

GENETIC DIFFERENTIATION BY SEXUAL CONFLICT

Takehiko I. Hayashi,^{1,2} Michael Vose,^{3,4} and Sergey Gavrilets^{5,6}

¹Research Center for Chemical Risk Management, National Institute of Advanced Industrial Science and Technology, 16-1 Onogawa, Tsukuba, Ibaraki 305-8589, Japan

²E-mail: ti-hayashi@aist.go.jp

³Department of Computer Sciences, University of Tennessee, Knoxville, Tennessee 37996

⁴E-mail: vose@cs.utk.edu

⁵Department of Ecology and Evolutionary Biology, Department of Mathematics, University of Tennessee, Knoxville, Tennessee 37996

⁶E-mail: gavrila@tiem.utk.edu

Received June 6, 2006

Accepted October 31, 2006

Sexual conflict has been suggested as a general cause of genetic diversification in reproductive characters, and as a possible cause of speciation. We use individual-based simulations to study the dynamics of sexual conflict in an isolated diploid population with no spatial structure. To explore the effects of genetic details, we consider two different types of interlocus interaction between female and male traits, and three different types of intra-locus interaction. In the simulations, sexual conflict resulted in at least the following five regimes: (1) continuous coevolutionary chase, (2) evolution toward an equilibrium, (3) cyclic coevolution, (4) extensive genetic differentiation in female traits/genes only, and (5) extensive genetic differentiation in both male and female traits/genes. Genetic differentiation was hardly observed when the traits involved in reproduction were determined additively and interacted in a trait-by-trait way. When the traits interacted in a component-by-component way, genetic differentiation was frequently observed under relatively broad conditions. The likelihood of genetic differentiation largely depended on the number of loci and the type of within-locus dominance. With multiple loci per trait, genetic differentiation was often observed but sympatric speciation was typically hindered by recombination. Sympatric speciation was possible but only under restrictive conditions. Our simulations also highlight the importance of stochastic effects in the dynamics of sexual conflict.

KEY WORDS: Diploid, genetic divergence, mathematical models, mating rate, multilocus, polymorphism, sympatric speciation.

Sexual conflict—a conflict between the evolutionary interests of individuals of the two sexes (Parker 1979, 2006)—occurs when the interests of the sexes differ with regard to certain characteristics of reproduction. For example, male fitness typically increases with mating rate, but a too high mating rate is often deleterious for females (e.g., Arnqvist and Nilsson 2000; Arnqvist and Rowe 2005; Maklakov et al. 2005). The difference in interests can cause an evolutionary arms race between the sexes in which females evolve resistance to increased mating rate whereas males evolve an ability to overcome female resistance (Holland and Rice 1998;

Arnqvist and Rowe 2005). Sexual conflict has been suggested as a general cause of continuous and rapid coevolution in traits controlling reproductive characters (reviewed in Rice and Holland 1997; Holland and Rice 1998; Rice 1998; Chapman et al. 2003; Pizzari and Snook 2003; Arnqvist and Rowe 2005).

Many empirical studies have supported the idea that sexual conflict can cause antagonistic coevolution between sexes. Arnqvist and Rowe (2002) provide strong empirical evidence for a coevolutionary arms race between male and female armaments (clasping and anti-clasping morphologies) in water striders.

Experiments with *Drosophila* (Rice 1996; Holland and Rice 1999; Pitnick et al. 2001a,b) and the dung fly *Sepsis cynipsea* (Martin and Hosken 2003) directly show that sexual conflict causes rapid coevolution in reproductive traits. Molecular studies also demonstrate rapid evolution in many genes that mediate sexual reproduction, such as those involved in gamete recognition (reviewed in Swanson and Vacquier 2002). At least some of these examples may be explained by selection for avoidance of polyspermy (e.g., Howard et al. 1998), which is an important example of conflict over mating rate.

Theoretical studies support the idea that sexual conflict can cause rapid antagonistic coevolution (reviewed in Gavrillets and Hayashi 2005). Models of conflict over mating rate have demonstrated a variety of coevolutionary dynamics including evolutionary chase (Gavrillets 2000; Gavrillets et al. 2001; Gavrillets and Waxman 2002; Gavrillets and Hayashi 2006), cyclic coevolution (Gavrillets et al. 2001; Haygood 2004; Rowe et al. 2005), stable equilibrium (Gavrillets et al. 2001; Rowe et al. 2005), and random genetic drift along a line of equilibria (Gavrillets 2000). Moreover, models suggest that sexual conflict can cause genetic differentiation in reproductive traits (Frank 2000; Gavrillets and Waxman 2002) and eventual sympatric speciation (Gavrillets and Waxman 2002). These earlier models have a number of important limitations, however. First, models adopting a quantitative genetics approach (e.g., Gavrillets 2000; Gavrillets et al. 2001; Rowe et al. 2003, 2005) treat underlying genetics in an implicit, approximate way, by assuming constancy of genetic variances and weak selection. To assume constant genetic variance prevents examination of genetic differentiation, a possible primary consequence of sexual conflict. Those models that consider explicit genetics allow for only a single locus per sex (e.g., Gavrillets and Waxman 2002; Haygood 2004), yet the number of loci involved in reproductive interaction can be large (Ritchie and Phillips 1998) and the number of loci can strongly affect the likelihood of sympatric speciation (Arnégard and Kondrashov 2004; Gavrillets 2004). Gavrillets and Waxman (2002) analyze only the haploid case, but dominance in diploid models can be an important factor determining coevolutionary dynamics (Haygood 2004).

Here, we use individual-based simulations to study diploid, multilocus, and multiallele models of sexual conflict over mating rate. We consider two different types of interlocus interaction between female and male traits, and three different types of intralocus interaction. The primary goal of this paper is to study the effects of these genetic details on the evolutionary dynamics of sexual conflict.

Model and Methods

We assume reproductive interactions are mediated by two independent sex-limited traits: a female (or egg) trait x and a male (or

sperm) trait y (see Table A1 for a glossary of notation). Each trait is controlled by $\mathcal{L}/2$ unlinked diploid loci, each with 32 alleles, labeled by integers from 0 to 31 (the numeric value is the allelic effect). Mutation patterns are specified by the stepwise mutation model (e.g., Nei et al. 1983), so that allele i can only mutate to alleles $i - 1$ or $i + 1$, each with probability $\mu/2$. Alleles 0 and 31 can mutate only to alleles 1 and 30, respectively, with probability $\mu/2$.

The probability that a female with trait x will mate upon encountering a male with trait y is

$$\psi(d) = \exp(-\alpha d^2), \quad (1)$$

where d is the genetic or phenotypic distance between the female and male (defined below), and where the parameter α controls the range of compatibility between x and y . The function $\psi(d)$ is analogous to the so-called preference function common in models of sexual selection (e.g., Lande 1981; Gavrillets 2004). A female's mating rate is the proportion P of males she mated with (out of the number of males she encountered). Female fitness should increase with P at small values of the mating rate (with more acceptable/compatible males comes an increased probability of successful fertilization), whereas high values of P should depress female fitness because of various costs of mating (reviewed in Arnqvist and Rowe 2005). Following previous work (Gavrillets 2000; Gavrillets et al. 2001; Gavrillets and Waxman 2002), female fertility is defined by a function maximized at P_{opt} ($0 < P_{\text{opt}} < 1$): the overall number W_f of offspring produced by a female having mating rate P is

$$W_f = B_{\text{max}} \exp[-s_c(P - P_{\text{opt}})^2], \quad (2)$$

where B_{max} is the maximum possible number of offspring, and s_c is the strength of selection (by sexual conflict) in females.

GENETIC AND PHENOTYPIC DISTANCES BETWEEN FEMALE AND MALE TRAITS

We use two different models to calculate the genetic and phenotypic distance d between reproductive traits.

Genetic distance models

In general, compatibility between the sexes (e.g., genetic, behavioral, etc.) is expected to depend on the degree of matching between a number of specific components. In the genetic distance models, distance is given by the sum $d = \sum_{i=1}^{\mathcal{L}/2} d_i$ of locus-by-locus differences between female and male loci (d_i is the genetic distance between the i th female locus and the corresponding i th male locus). In the calculation of d_i , we assumed three different types of dominance. In the additive case, alleles contribute additively at each locus, so that $d_i = |(f_i + f'_i) - [m_i + m'_i]|/2$, where f_i and f'_i [m_i and m'_i] are the allelic values of maternal and

paternal alleles at the i th female [male] locus. In the directional dominance case, alleles with larger effect are always dominant over alleles with smaller effect, $d_i = | \max(f_i, f'_i) - \max(m_i, m'_i) |$. In the codominance case, d_i is the average of the distances over four pairs of allele combinations, $d_i = (|f_i - m_i| + |f_i - m'_i| + |f'_i - m_i| + |f'_i - m'_i|)/4$.

