Fluctuating Population Dynamics Promotes the Evolution of Phenotypic Plasticity

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Submitted May 12, 2008; Accepted February 20, 2009; Electronically published June 11, 2009

Online enhancements: appendixes.

abstract: Theoretical and empirical studies are showing evidence in support of evolutionary branching and sympatric speciation due to frequency-dependent competition. However, phenotypic diversification due to underlying genetic diversification is only one possible evolutionary response to disruptive selection. Another potentially general response is phenotypic diversification in the form of phenotypic plasticity. It has been suggested that genetic variation is favored in stable environments, whereas phenotypic plasticity is favored in unstable and fluctuating environments. We investigate the "competition" between the processes of evolutionary branching and the evolution of phenotypic plasticity in a predator-prey model that allows both processes to occur. In this model, environmental fluctuations can be caused by complicated population dynamics. We found that the evolution of phenotypic plasticity was generally more likely than evolutionary branching when the ecological dynamics exhibited pronounced predator-prey cycles, whereas the opposite was true when the ecological dynamics was more stable. At intermediate levels of density cycling, trimorphisms with two specialist branches and a phenotypically plastic generalist branch sometimes occurred. Our theoretical results suggest that ecological dynamics and evolutionary dynamics can often be tightly linked and that an explicit consideration of population dynamics may be essential to explain the evolutionary dynamics of diversification in natural populations.

Keywords: individual-based model, evolutionary model, adaptive dynamics, evolutionary branching, adaptive radiation, resource polymorphism.

Introduction

Trophic polymorphisms are common in nature (e.g., Skúlason and Smith 1995; Robinson and Schluter 2000; Svanbäck et al. 2008) where the polymorphic populations are thought to be on the evolutionary track from a monomorphic population to sympatric speciation, which includes increased phenotypic variation and formation of two or more phenotypic clusters (Skúlason and Smith 1995; Dieckmann et al. 2004). Generally, divergent natural selection on traits that enhance the efficient use of resources is thought to be a major mechanism in the evolution of trophic polymorphisms (Robinson and Schluter 2000; Snorrason and Skúlason 2004).

Trade-offs in foraging efficiencies on different resources are also common in ecological models of adaptive speciation (e.g., Dieckmann and Doebeli 1999; Kondrashov and Kondrashov 1999; Doebeli and Dieckmann 2000). In clonal models, evolutionary branching occurs if populations evolve to a point in phenotype space at which selection turns disruptive as a result of frequency-dependent interactions. In sexual populations, evolutionary branching also requires some form of assortative mating (see discussion in references above). It has recently been pointed out by Rueffler et al. (2006*a*; see also Leimar 2005) that whenever the ecological conditions for adaptive speciation are satisfied, there are also alternative evolutionary scenarios to sympatric speciation. For example, adaptive dynamics models that can give rise to evolutionary branching can also give rise to sexual dimorphisms (Bolnick and Doebeli 2003) or to the evolution of random assignment of phenotypes during development (Leimar 2005) as alternative mechanisms of diversification. Another alternative to evolutionary branching is phenotypic plasticity, where a single genotype produces different phenotypes, depending on the requirements in variable environments (e.g., Stearns 1989; Scheiner 1993; Leimar 2005; Leimar et al. 2006). Phenotypic plasticity was once thought to result primarily from developmental accidents, but recent evidence suggests that environmentally induced phenotypic variation can be selectively advantageous (e.g., Brönmark and Miner 1992; Day et al. 1994; Robinson and Wilson 1996; Svanbäck and Eklöv 2006). Therefore, phenotypic

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Am. Nat. 2009. Vol. 174, pp. 176–189.
DOI: 10.1086/600112

plasticity has come to be viewed as a trait subject to selection, just like any other phenotypic character (Schlichting and Levin 1986; Scheiner 1993; Schlichting and Pigliucci 1998).

There are many examples in nature of either a resource polymorphism, where a population consists of two or more discrete sympatric phenotypic clusters specializing on different prey types (reviewed in Skúlason and Smith 1995), or a more continuous adaptive morphological variation (i.e., individual specialization) along a resource axis (Bolnick et al. 2003). Where investigated, the morphological variations in these populations are due to either genetic differences or phenotypic plasticity (Skúlason and Smith 1995; Bolnick et al. 2003). However, few studies of resource polymorphisms within populations have addressed the combined effects of genetic differentiation and phenotypic plasticity in forming individuals' phenotypes. For example, in both pumpkinseed sunfish (*Lepomis gibbosus*; Robinson et al. 1996) and Eurasian perch (*Perca fluviatilis*; Svanbäck and Eklöv 2003), individuals that occupy the littoral zone in lakes (near-shore vegetated habitat) are deeper bodied compared with the more streamlined individuals that occupy the pelagic zone in lakes (open-water sparsely vegetated habitat). Robinson and Wilson (1996) and Svanbäck and Eklöv (2006) showed that the majority of the morphological variation in these species is due to phenotypic plasticity and only a small amount to genetic variation. It remains unclear, however, why some populations adapt to resource polymorphism by increasing phenotypic plasticity while others undergo genetic differentiation for fixed phenotypes. It has been hypothesized that genetic variation for a fixed phenotype is favored in stable environments (Hori 1993; Smith 1993), whereas phenotypic plasticity is favored in unstable environments (Stearns 1989; Scheiner 1993; Snorrason and Skúlason 2004). Many natural populations fluctuate in density over time as a result of variation in resource levels (Mittelbach et al. 1995; Smith et al. 1999; Grant and Grant 2002; Persson et al. 2003), and the variations in resource levels can be related to both external environmental factors (e.g., Grant and Grant 2002) and intrinsic biotic factors, that is, population dynamics (e.g., Persson et al. 2003). In both cases, population fluctuations will influence diet and habitat choices and thus the selective forces acting on the population (Svanbäck and Persson 2004, 2009; Svanbäck and Bolnick 2005, 2007).

Based on classic work on niche polymorphisms by Levene (1953) and Maynard Smith (1966), several recent theoretical studies used adaptive dynamics theory to show that consumer populations exploiting two distinct food resources or habitats can evolve to an evolutionary branching point, where further evolution could give rise to either a genetic polymorphism or speciation (e.g., Kisdi and Geritz 1999; Rueffler et al. 2006*b*; Claessen et al. 2007). The condition for evolutionary branching in such models is that there are trade-offs in foraging efficiencies where the generalist strategy can utilize both resources but at a lower foraging efficiency compared with either of the specialist strategies. Most previous models (e.g., references above) have assumed very simple population dynamics, with populations always exhibiting a stable (ecological) equilibrium, and they have not allowed for the possibility of single genotypes exhibiting different phenotypes due to phenotypic plasticity. The aim of this article is to study the two competing evolutionary processes of genetic diversification (classical evolutionary branching) and diversification through the evolution of phenotypic plasticity in niche models with possibly complicated population dynamics and hence potentially large environmental fluctuations. We develop a model of a predator population (consumer) utilizing two prey populations (resource) occurring in different habitats. The predator population is characterized by two clonally inherited quantitative traits, the average prey utilization phenotype and the amount of phenotypic plasticity in prey utilization. The model can give rise to evolutionary diversification either by evolutionary branching in the average prey utilization trait, leading to predator types specialized on the two different prey types, or through the evolution of phenotypic plasticity in prey utilization, allowing one predator genotype to give rise to a well-adapted phenotype, depending on the prey habitat in which it ends up. We are interested in the population dynamic conditions determining which of these two alternative evolutionary outcomes is more likely to occur.

