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LEARNING AND COLONIZATION OF NEW NICHES: A FIRST STEP TOWARD SPECIATION

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Abstract.—Learning processes potentially play a role in speciation but are often ignored in speciation models. Learning may, for instance, play a role when a new niche is being colonized, because the learning of niche features may cause niche-specific assortative mating and a tendency to produce young in this niche. Several animal species learn about their environmental features that may be important in finding or attracting mates. We use a gene-culture coevolutionary model to look into the effect of such learning on the colonization of new niches and on the genetic divergence between groups using different niches, which are steps necessary in achieving speciation. We assume that density is regulated separately in each of the two niches and that the viability of an individual depends on its genotype as well as on which niche it exploits. Our results show that genetic adaptation to the new niche is enhanced by a high female fecundity and a low viability selection against heterozygotes. Furthermore, when initial colonization (without genetic adaptation) fails, genetic divergence is more difficult when the mating preference is stronger. In contrast, when colonization without genetic adaptation is successful, a stronger mating preference makes genetic divergence easier. An increase in the number of egg-laying mistakes by females can have a positive or negative effect on the success of genetic adaptation depending on other parameters. We show that genetic divergence can be prevented by a niche shift, which can occur only if viabilities in the two niches are asymmetrical.

Key words.—Colonization of niches, genetic divergence, learning, speciation, Viduidae.

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Speciation processes attract a lot of attention from both experimentalists and theoreticians. Classically, researchers have focused on the geographical context of speciation ranging from allopatry through parapatry to sympatry (Mayr 1942, 1963; Schilthuizen 2001; Turelli et al. 2001). Kirkpatrick and Ravigné (2002) propose different classifications of speciation mechanisms, for example, by different forms of disruptive selection or by prezygotic isolating mechanisms. One factor that may play a role in speciation but that is not mentioned by Kirkpatrick and Ravigné (2002) is the influence of learning processes. The potential of learning to generate assortative mating may be equally large as that of geographical separation (see Kondrashov and Mina 1986). Indeed, several authors have suggested that learning plays a role in speciation and other evolutionary processes (ten Cate and Bateson 1988; Grant and Grant 1997; Irwin and Price 1999; Owens et al. 1999; ten Cate and Vos 1999; Slabbekoorn and Smith 2002). However, although a multitude of mathematical and simulation models has been employed to investigate whether speciation in the presence of gene flow between different lineages is feasible (e.g., Turner and Burrows 1995; Kawecki 1997; Payne and Krakauer 1997; Dieckmann and Doebeli 1999; Higashi et al. 1999; Kondrashov and Kondrashov 1999; Gavrilets 2000; Gavrilets et al. 2000; Takimoto et al. 2000; van Doorn and Weissing 2001; Gavrilets and Waxman 2002; Doebeli and Dieckmann 2003), relatively few theoretical studies have attempted to verify these suggestions (Laland 1994a,b; Lachlan and Slater 1999; Aoki et al. 2001; Beltman et al. 2003; Ellers and Slabbekoorn 2003). In this paper we focus on this neglected aspect of speciation mechanisms.

A compelling example where learning has been proposed to influence speciation is the case of finches of the genus *Vidua* (family Viduidae). They are specialist brood parasites of estrildid finches. The male viduids copy the songs of their foster father (Payne 1973b; Payne et al. 1998). Although female viduids do not sing, they do learn to recognize the heterospecific songs that they hear at a young age. They develop a mating preference for males singing a song resembling that of their foster father (Payne 1973a; Payne et al. 2000), and they prefer to lay their eggs in the nests of individuals that sing songs similar to those of their foster father (Payne et al. 2000). The learning of heterospecific song not only maintains the viduids as specialist brood parasites, but also yields a potential mechanism for speciation because it enables the colonization of new host species (Payne 1973b; Payne and Payne 1995; Klein and Payne 1998; Payne et al. 1998, 2000; ten Cate 2000; Sorenson and Payne 2001; Payne et al. 2002). When some females lay eggs in the nests of a new foster species, the cross-fostered young will learn the new song. These young will most likely mate assortatively with each other as a consequence of the learned song preference. The result of these events could be a new specialist brood-parasitic species.

The process of speciation that is thought to occur in the Viduidae is an example of a more general phenomenon. Important features of the mechanism are that a formerly unused niche is being colonized and the learning of niche characteristics causes both niche-specific assortative mating and a preference to produce young in that niche. In the case of brood-parasitic finches the new niche is constituted by a different foster species, and the learned characteristic of the niche is the foster species' song. Other groups in which this mechanism of speciation could play a role are fish and insects. For instance, in some anemonefish species that have a symbiotic relationship with anemones, it has been shown that the larvae imprint on chemicals secreted by the anemones to locate these hosts as adults (Arvedlund and Nielsen 1996; Arvedlund et al. 1999). Other fish species as well (e.g., salmon) are known to imprint olfactorily at a young age on their

local habitat (rivers or streams) and use this learned information as adults for homing (Hasler and Scholz 1983; Dittman et al. 1996). Another example is provided by insects feeding on particular food plants. Some insect species prefer to feed on the food plant they have experienced since their birth (reviewed by Papaj and Prokopy 1989; Bernays and Chapman 1994). Many species of parasitic wasps learn odors of the food substrate of their hosts, and use these to find hosts (for reviews, see Turlings et al. 1993; Vet et al. 1995). In all these cases, learned host, habitat, or feeding preferences can give rise to both assortative mating of the groups exploiting new and old niches as well as to production of young in these niches. Note that in these examples assortative mating and location of reproduction results from a learned habitat preference instead of a learned mating preference and egg-laying preference (which is the case for the brood-parasitic finches).

The colonization of a new niche is a first step toward speciation, but it is not the same as speciation. Although the individuals exploiting different niches may show assortative mating, they are not yet genetically different. Therefore, the different groups are best named ''cultural species'' instead of species. After the colonization process, one of the mechanisms that may generate genetic differences between the cultural species is drift, at least if mating is sufficiently assortative. Genetic divergence, however, is even more likely to evolve as a result of selection for adaptation to the new niche, if individuals carrying certain alleles survive better in the new niche, while these alleles decrease fitness in the old niche. When new advantageous alleles have invaded, further progress toward speciation can be made because of the increased specialization. At this stage of speciation, hybrids of the cultural species are less viable because of their intermediate genotype. As a consequence, the strength of the mating preference may increase in the course of evolution, resulting in fewer matings that produce hybrids.

In this paper we use a gene-culture coevolutionary model to look into the initial steps of the described speciation process, assuming that the learning of niche features causes both niche-specific assortative mating and a tendency to produce young in that niche. Starting from a population that is genetically specialized on the exploitation of a particular niche, we study under what circumstances colonization of a new niche is possible and whether genetic differences between the cultural species can arise.

