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MULTILOCUS GENETICS AND THE COEVOLUTION OF QUANTITATIVE TRAITS

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Abstract.—We develop and analyze an explicit multilocus genetic model of coevolution. We assume that interactions between two species (mutualists, competitors, or victim and exploiter) are mediated by a pair of additive quantitative traits that are also subject to direct stabilizing selection toward intermediate optima. Using a weak-selection approximation, we derive analytical results for a symmetric case with equal locus effects and no mutation, and we complement these results by numerical simulations of more general cases. We show that mutualistic and competitive interactions always result in coevolution toward a stable equilibrium with no more than one polymorphic locus per species. Victim-exploiter interactions can lead to different dynamic regimes including evolution toward stable equilibria, cycles, and chaos. At equilibrium, the victim is often characterized by a very large genetic variance, whereas the exploiter is polymorphic in no more than one locus. Compared to related one-locus or quantitative genetic models, the multilocus model exhibits two major new properties. First, the equilibrium structure is considerably more complex. We derive detailed conditions for the existence and stability of various classes of equilibria and demonstrate the possibility of multiple simultaneously stable states. Second, the genetic variances change dynamically, which in turn significantly affects the dynamics of the mean trait values. In particular, the dynamics tend to be destabilized by an increase in the number of loci.

Key words.—Coevolutionary cycling, disruptive selection, frequency-dependent selection, maintenance of genetic variation, multilocus genetics, victim-exploiter coevolution, weak-selection approximation.

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Coevolution between interacting species (Futuyma and Slatkin 1983; Thompson 1994, 2005) plays an important role in shaping biological diversity. For example, coevolution is thought to be associated with major events in the history of life, such as the evolution of eukaryotic cells (Margulis 1970) and the evolution of sex (e.g., Hamilton et al. 1990), and to have shaped macroevolutionary trends, such as the evolution of brain size (Jerison 1973) and limb morphology (Bakker 1983) in carnivores and ungulates. Coevolution between plants and herbivores may be responsible for a considerable proportion of biodiversity (Ehrlich and Raven 1964). On a more microevolutionary time scale, coevolution influences the strength of interspecific interactions (Thompson 1994, 2005; Dybdahl and Lively 1998; Benkman 1999; Brodie and Brodie 1999), which in turn might have important consequences for the dynamics of ecological communities (Thompson 1998). Coevolution—especially between hosts and parasites or pathogens—is also thought to play an important role in the maintenance of genetic variation (e.g., Hamilton et al. 1990; Sasaki 2000).

An increased understanding of coevolutionary processes is an important goal for evolutionary biology. Due to the inherent complexity of these processes and the long time scales involved, a particularly important role in this endeavor must be played by mathematical models (for reviews, see Maynard Smith and Slaktin 1979; Abrams 2000; Bergelson et al. 2001). Key theoretical questions concern the rate and direction of change in the phenotype of one species in response to changes in another species (e.g., Abrams 1986a,b), the influence of coevolution on population dynamics (reviewed in Abrams 2000), and the maintenance of genetic variation under different types of coevolutionary interactions (e.g., Kirzhner et al. 1999; Sasaki 2000). Furthermore, considerable effort has been made to understand the conditions under which coevolution between two species reaches a stable endpoint or equilibrium and when it results in continuing escalation or endless coevolutionary cycling (e.g., Dieckmann et al. 1995; Abrams and Matsuda 1997; Gavrilets 1997a; Gavrilets and Hastings 1998; Sasaki 2000). The latter two outcomes seem particularly likely when coevolution is between a victim (such as a prey or host) and an exploiter (such

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as a predator or parasite), and they are reflected by two well-known metaphors: the evolutionary arms race (Dawkins and Krebs 1979) and the Red Queen (van Valen 1973).

Many potentially coevolutionary interactions involve quantitative traits, that is, traits showing continuous variation in populations. Examples include typical morphological traits such as claw strength and shell thickness in crabs and gastropodes from Lake Tanganyika (West et al. 1991) or the morphologies of the bills of North American crossbills and the pine cones they feed upon (Benkman 1999). Quantitative variation has also been observed in biochemically mediated interactions, such as those between wild parsnip and the parsnip webworm (Beerenbaum et al. 1986; Beerenbaum and Zangerl 1992), aphids and parasitoid wasps (Henter 1995; Henter and Via 1995), and toxic newts and garter snakes (Brodie and Brodie 1999).

Quantitative traits are determined by the interaction of multiple genetic loci (Lynch and Walsh 1998). For the majority of traits, details of this interaction are unknown. However, even simple additive models depend on a considerable number of parameters, such as the number of loci and their relative contributions to the trait. Furthermore, models of the evolution of quantitative traits in single species have shown that these genetic details are very important, both in the case of constant selection (e.g., Nagylaki 1991; Bürger 2000) and in the case of within-population frequency-dependent selection (e.g., Gavrilets and Hastings 1995; Bürger 2002a,b, 2005). Similar effects should also be expected in models of coevolution, where selection is frequency dependent between populations.

However, mathematical models of coevolution involving quantitative traits have usually incorporated only very simple genetics. Models using phenotypic approximations (Abrams 2001), such as adaptive dynamics (e.g., Dieckmann and Law 1996; Doebeli and Dieckmann 2000; Dercole et al. 2003), game theory (e.g., Brown and Vincent 1992), and quantitative genetics (e.g., Saloniemi 1993; Abrams and Matsuda 1997; Gavrilets 1997a; Khibnik and Kondrashov 1997), describe the evolution of phenotypes directly, while skipping over the details of the underlying genetics. Explicit genetic models frequently consider only one locus (Gavrilets and Hastings 1998) or two loci (e.g., Bell and Maynard Smith 1987; Seger 1988; Preigel and Korol 1990; Kirzhner et al. 1999) per species. Only a few authors have analyzed the coevolution of quantitative traits using multilocus models. For example, Frank (1994) studied a multilocus model of the interaction between asexual hosts and parasites (see also Sasaki and Godfray 1999). Doebeli (1996a,b), Doebeli and Dieckmann (2000), and Nuismer and Doebeli (2004) investigated multilocus sexual models using simulations, and Nuismer et al. (2005) using analytical methods.

At least three of these models indicate that explicit multilocus genetics can, indeed, have important impacts in models of between-species coevolution. First, Doebeli (1996a) showed that a multilocus model of competitive coevolution predicts ecological character displacement where a comparable quantitative genetic model (Slatkin 1980) does not. Doebeli (1996b) also argued that quantitative genetic approximations (which assume constant genetic variances) do not show the full range of possible behaviors in models where ecological and evolutionary dynamics are coupled. However, Doebeli's models do not incorporate the full range of multilocus dynamics either, because he made the simplifying assumption that allele frequencies at all loci are identical at all time (for the conditions when this assumption can hold true, see Shpak and Kondrashov 1999; Barton and Shpak 2000). Second, Nuismer and Doebeli (2004) analyzed coevolution of simple three-species communities. They contrasted individual-based simulations of an explicit multilocus model to analytical solutions for a quantitative genetic (i.e., constant-variance) approximation. The explicit model yielded far richer results, including novel equilibria and cycles not possible in the simpler model. Finally, Nuismer et al. (2005) analyzed a haploid multilocus model of host-parasite coevolution and found that the evolution of genetic variances can drive coevolutionary cycles. Multilocus genetics also have been shown to be important in coevolutionary models of the gene-for-gene or matching-allele type (i.e., in host-parasite models where the interaction strength is determined by interspecific pairs of resistance and virulence alleles, as opposed to a single pair of quantitative traits; e.g. Seger 1988; Frank 1993; Hamilton 1993; Sasaki 2000; Sasaki et al. 2002).

In summary, explicit multilocus models of coevolving quantitative traits have been studied only sporadically. Furthermore, the existing models contain a number of assumptions, such as haploidy, asexual reproduction, or equal allele frequencies across loci, that are unlikely to be met in many systems. Most models rely on numerical simulations, and analytical results are extremely rare. Nevertheless, general arguments (Thompson 1994; Gavrilets 1997b) and some of the existing models suggest that the incorporation of explicit multilocus genetics into a coevolutionary model can significantly alter its predictions. Therefore, studying the effects of multilocus genetics promises to be an important step toward a better understanding of coevolution.

In the present paper, we investigate the effects of multilocus genetics on a simple model of coevolution between two species (mutualists, competitors, or victim and exploiter). We assume that these species interact via a pair of quantitative traits and that the interaction strength is maximal if the two traits have equal values. In addition, the traits are assumed to be under stabilizing selection toward intermediate optima. This model is a genetically explicit version of a quantitative genetic model by Gavrilets (1997a), as well as an extension of a one-locus model by Gavrilets and Hastings (1998). The behavior of the one-locus model is summarized in Figure 1. Typically, the phenotypes of competitors diverge, leading to ecological character displacement. In contrast, the phenotypes of mutualists tend to converge. Victim-exploiter coevolution can lead to four qualitatively different dynamic regimes. If the victim is under strong stabilizing selection, the outcome is similar to the one of a mutualistic interaction. If this is not the case and if the exploiter can evolve faster than the victim, the system may reach a stable equilibrium where the victim is trapped at a fitness minimum and experiences disruptive selection. If, in contrast, the victim can evolve faster than the exploiter, both species may undergo coevolutionary cycles. These may be either of small scale (so-called stable limit cycles) or span the entire phenotypic range (heteroclinic cycles). Finally, if the exploiter is under