The Model

The ecological model we are analyzing is a Rosenzweig-MacArthur predator-prey model (Rosenzweig and MacArthur 1963) that was extended with existing theory of habitat choice (de Roos et al. 2002) and individual-based evolutionary models (Dieckmann and Doebeli 1999; Doebeli and Dieckmann 2000). In our version of the model, the two resource types are different prey types that are assumed to inhabit different habitats. The deterministic model is given by the following equations:

$$\frac{dN_i}{dt} = r_i N_i\left(1 - \frac{N_i}{K_i}\right) - F_i P,$$

$$\frac{dP}{dt} = P(c_1F_1 + c_2F_2) - mP. \tag{1}$$

Here N_i and P are the densities of the two prey types i ($i = 1, 2$) and the predator, respectively; r_i is the habitat-

specific intrinsic per capita growth rate of the prey; K_i is the habitat-specific carrying capacity of the prey types; c_i is the prey-specific conversion efficiency of the predator (giving the average number of predator offspring produced per consumed prey of type *i*); m is the per capita death rate of the predator; and F_i is the specific foraging rate (or per capita intake rate) of the predator on prey type *i*. For a general discussion of the dynamic behavior of such predator-prey models, see, for example, Kot (2001). The foraging rates F_i are further defined as

$$F_1 = qf_1, \tag{2}$$

$$F_2 = (1 - q)f_2, \tag{3}$$

where q is the probability that a given predator type will forage on prey type 1 (i.e., choose habitat 1) and f_i is the functional response on prey type *i*. It should be noted that the expression $c_1F_1 + c_2F_2$ appearing in equation (1) for the predator dynamics is the expected per capita birth rate of a predator individual with foraging rates F_1 and F_2. For the functional response, we used a Holling type 2 function of the form

$$f_i = \frac{a_iN_i}{1 + a_ih_iN_i}, \tag{4}$$

where a_i is the attack rate and h_i is the handling time of prey type *i*. The attack rates and handling times are determined by the predator phenotype (u_i). In our model, a nonplastic predator individual will have the same phenotype in both habitats, whereas a predator individual that has the ability to change its phenotype due to plasticity will exhibit different phenotypes in the two habitats. Specifically, in each habitat there is an optimal phenotype $\hat{u}_i$, and a_i and h_i in habitat i depend on how closely u_i matches $\hat{u}_i$ in that habitat, according to

$$a_i(u_i) = \hat{a}_i \times \exp\left[-\frac{(\hat{u}_i - u_i)^2}{2\sigma_a^2}\right], \tag{5}$$

$$h_i(u_i) = \hat{h}_i + \left\{1 - \exp\left[-\frac{(\hat{u}_i - u_i)^2}{2\sigma_h^2}\right]\right\}, \tag{6}$$

where $\hat{a}_i$ and $\hat{h}_i$ are the maximal attack rate and the minimal handling time, respectively, and in each habitat the decline in predation efficiency departure from the optimum is scaled by the variances σ_a^2 and σ_h^2, respectively. Without loss of generality, we will assume the optimal phenotypes to be $\hat{u}_1 = -1$ and $\hat{u}_2 = 1$. An optimal phenotype on any of the two resources has a high attack rate and a low handling time on that resource. To keep the model as simple as possible, we have chosen to let both handling time and attack rate be related to only one morphological trait (u), but in general, they may be related to different and independently evolving traits.

The predator phenotype (u) is determined by two quantitative genetic traits, x and y, where x is the mean morphology of the predator and y is the phenotypic plasticity around x. The mean morphology can vary from negative to positive values ($-\infty \le x \le \infty$), whereas the plasticity is always ≥ 0. Genotype (x, y) produces the predator phenotypes $u_1 = x - y$ in habitat 1 and $u_2 = x + y$ in habitat 2. Thus, we follow a common definition of phenotypic plasticity, that is, the environmentally sensitive production of alternative phenotypes by given genotypes (Stearns 1989; DeWitt and Scheiner 2004), where the phenotype in our case can be either a morphological character or a behavioral phenotype. We assume that phenotypic plasticity cannot overshoot the optimal phenotype in the habitats; hence, if $x - y < -1$, then $u_1 = -1$, and if $x + y > 1$, then $u_2 = 1$.

Before we can describe the evolutionary dynamics of the genotypes x and y, we have to specify the probability q of foraging in habitat 1 (eqq. [2], [3]). Following de Roos et al. (2002), whether a given genotype (x, y) forages in habitat 1 or habitat 2 depends on the relative profitabilities Q_1 and Q_2 of the two habitats, given by

$$Q_i = \frac{c_if_i}{m}. \tag{7}$$

The profitabilities Q_i in turn determine the probability of foraging in habitat 1 as

$$q = \frac{1}{1 + \exp\left[-s(Q_1 - Q_2)\right]}. \tag{8}$$

Here s is a parameter describing the predator's sensitivity to habitat differences. If $s = 0$, the predator is insensitive to the differences in habitat quality and is equally likely to choose either of the habitats. When $s > 0$, the predator is sensitive to differences in habitat quality, and the more s deviates from 0, the more likely the predator is to choose the higher-quality habitat over the lower-quality habitat (fig. A1 in the online edition of the *American Naturalist*). The role of predator habitat choice is to have an optimally foraging predator that can change habitat to optimize energy intake. We have not analyzed how habitat choice would affect stability in our model because it was not within the scope of this article.

Finally, we introduced a cost to phenotypic plasticity by assuming that the conversion efficiencies c_i depend on the plasticity trait y. Specifically, we assumed that the conversion efficiencies decline linearly with the plasticity y, so that $c_i = \hat{c}_i(1 - yk)$, where $\hat{c}_i$ is the maximal conversion

efficiency for prey type i, achieved for $y = 0$, and k is a parameter measuring the severity of the costs to plasticity. When $\hat{c}_i(1 - yk) < 0$, we set $c_i = 0$.

The deterministic model described above can exhibit very complicated dynamics (Kot 2001). As a consequence, it is impossible to derive analytical results for the evolutionary dynamics of the two predator traits x and y, whose investigation thus necessitates the use of numerical simulations. Also, it is known that finite population sizes can have a significant effect on the dynamics of predator-prey models (Renshaw 1991; Pineda-Krch et al. 2007). We chose to explore the evolutionary dynamics of x and y by formulating an individual-based version of the deterministic model described above (eqq. [1]–[8]) as a stochastic birth-death process in finite populations (Donalson and Nisbet 1999; Pineda-Krch et al. 2007).