Model Description

We consider a population of individuals using either niche *A* or *B*. Females prefer to produce their young in the same niche as they were raised in. The young learn a characteristic of the niche they are raised in. This is a cultural trait denoted by c_A or c_B. Apart from their learned trait, both males and females carry a gene determining their viability when exploiting a certain niche. In reality, such traits will probably be determined by many genes, but for simplicity we use only one diploid locus. The three possible genotypes in the model are g_Ag_A, g_Ag_B, and g_Bg_B.

For mathematical convenience we only follow the dynamics of the females and assume that those of males are equal. There are six possible combinations of the culturally and genetically inherited traits. The density of females and males of these combinations is denoted by N_i ($i = 1, \ldots, 6$; see Table 1). The model consists of recurrence equations of these densities (see the appendix for the full set of equations). To derive the recurrence equations, we assume that the life history of the individuals is as follows.

Table 1. The variable names.

Variable	Cultural trait	Genetic trait
N_1	c_A	g_Ag_A
N_2	c_A	g_Ag_B
N_3	c_A	g_Bg_B
N_4	c_B	g_Ag_A
N_5	c_B	g_Ag_B
N_6	c_B	g_Bg_B

In step 1, individuals mate assortatively with respect to their cultural trait. We use polygynous mating as in Kirkpatrick (1982). Mating frequencies depend on the preference of females for males that have the same cultural trait and on male frequencies. A higher mating preference α means that mating is more assortative with respect to the cultural trait. We assume α to be at least one, because this value means that mating is random. For example, the probability of a $g_Ag_Ac_A$ female to mate with a $g_Ag_Ac_A$ male is:

$$\frac{\alpha N_1}{\alpha(N_1 + N_2 + N_3) + N_4 + N_5 + N_6}. \quad (1)$$

Note that we assume that there is no cost of female choice: females will mate with other males if the favored males are not present.

In step 2, the average fertility of females is denoted by *E*. We assume that *E* is sufficiently large to sustain a population in at least one of the niches (the exact condition for such a viable population is given in the results section). The genotype of the young is determined by Mendelian genetics. The young learn features of the niche they grow up in: young that grow up in niche *A* (*B*), have cultural trait c_A (c_B). Females prefer to produce their young in the same niche as they grew up in themselves, but with probability *p* they produce their young in the ''wrong'' niche. We only consider values of *p* between 0 and 0.5, because larger values for *p* would mean that females prefer to produce young in the niche they did not grow up in themselves. For simplicity we assume that generations are nonoverlapping.

In step 3, the density of the young growing up in niche *A* is regulated separately from those growing up in niche *B*. As an example we assume Beverton-Holt type density dependence. We expect that the exact type of density dependence does not qualitatively influence the results. At high densities, the number of young produced decreases due to a lower production or to an increased mortality. For example, the number of young of type 1 ($c_Ag_Ag_A$) after density dependence has acted is

$$\frac{Y_1}{1 + K(Y_1 + Y_2 + Y_3)}, \quad (2)$$

where *K* is a parameter determining the size of the population at equilibrium ($K > 0$), and Y_i (*i* refers to the types in Table

1) is the number of young of type i before density regulation. For simplicity we assume that at equilibrium the two niches can sustain an equal number of individuals.

In step 4, individuals of different genotypes are subject to viability selection, depending on the niche. In niche A, individuals carrying more g_A alleles have a higher viability than other individuals, whereas in niche B this is the case for individuals carrying more g_B alleles. We denote the viability of type i by w_i (the interpretation of i is as in Table 1). The parameters w_i can range from 0 (no individuals of this type survive) to one (all individuals of this type survive). We assume $w_1 = w_6 = 1$, $w_1 > w_2 > w_3$, and $w_6 > w_5 > w_4$.

Both density regulation and viability selection may cause a decrease in the number of young reaching reproductive age. For simplicity, we assume that density regulation and viability selection take place at different stages in development. In reality these two processes will usually not be independent. For example, individuals that have a low viability are most likely to die as a result of overcrowding. Still, in some cases density regulation and viability selection may occur at separate stages of development. For instance, nestling brood-parasitic Viduidae mimic the mouth markings of their host young (Nicolai 1964). This is adaptive because the foster parents feed chicks that have the ''correct'' mouth markings more often (Payne et al. 2001). Therefore, mortality due to nonmatching mouth markings (viability selection) is likely to take place especially in the nestling and fledgling stage. In this particular example, density regulation may act even earlier, as a result of competition for nest sites between the female brood parasites and as a result of competition between eggs in the nest. For the Viduidae, our model may thus be a reasonable approximation. In other cases, however, viability selection may act before density regulation. For instance, insect larvae specialized on certain food plants may have lower survival on other plants because of toxic substances they are not adapted to. This may play a role at earlier developmental stages than density-dependent death due to competition for resources. We are interested in the general effect of learning niche features on speciation instead of a specific biological example. For this reason we not only study the case that density regulation acts before viability selection, but also investigate whether a reversed order renders qualitatively different results (steps 3 and 4 above are exchanged in this case).

We assume that at the start of the evolutionary process only individuals carrying g_Ag_A alleles (adapted to niche A) are present. By performing a stability analysis of the recurrence equation model, we investigate under what circumstances a situation can be attained in which both niches are exploited by individuals that are adapted to their niches. To interpret the results in an evolutionary context, we additionally study the dynamics of rare heterozygotes in the initial population of g_Ag_A individuals.

Results

The recurrence equation model has several equilibria. First, there exists a trivial equilibrium where the population is extinct. We assume that the trivial equilibrium is unstable, that is the following condition for a viable population is fulfilled:

$$E > \frac{2}{1 + p(w_4 - 1) + w_4 + \sqrt{(1-p)^2(1-w_4)^2 + 4w_4p^2}}. \quad (3)$$

One of the nontrivial equilibria consists of only g_Ag_A and another of only g_Bg_B individuals. In these monomorphic equilibria, individuals of both cultural types exist. In the monomorphic equilibrium A both $c_Ag_Ag_A$ and $c_Bg_Ag_A$ individuals are present, and in the ''monomorphic equilibrium B'' both $c_Bg_Bg_B$ and $c_Ag_Bg_B$ individuals are present. The monomorphic equilibria always exist although they are not necessarily stable. We are interested in the existence, stability and attainability of equilibria where both alleles are present (polymorphic equilibria). Biologically, this represents a situation in which both niches are exploited by individuals that are adapted to their niches. Hence, in a polymorphic equilibrium both colonization of niche B and genetic divergence of the groups exploiting the different niches have succeeded.

