EVOLUTION

INTERNATIONAL JOURNAL OF ORGANIC EVOLUTION

PUBLISHED BY

THE SOCIETY FOR THE STUDY OF EVOLUTION

Vol. 57 **November 2003** **No. 11**

Evolution, 57(11), 2003, pp. 2433–2449

SEXUAL DIMORPHISM AND ADAPTIVE SPECIATION: TWO SIDES OF THE SAME ECOLOGICAL COIN

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Abstract.—Models of adaptive speciation are typically concerned with demonstrating that it is possible for ecologically driven disruptive selection to lead to the evolution of assortative mating and hence speciation. However, disruptive selection could also lead to other forms of evolutionary diversification, including ecological sexual dimorphisms. Using a model of frequency-dependent intraspecific competition, we show analytically that adaptive speciation and dimorphism require identical ecological conditions. Numerical simulations of individual-based models show that a single ecological model can produce either evolutionary outcome, depending on the genetic independence of male and female traits and the potential strength of assortative mating. Speciation is inhibited when the genetic basis of male and female ecological traits allows the sexes to diverge substantially. This is because sexual dimorphism, which can evolve quickly, can eliminate the frequency-dependent disruptive selection that would have provided the impetus for speciation. Conversely, populations with strong assortative mating based on ecological traits are less likely to evolve a sexual dimorphism because females cannot simultaneously prefer males more similar to themselves while still allowing the males to diverge. This conflict between speciation and dimorphism can be circumvented in two ways. First, we find a novel form of speciation via negative assortative mating, leading to two dimorphic daughter species. Second, if assortative mating is based on a neutral marker trait, trophic dimorphism and speciation by positive assortative mating can occur simultaneously. We conclude that while adaptive speciation and ecological sexual dimorphism may occur simultaneously, allowing for sexual dimorphism restricts the likelihood of adaptive speciation. Thus, it is important to recognize that disruptive selection due to frequency-dependent interactions can lead to more than one form of adaptive splitting.

Key words.—Adaptive dynamics, disruptive selection, evolutionary branching, resource partitioning, stable fitness minima, sympatric speciation.

Received October 9, 2002. Accepted May 29, 2003.

Traditionally, evolutionary biologists have thought that speciation is initiated by a phase of geographic isolation between subpopulations of an ancestral lineage (Mayr 1963). Over evolutionary time, these allopatric populations diverge genetically, either in response to drift or different selection regimes. As a by-product of the different evolutionary trajectories, the two emerging species become reproductively isolated. Even though adaptations may be the cause of the differentiation in such allopatric speciation scenarios, the initial process of splitting the gene pool is not itself adaptive and instead is generated by external forces leading to geographic isolation.

In contrast, the past years have seen a renewed interest in a process termed ‘‘adaptive speciation,’’ in which the splitting itself is an adaptation (Dieckmann et al. 2003). This interest has been spurred on the one hand by a number of empirical studies suggesting that speciation can occur under nonallopatric conditions (e.g., Schliewen et al. 1994; Bernatchez et al. 1996; Shaw et al. 2000; Wilson et al. 2000; Schliewen et al. 2001; Via 2001), and on the other hand by theoretical advances showing that adaptive speciation is a theoretically plausible process (reviewed in Turelli et al. 2001).

Theoretical models of adaptive speciation must specify the ecological mechanisms generating the disruptive selection regime that renders the splitting adaptive. Under the classical view of static fitness landscapes, disruptive selection (e.g., through bimodal niches) is unlikely to be important for adaptive splitting because a population whose mean phenotype is close to a fitness minimum will simply evolve directionally

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away from it, making evolutionary diversification unlikely. In contrast, frequency-dependent interactions induce fitness landscapes that change dynamically in response to changes in the phenotype distribution. In particular, if disruptive selection is generated by frequency-dependent interactions, a perturbation to the mean phenotype away from a fitness minimum can induce changes in the fitness landscape that drive the population back toward the state in which fitness turns disruptive. Such evolutionary stability of fitness minima has been found in several different models (e.g., Eshel 1983; Brown and Pavlovic 1992; Abrams et al. 1993; Christiansen 1991; Geritz et al. 1998; Doebeli and Dieckmann 2000; Doebeli and Dieckmann 2003). Consequently, models of adaptive speciation typically involve frequency-dependent intraspecific interactions.

In asexual models, the process of convergence to a regime of disruptive selection is often followed by adaptive splitting into separate lineages. This phenomenon is called ''evolutionary branching'' in the theoretical framework of adaptive dynamics, an analytic approximation for the evolution of mean phenotypes (Metz 1996; Geritz et al. 1998). An important insight gained from adaptive dynamics theory is that evolutionary branching is a generic and robust phenomenon in many different models of evolution due to frequency-dependent ecological interactions (e.g., Geritz et al. 1998; Kisdi 1999; Doebeli and Dieckmann 2000, 2003). The most common ecological setting used to illustrate evolutionary branching entails frequency-dependent competition for a limiting resource. In the corresponding models, populations typically evolve under directional selection toward the phenotype best suited to the most abundant resource, the density-dependent optimum (Bolnick 2001). Once there, this most common phenotype experiences disproportionately intense competition, and hence has lowest fitness. Accordingly, such models readily exhibit evolutionary branching in clonal populations (Dieckmann and Doebeli 1999; Kisdi and Geritz 1999; Doebeli and Dieckmann 2000). In sexual populations, however, adaptive splitting requires assortative mating mechanisms that prevent the production of phenotypically intermediate offspring. Consequently, a primary aim of recent models of adaptive speciation has been to demonstrate that the emergence of disruptive selection can favor the evolution of assortative mating and subsequent evolutionary branching in sexual populations (Dieckmann and Doebeli 1999; Doebeli and Dieckmann 2000, 2003). However, adaptive speciation is not the only possible outcome of convergence toward disruptive selection.

Other evolutionary mechanisms can widen the phenotypic distribution of the population, equalizing the effects of competition (and hence equalizing fitness) across all phenotypes (Roughgarden 1972). In particular, adaptive splitting can occur intraspecifically, as seen in ontogenetic niche shifts, resource polymorphisms, and ecological sexual dimorphisms (Slatkin 1984). By flattening the fitness function, these alternative forms of phenotypic expansion may eliminate the disruptive force that would have driven speciation. It thus becomes important to understand the genetic conditions that may give rise to alternative escape routes from stable fitness minima and the relative rates with which different alternatives might evolve.

In this paper we demonstrate that a single model of intraspecific competition can give rise to ecological sexual dimorphism and/or adaptive speciation, and we explore how mating behavior and genetic assumptions about trait determination in males and females affect the relative likelihood of these two outcomes. Using an adaptive dynamics model for quantitative traits in males and females determining competition for a limiting resource, we first show that frequency-dependent competition can favor an ecological sexual dimorphism and that the ecological conditions for evolutionary branching and for sexual dimorphism are identical. We then use individual-based models incorporating explicit genetics and assortative mating to examine the prerequisites for sexual dimorphism and speciation.

DETERMINISTIC MODEL OF SEXUAL DIMORPHISM

In Slatkin's (1984) quantitative genetic models sexual dimorphism is a consequence of competitive displacement, and the ecological mechanisms driving evolutionary change bear a striking resemblance to models of adaptive speciation (e.g., Doebeli 1996; Dieckmann and Doebeli 1999). Here we present a deterministic model of sexual dimorphism using the same underlying ecological dynamics as in Slatkin (1984) and Dieckmann and Doebeli (1999). The resulting equations serve as the basis for the individual-based models used in subsequent sections to examine the interaction between dimorphism and speciation.

Males and females are characterized by a quantitative character z (e.g., body size) that determines ecological interactions, denoted by z_m in males and by z_f in females. To derive the deterministic dynamics, we assume that there is no genetic covariance between z_m and z_f, for example, the ecological trait z is determined by independent sets of loci in males and females. We assume that males pass on their phenotype to sons and females pass on their phenotype to daughters, so the value of a parent's trait is only important to progeny of the same sex. Resources are most abundant for individuals with some intermediate character value z_0, which we arbitrarily set at $z_0 = 0$. Specifically, we assume that populations that are monomorphic for character value z have carrying capacity

$$K(z) = K_0 \exp\left[-\frac{(z - z_0)^2}{2\sigma_k^2}\right]. \qquad (1)$$

Here K_0 scales the maximal carrying capacity, and σ_k measures how fast resource availability decreases with increasing phenotypic distance from the optimal trait value z_0.

To incorporate frequency-dependence, the driving force of competitive displacement, we assume that the strength of competition between individuals with phenotypes z and z' decreases with phenotypic distance and is given by

$$c(z, z') = \exp\left[-\frac{(z - z')^2}{2\sigma_c^2}\right], \qquad (2)$$

where σ_c measures how fast competitive impacts decrease with an increase in the phenotypic distance between interacting individuals. Thus, σ_c determines the strength of frequency dependence in the competitive interactions, with small σ_c corresponding to a high degree of frequency dependence. Note that we assume that the carrying capacity (eq. 1) and effects of competition between two individuals (eq. 2) are independent of the sex of the competing individuals.

Deterministic adaptive dynamics models assume that evolution is mutation limited and that the phenotype distribution of a resident population determines the invasion success of new mutants (Dieckmann and Law 1996; Metz et al. 1996; Geritz et al. 1998). We first calculate the ecological equilibrium population sizes for a population in which females and males are each monomorphic for some trait value z_f and z_m, respectively. Based on this equilibrium state, on the carrying capacity function $K(z)$, equation (1), and on the competition function $c(z, z')$, equation (2), we then calculate the per capita growth rate of any given rare mutant phenotype in either males or females. In the limit of very small mutations, these growth rates yield selection gradients in both males and females, from which the adaptive dynamics are deduced.

Here we assume that the underlying model for the ecological dynamics is a discrete-time model with nonoverlapping generations given by the Beverton-Holt equation,

$$n(t + 1) = \frac{rn(t)}{1 + \frac{r - 1}{K}n(t)}, \tag{3}$$

where $n(t)$ and $n(t + 1)$ are population densities in successive generations, r is the per capita number of offspring, and K is the carrying capacity. In the Appendix, we describe how this model can be adopted to describe the dynamics of male and female population densities and calculate equilibrium population densities of monomorphic males and females.

