population into two. Fortunately, the hypergeometic phenotypic model[7–9] is applicable under the conditions required for sympatric speciation[10]. For a quantitative trait with the possible phenotypes 0, 1, …, n, this model provides, as a sum of certain hypergeometric functions, the probability $R(i, j, k)$ of two parents with phenotypes i and j producing an offspring with phenotype k (refs 8–10).

Implementation of the model. We considered haploid individuals with the phenotype in their m-th trait determined by the number of alleles 1 at the n_m corresponding loci, each with alleles 0 and 1. The dynamics with $n_m/2$ diploid loci are very similar[10]. The generations were discrete and the life cycle consisted of selection, mating and reproduction.

In the two-trait model, the frequency of (i_1, i_2) individuals of the i_1-th phenotype in trait 1 and i_2-th phenotype in trait 2 before selection was $p(i_1, i_2)$. After selection, this frequency become $p'(i_1, i_2) = w(i_1)p(i_1, i_2)/W$, where $w(i_1)$ is the fitness function and W is the mean fitness. An individual mated no more than once. Individuals paired randomly and mating of a pair occurred with the probability $M(d)$, where d is the ratio of the difference between their phenotypes in trait 2 expressed as a proportion of n_2. All unmated individuals paired again, and the process continued until less than 10^{-10} of the population remained unmated. In this way, $A(i_1, i_2, j_1, j_2)$, that is, the frequency of mating between (i_1, i_2) and (j_1, j_2) individuals, was calculated. During reproduction a pair of such parents produced an offspring (k_1, k_2) with the probability $R(i_1, j_1, k_1)R_2(i_2, j_2, k_2)$, where R_m describes the transmission of phenotypes in the m-th trait.

The three-trait model was analogous, but M depended on $d = |i_2/n_2 - j_3/n_3|$, where i_2 and j_3 were phenotypes of the first (female) and second (male) potential partners in traits 2 and 3, respectively. THINK C programs are available on request.

Parameters. During selection, 10% of individuals with the highest and lowest values of their phenotypes in trait 1 had fitness 1.0, and the fitness of the rest was $D < 1.0$. Such selection causes sympatric speciation most efficiently[18].

In both models, $M(d) = 1 - d$, that is, the degree of reproductive isolation, grew linearly with the difference between the potential mates, but only the individuals that were maximally different were completely isolated. If such individuals were isolated but all others mated freely, speciation never occurred.

In most runs, the initial population consisted of 99.99% of individuals with phenotype 0 in all two or three traits. The rest of the population had phenotypes no more than 1 in every trait and the covariances between the traits were very small.

Received 20 January; accepted 10 May 1999.

