Early learning and speciation : the effects of early experience on sexual and aggressive behaviour in Lake Victoria cichlid fish

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Chapter 2

Female mate choice behavior and sympatric speciation.

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Abstract

Many models have investigated how the process of speciation may occur in sympatry. In these models individuals are either asexual or mate choice is determined by very simple rules. Females, for example, may be assumed either to compare their phenotype to that of a potential mate, preferring to mate with similar males ("phenotype matching"), or to possess "preference genes" which determine which male phenotype they prefer. These rules often do not reflect the mate choice rules found in empirical studies. In this paper, we compare these two modes of female choice with various types of sexual imprinting. We examine the efficacy of different mate choice behavior in causing divergence in male traits under simple deterministic one-locus population genetic models as well as under polygenic, individual based simulations based on the models of Dieckmann and Doebeli (1999). We find that the inheritance mechanism of mate choice can have a large effect on the ease of sympatric speciation. When females imprint on their mothers, the result of the model is similar to phenotype matching, where speciation can occur fairly easily. When females imprint on their fathers or imprint obliquely, speciation becomes considerably less likely. Finally, when females rely on preference genes, male trait evolution occurs easily, but the correlation between trait and preference can be weak, and interpreting these results as speciation may be suspect.

Key words: cultural evolution, female preference, phenotype matching, sexual imprinting, sympatric speciation.

Introduction

Using mathematical models to explore complex evolutionary phenomena always requires a number of simplifying assumptions. Many sympatric speciation models, for example, include a simple behavioral basis of assortative mating. In some models, mate choice is based upon phenotypic matching between the same trait in males and choosy females (Dieckmann & Doebeli 1999). In others, mate choice depends upon a match between an inherited female preference and a separate male trait (i.e. Kondrashov & Kondrashov 1999). Whether or not traits (or traits and preferences) match is simply a function of the difference between their phenotypic values. These simplified models can help to determine whether sympatric speciation is possible under a variety of circumstances.

We know, however, that assortative mating in nature is often not a simple matter of matching phenotypes. Learning, or sexual imprinting, often influences the mating choices of females. Some early work on the impact of learning on the divergence of populations had conflicting outcomes, and used assumptions now found to be unlikely or rare, such as male dominated mate choice and absolute preferences (Seiger 1967; O'Donald 1960; Kalmus & Smith 1966). A number of recent papers have stressed the importance of investigating how imprinting processes may have influenced evolution at various levels (Aoki et al. 2001; Laland 1994; Owens et al. 1999; ten Cate & Bateson 1988; Weary et al. 1993). Here, we will assess how assumptions about the ontogeny of female mating preferences influence models of sympatric speciation. The goal is not to explore every facet of female behavior, or to create a necessarily realistic model of sympatric speciation, but to broadly demonstrate how certain simple assumptions may influence conclusions drawn about sympatric speciation.

In our most basic model of female behavior, "phenotype matching", we assume that females prefer to mate with a male that shares their trait (we use this term as shorthand for "self-referent phenotype matching"). Although there is limited evidence for this exact mechanism (for review see Hauber & Sherman 2001), it could occur if females assess their own phenotype and use it as a basis for mate choice. Juvenile brown headed cowbirds, for example prefer to associate with adults that have the same feather colour and vocalisations as themselves, even though they have never seen a conspecific individual (Hauber et al. 2001). Likewise, females in the Australian frog (*Uperoleia rugosa*) prefer to mate with males that are about 70% (range 65 - 79 %) of their own body weight; correspondingly, clutches are only successful when the male is within 64 – 80 % of the females bodyweight (Robertson 1990). A similar mechanism of phenotype matching has also been invoked in the sympatric speciation of fish in Cameroonian volcanic lakes (Schliewen et al. 1994; Schliewen et al. 2001). In addition to self-reference, phenotype matching could also occur if females differentially aggregated with or encountered males with a similar phenotype to their own (dependent on the particulars of encounter probabilities). One final possible mechanism that would result in this mating pattern is a pleiotropic effect of genes on the mating trait and the mating preference. The general mechanism of phenotype matching forms the basis of assortative mate choice in the models of Kondrashov and Kondrashov (1999) and Dieckmann and Doebeli (1999). We examine several variants of this basic model.

The first three variants assess the effect of sexual imprinting. Females can 1) imprint on the phenotype of their mothers, 2) imprint on the phenotype of their fathers, or 3) imprint obliquely on members of the population at large. Although these three patterns differ in whom females learn from, they might entail somewhat similar social learning mechanisms. Evidence for sexual imprinting has been found in a wide variety of animals, mostly in birds and mammals (ten Cate & Vos 1999). Few studies of parental imprinting, however, have determined which parent the young imprint upon. Imprinting on the maternal phenotype has been shown in species where only females provide care for their offspring, such as in various geese and duck species (e.g. Kruijt et al. 1982). In a study on imprinting on novel traits, Witte et al (2000) also showed that both sexes of the Javanese Mannikin (*Lonchura Leucogastroides*) imprint on the maternal phenotype, rather than on the paternal phenotype. Less evidence exists that female offspring imprint on their fathers. Weisman et al. (1994) found that female zebra finches (*Taeniopygia guttata*) imprint on their fathers' beak color if the parents were not alike. Female zebra finches also were found to sexually imprint on novel traits of their father, but not their mother (Witte & Sawka 2003). However, another study also showed evidence for maternal imprinting by females in this species (Vos 1995). The scarcity of evidence for a paternal imprinting mechanism in female offspring probably reflects a dearth of studies investigating this phenomenon (Vos 1995). Oblique imprinting also appears somewhat uncommon, and has received little attention in classical imprinting studies. However, a recent study on wolf spiders (Hebets 2003) provides some evidence that this mechanism is found in nature. In this study sub-adult female wolf spiders exposed to a sexually active male with a certain phenotype later biased their adult mate choice towards this phenotype. The phenomenon of oblique imprinting also bears some resemblance to the processes of mate choice copying, in the sense that females obtain their preference from an unrelated individual in the population. Mate choice copying has been found in species including guppies (*Poecilia reticulata*) (e.g. Dugatkin & Godin 1992) and mollies (*Poecilia latipinna*) (Witte & Noltemeier 2002).

In the final variant, we consider a model where assortative mating is based on specific female preference alleles. Here females with one preference allele prefer a certain trait, while females with an alternative preference allele prefer another trait. In order to conclusively prove that female preferences work this way in a certain system, one would have to assess the genetic basis of preferences and/or rule out that preferences are based on imprinting or assessment of the female's own phenotype; few studies go to these lengths. Genetic variation in preferences that strongly suggests this type of genetic control has, however, been found in a variety of taxa, including Drosophila (e.g. Noor et al. 2001), swordtails (*Xiphophorus cortezi*) (Morris et al. 2003), and crickets in the genera Ephipigger and Laupala (e.g. Ritchie 2000; Shaw 2000).

We investigate the effect of preference inheritance on sympatric speciation in two ways. First, each mode of preference behavior is analyzed in a simple population genetic model. Secondly, we implemented each behavior in individual based simulation models based on those developed by Dieckman and Doebeli (Dieckmann & Doebeli 1999; Doebeli & Dieckmann 2000).

We find that these behavioral details of preference inheritance can have a profound effect on the chance of sympatric speciation. The ‘phenotype matching’ model is most favorable to speciation, under both modeling methods. The results of the variant where females imprint on their mothers are similar to those of phenotype matching. However, when females imprint on their fathers, conditions for sympatric speciation become more stringent. Furthermore, we find that populations with oblique imprinting are incapable of sympatric speciation. Finally, when female choice depends on preference genes the male trait may evolve easily, but the correlation between the trait and preference is not always very strong. We discuss the differences between the model variants and the implications for sympatric speciation.

