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Sexual selection and sympatric speciation

van Doorn, Gerrit Sander

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Sexual Selection & Sympatric Speciation

G. Sander van Doorn

Sexual Selection & Sympatric Speciation

RIJKSUNIVERSITEIT GRONINGEN

Sexual Selection and Sympatric Speciation

PROEFSCHRIFT

ter verkrijging van het doctoraat in de
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aan de Rijksuniversiteit Groningen
op gezag van de
Rector Magnificus, dr. F. Zwarts,
in het openbaar te verdedigen op
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Sympatric speciation is like the measles;
everyone gets it, and we all get over it.

(attributed to THEODOSIUS DOBZHANSKY)

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General Introduction

How selection creates new species, and why it usually does not

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G. Sander van Doorn

"That Mystery of Mysteries"

Speciation – the origin of new species – is the source of the diversity of life. A theory of speciation is essential to link poorly understood macro-evolutionary processes, such as the origin of biodiversity and adaptive radiation, to well understood micro-evolutionary processes of allele frequency change due to selection and drift within single populations. Such is the importance of understanding speciation that many illustrious biologists have worked on the problem. The fact that this thesis, written nearly 150 years after Darwin published his famous book 'On the Origin of Species' (Darwin, 1859), deals mainly with speciation, indicates that these biologists failed to find a satisfying solution to the problem. Indeed, Darwin himself probably questioned his own ideas about speciation, since he referred to the birth of new species as "that mystery of mysteries" on several occasions. Darwin's puzzlement is understandable, since he lacked understanding of population genetics, an important pillar of the modern theory of evolution. In fact, at the time Darwin's book was published, Mendel was about to discover the laws of inheritance. However, at present, "speciation still remains a major challenge faced by evolutionary biologists" (Gavrilets, 2003), and more than 50 years after the 'Modern Synthesis' of Darwinian evolution with Mendelian genetics (e.g., Fisher, 1930; Dobzhansky, 1937), Kirkpatrick and Ravigné (2002), in their recent review on speciation, address "why we still lack coherent understanding of speciation in terms of population genetic principles".

WHY UNDERSTANDING SPECIATION REMAINS A CHALLENGE

In defense of the numerous biologists that worked on speciation, I should say that there are several good excuses for our poor understanding of the issue. I mention four, which all are of relevance to the work presented in this thesis:

SPECIATION IS INHERENTLY COMPLEX

Speciation involves (ecological) diversification accompanied by the evolution of reproductive isolation. Diversification often relies on disruptive selection, a type of selection that is inherently more difficult to understand from an ecological perspective than directional or stabilizing selection. To address the evolution of reproductive isolation, speciation theory is forced to deal explicitly with sexual reproduction and all the genetic intricacies it involves.

Hence, speciation is contingent on the interaction of ecological and genetic processes, which are already difficult enough to understand in isolation. Moreover, both may depend on many different factors. Each individual study on speciation necessarily can only address a few of these factors. As a consequence, the literature on speciation is fragmented into a large number of contributions that each focus on a highly specific scenario. The field is waiting for a unification of these separate contributions (Kirkpatrick and Ravigné, 2002), but this has proven to be difficult.

Even to the extent that there is consensus as to the basic processes involved in speciation (selection, genetic drift, spatial isolation and chance), important controversies revolving around the relative importance of these factors and their interactions remain to be solved. A thorough understanding of these issues is crucial to answer fundamental questions about biodiversity. For example, different opinions on the relative importance of these different factors in speciation, may lead to widely different theories for patterns of species richness and abundance (Hutchinson, 1959; Felsenstein, 1981; Hubell, 2001).

THE SPECIATION DEBATE FOCUSES MORE ON STRUCTURES THAN ON PROCESSES

Historically, the speciation debate is organized around the geographical context of speciation. This is reflected by the primary classification of speciation theories along a spectrum ranging from allopatric speciation (speciation in geographically separated populations) to sympatric speciation (speciation within spatially well-mixed populations). Proponents of the early allopatric theory (Mayr, 1942) fervently defended the view that speciation required externally imposed geographical barriers to gene flow, thereby attributing a prominent role to chance events and genetic drift. Proponents of the early sympatric theory, however, maintained that reproductive isolation could evolve from scratch within spatially well-mixed populations, without any pre-existing barriers to gene flow (Bush, 1994). Sympatric speciation models traditionally rely on selection as a cause of divergence.

Although the dichotomy based on geographic speciation modes traditionally coincided with one based on speciation mechanisms, this is nowadays no longer the case. Models of sympatric speciation by genetic drift have been published (e.g., Nei *et al.*, 1983; Wu, 1985; Higgs & Derrida, 1992), and also the importance of selection in causing divergence of allopatric populations is widely appreciated (e.g., Lande, 1981; Schluter & Price, 1993; Gavrilets, 2000). This clearly indicates that a classification based on geographic speciation modes is phenomenological and implies nothing about the mechanisms involved in speciation (Templeton, 1981; Gavrilets, 2003; Dieckmann *et al.*, 2004).

Yet, a mechanistic understanding of speciation is important, since the crucial question is no longer simply whether or not spatial isolation is essential to create reproductive isolation from other species. This question has already been settled in the negative by Bush and others, who, in a classical study on host race formation in flies of the genus *Rhagoletis* (reviewed in Bush, 1994), convincingly demonstrated that reproductive isolation can evolve as a consequence of selection within a spatially well-mixed population. Accompanied by an increasing appreciation of the role of ecological processes in evolution, the focus of the speciation debate has shifted gradually from the geographical context of speciation to a discussion of the mechanisms of speciation (Via, 2001; Dieckmann *et al.*, 2004). The debate now focuses on the question what exactly the relative roles are of selection, genetic drift, and external chance events, such as the formation of geographical barriers, in the speciation process.

However, the distinction between the geographical mode and the mechanism of speciation is often not fully appreciated. Much of the discussion still emphasizes the geographical context of speciation, and, as a consequence of that, the emergence of a mechanistic understanding of speciation is impeded. How can we expect to draw conclusions about, say, the role of selection in speciation from models that rely on both adaptive and non-adaptive processes to cause divergence? It is very easy to contrast speciation models with respect to the geographic mode of speciation, but very difficult to do so relative to the mechanisms involved in speciation. Yet, it is to be expected that a mechanistic approach to speciation is much more powerful to gain insight than a phenomenological one based merely on the geographic modes of speciation, even though the latter might seem to be more practical (Gavrilets, 2003). It is precisely the goal to understand the mechanisms of speciation, and with such a goal, a phenomenological approach will prove counterproductive.

A full mechanistic theory of speciation would require a detailed understanding of the separate roles of selection, genetic drift and external factors in speciation, as well as insight into their interactions. This is more than can be accomplished here. In this thesis, I restrict myself mainly to investigating the role of selection, and sexual selection in particular, in causing the origin and maintenance of variation in characters responsible for reproductive isolation. By gradually isolating the role of selection from that of other mechanisms, such as mutation and genetic drift, we will eventually arrive at a set of conditions for adaptive speciation by sexual selection.

EVIDENCE FOR SPECIATION IS INDIRECT

Speciation is a slow process that cannot be observed directly in the wild (sudden speciation in plants involving genome duplication provides an exception to this rule). Consequently, speciation research is forced to rely on indirect methods, rather than on (manipulative) field studies. Molecular biology has made many such methods available, and they have provided fascinating data (e.g., Meyer *et al.* 1990; Schliewen *et al.*, 1994; Klicka & Zink, 1997; Wilson *et al.*, 2000). Yet, irrespective of the sophistication of the empirical techniques, it is notoriously difficult to infer past processes from present phylogenetic patterns.

Experiments in a simplified laboratory setting (reviewed in Rice & Hostert, 1993; Kirkpatrick & Ravigné, 2002) provide an alternative to studying speciation in the wild. Several such experiments were aimed at demonstrating the evolution of reproductive isolation in laboratory populations that had been subjected to artificially imposed selection against interbreeding. Since conclusive evidence for speciation without spatial isolation was only found under restrictive experimental conditions (Rice & Hostert, 1993), the outcome of these experiments has been used to argue against sympatric speciation. However, this interpretation of the results is uncertain, due to the possibility that the effective population size of laboratory populations is too small for speciation to occur (Ödeen & Florin, 2000).

A third alternative to study speciation is to use mathematical modeling (reviewed in Johnson & Gullberg, 1998; Turelli *et al.*, 2001; Kirkpatrick & Ravigné, 2002; Gavrilets, 2003). Mathematical models can be extremely effective to structure our thinking about a phenomenon as complex as speciation. The work presented in this thesis, which is exclusively theoretical in nature, aims at doing just that. Put sharply, it addresses, first and foremost, our thinking about speciation rather than the biological process of speciation itself. In my personal opinion, this is true for all existing theoretical work on speciation; ultimately, a theory of speciation can only rest on empirical data. With this inherent limitation of theoretical work in mind, mathematical models can be useful to reveal the hidden assumptions or inconsistencies of verbal arguments, and they can be used to identify gaps in our knowledge as well as fruitful directions for further empirical research.

THEORETICAL TOOLS ARE LACKING

Unfortunately, a lack of theoretical tools to study speciation forces theoreticians to rely heavily on numerical or individual-based simulation methods. Such simulations are very useful to check the results of more general analytical methods, which often involve restrictive assumptions (Taylor, 1996a; Weissing, 1996). However, when used on their own, they have serious disadvantages (Gavrilets, 2003): simulation models are often highly specific, and the interpretation and generalization of their results is very difficult.

The standard analytical methods available to the theoretical biologist can often not be applied straightforwardly in the context of speciation. Population-genetic models (Hartl & Clark, 1997), for example, are suitable to study the dynamics of allele frequencies at a small number of loci, each with a small number of alleles. A major advantage of these models is that they allow for a detailed description at the genetic level: genetic linkage between loci, dominance-recessivity relations between alleles and pleiotropic interactions can be dealt with explicitly. Yet, in the context of speciation, where multiple characters and many different loci are likely to play a role, the applicability of population genetic models is limited. This is due to the fact that these models have the tendency to become too complex to be tractable, as soon as more than two loci are involved (e.g., Maynard Smith, 1966).

Quantitative genetics (Lynch & Walsh, 1998) can be used to model the evolution of characters that are based on a large number of loci. This method describes evolution at the phenotypic level through postulating statistical relations between the breeding values of parents and their offspring. Such statistical relations strictly apply only under highly idealized genetic assumptions. Moreover, quantitative genetic models are accurate only when the distribution of breeding values is unimodal and Gaussian. This is obviously problematic, since speciation naturally involves the evolution of bimodal character distributions.

A third alternative is provided by the method of adaptive dynamics (Metz *et al.*, 1996). Adaptive dynamics theory considers evolution to be a mutation-limited process. Rare mutation events lead to the introduction of a new mutant allele. The mutant allele may disappear, replace one of the resident alleles or coexist with the resident alleles that are already present, depending on a fitness measure that can be derived from ecological considerations (Metz *et al.*, 1992). If no coexistence is possible, subsequent mutation and allele-substitution events will lead to gradual directional evolution. In the case that coexistence of multiple alleles is attained, further mutation and allele-substitution events will often lead to increasing differentiation of phenotypes. Adaptive dynamics theory offers analytical tools to study such adaptive emergence of polymorphism (Geritz *et al.*, 1998), which makes it an attractive method to study speciation. However, it has a number of limitations (Waxman & Gavrilets, 2004). The most important one for the research presented here is that adaptive dynamics is a local theory, which relies on restrictive assumptions on the frequency of mutations and the magnitude of their phenotypic effects. An additional practical disadvantage is that the derivation of the fitness function is usually feasible only under idealized genetic assumptions.

In view of the strengths and weaknesses of the different methods, my research strategy has generally been to complement multi-locus individual-based simulations with genetically idealized analytical investigations based on adaptive dynamics theory. This combined approach offers general insights and gives an impression of the robustness of the results under less idealized genetic assumptions.

SIX OBSTACLES TO SPECIATION

In order to fully appreciate the inherent difficulties associated with speciation, it is revealing to consider a prototypic example that illustrates six different general obstacles on the road to speciation. Consider a seed-eating bird species that lives in a habitat where different types of seeds are available. The distribution of seed sizes is such that small and large seeds are abundant, whereas seeds of intermediate size are rare. Birds cannot feed on all seeds equally well. Which seeds can be handled most efficiently is determined by genetic factors that affect the size of the bill. Small-billed individuals feed most efficiently on small seeds; large-billed birds feed most efficiently on large seeds. One would expect this to lead to a situation where birds with an intermediate bill size have the lowest feeding rate, as in FIGURE 1.

Under the assumption that the fitness of an individual is determined by its feeding rate, the population would then experience disruptive selection. One could easily imagine such disruptive selection to split the population into two specialized species, one feeding on the abundant small seeds, the other feeding on the abundant large seeds. Unfortunately, however, it is not self-evident that disruptive selection is generated in our example situation. In fact, the establishment of a selective regime as in FIGURE 1, where intermediate phenotypes have a selective disadvantage and where the highest fitness values are associated with extreme phenotypes, can be considered an important first obstacle to speciation.

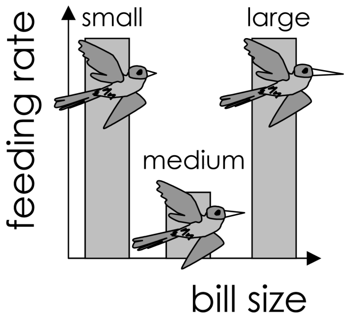

FIGURE 1 – DISRUPTIVE SELECTION ON BILL SIZE

For simplicity, I assume that three different bill-size phenotypes are present in the population. Assuming that the fitness of an individual is determined by its feeding rate, individuals with intermediate bill size have the lowest fitness, which implies that selection is disruptive.

OBSTACLE 1 — THE ESTABLISHMENT OF A DISRUPTIVE SELECTION REGIME

To be sure, disruptive selection can only be generated when there exists variation in seed size and when there exist feeding trade-offs such that no type of bill is suited to forage on all types of seeds with high efficiency. However, as illustrated in FIGURE 2, these two conditions are not sufficient. The figure shows the relative feeding efficiencies of individuals with different bill sizes on seeds of different size (light-gray bars). Small seeds are most likely to be consumed by birds with a small bill, medium size seeds by birds with an intermediate bill, and large seeds by birds with a large bill. Yet, the feeding rate (dark-gray bars on the left) of individuals with intermediate bill size is higher than that those with small or large bills, even when the distribution of seed sizes is bimodal (dark-gray bars in the back). This is a consequence of the fact that, for all seed sizes, the average feeding efficiency of individuals with large and small bills (indicated by the dashed lines) is lower than that of the individuals with intermediate-sized bills; in such a case, the feeding trade-off is 'weak' (Levins, 1962).

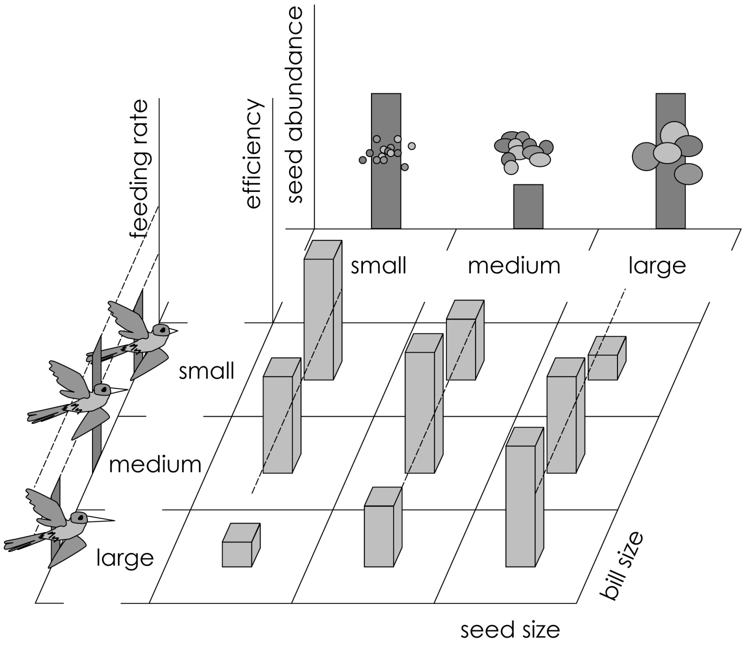

FIGURE 2 – A WEAK TRADE-OFF IS INSUFFICIENT TO GENERATE DISRUPTIVE SELECTION

Feeding efficiencies (light bars) are normalized within each seed size category, such that they represent the probability that a seed of a certain size is consumed by a bird with a certain bill size. The feeding rate of individuals (dark bars on the left) was calculated as a weighted average of the efficiency on seeds of different size, where the weighting factors are given by the seed abundances (dark bars in the back).

Apparently, a special kind of trade-off between the feeding efficiencies on seeds of different sizes is required in order to generate disruptive selection. Indeed, it is well established that, without more complex assumptions on foraging behavior (Rosenzweig, 1987), a disruptive selection regime can only arise when the feeding efficiencies are subject to a 'strong' trade-off (Levins, 1962; FIGURE 3). Such is the case when the feeding efficiency of an individual with intermediate bill size on small and large seeds is less than the average feeding efficiency of individuals with small and large bills on the same seeds (dashed lines). If these seeds are abundant,

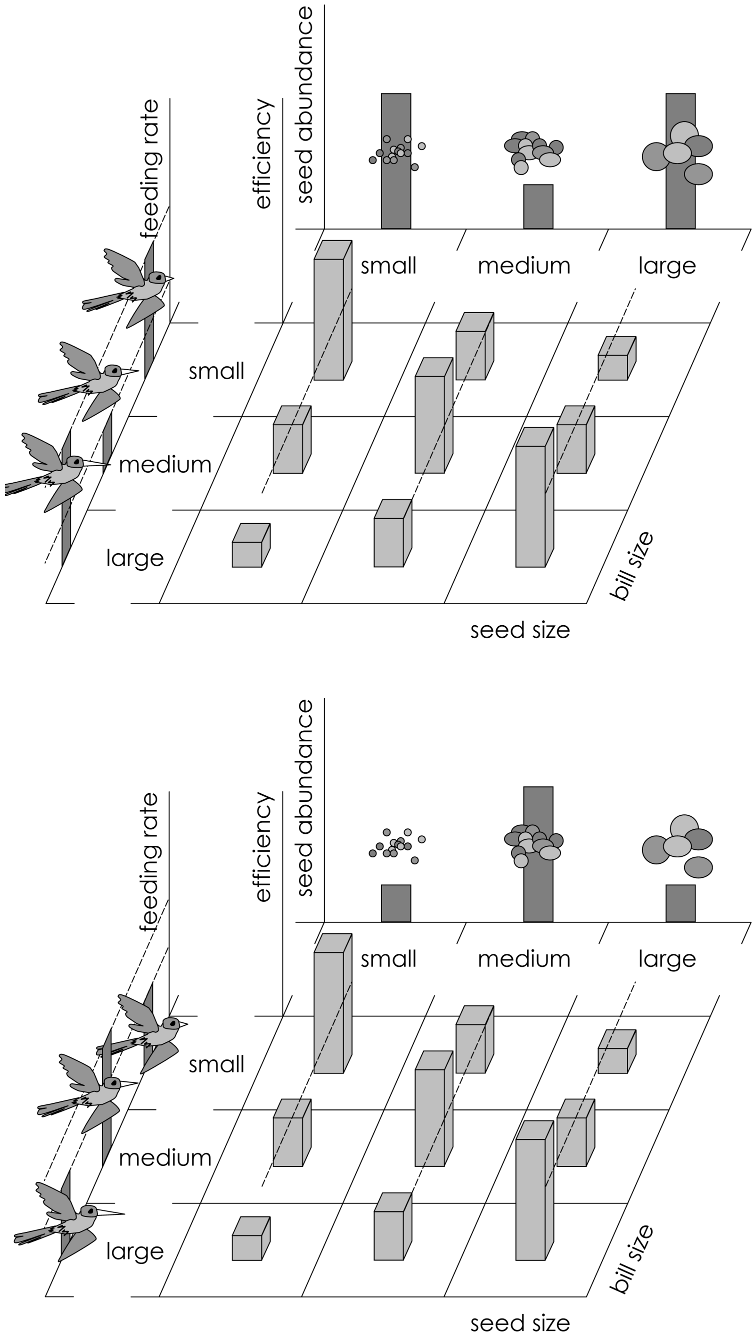

FIGURE 3 – A STRONG TRADE-OFF

With a strong trade-off for the feeding efficiencies on small and large seeds, individuals with large and small bills do better on average than individual with an intermediate bill size (dashed line). The latter do better on seeds of intermediate size. Depending on the abundance of small and large seeds relative to that of seeds of intermediate size, intermediate types have a lower (upper panel) or higher (lower panel) feeding rate.

the feeding rate of individuals with intermediate bill size will be lowest (upper panel; as in FIGURE 1). In this case, the population will experience disruptive selection with respect to bill size. If, however, seeds of intermediate size are more abundant (lower panel), individuals with intermediate bill size will have the highest feeding rate. Note that I so far ignored any effects of the population density of birds with different bill sizes on the distribution of seed abundances. A discussion of these effects is postponed to a later point in this chapter.

The establishment of a disruptive selection regime presents an obstacle to speciation that is usually ignored in speciation models. Despite the fact that there are good reasons for doing so, it is important to realize that the presence of disruptive selection cannot be taken for granted. Disruptive selection usually arises from strong constraints on the evolving system. Evolution in natural populations has many different degrees of freedom, and it may very often succeed in circumventing the type of constraints and trade-offs that are implemented in simple models (Van Dooren *et al.*, in press). The establishment of a disruptive selection regime may therefore be even more complicated than simple models would appear to suggest. Yet, a definite answer to this question requires a theory of evolution under complex constraints, which is still largely lacking at the moment.

OBSTACLE 2 — THE MAINTENANCE OF POLYMORPHISM UNDER DISRUPTIVE SELECTION

Let me suppose from here on that the bird species in my example does indeed experience disruptive selection, as in FIGURE 1. One would intuitively expect this to lead to the establishment of two specialist species. To check formally this intuition, let me suppose that the size of the bill is determined by a single locus with two alleles, *A* and *a*. Individuals with the genotype *AA* develop a large bill, those with genotype *aa* develop a small bill, and heterozygote individuals develop a bill of intermediate size. I will use p_t and q_t to denote the frequencies of the allele *A* and *a*, respectively, in generation t. Furthermore, let w_{AA}, w_{Aa} and w_{aa} denote the fitness values associated with the three bill size genotypes. For the moment, I take these fitness values to be fixed and proportional to the feeding rates. Individuals mate at random.

Under these assumptions, the allele frequencies change from one generation to the next according to the standard replicator equation

$$p_{t+1} = p_t \frac{w_A}{\bar{w}} \qquad [1]$$

where $w_A = p_t\, w_{AA} + q_t\, w_{Aa}$ and $w_a = q_t\, w_{aa} + p_t\, w_{Aa}$ are the fitness values of the two alleles, and where $\bar{w} = p_t\, w_A + q_t\, w_a$ is the average fitness.

Numerical iteration of equation [1] shows that disruptive selection indeed leads to the evolution of a specialist phenotype. However, one always observes the evolution of only a single specialist phenotype, instead of two, due to the fact that one of the alleles is rapidly lost from the population. As shown in FIGURE 4, it depends on the initial allele frequencies, which one of the alleles will disappear.

The threshold initial frequency of the allele *A* that separates between those cases where the allele *A* disappears and those were the allele *a* is lost, depends on the genotype fitness values. It is given by $\left(w_{aa} - w_{Aa}\right)/\left(w_{AA} + w_{aa} - 2w_{Aa}\right)$. For the parameters used in FIGURE 4, this formula predicts a threshold frequency of $4/9 \approx 0.44$.

The loss of polymorphism observed in the simulations is caused by an intrinsic disadvantage of rarity. A rare allele, be it either *A* or *a*, will occur relatively often in combination with the other, more frequent allele. This implies that a rare allele is relatively often expressed as part of the heterozygote genotype, which has the lowest fitness. Stable polymorphism can be maintained with an intrinsic advantage of rarity (Lewontin, 1958); i.e., when the rare allele has a higher fitness than the common allele. This requires for the genotype fitness values that $w_{Aa} > w_{aa}$ when the allele *A* is rare, and, similarly, that $w_{Aa} > w_{AA}$ when the allele *a* is rare. With fixed fitness values for the genotypes, these conditions cannot be reconciled with the conditions for disruptive selection ($w_{Aa} < w_{AA}$ and $w_{Aa} < w_{aa}$). As a consequence, the establishment of a stable polymorphism is precluded when the genotype fitness values do not vary with the frequencies of the genotypes. Since speciation is always accompanied by the establishment of genetic polymorphism, speciation requires frequency-dependent selection (Udovic, 1980).

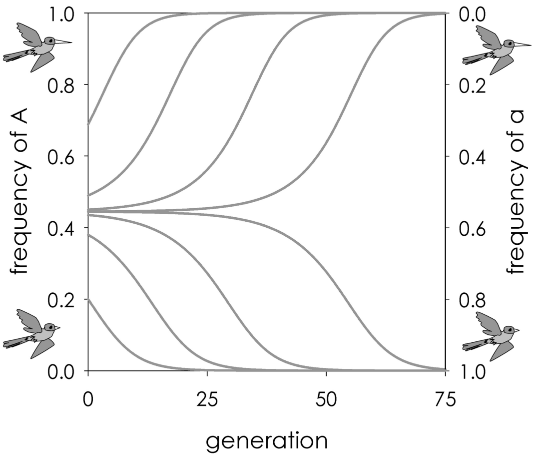

FIGURE 4 – ALLELE-FREQUENCY CHANGE DUE TO DISRUPTIVE SELECTION

The lines represent replicate simulations, started from different initial conditions. The genotype fitness values were $w_{AA} = 1$, $w_{Aa} = 0.75$ and $w_{aa} = 0.95$.

The contrast between frequency-independent and frequency-dependent selection is explained in FIGURE 5. The left panel of this figure illustrates the disadvantage for rare alleles under frequency-independent disruptive selection. The panel shows the allele fitness values (black lines) as function of the allele frequencies in a situation where the genotype fitness values (gray lines) are constant as in the earlier simulations (FIGURE 4). The dashed line demarcates the equilibrium allele frequency where the fitness of both alleles is equal. Left of the dashed line, the frequency of the allele *A* decreases over time (as indicated by the arrow) due to the fact that $w_A < w_a$. Right of the dashed line, the opposite will occur, because $w_A > w_a$. Clearly, a polymorphism of alleles cannot be maintained in this case.

This is different in the right panel of the figure, which shows a situation where the genotype fitness values vary with the genotype frequencies (for a randomly mating population in Hardy-Weinberg equilibrium, the latter are uniquely determined by the allele frequencies). Again, the dashed line demarcates the equilibrium allele frequency at which both alleles have equal fitness. Contrary to the situation in the left panel, the allele frequencies converge on the equilibrium value (arrows), due to the fact that $w_A > w_a$ left of the dashed line, and $w_A < w_a$ right of the dashed line. Note that the genotype fitness values satisfy the conditions for maintenance of polymorphism as well as the conditions for disruptive selection. Neither allele will disappear when rare, due to the fact that $w_{Aa} > w_{aa}$, when the allele *A* is rare, and $w_{Aa} > w_{AA}$, when the allele *a* is rare. At the same time, the population experiences disruptive selection once the equilibrium allele frequencies have been attained: in the vicinity of the stable equilibrium, $w_{Aa} < w_{AA}$ and $w_{Aa} < w_{aa}$.

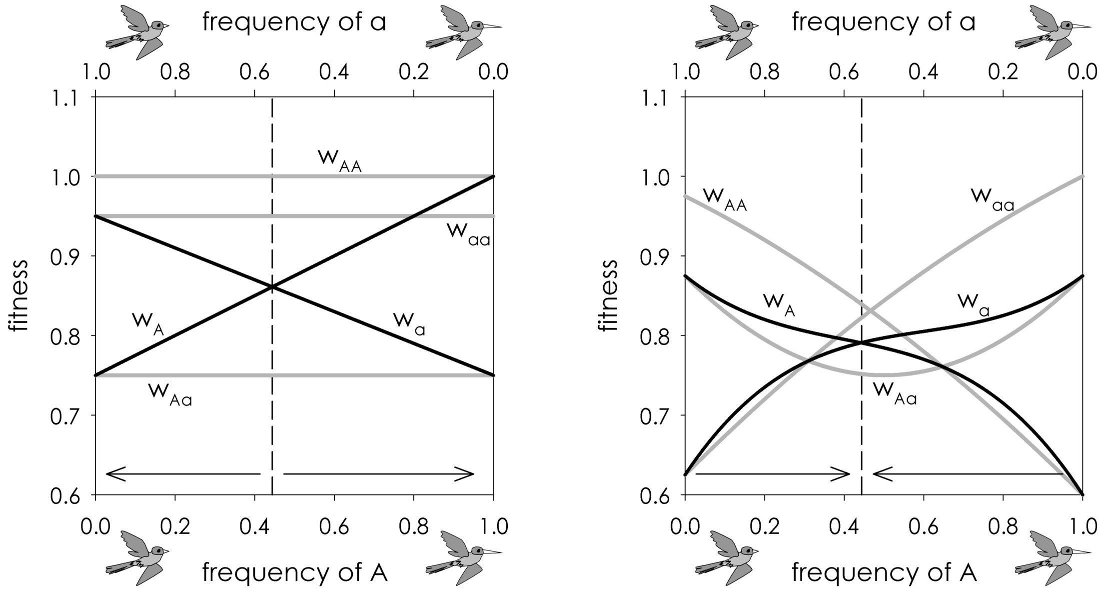

FIGURE 5 – FREQUENCY-INDEPENDENT VERSUS FREQUENCY-DEPENDENT DISRUPTIVE SELECTION

When selection is frequency-independent (left panel), underdominance ($w_{Aa} < w_{AA}$, $w_{Aa} < w_{aa}$) cannot lead to a stable polymorphism of alleles. By contrast, if the genotype-fitness values vary with the allele frequencies, a protected polymorphism of alleles can be established. This can be accomplished when both alleles have a selective advantage when rare. In the right panel, this is even accompanied by underdominance in the vicinity of the equilibrium.

It will be clear that the combination of disruptive selection and an advantage of rarity requires a specific relation between the fitness values associated with the different phenotypes and their respective frequencies and, hence, a specific type of frequency-dependent selection. To be precise, it requires the fitness of a phenotype to be strongly depressed when the phenotype occurs at a high frequency. Several authors have proposed ecological mechanisms that could give rise to such fitness relations. A classical example is that of ecological resource competition (Rosenzweig, 1978; Dieckmann & Doebeli, 1999; Drossel & McKane, 2000). To illustrate its action in the context of the example model (FIGURE 6), let me suppose that individuals compete for seeds; foraging birds deplete the available seed stock, which also acts as a limiting factor to population growth. If, for some reason, individuals with a large bill are common (A), particularly the abundance of large seeds will be depressed (B). Large-billed individuals will then experience difficulties to find suitable seeds and their feeding rate will be lower (C). As a consequence, the fitness of the allele *A* will be less than that of *a* (D). One would expect this to induce a decrease of the frequency of large-billed individuals, perhaps leading to a situation where individuals with small bills are more common (E). This would, however, rapidly lead to a depletion of small seeds (F), a lower feeding rate for small-billed individuals (G), and a lower allele fitness of the allele *a*. As a general outcome of this feedback mechanism, one would sooner or later expect the allele fitness values to become identical (I).

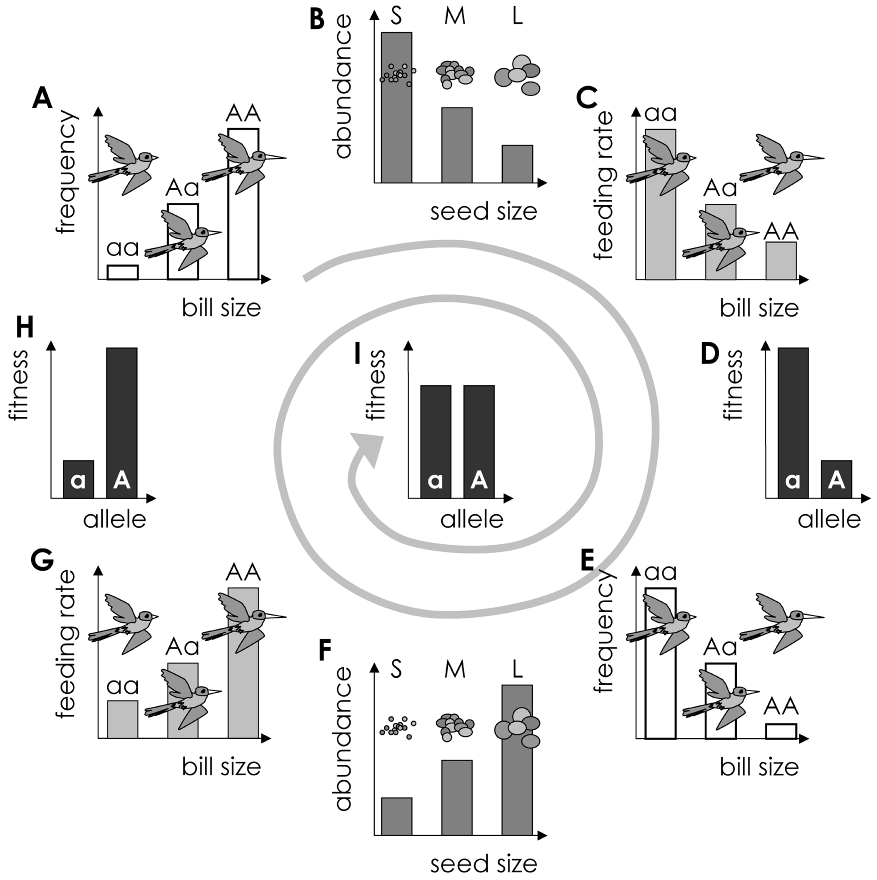

FIGURE 6 – COMPETITION FOR SEEDS GENERATES FREQUENCY-DEPENDENT SELECTION

Ecological resource competition is certainly not the only mechanisms that can give rise to a stable disruptive selection regime. A few additional examples (more can be found in Udovic, 1980; Weissing, 1996) are separate density regulation of the population in two different niches (Levene, 1953; Maynard Smith, 1966; references in Kirkpatrick & Ravigné, 2002), multi-species interactions such as mutualism and predator-prey interactions (Doebeli & Dieckmann, 2000), and mating behavior (Ehrmann & Spiess, 1969; Knoppien, 1985). The latter possibility will receive detailed attention in subsequent chapters of this thesis.

On a more abstract level, the potential for protected polymorphism can be related to characteristics of the feedback between population growth and the environment (the so-called effective environmental dimensionality; Meszéna & Metz, in press). Roughly speaking, there can be no polymorphism under disruptive selection if the population is regulated through only a single environmental factor (e.g., if a single resource limits growth). If, however, population regulation acts through multiple environmental factors, such as the abundances of distinct resources, the coexistence of multiple alleles is possible in principle.

OBSTACLE 3 — THE EVOLUTION OF REPRODUCTIVE ISOLATION

In what follows, I do not model the biological interactions that give rise to frequency-dependent selection in detail. Instead, I simply assume frequency-dependent genotype fitness values as in FIGURE 5. Numerical simulations of the recurrence equations [1] with these fitness relations, confirm the expectation that frequency-dependent selection can maintain polymorphism (FIGURE 7, left panel). However, these simulations do not show the evolution of two separate specialist species. The distribution of genotypes remains unimodal. Both homozygote genotypes are present, but the majority of individuals are heterozygote. The reason for this is straightforward. Since individuals mate at random, the population genotype frequencies cannot deviate from the Hardy-Weinberg proportions $f_{AA} : f_{Aa} : f_{aa} = p_t^2 : 2p_tq_t : q_t^2$. Obviously, speciation requires a mechanism to account for reproductive isolation between the daughter-species.

In fact, the biological species concept defines species as groups of actually or potentially interbreeding natural populations *reproductively isolated* from other such groups (Mayr, 1942). Reproductive isolation may be caused by a variety of factors, such as spatial isolation, behavioral isolation, mechanical isolation and hybrid inviability or sterility. Here, I concentrate on reproductive isolating mechanisms that act before zygote production. Cases where species remain distinct only because of postzygotic isolation are extremely rare (Kirkpatrick & Ravigné, 2002).

In many cases, prezygotic isolating mechanisms give rise to barriers to gene flow between species by effectuating a higher incidence of mating between individuals that are phenotypically similar ('positive assortative mating', abbreviated as 'assortative mating' from here on). Assortative mating reduces the probability of crosses between dissimilar individuals. As a consequence, the frequency of heterozygote individuals strongly decreases. The stronger the tendency to mate with phenotypically similar individuals, and the stronger selection against heterozygote individuals, the lower their frequency will become.

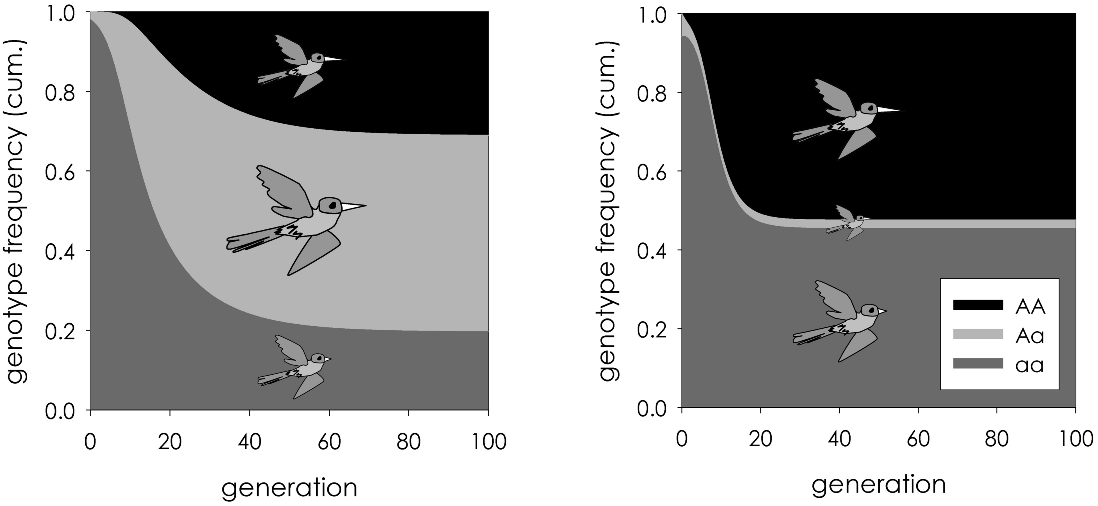

FIGURE 7 – DISRUPTIVE SELECTION WITH AND WITHOUT ASSORTATIVE MATING

Disruptive selection cannot generate two species when individuals mate at random (left panel). Random mating produces a large number of maladapted heterozygote individuals. In the right panel, I implemented strong assortative mating with respect to bill size: individuals had a 50 times higher tendency to mate with a phenotypically similar partner.

To illustrate this effect, I extended the simulation model to include assortative mating (FIGURE 7, right panel). Assortative mating can be implemented in many different ways. I assumed a polygynous mating system where females choose a partner from the available males after viability selection has occurred. The probability that a female selects a male with a particular bill size is determined by the frequencies f_i of the different bill size genotypes, as well as by mate choice coefficients $c_{i\times j}$ which measure the tendency of a female with bill size i to mate with a male with bill size j ($i, j = AA, Aa$ or aa). Mating is assortative with respect to bill size when $c_{i\times i} > c_{i\times j}$ for all $j \neq i$, that is, when individuals have the highest tendency to mate with phenotypically similar mating partners. The frequency $C_{i\times j}$ of crosses between females of genotype i and males of genotype j was taken as

$$C_{i\times j} = f_i\, f_j \frac{c_{i\times j}}{\overline{c}_i} = f_i \frac{c_{i\times j}\, f_j}{c_{i\times AA}\, f_{AA} + c_{i\times Aa}\, f_{Aa} + c_{i\times aa}\, f_{aa}}, \qquad [2]$$

such that all females had an equal probability to reproduce, irrespective of their bill size.

It is selectively advantageous to evolve stronger assortative mating, if individuals mate assortatively with respect to the same trait that is also under disruptive selection (Udovic, 1980; Kondrashov, 1986; Dieckmann & Doebeli, 1999). In this case, mating between individuals with opposite phenotypes is disfavored, since the offspring of such a mating will express the selectively disadvantageous heterozygote phenotype. In our example, assortative mating based on bill size is selectively advantageous, because it reduces the frequency of matings between individuals with a large and a small bill. Such matings lead to the production of offspring with intermediate bill size, which have a low fitness. If no intrinsic costs are associated with assortative mating (e.g., mate choice costs), selection will favor it to become ever stronger, until full reproductive isolation is attained eventually.

This conclusion derives from so-called 'one-allele' models for speciation (Felsenstein, 1981), which consider the evolution of characters that modify the strength of assortative mating (see, e.g., Felsenstein, 1981; de Meeûs *et al.*, 1993; Kawecki, 1996, 1997; Dieckmann & Doebeli, 1999). The qualification 'one-allele' is to reflect that reproductive isolation can be strengthened in these models by the spread of the same allele in both daughter species (FIGURE 8, left panel). For example, reproductive isolation between two host races of a phytophagous insect can be strengthened by an allele that increases the tendency to mate on the host plant. With the exception of a few similarly specific examples, it seems, however, that one-allele mechanisms are not very common in nature, presumably because genetic variation for modifiers of the strength of assortative mating is rare (Felsenstein, 1981; Kirkpatrick & Ravigné, 2002).

In most cases, assortative mating must be strengthened by the substitution of different alleles in the two daughter-species (i.e., by a so-called two-allele mechanism; Felsenstein, 1981; FIGURE 8, right panel). Examples of two-allele mechanisms include the evolution of different host plant preferences in phytophagous insects that mate on their host plant, or the evolution of different flowering times in a plant species, giving rise to temporal isolation. Speciation models where two-allele mechanisms are investigated include Udovic, 1980, Felsenstein (1981), Rice (1984), Dieckmann & Doebeli (1999) and Kondrashov & Kondrashov (1999).

One- and two-allele mechanisms may act the same time. In fact, one-allele mechanisms require that there already be differentiation based on a two-allele mechanism. This important point is illustrated in FIGURE 8, which shows the two ways in which assortative mating can be strengthened in the example of our seed-eating bird species. Since only under rather specific conditions it seems plausible to presume that individuals mate assortatively with respect to the trait under disruptive selection (as in FIGURE 7), I will suppose from here on that assortative mating is based on the color of the plumage, an arbitrary character that I assume to be completely independent of an individual's bill size. In the left part of FIGURE 8, it is depicted how assortative mating can be strengthened by a one-allele mechanism, in this case, the invasion of a single modifier allele $\tilde{m}$ in both species, which increases the tendency to mate with an individual that exhibits the same plumage color. In the right part of FIGURE 8, assortative mating is strengthened by a two-allele mechanism, i.e., by the invasion of different alleles that increase the difference in plumage color between the daughter species. In the one species, the allele $\tilde{Q}$ causes the plumage to become whiter; in the other species, the allele $\tilde{q}$ causes the plumage to become blacker. Crucially, the one-allele mechanism requires there to be variation in plumage color to start with.

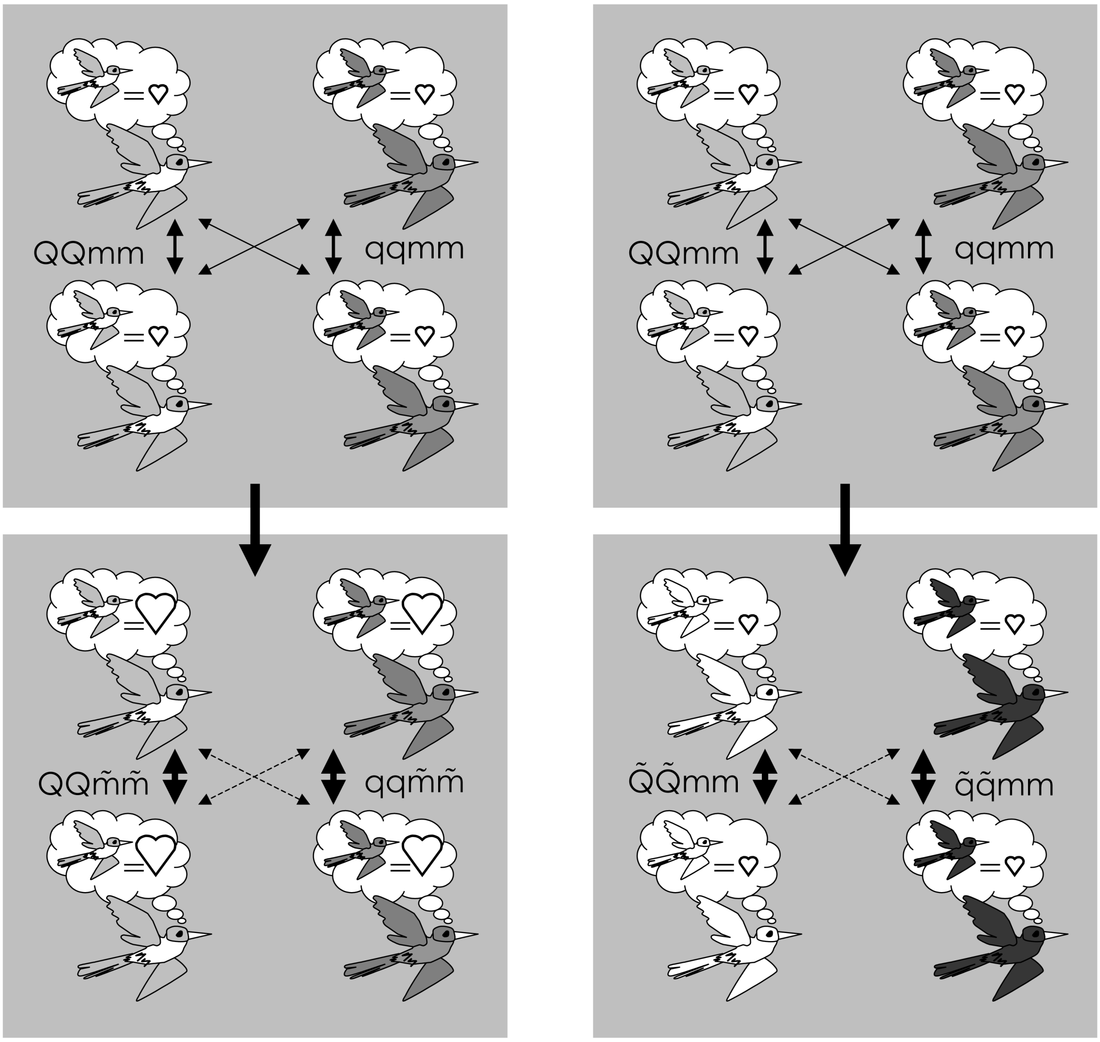

FIGURE 8 – TWO WAYS TO STRENGTHEN ASSORTATIVE MATING

Left: in a population that is variable for plumage color (the allele Q codes for a light plumage; the allele q codes for a dark plumage), assortative mating based on the color of the plumage can be strengthened by the invasion of a single modifier allele $\tilde{m}$ (one allele mechanism) that increases the tendency to mate with a phenotypically similar individual (as indicated by the size of the hearts). Right: alternatively, assortative mating can be strengthened by the invasion of different alleles (two allele mechanism) in the two daughter species ($\tilde{Q}$ codes for white plumage; $\tilde{q}$ codes for black plumage) that increase the differentiation of the plumage colors. Both mechanisms will increase the probability of crosses (indicated by the width of arrows) between similar individuals, and decrease the probability of interbreeding between the daughter species.

OBSTACLE 4 — TRANSMITTING THE FORCE OF DISRUPTIVE SELECTION TO THE GENES RESPONSIBLE FOR REPRODUCTIVE ISOLATION

The preceding discussion leaves two important issues unresolved. First, why should we expect there to be variation in plumage color? It is not obvious that the frequency-dependent selection that maintains variation in bill size, will have the same effect on plumage color, unless it would, in some way, act on the genes for plumage color as well. Second, why should we expect the strength of assortative mating based on plumage color to increase at all – irrespective of whether this occurs through a one- or two-allele mechanism? In fact, I argued that enhancing assortative mating is selectively advantageous when this allows individuals to avoid the production of less-fit heterozygote individuals. Without this selective advantage, it is difficult to explain why assortative mating should become strong, especially given the fact that assortative mating is likely to involve costs. Yet, in the case that assortative mating is based on an arbitrary trait (i.e., not the character under disruptive selection) it is not at all obvious that it will lead to a lower frequency of heterozygotes for the character under disruptive selection. In terms of our example, why should assortative mating with respect to plumage color reduce the frequency of individuals with an intermediate bill size? A selective advantage for assortative mating based on plumage color would arise only when the heterozygotes for plumage color are less fit.

It will be clear that these problems do not play a role when the character under disruptive selection is also the character on which assortative mating is based. As a matter of fact, the most convincing evidence for sympatric speciation comes from natural systems where this appears to be the case (Rice, 1984; Rice & Hostert, 1993). For example, in many herbivorous arthropods, specialization to new host plants often leads to reproductive isolation as a by-product, since individuals mate on their host plant (Bush, 1994).

Notwithstanding these specific cases, it is, however, safe to assume that the average trait under disruptive selection does not simultaneously act as a basis for reproductive isolation. When assortative mating and disruptive selection act on different sets of genes, speciation can only proceed when the genes subject to assortative mating are genetically correlated, i.e., in linkage disequilibrium, with the genes that are the target of disruptive selection. This linkage disequilibrium is instrumental in transmitting the force of frequency-dependent disruptive selection to the genes responsible for reproductive isolation, which, in view of the above, is needed to maintain variation in these genes and to strengthen assortative mating.