Phenotypic distance model

In the phenotypic distance model, values of traits are given by sums $x = \sum_{i=1}^{\mathcal{L}/2} (f_i + f'_i)$ and $y = \sum_{i=1}^{\mathcal{L}/2} (m_i + m'_i)$ of the allelic effects of the corresponding alleles. Phenotypic distance d between female and male traits is $d = |x - y|$ (Gavrilets 2000; Gavrilets et al. 2001). Note that in two-locus models ($\mathcal{L} = 2$), the additive genetic distance model is qualitatively equivalent to the phenotypic distance model.

DIRECT NATURAL SELECTION ON THE TRAITS

Direct natural selection on female and male traits is incorporated as follows. In the phenotypic distance model, female viability is given by $W_{\text{nat}} = \exp(-s_x(x - x_{\text{opt}})^2)$, where x is the phenotypic value of the female trait, s_x is the strength of natural selection, and x_{opt} is the optimum trait value for x with respect to natural selection. In the genetic distance model, stabilizing selection acts on allelic effects at each individual locus. Female viability is given by $W_{\text{nat}} = \exp(-s_x d_{\text{nat}}^2)$, where $d_{\text{nat}} = \sum_{i=1}^{\mathcal{L}/2} |(f_i + f'_i)/2 - x_{\text{opt}}|$ in the additive case, $d_{\text{nat}} = \sum_{i=1}^{\mathcal{L}/2} |\max(f_i, f'_i) - x_{\text{opt}}|$ in the directional dominance case, and $d_{\text{nat}} = \sum_{i=1}^{\mathcal{L}/2} (|f_i - x_{\text{opt}}| + |f'_i - x_{\text{opt}}|)/2$ in the codominance case. Male viability is defined similarly.

NUMERICAL PROCEDURE

We performed individual-based simulations based on constant size populations containing N individuals. Generations are distinct and nonoverlapping, produced according to the following procedure. Each female encounters n uniformly sampled males. At each encounter, the mating probability is $\Psi(d) = \exp(-\alpha d^2)$. The overall number of offspring produced by a female is $B_{\text{max}} \exp[-s_c(P - P_{\text{opt}})^2]$, except if she mates with no male ($P = 0$), in which case she has no offspring. For each offspring, a male is randomly chosen as the father from those males who mated with the mother. Offspring are produced with account of mutation and recombination (all loci are unlinked), and offspring sex is assigned randomly. Viability of offspring is taken into account by discarding each offspring with probability $1 - W_{\text{nat}}$. After producing all offspring (if any) from all females by the procedure above, N offspring are sampled to form the next generation.

Simulations started with a monomorphic population where all individuals have allele 16 at all loci. We ran each simulation for 25,000 generations, and performed 20 simulations for each

parameter set. The following parameter values were used: mutation rate per locus $\mu = 5.0 \times 10^{-5}$ or 10^{-5} ; strength of female preference $\alpha = 0.01$ or 0.05 ; optimum female mating rate $P_{\text{opt}} = 0.2, 0.4, 0.6,$ or 0.8 ; strength of selection (by sexual conflict) in females $s_c = 1.02$ or 4×1.02 ; number of loci $\mathcal{L} = 2, 8,$ or 32 ; population size $N = 1000$ or $10,000$. For the simulations discussed in this article, the number of males each female encounters is $n = 20$, and the maximum possible number of offspring is $B_{\text{max}} = 5$. Unless specified otherwise, there is no natural selection on the traits (i.e., $s_x = s_y = 0$).

STATISTICS AND DATA ANALYSIS

We used the following characteristics and methods to describe the evolutionary dynamics and genetic structure of each population.

Locus effect

In the phenotypic distance model, the locus effect x_i of the i th female locus is the sum $x_i = f_i + f'_i$ of allelic effects at the locus. For the three genetic distance models the locus effect is $x_i = (f_i + f'_i)/2$ in the additive case, $x_i = \max(f_i, f'_i)$ in the directional dominance case, and x_i is an allele randomly chosen from either f_i or f'_i in the codominance case. The locus effect y_i of the i th male locus is defined similarly.

Frequency of genetic differentiation

To measure the likelihood of genetic differentiation, we used the number of simulations (out of 20 runs) in which the population exhibits a distinct bimodal or multimodal distribution of allelic effects in any locus at generation 25,000. To measure the likelihood of sympatric speciation, we used the number of simulations in which the population exhibits sympatric speciation at generation 25,000. Sympatric speciation is considered to occur when a population meets all of the following conditions: (1) there are distinct genetic clusters, (2) the average probability of mating between individuals from different clusters is less than 0.001, (3) the cumulative frequency of recombinants between different clusters is less than 5%.

Haplotype network

We used TCS (Clement et al. 2000) to create a haplotype network of each population. Our goal was not to estimate the “phylogenetic relationship” among individuals, but to graphically illustrate genetic structure of multilocus populations. Given that purpose, we ignored the historical process underlying differences among haplotypes and considered all differences as parsimonious. In this case, the estimation algorithm of TCS based on statistical parsimony (Templeton et al. 1992) links up nearest haplotypes step by step until all haplotypes are connected in a single network (Templeton et al. 1992; Clement et al. 2000). TCS also identifies potential products of recombination, inferred by loops in a haplotype network (Templeton et al. 1992). To analyze with TCS,

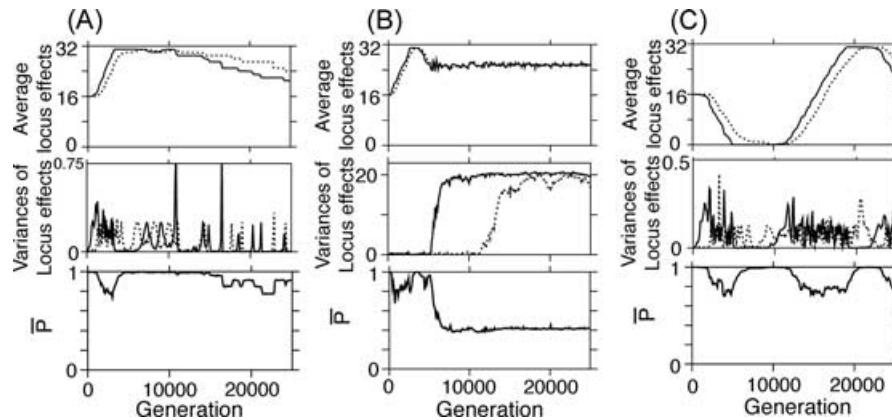


Figure 1. Examples of coevolutionary dynamics in the genetic distance model with two loci. Average locus effects (upper row) and variances of locus effects (middle row) of the female (solid lines) and male (dashed lines) loci, and average mating rate \bar{P} (lower row). Within-locus interaction is (A) directional dominance, (B) codominance, or (C) additive. Parameters: $P_{\text{opt}} = 0.4$, $\alpha = 0.01$ (A, C) or 0.05 (B), $s = 1.02$, $\mu = 0.5 \times 10^{-5}$, $N = 10,000$.

we converted haplotypes into a compatible format (PHYLIP sequence file format) keeping character distances among haplotypes unchanged.

Results

GENETIC DISTANCE MODELS

Two-locus model

In the genetic distance models with two loci ($\mathcal{L} = 2$), the dynamics depend largely on the type of dominance.

The directional dominance case.—Evolutionary chase (i.e., continuous change in the average allelic effects of female and male traits) without within-population genetic differentiation was the most common outcome (Fig. 1A and Table 1a). The direction of chase changes to the opposite once a boundary of possible trait values is reached. During the chase, evolution toward larger allelic values was much faster than in the opposite direction (Fig. 1A). This reflects the fact that dominant alleles are easier to fix than recessive ones (e.g., Crow and Kimura 1970). When sexual conflict was weak (i.e., $P_{\text{opt}} = 0.8$), genetic differentiation only in the female locus was also frequently observed (Table 1a).

The codominance case.—Genetic differentiation in the female locus was frequently observed (Table 1b). The frequency of differentiation increased as the strength of sexual conflict decreased (i.e., P_{opt} increased) (Table 1b). However, given that some differentiation occurred, the degree of differentiation (variance in locus effect) increased as P_{opt} decreased. In the simulations described in Table 1b, the average variance of locus effect in the female locus (at generation 25,000) when differentiation in the female locus occurred was 152.26 for $P_{\text{opt}} = 0.2$ and 17.48 for $P_{\text{opt}} = 0.8$. When differentiation occurred only in the female locus, males got trapped between the two diverged female allele

clusters (Fig. 2A), and the average mating rate decreased to values relatively close to P_{opt} (Fig. 1B). We call this the “Buridan’s Ass regime,” which is characterized by extensive differentiation in females with no corresponding differentiation in males (Gavrilets and Waxman 2002). Once this regime begins, the speed of coevolutionary chase is significantly reduced (see Fig. 1B around generation 7500 when alleles in the female locus diverge). When P_{opt} was small enough (i.e., sexual conflict was strong), subsequent genetic differentiation in the male locus often followed the differentiation in the female locus (Figs. 1B, 2B–C, and Table 1b). When both female preference and sexual conflict were strong ($\alpha = 0.05$ and $P_{\text{opt}} = 0.2$), differentiation in the loci of both sexual traits often increased to a level that provided strong reproductive isolation between the differentiating genetic clusters, that is, sympatric speciation (Table 1c, Figs. 3A and 2C). In some simulation runs, the distribution of locus effect in the female loci exhibited more than two peaks (Fig. 2C).