The Models

In all of our models, we assume that males express a genetically transmitted trait that is used as a mating cue by females. The different inheritance mechanisms of mating preferences (whether learned or genetic) for this mating cue define the differences between the models. We examine the impact of these differences on the likelihood of speciation via two different approaches.

The first approach is to construct a very simple population genetic model that incorporates the female choice behavior. We are interested in the degree of facilitation of sympatric speciation by various forms of mate choice, and not in the various sources of selection and population dynamics that may produce and maintain two newly speciated populations in sympatry. Furthermore, recent work shows that sexual selection alone is unlikely to drive sympatric speciation under most conditions (Arnegard & Kondrashov 2004). We therefore simply assume that there is ecologically based negative frequency dependent selection on a male mating character that maintains the necessary genetic variation in a speciating population. Our analysis of the resulting models allows us to make some general conclusions about the influence of the different mate choice behaviors. The second approach that we use in the paper is to implement more complex individual-based simulation models of the evolution of reproductive isolation based on Dieckmann and Doebeli (1999), one of the more successful recent attempts to model sympatric speciation. In these models, individuals’ mating traits are determined by a finite number of diploid loci, acting additively.

These two approaches examine the same preference inheritance mechanisms, however they ask slightly different questions about the speciation process. In the analytical approach, for example, we assess speciation by examining the relative stability of a polymorphism in the mating trait and by determining the degree of non-random mating in the population. In the simulation approach, on the other hand, we study speciation occurring as bifurcation of the population based on the mating trait.

Analysis of Population Genetic Models

The models describe evolution at a single trait locus by sexual selection in very large populations. We assume individuals are haploid; on the basis of simulations, we believe that this assumption does not qualitatively affect our conclusions regarding comparisons between the models. We also assume that all females mate once, and that generations are discrete and non-overlapping.

Females mate according to their preferences, based on our specific models of female behavior. We examine variation at the trait that acts as a mating cue, which is expressed in both sexes. The mating trait is controlled by locus T with alleles T1 and T2. We form three recursion equations for each model (details are presented in models 1-4 below): 1) the change from one generation to the next in the frequency of trait T1, 2) the change in the frequency with which females prefer T1, which we denote by Tx1, and 3) the change in D. D is a measure of disequilibrium between the locus T and the female preference for T. In other words, it indicates the level of association between preferences and traits. It is analogous to, and calculated in the same way as, the more familiar linkage disequilibrium between two genetic loci (used in model 5 below), or the "gene-culture disequilibrium" of Feldman & Cavalli-Sforza (1984).

As stated above, sexual selection alone is unlikely to drive sympatric speciation. These simple models are unable to result in a stable polymorphism for the trait with only divergent sexual selection as driving force. We therefore incorporate negative frequency dependent natural selection, *s*, to maintain genetic variation at this mating trait. This parameter selects against the mating trait, T1 or T2, that has a larger frequency than 1/2 in the total population. We will use the strength of s needed to maintain this polymorphism as one of the measures to compare the relative ease with which each assortative mating regime maintains two incipient species. Frequency dependent natural selection affects the frequencies of T1, Tx1 and D in models 1-4 after the formation of the zygotes, but before sexual reproduction, as follows:

$$t_1^* = t_1 \frac{1-2s(t_1-1/2)}{1-4s(t_1-1/2)^2} \qquad [1a]$$

$$t_{x1}^* = t_{x1} + D\frac{2s(1-2t_1)}{1-s(1-2t_1)^2} \qquad [1b]$$

$$D^{*} = D\frac{1-2s^{2}(t_{1}-1/2)^{2}}{(1-4s(t_{1}-1/2)^{2})^{2}}$$

[1c]

Where t_1 is the frequency of T1 individuals before selection at time t and $t_1{}^*$ is their frequency after selection; t_{x1} is the frequency at which trait T1 is preferred and $t_{x1}{}^*$ is that frequency after selection; D* is the disequilibrium between $t_1{}^*$ and $t_{x1}{}^*$.

Model 1: phenotype matching

This model, over the others in this study, bears most resemblance to several earlier models of speciation (Maynard Smith 1966; Udovic 1980; Kondrashov & Kondrashov 1999; Dieckmann & Doebeli 1999; Servedio 2000). Here a female's preference is determined by comparing her own expression of the trait T with the expression of that same trait in a potential mate. Thus T1 females prefer to mate with T1 males over T2 males by a factor $1+\alpha$ (and likewise, T2 females prefer to mate with T2 males over T1 males by the same amount). Here the preference specifically defines how much more likely a female would be to mate with one type of male over another type if she were to encounter one of each. Mating occurs assortatively, according to the described preferences above, resulting in the following recursion equations for the frequency of T1 and the preference for T1 at time t+1:

$$t_{1}(t+1) = t_{1}^{*} + \frac{\alpha t_{1}^{*} t_{2}^{*}\left(t_{1}^{*}-t_{2}^{*}\right)}{2\left(1+t_{1}^{*}\alpha\right)\left(1+t_{2}^{*}\alpha\right)}$$

[2a]

$$t_{x1}(t+1) = t_{1}(t+1)$$

[2b]

Table 1. Mating table for models 2-4: imprinting on mother, father and oblique imprinting models. Females are separated by trait and preference, where T12 is a female bearing trait T1 and preferring to mate with a male with trait T2. The matings are normalised for each female gene-preference combination, ensuring that each female mates once.

	Males	
Females	T1	T2
T11	$\frac{t_1^* t_{11}^* (1+\alpha)}{1+t_1^*\alpha}$	$\frac{t_2^* t_{11}^*}{1+t_1^*\alpha}$
T12	$\frac{t_1^* t_{12}^*}{1+t_2^*\alpha}$	$\frac{t_2^* t_{12}^* (1+\alpha)}{1+t_2^*\alpha}$
T21	$\frac{t_1^* t_{21}^* (1+\alpha)}{1+t_1^*\alpha}$	$\frac{t_2^* t_{21}^*}{1+t_1^*\alpha}$
T22	$\frac{t_1^* t_{22}^*}{1+t_2^*\alpha}$	$\frac{t_2^* t_{22}^* (1+\alpha)}{1+t_2^*\alpha}$

The recursion for the frequency of the preference for T1, t_{x1} is the same as the recursion for t_1, since each individual has a preference for its own trait by definition. Disequilibria between the preference and the trait are calculated in a way analogous to genetic disequilibrium (e.g., D = $t_{11}t_{22} - t_{12}t_{21}$ where t_{21} is the frequency of individuals with trait T2 and preference Tx1). The value of D for phenotype matching will be t_1t_2. This is a trivial consequence of the fact that under phenotype matching t_{12} and t_{21} are 0 by definition; the relevance of D will become apparent in comparison with the other models.

The following three models, where females imprint on their mother, father, or obliquely on males in the parental generation, are modifications of the phenotype matching model. In all three of these models, each phenogenotype of preference and trait is affected through selection on the trait alone. The only difference between the models is the subject of sexual imprinting for the females.

Model 2: maternal imprinting

In this model, a female's preference for either T1 or T2 is determined by the phenotype of her mother. Females that had a mother bearing the T1 trait will prefer to mate with a T1 male over a T2 male by a factor 1+α, while females with a mother bearing T2 would prefer to mate with a T2 male by the same factor. Table 1 shows how the frequencies of the crosses are determined. In the appendix, section 1, we show which cells of the table contribute to each recursion equation. The resulting recursion equations are:

$$t_1(t+1) = t_1^* + \frac{1}{2}F \quad , \qquad [3]$$

where

$$F = \frac{\alpha t_1^* t_2^*}{(1+t_1^*\alpha)(1+t_2^*\alpha)}\left(t_{x1}^* - t_{x2}^* + \alpha(t_{x1}^* - t_1^*)\right) \quad . \qquad [3a]$$

$$t_{x1}(t+1) = t_1^* \qquad [4]$$

$$D(t+1) = \frac{t_2^* t_1^* (1+\alpha+2\alpha D^* + \alpha^2 (D^* + t_1^* - t_1^{*2}))}{2(1+t_1^*\alpha)(1+t_2^*\alpha)} \qquad [5]$$

Here the number of females preferring T1 is simply the frequency of mothers with T1.