Indeed, when the genes for plumage color are in linkage disequilibrium with the genes for bill size, as in panel C of FIGURE 9, individuals with intermediate plumage color are likely to be heterozygous for bill size too. Selection against such individuals favors the evolution of stronger assortative mating (e.g., through increasing differentiation of the plumage), which may eventually lead to complete reproductive reproduction isolation and speciation (panel D). Unfortunately, linkage disequilibrium is eroded by recombination. Accidental mating between dissimilar homozygote individuals creates intermediate types. Crosses between these intermediate types create all possible combinations between bill-size and plumage-color alleles, which very rapidly leads to a situation where plumage-color and bill-size alleles are genetically uncorrelated (FIGURE 9, panel B). Without linkage disequilibrium, we are far from speciation: mating is effectively random with respect to bill size. In addition, variation in plumage color can no longer be maintained by frequency-dependent selection on bill size, with the consequence that plumage-color alleles may be lost from the population (FIGURE 9, panel A).

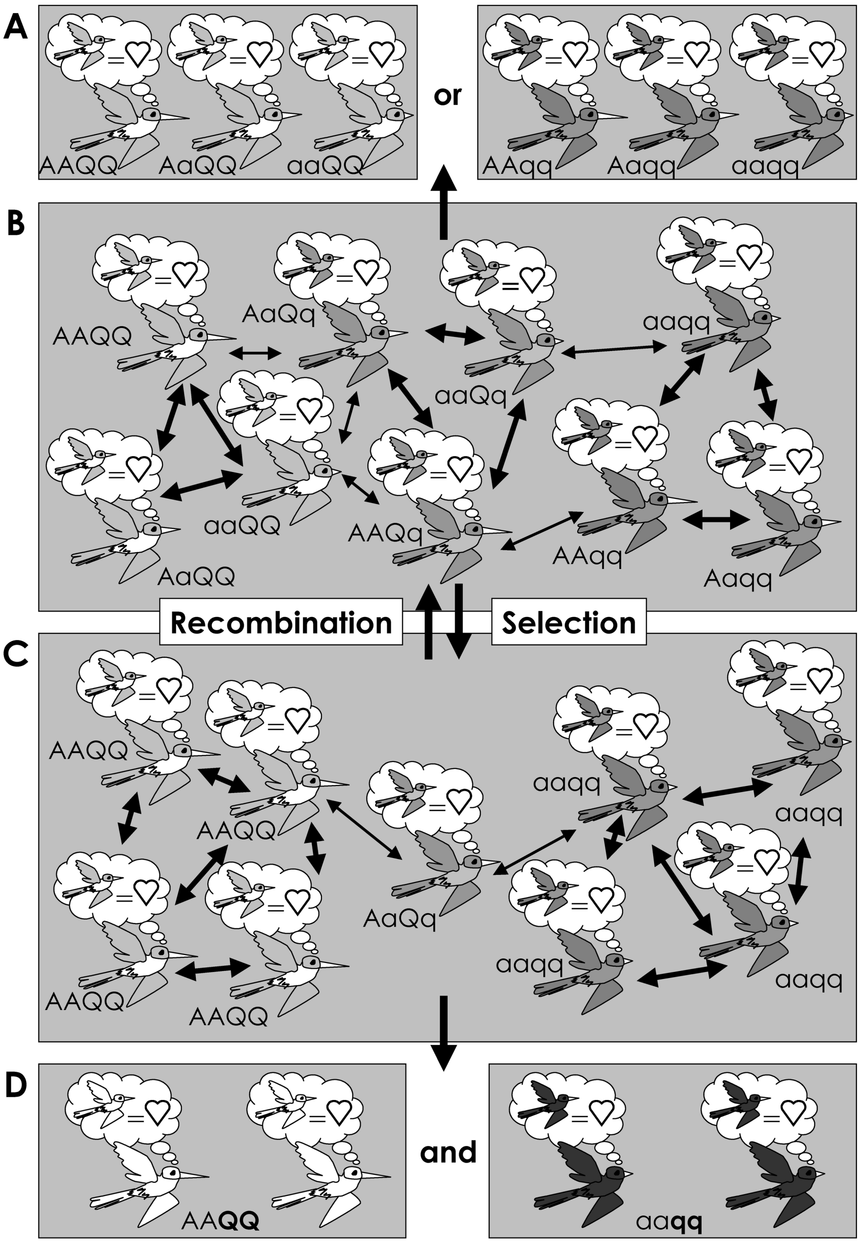

FIGURE 9 – RECOMBINATION OPPOSES SPECIATION

Linkage disequilibrium can only build up when selection can remove heterozygote individuals faster than recombination creates them. This requires that assortative mating be strong enough, such that crosses between dissimilar individuals are rare, and that selection against heterozygotes be intense, such that they have a low probability of contributing to the next generation. FIGURE 10 illustrates this point for the example model. The figure shows the evolution of the gamete frequencies in a situation where disruptive selection acts on bill size and where individuals mate assortatively with respect to plumage color, as in FIGURE 9. Assortative mating was implemented in a manner analogous to that used in the simulations shown in FIGURE 7, except, of course, that assortative mating was not based on bill size, but on plumage color. In the two panels of FIGURE 10, I kept the strength of disruptive selection constant; genotype fitness values were taken as in FIGURE 5.

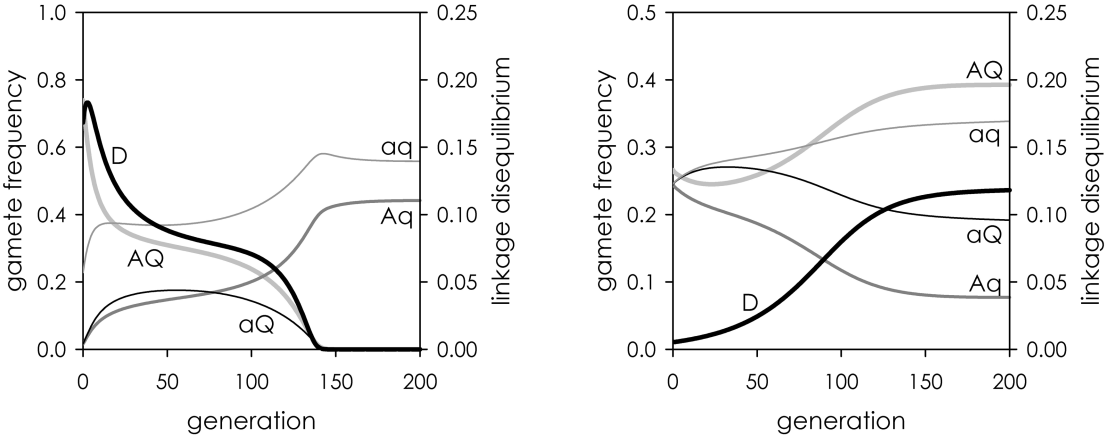

FIGURE 10 – STRONG ASSORTATIVE MATING IS REQUIRED TO OVERCOME RECOMBINATION

Although the tendency to mate with phenotypically similar individuals is already high in the left panel (individuals have a 25 times higher tendency to mate with a phenotypically similar partner), the combined strength of assortative mating and disruptive selection is not sufficient to maintain an association between bill-size and plumage-color alleles. The strength of such an association is measured by the linkage disequilibrium *D*. In the right panel, assortative mating is strong enough (individuals have a 50 times higher tendency to mate with a phenotypically similar partner) to allow a linkage disequilibrium to build up. I assumed free recombination between the bill-size and plumage-color locus.

In the left panel of FIGURE 10, assortative mating, though strong, is not strong enough. The simulation starts from initial conditions where the linkage disequilibrium (*D*) between bill size and plumage color alleles is large (cf. FIGURE 9C). However, recombination erodes the existing linkage disequilibrium (cf. FIGURE 9B), and, eventually, the allele *Q* is even lost from the population (cf. FIGURE 9A; which allele is lost depends on arbitrarily small initial asymmetries between the alleles). In the right panel, assortative mating is stronger. The combined strength of selection against intermediate bill sizes and assortative mating is now large enough to oppose the homogenizing force of recombination, allowing a linkage disequilibrium to build up.

In view of the overwhelming force of recombination to destroy linkage disequilibria, the combined strength of assortative mating and disruptive selection must be very large for speciation to occur (Udovic, 1980; Felsenstein, 1981; Gavrilets, 2003). This condition has led many to reject the possibility that disruptive selection can generate reproductive isolation when it does not directly act on genes responsible for assortative mating (e.g., Felsenstein, 1981).

The conditions for speciation become slightly less stringent when there are pleiotropic interactions between the character under disruptive selection and the assortative mating trait. Such pleiotropic interactions arise, for example, in situations where individuals choose between two habitats. A genetic character coding for habitat preference serves as a basis for assortative mating (assuming that mating occurs within the habitat). At the same time, it may influence ecological success: when each habitat requires specific adaptations, individuals will have high fitness only when they express a preference for the habitat to which they are optimally adapted. By transferring some of the force of disruptive ecological selection to the assortative mating trait, pleiotropic interactions somewhat lower the combined strength of selection and assortative mating needed to overcome the effect of recombination (Fry, 2003). However, the ecological circumstances under which one would expect strong interactions between the character under disruptive selection and the assortative mating trait are only slightly less specific than those required for the case where disruptive selection acts directly on the reproductive isolating mechanism.

I emphasize that recombination not only destroys associations between the genes for assortative mating and those subject to disruptive selection. It also affects linkage disequilibria within each of these two types of genes. This is not relevant for the example I used, where bill size and plumage color are coded by a single locus each. However, in a situation where these characters are coded by multiple loci, it will be even easier for recombination to create individuals with intermediate bill sizes and plumage color. By decreasing the effectiveness of assortative mating and disruptive selection, this will facilitate, in a self-reinforcing manner, the further erosion of existing linkage disequilibria. In multilocus models, it is therefore possible that both a population state with high linkage disequilibria and a state with random associations between alleles are stable at the same time (Liou & Price, 1994; Dieckmann & Doebeli, 1999; Kirkpatrick & Ravigne, 2002). In such cases, the strength of selection needed to initiate speciation is larger than that needed to complete it. For a reasonable strength of selection, speciation will therefore only occur when linkage equilibria are initially large. This could be the result of stochastic fluctuations in small populations (Dieckmann & Doebeli, 1999), or it could arise in the context of secondary contact between two incompletely isolated species, where it might lead to reinforcement of premating isolation in situations where *de novo* sympatric speciation cannot occur (Liou & Price, 1994).

OBSTACLE 5 — A MECHANISM FOR ASSORTATIVE MATING

Up to here, I have taken for granted that assortative mating with respect to bill size or plumage color can easily evolve. This is not unlike the usual practice in speciation models, where the presence of assortative mating based on some phenotypic character is often presupposed. Although assortative mating is sometimes generated as an automatic by-product, e.g., as a consequence of spatial or temporal isolation, it is often not obvious why individuals should mate assortatively in the first place. Why, for instance, should individuals of the example bird species mate assortatively with respect to plumage color?

A general mechanism that could lead to the evolution of assortative mating is female mate choice. Females exert mate choice in a wide variety of species. This is often explained as a consequence of the characteristic difference between the potential reproductive rates of males and females (Clutton-Brock & Vincent, 1991). Females are often limited by the time required to secure resources for the production of large gametes and for maternal care. They typically have a lower potential rate of reproduction than males, which invest less into gamete production and parental care. This leads to competition among males for access to receptive females and it provides the opportunity for females to choose among potential mates (Andersson, 1994).

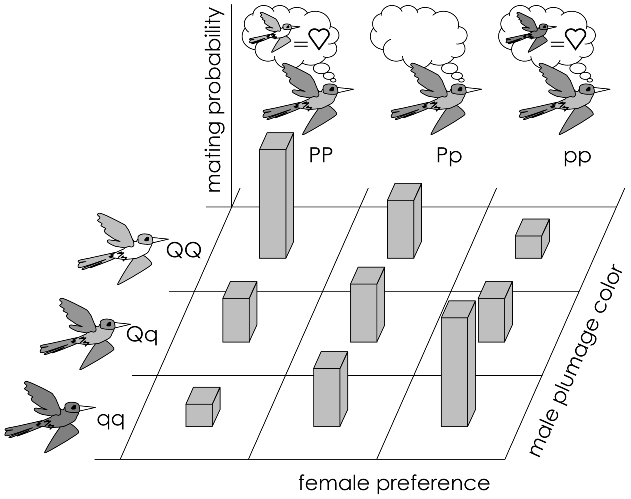

FIGURE 11 – FEMALE PREFERENCE FOR PLUMAGE COLOR

Suppose, for the sake of concreteness, that, in the example species, female preference is genetically determined by a single locus with two alleles. The allele P conveys a preference for a light plumage; the allele p conveys preference for a dark plumage (FIGURE 11). As before, plumage color is encoded by a single locus with two alleles, Q inducing light plumage, and q inducing dark plumage. For simplicity, I assume sex-limited expression of preference (in females) and plumage color (in males). The probability $M_{i\times j}$ that a female with preference i ($i = PP, Pp$ or pp) will mate with a male with plumage color j ($j = QQ, Qq$ or qq) depends on the frequencies f_j of the different types of males, as well as on the 'attractiveness' $m_{i\times j}$ of these males as perceived by the female. Females that carry the genotype *PP* prefer to mate with lighter males, implying that $m_{PP\times QQ} > m_{PP\times Qq} > m_{PP\times qq}$. Similarly, *pp* females prefer to mate with darker males, such that $m_{pp\times QQ} < m_{pp\times Qq} < m_{pp\times qq}$. I furthermore assume that heterozygote females exhibit no mating preference and, hence, that $m_{Pp\times QQ} = m_{Pp\times Qq} = m_{Pp\times qq}$. Analogously to equation [2], the frequency of crosses between females with preference i and male with plumage color j is given by

$$C_{i\times j} = f_i\, M_{i\times j} = f_i\, f_j \frac{m_{i\times j}}{\bar{m}_i} = f_i \frac{f_j\, m_{i\times j}}{f_{QQ}\, m_{i\times QQ} + f_{Qq}\, m_{i\times Qq} + f_{qq}\, m_{i\times qq}}, \qquad [3]$$

such that all females have an equal probability of mating, irrespective of their preference. The mating probabilities $M_{i\times j}$ are indicated by gray bars in FIGURE 11. The similarity between equations [2] and [3] is indicative of the fact that assortative mating can be considered as a special kind of preference where the mating preference acts on itself (Kirkpatrick & Ravigné, 2002).

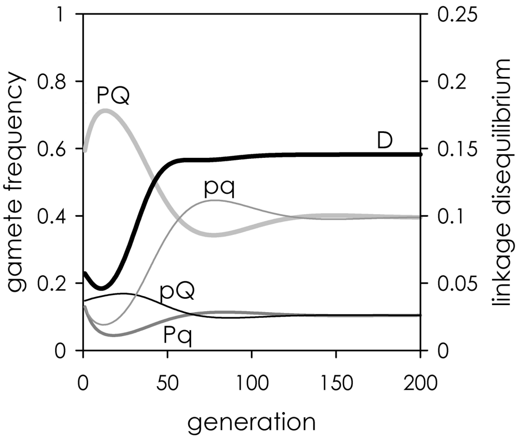

FIGURE 12 – FEMALE CHOICE GENERATES LINKAGE DISEQUILIBRIUM

Non-random mating, as defined in equation [3], generates non-random associations, or linkage disequilibrium (*D*), between female preference and male plumage color alleles. In the simulation, I assumed free recombination between the preference and plumage color loci. Female choice was assumed to be costly: *PP* and *pp* females had a 5% lower viability than *Pp* females. Similarly, the expression of light or dark plumage carried a viability cost for males: *QQ* and *qq* males had a 20% lower viability than *Qq* males. The attractiveness coefficients were taken as follows: $m_{PP\times QQ}=m_{pp\times qq}=10$, $m_{PP\times qq}=m_{pp\times QQ}=0.1$, all other $m_{i\times j}=1$.

Female mate choice can give rise to assortative mating, since it generates linkage disequilibrium between the genes for preference and plumage color (FIGURE 12). As a result of this linkage disequilibrium, females with a preference for light plumage (genotype *PP*) are likely to select a mating partner, which itself carries *P* alleles. In other words, due to the non-random association between preference and plumage color alleles, mating becomes assortative with respect to these characters. Assortative mating will be stronger when the linkage disequilibrium is larger, i.e., when females are choosier.

The preceding argument explains how female choice can generate assortative mating, but it leaves unresolved why females should be choosy to start with. At first sight, it may even seem rather strange that females exhibit costly preferences for male ornaments that harm the male's survival, as often appears to be the case. Two theories have been proposed for those cases where females obtain no direct benefits (e.g., nuptial gifts or paternal care) by being choosy (Andersson, 1994). The first suggests that the advantage of choosiness lies in the fact that attractive males will father attractive sons, which will enjoy a high mating success (Fisher, 1930). As a consequence of the linkage disequilibrium between female-preference and male-ornamentation alleles, the high mating success of ornamented males confers a selective advantage to the alleles that determine a preference for the ornament. In a self-reinforcing manner, this may lead to the rapid establishment of male ornamentation and corresponding female mate preferences (Lande, 1981), a process also known as Fisher's runaway process (Fisher, 1930; Andersson, 1994). The second theory proposes that the benefit of choice lies in the fact that choosy females produce offspring with increased survival probabilities. According to this view, the male ornament provides information about the genetic quality of the male (Zahavi, 1975). By being choosy, females are more likely to mate with a male that carries 'good genes', which, once transmitted to the offspring, promote their survival.

The difficulty in speciation models is that speciation requires there to be not just a single female mate preference but variation in preferences (in the example, preference for light and dark plumage). Since sexual selection for good genes acts on a specific trait that acts as an indicator of genetic quality, it is not easy to see how good-genes sexual selection could, by itself, give rise to reproductive isolation. For instance, if plumage color provides information about good genes, then one would expect either light or dark plumage to signal high genetic quality, but not both at the same time. In contrast, Fisherian sexual selection acts on whatever character is currently perceived attractive by females. This may, in fact, be any arbitrary character, which makes it conceivable, at least in principle, that multiple Fisherian runaway processes occur at the same time within the same population (Lande, 1981). This can then lead to the rapid evolution of reproductive isolation within a single population (Turner & Burrows, 1995; Van Doorn *et al.*, 1998; Higashi *et al.*, 1999).

Apart from female mate choice based on genetic female mate preferences and genetic male secondary sexual characters, there are many other mechanisms that could give rise to assortative mating. An interesting example of such a mechanism is mate choice based on sexual imprinting (Aoki *et al.*, 2001), where preferences or display characters are acquired through exposure at a young age to the parents or other individuals (this appears to be widespread among birds). Also other aspects of cultural evolution, such as the learning of niche features, may generate strong assortative mating (Beltman *et al.*, 2004). As a third alternative, I mention mutual mate choice. Mutual mate choice can give rise to assortative mating in combination with good-genes sexual selection, even when all individuals have the same mate preference (high-quality individuals will only mate with a high-quality partner, forcing low-quality individuals to mate with another low-quality individual). Almeida & Vistulo de Abreu (2003) recently demonstrated the potential of mutual mate choice to drive speciation.

OBSTACLE 6 — FURTHER EVOLUTION TOWARDS FULL REPRODUCTIVE ISOLATION

The preceding five obstacles to speciation relate to conditions for the maintenance of genetic variation in a character responsible for reproductive isolation on a relatively short timescale (up to a few hundreds of generations; the ecological or population-dynamical timescale). However, the maintenance of genetic variation in a character that serves as basis for assortative mating is merely a first step towards speciation. Consider, for example, the right panel of FIGURE 10. Strong assortative mating and disruptive selection maintain a bimodal distribution of genotypes (*AAQQ* and *aaqq* are most common) but there are still many hybrid individuals and a high level of gene flow (as evidenced by the considerable frequency of *Aq* and *aQ* gametes). Full reproductive isolation can only be attained when assortative mating and disruptive selection are strengthened further. One could imagine this to occur through the substitution of the current alleles by new ones that increase the variation between the daughter species. This process lies beyond the scope of the model that was used. As in the other population genetic models used to illustrate the different obstacles to speciation (FIGURES 4, 8, 10 & 12), I assumed a fixed set of alleles to be initially present, and I did not explicitly model the origin of novel alleles by mutation. The substitution of existing alleles by novel ones typically occurs on a longer timescale (up to many thousands of generations; the evolutionary timescale).

Conditions for the maintenance of polymorphism on the ecological timescale are also necessary conditions for the long-term increase of phenotypic differentiation by subsequent mutation and allele-substitution events on the evolutionary timescale. However, they are not sufficient (Geritz *et al.*, 1998). In other words, it is conceivable that long-term evolution will lead to a gradual decrease of phenotypic variation, while a genetic polymorphism of alleles is all the time maintained. For instance, in the example of the seed-eating bird species, one could imagine the differentiation in bill sizes to decrease as the result of the evolution of alleles that allow for a more generalist feeding strategy.

The conditions for the long-term growth of phenotypic variation are not well characterized in a multi-locus context, except under idealized assumptions regarding the rate and phenotypic effects of mutations. Due to its technical nature, I defer a more detailed discussion of this issue to a later point in this thesis (CHAPTER 5). It suffices to say here that the strengthening of reproductive isolation and the completion of speciation are not straightforward consequences of the establishment of genetic variation in a reproductive isolating character. Increasing the genetic divergence between daughter species can usually only occur when additional conditions (e.g., on the precise nature of frequency-dependent selection or the structure of the mutation process) are met.

It is obvious that it will only be under rather special circumstances that selection can overcome all of the above six obstacles to speciation. This need not be problematic in itself: if disruptive selection would all but inevitably lead to speciation, we would probably find "nearly infinite numbers of species, a different species on every bush" (Felsenstein, 1981). The important question is whether selection is a more or less potent force in speciation than genetic drift and spatial isolation, the two main alternative factors that promote divergence. This question is still open to debate. It is immediately clear, however, that spatial isolation in one go creates strong reproductive isolation and conditions favorable to the maintenance of polymorphism. Hence, it may well be essential in the majority of speciation events.

DIVIDE AND BE RULED — A PLEA FOR INTEGRATION

The reductionist strategy – to solve problems by dividing them into smaller parts that can more easily be solved – has proven to be a powerful approach to gain understanding of complex systems. In speciation research, it has provided (partial) solutions to all of the obstacles discussed above. Unfortunately, an integration of these partial solutions is largely lacking, with the consequence that our understanding of speciation as a whole remains fragmented. To illustrate this, I will now briefly discuss two recent and widely cited models of sympatric speciation. The two models address the problem of speciation in two completely different ways and both offer important new insights. Rather than opposing the models based on their different view on speciation, I will argue that they in fact address complementary issues: the insights gained in each of the models are needed to resolve the weaknesses of the other.

'ON THE ORIGIN OF SPECIES BY SYMPATRIC SPECIATION' (DIECKMANN & DOEBELI, 1999)

Dieckmann & Doebeli (1999) consider a population in which individuals compete for a continuum of ecological resources (e.g., seeds of different sizes), distributed according to some fixed resource distribution function (FIGURE 13; gray distribution). Individuals do not consume all resources, but rather they are specialized to some extent on particular resources. This is reflected by an individual's resource utilization function (FIGURE 13; transparent distribution). The width of this function is equal for all individuals, but its optimum may be located at different points, depending on the ecological character of the individual (e.g., bill size), a character that is assumed to be heritable.

For speciation to occur, it has to be assumed that individuals can only efficiently consume a relatively narrow spectrum of resources (i.e., the width of the resource utilization function must be smaller than that of the resource distribution). If this is the case, directional evolution (FIGURE 13A) first drives the population to an optimum of the resource distribution function, where the carrying capacity of the population is maximized. Once there, however, individuals with deviating ecological characters strongly benefit from a reduced intensity of competition while only mildly suffering from the reduced availability of alternative resources (this is a consequence of the fact that the width of the resource utilization function is narrower than that of the resource distribution function). Hence, competition for resources induces disruptive selection (FIGURE 13B). At the same time, it intrinsically favors rare phenotypes over more common ones, a situation required for the maintenance of polymorphism.

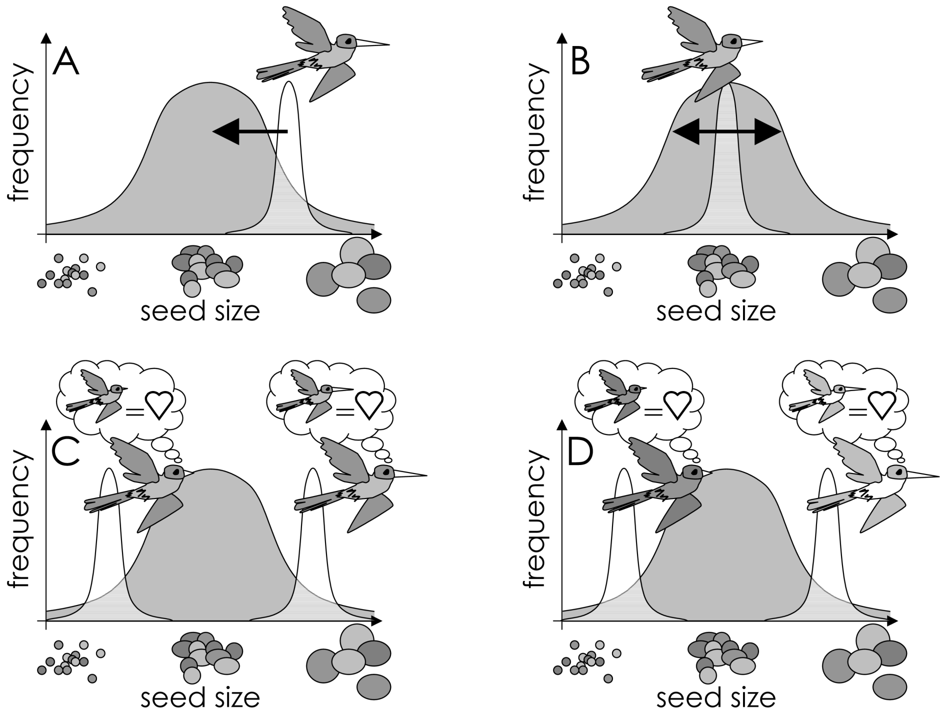

FIGURE 13 – SPECIATION À LA DIECKMANN & DOEBELI

After convergence to the ecological optimum (panel A), resource competition for a distribution of resources (gray distribution) generates disruptive selection (panel B) when the resource utilization function (transparent distribution) is narrow relative to the distribution of resources. Disruptive selection induces speciation only when individuals evolve to mate assortatively with respect to bill size (panel C), or plumage color, a neutral marker trait (panel D). The latter requires that a linkage disequilibrium develop between bill-size and plumage-color alleles.

In asexual populations this is sufficient for diversification to occur. Indeed, asexual populations will diverge into two or more discrete phenotypes after first having converged on the resource optimum (Metz *et al.*, 1996). This process, which has been named evolutionary branching (Metz *et al.*, 1996; Geritz *et al.*, 1998), cannot occur in sexual populations, at least as long as there is no assortative mating to counteract the homogenizing force of recombination (cf. FIGURE 7). Dieckmann & Doebeli (1999) considered assortative mating based on the ecological character

(FIGURE 13C) or on a selectively neutral marker trait (e.g., plumage color; FIGURE 13D). The strength of assortative mating was modified by another heritable character (the mating character). By selecting against intermediate phenotypes, resource competition provides a selective advantage to alleles that increase the tendency to mate with a phenotypically similar individual. As demonstrated by Dieckmann & Doebeli, this may lead to the evolution of strong assortative mating, allowing sexual populations to undergo speciation by evolutionary branching.

Together with its asexual predecessors (e.g., Metz *et al.*, 1996), Diekmann & Doebeli's model offers insights into the ecological and evolutionary processes that lead to the origin, the maintenance and long-term growth of polymorphism under disruptive selection (obstacles 2 & 6). By demonstrating that these processes at the same time generate selection for stronger assortative mating (obstacle 3), the authors established evolutionary branching as a paradigm for adaptive speciation in sexual populations, although a somewhat unsatisfactory feature of the model is that it relies heavily on a one-allele mechanism to strengthen assortative mating. When assortative mating is not based on the ecological character, but on a neutral marker trait, the model reveals the potential of stochastic fluctuations to generate initial linkage disequilibrium between the ecological character and the neutral marker. It is quite interesting that such stochastic fluctuations can apparently facilitate the evolution of substantial linkage disequilibrium between these characters. In view of obstacle 4, this effect deserves a more detailed investigation.

A weakness of the model is that it provides no mechanistic explanation for the presence of assortative mating itself (obstacle 5). It is simply assumed that individuals mate assortatively with respect to either the ecological trait or the marker trait, and that assortative mating can become very strong without any costs. Especially in the case that assortative mating is based on a neutral marker character, i.e., not on the character under disruptive selection, direct selection for random mating, which is likely to minimize the costs searching for suitable mates and so on, could easily override the indirect selection for stronger assortative mating generated by disruptive selection on the ecological character (a point already made by Udovic, 1980). In fact, it is well known from sexual selection theory that small costs of mate choice can dramatically decrease the potential for the evolution of non-random mating (Bulmer, 1989).

As a final point, Dieckmann & Doebeli preclude a number of possible responses to disruptive selection that one could imagine to interfere with speciation. For example, by considering the width of the resource utilization function as fixed, the model does not allow individuals to evolve broader resource utilization strategies in response to disruptive selection. By doing so, the model does not mechanistically address the establishment of a disruptive selection regime (obstacle 1). Similarly, one could consider the evolution of sexual dimorphism as a possible response to accommodate disruptive selection (Bolnick & Doebeli, 2003; Van Dooren *et al.*, in press). It is important to consider these alternative responses to disruptive selection and the constraints on evolution that are needed to preclude them, in order to assess the relevance of evolutionary branching as a general mechanism for speciation in sexual populations (Van Dooren *et al.*, in press).

'SYMPATRIC SPECIATION BY SEXUAL SELECTION' (HIGASHI *ET AL.*, 1999)

Higashi *et al.* (1999) focus on sexual selection as the driving force for divergence. They consider a species with a polygynous mating system. Each female chooses one male to mate with and reproduces once. Males may reproduce many times; all males are available to mate with every female. Mate choice is governed by two quantitative heritable characters. Females express a mating preference and males express a secondary sexual trait (e.g. plumage color) upon which female preference acts. A female maximizes her viability when she mates at random (i.e., when here preference is zero). Similarly, males maximize their survival when they do not express the secondary sexual trait (i.e., when their mating trait is zero). The expression of strong mating preferences is costly, as is the expression of an elaborated male mating trait. Yet, female preferences for exaggerated male mating traits can evolve due to Fisherian sexual selection. Starting from a randomly mating population (FIGURE 14A), the Fisherian runaway process may proceed either towards positive or to negative preference values; that is to say, females may either evolve preferences for positive (e.g., a light plumage; FIGURE 14B) or negative values (e.g., a dark plumage; FIGURE 14C) of the male mating trait. Under special circumstances, however, it is possible that Fisherian sexual selection proceeds in both directions at the same time. The simultaneous occurrence of two diverging runaway processes rapidly results in nearly complete linkage disequilibrium between female-preference and male-mating-trait alleles, which leads to strong assortative mating and reproductive isolation between the daughter species (FIGURE 14E).

Multiple runaway processes occur when the initial genetic variation of female preference is large (FIGURE 14D). This prerequisite is not surprising, since classical female-choice models (see Andersson, 1994) demonstrate that already a single runaway process will only occur if the initial level of choosiness exceeds a certain threshold value. It is therefore to be expected that, in order to trigger two simultaneous runaway processes, the level of choosiness for two distinct male traits has to be sufficiently high. Multiple preference alleles, coding for choosiness with respect to different male traits must therefore be present in sufficiently high frequencies, which requires that the population be highly variable for female preference. Higashi *et al.* (1999) account for the required high levels of initial variation by arguing that rapid and large changes in the environment may suddenly change the parameters of the model. This may cause previously hidden variation of female preference to become exposed, for instance, when males can suddenly be discriminated more easily.

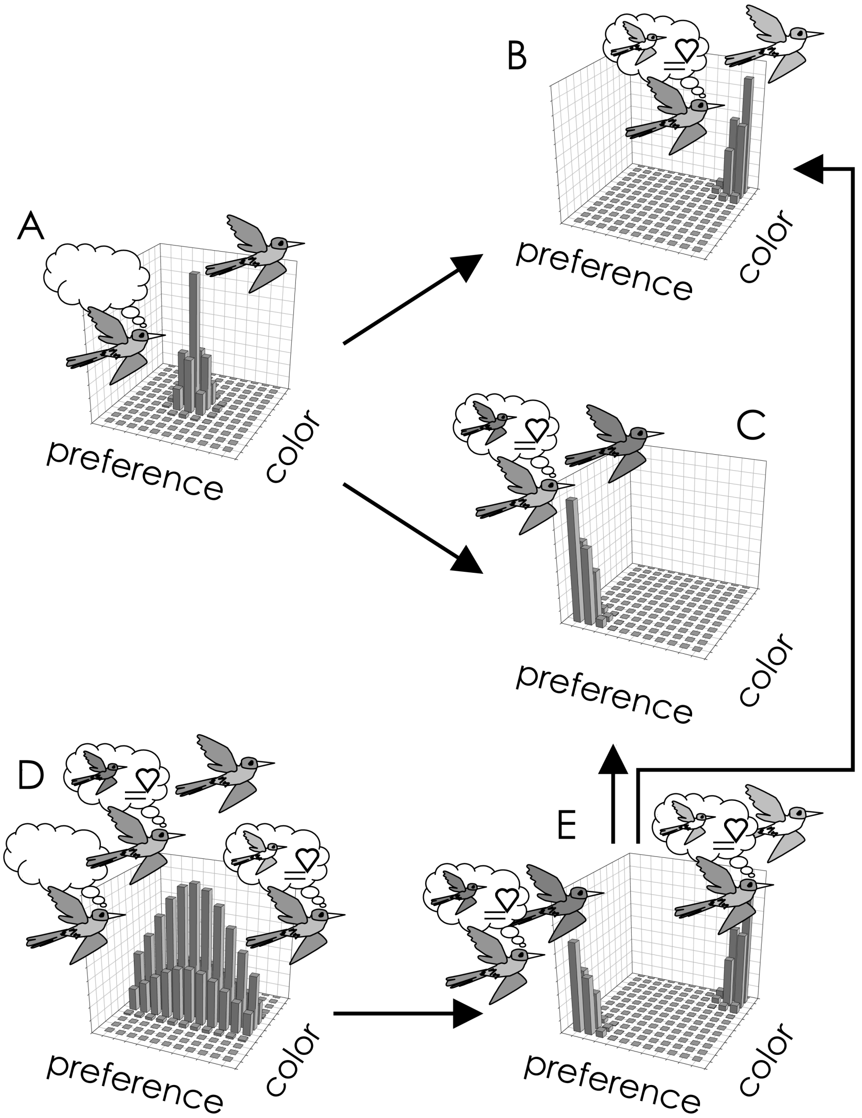

FIGURE 14 – SPECIATION À LA HIGASHI *ET AL*.

Starting from a population that mates at random (A), Fisherian runaway may lead to the establishment of mating preferences for light (B) or dark plumage (C). When the population is initially highly variable for female preference (D), two runaway processes may occur at the same time, leading to the rapid evolution of reproductive isolation (E). Unfortunately, coexistence of the two daughter species is not ensured, implying that one of them will rapidly go extinct.

Higashi *et al.* have provided proof-of-principle that sexual selection can generate disruptive selection (obstacle 1) and that it can rapidly generate nearly complete reproductive isolation (obstacle 3). Most importantly, their model offers an adaptive and mechanistic explanation for the evolution of strong assortative mating (obstacle 5). Sexual selection by mate choice acts directly on the characters responsible for reproductive isolation, which, in view of obstacle 4, renders it a potentially important driving force of speciation. However, Higashi *et al.* fail to provide an adaptive explanation for the origin and maintenance of variation in mating preferences. This not only forces the authors to rely on changes in the environment to initiate speciation. Also the end-state of the speciation process is unstable, a fact that becomes clear in a deterministic version of the model by the same authors (Takimoto *et al.*, 2000). All of this is due to a lack of frequency-dependent selection to promote the stable coexistence (obstacle 2) and further diversification (obstacle 6) of the daughter species.

A NICHE FOR THIS THESIS

Given a basis for reproductive isolation, ecological models of speciation illustrate that frequency-dependent selection is pivotal in creating and maintaining genetic diversity. Given the presence of genetic diversity, sexual selection models illustrate the potential of sexual selection to create a basis for reproductive isolation. Hence, in order to give full right to all obstacles to speciation, it is necessary to integrate the insights gained from ecological models (Dieckmann & Doebeli, 1999, and also Maynard Smith, 1966; Rosenzweig, 1978; Udovic, 1980; Felsenstein, 1981; Rice, 1984; Kondrashov, 1986; De Meeûs *et al.*, 1993; Doebeli & Dieckmann, 2000; Drossel & McKane, 2000; Fry, 2003) with those gained from sexual selection models of speciation (Higashi *et al.*, 1999, and also Karlin & Scudo, 1969; Lande, 1982; Wu, 1985; Turner & Burrows, 1995; Payne & Krakauer, 1997; Takimoto *et al.*, 2000; Takimoto, 2002). By the work presented in this thesis I aim to contribute to such integration, specifically by investigating frequency-dependent disruptive selection in a sexual-selection context.

The focus of this thesis on sexual selection is motivated by the empirical observation that many morphologically and genetically similar species differ markedly in secondary sexual characters (West-Eberhard, 1983; Seehausen & Van Alphen, 1999; Wilson *et al.*, 2000; Panhuis *et al.*, 2001). Molecular studies have shown a remarkable divergence of sex-related genes between closely related species (e.g., Vacquier, 1998). Comparative studies in birds have demonstrated species richness to correlate with the mating system (Mitra *et al.*, 1996), the degree of sexual differences in plumage (Barraclough *et al.*, 1995) and the degree of feather ornamentation (Møller & Cuervo, 1998). All of this suggests that sexual selection can act as a potent force in speciation, a suggestion that is supported by the theoretical argument that sexual selection is predisposed to generate reproductive isolation, since it acts directly on genes involved in mate recognition. This circumvents the problem of transmitting the force of disruptive selection to a reproductive isolating mechanism through linkage disequilibrium. Moreover, once a stable disruptive sexual selection regime has been established, a population has few other options than to undergo speciation. This is not so for disruptive natural selection, to which a population may also respond by evolving sexual dimorphism (Bolnick & Doebeli, 2003; Van Dooren *et al.*, in press), an option that, in fact, seems more likely to be realized than speciation (Van Dooren *et al.*, in press).

This thesis contributes in three different ways to a conceptual integration of ecological and sexual-selection theories for speciation. First, it demonstrates the need for such integration, by studying the evolution of ecological and mating strategies in a combined speciation model. We illustrate that the evolution of reproductive isolation through sexual selection, and the origin and maintenance of variation through frequency-dependent ecological selection are complementary processes that are both required for speciation.

Second, we identify an analogy between ecological resource competition, which has been shown to be capable of generating frequency-dependent disruptive selection (FIGURE 6), and competition for mating partners (FIGURE 15). In CHAPTER 3 of this thesis, we show that the similarity is more than superficial and that the analogy can be exploited, for instance, by deriving from the ecological literature on specialization conditions for the emergence of disruptive selection on male secondary sexual characters. Moreover, the analogy illustrates that an integration of ecological and sexual-selection theories for speciation is not only desirable but also natural.

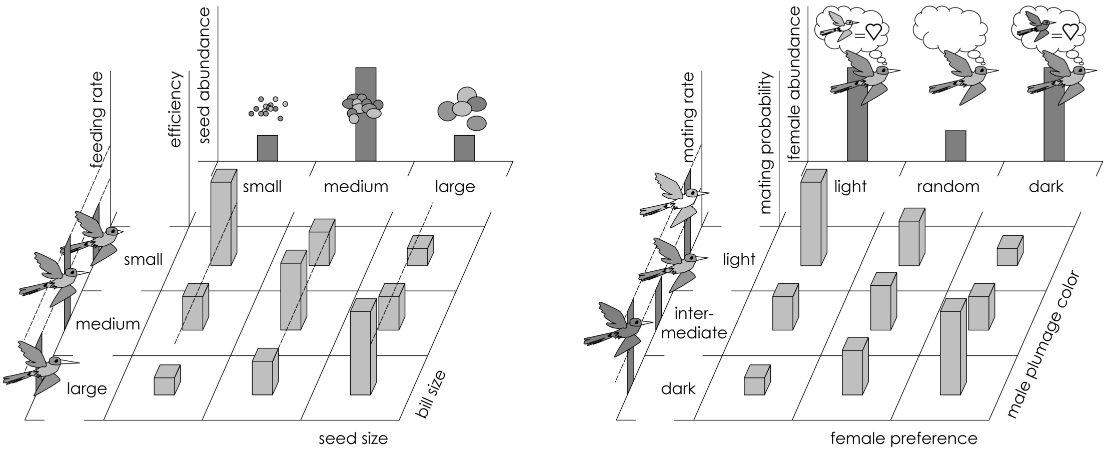

FIGURE 15 – COMPETITION FOR MATES IS AKIN TO ECOLOGICAL RESOURCE COMPETITION

Just as individuals compete for seeds (left), males compete for mating opportunities (right): there is a formal resemblance between the distribution of an ecological resource and the distribution of female preferences, between the feeding rates and the mating rates, and between the feeding efficiencies of individuals with different ecological strategies and the mating probabilities of males with different plumage characteristics.

Third, and finally, this thesis outlines conditions under which disruptive and frequency-dependent sexual selection is generated. By means of an example model, it is shown that such selection is capable of initiating speciation independently of non-adaptive processes, without the need to rely on high mutation rates or pre-existing variation in preferences. Moreover, at the end of the speciation process, stable coexistence of the daughter species is attained. Adaptive speciation by sexual selection occurs under conditions that are more restrictive than earlier models of speciation by sexual selection would appear to suggest. The main reason for this is that it relies on processes other than mate choice (e.g., intra-sexual competition or sexual conflict) to generate frequency-dependence. By identifying such processes as essential factors in the adaptive speciation process, we argue for an increased appreciation for previously ignored aspects of speciation by sexual selection, such as male-male competition, (indirect) competition between females, and various processes that limit the potential reproductive rates of males.

OUTLINE OF THE THESIS

This thesis consists of three parts. The first and largest part deals with speciation through sexual selection. The other parts contain contributions to aspects of sexual selection theory that are important to appreciate the role of sexual selection in speciation, but which are themselves not sufficiently well understood.

PART 1 – SPECIATION THROUGH SEXUAL SELECTION

In CHAPTER 2, we study the specific situation of marine invertebrate broadcast spawners. The model is used to explain the rapid evolution of gamete-recognition proteins that has been observed in several of these organisms. Gamete-recognition proteins appear to diversify most rapidly between closely related sympatric species, which suggests that they may play a role in speciation. The model demonstrates that competition for fertilizations can indeed induce strong disruptive selection on sperm proteins, which may lead to rapid diversification at the moment of speciation. The results of the model can be explained from the analogy between ecological resource competition and competition for mates: once mutation has caused the variation of egg receptor proteins (which act as 'resource' to the males) to exceed a certain threshold level, it pays males to specialize on extreme egg receptor types, since this allows them to avoid competition for fertilizations with other males.

The analogy between ecological resource competition and competition for fertilizations is formally demonstrated in CHAPTER 3. In addition, this chapter motivates the need to integrate ecological and sexual selection models of sympatric speciation. We investigate a combined model that incorporates ecological interactions and sexual selection. Speciation is initiated by the simultaneous diversification of ecological and mating strategies. Both types of diversification can be understood as the outcome of a competition process in which individuals compete for a spectrum of either ecological resources or mating opportunities. We illustrate that speciation relies crucially on the interplay between the evolution of ecological and mating strategies. This chapter also provides an introduction to the combined approach of individual-based simulations complemented by analytical approximations based on adaptive dynamics theory, a powerful approach that is also used in other chapters of this thesis.

The models investigated in chapter 2 and 3 rely on mutation pressure to create variation in female mating preferences. Speciation cannot occur when the variation in female mating preference is small. In order to create sufficient variation, selection on female preference must therefore be very weak. Though it is probably accurate to assume weak selection on female preference in specific situations (e.g., in marine broadcast spawners), one would in general not expect mutation pressure to be able to create sufficient variation in female preference.

For that reason, CHAPTER 4 introduces a model where mutation pressure cannot sustain appreciable levels of variation in mating preference. Hence, the required variation of female preference can only arise through selection. It is formally demonstrated that sexual selection cannot create and maintain variation of female mating preferences under the assumptions of standard mate-choice models. The reason for this is that mate choice (under the standard assumptions) does not generate frequency-dependent selection on female preference. Therefore, it does neither allow for the origin of variation in mating preference (speciation is not initiated), nor for its maintenance (even if speciation would be initiated, the daughter-species would not stably coexist). The problem can be fixed by deviating from the standard assumptions of mate-choice models such that competition between females is introduced. This creates a potential for the adaptive emergence of variation in mating preferences, but, at the same time, it counteracts the divergence of the male mating trait. Speciation can only occur when yet an additional source of disruptive selection, such as male-male competition, is included. In the final version of the model, where mate choice, competition between females and male-male competition act simultaneously, speciation is initiated and completed independently of non-adaptive processes. Such adaptive speciation does not suffer from the theoretical weaknesses associated with the current sexual selection models, but, obviously, it occurs under rather restrictive biological conditions. In the AFTERTHOUGHTS ON CHAPTER 4, we provide additional simulation results to support the assumption that male-male competition generates disruptive selection on males. We illustrate that, under suitable conditions, the strategies of males in male-male competition will evolve in such a way that rare male types are intrinsically favored in male-male competition.

The conditions for adaptive speciation through sexual selection are further investigated in CHAPTER 5, where an attempt is made to link the potential for adaptive speciation to the type and the structure of the interactions between individuals in the mating process. A mechanistic description of a broad class of mating processes is used to delineate two classes of models in which adaptive speciation by sexual selection can be observed: intra-sexual selection models, where intra-sexual interactions generate frequency-dependent selection in both sexes and inter-sexual selection models, where interactions between individuals of opposite sexes (particularly sexual conflict) give rise to frequency-dependent selection. The chapter offers general insights at a somewhat more abstract level, and it suggests many directions for further research. An effort has been made to make the general conclusions of the chapter accessible to a broad audience, but due to its technical nature, this chapter will nevertheless be quite demanding for readers without a background in mathematical biology (to whom I apologize for this).

Much of the analytical work presented in the chapters 2, 4 and 5, is based on approximations of multi-locus individual-based simulations. These approximations assume highly idealized genetics, which is often necessary to keep the mathematical analysis tractable. Although the good agreement between the simulations and their approximations gives some confidence in the robustness of the conclusions, it is, at present, unclear how accurate these approximations are. This is due to the fact that frequency-dependent disruptive selection has not been thoroughly studied in a multi-locus context.

The results presented in CHAPTER 6 help to understand the evolutionary dynamics of multi-locus traits under frequency-dependent disruptive selection. A multi-locus soft-selection model is studied as a simple example of a model where frequency-dependent disruptive selection is acting. The evolutionary dynamics observed in this model, combines features known from quantitative genetics models, where frequency-dependent disruptive selection leads to a broad continuous distribution of phenotypes, with those known from adaptive dynamics models, where it leads to a small number of discrete phenotypes. Overall, the potential of frequency-dependent disruptive selection to generate genetic polymorphism is smaller than one would naively expect: long-term evolution typically leads to genetic polymorphism at the smallest possible number of loci. This outcome somewhat justifies the use of single-locus approximations in speciation models.

PART 2 – GOOD GENES

Good-genes sexual selection is inherently less likely than Fisherian sexual selection to drive speciation through the divergence of mating preferences. As previously mentioned, this is due to the fact that Fisherian sexual selection acts on arbitrary traits, whereas sexual selection on good genes acts on specific traits that act as indicators of genetic quality. It is difficult to imagine multiple realizations of the same character to act as a signal of quality, and this makes it difficult to explain the divergence of mating preference for a quality indicator. Yet, sexual selection for good genes occurs in many species, and many empirical biologists judge it to act commonly alongside Fisherian sexual selection.

Even though sexual selection on a single indicator of genetic quality is perhaps unlikely to drive speciation on its own, it may still play a major facilitating role in speciation. As shown in CHAPTER 7, this is especially true for indicator traits that act as signals for local adaptedness. The evolution of mating preferences for such an indicator gives a mating advantage to locally adapted males, which reinforces the effect of disruptive natural selection. At the same time, it enables females to avoid maladapted males, which reduces the frequency of interbreeding between eco-morphs and strengthens assortative mating. Due to this double effect, sexual selection on good genes may act as a potent facilitator of speciation.

Another possibility for good-genes sexual selection to play a role in speciation is suggested by the observation that mate choice is often based on complex male courtship behavior or intricate morphological features, which, in most cases, involve multiple traits that signal genetic quality. Reproductive isolation could arise when some females in the population evolve strong mating preference for certain aspects of the male courtship display, whereas other females evolve preference for a different set of male characters.

Unfortunately, the evolution of female mating preferences for multiple indicators of quality is not yet well understood. For that reason, CHAPTER 8 investigates the conditions for the evolution of multiple preferences. Unlike previous models for the evolution of mating preferences for multiple indicators of genetic quality, we consider the possibility that different male ornaments provide information about different aspects of genetic quality, or the possibility that they provide different estimates of overall quality. This dramatically increases the potential for the evolution of female sexual preferences for multiple indicators of genetic quality even under the condition that the use of multiple preferences in mate choice is costly. We discuss our results in relation to different hypotheses for ornament diversity and identify parallels between Fisherian and good-genes mechanisms for the evolution of multiple ornaments.

The results of chapter 8 are extended in CHAPTER 9, where males are allowed to evolve optimal patterns of condition-dependent investment into their ornament. Although much is known about the evolutionary equilibrium properties of condition-dependent male signaling strategies, the joint evolutionary dynamics of female preference and male condition-dependent signaling has not been thoroughly characterized. Indeed, it is shown that sexual conflict over the information content of signals can lead to the continual evolution of female mating preference and male ornament expression. This phenomenon causes qualitative discrepancies with the predictions of existing theory, which is based on the assumption of equilibrium dynamics. Furthermore, it leads to the evolution of highly dynamic and complex signaling strategies and preferences, which may help to explain the apparent frequent loss of sexually selected traits and the evolution of intricate courtship displays.