The additive case.—Continuous evolutionary chases without genetic differentiation were always observed (Fig. 1C and Table 1f). With the same parameter configuration, the likelihood of genetic differentiation was smaller in the additive case than in the codominance or direct dominance cases (compare Table 1f, a, and b). This observation can be explained by how the type of dominance affects the “incentive” for females to differentiate. In the Buridan’s Ass regime, which is characterized by the extensive differentiation in females without corresponding differentiation in males, females “try” to reduce their mating rate by increasing their genetic variance. In this regime, allelic effects of a male locus in the genetic distance model is typically intermediate between those of diverged female alleles at the corresponding female locus. Linkage disequilibrium is nearly absent (because mating is effectively random) and heterozygous female loci are always common. In this situation, the additive allelic interaction provides smaller incentive

Table 1. The effect of the optimal mating rate for females (P_{opt}) and the type of dominance on the average mating rate (\bar{P}) and the likelihood of genetic differentiation in the genetic distance model with two loci. Shown is the median of \bar{P} , with the 10th and 90th percentiles in parentheses. \bar{P} is recorded at generation 25,000. Within-locus interaction is (a) directional dominance, (b–e) codominance, or (f) additive. Parameters are $s_c = 1.02$, $\mu = 5 \times 10^{-5}$, $N = 10,000$, and $\alpha = 0.01$ except (c): $\alpha = 0.05$, (d): $\mu = 10^{-5}$, and (e): $s_c = 4 \times 1.02$.

	P_{opt}	\bar{P}	Differentiation ¹		
			F	M	SS
(a)	.2	.91 (.84, .97)	0	0	0
	.4	.96 (.83, .99)	0	0	0
	.6	.96 (.91, .99)	1	0	0
	.8	.85 (.78, .99)	12	0	0
(b)	.2	.85 (.34, .98)	5	3	0
	.4	.91 (.40, 1.00)	7	3	0
	.6	.67 (.59, .98)	14	0	0
	.8	.81 (.80, .91)	19	0	0
(c)	.2	.68 (.32, .93)	9	9	8
	.4	.42 (.41, .47)	18	18	0
	.6	.64 (.63, .66)	20	0	0
	.8	.85 (.82, .89)	20	0	0
(d)	.2	.98 (.76, 1.00)	1	1	0
	.4	.99 (.94, 1.00)	0	0	0
	.6	.97 (.84, 1.00)	3	0	0
	.8	.98 (.85, 1.00)	5	0	0
(e)	.2	.95 (.52, 1.00)	0	0	0
	.4	.99 (.41, 1.00)	2	2	0
	.6	.78 (.70, .99)	1	0	0
	.8	.95 (.82, .99)	4	0	0
(f)	.2	.95 (.81, 1.00)	0	0	0
	.4	.99 (.90, 1.00)	0	0	0
	.6	.97 (.95, .99)	0	0	0
	.8	.99 (.98, 1.00)	0	0	0

¹The number of simulation runs (out of 20 runs) in which sympatric speciation (SS) or differentiation in any female locus (F) or any male locus (M) was observed at generation 25,000.

for females to differentiate than the codominance or directional dominance allelic interaction because the heterozygous female loci with diverged alleles still suffer high compatibility with the average male trait. To illustrate this, let us consider a population in the Buridan’s Ass regime under the genetic distance model in which a female locus has alleles 12 and 20 and the corresponding male locus has allele 16. For a homozygous female locus, the genetic distance between female and male loci is 4 (i.e., $|16 - 20|$ or $|16 - 12|$) irrespective of the type of dominance. For a heterozygous female locus, the distance remains 4 in the directional dominance case (i.e., $|16 - 20|$) and the codominance case (i.e., $(|16 - 20| + |16 - 12|)/2$). However, the genetic distance is zero

in the additive case (i.e., $|16 - (20 + 12)/2|$), which suggests that differentiation (into the Buridan’s Ass regime) is not an effective way to reduce mating rate in the additive case if heterozygous loci with diverged alleles are common (as observed under the Buridan’s Ass regime). This effect of heterozygous loci in the additive case predisposes females to evolve away from males (as in the continuous coevolutionary chase regime) rather than differentiate (as in the Buridan’s Ass regime) as a strategy for reducing their mating rate.

As for the effects of parameter values, strong female preference ($\alpha = 0.05$) increased the likelihood of genetic differentiation in both sexes in the codominance case (Table 1c), but only in the female locus in the directional dominance case (data not shown). In the additive case, strong female preference often caused small differentiation in females when sexual conflict was weak ($P_{opt} = 0.8$; data not shown). Decreased mutation rate (Table 1d) and stronger selection (by sexual conflict) in females (Table 1e) suppressed the likelihood of genetic differentiation. No genetic differentiation was observed when the population size was small ($N = 1000$; data not shown).

Eight-locus model

In the genetic distance model with eight loci ($\mathcal{L} = 8$), the dynamics depend on the type of dominance.

The directional dominance case.—Differentiation in both sexes was commonly observed when sexual conflict was sufficiently strong ($P_{opt} \leq 0.4$; Table 2a and Fig. 4A). The dynamics of coevolutionary chase and diversification was typically complex. First, each male–female pair of corresponding loci evolved largely independently (Fig. 4A). Different regimes were often observed at different loci at the same time, and more than one locus often exhibited differentiation at the same time (Figs. 4A and 5). In addition, rapid stochastic transitions between different regimes in a single locus (i.e., creation and breakdown of genetic diversification) were also frequently observed. For example, Figure 4A shows loss of genetic variation in the third and fourth pair of loci (near generations 17,000 and 12,000, respectively). This sudden loss of genetic variation was caused by the extinction of one cluster of diverging alleles. When sexual conflict was relatively weak ($P_{opt} \geq 0.6$), differentiation was observed only in the female trait (Table 2a). Decreased mutation rate ($\mu = 10^{-5}$) suppressed the likelihood of genetic diversification (data not shown). Stronger selection in females ($s_c = 4 \times 1.02$) did not significantly change the qualitative results (data not shown).

Even when genetic differentiation occurred in both sexes, the population did not form distinct reproductively isolated clusters; no sympatric speciation was observed. Genetic differentiation did occur in multiple loci, but formation of large and distinct genetic clusters was suppressed by recombination among diverged loci. Figure 3B is a typical example (for the directional dominance

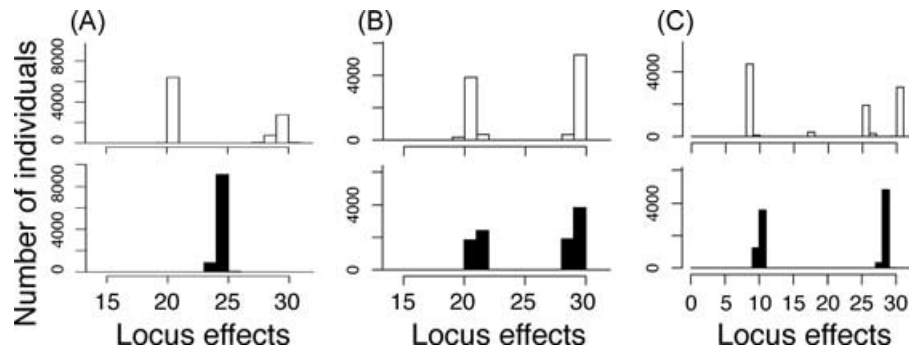


Figure 2. The distribution of locus effects in the genetic distance model with two loci. (A) Differentiation in the female locus only (the “Buridan’s Ass” regime). The population is from the simulation shown in Figure 1B at generation 7500. (B) Differentiation in both female and male loci. The population is from the simulation shown in Figure 1B at generation 25,000. (C) Sympatric speciation with strong sexual conflict ($P_{\text{opt}} = 0.2$). Upper white bars: female locus. Lower black bars: male locus. Parameters are the same as in Figure 1B unless specified.

case) of a haploid network when genetic diversification in both sexes occurred. It shows a population consisting of small, loosely connected clusters, rather than large and distinct clusters. The web structure among clusters shows that recombinant genotypes were common in the population.

With the same parameter configuration, genetic differentiation is more likely in the eight-locus model (Table 2a) than in the two-locus model (Table 1a). This partly reflects the difference in the strength of selection pressure on each locus. When the number of loci is small, the selection pressure on each locus is strong and tends to suppress the genetic variance required to initiate genetic differentiation. Moreover, differentiation rarely occurs once coevolutionary chase begins. In contrast, genetic differentiation is still possible even when coevolutionary chase occurs in one of the

pairs of loci, provided that the number of loci is moderate (e.g., $\mathcal{L} = 8$).