Using these equilibrium densities, one can calculate the growth rates of rare mutant males and females in a resident population (z_m, z_f), which are given by two functions $w_m(z'_m, z_m, z_f)$ for mutant males z'_m and $w_m(z'_f, z_m, z_f)$ for mutant females z'_f (see Appendix). From these growth rates, one obtains the selection gradients for male and female traits in a resident population (z_m, z_f) as

$$g_m(z_m, z_f) = \left.\frac{\partial w_m}{dz'_m}\right|_{z'_m=z_m} \quad \text{and} \tag{4a}$$

$$g_f(z_m, z_f) = \left.\frac{\partial w_f}{dz'_f}\right|_{z'_f=z_f}. \tag{4b}$$

These gradients describe the adaptive dynamics of the male and female traits z_m and z_f. In particular, equilibrium points of the adaptive dynamics are points z^*_m, z^*_f in phenotype space at which the gradients g_m and g_f vanish simultaneously. It is shown in the Appendix that the point $(z^*_m, z^*_f) = (0,0)$ is always an equilibrium point of the adaptive dynamics (recall that we assumed that $z_0 = 0$ is the trait value maximizing the carrying capacity). This equilibrium (0, 0) is locally stable if and only if $\sigma_c > \sigma_k$, where σ_k and σ_c are the parameters determining the width of the carrying capacity function, equation (1), and the strength of frequency dependence in the competition function, equation (2), respectively. Thus, if the effect of frequency dependence is weak relative to the stabilizing selection imposed by the unimodal resource distribution, both sexes will adapt to the modal resource and no dimorphism will occur. We note that if the equilibrium $(z^*_m, z^*_f) = (0,0)$ is locally stable for the adaptive dynamics, then it is also evolutionarily stable in the sense that the invasion fitness functions w_m and w_f have a maximum with respect to the mutant trait values at the equilibrium.

Conversely, if frequency dependence is relatively strong ($\sigma_c < \sigma_k$), then the symmetric equilibrium (0,0) is unstable, and instead there is a new equilibrium (z^*_m, z^*_f) of the adaptive dynamics given by

$$z^*_m = -z^*_f = \sigma_c\sqrt{\frac{1}{2}\log\left(\frac{2\sigma^2_k}{\sigma^2_c} - 1\right)}. \tag{5}$$

This equilibrium represents a sexual dimorphism, and it exists if and only if

$$\sigma_c < \sigma_k. \tag{6}$$

If the dimorphic equilibrium exists, then so does the equilibrium with the male and female trait values reversed, and both equilibria are locally stable. Note that inequality (6) is essentially the same condition as that found by Slatkin (1984) for the evolution of sexual dimorphism, namely $V < \sigma_k - \sigma_c$, where V is the phenotypic variance. In the present model $V = 0$ because of our assumption, necessary to derive the adaptive dynamics, that each sex is monomorphic. With $V = 0$, Slatkin's (1984) condition and condition (6) above are identical. Our analysis also confirms Slatkin's (1984) conjecture that if the symmetric equilibrium $(z^*_m, z^*_f) = (0,0)$ is unstable, then there is a stable asymmetric equilibrium (z^*_m, z^*_f) given by equation (5), inducing convergence of the evolutionary dynamics toward a sexually dimorphic state. It is important to note, however, that while this equilibrium is an attractor in the two-dimensional phenotype space of male and female trait values, the equilibrium is not evolutionarily stable. This can be seen by considering the second derivatives of the fitness functions w_m and w_f with respect to mutant trait values: one can show that both these functions actually have a minimum at the equilibrium (5) whenever this equilibrium exists. In other words, the dimorphic equilibria are themselves potential evolutionary branching points for further niche partitioning. Nevertheless, the traits will not undergo evolutionary branching in randomly mating populations, for which the dimorphic equilibrium (5) therefore represents the evolutionary end state.

However, evolutionary branching could occur in principle when mating is assortative. This is exemplified by models of adaptive speciation (Dieckmann and Doebeli 1999). These speciation models use the same basic ecological assumptions as the dimorphism model above, but they are different in that males and females are assumed to always have identical ecological phenotypes but may exhibit nonrandom mating. Thus, sexual dimorphism and adaptive speciation are expected to occur under similar ecological conditions, but for different genetic assumptions.

There are two main reasons why one might expect that the two processes would be mutually exclusive. First, whichever form of divergence evolves first would tend to eliminate the disruptive selection necessary to drive the other. Second, positive assortative mating, which is generally deemed necessary for speciation, might be incompatible with dimorphism as long as it is based on male and female similarity in ecological traits. Nevertheless, the fact that the dimorphic equilibrium (5) is an evolutionary branching point could lead to interesting interactions between sexual dimorphism and assortative mating. To explore these issues we develop an individual-based numerical model with explicit genetics in which both processes can be studied simultaneously.

STOCHASTIC MODEL OF SEXUAL DIMORPHISM

We first assume that mating is random and concentrate on the dynamics of sexual dimorphism using individual-based models with explicit multilocus genetics. As before, the population is subject to the ecological dynamics in equations (1–3). Rather than simply assuming each sex is monomorphic, however, each individual is now assigned a genotype that in turn determines its ecological trait value.

In their numerical models of sympatric speciation, Dieckmann and Doebeli (1999) assigned each diploid individual N loci of equal additive effect. Each locus had two alleles, with phenotypic value -1 or 1. An individual's phenotype value was the sum of the additive values of all $2N$ alleles, ranging from $-2N$ to $2N$. We refer to this as the basic multilocus approach and use it later in this paper to model the degree of assortative mating and an unlinked mating phenotype. However, this approach is insufficient for modeling sexual dimorphism, because it does not allow one to vary the degree to which male and female phenotypes are genetically independent.

To model sexual dimorphism, we used a similar additive diploid multilocus approach but incorporated loci that are only expressed in one sex or another. The total number of loci expressed by any one individual (N_{total}) can be divided into those loci that are expressed in both sexes (N_{shared}) and those that are only expressed in males (N_{male}) or females (N_{female}), so $N_{total} = N_{shared} + N_{male} = N_{shared} + N_{female}$. This scheme reflects recent quantitative trait loci (QTL) studies of sexually dimorphic quantitative traits that have revealed numerous sex-specific QTL (Mogil et al. 1997; Nuzhdin et al. 1997; Agulnik et al. 1998; Gurganus et al. 1999; Ramos et al. 1999; Kopp et al. 2003). We assume that $N_{male} = N_{female}$ so that N_{total} is the same for both sexes. As with the basic model, each locus has two alleles, with allele j at locus i having two possible values:

$$locus_{ij} = \begin{cases} 1 \\ -1. \end{cases} \tag{7}$$

The value of an individual's ecological phenotype is then the sum of the allele values at all the loci that the individual expresses (shared loci plus the loci for the relevant sex):

$$z_m = \sum_{i}^{N_{shared}} \sum_{j}^{2} locus_{ij} + \sum_{i}^{N_{male}} \sum_{j}^{2} locus_{ij} \quad \text{and} \tag{8}$$

$$z_f = \sum_{i}^{N_{shared}} \sum_{j}^{2} locus_{ij} + \sum_{i}^{N_{female}} \sum_{j}^{2} locus_{ij}. \tag{9}$$

Phenotypes may range from $-2N_{total}$ to $2N_{total}$. In the simulations we standardized the range of traits from -1 to 1, for example, we divided the values obtained from equations (8) and (9) by $2N_{total}$.

This scheme for explicitly modeling male and female ecological character values allows us to manipulate a population's genetic capacity for sexual dimorphism. When $N_{shared} = N_{total}$, there are no sex-specific loci, and hence male and female trait means cannot possibly diverge. As N_{shared} becomes smaller the sexes can diverge more, until their traits are completely independent at $N_{shared} = 0$. Hence by manipulating the proportion of shared loci (N_{shared}/N_{total}) we can control a population's potential for sexual dimorphism.

For the numerical simulations, we initialize populations by assigning individuals a sex and a genotype, which in turn determines the individuals' ecological phenotype. The distribution of the ecological phenotypes then determines individual survival probabilities, and hence the ecological dynamics of the population. At each time step t, each individual survives with probability

$$P(z) = \frac{1}{1 + \dfrac{r - 1}{K(z)} n_{eff,z}(t)}, \tag{10}$$

where z is the individual's trait value, $K(z)$ is the carrying capacity of the phenotype (eq. 1), and $n_{eff,z}(t)$ is the effective population size that the individual experiences in the current population. Using the competition function $c(z, z')$, equation (2), this effective density is calculated as

$$n_{eff,z}(t) = \sum_{z'} c(z, z')n_{z'}(t), \tag{11}$$

where the sum runs over all possible trait values z', and where $n_{z'}(t)$ is the number of individuals with trait value z'. Note that, in slight abuse of notation, we now use the symbol n to denote actual population size rather than population density, because in the individual-based models we are dealing with actual numbers of individuals. Note also that $n_{eff,z}(t)$ includes both males and females.

Each of the surviving females reproduces by randomly choosing one of the surviving males as a mate and then producing a number of offspring drawn from a Poisson distribution with mean r. The genotype of each offspring is determined probabilistically from the parent genotypes under the assumptions of Mendelian segregation within loci and free recombination between loci. Each allele in the offspring has a probability $\mu = 0.001$ of reversing its value due to mutation. This unusually high mutation rate was chosen to speed up the simulations. (We note that our genetic architecture can be understood more generally as describing independent stretches of DNA of variable length that affect the trait under consideration additively and that recombine freely with other such stretches of DNA. In particular, such stretches might be much longer than a single locus, hence the mutation rate per such stretch might be quite high.) The offspring is randomly made male or female with equal probability, which then determines the subset of loci it will use to express its ecological trait z. The resulting population of offspring is then subjected again to ecological dynamics and mating, and this generational cycle is repeated iteratively.

An example of the evolutionary dynamics of male and female trait values emerging from this individual-based model is shown in Figure 1 for a case when $N_{shared}/N_{total} = 0.4$. The population was initialized with all individuals having an ecological phenotype of $z = -1$ (all loci fixed with