We return to the subject of speciation in the AFTERTHOUGHTS ON CHAPTER 8 & 9. We investigate the potential for speciation through the divergence of mating preferences for multiple quality indicators by analyzing a two-patch model, where the information provided by different ornaments varies over the patches. The preliminary results suggest that speciation will only occur when migration between the patches is highly restricted. A similar conclusion can be drawn from simulations of the model of chapter 9 in a meta-population context. Divergence of mating preferences between the patches will only occur for extremely low migration rates.

PART 3 – MALE-MALE COMPETITION

The third part of this thesis concentrates on male-male competition. Unlike chapter 4 and 5, part 3 does not focus on the role of male-male competition in adaptive speciation, but on an issue that has no immediate link with speciation. CHAPTER 10 and 11 deal with the evolution of competitive strategies that lead to the establishment of dominance hierarchies, a phenomenon that has important consequences for sexual selection.

There exists disagreement as to which mechanisms are responsible for the emergence of dominance hierarchies. One could explain dominance hierarchies as a straightforward consequence of difference in fighting ability, but convincing arguments have been proposed to demonstrate that this explanation is incomplete. As an alternative, it has been argued that dominance hierarchies result from behavioral strategies referred to as winner- and loser effects. If winners of previous conflicts are more likely to escalate the current conflict, whereas losers are less likely to do so, arbitrary historical asymmetries between individuals can be reinforced to give rise to stable dominance relations.

The evolution of winner- and loser effects is investigated in CHAPTER 10. An idealized model of repeated aggressive interactions between pairs of individuals is developed, and the evolutionary equilibria of the model are characterized. It is shown that winner- and loser effects can indeed be stable endpoints of evolution, but alternative behavioral conventions are also possible. Asymmetries in fighting ability cause evolution to favor winner- and loser effects over these alternative solutions.

The model is extended to more than two players in CHAPTER 11. Repeated aggressive interactions among multiple players again favor the evolution of conflict resolution based on behavioral conventions, to avoid the costs associated with escalated fights. As in chapter 10, there exist several evolutionary solutions, which can give rise to either stable or highly dynamic social relations. Winner- and loser effects, which give rise to linear dominance hierarchies, are a likely outcome of evolution, especially when some individuals consistently have an advantage in escalated fights.

Part 1

Speciation through Sexual Selection

Sexual selection at the protein level drives the extraordinary divergence of sex-related genes during sympatric speciation

2

G. Sander van Doorn, Pieternella C. Luttikhuizen & Franz. J. Weissing

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ABSTRACT

An increasing number of molecular studies indicates that, in a wide variety of species, genes directly related to fertilization evolve at extraordinarily high rates. By means of a simple theoretical model, we try to gain insight in the dynamics of this rapid evolution and the underlying mechanisms. In the model, sexual selection and sympatric speciation act together to drive rapid divergence of gamete-recognition proteins. In this process, intraspecific competition for fertilizations enlarges male gamete protein variation by means of evolutionary branching, which initiates sympatric speciation. Additionally, avoidance of competition for fertilizations between the incipient species drives the rapid evolution of gamete-recognition proteins. This mechanism can account for both strong stabilizing selection on gamete-recognition proteins within species and rapid divergence between species. Moreover, it can explain the empirical finding that the rate of divergence of fertilization genes is not constant, but highest between closely related species.

INTRODUCTION

In a variety of species, sex-related genes show a remarkable pattern of molecular evolution. DNA sequence analysis has shown extraordinary divergence of fertilization proteins among closely related marine invertebrate species (Lee *et al.*, 1995; Metz & Palumbi, 1996; Biermann, 1998; Metz *et al.*, 1998; Hellberg *et al.*, 2000), in *Drosophila* (Tsaur *et al.*, 1998) and between higher primates (Wyckoff *et al.*, 2000). Other examples of rapidly evolving sex-related genes include mating pheromones in ciliates (Luporini *et al.*, 1995), mate recognition genes in *Chlamydomonas* (Ferris *et al.*, 1997) and several sex determining loci (Tucker & Lundrigan, 1993; Whitfield *et al.*, 1993). In many of these studies, selection pressures on the gene of interest were quantified by comparing the rate of substitutions per nonsynonymous site (D_n), with the rate of substitutions per synonymous site (D_s). A larger substitution rate at nonsynonymous sites ($D_n>D_s$) indicates rapid directed evolution (positive selection). For neutrally evolving genes, one expects $D_n \approx D_s$, whereas stabilizing selection translates into $D_n<D_s$. One would expect that sex-related genes are under stabilizing selection, but in fact -especially for male reproductive genes- D_n/D_s ratios larger than one (positive selection), sometimes even exceeding those of the rapidly evolving proteins of the immune system (Vacquier *et al.*, 1999), are frequently reported.

The evolutionary mechanism that causes rapid divergence of sex-related genes is poorly understood. Most empirical data are available for gamete recognition systems of marine invertebrate species. For these systems, several (mostly verbal) models have been suggested in order to explain positive selection on male gamete-recognition proteins. They all propose that sperm surface proteins evolve rapidly in order to maintain proper interaction with their continually changing cognate egg surface protein. For sperm proteins low degrees of polymorphism within species (Ferris *et al.*, 1997; Metz *et al.*, 1998) (indicating stabilizing selection) together with rapid divergence between species (positive selection) can then be explained as the result of a series of selective sweeps of favorable sperm-protein mutations in reaction to changes in the egg receptor.

However, a closer look at the available empirical data (summarized in FIGURE 1) reveals some aspects that cannot easily be accounted for by the explanations mentioned above.

1 – It is unclear which mechanism underlies the proposed continual change in the egg receptor. If selection, for instance caused by microbial attack of the egg cell surface (Vacquier & Lee, 1993) or sexual conflict (see Gavrilets, 2000) for a general mathematical model), drives the evolution of the egg receptor, one would expect to find positive selection in egg proteins too. In fact, the current limited amount of data indicates that egg surface proteins evolve under weakly stabilizing selection close to neutrality (Swanson & Vacquier, 1998). Alternatively, if genetic drift, possibly accelerated by concerted evolu-

tion (Swanson & Vacquier, 1998), underlies the continual change of the egg receptor, one can wonder how these neutral processes can drive the very rapid evolution of sperm proteins, and in our opinion a general solution of this paradox is impossible without a clear mechanistic explanation.

2 – If the rapid evolution of sperm proteins occurs continuously, one expects to find a more or less constant high *rate* of divergence over evolutionary time (Gavrilets, 2000), with the highest *degree* of divergence between the oldest species (note the distinction between *rate* of divergence and *degree* of divergence!). However, there is a relation between the *rate* of divergence and the time since speciation, with the strongest positive selection between the most recently speciated species (FIGURE 1).

3 – One expects to find divergence of gamete-recognition proteins within a species (between allopatric populations) and between allopatrically speciated species, but rapid divergence is found only between sympatric species and not between allopatric species. In Chlamydomonas, mating type and mate recognition genes are highly divergent between recently speciated species, but strictly conserved within a species, even for allopatric populations that have been separated for over 1 million years (Ferris *et al.*, 1997). A similar observation can be made from FIGURE 1, when comparing the allopatric *Arbacia* species with the sympatric *Strongylocentrotus* and *Echinometra* species. For abalones, the same pattern is present, although interpretation of the data is complicated by the fact that comparisons between allopatric abalone species typically involve more distantly related species for which positive selection may be more difficult to detect due to saturation effects (Lee *et al.*, 1995). Moreover, species that are now allopatric may have been sympatric at the time of speciation (and vice versa).

Based on these observations, we argue that the rapid evolution of gamete-recognition genes coheres with sympatric speciation and arises from interactions between the incipient species during the speciation process (for similar ideas see Palumbi, 1992; Metz & Palumbi, 1996; Ferris *et al.*, 1997). An important question to be answered here is whether sympatric speciation is the cause or consequence of the rapid evolution of gamete-recognition genes, or, what is the mechanism linking speciation and rapid evolution? On the one side of the spectrum of hypotheses is the idea that diversification of sex-related genes is promoted by selection against hybrids (i.e. as a consequence of speciation), on the other side is the idea that sexual selection on polymorphic mate recognition loci drives speciation (Wu, 1985). Here a theoretical approach can give useful insights in the underlying dynamics and evolutionary mechanisms. We therefore constructed a theoretical model in order to determine whether and how sexual selection and speciation are related to the rapid evolution of gamete-recognition genes.

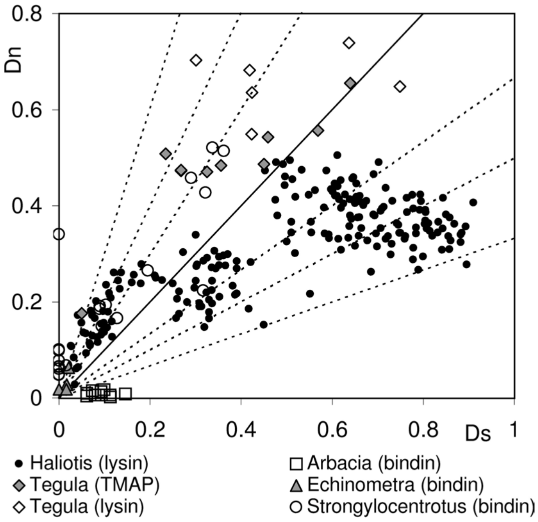

FIGURE 1 – SUMMARY OF EMPIRICAL DATA

For a number of marine invertebrate genera we plotted D_n and D_s values for a specific gamete-recognition protein (see legend). Data were taken from the literature (abalones: *Haliotis spp.* (Lee and Vacquier, 1992; Lee *et al.*, 1995), top snails: *Tegula spp.* (Hellberg and Vacquier, 1999; Hellberg *et al.*, 2000) and sea urchins: *Strongylo centrotus spp.* (Biermann, 1997, 1998), *Echinometra spp.* (Metz & Palumbi, 1996; Palumbi, 1999) and *Arbacia spp.* (Metz, Gómez-Gutiérrez and Vacquier, 1998)). Strongylocentrotus data are computed for the variable regions upstream and downstream of the conserved region of bindin (Biermann, 1997, 1998). Each point represents a comparison between two species. The solid line represents the neutral expectation (D_n=D_s), dashed lines are arbitrary reference lines of constant D_n/D_s ratio. For *Haliotis*, *Tegula* and *Strongylocentrotus* D_n/D_s ratios decrease with D_s. The estimated time of divergence based on mitochondrial DNA correlates with D_s for these genera, and therefore the data suggest that the signal of positive selection is highest for the most recently speciated species. The *Echinometra* species are too closely related to show this pattern. The genus *Arbacia* is the only group for which D_n/D_s ratios are very low. This genus also contrasts with the other examples in that the data concerns allopatric species.

The model aims to be general but is inspired by marine broadcast spawners (e.g. sea urchins, abalones). In these organisms, hybridization, mate selection and intrasexual competition for mates is determined largely by species-specific interactions between sperm and egg gamete-recognition proteins, without being blurred by complex behavioral interactions. Detailed knowledge is available on these gamete-recognition proteins and their interaction during fertilization (Vacquier, 1998).

THE MODEL

Our model incorporates a minimal description of the interaction between gamete-recognition proteins and an ecological component that allows for diversification without competitive exclusion, a prerequisite for speciation (Van Doorn *et al.*, 1998). We use an individual oriented model in which each individual is represented by three characters: a sperm protein gene, S, an egg surface protein gene, E and an ecological character, z. For simplicity, individuals are taken to be haploid and hermaphroditic, generations are overlapping.

Let N denote the population size, and let i, j, and so on, denote arbitrary individuals. An individual i is randomly selected from the population and it is determined whether i survives until reproduction. If so, i produces eggs, and the resulting offspring are added to the population. If not, i is removed from the population. This procedure is repeated N times per time step τ, for a large number of time steps.

More precisely, individuals survive with a probability depending on the amount of resource competition the individual experiences. Individuals with similar ecological characters compete stronger with each other than individuals with dissimilar z-values. Accordingly, i dies with probability d_i, where

$$d_i = \tau \left(d + c \sum_j \exp\left(-\frac{1}{2} \left(\frac{z_i - z_j}{\sigma_c} \right)^2 \right) \right). \quad [1]$$

Here, d denotes the basal death rate (from here on scaled to one) and c determines competition intensity.

Surviving individuals produce $b \cdot \tau$ eggs (b denoting the birth rate). We assume that all individuals compete to fertilize the eggs. The probability that j succeeds to fertilize i's eggs depends on the fertilization efficiency of j's sperm – a function $f\left(S_j, E_i\right)$ of the male sperm protein gene and the female egg surface protein gene – and the fertilization efficiencies of all competing sperm. More precisely, the probability $p_{i,j}$ that j's sperm will fertilize i's egg is taken as

$$p_{i,j} = \frac{f\left(S_j, E_i\right)}{\eta + \sum_k f\left(S_k, E_i\right)}. \quad [2]$$

Here, η is a constant determining the amount of sperm limitation. The larger η, or the lower the number of efficient sperm, the larger the chance that an egg is left unfertilized.

Characters S and E are modeled as bit strings of length L. In order to mimic the situation at the DNA level, odd bits in the bit string are defined to be nonsynonymous sites whereas even bits are synonymous sites, which have no phenotypic effect. The interaction between sperm protein and egg surface receptor during fertilization is modeled by bit string matching. Bit string S_j is bit wise compared with E_i, and the number of differences at **nonsynonymous** sites, $\delta\left(S_j, E_i\right)$ between the two is counted. Fertilization efficiency is taken to decay exponentially with $\delta\left(S_j, E_i\right)$ at a rate u, or

$$f\left(S_j, E_i\right) = u^{-\delta\left(S_j, E_i\right)}. \quad [3]$$

After fertilization, mutation occurs and the resulting offspring is added to the population. The ecological character z is assumed to be polygenic and inherits according to simple quantitative genetics: mean offspring character is the mean of the parent types and offspring variance is taken to be a constant σ_v^2. Characters S and E are treated as single genes. We assume full recombination between all characters (ecological trait z, S and E loci). Crossing-over within S and E loci is ignored. Bits in the bit string mutate at a rate μ (site^{-1} generation^{-1}).

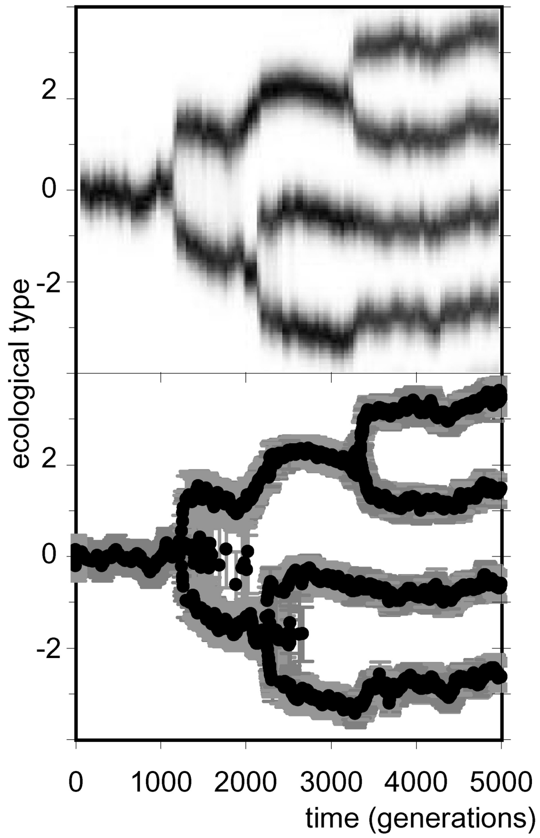

FIGURE 2 – EVOLUTION OF ECOLOGICAL TYPES AND MATING TYPES

The upper panel shows the frequency distribution of ecological types in time, with darker gray-levels indicating higher densities. Lower panel: analogously to the biological species concept, individuals were subdivided into reproductively isolated clusters according to dissimilarities between their gamete-recognition proteins. Mean ecological type (black dots) and standard deviation (gray bars) were calculated for each of these clusters separately. We used a single linkage clustering algorithm with $f(S_j,E_i)+f(S_i,E_j)$ as a distance measure between i and j. This algorithm sorts individuals into clusters for which the following is true: if individuals are assigned to different species, their fertilization efficiency is below a certain small threshold value. Ecological space is bounded from -4 to 4 with periodical boundary conditions. Parameters are $b=15$, $c=0.01$, $\sigma_v=0.2$, $\sigma_c=1.0$, $\mu=1.0\cdot10^{-4}$, $u=4.0$, $\eta=0.1$, $\tau=0.05$, $L=120$. For this choice of parameters, species consist of about 800 individuals.

SYMPATRIC SPECIATION

FIGURE 2 shows a representative run of our simulation program. The upper panel shows how the population splits into distinct ecological types, starting from identical individuals. In order to determine whether these ecological types are also reproductively isolated from each other, we divided the population into reproductively isolated groups according to a clustering procedure (Van Doorn *et al.*, 1998) on the gamete recognition sequences. This revealed distinct mating types, reproductively isolated from other mating types. As shown in the lower panel of FIGURE 2, these mating types correspond exactly with the ecological types, indicating that the population has split into ecologically distinct, reproductively isolated groups, and consequently, that sympatric speciation has occurred.

In order to determine the strength and nature of selection pressures we compared the evolutionary rates of substitution at synonymous and nonsynonymous sites of the sperm and egg receptor sequences. From all possible pair wise comparisons between individuals, the average number of synonymous and nonsynonymous substitutions in the sperm and egg receptor sequence (denoted $\Delta_s(S)$, $\Delta_s(E)$, $\Delta_n(S)$ and $\Delta_n(E)$) was determined. From this, the rate of nonsynonymous substitutions per nonsynonymous site (D_n) and the rate of synonymous substitutions per synonymous site (D_s) can be calculated.

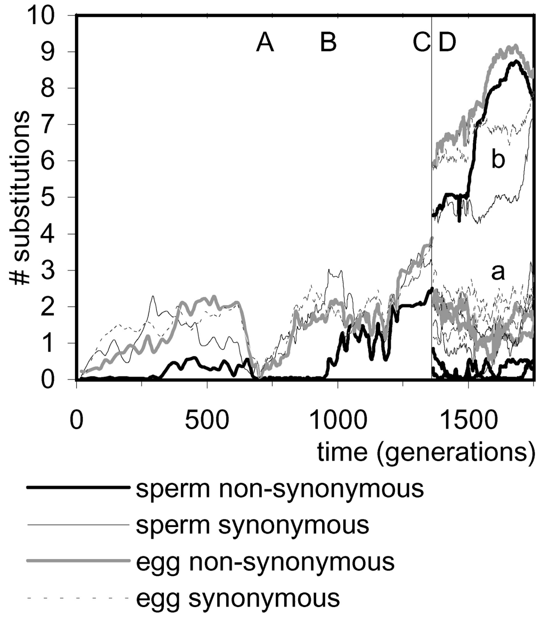

FIGURE 3 – TIME SERIES OF THE AVERAGE NUMBER OF SUBSTITUTIONS

Before speciation occurs (time = 1360 generations), the graph shows a trait substitution (time = 0 – 700 generations) and the onset of speciation. After speciation, substitutions were counted within (a) and between (b) species. Letters A-D are used in TABLE 1.

For a number of simulations (summarized in TABLE 1), the evolution of the gamete recognition sequences was followed. The evolutionary dynamics shows characteristic patterns, which occur periodically and may or may not result in speciation. As an example, FIGURE 3 shows the pattern resulting from the simulation in FIGURE 2. At time 0, the population starts with a period of low sequence variation. In the course of time (0…300 generations) neutral variation $(\Delta_s(S),\Delta_s(E))$ increases, together with $\Delta_n(E)$, because sperm availability, which is limiting only at very low fertilization efficiencies, produces only weak selection on the egg receptor.

Sperm protein variation $\Delta_n(S)$ at first remains small, but increases suddenly as egg receptor protein variation exceeds a certain threshold value (time=330 generations). This can be understood by realizing that for sperm not an absolute measure of fertilization efficiency is important but a relative one: sperm has to compete with other sperm to fertilize the eggs. Therefore, the optimal sperm protein type depends on the strategies of other sperm and the distribution of egg receptor types. When egg receptor variation is limited, there is a single optimal sperm type. Then, selection on sperm proteins is strongly stabilizing and the population is almost monomorphic for sperm protein. As egg receptor variation increases, mutant sperm proteins can invade, which specialize on egg receptors that are inefficiently fertilized by the wild-type sperm. Such mutants are less general, but they partly avoid competition with the wild-type sperm. Now, selection on the sperm protein is suddenly positive, because competition for fertilizations between wild-type and mutant sperm favors mutants that are more different from the wild type and vice versa.

In the meantime, ecological resource competition is in play. If the different gamete-recognition proteins do not become correlated with ecological types, competitive exclusion occurs, which may result in a substitution of the wild type by the mutant (trait substitution, FIGURE 3, time=700 generations). This process results in low overall sequence variation and restores the population to a state qualitatively similar to the initial state. Alternatively, as shown in FIGURE 4, a correlation between ecological types and gamete-recognition proteins arises. In that case, the different mating types in the population start to specialize on different ecological resources, and the subpopulations separate in ecological space too, eventually evolving into different species (FIGURE 2 & 3, time=1360 generations). In a typical run, cycles of trait substitutions alternate with speciation events.

TABLE 1 – SELECTION PRESSURES BEFORE, DURING AND AFTER SPECIATION[a]

	time period[b]					
	within species			between species		
	A-C	A-B	B-C	D-F	D-E	E-F
$\Delta_n(S)$	2.21±0.14	0.02±0.001	2.19±0.14	6.79±0.28	3.56±0.14	3.22±0.25
$\Delta_s(S)$	2.57±0.21	1.04±0.13	1.53±0.23	7.05±0.60	2.50±0.31	4.55±0.48
$\Delta_n(E)$	2.85±0.15	0.97±0.08	1.88±0.09	6.29±0.33	2.45±0.22	3.84±0.34
$\Delta_s(E)$	2.66±0.19	1.04±0.16	1.62±0.18	6.89±0.45	1.85±0.26	5.04±0.46
selection on sperm[c] (D_n/D_s)	weakly stabilizing (0.86)	strongly stabilizing (0.02)	positive (1.43)	neutral (0.96)	positive (1.42)	weakly stabilizing (0.71)
selection on egg[c] (D_n/D_s)	neutral (1.07)	neutral (0.93)	positive (1.16)	neutral (0.91)	positive (1.32)	weakly stabilizing (0.76)

a) Averaged over 15 simulations we computed the number of substitutions (±SEM) and average rates of substitution for sperm and egg synonymous and nonsynonymous sites.

b) A-F denote characteristic points in time: A,B,C,D as in FIGURE 3; E: 500 generations after speciation, F: start of the next speciation event.

c) Selection regimes are classified as follows: positive ($D_n/D_s \geq 1.1$), neutral ($0.9 \leq D_n/D_s < 1.1$), weakly stabilizing ($0.5 \leq D_n/D_s < 0.9$), strongly stabilizing ($D_n/D_s < 0.5$).

During speciation, the two daughter species continue to segregate in ecological and protein-sequence space, until both interspecific resource competition and interspecific sperm competition for eggs are minimized. The latter is the driving force behind the rapid divergence of sperm protein types. In this process, the distribution of egg receptor sequences widens further and gradually evolves into a bimodal distribution matching the diversifying sperm proteins. TABLE 1 summarizes how the selection pressures on sperm and egg proteins vary over time. For the sperm protein, selection pressures change from strongly stabilizing before speciation (TABLE 1, time period A-B) to positive during speciation (time period B-C). For the egg receptor, these differences are far less pronounced. Between species comparisons reveal that selection is positive during the initial phase after speciation (TABLE 1, time period D-E), whereas it becomes weakly stabilizing afterwards (time period E-F).

The mechanism of speciation, as explained above, is shown schematically in FIGURE 5.

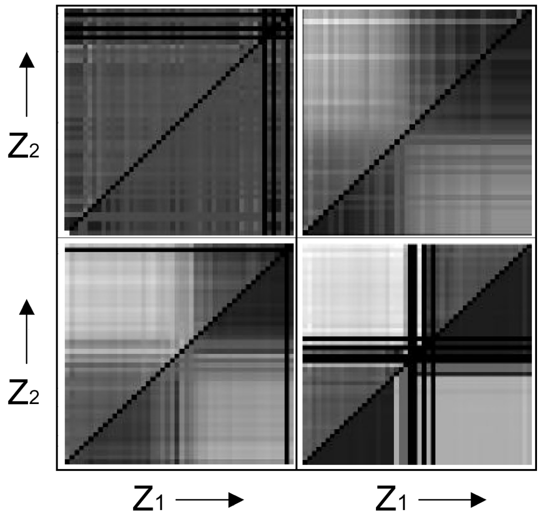

FIGURE 4 – THE DEVELOPMENT OF A CORRELATION BETWEEN MATING TYPES AND ECOLOGICAL TRAITS DURING A SPECIATION EVENT

The dissimilarity between individuals of ecological type z_1 and individuals of ecological type z_2, measured as the average number of non-synonymous substitutions, is indicated as a gray-level at (z_1,z_2) in a two dimensional space (above the diagonal: egg receptor, below the diagonal: sperm protein). Lighter gray-levels indicate a larger dissimilarity (larger number of substitutions), completely black indicates that there were no individuals of that particular ecological trait present at that time. Before speciation (upper left, 200 generations before speciation) there is no correlation between ecological traits and recognition proteins. During the speciation process (upper right, 100 generations before speciation; lower left, at speciation) the population splits into two groups with low variation within the groups, and larger variation between groups. Finally, (lower right, 100 generations after speciation) the two groups have completely separated and intermediate types start to disappear.

ROBUSTNESS OF THE RESULTS

Here we only show simulations for one set of parameters, and parameters were chosen such that speciation occurs on a short timescale. However, the proposed mechanism is general, and therefore the results presented here are not expected to depend sensitively on the details of our model or on the precise choice of parameters. In fact, additional simulations, for other parameter combinations and other model assumptions (such as diploid organisms and different underlying genetics) together with analytical results (CHAPTER 3) have confirmed the results presented here. Speciation occurs for a wide range of parameters, provided that egg receptor variation can get sufficiently large. Quantitatively this means that the variation in egg receptor types has to exceed the variation of the egg utilization distribution (FIGURE 5), a scale that is determined by u. For a given set of parameters, this condition requires that selection on the egg receptor must be sufficiently weak so as to allow the variation of egg receptor types to become sufficiently large (CHAPTER 3). This implies that η must be sufficiently small and consequently, sperm limitation is not severe. Although sperm limitation is considered a major selective force, recent empirical work on natural populations of marine free-spawning organisms suggests that sperm limitation might not be as severe as initially suspected (Yund, 2000). Furthermore, sequence comparisons of the egg receptor gene in abalones revealed that it is indeed subjected to weak selection and that it is polymorphic in several species (Swanson & Vacquier, 1998; W.J. Swanson, personal communication). The other parameters are important in determining the timescale on which speciation occurs: the rate of speciation will be higher in larger populations and for higher mutation rates. Moreover, other factors, such as spatial structure or the details of the molecular structure of the egg receptor, which were left out of consideration here, are likely to play an important role in determining the timescale of speciation.

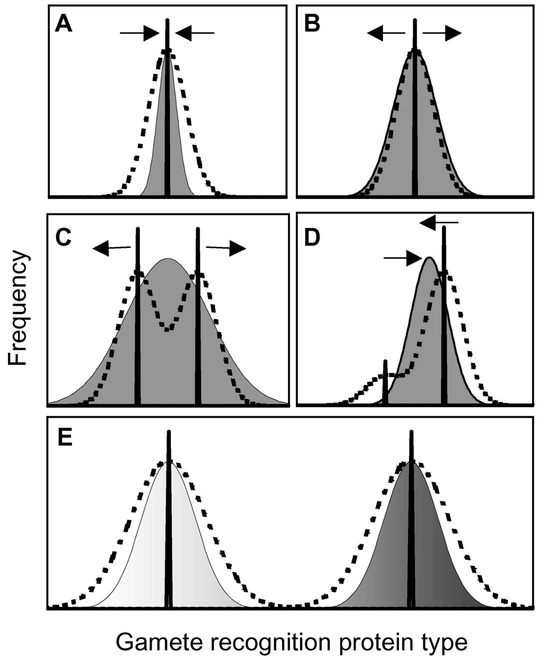

FIGURE 5 – A SCHEMATIC REPRESENTATION OF THE MECHANISM OF SPECIATION

A) when the width of the distribution of egg receptor sequences (gray) is small, selection on sperm will be stabilizing (arrows) and consequently, the distribution of sperm protein types will be very narrow (thick black line). The spectrum of egg receptor proteins that are efficiently fertilized by the available sperm, from here on denoted as egg utilization distribution is drawn as a dashed line. B) as soon as the variation of egg receptor types exceeds the width of the egg utilization distribution, selection on sperm becomes disruptive and evolutionary branching of the sperm protein type occurs. This process is driven by competition between males for fertilizations similar to the way in which competition for ecological resources causes evolutionary branching in Dieckmann & Doebeli's (1999) model of sympatric speciation. C) after branching, the variation of egg receptor sequences increases further, which allows sperm protein sequences to diverge further, thus increasingly lowering competition for fertilizations between the two male types. During this stage of the speciation process the population evolves into two increasingly reproductively isolated groups, which can only persist if ecological traits become correlated with the gamete-recognition types (E, ecological trait depicted as grayscale). Otherwise, one of the sperm protein types outcompetes the other, resulting in a trait substitution (D).

CONCLUDING REMARKS

In our model, sexual selection at the level of gamete-recognition proteins and sympatric speciation are interwoven processes. Intraspecific competition for fertilizations enlarges sperm protein variation, which initiates sympatric speciation. Additionally, avoidance of competition for fertilizations between the incipient species drives the rapid divergence of gamete-recognition proteins. This single mechanism can account for the different selective regimes for male and female gamete-recognition proteins, the paradox between stabilizing selection within a species versus positive selection between species, the link between sympatric speciation and the rapid evolution of gamete-recognition genes and the patterns of divergence in evolutionary time.

ACKNOWLEDGEMENTS

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Ecological versus Sexual Selection models of Sympatric Speciation: a Synthesis

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G. Sander van Doorn & Franz J. Weissing

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ABSTRACT

Sympatric speciation is a composite phenomenon requiring both ecological differentiation and the evolution of a mating structure that induces reproductive isolation. Ecological and sexual selection models have addressed these two aspects of sympatric speciation separately. We briefly discuss the recent results of these models and argue that the evolution of ecological and mating strategies are mutually dependent processes rather than independent phenomena corresponding to incompatible views of sympatric speciation. Then, we consider a combined model incorporating ecological interactions and sexual selection. In this model, sympatric speciation is initiated by simultaneous evolutionary branching of ecological strategy, leading to ecological differentiation, and mating strategies, resulting in assortative mating. Both types of evolutionary branching can be understood as the outcome of a competition process in which individuals compete for a spectrum of either ecological resources or mating opportunities. Speciation is completed when a linkage disequilibrium between ecological and mating types splits the population into two ecologically differentiated and reproductively isolated groups. Using a combined analytical and individual-based simulation approach, we illustrate the different dynamical regimes and characterize the necessary conditions for sympatric speciation in the model.

INTRODUCTION

The recent empirical and theoretical interest in sympatric speciation has produced a multitude of theoretical models (e.g., Kawecki, 1997; Payne & Krakauer, 1997; Van Doorn *et al.*, 1998; Dieckmann & Doebeli, 1999; Higashi *et al.*, 1999; Kondrashov & Kondrashov, 1999; Drossel & McKane, 2000). Some of these models are very specific (e.g., Van Batenburg & Gittenberger, 1995), others are more general, but all of them conclude that sympatric speciation is theoretically very well feasible (for recent reviews see Via, 2001, Turelli *et al.*, 2001). This conclusion is in striking contrast to the conclusions based on classical models of sympatric speciation (e.g., Maynard Smith, 1966; Felsenstein, 1981; Rice, 1984), which almost universally discarded sympatric speciation as a plausible mode of speciation (Via, 2001). Yet, superficially at least, the recent models are quite similar to the classical models.

This paradox is resolved by two recent theoretical developments. These developments originate from different lines of research, which address two long-standing difficulties in the theory of sympatric speciation (Kondrashov & Mina, 1986). First, sympatric speciation requires, almost by definition, the evolution of a specific mating structure enabling reproductive isolation. Classical models (Maynard Smith, 1966; Felsenstein, 1981; Rice, 1984) had problems to explain the evolution of assortative mating under general and plausible conditions. More recently (Wu, 1985; Liou & Price, 1994; Van Doorn *et al.*, 1998; Higashi *et al.*, 1999; Takimoto *et al.*, 2000), it has been shown that these problems can be overcome if sexual selection is the driving force behind the evolution of reproductive isolation. Second, reproductive isolation is not sufficient to ensure the sympatric coexistence of daughter species. In view of the ecological principle of competitive exclusion, the species can only survive if reproductive isolation is associated with ecological differentiation. Only recently (Metz *et al.*, 1996; Geritz *et al.*, 1998), evolutionary branching theory has provided a plausible mechanism for the evolution of ecological polymorphism in the presence of disruptive selection.

Based on these new insights, a new generation of 'ecological' and 'sexual selection' models of sympatric speciation has been developed. These approaches will be briefly reviewed below. Unfortunately, an integration of both research lines has not yet been achieved. Ecological speciation models (reviewed by Schluter, 2001) focus on ecological differentiation without much attention for the mechanisms underlying the evolution of mating structure. Sexual selection models (reviewed by Panhuis *et al.*, 2001) focus on the process leading to reproductive isolation, usually neglecting ecological divergence. We will argue that both approaches present mutually dependent rather than conflicting explanations of sympatric speciation. To provide a conceptual bridge between them, we will present and analyze a model that integrates the ecological and sexual selection aspects of sympatric speciation. Our main objectives are to investigate the origin of ecological polymorphism and the evolution of mating strategies within the same formal model, in order to characterize the conditions under which sympatric speciation occurs, to investigate the mutual dependence of ecological differentiation and the evolution of assortative mating, and to identify the common mechanism underlying these two aspects of sympatric speciation.

THE EVOLUTION OF POLYMORPHISM IN THE PRESENCE OF DISRUPTIVE SELECTION

The starting point of ecological models is that sympatric speciation results from disruptive selection. However, disruptive selection alone is not sufficient for speciation to occur. Consider, for example, a species that has access to a range of alternative habitats (e.g., from wet to dry) and assume that, due to external factors, individual fitness is highest in the extreme habitat types and lower in intermediate habitat types. Accordingly, selection is disruptive and one might expect that the population will split into two ecotypes, one specialized on living under wet conditions, and the other specialized on living under dry conditions. In contrast to this expectation, however, such a population will become monomorphic for one of the specialist strategies. In fact, the population will only experience disruptive selection if it starts exactly at the fitness minimum. If the initial state is slightly shifted towards one of the extremes, the population will experience directional selection enhancing the initial bias.

Hence, at first sight at least, populations tend to evolve away from fitness minima where selection is disruptive. This fundamental problem has only recently been resolved (Abrams *et al.*, 1993; Metz *et al.*, 1996), at least for asexual populations. The resolution is based on the insight that selection is usually not externally imposed, as in our example, but frequency dependent. Moreover, selection pressures may vary in strength and direction in the course of evolution, as a result of a feedback between evolutionary and ecological processes. Under such circumstances, evolution may drive the population towards a point where it experiences disruptive selection (Abrams *et al.*, 1993), which subsequently induces polymorphism (Metz *et al.*, 1996). This phenomenon is named 'evolutionary branching'.

To explain this further, we will now consider the example of resource competition as a general ecological interaction that can give rise to evolutionary branching (FIGURE 1). In line with recent models (e.g., Metz *et al.* 1996; Doebeli & Dieckmann, 2000), let us assume that individuals compete for a continuum of ecological resources, distributed according to some fixed resource distribution function (shown in gray in FIGURE 1). Individuals compete for resources locally in resource space, that is to say, individuals do not consume all resources but rather they are specialized to some extent on particular resources. This is reflected by an individual's resource utilization function, the location of which is determined by a quantitative, heritable trait that we will refer to as the individual's ecotype. As competition affects fitness, ecotype is under natural selection, the direction and intensity of which depends on the resource distribution and on the frequency and resource utilization characteristics of the set of ecotypes present in the population.

Consider a population that is monomorphic for a certain ecotype that does not match the ecological optimum of maximal resource availability (FIGURE 1A). Such a population is not evolutionarily stable: a mutant that is closer to the ecological optimum will be favored by selection because it utilizes resources that are more abundant. Eventually, such a mutant will outcompete the resident ecotype, and in a series of such mutation/substitution events, evolution will drive the population towards the peak of the resource distribution.

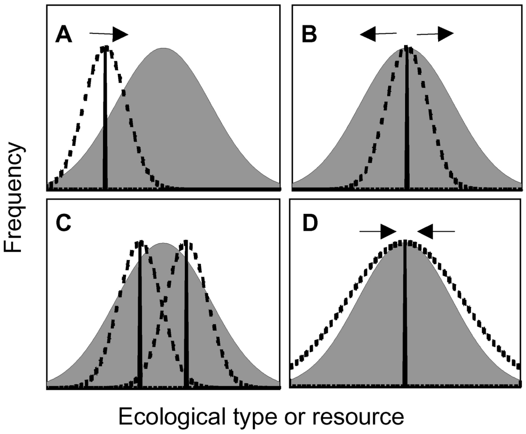

FIGURE 1 – EVOLUTIONARY BRANCHING IN COMPETITION MODELS

Individuals compete for resources that are distributed according to a fixed resource distribution function (shown in gray). An individual's ecotype corresponds to the location of its resource utilization curve (dashed line), which delimits the spectrum of resources that can be utilized. (A) A population that is monomorphic for ecotype (solid line) will evolve towards the peak of the resource distribution. (B) In the case of an ecological specialist, i.e. if the width of the resource distribution is larger than the width of the resource utilization curve, the population experiences disruptive selection once located at the peak of the resource distribution. (C) This leads to evolutionary branching, after which the population becomes dimorphic for ecological type. (D) In the case of an ecological generalist, the population will evolve towards the peak of the resource distribution and remain there.

Once there, however, the population experiences disruptive selection (FIGURE 1B): because of the specialist resource utilization strategy of the population, the resources in the tails of the resource distribution are hardly competed for. Mutants that utilize the tails of the resource distribution gain a competitive advantage, which more than outweighs the lesser availability of those resources. Such mutants can invade the population, which is therefore evolutionarily unstable, and, in this sense, located at a fitness minimum.

The population can only escape from this fitness minimum if it undergoes evolutionary branching and becomes dimorphic for ecotype (FIGURE 1C), since any monomorphic population would be driven back to the ecological optimum again. Evolution eventually leads to a stable situation where selection for avoidance of competition with the other ecotype balances selection towards the ecological optimum.

The mechanism sketched above works only for ecological specialists, with a narrow utilization curve relative to the distribution of available resources. For an ecological generalist (FIGURE 1D) the competitive advantage of utilizing resources in the tails of the resource distribution will be much smaller than in the specialist case, since the generalist still competes for those resources rather efficiently. Therefore, a mutant that utilizes resources not at the ecological optimum will suffer more from the disadvantage of the lesser availability of those resources than it benefits from its competitive advantage, and selection will therefore be stabilizing towards the ecological optimum. Consequently no mutants can replace the resident ecotype at the ecological optimum, and in this sense, the monomorphic resident population is evolutionarily stable.

The intuition behind the occurrence of evolutionary branching of ecological strategies in resource competition was confirmed, at least for asexual populations, by a mathematical formulation based on Lotka-Volterra type population dynamics (Metz *et al.*, 1996). In fact, evolutionary branching has been shown to occur in a variety of mathematical models of asexual populations in different ecological settings, and can therefore be considered a general explanation for the evolution of polymorphism in the presence of disruptive selection (Doebeli & Ruxton, 1997; Geritz *et al.*, 1999; Kisdi, 1999).

THE PROBLEM OF RECOMBINATION

A solution of the problem of the origin of polymorphism under disruptive selection does not solve the whole problem of sympatric speciation. In sexual populations, a second problem arises. As soon as a polymorphism originates in a sexual population, it will immediately be destroyed when mating is random. This is because mating between different ecotypes will yield intermediary and less fit hybrids, and the random recombination of genotypes will reshuffle co-adapted gene complexes. In order to overcome these problems, assortative mating is required.

However, it is not self-evident that assortative mating will evolve and whether the specific association between ecological and mating type loci will develop. These issues were addressed in several classical models of sympatric speciation (Felsenstein, 1981; Rice, 1984). These models have shown that the required association between ecological type and mating type can, in principle, evolve, but only under conditions of strong linkage or pleiotropy between ecological and mating loci, such that, essentially, ecological and mating characters are determined by a single locus or trait. Such a scenario may apply to certain biological systems, but in general weaker pleiotropic interactions are to be expected (Felsenstein, 1981). For weak pleiotropic interactions between ecological and mating loci it requires unrealistically strong disruptive selection to overcome the randomizing effects of recombination.

Dieckmann & Doebeli (1999) argue that these problems can partially be overcome by a stochastic, individual based description of the process. In their model, assortative mating did evolve in a finite population located at a branching point, leading to both divergence of ecotypes and reproductive isolation. This occurred for competitive and various other types of ecological interactions (Doebeli & Dieckmann, 2000). Hence, it is possible, at least in principle, that a sexual population undergoes evolutionary branching. However, selection for assortative mating is weak in Dieckmann & Doebeli's models, and only acting at the branching point. This is because only a phenomenological description of mating behavior is given and the mechanism underlying assortative mating is not specified. It is more plausible that assortative mating is the outcome of the evolution of male and female mating strategies. This issue is being addressed in the recent sexual selection models of sympatric speciation.

SEXUAL SELECTION AND THE EVOLUTION OF ASSORTATIVE MATING

Sexual selection models, which explicitly take into account the interaction of male and female mating strategies, typically assume different male and female sex roles: female reproductive success is largely determined by the quality of the offspring produced, whereas male reproductive success is limited by the number of females that can be fertilized. Because of these asymmetries, there will be strong competition for fertilizations among the males and females will exert mate choice if this enables them to mate with a higher quality male (Andersson, 1994).

In the context of speciation, models have focused on the evolution of female preferences for male ornaments by runaway sexual selection. This mechanism, originally proposed by Fisher (1930) as a verbal argument, and later confirmed by theoretical models (O'Donald, 1980; Lande, 1981; Kirkpatrick, 1982), proposes that male traits and female preferences for those traits will become genetically correlated because of non-random mate choice. Once this correlation is established, female preference for more extreme male traits will result in more extreme female preferences, because of a correlated selection response. Because of this positive feedback, preference and trait coevolve in a rapid runaway process, until halted by counteracting natural selection pressures.

Runaway sexual selection is interesting for speciation for two reasons. First, the evolution of preferences for male ornaments provides a mechanism of strong pre-zygotic reproductive isolation. Second, and in contrast with good genes models, the direction of the runaway process is arbitrary. This could result in rapid evolution of reproductive isolation between allopatric populations (Lande, 1981). For sympatric speciation however, there would have to be simultaneous runaway processes in different directions within a single population. In a previous model, specifically addressing speciation of cichlid species (Van Doorn *et al.*, 1998), we showed that simultaneous runaway processes within a single population are attainable. Moreover, Higashi *et al.* (1999) and Takimoto *et al.* (2000) provided an important proof of principle that sexual selection alone is sufficient to split a population into two reproductively isolated groups.

There are however a number of problems. First, it is not clear how the reproductively isolated daughter species can coexist in the absence of ecological differentiation. Second, there is the fundamental problem that disruptive sexual selection has similar properties as disruptive natural selection, and it is a similarly delicate affair to maintain a long-term polymorphism in the presence of disruptive sexual selection as it is for disruptive natural selection. In the models, this translates for instance into neutral stability of relevant equilibria and requirements of symmetric parameter conditions, large initial genetic variance of female preference and male trait or very strong selection.

EVOLUTIONARY BRANCHING OF MATING STRATEGIES

The problem of maintaining polymorphism in the presence of disruptive selection is similar for both natural and sexual selection. This similarity leads one to wonder whether processes analogous to the evolutionary branching in the ecological models could also occur as a consequence of sexual selection, resulting in the evolutionary branching of mating strategies. In fact, in another paper (CHAPTER 2 of this thesis) we analyzed a model in which such mating type branching does indeed occur, resulting in a stable polymorphism of mating strategies.

In order to explain this further, let us now consider a verbal model in which male and female mating strategies are determined by heritable mating types. Let us also assume the typical sex roles: all males compete to fertilize a female, and a female chooses a male (actively or passively) based on the compatibility of male and female mating strategies, according to some mate choice or fertilization efficiency function. The model is very general and reflects a variety of specific examples ranging from female preference (female mating type) for male ornaments (male mating type) in lekking birds to the interaction between gamete recognition proteins in marine broadcast spawners (Vacquier, 1998), where sperm proteins (male mating type) interact with egg surface proteins (female mating type) during fertilization.

Under these assumptions, selection on males will be much stronger than selection on females. In FIGURE 2A we consider an extreme case, in which there is a variety of female mating types present in the population (gray distribution), and, because of stronger selection, just a single male mating type (solid black). The spectrum of female mating types that can be efficiently fertilized by the male mating type is indicated by the dashed line, representing the fertilization efficiency function. If we assume that fertilization efficiency is highest when male and female mating types match, then selection on male mating type will drive it towards the maximum of the female mating type distribution. In addition, the distribution of female mating types is not fixed, and evolution will act on it towards optimal matching with male mating type, although the selection pressure on female mating type will be much weaker than on male mating type. When male and female mating type are matched, selection will be stabilizing as long as the distribution of female mating types is narrow, as in FIGURE 2B. In that case, males can fertilize the full distribution of female mating types with reasonable efficiency. Hence, the competitive advantage of specializing on extreme female mating types does not outweigh the disadvantage of the lesser abundance of those female mating types.

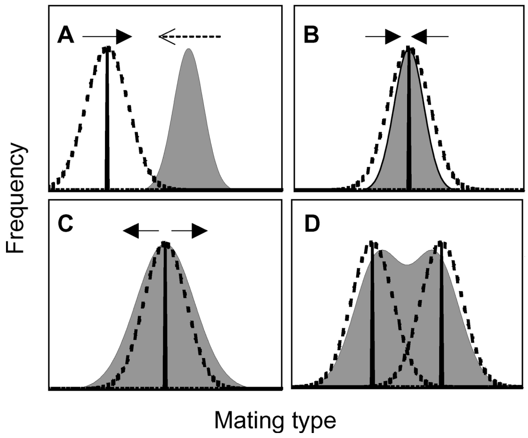

FIGURE 2 – EVOLUTIONARY BRANCHING OF MATING STRATEGIES

In (A), we consider a population that is monomorphic for male mating type (solid black line). Males compete to fertilize a spectrum of female mating types (shown in gray). Fertilization efficiency is highest when male and female mating type match, as reflected by the fertilization efficiency function (shown as a dashed line). This results in strong selection on male mating type, and similar, but much weaker, selection on the female mating type distribution, towards optimal matching of male and female mating types. (B) If the variation of female mating types is small, there is a single optimal male mating type and the population will experience stabilizing selection. (C) When the variation of female mating types becomes larger, however, it pays to specialize on females that are not that efficiently fertilized by the resident male mating type. Then selection becomes disruptive, and the population undergoes evolutionary branching (D), inducing further widening of the female mating type distribution, and the subsequent evolution of assortative mating.

However, when selection of female mating type is sufficiently weak, the distribution of female mating types may widen by mutation pressure, beyond a point where selection on male mating types becomes disruptive (FIGURE 2C). Then, mutant males that specialize on the extreme female mating types can invade, since these are hardly competed for by the resident males, and the population will undergo evolutionary branching of male mating types.

After evolutionary branching, the distribution of female mating types slowly adapts to the dimorphic distribution of male mating types, widening further, which allows male mating types to separate even more, thus lowering competition for fertilizations (FIGURE 2D). In the end, assortative mating can evolve, as a result of linkage disequilibrium between male and female mating type genes.

Note that there is a biological analogy between competition for ecological resources and competition for fertilizations (as also reflected by the analogous choice of notation in FIGURES 1 & 2), which –in the verbal models at least- extends to analogous evolutionary dynamics for ecotype and male mating type.

A SYNTHESIS OF ECOLOGICAL AND SEXUAL SELECTION MODELS

In order to understand sympatric speciation, we will eventually have to analyze the interplay between ecological and mating type branching. As argued above, ecological branching requires assortative mating, the evolution of which might be explained by sexual selection. On the other hand, sexual selection models require ecological differentiation for the coexistence of incipient species. Therefore, it is very likely that in real world systems, both sexual selection and ecological processes will play a role in sympatric speciation (Galis & Metz, 1998). Based on these arguments, we will now proceed by writing a formal model of sympatric speciation. The model will incorporate a minimal description of mating behavior and ecological interactions, which will allow us to further analyze evolutionary branching of mating strategies and ecological branching within the same framework.

GENERAL MODEL STRUCTURE

We consider the evolutionary dynamics of three continuous, heritable, phenotypic traits: ecotype (denoted x), female mating type (denoted p) and male mating type (denoted q). Let us focus on an arbitrary female i. When the female is ready to mate, all males compete to fertilize her. The probability that a particular male j succeeds to fertilize the female is proportional to the male's 'attractivity' for female i, denoted a_{ij}. We keep the model as general as possible and make no assumptions regarding the mechanism of female choice: attractivity and mate choice may be based on any active or passive process (behavioral, morphological or other) that affects the probability that a female mates with a particular male. We assume that attractivity is highest when male and female mating types match. Moreover, we allow for the possibility that attractivity might also be higher when i and j are of similar ecotype, for instance, when individuals occupying similar ecological niches are more likely to meet one-another. Therefore, we take

$$a_{ij} = g_m(p_i - q_j)\, g_e(x_i - x_j), \qquad [1]$$

where here and henceforth g_a denotes a Gaussian function with mean zero and standard deviation σ_a. In particular, the standard deviations of the Gaussian distributions used here, σ_m and σ_e, determine the specificity of mate choice with respect to mating type and ecotype differences respectively (for an overview of the parameters used in the model, consult TABLE 1). In the limit of large σ_e, mating probabilities are independent of ecological differences and determined solely by male and female mating types. Alternatively, in the limit of large σ_m, mating is assortative with respect to ecotype without any dependency on male and female mating types.