The (integer) numbers of prey and predator individuals present at any given point in time are N_i and P, respectively. To implement the individual-based models, we first note that for any given predator individual j, the probability q, the foraging rates f_i, and the conversion efficiencies c_i all depend on the phenotype of the predator individual. In the individual-based model, these quantities therefore receive a subscript j, so that q_j is the probability that predator individual j chooses habitat 1, $f_{i,j}$ is its foraging rate in the two habitats, and $c_{i,j}$ is the corresponding conversion efficiency. Similarly, the expressions for the foraging rates in the two habitats (eqq. [2], [3]) are now individual quantities ($F_{1,j}$, $F_{2,j}$). These should be viewed as expectations (i.e., with probability q_j, the foraging rate is $f_{1,j}$, and with probability $1 - q_j$, the foraging rate is $f_{2,j}$), so that the total expected birth rate of predator individual j, b_j, is given by $b_j = c_{1,j}F_{1,j} + c_{2,j}F_{2,j}$. The individual predator death rates are m.

The per capita intrinsic growth rate in prey population i is r_i. The per capita death rate in prey population i is the sum of two components: (1) intraspecific competition, which generates an individual death rate of $\sum r_iN_i/K_i$, and (2) predation, which generates an individual death rate of $\sum_j F_{i,j}/N_i$, where $F_{i,j}$ is the foraging rate of predator individual j on prey type i and the sum runs over all predator individuals j. Because $F_{i,j}$ is the total foraging rate of predator individual j on prey population i, we have to use the ratio $F_{i,j}/N_i$ to get the effect of predator individual j on a single member of prey population i. Because the predator population is generally polymorphic, the total death rate of an individual of prey type i caused by predators is obtained by taking the sum of the death rates due to predation over all predator individuals, where each predator contributes to the sum according to its genotype.

Given these individual birth and death rates in the prey and predators, the total rates are derived by summing over the populations. Thus, the total birth rate of prey type i is

$$B_i = r_iN_i, \tag{9}$$

and the total death rate of prey type i is

$$D_i = \frac{r_iN_i^2}{K_i} + \sum_j F_{i,j}. \tag{10}$$

The total birth rate of the predator population is

$$B_P = \sum_j (c_{1,j}F_{1,j} + c_{2,j}F_{2,j}), \tag{11}$$

and the total predator death rate is

$$D_P = mP. \tag{12}$$

In equations (9)–(12), N_i and P are the actual (finite) number of prey and predator individuals at a given point in time. The dynamics of the stochastic model unfolds according to Gillespie's (1977) stochastic simulation algorithm. At any given point in time, with current population sizes N_i and P, the total population birth and death rates for the two prey types and the predator are calculated as above, and the next event occurring is chosen stochastically according to the probabilities B_1/E, D_1/E, B_2/E, D_2/E, B_P/E, and D_P/E, where $E = B_1 + D_1 + B_2 + D_2 + B_P + D_P$ is the total event rate. If the chosen event is birth of prey type i, N_i is increased by 1; if it is a death, N_i is decreased by 1. Note that all individuals in a given prey species have the same individual birth and death rates. This is not the case in the predator, where the per capita birth rate depends on the genotype. Thus, if the chosen event is the birth of a predator, then the predator individual giving birth has to be chosen probabilistically according to the individual birth rates, that is, according to the probabilities b_j/B_P.

The chosen predator individual then produces one offspring with genotype (x', y'), which is derived from the parental phenotype under the assumption of clonal reproduction subject to mutation, where the mutation probabilities of the two traits are μ_x and μ_y, respectively. In case a mutation occurred, the new offspring phenotype was drawn from a Gaussian distribution with mean equal to the corresponding parent genotype (x or y) and variance σ_x or σ_y, respectively. Because phenotypic plasticity cannot be less than 0, we redrew any mutation that would have yielded a negative y value. Finally, if the chosen event is the death of a predator, the predator individual to be removed was chosen at random because all predators have the same death rate.

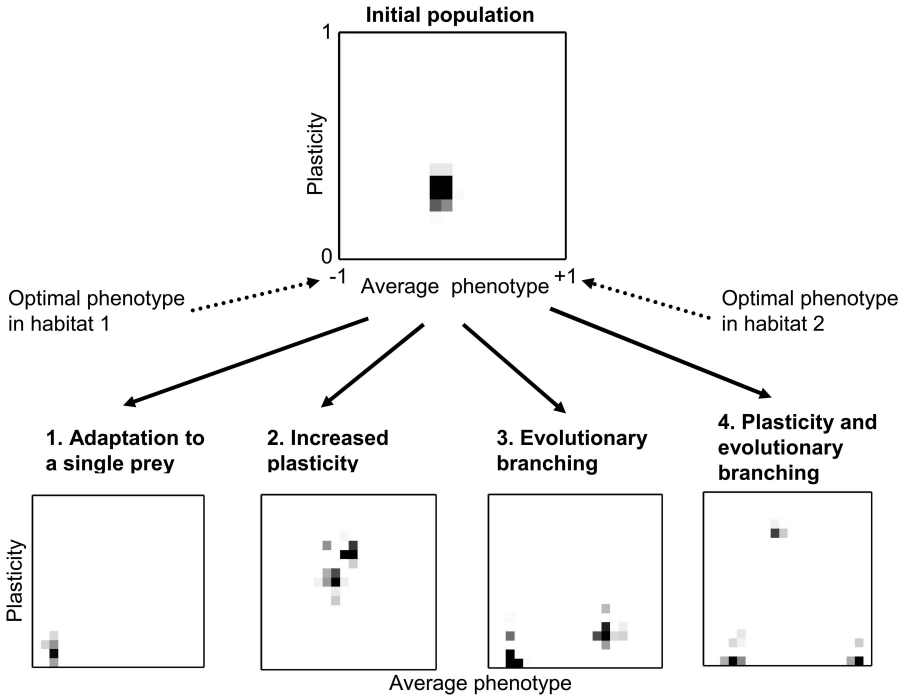

Figure 1: Examples of different evolutionary outcomes from the model. The initial population has an intermediate phenotype with a small amount of phenotypic plasticity (*top*). *Bottom*, evolutionary outcomes in the two traits after 10,000 time units. *1*, Predator population becomes perfectly adapted to the optimal phenotype in one of the two habitats, with virtually no ability for phenotypic plasticity. *2*, Predator evolves an increased phenotypic plasticity, allowing it to efficiently prey on both prey types. *3*, Predator undergoes evolutionary branching such that each of the emerging branches is well-adapted to one of the two habitats but exhibits little phenotypic plasticity. *4*, Predator diversifies into one generalist genotype with high plasticity and two specialist genotypes with low plasticity.