The codominance case.—Genetic differentiation in the loci of both sexes was often observed (Fig. 4B and Table 2b), although the frequency of differentiation in male loci was smaller than in the directional dominance case. When selection in females was not sufficiently strong (i.e., $s_c = 1.02$), no sympatric speciation was observed (because of recombination among diverging loci). Strong female preference ($\alpha = 0.05$) enhanced genetic differentiations in the loci of both sexes but did not cause sympatric speciation (Table 2c). Decreased mutation rate ($\mu = 10^{-5}$) suppressed genetic differentiation, especially in male loci (Table 2d). No diversification was observed when population size was small ($N = 1000$).

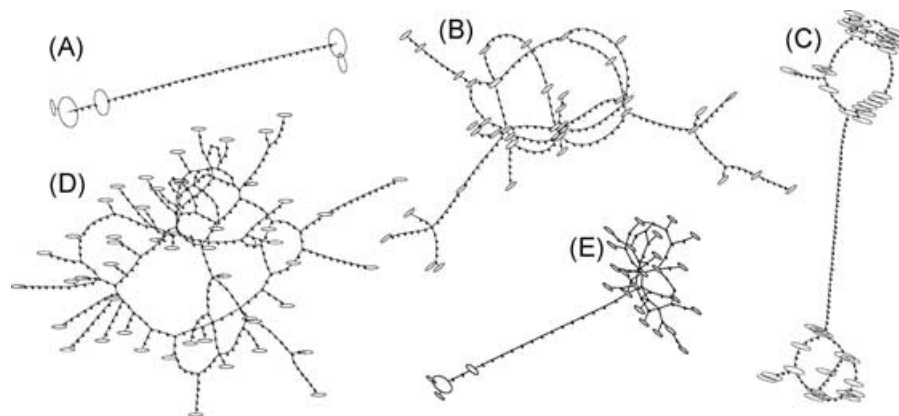


Figure 3. Examples of haplotype networks. The ovals represent groups of individuals with the same haplotype. The length of a branch (more precisely, the number of nodes between clusters) represents the distance between haplotypes. These haplotype networks were computed based on 50 individuals randomly sampled at generation 25,000. Parameters: $P_{\text{opt}} = 0.2$, $\alpha = 0.01$, $s_c = 1.02$, $\mu = 0.5 \times 10^{-5}$, $N = 10,000$, unless specified. (A) Sympatric speciation in the two-locus genetic distance model with codominance. The data are from the simulation run shown in Figure 2C, $\alpha = 0.05$. (B) Genetic diversification without speciation in the eight-locus genetic distance model with directional dominance. The data are from the simulation run shown in Figure 4A. (C) Sympatric speciation in the eight-locus genetic distance model with codominance, $s_c = 4 \times 1.02$. (D) Genetic diversification without speciation in the 32-locus genetic distance model with codominance. (E) Sympatric speciation in the phenotypic distance model with 32 loci, $\alpha = 0.05$.

Table 2. The effect of the optimal mating rate for females (P_{opt}) and the type of dominance on the average mating rate (\bar{P}) and the likelihood of genetic differentiation in the genetic distance model with eight loci. Shown is the median of \bar{P} , with the 10th and 90th percentiles in parentheses. \bar{P} is recorded at generation 25,000. Within-locus interaction is (a) directional dominance, (b–e) codominance, or (f) additive. Parameters are $s_c = 1.02$, $\mu = 5 \times 10^{-5}$, $N = 10,000$, and $\alpha = 0.01$ except (c): $\alpha = 0.05$, (d): $\mu = 10^{-5}$, and (e): $s_c = 4 \times 1.02$.

	P_{opt}	\bar{P}	Differentiation ¹		
			F	M	SS
(a)	.2	.30 (.28, .36)	20	20	0
	.4	.43 (.42, .49)	20	17	0
	.6	.66 (.60, .70)	20	0	0
	.8	.84 (.81, .87)	20	0	0
(b)	.2	.33 (.28, .41)	20	8	0
	.4	.42 (.40, .49)	20	1	0
	.6	.63 (.59, .66)	20	0	0
	.8	.82 (.79, .86)	20	0	0
(c)	.2	.29 (.27, .30)	20	20	0
	.4	.42 (.39, .45)	20	10	0
	.6	.64 (.62, .67)	20	0	0
	.8	.84 (.83, .86)	7	0	0
(d)	.2	.38 (.28, .66)	18	0	0
	.4	.44 (.42, .65)	19	0	0
	.6	.63 (.61, .69)	19	0	0
	.8	.85 (.83, .89)	19	0	0
(e)	.2	.27 (.24, .34)	20	16	7
	.4	.41 (.40, .48)	19	8	0
	.6	.64 (.62, .69)	20	0	0
	.8	.83 (.83, .85)	20	0	0
(f)	.2	.75 (.70, .82)	0	0	0
	.4	.83 (.79, .91)	0	0	0
	.6	.90 (.82, .95)	0	0	0
	.8	.88 (.87, .90)	3	0	0

¹The number of simulation runs (out of 20 runs) in which sympatric speciation (SS) or differentiation in any female locus (F) or any male locus (M) was observed at generation 25,000.

When both selection in females and sexual conflict were strong ($s_c = 4 \times 1.02$ and $P_{opt} = 0.2$), sympatric speciation was observed in several simulation runs (Table 2e). Genetic differentiation occurred in multiple loci for these cases, but no recombinant among diverging clusters was present (Fig. 3C). Large distinct clusters form as follows. Extensive differentiation in allelic effects within a single pair of female and male loci initially creates two distinct clusters having some degree of reproductive isolation. Subsequent differentiation in other loci occurs within clusters, further increasing their distance to a degree that reproductively isolates them.

The additive case.—Continuous coevolutionary chase without genetic differentiation was the most common outcome (Fig. 4C and Table 2f). The difficulty of genetic differentiation is explained by the same mechanism discussed above for the two-locus model. When sexual conflict is weak ($P_{opt} = 0.8$), small genetic differentiation was observed in a few simulation runs (Table 2f), but only in female loci.

32-locus model

In the genetic distance model with 32 loci ($\mathcal{L} = 32$), the dynamics were weakly affected by dominance type. Genetic differentiation was frequently observed only in the directional dominance and codominance cases (Table 3). However, the degree of differentiation was so small as to not significantly affect the overall phenotypic variances or resulting evolutionary dynamics. (In Table 3 with $P_{opt} = 0.2$, e.g., the median genic variance in the female trait is 12.31 for the directional dominance case [Table 3a] and 11.18 for the additive case [Table 3c], where genic variance is the sum over loci of variances of locus effects.) Irrespective of the type of dominance, average mating rates (\bar{P}) were always close to the females' optimum (P_{opt}) (Table 3). Typically, many female loci exhibited unimodal phenotypic distributions with moderate variance. The mating rate was reduced by phenotypic variation created by many loci each having small effect. Average locus effects of both sexes were almost equal, and the changes in the average allelic effects were typically slow; no apparent coevolutionary chase was observed (Fig. 6). No sympatric speciation was observed in any run of the 32-locus model. Most populations showed extensive genetic differentiation without forming any distinct subclusters (Fig. 3D). Decreased mutation rate ($\mu = 10^{-5}$), strong female preference ($\alpha = 0.05$), or strong selection in females ($s_c = 4 \times 1.02$) did not significantly affect the qualitative results (data not shown).

The effects of a different scaling of allelic effects.—In our model, increasing the number of loci increases the maximum possible distance between female and male traits. However, the same distance between traits results in the same mating probability irrespective of the number of loci; the (maximum) effect of a single mutation on mating probability is independent of the number of loci. Alternative models are possible. In particular, the maximum possible distance between traits could be kept the same irrespective of the number of loci, and the (maximum) effect of a single mutation on mating probability could be scaled with the number of loci. Our model can effectively simulate this scenario by choosing α so as to keep the maximum value of αd^2 constant as the number of loci changes.