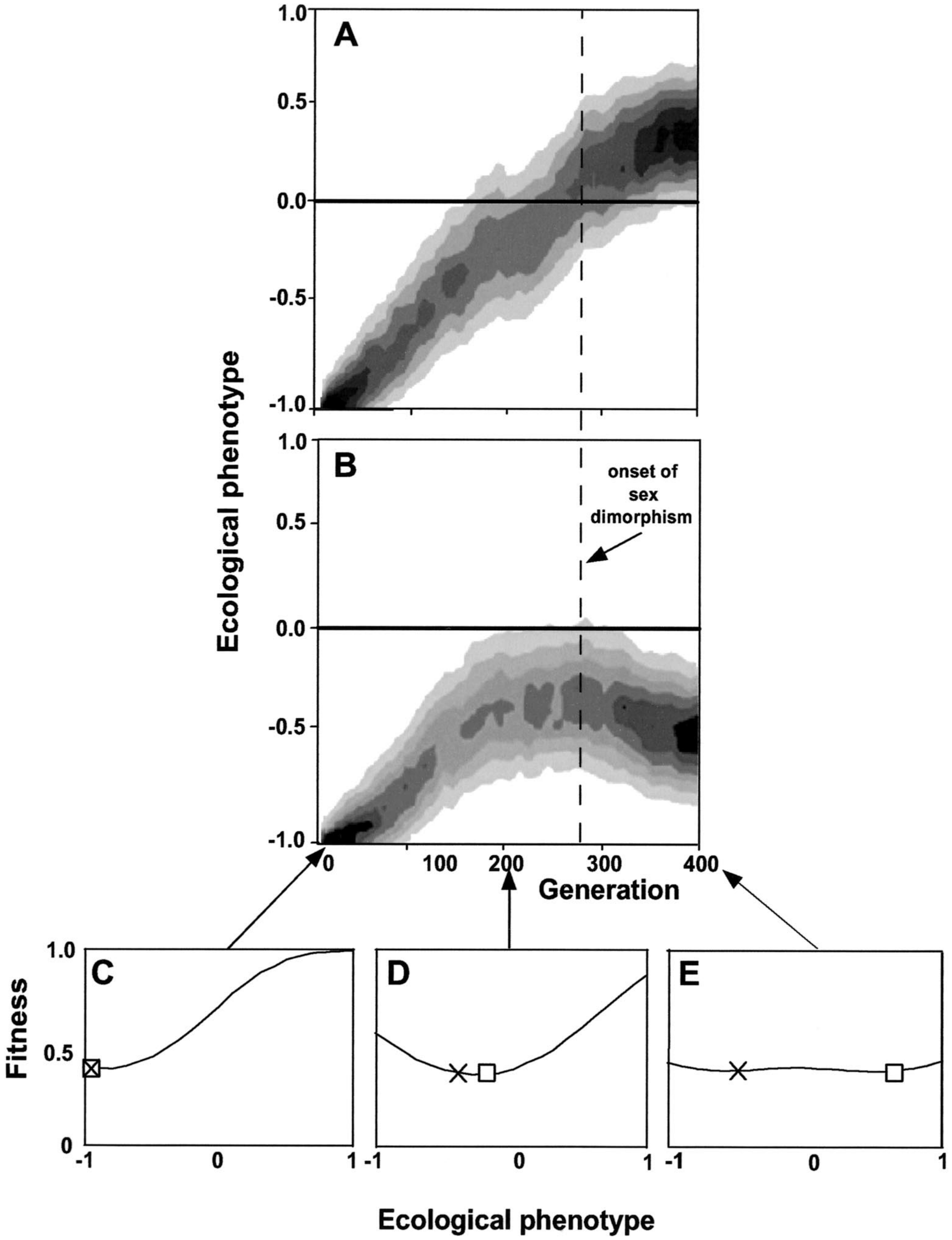

FIG. 1. Simulation of the evolution of sexual dimorphism as male (A) and female (B) phenotype distributions change over time in response to resource competition. Darker shading indicates a greater number of individuals of a given phenotype at a given time. The horizontal line at 0.0 in (A) and (B) indicates the location of the maximum of the carrying capacity curve. Fitness functions are shown for generation 10 (C), 200 (D), and 400 (E), with the mean phenotypes for males (□) and females (×). The dashed vertical line marks the onset of sexual dimorphism. Both sexes were initially monomorphic for the ecological trait $z = -1.0$. $N_{shared}/N_{total} = 0.4$, $s = 10$, $K_0 = 5000$, $\sigma_k = 1.0$, $\sigma_c = 0.25$, $r = 5$, $\mu = 0.001$, $N_{total} = 10$, $N_{assort} = 5$.

allele values of -1), and both males (Fig. 1A) and females (Fig. 1B) quickly evolved under directional selection (Fig. 1C) toward the density-dependent phenotypic optimum of $z = 0$. As the population mean approached this optimum, it came under disruptive selection due to frequency-dependent competition (Fig. 1D). A sexual dimorphism then evolved (Fig. 1A, B), flattening the fitness function so that all phenotypes had nearly equal fitness (Fig. 1E). Similar dynamics are seen for any run in which $\sigma_c < \sigma_k$ and N_{shared}/N_{total} is less than one. The direction of the resulting dimorphism ($\bar{z}_m < \bar{z}_f$ or $\bar{z}_m > \bar{z}_f$) is arbitrary and varies between replicate simulations.

The degree of sexual dimorphism that results from disruptive selection is sensitive to two sets of parameters. The ecological parameters σ_k and σ_c determine the location of the dimorphic equilibrium given by equation (5), to which males and females will evolve in the nongenetic, analytical model of the previous section. However, in the genetic in-

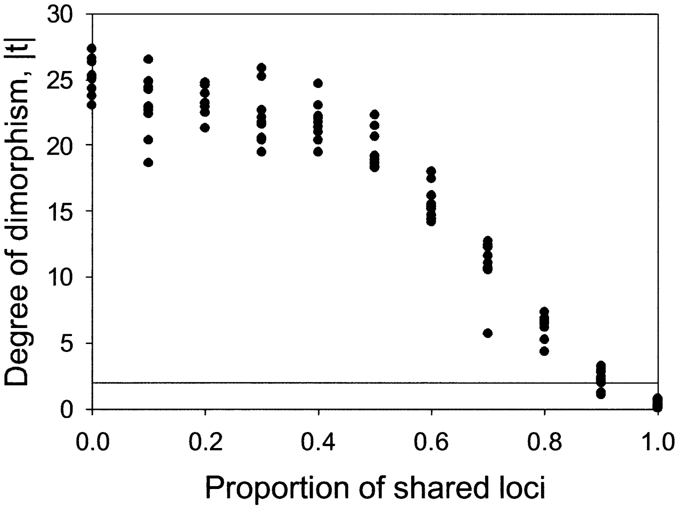

FIG. 2. The magnitude of ecological sexual dimorphism depends on the proportion of loci shared between males and females (N_{shared}/N_{total}).The magnitude of dimorphism is measured by the absolute value of Student's t-statistic. The horizontal line at $|t| = 2$ indicates the cut-off below which dimorphism is not statistically significant. Parameter values were: $s = 10$, $K_0 = 1000$, $\sigma_k = 0.75$, $\sigma_c = 0.25$, $r = 5$, $\mu = 0.001$, $N_{total} = 10$, $N_{assort} = 5$, with 10 replicates for each value of N_{shared}/N_{total}, run for 1000 generations each.

dividual-based model, N_{shared}/N_{total} can constrain whether the sexes are able to evolve to these values, as illustrated in Figure 2.

When between-sex covariance is high, the sexes are unable to diverge and so the population remains at the intermediate phenotype value z_0, subject to a stable fitness minimum. Such persistent disruptive selection is the starting point for adaptive speciation (Dieckmann and Doebeli 1999). In the next section we incorporate assortative mating mechanisms into our individual-based model to illustrate that when $N_{shared}/N_{total} = 1$ the population can escape from the fitness minimum via speciation. We then combine the assortative mating model with variable values of N_{shared}/N_{total} to investigate how speciation and sexual dimorphism interact.

STOCHASTIC MODEL OF ADAPTIVE SPECIATION

As before, all individuals possess an ecological phenotype determined by the additive effect of N_{total} diallelic loci. The ecological dynamics remain the same, but in contrast to the preceding section, $N_{shared} = N_{total}$ so dimorphism is impossible, and females may choose their mate nonrandomly. Assortative mating is described by a mate-choice function (Fig. 3) that determines the probability of a given female accepting a given male as a mate. Females vary with respect to a new quantitative genetic trait for assortability, a, which determines the degree to which a female mates assortatively. As with the ecological trait, the value of a represents the sum of allele values of N_{assort} independent diallelic loci, standardized to range from -1 to 1. While the N_{assort} loci are present in both males and females and inherited according to normal Mendelian rules, only females express their assortability, in keeping with female-limited mating (see Appendix). Negative values of a confer disassortative mating, positive values lead to positive assortative mating, and values near zero produce random mating.

The probability that a female with assortative phenotype a and ecological trait z_f will mate with a male with ecological trait z_m is described by the following mate-choice function (Fig. 3):

$$P(a, z_f, z_m) = \begin{cases} \exp\left[-\frac{1}{2}\left(\frac{a^2}{s}\right)^2 |z_f - z_m|^2\right] & \text{for } a > 0 \\ 1 & \text{for } a = 0 \\ \exp\left[-\frac{1}{2}\left(\frac{a^2}{s}\right)^2 (2 - |z_f - z_m|)^2\right] & \text{for } a < 0. \end{cases} \qquad (12)$$

Here s is a scaling parameter that determines the slope of the mate-choice function. Large values of s flatten the function $P(a, z_f, z_m)$ for all a (Fig. 3B), so that all phenotypes are equally acceptable, in which case even females with assortative trait values a close to -1 or 1 will mate randomly. As s decreases, the probability that a female mates with a phenotypically different (phenotypically similar) male drops off more and more rapidly for females with positive (negative) values of a (see Fig. 3A). In the numerical simulations, we assumed that every surviving female mates, implying that the mate-choice function (12) only determines the relative probabilities with which each male is chosen by a particular female. Thus, we assumed that there is no cost to a female for assortative mating (see Discussion), though males with rare ecological phenotypes are penalized.

Simulations in which dimorphism was restricted (i.e., $N_{shared}/N_{total} = 1$), and females initially mated randomly, on average, confirmed that adding the potential for assortative mating allowed populations to escape their stable fitness minima at $z = 0$ via speciation. The results of one such simulation are illustrated in Figure 4. The population was initialized with equal probabilities of -1 and $+1$ alleles at all mating loci so trait a is polymorphic with mean zero. The population initially evolves toward the fitness peak corresponding to the most abundant resource ($z = 0$) and the directional selection changes to disruptive selection as the population approaches $z = 0$. Assortative mating then evolves in a process analogous to reinforcement (Fig. 4C). This allows the population to split into two ecologically distinct species, as shown by the branching in both males and females (Fig. 4A, B). Note that because of strong assortative mating, the two emerging species are reproductively isolated. The increase in phenotypic variation resulting from evolutionary branching equalizes the fitness across different phenotypes, flattening the fitness function (Fig. 4F).

SIMULTANEOUS MODEL OF DIMORPHISM AND SPECIATION

Given that disruptive selection can result in either sexual dimorphism or sympatric speciation, we now turn to the question of which outcome is more likely when considered simultaneously. Because both forms of divergence have identical ecological prerequisites ($\sigma_c < \sigma_k$), we focus our attention on the potentially important genetic parameters of this model: N_{shared}/N_{total} and s. The former parameter puts an upper bound

