulations revealed several additional features of coevolutionary alternation. First, simulation results demonstrated that the sensitivity of parasite host preference to parasite preference phenotype (ω) substantially influences the likelihood of coevolutionary alternation. However, the effect of ω is not universal, generating a consistent impact in only the phenotypic difference model where the frequency of coevolutionary alternation was greatest for the largest value of ω (Appendix Tables A1–A6). Second, simulation results reveal that parasite preference can change significantly because of coevolutionary alternation (Fig. 2; Appendix Tables A1–A6). For instance, the average amplitude of coevolutionary alternation across all simulation runs was 0.1335 in the matching model and 0.2421 in the difference model, which, given that the maximum possible change in preference phenotype is one, represents significant evolutionary change in the preference phenotype. For some combinations of parameter values, particularly those where ω is large, these changes in parasite preference phenotype correspond to rapid evolutionary shifts between virtually complete specialization on host 1 and virtually complete specialization on host 2.

Finally, the simulation results demonstrate that when coevolutionary alternation occurs, coevolutionary dynamics between resistance and attack traits can be significantly altered. This effect is particularly striking for the model of phenotype difference, which, in the absence of evolutionarily labile parasite preference generally results in host resistance phenotypes that approach stable values. Allowing parasite preference to evolve can lead to shifts in this behavior, promoting cyclical dynamics of host resistance traits (Fig. 2). Although parasite preference can also alter the coevolutionary dynam-

ics of the matching model, the effects appear to be generally less pronounced.

Discussion

Our simple model of multispecific coevolution between a parasite and two host species demonstrates that coevolutionary alternation is possible and, in some cases, quite likely. When coevolutionary alternation occurs, the result is repeated, and potentially rapid, evolutionary change in patterns of parasite specialization (see Fig. 2; Appendix Tables A1–A6) and relative levels of host defense. Hence, coevolutionary alternation provides a specific mechanism by which a community of interacting species can become compartmentalized into pairwise interactions with constantly shifting species compositions.

The likelihood of coevolutionary alternation depends upon the functional form of host resistance (phenotype matching vs. phenotype difference), the potential for parasite preference to evolve, and the ecological equivalence of host species. Specifically, coevolutionary alternation tends to be most common when interactions are mediated by phenotype matching, predator preference can evolve significantly, and the two host species occur with equal frequency and provide equivalent fitness benefits to the parasite. Coevolutionary alternation can also occur with reasonable frequency (26.81%) in the phenotypic difference model, although the required conditions are more restrictive. When alternation does occur in the phenotypic difference model, however, its amplitude tends to be greater—and the changes it causes in the coevolutionary dynamics of host resistance and parasite attack traits more striking—than in the phenotype matching model.

There are two reasons that the frequency of coevolutionary alternation differs between the two models of host resistance that we have considered. First, the functional relationships between parasite attack and host resistance traits that define each model provide different scope for the evolution of variable resistance levels across the two host species (Fig. 1). Specifically, the function that defines phenotype matching provides greater scope for variable levels of host resistance to evolve than does the function that defines phenotype differences. Consequently, the evolution of host preference is more likely to be driven by variable levels of resistance across host species than by static differences in overall host quality in the matching model. Second, the phenotype matching and phenotype difference models differ in their propensity to cycle in the absence of evolutionarily labile parasite preference. Specifically, cycles of parasite attack and host resistance traits are more common in the matching than difference model. As a result, at least some cases of coevolutionary alternation observed in the matching model are likely the result of parasite preference tracking fluctuating levels of host resistance rather than reciprocal evolutionary changes in parasite preference and host resistance.

Our results also have important consequences for longstanding arguments over the potential for coevolution to drive the evolution of complex biological communities (Hougeneitzman and Rausher 1994; Rausher 1996; Iwao and Rausher 1997; Juenger and Bergelson 1998; Thompson 1998; Stinchcombe and Rausher 2001; Strauss et al. 2005; Bascompte et al. 2006). For example, coastal regions of oceans have a wide range of invertebrate species that defend themselves against predators by using thick calcareous shells, and predators have evolved a diverse array of mechanisms to overcome these defenses. It has been argued that the escalating defenses of these invertebrates result from predator attack but not through a mechanism of reciprocal selection (Vermeij 1994). Rather, prey species collectively have been subject to overall selection for escalation in defenses over evolutionary time in an increasingly dangerous world. Adopting a different tack, others have argued that coevolutionary selection may often be diffusely and nonlinearly distributed across multiple species (Iwao and Rausher 1997; Inouye and Stinchcombe 2001; Strauss et al. 2005). Although this must surely be true in some interactions, coevolutionary alternation provides a specific mechanism by which diffuse coevolution can be repeatedly broken down into compartmentalized pairwise interactions with different component species.