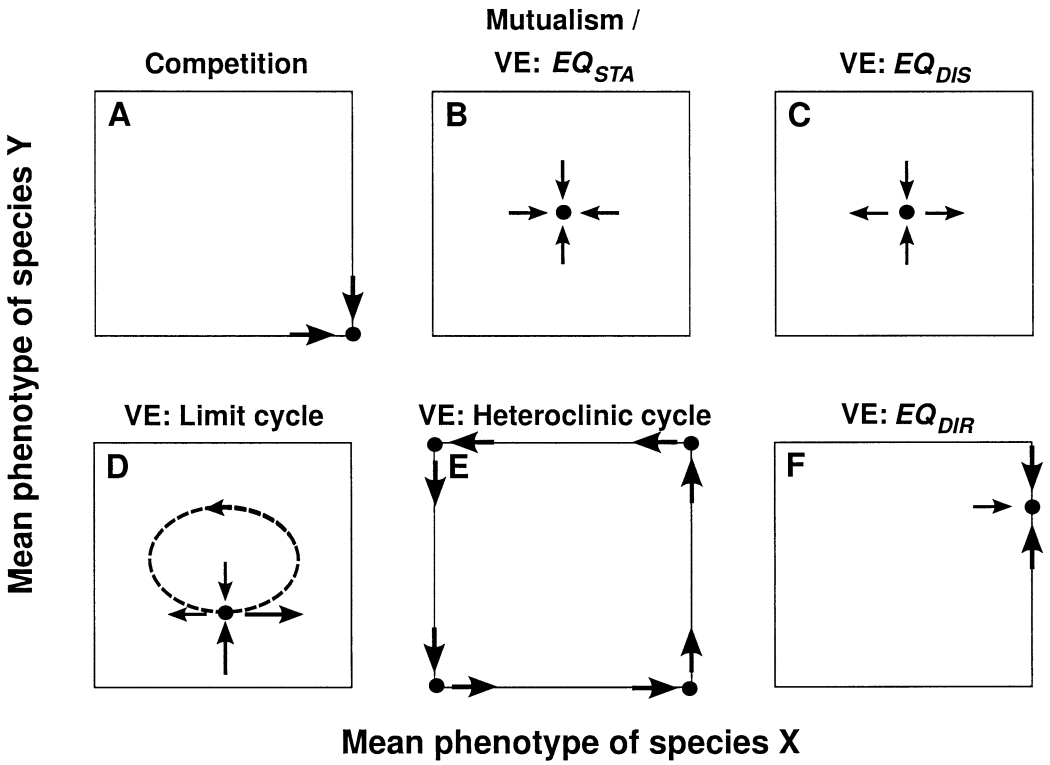

FIG. 1. Schematic overview of possible outcomes of coevolution in the one-locus model (Gavrilets and Hastings 1998). VE stands for victim-exploiter coevolution (where species X is the victim and species Y the exploiter). EQ_{STA}, EQ_{DIS}, and EQ_{DIR} denote the three types of equilibria possible in this case (see Table 1). Dots represent mean phenotypes and arrows indicate the net direction of selection. (A) Stable equilibrium with net directional selection and opposite extreme phenotypes in two competitors. (B) Stable intermediate equilibrium with net stabilizing selection in both species. (C) Stable intermediate equilibrium with net disruptive selection in the victim. (D) Stable limit cycle: the mean phenotypes cycle counter-clockwise along the dashed line. (E) Heteroclinic cycle: the mean phenotypes cycle counter-clockwise along the boundaries of the graph. (F) Stable equilibrium with net directional selection in the victim, leading to an extreme mean victim phenotype.

strong stabilizing selection or if it has a constrained phenotypic range, the victim may permanently escape to an extreme phenotype. Similar conclusions can be drawn from the quantitative genetic model by Gavrilets (1997a). By comparing the results of our multilocus model with those of the two previous approaches, we are able to directly assess the effects of multilocus genetics on the stability of coevolutionary equilibria, the likelihood of coevolutionary cycling, and the role of coevolution in the maintenance of genetic variation.

THE MODEL

We consider a system of two coevolving species, X and Y, whose interaction is governed by a pair of quantitative traits, x in species X and y in species Y. The values of x and y will be referred to as trait values or phenotypes. At the population level, the mean phenotypes are denoted by $\bar{x}$ and $\bar{y}$, respectively, and the phenotypic variances (also called genetic variances here) by G_x and G_y.

Assumptions on Fitness

The traits x and y are subject to two sources of selection: direct stabilizing selection due to, for example, abiotic factors or genetic constraints, and frequency-dependent selection due to between-species interactions. Stabilizing selection favors optimal phenotypes θ_x and θ_y in species X and Y, respectively. These optima can be thought of as physiological optima that are selected for in the absence of the other species. The fitness component due to stabilizing selection decreases with increasing distance of a trait from its physiological optimum, following the Gaussian functions

$$w_x^s(x) = \exp[-\sigma_x(x - \theta_x)^2] \quad \text{and} \tag{1a}$$

$$w_y^s(y) = \exp[-\sigma_y(y - \theta_y)^2], \tag{1b}$$

where the strength of selection is determined by the positive parameters σ_x and σ_y. The second source of selection is the interspecific interaction. The strength of this interaction decreases with the phenotypic distance $|x - y|$ (e.g., Roughgarden 1979) between interacting individuals. The fitness component due to the interaction depends on the average interaction strength experienced by an individual and can be written as

$$w_x^c(x) = \sum_y \exp[-\gamma_x(x - y)^2] f_y(y) \quad \text{and} \tag{2a}$$

$$w_y^c(y) = \sum_x \exp[-\gamma_y(y - x)^2] f_x(x). \tag{2b}$$

Here, $f_x(x)$ and $f_y(y)$ are the frequencies of the respective

phenotypes in the two populations, and the strength of selection is controlled by the absolute values of γ_x and γ_y. The signs of γ_x and γ_y determine the type of the interaction. If both γ_x and γ_y are positive, the interaction is mutualistic, and both species benefit from matching each other's phenotype. If both are negative, the interaction is competitive, and both species benefit from phenotypically diverging from each other. If γ_x and γ_y have different signs, the interaction is of victim-exploiter type and the species with the negative γ is the victim. In this case, the victim benefits from being phenotypically different from the exploiter, whereas the exploiter benefits from being similar to the victim. Thus, the fitness values of victim and exploiter depend only on their absolute phenotypic distance $|x - y|$, but not on whether $x > y$ or $y > x$. It should be noted that such a ''bidirectional axis of vulnerability'' (Abrams 2000) is an important assumption, which may hold true for traits such as size or habitat choice (or any case where the predation process involves a kind of pattern matching or lock-and-key mechanism), but not for other traits such as speed or the ability to detect individuals of the other species (see Discussion).

Finally, we assume that the two fitness components act multiplicatively. This is appropriate, for example, if the corresponding selective forces act at different points in time. Thus, the overall fitness functions are $w_x(x) = w_x^s(x) \cdot w_x^c(x)$ and $w_y(y) = w_y^s(y) \cdot w_y^c(y)$.

Assumptions on Genetics

We assume that the trait x is controlled additively by L_x diploid diallelic loci. (A corresponding haploid model is analyzed in Appendix 3, which is available online only at http://dx.doi.org/10.1554/05-581.1.s3). At locus i, alleles 0 or 1 have effects $\alpha_i/2$ and $-\alpha_i/2$, respectively (all $\alpha_i > 0$). Similarly, y is controlled additively by L_y diallelic loci with locus effects $\beta_j/2$ and $-\beta_j/2$, (all $\beta_j > 0$). The α_i and β_j will also be referred to as locus effects, and the indices will be skipped if the locus effects within a species are identical. Furthermore, we denote the midrange values of the traits as x_m and y_m, so that the phenotypic range (i.e., the range of possible trait values) in species X is from $x_m - \Sigma_i \alpha_i$ to $x_m + \Sigma_i \alpha_i$ and that in species Y is from $y_m - \Sigma_j \beta_j$ to $y_m + \Sigma_j \beta_j$. For simplicity of notation, we will neglect the effects of the microenvironment on the phenotypes, as these can be incorporated in the model by adjusting the coefficients controlling the strength of selection (e.g., Bürger 2000).

We assume that generations are discrete and nonoverlapping and mating is random in both species. Population sizes are constant (or at least regulated by factors different from those considered here) and sufficiently large to exclude stochastic factors such as genetic drift. Each generation, both species undergo a sequence of selection, recombination, segregation, and mutation. Haplotype frequencies after selection and recombination are calculated from the standard recursion relation

$$f_r' = \bar{w}^{-1} \sum_{s,t} w_{st} f_s f_t R(st \to r) \tag{3}$$

(e.g., Bürger 2000). Here, haplotypes are labeled r, s, and t; f_r and f_r' are the frequencies of haplotype r before and after selection and recombination, respectively, $\bar{w}$ is the mean fitness; w_{st} is the fitness of the diploid genotype containing haplotypes s and t; and $R(st \to r)$ is the probability that recombination between haplotypes s and t results in haplotype r. In this paper, we only consider the case of free recombination, where the recombination rate between adjacent loci equals 0.5. Mutation can turn allele 0 into allele 1, and vice versa, at the rate of 10^{-5} per locus and generation.

The Weak-Selection Approximation

In the above form and with more than two loci per species, the model is analytically intractable and can only be investigated by numerical simulations. However, considerable simplifications can be achieved by assuming that selection is weak relative to recombination, but still strong relative to mutation. In this case, linkage disequilibria and mutation can be neglected, and the evolutionary dynamics can be sufficiently described in terms of allele frequencies (e.g., Bürger 2000), using the standard equations

$$\frac{dp_i}{dt} \approx \frac{p_i(1 - p_i)}{2} \frac{\partial \bar{w}_x}{\partial p_i} \quad \text{and} \tag{4a}$$

$$\frac{dq_j}{dt} \approx \frac{q_j(1 - q_j)}{2} \frac{\partial \bar{w}_y}{\partial q_j}, \tag{4b}$$

where p_i is the frequency of allele 1 at the ith locus of species X and q_j the frequency of allele 1 at the jth locus of species Y. $\bar{w}_x$ and $\bar{w}_y$ denote the mean fitness of the two species. Approximating the Gaussian functions in equations (1) and (2) by quadratics and neglecting terms of quadratic and higher order in σ and γ, the mean fitness values can be written as

$$\bar{w}_x = 1 - (\gamma_x + \sigma_x)\{[\bar{x} - \tilde{\theta}_x(\bar{y})]^2 + G_x + G_y\} + \cdots \tag{5a}$$

and

$$\bar{w}_y = 1 - (\gamma_y + \sigma_y)\{[\bar{y} - \tilde{\theta}_y(\bar{x})]^2 + G_x + G_y\} + \cdots \tag{5b}$$

for species X and Y, respectively. Here, the phenotypic means and variances are given by $\bar{x} = x_m + 2\Sigma_i \alpha_i(p_i - 1/2)$ and $G_x = 2\Sigma_i \alpha_i^2 p_i(1 - p_i)$ for species X and by analogous expressions for species Y. Dots stand for terms that do not depend on the population genetic state of the two species. The variables

$$\tilde{\theta}_x(\bar{y}) = \theta_x + \frac{\gamma_x}{\gamma_x + \sigma_x}(\bar{y} - \theta_x) \quad \text{and} \tag{6a}$$

$$\tilde{\theta}_y(\bar{x}) = \theta_y + \frac{\gamma_y}{\gamma_y + \sigma_y}(\bar{x} - \theta_y) \tag{6b}$$

represent the phenotypic values at which the mean fitness of a species has an extremum, which can be a maximum or a minimum depending on the sign of $\gamma + \sigma$. Thus, equations (5) and (6) state that both species are subject to quadratic selection with respect to a phenotype that is a weighted mean of the phenotypes maximizing or minimizing fitness with regard to the two selection components (direct stabilizing selection and selection due to the between-species interaction). Equations (6a,b) are undefined for $\gamma + \sigma = 0$, in which case there is no net selection at all.