Model 3 Paternal Imprinting

Female preferences for either T1 or T2 are determined by the phenotype of the father, where a female with a T1 father will prefer to mate with a T1 male over a T2 male by a factor $1+a$. This model is otherwise the same as model 2. The change in the ontogeny of the female preference results in different recursion equations for t_{x1} and D, while the expression for t_1(t+1) remains the same as in model 2, equation [3] (see appendix section 1).

$$t_{x1}(t+1) = t_1^* + F \tag{6}$$

$$D(t+1) = \frac{1}{2}\left(t_1^* \left(\frac{(1+\alpha)(t_1^* t_{x1}^* + D^* + t_{x1})}{1+\alpha t_1^*} + \frac{t_{x2}^*(1+t_1^*) - D^*}{1+\alpha t_2^*} - 2t_1^* - 3F \right) - F^2 \right) \tag{7}$$

In equations [6] and [7], F is the same expression given in equation [3a]. This difference between model 2 and model 3 stems from the fact that fathers, unlike mothers, have unequal mating success, so t_{x1}(t+1) attains the more complicated form seen here.

Model 4: oblique Imprinting

Here, females 'imprint' obliquely on the phenotype of the males of the previous generation. In other words, the chance that a female will have a preference for trait T1 is proportional to the frequency of that trait in her parents' generation. There are two plausible biological scenarios that might cause this: either females choose (or encounter) a random male from their parents' generation to imprint upon; or females simply choose whether they prefer T1 or T2 based on how common these phenotypes were in their parents' generation.

The recursion equations for t_1 and t_{x1} in this model are the same as in model 2 (see appendix section 1). The recursion for D is different, however. Because the chance that a female will imprint on T1 has no relation to the chance that she also bears this trait, D is always 0.

Model 5: genetic preference for mating trait

In this model, female preferences are based on a separate genetic locus P (similar to Kirkpatrick 1982). This preference locus has two alleles, P1 and P2, which correspond to a preference for either of the two male traits. Unlike Kirkpatrick (1982), and the models above, we analyze the simple situation where natural selection on males is absent. Negative frequency dependence is not necessary to maintain a polymorphism, as in the other models, because here there is a line of stable polymorphic equilibria even without selection. The strength of preference of P1 females for T1 males over T2 males is $1+a$, and vice versa, where the strength determines the likelihood of mating with the preferred male when one male of each type is encountered.

The frequencies of mating are determined following the mating table (table 2), which is a modification of the mating table in Kirkpatrick (1982, table 1). When individuals have mated, the genotype of their offspring is affected by the

	Males			
Females	T1P1	T1P2	T2P1	T2P2
T1P1	$\frac{x_1^2(1+\alpha)}{1+t_1^*\alpha}$	$\frac{x_1x_2(1+\alpha)}{1+t_1^*\alpha}$	$\frac{x_1x_3}{1+t_1^*\alpha}$	$\frac{x_1x_4}{1+t_1^*\alpha}$
T1P2	$\frac{x_2x_1}{1+t_2^*\alpha}$	$\frac{x_2^2}{1+t_2^*\alpha}$	$\frac{x_2x_3(1+\alpha)}{1+t_2^*\alpha}$	$\frac{x_2x_4(1+\alpha)}{1+t_2^*\alpha}$
T2P1	$\frac{x_3x_1(1+\alpha)}{1+t_1^*\alpha}$	$\frac{x_3x_2(1+\alpha)}{1+t_1^*\alpha}$	$\frac{x_3^2}{1+t_1^*\alpha}$	$\frac{x_3x_4}{1+t_1^*\alpha}$
T2P2	$\frac{x_4x_1}{1+t_2^*\alpha}$	$\frac{x_4x_2}{1+t_2^*\alpha}$	$\frac{x_4x_3(1+\alpha)}{1+t_2^*\alpha}$	$\frac{x_4^2(1+\alpha)}{1+t_2^*\alpha}$

Table 2. Mating table for model 5: preference genes model.
The frequency of T1P1 after mutation is noted by x_1, T1P2 by x_2, T2P1 by x_3 and T2P2 by x_4. Matings are normalised so that each female mates once.

recombination rate, r, between the preference and the trait loci.

Modifying the analysis in Kirkpatrick (1982) to determine the recursion equations for t_1, p_1 and D, where D in this case is the traditional gene-linkage disequilibrium, and p_1 is the frequency of P1 individuals at time t, we find that

$$t_{1(t+1)} = t_1^* + \frac{1}{2}t_1^*(H-1) \qquad [8]$$

$$p_{1(t+1)} = (t_{1(t+1)} - t_1^*)\left(\frac{D}{t_1^*(1-t_1^*)}\right) \qquad [9]$$

$$D_{(t+1)} = D + \frac{1}{4}[G+H+GH-3] - \frac{1}{4}S[1+Gt_1^* + Ht_2^*] \quad , \qquad [10]$$

where

$$G = \frac{(\alpha+1)p_1^*}{1+t_1^*\alpha} + \frac{p_2^*}{1+t_2^*\alpha} \quad ,$$

$$H = \frac{p_1^*}{1+t_1^*\alpha} + \frac{(\alpha+1)p_2^*}{1+t_2^*\alpha} \quad \text{, and}$$

$$S = r\left[\left(\frac{2-\alpha}{1+t_1^*\alpha} - \frac{\alpha}{1+t_2^*\alpha}\right)\left(t_2^* t_1^* p_2^* p_1^* + D^2\right) + D\left(\frac{1}{1+t_1^*\alpha} + \frac{1}{1+t_2^*\alpha}\right)\left[\left(t_1^* p_1^* + t_2^* p_2^*\right) + \left(t_1^* p_2^* + t_2^* p_1^*\right)(\alpha+1)\right]\right]$$

Equilibria and Stability

We solved these models for their equilibria, on which we performed a local stability analysis. We were then able to compare the stability of the models by comparing the strength of the frequency dependent selection coefficient, *s*, that switches a polymorphic equilibrium, found in all models, from stable to unstable (bifurcation point; see appendix sections 2 and 3). This produces a curve (the "*s*-curve") for each model, expressing the switch point for *s* in terms of *α*. We used the level of disequilibrium, D, at these stable polymorphic equilibria as another indication of progress towards sympatric speciation. Models 1-4 all have several equilibria (see appendix), of which all models have three biologically relevant equilibria in common; two where one of the two trait alleles is extinct, and one where the frequencies are $t_1 = t_2 = ½$. However, the equilibrium level of disequilibrium, $\hat{D}$, at this polymorphic equilibrium differs between all models (table 3).

Model 5 behaves somewhat differently than the other models that we have introduced. Instead of one internal equilibrium, there is a line of stable equilibrium which is expressed by the following equations:

When: $\frac{1}{2+\alpha} < \hat{p}_1 > \frac{1+\alpha}{2+\alpha}$, $\hat{t}_1 = \hat{p}_1 \frac{2+\alpha}{\alpha} - \frac{1}{\alpha}$

Otherwise: $\hat{t}_1 = 0,\ \hat{p}_1 \le \frac{1}{2+\alpha}$ or $\hat{t}_1 = 1,\ \hat{p}_1 \ge \frac{1+\alpha}{2+\alpha}$

The minimum frequency dependent selection, *s*, required for the polymorphic equilibria to be stable depends differently on the strength of the preference, α, in all learning models. Increasing *s* makes the polymorphic equilibria more likely to be locally stable and the edge equilibria less likely to be locally stable. The maximum frequency dependent selection value, *s*, at which the edge equilibria are stable, however, is the same among these models. The equations of these relations are shown in table 3, and the corresponding curves are plotted in figure 1.