Although our central result, namely that coevolutionary alternation is feasible, is likely to be quite robust, the frequency with which it occurs may be sensitive to our model assumptions. Of potential importance are our assumptions that phenotypes are determined by the additive action of diallelic haploid loci and that a single predator trait controls interactions with both prey species. The latter assumption may not be critically important, however, because the model of Kawecki (1998), in which resistance to different host species was controlled by different traits, still produced what appears to be coevolutionary alternation in at least some cases.

In addition to these genetic assumptions, our model assumes that coevolution occurs within a single locality and thus ignores the potential for the ongoing dynamics of coevolutionary alternation to be shaped by the geographic mosaic of coevolution (Thompson 1994, 2005). For instance, different prey or host populations are likely to harbor different levels of defense at any moment in evolutionary time, as defenses wax and wane at different rates and to different degrees among regions. Moreover, during some periods, some populations could simply be in coevolutionary cold spots, outside the geographic range of a particular parasite or predator with which they coevolve elsewhere. Hence, gene flow among populations and the continually changing geographic ranges of interacting species are likely to keep the evolution of specialization and defense highly dynamic both within and among populations, often preventing fixation at the species level of any particular levels of specialization or defense. Coevolutionary alternation may occur in only a subset of an interacting population at any moment in evolutionary time, as suggested by the models, but the overall geographic mosaic of coevolution will continue to drive ongoing change at the species level.

Consider, for example, the geographically variable interactions between avian brood parasites and their hosts. Davies and Brooke (1989) found that white wagtails in Britain reject cuckoo eggs, whereas in Iceland, where cuckoos do not occur, they do not. Lahti (2005) found evidence of relaxed selection over 100–200 years in introduced populations of African village weaverbirds introduced onto islands where cuckoos were absent. Soler and colleagues (Soler et al. 1999, 2001; Soler

and Soler 2000) found complex interactions between current coevolutionary selection and gene flow in the patterns of defense shown by European magpies in response to brood parasitism by great spotted cuckoos. Based upon their combined genetic and ecological analyses, they argued that the patterns they observed among European magpie populations that serve as cuckoo hosts are consistent with the occurrence of some populations in coevolutionary hotspots that exchange genes with populations in coevolutionary coldspots (i.e., regions in which coevolutionary selection is reduced or absent; Soler et al. 2001). In addition, the number of potential local host species varies geographically across the ranges of brood parasites, thereby creating additional geographic variation in the potential for coevolutionary alternation. Consequently, these interactions, which were the first to show evidence of coevolutionary alternations in some regions, show strong differences among populations in levels of defenses and even the potential for coevolutionary alternation. That potential for coevolutionary alternation will itself continue to change through changing patterns of gene flow and continually changing geographic ranges over evolutionary time.

We have considered a model of one predator or parasite that interacts with two prey or host species in an effort to elucidate the conditions that lead to coevolutionary alternation. Adding additional species could significantly alter the frequency of coevolutionary alternation or create novel dynamical regimes not possible in our simple three-species framework. Exploring how the frequency and structure of coevolutionary alternation changes as the number of species increases will be critical to understanding the extent to which coevolutionary alternation shapes the network structure of entire food webs.

Acknowledgments

We thank M. Dybdahl, B. Ridenhour, C. Fernandez, K. Horjus, P. Abrams, M. Servedio, and two anonymous reviewers for helpful comments. This work was supported by National Science Foundation grants DEB 0343023 and DMS 0540392 to SLN and DEB 344147 to JNT.

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Corresponding Editor: M. Servedio

Appendix: Simulation Data in Tabular Format

Table A1. Summary of simulation data for the model of phenotype difference and the genetic architecture described by case 1: $n_{R,1} = n_{R,2} = 3$, $n_A = 3$, $n_P = 3$, $n_{tot} = 6$. The first entry in each cell is the percentage of cases in which coevolutionary alternation occurred. The second entry in each cell is the average amplitude of coevolutionary alternation for those cases where it occurred (NA indicates that the amplitude could not be determined because there were no cases of coevolutionary alternation).