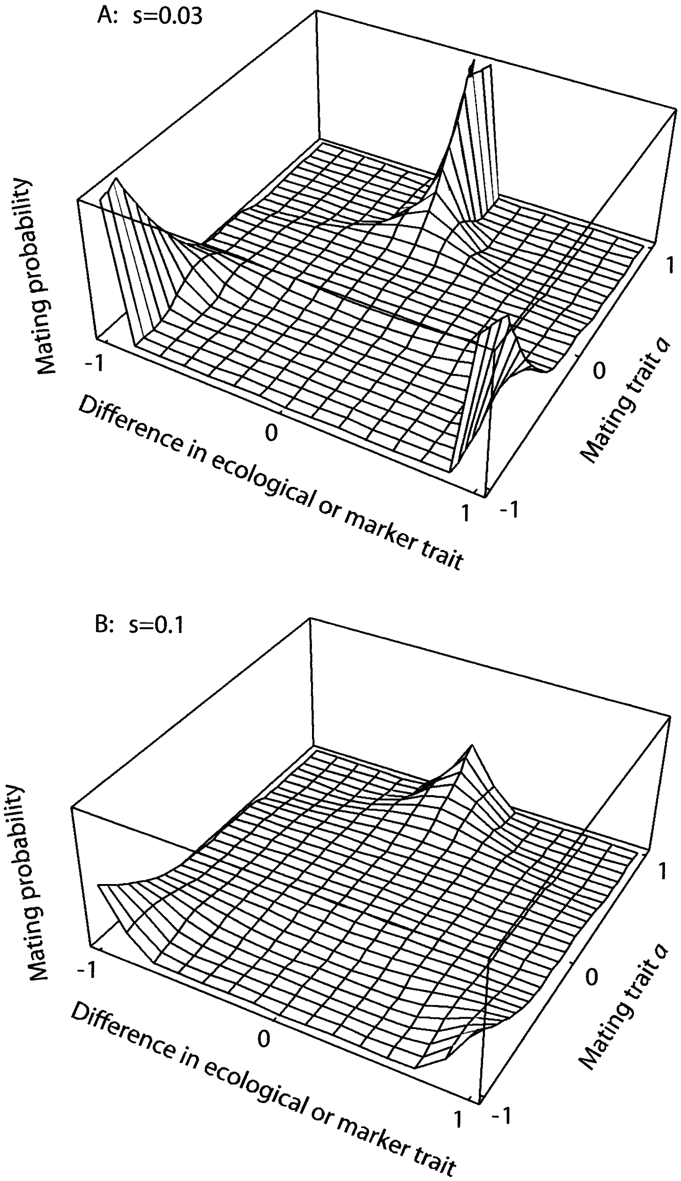

FIG. 3. The probability that a female mates with a male is given by the mating function, equation (12), and depends on the difference in their ecological (or marker) phenotypes, and on the female's degree of assortative mating, *a*. In addition, the parameter *s* in equation (12) determines the shape of the mate choice function. Lower values of *s* steepen the surface and correspond to a higher genetic potential for assortative mating (A), whereas higher values of *s* flatten the surface and correspond to a lower genetic potential for assortative mating (B).

on the extent of phenotypic divergence between the sexes, and the latter determines how fast mating becomes strongly assortative when the mating trait changes from 0 to 1 or -1. We investigated the relative robustness of each evolutionary outcome by factorially varying each parameter: (N_{shared}/N_{total} = 0, 0.1, 0.2, . . . , 1.0; s = 0.02, 0.025, 0.03, . . . , 0.1). This was done for a fixed ecological scenario. Our extensive numerical simulations showed that all the results reported below hold qualitatively for any ecological scenario favoring divergence. For each combination of the two parameters N_{shared}/N_{total} and s we ran 10 replicate numerical simulations. All simulations started with populations that were phenotypically variable with a mean of zero (equal probability for each allele at all ecological and mating loci) for the evolving characters and were run for 2000 generations.

Three main observations emerge from these simulations. First, speciation is inhibited in populations with a high capacity for sexual dimorphism (Fig. 5A). Second, dimorphism is inhibited in populations with a high capacity for assortative mating (Fig. 5B). Third, contrary to our initial expectations it is possible to simultaneously achieve dimorphism and speciation (Fig. 5C). We discuss each of these conclusions in turn.

It is not surprising that speciation was most common in the area of parameter space with the highest potential assortative mating ($s < 0.04$, Fig. 5A). However, the frequency of speciation declines as the population's potential for sexual dimorphism increases (lower values of N_{shared}/N_{total}, Fig. 5A), even when s is very small. This inhibition could reflect either a direct conflict between assortative mating and dimorphism or an indirect effect mediated via the fitness function. As noted above, dimorphism eliminates disruptive selection (Fig. 1E), so if dimorphism is faster, it may remove the impetus for speciation.

Additional simulations confirmed that dimorphism tends to evolve more quickly than speciation. We ran 50 replicate simulations for each of three genetic systems, noting the time to dimorphism or speciation in each run. When dimorphism was the only possible result ($N_{shared}/N_{total} = 0$, $s = 10$), it evolved faster than the mean time to speciation when speciation was the only possibility ($N_{shared}/N_{total} = 1$, $s = 0.05$; cf. Fig. 6A and 6B). Similarly, when the two processes were allowed to compete within a single simulation ($N_{shared}/N_{total} = 0.4$, $s = 0.05$), the mean time to dimorphism was much shorter than the time to speciation (cf. Fig. 6C and 6D). This difference reflects the fact that disruptive selection can act directly on male and female ecological traits, but only indirectly on the level of assortative mating.

Although the potential for dimorphism reduces the probability of speciation, the reverse is also true. When the potential for assortative mating was weak ($s > 0.1$), dimorphism evolved for all values of N_{shared}/N_{total} except $N_{shared}/N_{total} = 1$ (Fig. 5B), where dimorphism is impossible. However, as the potential for assortative mating increased (lower values of the mating parameter s), dimorphism became less common, restricted to situations where the sexes were largely independent ($N_{shared}/N_{total} < 0.5$).

Speciation can inhibit sexual dimorphism in several ways. First, because both processes rely on stochastic effects, speciation occasionally occurs before, and thus preempts, sexual dimorphism (16 of 50 simulations for Fig. 6C, D). Second, there is a fundamental antagonism between positive assortative mating and a sexual dimorphism: females cannot simultaneously prefer mates that are most like themselves ecologically and still maintain an ecological sexual dimorphism. If some degree of positive assortative mating (but not full speciation) evolves before a sexual dimorphism, this will limit the degree to which the sexes may partition resources, because ecologically divergent males will be eliminated by sexual selection.

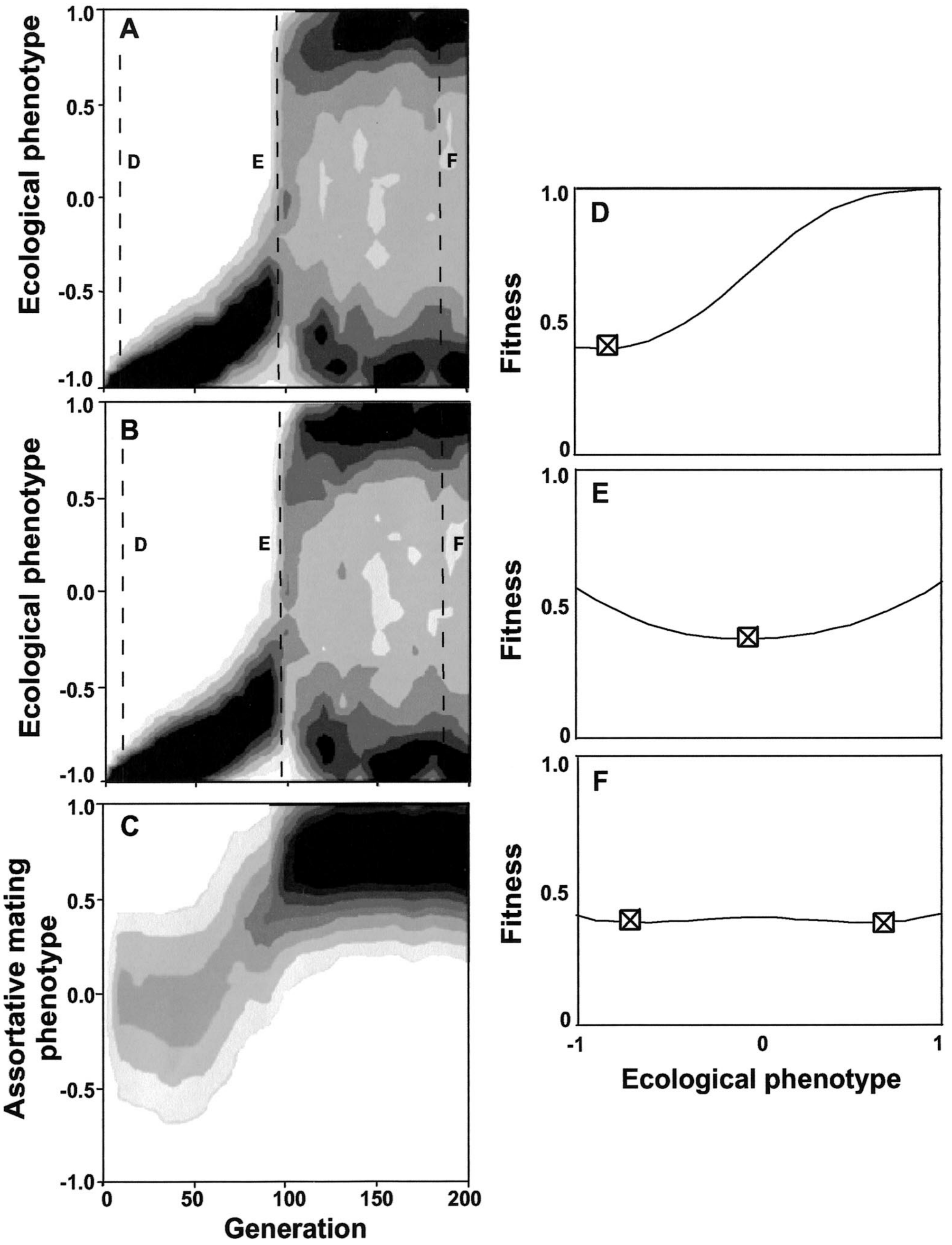

FIG. 4. Simulation of speciation in response to resource competition. Male (A) and female (B) phenotype distributions initially evolve toward the mode of the resource distribution, after which the level of assortative mating increases (C) and branching occurs in both sexes, indicating speciation. Darker shading indicates a greater number of individuals of a given phenotype at a given time. Fitness functions are shown for generation 10 (D), 100 (E), and 190 (F). Dashed vertical lines indicate the points in time corresponding to each fitness function. The mean phenotypes for males (□) and females (×) are marked on the fitness functions. Both sexes were initially monomorphic for the ecological trait $z = -1.0$ and polymorphic for assortativeness with mean $a = 0$. Parameter values were: $N_{shared}/N_{total} = 1.0$, $s = 0.05$, $K_0 = 5000$, $\sigma_k = 1.0$, $\sigma_c = 0.25$, $r = 5$, $\mu = 0.001$, $N_{total} = 10$, $N_{assort} = 5$.

The third limit to sexual dimorphism depends heavily on N_{shared}/N_{total}. If the maximum distance between the sexes is genetically constrained to be less than the distance between the two stable phenotypic optima, equation (5), sexual dimorphism will be insufficient to eliminate the disruptive selection. Dimorphism might then evolve temporarily (because it is faster than speciation), but be replaced by speciation, which can more effectively equalize fitness across phenotypes.