After an event has occurred (birth or death in either prey or predator), all birth and death rates are calculated anew (because they depend on the population size and on which genotypes are present in the predator), and the next event is chosen on the basis of the new rates. The stochastic model thus unfolds in discrete steps from one single birth or death event to the next. In order to translate the passage of real time, the lapse in real time between two successive events is drawn from an exponential distribution with mean $1/E$. Thus, when E is high, little time passes until the next event occurs, and vice versa when E is low. The simulation model was coded in C++ and is available under a GNU General Public Licence (ver. 3 or higher) at http://pineda-krch.com/code.

To characterize the ecological and evolutionary behavior of the stochastic model, we ran a large number of simulations throughout parameter space, where we explored the effect of the cost of plasticity (k), predator mortality (m), and the predators's habitat sensitivity (s). We varied cost of plasticity to investigate the hypothesis that without a cost of plasticity, evolution would lead to increased plasticity, whereas at a high cost, plasticity would be disfavored and hence phenotypic diversification would occur through evolutionary branching. We were interested in how population dynamics and habitat choice influenced the evolutionary dynamics in our model, so we focused on predator mortality and habitat sensitivity because these parameters are known to have large effects on population dynamics and habitat choice (e.g., Claessen et al. 2002; de Roos et al. 2002). Thus, we varied m to create simulations with different types of predator-prey dynamics, ranging from stable equilibrium dynamics to strongly oscillating dynamics. Apart from k, m, and s, all parameters and initial conditions were identical in all simulations (table B1 in the online edition of the *American Naturalist*), unless stated otherwise. The simulations started with $P = N_1 = N_2 = 500$, $\sigma_a = \sigma_h = 1$, and were run for 10,000 time units. Roughly, the predator generation time is $1/m$, and therefore one unit of time would correspond to m predator generations. Ten simulations were run for each parameter combination. The population sizes and the distribution of trait values in the predator population were censused every time unit. The initial average morphology (x) and phenotypic plasticity (y) of a predator were 0 and

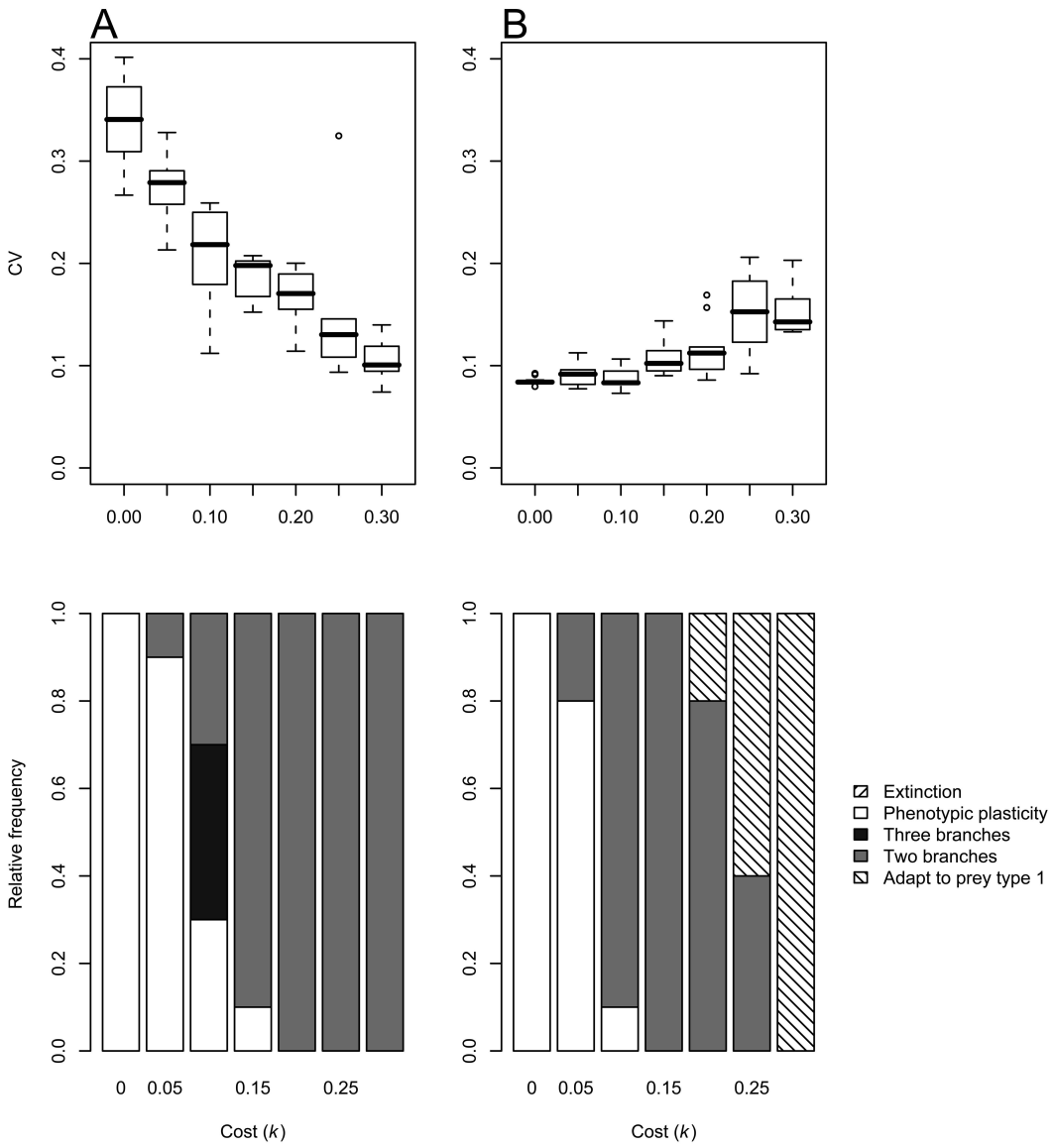

Figure 2: Mean coefficient of variation (CV) in predator population numbers during the first 500 time units and the relative frequency of evolutionary outcomes, depending on cost of plasticity (k) for different predator mortalities (m). In *A*, $m = 0.22$; $s = 10$. In *B*, $m = 0.30$; $s = 10$. For the box plots, whiskers represent the range between the lowest and the highest values, boxes represent the seventy-fifth and twenty-fifth percentiles (the interquartile range), and thick lines represent the median CV. Circles indicate outliers, that is, points located farther than 1.5 times the interquartile range.

0.5 respectively, with a small amount of variation, $\sigma = 0.05$. We calculated the coefficient of variation (CV) on the predator population to characterize the population fluctuations, though the results would have been similar if any of the prey populations had been used because they were all strongly correlated. We did this for the first 500 time steps in each simulation before substantial evolution would have any effect on the population fluctuations. To confirm that our CV results were not biased by either transient dynamics or substantial evolution, we used sliding windows of various sizes. We found no evidence that there were any transient dynamics or evolution biasing our CV results during the first 500 time steps (results not shown).