		$(\alpha_1 + \alpha_2)/2 = 0.1$	$(\alpha_1 + \alpha_2)/2 = 0.55, 1$	$(\alpha_1 + \alpha_2)/2 = 5.05, 5.5$	$(\alpha_1 + \alpha_2)/2 = 10$	Row average
$\omega = 0.1$	$\lvert f_1\kappa_1 - f_2\kappa_2\rvert = 0.002$	0, NA	0, NA	0.69, 0.0006	30.56, 0.0032	7.81, 0.0019
	$\lvert f_1\kappa_1 - f_2\kappa_2\rvert = 0.398, 0.402$	0, NA	0, NA	2.43, 0.2817	11.81, 0.0001	3.56, 0.1409
$\omega = 1$	$\lvert f_1\kappa_1 - f_2\kappa_2\rvert = 0.002$	0, NA	0, NA	22.92, 0.6503	25.00, 0.4231	11.98, 0.5367
	$\lvert f_1\kappa_1 - f_2\kappa_2\rvert = 0.398, 0.402$	0, NA	0, NA	6.60, 0.4690	25.00, 0.5028	7.90, 0.4859
$\omega = 10$	$\lvert f_1\kappa_1 - f_2\kappa_2\rvert = 0.002$	70.83, 0.0652	97.22, 0.1952	100.00, 0.4902	100.00, 0.0732	92.01, 0.2060
	$\lvert f_1\kappa_1 - f_2\kappa_2\rvert = 0.398, 0.402$	0, NA	0, NA	18.75, 0.5320	54.86, 0.6634	18.40, 0.5977
	Column average	11.81, 0.0652	16.20, 0.1952	25.23, 0.4040	41.20, 0.2776	23.61, 0.3282

Table A2. Summary of simulation data for the model of phenotype matching and the genetic architecture described by case 1: $n_{R,1} = n_{R,2} = 3$, $n_A = 3$, $n_P = 3$, $n_{tot} = 6$. The first entry in each cell is the percentage of cases in which coevolutionary alternation occurred. The second entry in each cell is the average amplitude of coevolutionary alternation for those cases where it occurred (NA indicates that the amplitude could not be determined because there were no cases of coevolutionary alternation).

		$(\alpha_1 + \alpha_2)/2 = 0.1$	$(\alpha_1 + \alpha_2)/2 = 0.55, 1$	$(\alpha_1 + \alpha_2)/2 = 5.05, 5.5$	$(\alpha_1 + \alpha_2)/2 = 10$	Row average
$\omega = 0.1$	$\lvert f_1\kappa_1 - f_2\kappa_2\rvert = 0.002$	0, NA	37.50, 0.0328	88.19, 0.0088	70.83, 0.0226	49.13, 0.0214
	$\lvert f_1\kappa_1 - f_2\kappa_2\rvert = 0.398, 0.402$	0, NA	1.74, 0.0003	45.83, 0.0026	68.75, 0.0171	29.08, 0.0067
$\omega = 1$	$\lvert f_1\kappa_1 - f_2\kappa_2\rvert = 0.002$	0, NA	38.19, 0.1170	79.17, 0.1762	66.67, 0.0966	46.01, 0.1299
	$\lvert f_1\kappa_1 - f_2\kappa_2\rvert = 0.398, 0.402$	0, NA	3.47, 0.0000	43.75, 0.0859	75.00, 0.1741	30.56, 0.0867
$\omega = 10$	$\lvert f_1\kappa_1 - f_2\kappa_2\rvert = 0.002$	61.11, 0.1440	54.17, 0.2872	75.00, 0.2620	100.00, 0.2366	72.57, 0.2324
	$\lvert f_1\kappa_1 - f_2\kappa_2\rvert = 0.398, 0.402$	0, NA	0, NA	50.00, 0.1098	75.00, 0.1315	31.25, 0.1207
	Column average	10.19, 0.1440	22.51, 0.0875	63.66, 0.1075	76.04, 0.1131	43.10, 0.0996

Table A3. Summary of simulation data for the model of phenotype difference and the genetic architecture described by case 2: $n_{R,1} = n_{R,2} = 4$, $n_A = 4$, $n_P = 4$, $n_{tot} = 6$. The first entry in each cell is the percentage of cases in which coevolutionary alternation occurred. The second entry in each cell is the average amplitude of coevolutionary alternation for those cases where it occurred (NA indicates that the amplitude could not be determined because there were no cases of coevolutionary alternation).