Symmetrical Viabilities

Nonadapted homozygotes do not survive

We first analyze the case with $w_3 = w_4 = 0$ and $w_2 = w_5$ (and refer to this as w_{het}, the viability of heterozygotes). Hence, individuals that have two maladapted alleles never survive, and individuals that have only one maladapted allele die with probability w_{het}. Note that because of these assumptions, only one of the niches is exploited in the monomorphic equilibria. In the monomorphic equilibrium A(B) $c_Ag_Ag_A$ ($c_Bg_Bg_B$) individuals are present with density

$$\frac{-1 + (1-p)E}{(1-p)KE}. \quad (4)$$

The assumption of symmetry assures that as soon as one of the monomorphic equilibria is unstable, the other one is as well. If both monomorphic equilibria are unstable, there exists at least one stable polymorphic equilibrium where both niches are being exploited by individuals with genotypes adapted to their niche. When density regulation takes place before viability selection, this situation occurs when

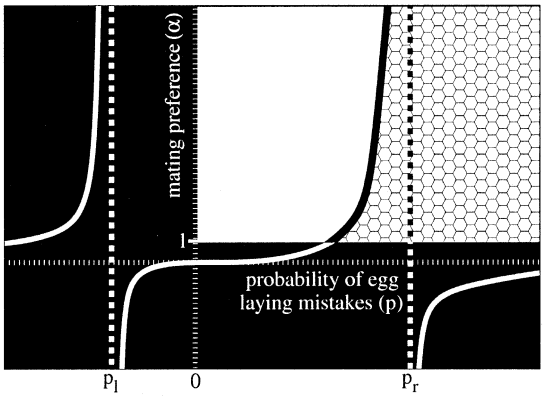
$$\alpha < [(1-p)pE(1-w_{het})] \div \{2p[2 - E(w_{het}+1)](1-w_{het}) + p^2E(2-w_{het}) + (1-w_{het})(-2 + Ew_{het})\}. \quad (5)$$

When viability selection acts before density regulation, this condition becomes

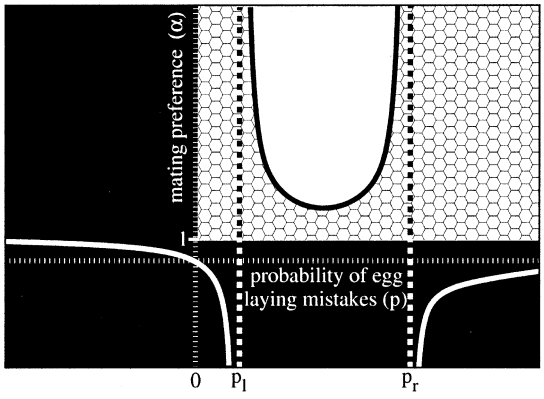
$$\alpha < [-(1-p)pEw_{het}] \div \{2p[1 - E(w_{het})](1-w_{het}) + p^2Ew_{het} - (1-w_{het})(-2 + Ew_{het})\}. \quad (6)$$

The parameter conditions that are necessary to fulfill inequalities (5) and (6) are qualitatively similar. Figure 1 shows in what way the conditions change when female fertility E increases. Biologically unrealistic parameters are included in the figure to give a clearer picture of what is happening mathematically. When $Ew_{het} < 2$ there are two vertical asymptotes, one at positive p and one at negative p (Fig. 1A). When E

(A) $Ew_{het} < 2$



(B) $Ew_{het} > 2$



(C) $Ew_{het} >> 2$

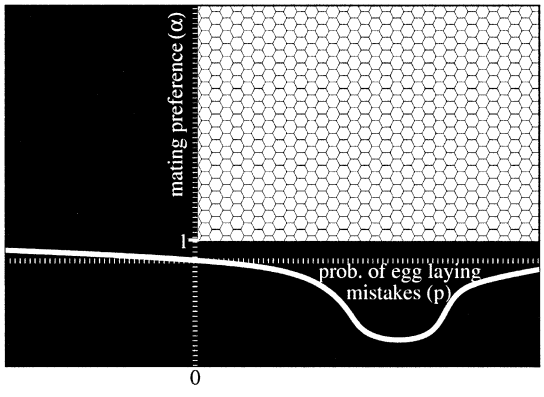


FIG. 1. Attainability of a polymorphic equilibrium in the recurrence equation model for symmetrical viabilities ($w_3 = w_4 = 0$ and $w_2 = w_5 = w_{het}$). Biologically unrealistic parameter values are included (black background) for mathematical clarity. In the white background with hexagonals a polymorphic equilibrium is attained; without hexagonals the system remains in the monomorphic equilibrium *A*. As a result of changes in heterozygote viability w_{het} and female fertility *E*, the vertical asymptotes (dashed lines) shift to the right (A, B), and both asymptotes may disappear completely (C). In (A) and (B) it depends on other parameter values whether the maximum value of p (0.5) is to the right or to the left of the rightmost asymptote. When viability selection occurs before density regulation, p_l and p_r are $[-1 + (1 + E)w_{het} - Ew_{het}^2 \mp \{(w_{het} - 1)(E^2w_{het}^3 - 2Ew_{het}^2 + w_{het} - 1)\}^{1/2}]/(Ew_{het})$, where p_l corresponds to the negative and p_r to the positive root. When density regulation occurs first, p_l and p_r are $[2(1 - w_{het}) + E(w_{het}^2 - 1) \pm \{(w_{het} - 1)(4(w_{het} - 1) + 2E(1 - 2w_{het})w_{het} + E^2(w_{het}^3 + w_{het} - 1))\}^{1/2}]/[E(w_{het} - 2)]$ where p_l corresponds to the positive and p_r to the negative root.

is increased, the left asymptote shifts toward positive p (at $Ew_{het} < 2$, Fig. 1B). If E is increased even further, the two asymptotes disappear (Fig. 1C) when viability selection acts before density regulation or when density regulation occurs before viability selection and heterozygote viability w_{het} is larger than approximately 0.68. Although the two models have similar qualitative outcomes, they are quite different quantitatively. For instance, when the regulation of density acts before viability selection, the disappearance of the area where the monomorphic equilibrium *A* is stable (Fig. 1C) occurs at much higher values of E than when the order of the processes is reversed (Fig. 2).

The parameters w_{het}, E, α and p influence the stability of the monomorphic equilibria (Figs. 1, 2), and hence the attainability of a polymorphic equilibrium. Attaining a polymorphic equilibrium is enhanced by: (1) an increase in heterozygote viability w_{het}; (2) an increase in female fertility E; (3) a decrease in the mating preference α, (4) a high probability of egg-laying mistakes p when $Ew_{het} \leq 2$; and (5), high or low, but not intermediate, values of p when $Ew_{het} > 2$.

The monomorphic equilibrium *A* is unstable when the per capita growth rate of g_Ag_B mutants (heterozygotes) in a resident population of $c_Ag_Ag_A$ individuals is positive, that is, if each heterozygote produces on average more than one heterozygote in the next generation. Because initially the number of heterozygotes is very small, they will experience an environment of solely $c_Ag_Ag_A$ individuals (residents). Hence, the heterozygotes will initially only mate and compete with $c_Ag_Ag_A$ individuals. An intuitive insight into the results can be obtained by considering the influence of the parameters on the number of heterozygotes produced by those initial matings. These include four types of matings, because heterozygotes with both cultural traits should be considered (hereafter referred to as niche *A* and niche *B* heterozygotes), as well as male and female heterozygotes. The contribution of each of these matings to the number of heterozygote young in the next generation helps to explain the influence on the occurrence of genetic divergence (Fig. 3A):

• A fraction w_{het} of all heterozygote young survives viability selection (A–H in Fig. 3A). Hence, an increase in heterozygote viability w_{het} has a positive impact on the attainability of the polymorphic equilibrium.