Because the *s*-curves for the edge equilibria are not identical to any of the *s*-curves for the polymorphic equilibra in the models, there is an area in each model where there are 3 stable equilibria. Numerical iterations have shown however, that in this area the range of t_1 for which the edge equilibria are locally stable is marginal and that the separatrix determining movement towards an edge equilibrium is close to the edge. This indicates that once the

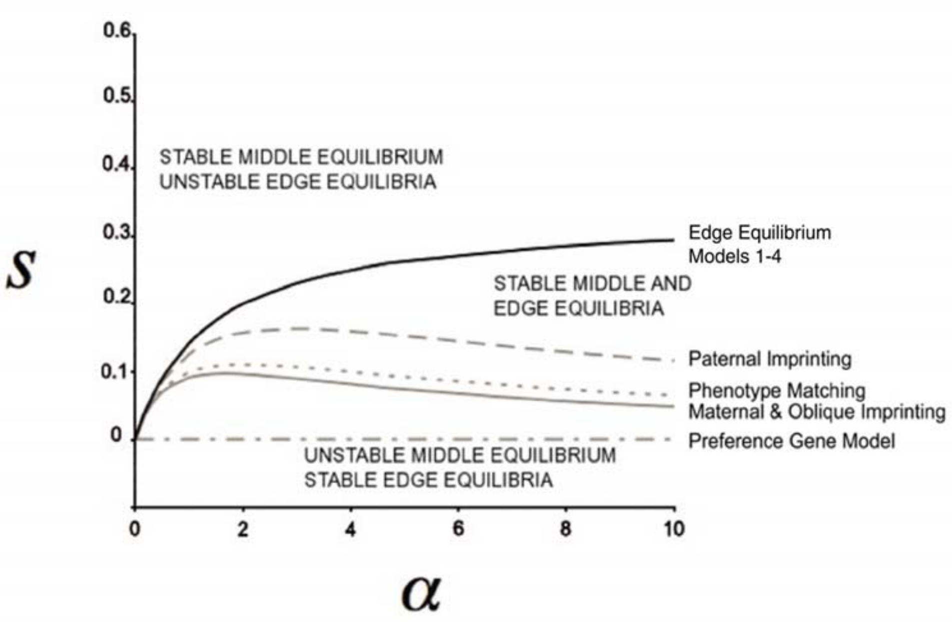


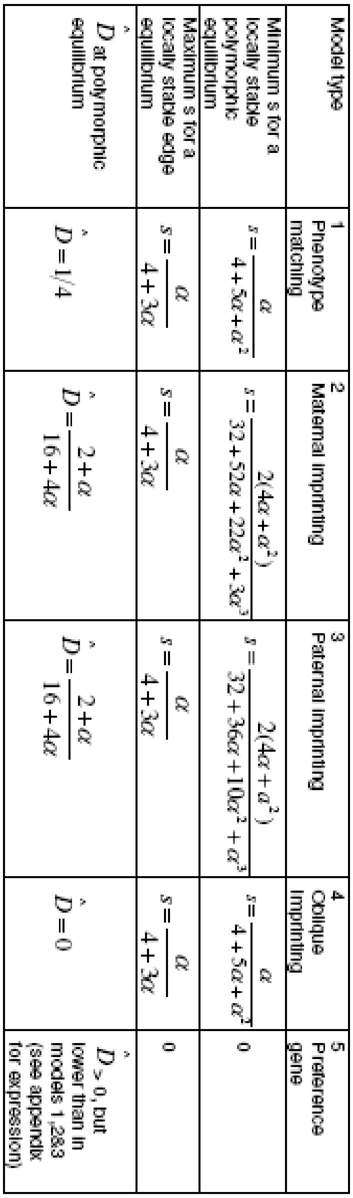
Figure 1. The dependence of *s*, the value of frequency dependent selection needed for each equilibrium point to be stable, on *α*, the strength of the preference for a certain trait. The middle equilibrium is the polymorphic equilibrium. All values of *s* above the gray line for each model will give a locally stable polymorphic equilibrium, while all values of *s* below the gray line will make the polymorphic equilibrium locally unstable. Models 1-4 have the same curve for the edge equilibria, which also shows the dependence of *s* on *α*, where here the curve shown is the maximum frequency dependent selection value for which the edge equilibria will be locally stable. The preference gene model has a line of stable equilibria without frequency dependent selection, and thus does not depend on *s*.

conditions for a stable polymorphic equilibrium are met, the models move towards the polymorphic equilibrium over most of the parameter space.

We concentrate our comparisons between the models on the stability of the polymorphic equilibrium because this shows the ability of each model to maintain two species in sympatry. The ability to maintain a polymorphism is both a prerequisite for speciation and potentially an important determinant of whether two incipient species can continue to co-occur. Stability of the polymorphic equilibrium is also particularly important in our model because of the relatively small area of the parameter space in which the population will evolve towards the edge equilibria. We therefore use the range of parameters for which the polymorphic equilibrium is stable as a measure of the ease of the generation and maintenance of speciation in sympatry.

Table 3. Summary of results of population genetics models.

The minimum s is the lowest value of the frequency dependent selection coefficient, s, required to maintain a polymorphism for the mating trait. See appendix for derivation of these equations. ‘D’ at equilibrium results in equal equations for maternal and paternal imprinting models. Outside this equilibrium point, however, the disequilibrium in the paternal imprinting model is lower than in the maternal imprinting model.



Model type	1 Phenotype matching	2 Maternal imprinting	3 Paternal imprinting	4 Oblique Imprinting	5 Preference gene
Minimum s for a locally stable polymorphic equilibrium	$s=\frac{\alpha}{4+5\alpha+\alpha^2}$	$s=\frac{2(4\alpha+\alpha^2)}{32+52\alpha+22\alpha^2+3\alpha^3}$	$s=\frac{2(4\alpha+\alpha^2)}{32+36\alpha+10\alpha^2+\alpha^3}$	$s=\frac{\alpha}{4+5\alpha+\alpha^2}$	0
Maximum s for a locally stable edge equilibrium	$s=\frac{\alpha}{4+3\alpha}$	$s=\frac{\alpha}{4+3\alpha}$	$s=\frac{\alpha}{4+3\alpha}$	$s=\frac{\alpha}{4+3\alpha}$	0
$\hat{D}$ at polymorphic equilibrium	$\hat{D}=1/4$	$\hat{D}=\frac{2+\alpha}{16+4\alpha}$	$\hat{D}=\frac{2+\alpha}{16+4\alpha}$	$\hat{D}=0$	$\hat{D}>0$, but lower than in models 1,2&3 (see appendix for expression)

Results of the population genetic models

As mentioned above, we considered two parameters of the models important in the assessment of the evolution of reproductive isolation in sympatry. The first of these is the tendency to maintain a polymorphism in the mating trait, which we assessed by measuring the strength of negative frequency dependent selection needed for local stability of the polymorphism. This selection prevents the positive frequency dependent selection created by mating from causing the population to converge on one mating trait. The second measure is the strength of disequilibrium between trait and preference. This is a critical factor, because without this association there would be one polymorphic population instead of two, reproductively isolated, emerging subpopulations or species. It is not immediately apparent how these two parameters trade off to determine whether speciation can be considered to be occurring, although we discuss the significance of these measures below.

In table 3 and figures 1 and 2 we have summarized the analytical evaluation of our models in terms of the trait-preference disequilibrium and the minimum amount of *s* required to produce an internal equilibrium.