An example of this sequential sexual dimorphism and speciation is shown in Figure 7. A population initially centered on the resource optimum ($z = 0$) is subject to disruptive selection (Fig. 7D) in period **a** (generations 0 to 290), after which a slight dimorphism evolves (period **b**). The degree of dimorphism is limited by genetic constraints ($N_{shared}/N_{total} = 0.7$). Because phenotypic divergence is limited, the fitness function does not flatten greatly and the total population size

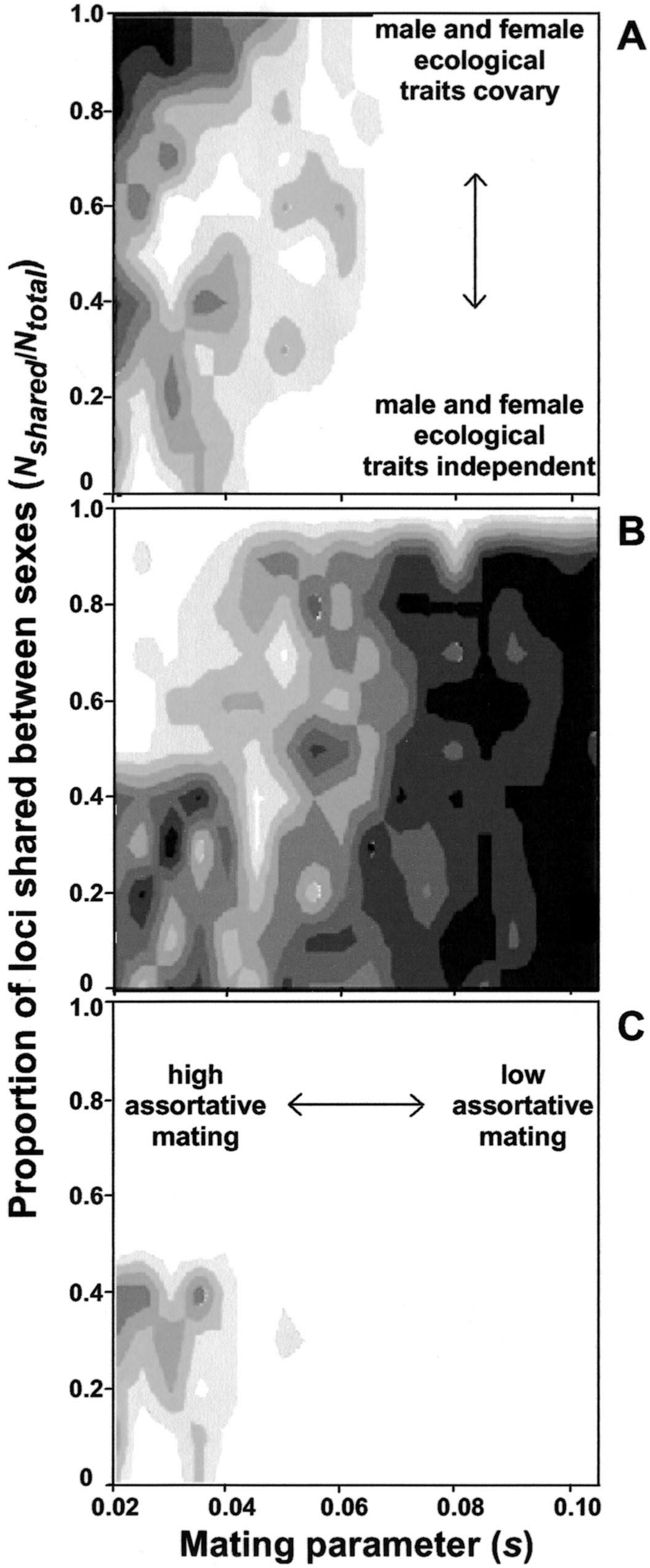

FIG. 5. Shaded contour plots indicate frequency of speciation (A), sexual dimorphism (B), and cases of simultaneous dimorphism and speciation (C) as a function of the potential for assortative mating (s) and the genetic independence of the sexes (N_{shared}/N_{total}). For each combination of parameter values, we ran 10 simulations for 2000 generations. The proportion of runs resulting in a particular outcome is indicated by the shading, ranging from white (0%) to black (100%). Populations were initially polymorphic for both ecological and mating traits with means of zero. $K_0 = 1500$, $\sigma_k = 1.0$, $\sigma_c = 0.25$, $r = 5$, $\mu = 0.001$, $N_{total} = 10$, $N_{assort} = 5$.

remains low, though marginally higher than in period **a** (Fig. 7F). This sexual dimorphism then collapses, returning to a period of intermediate ecological phenotypes, disruptive selection, low population size, but increasing assortative mating (period **c**). When assortative mating is strong enough speciation occurs (period **d**). With the advent of speciation, the phenotype distributions of both sexes bifurcate, assortative mating remains consistently high, disruptive selection ceases (Fig. 7E), and population sizes increase dramatically (Fig. 7F). This last effect is particularly noteworthy because it indicates that the higher phenotypic variance has increased the overall carrying capacity of the population—an effect that was not achieved by the genetically constrained sexual dimorphism.

So far we have shown that speciation and ecological sexual dimorphism are mutually antagonistic. Dimorphism can preempt speciation, whereas assortative mating can restrict or sometimes replace dimorphism. It was therefore surprising to find some simulations that resulted in both sexual dimorphism and speciation (Fig. 5C). In contrast to previous models of adaptive speciation, in this case speciation entails strong negative, rather than positive, assortative mating. The ecological phenotype distributions of both sexes become bimodal, with the two male modes toward the phenotype extremes, and the female modes closer to the interior (Fig. 8). Thus, each group of females is furthest from a different group of males. Due to strong negative assortative mating, each group of females mates with the males ecologically least like them, rejecting the more phenotypically similar males.

Note that the existence of four phenotypic clusters in this scenario is a reflection of the fact the dimorphic equilibrium (5) in the analytical model is an evolutionary branching point: with negative assortative mating and concomitant speciation into two sexually dimorphic species, a finer partitioning of niche space is possible than with sexual dimorphism alone. However, this particular outcome is rare because it requires a finely balanced set of parameters. Assortative mating must be strong enough to maintain two species, while N_{shared}/N_{total} must be small enough to allow dimorphism yet not so small that dimorphism preempts speciation.

Assortative Mating Based on Marker Phenotypes

In this section we investigate to what extent the conflict between dimorphism and speciation carries over to cases in which assortative mating is not based on traits under ecological selection, but instead on ecologically neutral marker traits, such as coloration. For speciation to occur in this situation, a linkage disequilibrium between the marker trait and the ecological trait must develop, so that assortative mating can indirectly latch onto the ecological trait. Classically, recombination between the marker and the ecological trait is expected to severely impede speciation with this type of as-

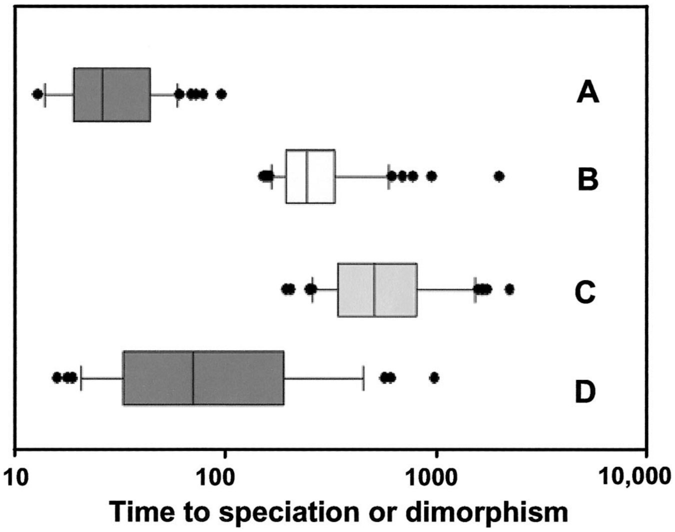

FIG. 6. Box plots of the time to dimorphism (dark boxes) or speciation (light boxes), showing quartiles (boxes), 95% confidence intervals (horizontal lines), and outliers (dots) for 50 replicates of each of three scenarios (A, B, and C/D). (A) Dimorphism only, due to random mating ($s = 10$) and complete independence between male and female ecological phenotypes ($N_{shared}/N_{total} = 0$). (B) Speciation only, due to potentially strong assortative mating ($s = 0.05$) and sexes are not independent ($N_{shared}/N_{total} = 1$). Speciation (B) occurs an order of magnitude more slowly than sexual dimorphism (A), $t_{98} = -7.37$, $P < 0.0001$. When both speciation (C) and dimorphism (D) are possible outcomes of a single simulation ($s = 0.05$, $N_{shared}/N_{total} = 0.4$), dimorphism still occurs more rapidly ($t_{82} = -6.04$, $P < 0.0001$), although 16 of 50 replicate simulations led to speciation first and failed to evolve a sexual dimorphism. Parameter values $K_0 = 1000$, $\sigma_k = 0.75$, $\sigma_c = 0.25$, $r = 5$, $\mu = 0.001$, $N_{total} = 10$, $N_{assort} = 5$, with 5000 generations per simulation.

sortative mating (Felsenstein 1981). However, Dieckmann and Doebeli (1999) have shown that this expectation is in fact unwarranted, and that speciation can easily occur even in this scenario, albeit for slightly more restrictive ecological parameters than in the case where assortative mating is based on the ecological trait.

Following Dieckmann and Doebeli (1999), we incorporate marker-based assortative mating by assuming that there is a third set of N_{marker} diallelic loci determining an ecologically neutral trait that is expressed in both sexes. As before, mating depends on assortability trait a, and may be disassortative, random, or assortative to varying degrees, but now mate choice, equation (12), is based not on similarity in the ecological trait, but in the neutral marker trait. Thus, in Figure 3 the x-axis now represents the difference in the value of the marker trait between two potential mating partners, which varies between $-2N_{marker}$ and $2N_{marker}$. It is assumed that the marker loci freely recombine with all the other loci.

In accordance with the results of Dieckmann and Doebeli (1999), adaptive speciation occurs in our models with marker-based assortative mating if $N_{shared} = N_{total}$, and if the parameter s scaling the assortative mating function is low enough.. However, if $N_{shared} < N_{total}$, a new type of evolutionary dynamics can be seen, during which the ancestral population splits into two sexually dimorphic descendant species with positive assortative mating (Fig. 9). Two clusters of individuals exist at opposite ends of the marker trait axis, representing two reproductively isolated species with high assortativeness. Within each species male and female ecological traits are dimorphic. Thus, assortative mating based on a marker trait can alleviate the antagonism between sexual dimorphism and positive assortative mating: as long as the sexes have the same marker trait (and hence mate with each other), they form a species even if they are ecologically differentiated. Note again that sexual dimorphism together with speciation due to marker-based assortative mating allows for finer niche partitioning than either sexual dimorphism or speciation would if they occurred alone.

Although mating based on a marker trait alleviates one of the sources of conflict between dimorphism and speciation, the region of parameter space in which speciation occurs is more limited than seen for mating based on ecological traits. This is illustrated in Figure 10, for which we used the same numerical procedure as for Figure 5, except that single runs were continued for 5000 generations (to allow for the potentially slow process of linkage disequilibrium build-up). Even for low values of the scaling parameter s, speciation will not occur when N_{shared}/N_{total} is much less than one. Instead it appears that sexual dimorphism is facilitated, as it is no longer countered by any positive assortative mating. Dieckmann and Doebeli (1999) noted that the time to speciation was much longer when assortative mating was based on a marker trait than when based on the ecological traits, a result also seen in our simulations. Consequently sexual dimorphism will almost always occur before speciation in this case and eliminate or reduce the disruptive selection needed to drive speciation. The exception is when N_{shared}/N_{total} restricts the degree of dimorphism, so that the sexes cannot diverge all the way to the ecologically determined optima. Disruptive selection is then maintained until speciation occurs in addition to the dimorphism, more effectively equalizing competition across phenotypes.