• Higher female fertility E contributes to the production of niche *B* heterozygotes (*E–H* in Fig. 3A) but not to the production of niche *A* heterozygotes (*A–D* in Fig. 3A). Although more niche *A* heterozygote young are produced as a result of higher fertility, the production of niche *A* homozygotes is

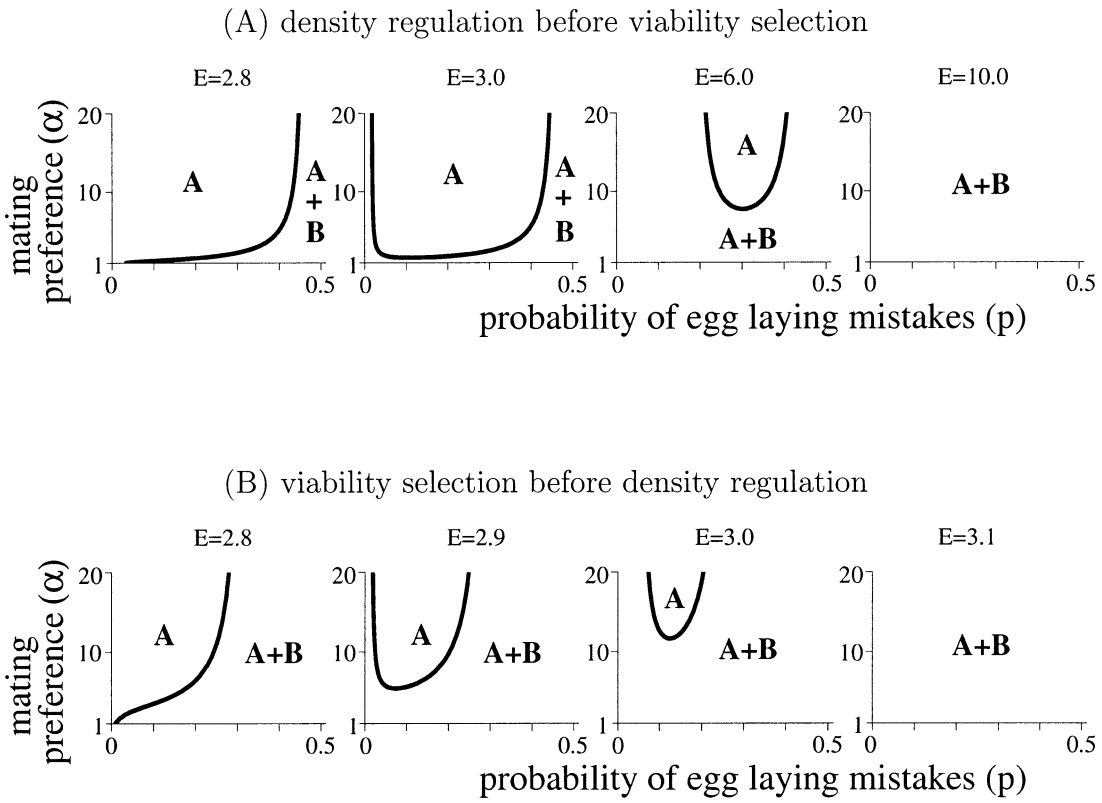


FIG. 2. Comparison of the attainability of a polymorphic equilibrium in the recurrence equation models with orders of density regulation and viability selection exchanged, for symmetrical viabilities ($w_3 = w_4 = 0$ and $w_2 = w_5 = w_{het}$). Capital letters denote which alleles are present in the attained equilibria. The effect of changing female fertility E is demonstrated, when heterozygote viability w_{het} is held constant at 0.7.

increased with the same factor. Density regulation subsequently kills the extra young before they reach maturity, because niche *A* is already maximally exploited. Niche *B* is not yet exploited, hence female fertility does increase production there (*E–H* in Fig. 3A). As a result, an increase in female fertility *E* makes attainability of the polymorphic equilibrium easier.

• The mating preference α influences the production of heterozygotes only as a result of matings between heterozygote niche *B* males and resident females (C and G in Fig. 3A). This happens because an increase in α results in a higher reluctance of c_A females to mate with c_B males. Instead, the resident females will mate with resident males relatively more often. The mating preference does not influence the mating frequency of resident females with heterozygote niche *A* males (A and E in Fig. 3A), because the females' preference for these males does not change in comparison to that for resident males. Finally, the mating preference does not change which males are chosen by heterozygote females (B, D, F, and H in Fig. 3A), because initially the only option for these females is to mate with resident males. In conclusion, an increase in mating preference makes the attainability of the polymorphic equilibrium more difficult.

• The probability of egg-laying mistakes *p* has a complicated impact on the production of heterozygotes. The production of niche *B* heterozygotes by matings between resident males and heterozygote niche *B* females is negatively influenced by *p* (H in Fig. 3A), because mistakes by these females result in the young ending up in niche *A* (D in Fig. 3A). The probability of egg-laying mistakes *p* does not alter the production of niche *A* heterozygotes for the other three types of mating involving c_A females (A–C in Fig. 3A). This is because *p* influences young production by resident females in the same way. Hence, because niche *A* is already in use, density regulation in the maximally exploited niche kills the extra young. On the contrary, the production of niche *B* individuals by c_A females is positively influenced by *p* (E–G in Fig. 3A), because niche *B* is not yet exploited. The net effect of changing *p* is a mixture, but not a simple summation, of all the contributions mentioned.

The reason why the area where the monomorphic equilibrium *A* is stable does not disappear for low heterozygote viability in the model where density regulation occurs first can be understood by comparing the contribution of the initial matings to the number of heterozygote young in the next generation of the two models. The positive influence of female fertility *E* on the number of mutant young in the next generation is present in both models (cf. Figs. 3A and 3B). However, when density regulation acts earlier than viability selection the impact of *E* is much smaller than in the reversed case (compare E–H in Figs. 3A, B). This is due to the competition that niche *B* heterozygote young experience from $c_B g_A g_A$ young that result from egg-laying mistakes by resident females. Thus, on the one hand *E* positively influences the