1. Mina, M. V., Mironovsky, A. N. & Dgebuadze, Y. Y. Lake Tana large barbs: phenetics, growth and diversification. *J. Fish Biol.* **48,** 383–404 (1996).
2. Berrebi, P. & Valiushok, D. Genetic divergence among morphotypes of Lake Tana (Ethiopia) barbs. *Biol. J. Linn. Soc.* **64,** 369–384 (1998).
3. Schliewen, U. K., Tautz, D. & Paabo, S. Sympatric speciation suggested by monophyly of crater lake cichlids. *Nature* **368,** 629–632 (1994).
4. Johnson, T. C. *et al.* Late pleistocene desiccation of Lake Victoria and rapid evolution of cichlid fishes. *Science* **273,** 1091–1093 (1996).
5. Ritchie, M. G. & Phillips, S. D. F. in *Endless Forms: Species and Speciation* (eds Howard, D. J. & Berlocher, S. H.) 291–308 (Oxford Univ. Press, New York, 1998).
6. Kondrashov, A. S. & Mina, M. V. Sympatric speciation: when is it possible? *Biol. J. Linn. Soc.* **27,** 201–223 (1986).
7. Kondrashov, A. S. On the intensity of selection for reproductive isolation at the beginnings of sympatric speciation. *Genetika* **20,** 408–415 (1984).
8. Barton, N. H. On the spread of new gene combinations in the third phase of Wright's shifting-balance. *Evolution* **46,** 551–557 (1992).
9. Doebeli, M. A quantitative genetic competition model for sympatric speciation. *J. Evol. Biol.* **9,** 893–909 (1996).
10. Shpak, M. & Kondrashov, A. S. Applicability of the hypergeometric phenotypic model to haploid and diploid populations. *Evolution* **53,** 600–604 (1999).
11. Darwin, C. *The Origin of Species by Means of Natural Selection* (Murray, London, 1859).
12. Kondrashov, A. S. & Shpak, M. On the origin of species by means of assortative mating. *Proc. R. Soc. Lond. B* **265,** 2273–2278 (1998).
13. Wallace, A. R. *Darwinism.* (Macmillan, London, 1889).
14. Rice, W. R. Disruptive selection of habitat preference and the evolution of reproductive isolation: a simulation study. *Evolution* **38,** 1251–1260 (1984).
15. Kondrashov, A. S. Multilocus model of sympatric speciation. III. Computer simulations. *Theor. Popul. Biol.* **29,** 1–15 (1986).
16. Maynard Smith, J. Sympatric speciation. *Am. Nat.* **100,** 637–650 (1966).
17. Rice, W. R. & Hostert, E. E. Laboratory experiments on speciation: what have we learned in 40 years? *Evolution* **47,** 1637–1653 (1993).
18. Kondrashov, A. S., Yampolsky, L. Yu. & Shabalina, S. A. in *Endless Forms: Species and Speciation* (eds Howard, D. J. & Berlocher, S. H.) 90–98 (Oxford Univ. Press, New York, 1998).
19. Felsenstein, J. Skepticism towards Santa Rosalia, or why are there so few kinds of animals? *Evolution* **35,** 124–138 (1981).
20. Karlin, S. General two-locus selection models: some objectives, results and interpretations. *Theor. Popul. Biol.* **7,** 364–398 (1975).
21. Turner, G. F. & Burrows, M. T. A model of sympatric speciation by sexual selection. *Proc. R. Soc. Lond. B* **260,** 287–292 (1995).
22. Galis, F. & Metz, J. A. J. Why are there so many cichlid species? *Trends Ecol. Evol.* **13,** 1–2 (1998).
23. van Doorn, G. S., Noest, A. J. & Hogeweg, P. Sympatric speciation and extinction driven by environment dependent sexual selection. *Proc. R. Soc. Lond. B* **265,** 1915–1919 (1998).
24. Axelrod, H. R. *The Most Complete Colored Lexicon of Cichlids* (TFH Publications, Neptune City, 1996).
25. Seehausen, O., van Alphen, J. J. M. & Witte, F. Cichlid fish diversity threatened by eutrophication that curbs sexual selection. *Science* **277,** 1808–1811 (1997).
26. Seehausen, O. & van Alphen, J. J. M. The effect of male coloration on female mate choice in closely related Lake Victoria cichlids (*Haplochromis nyererei* complex). *Behav. Ecol. Sociobiol.* **42,** 1–8 (1998).
27. Lewontin, R. C., Kirk, D. & Crow, J. F. Selective mating, assortative mating and inbreding: definitions and implications. *Eugen. Quart.* **15,** 141–143 (1966).

Acknowledgements. This study was supported by a grant from the NSF.

Correspondence and requests for materials should be addressed to F.A.K. (e-mail: fedya@simons-rock.edu).

On the origin of species by sympatric speciation

Ulf Dieckmann & Michael Doebeli

Adaptive Dynamics Network, International Institute for Applied Systems Analysis, A-2361 Laxenburg, Austria
Zoology Institute, University of Basel, Rheinsprung 9, CH-4051 Basel, Switzerland

Understanding speciation is a fundamental biological problem. It is believed that many species originated through allopatric divergence, where new species arise from geographically isolated populations of the same ancestral species[1–3]. In contrast, the possibility of sympatric speciation (in which new species arise without geographical isolation) has often been dismissed, partly because of theoretical difficulties[2,3]. Most previous models analysing sympatric speciation concentrated on particular aspects of the problem while neglecting others[4–10]. Here we present a model that integrates a novel combination of different features and show that sympatric speciation is a likely outcome of competition for resources. We use multilocus genetics to describe sexual reproduction in an individual-based model, and we consider the evolution of assortative mating (where individuals mate preferentially with like individuals) depending either on an ecological character affecting resource use or on a selectively neutral marker trait. In both cases, evolution of assortative mating often leads to reproductive isolation between ecologically diverging subpopulations. When assortative mating depends on a marker trait, and is therefore not directly linked to resource competition, speciation occurs when genetic drift breaks the linkage equilibrium between the marker and the ecological trait. Our theory conforms well with mounting empirical evidence for the sympatric origin of many species[10–18].