Using a 32-locus model with $\alpha = 0.05 \times 16^{-2}$ provides the same maximum value for αd^2 as do two-locus models with $\alpha = 0.05$ (Table 1c). We have examined the effect of this alternative scaling on evolutionary dynamics (Table 3d–e). The simulations revealed

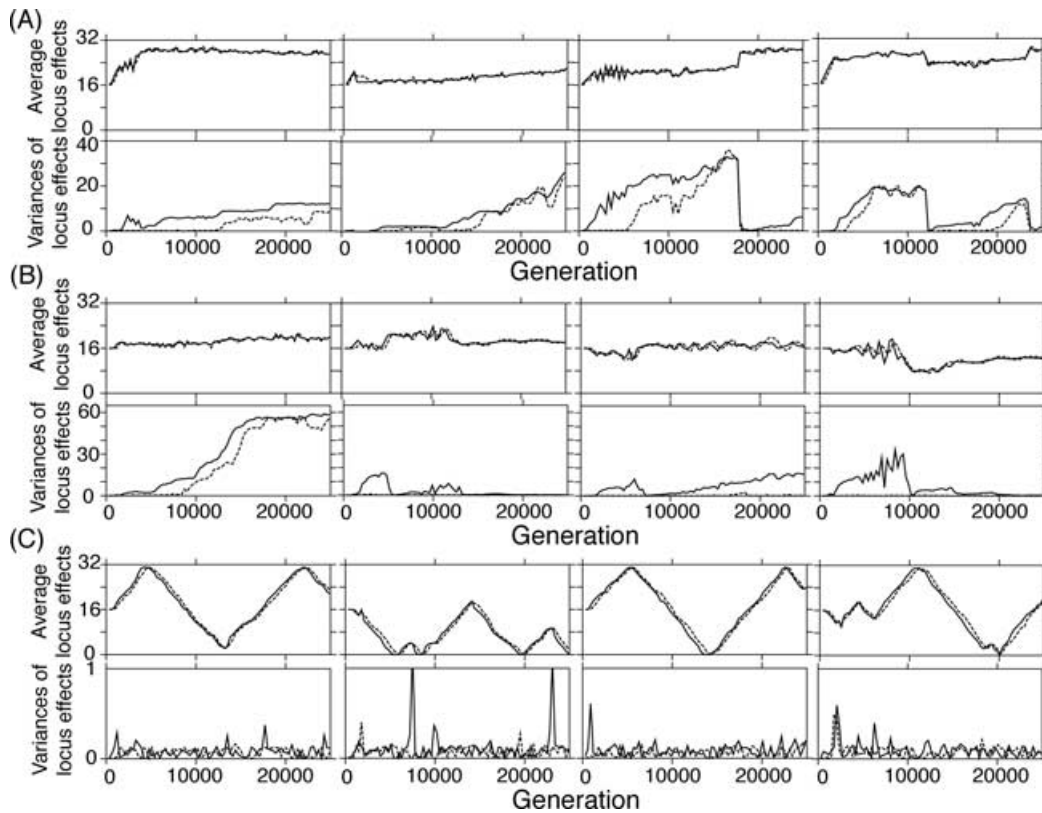


Figure 4. Examples of coevolutionary dynamics in the genetic distance model with eight loci. Average locus effects (upper row) and variances of locus effects (lower row). Each column represents the first to fourth (left to right) female (solid lines) and male (dashed lines) loci. The type of dominance is (A) directional dominance, (B) codominance, or (C) additive. Parameters: $P_{opt} = 0.2$, $\alpha = 0.01$, $s_c = 1.02$, $\mu = 0.5 \times 10^{-5}$, $N = 10,000$.

two qualitative differences. In contrast to the result shown in Figure 6, the alternative scaling often caused rapid coevolutionary chase in the loci that did not previously exhibit genetic differentiation. Also, a significant decrease in the average mating rate (\bar{P}) in the additive case (Table 3c) was not observed when alternative scaling was used (Table 3e).

PHENOTYPIC DISTANCE MODEL

In the phenotypic distance model, continuous coevolutionary chase without genetic differentiation was the most common out-

come when the number of loci was not too large ($\mathcal{L} \leq 8$; Table 4a). The phenotypic variances were small during the chase. For example, the data of Table 4a for $\mathcal{L} = 2$ and $P_{opt} = 0.2$ show that the average phenotypic variances in the female and male traits are 1.36 and 1.00, respectively. During the chase, allelic effects evolved in the same direction in all loci, in contrast to the genetic distance model. The small likelihood of genetic differentiation in the phenotypic distance model can partly be explained by the additive allelic interaction of loci discussed above for genetic distance models. The coincidence in the direction of change of allelic

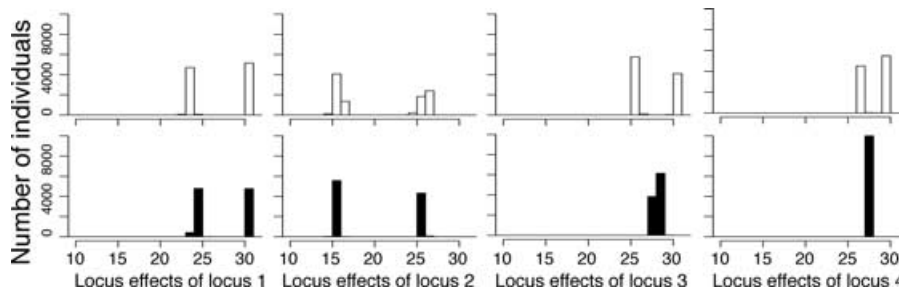


Figure 5. Differentiation in the locus effects in both sexes in the genetic distance model with eight loci. The directional dominance case. Each column represents first to fourth (left to right) female (upper white bars) and male (lower black bars) loci. The data are from the simulation run shown in Figure 4A.

Table 3. The effect of the optimal mating rate (P_{opt}) and the type of dominance on average mating rate (\bar{P}) and the likelihood of genetic differentiation in the genetic distance model with 32 loci. Shown is the median of \bar{P} , with the 10th and 90th percentiles in parentheses. \bar{P} is recorded at generation 25,000. Within-locus interaction is (a) directional dominance, (b, d) codominance, or (c, e) additive. Parameters are $s_c = 1.02$, $\mu = 5 \times 10^{-5}$, $N = 10,000$, and $\alpha = 0.01$ except (d–e): $\alpha = 0.05 \times 16^{-2}$. No sympatric speciation was observed.

	P_{opt}	\bar{P}	Differentiation ¹	
			F	M
(a)	.2	.30 (.29, .32)	20	18
	.4	.43 (.40, .44)	20	8
	.6	.63 (.61, .66)	12	0
	.8	.84 (.83, .85)	8	0
(b)	.2	.29 (.28, .29)	20	0
	.4	.41 (.40, .42)	16	0
	.6	.61 (.61, .62)	12	0
	.8	.83 (.82, .84)	0	0
(c)	.2	.32 (.31, .32)	2	0
	.4	.43 (.42, .44)	0	0
	.6	.62 (.62, .63)	0	0
	.8	.83 (.82, .84)	0	0
(d)	.2	.42 (.33, .47)	20	0
	.4	.51 (.47, .55)	20	0
	.6	.70 (.69, .73)	20	0
	.8	.86 (.85, .88)	20	0
(e)	.2	.89 (.84, .91)	0	0
	.4	.86 (.84, .89)	0	0
	.6	.92 (.90, .94)	0	0
	.8	.95 (.94, .97)	0	0

¹The number of simulation runs (out of 20 runs) in which differentiation in any female locus (F) or any male locus (M) was observed at generation 25,000.

effects also reduces the potential for differentiation at any of these loci. When sexual conflict is weak to moderate ($0.6 \leq P_{opt} \leq 0.8$), differentiation only in the female trait was observed in several simulation runs (Table 4a). The degree of differentiation was typically small. For example, the data of Table 4a for

$\mathcal{L} = 2$ and $P_{opt} = 0.8$ show that when diversification occurred, the average phenotypic variance in the female trait was 5.18. The mating rate in the populations was not greatly affected by differentiation.

When the number of loci was large ($\mathcal{L} = 32$), phenotypic differentiation only in the female trait (i.e., the Buridan’s Ass regime) was observed frequently when sexual conflict was moderate ($0.4 \leq P_{opt} \leq 0.6$; Table 4a). The degree of differentiation was relatively large. For example, the data of Table 4a for $\mathcal{L} = 32$ and $P_{opt} = 0.6$ show that when diversification occurred, the average phenotypic variance in the female trait was 21.38. The differentiation in the phenotypic distribution typically resulted in three peaks (corresponding to two homozygotes and their heterozygote), and males were typically trapped between the (largest and second largest) female subclusters. Such differentiation typically occurred only in a single female locus, and all other loci had very low genetic variation. One may interpret this outcome as the evolution of an allele of major genetic effect (because nearly all genetic variation in the female trait is attributed to a single locus). The observed increased likelihood of differentiation with increasing number of loci is reasonable; increasing the number of loci increases the overall mutation rate per trait and decreases selection pressure per locus. These effects increase phenotypic variance of traits, helping initiate differentiation. Moreover, increasing the number of loci increases the number of sites available for differentiation.

When the number of loci was large, and both female preference and sexual conflict were strong ($\mathcal{L} = 32$, $\alpha = 0.05$, and $P_{opt} = 0.2$), differentiation was frequently observed in both sexes (Table 4b), and a single pair of loci always contributed most to the phenotypic variance. This differentiation typically occurs at boundaries (allele 0 or allele 31), initiated by a small cluster of females who split away (they consequently do not suffer high compatibility with males) while the majority remain at the boundary. Males are concentrated near the boundary during this initial stage (because the majority of females are there) and subsequently get trapped between the (largest and second largest) female subclusters. In some simulations (Table 4b and Fig. 3E), differentiation in both traits resulted in sympatric speciation (i.e., the formation of reproductively isolated clusters).