In order to investigate the effects of the different model parameters (m, s, and k) and the CV on the probability of plasticity versus branching, we analyzed the simulation data by using partial least squares (PLS) regression (for

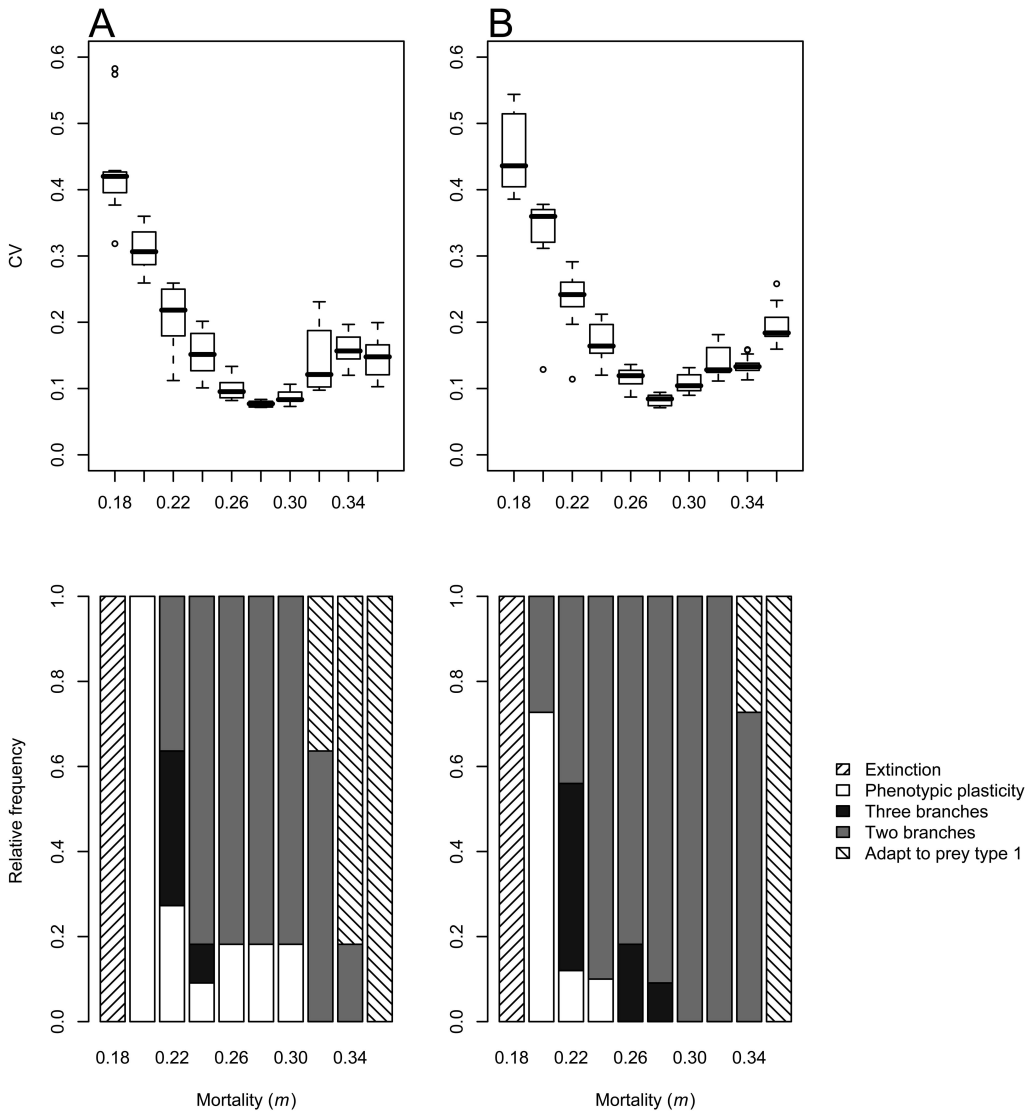

Figure 3: Mean coefficient of variation (CV) in predator population numbers during the first 500 time units and the relative frequency of evolutionary outcome, depending on background predator mortality (m) for two different habitat sensitivities (s). $k = 0.10$. In *A*, $s = 10$. In *B*, $s = 100$. Box plots as in figure 2.

details, see app. C in the online edition of the *American Naturalist*).

Results

Our simulations revealed four main regimes of evolutionary dynamics: (1) predator adaptation to a single prey: the average predator phenotype adapts to one of the prey types while its phenotypic plasticity declines; (2) increased plasticity: the predator increases its phenotypic plasticity without branching in the average phenotype; (3) evolutionary branching: diversification of the average morphology, resulting in two predator branches, both with low phenotypic plasticity; and (4) plasticity and evolutionary branching: branching in the average morphology, resulting in two specialist branches with low plasticity, coexisting with a generalist average type exhibiting a high degree of plasticity (fig. 1). In addition to these evolutionary outcomes, there is also the ecological outcome of extinction of one or both prey types, the predator, or both. In principle, if run long

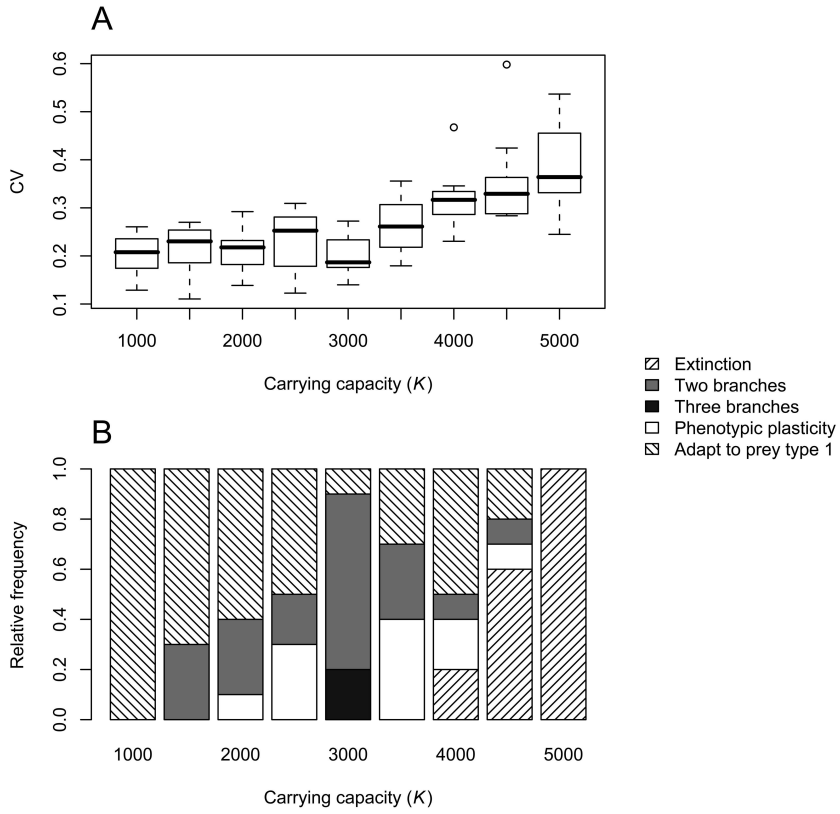

Figure 4: *A*, Coefficient of variation (CV) in predator population numbers during the first 500 time units, depending on variation in carrying capacity (K). Box plots as in figure 2. *B*, Relative frequency of evolutionary outcome, depending on carrying capacity. $m = 0.26$; $k = 0.10$; $s = 10$.

enough, all simulations would eventually yield extinction of all three populations because, technically speaking, this is the only absorbing state of our stochastic model. In our simulations, extinction was, however, recorded only if it occurred within the first 10,000 time units.