The theory of adaptive dynamics[19–22] is a general framework for studying phenotypic evolution driven by ecological interactions. One of the phenomena unravelled by adaptive dynamics is evolutionary branching, during which directional selection drives a monomorphic population to a phenotype where ecological interactions induce disruptive selection and a subsequent split into two coexisting phenotypic clusters (Fig. 1a). Evolutionary branching explains the dynamic emergence and perpetuity of disruptive selection and serves as a unifying concept for understanding the evolution of polymorphisms. It is found in a wide range of models of asexual populations (see refs 22 and 23 for examples). Here we show that evolutionary branching also occurs in sexual populations and thus leads to a general theory for sympatric speciation.

We start from assumptions that are likely to be satisfied in many natural populations. Individuals vary in a quantitative character x determining resource use, as for example when beak size in birds determines the size of seeds consumed. Populations consisting of individuals of a given trait value x have density-dependent logistic growth with carrying capacity $K(x)$. We assume that the resource

distribution $K(x)$ is unimodal and varies according to a gaussian function $N(x_0,\sigma_K)$, with the maximum at an intermediate phenotype x_0 and variance σ_K^2. In polymorphic populations consisting of individuals with different trait values, dissimilar individuals interact only weakly, as, for example, when birds with different beak sizes eat different types of seed. That is, competition is not only density- but also frequency-dependent, and rare phenotypes experience less competition than common phenotypes. Specifically, we assume that the strength of competition between individuals declines with phenotypic distance according to a gaussian function $N(0,\sigma_C)$, with a maximum at zero and variance σ_C^2.

These assumptions are integrated into an asexual individual-based model in which each individual is characterized by its trait value x. Individuals give birth at a constant rate and die at a rate that is determined by frequency- and density-dependent competition (see Methods). Evolutionary dynamics occur because offspring phenotypes may deviate slightly from parent phenotypes. The quantitative character first evolves to the value x_0 with maximal carrying capacity. After that, two things can happen: either x_0 is evolutionarily stable and evolution comes to a halt at x_0, or x_0 is actually a fitness minimum and can be invaded by all nearby phenotypes[19,21,22]. In the latter case, evolutionary branching occurs

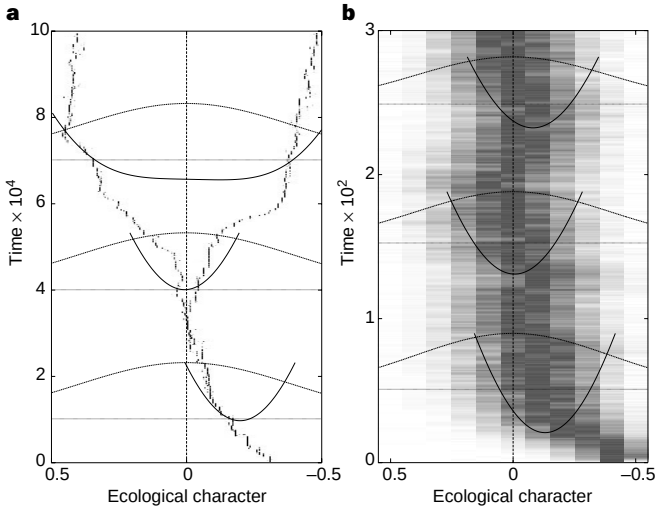

Figure 1 Convergence to disruptive selection **a**, Evolutionary branching in the individual-based asexual model: at the branching point $x_0 = 0$, the population splits into two morphs. Three insets show fitness functions (continuous curves) generated by the ecological interactions at different points in time (indicated by horizontal dotted lines). Selection changes from directional to disruptive when evolution reaches x_0. The resource distribution $K(x)$ has its maximum at x_0 and is shown for comparison (dashed curve). **b**, As in **a**, but with multilocus genetics for the ecological character and random mating. Shading represents phenotype distributions (5 diploid and diallelic loci result in 11 possible phenotypes). Despite disruptive selection at the branching point (see insets), branching does not occur.