The preference gene model (model 5) has the easiest conditions for a stable internal equilibrium, since in this model there exists a stable line of equilibrium between the loss and fixation of the allele T1. This stable internal equilibrium exists even without negative frequency dependent selection. This results from the fact that all females have equal mating success, regardless of their preference allele. Preference alleles can therefore be stable at any frequency, and a corresponding stable frequency of the trait allele will result. However, disequilibrium in this model is relatively weak, since an individual's preference is not directly related to its own trait phenotype. In comparison, the phenotype-matching model (model 1) obviously has a very high disequilibrium value, but on the other hand, requires more stringent conditions for stability.

Both the maternal and paternal imprinting models (models 2 and 3, respectively) have slightly lower disequilibrium values than does phenotype matching. As long as there is any interbreeding between mating types, either of an individual's parents might not possess the same mating trait as the individual itself. The disequilibrium in these models is still considerably higher than that in the preference gene model. The stability of the internal equilibrium in the maternal imprinting model is similar to that of the phenotype matching model, however, it requires slightly lower negative frequency dependent selection to be stable. Paternal imprinting on the other hand, seems to cause greater positive frequency dependence, and hence requires much stronger

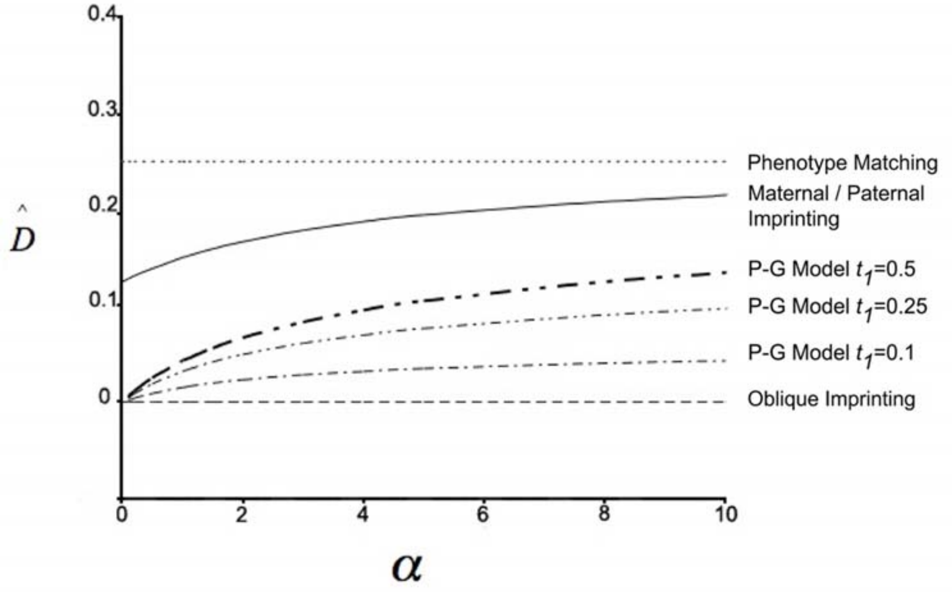


Figure 2. The level of preference trait disequilibrium at the polymorphic equilibrium, t_1=1/2. The phenotype matching model is at maximal disequilibrium. For the preference gene model three examples of disequilibrium along the equilbrium line are shown and their corresponding values of p_1 are given in the appendix, figure A-1.

negative frequency dependence for stability of its internal equilibrium (see figure 1).

The conditions for speciation are most stringent with oblique imprinting. Despite the relatively lax conditions for maintaining a stable internal equilibrium, in this model, disequilibrium cannot be established. We therefore consider oblique imprinting incapable of driving speciation.

Individual Based Simulations

The results of the analytical models above have several limitations. First, it is not obvious how the two factors of gene-preference disequilibrium and the stability of the polymorphic equilibria will trade-off to determine the ease of speciation. Second, the models are very simplified. For example, phenotypic differences between two species, including sexual signals, are often controlled by multiple genetic differences. Recent models with this level of complexity have used individually-based computer simulations (Dieckmann and Doebeli 1999, 2000), following earlier individual-based simulations of speciation (Kulagina & Lyapunov, 1966, Menshutkin, 1977, Kondrashov, 1980, 1986). We implemented an individual-based model of this type, based on a simplified version of the Dieckmann and Doebeli (1999, 2000) model. Individuals possessed 5 unlinked diploid loci (with alleles T and t). An individual's phenotype, x, is the sum of all the T genes it possesses. In other words, genetic interactions are strictly additive and the effects of all loci on the phenotype are equal. The mutation rate of these genes was 0.00001 per locus. All females mated once per "year". Females possessed a preference for one type of phenotype, y. This type of phenotype was either their own phenotype (phenotype matching, corresponding to model 1 above); the phenotypic output of their preference loci (corresponding to model 5 above); either their mother or father's phenotype (corresponding to models 2 and 3 above respectively); or the phenotype of a randomly selected male from the population (oblique imprinting, corresponding to model 4 above). In the case, similar to model 5 above, where females possessed separate preference loci, individuals also possessed 5 further unlinked, additive loci to determine the preference phenotype. The chance that a female would mate with a given male depended on the difference between her preferred phenotype and his phenotype. As this difference increased, the probability of mating declined according to a Gaussian distribution (Dieckman and Doebeli 1999), with variance a (a measure similar to a in the models described above). The preference, p, of a female with preferred phenotype yf for a male with phenotype xm was therefore:

$$p = e^{\frac{-(x_m + y_f)^2}{a^2}}$$

The preference of a female for a given male phenotype was normalized over the distribution of male phenotypes to ensure that all females mated, even if their preference was very different from the available male phenotypes (this has a conservative effect on speciation). In our models, we investigated different parameter values of a, but within a simulation, female preferences did not evolve. This was different from Dieckman & Doebeli's simulations, where female preferences evolved, but to a maximum of a=0.05, a value that was fixed in all the results they reported.

We modified Dieckman & Doebeli's model by removing ecological variation. The model incorporates overlapping generations, with all individuals facing the same risk of dying in each "year", irrespective of their phenotype. The mortality rate was determined by a simple frequency-dependent relation that ensured a relatively constant population size: rN/K . Here r is the birth rate, N is the population size, and K is a parameter of the carrying capacity of the population. r was set to 1, and K to 15000 by default, causing an equilibrium population size of around 5000 individuals.

The evolutionary trajectory of such a system can be followed. In the case of speciation, it is necessary that polymorphisms are maintained and that disequilibrium builds up between these loci such that some individuals have a preponderance of T alleles, while others have primarily t alleles, causing a bifurcation of phenotypes. From a phenotypic perspective, this results in reproductive isolation between the two groups of individuals. In this model, even without ecological competition, assortative mating can still cause bifurcation of a population into two subpopulations, as predicted by Shpak & Kondrashov (1999).

In the simulations we measured whether the population equilibrated in one of three states: fixation of the population on one genotype; or on a range of genotypes without reproductive isolation; or finally, fixation of the population on two (or more) reproductively isolated genotypes. Only the latter case counts as speciation.

At the beginning of each run of the simulation, each individual's genotype was determined at random (for each gene there was a 50% probability of acquiring the T allele). Female preferences were set to be the same as their own phenotype in the cases of imprinting as well as assortative mating; in the case of preference genes, they were allocated the same values as the signal genes (these assumptions were not critical since rapid association of trait and preferences developed in the models anyway). Simulations ran for a period of 1000 "years" (equivalent to approximately 300 generations). To establish the threshold value, we carried out 10 repetitions of each parameter setting.

Our simulations can be found online at http://website.leidenuniv.nl/~lachlanrf/Simulation.html.