DISCUSSION

Although it is now widely accepted that adaptive speciation is both theoretically plausible (Turelli et al. 2001; Dieckmann et al. 2003) and has been established empirically in at least a few cases (Schliewen et al 1994, 2001; Berlocher and Feder 2002; Dres and Mallet 2002), its generality remains contentious (Barraclough and Vogler 2000; Coyne and Price 2000). We therefore feel that it is useful to move theoretical work away from showing that adaptive speciation is possible to more directly considering the conditions under which it is more and less likely. For example, the model by Dieckmann and Doebeli (1999) highlighted the importance of within-population niche variation ($\sigma_c < \sigma_k$) in generating frequency-dependent disruptive selection. This facilitates empirical study because information about the degree and frequency of within-population variation (Bolnick et al. 2002, 2003) then tells us about the range of species likely to experience the ecologically mediated disruptive selection necessary for adaptive speciation.

In this paper we have illustrated one way in which theory can shift from considering feasibility of adaptive speciation toward considering its expected frequency of occurrence. Previous studies lead one to believe that speciation is likely

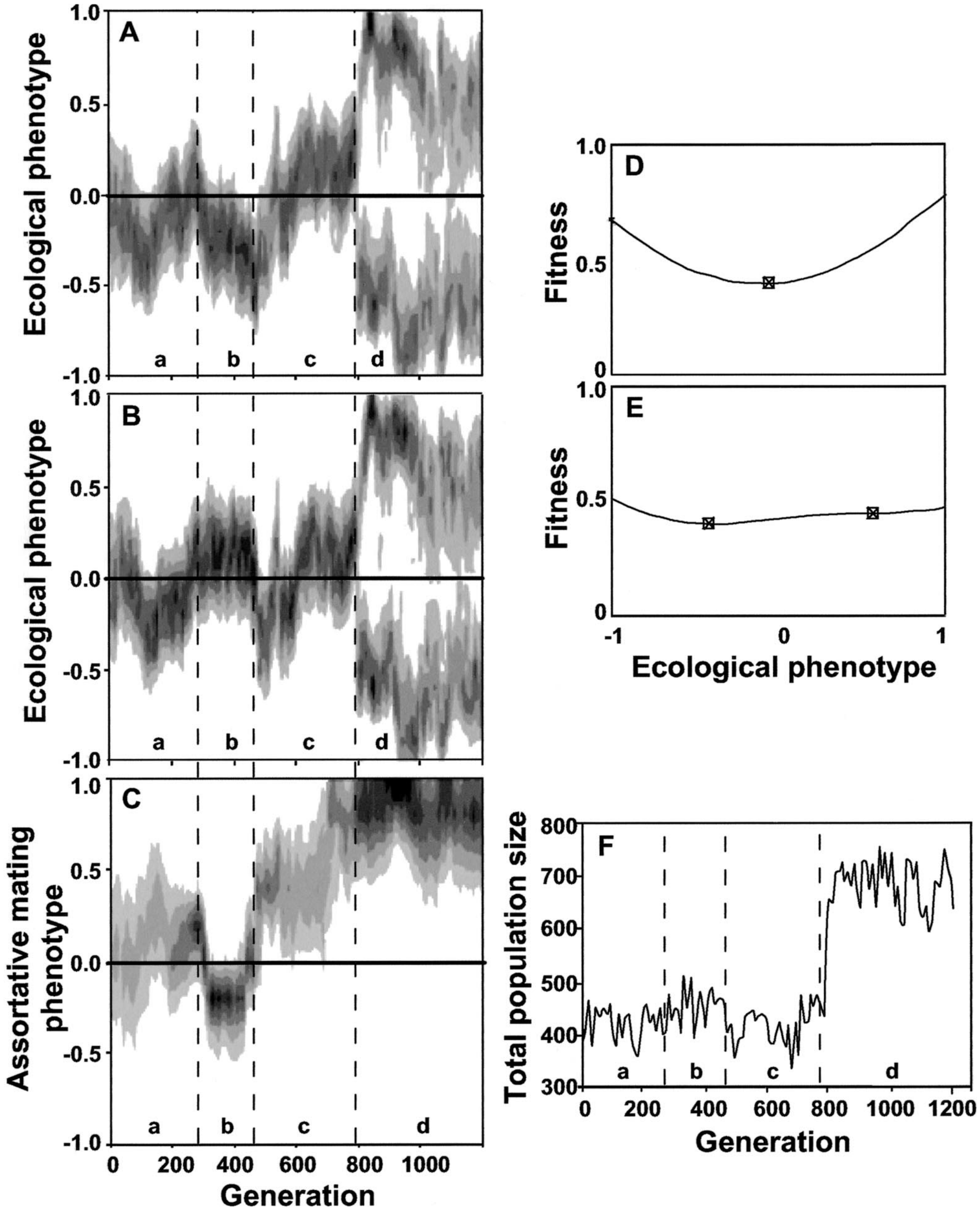

FIG. 7. A simulation resulting in successive sexual dimorphism and speciation. The male (A) and female (B) ecological phenotype distributions, and assortative mating distributions (C) are divided into four temporal regions, **a, b, c,** and **d.** These regions correspond to **a**: a period of disruptive selection (D), **b**: a period of weak sexual dimorphism. Note that the male distribution (A) is below the dark horizontal reference line, whereas the female distribution (B) is above the reference line. Assortative mating becomes negative (C), because females must choose males with phenotype traits different from their own. The dimorphism is weak due to genetic constraints. **c**: sexual dimorphism is lost, **d**: speciation, indicated by bifurcation of both sexes ecological traits (A, B), and high assortative mating (C). Following speciation (in period **d**), the fitness function is flattened (E), and population sizes increase due to reduced intraspecific competition (F). Populations were initially polymorphic for both ecological and mating traits with means of zero. $N_{shared}/N_{total} = 0.7$, $s = 0.05$, $K_0 = 1000$, $\sigma_k = 0.75$, $\sigma_c = 0.25$, $r = 5$, $\mu = 0.001$, $N_{total} = 10$, $N_{assort} = 5$.

whenever the necessary ecological conditions are fulfilled (e.g., when $\sigma_c < \sigma_k$). In contrast, we have taken the view here that once disruptive selection has emerged from the ecological interactions, it can have more than one outcome. Specifically, we have studied the relative likelihood with which disruptive selection leads to sexual dimorphism or adaptive speciation. In doing so we have found that the genetic basis of male and female ecological traits greatly affects whether a population will undergo speciation even when the ecological dynamics are sufficient. Under the assumptions of our model, a population with a large capacity for sexual dimorphism is less likely to undergo speciation.

Our simulations suggest two main reasons why sexual dimorphism and speciation are mutually antagonistic outcomes of frequency-dependent disruptive selection. First, there is a fundamental conflict between sexual dimorphism and a fe-

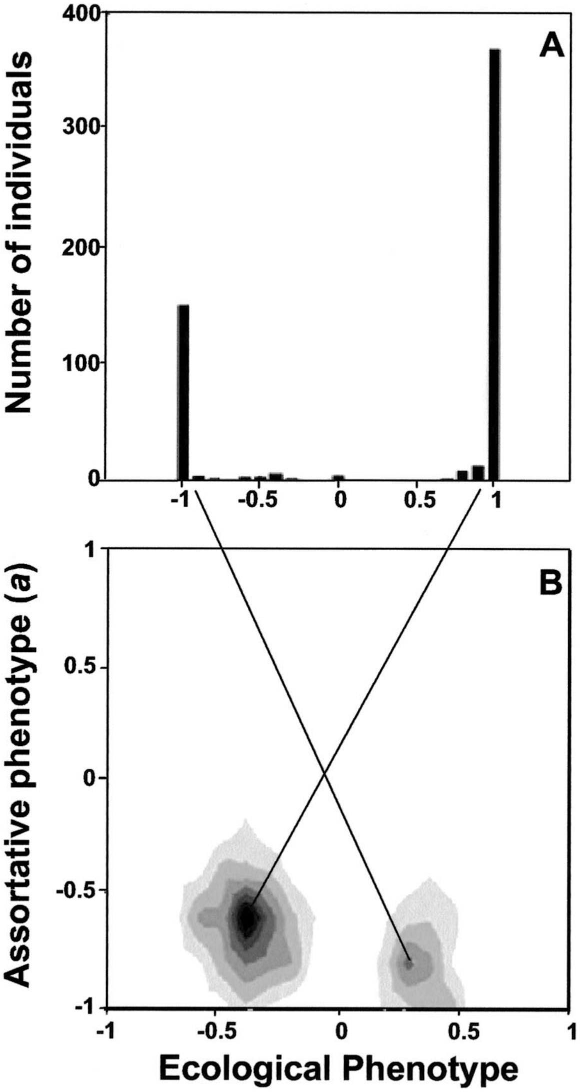

FIG. 8. Simultaneous speciation and sexual dimorphism in which assortative mating is based on ecological traits. A histogram of male ecological traits (A) shows two ecological groups at $z = -1$ and $z = 1$. Females (B) express both ecological and assortative mating traits, so their phenotype distribution is shown as a density plot (darker shading indicates more individuals). Females have split into two ecological groups, each with strong negative assortative mating. Straight lines connect conspecific males and females, whereas phenotypically similar males and females fail to mate due to negative assortative mating. $N_{shared}/N_{total} = 0.2$, $s = 0.025$, $K_0 = 1500$, $\sigma_k = 1.0$, $\sigma_c = 0.25$, $r = 5$, $\mu = 0.001$, $N_{total} = 10$, $N_{assort} = 5$.

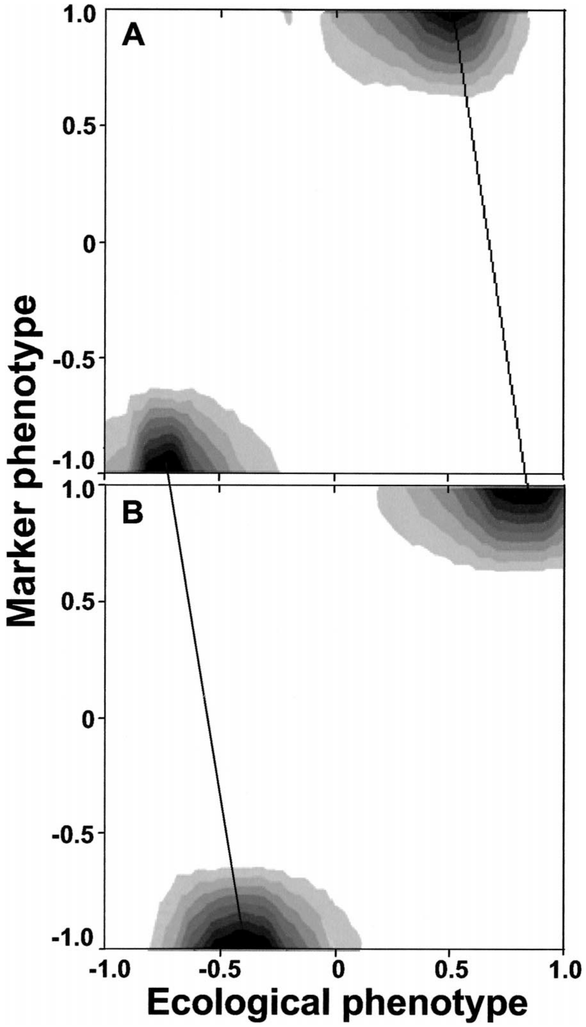

FIG. 9. Simultaneous speciation and sexual dimorphism in which assortative mating is based on a neutral marker trait. Joint ecological and mating marker phenotype distributions are shown for males (A) and females (B). Due to strong positive assortative mating, males and females with high marker values (connected by a line) constitute one species, and males and females with low marker values (connected by a line) constitute a second species. Each of these species is mildly sexually dimorphic. $N_{shared}/N_{total} = 0.4$, $s = 0.05$, $K_0 = 1000$, $\sigma_k = 1.0$, $\sigma_c = 0.25$, $r = 5$, $\mu = 0.001$, $N_{total} = 10$, $N_{assort} = 5$, $N_{marker} = 5$.

male's ability to choose a mate phenotypically like herself. This conflict can be resolved if females' mate preferences are based on independent marker cues (e.g., color), rather than ecologically important dimorphic traits. Alternatively, it is possible for speciation and dimorphism to occur simultaneously when the speciation is based on negative assortative mating. Such dual diversification is consistent with our analytical and simulation observation that each two-species or dimorphic equilibrium is itself a slight fitness minimum (see Figs. 1E, 4F) and hence may lead to still finer niche partitioning through successive speciation events. Our genetic simulations are too coarse to observe finer partitioning than the combined speciation and dimorphism.