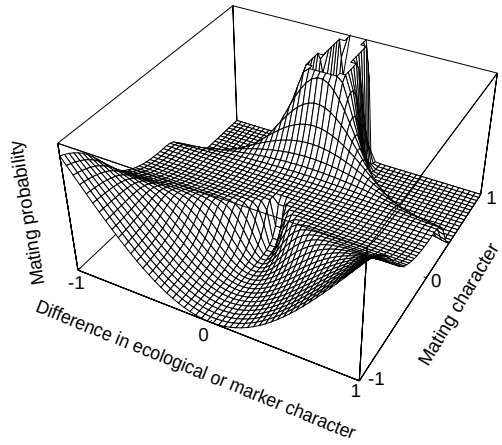

Figure 2 Mating probabilities as determined by mating character and difference in ecological or marker character between mates. The mating character m is scaled to vary between –1 (all – alleles) and +1 (all + alleles). Mating probabilities vary with differences in either ecological or marker character, depending on the scenario. If the mating character in the focal individual is close to +1, it has a high probability of mating with similar individuals. If its mating character is close to –1, it is more likely to mate with dissimilar individuals. Intermediate mating characters (close to 0) correspond to random mating.

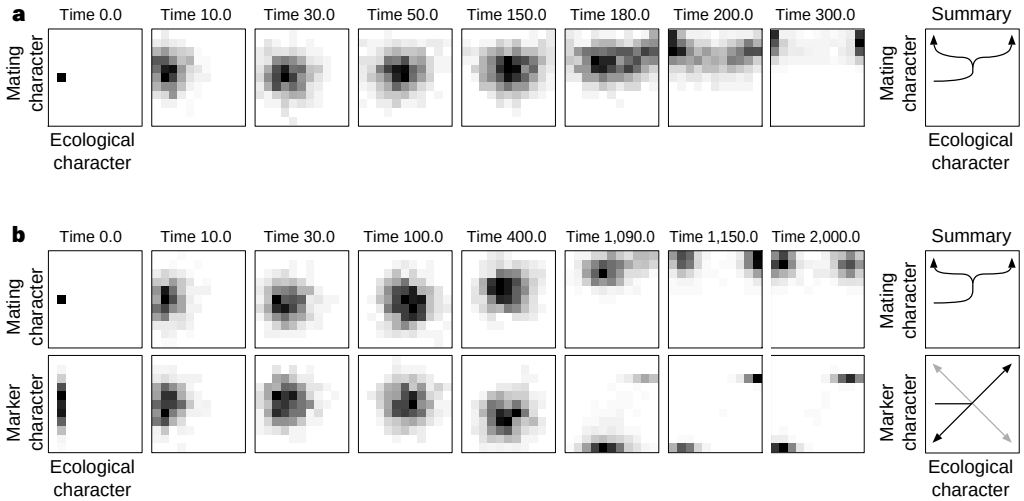

Figure 3 Evolutionary branching in sexual populations. **a**, First scenario: mating probabilities (vertical axes) depend on the ecological character (horizontal axes), which first evolves to intermediate values (50 generations). Then the mean mating character increases to positive values (180 generations) and induces a bimodal split in the ecological character (200 generations). **b**, Second scenario: mating probabilities (vertical axes in upper panels) depend on a marker trait (vertical axes in lower panels). The ecological trait (horizontal axes in all panels) first evolves to intermediate values (100 generations). Owing to temporary correlations between marker and ecological trait, assortative mating increases, which in turn magnifies these correlations (generations 400–1,090). This positive feedback eventually leads to speciation (1,150 generations). In **b** branching typically takes longer than in **a**. The summary panels depict the evolution of mean character values schematically. Grey arrows in the bottom summary panel show an alternative, equally likely, evolution of linkage disequilibrium between ecological and marker character.

(Fig. 1a). This happens for $\sigma_C < \sigma_K$, that is, if the curvature of the carrying capacity at its maximum is less than that of the competition function. Then the advantage of deviating from the crowded optimal phenotype x_0 more than compensates for the disadvantage of a lower carrying capacity.

Sexual reproduction is incorporated by assuming that character values are determined by many additive, diploid loci with two alleles, + and −, and are proportional to the number of 'plus' alleles. Offspring inherit maternal and paternal alleles at each locus independently (free recombination). As in the asexual case, the sexual population evolves to a mean phenotype x_0. If mating is random, however, evolutionary branching does not occur for any values of σ_K and σ_C: the split into two distinct phenotypic morphs is prevented by the continual generation of intermediate phenotypes through recombination (Fig. 1b). Thus, in sexual populations, non-random mating is a prerequisite for evolutionary branching[24].