Simulation Results

Whether speciation occurred depended on several variables in the model: larger population sizes and higher mutation rates increased the genetic diversity in the population and tended to facilitate speciation. Of more interest was the effect of the preference variance (a): how quickly a female's preference dropped off as a male's phenotype became more unlike her ideal phenotype. At high levels of this parameter, when individuals tended not to have a strong preference for their preferred phenotype, speciation was prevented since too much recombination between individuals with different phenotypes occurred. We therefore measured the maximum level of a that resulted in speciation, and used this as an assessment of how easily a given set of parameters led to speciation (a higher a-max represents more permissive conditions for speciation).

Speciation occurred under all modes of female preference behavior we examined, except for oblique transmission. In this latter case, no linkage can develop between preference and mating trait, as discussed in the population genetics section above. In the remaining four models of mate choice, however, bifurcation occurred under different parameter ranges and to different degrees. Speciation occurred particularly easily with phenotype matching (a-max=0.15). From an initial central range of phenotypes, the population evolved so that there were two sub-populations that existed at

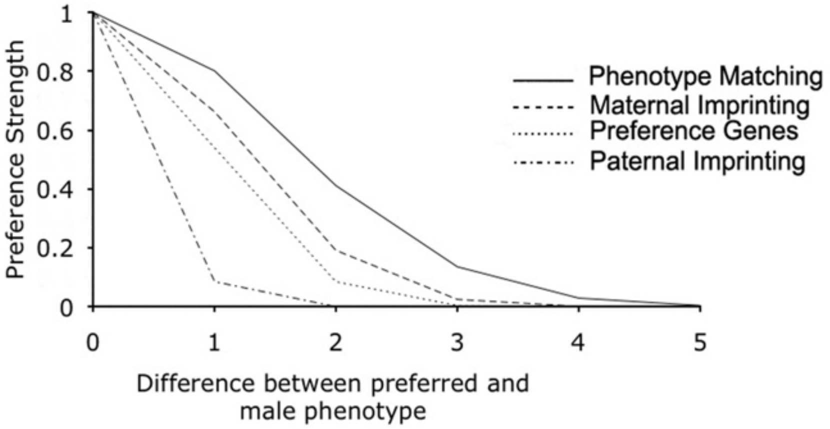


Figure 3. Shows the female preference functions at the speciation thresholds (amax) in the individual-based model. The more effective a type of female preference is at causing speciation, the weaker the female preference strength has to be to cause speciation. The figure shows the distribution of female preferences at the threshold values of a-max required to allow speciation. The y-axis values represent the strength of preference a female has for a given phenotype, relative to her preference for the most-preferred phenotype. For example if a female has to choose between two males, one with her most-preferred phenotype, and one with a preference score of 0.5, her probability of choosing the most-preferred phenotype is (1+0.5)/2=0.75. The x-axis shows the phenotype value of a male, in terms of how different to the most-preferred phenotype of the female.

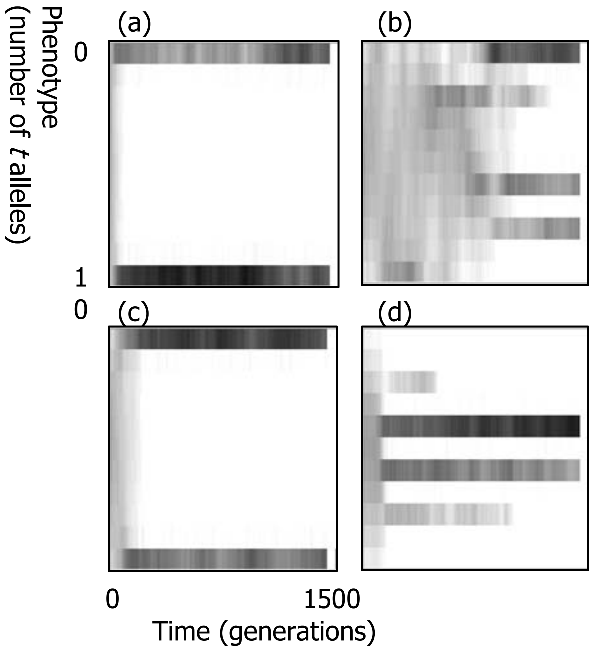


Figure 4. Representative runs of the simulation under the four mating modes (a: phenotype matching; b: preference loci; c: maternal imprinting; d: paternal imprinting). The x-axis represents time (the simulation proceeded for 1500 years); while the y-axis represents the value of the phenotype. The gray-scale represents how many individuals had that phenotype at that time (black = all individuals had that phenotype, white = no individuals had that phenotype). Under phenotype matching and maternal imprinting, bifurcation creates two subpopulations at extreme phenotype values, while different patterns are found with preference genes and paternal imprinting.

either extreme of the distribution, with all loci fixed for T in one sub-population and t in the other. When females imprinted on their mother there was a similar pattern of bifurcation, although the value of a-max, 0.11, was somewhat lower than for phenotype matching, translating into more stringent conditions for bifurcation (figure 3). When females imprinted on their fathers, however, there was a different pattern of bifurcation (figure 4), and a-max=0.045.

Rather than evolving to the extremes of the phenotypic space, when bifurcation occurred, populations evolved such that the two phenotypes were somewhat similar. In this case, there nevertheless tended to often be two genotypes fixed in the population, and each locus tended to lose variation. Finally, when females relied on preference loci to determine their mate choices, another pattern of bifurcation was observed (figure 4). Bifurcation did not lead to two subpopulations that were equally distant from the median phenotype; instead, the phenotypes seemed to be determined more or less at random. In this case too, a-max was considerably more stringent than for phenotype matching at 0.09.

Figure 3 shows how these values of a-max affect each model. With phenotype matching speciation occurred even if females were unable to clearly distinguish their most preferred phenotype from its neighbors in phenotype space. With maternal imprinting and preference loci the conditions for speciation are somewhat more restrictive. Under paternal imprinting, the conditions for speciation require that females nearly always mate with males whose phenotype matches their preference exactly. Finally, with oblique imprinting no speciation could occur at all.

Discussion

The main conclusion that can be drawn from our models is that the sympatric divergence of mating traits, determining the development of prezygotic isolation and hence the process of sympatric speciation, can be very sensitive to the behavioral basis of mate choice preference. The five different manners in which females acquired a mating preference all differed in how easily they maintained a polymorphism in the mating trait, in the linkage disequilibrium between preference and trait, and in how likely they were to bifurcate in a multilocus model. All three of these measures are likely to be important indicators of the potential to speciate.

Phenotype matching was our reference behavior, since this is the form of mate choice that most previous models incorporate (Maynard Smith 1966; Udovic 1980; Kondrashov & Kondrashov 1999; Dieckmann & Doebeli 1999; Servedio 2000). It is not clear how common this mechanism is in nature. It may be difficult, for example, for individuals of many species to assess their own color patterns or other mating cues. However, a review of the evidence for self-referent phenotype matching suggests that it may be a more common phenomenon than previously thought (Hauber & Sherman 2001). In the context of individual based simulations, this model facilitated ‘speciation’ the most, leading to bifurcation in the widest range of parameter values (see figures 3 and 4). By definition, the trait preference disequilibrium is always maximal in this model.

Maternal imprinting provided the closest approximation to phenotype matching. With phenotype matching, there is obviously a perfect match between a female’s preference and her own trait, while with maternal imprinting, the disequilibrium must be diluted to some extent. Therefore, the trait preference disequilibrium was slightly lower in the maternal imprinting model. Interestingly, however, the stability of the polymorphism was achieved with less help of negative frequency dependent natural selection (figure 1) in the maternal imprinting model than in the phenotype matching model. An explanation for this counterintuitive result lies in the difference between the two models in how the frequency of the female preference is determined. In the phenotype matching model, this is equal to the frequency of each trait in the current generation. In the maternal imprinting model, the frequency of the preference for a trait is equal to the frequency of that trait in the previous (parental) generation. This results in a time lag in the evolution of the preference distribution in the maternal imprinting model over that in the phenotype matching model. This in turn leads to an increase in the parameter range for stability of the polymorphic equilibrium, because the time lagging preference slows any movement of the population away from the equilibrium frequency.