Inserting equations (5a,b) into equations (4a,b) and per-

forming some algebraic manipulations yields the dynamics of allele frequencies under the weak-selection approximation:

$$\frac{dp_i}{dt} = p_i(1 - p_i)\alpha_i^2(\gamma_x + \sigma_x)\left[2p_i - 1 - 2\frac{\bar{x} - \tilde{\theta}_x(\bar{y})}{\alpha_i}\right] \quad (7a)$$

and

$$\frac{dq_j}{dt} = q_j(1 - q_j)\beta_j^2(\gamma_y + \sigma_y)\left[2q_j - 1 - 2\frac{\bar{y} - \tilde{\theta}_y(\bar{x})}{\beta_j}\right]. \quad (7b)$$

Here, the interspecific interaction enters only through the mean phenotypes of the two species.

It is also illuminating to consider the evolution of mean phenotypes. By summing up equations (7a) and (7b) with appropriate weights, one finds that

$$\frac{d\bar{x}}{dt} = 2G_x(\gamma_x + \sigma_x)[\tilde{\theta}_x(\bar{y}) - \bar{x}] - (\gamma_x + \sigma_x)M_{3,x} \quad \text{and} \quad (8a)$$

$$\frac{d\bar{y}}{dt} = 2G_y(\gamma_y + \sigma_y)[\tilde{\theta}_y(\bar{x}) - \bar{y}] - (\gamma_y + \sigma_y)M_{3,y}, \quad (8b)$$

where the M_3 values are the third central moments of the phenotypic distribution, which measure asymmetry (cf., Barton and Turelli 1987). Note, however, that equations (8a,b) cannot be used directly to study the long-term dynamics of the mean phenotypes, because both the genetic variances and the third moments change over time.

Two previous models can be derived as special cases of the weak-selection approximation. First, the one-locus case of equations (7a,b) with the simplifying assumption $x_m = y_m = \theta_x = \theta_y$ is structurally identical to a haploid model for the evolution of Batesian mimicry analyzed by Gavrilets and Hastings (1998). Second, taking equations (8a,b) and making the common assumptions that genetic variances G_x and G_Y are constant and the phenotypic distributions symmetric (i.e., $M_{3,x} = M_{3,y} = 0$) leads to the quantitative genetic model by Gavrilets (1997a).

Analysis

To analyze the dynamic behavior of the multilocus model, we used two complementary approaches. First, we studied the weak-selection approximation (7). To get analytical results concerning the (local) stability of equilibria, we made the simplifying assumption that the phenotypic effects of all loci within a species are identical ($\alpha_i = \alpha$, $\beta_j = \beta$ for all *i, j*). Details of this analysis are given in Appendices 1 and 2 (available online only at http://dx.doi.org/10.1554/05-581.1.s1 and http://dx.doi.org/10.1554/05-581.1.s2, respectively). Second, we performed numerical simulations of the exact model, using the nonapproximated fitness functions (1) and (2) together with the recursion (3). For comparison, we also ran some simulations based on the weak-selection approximation (7) with an added term for mutation.

Results

Types of Selection and the Evolution of Phenotypic Means and Variances

Before analyzing in detail the coevolutionary dynamics arising from various ecological scenarios, we use the weak-selection approximation to derive some general predictions about the evolution of phenotypic means and variances.

In general, the evolution of mean phenotypes reflects a balance between direct stabilizing selection and the selection pressures arising from the interspecific interaction. For internal equilibria (i.e., equilibria with intermediate mean phenotypes), the mean phenotypic distance between the two species can be approximated from equations (8a,b) if one makes the simplifying assumption that phenotypic distributions are symmetric (cf., Gavrilets 1997a). Then, at equilibrium, the difference in the mean phenotypes relative to the difference in the physiological optima is

$$\frac{|\bar{x} - \bar{y}|}{|\theta_x - \theta_y|} = \frac{1}{|1 + \gamma_x/\sigma_x + \gamma_y/\sigma_y|}. \quad (9)$$

This result shows that the mean phenotypes of mutualists tend to converge and those of competitors to diverge. The mean phenotypes in a victim-exploiter interaction diverge or converge, depending on whether the relative strength of selection arising from the interaction is stronger in the victim or in the exploiter (i.e., whether $|\gamma_x|/\sigma_x$ is smaller or larger than γ_y/σ_y).

The evolution of phenotypic variances depends on the type of net selection experienced by the two species, which can be seen from equations (5) and (6). Mutualists and exploiters (which have positive γ values) are always under net stabilizing selection, which tends to remove genetic variation (Wright 1935; Barton 1986; Spichtig and Kawecki 2004). Their mean fitness decreases with the deviation from the corresponding fitness maximum θ and with genetic variance. (Note the difference between direct stabilizing selection, which refers to a fitness component, and net stabilizing selection, which refers to the overall fitness function.) For the same reason, competitors and victims (which have negative γ values) are under net stabilizing selection if $|\gamma| < \sigma$, but under net disruptive selection in the opposite case. Net disruptive selection tends to increase genetic variation (e.g., Bürger and Gimelfarb 2004; Spichtig and Kawecki 2004; Bürger 2005). Finally, each species can be under net directional selection if the respective fitness maximum or minimum is outside of the range of phenotypes currently present in the population. Net directional selection drives mean phenotypes toward extreme values, that is toward the edge of the phenotypic range, where genetic variation is destroyed.

Coevolution of Competitors

In a competitive interaction (i.e., when $\gamma_x, \gamma_y < 0$), the mean phenotypes of the two species tend to diverge (eq. 9), leading to ecological character displacement. The two species are either under net directional selection (if $|\gamma_x| > \sigma_x, |\gamma_y| > \sigma_y$) or under net stabilizing selection (if the above conditions are reversed). Analysis of the weak-selection approximation with equal locus effects shows that the resulting equilibrium is always stable. Species under net directional selection evolve to the extreme phenotype that is furthest away from the phenotype of the other species (see Fig. 1A). Species under net stabilizing selection evolve to an intermediate mean phenotype and are polymorphic in no more than one locus. A slight complication occurs if both species

TABLE 1. The three types of stable equilibria for victim-exploiter coevolution. The table shows the characteristics of the equilibria with respect to the victim. The exploiter is always under net stabilizing selection and has an intermediate mean phenotype with low phenotypic variance.

	EQ_{STA}	EQ_{DIS}	EQ_{DIR}
Net selection in victim	stabilizing	disruptive	directional
Victim mean phenotype $\bar{x}$	intermediate	intermediate	extreme
Victim phenotypic variance G_x	low or zero	high	zero
Intuitive explanation	victim dominated by stabilizing selection	victim trapped	victim escaped

are under net stabilizing selection, but direct stabilizing selection is relatively weak. In this case, at most one species can be polymorphic at equilibrium, and the resulting equilibrium is not necessarily unique (for further details, see online Appendix 2).

Coevolution of Mutualists

In a mutualistic interaction (i.e., when $\gamma_x, \gamma_y > 0$), the mean phenotypes of the two species tend to converge (eq. 9), while both species are subject to net stabilizing selection (see Fig. 1B). Analysis of the weak-selection approximation with equal locus effects shows that the system evolves to a stable equilibrium at which each species is polymorphic in no more than one locus. If direct stabilizing selection is relatively weak in both species, at most one species can be polymorphic at equilibrium, and the resulting equilibrium state is not necessarily unique (for further details, see online Appendix 2).

Coevolution between Victim and Exploiter

Victim-exploiter coevolution is considerably more complex than coevolution between mutualists or competitors. We first present results for the weak-selection approximation with equal locus effects (both analytical and numerical) and later analyze situations with strong selection and unequal locus effects (numerically). In all of the following, we assume that species X is the victim and species Y the exploiter (i.e., $\gamma_x < 0, \gamma_y > 0$).

Weak selection and equal locus effects

Analysis of the weak-selection approximation with equal locus effects shows that victim-exploiter coevolution can lead to four qualitatively different dynamic regimes: three types of equilibria (characterized by the type of net selection acting on the victim and referred to as EQ_{STA}, EQ_{DIS}, and EQ_{DIR}, respectively; Table 1) plus coevolutionary cycles. These regimes are qualitatively similar to those found in the one-locus case. We will present them in the same order as in Figure 1, which reflects increasing success of the victim at evolutionary escape from the exploiter.

Equilibria with net stabilizing selection in the victim (EQ_{STA}).—The first type of stable equilibria occurs if, in the victim, direct stabilizing selection is stronger than selection due to the between-species interaction (i.e., $|\gamma_x| < \sigma_x$). In this case, both species are under net stabilizing selection (see Fig. 1B), and the system evolves to a stable equilibrium, where the mean phenotypes of the two species are closer together than the respective physiological optima if $|\gamma_x|/\sigma_x < \gamma_y/\sigma_y$ and further apart from each other otherwise. At the equilibrium, each species is polymorphic in no more than one locus (similar to a mutualistic system). Unlike in the cases of mutualism and competition, the equilibrium is always unique. Gradual variation of parameters affecting the physiological optima results in an alternation of monomorphic and polymorphic states in both species (see online Appendix 2 and Figure A1 for additional details).

The other three dynamic regimes occur if direct stabilizing selection in the victim is weaker than selection due to the between-species interaction ($|\gamma_x| > \sigma_x$).