To model the evolution of assortative mating we assume that individuals express an additional quantitative character that determines mating probabilities according to two scenarios. In the first, mating probabilities are based on similarity in the ecological character, and in the second they are based on similarity in a third, ecologically neutral 'marker' trait (see Methods). Mating character and marker trait are also determined by many additive diallelic loci. Individuals with an intermediate mating character mate randomly. Individuals carrying mostly 'minus' alleles at the mating loci mate disassortatively, and hence are more likely to mate with individuals with very different ecological or marker phenotypes. Individuals carrying mostly plus alleles at the mating loci mate assortatively: the probability of mating increases with phenotypic similarity to the partner (Fig. 2).

Figure 3a shows the evolutionary dynamics of an initially randomly mating population when mating probabilities depend on the ecological character. While this character evolves to x_0, the mating character initially changes only slowly, but it picks up speed and evolves towards positive assortativeness when the ecological character reaches x_0. Once assortativeness is strong enough, the population splits into two ecologically different morphs which eventually are almost completely reproductively isolated. These results confirm and extend those of ref. 24 and occur because, near the dynamically emerging fitness minimum at x_0, selection favours mechanisms that allow for a split in the phenotype distribution and hence for a departure from the fitness minimum. Assortative mating is such a mechanism, because it prevents the generation of intermediate offspring phenotypes from extreme parent phenotypes. Parameter requirements for evolutionary branching in sexual populations appear to be only slightly more restrictive than in the asexual case (Fig. 4).

When assortative mating depends on the ecological character, speciation is not hindered by recombination between mating loci and ecological loci. However, when mating depends on an ecologically neutral marker trait, a linkage disequilibrium between marker loci and ecological loci, leading to a correlation between marker trait and ecological character, is required for the evolution of assortative mating and for speciation. Classical, deterministic models (such as Felsenstein's 'two-allele' models[6]) predict that such linkage disequilibria are unlikely because of recombination between ecological and marker loci[3,6]. In our individual-based model, however, genetic drift due to stochastic demographic effects readily leads to speciation despite the opposing force of recombination. Figure 3b shows the adaptive dynamics when mating probabilities depend on a neutral marker trait. Genetic drift temporarily results in small and localized linkage disequilibria between some marker loci and some ecological loci. Positive and negative correlations both select for assortative mating, which in turn magnifies the local disequilibria into a global linkage disequilibrium between marker and ecological trait. This feedback eventually induces the sympatric split into reproductively isolated phenotypic clusters. Thus, stochastic fluctuations in finite populations can spontaneously break the symmetry of linkage equilibria seen in determi-

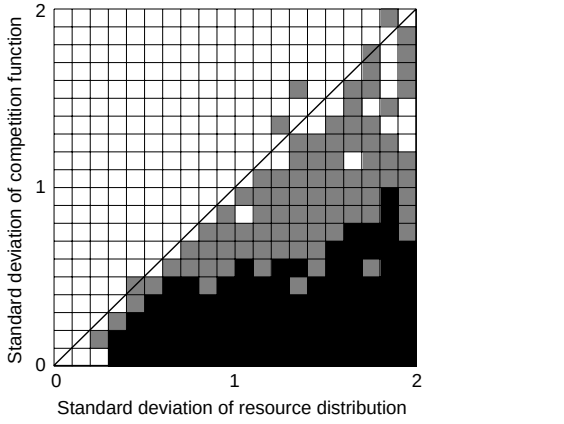

Figure 4 Combinations of standard deviations σ_K and σ_C of the resource distribution $K(x)$ and competition function $C(x)$, respectively, that allow for evolutionary branching. Analytical results are available for the asexual model (see Methods) and predict branching for $\sigma_C < \sigma_K$, that is, below the diagonal. Conditions for branching in sexual populations (within 20,000 generations) are shown in grey when mating probabilities depend on the ecological character and in black when they depend on a marker trait.