Population Fluctuations and Evolutionary Dynamics

When there is no cost to plasticity, our simulations always led to an increase in plasticity and, consequently, no evolutionary branching (fig. 2). As the cost of plasticity increased, however, the probability of evolutionary branching increased as well. Given a cost of plasticity, the nature of the population dynamics was the main determinant of whether phenotypic plasticity evolved or whether the population exhibited evolutionary branching.

The population fluctuation of the predator was influenced by predator mortality, where initially an increase in mortality of the predator led to more stable predator densities, whereas at higher mortalities, the CV increased again (fig. 3). In general, phenotypic plasticity was favored when the density fluctuations were high, whereas branching was favored at more stable populations. For the highest-amplitude fluctuations observed in the predator ($m = 0.18$), the fluctuations eventually led to stochastic extinctions (fig. 3). Before this, however, phenotypic plasticity increased without any sign of branching in the average phenotype. With a small increase in mortality ($m = 0.20$), extinction gave way to persistent high-amplitude population fluctuations. These conditions always promoted the evolution of increased phenotypic plasticity and never led to branching in the average phenotype (fig. 3). The probability of branching started to increase only when the population fluctuation decreased to intermediate levels (for $m = 0.22$–0.26). At this mortality level, evolution occasionally led to three coexisting genotypic clusters, two of which were specialist branches with a low degree of plasticity and the third of which was a generalist average phenotype that was maladapted in both habitats but exhibited a high degree of phenotypic plasticity (fig. 3). With even smaller density fluctuations in the consumer (for $m = 0.28$–0.32), evolution in the consumer led to branch-

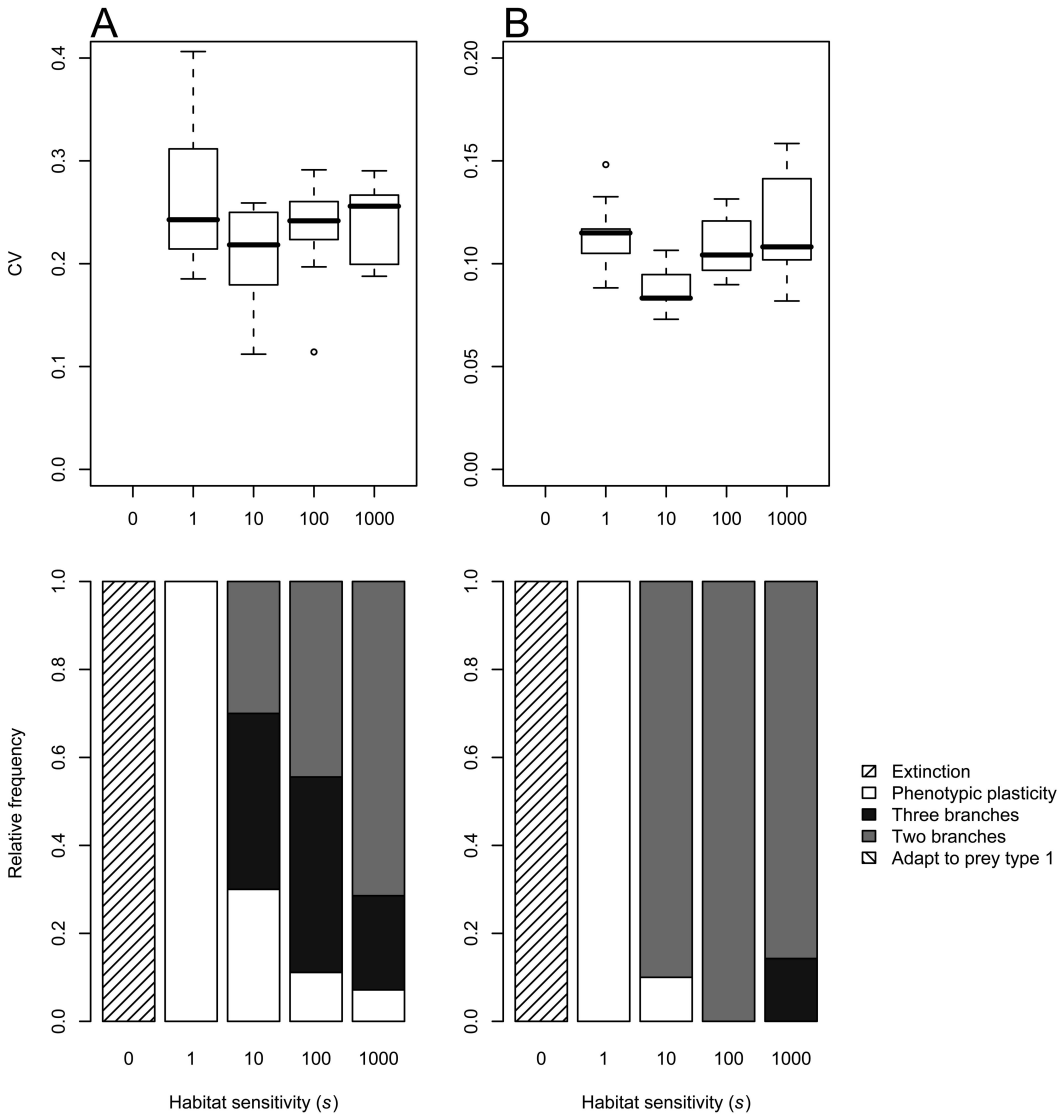

Figure 5: Mean coefficient of variation (CV) in predator population numbers during the first 500 time units and the relative frequency of evolutionary outcome for the different sensitivities to differences in habitat quality (s) for two different consumer mortalities (m). $k = 0.10$. In *A*, $m = 0.22$. In *B*, $m = 0.30$. Box plots as in figure 2.

ing most of the time, with two specialist branches with a low degree of plasticity (fig. 3). At mortalities of 0.34 and 0.36, the predator phenotype evolved to be adapted to prey 1 with a low degree of plasticity due to the high mortality rate of the predator and the low profitability of prey type 2 (fig. 3).