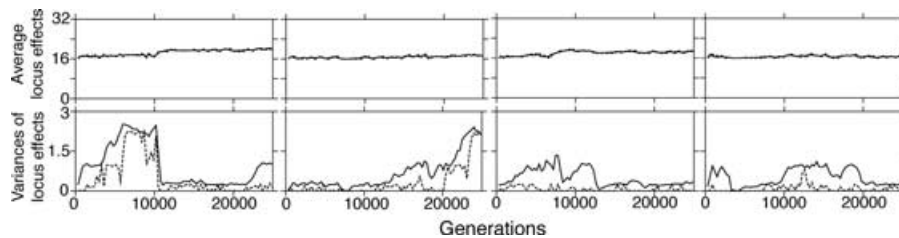


Figure 6. Coevolutionary dynamics in the genetic distance model with 32 loci. Examples for four pairs of loci are shown. Average locus effects and variances of locus effects of female (solid lines) and male (dashed line) loci. Average locus effects of both sexes are almost coincident. Parameters: $P_{opt} = 0.2$, $\alpha = 0.01$, $s = 1.02$, $\mu = 0.5 \times 10^{-5}$, $N = 10,000$.

Table 4. The effects of the optimal mating rate for females (P_{opt}) and the number of loci (\mathcal{L}) on the average mating rate (\bar{P}) and the likelihood of genetic differentiation in the phenotypic distance model. Shown is the median of \bar{P} , with the 10th and 90th percentiles in parentheses. \bar{P} is recorded at generation 25,000. Parameters: $s_c = 1.02$, $\mu = 5 \times 10^{-5}$, $N = 10,000$. (a): $\alpha = 0.01$. (b): $\alpha = 0.05$.

	\mathcal{L}	P_{opt}	\bar{P}	Differentiation ¹		
				F	M	SS
(a)	2	.2	.79 (.69, .88)	0	0	0
		.4	.91 (.86, .95)	0	0	0
		.6	.95 (.92, .98)	0	0	0
		.8	.96 (.94, .98)	9	0	0
	8	.2	.85 (.75, .90)	0	0	0
		.4	.91 (.84, .93)	0	0	0
		.6	.88 (.82, .95)	5	0	0
		.8	.94 (.93, .95)	4	0	0
32	.2	.85 (.77, .97)	0	0	0	
	.4	.77 (.70, .88)	7	0	0	
	.6	.81 (.81, .84)	18	0	0	
	.8	.91 (.90, .92)	0	0	0	
(b)	32	.2	.58 (.40, .60)	16	12	8
		.4	.65 (.64, .66)	0	0	0
		.6	.77 (.75, .78)	0	0	0
		.8	.91 (.90, .92)	0	0	0

¹The number of simulation runs (out of 20 runs) in which sympatric speciation (SS) or differentiation in any female locus (F) or any male locus (M) was observed at generation 25,000.

When the number of loci was not sufficiently large ($\mathcal{L} \leq 8$) or sexual conflict was not strong enough ($P_{opt} \geq 0.4$), strong female preference ($\alpha = 0.05$) did not significantly change qualitative results (data not shown). Decreasing mutation rate ($\mu = 10^{-5}$) decreased the frequency of genetic differentiation (data not shown). Increasing selection in females ($s_c = 4 \times 1.02$) greatly enhanced the speed of coevolutionary chase but suppressed genetic differentiation (data not shown). No differentiation was observed whenever the population size was small ($N = 1000$; data not shown). When we used the alternative scaling with the 32-locus model with $\alpha = 0.05 \times 16^{-2}$, the phenotypic distance model always exhibited continuous coevolutionary chase (data not shown).

DIRECT NATURAL SELECTION ON THE TRAITS

Natural selection applied only to males resulted in coevolution terminating in an equilibrium in which average mating rate was optimal for females (P_{opt}); females won the sexual conflict (Table 5a). In this case, a coevolutionary chase was initially observed wherein the average genetic or phenotypic distance between the sexes continuously increased. This terminated once the optimal mating rate for females was achieved (or allelic effects reached the boundary;

Table 5. The effects of natural selection on the female and male traits. Shown is the median of \bar{P} , with the 10th and 90th percentiles in parentheses. \bar{P} is recorded at generation 25,000. Parameters are the same as in the Table 2a unless specified. The genetic distance model with directional dominance is used unless specified. (a) Natural selection only in males: $s_x = 0$, $s_y = 0.02$, $\theta_y = 16$. (b) Natural selection only in females: $s_x = 0.02$, $s_y = 0$, $\theta_x = 16$. (c) Natural selection only in females in the phenotypic distance model: $s_x = 0.02$, $s_y = 0$, $\theta_x = 128$. (d) Natural selection in both sexes: $s_x = 0.02$, $s_y = 0.02$, $\theta_x = 16$, $\theta_y = 16$. No sympatric speciation was observed.

	P_{opt}	\bar{P}	Differentiation ¹		
			F	M	
(a)	.2	.36 (.36, .37)	0	0	
	.4	.41 (.41, .43)	0	0	
	.6	.61 (.61, .62)	0	0	
	.8	.85 (.83, .86)	6	0	
	(b)	.2	.47 (.44, .51)	20	20
		.4	.60 (.57, .61)	20	6
		.6	.76 (.75, .77)	20	0
		.8	.95 (.93, .96)	9	0
(c)	.2	.90 (.86, .94)	0	0	
	.4	.90 (.86, .95)	0	0	
	.6	.83 (.81, .84)	20	0	
	.8	.97 (.96, .97)	0	0	
(d)	.2	.46 (.43, .49)	20	7	
	.4	.59 (.57, .60)	20	0	
	.6	.76 (.73, .78)	20	0	
	.8	.94 (.92, .95)	8	0	

¹The number of simulation runs (out of 20 runs) in which differentiation in any female locus (F) or any male locus (M) was observed at generation 25,000.

allele 0 or 31). In the equilibrium state, no genetic differentiation was observed. This outcome was robust to differences in genetic details (i.e., the number of loci, the type of dominance, and the type of interaction between male and female traits). Given natural selection in males only, the “females win” outcome is reasonable; qualitatively similar results have already been shown in previous models (e.g., Gavrillets et al. 2001; Rowe et al. 2005).

When there was natural selection in females only, genetic differentiation was frequently observed in the genetic distance model both with directional dominance and codominance (Table 5b). The range of models and parameter configurations that caused genetic differentiation did not largely differ from what was observed without natural selection. When genetic differentiation occurred, average genetic distance between the sexes initially decreased to zero, followed by genetic differentiation in female loci. The average female and male locus effects were typically equal and optimal for female natural selection (i.e., equal to x_{opt}). With the same parameter configuration, average mating rates were typically higher

with natural selection than without natural selection (compare Table 2a and Table 5b); natural selection applied to females reduced the degree of differentiation in female loci (i.e., the variance in locus effects). This decrease in differentiation of female loci in turn decreases the likelihood of differentiation in male loci (compare Table 2a and Table 5b). In the phenotypic distance model and the genetic distance model in the additive case, cyclic coevolution (or continuous coevolutionary chase with frequent changes in direction) without genetic differentiation was commonly observed (Table 5c). When sexual conflict was moderate (i.e., $P_{\text{opt}} = 0.6$), genetic differentiation only in females was also frequently observed in the phenotypic distance model (Table 5c).

Natural selection in both sexes yields at least four possible outcomes. When natural selection in females was sufficiently stronger than that in males, or when optimal locus effects (x_{opt} and y_{opt}) were close, genetic differentiation was frequently observed in female loci (and, less frequently, in both female and male loci; Table 5d). These outcomes are similar to those under natural selection in females only (discussed above). If natural selection in females was not sufficiently stronger than that in males, and optimal locus effects were not sufficiently close, populations exhibited either cyclic coevolution or coevolution toward an equilibrium state, without genetic differentiation. In the equilibrium state, neither sex wins the conflict and the average mating rate \bar{P} is intermediate between P_{opt} and 1. The last two outcomes have already been shown in previous models (e.g., Gavrillets et al. 2001; Rowe et al. 2005).

Discussion

DYNAMICS OF REPRODUCTIVE TRAITS UNDER SEXUAL CONFLICT

Dynamic regimes

We used individual-based simulations to explore the dynamics of sexual conflict under diploid, multilocus, and multiallelic genetic conditions. Our results show that sexual conflict can exhibit at least the following five regimes: (1) continuous coevolutionary chase, (2) evolution toward an equilibrium, (3) cyclic coevolution, (4) extensive genetic differentiation in female traits/genes only, and (5) extensive genetic differentiation in both male and female traits/genes. These dynamics have been predicted in the previous models of sexual conflict based on simple genetics and/or analytical approximations (reviewed in Gavrillets and Hayashi 2005). Results from the individual-based simulations presented here support the generality of the theoretical conclusions from those previous models.