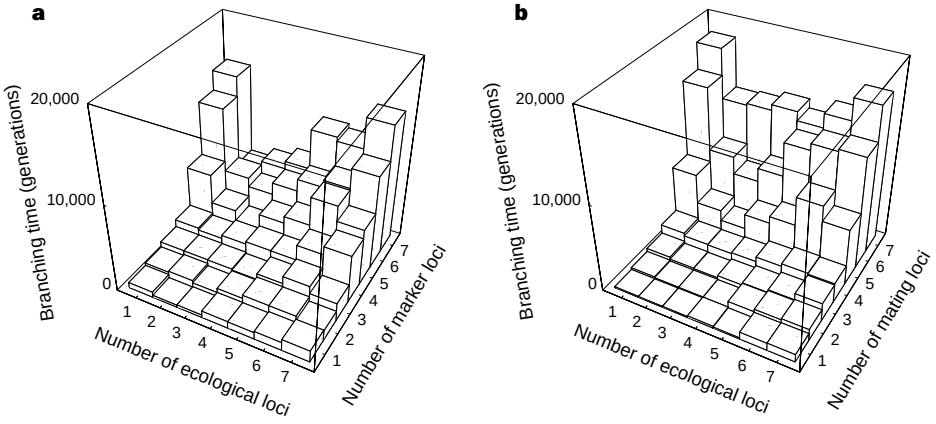

Figure 5 Average waiting times for evolutionary branching with different numbers of loci when assortative mating depends on a marker trait. **a**, Variable numbers of loci for marker and ecological trait with a fixed number of loci (5) for assortative mating. **b**, Variable numbers of loci for assortative mating and ecological trait with a fixed number of loci (5) for the marker trait. Other parameters are as in Fig. 3; each column represents the average waiting time from 60 simulation runs.

nistic models. Recombination between marker loci and ecological loci implies that parameter requirements for evolutionary branching are more restrictive when mate choice is based on a neutral marker than when it is based on the ecological trait (Fig. 4).

The effects of stochasticity on evolutionary branching are further illustrated by varying the number of loci determining the quantitative characters (Fig. 5). Evolutionary branching is more likely when there are fewer loci, because then the phenotypic effects of genetic drift are larger (an exception occurs with only one ecological locus: with only three phenotypes, sufficiently strong fluctuations arise more rarely). Branching triggered by drift becomes less likely in very large populations where stochastic effects become small.

Our results extend and contrast previous insights[6,8,9,24–26] by showing that competition for unimodal resources can initiate sympatric speciation even if assortative mating depends on an ecologically neutral marker trait. The results are robust against changes in the models such as varying numbers of loci (Fig. 5), assuming different mutation rates per locus, different relations between the number of plus alleles on the mate choice loci and the degree of assortativeness (see Methods), and different functions for the carrying capacities, $K(x)$, and for the strength of competition, $C(x)$, while maintaining their qualitative characteristics. Evidence is accumulating that ecology is important for speciation[18,27,28], and our theory may provide an integrative framework for understanding otherwise puzzling evidence for monophyletic origins of many sympatric species, including cichlids[11,12], sticklebacks[13,16,27], snails[14], giant senecios[15] and anolis lizards[17]. In all these cases, it is likely that frequency-dependent mechanisms are important determinants of the species' ecologies. Therefore, assortative mating based on ecologically important traits such as body size (as in sticklebacks[29]) or on marker traits that co-vary with ecological traits (such as coloration or breeding behaviour in cichlids[30]) could have led to the formation of new species in accordance with the theory presented here. We expect our theory to work best in relatively recently colonized habitats, in which sympatric divergence is not strongly opposed by competition from other species already present. In fact, a striking example of incipient sympatric speciation due to ecological interactions in a new habitat has recently been documented in a pair of cichlid morphs (U. K. Schliewen *et al.*, submitted), in which restricted gene flow has evolved through size-assortative mating. The mechanisms of speciation are rarely as clear as in this example, but our theoretical evidence generally suggests a prominent role for ecologically driven speciation in sympatry. □

Methods

The deterministic dynamics of a resident population of phenotype x are

$$\frac{dN(x,t)}{dt} = r \cdot N(x,t) \cdot \left[1 - \frac{N(x,t)}{K(x)}\right]$$