Equilibria with net disruptive selection in the victim (EQ_{DIS}).—At the second type of equilibria, the mean phenotypes of both species are intermediate, the victim is polymorphic at all L_x loci (and has equal allele frequencies at them), and the exploiter is polymorphic at a single locus. This reflects net disruptive selection in the victim and net stabilizing selection in the exploiter (see Fig. 1C). An equilibrium of this type is stable if the following two conditions are met:

$$\frac{\gamma_y}{\sigma_y} > \frac{|\gamma_x| - \sigma_x}{\frac{2L_x + 1}{2L_x - 1}|\gamma_x| + \sigma_x} \quad \text{and} \tag{10a}$$

$$R \equiv \frac{G_x|\gamma_x + \sigma_x|}{G_y|\gamma_y + \sigma_y|} < \frac{L_x}{2L_x - 1}, \tag{10b}$$

where the genetic variances $G_x = 2L_x\alpha^2 p(1 - p)$ and $G_y = 2\beta^2 q(1 - q)$ are evaluated at equilibrium. Condition (10a) states that the exploiter must not be too constrained by direct stabilizing selection. From equations (8a,b), the composite parameter R can be interpreted as the ratio of potential evolutionary rates of the two species. Therefore, condition (10b) states that, close to the equilibrium, the exploiter must be able to evolve faster than the victim. Using the arms-race metaphor, one might say that the victim is evolutionarily trapped and cannot escape the exploiter. At the equilibrium, the victim's mean phenotype is close to a fitness minimum, which is stabilized by a negative feedback between the evolutionary dynamics of the two species, that is, by (between-species) frequency-dependent selection (Abrams and Matsuda 1997). Stable fitness minima are closely related to the ''evolutionary branching points'' described by the theory of adaptive dynamics (e.g., Geritz et al. 1998; reviewed by Waxman and Gavrilets 2005). As shown in Appendix 2 (available online), there can be up to L_y such equilibria, and several of them can be stable simultaneously.

These results yield several insights into the effects of multilocus genetics on victim-exploiter coevolution. First, with

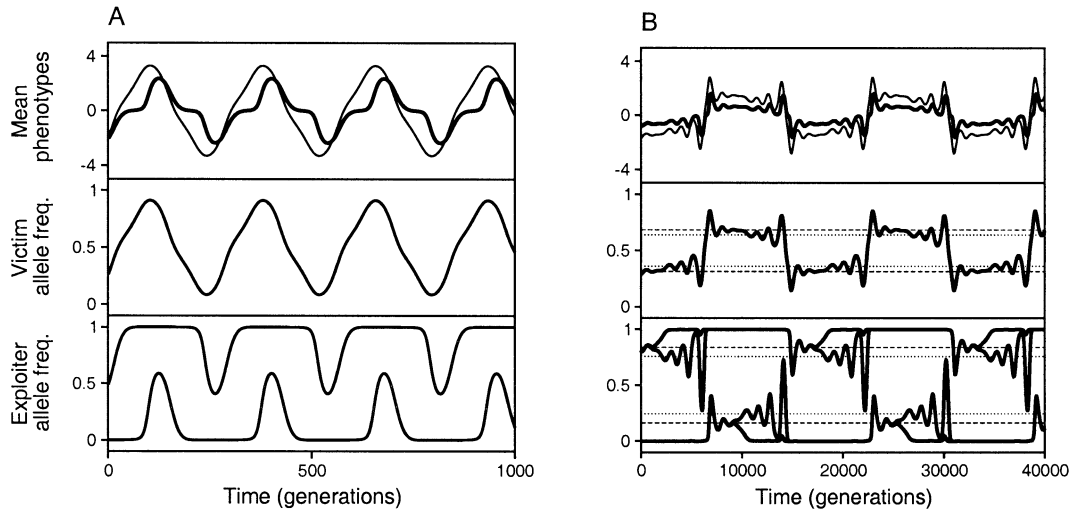

FIG. 2. Heteroclinic cycles in a four-locus system with symmetric stabilizing selection and equal locus effects within and across species. (A) Simple cycles. Top panel: victim (thin line) and exploiter (thick line) mean phenotypes; middle panel: victim allele frequencies (equal in all loci); bottom panel: exploiter allele frequencies (two pairs of loci with nearly identical allele frequencies within each pair). Note that allele frequencies cycle synchronously in the victim but asynchronously in the exploiter. Therefore, the cycles in the exploiter's mean phenotypes do not span the entire phenotypic range. (B) Complex cycles. The system periodically approaches equilibria with one (dashed lines) and two (dotted lines) polymorphic exploiter loci. Oscillations around equilibria with one polymorphic exploiter locus are diverging, because the victim can evolve faster than the exploiter (i.e., condition 10b is fulfilled). Oscillations around equilibria with two polymorphic exploiter loci are converging, because with two polymorphic loci, the exploiter can evolve faster than the victim (i.e., the generalized condition A2–13 holds; see online Appendix 2). However, as shown in online Appendix 1, the two-locus polymorphism is unstable and can be maintained only temporarily. Parameters: (A) $\gamma_x = -0.02$, $\sigma_x = 0.0$, $\gamma_y = 0.03$, $\sigma_y = 0.0$; (B) $\gamma_x = -0.003$, $\sigma_x = 0.0015$, $\gamma_y = 0.006$, $\sigma_y = 0.003$;, both plots $L_x = L_y = 4$, $\theta_x = \theta_y = 0$, $x_m = y_m = 0$, $\alpha = \beta = 1$.

multiple loci, the equilibrium genetic variance of the victim is typically much larger than that of the exploiter (unless $\alpha \ll \beta$), and this increases the mean fitness of the victim (see eq. 5). Second, multilocus genetics often foster instability. Fulfillment of condition (10a) becomes less likely as L_x increases. Fulfillment of condition (10b) depends not only on L_x and L_y, but also on the locus effects α and β. If the locus effects are scaled such as to keep the phenotypic range constant (i.e., $\alpha = 1/L_x$, $\beta = 1/L_y$), then increasing L_x alone will increase stability, but increasing L_x and L_y together will decrease stability. If, instead, the locus effects are fixed (such that the phenotypic range increases with the number of loci), then increasing L_x decreases stability; whereas increasing L_y has no effect. Third, compared to the one-locus case, multilocus genetics significantly increase complexity, as evidenced by the existence of multiple simultaneously stable equilibria.

Coevolutionary cycles.—Coevolutionary cycles can be interpreted as temporary 'escapes' of the victim. We do not have analytical results for this regime, but instead investigated it by simulations. As in the one-locus case, there are two qualitatively different types of cycles: stable limit cycles (Fig. 1D) and heteroclinic cycles (Fig. 1E).

Stable limit cycles are small-scale oscillations centered around equilibria with net disruptive selection in the victim (EQ_{DIS}). Allele frequencies at all victim loci perform (synchronized) cycles, whereas only one locus cycles in the exploiter. In our simulations, stable limit cycles were observed very rarely. A likely explanation can be gained from the one-locus case. Using the results from Gavrilets and Hastings (1998), it can be shown that stable limit cycles are possible only if the phenotypic range of the exploiter is sufficiently large (often larger than that of the victim). This condition is unlikely to be fulfilled in the multilocus case, where the effective phenotypic range of the exploiter is reduced by the fixation of all loci but one.

Therefore, most coevolutionary cycles are heteroclinic cycles, which typically have a large amplitude. Heteroclinic cycles are defined as cycles that temporarily approach unstable equilibria (e.g., at the edge of the phenotypic range). In the one-locus case, a heteroclinic cycle is a simple chase between extreme phenotypes. A similar behavior is shown for the multilocus case in Figure 2A. However, because the allele frequencies in the exploiter loci do not always cycle synchronously, the cycles in the mean phenotypes do not necessarily span the entire phenotypic range of the two species (Fig. 2A) and a considerable amount of genetic variation can be maintained (see below, Fig. 8).

In other cases, heteroclinic cycles temporarily approach unstable equilibria with intermediate mean phenotypes. The basic pattern can be best seen in cases where selection is very weak. In the simulation shown in Figure 2B, the system alternately approaches unstable equilibria where the exploiter has one or two polymorphic loci, respectively, leading to second- and higher order oscillations. In the following, cycles of this kind will be referred to as ''complex'' heteroclinic cycles, as opposed to ''simple'' cycles like those shown in Figure 2A. We observed complex cycles only in simulations with more than two exploiter loci.

Equilibria with net directional selection in the victim (EQ_{DIR}).—Finally, the victim can permanently escape to an extreme phenotype, where the exploiter cannot follow (see