The population fluctuations in the predator can also be influenced by the carrying capacities of the prey, so that an increase in carrying capacities of the prey led to an increase in the CV in predator density (fig. 4*A*). This is in accordance with the well-known paradox of enrichment (Rosenzweig 1971). In general, phenotypic plasticity was favored over branching at intermediate carrying capacities, for which predator density fluctuations increased compared with low carrying capacities, whereas branching was favored over plasticity when population dynamics exhibited smaller fluctuations (fig. 4*B*). We also note that at high prey carrying capacity ($K \geq 4{,}000$), large-amplitude fluctuations observed in the predator often led to stochastic extinctions. Before this, however, phenotypic plas-

ticity increased without any sign of branching in the average phenotype.

Effect of Habitat Sensitivity to the Evolutionary Outcome

Variation in habitat sensitivity (s) had no major effect on variation in predator population fluctuations (fig. 5). Nevertheless, habitat sensitivity affected the ecological and evolutionary outcome of our model. When predators were ignorant of habitat differences ($s = 0$), this always led to extinction of one of the prey types. Before extinction, however, the predator evolved increased plasticity (data not shown). When predators were relatively poor at sensing habitat differences ($s = 1$), then the evolutionary outcome always led to phenotypic plasticity and no branching (fig. 5). As habitat sensitivity increased, the frequency of branching also increased (fig. 5). However, even with high habitat sensitivity, low mortality ($m = 0.22$) led to high-amplitude fluctuations that decreased the chance of branching compared with when there was more stable equilibrium dynamics ($m = 0.30$; fig. 5).

Relative Importance of Different Model Variables to the Evolution of Plasticity

The PLS model identified k as the most important variable explaining increases in phenotypic plasticity over adaptive branching in our model, followed by the CV in population density, m, and, last, s (table C1 in the online edition of the *American Naturalist*). The results of the PLS model were in agreement with what is shown in figures 2, 3, and 5; that is, the probability of plasticity increases with (i) a reduced cost of plasticity, (ii) an increase in CV, (iii) lower mortality, and (iv) lower habitat sensitivity (for a more detailed description of the results from the PLS model, see app. C).

Evaluation of Model Assumptions

To evaluate to what extent the assumption that attack rate and handling time are positively correlated influences our results, we performed control simulations, where we kept the handling times fixed while the attack rate evolved and vice versa. The results of these simulations are qualitatively similar to those from the original simulations. In particular, high-amplitude ecological fluctuations in the predator-prey dynamics promote the evolution of phenotypic plasticity, whereas more stable dynamics tends to lead to evolutionary branching (fig. D1 in the online edition of the *American Naturalist*).

Another model assumption that may influence evolutionary outcomes is the assumption that at every point in time, expected predator foraging rates are calculated on the basis of the potential phenotypes an individual would attain in each habitat. This corresponds to assuming behavioral plasticity, according to which phenotypes can change within short periods of time. In contrast, with morphological plasticity, phenotypes may be fixed at birth (or at least change only on long timescales). We modeled this by assuming that a predator's phenotype is determined at birth (by the same procedure used before; i.e., at birth, habitats are chosen with probabilities determined by the potential phenotypes in each habitat, and throughout its life, the predator individual then has the phenotype corresponding to the habitat chosen at birth). The results of these simulations are qualitatively very similar to those from the original simulations, where high-amplitude ecological fluctuations in the predator-prey dynamics promote the evolution of phenotypic plasticity, whereas more stable dynamics tends to lead to evolutionary branching (figs. E1, E2 in the online edition of the *American Naturalist*).

Discussion

In this article, we used a stochastic individual-based predator-prey model to investigate the evolutionary dynamics of two predator traits that affect predation performance on two different types of prey. The first trait affects the mean performance on each prey type under the assumption that there is a trade-off between the performances on the prey types. The second trait affects the amount of phenotypic plasticity by which predators can adjust their performance phenotype according to the prey type encountered. It is known that the type of two-niche model underlying our study can give rise to evolutionary diversification. In our models, evolutionary branching in the performance trait, on the one hand, and the evolution of phenotypic plasticity, on the other hand, are alternative ways of evolutionary diversification, and by including the potential for both processes to occur, we investigated the conditions that are more conducive to one or the other. A PLS regression (see app. C) identified that, in our model, cost of plasticity (k) was the most important variable explaining the increase in phenotypic plasticity, followed by the ecological dynamics (CV). Mortality rate and habitat sensitivity played minor roles in explaining the increase in plasticity. Without a cost of plasticity, populations always evolved to be composed of individuals with high degree of phenotypic plasticity, and no branching occurred, whereas at a high cost of plasticity, evolutionary branching was a more likely outcome. Besides the obvious effect of cost of plasticity, the ecological dynamics of the predator-prey system is the main determinant of the evolutionary outcome. With high-amplitude ecological fluctuations in the predator-prey dynamics, the probability of

evolutionary branching in the performance trait was low, and instead phenotypic plasticity tended to evolve. On the other hand, with low-amplitude ecological fluctuations, the probability of branching increased. Habitat sensitivity, that is, the ability to distinguish between profitable and nonprofitable habitats, played a minor role in both the ecological and the evolutionary dynamics in our model populations, where an increase in habitat sensitivity decreased population fluctuations and thus increased the chance of evolutionary branching.

Population Dynamics and Evolution

The formation of discrete genetic clusters has been shown to be possible in a number of different ecological models, for both asexual systems and sexual systems (e.g., Dieckmann and Doebeli 1999; Kondrashov and Kondrashov 1999; Doebeli and Dieckmann 2000). Genetically determined diversification is thought to be favored in relatively stable environments, whereas phenotypic plasticity is thought to be more likely when the environment is changing and unpredictable or if the environment fluctuates in a predictable way (e.g., Stearns 1992; Hori 1993; Scheiner 1993; Smith 1993). Our results confirm these expectations in a model in which environmental fluctuations are generated by fluctuating population densities. The generality of this result is supported by the fact that it is independent of whether changes in predator fluctuations are caused by changing predator mortality rate or by changing prey carrying capacity (figs. 3, 4). It might be worth noting that our model assumes clonal inheritance. In a sexual population, phenotypic diversification typically requires assortative mating (see, e.g., Dieckmann and Doebeli 1999; Doebeli et al. 2007). In the absence of assortative mating, formation of a phenotypic polymorphism is more difficult (though not impossible; see, e.g., Kopp and Hermisson 2006; van Doorn and Dieckmann 2006), which could favor the evolution of phenotypic plasticity.

There are many examples of resource polymorphisms and individual diet specialization in nature, among which some represent genetic differentiation and others are examples of phenotypic plasticity (Skúlason and Smith 1995; Bolnick et al. 2003). Our model suggests that population dynamics might play an important role in the evolution of resource polymorphisms and ultimately in sympatric speciation. Interestingly, a recent review suggests that more stable population fluctuations promote resource polymorphisms in fish (Andersson et al. 2007).