In the individual based simulations, maternal imprinting also behaved most similarly to phenotype matching. Maternal imprinting is known to be present in birds (Kruijt et al. 1982; ten Cate & Vos 1999; Witte et al. 2000) and also occurs in a number of mammals (Kendrick et al. 1998), and is, to our knowledge, more prevalent than phenotype matching mechanisms. Its similarity to phenotype matching implies that the results of many previous phenotype matching models (e.g. Kondrashov & Kondrashov 1999; Dieckmann & Doebeli 1999) may apply to species that demonstrate maternal imprinting as well.

One of the principal results of this paper is that it matters greatly whom females imprint upon. In contrast to maternal imprinting, polymorphisms were less easily maintained with paternal imprinting. Correspondingly, bifurcation did not occur with paternal imprinting except under relatively extreme conditions in the individual based simulation. The difference between paternal and maternal imprinting in our models stems from the fact that all females had equal reproductive success, while males' reproductive success varied according to females' preferences. This meant that, with paternal imprinting, only successful males were imprinted upon, which increased the number of females preferring that type of male. This generates greater positive frequency dependence compared to the other models, which makes it difficult to maintain polymorphisms.

Finally, imprinting on unrelated males, unsurprisingly, made it impossible to establish prezygotic isolation; under these conditions no disequilibrium between trait and preference can ever develop. In summary, while phenotype matching is unlikely in reality, imprinting may cause similar evolutionary patterns, but only when females imprint on their own mothers.

Our final comparison was with a model incorporating preference genes. In this case, polymorphisms are maintained very easily indeed, because preferences are selectively neutral as long as all females have equal reproductive success (see Kirkpatrick 1982). On the other hand, recombination between the preference loci and the trait loci keeps the level of trait preference linkage disequilibrium in this model substantially lower than the trait - learned preference disequilibria in the sexual imprinting and phenotype matching models. It is unclear how to interpret the combination of these two factors in influencing the probability of speciation. We suggest, from the results of the individual based simulations, that speciation is considerably less likely with preference genes.

The ability of a system to maintain a polymorphism over time is a requirement for the formation of two distinct and isolated populations. The relative ability of a model to maintain a polymorphism is often seen in speciation models as the most important factor in influencing sympatric speciation. By contrast, trait-preference disequilibrium is seen as a less critical factor. Any disequilibrium at all that is measured in the analysis of such an equilibrium is interpreted as progress towards isolation of two incipient species, since it is assumed that further evolution of mate preferences will cause the disequilibrium to increase. In our deterministic models, we considered both the stability and the disequilibrium measures, and in comparing with the individual based models, it is clear that the level of disequilibrium does play a critical role. Even though there is significant disequilibrium in the preference gene model, it is less than in the phenotype matching model, and this translated to speciation, measured as bifurcation, occurring more readily with phenotype matching in the individual based models (figures 3 and 4).

The assumption that only females are choosy ensures that all females, unlike males, have equal reproductive success regardless of their phenotype or choice of mate. This in turn is important to the difference between the maternal and paternal imprinting models, as discussed above. In nature, females with an unusual preference may waste time, or may not even mate at all if they cannot find a suitable mate. A cost of having an unusual preference would make it less likely for all females to have equal reproductive success. This would reduce the difference in results from the maternal and paternal imprinting models. Males on the other hand, are not exerting a choice at all in our models. Mutual mate choice is another factor that could change the equal reproductive success of females, because a female with an unusual phenotype would have a reduced chance of finding a suitable mate. This last factor would affect the predictions for establishing a polymorphism, if we were to study how a new trait invading a population would fare. However, in our analysis we focus on the stability of an existing polymorphism, hence largely ignoring the dynamics associated with trait variation establishment.

Another aspect of speciation that was largely ignored in this paper is the evolution of the strength of mating preferences. This would probably affect our more detailed conclusions. A partially reproductively isolated population could become more isolated either by continuing divergence of the mating traits, or alternatively, by female preferences becoming more acute. In the case of the preference gene model (model 5 above), the mating traits did not always continue to diverge in the individual based simulations, because of the low trait-preference disequilibrium. This disequilibrium would have increased if the female preferences had been allowed to become stronger over time.

The mechanisms by which the females obtained their preferences were also not allowed to evolve in this study. We assumed that the evolution of these mechanisms is constrained. The pattern of occurrence of sexual imprinting in the phylogeny of birds suggests that imprinting is widespread, but the role it plays in the formation of sexual preferences varies (Immelmann 1975; ten Cate & Vos 1999). This may be an indication that the function of sexual imprinting can evolve, but the mechanism itself does not easily disappear. Furthermore, a few models show that learning of sexual preferences can indeed evolve (Servedio & Kirkpatrick 1996; Todd & Miller 1993), even without direct fitness benefit (Servedio & Kirkpatrick 1996).

Well-known examples of species that may have formed in sympatry are the cases of the haplochromine cichlids from the East African lakes. A long standing debate on the origin of the species richness in these lakes has recently focussed on sympatric speciation with sexual selection as a major driving force. Numerous sympatric species within a trophic group show remarkable radiation in color patterns, and there is growing evidence that many of these are the result of sympatric speciation (Seehausen et al. 1999; Allender et al. 2003; Albertson et al. 1999; Salzburger & Meyer 2004). Within these trophic groups often little ecological differentiation is found (Seehausen & Bouton 1997; Danley & Kocher 2001). Several experiments indicate the presence of disruptive sexual selection (Maan et al. 2004; Knight & Turner 2004; Seehausen & van Alphen 1998). For these cichlids it is largely unknown how their mate choice preferences are formed, however where tested their mate preference was found to be assortative.

In our analysis, we presume there is ample variation in mating traits by concentrating on the stability of a polymorphism. Radiation by hybridization may be one scenario that would provide this variation in traits. In a recent review, it was argued that hybridization could potentially even fuel adaptive radiation (Seehausen 2004). Hybridization of two species would create sudden (in only a few generations), broad variation in male traits in the new hybrid population. This broad variation may be a prerequisite for sympatric speciation, according to theory (e.g. Kondrashov & Kondrashov 1999; Dieckmann & Doebeli 1999; Higashi et al. 1999). The cichlid flock of Lake Victoria may have originated from a hybrid swarm that colonized the lake after a period of drought (Seehausen et al. 2003); the scenario of speciation by hybrid adaptive radiation has also been suggested for cichlid species from Lake Malawi (Smith et al. 2003). These aspects render our model potentially applicable to cichlid speciation.

Guppies are another example of species with a high degree of polymorphism in male color patterns. This polymorphism in nuptial coloration appears both between different populations and within certain populations, but does not appear to result in speciation. It has been shown that females' mate choice preferences in guppies are influenced by mate choice copying (Dugatkin & Godin 1992), although they appear to have a genetic basis for preference ranges as well (Brooks 2002). Mate choice copying has a crucial property in common with our model of oblique imprinting, in that it involves preference learning from an unrelated individual. This hinders the build up of any trait-preference disequilibrium, making divergence based on sexual selection nearly impossible as long as there is any degree of gene flow between populations.

In conclusion, we show that assumptions concerning the way females form their mate choice preferences can have potentially profound influences on the predictions for speciation. Although phenotype matching mechanisms have been used very often in general models of speciation, they are probably not common mechanisms in nature. Sexual imprinting, on the other hand, is quite widespread. We find that the type of sexual imprinting can affect speciation. While speciation occurs almost as readily in our maternal imprinting model as with phenotype matching, we find it much less likely to obtain with paternal imprinting. We also conclude that speciation cannot be driven by oblique imprinting. Finally, we find that the model with a genetic basis for mate choice preference behaved very differently from our other models, and may not always facilitate speciation. In nature, many species may have a mixture of these mechanisms to form their preferences, such as genetic predispositions to imprint on certain traits more than on others (Bolhuis 1996; ten Cate 1989). This study shows that the knowledge about how species form their preferences will improve our understanding of speciation processes.