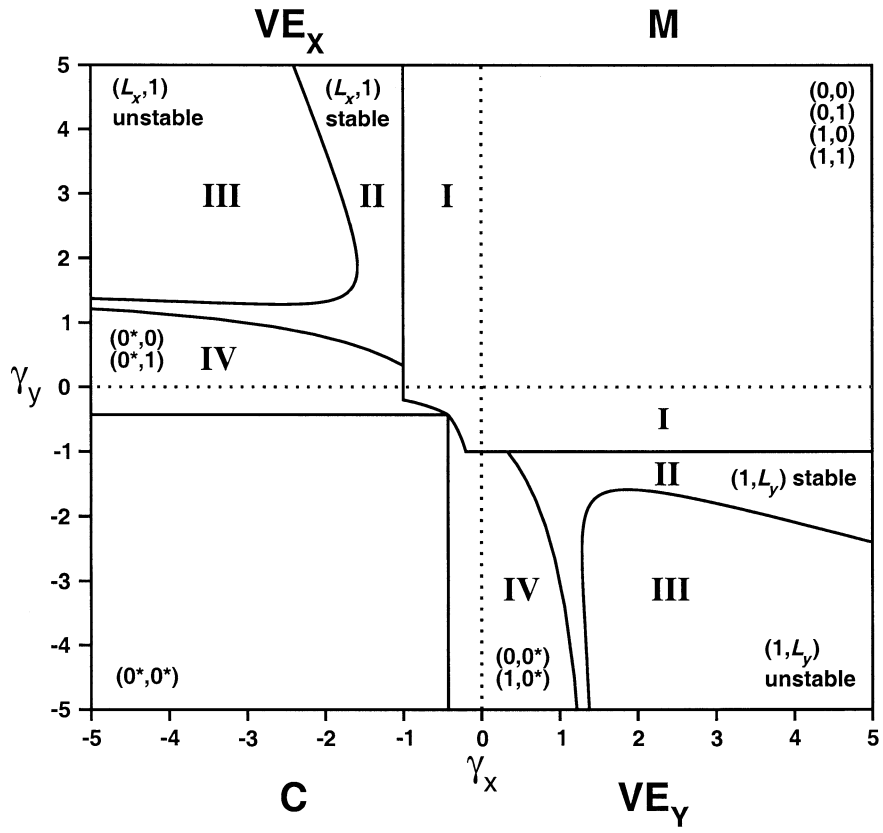

FIG. 3. Possible outcomes of coevolution in a two-locus system ($L_x = L_y = 2$) with symmetric stabilizing selection ($\theta_x = \theta_y = x_m = y_m$) and equal locus effects within and across species ($\alpha = \beta = 1$), shown as a function of the parameters γ_x and γ_y (and assuming $\sigma_x = \sigma_y$). Dotted lines delimit areas corresponding to different ecological interactions (marked at the top and bottom margins): C, competition; M, mutualism; VE_X, victim exploiter interaction with species X being the victim; and VE_Y, victim exploiter interaction with species Y being the victim. Solid lines delimit areas with qualitatively different types of equilibria. These equilibria are characterized by the numbers of polymorphic loci in species X and species Y, (m, n). A 0* means that a species is monomorphic and all loci are fixed for the same allele, leading to an extreme phenotype. Finally, Roman numerals mark the areas corresponding to the four dynamic regimes possible in victim-exploiter interactions: (I) stable equilibria with net stabilizing selection in the victim (EQ_{STA}); (II) stable equilibria with net disruptive selection in the victim (EQ_{DIS}); (III) coevolutionary cycles; and (IV) stable equilibria with net directional selection in the victim (EQ_{DIR}). Note that marks I to IV apply only to victim-exploiter interactions, that is, only to the upper-left and lower-right quadrants (delimited by the dotted lines).

Fig. 1F). That is, the system evolves to a stable equilibrium at which the victim is monomorphic at an extreme mean trait value and the exploiter is either monomorphic or polymorphic at a single locus, with a mean phenotype less extreme than that of the victim. At the equilibrium, the victim experiences net directional selection, and the exploiter experiences either net directional or net stabilizing selection. The exploiter fails to match the victim trait more closely due to one of three mechanisms. First, the exploiter may be constrained by direct stabilizing selection (small γ_y/σ_y). Second, the extreme victim phenotype, say $x_{\max}$, may be outside the range of the exploiter's trait values (which implies either $\alpha > \beta$ or $x_m > y_m$). Third, the difference between possible exploiter phenotypes may the larger than that between possible victim phenotypes ($\beta > \alpha$). As net stabilizing selection tends to draw the mean exploiter trait value toward the nearest trait of a homozygote (see online Appendix 2), this can lead to a stable $\bar{y}$ that is considerably smaller than $x_{\max}$.

Figure 3 gives an overview of the behavior of the weak-selection approximation with equal locus effects as a function of the parameters γ_x and γ_y for the simplest case with two loci per species and symmetric stabilizing selection. Figure 4 presents the results of simulations demonstrating the effects of increasing the number of loci (while keeping the phenotypic range constant). The plots show the frequency distribution of dynamic regimes as a function of γ_y (assuming $|\gamma_x| > \sigma_x$). Each simulation was run for 10,000 generations and repeated 10 times with random initial allele frequencies. As predicted by our analytical results, increasing the number of loci decreases stability. Furthermore, Figure 4 compares simulations using the weak-selection approximation (7) with those using the exact model (eqs. 1–3). As long as selection is moderately weak, system (7) is a good approximation to the exact model. The slight differences in the two approaches are probably due to the explicitely discrete time steps in the exact model. In Appendix 3 (available online), we analyze the haploid version of the weak-selection approximation with equal locus effects. The behavior of the haploid model is generally similar to that of the diploid model. The most significant difference is that equilibria with net disruptive selection in the victim (EQ_{DIS}) can never be stable.

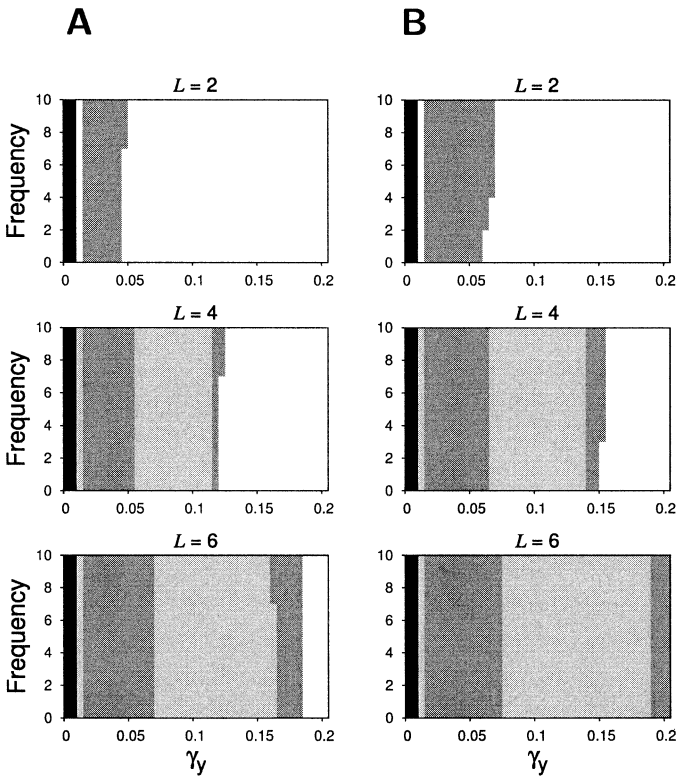

FIG. 4. The influence of the number of loci in the two species ($L_x = L_y = L$) on victim-exploiter coevolution with symmetric stabilizing selection and equal locus effects within and across species ($\alpha = \beta = 1/L$). Each plot shows the frequency of different dynamic regimes in 10 replicated simulations with random initial conditions as a function of the parameter γ_y. White, stable equilibrium with disruptive selection in the victim (EQ_{DIS}); light gray, complex heteroclinic cycles (complex cycles where defined as cycles showing second- or higher order oscillations and were determined visually); dark gray, simple heteroclinic cycles; black, stable equilibrium with directional selection and extreme phenotype in the victim (EQ_{DIR}). (A) Numerical solutions of the weak-selection approximation (7). (B) Simulations based on the exact model (1) to (3). Increasing the number of loci decreases stability. Stability is slightly more likely in the weak-selection approximation than in the exact model. Other parameters were $\sigma_x = \sigma_y = 0.01$, $\gamma_x = -0.02$, $\theta_x = \theta_y = x_m = y_m = 0$.

Strong selection and unequal locus effects

We relax the assumptions from the previous section and allow for strong selection and unequal locus effects. All of the following results are based on simulations using equations (1) to (3). Our focus is on a four-locus system ($L_x = L_y = 4$) with symmetric stabilizing selection ($\theta_x = \theta_y = x_m = y_m = 0$).

Unequal locus effects.—In nature, the phenotypic effects of different loci will never be completely equal. Unequal locus effects increase the number of phenotypes and, therefore, the number of possible equilibria. This has several consequences. First, in species under net stabilizing selection, alternative equilibria with similar mean phenotypes (close to the $\tilde{\theta}$ values) may be stable simultaneously (see Barton 1986; Bürger and Gimelfarb 1999). Second, in victims experiencing net disruptive selection, the equilibrium allele frequencies generally are no longer identical, and loci with weak effects may become fixed for one of the two alleles (see Bürger and Gimelfarb 2004; Spichtig and Kawecki 2004; Bürger 2005). Finally, both simple and complex heteroclinic cycles are less regular than those shown in Figure 2, more than one type of either class of cycles can coexist for the same parameter values, and complex cycles can be truly chaotic. In some cases, complex cycles were transient, and we also observed cases of intermittency (alternation of chaotic and nonchaotic behavior) and transient chaos, similar to those described by Gavrilets and Hastings (1995) for frequency-dependent selection in single populations. Besides these differences, however, our simulations did not show any changes in the nature of the dynamic regimes described in the previous section. For example, we did not observe novel types of stable equilibria (such as equilibria with more than one polymorphic locus in the exploiter).

Unequal locus effects do, however, influence the prevalence of the various regimes and the sensitivity of the system to initial conditions. This is demonstrated in Figure 5, which shows the frequency distribution of dynamic regimes as a function of γ_y (assuming $|\gamma_x| > \sigma_x$) for 16 combinations of locus effects. Several results are noteworthy. First, unequal locus effects in the exploiter have a larger effect on coevolutionary dynamics than unequal locus effects in the victim. Second, increasing the difference between locus effects in the exploiter increases the likelihood of stable equilibria with disruptive selection in the victim (EQ_{DIS}) and of complex heteroclinic cycles. Most likely, this is because of the increase in the effect of the strongest exploiter locus, which leads to an increase in the genetic variance of the exploiter if this locus is polymorphic. Third, intermediate differences between the exploiter loci dramatically increase the dependence of the dynamics on initial conditions (i.e., the coexistence of various regimes under a given set of parameters). We have to note, though, that these effects appear to be less obvious in cases with stronger selection in the victim (results not shown). However, the interaction between unequal locus effects and the strength of selection is something we have not investigated in detail.

Strong selection.—While selection in natural populations is often found to be weak (Kingsolver et al. 2001), it can arguably be quite strong in specialized victim-exploiter interactions. In Figure 5, the range of γ_y already includes rather strong selection in the exploiter. However, results qualitatively similar to those in Figure 5 can be obtained by dividing all selection coefficients and the mutation rates by 10 (not shown). Therefore, the effects of high γ_y are due to the relative strength of selection in the two species, not to strong selection per se. In the following simulations, we investigated the effect of strong selection in the victim.

We started by investigating the conditions for a stable equilibrium with net stabilizing selection in the victim (EQ_{STA}). Under the weak-selection approximation, this equilibrium is stable whenever $\sigma_x > |\gamma_x|$. However, for $|\gamma_x| > 0.1$, the critical value of σ_x is significantly increased (Fig. 6). For σ_x below this critical value, the dynamics are dominated by simple heteroclinic cycles.