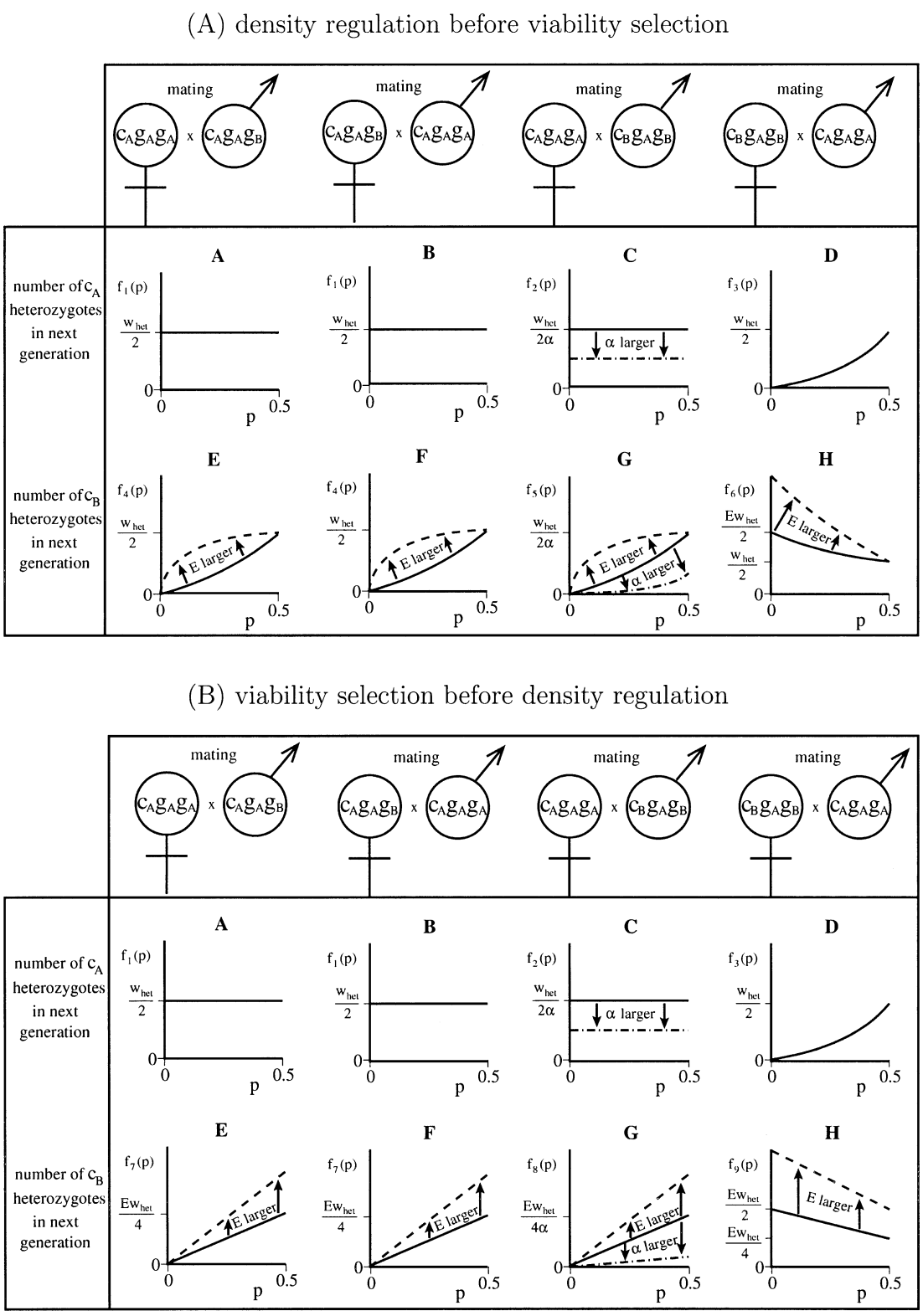


FIG. 3. Contribution of different types of matings to the number of heterozygotes in the next generation. At the monomorphic equilibrium *A* only the four shown types of mating (shown in columns) can give rise to new heterozygotes. The numbers of produced heterozygotes having cultural trait c_A or c_B (shown in rows) depend on female fertility E, heterozygote viability w_{het}, the probability of egg-laying mistakes p, and the mating preference α. Viabilities are symmetrical ($w_3 = w_4 = 0$ and $w_2 = w_5 = w_{het}$). Functions: $f_1(p) = w_{het}/2$, $f_2(p) = w_{het}/(2\alpha)$, $f_3(p) = pw_{het}/[2(1 - p)]$, $f_4(p) = p(1 - p)\, Ew_{het}/[2(1 - 2p + p(1 - p)E)]$, $f_5(p) = p(1 - p)\, Ew_{het}/[2\alpha(1 - 2p + p(1 - p)E)]$, $f_6(p) = (1 - p)^2\, Ew_{het}/[2(1 - 2p + p(1 - p)E)]$, $f_7(p) = pEw_{het}/2$, $f_8(p) = pEw_{het}/(2\alpha)$, $f_9(p) = (1 - p)Ew_{het}/2$.

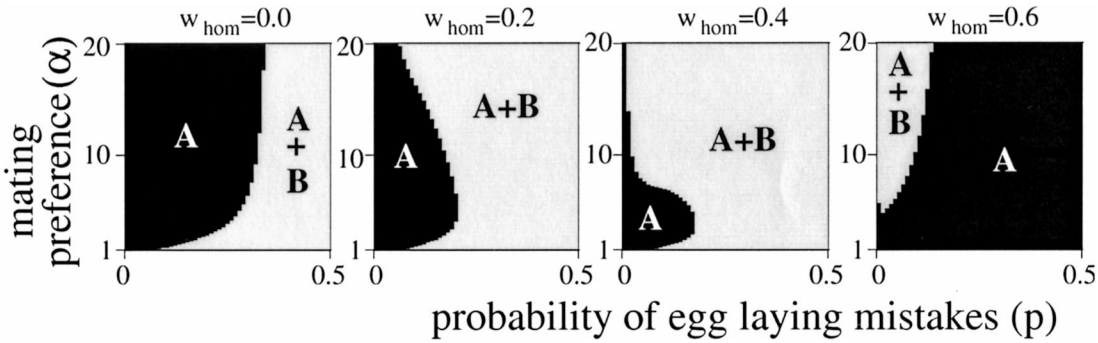


FIG. 4. Attainability of a polymorphic equilibrium in the recurrence equation model for symmetrical viabilities ($w_3 = w_4 = w_{hom}$ and $w_2 = w_5 = w_{het}$). Viability of homozygotes having two maladapted alleles (w_{hom}) increases from left to right. Capital letters denote which alleles are present in the equilibria attained. Other parameters: female fertility $E = 2$, heterozygote viability $w_{het} = 0.8$.

production of heterozygote young, but, on the other hand, it increases death caused by density regulation. As a result, the negative effect of low viability of heterozygote young cannot be compensated for by an increase in female fertility. This is not the case when viability selection takes place before density regulation: then all $c_B g_A g_A$ young die and will thus not interfere with the growth of the niche *B* heterozygote young.

Nonadapted homozygotes can survive

So far we have assumed that individuals having two maladapted alleles could never survive viability selection. Hence, $g_A g_A$ individuals could not survive in niche *B*, and $g_B g_B$ individuals could not survive in niche *A*. In the following this assumption is relaxed: individuals having two maladapted alleles have viability $w_{hom} > 0$. Note that the viabilities are still assumed to be symmetric ($w_{hom} = w_3 = w_4$ and $w_{het} = w_2 = w_5$).