When the female cannot find an attractive male, she does not mate. This occurs with a probability that increases with η. This parameter determines the strength of direct selection on female mating preference. When $\eta = 0$, females will always mate, regardless of their mating preference, and hence there is no direct selection on female mating type p. In contrast, when $\eta > 0$, for instance when searching for mates is costly, or when sperm is limiting, selection will act to match female with male mating type.

Under these assumptions, the probability that female i mates with male j, denoted as α_{ij} is given by

$$\alpha_{ij} = \frac{a_{ij}}{\eta + \sum_{\text{males } k} a_{ik}}. \tag{2}$$

A fertilized female produces b offspring. Offspring mortality until reproductive age is determined by the intensity of ecological resource competition. Individuals compete for a continuum of ecological resources distributed according to a fixed Gaussian function $g_K(x)$. The intensity of resource competition between two individuals i and j is taken to decline as a Gaussian function $g_c(x_i - x_j)$ of the difference between their ecotypes. This reflects the assumption that individuals with dissimilar resource utilization strategies compete less intensely. More precisely, competition induced mortality, m_i, is taken to be directly proportional to the intensity of competition with all other individuals and inversely proportional to resource availability

$$m_i = \gamma \frac{\sum_k g_c(x_i - x_k)}{g_K(x_i)}, \tag{3}$$

where the parameter γ scales the carrying capacity of the system. As argued before (see FIGURE 1), the width of the resource utilization function, σ_c, relative to the width of the resource distribution, σ_K, determines the competitive regime.

We also include direct viability selection on male mating type as an extra source of mortality for males. We normalize male mating type in such a way that $q = 0$ is the optimal mating type for survival, and multiply the survival rate, $1 - m_i$, by an extra Gaussian factor, $g_s(q)$, for males. Adult males and females die at a constant rate. We assume that females reproduce only once during their lifetime. Males may reproduce several times, by fertilizing multiple females.

We will analyze the model by combining two approaches: individual based computer simulations and mathematical analysis. Using the computer simulations, we will illustrate the different types of dynamical behavior of the model. Subsequently, we will try to gain more insight in the processes underlying sympatric speciation by studying a special case of the model using adaptive dynamics methods. In this analysis, we will derive predictions for the parameter conditions under which sympatric speciation occurs. Finally, we will test the robustness of these predictions in the simulation model again, which will enable us to study the effects of stochasticity and more complicated genetics on the model outcome.

TABLE 1 – IMPORTANT MODEL PARAMETERS AND THEIR BIOLOGICAL INTERPRETATION

parameter	biological interpretation
b	birth rate
ε, η	cost of mate choice ($\varepsilon = \eta/\text{number of males}$)
σ_K	width of the ecological resource distribution
σ_c	width of the resource utilization function
σ_e	specificity of mate choice with respect to ecological type
σ_m	specificity of mate choice with respect to mating type
σ_ν	width of the female mating type distribution
σ_s	strength of viability selection on male mating type.
σ_v	width of the distribution of mutation sizes
μ	population average female mating type

SIMULATION RESULTS

Simulations were run with overlapping generations, with the following additional assumptions: x, p and q are fully heritable traits that are each determined by a diploid locus. All genes are unlinked and alleles interact additively. Offspring genotypes are determined according to normal Mendelian genetics. We assume a continuum of alleles, that is, the phenotypic effect of each allele is a continuous quantity. Mutation is modeled by altering the phenotypic effect of each allele every generation and independently by a number drawn from a normal distribution with a narrow width σ_v.

We ran simulations for parameter combinations that either precluded or allowed for evolutionary branching. Based on our verbal arguments, we may expect branching of ecotypes (FIGURE 1) to occur in a specialist resource utilization scenario. Quantitatively, as was shown in Doebeli & Dieckmann (2000), this means that the width of the resource utilization function should not exceed the width of the resource distribution, that is, $\sigma_c < \sigma_K$. Moreover, the specificity of mate choice with respect to ecotype, σ_e, may also affect the conditions for ecological branching. We chose to vary the width of the ecological resource distribution, σ_K, in order to simulate a specialist and a generalist resource utilization scenario. Similarly, conditions for mating type branching (FIGURE 2) are likely to be affected by the width of the female mating type distribution relative to the width of the fertilization efficiency function, σ_m. This balance is affected by the width of the mutation distribution, σ_v, the cost of female mate choice, η, and, of course, σ_m. The latter parameter was varied in order to simulate high and low specificity of mate choice.

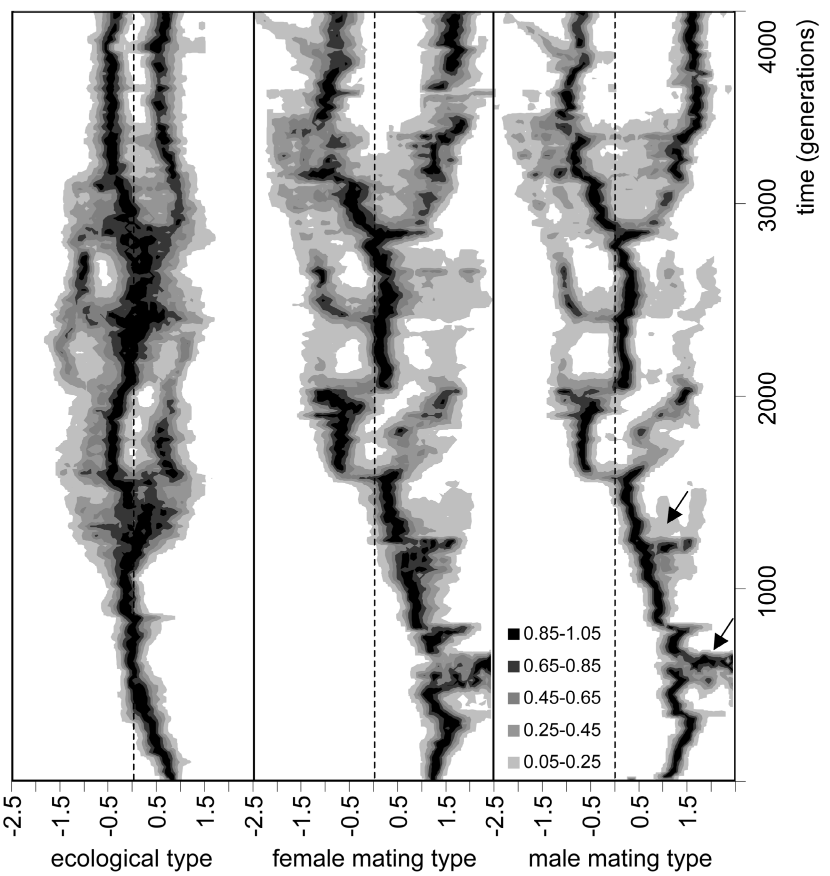

FIGURE 3 – SCENARIO 1: SPECIATION

The three panels show the distribution of ecological and mating types (grayscale) during 4000 generations of evolution. In the first phase of evolution, ecotype evolves towards the ecological optimum (dashed line). There, the population experiences disruptive selection (the variation of ecological types increases), but cannot undergo evolutionary branching, because assortative mating has not yet evolved. During this initial stage, (<1500 generations) male and female mating type evolve jointly towards the optimum for male survival (dashed line). Several times, polymorphisms of mating types originate (arrows), but these are unstable due to competitive exclusion and viability selection against extreme male mating types. After 1500 generations, simultaneous branching of ecotype and mating strategies repeatedly splits the population into groups that are ecologically differentiated and, at the same time, reproductively isolated. Such branching events may therefore be interpreted as sympatric speciation events. Parameters were $\sigma_c = 0.4$, $\sigma_K = 1.2$, $\sigma_e = 0.6$, $\sigma_m = 0.2$, $\sigma_v = 0.02$, $\sigma_s = 1.0$ $\eta = 1.0$, $b = 4.0$. Furthermore, parameter $\gamma = 5 \cdot 10^{-4}$ kept the population sizes in the simulations close to about 1000 individuals per species.

Sympatric speciation occurs only under conditions that allow for evolutionary branching of both ecotype and male and female mating type (FIGURE 3). In that case, the population splits into distinct clusters, where each cluster can be interpreted as a species, since it is characterized by a unique combination of ecotypes and mating types. Within a species, male and female mating type match with one another, while there are large differences in mating types between species. Because of these mating type differences, species are reproductively isolated from each other. Male and female mating types are highly correlated with each other across the population, as a result of the evolutionary dynamics. This effectively results in assortative mating to a degree high enough to allow for the evolutionary branching of ecotypes and the simultaneous build up of a linkage disequilibrium between the ecological and mating loci. The same processes occur in multilocus simulations (data not shown) where ecological and mating types are coded by multiple loci. In that case, assortative mating is strong enough to overcome the randomizing effects of recombination between ecological loci, which allows for evolutionary branching of ecotypes and speciation.

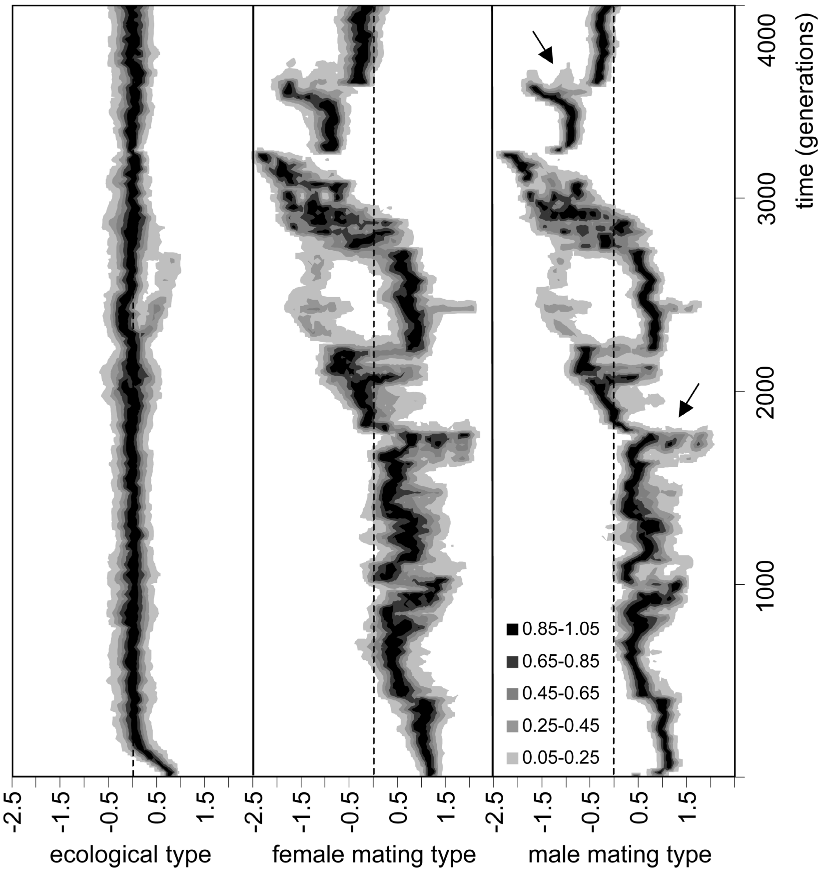

FIGURE 4 – SCENARIO 2: COMPETITIVE EXCLUSION

In this simulation, parameters are as in FIGURE 3, except that $\sigma_K = 0.6$. Now, individuals are ecological generalists and branching of ecological type does not occur. Because of this, polymorphisms in mating types (arrows) cannot persist, due to competitive exclusion.

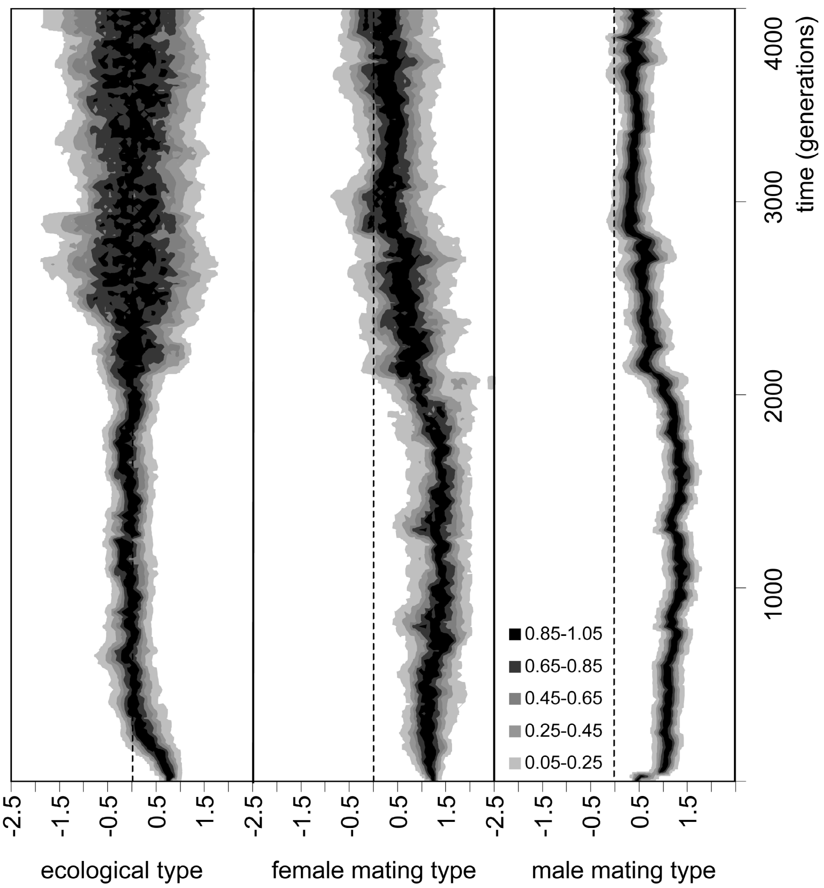

FIGURE 5 – SCENARIO 3: RANDOM MATING

When male and female mating types do not undergo evolutionary branching, as in this simulation, branching of ecological type is excluded. This is because mating is random without polymorphisms of mating types, and random recombination of ecological type genes prevents evolutionary branching. Note that selection on ecological types is still disruptive after convergence to the point of highest resource abundance, as evidenced by the large variance of ecological types. Parameters are as in FIGURE 3, except that $\sigma_m = 0.4$.

Under conditions that preclude the occurrence of ecological branching, the different mating types cannot stably coexist, because competition for ecological resources will drive all but one of the mating types to extinction (FIGURE 4). Alternatively, if there is no polymorphism of mating types, individuals mate randomly. In that case, the evolution of distinct, reproductively isolated ecotypes is prevented (FIGURE 5). Nevertheless, random mating does not preclude the evolution of ecological polymorphism, as evidenced by the broadening of the distribution of ecological types after the ecological optimum has been reached (FIGURE 5). This observation is in line with other models (Kisdi & Geritz, 1999), which suggest for our single locus simulation that disruptive selection at the ecological optimum should result in the evolution of distinct allele types and a stable polymorphism of genotypes. Note that, in our simulations, we do not observe a polymorphism of discrete types (as in Kisdi & Geritz, 1999), but a broad continuous distribution of ecotypes. This is explained by the smearing that occurs as a result of a rather strong mutation pressure. In multilocus simulations a similarly broad distribution is observed, even under weak mutation pressure, due to recombination.

In order to get more insight in the process of speciation, we need to study the underlying processes of evolutionary branching of ecotype and mating types in more detail. In the next section, we will attempt to find the conditions that are required for both types of evolutionary branching by considering a special case of the simulation model, which will allow us to use the methods of adaptive dynamics.

ADAPTIVE DYNAMICS APPROXIMATION

In our simulations, the variation of female mating types is typically larger than the variation of male mating types (FIGURES 3–5). This can be understood by realizing that the selection pressures on male and female mating types are qualitatively different. For males, the number of females fertilized is the main determinant of fitness, and therefore there is strong competition for fertilizations. Selection is strongly frequency dependent, since a male's reproductive success depends not on his own mating type per se, but rather on its performance in competition relative to the other male mating types present. For females, reproductive success is independent of the strategies of other females and depends solely on the compatibility between female mating type and the male mating types present. If, as we assumed, females are not severely limited by the availability of suitable males (i.e. η is small), then selection pressures on female mating type will be weak. Consequently, the distribution of female mating types will be wider than the distribution of male mating types.

As an approximation of this situation, we will now consider a model in which female mating types vary according to some continuous distribution and where male mating type and ecological type are monomorphic. In order to keep the analysis of the model tractable, we furthermore restrict ourselves to a special case of the simulation model, where traits x, p and q are coded by a single-locus haploid genotype. As in the simulation model, individuals reproduce sexually, allowing for recombination between genotypes. We assume that population size is sufficiently large to allow for a deterministic description of the evolutionary dynamics. Later, we will show that important qualitative as well as some quantitative results derived for this special case apply in general.

INVASION-PROOFNESS AND ATTAINABILITY

We apply standard adaptive dynamics theory (Metz *et al.,* 1996; Geritz *et al.,* 1998) and consider the dynamics of a rare mutant, which differs in either male mating type or ecological type from the resident population. The question is under what conditions this mutant can invade the resident population. The answer to this question will depend on the mutant phenotype (denoted as y), but also on the resident phenotype (denoted $\hat{y}$), reflecting the effect that the resident has on the biotic and abiotic environment in which the mutant invades. Formally, the invasion prospects of a rare mutant depend on the mutant's long term per capita growth rate $\lambda(y,\hat{y})$, also referred to as invasion fitness (Metz *et al.*, 1996; Geritz *et al.*, 1998). If $\lambda(y,\hat{y})>\lambda(\hat{y},\hat{y})$, the mutant can invade and a new population dynamical equilibrium will be established, where, usually but not always, the resident is replaced by the mutant. Otherwise, the resident population is proof against invasion by y. If we assume that mutations occur only rarely and in small, discrete steps, this will result, on a longer timescale, in an evolutionary dynamics that consists of a series of such invasion events each followed by the establishment of a new population dynamical equilibrium.

In the case that mutants are only slightly phenotypically different from the resident, the evolutionary dynamics can be derived from the local behavior of the invasion fitness function. In order to do so, we compute the selection gradient

$$\lambda_y(\hat{y})=\left.\frac{\partial\lambda(y,\hat{y})}{\partial y}\right|_{y=\hat{y}}, \qquad [4]$$

which can be interpreted as follows: if the selection gradient is positive (negative), mutant types that have a higher (lower) phenotypic value than the resident will have a higher fitness, and therefore a selective advantage with respect to the resident. Such a mutant can invade the population and replace the resident. This process is repeated when new mutants arise, and, in a series of mutation/substitution events, evolution will proceed in the direction of the selection gradient.

Interesting resident strategies are those strategies for which directional selection is absent, i.e. strategies for which the selection gradient is zero. Such strategies are referred to as evolutionarily singular strategies (Metz *et al.*, 1996). In generic cases, an evolutionarily singular strategy either cannot be invaded by any mutant strategy, or, alternatively, it can be invaded by all mutants. In the former case, all mutant strategies will have a lower fitness than the evolutionarily singular strategy, which can therefore be characterized mathematically as a fitness maximum with respect to the mutant strategy. In other words, an evolutionarily singular strategy y^* is invasion-proof, when

$$\left.\frac{\partial^2\lambda(y,\hat{y})}{\partial y^2}\right|_{y=\hat{y}=y^*}<0. \qquad [5]$$

In the latter case, when the evolutionarily singular strategy can be invaded by all mutants, y^* corresponds to a fitness minimum with respect to the mutant strategy and the sign in [5] is reversed.

In the literature, an invasion-proof strategy is often called an evolutionarily stable strategy (ESS; Maynard Smith & Price, 1973). However, an ESS is not necessarily stable in the dynamical sense (Eshel, 1983; Metz *et al.*, 1996; Taylor, 1996; Weissing, 1996). In fact, condition [5], which characterizes invasion proofness, does not ensure that the ESS will actually be attainable as the endpoint of a series of mutation/substitution events. There are examples of systems, in which arbitrarily small perturbations away from an ESS will cause the evolutionary dynamics to diverge from that ESS. Therefore, in addition to the invasion-proofness, we also need to distinguish attainable (or 'convergence stable' *sensu* Taylor, 1996a) singular points (evolutionary attractors) from dynamically unstable singular points. An evolutionarily singular strategy y^* is attainable if evolution proceeds towards higher $\hat{y}$ when $\hat{y} < y^*$, and towards lower $\hat{y}$ when $\hat{y} > y^*$. Since the direction of evolution is given by the sign of the selection gradient, equation [4], attainable evolutionarily singular points can be characterized by the condition

$$\left. \frac{d\lambda_y(\hat{y})}{d\hat{y}} \right|_{\hat{y}=y^*} < 0 . \qquad [6]$$

Using conditions [5] and [6], the local evolutionary dynamics around any resident strategy $\hat{y}$ can be classified (Geritz *et al.*, 1998). An interesting phenomenon occurs when an evolutionarily singular strategy is attainable but not invasion-proof. That is to say, a series of mutation/substitution events converges to the evolutionarily singular strategy, but at that strategy, the population can be invaded by all mutants. In that case, the population is trapped at a fitness minimum, from which it can only escape when it undergoes evolutionary branching and becomes dimorphic, since any monomorphic population would be driven back to the evolutionarily singular point again (Metz *et al.*, 1996).

CONDITIONS FOR ECOLOGICAL BRANCHING

After these general arguments, let us now proceed to derive the adaptive dynamics of ecotype and male mating type in our model. As is derived in detail in APPENDIX A, the growth rate of an ecological type mutant $(x,\hat{q})$, in a resident population $(\hat{x},\hat{q})$ is given by the expression (approximated for weak selection on females)

$$\lambda(x,\hat{x}) \approx -1 + \frac{1}{4}\left(b - (b-2)\frac{g_c(x-\hat{x})\,g_K(\hat{x})}{g_K(x)} \right)\left(1 + g_e(x-\hat{x})\right). \qquad [7]$$

This equation can best be understood by considering the two extreme regimes of small and very large birth rates. When b is small, the population can just sustain itself, and in that case the reduction of mate encounter rate caused by ecological differences governs the evolution of ecotype. Indeed, in that case equation [7] reduces to

$$\lambda(x,\hat{x}) \approx -\frac{1}{2} + \frac{1}{2} g_e(x-\hat{x}), \ \text{for}\, b \approx 2 . \qquad [8]$$

As a consequence, $\lambda(x,\hat{x}) < \lambda(\hat{x},\hat{x})$. Selection will act against all mutant strategies, since all mutant males will suffer from reduced mate encounter rates.

In the regime of a very large birth rate the expression for the mutant growth rate reduces to

$$\lambda(x,\hat{x}) \propto 1 - \frac{g_c(x-\hat{x})\,g_K(\hat{x})}{g_K(x)} \quad \text{for large } b. \qquad [9]$$

Note that the sign of the invasion fitness, and therefore the fate of the mutant, is completely determined by the competitive advantage of the mutant relative to the resident.

In the general case [7], the evolutionarily singular ecotypes can be found by computing the selection gradient

$$\left.\frac{\partial\lambda(x,\hat{x})}{\partial x}\right|_{x=\hat{x}} \approx -\frac{b-2}{2\sigma_K^2}\hat{x}. \qquad [10]$$

By checking the signs of the selection gradient for positive and negative $\hat{x}$, or according to condition [6], it can be seen that the ecological strategy $x^* = 0$ is an evolutionary attractor (i.e. an attainable evolutionarily singular point), provided that the population is viable ($b > 2$). Biologically, this means that a monomorphic population evolves towards the ecotype that matches the most abundant resources.

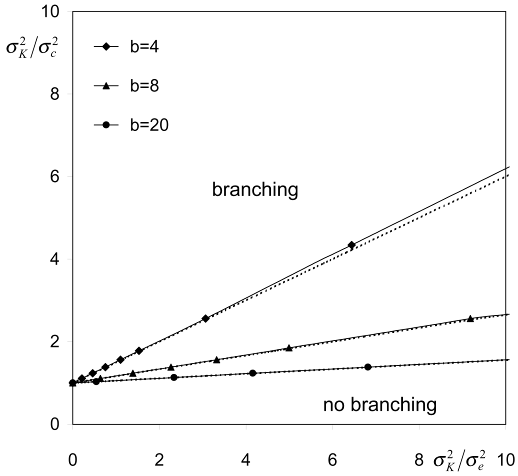

FIGURE 6 – CONDITIONS FOR BRANCHING OF ECOLOGICAL TYPE

The solid straight lines delimit the region of evolutionary branching of ecological type for different values of birth rate b. To the right on the σ_K^2/σ_e^2-axis, the effect of ecological differences on mate encounter rates becomes more important. Then, ecological branching occurs only when the width of the resource utilization function, σ_c, becomes increasingly smaller than the ecological resource variation σ_K. Branching occurs for a wider range of parameters when the birth rate is larger. The dashed lines correspond to the analytical predictions resulting from expression [12].

In order to determine the invasion-proofness of this strategy, we compute, again approximated for weak selection on female mating type, the second order derivative (all these results are derived in detail in APPENDIX A)

$$\left.\frac{\partial^2\lambda(x,\hat{x})}{\partial x^2}\right|_{x=\hat{x}=0} \approx \frac{1}{2}(b-2)\left(\frac{1}{\sigma_c^2}-\frac{1}{\sigma_K^2}\right)-\frac{1}{2}\frac{1}{\sigma_e^2}. \qquad [11]$$

From this equation, it follows that evolutionary branching of ecological type occurs when

$$\frac{\sigma_K^2}{\sigma_c^2} > 1 + \frac{1}{b-2}\frac{\sigma_K^2}{\sigma_e^2}. \qquad [12]$$

In the limit where mating probabilities are independent of ecological differences (infinite σ_e), this expression reduces to the criterion $\sigma_c < \sigma_K$ (Doebeli & Dieckmann, 2000; see also FIGURE 1). In the general case, branching of ecological type occurs when the width of the resource utilization function is sufficiently smaller than the width of the resource distribution -'sufficiently' depending on the influence of ecological differences on mate encounter rates (FIGURE 6).

CONDITIONS FOR MATING TYPE BRANCHING

As is also derived in APPENDIX A, the invasion fitness, $\lambda(q,\hat{q})$ of a male mating type mutant $(\hat{x},q)$ in a resident population of type $(\hat{x},\hat{q})$ is given by the expression

$$\lambda(q,\hat{q}) = -\frac{1}{2} + \frac{1}{2}\frac{g_s(q)}{g_s(\hat{q})}\frac{Q(q,\hat{q})}{Q(\hat{q},\hat{q})}. \qquad [13]$$

Recall that the Gaussian function $g_s(q)$ represents viability selection on male mating type. The function $Q(q,\hat{q})$ denotes the expected number of q type offspring, produced by an arbitrary female, which is proportional to the probability that a female chooses a q- type male to mate with, and which depends on the distribution of female mating types in the population. Here this dependency is not made explicit: for details regarding this and all other results derived in this section, the reader is referred to APPENDICES A & B.

In particular, male mating type mutants can invade when

$$g_s(q)\,Q(q,\hat{q}) > g_s(\hat{q})\,Q(\hat{q},\hat{q}), \qquad [14]$$

which has a straightforward biological interpretation: evolution will maximize the product of male survival and reproductive success. This is also reflected by the selection gradient

$$\left.\frac{\partial\lambda(q,\hat{q})}{\partial q}\right|_{q=\hat{q}} = -\frac{1}{2}\hat{q}\frac{1}{\sigma_s^2} + \frac{1}{2}(\mu-\hat{q})\frac{1}{\sigma_m^2}, \qquad [15]$$

where the first term represents the stabilizing effect of viability selection on male trait, and the second term represent selection for an optimal match with female mating type (μ denotes the population average female mating type). Therefore, the evolution of male mating type will converge towards a compromise value between the optimum for viability selection ($\hat{q}=0$) and the optimum for mate competition ($\hat{q}=\mu$). This latter optimum is not constant in the course of evolution, however, since the distribution of female mating types is itself under weak directional selection towards optimal matching with male mating type. So eventually, both $\hat{q}$ and μ will converge to zero, the optimum for viability selection.

Again, the invasion proofness of this endpoint of monomorphic evolution can be checked by considering the second derivative of the invasion fitness

$$\left.\frac{\partial^2\lambda(q,\hat{q})}{\partial q^2}\right|_{q=\hat{q}=0} = \frac{1}{2}\left(-\frac{1}{\sigma_s^2} - \frac{1}{\sigma_m^2} + \frac{\sigma_p^2}{\sigma_m^4}\right), \qquad [16]$$

where σ_p^2 denotes the variance of female mating types present in the population. According to this expression, evolutionary branching of male mating type occurs when

$$\sigma_p^2 > \sigma_m^2 \left(1 + \frac{\sigma_m^2}{\sigma_s^2} \right). \qquad [17]$$

In the simplest case, when viability selection on male mating type is absent ($\sigma_s \to \infty$), evolutionary branching occurs only when the width of the female mating type distribution, σ_p, exceeds the width of the fertilization efficiency function, σ_m. One would expect this to occur when selection on female mating type is weak and when the mutation rate of the female mating type gene is higher. Indeed, numerical and analytical analysis (FIGURE 7, APPENDICES A & B), taking into account the full evolutionary dynamics of female mating type, have confirmed this expectation. Note that, in the general case, conditions for evolutionary branching become more restrictive for smaller σ_s^2, that is, for stronger viability selection on males.

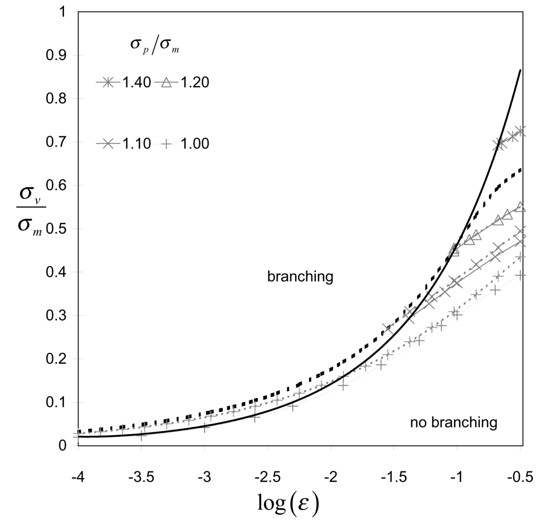

FIGURE 7 – CONDITIONS FOR BRANCHING OF MALE MATING TYPE

Along the horizontal axis, the cost of female mate choice varies (the parameter ε is a dimensionless quantity defined as η divided by the number of males, see APPENDIX A). The solid black line delimits the region of evolutionary branching of male mating type. As can be seen from the figure, branching occurs when selection on females is weak enough (as quantified by a small value of ε) and when the mutation rate of the female mating type gene is high enough relative to the mate choice specificity (upwards on the σ_v/σ_m -axis). The solid gray lines are lines of equal σ_p/σ_m. Note that the line $\sigma_p/\sigma_m = 1$, approaches the boundary of the branching region for small ε, that is to say, when selection on female mating type is weak, male mating type undergoes evolutionary branching as soon as the width of the female mating type distribution exceeds the width of the mating kernel. All solid lines result from numerical analysis of equations [A-7] and [B-1]. Also shown, as dashed lines, are the corresponding analytical approximations, resulting from equations [B-10] and [A-18]. This figure was computed for $\sigma_s/\sigma_m = 5.0$.

As mentioned before, there is an analogy between competition for ecological resources and competition for fertilizations. This analogy extends to the conditions for evolutionary branching: in the simplest case ($\sigma_s \to \infty, \sigma_e \to \infty$), conditions [12] and [17] reduce to $\sigma_c < \sigma_K$ and $\sigma_m < \sigma_p$, highlighting the analogy between resource utilization function and mate choice kernel (σ_c and σ_m) and between resource distribution and female mating type distribution (σ_K and σ_p). Note that, notwithstanding the analogy, an important distinction between the two types of competition lies in the fact that the distribution of ecological resources is fixed, whereas the distribution of female mating types evolves in response to the male mating type(s) present in the population.

CONDITIONS FOR SYMPATRIC SPECIATION

It is important to realize that the occurrence of both ecological and mating type branching is necessary but not sufficient for sympatric speciation. Besides polymorphism of mating types and ecotypes, it is also required that, during speciation, linkage disequilibrium develops between ecological and mating strategies (Felsenstein, 1981). Only then will evolutionary branching of ecotype and mating type result in the evolution of reproductively isolated and –at the same time– ecologically differentiated species.

We investigated the development of linkage disequilibrium after evolutionary branching by a technique similar to the one used above. Under the assumption that the resident population is dimorphic for both male mating type and ecotype, one again writes down the invasion fitness of a mutant and solves for the attractors of the dimorphic evolutionary dynamics. Together with equations for the evolution of female mating types, this is a complete description of the adaptive dynamics, from which the expected correlation between ecotype and male mating type (which is a measure for the linkage disequilibrium) can be derived (APPENDIX C). The results of this analysis show that there is a region in parameter space where male mating type and ecotype become correlated with another (FIGURE 8). On the other hand, FIGURE 8 also illustrates that evolutionary branching indeed not inevitably results in sympatric speciation since, in the complementary region of parameter space, linkage disequilibrium does not build up.

Two processes will determine whether linkage disequilibrium can develop. On the one hand, competition within ecotype (i.e. between individuals of identical ecotype) will tend to eliminate polymorphism of male mating type within ecotype and enlarge the correlation between ecotypes and mating types. On the other hand, mating between ecotypes will tend to destroy any linkage disequilibrium. Because of this, one would intuitively expect that linkage disequilibrium would develop more easily when the encounter rate between individuals of different ecological types is reduced (smaller σ_e). Then, mating between different ecotypes will occur only rarely, even if individuals are of matching mating type. Indeed, Figure 8 shows that correlations develop only for sufficiently small σ_e. Here, 'sufficiently small' relates to the ecological separation between ecotypes after ecological branching, Δx: in the region where linkage disequilibrium develops, we have $\sigma_e < \Delta x$, and at its border $\sigma_e = \Delta x$.

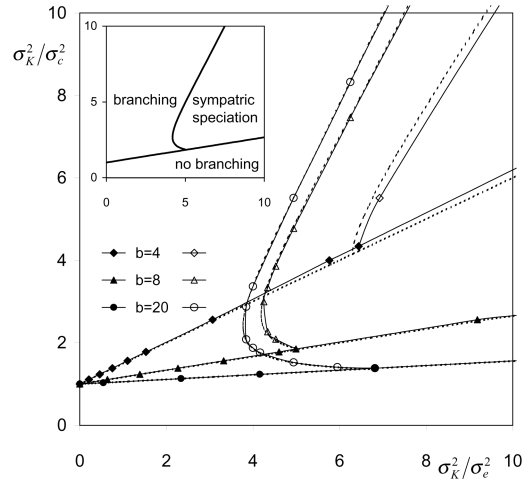

FIGURE 8 – CONDITIONS FOR SYMPATRIC SPECIATION

When ecotype and male mating type are both dimorphic, correlations may develop between ecotype and mating types, resulting in the evolution of reproductively isolated and ecologically differentiated species. In an infinite population, such a correlation develops only in a limited region of parameter space (inset). This region is delimited by a straight line bordering the region of evolutionary branching (filled symbols) and another line (open symbols), which can be computed by considering the adaptive dynamics of a population dimorphic for both ecotype and male mating type (see the main text and APPENDIX C for details). In the remaining part of the parameter space where ecological branching is possible, correlations can only evolve in finite populations. All solid lines result from numerical analysis. The dashed lines represent the corresponding analytical predictions (resulting from equations [12] and [C-5] where $x^* = \sigma_e$).

Simulations indicate that the region of parameter space where a linkage disequilibrium does build up corresponds to those conditions under which male mating type branching results in a bimodal female mating type distribution (as suggested in FIGURE 2D) and, correspondingly, in a high degree of assortative mating. For parameters outside this region, male mating type branching usually results in a broad unimodal distribution of female mating types. The evolution of a bimodal female mating type distribution, corresponding to branching in female mating types, requires diversifying frequency dependent selection on female mating type. In our case, this is caused by the association between mating types and ecological type, giving extreme mating types an advantage due to competition avoidance. We conjecture that other forms of frequency-dependent selection will have similar effects, but this requires further investigation.

GENERALIZATION OF THE MATHEMATICAL RESULTS

DIPLOID/MULTILOCUS GENETICS AND SMALL POPULATION SIZE

The results presented in the previous section are strictly valid only for single locus haploid genetic systems and for sufficiently large populations (such that stochasticity can be ignored). In order to check whether the analytical results hold in a more general context of multilocus genetics we ran simulations with different genetic systems underlying the traits, each for a large number of parameter combinations, and we measured the correlation between ecological and mating types as an indicator of the occurrence of speciation. FIGURE 9 shows the result of one such experiment, where we assumed single locus diploid genetics underlying all traits.

The conditions for branching of mating types are predicted well by the theory (data not shown). For ecological branching and subsequent speciation we find that there are two important qualitative differences between theoretical predictions and simulation results.

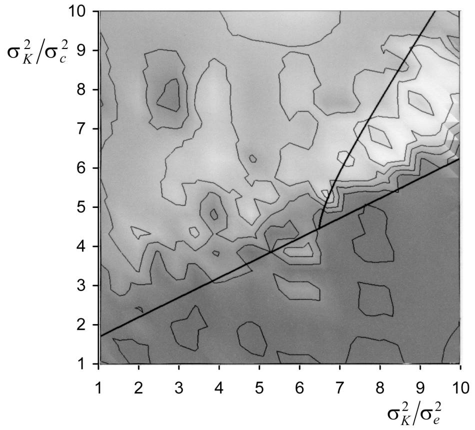

FIGURE 9 – A COMPARISON OF THE ANALYTICAL PREDICTIONS WITH INDIVIDUAL-BASED SIMULATIONS

A large number of simulations were run for different combinations of the parameters σ_c, σ_e and σ_K, thus varying the ecological selection regime. For these parameter combinations (other parameters as in FIGURE 3), the figure shows the association between ecological type and mating type. This is measured as the correlation between these traits (averaged over 1000 generations) and indicated on a gray scale with contour lines at the values 0, 0.2, 0.4, 0.6 and 0.8 (white indicates high, black indicates low correlation). Also shown in thick black lines are the analytical predictions for the boundaries of the branching region and the region where a correlation between ecological type and mating type exists (also shown in FIGURE 8). These analytical predictions are based on a large population approximation where stochasticity is ignored. The region where a strong association between mating types and ecotype evolves -this is the light gray / white area- falls within the analytically predicted region, but does not completely fill it, particularly not for small σ_K^2/σ_e^2. Another striking feature is that speciation occurs for a considerably larger range of parameters than would be predicted from the deterministic approximation of the population dynamics. As in the FIGURES 3–5, population sizes in the simulations were about 1000 individuals per species.

First, conditions for branching are more restrictive than predicted when mate encounter rates are not affected by ecological differences (for large σ_e). This observation is in accordance with the results of Dieckmann & Doebeli (1999), who noted that the conditions for branching agree well with predictions based on an asexual model when mating probabilities depend on ecological type, but are more restrictive when mating probabilities are independent of ecological traits, instead depending on a neutral marker trait (in our model male and female mating type).

Second, the parameter region in which mating types and ecological type become correlated with another –this is the region where sympatric speciation occurs– is much larger than predicted. This discrepancy can partly be explained by the fact that the population sizes in our simulations are not so large that stochasticity can be ignored, as we assumed in the mathematical analysis. In the simulations, random fluctuations of the linkage disequilibrium between ecological and mating type loci occur, caused by the stochasticity of demographic processes in finite populations. Due to the evolutionary dynamics, such small fluctuations may be enlarged, if they exceed a certain threshold, resulting in a larger linkage disequilibrium and eventually speciation. This effect was also described by Dieckmann & Doebeli (1999). Another factor that may explain why speciation seems to occur for a wider range of parameters than expected, is that there is some amount of genetic variation of male mating type in the individual based simulations (which was neglected in the adaptive dynamics approximation). This genetic variation enlarges the 'assortativeness' of mating, which may facilitate speciation.

For larger populations and multi-locus genetics, results are similar, except that the waiting time until speciation increases. For a very large number of loci, results were also checked using a quantitative genetics approximation. Again, sympatric speciation occurred for a wide range of parameters (similar to FIGURE 9).

DISCUSSION

Our model represents a first step towards understanding the multitude of entangled processes that underlie sympatric speciation. We have focused on the interplay between ecological interactions as a source of biological diversity and sexual selection as the mechanism underlying reproductive isolation. The results of our analysis show that ecological differentiation and the evolution of assortative mating are mutually dependent processes that are both required for sympatric speciation. Ecological differentiation arises naturally from evolutionary branching of competitive strategies (as determined by ecotype). Assortative mating results from evolutionary branching of mating strategies (male mating type) automatically followed by the genetic association of matching male and female mating types. Sympatric speciation is completed when, in addition, a linkage disequilibrium develops between ecotypes and mating types, giving rise to reproductively isolated and, at the same time, ecologically differentiated daughter species.

On an abstract level, ecological and mating type branching can both be understood as the outcome of a competition process in which the optimal competitive strategy is determined by the distribution of resources (ecological resources or mating opportunities, respectively) and by the competitive behavior of other individuals. At first sight, it might seem an optimal competitive strategy to specialize on the most abundant resource. However, it might also pay to specialize on less abundant resources, thus avoiding competition with other individuals. The latter applies particularly when the variation of resources is large. These considerations translate into a feedback between ecological and evolutionary processes, which drive a monomorphic population towards the optimum of the resource distribution, but may there induce disruptive selection resulting in evolutionary branching. For ecological resource competition and mate competition alike, this occurs only when the variation 'resources' (ecological resources or female mating types, respectively) is large enough. For ecological competition, this implies that individuals should be ecological specialists rather than generalists. For mate competition, it implies that mate choice should be sufficiently specific or that selection on female mate choice should be weak.

In very large populations, the required linkage disequilibrium between ecotype and mating types will only develop when individuals that differ ecologically also have a lower probability of mating with each other. This mate choice with respect to ecotype has to be specific enough in order to establish a linkage disequilibrium, but not too specific, since, in that case, evolutionary branching of ecotype would be prohibited. In small populations, however, the conditions for sympatric speciation are far less stringent, as a result of stochasticity. Small random fluctuations of genetic correlations between ecotype and mating types can be enlarged by the ecological dynamics to a strong linkage disequilibrium. This important effect of stochasticity was also noted by Dieckmann & Doebeli (1999), and it illustrates the usefulness of a combined computer-simulation and analytical approach.

Although we have chosen for a rather general modeling approach, we would like to stress that we have imposed a number of restrictions, the alleviation of which provides ample opportunities for future research. For example, in our analysis we have treated ecological branching and mating type branching as largely separate processes, assuming that the coupling between ecotype dynamics and mating type dynamics becomes important only later, when the linkage disequilibrium between ecotype and mating types establishes itself. This approach has been motivated and checked by numerical studies, but from a methodological point of view, it is important to note that this leaves out of consideration modes of evolutionary branching that result from the coupling of ecotype and mating type dynamics. The theory of such higher dimensional branching is rather complicated and is subject of current theoretical research.

A number of key parameters of our model were assumed to be constant and not subject to evolution. This may not be adequate, particularly not for the parameters σ_c and σ_m, since resource utilization characteristics (σ_c) and the specificity of mate choice (σ_m) are themselves likely to be subject to evolutionary change. It would therefore be interesting to consider as a second step models that include more of the mechanisms and trade-offs associated with resource utilization and mate choice characteristics.

Other elaborations may include the modeling of different ecological interactions or other mechanisms of mate choice. Moreover, a spatial version of the model may be used to gain insight in the influence of spatial pattern formation on sympatric speciation. In order to study sympatric speciation in its purest form, models usually consider only well-mixed populations. This excludes the possibility of spatial pattern formation, which is all but absent from most biological systems. In addition, from a theoretical viewpoint, it is well conceivable that adding a small spatial component may greatly enlarge the potential for sympatric speciation. This is, for instance, because the stochasticity of local interactions with a limited number of neighbors may greatly facilitate the development of linkage disequilibrium required for speciation or because spatial heterogeneity may enhance evolutionary branching.

APPENDIX A — CONDITIONS FOR EVOLUTIONARY BRANCHING

To find the invasion fitness of a mutant (x,q) in a resident population $(\hat{x},\hat{q})$, we write a differential equation describing the dynamics of the number of mutants when rare

$$\frac{dn}{dt}=\underbrace{-n}_{\text{death}}+\underbrace{\tfrac{1}{2}nS(q)\left(1-M(x,\hat{x})\right)Q_M(x,q,\hat{x},\hat{q})}_{\text{production and survival of male mutants}}+\underbrace{\tfrac{1}{2}n\left(1-M(x,\hat{x})\right)Q_F(x,q,\hat{x},\hat{q})}_{\text{production and survival of female mutants}}+O\left(n^2\right), \text{[A-1]}$$

where n denotes the density of the mutant before selection, and Q_M (Q_F) denotes the number of mutant offspring produced per mutant male (female). Analogously to the stochastic simulation model, $S(q)=g_s(q)$ represents direct viability selection on male mating type and $M(x,\hat{x})$ defines density dependent mortality due to ecological resource competition:

$$M(x,\hat{x})=\gamma\,\frac{(N_M+N_F)g_c(x-\hat{x})}{g_K(x)}, \qquad \text{[A-2]}$$

where N_M and N_F are the densities of resident males and females.

Formally, we should have written more complicated equations for the mutant dynamics, at least, when we want to consider mutants that differ from the resident in both mating type and ecological type. Such double mutants can however be neglected, since we will consider only single mutants (x,q) where either $x=\hat{x}$ or $q=\hat{q}$.

Now, let us define the mutant per capita birth rates Q_M and Q_F. The probability that a mutant male of type (x,q) mates with a female of type $(\hat{x},\hat{q})$ and female mating type p is given by the expression

$$\frac{g_m(p-q)g_e(x-\hat{x})}{\eta+N_M\,g_m(p-\hat{q})}=g_e(x-\hat{x})\frac{1}{N_M}\frac{g_m(p-q)}{\varepsilon+g_m(p-\hat{q})}, \qquad \text{[A-3]}$$

where $\varepsilon=\eta/N_M$. Formally, N_M depends on $\hat{x}$ and $\hat{q}$, but since ε will generally be small we will ignore these higher order dependencies and treat ε as a parameter from here on.

To find Q_M, the mating probability [A-3] has to be multiplied by the density of mating type p females, and integrated over all possible female mating types. If we denote the frequency distribution of female mating types in the population as $f(p)$, then

$$Q_M(x,q,\hat{x},\hat{q})=g_e(x-\hat{x})\frac{N_F}{N_M}Q(q,\hat{q}), \qquad \text{[A-4]}$$

where

$$Q(q,\hat{q}) = \frac{b}{2}\int_{-\infty}^{\infty} f(p)\frac{g_m(p-q)}{\varepsilon + g_m(p-\hat{q})}dp\,. \qquad \text{[A-5]}$$

The function $Q(q,\hat{q})$ can be interpreted as the expected number of q-offspring that will be produced by an average resident female. In particular, we will use that, if selection on females is weak, $Q(\hat{q},\hat{q}) \approx b/2$. This corresponds to saying that females will produce close to b offspring when they mate with a resident male.

Similarly, to find Q_F, the probability that a mutant female mates, is integrated over all possible female mating types, weighted with respect to their frequency, which yields

$$Q_F(x,q,\hat{x},\hat{q}) = \frac{b}{2}\int_{-\infty}^{\infty} f(p)\frac{N_M\, g_m(p-\hat{q})g_e(x-\hat{x})}{\eta + N_M\, g_m(p-\hat{q})g_e(x-\hat{x})}dp = Q(\hat{q},\hat{q}) + O(\varepsilon)\,. \qquad \text{[A-6]}$$

Equation [A-1] can now be rewritten using equations [A-4] and [A-6] together with $N_M / N_F = g_s(\hat{q})$, to give $\lambda(x,q,\hat{x},\hat{q})$, the per capita growth rate of the mutant in the resident population $(\hat{x},\hat{q})$:

$$\lambda(x,q,\hat{x},\hat{q}) = \frac{1}{n}\frac{dn}{dt} = -1 + \frac{1}{2}\big(1 - M(x,\hat{x})\big)\left(g_e(x-\hat{x})\frac{g_s(q)}{g_s(\hat{q})}Q(q,\hat{q}) + Q(\hat{q},\hat{q}) + O(\varepsilon)\right). \qquad \text{[A-7]}$$

We assume that the resident population is in population dynamical equilibrium, which implies that a 'mutant' $(x = \hat{x}, q = \hat{q})$ should have a per capita growth rate equal to zero. According to equations [A-2] and [A-7], this condition implies that

$$N = \frac{g_K(\hat{x})}{\gamma}\left(1 - \frac{1}{Q(\hat{q},\hat{q})}\right). \qquad \text{[A-8]}$$

To find the invasion fitness of an ecological type mutant, we substitute $q = \hat{q}$ into equation [A-7], which yields

$$\lambda(x,\hat{q},\hat{x},\hat{q}) = -1 + \frac{1}{2}\left(Q(\hat{q},\hat{q}) - \big(Q(\hat{q},\hat{q}) - 1\big)\frac{g_c(x-\hat{x})g_K(\hat{x})}{g_K(x)}\right)\big(g_e(x-\hat{x}) + 1 + O(\varepsilon)\big). \qquad \text{[A-9]}$$

For small ε, this equation becomes independent of $\hat{q}$, and approaches equation [7], which is interpreted biologically in the main text.

For a male mating type mutant, the invasion fitness reduces to

$$\lambda(\hat{x},q,\hat{x},\hat{q}) = -\frac{1}{2} + \frac{1}{2}\frac{g_s(q)}{g_s(\hat{q})}\frac{Q(q,\hat{q})}{Q(\hat{q},\hat{q})}, \qquad \text{[A-10]}$$

which is independent of $\hat{x}$ and identical to equation [13].