Next, we investigated the effect of strong selection on the coevolutionary dynamics if $|\gamma_x| > \sigma_x$. Figure 7 shows the

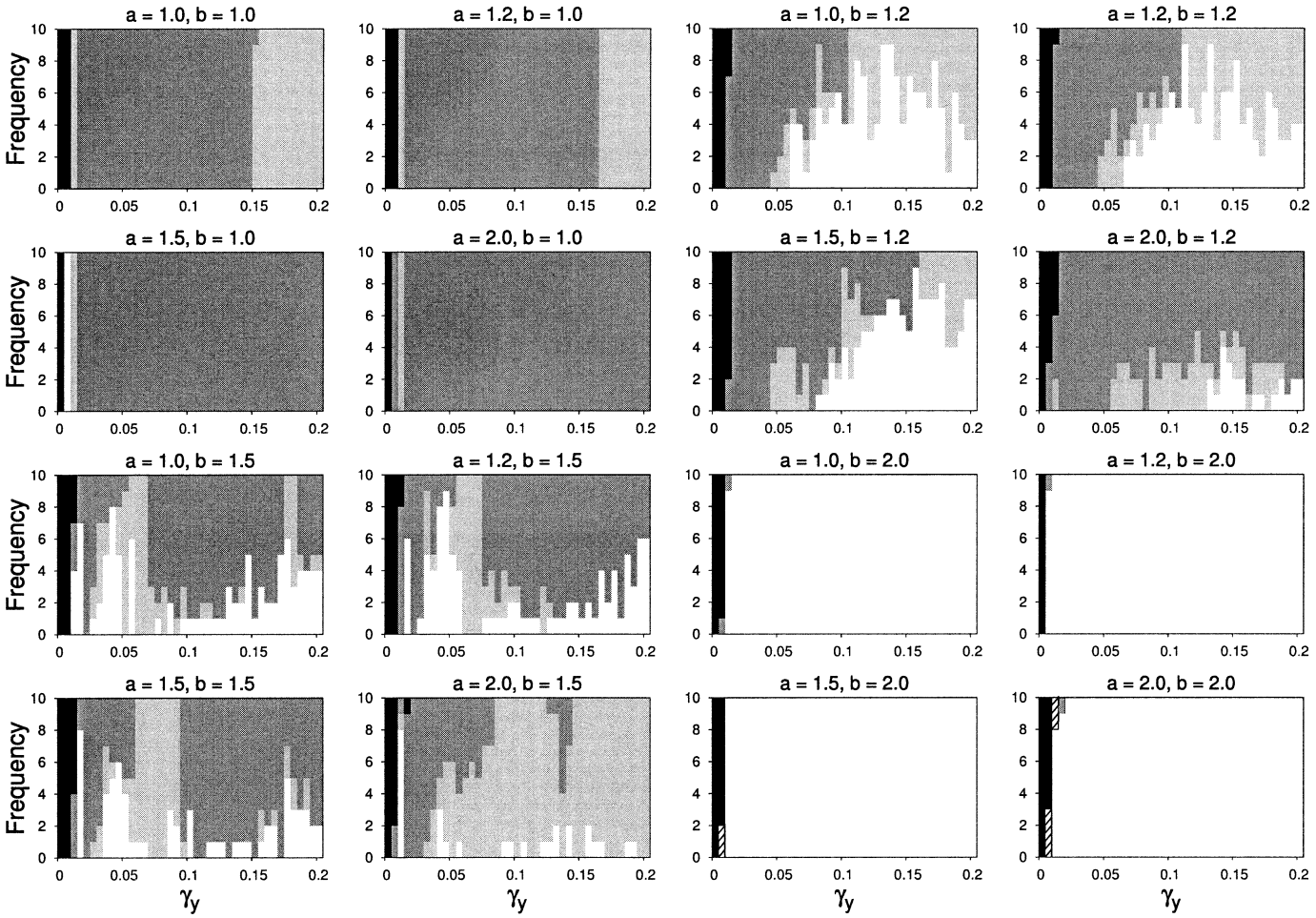

FIG. 5. The influence of unequal locus effects on victim-exploiter coevolution in a four-locus system. The color code is as in Figure 4. In addition, the hatched area signifies stable limit cycles. Plot titles indicate the locus effects, with *a* and *b* specifying the degree to which locus effects in the victim (*a*) and exploiter (*b*) are unequal. For example, in the victim, $a = 1$ stands for equal locus effects and $a = 2$ for highly unequal locus effects. More precisely, the mean locus effect is always equal to 1, and *a* specifies the ratio of the effects of adjacent loci. Thus, $a = 1$ leads to $\alpha_i = [1, 1, 1, 1]$; $a = 1.2$ to $\alpha_i = [0.745, 0.894, 1.073, 1.288]$; $a = 1.5$ to $\alpha_i = [0.492, 0.738, 1.108, 1.662]$; and $a = 2$ to $\alpha_i = [0.267, 0.533, 1.067, 2.133]$ (analogous for *b* and β_i's values). Unequal locus effects in the exploiter increase the likelihood of stable intermediate equilibria and complex coevolutionary cycles. Intermediate differences between locus effects increase the sensitivity of the system to initial conditions. Other parameters were $\sigma_x = \sigma_y = 0.01$, $\gamma_x = -0.02$, $\theta_x = \theta_y = x_m = y_m = 0$, $L_x = L_y = 4$.

distribution of dynamic regimes as a function of γ_y for increasing absolute values of γ_x. In Figure 7A, $\sigma_x = 0.01$ is held constant, whereas in Figure 7B, $\sigma_x = |\gamma_x| - 0.01$ increases with γ_x. The locus effects within both species are slightly different from each other, which generally favors stability (see above). The simulations show that stable equilibria with net disruptive selection in the victim (EQ_{DIS}) and complex cycles occur only if $|\gamma_x|$ is relatively small (<0.1) and σ_x is not much less than $|\gamma_x|$ (i.e., selection in the victim is weak overall and the two components of selection are almost equally strong). With even moderately strong selection in the victim, the system invariably shows either equilibria with net directional selection in the victim (EQ_{DIR}) or simple heteroclinic cycles, depending on the strength of selection in the exploiter. Asymmetric stabilizing selection (e.g., $\theta_x = -2$, $\theta_y = 2$) slightly increases the likelihood of stability and complex cycles, but does not alter the general conclusions (results not shown).

In summary, strong selection in the victim has much stronger effects on coevolutionary dynamics than strong selection in the exploiter. Most likely, this is because it is the victim that is running away and, thereby, setting the pace of the coevolutionary arms race. More precisely, with strong selection in the victim, the assumption of nonoverlapping generations becomes critical. Because of the large phenotypic changes between generations (a full cycle can be completed in about 10 generations), potentially stable intermediate equilibria are never approached closely enough to become attracting. Thus, by making simple heteroclinic cycles the predominant regime, strong selection in the victim considerably simplifies the coevolutionary dynamics.

Genetic variation.—Finally, we were interested in the amount of genetic variation maintained by coevolutionary cycling. In Figure 8, we show the average genetic variance over time in some of the simulations from Figure 7. Several conclusions can be drawn. First, for most parameter values

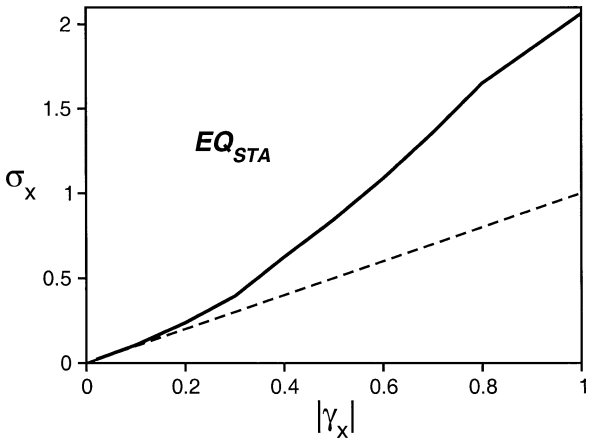

FIG. 6. The effect of strong selection in the victim on the prevalence of stable equilibria with net stabilizing selection in the victim (EQ_{STA}). For parameter combinations above the solid line, a EQ_{STA} equilibrium occurred in more than five of 10 replicated simulations with random initial allele frequencies (in most case, in all 10). The weak-selection approximation predicts that this regime prevails whenever $\sigma_x > |\gamma_x|$ (dotted line), but strong selection increases the value of σ_x necessary for its stability. For $|\gamma_x| > 0.1$ and σ_x below the solid line, the system mostly showed simple heteroclinic cycles. Parameters: $L_x = L_y = 4$, $\alpha = \beta = 1$ (equal locus effects), $\theta_x = \theta_y = x_m = y_m$, $\gamma_y = 2|\gamma_x|$ (identical results where found for $\gamma_y = 2$ = constant).

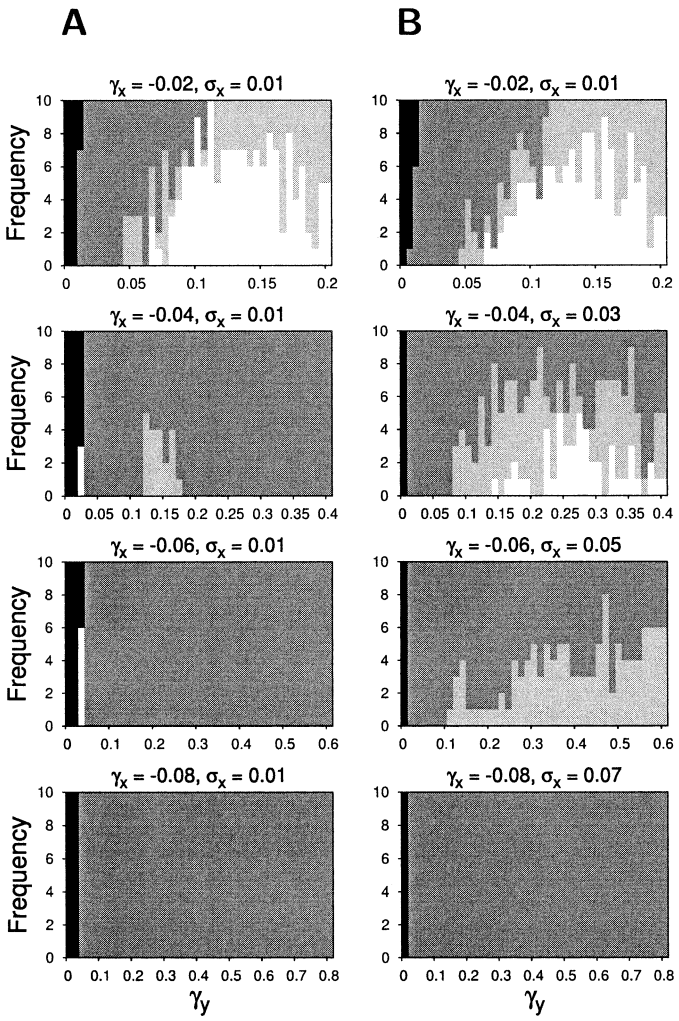

FIG. 7. The effect of the strength of selection in the victim on the coevolutionary dynamics of a four-locus victim-exploiter system. In (A) $\sigma_x = 0.01$ is constant, whereas in (B), $\sigma_x = |\gamma_x| - 0.01$ increases with $|\gamma_x|$. The color code is as in Figure 4. EQ_{DIS} equilibria and complex cycles are frequent only if selection in the victim is weak. In both species, the locus effects were slightly unequal with $\alpha_i = \beta_i = [0.745, 0.894, 1.073, 1.288]$. Note that the scale of the horizontal axes varies, because γ_y always ranges from 0 to $10|\gamma_x|$. Other parameters were $\gamma_x = -0.02$, $\theta_x = \theta_y = x_m = y_m = 0$, $L_x = L_y = 4$.