The population constitution of the monomorphic equilibria and hence their stability cannot be determined analytically for the case $w_{hom} > 0$. Instead, to investigate the attainability of a polymorphic equilibrium, we apply a procedure which is the numerical analogue to the previous stability analysis: we initialize the population such that only individuals of genotype $g_A g_A$ are present. Then we iterate the recurrence equations until the densities $N_1, \ldots, N_6$ no longer change. Because we have not yet introduced heterozygotes, the population will now have attained the monomorphic equilibrium *A* where only genotype $g_A g_A$ but both cultural types will be present (given that $w_{hom} > 0$ and $p > 0$). The density of the two types of individuals at equilibrium depends on the probability of egg-laying mistakes p and on the viability of nonadapted homozygotes w_{hom}. An increase in p results in a higher density of c_B individuals and a lower density of c_A individuals at equilibrium, while an increase in w_{hom} results in a higher density of both types of individuals at equilibrium (although mainly the density of c_B individuals is affected in the latter case). Having attained the monomorphic equilibrium *A* (a process typically achieved in five to 10 generations), we add to the population a very small frequency (0.0001) of $c_B g_A g_B$ individuals. Then we again iterate the recurrence equations until a new equilibrium is attained, and we examine the population constitution of this equilibrium. This can either be the monomorphic equilibrium *A* or a polymorphic equilibrium.

Increasing viability w_{hom} of individuals having two maladapted alleles can have a positive or negative effect on attaining a polymorphic equilibrium (Fig. 4). This is a consequence of the fact that two types of residents are now present in the monomorphic equilibrium *A* (before, at $w_{hom} = 0$, only $c_A g_A g_A$ residents were present). The presence of the additional $c_B g_A g_A$ residents may on the one hand increase the probability with which niche *B* male heterozygotes are chosen as mates by female residents. This counteracts the negative effect that the mating preference α has in the case $w_{hom} = 1$. However, as a consequence of the presence of the additional resident type that exploits niche *B*, niche *B* heterozygotes also experience more severe competition from these residents. As illustrated in Figure 4, the positive effect (due to more matings obtained by male heterozygotes) prevails when w_{hom} is only slightly increased, while the negative effect (due to increased competition) starts to dominate at much higher values of w_{hom}.

Asymmetrical Viabilities

Next we study whether the effects of the parameters on the attainability of a polymorphic equilibrium changes if viabilities in the two niches are asymmetrical. Hence, we relax the assumption that $w_2 = w_5$ and $w_3 = w_4$, which means that $g_A g_B$ individuals no longer have the same viability in niche *A* and niche *B*, and $g_B g_B$ individuals in niche *A* no longer have the same viability as $g_A g_A$ individuals in niche *B*. For example, not all hosts will have equal acceptance probabilities for odd-looking, brood-parasitic young. As a consequence, brood parasites using host *A* but having a genotype adapted to host *B* may have a lower viability than brood parasites using host *B* that have a genotype adapted to host *A*. Note that the initial assumptions that $w_1 = w_6 = 1$, $w_1 > w_2 > w_3$, and $w_6 > w_5 > w_4$ are not changed.

To investigate the attainability of a polymorphic equilibrium, it is now no longer sufficient to study the stability of the monomorphic equilibrium *A*. In the case of symmetrical viabilities, instability of the monomorphic equilibrium *A* guaranteed that the monomorphic equilibrium *B* was unstable as well. Because this is not true in the asymmetrical case, the system can now also end up in the monomorphic equi-

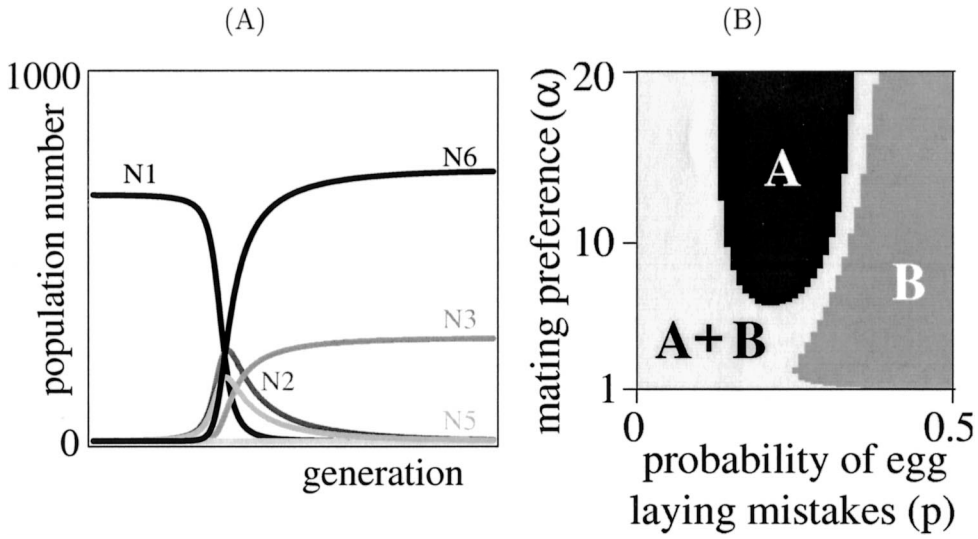


FIG. 5. (A) Time plot demonstrating the occurrence of a shift from the monomorphic equilibrium *A* toward the monomorphic equilibrium *B*. The density of individuals of the different types is denoted by N_i (see Table 1). At the start of the iteration a very small frequency of $c_B g_A g_B$ individuals ($N_5 = 0.0001$) is introduced in the monomorphic equilibrium *A* where only N_1 has nonzero density. Parameters: mating preference $\alpha = 10$, probability of egg-laying mistakes $p = 0.4$. (B) Attainability of equilibria in the recurrence equation model. Capital letters denote which alleles are present in the equilibria attained. Other parameters of (A) and (B): female fertility $E = 5$, viability of $c_A g_A g_B$ individuals $w_2 = 0.8$, viability of $c_A g_B g_B$ individuals $w_3 = 0.4$, viability of $c_B g_A g_A$ individuals $w_4 = 0.0$, viability of $c_B g_A g_B$ individuals $w_5 = 0.6$, $K = 0.001$.

librium *B*. Attaining the polymorphic equilibrium can thus be prevented by stability of the monomorphic equilibrium *A* or because the monomorphic equilibrium *B* is reached.