Up to now, we have ignored the dynamics of the frequency distribution of female mating types $f(p)$. As it turns out, we do not need to know the full distribution $f(p)$. Instead, we can suffice with the functions m_0, m_1 and m_2, where

$$m_k(\hat{q}) = \int_{-\infty}^{\infty} (p-\hat{q})^k f(p)\frac{g_m(p-\hat{q})}{\varepsilon + g_m(p-\hat{q})}dp\,. \qquad \text{[A-11]}$$

As will be shown in detail in APPENDIX B, where the dynamics of $f(p)$ is treated, we have

$$\text{for all } \hat{q}: \quad \frac{m_1(\hat{q})}{m_0(\hat{q})} = \mu - \hat{q} \quad \text{and} \quad \frac{m_2(\hat{q})}{m_0(\hat{q})} \approx \sigma_p^2 \, . \qquad \text{[A-12]}$$

These expressions can be interpreted as the first and second moment of the distribution of mating opportunities, which turn out to be related to the mean (μ) and variance (σ_p^2) of the female mating type distribution (APPENDIX B).

To find the evolutionary attractors $(\overline{x},\overline{q})$ of the monomorphic dynamics, we need to compute the selection gradients

$$\lambda_x\left(\hat{x},q^*\right) = \left.\frac{\partial\lambda(x,q,\hat{x},\hat{q})}{\partial x}\right|_{\substack{x=\hat{x}\\ q=\hat{q}=q^*}} = -\hat{x}\,\frac{Q\left(q^*,q^*\right)-1}{\sigma_K^2} \, , \qquad \text{[A-13]}$$

and

$$\lambda_q\left(\hat{q},x^*\right) = \left.\frac{\partial\lambda(x,q,\hat{x},\hat{q})}{\partial q}\right|_{\substack{x=\hat{x}=x^*\\ q=\hat{q}}} = -\frac{1}{2}\hat{q}\frac{1}{\sigma_s^2} + \frac{1}{2}(\mu-\hat{q})\frac{1}{\sigma_m^2} \, , \qquad \text{[A-14]}$$

where we have used the expression [A-12] for the first moment of the distribution of mating opportunities.

When $\lambda_x\left(\hat{x},q^*\right) > 0$, selection acts to increase $\hat{x}$. Alternatively, when $\lambda_x\left(\hat{x},q^*\right) < 0$, selection acts to decrease $\hat{x}$. With similar conditions for $\hat{q}$, where we additionally postulate that $\mu \to 0$ in the course of evolution (see APPENDIX B), it follows that $\left(x^*,q^*\right)$ must satisfy $\lambda_x\left(x^*,q^*\right)=0$ and $\lambda_q\left(q^*,x^*\right)=0$, implying that $\left(x^*,q^*\right)=(0,0)$. This evolutionarily singular point is an evolutionary attractor when

$$\left.\frac{\partial\lambda_x(\hat{x},0)}{\partial\hat{x}}\right|_{\hat{x}=0} = -\frac{Q(0,0)-1}{\sigma_K^2} < 0 \quad \text{and} \quad \left.\frac{\partial\lambda_q(\hat{q},0)}{\partial\hat{q}}\right|_{\hat{q}=0} = -\frac{1}{2}\frac{1}{\sigma_s^2} < 0 \quad \Leftrightarrow$$

$$b > \frac{2}{\int_{-\infty}^{\infty} f(p)\frac{g_m(p)}{\varepsilon + g_m(p)}\,dp} \approx 2 \, , \qquad \text{[A-15]}$$

the latter condition implying that there should be a viable population.

Evolutionary branching occurs only when the population at $\left(x^*,q^*\right)=(0,0)$ is located at a fitness minimum with respect to the mutant strategy, in other words, when

$$\lambda_{xx}\left(x^*,q^*\right) = \left.\frac{\partial^2\lambda(x,q,\hat{x},\hat{q})}{\partial x^2}\right|_{\substack{x=\hat{x}=x^*\\ q=\hat{q}=q^*}} > 0 \, , \qquad \text{[A-16]}$$

and

$$\lambda_{qq}\left(q^*,x^*\right) = \left.\frac{\partial^2\lambda(x,q,\hat{x},\hat{q})}{\partial q^2}\right|_{\substack{x=\hat{x}=x^*\\ q=\hat{q}=q^*}} > 0 \, . \qquad \text{[A-17]}$$

Substituting the evolutionary attractor $\left(x^*,q^*\right)=(0,0)$ into equation [A-17] yields the following condition for branching of male mating type

$$\lambda_{qq}(0,0)=\frac{1}{2}\left(-\frac{1}{\sigma_s^2}-\frac{1}{\sigma_m^2}+\frac{1}{\sigma_m^4}\frac{M_2(0)}{M_0(0)}\right)>0\,. \qquad \text{[A-18]}$$

Equations [A-12] and [A-18], approximated for small mutation rates, combine into the condition [17]

$$\sigma_p^2>\sigma_m^2\left(1+\frac{\sigma_m^2}{\sigma_s^2}\right). \qquad \text{[A-19]}$$

Consequently, male mating strategy undergoes evolutionary branching when the variation of female mating strategies exceeds a threshold of the order of σ_m, the scale that determines the specificity of mate choice.

Furthermore, branching of ecological type x occurs when

$$\lambda_{xx}(0,0)=\left(Q^*-1\right)\left(\frac{1}{\sigma_c^2}-\frac{1}{\sigma_K^2}\right)-\frac{1}{2}\frac{1}{\sigma_e^2}\left(1+O(\varepsilon)\right)>0\,, \qquad \text{[A-20]}$$

with $Q^*=Q(0,0)$.

In the limit of infinite σ_e, the condition for branching reduces to the well-known form $\sigma_c<\sigma_K$. In the general case, we find condition [12]

$$\frac{\sigma_K^2}{\sigma_c^2}>1+\frac{1}{2\left(Q^*-1\right)}\frac{\sigma_K^2}{\sigma_e^2}+O(\varepsilon)\approx 1+\frac{1}{b-2}\frac{\sigma_K^2}{\sigma_e^2}\,. \qquad \text{[A-21]}$$

APPENDIX B — DYNAMICS OF FEMALE MATING TYPE

If the female mating type p is determined by a single locus haploid genotype, then $f(p)$ must satisfy

$$f(p)=\underbrace{\frac{1}{2}\bar{g}_v*f(p)}_{\text{contribution via male parent}}+\underbrace{\frac{1}{2Q(\hat{q},\hat{q})}\bar{g}_v*\left(\frac{b}{2}f(p)\frac{g_m(p-\hat{q})}{\varepsilon+g_m(p-\hat{q})}\right)}_{\text{contribution via female parent}}, \qquad \text{[B-1]}$$

where $\bar{g}_v$ denotes the mutation kernel

$$\bar{g}_v(p)=\frac{1}{\sigma_v\sqrt{2\pi}}\exp\left(-\frac{1}{2}\frac{p^2}{\sigma_v^2}\right), \qquad \text{[B-2]}$$

and $*$ denotes the convolution operator

$$u*v(p)=\int_{-\infty}^{\infty}u(z)\,v(z-p)\,dz\,. \qquad \text{[B-3]}$$

Let us now write $f(p)$ as a series expansion.

$$f(p)=g_p(p-\mu)\left(1+\sum_{n=3}^{\infty}\alpha_n H_n\left(\frac{p-\mu}{\sigma_p}\right)\right). \qquad \text{[B-4]}$$

The functions $H_n(p)$ are so called Hermite polynomials, defined as

$$H_n(p) = (-1)^n e^{\frac{1}{2}p^2} \frac{d^n}{dp^n} e^{-\frac{1}{2}p^2} . \qquad \text{[B-5]}$$

In particular, $H_0(p) = 1$, $H_1(p) = p$ and $H_2(p) = p^2 - 1$. It can be shown that the series at the right hand side of equation [B-4] converges uniformly towards $f(p)$.

Multiplying both sides of equation [B-1] with $H_1\left((p-\mu)/\sigma_p\right)$ and integrating over all p yields

$$\mu = \frac{\int_{-\infty}^{\infty} p\, f(p) \frac{g_m(p-\hat{q})}{\varepsilon + g_m(p-\hat{q})} dp}{\int_{-\infty}^{\infty} f(p) \frac{g_m(p-\hat{q})}{\varepsilon + g_m(p-\hat{q})} dp}, \qquad \text{[B-6]}$$

which is an expression that relates the first moment of the distribution of mating opportunities to the average female mating type (equation [A-12]). Stationary solutions $f(p)$ are obtained only when $\mu = \hat{q}$. Here we omit the mathematical proof, but this result can explained biologically by the fact that only one directed selection pressure acts on the female mating type distribution, and this will lead towards exact matching of the mean of the female mating type distribution with the resident male mating type. In deriving equation [B-6], we have used equation [B-4] as an approximation for $f(p)$ together with the orthogonality property of Hermite polynomials

$$\int_{-\infty}^{\infty} H_n(p) H_m(p) e^{-\frac{1}{2}p^2} dp = 0 \quad \text{if} \quad n \neq m . \qquad \text{[B-7]}$$

Similarly, multiplying both sides of equation [B-1] with $H_2\left((p-\mu)/\sigma_p\right)$ and integrating over all p gives an expression for σ_p^2

$$\sigma_p^2 = 2\sigma_v^2 + \frac{\int_{-\infty}^{\infty} (p-\hat{q})^2 f(p) \frac{g_m(p-\hat{q})}{\varepsilon + g_m(p-\hat{q})} dp}{\int_{-\infty}^{\infty} f(p) \frac{g_m(p-\hat{q})}{\varepsilon + g_m(p-\hat{q})} dp} . \qquad \text{[B-8]}$$

Now it also follows immediately that $m_2(q)/m_0(q) = \sigma_p^2 - 2\sigma_v^2 \approx \sigma_p^2$ (equation [A-12]).

In order to express σ_p in the parameters of the model, we can make the following approximation

$$\frac{g_m(p)}{\varepsilon + g_m(p)} \approx \beta \int_{-\vartheta}^{\vartheta} \exp\left(-\frac{1}{2} \left(\frac{z-p}{\frac{1}{2}\sigma_m} \right)^2 \right) dz , \qquad \text{[B-9]}$$

where $\vartheta = \sigma_m \sqrt{-2\ln(\varepsilon)}$ and where β is an unimportant proportionality constant. Under this approximation and neglecting the higher order Hermite polynomial terms in equation [B-4], it can be derived that σ_p satisfies

$$\tilde{\sigma}_v^2 = \frac{\tilde{\sigma}_p^4\,\tilde{\vartheta}}{\sqrt{2\pi}\left(\tilde{\sigma}_p^2 + 1/4\right)^{3/2}} \frac{e^{-\frac{1}{2}\frac{\tilde{\vartheta}^2}{\tilde{\sigma}_p^2+1/4}}}{\operatorname{erf}\left(\frac{1}{2}\sqrt{2}\frac{\tilde{\vartheta}}{\sqrt{\tilde{\sigma}_p^2+1/4}}\right)}, \qquad \text{[B-10]}$$

with $\tilde{\vartheta} = \sqrt{-2\ln(\varepsilon)}$, $\tilde{\sigma}_p = \sigma_p/\sigma_m$ and $\tilde{\sigma}_v = \sigma_v/\sigma_m$. FIGURE 7 shows the parameter region in which branching occurs, following from equation [B-10] as well as numerically calculated from equations [A-7] and [B-1].

APPENDIX C — DIMORPHIC DYNAMICS

Suppose that the resident population is dimorphic for ecological type and male mating type with character values x_1, x_2, q_1 and q_2. Moreover, consider the female mating type distributions $f_{i,j}(p)$ for genotype (x_i, q_j). Because of the symmetry of the model, we will consider only symmetric cases here, and therefore denote

$$\begin{aligned} \hat{q} &= \hat{q}_1 = -\hat{q}_2\,, \\ \hat{x} &= \hat{x}_1 = -\hat{x}_2\,, \\ f_+(p) &= 2f_{1,1}(p) = 2f_{2,2}(-p)\,, \\ f_-(p) &= 2f_{1,2}(p) = 2f_{2,1}(-p)\,, \\ \text{with} &\int_{-\infty}^{\infty} f_+(p) + f_-(p)\,dp = 1\,. \end{aligned} \qquad \text{[C-1]}$$

As a measure of correlation between ecological and male mating type, we define

$$\rho = \int_{-\infty}^{\infty} f_+(p)\,dp\,. \qquad \text{[C-2]}$$

A derivation similar to the one presented in APPENDIX A, enables us to find the invasion fitness of a rare mutant (x,q) in a resident population consisting of the types $(\hat{x},\hat{q})$, $(\hat{x},-\hat{q})$, $(-\hat{x},\hat{q})$ and $(-\hat{x},-\hat{q})$. Analogously to equation [A-7], we find

$$\begin{aligned} &\lambda(x,q,\hat{x},\hat{q}) = -1 + \frac{1}{2}\left(1 - \frac{\left(g_c(x-\hat{x}) + g_c(x+\hat{x})\right)g_K(\hat{x})}{\left(1+g_c(2\hat{x})\right)g_K(x)}\left(1 - \frac{1}{Q_2(\hat{q},\hat{x})}\right)\right)\cdot \\ &\left(\frac{g_s(q)}{g_s(\hat{q})}\int_{-\infty}^{\infty}\left(f_+(p)+f_-(p)\right)\cdot\right. \\ &\frac{g_m(p-q)\left(\rho\, g_e(x-\hat{x}) + (1-\rho)\,g_e(x+\hat{x})\right) + g_m(p+q)\left(\rho\, g_e(x+\hat{x}) + (1-\rho)\,g_e(x-\hat{x})\right)}{2\varepsilon + g_m(p-\hat{q})\left(\rho + (1-\rho)\,g_e(2\hat{x})\right) + g_m(p+\hat{q})\left(\rho\, g_e(2\hat{x}) + (1-\rho)\right)}dp \\ &+\int_{-\infty}^{\infty}\left(f_+(p)+f_-(p)\right)\cdot \\ &\left.\frac{g_m(p-\hat{q})\left(\rho\, g_e(x-\hat{x}) + (1-\rho)\,g_e(x+\hat{x})\right) + g_m(p+\hat{q})\left(\rho\, g_e(x+\hat{x}) + (1-\rho)\,g_e(x-\hat{x})\right)}{2\varepsilon + g_m(p-\hat{q})\left(\rho\, g_e(x-\hat{x}) + (1-\rho)\,g_e(x+\hat{x})\right) + g_m(p+\hat{q})\left(\rho\, g_e(x+\hat{x}) + (1-\rho)\,g_e(x-\hat{x})\right)}dp\right), \end{aligned}$$

$$\text{[C-3]}$$

with

$$Q_2(\hat{q},\hat{x}) = \frac{b}{2}\int_{-\infty}^{\infty}\left(f_+(p)+f_-(p)\right)\cdot$$

$$\frac{g_m(p-\hat{q})\left(\rho+(1-\rho)g_e(2\hat{x})\right)+g_m(p+\hat{q})\left(\rho\, g_e(2\hat{x})+(1-\rho)\right)}{2\varepsilon+g_m(p-\hat{q})\left(\rho+(1-\rho)g_e(2\hat{x})\right)+g_m(p+\hat{q})\left(\rho\, g_e(2\hat{x})+(1-\rho)\right)}dp\,. \qquad \text{[C-4]}$$

Attractors (x^*,q^*) of the dimorphic dynamics satisfy

$$\left.\frac{\partial\lambda(x,q,\hat{x},\hat{q})}{\partial q}\right|_{\substack{x=\hat{x}=x^*\\ q=\hat{q}=q^*}} = -\frac{1}{2}\left(\frac{q^*}{\sigma_s^2}+\frac{q^*}{\sigma_m^2}-\frac{1}{\sigma_m^2}\frac{L_1(x^*,q^*)}{L_0(x^*,q^*)}\right)=0 \quad \text{and}$$

$$\left.\frac{\partial\lambda(x,q,\hat{x},\hat{q})}{\partial x}\right|_{\substack{x=\hat{x}=x^*\\ q=\hat{q}=q^*}} = x^*\left(\frac{1}{\sigma_c^2}\frac{2g_c(2x^*)}{1+g_c(2x^*)}-\frac{1}{\sigma_K^2}\right)\left(Q_2(q^*,x^*)-1\right) \qquad \text{[C-5]}$$

$$-x^*\frac{1}{2}\frac{1}{\sigma_e^2}\frac{2g_e(2x^*)}{1+g_e(2x^*)}\left(\frac{\tilde{L}_0(x^*,q^*)}{L_0(x^*,q^*)}+O(\varepsilon)\right)=0\,,$$

with

$$L_k(x^*,q^*) = \int_{-\infty}^{\infty}(p-q^*)^k\left(f_+(p)+f_-(p)\right)\cdot$$

$$\frac{g_m(p-q^*)\left(\rho+(1-\rho)g_e(2x^*)\right)+g_m(p+q^*)\left(\rho\, g_e(2x^*)+(1-\rho)\right)}{2\varepsilon+g_m(p-q^*)\left(\rho+(1-\rho)g_e(2x^*)\right)+g_m(p+q^*)\left(\rho\, g_e(2x^*)+(1-\rho)\right)}\,, \qquad \text{[C-6]}$$

$$\tilde{L}_0(x^*,q^*) = \int_{-\infty}^{\infty}\left(f_+(p)+f_-(p)\right)\cdot$$

$$\frac{(1-\rho)g_m(p-q^*)\left(1+g_e(2x^*)\right)+\rho\, g_m(p+q^*)\left(1+g_e(2x^*)\right)}{2\varepsilon+g_m(p-q^*)\left(\rho+(1-\rho)g_e(2x^*)\right)+g_m(p+q^*)\left(\rho\, g_e(2x^*)+(1-\rho)\right)}\,.$$

Note that for $\rho=1/2$, $\tilde{L}_0(x^*,q^*)=L_0(x^*,q^*)$.

We can describe the dynamics of $f_+(p)$ and $f_-(p)$ by deriving equations for the genotypes (x_i,q_j) similar to equation [B-1]

$$f_{i,j}(p)=\frac{1}{8\,Q_2(q^*,x^*)}\sum_{\substack{k\in\{1,2\}\\ l\in\{1,2\}}}\bar{g}_v * \int_{-\infty}^{\infty} T^{i,j}_{k,l}(p,z)+T^{i,j}_{k,l}(z,p)+T^{k,j}_{i,l}(p,z)+ \qquad \text{[C-7]}$$

$$T^{k,j}_{i,l}(z,p)+T^{i,l}_{k,j}(p,z)+T^{i,l}_{k,j}(z,p)+T^{k,l}_{i,j}(p,z)+T^{k,l}_{i,j}(z,p)\,dz\,,$$

where the terms $T^{k,l}_{i,j}(p_1,p_2)$ denote the contributions by a mating of a (p_1,x_i,q_j) female with a (p_2,x_k,q_l) male:

$$T^{i,j}_{k,l}(p_1,p_2)=f_{i,j}(p_1)f_{k,l}(p_2)g_m(p_1-q_l)g_e(x_i-x_k)\Big/\Big(2\varepsilon+g_m(p_1-\hat{q})\big(\rho\, g_e(x_i-\hat{x}) \qquad \text{[C-8]}$$

$$+(1-\rho)g_e(x_i+\hat{x})\big)+g_m(p_1+\hat{q})\big(\rho\, g_e(x_i+\hat{x})+(1-\rho)g_e(x_i-\hat{x})\big)\Big)\,.$$

In order to investigate the conditions under which a correlation between ecological types and mating types develops, equations [C-5] and [C-7] can be solved numerically, yielding the equilibrium distributions of female mating types, from which ρ can be calculated (equation [C-2]).

Sympatric speciation by sexual selection: a critical reevaluation

4

G. Sander van Doorn, Ulf Dieckmann & Franz J. Weissing

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ABSTRACT

Several empirical studies put forward sexual selection as an important driving force of sympatric speciation. This idea agrees with recent models suggesting that speciation may proceed by means of divergent Fisherian runaway processes within a single population. Notwithstanding this, the models so far have not been able to demonstrate that sympatric speciation can unfold as a fully adaptive process, driven by sexual selection alone. Implicitly or explicitly, most models rely on non-selective factors to initiate speciation. In fact, they do not provide a selective explanation for the considerable variation in female preferences required to trigger divergent runaway processes. We argue that such variation can arise by disruptive selection, but only when selection on female preferences is frequency-dependent. Adaptive speciation is therefore unattainable in traditional female choice models, which assume selection on female preferences to be frequency-independent. However, when frequency-dependent sexual selection processes act alongside mate choice, truly adaptive sympatric speciation becomes feasible. Speciation is then initiated independently of non-adaptive processes, and does not suffer from the theoretical weaknesses associated with the current Fisherian runaway model of speciation. However, adaptive speciation requires the simultaneous action of multiple mechanisms, and therefore it occurs under conditions far more restrictive than earlier models of sympatric speciation by sexual selection appear to suggest.

INTRODUCTION

Classical research into the potential mechanisms of sympatric speciation has sought to explain sympatric speciation primarily from ecological causes (reviewed, e.g., in Johnson & Gullberg, 1998; Schluter, 2001), often presupposing the presence of an assortative mating structure allowing for a high degree of reproductive isolation. Recent research, on the other hand, has put emphasis on the evolution of the mating structure itself and focuses on sexual selection as a driving force of sympatric speciation (reviewed in Panhuis *et al.*, 2001).

The view that sexual selection plays a significant role in sympatric speciation is supported by comparative studies, which indicate that closely related species often differ most pronouncedly in their secondary sexual characters, rather than in other, ecologically relevant, morphological traits (Eberhard, 1985; Wilson *et al.*, 2000). Moreover, DNA sequence analysis has revealed an extraordinary divergence of sex-related genes, particularly between closely related species (e.g., Vacquier, 1998; Wyckoff *et al.*, 2000), lending support to the hypothesis that strong (sexual) selection has acted on these genes during speciation (CHAPTER 2 of this thesis).

Also from a theoretical point of view, the involvement of sexual selection in sympatric speciation seems plausible. It is now well established that sexual selection by female mate choice may lead to rapid evolution of exaggerated male traits and corresponding female preferences by means of a Fisherian runaway process (Fisher, 1930; Lande, 1981; Kirkpatrick, 1982). In contrast to good-genes processes, female preferences in a runaway process may be based on arbitrary male traits, conferring no inherent fitness advantage. In principle, it is therefore conceivable that multiple runaway processes simultaneously occur within the same population. A theoretical study by Higashi *et al.* (1999) has shown that this is a feasible scenario and that sexual selection alone can split a population into two reproductively isolated parts.

As indicated by Higashi *et al.* (1999), sufficient initial genetic variation of female preferences has to be present in order to trigger two simultaneous runaway processes. This prerequisite is not surprising, since classical female-choice models demonstrated that already a single runaway process will only occur if the initial level of choosiness exceeds a certain threshold value (Kirkpatrick, 1982; Andersson, 1994). It is therefore to be expected that, in order to trigger two simultaneous runaway processes, the level of choosiness for two distinct male traits has to be sufficiently high. Consequently, multiple preference alleles, coding for choosiness with respect to different male traits will have to be present in sufficiently high frequencies, which implies that there should be considerable variation of female preferences in the initial population. Although female preference variation has been documented (Kirkpatrick, 1987; Bakker, 1990), the origin and maintenance of such a large amount of variation in natural systems is not self-evident.

Two paths along which sufficient genetic variation in female preferences could arise have been discussed in the literature. First, there is the possibility that a sudden change in environmental conditions changes the parameters of mate choice in such a way that previously hidden genetic variation of female preferences is suddenly exposed (Higashi *et al.*, 1999). For example, it has been argued that the deterioration of the underwater light conditions in Lake Victoria has led to a decline in haplochromine cichlid diversity, since the increased turbidity of the water has severely compromised female mate choice based on male coloration (Seehausen *et al.*, 1997). If the water would suddenly become clear again, a large variation of female preferences that was hidden under the turbid water conditions would be expressed, possibly leading to new speciation events. It is hard to determine whether such sudden environmental changes are very likely to occur. Moreover, if such events were required to induce sympatric speciation, then sympatric speciation, like allopatric speciation requiring imposed geographic isolation, would largely be dependent on unpredictable external events. This conclusion not only conflicts with the historical interpretation of sympatric speciation as an internally driven and adaptive process, but also has implications for several of the arguments commonly raised in favor of sympatric speciation and against allopatric speciation. For example, the argument that allopatric speciation, unlike sympatric speciation, is too slow to be able to account for the presently observed biodiversity, loses much of its strength when also sympatric speciation is driven by external processes. In fact, the supposed higher rate of sympatric speciation is commonly substantiated by arguing that sympatric speciation is internally driven by selection.

The second possibility that has been considered is that the mutation-selection balance on female preference allows for the maintenance of significant preference variation. If selection on female preference were very weak or absent and if the mutation rate were sufficiently high, mutations in female preference genes would accumulate over time, resulting in a sufficiently broad distribution of female preferences in the population (as illustrated in Wu, 1985; Takimoto, 2002; CHAPTER 3 of this thesis). The condition of weak selection (and/or high mutation rate) is not likely to hold in general, although in some species (e.g., marine invertebrates) selection on female preference has been shown to be very weak (Swanson & Vacquier, 1998).

Surprisingly, an obvious third possibility has largely been overlooked in the literature on sympatric speciation (but see mutual mate choice models by Lande *et al.*, 2001; Almeida & Vistulo de Abreu, 2003): genetic variation of female preferences could be maintained by disruptive selection. In contrast to the other possibilities, this option allows sympatric speciation to be described as a directed and adaptive process governed by selective forces – thus eliminating a critical dependence on external events, weak preference selection, or high mutation rates.

The aim of this paper is to critically investigate whether sexual selection by female mate choice can drive adaptive speciation. Specifically, we ask whether female mate choice, through its effect on the joint evolution of male and female mating characters, is capable of generating the conditions under which a polymorphism of female preferences can arise and be maintained. As we will show, by means of individual-based simulations and numerical analysis of a model for the evolution of male and female mating types, the answer to this question is negative. In the traditional models of female choice, mate choice cannot induce *frequency-dependent* disruptive selection on female preferences, which, as we argue, precludes the occurrence of adaptive speciation (Dieckmann et al., 2004). We subsequently propose specific inter- and intrasexual interactions that do generate frequency-dependent disruptive selection. With these additional sources of sexual selection, acting alongside mate choice, sympatric speciation by sexual selection becomes feasible, without any dependence on non-adaptive processes. However, since it is far from trivial to generate frequency-dependent disruptive selection in both sexes simultaneously, we expect that the occurrence of sympatric speciation by sexual selection will be limited to rather specific biological conditions.

A MODEL OF FEMALE CHOICE

We consider the evolution of two continuous phenotypic traits: female preference (denoted p) and the male trait on which female preference acts (denoted q). In every generation, a constant number of N offspring is produced (other forms of population density regulation give identical results, as long as female preference and male trait are ecologically neutral traits; see CHAPTER 5 of this thesis). For every offspring, a female (denoted i) is randomly selected from the population. She is then allowed to choose a mate (denoted j) from the available males. The probability that a particular male j succeeds to fertilize the female is proportional to the male's 'attractiveness' to female i, denoted a_{ij}, which depends on both the female preference value p_i and the male trait value q_j. We keep the model as general as possible and make no assumptions regarding the mechanism of female choice: attractiveness and mate choice may be based on any active or passive process (behavioral, morphological, or other) affecting the probability that a female is successfully fertilized by a particular male. For convenience however, our terminology will not always reflect this general interpretation of the model. 'Mating', for example, will often be used as shorthand for 'successful fertilization'.

We assume that attractiveness is highest when the male trait q_j matches the value preferred by the female. Naively, one could be tempted to specify the male trait q that optimally matches a given female preference p as $q = p$, thereby equalizing traits with preferences. However, this convenient choice is only justified if male trait and female preference phenotypes can be measured on the same scale. This assumption is implicitly made in many models of sexual selection – yet it is problematic, since the choice of scale is not arbitrary, but prescribed by the assumptions made on the mutation process at the genetic level. Since trait and preference represent different entities that are governed by different biological processes (e.g., a color trait may be governed by pigment formation, while a color preference may be governed by processes at the level of color receptors), it is unlikely that a convenient choice of scale at the genotypic level, will also allow us to measure trait and preference on the same scale at the phenotypic level.

There are two more or less equivalent ways to deal with this issue. One could simply define trait and preference such that they are measured on the same scale on the phenotypic level. However, this would require a relatively complicated description of processes at the genetic level, involving, e.g., mutation biases. It has been shown previously that the latter may strongly affect the outcome of sexual selection models (Bulmer, 1989). Alternatively, one could choose to measure trait and preference on a scale determined by their respective mutation processes, rendering the description at the genetic level simple. In that case, one has to assume, as we do in our model, that female preferences are 'translated' into (preferred values of) male traits by means of a 'choice function' c, where $q=c(p)$ is the male trait preferred by a female with preference p. By identifying traits with preferences, most classical models implicitly assume that this choice function is linear, but it is easily conceivable that developmental processes and the mechanisms of perception create all kinds of nonlinearities. Such nonlinearities can have important consequences, since the shape of the function c determines the strength and direction of sexual selection. This can be illustrated as follows. Consider the male trait value that is, on average, preferred by the females in the population. It follows from standard error analysis theory that this quantity, which we denote $\overline{c(p)}$, is approximated by

$$\overline{c(p)} \approx c(\overline{p}) + \frac{1}{2} c''(\overline{p}) \operatorname{var}(p). \qquad [1]$$

If the choice function is linear, its second and higher derivatives will be zero, which, according to equation [1], implies that $\overline{c(p)} = c(\overline{p})$. Hence, the male type that is on average preferred by the females is the same as the male type that is preferred by the female with the average preference. By contrast, nonlinearities in the female choice function translate into a discrepancy between $\overline{c(p)}$ and $c(\overline{p})$. Such a discrepancy generates directed sexual selection, since it directly results in a discrepancy between the optimal and the mean trait and preference values. The direction and intensity of sexual selection depend, respectively, on the sign and magnitude of $c''(\overline{p})$, that is, on the local curvature of the female choice function. For illustration we will choose a particular function c allowing for divergent evolution (see FIGURE 2 later on).

We assume that females tolerate some deviation of male traits from their preferred value, such that attractiveness is described by

$$a_{ij} = g_m\left(c(p_i) - q_j\right), \qquad [2]$$

where here and henceforth g_a denotes a Gaussian function with mean zero and standard deviation σ_a. In particular, the standard deviation of the Gaussian distribution used here, σ_m, determines the specificity of mate choice: higher values of

σ_m correspond to less discriminate mate choice. This model of mate choice assumes fixed absolute preferences and is more conservative than models based on fixed relative, or open-ended preferences (Lande, 1981).

Preferences are potentially costly, especially if a female does not mate when she cannot find a sufficiently attractive male. This occurs, for instance, when searching for mates is time-consuming, or when sperm is limiting. Let us assume that a female encounters any given male at rate $1/\eta$, such that the female can locate and evaluate at most N/η potential mates in a time unit (a time unit is conveniently defined as the time needed to produce a single offspring). Every time the female encounters a male, she may reject him or accept him as a mate. The latter occurs with probability a_{ij}, defined in [2]. A female will produce a single offspring per time unit, as long as she has mated at least once in the previous time interval. Under these assumptions, we can easily compute α_i, the offspring production rate of female i. We find

$$\alpha_i = \frac{\sum\limits_{\text{males } k} a_{ik}}{\eta + \sum\limits_{\text{males } k} a_{ik}} . \qquad [3]$$

If a female encounters many attractive males, she mates multiple times per time unit. In that case, all males that were accepted by the female have an equal probability to father the offspring, such that the probability that a particular male j succeeds to fertilize female i, denoted α_{ij}, is given by

$$\alpha_{ij} = \frac{a_{ij}}{\eta + \sum\limits_{\text{males } k} a_{ik}} . \qquad [4]$$

The parameter η can be interpreted as the time needed to locate and evaluate a particular potential male. When $\eta = 0$, females are not time-constrained and they will always find an attractive mate, regardless of their mating preference. Consequently, there is no direct selection on female mating type p. By contrast, when $\eta > 0$, there is a time-cost associated with mate rejection. Females with deviating preferences will reject most of the potential mates they encounter. Such females will produce offspring at a lower rate, since they waste time searching for more attractive mates. Consequently, when $\eta > 0$, selection will act to match female preference with the predominant male trait.

In this model for female choice, females with different mating types differ only in their preferred male trait value, not in the effort invested in mate choice or the degree of choosiness. All females encounter potential mating partners at the same rate, and the average probability that the female will accept a male as mating partner, which is defined as the integral of a_{ij} over q_j, is independent of female preference (the integral of a Gaussian function is independent of its mean). Consequently, no female preference type is inherently favored. Rather, the selective advantage or disadvantage of a particular preference type is dependent on its match with the male types that are currently present in the population.

Underlying equations [3] and [4] is the assumption that females are limited in the total number of offspring they may produce and that males, on the other hand, may potentially father an unlimited number of offspring, since their reproductive success is limited only by the number of females they succeed to fertilize. This assumption is habitually made in many models of sexual selection, and we will therefore refer to it as the 'typical sex-role assumption'. Note that, under this typical sex-role assumption, the sex roles themselves are hardly 'typical' but rather extremely asymmetric. Later on, we will therefore relax this restrictive assumption.

For simplicity, we assume discrete and non-overlapping generations. After a new generation of offspring has been produced, viability selection occurs. We assume that male survival probabilities vary according to a Gaussian function $g_s(q)$, such that the male trait value $q = 0$ is optimal for survival and extreme male traits suffer a viability disadvantage. Viability selection is stabilizing, and the width of the viability selection function, σ_s, is inversely related to the intensity of direct selection on the male trait.

The model was implemented as an individual-based simulation program. We assumed multi-locus genetics underlying male trait and female preference. Specifically, phenotypic preference and trait values both consist of a genetic and an environmental component. The genetic components are determined by L diploid loci for male trait and another L diploid loci for female preference. We assume a continuum of alleles, that is, the phenotypic effect of each allele is a continuous quantity. All genes are unlinked and alleles interact additively within and between loci, that is, genotypic trait and preference values are the average of the phenotypic effects of the trait and preference alleles. Trait and preference genes are transmitted according to normal Mendelian genetics. Mutations occur with a frequency of μ per allele per generation and are modeled by altering the phenotypic effect of an allele by a number drawn from a normal distribution with a narrow width $\sigma_v \sqrt{2L}$. Under this scaling of the size of mutations, the phenotypic variation caused by mutation is independent of the number of loci L. The environmental component of trait and preference is drawn from a normal distribution with width σ_p for the female preference and σ_q for the male trait. Hence, the non-heritable environmental variance of female preference and male trait is σ_p^2 and σ_q^2, respectively.

In addition, we approximated the individual-based simulation model by assuming mutation-limited evolution and single-locus haploid genetics underlying female preference and male trait (APPENDIX A). This approximation yields equations for the expected growth rate of rare female preference or male trait mutants interacting with a monomorphic resident population. We then use adaptive dynamics theory (Metz *et al.*, 1996; Dieckmann & Law, 1996; Geritz *et al.*, 1998) to numerically calculate the expected evolutionary trajectories of female preference and male trait (APPENDIX B). Throughout the manuscript, results based on this adaptive dynamics approximation will be used to complement results obtained from the individual-based simulations.

DISRUPTIVE SEXUAL SELECTION, BUT NO SYMPATRIC SPECIATION

The individual-based simulations show that populations starting out from arbitrary initial conditions quickly converge to combinations of male trait and female preference values such that $q \approx c(p)$ (FIGURE 1). This is not surprising, since, at those combinations of trait values, the trait expressed by the males optimally matches the mating preference exerted by the females, and sexual selection on the male trait selects for such optimal matching. The same is true for direct selection on female preference, since females with deviating preferences suffer more from the cost of mate choice.

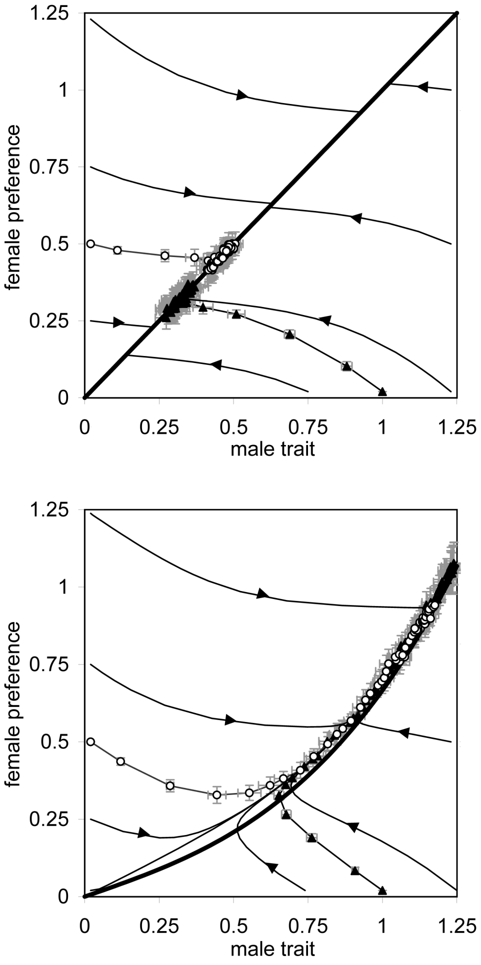

FIGURE 1 – EVOLUTION ALONG THE FEMALE-CHOICE FUNCTION

For a given female-choice function (thick black curves), the two panels show the evolutionary trajectories of male trait and female preference from different initial conditions as obtained by individual-based simulations (open circles and filled triangles show simulations from two different starting conditions) and numerical integration of the deterministic equations derived in Appendices A and B; equation [B-1] (thin black curves with arrows). Parameters: $N = 500$, $\sigma_m = 0.15$, $\sigma_p = \sigma_q = 0.2$, $\eta = 25.0$, $L = 2$, $\mu = 0.025$, $\sigma_v = 0.05$, and $\sigma_s \to \infty$ (no viability selection on male trait). Individual-based simulations lasted for 3000 generations, with data plotted every 50 generations. The resulting individual-based trajectories represent averages over five independent simulations; error bars indicate the standard errors of the mean across the replicate simulations. For details about the nonlinear female-choice function see APPENDIX B.

After this initial phase of rapid evolution, a slower phase of adaptive change along the female-choice function sets in. In the absence of viability selection on the male trait, the direction of evolution along the female-choice function is completely determined by the local curvature of that function (FIGURE 1). If the function is linear, the female choice function defines a line of equilibria, along which the population drifts neutrally (FIGURE 1, upper panel). This line of equilibria disappears as soon as the female choice function becomes nonlinear (FIGURE 1, lower panel). In that case, the local curvature of the female choice function generates directed sexual selection, thus forcing the population to move slowly along the female choice function. This can be understood from the fact that the local curvature of the female choice function translates into a discrepancy between the average preferred male trait value and the male trait value that is preferred by a female expressing the average preference value (equation [1]; Van Doorn *et al.*, 1998). Because of this discrepancy, the optimal male trait value will be different from the population mean male trait value, which will therefore shift towards the optimal value. However, this will immediately induce a corresponding change in the population mean female preference value, since the female preference value at which the costs of choice are minimized is the one that matches the population mean male trait value.

In general, the female-choice function could have any shape and it need not necessarily be smooth, as in FIGURE 1. For different biological systems, the shape of the respective female choice functions will vary with specific nonlinearities induced by processes such as development and perception. Here we will not attempt to model this biological complexity in any detail. Instead, without harm to our argument, we will simply choose an example female-choice function in such a way that disruptive sexual selection is generated. In FIGURE 2, the female-choice function (thick black line) is shaped such that sexual selection can drive the population in two different directions, towards two possible endpoints of evolution. The latter are located at the intersection points of the null-isoclines for the rate of change of trait and preference (thick gray lines). Females prefer costly and exaggerated male traits in both of these endpoints. However, despite a potential for the occurrence of evolution in multiple directions, diversification of female mate preferences was never observed in our simulations. The two replicate runs (circles and triangles) represented in FIGURE 2 were both started from the initial conditions $p_0 = q_0 = 0$, i.e., exactly at the point where sexual selection is disruptive. Nevertheless, the two simulations show no speciation, but evolution towards either one of the two possible stable endpoints of evolution. For some parameter conditions, a polymorphic transient (as in FIGURE 2) or a permanent genetic polymorphism of male trait arose, but the distribution of female preference always remained unimodal. In fact, there is always a clear boundary line (dashed curve IN FIGURE 2) that separates the initial conditions from which the respective endpoints of evolution are reached.

Why does the distribution of female preference remain unimodal in all of our simulations, whereas genetic polymorphism in the male trait does arise under suitable conditions? The answer to this question lies in the fact that the selective forces acting on female preference are fundamentally different from those acting on the male trait. This difference derives from a basic assumption of our model: females are limited in the number of offspring they may produce by time or energy constraints. These factors are not influenced by the preferences of the other females in the population. As a consequence, female fitness is not affected at all by the strategies of other females, and, therefore, selection on female preference is independent of the frequencies of other preference strategies in the population. Male fitness, on the other hand, varies with the strategies of other males, since it is determined mainly by success in competition between the males for access to the females.

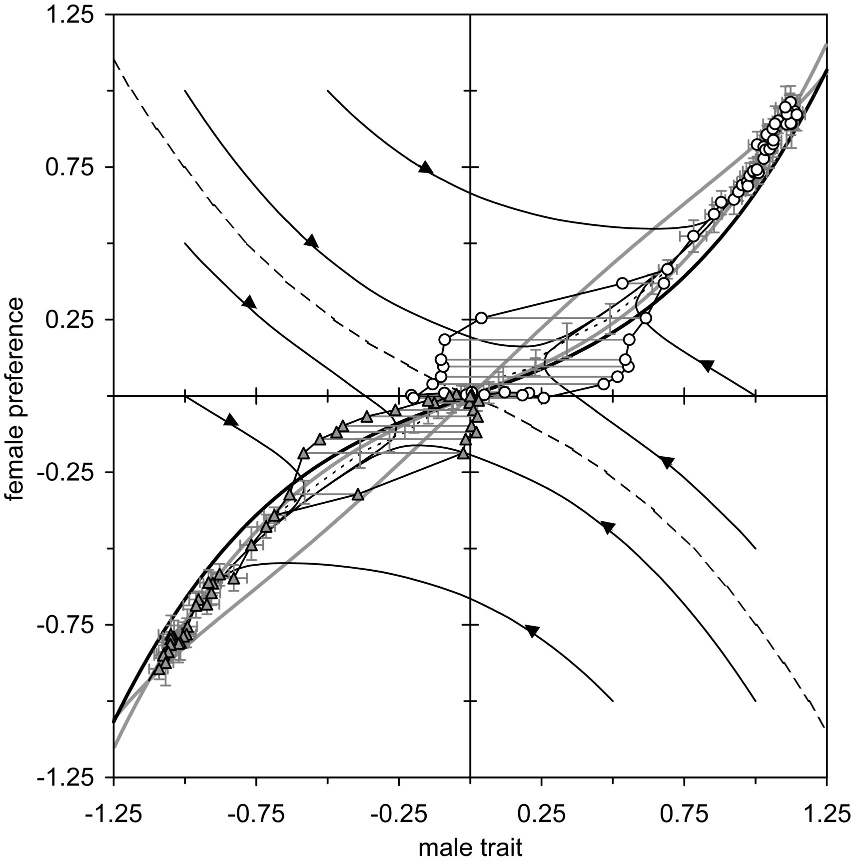

FIGURE 2 – DISRUPTIVE SEXUAL SELECTION, BUT NO SPECIATION

Two replicate individual based simulations (circles and triangles) were started from the initial conditions $p_0 = q_0 = 0$, i.e., exactly at the point where sexual selection is disruptive. The simulations do not show speciation, but evolution towards one of two possible endpoints of evolution. At the start of both simulations, male traits are polymorphic. There are two clearly distinct male trait alleles, indicated separately in the graph, as circles or triangles joined by horizontal gray lines. Later, the populations again become monomorphic for male trait. Grey error bars indicate within-population variation of preference and trait (not, as in FIGURE 1, variation between replicate runs). Other lines in this graph represent the female choice function (thick black line), null-isoclines for the rate of change of trait and preference, corresponding to the solutions of the separate equations [B-1] in APPENDIX B (thick gray lines), evolutionary trajectories predicted by the adaptive dynamics approximation (thin black lines with arrows), and the boundary line that separates the basins of attraction of the two stable equilibria (dashed curve). Parameters: $N = 1000$, $\sigma_m = 0.15$, $\sigma_p = \sigma_q = 0.3$, $\eta = 25.0$, $L = 1$, $\mu = 0.0125$, $\sigma_v = 0.01$, and $\sigma_s = 1.0$. Individual-based simulations lasted for 10000 generations, with data plotted every 200 generations. For details about the nonlinear female-choice function see APPENDIX B.

This difference has important consequences. Frequency-independent selection, such as the selection on female preference in the model above, is unable to support genetic polymorphism (APPENDIX C), at least when the underlying genetics is not governed by strong constraints (as, for example, in the case of over-dominance). This point is illustrated in the upper panel of FIGURE 3. For this figure, we first calculated an evolutionary trajectory of female preference (thick black curve). At every point in time we subsequently computed the fitness of rare female preference mutants in an equilibrium population with the current resident female preference. As reflected by the bimodal shape of the resulting female-preference fitness landscape, selection on female preference is disruptive at the start of the simulation. Due to the fact that selection on female preference is frequency-independent, the fitness landscape does not change in response to changes in the resident female strategy. As a result, the population can easily escape from a point where selection is disruptive and will do so without polymorphism being generated.

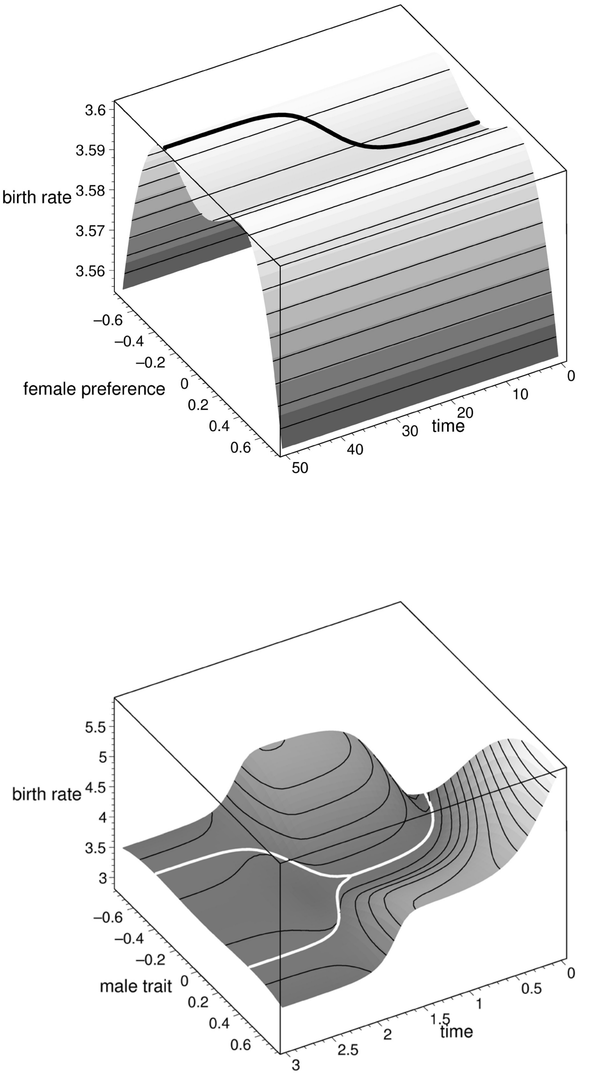

FIGURE 3 – DIFFERENCES BETWEEN FREQUENCY-INDEPENDENT AND FREQUENCY-DEPENDENT SELECTION

The adaptive dynamics approximation was used to calculate an evolutionary trajectory of female preference (upper panel, thick black curve). For this illustration, a fixed dimorphism of male trait was considered, with two equally frequent male types at $q_1 = -q_2 = 0.75$. At every point in time, we determined the birth rate of rare female preference mutants in an equilibrium population with the current resident female preference; Selection on female preferences is frequency-independent. Consequently, the fitness landscape does not change in response to changes of the resident female preference. The lower panel shows evolutionary branching of the male trait (evolutionary trajectories are represented by white curves), and the associated dynamic change of the fitness landscape caused by frequency-dependent selection. For this simulation, female preference was kept at a constant value, $p = 0$. Parameters are as in FIGURE 2.

Frequency-dependent selection, by contrast, allows for the origin and maintenance of stable genetic polymorphisms under far more general conditions. The effects of frequency-dependent disruptive selection are highlighted in the lower panel of FIGURE 3. First, an evolutionary trajectory of the male trait (white curves) was calculated. In the simulation, the male trait first converges to $q=0$, where matching with the average female preference is maximal. Then a stable polymorphism of two male-trait genotypes emerges. This course of events is typical of the process of evolutionary branching (Metz *et al.*, 1996; Geritz *et al.*, 1998; Dieckmann *et al.*, 2004), as formulated by the theory of adaptive dynamics. The underlying male-trait fitness landscape, calculated in analogy to the female-preference fitness landscape described above, drastically changes over time, responding to changes in the resident male trait because of frequency-dependent selection on that trait. The dynamically changing fitness landscape makes it possible that evolution, even though always moving uphill, first converges to the bottom of a fitness valley, that is, to a point where selection turns disruptive. A monomorphic population cannot escape from such a valley, since any step away from the bottom of the valley would change the landscape in such a way that the population is driven back. The only way for the population to escape from the fitness minimum is to become dimorphic in the male trait.

If selection on female preferences is frequency-independent, three interrelated problems arise, all potentially preventing speciation:

1 – Sexual selection on female preference is disruptive only when the population mean trait values are close to the boundary line between the two stable equilibria (of the order of a standard deviation away), but directional everywhere else.

2 – A population tends to evolve away from the area in which selection is disruptive.

3 – Even if a population spends a long time in the area of disruptive sexual selection and a polymorphism arises, this polymorphism quickly disappears because there are no selective forces to stabilize it.

Although the first and second problem can be overcome if the initial conditions are suitably chosen (the initial population should exhibit considerable variation and it should be perched on the boundary line between the two stable equilibria), the third problem cannot. At best, female preferences may transiently diversify, but due to the lack of stabilizing forces, the two resulting daughter species can only coexist ephemerally. Note that this transient phase might seem deceptively long in deterministic models (as in Takimoto *et al.*, 2000), an artifactual feature that disappears as soon as only a minimal amount of stochasticity is introduced. This aggravates the problem mentioned in the introduction: even if a large amount of genetic variation of female preferences is initially present – by a sudden change of the environment, or by mutation pressure – speciation is still impossible, since there is no selection that will stabilize the coexistence of the daughter species.