Stochastic effects and multilocus dynamics

Our simulations demonstrate that stochastic effects in multilocus systems can result in very complex dynamics. Different dynamic

regimes can emerge even within the same set of parameter values and initial conditions. In addition, the same population can switch from one regime to another as a result of stochastic perturbations. Moreover, different sets of loci controlling mating and fertilization in the same population can follow different dynamic regimes. These observations suggest that making precise predictions about the evolutionary dynamics of traits involved in sexual conflict may be difficult.

EFFECTS OF UNDERLYING GENETICS ON THE DYNAMICS OF SEXUAL CONFLICT

Our results demonstrate that evolutionary dynamics of sexual conflict strongly depend on genetic details. First, our simulations show that interaction between reproductive traits in a component-by-component manner (as in the genetic distance model) is more likely to cause genetic differentiation than trait-by-trait interaction (as in the phenotypic distance model). Second, the type of dominance strongly affects the likelihood of genetic differentiation. When alleles work in a dominant fashion (e.g., the directional dominance case in the genetic distance model) or codominant fashion (e.g., the codominance case in the genetic distance model), genetic differentiation is more frequently observed than when alleles work in an additive fashion (e.g., the phenotypic distance model and the additive case in the genetic distance model). Gavrillets and Waxman (2002) observed that genetic differentiation occurred under relatively broader conditions than those shown here. The ease of genetic differentiation in their model is partly due to their using a haploid model in which formation of heterozygous loci is not possible.

Third, our simulations show that the number of loci is also very important. When the interaction between reproductive traits occurs in a trait-by-trait way (as in the phenotypic distance model), the likelihood of differentiation increases with the number of loci (if we assume the maximum effect of a single mutation is independent of the number of loci). When the interaction between reproductive traits occurs in a component-by-component way (as in the genetic distance model) and the (maximum) effect of a single mutation is independent of the number of loci, increasing the number of loci typically results in female mating rates being closer to their optimal value. This implies that increasing the number of loci potentially benefits females. This is consistent with studies of abalone fertilization proteins that show that a sperm-protein-binding receptor molecule on the egg envelope (VERL) consists of a number of repetitive sequences that are apparently redundant (e.g., Galindo et al. 2002). The maintenance of these repetitive elements may be explained by the benefit of mediating an egg's fertilization rate.

In the numerical simulations presented here, each locus had 32 alleles. Our preliminary simulations (not presented here) suggested that differences in the number of alleles would not largely

affect our conclusions. However, those simulations also suggested that the effect of a small number of alleles (say, four or eight alleles for each locus) was to stabilize genetic differentiation, typically expressed as a bimodal distribution consisting of alleles having the smallest and largest effects. This increase in stability increased the likelihood of differentiation. Sympatric speciation was strongly suppressed, however, due to the limited possible degree of differentiation in a locus.

GENETIC DIFFERENTIATION BY SEXUAL CONFLICT

Genetic differentiation in female reproductive traits

Genetic variation in female reproductive traits has received relatively less attention than that in males (Jennions and Petrie 1997). However, empirical studies show that variation in female reproductive traits within a population is not rare (e.g., Wagner et al. 1995; Kodric-Brown and Nicolette 1996; Sota et al. 2000; Brooks and Endler 2001; Huber and González 2001; Swanson et al. 2001; Terai et al. 2002; Morris et al. 2003). In particular, Bergsten et al. (2001) suggest that variation of female dorsal structure in diving beetles is maintained by sexual conflict. Svensson et al. (2005) also conclude that sexual conflict maintains a color-polymorphism in females in damselfly species. Our results show that sexual conflict can cause differentiation in female reproductive traits under relatively broad conditions and parameter ranges. Female differentiation occurs even under seemingly unfavorable parameter values, such as weak sexual conflict ($P_{\text{opt}} = 0.8$ in Table 1), relatively low mutation rate ($\mu = 10^{-5}$ in Table 2), and the presence of direct natural selection in females (Table 5). Results presented here, and previously (Frank 2000; Gavrilets and Waxman 2002), suggest that sexual conflict is an important and plausible cause of differentiation in female reproductive traits.

Genetic differentiation in male reproductive traits

Genetic variation in male sexual traits has been of interest in modeling sexual selection (e.g., Pomiankowski and Moller 1995; Rowe and Houle 1996; Kotiaho et al. 2001). Our results show that sexual conflict can explain extensive genetic variation without assuming condition-dependent expression in male traits (Rowe and Houle 1996), modifiers that increase phenotypic variation (Pomiankowski and Moller 1995), or biased mutation (Pomiankowski and Iwasa 1991). Our simulations suggest that differentiation in males requires differentiation in females and strong sexual conflict (i.e., small P_{opt}); disruptive selection in males is caused by differentiation in females. When sexual conflict is weak, differentiation in females can occur, but the degree of differentiation is not enough to initiate differentiation in males. Strong female preference, directional dominance, moderate (in the genetic distance model) or large (in the phenotypic distance model) number of loci, high mutation rates, and large population size all promote differentiation in male reproductive traits.

Sympatric speciation by sexual conflict

Our results show that sympatric speciation by sexual conflict requires more strict conditions than previously suggested in Gavrilets and Waxman (2002). Gavrilets and Waxman (2002) used a deterministic, haploid, two-locus, and multiallele model. The models studied here incorporate diploidy, multiple loci, explicit stochastic effects (e.g., random genetic drift), and natural selection. It turns out that all of these factors act against sympatric speciation. First, sympatric speciation often requires a particular type of dominance. Second, the presence of multiple loci (per trait) can suppress sympatric speciation because of recombination. Third, random genetic drift can hinder the creation and maintenance of distinct allele clusters. Fourth, natural selection in reproductive traits suppresses sympatric speciation by limiting the degree of differentiation in the traits.

EVOLUTIONARY SIGNIFICANCE OF SEXUAL CONFLICT

Previous models of sexual conflict (reviewed in Gavrilets and Hayashi 2005; Rowe et al. 2005) highlighted the significance of sexual conflict and showed its various possible evolutionary outcomes. Based on the results from our individual-based simulations and the previous models, some generalizations about the plausibility of these outcomes can be made. Sympatric speciation is possible only under strict conditions and would not be expected as a common outcome of sexual conflict. Genetic differentiation (without sympatric speciation) occurs under relatively broad conditions, and the likelihood of differentiation primarily depends on the genetic architecture/mechanism. When there is no natural selection on the reproductive traits and no (extensive) genetic differentiation occurs, populations commonly exhibit continuous coevolutionary chase. It is also possible to achieve the outcome in which average female and male traits match the optimum (yet the mating rate can still be reduced by variance in females) and no coevolutionary chase occurs (e.g., figure 1 in Gavrilets 2000 and Fig. 6 in this article). Coevolution toward an equilibrium point is the most common outcome under natural selection on reproductive traits (cyclic coevolution or genetic differentiation can also frequently occur under some conditions). There are also possibilities that sexual conflict mechanisms not considered in this article cause additional outcomes (Rowe et al. 2005; Gavrilets and Hayashi 2006). For example, females can evolve indifference to male traits if female preference consists of two independent components, sensitivity and threshold (Rowe et al. 2005). In summary, the generalizations above suggest that sexual conflict can cause various outcomes whose plausibility depends on the underlying genetic/behavioral/physiological mechanisms. Revealing the underlying mechanisms is therefore required to assess the evolutionary significance of sexual conflict.

ACKNOWLEDGMENTS

We thank M. Shpak, C. Eaton, R. Dilling, L. Rowe, and two anonymous reviewers for providing constructive comments on this article. This work was supported by National Institutes of Health grant GM56693 and by National Science Foundation grant DEB-0111613.