We first study when stability of the monomorphic equilibrium *A* prevents attaining a polymorphic equilibrium, and whether the effects of the parameters are qualitatively different from the case of symmetrical viabilities. Because the stability of the monomorphic equilibria can again not be determined analytically, we use iteration as described at the end of the previous section to investigate the attainability of a polymorphic equilibrium. As before, we study the case that niche *A* is already in use at the start of the iterations. The effects of the parameters female fertility *E*, probability of egg-laying mistakes *p*, and mating preference α on the stability of the allele *A* equilibrium are not qualitatively different from the symmetrical case. Increasing viability of heterozygotes, that is, parameters w_2 and w_5, which in the symmetrical case were equal, makes it easier to attain the polymorphic equilibrium. Changing viability of $c_A g_B g_B$ individuals, w_3, has no effect on the stability of the monomorphic equilibrium *A*. This is due to the absence of $g_B g_B$ individuals at the time of initial invasion. Changing viability of $c_B g_A g_A$ individuals, w_4, does influence the stability of the monomorphic equilibrium *A*, because this parameter determines the constitution of the resident population. The effect of changing w_4 is qualitatively the same as in the symmetrical case (where $w_3 = w_4 = w_{hom}$). In summary, the effect of all parameters on the stability of the monomorphic equilibrium *A* is not qualitatively different from the symmetrical case, and can be intuitively well understood by the invasion analysis of Figure 3.

Finally, we investigate when attaining a polymorphic equilibrium is prevented because instead the system reaches the monomorphic equilibrium *B*. This means that starting from a situation where mainly niche *A* is exploited, a shift to the exploitation of mainly niche *B* takes place (Fig. 5A). A parasite that shifts to the parasitization of a new host is an example. This situation can occur for a wide range of parameter conditions (an example for fixed w_i and *E* is shown in Fig. 5B). The occurence of the niche shift phenomenon is enhanced by an increase in parameters w_3 and *p*, and by a decrease in parameters w_2, w_5 and *E* (results not shown).

The influence of the parameters on the attainability of a polymorphic equilibrium does not depend qualitatively on the order of viability selection and density regulation (not shown).

DISCUSSION

We used a recurrence equation model to investigate the colonization of a new niche and genetic divergence of the groups exploiting different niches when the learning of niche features causes both niche-dependent assortative mating and a tendency to produce young in this niche. Assuming that individuals exploit only one niche during their lifetime and that individuals are initially adapted to the old niche, at the start of the evolutionary process mainly the old niche is exploited. To achieve an increased exploitation of the new niche, it is necessary for individuals to adapt to the new niche. In the simplified case of genetic divergence at one locus that we study, the success of the adaptation process depends on the per capita growth rate of heterozygotes. Hence, high female fecundity and high viability of heterozygotes make genetic divergence easier (Fig. 2). Another factor that influences the occurrence of genetic adaptation to the new niche is the success of the initial colonization process (without genetic adaptation), because this determines the types of individuals that heterozygotes mate and compete with (Fig. 4). The suc-

cess of initial colonization increases when the viability of nonadapted individuals is higher and the probability of egg-laying mistakes is higher. When initial colonization is not successful, genetic divergence is more difficult when the mating preference is stronger. In contrast, when colonization without genetic adaptation is successful, a stronger mating preference makes genetic divergence easier. An increase in the number of egg-laying mistakes of females can have a positive or negative effect on the success of genetic adaptation (Fig. 2). The order of the two processes of viability selection and density regulation does not qualitatively influence the effects of the model parameters. However, when density regulation takes place earlier than viability selection, it is generally more difficult for the lineages to diversify genetically than when the order of these processes is reversed. This is due to the competition that individuals experience from less viable individuals. Another phenomenon that can obstruct genetic divergence is a niche shift (Fig. 5), which can occur when viabilities in the two niches are asymmetrical. All results depend on our assumption that individuals compete for resources only with individuals using the same niche. If all individuals compete with one another, only one of the genotypes can survive because of the principle of competitive exclusion and genetic differences can thus not be achieved.

In the monomorphic equilibria of our model, in which individuals are genetically identical but both cultural traits are present, the individuals exploiting the different niches rarely interbreed if the mating preference is strong and few egg-laying mistakes are made. Still, the groups in this situation are best referred to as ''cultural'' species, because there are no genetic differences between them. The polymorphic equilibrium represents a situation in which two niches are exploited by individuals specialized on the different niches. Unless the mating preference is strong and few egg-laying mistakes are made, a lot of genetic mixing between the different lineages takes place. In this case the polymorphic equilibrium may best be viewed as one polymorphic species. Thus, attaining a polymorphic equilibrium is in itself not sufficient for speciation. However, once this equilibrium has been reached, further progress toward speciation can be made if evolutionary changes in the strength of the mating preference and the frequency of egg-laying mistakes result in a decreased genetic mixing of the lineages. We are currently using an adaptive dynamics approach (Metz et al. 1996; Geritz et al. 1998) to study the direction of changes in the two traits. Our results (which we will report elsewhere) indicate that the final steps in the speciation process occur easily. Note that to attain the polymorphic equilibrium, in some cases the strength of the mating preference should be low and the number of egg-laying mistakes should be high to attain the polymorphic equilibrium (Figs. 2, 4). Hence, the conditions necessary for different steps of the speciation process do not necessarily coincide.

Interestingly, our model can also be interpreted in a different context, namely as a model for different geographical modes of speciation when there is selection for local adaptation. The learning of niche features plays no role in this interpretation. If the niches are not evenly distributed over space but rather are more or less separated areas, the mating preference and probability of egg-laying mistakes determine the geographical mode. The probability of egg-laying mistakes can in this context be interpreted as the migration probability between different areas at a young age. A large mating preference and low migration probability are synonymous to allopatric speciation. At the other extreme (low mating preference and high migration) the model is equal to a model for genetic divergence in sympatry. All situations in between represent the more general form of parapatric speciation. This alternative interpretation, on the one hand, nicely demonstrates the power of allopatric speciation in generating perfect assortative mating (see also Kirkpatrick and Ravigné 2002). On the other hand, our interpretation shows that the learning of niche features is an equally powerful mechanism of speciation. Whether speciation by the learning of niche features can be called sympatric speciation is a matter of definition. According to the commonly used definition that the sister species should evolve within the dispersal range of the offspring of a single deme, the speciation mechanism we study is beyond doubt sympatric. However, the definition of Kondrashov and Mina 1986 restricts it to cases where ''the probability of mating between two individuals depends on their genotype only.'' According to their definition, speciation by the learning of niche features is thus parapatric rather than sympatric.