(unless γ_y is small), genetic variance is much larger in the victim than in the exploiter. This is, of course, in accordance with our analytical results. Second, the average genetic variance is lower for coevolutionary cycles than for stable equilibria with disruptive selection in the victim (EQ_{DIS}). This is because, during cycles, the population periodically approaches states near the edge of the phenotypic range, where genetic variance is low. There is no significant difference in the amount of genetic variance maintained during simple and complex heteroclinic cycles. Third, average genetic variance during coevolutionary cycles in the victim increases with the potential evolutionary rate of the exploiter (i.e., with γ_y) and decreases with the potential evolutionary rate of the victim (i.e., it decreases with $|\gamma_x|$ and increases with σ_x). This is because, with a relatively fast exploiter, the victim can spend less time at extreme phenotypes (because the exploiter catches up more quickly). For low $|\gamma_x|$ and high γ_y, the genetic variance in the victim during cycles almost matches that at equilibria with disruptive selection (which, in turn, is close to the maximum genetic variance at linkage equilibrium). In contrast to the situation in the victim, the average genetic variance in the exploiter slightly decreases with both $|\gamma_x|$ and γ_y, probably because, for high values of these parameters, the exploiter gets closer to its phenotypic extremes.

DISCUSSION

The primary goal of this study has been to investigate the effects of explicit multilocus genetics in models of coevolution involving quantitative traits. For this purpose, we developed and analyzed a multilocus model of two coevolving species, which may play the roles of mutualists, competitors, or victim and exploiter. This model can be compared to two previously published analyses: a quantitative genetic model by Gavrilets (1997a), which assumes constant genetic variances and symmetric phenotypic distributions, and a one-locus model by Gavrilets and Hastings (1998). The three models differ in their assumptions about the genetic basis of the coevolving traits, but they consider the same ecological scenario (i.e., the same fitness functions): two species interacting via a pair of quantitative traits, which are also subject to direct stabilizing selection.

All three models make similar qualitative predictions about the outcome of coevolution. This shows that the basic behavior of the system is unaffected by genetic details. In particular, the models predict that the trait values of mutualists converge and those of competitors diverge, in both cases reaching an equilibrium with low genetic variation. Coevolution in victim-exploiter systems can lead to four qualita-

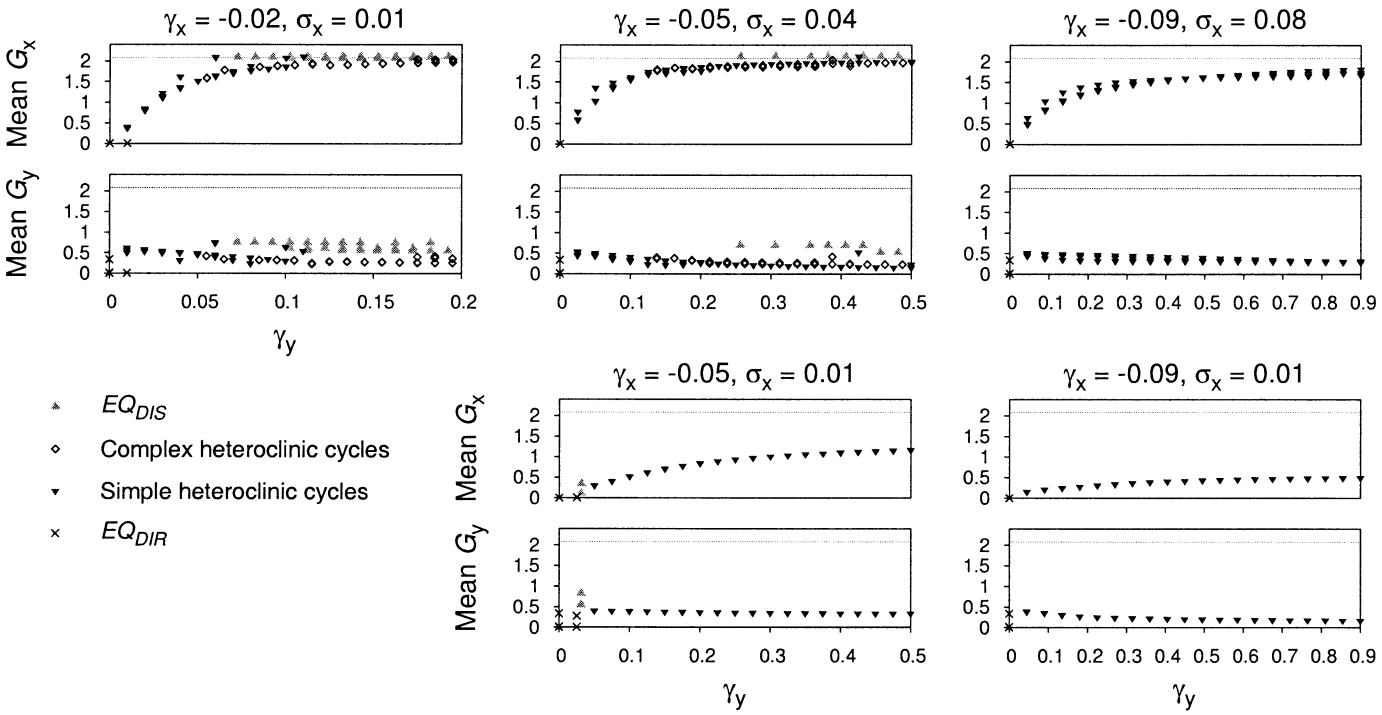

FIG. 8. The maintenance of genetic variation by victim-exploiter coevolution if $|\gamma_x| > \sigma_x$. Each datapoint shows the mean value of genetic variance for victim (G_x) and exploiter (G_y), respectively, taken over the course of one simulation run. For comparison, the horizontal dashed lines mark the maximum genetic variance possible at linkage equilibrium. In general, genetic variance is much higher in the victim than in the exploiter. Data are from the simulations shown in Figure 7. The symbols for EQ_{STA} equilibria and complex heteroclinic cycles have been shifted slightly to the right to reduce overlap.

tively different dynamic regimes. These regimes differ in how successful the victim is at evolutionarily escaping from the exploiter, which in turn depends on the strength of direct stabilizing selection in the two species and on their relative potential evolutionary rates. If the victim is strongly constrained by direct stabilizing selection, both species approach stable equilibria with intermediate trait values and low genetic variation (EQ_{STA}). If neither species is strongly constrained by direct stabilizing selection, the system may reach a stable equilibrium with disruptive selection and high genetic variation in the victim (EQ_{DIS}). Stability of this equilibrium requires that the exploiter is able to evolve faster than the victim (see also Dieckmann et al. 1995; Marrow et al. 1996; Gavrilets 1997a; Gavrilets and Hastings 1998; Doebeli and Dieckmann 2000). Alternatively, victim and exploiter may engage in coevolutionary cycles. Finally, if the exploiter is strongly constrained by direct stabilizing selection or by the set of available phenotypes, then the victim can escape to an extreme phenotype (EQ_{DIR}).

Quantitatively, however, the multilocus model makes predictions that cannot be obtained from either of the simpler models. In particular, the multilocus model yields four central new results. First, the coevolutionary systems may have multiple internal equilibria and simultaneously stable states (i.e., dependence on initial conditions). Second, genetic variances change dynamically, and they may evolve in opposite directions in victims and exploiters. Third, coevolutionary equilibria in victim-exploiter systems tend to be destabilized by increasing the number of loci contributing to the exploiter trait. Fourth, the resulting nonequilibrium dynamics are characterized by large-amplitude cycles, which can be either simple or complex. In the following, we will discuss these results and their implications in greater detail.

In the multilocus model, selection determines not only the mean trait value of a species but also its genetic variance (see Bürger and Gimelfarb 2004; Spichtig and Kawecki 2004; Bürger 2005). In particular, genetic variance depends on the type of net selection that the species experiences (where net selection refers to the combined effect of direct stabilizing selection and selection due to the between-species interaction). Net directional selection leads to an extreme mean trait value with zero genetic variance (competitors, victims at EQ_{DIR} equilibria, sometimes exploiters at EQ_{DIR} equilibria). Net stabilizing selection leads to an intermediate mean trait value with low genetic variance, that is polymorphism in no more than one locus (mutualists, typical competitors, victims at EQ_{STA} equilibria, exploiters). Finally, net disruptive selection leads to an intermediate mean trait value with high genetic variance, that is, polymorphism at all loci (victims under net disruptive selection at EQ_{DIS} equilibria or during coevolutionary cycles).

At equilibrium, populations under net stabilizing selection are either monomorphic or polymorphic at exactly one locus. Which of these states is reached depends on the location of the (overall) phenotypic optimum relative to the phenotypes of homozygotes (see online Appendix 2). Gradual variation of parameters affecting the phenotypic optimum results in an alternation of monomorphic and polymorphic states, which is a well-known result for weak stabilizing selection with quadratic fitness functions in single populations (Wright

1935; Barton 1986; Spichtig and Kawecki 2004). For the case of two coevolving species, which are both under net stabilizing selection, our results (Fig. A1) can be viewed as a two-dimensional extension to the these classical results.