The second reason for the antagonism is indirect, mediated by changes in the shape of the fitness function. Both ecological sexual dimorphism and adaptive speciation reduce or eliminate disruptive selection by equalizing the effect of competition across phenotypes. Consequently, the outcome that occurs first will eliminate the selective force that is needed to drive the alternative evolutionary outcome. Our simulations consistently found that sexual dimorphism evolved faster than speciation, presumably because selection acts directly on the ecological traits but only indirectly on mating behavior. Only if dimorphism is insufficient to eliminate disruptive selection, such as when the sexes cannot diverge enough to reach the ecologically determined optima due to genetic con-

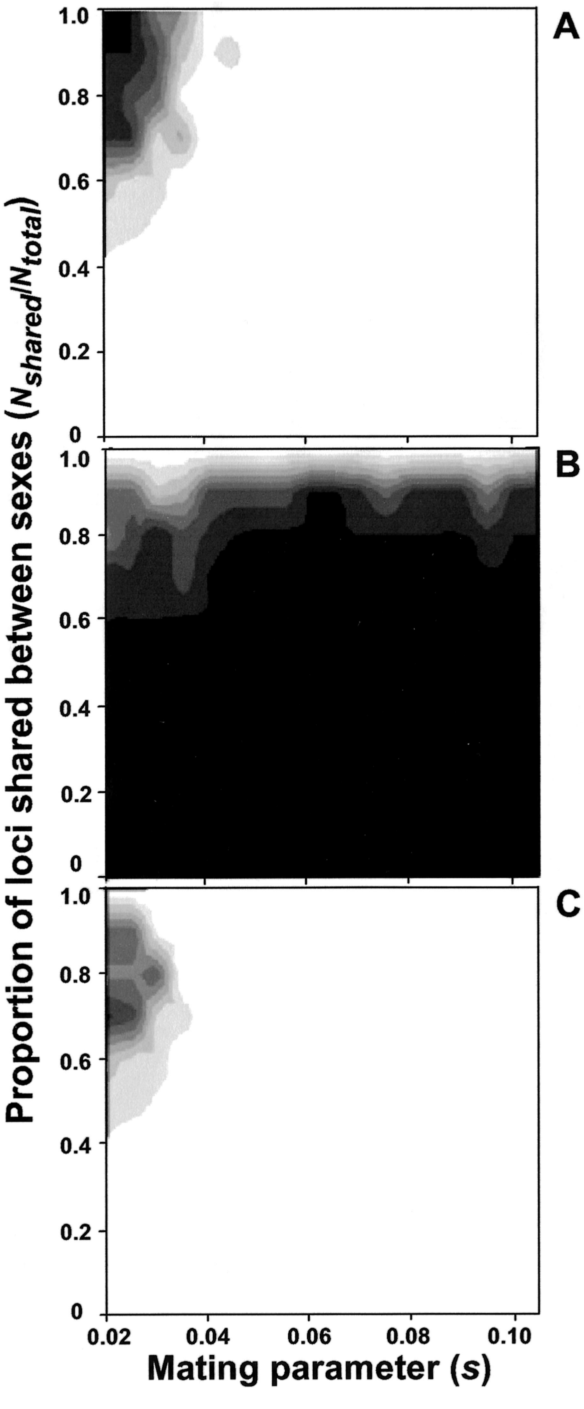

FIG. 10. Shaded contour plots indicate frequency of speciation (A), sexual dimorphism (B), and simultaneous speciation and dimorphism (C), when mate choice is based on an independent marker trait such as color, as a function of the potential for assortative mating (s), and the genetic independence of the sexes (N_{shared}/N_{total}). For each combination of parameter values, we ran 10 simulations for 5000 generations. The proportion of runs resulting in a particular outcome is indicated by the shading, ranging from white (0%) to black (100%). Populations were initially polymorphic for both ecological and mating traits with means of zero. $K_0 = 1500$, $\sigma_k = 1.0$, $\sigma_c = 0.25$, $r = 5$, $\mu = 0.001$, $N_{total} = 10$, $N_{assort} = 5$, $N_{marker} = 5$.

straints, can speciation follow, either replacing or complementing the dimorphism.

As with any theoretical model, it is important to bear in mind the underlying assumptions and their consequences. In the context of this model, some noteworthy assumptions include the choice of particular ecological equations, the symmetrical resource distributions and competition functions, constant individual niche widths (σ_c), the absence of resource population dynamics or evolution, the scheme for simulating genetic independence of male and female traits, and the lack of any cost of assortative mating for females.

Our results are robust to changes in many of these assumptions. For example, we chose one of a number of alternative ways to model genetic divergence between sexes. Alternative schemes could include loci that have opposite sex-dependent effects, specifically control dimorphism, or arrest growth earlier in one sex than another. While our choice appears to be biologically reasonable based on QTL studies of dimorphic traits (Mogil et al. 1997; Nuzhdin et al. 1997; Agulnik et al. 1998; Gurganus et al. 1999; Ramos et al. 1999; Kopp et al. 2003), the particular choice of genetic model is not likely to be critical. Slatkin's (1984) model of ecological sexual dimorphism concluded that a sexual dimorphism is possible whenever the genetic correlation between sexes is less than one, so different approaches to incorporate explicit genetics should yield equivalent results. Similarly, adding a (small) cost to assortative mating does not qualitatively change our results (D. I. Bolnick and M. Doebeli, unpubl. simulations). Also, asymmetrical competition has been shown to induce evolutionary branching (Kisdi and Geritz 1999; Doebeli and Dieckmann 2000), and hence the ecological preconditions for sexual dimorphism as well. However, with asymmetrical competition the evolutionary branching point is not located at the resource maximum K_0, and the emerging phenotypic clusters have different population sizes, which may have quantitative effects on the evolution of sexual dimorphism because one sex will tend to be rarer than the other.

Other assumptions may prove to be more critical to our results. For instance, if individual niche widths (σ_c) were allowed to evolve, niche expansion might occur most quickly through increased within-phenotype niche width rather than increased between-phenotype variation (Taper and Case 1985). However, it is empirically quite reasonable to assume an upper limit on σ_c reflecting functional or cognitive trade-offs that impose limits on individual niche breadth. Such trade-offs are known to maintain individual specialization in a wide range of taxa (Bolnick et al. 2003). Our results might also be sensitive to removing our assumption that the resource distribution neither evolves nor shows a numerical response to predation. The addition of interspecific competitors would also greatly change the dynamics, as rare phenotypes that would otherwise have been favored by disruptive selection are subject to competitive exclusion by other species. Finally, we have assumed that the ecological traits whose evolution we study affect viability, but not fertility, of individuals. This assumption is in line with many previous theoretical studies, and it ultimately originates in the observation that the fertility r has no qualitative effect on the dynamics of two-species Lotka-Volterra competition models.

Another question is whether we chose an appropriate range of parameter space to explore in our simulations. The relative likelihood of dimorphism or speciation depends on the genetic independence of ecological traits in males and females and on the potential for assortative mating. Within the parameter space we examined, dimorphism tends to be more likely than speciation. Speciation is restricted to cases where the male-female covariance is high, and assortative mating is potentially very strong. Without data on what constitutes empirically realistic values of these parameters, it is impossible to tell how the parameter space used in this paper relates to natural systems. The maximum slope of assortative mating may never reach that required for speciation, or the between-sex correlation may rarely be so low as to permit dimorphism (Merilä et al. 1998). Such data is, in principle, possible to collect, through quantitative genetic studies of dimorphic and nondimorphic traits and behavioral studies of mate choice.

The high frequency of sexual dimorphism in nature would seem to argue that male-female covariance is potentially quite low. Based on our models, we would thus expect to find cases of resource partitioning between the sexes more often than adaptive speciation. However, both phenomena are difficult to demonstrate empirically. While resource partitioning between the sexes has been observed (e.g., Feduccia and Slaughter 1973; Bowers and Smith 1979; Ebenman and Nilsson 1982; Shine 1989, 1991; Dayan and Simberloff 1994; Voight 1995; Gvozdik and Boukal 1998; Temeles et al. 2000; Pasinelli 2002; Pearson et al. 2002; Shine et al. 2002), such dimorphisms may be the result of sexual selection instead of ecological character displacement. Intra- or intersexual selection on male or female body size could lead indirectly to resource partitioning if only because the relatively larger sex can consume larger prey (Shine 1989, 1991; Katsikaros and Shine 1997). Sex-specific energetic or nutritive requirements might lead to differential resource use (Slatkin 1984), as in female moose (*Alces alces*), which consume energetically suboptimal aquatic vegetation, avoided by males, to acquire sodium needed for lactation (Belovsky 1978). Differences in parental care responsibilities might also lead to between-sex differences in foraging (Williamson 1971). Of course, sexual selection and ecological interactions are not mutually exclusive and might interact synergistically during the evolution of sexual dimorphism.

An ecological cause of sexual dimorphism is more credible when the dimorphism occurs in a phenotypic trait that is thought to be free from sexual selection or in a direction opposite of that expected by sexual selection (Shine 1989, 1991). For example, many populations of the three-spine stickleback (*Gasterosteus aculeatus*), have sexually dimorphic gill rakers, internal structures that are tightly tied to resource use (Reimchen and Nosil 2001). The degree of gill raker dimorphism varies widely among lake populations and is subject to disruptive selection due to resource competition (D. I. Bolnick, unpubl. ms.). It is intriguing that sticklebacks have speciated in some contexts, but evolved substantial sexual dimorphisms in other populations. The degree of sexual dimorphism can also show a pattern of character release, increasing when a population is freed from pressure by interspecific competitors (Lister 1976; Schoener 1977; Ebenman and Nilsson 1982; Mysterud 2000; but see Stamps et al. 1997). Finally, in a few cases there has been direct evidence for ecological causation of a sexual dimorphism (e.g., Temeles et al. 2000).