The learning of niche features occurs in many animal species (see introduction), and in principle it is possible to estimate parameter values for all these examples. However, in most cases such detailed information is not available. Indeed, the goal of our modeling approach was not to render a detailed description of the speciation process in any specific example, but rather to examine for what type of circumstances the mechanism can work. This approach allows us to to draw general conclusions. As an example, we look in more detail at the case of the brood-parasitic finches of the genus *Vidua*, our source of inspiration. Observations and experimental work by Payne and coworkers allows a very rough estimate of some of the parameters. First, the probability of mistakes in egg laying is very low in viduid finches, presumably approximately 1% (estimated from males found in the field that sing the wrong song in comparison with their appearance; Payne 1973b). Second, hybridization between different species of brood-parasitic finches is known to occur occasionally but is very rare (Payne 1980). Indeed, the strength of the song-based mating preference of females is large: when measuring female response to the playback of songs of males raised by different foster species, females preferred the songs that resembled those of their foster father in almost all cases (Payne et al. 2000). In the field the mating preference will probably even be a lot stronger as it can then be based on other cues as well (e.g., plumage characteristics). However, at the time of the colonization process no additional cues except song are available to the females to make a distinction between males raised by either of the two foster species. Finally, foster parents distinguish between their own chicks and parasitic chicks on the basis of their mouth patterns and feed chicks with mismatching mouth patterns less often. However, the selective disadvantage for these chicks is not very high (Payne et al. 2001). At the start of a colonization process, selection against odd-looking young is likely to be absent, at least in hosts that have not been parasitized before

and therefore did not yet evolve defense behaviors. Hence, during the initial stages of colonization of new hosts, the brood parasites can split into two cultural species very quickly. Only after hosts have evolved ways to discriminate between their own and parasitic young can genetic divergence between the culturally different brood parasites evolve in response.

Considering the above described parameter estimates for the contemporary Viduidae, our results confirm that the suggested speciation mechanism could be occurring. However, it is probable that circumstances for ancestors of the viduids were different, and the learning of the foster species' song may not always have occurred (see Sorenson and Payne 2001, 2002; Beltman et al. 2003). Hence, the parameter values are likely to have evolved to the present-day values at a relatively late evolutionary stage. Our results show that for many parameter combinations the initial steps of speciation do not occur. Hence, during the whole evolutionary history of the Viduidae, colonization of new foster species may often have failed or shifts toward the parasitization of other hosts may have occurred. Additionally, the presence of host defenses against brood parasitism may have influenced these processes. Defense behaviors may be present in hosts that have been parasitised before, either by other brood-parasitic species or by conspecific nest parasites. This would influence viability of the parasitic young, and our results show that this plays a crucial role in the initial steps of colonization and genetic divergence.

There are several differences in assumptions between our approach and that of other models on speciaton. One factor that we did not consider is the cost of female choosiness. This cost is usually assumed to be constant (Pomiankowski 1987; Higashi et al. 1999; Hall et al. 2000; Takimoto et al. 2000), but alternatively, and in some cases probably more realistically, it is assumed to depend on the frequency of the preferred males (Janetos 1980; Gavrilets and Boake 1998; Beltman et al. 2003). Both variants of costly female choosiness probably make speciation less likely. We expect that the former of the implementations has a uniform impact across all parameter combinations, whereas the latter only has an effect when preferred males are initially rare (as in Beltman et al. 2003).

Many studies have previously investigated the invasion of empty niches explicitly (Ludwig 1950; Rosenzweig 1978; Pimm 1979; Wilson and Turelli 1986; Kawata 2002) or the origin and maintenance of genetic variation between lineages using different niches (Maynard Smith 1966; Christiansen 1975, 1985; Felsenstein 1981; Rice 1984; Johnson et al. 1996; Day 2000). In these models, as well as in most other models of speciation, characteristics such as mating preferences or habitat preferences are genetically determined. Indeed, the most important aspect distinguishing this study from others is that learning influences both which matings occur and in which niche the young are produced. The idea that the learning of habitat or niche features may lead to speciation is not new (e.g., Maynard Smith 1966; Rice 1984; Schilthuizen 2001, p.138). However, in our view this phenomenon has not received the attention it deserves, and, to our knowledge, the present study is the first to investigate it theoretically. The learning of niche features influencing mating behavior and location of young production is known to occur in animals as diverse as birds, fish, and insects. Whether it is the rule rather than the exception is difficult to say, because experiments are not usually designed to investigate this question. We believe that, once given more attention both theoretically and experimentally, learning will turn out to play a crucial role in the formation of new species.

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APPENDIX

The recurrence equations for the model with density dependence acting at an earlier stage in development than viability selection are:

$$N_1' = \frac{E}{4D_a}\{(1-p)(N_a+\alpha N_b)[\alpha(2N_1+N_2)^2 + (2N_1+N_2)(2N_4+N_5)] + p(\alpha N_a+N_b)[\alpha(2N_4+N_5)^2 + (2N_1+N_2)(2N_4+N_5)]\}, \quad \text{(A1)}$$

$$N_2' = \frac{Ew_2}{2D_a}\{(1-p)(N_a+\alpha N_b)[\alpha(2N_1+N_2)(N_2+2N_3) + (N_2+2N_3)N_4 + N_aN_5 + (2N_1+N_2)N_6] + p(\alpha N_a+N_b)[\alpha(2N_4+N_5)(N_5+2N_6) + (N_2+2N_3)N_4 + N_aN_5 + (2N_1+N_2)N_6]\}, \quad \text{(A2)}$$

$$N_3' = \frac{Ew_3}{4D_a}\{(1-p)(N_a+\alpha N_b)[\alpha(N_2+2N_3)^2 + (N_2+2N_3)(2N_6+N_5)] + p(\alpha N_a+N_b)[\alpha(2N_6+N_5)^2 + (2N_3+N_2)(2N_6+N_5)]\}, \quad \text{(A3)}$$

$$N_4' = \frac{Ew_4}{4D_b}\{(1-p)(\alpha N_a+N_b)[\alpha(2N_4+N_5)^2 + (2N_1+N_2)(2N_4+N_5)] + p(N_a+\alpha N_b)[\alpha(2N_1+N_2)^2 + (2N_1+N_2)(2N_4+N_5)]\}, \quad \text{(A4)}$$

$$N_5' = \frac{Ew_5}{2D_b}\{(1-p)(\alpha N_a+N_b)[\alpha(2N_4+N_5)(N_5+2N_6) + (N_2+2N_3)N_4 + N_aN_5 + (2N_1+N_2)N_6] + p(N_a+\alpha N_b)[\alpha(2N_1+N_2)(N_2+2N_3) + (N_2+2N_3)N_4 + N_aN_5 + (2N_1+N_2)N_6]\}, \quad \text{(A5)}$$

and

$$N_6' = \frac{E}{4D_b}\{(1-p)(\alpha N_a+N_b)[\alpha(2N_6+N_5)^2 + (2N_3+N_2)(2N_6+N_5)] + p(N_a+\alpha N_b)[\alpha(N_2+2N_3)^2 + (N_2+2N_3)(2N_6+N_5)]\}, \quad \text{(A6)}$$

where

$$D_a = [1 + EK((1-p)N_a + pN_b)](\alpha N_b + N_a)(\alpha N_a + N_b), \quad \text{(A7)}$$

$$D_b = [1 + EK(pN_a + (1-p)N_b)](\alpha N_b + N_a)(\alpha N_a + N_b), \quad \text{(A8)}$$

$$N_a = N_1 + N_2 + N_3, \quad \text{and} \quad \text{(A9)}$$

$$N_b = N_4 + N_5 + N_6. \quad \text{(A10)}$$