In victim-exploiter systems where the exploiter is subject to net stabilizing selection and the victim is subject to net disruptive selection, the genetic variance in the two species evolves in opposite directions: it is maximized in the victim and (nearly) minimized in the exploiter. This effect has three important consequences. First, at equilibrium the mean fitness of the victim is increased even though its mean phenotype is close to a fitness minimum. The exploiter cannot counter this diversification and is itself trapped in the middle of the victim's phenotypic distribution—a situation termed ''Buridan's Ass regime'' by Gavrilets and Waxman (2002). Second, the high genetic variance of the victim increases its relative evolutionary rate, which frequently allows temporary or permanent escapes from the exploiter. In particular, multilocus genetics foster coevolutionary cycles, and the likelihood of cycles increases with the number of loci contributing to the coevolving traits. The appearance of coevolutionary cycles due to the evolution of genetic variances was also observed by Nuismer and Doebeli (2004) and Nuismer et al. (2005). Third, the fixation of alleles in the exploiter reduces its effective phenotypic range, and this destabilizes small-scale limit cycles and induces large-scale heteroclinic cycles. In most cases, these cycles are simple oscillations between low and high trait values (Fig. 2A). If selection in the victim is weak, the cycles can include more complex behavior, such as temporary approach of unstable equilibria and chaos (Fig. 2B).

It is important to note that the qualitative behavior of our model strongly depends on the bidirectional axis of vulnerability (sensu Abrams 2000), that is, on the assumption that the strength of the interspecific interaction depends only on the absolute distance between phenotypes and not on which species has the larger phenotype. This assumption is particularly critical in victim-exploiter interactions, where often ''more is better'' (such as with speed, strength). A bidirectional axis might apply with traits such as size or habitat choice, or whenever some sort of pattern matching or lock-and-key mechanism is involved. For example, pattern matching occurs in Batesian mimicry systems, where the mimic exploits the model, and in systems of brood parasitism, where the host must recognize parasite eggs (Soler et al. 2001). Lock-and-key mechanisms can be found in predators with specialized feeding morphologies. For example, the subspecies of crossbills studied by Benkman (1999) differ in the size and shape of their beaks. Each beak is specialized for feeding on the seeds of a particular conifer species, and no one beak morphology is optimal for exploiting all conifer species. Because a bidirectional axis of vulnerability is important for creating disruptive selection and, in general, is much more favorable for coevolutionary cycles than a unidirectional axis (Abrams 2000), the results of a multilocus model with a unidirectional axis might be markedly different from those of the present model. Clearly, this is worth further study.

Implications

Our multilocus model sheds light on at least two important theoretical questions. First, to what extent can coevolution contribute to the maintenance of genetic variation? Our model predicts that competition or mutualism always lead to very low levels of genetic variation. Victim-exploiter coevolution, in contrast, can lead to the maintenance of high levels of genetic variation in the victim but not in the exploiter. Two points are noteworthy. First, high genetic variation in the victim requires that ongoing coevolution is intense, in the sense that the victim is either trapped at an intermediate equilibrium (EQ_{DIS}) by frequency-dependent selection or is engaged in coevolutionary cycles (see Fig. 8). No or low variation is maintained if the victim is either strongly constrained by direct stabilizing selection (EQ_{STA}) or has permanently escaped to an extreme phenotype (EQ_{DIR}). Second, the maintenance of genetic variation due to antagonistic coevolution is strongly asymmetric, as it occurs only in the victim but not in the exploiter (Fig. 8). These results are in marked contrast to recent studies of host-parasite coevolution that have used the multilocus gene-for-gene (Sasaki 2000) or matching-allele (Sasaki et al. 2002) models and have shown that, in these models, coevolutionary cycles lead to the maintenance of multilocus polymorphisms in both species.

Second, what is the likelihood of observing coevolutionary cycles in victim-exploiter interactions? As mentioned above, this likelihood is generally high, at least in systems that meet our assumption of a bidirectional axis of vulnerability and that have the following properties: the traits under consideration are truly polygenic with small effects of individual loci, and neither species is strongly constrained by direct stabilizing selection or by the set of available phenotypes. Cycles are almost inevitable if the selection pressure is strong in the victim and not too weak in the exploiter.

The above predictions are empirically testable, at least in principle. Levels of genetic variation can be readily observed in single populations. In contrast, direct observation of coevolutionary cycles is likely to be difficult, because most time series data do not extend over a sufficiently long period. However, considerable progress can be made by comparing data from several geographic locations (Thompson 1994, 2005). If cycles in different populations are asynchronous, our model predicts a mosaic of populations showing local adaptation of the exploiter (i.e., matching phenotypes) or the victim (i.e., diverging phenotypes), respectively. Spatially variable outcomes of coevolution despite similar fitness functions may also reflect convergence to alternative stable states.

Furthermore, key parameters of the model can also principally be determined empirically. The strengths of stabilizing selection (σ_x, σ_y) and competition (γ_x, γ_y), can be estimated using standard approaches (Roughgarden 1972; Endler 1986; Kingsolver et al. 2001). If this is not possible, the composite parameters $2G_x(\gamma_x + \sigma_x)$ and $2G_y(\gamma_y + \sigma_y)$, which determine the potential evolutionary rates of the two species, can be estimated by observing the evolutionary response in one species when the other species is prevented from coevolving by some experimental manipulation, for example, similar to those used by Rice (1996) in his studies of antagonistic coevolution between sexes within the same species.

Finally, the number of loci influencing the trait of interest and their relative effects can be estimated using standard methods for the analysis of quantitative trait loci (Lynch and Walsh 1998). In addition, empirical patterns of coevolution might sometimes provide clues about the genetic basis of the evolving traits. For example, if the system is at an intermediate equilibrium with high genetic variance (EQ_{DIS}), it is more likely that the underlying genetics are simple (i.e., the number of loci is small).

Comparison of Modeling Approaches

Two previous models—the quantitative genetic model by Gavrilets (1997a) and the one-locus by Gavrilets and Hastings (1998)—yield results qualitatively similar to those of the multilocus model, but their quantitative predictions regarding dynamic details are different.

Under the assumptions of the quantitative genetic model (constant genetic variances and symmetric phenotypic distributions), the coevolutionary model reduces to a very simple system of linear differential equations (Gavrilets 1997a). The dynamic behavior of this kind of system is very limited. It supports neither multiple equilibria nor coevolutionary cycles with constant amplitude. Instead it can lead to the biologically unrealistic evolution of infinite trait values. Obviously, the quantitative genetic model cannot be used to study the evolution of genetic variances nor its interaction with the evolution of phenotypic means. In summary, while the quantitative genetic model can reproduce the basic qualitative results of the multilocus model, it fails to convey any of its dynamic details. Therefore, our results highlight some of the limitations of the constant-variance assumption in models of long-term phenotypic evolution (see Pigliucci and Schlichting 1997).

The one-locus model is closer to the multilocus model than is the quantitative genetic model (Fig. 1; Gavrilets and Hastings 1998). It prevents evolution of infinite phenotypes and it allows for changes in genetic variances. In consequence, it supports equilibria with both intermediate and extreme trait values and, for the victim-exploiter interaction, it is able to produce different types of coevolutionary cycles (stable limit cycles and heteroclinic cycles). The relative simplicity of the one-locus model allows for a more detailed mathematical analysis than that possible for the multilocus model. However, in the one-locus model, genetic variances cannot vary independently of the mean trait values, and therefore, they cannot be shaped by the type of selection (stabilizing versus disruptive). In consequence, there is no difference between intermediate equilibria with net stabilizing and disruptive selection in the victim (see Spichtig and Kawecki 2004). Equilibria with net disruptive selection are as likely as not to be stable, and stable limit cycles have the same likelihood as heteroclinic cycles.

The new results reported in this paper are mostly derived from two key properties of the multilocus model. First, the multilocus model supports a complex equilibrium structure, which in turn gives rise to a rich set of dynamic behaviors, including complex heteroclinic cycles and alternative stable states (note, however, that stable equilibria and heteroclinic cycles can also coexist in the one-locus model; see Gavrilets and Hastings 1998). Second, in the multilocus model, genetic variances can vary partly independently of the trait means (Spichtig and Kawecki 2004). Thus, the multilocus model can be used not only to investigate the role of coevolution in the maintenance of genetic variation, but also to make predictions about the likelihood and the nature of coevolutionary cycles. Indeed, while all three models agree on the conditions for cycles (high relative evolutionary rate of the victim), only the multilocus model is able to predict that this condition is actually likely to be fulfilled (because the victim tends to have a higher genetic variance), and that cycles tend to span a large proportion of the phenotypic range. In short, the interaction between the evolution of genetic variances and the coevolutionary dynamics of the mean trait values is a key results of this paper, and it can only be studied in a multilocus context.

In summary, our analysis has shown that genetic details are very important for the dynamics of coevolutionary interactions. We have focused on the number of loci and the distribution of locus effects. Furthermore, in Appendix 3 (available online) we show that the majority of our conclusions is independent of whether the interacting species are haploid or diploid. Future studies should investigate the effects of additional factors such as linkage, epistasis, or genetic drift and try to integrate multilocus models of coevolution with models of population dynamics.

Comparison to Nuismer et al. (2005)

In a recent paper, which appeared after the present study had been submitted, Nuismer et al. (2005) also analyzed the effects of explicit multilocus genetics on coevolutionary dynamics (using a haploid model with equal locus effects). Their model is complementary to ours, in that they do not include direct stabilizing selection but instead allow a significant role for mutation and analyze linkage disequilibrium (assuming weak selection). Their analytical treatment focuses on a polymorphic equilibrium at which, in both species, allele frequencies at all loci are maintained at one-half by mutation. As shown by Nuismer et al. (2005), this equilibrium exists if the phenotypic midranges of the two species are identical, and it can only be stable if mutation rates are high enough relative to the strength of per locus selection (otherwise, the polymorphism in the exploiter is destroyed by stabilizing selection, as described above). This assumption might hold true if the number of loci is very large and each locus has only a marginal effect on the phenotype. In contrast to Nuismer et al. (2005), we assume that mutation is weak relative to selection. As shown above, this approach allows us to perform a complete stability analysis of all possible equilibria and to include direct stabilizing selection. In particular, it allows us to analytically study the coevolutionary dynamics for parameter values where the fully polymorphic equilibrium analyzed by Nuismer et al. (2005) becomes unstable.

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