Eurasian perch (*Perca fluviatilis*) have been shown to exhibit habitat differences in phenotype, where deeper-bodied individuals are found in the littoral habitat of lakes and the more streamlined individuals are found in the pelagic habitat (Svanbäck and Eklöv 2003). Deeper-bodied individuals of perch, compared with the more streamlined individuals, are superior foragers in the littoral habitat, whereas the more streamlined individuals are better foragers in the pelagic habitats of lakes (Svanbäck and Eklöv 2003, 2004). Furthermore, perch population densities have been shown to fluctuate as a result of predatory and competitive interactions (Claessen et al. 2000; Persson et al. 2000), such that at low density, all perch are found in the littoral zone, feeding on littoral prey types, whereas at high density, a part of the population moves out to the pelagic zone to feed on pelagic prey types (Persson et al. 2000; Svanbäck and Persson 2004). In accordance with our model, a common-garden experiment has shown that the morphological variation in perch is mainly due to phenotypic plasticity (Svanbäck and Eklöv 2006). Interestingly, it has been suggested that Arctic char (*Salvelinus alpinus*) show more stable population dynamics (Johnson 1994; Claessen et al. 2002) than perch. Correspondingly, Arctic char also show much more pronounced differences between different morphs than do perch (Hindar and Jonsson 1982; Parker and Johnson 1991; Svanbäck and Eklöv 2003). Genetic studies also suggest that Arctic char populations may be more genetically differentiated than perch populations (Hindar et al. 1986; Magnusson and Ferguson 1987; Gerlach et al. 2001). This might suggest that the differences in population dynamics between perch and Arctic char could explain why perch morphology is mainly determined by phenotypic plasticity whereas Arctic char morphology is determined more by genetic variation.

Interestingly, the evolutionary dynamics in our models generated coexistence of a phenotypically plastic generalist and two nonplastic specialists under certain conditions. Abrams (2006) investigated the evolution and coexistence of a generalist and specialists and found that in systems with stable ecological equilibria, evolution produces one generalist or two specialists, depending on the trade-off relationship in food acquisition. When there were sustained population fluctuations, evolution led to dimorphism, with two relatively specialized consumers, or trimorphism, with a single generalist and two specialists, again depending on the trade-off function. This is reminiscent of our model, in which trimorphism occurred at intermediate levels of population fluctuations. Wilson and Yoshimura's (1994) purely ecological study also showed that moderate variation in carrying capacities in different habitats promoted the coexistence of generalists and specialists. Furthermore, in their model, specialists were favored at low variations, whereas generalists were favored at high variations, just as in our evolutionary models.

Cost of Plasticity

Adaptive phenotypic plasticity is widespread, and there is good evidence that expression of plasticity as a response

to particular ecological conditions can lead to dramatic fitness benefits, compared with a lack of a plastic response (Newman 1992; Stearns 1992; Scheiner 1993; Schlichting and Pigliucci 1998). Inherent in discussions about adaptive phenotypic plasticity is the idea that there are costs to plasticity (e.g., DeWitt 1998; DeWitt et al. 1998; Scheiner and Berrigan 1998; Relyea 2002; Merilä et al. 2004). Cost of plasticity is defined as fitness deficits associated with plastic genotypes relative to fixed genotypes producing the same average phenotype in a focal environment (DeWitt et al. 1998; Scheiner and Berrigan 1998). There are only a few data, however, on the energetic cost of plasticity, showing mixed results (e.g., Nguyen et al. 1989; Krebs and Feder 1997; DeWitt 1998; Scheiner and Berrigan 1998; Relyea 2002). For example, Nguyen et al. (1989) found no cost of plasticity in *Escherichia coli* in reacting to tetracycline environments, but *Drosophila* exhibits a cost of plasticity for heat shock (Krebs and Feder 1997). DeWitt (1998) found weak evidence of a cost of plasticity in snails (*Physa heterospora*) for predator-induced shell plasticity and growth rate plasticity. One obvious explanation for these discrepancies might be that different traits are associated with different costs. For example, behavioral plasticity might have a lower cost than morphological plasticity because the latter may often require structural changes. Morphological plasticity might also come with different costs, depending on what sorts of tissues need to be redefined, for example, muscle tissue versus bony parts. It is also generally unclear how costs of plasticity will manifest themselves. In our models, such costs were assumed to affect the predation conversion efficiency, but they could of course also affect other demographic parameters, such as the background mortality. Nevertheless, our results suggest that if costs of plasticity are low, phenotypic plasticity may often be a more likely form of phenotypic diversification than genetic diversification. Whether this is also true in natural systems will also depend on the relative ranges of phenotypic variation that can be achieved with plasticity, on the one hand, and genetic differentiation, on the other hand. In our model, we have assumed that a phenotypically plastic individual can, in principle, attain any phenotype in the range between the two niche optima, an assumption that may often be violated in real systems.

Another cost of plasticity is the loss of fitness by displaying the wrong phenotype at the wrong time, as a result of slow adaptation rates in relation to the rate of environmental change (Stomp et al. 2008). Stomp et al. (2008) showed that when phenotypic plasticity needs time (e.g., for the building up of new structures), the flexible phenotype benefits from its plasticity only when environmental fluctuations are relatively slow. While Stomp et al.'s (2008) argument is valid in principle, it seems important to note that even if plasticity is thought to be mostly morphological, the timescale of plastic change may be short compared with the timescale of changes in population size. For example, in perch, plasticity is thought to be mostly morphological, and it has been shown that individual perch can change their phenotype in 4 weeks (Olsson and Eklöv 2005), which is a much shorter timescale than the density changes in perch (7–8 years for a full density cycle; see Persson et al. 2000, 2003; Svanbäck and Persson 2004). Interestingly, in our alternative model in which phenotypes were fixed at birth (app. D in the online edition of the *American Naturalist*), we obtained results qualitatively similar to those from the original simulations, suggesting that in our model, the timescale of plastic change does not greatly affect the evolution of plasticity.

In conclusion, our model shows that ecological dynamics may greatly influence the evolutionary dynamics of diversification in a predator population foraging in two different prey niches. Diversification through the evolution of phenotypic plasticity was favored when the predator-prey system exhibited large fluctuations in population size, whereas evolutionary branching in habitat performance was favored when the predator-prey dynamics was more stable. For intermediate complexities of the ecological dynamics, evolution sometimes generated trimorphisms in which two nonplastic habitat specialists coexisted with a phenotypically plastic generalist. Our results suggest that an explicit consideration of population dynamics may be essential for understanding the evolutionary dynamics of diversification in natural populations.

Acknowledgments

This article was greatly improved by valuable comments on previous drafts of the manuscript by P. Eklöv, M. Kopp, J. Olsson, and M. Stomp. The simulations were performed on the Von Neumann cluster at the Department of Physics and the Driftwood cluster at the Institute of Applied Mathematics of the University of British Columbia. This study was supported by grants from the Swedish Research Council to R.S., from the Pacific Institute for the Mathematical Sciences (Canada) to M.P.-K., and from the Natural Sciences and Engineering Research Council of Canada and the James S. McDonnell Foundation to M.D.

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