FEMALE COMPETITION FOR MALES RENDERS SELECTION ON FEMALE PREFERENCE FREQUENCY-DEPENDENT

The solution to the problems highlighted above might seem to be straightforward: in order to allow for sympatric speciation, selection on female preference must be made dependent on the strategies of other females in the population. It is not at all unlikely that such dependence exists. For example, if we replace the assumption that males can potentially father an unlimited number of offspring by the more realistic assumption that also male matings are limited (to a small extent) by time or energy constraints, then selection on female preference immediately becomes dependent on the strategies of other females in a population.

Male limitation of this type can arise in many different ways. For example, males may be limited in the amount of time they can invest in parental care, such that a male that has fathered many offspring cannot provide paternal care for all of them. Alternatively, males may have to spend time on courting a female, which makes them temporarily unavailable for other females. Another possibility that may be of relevance for specific natural systems is that males are limited in the amount of sperm they can produce. In all these cases, male limitation introduces (indirect) mate-competition between the females, such that females preferring males not already chosen by other females will enjoy elevated fitness.

Although the above examples show that male limitation is biologically plausible, work is needed to delineate the biological conditions under which male limitation is also strong enough to result in appreciable intensities of competition between females. We leave this issue unresolved, and, for the sake of our argument, examine an example in which competitive interactions between females are quite strong. For this purpose, we slightly extend our model by allowing for the fact that the quality of a male partner may deteriorate with the number of times the male has already mated during a season (for example, because the male can only offer a fixed amount of parental care, which has to be shared among all his offspring); we also assume that a female cannot ascertain how many times a male has mated before. The female therefore still selects a male on the basis of her preference. We assume that if she selects a male that has mated n times before, she produces viable offspring with probability φ^n $(0 < \varphi < 1)$. The parameter φ determines how fast male quality deteriorates with the number of matings. For $\varphi = 1$, we recover the model analyzed above.

With this modification of the model, there are parameter conditions under which a genetic polymorphism of female preference evolves. In FIGURE 4, we systematically varied the environmental variances of male trait and female preference (σ_q^2 and σ_p^2, respectively), thereby manipulating the population variances of trait and preference (when mutations are rare, genetic variation can be neglected). As we will explain shortly, the latter variances determine, relative to the other parameters, whether selection on the male trait and the female preference will be stabilizing or disruptive.

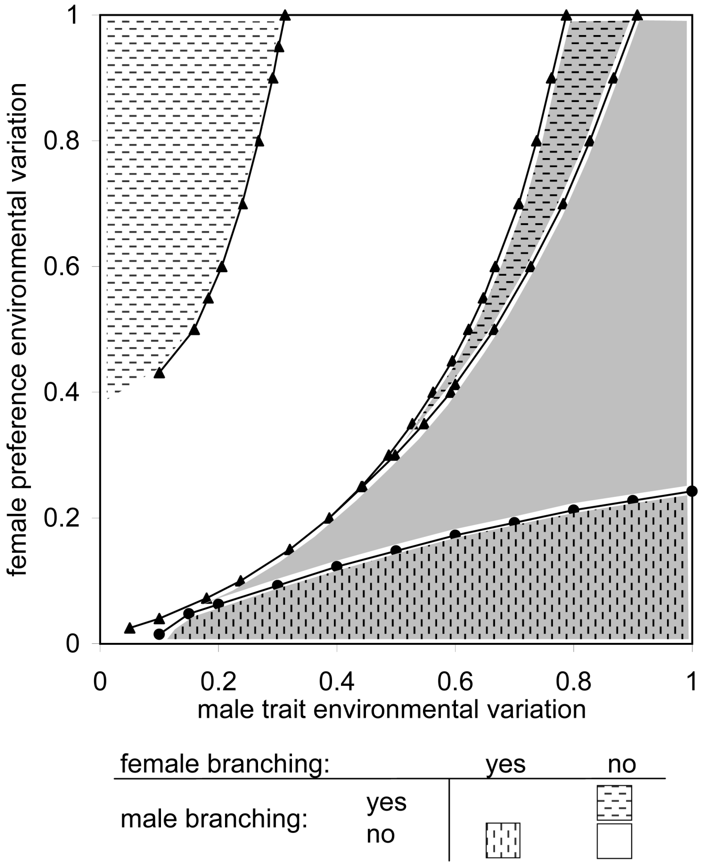

FIGURE 4 – IMPOSSIBILITY OF SIMULTANEOUS EVOLUTIONARY BRANCHING

As explained in the text, male branching occurs when the (environmental) variation in female preference (σ_p) is large relative to the (environmental) variation in male trait (σ_q). By contrast, female branching occurs in the opposite corner of parameter space. The picture is complicated slightly by the fact that, in the initial phase of evolution, females do not always evolve preferences for costly male traits (this depends on the stability of the equilibrium $p = q = 0$, which can be assessed from equation [B-1] in Appendix B). In the white region, monomorphic evolution leads to an equilibrium at which female preference for costly male traits has been established. In the gray region, this does not occur, and the endpoint of monomorphic evolution is the equilibrium that optimizes male survival. When male-trait environmental variation is large, extreme male traits suffer (on average) more from viability selection, and therefore female preferences for costly male traits evolve less easily. For a similar reason, male-trait branching requires more extreme parameter combinations when females exhibit preference for costly male traits, since branching will then on average lead to larger viability disadvantages. All boundary lines in this plot (triangles: male primary branching, circles: female primary branching) were calculated using the adaptive dynamics approximation. Parameters are as in FIGURE 2, with $\varphi = 0.75$. Numerical instabilities prevented accurate calculation of selection gradients for very small σ_q; no points are therefore shown for the leftmost region of parameter space.

A polymorphism of female preference can evolve when the variation of female preference in the population is small with respect to the population variation of male trait (FIGURE 4). Under these conditions, males in the tails of the distribution of trait values are rarely chosen, and, therefore, females that choose such males are favored. Because of the fitness advantage of females that prefer extreme male types, female preference is expected to diversify, and this is accomplished by evolutionary branching. Notice that in this situation the evolution of female preference is governed by the same mechanism underlying the evolution of resource-utilization traits: the population-level phenotypic distribution of male trait acts like a resource availability spectrum, the individual-level phenotypic distribution of female preference acts like a resource utilization spectrum, and evolutionary branching occurs if the former is wider than the latter (see, e.g., Dieckmann & Doebeli 1999). This highlights a structural similarity between selection pressures acting on ecological characters and those acting on sexual traits (CHAPTER 3 of this thesis).

The conceptual relevance of this similarity, however, is much undermined by the following observation: parameter conditions under which female-preference branching occurs do not overlap with those under which the male trait undergoes evolutionary branching (FIGURE 4). Indeed, male branching occurs when the variation of female preference in the population is large with respect to the population variation of the male trait. In that case, disruptive selection favors male-trait specialization on females in the tails of the relatively broad distribution of preferences, since those females are hardly competed for in a monomorphic male population (CHAPTER 3 of this thesis). Irrespective of the strength of competition between females (determined by the parameter φ), we never found overlapping conditions for female preference and male trait branching. Since there is no reproductive isolation without a polymorphism evolving in both female preference and male trait, speciation does not occur.

The reason for the mutual exclusiveness of the conditions for male-trait and female-preference branching lies in the fact that male fitness increases when a male mates with more females, whereas female fitness decreases in the same situation. This fundamental conflict between the sexes translates into opposing selective forces: when it pays the males to diversify and undergo branching, the females will experience stabilizing selection, and vice versa. A simple calculation shows that this intuitive explanation applies under general conditions (APPENDIX D).

MALE-MALE COMPETITION PROVIDES ADDITIONAL DISRUPTIVE SELECTION

The results of the previous section show that the areas in parameter space in which female preference and male trait undergo evolutionary branching do not overlap and must thus be enlarged in order to create a potential for sympatric speciation. This can only be accomplished by assuming an additional source of disruptive selection acting on either females or males, separate from the disruptive selection already generated by mate choice and female-female competition. Such additional disruptive selection could be caused by a variety of mechanisms, and any process that leads to an intrinsic advantage of rarity (Weissing, 1996) would be adequate.

For the sake of concreteness, we consider a particular example, in which the trait subject to female preference is also involved in male-male competition. This is, for example, well known for sticklebacks, where the red coloration of the male is used as a signal by females (in the context of mate choice) as well as by males (in the context of aggressive interactions). Intuitively, one would also expect males to make their competitive strategies dependent on the traits on which female preference acts, at least, as long as males compete for no other resource than the female's attention. In that case, it makes no sense to waste valuable energy in fighting a male with whom no potential partners are shared (Verkiel, 2002). More specifically, one would expect that males behave less aggressively towards one another if their mating traits are less similar. This idea is currently under empirical investigation for haplochromine cichlids, where it has been suggested that males behave less aggressively towards rare male color morphs and that these rare morphs are therefore favored in male-male competition (Dijkstra & Groothuis & Dijkstra, *ms. in prep.*).

Inspired by this biological example, we therefore assume that all males compete to establish mating territories. When a male tries to establish a territory, he has to compete with the other males already owning a territory. In accordance with the preceding discussion, the intensity of competition, f_{ij}, between two males i and j is taken to be dependent on the difference between their trait values,

$$f_{ij} = g_c\left(q_i - q_j\right) \ . \qquad [5]$$

The width of the Gaussian g_c, σ_c, determines how strongly male aggression is influenced by male trait differences. Male aggression is independent of male trait differences when σ_c approaches infinity, whereas males fight only with identical males when σ_c is very small.

A male experiencing very intense competition is assumed not to be able to establish or maintain a territory. The total strength of competition experienced by a male, $\overline{f}_i$, is given by

$$\overline{f}_i = \sum_{\substack{\text{territory owners } j \\ j \neq i}} f_{ij} \ . \qquad \textbf{[6]}$$

When this total strength exceeds a threshold value θ, the male loses his territory. If, on the other hand, $\overline{f}_i$ falls below the threshold, a male is allowed to establish a territory, if he does not already possess one. Throughout the rest of this paper the threshold value θ is chosen such that one quarter of a population consisting of identical males is able to maintain a mating territory (the precise numerical value of θ depends on the model parameters σ_c, σ_s and the population size N). In the individual-based simulations, all males were given several opportunities to establish a territory, allowing for a stable composition of territory owners to be reached. After that, females were allowed to choose a mate from the males that had succeeded to obtain a territory.

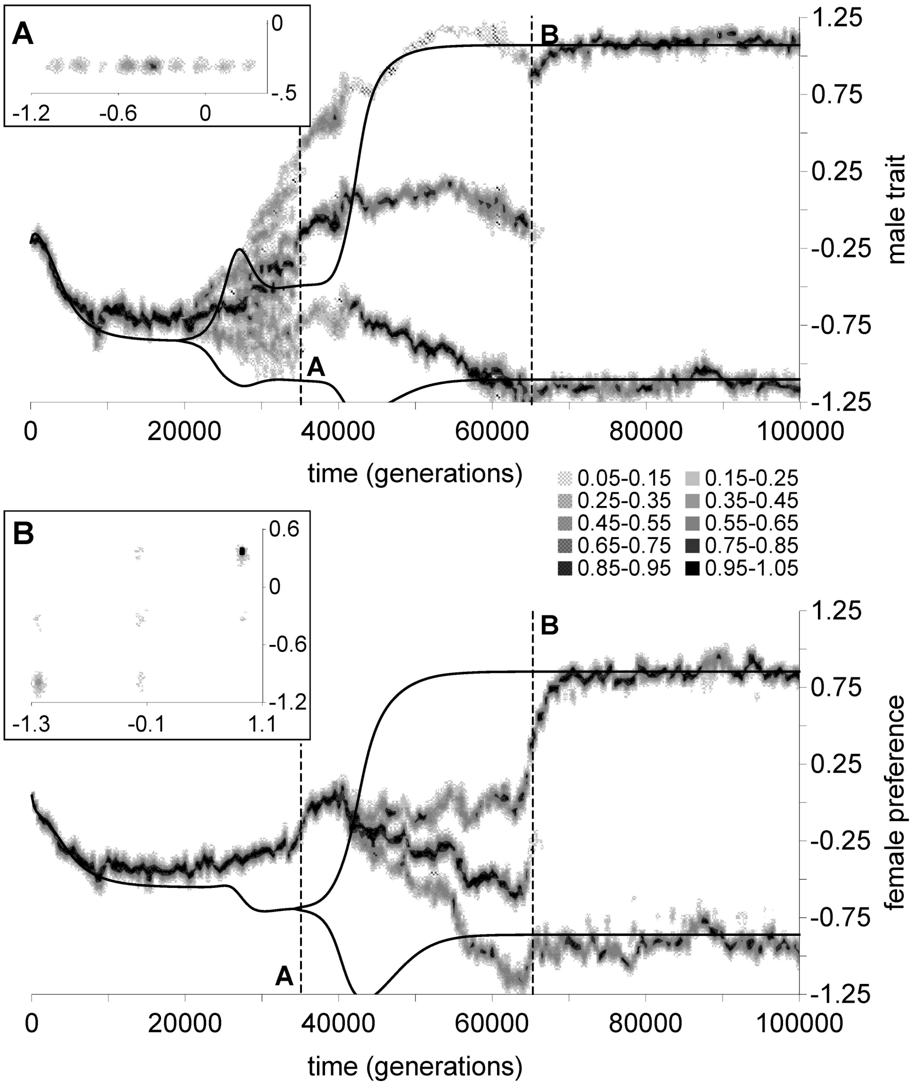

FIGURE 5 – SYMPATRIC SPECIATION

Two plots show the relative frequency distributions of male-trait and female-preference genotypes (indicated on a gray-scale) in a population with $N = 1000$ in which both trait and preference are based on four diploid loci, $L = 4$. The thin black curves represent trajectories of the corresponding adaptive dynamics approximation. Small insets depict the frequency distribution of male trait (horizontal axis) and female preference (vertical axis) at two moments during the simulation (inset A: $t = 35,000$ generations, just before polymorphism is lost at all but one male trait locus, inset B: $t = 65,000$ generations, just before full linkage disequilibrium develops). Parameters are as in FIGURE 4, with $\sigma_c = 1$.

As shown in FIGURE 5, the additional disruptive selection generated by male-male competition may indeed result in sympatric speciation. The figure shows a complicated sequence of events, eventually resulting in speciation: over the first 10,000 generations, females evolve preferences for costly male traits, after which a polymorphism of male trait arises ($t = 20{,}000$ to 35,000 generations). Each of the four loci coding for the male trait undergoes evolutionary branching, transiently giving rise to nine genotypic clusters (inset A). Triggered by a sudden change of female preference around that time, the polymorphism in male trait is lost at three of the four loci, whereas the polymorphism at the remaining locus continues to grow. This divergence induces female-preference branching around $t = 40{,}000$ generations. Females specialize on one of the extreme male traits, leading to a highly skewed distribution of male-trait alleles in the population. At $t = 65{,}000$ generations, the female-preference polymorphism has grown sufficiently in order to allow for the build-up of linkage disequilibrium of trait and preference alleles; the distribution of genotypes at this moment in time is shown in inset B. Full linkage disequilibrium then evolves quickly, and as, a consequence, the heterozygotes (middle branches) carrying two different male-trait or female-preference alleles at the polymorphic locus disappear. In the end, two stably coexisting and reproductively isolated daughter species remain.

For the parameters used in the multi-locus simulation represented in FIGURE 5, there is good agreement between simulation and the corresponding adaptive dynamics approximation (APPENDIX E; also shown in FIGURE 5), even though our adaptive dynamics approximation is based on haploid single-locus genetics and on the assumption of mutation-limited evolution. We tested other parameter conditions and found that the adaptive dynamics approximation correctly predicted the evolutionary equilibrium eventually attained in the individual-based simulations. For the transient behavior, we found better quantitative agreement between adaptive dynamics approximation and the simulations for smaller mutation step size σ_v. Larger mutation step sizes result in increasingly rapid evolutionary branching in the simulations, such that the population already undergoes evolutionary branching before the predicted evolutionary equilibrium for the monomorphic population is reached. These observations are in line with theoretical results predicting that the rate of evolutionary change in a monomorphic population is proportional to $\mu\sigma_v^2$, whereas the rate of evolutionary branching is proportional to $\mu\sigma_v^3$ (Metz *et al.*, 1996). For this reason, we use small mutation step sizes ($\sigma_v = 0.01$) in our simulations, and a high mutation rate ($\mu = 1.25 \cdot 10^{-2}$, due to limitations on computer time). Simulations with a more realistic mutation rate ($\mu = 1 \cdot 10^{-5}$), and a larger mutation step size ($\sigma_v = 0.15$), however, reach the same evolutionary end state and show speciation on the same timescale as the simulation represented in FIGURE 5.

We have used the more tractable adaptive dynamics approximation to further explore the parameter space of our model. FIGURE 6 shows that the parameter space is subdivided in two regions. In the first region (white background), sexual selection drives the monomorphic evolution (i.e., the adaptive change before branching has occurred) to an equilibrium characterized by female preference for

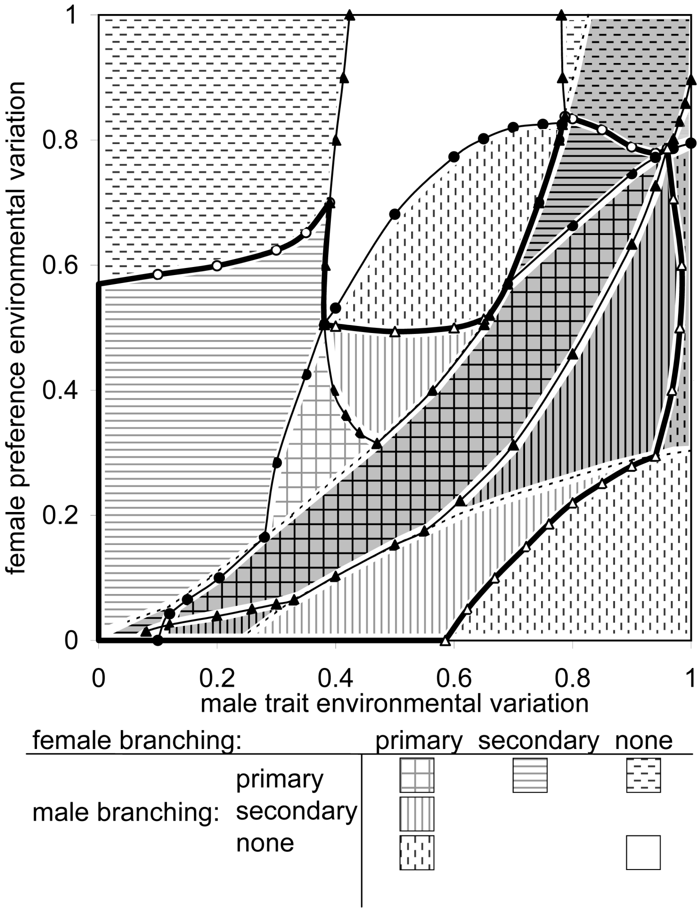

FIGURE 6 – Possible outcomes of female-female and male-male competition

In comparison with FIGURE 4, additional disruptive selection on the male trait has enlarged the regions in parameter space in which male and female branching occur, now allowing for sympatric speciation in the region delimited by the thick black curve. There are now regions in which female and male branching are possible simultaneously or in arbitrary sequence (male *and* female primary branching), and, in addition, regions in which branching in one of the traits induces branching in the other one (secondary branching). Sympatric speciation is possible in all these regions, but not in regions where only a single trait or none of the traits undergoes branching. Like in FIGURE 4, the gray background extends over parameter combinations where females do not evolve preferences for costly male traits. As in FIGURE 4, lines with triangles delimit male branching areas, and circles delimit female branching areas. Filled and open symbols are used to distinguish between primary and secondary branching, respectively. Parameters are as in FIGURE 4, and male-male competition was incorporated as explained in the text.

exaggerated (costly) male traits (as in the first 2000 generations in FIGURE 5). In the second region (gray background), this process does not occur and monomorphic evolution converges to the equilibrium $p = q = 0$, where the male trait value is optimal for survival. In both regions (white and gray), there are large areas in which female preference and male trait undergo branching. Crucially, there now is a substantial overlap between these areas, in which speciation is possible. Depending on parameter values, the speciation process may unfold in different ways.

First, the order in which female preference and male trait undergo evolutionary branching may vary. In some regions, both male trait and female preference may undergo branching from a monomorphic population (both traits are capable of 'primary branching'), in other regions, branching of one of the traits is possible only after the other trait has undergone branching and has diversified sufficiently (see Doebeli & Dieckmann (2000) for another model in which such 'secondary branching' occurs).

Second, speciation may occur with or without the initial establishment of mating preferences for costly male traits. In the former case the population first evolves towards a stable equilibrium at which females show preference for costly male traits, in the latter case monomorphic evolution converges to the viability optimum for the male trait. Surprisingly, the outcome of this initial monomorphic phase of evolution does not seem to influence the further speciation process qualitatively. Quantitatively, there is an effect, though. Ironically, the establishment of female preferences for costly male traits inhibits male-trait branching. When males express costly mating traits, male-trait branching occurs only when there is a large difference between the environmental variances of male trait and female preference (FIGURES 4 & 6), indicating that male-trait branching now requires much stronger disruptive selection. The reason for this effect is that branching after the establishment of female preferences for costly male traits results in two male types that, on average, suffer more from viability selection. This stabilizing force counteracts male-trait branching. Not surprisingly, female preference branching is facilitated under these conditions (FIGURE 6). When females have evolved preferences for costly male traits, females, on average, prefer extreme males from one of the tails of the male distribution, rather than the most common males. This increases the intensity of competition between the females, and hence the intensity of disruptive selection on female preference.

DISCUSSION

The essential ingredients of sympatric speciation by sexual selection are selective forces that not only account for the evolution of male-trait polymorphism, but also generate and maintain a polymorphism of female preference. It is already well established that female mate choice can cause frequency-dependent disruptive selection on male traits, allowing for evolutionary branching of male secondary sexual characters. However, under the typical sex-role assumption, female mate choice does not result in any dependence of female mating success on the strategies of other females present in the population. Consequentially, female choice cannot generate frequency-dependent selection on female preference, and, therefore, the emergence and maintenance of a polymorphism in female mating preferences is precluded. Even when one is willing to accept that non-selective agents, such as sudden changes of environmental conditions or strong mutation pressure, are responsible for generating female preference polymorphism, the problem of maintaining such polymorphisms remains unresolved. This is a neglected but fundamental problem (APPENDIX C) for the theory of sympatric speciation by sexual selection, which underlies and explains several undesirable features of current models, such as the unstable coexistence of daughter species after divergent runaway processes.

Even though frequency-dependent interactions between females are neglected in traditional models, there are many ways in which female fitness could be dependent on the strategies of other females. One obvious mechanism, investigated in the present paper, is competition between females, which occurs as soon as males are limited in the number of offspring they can father. This immediately results in (indirect) competition between the females, generating frequency-dependent disruptive selection on female preference. Under suitable conditions, this disruptive selection is sufficiently strong to maintain a stable polymorphism in female preference. As FIGURE 4 showed, however, competition for males among females can only generate disruptive selection on female preference under conditions for which indirect competition for females between males (by means of female choice) results in stabilizing selection on male trait (and vice versa). Speciation, requiring both female-preference and male-trait polymorphism, therefore remains impossible under this relaxation of the typical sex-role assumption. The mutual exclusion between the conditions under which selection on males is disruptive and of those under which females experience disruptive selection derives from a fundamental conflict between the sexes regarding the mating rate of males (APPENDIX D): males benefit from mating as often as possible, whereas females benefit when they mate with males that have not mated very often before.

Because of the non-overlapping conditions for male-trait and female-preference branching, additional and independent disruptive selection is required to make sympatric speciation possible. Direct competition between males was presented here as a possible selective agent favoring rare male-trait varieties. This source of additional disruptive selection, acting together with sexual selection by female choice and with competition between females for mates, can then drive adaptive sympatric speciation (Dieckmann et al., 2004), without requiring a dependence on high mutation rates or external events. We have shown that this conclusion applies for a range of model parameters and even when trait and preference are based on several diploid loci with free recombination. Because of the frequency-dependent nature of the combined selection pressures, the daughter species stably coexist after speciation, even without ecological divergence.

These results permit us to conclude that the sympatric speciation observed in our simulations is a robust phenomenon, as far as genetic details and parameter conditions are concerned. However, this does not imply that sympatric speciation by sexual selection will occur under general biological conditions. For that, one needs to consider the robustness of speciation on the level of the processes involved in speciation. We have shown that sympatric speciation occurs only when several independent biological processes are acting simultaneously on the same mating traits. In our example model, the required interactions are represented by mate choice, significant female-female competition for males and male-male competition based on the trait also used in mate choice. Although the individual components of this cocktail appear to act in a wide variety of species, their simultaneous presence seems to be restricted to a few specific cases.

Therefore, we conclude that sympatric speciation by sexual selection is possible, but unlikely in general, since it requires rather specific conditions: first of all, selection must be frequency-dependent in *both* sexes, and second, selection must be disruptive in both sexes *simultaneously*. The latter will often require an additional and independent source of disruptive selection acting on the same traits. That these conditions are independent of the specific model structure and the biological scenarios used to illustrate our points follows from verbal and formal arguments (Appendices C and D). In particular, our arguments are equally valid for models with different assumptions regarding the mate choice process (open-ended preferences, relative preferences). Moreover, the core of our argumentation applies to three recent models of sympatric speciation that include interactions between the sexes other than (just) female mate choice. We will now discuss these models in some detail, in order to illustrate that the processes underlying frequency-dependent selection on females could be diverse in nature and that competition between females for males, although a likely factor, is certainly not the only candidate mechanism.

The first model describes sympatric speciation by sexual conflict (Gavrilets & Waxman, 2002). The basic assumptions are that mating rates, as in our present model, depend on the match between male and female mating characters. However, the sexes have conflicting interests, since mating is assumed to be costly for females but advantageous for males. As a consequence, the male mating character evolves to optimally match the female mating character, but the female mating character evolves away from the male mating character, resulting in a coevolutionary chase between the sexes. Under suitable parameter conditions, however, this coevolutionary chase can be stopped. This occurs when a female mutant arises by a large mutational step, such that the males are now trapped between the old female mating character and the new mutant type. The females then diversify into two separate clusters, which may subsequently also trigger diversification in the male mating character, resulting in sympatric speciation. Gavrilets and Waxman observed speciation while assuming unlimited availability of males and without introducing any additional processes to generate frequency dependence, an observation that, at first sight, would appear to contradict our conclusions. However, Gavrilets and Waxman assumed a large population, in which several mutants with rather different phenotypes were already present in low densities. As a result, frequency-dependent selection on female preference could arise from the *antagonistic interactions between females and a genetically polymorphic male population*. In a polymorphic male population, the fitness of a female mating strategy depends on the shape of the frequency distribution of male mating characters in the population. At the same time, the frequency distribution of male mating characters will always accommodate itself to the mating strategies of females in the population in such a way that male fitness is maximized. Due to this feedback on the population dynamical timescale between female mating strategies and the 'environment' (i.e., the frequency distribution of male mating characters), selection on female mating characters is clearly frequency-dependent. We emphasize that interactions with genetically polymorphic populations result in frequency-dependent selection, but not necessarily frequency-dependent disruptive selection. In the Gavrilets and Waxman model, however, selection is disruptive due to the nature of the interactions between males and females: the frequency distribution of male mating characters will tend to be skewed in such a way that it matches with the most abundant female mating character, and therefore, due to the sexual conflict, rare female mating characters are favored. Whereas our model focused on mechanisms influencing the availability of potential partners, the model of Gavrilets and Waxman illustrates that also mechanisms influencing the density of potential partners can generate the required frequency-dependent disruptive selection on female mating characters.

The second model (Almeida & Vistulo de Abreu, 2003) is again a model of sympatric speciation by mate choice, but it deviates from traditional sexual selection models in that it analyses the consequences of *mutual mate choice*. In this model, both females and males engage in mate choice, and both sexes may abandon their current partner when encountering one that better matches their mate choice criteria. Only pairs that persist for some minimal period of time produce offspring. Mutual mate choice generates competition between males for females as well as competition between females for males. The simultaneous action of these two types of competition can drive sympatric speciation, since it leads to an intrinsic advantage of rarity for both male and female mating characters. Individuals exhibiting rare mate choice criteria are favored because those individuals will be less likely to abandon their partner or to be abandoned by their partner before the minimal period required to produce offspring has elapsed. Although Almeida and Vistulo de Abreu modeled quite different biological processes than we did, the two models are almost identical at the level of the mechanisms involved in speciation. In both models, the source of frequency-dependent disruptive selection on both sexes is competition for mates. In our model this competition is caused by direct male-male competition for mating territories and indirect competition between females due to limited male availability. In the model of Almeida and Vistulo de

Abreu, there is indirect competition within both sexes due to the fact that the lifetime of a pair bond is determined by the mate choice criteria of other individuals in the population.

The third model (CHAPTER 3 of this thesis) integrates sexual selection with the ecological approach to sympatric speciation, and, as such, builds on classical (Felsenstein, 1981) and recent models (Dieckmann & Doebeli, 1999) of ecological speciation. The model does not deviate from the typical sex role assumption and, as in the present paper, it is assumed that mating rates are determined by the match between male and female mating characters. In addition to female preferences and male mating traits, the model also involves ecological characters, which determine an individual's success in competing for ecological resources and, through processes like habitat choice, also pleiotropically affect mating rates. On this basis Van Doorn & Weissing (see CHAPTER 3 of this thesis) show that sympatric speciation can be initiated by the simultaneous and mutually dependent diversification of mating characters and ecological characters. This option only exists when the pleiotropic interaction between ecological characters and mating rates is sufficiently strong. In the light of the conclusions of the present study, these results, which are in line with those based on earlier models (e.g., Felsenstein, 1981), can now be explained as follows. If sufficiently strong, the pleiotropic interaction between ecological characters and mating types allows for the development of a linkage disequilibrium between ecological characters and mating characters. The linkage disequilibrium, in turn, is responsible for generating the necessary frequency-dependent selection on female preference. Rare preference alleles are favored, not because of processes related to mate choice, but because rare preference alleles are, due to the linkage disequilibrium, often associated with rare ecological characters, which are favored in ecological resource competition. This illustrates that, through *linkage disequilibria*, frequency dependent disruptive selection on characters unrelated to mate choice can indirectly generate frequency-dependent selection on female preferences. It is clear that models of this type (Felsenstein, 1981; Dieckmann & Doebeli, 1999; CHAPTER 3 of this thesis) exhibit sympatric speciation not so much 'through' sexual selection as merely 'involving' sexual selection, since the speciation process is driven primarily by the disruptive selection acting on the (ecological) characters and not in the first place by disruptive sexual selection.

Also mate choice itself tends to generate non-random genetic associations (i.e., linkage disequilibria), particularly between female preference and male trait alleles. In fact, this is what actually drives the Fisherian runaway process of sexual selection (Fisher, 1930; Lande, 1981; Andersson, 1994). Given that linkage disequilibria with other traits under disruptive selection can generate frequency-dependent selection on female preferences, the above would seem to imply that Fisherian runaway sexual selection could generate the frequency-dependent and disruptive selection needed for adaptive sympatric speciation. Although this possibility exists in theory (see Takimoto, 2002), it does in practice not conflict with our conclusion that, as a general rule, mate choice alone is insufficient to support sympatric speciation. We provide three arguments to support this claim. First, sympatric speciation models based on divergent Fisherian runaway processes (Higashi *et al.* 1999; Takimoto, 2000) and also the simulations presented in this paper (FIGURE 2), illustrate that the indirect frequency-dependent selection on female preference generated through its genetic covariance with the male mating trait is typically only weak and unable to support stable coexistence of the daughter species. Second, although our adaptive dynamics approximation does not take into account the genetic covariances between traits, our analysis provides a valid limit for the case in which genetic covariances are small. Third, an extended analysis, which does take into account genetic covariances, reveals that the establishment of linkage disequilibrium between female preference and male trait will not qualitatively affect the outcome when both male trait and female preference are capable of undergoing evolutionary branching, or, alternatively, when evolutionary branching is precluded for both traits. Consequently, qualitatively different outcomes can only be expected when female choice generates frequency-dependent disruptive selection on males. This, however, requires selection on female preferences to be very weak, such that considerable genetic variation of female preferences can build up through mutation pressure (CHAPTER 3 of this thesis). In other words, unless genetic covariances are large and selection on female preferences is very weak, the effects of indirect selection on female preferences through genetic linkage with male mating traits can be neglected.

Although we have shown that sympatric speciation by sexual selection is feasible, this by no means suggests that it is ubiquitous. On the contrary, our main point here is that sympatric speciation by sexual selection requires far more specific biological conditions than is generally recognized. We argue that essential ingredients of the sympatric speciation process have been overlooked. For several empirical systems it has been investigated in quite some detail how female mate choice may exert frequency-dependent disruptive sexual selection on males (Andersson, 1994), but the analysis of processes capable of generating such selection on female preference has been neglected so far. Moreover, since it is far from trivial, as we have seen, that the frequency-dependent interactions in both sexes are capable of inducing disruptive selection simultaneously, empirical and theoretical attention needs to be devoted to additional sources of disruptive selection that may well be required to explain sympatric speciation. Only after these issues have been clarified will we be able to decide in which biological systems and under what conditions sympatric speciation by sexual selection, rather than another mode of speciation, is indeed the more plausible alternative.

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APPENDIX A — DERIVATION OF MUTANT INVASION FITNESS

We approximate the dynamics of our stochastic individual-based model by deterministic equations using methods derived from adaptive dynamics theory (Metz *et al.*, 1996; Dieckmann & Law, 1996; Geritz *et al.*, 1998). To enable this complementary treatment we make a number of simplifying assumptions. First of all, we assume that population sizes are sufficiently large such that we may neglect stochasticity in the dynamics of the resident population. Second, we consider mutation-limited evolution, so that mutants arise in genetically monomorphic resident populations. Third, we assume single-locus haploid genetics to underlie male trait and female preference (our approach can be extended to more complicated diploid genetics, but we refrain from doing so to keep our analysis tractable). Our aim is to derive the invasion fitness $\lambda(p,q,\hat{p},\hat{q})$ of a rare male trait or female preference mutant with genotypic values (p,q) that has arisen at low frequency in a resident population characterized by the genotypic values $(\hat{p},\hat{q})$. The invasion fitness measures the initial exponential growth rate of the mutant: only when it is larger than zero, i.e., larger than the resident growth rate at equilibrium, the mutant generically can replace the resident (Metz *et al.*, 1996; Geritz *et al.*, 2002). Moreover, the evolutionary rates of change in male trait and female preference can be derived from the invasion fitness (Dieckmann & Law 1996).

As introduced above, we denote *genotypic* trait and preference values p and q, and use hats to distinguish resident from mutant traits. The *phenotypic* trait and preference values, which consist of a genotypic component and added environmental noise, are denoted x and y for preference and trait, respectively. In the following, the variables $\tilde{x}$ and $\tilde{y}$ are always used as auxiliary integration variables. Finally, we use the notation g_i and $\overline{g}_i$ to denote, respectively, Gaussian functions $g_i(x) = \exp\left(-\frac{1}{2}x^2/\sigma_i^2\right)$ and normalized Gaussian functions $\overline{g}_i(x) = g_i(x)\big/\left(\sigma_i\sqrt{2\pi}\right)$.

Analogous to equation [4], the probability $\alpha(x,y,\hat{q})$ that a female with phenotypic preference x chooses a particular male with phenotypic trait y, when she also encounters males from a resident population with genotypic trait value $\hat{q}$, is given by

$$\alpha(x,y,\hat{q}) = \frac{a(x,y)}{\eta + A(x,\hat{q})} \quad , \qquad \text{[A-1]}$$

where, as in equation [2], $a(x,y) = g_m(c(x) - y)$ is the 'attractiveness' of the focal male to the female, and $A(x,\hat{q})$ sums the attractivities of all competing resident males to the female, that is,

$$A(x,\hat{q}) = \sum_{\text{males } \tilde{y}} \mathrm{N}_{\text{male}}(\tilde{y},\hat{q})\, a(x,\tilde{y}) \,. \qquad \text{[A-2]}$$

In a resident population in which all males posses the genotypic trait value $\hat{q}$, the number of males with a phenotypic trait value between $\tilde{y}$ and $\tilde{y} + d\tilde{y}$ is $\mathrm{N}_{\text{male}}(\tilde{y},\hat{q}) = N\, \bar{g}_q(\hat{q} - \tilde{y})\, g_s(\tilde{y})\, d\tilde{y}$. Recall that we assumed the environmental component of the male trait to be distributed according to a normal distribution with width σ_q. This distribution is described by the normalized Gaussian function $\bar{g}_q$. The Gaussian function g_s gives the probability density that a male survives viability selection. Finally, the constant N denotes the population size of the resident, expressed as the number of males or females before viability selection. These considerations allow us to write

$$A(x,\hat{q}) = N \int_{-\infty}^{\infty} \bar{g}_q(\hat{q} - \tilde{y})\, g_s(\tilde{y})\, a(x,\tilde{y})\, d\tilde{y} \,. \qquad \text{[A-3]}$$

To find the per capita mating rate of a mutant male interacting with the female resident population, denoted $M_m(q,\hat{p},\hat{q})$, the expression in equation [A-1] has to be weighted according to the frequencies of the phenotypes x and y, and integrated over all possible phenotypic values y of the mutant male trait and over all possible phenotypic preference values x resulting from the resident female preference with genotypic value $\hat{p}$. The density of values x is described by the normalized Gaussian $\bar{g}_p(\hat{p} - x)$. This yields

$$M_m(q,\hat{p},\hat{q}) = \int_{-\infty}^{\infty}\int_{-\infty}^{\infty} \bar{g}_p(\hat{p} - x) N \bar{g}_q(q - y)\, g_s(y) \frac{a(x,y)}{\eta + A(x,\hat{q})} dx\, dy \,, \qquad \text{[A-4]}$$

which can be written more concisely as

$$M_m(q,\hat{p},\hat{q}) = \int_{-\infty}^{\infty} \bar{g}_p(\hat{p} - x) \frac{A(x,q)}{\eta + A(x,\hat{q})} dx \,. \qquad \text{[A-5]}$$

Similarly, we may compute the per capita mating rate of a mutant female with preference p interacting with the resident male population as

$$M_f(p,\hat{q}) = \int_{-\infty}^{\infty} \bar{g}_p(p - x) \frac{A(x,\hat{q})}{\eta + A(x,\hat{q})} dx \,. \qquad \text{[A-6]}$$

Note that $M_f(p,\hat{q})$ does not depend on the resident female preference $\hat{p}$, and that $M_m(\hat{q},\hat{p},\hat{q}) = M_f(\hat{p},\hat{q})$.

We may now establish a recurrence equation for the number of mutants $n_t(p,q,\hat{p},\hat{q})$ when rare. Under our assumption of constant population size N, the number of mutants n_t changes according to

$$n_{t+1}(p,q,\hat{p},\hat{q}) = n_t(p,q,\hat{p},\hat{q})\,\frac{1}{2}\left(M_m(q,\hat{p},\hat{q}) + M_f(p,\hat{q})\right) S(\hat{p},\hat{q})\,. \qquad \text{[A-7]}$$

The factor 1/2 reflects that only half of the offspring of a mutant parent will inherit the mutant strategy. The function $S(\hat{p},\hat{q})$ captures the effects of density dependence. It absorbs the per capita survival probabilities, as well as the number of matings per female per generation.

When the mutant is identical to the resident ($p=\hat{p}, q=\hat{q}$) and the resident population is at equilibrium, the mutant's density must neither decline nor increase, which means that

$$\frac{1}{2}S(\hat{p},\hat{q}) = \frac{1}{M_m(\hat{q},\hat{p},\hat{q}) + M_f(\hat{p},\hat{q})} = \frac{1}{2\,M_f(\hat{p},\hat{q})}\,. \qquad \text{[A-8]}$$

Equations [A-7] and [A-8] show that the geometric rate of increase of the number of mutants equals the ratio of mutant mating rates over resident mating rates. Since the invasion fitness is defined as the natural logarithm of the geometric rate of increase, we find that

$$\lambda(p,q,\hat{p},\hat{q}) = \ln\left(\frac{M_m(q,\hat{p},\hat{q}) + M_f(p,\hat{q})}{2\,M_f(\hat{p},\hat{q})}\right). \qquad \text{[A-9]}$$

APPENDIX B — NUMERICAL PROCEDURES

In generic cases, the fact that $\lambda(p,q,\hat{p},\hat{q}) > 0$ implies that $\lambda(\hat{p},\hat{q},p,q) < 0$ and that the mutant will go to fixation (Geritz *et al.*, 2002). This means that mutants with positive invasion fitness can grow in the resident population until the resident is completely replaced. A series of such trait substitution events results in directed evolution, the direction and expected rate of which is dependent on the sign and magnitude of the local fitness gradients (Dieckmann & Law, 1996). Specifically, it can be shown that

$$\begin{aligned} \frac{d\hat{p}}{dt} &= \kappa \left.\frac{\partial \lambda(p,q,\hat{p},\hat{q})}{\partial p}\right|_{\substack{p=\hat{p}\\ q=\hat{q}}} = \frac{\kappa}{2\,M_f(\hat{p},\hat{q})} \left.\frac{\partial M_f(p,\hat{q})}{\partial p}\right|_{p=\hat{p}}\,, \\ \frac{d\hat{q}}{dt} &= \kappa \left.\frac{\partial \lambda(p,q,\hat{p},\hat{q})}{\partial q}\right|_{\substack{p=\hat{p}\\ q=\hat{q}}} = \frac{\kappa}{2\,M_f(\hat{p},\hat{q})} \left.\frac{\partial M_m(q,\hat{p},\hat{q})}{\partial q}\right|_{q=\hat{q}}\,. \end{aligned} \qquad \text{[B-1]}$$

Similar equations can be derived within a quantitative genetics framework (Lande, 1976). The constant $\kappa = \frac{1}{2}N\sigma_v^2\mu$ captures the effects of population size, mutation ratio, μ, and mutational variance, σ_v^2, on the rate of evolution. An assumption underlying equations [B-1] is that mutations do not have pleiotropic effects and that mutation ratio and variance are equal for female preference and male trait. Generalizations can be readily considered; in particular, unequal mutation rates and/or mutational variances can be dealt with by rescaling the female-choice function.

Equations [B-1] were solved numerically using a standard algorithm for the numerical integration of ordinary differential equations (the Cash-Karp Runge-Kutta algorithm with adaptive step size control, Press *et al.*, 1992). The resident trajectories were calculated until a monomorphic evolutionary equilibrium was attained. We then determined whether further polymorphic evolution would occur by checking the local evolutionary stability of the monomorphic equilibrium. If so, numerical integration was then continued with an extended system of equations, with initial conditions slightly displaced around the monomorphic equilibrium. For a population that is dimorphic in both trait and preference, the evolutionary dynamics can be described by a system of four equations

$$\begin{aligned}
\frac{d\hat{p}_1}{dt} &= \frac{\kappa\psi}{2\,M_{\mathrm{pol},f}(\hat{p}_1,\hat{q}_1,\hat{q}_2)} \left.\frac{\partial M_{\mathrm{pol},f}(p,\hat{q}_1,\hat{q}_2)}{\partial p}\right|_{p=\hat{p}_1}, \\
\frac{d\hat{p}_2}{dt} &= \frac{\kappa\,(1-\psi)}{2\,M_{\mathrm{pol},f}(\hat{p}_1,\hat{q}_1,\hat{q}_2)} \left.\frac{\partial M_{\mathrm{pol},f}(p,\hat{q}_1,\hat{q}_2)}{\partial p}\right|_{p=\hat{p}_2}, \\
\frac{d\hat{q}_1}{dt} &= \frac{\kappa\phi}{2\,M_{\mathrm{pol},f}(\hat{p}_1,\hat{q}_1,\hat{q}_2)} \left.\frac{\partial M_{\mathrm{pol},m}(q,\hat{p}_1,\hat{p}_2,\hat{q}_1,\hat{q}_2)}{\partial q}\right|_{q=\hat{q}_1}, \\
\frac{d\hat{q}_2}{dt} &= \frac{\kappa\,(1-\phi)}{2\,M_{\mathrm{pol},f}(\hat{p}_1,\hat{q}_1,\hat{q}_2)} \left.\frac{\partial M_{\mathrm{pol},m}(q,\hat{p}_1,\hat{p}_2,\hat{q}_1,\hat{q}_2)}{\partial q}\right|_{q=\hat{q}_2}.
\end{aligned} \qquad \text{[B-2]}$$

The constants ψ and φ determine the relative abundance of, respectively, the two resident female preferences and male traits. The mating rates in a polymorphic population are straightforward generalizations of the mating rates in a monomorphic population. For example,

$$\begin{aligned}
&M_{\mathrm{pol},m}(q,\hat{p}_1,\hat{p}_2,\hat{q}_1,\hat{q}_2) = \\
&\int_{-\infty}^{\infty} \left[\psi\,\bar{g}_p(\hat{p}_1-x)+(1-\psi)\,\bar{g}_p(\hat{p}_2-x)\right] \frac{A(x,q)}{\eta+\varphi\,A(x,\hat{q}_1)+(1-\varphi)\,A(x,\hat{q}_2)}\,dx\,.
\end{aligned} \qquad \text{[B-3]}$$

The stable coexistence of two resident female-preference types requires that both types have equal fitness. The same applies for the coexistence of two resident male-trait types. In other words, in a polymorphic population,

$$\begin{aligned}
M_{\mathrm{pol},f}(\hat{p}_1,\hat{q}_1,\hat{q}_2) &= M_{\mathrm{pol},f}(\hat{p}_2,\hat{q}_1,\hat{q}_2), \\
M_{\mathrm{pol},m}(\hat{q}_1,\hat{p}_1,\hat{p}_2,\hat{q}_1,\hat{q}_2) &= M_{\mathrm{pol},m}(\hat{q}_2,\hat{p}_1,\hat{p}_2,\hat{q}_1,\hat{q}_2).
\end{aligned} \qquad \text{[B-4]}$$

These two equations define the values of the constants ψ and φ for any given set of resident preference and trait values.

We used an efficient way (based on fast Fourier transforms, details available upon request) to calculate the mating rate gradients. However, our algorithm required us to specify the inverse of the female choice function $c^{\mathrm{inv}}(q)$, rather than the female choice function $c(p)$ itself. In all simulations where the female choice function was nonlinear, we used

$$c^{\mathrm{inv}}(q)=\frac{q+q^3}{3} \qquad \text{[B-5]}$$

The graph of this function can clearly be recognized in FIGURE 2. This simple function is convex to the right of $q=0$, and concave to the left. Therefore it allows for divergent evolution: sexual selection will drive the population towards higher values of q when $q>0$, and to lower values when $q<0$. At $q=0$, sexual selection is disruptive.

APPENDIX C — CONSEQUENCES OF FREQUENCY-INDEPENDENT SELECTION ON FEMALE PREFERENCE

Here we show in general that a polymorphism of female preference can never arise without frequency-dependent selection on female preference. As will become apparent, these general arguments are valid for a whole class of models sharing the property that the mating rate of females is independent of the resident female strategy. Our arguments can also be worked out on a more abstract and even more general level by considering the dimensionality of the environmental feedback (Meszéna & Metz, in press).

From equations [B-1], it can be seen that the endpoints of monomorphic evolution are strategy pairs $(\hat{p},\hat{q})=(p^*,q^*)$ at which the fitness gradients with respect to both trait and preference are zero. So, at those points

$$\left.\frac{\partial\lambda(p,q,\hat{p},\hat{q})}{\partial p}\right|_{\substack{p=\hat{p}=p^*\\ q=\hat{q}=q^*}}=0 \quad \text{and} \quad \left.\frac{\partial\lambda(p,q,\hat{p},\hat{q})}{\partial q}\right|_{\substack{p=\hat{p}=p^*\\ q=\hat{q}=q^*}}=0. \qquad \text{[C-1]}$$

In view of equation [A-9], this is equivalent with

$$\left.\frac{\partial M_f(p,q^*)}{\partial p}\right|_{p=p^*}=0 \quad \text{and} \quad \left.\frac{\partial M_m(q,p^*,q^*)}{\partial q}\right|_{q=q^*}=0\,. \qquad \text{[C-2]}$$

These conditions ensure that (p^*,q^*) is a pair of so-called evolutionarily singular strategies (Metz *et al.*, 1996), at which the rates of monomorphic evolution vanish. However, not all singular strategy pairs are relevant as endpoints of monomorphic evolution, since not all singular strategy pairs are attainable by gradual evolutionary change. Attainability of a singular strategy pair must be assessed by investigating its convergence stability. To ensure that evolution converges to the singular strategy pair irrespective of the mutational variance-covariance matrix, the singular strategy pair must satisfy the conditions for strong convergence stability (Leimar, 2001). Necessary (but not sufficient) conditions for strong convergence stability are

$$\left.\frac{\partial}{\partial\hat{p}}\left(\left.\frac{\partial\lambda(p,q,\hat{p},\hat{q})}{\partial p}\right|_{\substack{p=\hat{p}\\ q=\hat{q}}}\right)\right|_{\substack{\hat{p}=p^*\\ \hat{q}=q^*}}<0 \quad \text{and} \quad \left.\frac{\partial}{\partial\hat{q}}\left(\left.\frac{\partial\lambda(p,q,\hat{p},\hat{q})}{\partial q}\right|_{\substack{p=\hat{p}\\ q=\hat{q}}}\right)\right|_{\substack{\hat{p}=p^*\\ \hat{q}=q^*}}<0, \qquad \text{[C-3]}$$

which, in our case, following from equation [A-9], translate into

$$\left.\frac{\partial^2 M_f\left(p,q^*\right)}{\partial p^2}\right|_{p=p^*}<0 \quad \text{and} \quad \left.\frac{\partial^2 M_m\left(q,p^*,q^*\right)}{\partial q^2}\right|_{q=q^*}+\left.\frac{\partial^2 M_m\left(q,p^*,q^*\right)}{\partial q\,\partial q^*}\right|_{q=q^*}<0\,. \qquad \text{[C-4]}$$

The fact that monomorphic evolution comes to a halt at convergence stable singular strategy pairs does not imply that further evolution from such singular strategies is impossible. In fact, convergence stable singular strategy pairs need not be evolutionarily stable, that is, they need not be resistant against invasion by alternative strategies. In particular, the resident population at the singular strategy can be invaded by female preference or male trait mutants, when, respectively,

$$\left.\frac{\partial^2 \lambda\left(p,q,p^*,q^*\right)}{\partial p^2}\right|_{\substack{p=p^*\\ q=q^*}}>0 \quad \text{or} \quad \left.\frac{\partial^2 \lambda\left(p,q,p^*,q^*\right)}{\partial q^2}\right|_{\substack{p=p^*\\ q=q^*}}>0\,, \qquad \text{[C-5]}$$

or, equivalently, when

$$\left.\frac{\partial^2 M_f\left(p,q^*\right)}{\partial p^2}\right|_{p=p^*}>0 \quad \text{or} \quad \left.\frac{\partial^2 M_m\left(q,p^*,q^*\right)}{\partial q^2}\right|_{q=q^*}>0\ . \qquad \text{[C-6]}$$

When this situation occurs, the population will undergo evolutionary branching. It can only escape from the evolutionarily *un*stable singular strategy by becoming dimorphic, since, because of convergence stability, a monomorphic population is rapidly driven back to the singular strategy.