where $N(x,t)$ is the population size at time t. The carrying capacity, $K(x) = K_0 \cdot \exp(-\frac{(x-x_0)^2}{2\sigma_K^2})$, is the stable equilibrium. When a rare mutant y appears in a resident x at carrying capacity $K(x)$, it competes with the discounted density $C(x-y) \cdot K(x)$, where $C(x-y) = \exp(-\frac{(x-y)^2}{2\sigma_C^2})$ describes the strength of competition between phenotypes. Therefore, the per capita growth rate $s(y,x)$ of the rare mutant y is $r \cdot [1 - \frac{C(x-y) \cdot K(x)}{K(y)}]$. The derivative $\frac{\partial s(y,x)}{\partial y}|_{y=x} = r \cdot \frac{K'(x)}{K(x)}$ of $s(y,x)$ with respect to the mutant y and evaluated at the resident x is positive for $x < x_0$ and negative for $x > x_0$. Therefore, x_0 is an attractor for the adaptive dynamics[19,21,22]. In addition, if $s(y,x_0)$ has a minimum at $y = x_0$, then x_0 is a branching point[19,21,22]. This happens if and only if $\sigma_C < \sigma_K$.

These analytical predictions are confirmed by the individual-based asexual model, in which individuals are assigned a phenotype x, give birth at a rate r and die at a rate $\frac{r}{K(x)} \cdot \Sigma_y N(y,t) \cdot C(x-y)$, where the sum weighs all individuals by their competitive impact on x. Offspring have the same phenotype as their parent, except when a mutation occurs (at rate 0.001), in which case their phenotype is chosen from a normal distribution $N(x, \frac{1}{20})$, where x is the parent phenotype.

In sexual populations, birth and death rates are calculated similarly. Individuals are assigned up to three diploid genotypes with five diallelic loci each (variation in loci number is analysed in Fig. 5). The first set of loci determines the ecological character x, the second set determines mating probabilities and the third encodes the marker trait. The mating character m is given by the difference between the number of + and − alleles divided by the total number of alleles. If assortative mating depends on the ecological trait, then, for $m > 0$, mating probabilities fall off with a difference in the ecological trait according to a gaussian function $N(x,\sigma_a)$ with mean equal to the focal individual's ecological trait and variance $\sigma_a = \frac{1}{20m^2}$. If $m = 0$, the focal individual mates randomly. If $m < 0$, then mating probabilities increase with ecological difference according to the function $1 - N(x,\sigma_d)$, where $\sigma_d = \frac{1}{m^2}$ (Fig. 2). If assortative mating depends on the marker trait, then the third set of loci replaces the ecological trait in determining mating probabilities, which then depend on similarity in the marker trait. To avoid a bias against marginal phenotypes in the population, mating probabilities are normalized, so that the sum of mating probabilities over all potential partners is 1 for all phenotypes. A 50:50 sex ratio is assumed at all times. At each locus, one offspring allele is chosen randomly from the two maternal alleles and the other from the two paternal alleles at this locus. With a small probability (0.001), a mutation occurs in the inherited alleles and reverses their value. Other parameter values used for the figures are $r = 1$, $K_0 = 500$, $\sigma_K = 1$ and $\sigma_C = 0.4$ (variation in the last two parameters is analysed in Fig. 4).

Received 28 January; accepted 1 June 1999.