		$(\alpha_1 + \alpha_2)/2 = 0.1$	$(\alpha_1 + \alpha_2)/2 = 0.55, 1$	$(\alpha_1 + \alpha_2)/2 = 5.05, 5.5$	$(\alpha_1 + \alpha_2)/2 = 10$	Row average
$\omega = 0.1$	$\lvert f_1\kappa_1 - f_2\kappa_2\rvert = 0.002$	0, NA	0, NA	49.31, 0.1366	40.28, 0.0965	22.40, 0.1166
	$\lvert f_1\kappa_1 - f_2\kappa_2\rvert = 0.398, 0.402$	0, NA	0, NA	36.11, 0.1250	46.53, 0.1155	20.66, 0.1202
$\omega = 1$	$\lvert f_1\kappa_1 - f_2\kappa_2\rvert = 0.002$	0, NA	0.69, 0.2574	19.44, 0.1198	43.06, 0.1960	15.80, 0.2177
	$\lvert f_1\kappa_1 - f_2\kappa_2\rvert = 0.398, 0.402$	0, NA	0, NA	28.13, 0.1141	23.61, 0.1642	12.93, 0.1392
$\omega = 10$	$\lvert f_1\kappa_1 - f_2\kappa_2\rvert = 0.002$	44.44, 0.0545	83.33, 0.0648	95.83, 0.3038	87.50, 0.1011	77.78, 0.1310
	$\lvert f_1\kappa_1 - f_2\kappa_2\rvert = 0.398, 0.402$	0, NA	0, NA	31.25, 0.2964	64.58, 0.4670	23.96, 0.3817
	Column average	7.41, 0.0545	14.00, 0.1611	43.34, 0.1959	50.93, 0.1900	28.92, 0.1844

TABLE A4. Summary of simulation data for the model of phenotype matching and the genetic architecture described by case 2: $n_{R,1} = n_{R,2} = 4$, $n_A = 4$, $n_P = 4$, $n_{tot} = 6$. The first entry in each cell is the percentage of cases in which coevolutionary alternation occurred. The second entry in each cell is the average amplitude of coevolutionary alternation for those cases where it occurred (NA indicates that the amplitude could not be determined because there were no cases of coevolutionary alternation).

		$(\alpha_1 + \alpha_2)/2 = 0.1$	$(\alpha_1 + \alpha_2)/2 = 0.55, 1$	$(\alpha_1 + \alpha_2)/2 = 5.05, 5.5$	$(\alpha_1 + \alpha_2)/2 = 10$	Row average
$\omega = 0.1$	$\lvert f_1\kappa_1 - f_2\kappa_2\rvert = 0.002$	12.50, 0.0242	36.11, 0.3936	98.61, 0.1959	100.00, 0.1429	61.81, 0.1892
	$\lvert f_1\kappa_1 - f_2\kappa_2\rvert = 0.398, 0.402$	0, NA	22.57, 0.3428	81.60, 0.1546	100.00, 0.1649	51.04, 0.2207
$\omega = 1$	$\lvert f_1\kappa_1 - f_2\kappa_2\rvert = 0.002$	8.33, 0.0441	29.86, 0.4695	84.03, 0.3142	100.00, 0.1585	55.56, 0.2466
	$\lvert f_1\kappa_1 - f_2\kappa_2\rvert = 0.398, 0.402$	0, NA	22.57, 0.2625	73.61, 0.1695	100, 0.2252	49.05, 0.2190
$\omega = 10$	$\lvert f_1\kappa_1 - f_2\kappa_2\rvert = 0.002$	23.61, 0.1890	84.72, 0.1494	93.06, 0.1919	93.06, 0.1501	73.61, 0.1701
	$\lvert f_1\kappa_1 - f_2\kappa_2\rvert = 0.398, 0.402$	0, NA	0, NA	44.10, 0.1441	79.17, 0.1783	30.82, 0.1612
	Column average	7.41, 0.0858	32.64, 0.3236	79.17, 0.1950	95.37, 0.1700	53.65, 0.2011

TABLE A5. Summary of simulation data for the model of phenotype difference and the genetic architecture described by case 3: $n_{R,1} = n_{R,2} = 4$, $n_A = 4$, $n_P = 4$, $n_{tot} = 6$. The first entry in each cell is the percentage of cases in which coevolutionary alternation occurred. The second entry in each cell is the average amplitude of coevolutionary alternation for those cases where it occurred (NA indicates that the amplitude could not be determined because there were no cases of coevolutionary alternation).