It is obvious that the left halves of conditions [C-4] and [C-6] are mutually exclusive, implying that there can be no female-preference branching in this model. By contrast, male-trait branching is possible, and occurs when

$$-\left.\frac{\partial^2 M_m\left(q,p^*,q^*\right)}{\partial q\,\partial q^*}\right|_{q=q^*}>\left.\frac{\partial^2 M_m\left(q,p^*,q^*\right)}{\partial q^2}\right|_{q=q^*}>0\,. \qquad \text{[C-7]}$$

Note that the contradiction between conditions [C-4] and [C-6] with regard to female-preference branching is unavoidable and simply results from the fact that M_f is independent of the resident female strategy.

APPENDIX D — EXTENSION OF THE MODEL WITH FEMALE-FEMALE COMPETITION

Competition between females can easily be incorporated in the equations by multiplying all mating rates with a function Φ, measuring the number of offspring produced per mating and decreasing with increasing mating rates of the male. Using the shorthand notation

$$\xi\left(y,p,q\right)=\int_{-\infty}^{\infty}\frac{N\,\bar{g}_p\left(p-x\right)a\left(x,y\right)}{\eta+A\left(x,q\right)}dx \qquad \text{[D-1]}$$

for the mating rate of a male with trait y, the mutant male mating rate transforms into

$$\tilde{M}_m(q,\hat{p},\hat{q})=\int_{-\infty}^{\infty}\bar{g}_q(q-y)\,g_s(y)\xi(y,\hat{p},\hat{q})\Phi(\xi(y,\hat{p},\hat{q}))dy \qquad \text{[D-2]}$$

and the mutant female mating rate becomes

$$\tilde{M}_f(p,\hat{p},\hat{q})=\int_{-\infty}^{\infty}\bar{g}_q(\hat{q}-y)\,g_s(y)\xi(y,p,\hat{q})\Phi(\xi(y,\hat{p},\hat{q}))dy\,. \qquad \text{[D-3]}$$

This expression now also depends on the resident female preference, the importance of which was shown in APPENDIX C. Under our assumptions, the function Φ decreases with ξ, that is, a female will produce less and less offspring as her partner has mated more often. It seems biologically reasonable, however, to assume that males will still benefit from additional matings. Mathematically, this implies that

$$\text{for all } \xi: \quad \frac{d\Phi(\xi)}{d\xi}<0 \quad \text{and} \quad \frac{d(\xi\ \Phi(\xi))}{d\xi}>0 \qquad \text{[D-4]}$$

Using only these two properties of Φ, the semi-formal argumentation given below demonstrates that the conditions for male and female mating-type branching are mutually exclusive. A more rigorous proof, involving expansions of ξ and $\Phi(\xi)$ in terms of Hermite polynomial series, can be given, but this proof involves lengthy and complicated calculations and is therefore omitted here.

Let us suppose first that male mating-type branching is possible. This requires that, at the singular strategy (p^*,q^*),

$$\left.\frac{\partial^2\tilde{M}_m(q,\hat{p},\hat{q})}{\partial q^2}\right|_{\substack{\hat{p}=p^*\\ q=\hat{q}=q^*}}=\int_{-\infty}^{\infty}\bar{g}''_q(q^*-y)g_s(y)\xi(y,p^*,q^*)\Phi(\xi(y,p^*,q^*))dy>0\,. \qquad \text{[D-5]}$$

By repeatedly applying the product rule for integration, one can easily see that the integral in equation [D-5] represents the smoothed second derivative with respect to y and evaluated at $y=q^*$ of the product $g_s(y)\xi(y,p^*,q^*)\Phi(\xi(y,p^*,q^*))$. Consequently, the inequality [D-5] can only be fulfilled when $\xi(y,p^*,q^*)\Phi(\xi(y,p^*,q^*))$ has a minimum with respect to y close to $y=q^*$. Because of conditions [D-4], this implies that $\Phi(\xi(y,p^*,q^*))$ must have a maximum close to $y=q^*$. This in turn means that

$$\begin{aligned}\left.\frac{\partial^2\tilde{M}_f(p,\hat{p},\hat{q})}{\partial p^2}\right|_{\substack{p=\hat{p}=p^*\\ \hat{q}=q^*}}&=\int_{-\infty}^{\infty}\bar{g}_q(q^*-y)g_s(y)\left.\frac{\partial^2\xi(y,p,q^*)}{\partial p^2}\right|_{p=p^*}\Phi(\xi(y,p^*,q^*))dy\\ &<\int_{-\infty}^{\infty}\bar{g}_q(q^*-y)g_s(y)\left.\frac{\partial^2\xi(y,p,q^*)}{\partial p^2}\right|_{p=p^*}dy=\left.\frac{\partial^2 M_f(p,\hat{q})}{\partial p^2}\right|_{\substack{p=p^*\\ \hat{q}=q^*}}\,.\end{aligned} \qquad \text{[D-6]}$$

In most models of female choice without female-female competition, the only selective force on female preference is the cost of choosiness. Usually this source of stabilizing selection will drive female preference to a *stable* evolutionary equilibrium value at which the costs of choice are minimized. Because of condition [C-4], convergence stability of equilibria in the preference direction implies that

$$\left.\frac{\partial^2 M_f(p,\hat{q})}{\partial p^2}\right|_{\substack{p=p^* \\ \hat{q}=q^*}} < 0 \tag{D-7}$$

and therefore

$$\left.\frac{\partial^2 \tilde{M}_m(q,\hat{p},\hat{q})}{\partial q^2}\right|_{\substack{\hat{p}=p^* \\ q=\hat{q}=q^*}} > 0 \;\Rightarrow\; \left.\frac{\partial^2 \tilde{M}_f(p,\hat{p},\hat{q})}{\partial p^2}\right|_{\substack{p=\hat{p}=p^* \\ \hat{q}=q^*}} < 0\,. \tag{D-8}$$

A similar reasoning shows that the reverse inference is also true. This demonstrates that there can be no simultaneous male and female mating-type branching, and, hence, no sympatric speciation in our model involving only female choice and female-female competition.

APPENDIX E — EXTENSION OF THE MODEL WITH MALE-MALE COMPETITION

Competition between males (e.g., for mating territories) can be dealt with by replacing in equations [D-1]-[D-3] the distribution of males after viability selection by the distribution $z(y,q,\hat{q})$, which denotes the distribution of males after both viability selection and male-male competition.

The distribution $z(y,q,\hat{q})$ was determined as follows. Following equation [6], the competition experienced by a male trying to establish a territory is

$$\bar{f}(y,\hat{q}) = \int_{-\infty}^{\infty} z(\tilde{y},\hat{q},\hat{q})\, g_c(y-\tilde{y})\, d\tilde{y}\,. \tag{E-1}$$

If $\bar{f}(y,\hat{q})$ falls below the threshold θ, the density of males of type y that occupy a territory grows, otherwise it decreases, such that an equilibrium is reached when

$$z(y,q,\hat{q}) = H\big(\theta - \bar{f}(y,\hat{q})\big)\,\bar{g}_q(q-y)\,g_s(y)\,. \tag{E-2}$$

The function H is the Heaviside step function ($H(x)=0$ for $x<0$; $H(x)=1$ for $x>0$, and $H(x)$ is undefined at $x=0$), which we approximated for numerical reasons by the smoothed threshold function Θ, where

$$\Theta(y) = \frac{1}{2}\left(1+\tanh\left(\frac{y}{\sigma_w}\right)\right). \tag{E-3}$$

The smoothing parameter σ_w was chosen as $\sigma_w = 0.01$.

In our adaptive dynamics simulations, we solved equations [E-1] and [E-2] efficiently using Fast Fourier Transforms for the resident male distribution $z(y,\hat{q},\hat{q})$. The mutant male distribution $z(y,q,\hat{q})$ was then determined by using that

$$z(y,q,\hat{q}) = z(y,\hat{q},\hat{q})\frac{\overline{g}_q(q-y)}{\overline{g}_q(\hat{q}-y)}. \qquad \text{[E-4]}$$

Equation [E-4] follows from the fact that $z(y,\hat{q},\hat{q})$ is a solution of [E-2] when $q = \hat{q}$.

Afterthoughts on chapter 4

The evolution of male competitive strategies

This section summarizes results presented in a MSc-thesis by Han Verkiel (2002), who studied an alternative version of the model of CHAPTER 4. His analysis, which explicitly deals with the evolution of male competitive strategies, corroborates the previous assumptions on the behavior of males in male-male competition by demonstrating that there is indeed selection for increased intensity of competition between males that compete for the same females.

As before, we assume that males compete for mating territories. For simplicity, the number of mating territories, denoted M, is kept fixed. Whether or not a male will secure a mating territory is determined by his success in pair-wise aggressive interactions with other males. Males may influence their success by strategically varying their effort in these aggressive interactions, in relation to their own mating trait and that of their opponent.

Specifically, we think of a situation where males strategically choose the amount of time (alternatively, energy or resources) invested in aggressive interactions with a certain opponent. As long as two individuals, say, i and j, are willing to spend more time on their mutual aggressive interaction, they engage in an escalated fight. As soon as one of the individuals gives up, it is chased by its opponent for as long as the latter is willing to spend time in pursuit. Escalated fights can easily result in physical damage, and being chased induces considerably more stress than chasing an opponent.

To model this situation, we use τ_{ij} to denote the time spent by male i on aggressive behavior (e.g., biting and chasing) against male j, and define the cost C_{ij} to individual i of its aggressive interactions with individual j as

$$C_{ij} = c_{\text{fight}}\,\tau_{ij}\,\tau_{ji} + c_{\text{chase}}\,\tau_{ij}\left(1-\tau_{ji}\right) + c_{\text{flee}}\left(1-\tau_{ij}\right)\tau_{ji}\;, \qquad [1]$$

where c_{fight}, c_{flee} and c_{chase} represent the costs of fighting, being chased and chasing, respectively. We assume that $c_{\text{fight}} > c_{\text{flee}} > c_{\text{chase}}$, such that behaving more aggressively to an opponent (i.e., increasing τ_{ij}) is costly to oneself, but even more costly to the opponent. To ensure that all males have equal intrinsic competitive ability, we impose that

$$\sum_{\substack{\text{males } j \\ j \neq i}} \tau_{ij} = 1 , \qquad [2]$$

for all males.

A male's competitive success is determined by the overall intensity of competition, C_i, that he has experienced, where

$$C_i = \sum_{\substack{\text{males } j \\ j \neq i}} C_{ij} . \qquad [3]$$

Males that suffered less from competition have a higher probability P_i of obtaining a mating territory. We assumed a linear relation between P_i and C_i, with $P_i = 0$ for the male with the highest C_i and $P_i = 1$ for the male with the lowest C_i.

To allow evolution of the male competitive strategy, we assume that the τ_{ij} are influenced by an additional quantitative heritable character, denoted x, which determines the extent to which males behave more aggressively towards competitors with a similar appearance. To be precise, we assume that

$$\tau_{ij} = a_i \exp\left(-\frac{1}{2} x_i \left(\frac{q_i - q_j}{\sigma_c} \right)^2 \right) , \qquad [4]$$

where the proportionality constant a_i is chosen such that condition [2] is satisfied. The character x is coded by four additive diploid loci and subject to mutation exactly as previously defined for the mating traits. Males with positive x_i spend more time on aggressive interactions with males that express a male mating character similar to their own. By contrast, males with negative x_i spend more time on aggressive interactions with dissimilar males. Males with $x_i = 0$, finally, do not make their behavior in male-male competition dependent on male mating trait differences.

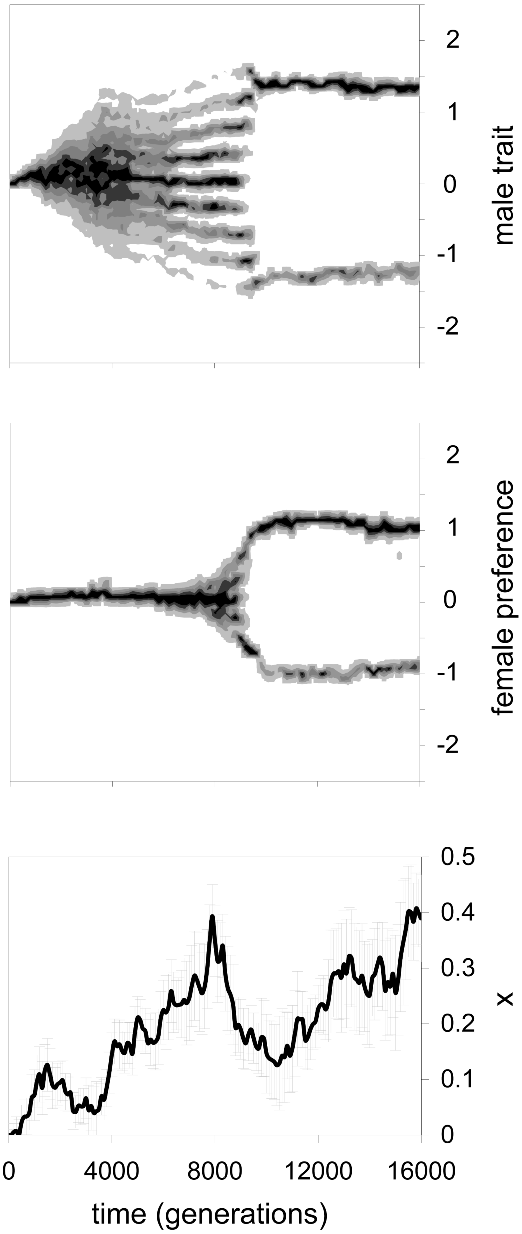

FIGURE 1 – SIMULATION RESULTS

Speciation occurs in the model with evolving male competitive strategies (upper two panels) for the same parameters as in FIGURE 5 of CHAPTER 4. Speciation is accompanied by the evolution of discriminatory aggressive behavior among males (lower panel): males preferentially direct their aggression to phenotypically similar competitors. Error bars represent the population standard deviation of x. This simulation is for $M = 300$, $c_{\text{fight}} = 1.0$, $c_{\text{flee}} = 0.8$, $c_{\text{chase}} = 0.2$

After male-male competition has occurred, females choose a mate from the males that secured a mating territory. Female mate choice and the production of offspring were modeled exactly as described previously.

Individual-based simulations of this extended model (FIGURE 1) show speciation under the same parameter conditions as the original model in CHAPTER 4. Additional disruptive selection on the male mating trait is again generated by male-male competition. This time, however, we did not presuppose that males preferentially direct their aggression towards phenotypically similar competitors. Instead, the model allows such a strategy to evolve. In fact, the simulations illustrate, that it is selectively advantageous to evolve discriminatory behavior in male-male competition. This finding is in line with our earlier verbal argument that it makes sense to behave most aggressively towards males with whom one competes for the same females. Of course, this presupposes that males compete predominantly for female attention, and not for other limiting resources that are not subject to female choice.

A general preliminary conclusion that could be drawn from these results is that traits important in female mate choice are also likely to become involved in male-male competition. We speculate that the converse could also be true: one could imagine traits that are used as signals in male-male competition, to become involved in female mate choice as the result of sexual selection for good-genes (in this case, indicators of male competitive ability). Indeed, there are many examples of species where secondary sexual signals are used in the context of male-male competition as well as in the context of female mate choice (e.g., red coloration in sticklebacks, spurs of pheasants; see Andersson, 1994 for more examples). Yet, theory has traditionally considered intra- and inter-sexual selection as largely separate processes. Certainly, it will be worthwhile to further investigate the mutual interactions between intra- and inter-sexual selection.

Sympatric Speciation through Inter- and Intra-Sexual Selection: A Unified Analysis

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G. Sander van Doorn & Ulf Dieckmann

unpublished manuscript

ABSTRACT

Several empirical studies have suggested sexual selection to be a potent driving force of sympatric speciation. In this vein, recent theoretical models have demonstrated that sexual selection can result in assortative mating of sufficient strength to oppose recombination, giving rise to reproductive isolation. However, with their emphasis on genetic constraints on speciation, sexual selection models have as yet ignored the restrictions associated with the origin of stable polymorphisms under disruptive selection. Also, sexual selection models often do not offer an evolutionary explanation for the origin of variation in mating characters, and for this reason often rely on non-adaptive processes to initiate speciation. These two limitations explain why many models of speciation by sexual selection cannot account for the stable coexistence of daughter species, and why their dynamics often critically depends on peculiar initial conditions. In this paper, we systematically investigate the consequences of selective restrictions on speciation by sexual selection. We derive general conditions under which speciation can proceed as an adaptive process driven by a stable regime of disruptive sexual selection. The frequency-dependent selection pressures needed for such adaptive speciation can be generated either by intra-sexual interactions, such as male-male and female-female competition, or by antagonistic inter-sexual interactions. We show that in both cases adaptive speciation requires conditions far more restrictive than earlier models of sympatric speciation by sexual selection appear to suggest. Our analytical treatment allows us to generalize this conclusion to a wide range of mating dynamics and types of sexual selection.

INTRODUCTION

Sexual selection has been put forward as a potent driving force of speciation. Evidence supporting this view comes from comparative studies (reviewed in Panhuis *et al.*, 2001) that highlight higher species richness in clades with more intense sexual selection. Moreover, closely related species often differ most pronouncedly in their secondary sexual characters (Eberhard, 1985; Vacquier, 1998), an observation that has been used to argue that sexual differentiation contributes earlier and more strongly to speciation than ecological diversification (Seehausen *et al.*, 1999; Wilson *et al.*, 2000). Also intuitively, an involvement of sexual selection in speciation is plausible, since sexual selection is capable of driving rapid evolutionary changes in traits responsible for prezygotic reproductive isolation (Andersson, 1994).

Given its predisposition to generate reproductive isolation, sexual selection has not only been suggested to facilitate allopatric speciation through divergent evolution of sexual characters in isolated populations (Lande, 1981; Gavrilets, 2000), but has also been proposed as a mechanism responsible for sympatric speciation (Howard & Berlocher, 1998; Via, 2001). Formal models (reviewed by Turelli *et al.*, 2001; Kirkpatrick & Ravigné, 2002) have demonstrated that sexual selection is indeed capable of driving the rapid evolution of reproductive isolation through female choice (Wu, 1985; Turner & Burrows, 1995; Payne & Krakauer, 1997; Van Doorn *et al.*, 1998; Higashi *et al.*, 1999; Takimoto *et al.*, 2000; Takimoto, 2002), sexual conflict (Gavrilets & Waxman, 2002), mutual mate choice (Almeida & Vistulo de Abreu, 2003), intra-sexual selection (CHAPTER 4 of this thesis), or sex-ratio selection (Lande *et al.*, 2001). By illustrating that sexual selection can generate strong assortative mating, these models successfully address one of the longstanding theoretical difficulties associated with sympatric speciation: recombination, which acts as a major force prohibiting speciation (Udovic, 1980; Felsenstein, 1981; Rice, 1984), can be overcome by assortative mating. Recombination otherwise hinders speciation by creating intermediary and less fit hybrid phenotypes, by eroding linkage disequilibria within and between genes responsible for diversification and reproductive isolation, and by breaking up co-adapted gene complexes.

SELECTIVE RESTRICTIONS ON SPECIATION

Sexual selection models of speciation provide a mechanistic explanation for the evolution of assortative mating itself. This issue is usually not considered in other (ecological) models of speciation, where the presence of a mechanism for assortative mating is often taken for granted. However, with their natural emphasis on reproductive isolation and the genetic restrictions imposed by recombination, sexual selection models have largely ignored selective restrictions on sympatric speciation, which, by contrast, have been investigated extensively in the context of ecological models of speciation (e.g., Dieckmann & Doebeli, 1999; Doebeli & Dieckmann, 2000).

Surprisingly, many sexual selection models do not offer an adaptive explanation for the origin and maintenance of variation in sexual characters. Instead, the models simply assume sufficient variation to be present initially (Payne & Krakauer, 1997; Higashi *et al.*, 1999; Takimoto *et al.*, 2000) or rely on non-adaptive mechanisms, such as genetic drift (Wu, 1985), mutation-selection balance (Van Doorn *et al.*, 1998; CHAPTER 3 of this thesis; Gavrilets & Waxman, 2002; Takimoto, 2002), or mutations with large phenotypic effects (Turner & Burrows, 1995) to initiate speciation. In addition, and perhaps even more importantly, many sexual selection models do not account for the stable coexistence of daughter species after speciation has occurred (Wu, 1985; Turner & Burrows, 1995; Payne & Krakauer, 1997; Higashi *et al.*, 1999; Takimoto *et al.*, 2000).

The fact that selective restrictions on sympatric speciation are hardly appreciated in models of sympatric speciation by sexual selection is a bit unexpected, since already classical papers identified the origin and maintenance of a polymorphism as an important first step towards sympatric speciation (Maynard Smith, 1966; Udovic, 1980). In fact, Maynard Smith (1966) originally thought the evolution of reproductive isolation to be a trivial affair, while considering the establishment of a stable polymorphism as the crucial step in sympatric speciation. When the origin of stable polymorphism is not supported by selection, it becomes necessary to concede a major role to non-adaptive processes as driving forces of speciation. This certainly conflicts with the interpretation of sympatric speciation as an adaptive process and has implications for several of the arguments commonly put forward in favor of sympatric speciation and against allopatric speciation. For example, the suggestion that allopatric speciation, unlike sympatric speciation, is too slow to be able to account for the presently observed biodiversity, loses much of its strength when non-adaptive, external processes were required to drive sympatric speciation: a higher rate of sympatric speciation can only be expected if sympatric speciation is an adaptive process, internally driven by selection (Bush, 1975).

EVOLUTION OF POLYMORPHISM UNDER DISRUPTIVE SELECTION

Selective restrictions on speciation arise from the fact that disruptive selection per se is not sufficient to generate and maintain stable polymorphisms. This can already be observed in the standard textbook example of a one-locus two-allele population genetic model with heterozygote inferiority and fixed fitness values associated with each of the genotypes. In such a setting, polymorphisms are always transient, and the population will become fixed for either one of the alleles, depending on the fitness values of the homozygous types and the initial allele frequencies.

In terms of a fitness landscape metaphor, points at which selection is disruptive correspond to fitness minima, that is, to valleys of the fitness landscape. A population located exactly at a fitness minimum will experience disruptive selection, but as soon as the population is only slightly shifted away from the minimum, it will instead experience directional selection, allowing it to easily escape from the fitness minimum without splitting. Exactly this problem explains why the model of Higashi *et al.* (1999) yields speciation only when the population is initially perched exactly on the point where selection is disruptive (Turelli *et al.*, 2001).

Already Maynard Smith (1966) and Udovic (1980) realized that, in order to overcome the destabilizing effects of disruptive selection, selection must be frequency-dependent, such that fitness is not only determined by an individual's own traits but also by the traits of other individuals in its population. In particular, a stable regime of disruptive selection requires that rare genotypes have a fitness advantage (Udovic, 1980). If selection is frequency-dependent, the strength and direction of selection (and, hence, the topology of the fitness landscape) is bound to vary in the course of evolution. This makes it possible that a population adaptively converges towards a fitness minimum (Abrams *et al.*, 1993), a rather counter-intuitive process. When this occurs, the population cannot escape from the fitness minimum through gradual evolution, since it will always be driven back by frequency-dependent selection. The population will therefore experience a stable and persistent regime of disruptive selection. In asexual populations, such a regime is expected to induce polymorphism, giving rise to a process that has been termed 'evolutionary branching' (Metz *et al.*, 1996; Geritz *et al.*, 1998).

In sexual populations, evolutionary branching is opposed by recombination. However, as soon as assortative mating is allowed to evolve, evolutionary branching can also occur in sexual populations: frequency-dependent disruptive selection favors increased levels of assortativeness. Dieckmann & Doebeli (1999) demonstrated this in the context of ecological resource competition, and later illustrated this effect for all other fundamental types of ecological interaction (Doebeli & Dieckmann, 2000).

FREQUENCY-DEPENDENT DISRUPTIVE SEXUAL SELECTION

Given the historical emphasis on ecological mechanisms as sources of disruptive selection during sympatric speciation (Maynard Smith, 1966; Udovic, 1980; Felsenstein, 1981), it is understandable that the selective restrictions on speciation have been extensively discussed in the context of ecological models of speciation, but were largely ignored in models of speciation by sexual selection. However, this historical bias has no biological basis. Frequency-dependent disruptive selection can be generated by ecological processes as well as by sexual selection. We can thus see no reason for judging by different standards models of speciation that are based on sexual versus ecological interactions, particularly not when issues like the mechanisms responsible for the origin of variation, the stable coexistence of the daughter species after speciation, and the sensitive dependence on initial conditions are concerned.

In this paper, we critically investigate consequences of selective restrictions for models of sympatric speciation by sexual selection. Our aim is to pry out the general biological conditions under which sexual selection can drive adaptive sympatric speciation (Dieckmann *et al.*, 2004). Processes of adaptive speciation are selection-driven from beginning to end (i.e., from the initial phase in which variation originates, up to the final phase in which two daughter-species stably coexist), without any crucial dependencies on non-adaptive processes. We therefore need to investigate which types of inter- and intra-sexual interactions can generate stable polymorphisms of sexual traits through evolutionary branching, i.e., driven by frequency-dependent disruptive sexual selection.

Comprehensive analysis

Rather than presenting results for a single model of speciation by sexual selection, we follow a more encompassing strategy by developing a general modeling framework that allows us to evaluate the potential for adaptive sympatric speciation by sexual selection for whole classes of models.

This comprehensive treatment is based on recognizing that models of sympatric speciation by sexual selection have to deal with processes on three interlocking timescales. The process that occurs on the fastest timescale is the mating process. It comprises interactions between males and females such as competition for access to mating partners, mate assessment and rejection, and pair formation. The mating process eventually results in the production of offspring. It is therefore intimately connected with the population dynamics. Still, population dynamical processes, such as birth and death, typically occur on a slower timescale than the elements of the mating process. This is especially true for iteroparous species, in which individuals pass through the mating process several times during their lifetime. The slowest timescale in speciation models characterizes the process of evolutionary change through mutation and selection.

A feedback loop exists between processes on all three timescales. On the one hand, evolution determines the strategies of individuals in the mating process. On the other hand, the evolutionary fate of a new mutant is determined by its population dynamical growth rate in competition with an existing resident type, which ultimately depends on the mutant's performance in the mating process.

Structure of this paper

In the first part of this paper, we devise a general description of mating interactions, eventually to arrive at a quantitative description of the trait substitution sequences underlying evolution. Along the way, we explain the simplifying assumptions needed to dissect the complex feedback between processes at the three different timescales. We then consider a number of increasingly complex examples, in order to illustrate the general structure of feedback between mating process, population dynamics, and evolutionary change. Analysis of this general structure subsequently yields general conditions for adaptive speciation by sexual selection, which are finally discussed in relation to existing models.

MATING PROCESS

We start by explaining how to describe a given biological mating process in terms of mating interactions between females, males, and pairs. We then introduce the class of basic mating processes, and give a first example of how our framework can be applied.

MATING STATES AND TRANSITIONS

Females, males, and pairs may be in any finite number of different mating states, which will be denoted, for the i-th state, by the following symbols:

$\textcircled{i}$ for female states,

$\boxed{i}$ for male states, and

$\boxed{\textcircled{i}}$ for pair states.

Pairs always consist of one female and one male. The densities of individuals in these states will be denoted by F_i, M_i, and P_i, respectively. The total densities of females and males, F and M, are then given by

$$F=\sum F_i+\sum P_i\,, \quad M=\sum M_i+\sum P_i. \qquad [1]$$

A specific mating process can now be modeled as a set of transitions between individual mating states, together with the corresponding transition rates. We allow for two qualitatively different kinds of transitions: simple transitions on the one hand, and interactions on the other.

Simple transitions can be either

1 – Transitions between female states,

$$\textcircled{i} \rightarrow \textcircled{j}$$

at rate $\phi_{ij} F_i$,

2 – Transitions between male states,

$$\boxed{i} \rightarrow \boxed{j}$$

at rate $\mu_{ij} M_i$,

3 – Transitions between pair states,

$$\boxed{\textcircled{i}} \rightarrow \boxed{\textcircled{j}}$$

at rate $\psi_{ij} P_i$, or

4 – Pair dissociation,

$$\boxed{\textcircled{i}} \rightarrow \textcircled{j} + \boxed{k}$$

at rate $\eta_{ijk} P_i$.

Interactions can be either

5 – Pair formation,

$$\textcircled{i} + \boxed{j} \rightarrow \boxed{\textcircled{k}}$$

at rate $\gamma_{ijk} F_i M_j$,

6 – Male-female interactions,

$$\textcircled{i} + \boxed{j} \rightarrow \textcircled{k} + \boxed{l}$$

at rate $\alpha_{ijkl} F_i M_j$,

7 – Female-female interactions,

$$\textcircled{i} + \textcircled{j} \rightarrow \textcircled{k} + \textcircled{l}$$

at rate $\chi_{ijkl} F_i F_j$, or

8 – Male-male interactions,

$$\boxed{i} + \boxed{j} \rightarrow \boxed{k} + \boxed{l}$$

at rate $\nu_{ijkl} M_i M_j$.

For the purpose of this study, we do not consider transitions involving female-female or male-male pairs, or interactions between three or more individuals, such as female-pair, male-pair, or pair-pair interactions. Our approach can easily be extended to include such higher-order mating interactions.

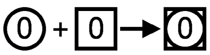
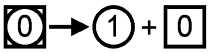
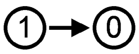
BASIC MATING PROCESSES

The eight transitions listed above allow us to describe a vast variety of biological interactions involving both inter- and intra-sexual selection. For example, our approach can be used to model female choice, sexual conflict, reproductive skew, male-male competition, mutual mate choice, and cooperative breeding. In the course of this study we will introduce a number of examples to illustrate this point.

As a first step, we will focus our mathematical analysis and examples on 'basic mating processes', an important subset of all the models that can be built within our general framework. We refer to a mating process as basic when males and females interact with other individuals in only one of their possible individual states. This initially excludes, for example, potentially interesting mating processes in which individuals can be engaged with multiple interactions at the same time, or mating processes in which individuals change their behavior in future interactions depending on previous experience. Nevertheless, as we will see below, the scope of basic mating process is already huge. Moreover, this class is rich enough to allow for adaptive sympatric speciation by sexual selection, while still remaining analytically tractable – a very helpful combination of features, which makes basic mating processes the primary target of our theoretical analysis. Basic mating processes can be defined more formally, based on the notion of interaction states defined in Appendix A, as the class of mating processes that do not require more than one interaction state per sex. In the later part of this study we will generalize our results to more complex mating processes involving multiple interaction states per sex.

EXAMPLE A

As a first example, suppose we want to describe the mating system of species A, in order to evaluate the potential for adaptive sympatric speciation in this species. In species A, males guard their female partner after mating for some period of time. The male deserts the female at the moment she lays eggs. From that moment onwards, the male is again available for mating, while the female has to take care of the eggs. She becomes available for mating again after the eggs have hatched. In this example, males are either available for mating or occupied with mate guarding. Females may be available for mating, guarded by a male while preparing to lay eggs, or taking care of the eggs. We can therefore distinguish one pair state (pair state 0, for the individuals involved in the mate guarding phase), one male state (male state 0, for males that are available for mating), and two female states (female state 0, for females available for mating, and female state 1, for females occupied with taking care of the eggs). Transitions between these states are then as follows:

	formation of mating pairs,
	dissociation of pairs at egg laying
	termination of egg hatching

In this example, only females in female state 0 and males in male state 0 interact with other individuals. Hence, species A follows a basic mating process, as defined above.

We stress that, already at this point, most of the biological modeling is done. The essential structure of the biological interactions is captured as soon as the mating process has been correctly characterized in terms of individual states and transitions between states. In what follows, we will merely switch from a diagrammatic to a mathematical representation of the mating process and we will apply a number of standard mathematical techniques in order to investigate the consequences of our biological assumptions.

From the diagrammatic representation of the mating process we can infer the set of equations describing changes in the densities of individuals in the different states. For species A, these equations would be as follows,

$$\begin{aligned} \frac{dF_0}{dt} &= -\gamma F_0 M_0 + \phi F_1 \,, & \frac{dF_1}{dt} &= \eta P_0 - \phi F_1 \,, \\ \frac{dM_0}{dt} &= -\gamma F_0 M_0 + \eta P_0 \,, & \frac{dP_0}{dt} &= \gamma F_0 M_0 - \eta P_0 \,. \end{aligned} \qquad [2]$$

Here and below, we omit the indices of the rate constants whenever this does not result in ambiguities.

POPULATION DYNAMICS

In addition to transitions between mating states, the densities associated with mating states also change due to births and deaths.

OFFSPRING PRODUCTION

The birth of new individuals results from specific interactions in the mating process, underlining the intimate connection between mating process and population dynamics. Birth events can occur after pair formation, pair dissociation, or male-female interactions. For any particular model, however, we must specify the moment in the sequence of transitions at which offspring is produced. We then associate with the corresponding transition in the mating process a parameter β, which measures the number of offspring produced.

In some species, there are several different ways in which offspring is produced. For example, in several bird species, males can father offspring through mating with their nest mate or through extra pair copulation. In such a case, there is more than one transition in the mating process in which offspring is produced: we then associate a specific birth coefficient β with each of these. The total offspring production rate B can then be found by adding the rates of all transitions in the mating process in which offspring is produced, weighted by the number of offspring produced in each transition, i.e., using the parameters β as the weighing factors in this sum.

Birth and death rates are assumed to be density-dependent, such that the population can attain a stable equilibrium size through density regulation. Density dependence could act on newly born individuals (through density-dependent survival until reproductive age), on adults (through density-dependent adult mortality), or on both. Similarly, density-dependent birth and mortality rates could vary with the number of newborns (e.g., when population size is limited by resource competition among juveniles), with the number of adult individuals (e.g., when population size is limited by the number of suitable nesting sites), or with both. We allow for all of these possibilities by assuming density-dependent survival until reproductive age, represented by the effective per capita birth rate b, as well as density-dependent mortality, represented by the per capita death rate d. Both rates are allowed to vary with B (a proxy for the number of newborns) and with $M+F$ (the number of adults). With these assumptions, we restrict ourselves to situations where all individuals are equally affected by density-dependent survival and mortality, irrespective of their state in the mating process. Moreover, all individuals are assumed to contribute equally to the density-dependent survival and mortality rates, irrespective of the distribution of individuals over mating states. These restrictions are appropriate here, since the focus of this paper is on sexual selection rather than on ecological processes as the driving force of sympatric speciation.

EXAMPLE A, CONTINUED

For the example of species A introduced above, we choose egg laying as the moment of offspring production. This implies

$$B = \beta \eta P_0 . \quad [3]$$

To specify the population dynamics of species A, we add birth and death rates to equations [2] as follows,

$$\begin{aligned}
\frac{dF_0}{dt} &= -\gamma F_0 M_0 + \phi F_1 + (1-Q) b(B, M+F) B - d(B, M+F)(F_0 - P_0), \\
\frac{dF_1}{dt} &= \eta P_0 - \phi F_1 - d(B, M+F) F_1 , \\
\frac{dM_0}{dt} &= -\gamma F_0 M_0 + \eta P_0 + Q b(B, M+F) B - d(B, M+F)(M_0 - P_0), \\
\frac{dP_0}{dt} &= \gamma F_0 M_0 - \eta P_0 - 2d(B, M+F) P_0 .
\end{aligned} \quad [4]$$

Since pairs consist of two individuals, the death rate per pair is twice as high as the per capita death rate of single individuals. When one individual in a pair dies, the remaining individual is assumed to return to state 0. We assume an equal sex ratio at birth, but we allow for a skewed sex ratio in adults, due to density-*in*dependent gender-specific juvenile survival probabilities. The sex ratio (proportion of males) after survival to the adult stage is denoted by Q, where Q is related to the gender-specific survival probabilities σ_f (for females) and σ_m (for males),

$$Q = \frac{\sigma_m}{\sigma_m + \sigma_f} . \quad [5]$$

Surviving offspring is assumed to enter the mating dynamics in state 0.

Typically, one would lump together the birth coefficients β and b. However, here we choose to conceptually separate the density-*in*dependent effects on the birth rate (as captured by β) that arise directly from interactions in the mating process (e.g., clutch size effects) from the density-dependent effects (as captured by b) that result from other interactions (e.g., density-dependent egg-to-adult survival).

SEPARATION OF ABUNDANCE DYNAMICS AND FREQUENCY DYNAMICS

We begin our general mathematical analysis by separating the differential equations for the densities of the individual mating states into two coupled systems of equations. The first system captures changes in total abundance and the second system describes the dynamics of the relative frequencies of individual states.

From equation [1] we obtain

$$\frac{dF}{dt} = \sum \frac{dF_i}{dt} + \sum \frac{dP_i}{dt} , \quad \frac{dM}{dt} = \sum \frac{dM_i}{dt} + \sum \frac{dP_i}{dt} , \quad [6]$$

which yields the abundance dynamics. For the example of species A, these are given by

$$\begin{aligned}\frac{dF}{dt} &= (1-Q)b(B,M+F)B - d(B,M+F)F,\\ \frac{dM}{dt} &= Qb(B,M+F)B - d(B,M+F)M.\end{aligned} \qquad [7]$$

These equations suggest that we may introduce a rescaled population density N,

$$N = \frac{F}{1-Q} = \frac{M}{Q}, \qquad [8]$$

which then satisfies the equation

$$\frac{dN}{dt} = N\big(\rho - d(B,N)\big), \qquad [9]$$

where ρ is an expression for the rescaled per capita birth rate,

$$\rho = b(B,N)\frac{B}{N}. \qquad [10]$$

In order to derive the second system of equations, we first define the relative frequencies of individuals in the different mating states (f_i, p_i, m_i, q_i) as follows

$$\begin{aligned} f_i &= \frac{F_i}{F}, \quad p_i = \frac{P_i}{F},\\ m_i &= \frac{M_i}{M}, \quad q_i = \frac{P_i}{M},\end{aligned} \qquad [11]$$

such that

$$\sum f_i + \sum p_i = \sum m_i + \sum q_i = 1. \qquad [12]$$

For species A, the equations for the relative frequencies of individuals in the different mating states can now be obtained from equations [4] and [9],

$$\begin{aligned}\frac{df_0}{dt} &= -\gamma f_0 m_0 QN + \phi f_1 + (f_1 + p_0)\rho + p_0 d(B,N),\\ \frac{df_1}{dt} &= \eta p_0 - \phi f_1 - \rho f_1,\\ \frac{dp_0}{dt} &= \gamma f_0 m_0 QN - \eta p_0 - \rho p_0 - p_0 d(B,N),\\ \frac{dm_0}{dt} &= -\gamma f_0 m_0 (1-Q)N + \eta q_0 + \rho q_0 + q_0 d(B,N),\\ \frac{dq_0}{dt} &= \gamma f_0 m_0 (1-Q)N - \eta q_0 - \rho q_0 - q_0 d(B,N).\end{aligned} \qquad [13]$$

As can be seen from equations [13], the dynamics of the relative frequencies of individuals in the different mating states are affected by terms for each of the transitions in the mating process, as well as by terms corresponding to birth and death events. Although one could further simplify equations [13] by rescaling, we refrain from doing so, in order not to obscure the link between population dynamics and evolutionary dynamics, as will become clear in the next section.

EVOLUTIONARY DYNAMICS

On the third level of our hierarchy of time scales, we now describe how to derive the evolutionary dynamics of mating strategies.

MATING STRATEGIES

The rate constants of the mating process (ϕ, η, γ etc.) as well as the parameters β, σ_f, and σ_m are bound to depend on some characteristics of the interacting individuals. In models for sympatric speciation by sexual selection, it is usually assumed that these parameters and rate constants are determined by heritable female and male mating strategies. We also make this assumption. As is common for models of sexual selection, we furthermore suppose that the female and male mating strategies can each be fully characterized by a single continuous mating trait character, which will be denoted by x and y, respectively. The phenotypic expression of x and y is taken to be sex-limited. Within the context of female-choice models, for example, one would typically interpret the female and male mating traits as, respectively, a female mating preference and some characteristic of a male ornament on which the female preference acts. In other models, the mating strategies could be interpreted, for example, in terms of female reluctance and male persuasiveness to mate (sexual conflict), male aggressiveness (male-male competition), investment in maternal and paternal care (parental care), or investment in breeding versus helping (cooperative breeding).

From here on, the rate constant of a given transition in the mating process is taken to be a function of the mating traits of the individual(s) involved in the transition. Therefore, rate constants ϕ will typically depend on x, rate constants α will depend on x and y, and so on. These rules would not apply for rescaled rate constants, which explains why we chose not to rescale equations [13]. The gender-specific survival probabilities σ_f and σ_m are allowed to vary with x and y, respectively, in accordance with the assumption that expression of the mating traits is sex-limited. Birth coefficients β are assumed to depend on the mating traits of the female and male involved in the offspring-producing interaction.

INVASION FITNESS

The primary goal of this study is to identify those biological conditions under which the evolutionary dynamics of mating strategies will result in sympatric speciation. In order to assess the potential for such adaptive speciation through sexual selection, we must find a way to predict the direction of evolutionary change for the mating traits x and y. In general, this is quite a difficult task, but the problem simplifies considerably if we assume that mutations occur only rarely. In that case, mutants arise in a resident population that will have had sufficient time to settle towards its population dynamical equilibrium by the time a new mutant appears. Consequently, variation in the resident population due to previous mutation events will be negligible, and the mutant will interact only with the predominant resident type (or types) that managed to oust previous mutants.

The evolutionary fate of the mutant mating strategy is then determined by its 'invasion fitness' (Metz *et al.*, 1992), that is, by the rare mutant's growth rate in the environment set by the predominant type in the resident population with which the mutant interacts. The mutant's abundance decreases if that growth rate is negative, resulting in the mutant's demise. Alternatively, if the mutant's invasion fitness is positive, its abundance is expected to increase (even though mutants are still highly likely to go extinct through demographic stochasticity; Dieckmann & Law, 1996). A mutant with positive invasion fitness will either replace the original resident or stably coexist with it, establishing, in our case, a mating dimorphism. It can be shown that, as long as mutations have small effects, positive invasion fitness implies mutant fixation, unless the population dynamics undergoes a bifurcation as a result of gradual evolution or evolution reaches points in trait space termed evolutionary branching points (Geritz *et al.*, 2002). The former phenomenon is rare and is actually absent in many models, while evolutionary branching points arise more readily. Gradual evolution then converges on such points, which is followed, first, by the establishment of a dimorphism around these points, and second, by the subsequent widening of that dimorphism in trait space. The feasibility of this process, known as evolutionary branching, is determined by the properties of the invasion fitness function (Metz *et al.*, 1996; Geritz *et al.*, 1998). We will therefore now proceed by showing how the invasion fitness of mating strategies can be derived within our general framework.

The invasion fitness $\lambda(x,y,\hat{x},\hat{y})$ measures the growth rate of a rare mutant with mating traits x and y, which has arisen in a population of residents with mating traits $\hat{x}$ and $\hat{y}$. It is thus clear that the invasion fitness can be derived from the mutant's population dynamics. We proceed as follows: we start by diagrammatically considering the interaction structure of the mating process for mutant females (FIGURE 1A) and mutant males (FIGURE 1B). At this point, we must take care to distinguish mutant-female/resident-male pairs from resident-female/mutant-male pairs. Assuming that the density of mutants is low, we may neglect the interactions between mutants, including those between mutant females and mutant males. From the structure and transitions of the mating process, we subsequently derive the equations for the densities of resident and mutant individuals in the different states. We then proceed as we did above by adding birth and death terms and separating the dynamics of abundances and frequencies.

FIGURE 1 – MATING DIAGRAM FOR SPECIES A

The interactions involved in the mating process for individuals of species A are represented as mating diagrams, showing the transitions between individual male states (squares), female states (circles), or pair states (circles within squares). (A) Interactions of a mutant female individual. (B) Interactions of a mutant male individual. In (A) and (B) the individual states of the focal mutant individual are represented by gray-filled symbols, while the states of resident individuals are represented by open symbols. Transitions between resident states that are irrelevant for the behavior of the mutant are shown in light gray. See the main text for the biological interpretation of the individual states.

Since the resident is assumed to be at population dynamical equilibrium when the mutant tries to invade, the right-hand side of the equation for the resident's abundance can be equated to zero, and the same applies to the equations for the resident's frequencies across states of the mating process. For species A, this results in the following equilibrium conditions for the resident,

$$\begin{aligned}
\hat{f}_1 &= \hat{f}_0 \hat{m}_0 \hat{Q} \hat{N} \frac{\eta(\hat{x},\hat{y})}{\phi(\hat{x})+\hat{\rho}} \frac{\gamma(\hat{x},\hat{y})}{\eta(\hat{x},\hat{y})+2\hat{\rho}}, \\
\hat{p}_0 &= \hat{f}_0 \hat{m}_0 \hat{Q} \hat{N} \frac{\gamma(\hat{x},\hat{y})}{\eta(\hat{x},\hat{y})+2\hat{\rho}}, \\
\hat{q}_0 &= \hat{f}_0 \hat{m}_0 \left(1-\hat{Q}\right) \hat{N} \frac{\gamma(\hat{x},\hat{y})}{\eta(\hat{x},\hat{y})+2\hat{\rho}}, \\
\hat{\rho}\hat{N} &= b\left(\hat{B},\hat{N}\right)\hat{B} = d\left(\hat{B},\hat{N}\right)\hat{N}.
\end{aligned} \qquad [14]$$

From here on, hats are used to denote resident traits, frequencies, densities, and sex ratios. Without explicitly expressing this in our notation, the latter quantities are functions of the resident mating traits. In particular, the population size $\hat{N}$, the offspring production rate $\hat{B}$, and the relative frequencies $\hat{f}_0$, $\hat{m}_0$, and $\hat{q}_0$ are functions of $\hat{x}$ and $\hat{y}$. Similarly,

$$\hat{Q}=\frac{\sigma_m\left(\hat{y}\right)}{\sigma_f\left(\hat{x}\right)+\sigma_m\left(\hat{y}\right)}. \tag{15}$$

Mutant traits, frequencies, or densities will be denoted by the corresponding symbols without hats. Again, without actually expressing this in our notation, mutant frequencies and densities vary with one or more mutant traits, as well as with the resident mating traits.

For the mutant, the procedure outlined above yields two differential equations for ε_f and ε_m, the densities of mutant females and males, respectively. One would expect the mating traits to be coded by separate sets of genes, such that every individual mutation affects either x or y, but not both at the same time. Yet, for the sake of generality, we consider the latter possibility in our analysis. The cases where individual mutations always affect only a single mating trait can be recovered from our more general analysis by choosing either $x=\hat{x}$ or $y=\hat{y}$.

$$\begin{aligned}
\frac{d\varepsilon_{fx}}{dt}&=\frac{1}{2}\left(1-\hat{Q}\right)\frac{\sigma_f\left(x\right)}{\sigma_f\left(\hat{x}\right)}b\left(\hat{B},\hat{N}\right)\left(\beta\left(x,\hat{y}\right)\eta\left(x,\hat{y}\right)\varepsilon_{fx}\,p_0+\beta\left(\hat{x},\hat{y}\right)\eta\left(\hat{x},\hat{y}\right)\varepsilon_{mx}\,\hat{q}_0\right)\\
&\quad-\varepsilon_{fx}\,d\left(\hat{B},\hat{N}\right),\\
\frac{d\varepsilon_{mx}}{dt}&=\frac{1}{2}\hat{Q}b\left(\hat{B},\hat{N}\right)\left(\beta\left(x,\hat{y}\right)\eta\left(x,\hat{y}\right)\varepsilon_{fx}\,p_0+\beta\left(\hat{x},\hat{y}\right)\eta\left(\hat{x},\hat{y}\right)\varepsilon_{mx}\,\hat{q}_0\right)\\
&\quad-\varepsilon_{mx}\,d\left(\hat{B},\hat{N}\right).
\end{aligned}\tag{16}$$

Similarly, the equations for male mutants are

$$\begin{aligned}
\frac{d\varepsilon_{fy}}{dt}&=\frac{1}{2}\left(1-\hat{Q}\right)b\left(\hat{B},\hat{N}\right)\left(\beta\left(\hat{x},\hat{y}\right)\eta\left(\hat{x},\hat{y}\right)\varepsilon_{fy}\,\hat{p}_0+\beta\left(\hat{x},y\right)\eta\left(\hat{x},y\right)\varepsilon_{my}\,q_0\right)\\
&\quad-\varepsilon_{fy}\,d\left(\hat{B},\hat{N}\right),\\
\frac{d\varepsilon_{my}}{dt}&=\frac{1}{2}\hat{Q}\frac{\sigma_m\left(y\right)}{\sigma_m\left(\hat{y}\right)}b\left(\hat{B},\hat{N}\right)\left(\beta\left(\hat{x},\hat{y}\right)\eta\left(\hat{x},\hat{y}\right)\varepsilon_{fy}\,\hat{p}_0+\beta\left(\hat{x},y\right)\eta\left(\hat{x},y\right)\varepsilon_{my}\,q_0\right)\\
&\quad-\varepsilon_{my}\,d\left(\hat{B},\hat{N}\right).
\end{aligned}\tag{17}$$

The factor $1/2$ in front of the birth term reflects the fact that only half of the offspring of a mutant individual will inherit the mutant strategy. Note that there are two contributions to the birth rate, corresponding to mutant-female/resident-male and to resident-female/mutant-male crosses, respectively. Due to the sex-limited expression of the mating traits, males carrying the mutant female mating trait are phenotypically equivalent to resident males. Consequently, the distribution of such mutant males over the individual states of the mating process is identical to the distribution of resident males over mating states (hence, in equations [16], we substituted $\hat{q}_0$ for the frequency of male mutants in the pair state). A similar argument explains why we may substitute $\hat{p}_0$ for the relative frequency of paired females carrying the mutant male mating trait in equations [17].