1. Mayr, E. *Animal Species and Evolution* (Harvard Univ. Press, Cambridge, Massachusetts, 1963).
2. Coyne, J. A. Genetics and speciation. *Nature* **355,** 511–515 (1992).
3. Rice, W. R. & Hostert, E. E. Laboratory experiments on speciation—what have we learned in 40 years. *Evolution* **47,** 1637–1653 (1993).
4. Maynard Smith, J. Sympatric speciation. *Am. Nat.* **100,** 637–650 (1966).
5. Rosenzweig, M. L. Competitive speciation. *Biol. J. Linn. Soc. (Lond.)* **10,** 275–289 (1978).
6. Felsenstein, J. Skepticism towards Santa Rosalia, or why are there so few kinds of animals? *Evolution* **35,** 124–138 (1981).
7. Seger, J. in *Evolution. Essays in Honour of John Maynard Smith* (eds Greenwood, P. J., Harvey, P. H. & Slatkin, M.) (Cambridge Univ. Press, Cambridge, 1985).
8. Kondrashov, A. S. Multilocus model of sympatric speciation III. computer simulations. *Theor. Pop. Biol.* **29,** 1–15 (1986).
9. Johnson, P. A., Hoppensteadt, F. C., Smith, J. J. & Bush, G. L. Conditions for sympatric speciation: a diploid model incorporating habitat fidelity and non-habit assortative mating. *Evol. Ecol.* **10,** 187–205 (1996).
10. Bush, G. L. Sympatric speciation in animals—new wine in old bottles. *Trends Ecol. Evol.* **9,** 285–288 (1994).
11. Meyer, A., Kocher, T. D., Basasibwaki, P. & Wilson, A. C. Monophyletic origin of Lake Victoria cichlid fishes suggested by mitochondrial DNA sequences. *Nature* **347,** 550–553 (1990).
12. Schliewen, U. K., Tautz, D. & Pääbo, S. Sympatric speciation suggested by monophyly of crater lake cichlids. *Nature* **368,** 629–623 (1994).
13. Schluter, D. Experimental evidence that competition promotes divergence in adaptive radiation. *Science* **266,** 798–801 (1994).
14. Johannesson, K., Rolan-Alvarez, E. & Ekendahl, A. Incipient reproductive isolation between two sympatric morphs of the intertidal snail *Littorina saxatilis*. *Evolution* **49,** 1180–1190 (1995).
15. Knox, E. B. & Palmer, J. D. Chloroplast DNA variation and the recent radiation of giant senecios (Asteraceae) on the tall mountains of Eastern Africa. *Proc. Natl Acad. Sci. USA* **92,** 10349–10353 (1995).
16. Taylor, E. B. & McPhail, J. D. Evolutionary history of an adaptive radiation in species pairs of threespine sticklebacks (*Gasterosteus*): insights from mitochondrial DNA. *Biol. J. Linn. Soc.* **66,** 271–291 (1999).
17. Losos, J. B., Jackman, T. R., Larson, A., de Queiroz, K. & Rodríguez-Schettino, L. Contingency and determinism in replicated adaptive radiations of island lizards. *Science* **279,** 2115–2118 (1998).
18. Orr, M. R. & Smith, T. B. Ecology and speciation. *Trends Ecol. Evol.* **13,** 502–506 (1998).
19. Metz, J. A. J., Geritz, S. A. H., Meszéna, G., Jacobs, F. J. A. & van Heerwaarden, J. S. in *Stochastic and Spatial Structures of Dynamical Systems* (eds van Strien, S. J. & Verduyn Lunel, S. M.) 183–231 (North Holland, Amsterdam, 1996).
20. Dieckmann, U. & Law, R. The dynamical theory of coevolution: a derivation from stochastic ecological processes. *J. Math. Biol.* **34,** 579–612 (1996).
21. Dieckmann, U. Can adaptive dynamics invade? *Trends Ecol. Evol.* **12,** 128–131 (1997).
22. Geritz, S. A. H., Kisdi, E., Meszéna, G. & Metz, J. A. J. Evolutionarily singular strategies and the adaptive growth and branching of the evolutionary tree. *Evol. Ecol.* **12,** 35–57 (1998).
23. Doebeli, M. & Ruxton, G. D. Evolution of dispersal rates in metapopulation models: branching and cyclic dynamics in phenotype space. *Evolution* **51,** 1730–1741 (1997).
24. Doebeli, M. A quantitative genetic competition model for sympatric speciation. *J. Evol. Biol.* **9,** 893–909 (1996).
25. Turner, G. F. & Burrows, M. T. A model of sympatric speciation by sexual selection. *Proc. R. Soc. Lond. B* **260,** 287–292 (1995).
26. Kondrashov, A. S. & Shpak, M. On the origin of species by means of assortative mating. *Proc. R. Soc. Lond. B* **265,** 2273–2278 (1998).
27. Schluter, D. & McPhail, J. D. Character displacement and replicate adaptive radiation. *Trends Ecol. Evol.* **8,** 197–200 (1993).
28. Schluter, D. & Nagel, L. M. Parallel speciation by natural selection. *Am. Nat.* **146,** 292–301 (1995).
29. Nagel, L. M. & Schluter, D. Body size, natural selection, and speciation in sticklebacks. *Evolution* **52,** 209–218 (1998).
30. Seehausen, O., van Alphen, J. J. M. & Witte, F. Cichlid fish diversity threatened by eutrophication that curbs sexual selection. *Science* **277,** 1808–1811 (1997).

Acknowledgements. We thank H. Metz, R. Law, M. Heino, S. Stearns, J. Maynard Smith and N. Knowlton for helpful comments, and U. Schliewen, K. Rasmussen and D. Tautz for discussions. The order of authors is alphabetical.

Correspondence and requests for materials should be addressed to U.D. (e-mail: dieckman@iiasa.ac.at).