		$(\alpha_1 + \alpha_2)/2 = 0.1$	$(\alpha_1 + \alpha_2)/2 = 0.55, 1$	$(\alpha_1 + \alpha_2)/2 = 5.05, 5.5$	$(\alpha_1 + \alpha_2)/2 = 10$	Row average
$\omega = 0.1$	$\lvert f_1\kappa_1 - f_2\kappa_2\rvert = 0.002$	0, NA	0, NA	50.00, 0.1437	45.83, 0.0831	23.96, 0.1134
	$\lvert f_1\kappa_1 - f_2\kappa_2\rvert = 0.398, 0.402$	0, NA	0, NA	28.82, 0.1518	45.83, 0.0911	18.66, 0.1215
$\omega = 1$	$\lvert f_1\kappa_1 - f_2\kappa_2\rvert = 0.002$	0, NA	0, NA	43.06, 0.1358	43.06, 0.1849	21.53, 0.1603
	$\lvert f_1\kappa_1 - f_2\kappa_2\rvert = 0.398, 0.402$	0, NA	0, NA	20.49, 0.2011	23.61, 0.1816	11.02, 0.1913
$\omega = 10$	$\lvert f_1\kappa_1 - f_2\kappa_2\rvert = 0.002$	37.50, 0.0463	72.22, 0.1908	89.58, 0.5785	73.61, 0.0988	68.23, 0.2286
	$\lvert f_1\kappa_1 - f_2\kappa_2\rvert = 0.398, 0.402$	0, NA	0, NA	31.25, 0.4656	64.58, 0.4684	23.96, 0.4670
	Column average	6.25, 0.0463	12.04, 0.1908	43.87, 0.2794	49.42, 0.1846	27.89, 0.2137

TABLE A6. Summary of simulation data for the model of phenotype matching and the genetic architecture described by case 3: $n_{R,1} = n_{R,2} = 4$, $n_A = 4$, $n_P = 4$, $n_{tot} = 6$. The first entry in each cell is the percentage of cases in which coevolutionary alternation occurred. The second entry in each cell is the average amplitude of coevolutionary alternation for those cases where it occurred (NA indicates that the amplitude could not be determined because there were no cases of coevolutionary alternation).

		$(\alpha_1 + \alpha_2)/2 = 0.1$	$(\alpha_1 + \alpha_2)/2 = 0.55, 1$	$(\alpha_1 + \alpha_2)/2 = 5.05, 5.5$	$(\alpha_1 + \alpha_2)/2 = 10$	Row average
$\omega = 0.1$	$\lvert f_1\kappa_1 - f_2\kappa_2\rvert = 0.002$	0, NA	37.50, 0.0328	88.19, 0.0087	70.83, 0.0226	49.13, 0.0214
	$\lvert f_1\kappa_1 - f_2\kappa_2\rvert = 0.398, 0.402$	0, NA	1.74, 0.0003	45.83, 0.0026	68.75, 0.0171	29.08, 0.0067
$\omega = 1$	$\lvert f_1\kappa_1 - f_2\kappa_2\rvert = 0.002$	0, NA	38.19, 0.1170	79.17, 0.1762	66.67, 0.0966	46.01, 0.1299
	$\lvert f_1\kappa_1 - f_2\kappa_2\rvert = 0.398, 0.402$	0, NA	3.47, 0.0000	43.75, 0.0859	75.00, 0.1741	30.56, 0.0867
$\omega = 10$	$\lvert f_1\kappa_1 - f_2\kappa_2\rvert = 0.002$	61.11, 0.1440	54.17, 0.2872	75.00, 0.2620	100.00, 0.2366	72.57, 0.2324
	$\lvert f_1\kappa_1 - f_2\kappa_2\rvert = 0.398, 0.402$	0, NA	0, NA	50.00, 0.1098	75.00, 0.1315	31.25, 0.1207
	Column average	10.19, 0.1440	22.51, 0.0875	63.66, 0.1075	76.04, 0.1131	43.10, 0.0996