LITERATURE CITED

- Arnegard, M. E., and A. S. Kondrashov. 2004. Sympatric speciation by sexual selection alone is unlikely. *Evolution* 58:222–237.
- Arnqvist, G., and T. Nilsson. 2000. The evolution of polyandry: multiple mating and female fitness in insects. *Anim. Behav.* 60:145–164.
- Arnqvist, G., and L. Rowe. 2002. Antagonistic coevolution between the sexes in a group of insects. *Nature* 415:787–789.
- . 2005. *Sexual conflict*. Princeton Univ. Press, Princeton, NJ.
- Bergsten, J., A. Töyrä, and A. N. Nilsson. 2001. Intraspecific variation and intersexual correlation in secondary sexual characters of three diving beetles (*coleoptera: Dytiscidae*). *Biol. J. Linn. Soc.* 73:221–232.
- Brooks, R., and J. A. Endler. 2001. Female guppies agree to differ: phenotypic and genetic variation in mate-choice behavior and the consequences for sexual selection. *Evolution* 55:1644–1655.
- Chapman, T., G. Arnqvist, J. Bangham, and L. Rowe. 2003. Sexual conflict. *Trends Ecol. Evol.* 18:41–47.
- Clement, M., D. Posada, and K. Crandall. 2000. TCS: a computer program to estimate gene genealogies. *Mol. Ecol.* 9:1657–1660.
- Crow, J.F. and M. Kimura. 1970. *An introduction to population genetic theory*. Harper and Row, New York.
- Frank, S. A. 2000. Sperm competition and female avoidance of polyspermy mediated by sperm-egg biochemistry. *Evol. Ecol. Res.* 2:613–625.
- Galindo, B. E., G. W. Moy, W. J. Swanson, and V. D. Vacquier. 2002. Full-length sequence of VERL, the egg vitelline envelope receptor for abalone sperm lysin. *Gene* 288:111–117.
- Gavrilets, S. 2000. Rapid evolution of reproductive isolation driven by sexual conflict. *Nature* 403:886–889.
- . 2004. *Fitness landscapes and the origin of species*. Princeton Univ. Press, Princeton, NJ.
- Gavrilets, S., and T. I. Hayashi. 2005. Speciation and sexual conflict. *Evol. Ecol.* 19:167–198.
- . 2006. The dynamics of two- and three-way sexual conflicts over mating. *Phil. Trans. Roy. Soc. Lond. B* 361:345–354.
- Gavrilets, S., and D. Waxman. 2002. Sympatric speciation by sexual conflict. *Proc. Natl. Acad. Sci. USA* 99:10533–10538.
- Gavrilets, S., G. Arnqvist, and U. Friberg. 2001. The evolution of female mate choice by sexual conflict. *Proc. R. Soc. B* 268:531–539.
- Haygood, R. 2004. Sexual conflict and protein polymorphism. *Evolution* 58:1414–1423.
- Holland, B., and W. R. Rice. 1998. Chase-away sexual selection: antagonistic seduction versus resistance. *Evolution* 52:1–7.
- . 1999. Experimental removal of sexual selection reverses intersexual antagonistic coevolution and removes a reproductive load. *Proc. Natl. Acad. Sci. USA* 96:5083–5088.
- Howard, D. J., M. Reece, P. G. Gregory, J. Chu, and M. L. Cain. 1998. The evolution of barriers to fertilization between closely related organisms. Pp. 279–288, in D. J. Howard and S. H. Berlocher, eds. *Endless forms: species and speciation*. Oxford Univ. Press, New York.
- Huber, B. A., and A. P. González. 2001. Female genital dimorphism in a spider (*Araneae: Pholcidae*). *J. Zool.* 255:301–304.
- Jennions, M. D., and M. Petrie. 1997. Variation in mate choice and mating preferences: a review of causes and consequences. *Biol. Rev.* 72:283–327.
- Kodric-Brown, A., and P. F. Nicolette. 1996. Consensus among females in their choice of males in the guppy *Poecilia reticulata*. *Behav. Ecol. Sociobiol.* 39:395–400.
- Kotiaho, J. S., L. W. Simons, and J. L. Tomkins. 2001. Towards a resolution of the lek paradox. *Nature* 410:684–686.
- Lande, R. 1981. Models of speciation by sexual selection on polygenic characters. *Proc. Natl. Acad. Sci. USA* 78:3721–3725.
- Maklakov, A. A., T. Bilde, and Y. Lubin. 2005. Sexual conflict in the wild: Elevated mating rate reduces female lifetime reproductive success. *Am. Nat.* 165:S38–S45.
- Martin, O., and D. Hosken. 2003. The evolution of reproductive isolation through sexual conflict. *Nature* 423:979–982.
- Morris, M. R., P. F. Nicolette, and E. Hesselman. 2003. A polymorphism in female preference for a polymorphic male trait in the swordtail fish *Xiphophorus cortezi*. *Anim. Behav.* 65:45–52.
- Nei, M., T. Maruyama, and C.-I. Wu. 1983. Models of evolution of reproductive isolation. *Genetics* 103:557–579.
- Parker, G. A. 1979. Sexual selection and sexual conflict. Pp. 123–166, in M. S. Blum and N. A. Blum, eds. *Sexual selection and reproductive competition in insects*. Academic Press, New York.
- . 2006. Sexual conflict over mating and fertilization: an overview. *Phil. Trans. Roy. Soc. Lond. B* 361:235–259.
- Pitnick, S., W. D. Brown, and G. T. Miller. 2001a. Evolution of female remating behavior following experimental removal of sexual selection. *Proc. R. Soc. B* 268:557–563.
- Pitnick, S., G. T. Miller, J. Reagan, and B. Holland. 2001b. Males' evolutionary responses to experimental removal of sexual selection. *Proc. R. Soc. B* 268:1071–1080.
- Pizzari, T., and R. R. Snook. 2003. Perspective: sexual conflict and sexual selection: chasing away paradigm shifts. *Evolution* 57:1223–1236.
- Pomiankowski, A., and Y. Iwasa. 1991. The evolution of costly mate preferences I. Fisher and biased mutation. *Evolution* 45:1422–1430.
- Pomiankowski, A., and A. P. Moller. 1995. A resolution of the lek paradox. *Proc. R. Soc. B* 260:21–29.
- Rice, W. R. 1996. Sexually antagonistic male adaptation triggered by experimental arrest of female evolution. *Nature* 381:232–234.
- . 1998. Intergenic conflict, interlocus antagonistic coevolution, and the evolution of reproductive isolation. Pp. 261–270, in D. J. Howard and S. H. Berlocher, eds. *Endless forms: species and speciation*. Oxford Univ. Press, New York.
- Rice, W. R., and B. Holland. 1997. The enemies within: intergenomic conflict, interlocus contest evolution (ICE), and intraspecific Red Queen. *Behav. Ecol. Sociobiol.* 41:1–10.
- Ritchie, M. G., and S. D. F. Phillips. 1998. The genetics of sexual isolation. Pp. 291–308, in D. J. Howard and S. H. Berlocher, eds. *Endless forms: species and speciation*. Oxford Univ. Press, New York.
- Rowe, L., and D. Houle. 1996. The lek paradox and the capture of genetic variance by condition dependent traits. *Proc. R. Soc. B* 263:1415–1421.
- Rowe, L., E. Cameron, and T. Day. 2003. Detecting sexually antagonistic coevolution with population crosses. *Proc. R. Soc. B* 270:2009–2016.
- . 2005. Escalation, retreat, and female indifference as alternative outcomes of sexually antagonistic coevolution. *Am. Nat.* 165:S5–S18.
- Sota T., F. Kusumoto, and K. Kubota. 2000. Consequences of hybridization between *Ohomopterus insulicola* and *O. arrowianus* (*Coleoptera, Carabidae*) in a segmented river basin: parallel formation of hybrid swarms. *Biol. J. Linn. Soc.* 71:297–313.
- Svensson, E. L., J. Abbot, and R. Hardling. 2005. Female polymorphism, frequency dependence, and rapid evolutionary dynamics in natural populations. *Am. Nat.* 165:567–576.

Swanson, W. J., and V. D. Vacquier. 2002. The rapid evolution of reproductive proteins. *Nat. Rev. Genet.* 3:137–144.

Swanson, W. J., C. F. Aquadro, and V. D. Vacquier. 2001. Polymorphism in abalone fertilization proteins is consistent with the neutral evolution of the egg's receptor for lysin (VERL) and positive Darwinian selection of sperm lysin. *Mol. Biol. Evol.* 18:376–383.

Templeton, A. R., K. A. Crandall, and C. F. Sing. 1992. A cladistic analysis of phenotypic associations with haplotypes inferred from restriction endonuclease mapping and DNA sequence data. *Genetics* 132:619–633.

Terai Y., W. E. Mayer, J. Kelin, H. Tichy, and N. Okada. 2002. The effect of selection on a long wavelength-sensitive (*LWS*) opsin gene of lake victoria cichlid fishes. *Proc. Natl. Acad. Sci. USA* 99:15501–15506.

Wagner, W. E. J., A. M. Murray, and W. H. Cade. 1995. Phenotypic variation in the mating preferences of female field crickets, *Gryllus integer*. *Anim. Behav.* 49:1269–1281.

Associate Editor: L. Rowe

APPENDIX

Table A1. Glossary of notation

x	female trait
y	male trait
ψ	preference function
P	mating rate of a female
\bar{P}	average mating rate of females in population
P_{opt}	optimal mating rate for females
\mathcal{L}	number of loci
α	strength of female preference
f_i, f'_i	allelic effects of the two (maternal and paternal) alleles in the i th female locus
m_i, m'_i	allelic effects of the two (maternal and paternal) alleles in the i th male locus
d	phenotypic or genetic distance between female and male traits.
d_i	genetic distance between i th female locus and corresponding i th male locus.
s_c	strength of selection (by sexual conflict) on female
μ	mutation rate per locus
N	population size
x_i	locus effect of the i th female locus
y_i	locus effect of the i th male locus
s_x	strength of natural selection on the female trait
s_y	strength of natural selection on the male trait
x_{opt}	optimal phenotype for the female trait with respect to natural selection
y_{opt}	optimal phenotype for the male trait with respect to natural selection