Although this paper has emphasized the potential conflict between sexual dimorphism and adaptive speciation, it is important to note that stable fitness minima might lead to any of a number of forms of evolutionary diversification. Ontogenetic niche shifts (Polis 1984), discrete polymorphisms (Smith and Skulason 1996), and character release (Van Valen 1965) all affect the level of competition experienced by any given phenotype, and thus may change the shape of the fitness function and interfere with speciation. The model presented in this paper is only a starting point, using sexual dimorphism and adaptive speciation out of a much larger toolkit of diversification mechanisms. Each outcome will have its own genetic requirements, and the interaction of these many possible outcomes is likely to reduce the probability of any one.

ACKNOWLEDGMENTS

We thank A. Blachford, J. Brown, B. Fitzpatrick, C. Hauert, C. McBride, L. Moyle, and M. Turelli for their comments on this manuscript. This work was done while DIB was supported by an Achievement Rewards for College Scientists Foundation Fellowship, the Center for Population Biology, and a University of California, Davis, Summer Research Fellowship. MD was supported by the National Science and Engineering Research Council (NSERC), Canada.

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Corresponding Editor: D. Houle

APPENDIX

Here we present the adaptive dynamics of the deterministic model of sexual dimorphism. We begin by formulating the ecological dynamics of male and female population densities in a population in which males and females are each monomorphic for ecological trait values z_m and z_f. We first modify the Beverton-Holt model, equation (3), to describe the effect of competition on males and on females. Let $n_{z_m}(t)$ and $n_{z_f}(t)$ be the population sizes of males and females at the beginning of generation t. Because we assume that the effects of competition are independent of the sex of the competing individuals, the males experience an effective total population size of $n_{z_m}(t) + c(z_m, z_f)\, n_{z_f}(t)$, where $c(z_m, z_f)$ is the strength of competition between a z_m individual and z_f individual, equation (2). Therefore, the population size of the males after death due to competition, but before reproduction, in generation t is given by

$$\tilde{n}_{z_m}(t) = \frac{n_{z_m}(t)}{1 + \dfrac{r-1}{K(z_m)}[n_{z_m}(t) + c(z_m, z_f)n_{z_f}(t)]}. \tag{A1}$$

Here we assume that the maximal reproductive output r, which occurs as a parameter in this equation, is independent of the trait value z and that the carrying capacity $K(z_m)$ is given by equation (1).

Similarly, the density of females after competition, but before reproduction, in generation t is given by

$$\tilde{n}_{z_f}(t) = \frac{n_{z_f}(t)}{1 + \dfrac{r-1}{K(z_f)}[n_{z_f}(t) + c(z_f, z_m)n_{z_m}(t)]}. \tag{A2}$$

To determine male and female densities at the beginning of generation $t + 1$, we have to specify how many matings result from the pre-reproduction densities of males and females, $\tilde{n}_{z_m}(t)$ and $\tilde{n}_{z_f}(t)$. That is, we have to specify the mating function $B[\tilde{n}_{z_m}(t), \tilde{n}_{z_f}(t)]$, and there are a number of ways in which this can be done. The simplest assumption is that of female dominance, in which the number of matings is simply equal to the number of females:

$$B[\tilde{n}_{z_m}(t), \tilde{n}_{z_f}(t)] = \tilde{n}_{z_f}(t). \tag{A3}$$

Other forms of the mating function have been used in the literature, for example, the harmonic mean of males and females (Caswell and Weeks 1986). However, for simplicity we will assume female dominance here, equation (A3).

To finally determine the number of male and female offspring, we assume that each mating gives rise to r offspring, half of which are male and half of which are female. That is, we assume a 1:1 sex ratio among the offspring. For the deterministic model, we also assume that there is no genetic covariance between male and female traits. Therefore, a female offspring of a mating between a z_m father and a z_f mother has trait value z_f, while a male offspring of such a mating has trait value z_m. With a 1:1 sex ratio, and for a general mating function $B[\tilde{n}_{z_m}(t), \tilde{n}_{z_f}(t)]$, the densities of males with trait value z_m and of females with trait value z_f at the start of the next generation are then

$$n_{z_m}(t+1) = n_{z_f}(t+1) = \frac{r}{2}B[\tilde{n}_{z_m}(t), \tilde{n}_{z_f}(t)]. \tag{A4}$$

With female mating dominance as assumed here, we end up with the following difference equation describing the ecological dynamics of males and females in a population in which each sex is monomorphic:

$$\begin{aligned} n_{z_m}(t+1) &= n_{z_f}(t+1) \\ &= \frac{r}{2}\,\frac{n_{z_f}(t)}{1 + \dfrac{r-1}{K(z_f)}[n_{z_f}(t) + c(z_f, z_m)n_{z_m}(t)]}. \end{aligned} \tag{A5}$$

Note that we necessarily have $n_{z_m}(t) = n_{z_f}(t)$ at all times t due to the 1:1 sex ratio assumptions, but $\tilde{n}_{z_m}(t)$ and $\tilde{n}_{z_f}(t)$ are not necessarily the same.

It is easy to see that these dynamics yield a stable equilibrium of male and female population sizes

$$n^* = n^*_{z_m} = n^*_{z_f} = \frac{r-2}{2}\,\frac{K(z_f)}{r-1}\,\frac{1}{1 + c(z_f, z_m)}. \tag{A6}$$

We now consider the fate of rare mutants in a resident population (z_m, z_f). If a mutant is rare, the effective density that it experiences is entirely determined by the resident equilibrium densities $n^*_{z_m}$ and $n^*_{z_f}$ given by equation (A6). Considering first female mutants z'_f, it follows from the assumption of female mating dominance that the ecological dynamics of the density of female mutants, $n_{z'_f}(t)$, is given by

$$\begin{aligned} n_{z'_f}(t+1) &= \frac{r}{2}\,\frac{n_{z'_f}(t)}{1 + \dfrac{r-1}{K(z'_f)}[c(z'_f, z_f)n^*_{z_f} + c(z'_f, z_m)n^*_{z_m}]} \\ &= \frac{r}{2}\,\frac{n_{z'_f}(t)}{1 + \dfrac{r-1}{K(z'_f)}n^*[c(z'_f, z_f) + c(z'_f, z_m)]} \end{aligned} \tag{A7}$$

In particular, the per capita number of offspring, and hence the long-term growth rate of the rare mutant z'_f in the resident population (z_m, z_f), which is denoted by $w_f(z'_f, z_m, z_f)$, is given by

$$w_f(z'_f, z_m, z_f) = \frac{r}{2}\,\frac{1}{1 + \dfrac{r-1}{K(z'_f)}n^*[c(z'_f, z_f) + c(z'_f, z_m)]}. \tag{A8}$$

Because we assume female dominance in mating, the per capita growth rate $w_m(z'_m, z_m, z_f)$ of rare male mutants z'_m in the resident (z_m, z_f) is determined by the dynamics of the frequency of mutant males during competition in a single generation. Therefore, $w_m(z'_m, z_m, z_f)$ is simply given by the ratio of the probabilities of survival for the resident and the mutant males:

$$w_m(z'_m, z_m, z_f) = \frac{1 + \dfrac{r-1}{K(z_m)}n^*[1 + c(z_m, z_f)]}{1 + \dfrac{r-1}{K(z'_m)}n^*[c(z'_m, z_m) + c(z'_m, z_f)]}. \tag{A9}$$

From the growth rates of rare mutants, w_m and w_f, given by equations (A8) and (A9), one obtains the selection gradients $g_m(z_m, z_f)$ and $g_f(z_m, z_f)$ for male and female traits in a resident population (z_m, z_f) that is at its ecological equilibrium as the partial derivatives of w_m and w_f with respect to the mutant trait values z'_m and z'_f and evaluated at the current resident values z_m and z_f. Thus,

$$g_m(z_m, z_f) = \left.\frac{\partial w_m}{dz'_m}\right|_{z'_m = z_m} \quad \text{and} \tag{A10}$$

$$g_f(z_m, z_f) = \left.\frac{\partial w_f}{dz'_f}\right|_{z'_f = z_f}. \tag{A11}$$

The selection gradients g_m and g_f then determine the rate of evolutionary change in males and females as

$$\frac{dz_m}{dt} = M_m g_m(z_m, z_f) \quad \text{and} \tag{A12}$$

$$\frac{dz_f}{dt} = M_f g_f(z_m, z_f). \tag{A13}$$

Here the quantities M_m and M_f describe the mutational process in male and female traits, respectively. In our situation it is reasonable to assume a symmetric scenario with $M_m = M_f$, in which case these quantities only determine the speed of evolutionary change but do not influence the location or stability of the equilibrium specified by equations (A12) and (A13). Therefore, we make the simplifying assumption that $M_m = M_f = 1$ in our analysis.

Equilibrium points of the adaptive dynamics given by equations (A12) and (A13) are points z_m^*, z_f^* in phenotype space at which the gradients g_m and g_f vanish simultaneously. To derive this equilibrium we assume that the optimal trait value z_0 in the carrying capacity function $K(z)$, equation (1), is located at $z_0 = 0$, and use the definition of $c(z_m, z_f)$ given in equation (2), the value of the resident equilibrium population size n^* given by equation (A6), as well as the derivation of the selection gradients in equations (A8–A11). It is then straightforward to see that the point $(z_m^*, z_f^*) = (0,0)$ is an equilibrium for the adaptive dynamics, that is,

$$g_m(0, 0) = g_f(0, 0) = 0. \tag{A14}$$

To determine the dynamic stability of this equilibrium, we have to consider the Jacobian matrix of the dynamical system given by (A12) and (A13), evaluated at the equilibrium, for example, the matrix

$$J(0, 0) = \begin{bmatrix} \frac{\partial g_m}{\partial z_m}(0, 0) & \frac{\partial g_m}{\partial z_f}(0, 0) \\ \frac{\partial g_f}{\partial z_m}(0, 0) & \frac{\partial g_f}{\partial z_f}(0, 0) \end{bmatrix}. \tag{A15}$$

The equilibrium (0, 0) is locally stable, and hence an evolutionary attractor, if and only if both eigenvalues of $J(0, 0)$ have negative real part, which is the case if and only if the trace of $J(0, 0)$ is negative and the determinant of $J(0, 0)$ is positive. By using a computer algebra system for symbolic calculations, one can easily see that these conditions are satisfied if and only if $\sigma_c > \sigma_k$, where σ_k and σ_c are the parameters determining the width of the carrying capacity function, equation (1), and the strength of frequency dependence in the competition function, equation (2), respectively.

Thus, the equilibrium (0, 0) is unstable if $\sigma_c < \sigma_k$, and one can similarly show that in this case the adaptive dynamics given by (A12) and (A13) has two locally stable equilibria (z_m^*, z_f^*) given by

$$z_m^* = -z_f^* = \pm\sigma_c\sqrt{\frac{1}{2}\log\left(\frac{2\sigma_k^2}{\sigma_c^2} - 1\right)}, \tag{A16}$$

both of which represent a sexual dimorphism.