Acknowledgments

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Appendix

1) Recursion equations models 2-4

The recursion equations in models 2-4 are determined using table 1. Which cells of the table make up each equation is explained below.
Individuals carrying trait T1 that have a preference for T2 individuals, are denoted by T12. Likewise, individuals with trait T2 and a preference for trait T1, are denoted by T21.

For each of the models:

$$t_1(t+1) = t_{11}(t+1) + t_{12}(t+1)$$

$$t_{x1}(t+1) = t_{11}(t+1) + t_{21}(t+1)$$

$$D(t+1) = t_{11}(t+1) * t_{22}(t+1) - t_{12}(t+1) * t_{21}(t+1)$$

Model 2: maternal imprinting

All T1 offspring from a T1 mother are T11 individuals. T2 offspring from a T1 mother are T21. Likewise, all T1 offspring from a T2 mother are T12 individuals and T2 offspring from a T2 mother are T22. Bracketed numbers indicate the corresponding cells [row, column] of table 1.

$$t_{11}(t+1) = [1,1] + [2,1] + \tfrac{1}{2}[1,2] + \tfrac{1}{2}[2,2]$$

$$t_{12}(t+1) = \tfrac{1}{2}[3,1] + \tfrac{1}{2}[4,1]$$

$$t_{21}(t+1) = \tfrac{1}{2}[1,2] + \tfrac{1}{2}[2,2]$$

$$t_{22}(t+1) = [4,2] + [3,2] + \tfrac{1}{2}[3,1] + \tfrac{1}{2}[4,1]$$

Model 3: paternal imprinting

All T1 offspring from a T1 father are T11 individuals. T2 offspring from a T1 father are T21. Likewise, all T1 offspring from a T2 father are T12 individuals and T2 offspring from a T2 father are T22.

$$t_{11}(t+1) = [1,1] + [2,1] + \tfrac{1}{2}[3,1] + \tfrac{1}{2}[4,1]$$

$$t_{12}(t+1) = \tfrac{1}{2}[1,2] + \tfrac{1}{2}[2,2]$$

$$t_{21}(t+1) = \tfrac{1}{2}[3,1] + \tfrac{1}{2}[4,1]$$

$$t_{22}(t+1) = [4,2] + [3,2] + \tfrac{1}{2}[1,2] + \tfrac{1}{2}[2,2]$$

Model 4: oblique imprinting

All T1 individuals, regardless of their parents' traits, may imprint on either a T1 or a T2 individual. The chance that a T1 individual imprints on trait T1 is dependent on the frequency with which this trait occurs in their parents' generation.

$$t_{11}(t+1) = t_1^*\Big([1,1]+[2,1]+\tfrac{1}{2}\big([1,2]+[2,2]+[3,1]+[4,1]\big)\Big)$$

$$t_{12}(t+1) = t_2^*\Big([1,1]+[2,1]+\tfrac{1}{2}\big([1,2]+[2,2]+[3,1]+[4,1]\big)\Big)$$

$$t_{21}(t+1) = t_1^*\Big([3,2]+[4,2]+\tfrac{1}{2}\big([1,2]+[2,2]+[3,1]+[4,1]\big)\Big)$$

$$t_{22}(t+1) = t_2^*\Big([3,2]+[4,2]+\tfrac{1}{2}\big([1,2]+[2,2]+[3,1]+[4,1]\big)\Big)$$

2) The equilibria of models 1-4

We were able to solve for the equilibria in the phenotype matching and the oblique imprinting model. The phenotype matching model has 6 equilibria for t_1, the oblique imprinting model has 7. Three of the equilibria of each of the models are mentioned in the main body of the paper (t_1=0, t_1=1/2, t_1=1). The others are expressed in terms of α and s. These will generate complex, negative values, as well as positive values for t_1, depending on how α and s are chosen. However, other than in a very small area of the parameter space discussed in the "Equilibria and Stability" section above, numerical simulations indicate there are no biologically relevant equilibria in these models other than the three already mentioned above.
We were not able to solve all of the equilbria of the maternal imprinting and paternal imprinting models. For maternal imprinting, there are at least 15 equilibria, and for paternal imprinting probably twice that number. However, these models also have the equilibria of t_1=0, t_1=1/2 and t_1=1, and simulations do not indicate the presence of any other biologically relevant equilibria.

3) Stability analysis of equilibria.

The stability of each equilibrium point was analysed with a linear stability analysis using Mathematica. The Jacobian matrix in each model was formed using the three recursion equations for t_1, t_{x1} and D. The leading eigenvalue of this matrix was then set equal to 1 (bifurcation point) and the resulting equation was solved for s, the level of positive frequency dependent selection that causes a switch of stability. This yielded an expression for s, with the strength of preference, α, as a second variable. These expressions were used to compare the relative strength of frequency dependent selection, as a proxy for the ease of speciation, necessary in each model to reach a stable polymorphic equilibrium (see table 3). The preference gene model does not include positive frequency dependent selection, so this analysis was not performed on that model.

4) D in the preference gene model at line of equilibria.

The genetic linkage disequilibrium in the preference gene model was calculated, following Kirkpatrick 1982, using our modifications to this model (absence of natural selection, and $\alpha1= \alpha2= \alpha+1$).

The equation for the $\hat{D}$ line is as follows:

$$\hat{D}=\frac{1}{2\alpha}(1+\alpha p_1+\alpha t_1-2\alpha p_1 t_1-\sqrt{1+2\alpha p_1+\alpha^2 p_1^2+2\alpha t_1-4\alpha p_1 t_1-2\alpha^2 p_1 t_1+\alpha^2 t_1^2})$$

Figure 2 shows the level of disequilibrium at three values of t_1 on the line of equilibria, dependent on α. The corresponding frequency of p_1 also depends on α, by the following equation:

$$p_1=\frac{1+t_1\alpha}{2+\alpha}$$

The curves for p_1 at three values of t_1 are shown in figure A-1.

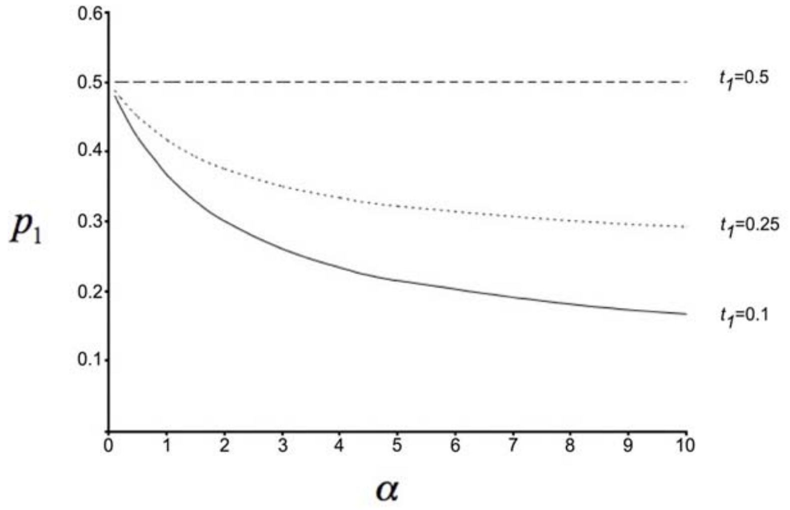

Figure A-1. Frequency of p_1 varying with the strength of preference, α, for three frequencies of t_1 on the line of stable equilibria.