As before, we reduce equations [16] and [17] by introducing rescaled mutant abundances ε_x and ε_y,

$$\begin{aligned} \frac{\varepsilon_x}{\sigma_f(\hat{x})+\sigma_m(\hat{y})} &= \frac{\varepsilon_{fx}}{\sigma_f(x)} = \frac{\varepsilon_{mx}}{\sigma_m(\hat{y})}, \\ \frac{\varepsilon_y}{\sigma_f(\hat{x})+\sigma_m(\hat{y})} &= \frac{\varepsilon_{fy}}{\sigma_f(\hat{x})} = \frac{\varepsilon_{my}}{\sigma_m(y)}. \end{aligned} \qquad [18]$$

The resulting equations may be further rewritten, using equations [14], to give

$$\begin{aligned} \frac{1}{\varepsilon_x}\frac{d\varepsilon_x}{dt} &= \rho_x - \hat{\rho}, \\ \frac{1}{\varepsilon_y}\frac{d\varepsilon_y}{dt} &= \rho_y - \hat{\rho}, \end{aligned} \qquad [19]$$

where ρ_x and ρ_y are shorthand notation for $\rho_x(x,\hat{x},\hat{y})$ and $\rho_y(y,\hat{x},\hat{y})$, the average per capita birth rates of female mutants and male mutants, respectively, in the environment set by the resident population. These quantities are given by

$$\begin{aligned} \rho_x(x,\hat{x},\hat{y}) &= \frac{1}{2}\left(\hat{\rho}(\hat{x},\hat{y}) + \left(1-\hat{Q}\right)\frac{\sigma_f(x)}{\sigma_f(\hat{x})} b\left(\hat{B},\hat{N}\right)\beta(x,\hat{y})\eta(x,\hat{y})p_0\right), \\ \rho_y(y,\hat{x},\hat{y}) &= \frac{1}{2}\left(\hat{\rho}(\hat{x},\hat{y}) + \hat{Q}\frac{\sigma_m(y)}{\sigma_m(\hat{y})} b\left(\hat{B},\hat{N}\right)\beta(\hat{x},y)\eta(\hat{x},y)q_0\right). \end{aligned} \qquad [20]$$

Note that

$$\hat{\rho}(\hat{x},\hat{y}) = \rho_x(\hat{x},\hat{x},\hat{y}) = \rho_y(\hat{y},\hat{x},\hat{y}), \qquad [21]$$

which follows from equations [3], [8], [10], [14], and [20].

The right-hand sides of the equations [19] are the per capita growth rates of female and male mutants, respectively. Since the invasion fitness λ is defined as the rare mutant's per capita growth rate, we find

$$\lambda(x,y,\hat{x},\hat{y}) = \left(\rho_x(x,\hat{x},\hat{y}) - \rho_x(\hat{x},\hat{x},\hat{y})\right) + \left(\rho_y(y,\hat{x},\hat{y}) - \rho_y(\hat{y},\hat{x},\hat{y})\right). \qquad [22]$$

Here we have conveniently combined the per capita growth rates of female and male mutants into a single expression for the invasion fitness that is valid for both types of mutants, as can easily be seen from equations [21].

As illustrated by equations [20], the mutant's invasion fitness depends on the relative frequencies of mutant individuals in the different mating states (in particular p_0 and q_0), which remain to be determined.

MUTANT FREQUENCIES

As it turns out, the dynamics of mutant frequencies are independent of mutant abundances, ε_x and ε_y. We may thus safely assume that, by the time the mutant reaches abundances ε_x or ε_y, it will have had ample time to attain a stable equilibrium distribution over mating states.

For species A, the equilibrium frequencies of mutant females are given by

$$\begin{aligned} f_1 &= f_0 \hat{m}_0 \hat{Q} \hat{N} \frac{\eta(x,\hat{y})}{\phi(x)+\rho_x} \frac{\gamma(x,\hat{y})}{\eta(x,\hat{y})+\hat{\rho}+\rho_x}, \\ p_0 &= f_0 \hat{m}_0 \hat{Q} \hat{N} \frac{\gamma(x,\hat{y})}{\eta(x,\hat{y})+\hat{\rho}+\rho_x}. \end{aligned} \qquad [23]$$

Likewise, the equilibrium frequency of mutant males is given by

$$q_0 = \hat{f}_0 m_0 \left(1-\hat{Q}\right) \hat{N} \frac{\gamma(\hat{x},y)}{\eta(\hat{x},y)+\hat{\rho}+\rho_y}. \qquad [24]$$

FEEDBACK STRUCTURE

The feedback between mating process, population dynamics, and evolutionary change is captured by the mutual dependencies between per capita birth rates, frequencies of mating pairs, and population size. Of course, we cannot eliminate these dependencies, but we may attempt to capture the structure of the feedback between the three levels in as few equations as possible.

For species A, a minimum of six equations is necessary to fully characterize the feedback structure. First of all, we need two equations for the equilibrium frequencies in the mating process,

$$\begin{aligned} f_0 &= 1 - f_0 \hat{m}_0 \hat{Q} \hat{N} \tau_{f\times m}\left(x,\hat{y},\rho_x,\hat{\rho}\right), \\ m_0 &= 1 - \hat{f}_0 m_0 \left(1-\hat{Q}\right) \hat{N} \tau_{m\times f}\left(\hat{x},y,\rho_y,\hat{\rho}\right). \end{aligned} \qquad [25]$$

Then, two equilibrium conditions to describe the resident population dynamics,

$$\begin{aligned} \hat{\rho} \hat{N} &= b\left(\hat{B},\hat{N}\right) \hat{B}, \\ b\left(\hat{B},\hat{N}\right) \hat{B} &= d\left(\hat{B},\hat{N}\right) \hat{N}. \end{aligned} \qquad [26]$$

And, finally, two more equations are required on the evolutionary level, for the mutant's per capita birth rates,

$$\begin{aligned} \rho_x &= \frac{1}{2}\left(f_0 \hat{m}_0 \hat{Q}\left(1-\hat{Q}\right) \hat{N} \frac{\sigma_f(x)}{\sigma_f(\hat{x})} b\left(\hat{B},\hat{N}\right) \xi\left(x,\hat{y},\rho_x,\hat{\rho}\right) + \hat{\rho} \right), \\ \rho_y &= \frac{1}{2}\left(\hat{f}_0 m_0 \hat{Q}\left(1-\hat{Q}\right) \hat{N} \frac{\sigma_m(y)}{\sigma_m(\hat{y})} b\left(\hat{B},\hat{N}\right) \xi\left(\hat{x},y,\rho_y,\hat{\rho}\right) + \hat{\rho} \right). \end{aligned} \qquad [27]$$

The functions $\tau_{f\times m}$ and $\tau_{m\times f}$ introduced here can be interpreted as the time fraction spent by females in interactions with a single male, and the time fraction spent by males in interaction with a single female, respectively. The function ξ measures the mutant's per capita offspring production rate. The definition of functions $\tau_{f\times m}$, $\tau_{m\times f}$, and ξ is given in equations (66) in Appendix B.

FURTHER EXAMPLES

Although we have so far focused attention on the particular example of species A, it turns out that the feedback structure captured in equations [25]-[27] can easily be generalized for the whole class of basic mating processes. In order to illustrate this point, we will now apply the procedure outlined in the previous section to three further examples of basic mating processes, adding a new level of biological complexity in each example.

EXAMPLE B

We start by demonstrating that the elaborateness of the mating process by itself does not affect the structure of the feedback. To illustrate this point, we consider another example, species B, with intricate mating interactions (FIGURE 2). The mating process of species B involves several different routes, along which offspring is produced. Males of species B may form mating pairs with a female and participate in parental care, or they may just mate with the female, without spending any time to take care of the offspring. In addition, each of these routes consists of several phases, reflected by sequential transitions between mating states.

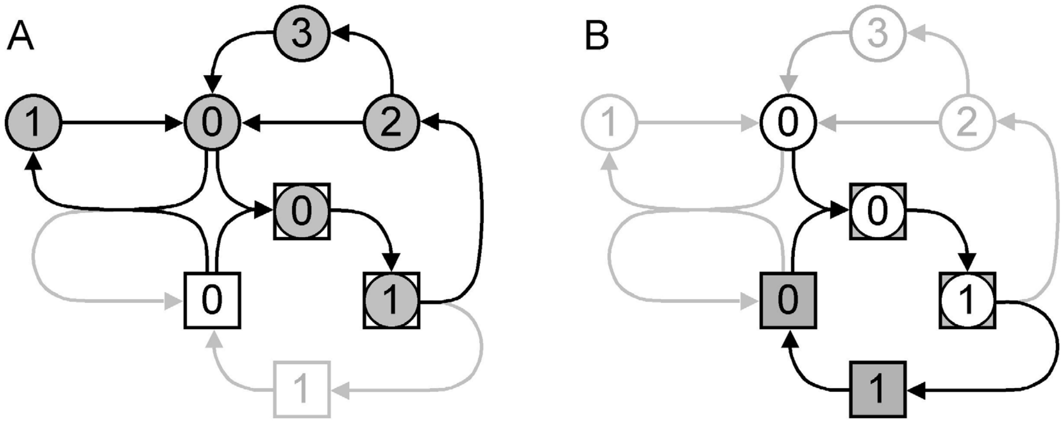


FIGURE 2 – MATING DIAGRAM FOR SPECIES B

(A) Interactions of a mutant female individual. (B) Interactions of a mutant male individual. See the main text for the biological interpretation of the individual states; see also the legend of FIGURE 1.

Given this degree of complexity it is remarkable that after working through all the steps involved in deriving the invasion fitness for species B one arrives at exactly the same set of equations for the mating process as derived earlier for species A and summarized in equations [25]-[27]. Surprisingly, all differences between the mating systems of species A and B can be absorbed in the three functions $\tau_{f\times m}$, $\tau_{m\times f}$, and ξ, which, for species B, are given in equations (67) in Appendix B.

EXAMPLE C

Our next example is meant to illustrate the effects of non-interactive processes that limit the availability of individuals for interactions. In the examples of species A and B, individuals were always available for interactions, unless they had not yet completed the sequence of transitions initiated by their previous interaction with another individual. Previous interactions with other individuals may, however, not be the only cause for (temporary) unavailability. Alternatively, individuals may also become unavailable for interactions as a result of processes that involve only the focal individual itself, independently of interactions with other individuals.

FIGURE 3 – MATING DIAGRAM FOR SPECIES C

(A) Interactions of a mutant female individual. (B) Interactions of a mutant male individual. See the main text for the biological interpretation of the individual states; see also the legend of FIGURE 1.

Such a process is illustrated by the example of species C (FIGURE 3). Females of species C periodically switch between a receptive state (female state 0) and an unreceptive state (female state 2), in which they do not interact with males or with other females. In other respects, the mating system of species C is identical to that of species A: males and females form mating pairs; the male guards the female, until she lays eggs; the male becomes available for mating immediately after he has stopped guarding the female; and the female takes care of the eggs until they hatch, returning to the unreceptive state afterwards.

The feedback structure derived for this example species differs from that in equations [25]-[27] in only one detail. The equations for the equilibrium distribution of individuals over mating states contain an additional term: specifically, we find that

$$\begin{gathered} f_0 = 1 - f_0\, \tau_f(x, \rho_x, \hat{\rho}) - f_0 \hat{m}_0 \hat{Q} \hat{N}\, \tau_{f\times m}(x, \hat{y}, \rho_x, \hat{\rho}), \\ m_0 = 1 - \hat{f}_0 m_0 \left(1 - \hat{Q}\right) \hat{N}\, \tau_{m\times f}\left(\hat{x}, y, \rho_y, \hat{\rho}\right). \end{gathered} \qquad [28]$$

These equations show that there are now two processes determining the availability of females for interactions (as measured by f_0). For a part of their time (measured by $\tau_{m\times f}$), females are caught up in interactions with males. For another part of their time (measured by τ_f), females are not available for interactions due to the fact that they are in their unreceptive state. This interpretation of the functions τ_f and $\tau_{m\times f}$ in terms of relative time costs follows from equations [B-3] in Appendix B, where the functions τ are expressed in terms of the rate constants of the mating process of species C. From these expressions it can be seen that the function τ_f measures the length of the unreceptive period, relative to the length of the receptive period.

EXAMPLE D

Since the mating structures discussed so far did not yet involve male-male or female-female interactions, we will now consider another example, species D (FIGURE 4), to illustrate the effects of intra-sexual selection. In species D, males compete amongst one another for mating territories. Males without mating territories establish themselves as territory owners by claiming their own mating territory. Territory owners may, however, lose their mating territory again when they are evicted due to conflicts with other territorial males. Females of species D choose a partner from the males that have succeeded in establishing a mating territory. The time needed for mating is negligible. After mating, females invest some time in maternal care.

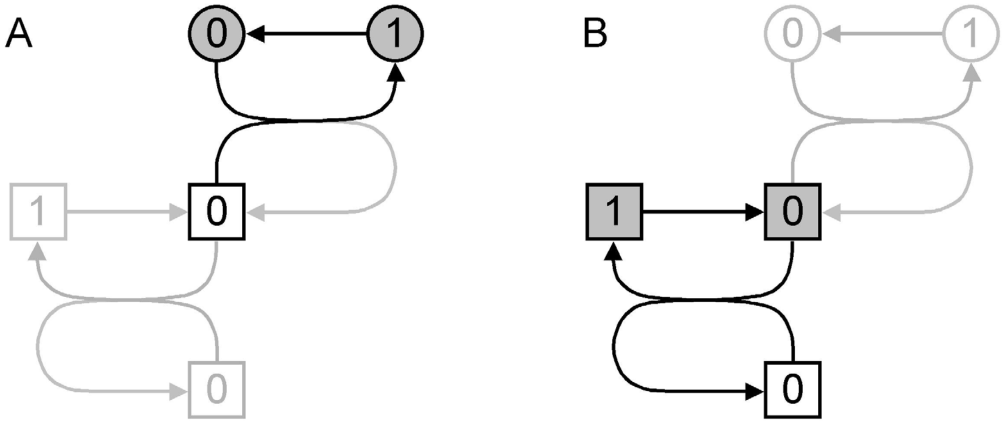


FIGURE 4 – MATING DIAGRAM FOR SPECIES D

(A) Interactions of a mutant female individual. (B) Interactions of a mutant male individual. See the main text for the biological interpretation of the individual states; see also the legend of FIGURE 1.

Based on these biological assumptions, we again obtain a feedback structure that is comparable to that in equations [25]-[27]. In fact, the feedback structure for species

D differs from the latter only in the equations for the mating process, which are now given by

$$\begin{aligned} f_0 &= 1 - f_0 \hat{m}_0 \hat{Q}\hat{N}\, \tau_{f\times m}\left(x, \hat{y}, \rho_x, \hat{\rho}\right), \\ m_0 &= 1 - m_0 \hat{m}_0 \hat{Q}\hat{N}\, \tau_{m\times m}\left(y, \hat{y}, \rho_y, \hat{\rho}\right). \end{aligned} \qquad [29]$$

Note that the term involving $\tau_{m\times f}$ is absent. Instead, there is now a new term, involving the relative time cost $\tau_{m\times m}$, which captures the effects of interactions between males: in species D, males interact with females when they mate. However, the duration of matings is negligible for males, and, therefore, male interactions with females do not show up in the male's time budget. Hence, $\tau_{m\times f} \equiv 0$. Interactions with other males, however, do have an impact on the male's availability for mating; if a male loses his mating territory after a conflict with another male, he temporarily loses his opportunity to mate. Correspondingly, as illustrated by the definition of $\tau_{m\times m}$ in equations (69) in Appendix B, the function $\tau_{m\times m}$ measures the time spent without a mating territory, relative to the time that a mating territory can be maintained in the face of competition with other males.

THE GENERAL FEEDBACK STRUCTURE OF BASIC MATING PROCESSES

We refrain from providing additional examples to illustrate the effects of intra-sexual interactions between females or non-interactive processes that could limit male availability for interactions. These effects can be deduced from the examples of species C and D by switching the roles of the two sexes. With this in mind, we note that the examples of species A-D collectively incorporate all possible types of inter-sexual and intra-sexual interactions between individuals. One can therefore conjecture that combining the feedback structures derived for the example species A, C, and D, will result in the general feedback structure covering all basic mating processes that can be constructed within our formalism.

CHARACTERIZATION OF THE GENERAL FEEDBACK STRUCTURE

Indeed, it can be shown formally that the feedback structure of basic mating processes can always be characterized in terms of the following six equations. The first two equations describe the relative time fractions f and m during which females and males, respectively, are available for interactions with other individuals

$$\begin{aligned} f &= 1 - f\,\tau_f\left(x, \rho_x, \hat{\rho}\right) - f\,\hat{m}\hat{Q}\hat{N}\,\tau_{f\times m}\left(x, \hat{y}, \rho_x, \hat{\rho}\right) - f\,\hat{f}\left(1-\hat{Q}\right)\hat{N}\,\tau_{f\times f}\left(x, \hat{x}, \rho_x, \hat{\rho}\right), \\ m &= 1 - m\,\tau_m\left(y, \rho_y, \hat{\rho}\right) - m\,\hat{f}\left(1-\hat{Q}\right)\hat{N}\,\tau_{m\times f}\left(\hat{x}, y, \rho_y, \hat{\rho}\right) - m\,\hat{m}\hat{Q}\hat{N}\,\tau_{m\times m}\left(y, \hat{y}, \rho_y, \hat{\rho}\right). \end{aligned} \qquad [30]$$

Each of the terms on the right-hand side above corresponds to a relative time cost associated with non-interactive processes, inter-sexual interactions, and intra-sexual interactions, respectively. These time costs collectively limit the availability of individuals for interactions.

The third and fourth equation of the feedback structure are given by

$$\begin{gathered}\hat{\rho}\hat{N}=b\big(\hat{B},\hat{N}\big)\hat{B}\,,\\ b\big(\hat{B},\hat{N}\big)\hat{B}=d\big(\hat{B},\hat{N}\big)\hat{N}\,,\end{gathered} \qquad [31]$$

and characterize the equilibrium of the resident population dynamics.

The final two equations of the feedback structure define the per capita birth rates of female and male mutants,

$$\begin{aligned}\rho_x&=\frac{1}{2}\left(f_0\hat{m}_0\hat{Q}\big(1-\hat{Q}\big)\hat{N}\frac{\sigma_f(x)}{\sigma_f(\hat{x})}b\big(\hat{B},\hat{N}\big)\xi\big(x,\hat{y},\rho_x,\hat{\rho}\big)+\hat{\rho}\right),\\ \rho_y&=\frac{1}{2}\left(\hat{f}_0m_0\hat{Q}\big(1-\hat{Q}\big)\hat{N}\frac{\sigma_m(y)}{\sigma_m(\hat{y})}b\big(\hat{B},\hat{N}\big)\xi\big(\hat{x},y,\rho_y,\hat{\rho}\big)+\hat{\rho}\right).\end{aligned} \qquad [32]$$

These six equations are sufficient to capture all basic mating processes. We can therefore assess the potential for sympatric speciation through evolutionary branching of mating strategies for the entire class of basic mating processes at once, by analyzing the general feedback structure in equations [30]-[32].

As illustrated by example of species B and backed up by the formal derivation, a particularly attractive feature of this approach is that the assessment thus obtained will be fully independent of all the potentially very intricate details of the underlying mating process, as long as the complexity of the mating process is not increased in terms of the number of interaction states (Appendix A; we will illustrate below that more equations are needed to characterize the feedback structure of mating processes that involve more than a single interaction state for males or females).

ANALYSIS OF THE GENERAL FEEDBACK STRUCTURE

The general feedback structure for basic mating processes, equations [30]-[32], will be analyzed by investigating the properties of the invasion fitness function

$$\lambda=\big(\rho_x-\hat{\rho}\big)+\big(\rho_y-\hat{\rho}\big). \qquad [33]$$

This will allow us to delineate the conditions under which sexual selection is capable of creating and maintaining polymorphisms of mating strategies through evolutionary branching. In order to evaluate the potential for evolutionary branching, we must determine certain first- and second-order derivatives of the invasion fitness function λ (see Appendix C for more details). In particular, we must calculate the components of the selection gradient,

$$G_x\big(\hat{x},\hat{y}\big)=\left.\frac{\partial\lambda\big(x,y,\hat{x},\hat{y}\big)}{\partial x}\right|_{\substack{x=\hat{x}\\ y=\hat{y}}} \quad\text{and}\quad G_y\big(\hat{x},\hat{y}\big)=\left.\frac{\partial\lambda\big(x,y,\hat{x},\hat{y}\big)}{\partial y}\right|_{\substack{x=\hat{x}\\ y=\hat{y}}}, \qquad [34]$$

in order to identify the endpoints $\left(x^*,y^*\right)$ of gradual evolution in populations with monomorphic mating traits. At such points, also referred to as singular strategies, the selection gradient vanishes,

$$G_x\left(x^*,y^*\right)=G_y\left(x^*,y^*\right)=0\,. \qquad [35]$$

Evolutionary branching of the female and male mating traits can only occur when the singular strategy $\left(x^*,y^*\right)$ is (1) convergence stable, that is, attainable by a series of small mutational steps (Eshel, 1983), and (2) locally evolutionarily unstable, that is, susceptible to invasion by some neighboring strategies (Geritz *et al.*, 1998). The first condition requires at least (see Appendix C) that both components of the selection gradient point towards the singular strategy,

$$\left.\frac{\partial G_x(\hat{x},\hat{y})}{\partial \hat{x}}\right|_{\substack{\hat{x}=x^*\\ \hat{y}=y^*}}<0 \quad\text{and}\quad \left.\frac{\partial G_y(\hat{x},\hat{y})}{\partial \hat{y}}\right|_{\substack{\hat{x}=x^*\\ \hat{y}=y^*}}<0\,. \qquad [36]$$

The second condition requires that the invasion fitness attains a minimum with respect to the mutant strategies at the singular strategy pair, that is,

$$\left.\frac{\partial^2 \lambda(x,y,\hat{x},\hat{y})}{\partial x^2}\right|_{\substack{x=\hat{x}=x^*\\ y=\hat{y}=y^*}}>0 \quad\text{and}\quad \left.\frac{\partial^2 \lambda(x,y,\hat{x},\hat{y})}{\partial y^2}\right|_{\substack{x=\hat{x}=x^*\\ y=\hat{y}=y^*}}>0\,. \qquad [37]$$

Evaluating conditions [35]-[37] is somewhat complicated, since there exist mutual dependencies between the derivatives of the fitness function and the derivatives of ρ_x, ρ_y, f, m, $\hat{B}$, and $\hat{N}$, as can be seen from equations [30]-[33]. These interdependencies can most easily be disentangled when the feedback equations are rescaled and replaced by suitable approximations. For example, close to the singular strategy the following second-order approximation holds,

$$\begin{aligned}\frac{f(x,\hat{x},\hat{y})}{f^*}\approx{}&1+f_x^*\left(x-x^*\right)+f_{\hat{x}}^*\left(\hat{x}-x^*\right)+f_{\hat{y}}^*\left(\hat{y}-y^*\right)\\ &+\frac{1}{2}\left(f_{xx}^*\left(x-x^*\right)^2+f_{\hat{x}\hat{x}}^*\left(\hat{x}-x^*\right)^2+f_{\hat{y}\hat{y}}^*\left(\hat{y}-y^*\right)^2\right)\\ &+f_{x\hat{x}}^*\left(x-x^*\right)\left(\hat{x}-x^*\right)+f_{x\hat{y}}^*\left(x-x^*\right)\left(\hat{y}-y^*\right)+f_{\hat{x}\hat{y}}^*\left(\hat{x}-x^*\right)\left(\hat{y}-y^*\right),\end{aligned} \qquad [38]$$

where $f^*=f\left(x^*,x^*,y^*\right)$. The constant coefficients f_x^*, $f_{\hat{x}}^*$, and so on, are related to derivatives of $f\left(x,\hat{x},\hat{y}\right)$ evaluated at the singular strategy, e.g.,

$$f_x^*=\frac{1}{f^*}\left.\frac{\partial f(x,\hat{x},\hat{y})}{\partial x}\right|_{\substack{x=\hat{x}=x^*\\ \hat{y}=y^*}}. \qquad [39]$$

By substituting equations [38] together with the approximations for the other functions listed above (for details, see Appendix D) in the general feedback structure of basic mating systems as captured by equations [30]-[33], we obtain equations from which the derivatives of the fitness function can be solved. In other words, the local slope and curvature of the fitness function can be expressed in terms of derivatives of the functions τ_f, τ_m, $\tau_{f\times f}$, $\tau_{f\times m}$, $\tau_{m\times f}$, $\tau_{m\times m}$, and ξ. As the reader may recall, these functions capture the structure of the mating process and therefore are uniquely determined by the specific biological assumptions defining this process.

For example, the coefficient f_x^* can be solved from the following equations

$$\begin{aligned} &\lambda_x^* = \rho_{x,x}^* \\ &\frac{\rho_{x,x}^*}{\rho^*} = \frac{1}{2} f_x^* + \frac{1}{2}\left(\xi_x^* + \xi_\rho^* \rho_{x,x}^*\right) + \frac{1}{2}\sigma_x^* , \\ &-f_x^* = \left(\tau_{f,x}^* + \tau_{fm,x}^* + \tau_{ff,x}^*\right) + \left(\tau_{f,\rho}^* + \tau_{fm,\rho}^* + \tau_{ff,\rho}^*\right)\rho_{x,x}^* , \end{aligned} \qquad [40]$$

which arise from equations [33], [32], and [30], respectively, by differentiating their right-hand and left-hand sides with respect to x. The three unknowns in the above equations are λ_x^*, $\rho_{x,x}^*$, and f_x^*. The coefficients ρ^*, ξ_x^*, $\tau_{f,\rho}^*$, and so on, are determined by the structure of the mating process and the population dynamical parameters. The coefficient λ_x^* represents the derivative of the fitness function $\lambda(x,y,\hat{x},\hat{y})$ with respect to x, evaluated at the singular strategy. Since the fitness gradient vanishes at the singular strategy, λ_x^* must be equal to zero. Hence, we can infer from equations [40] that

$$\lambda_x^* = \rho_{x,x}^* = 0 , \quad f_x^* = -\left(\tau_{f,x}^* + \tau_{fm,x}^* + \tau_{ff,x}^*\right). \qquad [41]$$

After applying a similar procedure to obtain other required derivatives of the fitness function (the full analysis is given in Appendix D), we find that the conditions for evolutionary stability and convergence stability of the singular strategy can be written as

$$\begin{aligned} &\left.\frac{\partial^2 \lambda(x,\hat{y},\hat{x},\hat{y})}{\partial x^2}\right|_{\substack{x=\hat{x}=x^* \\ \hat{y}=y^*}} = C_x H_x > 0 , \\ &\left.\frac{\partial G_x(\hat{x},\hat{y})}{\partial \hat{x}}\right|_{\substack{\hat{x}=x^* \\ \hat{y}=y^*}} = C_x\left(H_x - \left(\Delta_x^{f\times f} + \Delta_x^{f\times m} + \Delta_x^{B,N,Q}\right)\right) < 0 , \end{aligned} \qquad [42]$$

with similar expressions for the male mating trait (see Appendix D). The coefficients C_x, H_x, and so on, are complicated expressions of the biological parameters, which, however, have a straightforward biological interpretation. For example, the coefficient C_x scales the invasion fitness with the birth rate ρ^*, including any effects of the magnitude of the birth rate on the distribution of individuals over mating states,

$$C_x = \frac{\rho^*}{2 - \rho^*\left(\xi_\rho^* + \tilde{f}_\rho^*\right)} , \qquad [43]$$

At population dynamical equilibrium, the birth rate ρ^* is equal to the mortality rate d^*. In equation [43] and henceforth, we use $\tilde{f}_z^*$, (z can be either x, xx, ρ, or $\hat{\rho}$) as shorthand notation for $-\tau_{f,z}^* - \tau_{ff,z}^* - \tau_{fm,z}^*$. In biologically relevant cases,

$$C_x > 0 . \qquad [44]$$

The coefficient H_x measures the local curvature of the fitness function in the direction of the female trait, and therefore determines whether selection is stabilizing ($H_x < 0$) or disruptive ($H_x > 0$) at the singular strategy. Selection on the mating traits is generated through their effects on the availability for interactions in the mating process, as well as through their effects on the effective offspring production per interaction (given by the product of offspring production rate and offspring survival). Accordingly,

$$H_x = \tilde{f}^*_{xx} + \left(\sigma^*_{xx} + 2\sigma^*_x \, \xi^*_x + \xi^*_{xx}\right). \qquad [45]$$

Since we are interested in speciation by disruptive sexual selection rather than by disruptive natural selection, we assume, from here on, that viability selection is stabilizing, and hence, that $\sigma^*_{xx} < 0$. This means that H_x will be negative unless mating and offspring production generate disruptive selection.

The coefficients Δ, finally, which occur in the conditions for convergence stability of the singular strategy, measure how strongly the local topology of the fitness landscape responds to changes in the resident strategy. For example, in cases where selection is frequency-independent, the fitness landscape is fixed. This implies that the location of fitness maxima and minima is independent of the resident strategy and, consequently, that $\Delta_x^{f\times f} = \Delta_x^{f\times m} = \Delta_x^{B,N,Q} = 0$ (see below). By contrast, when selection is frequency-dependent the fitness landscape changes in response to evolution of the resident strategy. If this effect is large enough, it is possible that adaptive change leads to a singular strategy at which selection turns disruptive (Abrams *et al.*, 1993). In such a case, the population cannot escape from the fitness minimum through gradual evolution, other than by undergoing evolutionary branching (Metz *et al.*, 1996).

We decompose the frequency-dependent effect of selection into separate contributions representing intra-sexual interactions ($\Delta_x^{f\times f}$), inter-sexual interactions ($\Delta_x^{f\times m}$), and ecological effects of the mating traits mediated by effects on the population size, population sex ratio, and generation time ($\Delta_x^{B,N,Q}$). The first two of these separate contributions are given by

$$\Delta_x^{f\times f} = \tau^*_{ff,x\hat{x}} + \frac{\left(1+\tau^*_{mm}\right)\left(\left(\tilde{f}^*_x\right)^2 \tau^*_{ff} + \tilde{f}^*_x\left(\tau^*_{ff,x} + \tau^*_{ff,\hat{x}}\right) - \tau^*_{ff,x}\,\tau^*_{ff,\hat{x}}\right)}{\left(1+\tau^*_{ff}\right)\left(1+\tau^*_{mm}\right) - \tau^*_{mf}\,\tau^*_{fm}}, \qquad [46]$$

and

$$\begin{aligned}\Delta_x^{f\times m} = &-\frac{\left(\tilde{f}^*_x\right)^2 \tau^*_{fm}\,\tau^*_{mf} + \tilde{f}^*_x\left(\tau^*_{fm,x}\,\tau^*_{mf} + \tau^*_{fm}\,\tau^*_{mf,\hat{x}}\right)}{\left(1+\tau^*_{ff}\right)\left(1+\tau^*_{mm}\right) - \tau^*_{mf}\,\tau^*_{fm}} \\ &-\frac{\left(1+\tau^*_{ff}\right)\tau^*_{fm,x}\,\tau^*_{mf,\hat{x}} - \tau^*_{ff,x}\,\tau^*_{fm}\,\tau^*_{mf,\hat{x}} - \tau^*_{ff,\hat{x}}\,\tau^*_{fm,x}\,\tau^*_{mf}}{\left(1+\tau^*_{ff}\right)\left(1+\tau^*_{mm}\right) - \tau^*_{mf}\,\tau^*_{fm}},\end{aligned} \qquad [47]$$

which both contain weighted terms representing direct effects on the time fraction spent on female-female and female-male interactions, respectively, as well as terms representing indirect effects mediated by changes in the overall availability of individuals for interactions (as measured by $\tilde{f}_x^*$).

The indirect ecological effects of the mating traits, measured by $\Delta_x^{B,N,Q}$, will generally be quite small, at least as long as changes in mating behavior do not strongly affect the population size, the population sex ratio, or the generation time (which is inversely proportional to d^*),

$$\Delta_x^{B,N,Q} = \omega_1 + \frac{\left(\left(1+\tau_{mm}^*\right)\omega_2 - \tau_{fm}^*\,\omega_3\right)\left(\tau_{ff,x}^* - \tilde{f}_x^*\right) + \left(\left(1+\tau_{ff}^*\right)\omega_3 - \tau_{mf}^*\,\omega_2\right)\tau_{fm,x}^*}{\left(1+\tau_{ff}^*\right)\left(1+\tau_{mm}^*\right) - \tau_{mf}^*\,\tau_{fm}^*}, \qquad [48]$$

where

$$\begin{aligned}
\omega_1 &= \tilde{f}_x^*\left(\frac{-Q_{\hat{x}}^*}{1-Q^*} + \frac{N_{\hat{x}}^*}{N^*}\right) + \xi_x^*\left(\xi_\rho^* + \xi_{\hat{\rho}}^*\right)\left(d_N^*\,\frac{N_{\hat{x}}^*}{N^*} + d_B^*\,\frac{B_{\hat{x}}^*}{B^*}\right),\\
\omega_2 &= \frac{-Q_{\hat{x}}^*}{1-Q^*} + \frac{N_{\hat{x}}^*}{N^*} + \left(\tilde{f}_\rho^* + \tilde{f}_{\hat{\rho}}^*\right)\left(d_N^*\,\frac{N_{\hat{x}}^*}{N^*} + d_B^*\,\frac{B_{\hat{x}}^*}{B^*}\right), \qquad [49]\\
\omega_3 &= \frac{Q_{\hat{x}}^*}{Q^*} + \frac{N_{\hat{x}}^*}{N^*} + \left(\tilde{m}_\rho^* + \tilde{m}_{\hat{\rho}}^*\right)\left(d_N^*\,\frac{N_{\hat{x}}^*}{N^*} + d_B^*\,\frac{B_{\hat{x}}^*}{B^*}\right).
\end{aligned}$$

The coefficients ω_1, ω_2, and ω_3 measure the relative change in, respectively, the offspring production rate, the number of available females, and the number of available males, due to a change of the sex ratio, a change of the population size, or a change of the mortality rate and its effects on the distribution of individuals over mating states.

PATHWAYS OF ADAPTIVE SPECIATION BY SEXUAL SELECTION

The comprehensive analysis of basic mating processes in the preceding section now allows us to draw general conclusions about the feasibility of evolutionary branching through sexual selection in various classes of models.

CONDITIONS FOR EVOLUTIONARY BRANCHING

It follows from inequalities [42] and [44] that evolutionary branching of the female mating trait will only occur when

$$\Delta_x^{f\times f} + \Delta_x^{f\times m} + \Delta_x^{B,N,Q} > H_x > 0\,. \qquad [50]$$

Analogously (Appendix D), evolutionary branching of the male mating trait requires that

$$\Delta_y^{m\times f} + \Delta_y^{m\times m} + \Delta_y^{B,N,Q} > H_y > 0\,. \qquad [51]$$

Speciation through simultaneous evolutionary branching of the male and female mating traits therefore requires sufficiently strong disruptive selection on each of the mating traits, in order to overcome stabilizing natural selection, i.e., $H_x > 0$, $H_y > 0$. Without sufficiently strong disruptive selection, variation that arises in the population will not be maintained, and splitting will not occur. Of equal importance, however, is the requirement that the frequency-dependent components of selection ($\Delta_x^{f\times f}, \Delta_y^{m\times f}$, and so on) are sufficiently large, such that the population is actually driven towards a fitness minimum at which it experiences disruptive selection. Without sufficiently strong frequency-dependent selection, convergence to such minima is not ensured, allowing populations to escape from a minimum's neighborhood without splitting.

EFFECTS OF INTERACTION STATES ON CONDITIONS FOR EVOLUTIONARY BRANCHING

The decomposition of conditions [50] and [51] into separate components for the effects of intra-sexual interactions ($\Delta_x^{f\times f}$ and $\Delta_y^{m\times m}$) and inter-sexual interactions ($\Delta_x^{f\times m}$ and $\Delta_y^{m\times f}$) in the mating process, as well as for indirect ecological effects ($\Delta_x^{B,N,Q}$ and $\Delta_y^{B,N,Q}$), allows us to conveniently exploit the direct link between the structure of a mating process and the values of these coefficients. For example, if the mating process under consideration does not contain female-female interactions (more precisely, if there is no female-female interaction state; Appendix A), females will spend no time on interactions with other females, and hence $\tau_{f\times f}(x,\hat{x},\rho,\hat{\rho}) \equiv 0$. As can be seen from [46], this also implies that $\Delta_x^{f\times f}$ vanishes. In other words,

$$\text{no female-female interaction state} \Rightarrow \tau_{f\times f}(x,\hat{x},\rho,\hat{\rho}) \equiv 0 \Rightarrow \Delta_x^{f\times f} = 0. \qquad [52]$$

An analogous reasoning holds for male-male interactions,

$$\text{no male-male interaction state} \Rightarrow \tau_{m\times m}(y,\hat{y},\rho,\hat{\rho}) \equiv 0 \Rightarrow \Delta_y^{m\times m} = 0. \qquad [53]$$

For inter-sexual interactions, it is easy to see that

$$\begin{gathered} \text{no female-male interaction state} \Rightarrow \tau_{f\times m}(x,\hat{y},\rho,\hat{\rho}) \equiv 0, \\ \text{no male-female interaction state} \Rightarrow \tau_{m\times f}(\hat{x},y,\rho,\hat{\rho}) \equiv 0, \\ \tau_{f\times m}(x,\hat{y},\rho,\hat{\rho}) \equiv 0 \text{ or } \tau_{m\times f}(\hat{x},y,\rho,\hat{\rho}) \equiv 0 \Rightarrow \Delta_x^{f\times m} = \Delta_y^{m\times f} = 0. \end{gathered} \qquad [54]$$

In other words, inter-sexual selection can only give rise to evolutionary branching when the mating process contains a female-male and a male-female interaction state. We stress that from a general biological perspective a mating process need not necessarily contain a male-female interaction state if it contains a female-male interaction state, and vice versa. The example of species D may serve to illustrate this point. Although it is true that species-D males interact with females by mating, the interaction with females has no effect whatsoever on the availability of males, since the time males need for mating is assumed to be negligible. Females of species D, by contrast, spend time providing parental care after mating. Inter-sexual interactions are therefore time-consuming for females but not for males. Consequently, the mating process of species D contains a female-male interaction state, but it does not contain a male-female interaction state.

Finally, it is easy to see that

$$\begin{aligned}&\text{no female-female interaction state} \Rightarrow\\ &\tau_{f\times f}(x,\hat{x},\rho,\hat{\rho}) \equiv \tau_{f\times m}(x,\hat{y},\rho,\hat{\rho}) \equiv 0 \Rightarrow\\ &\Delta_x^{B,N,Q} = \left(\xi_x^* \xi_\rho^* - \tau_{f,x}^* \tau_{f,\rho}^*\right)\left(d_N^* \frac{N_{\hat{x}}^*}{N^*} + d_B^* \frac{B_{\hat{x}}^*}{B^*}\right),\end{aligned} \qquad [55]$$

and

$$\begin{aligned}&\text{no male-male interaction state} \Rightarrow\\ &\tau_{m\times m}(y,\hat{y},\rho,\hat{\rho}) \equiv \tau_{m\times f}(y,\hat{x},\rho,\hat{\rho}) \equiv 0 \Rightarrow\\ &\Delta_y^{B,N,Q} = \left(\xi_y^* \xi_\rho^* - \tau_{m,y}^* \tau_{m,\rho}^*\right)\left(d_N^* \frac{N_{\hat{y}}^*}{N^*} + d_B^* \frac{B_{\hat{y}}^*}{B^*}\right).\end{aligned} \qquad [56]$$

Although conditions [50] and [51] indicate that speciation may occur along several different pathways, it is clear from inferences [52]-[56] that the structure of the mating process (more precisely, the types of interaction states in the mating process) will strongly delimit the range of possibilities. Let us therefore now discuss several model types within the class of basic mating processes that qualitatively differ in the feasibility of speciation.

STRONGLY ASYMMETRIC MATING PROCESSES

We start by discussing models with a strong asymmetry between the potential reproductive rates of males and females. The asymmetry in potential reproductive rates of the two sexes is widely recognized as the raison d'être of sexual selection itself (Clutton-Brock & Vincent, 1991; Andersson, 1994), and is habitually incorporated in sexual selection models. In particular, it is often assumed that females cannot produce an arbitrarily large number of offspring, due to the fact that females are limited by time or energy constraints. Males, on the other hand, are habitually assumed not to be affected by such constraints. Male reproductive success is therefore taken to be limited only by the availability of females, implying that males may potentially father an unlimited number of offspring and that male investment into the production of a single offspring must be small. In the context of our model, this implies that $\tau_{m\times f}$ is small. If males, moreover, also do not spend time on direct interactions with other males ($\tau_{m\times m} \equiv 0$), but only compete for females indirectly through female choice, we can immediately see from [53]-[55] that the scope for adaptive sympatric speciation is minimal in this class of models. In particular, inequality (51) shows that if $\Delta_y^{m\times f}$ is small with respect to other terms and if $\Delta_y^{m\times m} = 0$, speciation can only occur when

$$\left(\xi_y^* \xi_\rho^* - \tau_{m,y}^* \tau_{m,\rho}^*\right)\left(d_N^* \frac{N_{\hat{y}}^*}{N^*} + d_B^* \frac{B_{\hat{y}}^*}{B^*}\right) > H_y > 0. \qquad [57]$$

Conflicting with the notion that the mating traits be ecologically neutral, speciation therefore requires that the male mating trait directly affects the population size or the offspring production rate ($N_{\hat{y}}^* \neq 0$ or $B_{\hat{y}}^* \neq 0$). In addition, the latter quantities must also affect the per capita death rates of adults ($d_N^* \neq 0$, $d_B^* \neq 0$), and hence, the generation time. Furthermore, the mortality rate must also influence the distribution of individuals over mating states ($\xi_\rho^* \neq 0$, $\tau_{m,\rho}^* \neq 0$), implying that the mating process must occur on the same timescale as birth and mortality (as, for example, in semelparous species).

Together, these conditions are highly restrictive and take us far outside the realm of (verbal) models of speciation by sexual selection. Indeed one has to conclude that, in this case, speciation can merely 'accidentally' involve mating traits under sexual selection, since speciation must be primarily driven by the ecological side effects of the mating traits, and thus by natural selection rather than by sexual selection.

Also when additional frequency-dependence is generated by interactions between males, that is, when $\tau_{m\times m} \neq 0$, these problems are not solved. Although the condition for evolutionary branching of the male mating trait in inequality [51] can now be satisfied under a more plausible range of conditions (since $\Delta_y^{m\times m} \neq 0$), difficulties remain when we consider the condition for evolutionary branching of the female mating trait in inequality (50). Without female-female interactions contributing to frequency dependence, female mating trait branching can only occur when

$$\Delta_x^{B,N,Q} > H_x > 0 . \qquad [58]$$

That is, considerable effects of the female mating trait on population size, adult sex ratio, or offspring production rate have to be assumed. In most cases, however, such effects can be ignored, particularly when the female mating trait has no ecological significance. We must therefore conclude that female mating trait branching in models with a strong asymmetry in sex roles requires additional frequency dependence generated by female-female interactions, such that $\Delta_x^{f\times f} > H_x > 0$.

The above considerations imply that adaptive sympatric speciation requires frequency-dependent intra-sexual interactions in both sexes simultaneously whenever there is a large difference between the potential reproductive rates of males and females. This conclusion holds, irrespective of the structure of the underlying mating process, as long as we restrict ourselves to cases where sexual selection rather than ecological effects of the mating traits drive speciation.

More symmetric mating processes

Let us now shift attention to mating processes in which the difference between the potential reproductive rates of males and females is small. In such cases, also intersexual selection contributes to the frequency dependence required for speciation. Specifically, when both males and females spend a considerable fraction of their time on interactions with individuals of the other sex, the coefficients $\Delta_x^{f\times m}$ and $\Delta_y^{m\times f}$ are not negligible. Since the magnitude of $\Delta_x^{f\times m}$ and $\Delta_y^{m\times f}$ scales with $\tau_{fm}^* \tau_{mf}^*$, the impact of inter-sexual selection is largest when the fractions of time spent on non-interactive processes (τ_f^* and τ_m^*) and intra-sexual interactions (τ_{ff}^* and τ_{mm}^*)

are small relative to the fractions of time spent on inter-sexual interactions (τ^*_{fm} and τ^*_{mf}). Under these ideal conditions,

$$\begin{aligned} \Delta_x^{f\times m} &\propto \left(\tau^*_{fm} - \frac{\tau^*_{mf,\hat{x}} \big/ \tau^*_{mf}}{\tau^*_{fm,x} \big/ \tau^*_{fm}} \right), \\ \Delta_y^{m\times f} &\propto \left(\tau^*_{mf} - \frac{\tau^*_{fm,\hat{y}} \big/ \tau^*_{fm}}{\tau^*_{mf,y} \big/ \tau^*_{mf}} \right), \end{aligned} \qquad [59]$$

illustrating that the effect of inter-sexual selection on the potential for speciation is not necessarily positive (note that, by definition, $0 \le \tau^*_{fm} \le 1,\ 0 \le \tau^*_{mf} \le 1$). Specifically, inter-sexual selection decreases the potential for speciation when the relative increase in time spent on inter-sexual interactions is equal for both females and males,

$$\frac{\tau^*_{mf,\hat{x}} \big/ \tau^*_{mf}}{\tau^*_{fm,x} \big/ \tau^*_{fm}} = \frac{\tau^*_{fm,\hat{y}} \big/ \tau^*_{fm}}{\tau^*_{mf,y} \big/ \tau^*_{mf}} = 1 . \qquad [60]$$

Such is the case in many models, including typical mate-choice models, where the mating traits influence only the rate at which individuals engage in interactions (determined by, e.g., the mating probability) but not the relative time costs of interactions for males and females (e.g., the time spent on parental care).

The positive effect of inter-sexual selection on the potential for speciation is largest when an increase in relative costs for females is associated with a decrease of the relative costs for males, and vice versa, that is, when

$$\frac{\tau^*_{mf,\hat{x}} \big/ \tau^*_{mf}}{\tau^*_{fm,x} \big/ \tau^*_{fm}} < 0 , \quad \frac{\tau^*_{fm,\hat{y}} \big/ \tau^*_{fm}}{\tau^*_{mf,y} \big/ \tau^*_{mf}} < 0 . \qquad [61]$$

Such is the case when the interaction between males and females is antagonistic and characterized by sexual conflict.

TWO SPECIATION PATHWAYS

Based on the results above, we conclude that there are two qualitatively different pathways along which sexual selection may drive adaptive speciation through evolutionary branching of mating traits. Speciation can be driven either by intrasexual selection or by inter-sexual selection. Necessary conditions for the intrasexual selection route are that both females and males spend a considerable fraction of their time on interactions with individuals of their own sex,

$$\tau_{f\times f}(x,\hat{x}) \not\equiv 0 \quad \text{and} \quad \tau_{m\times m}(y,\hat{y}) \not\equiv 0 . \qquad [62]$$

In addition, the intra-sexual interactions must be such that

$$\Delta_x^{f\times f} > H_x > 0 \quad \text{and} \quad \Delta_y^{m\times m} > H_y > 0 . \qquad [63]$$

Necessary conditions for the inter-sexual selection route are that both females and males spend a considerable fraction of their time on interactions with individuals of the opposite sex. Consequently, the potential reproductive rates of males and females should not be too different, and, at the very least,

$$\tau_{f\times m}(x,\hat{y}) \not\equiv 0 \quad \text{and} \quad \tau_{m\times f}(\hat{x},y) \not\equiv 0\,. \qquad [64]$$

In addition, the inter-sexual interactions must be such that

$$\Delta_x^{f\times m} > H_x > 0 \quad \text{and} \quad \Delta_y^{m\times f} > H_y > 0\,. \qquad [65]$$

The latter conditions are most easily satisfied when the inter-sexual interactions are antagonistic.

Apart from their role in sexual selection, the mating traits may also have an ecological role, resulting from effects on a population's abundance, sex ratio, or generation time. In typical verbal and formal models of speciation by sexual selection, these indirect effects are neglected, since the mating traits are assumed to be ecologically neutral. However, beyond the scope of these standard models, one can think of biological conditions under which the ecological effects of mating traits are significant and capable of driving speciation. In such cases, speciation does not occur through sexual selection but through natural selection and merely 'accidentally' involves traits that also play a role in sexual selection. Although this illustrates an interesting link between sexual selection and ecological models of speciation, it is recommendable to clearly separate such an ecological route to speciation from the two pathways characterized above, in which speciation is driven by sexual selection itself.

BEYOND BASIC MATING PROCESSES

Even though the comprehensive analysis of basic mating processes presented above is likely to encompass a majority of models of speciation by sexual selection, it is interesting to discuss the robustness of our conclusions for models that are too complex to belong to the class of basic mating processes.

EXAMPLE E

To investigate a complex mating process outside the class of basic mating processes, let us now consider the example of species E (FIGURE 5). The key assumption here is that, after a female of species E has mated with a male, she tries to find another female that has just mated and the two females then breed cooperatively. For males of species E mating is energetically costly, so that males are not immediately available for mating again after a mating event. In species E, there are two female interaction states: females engage in interactions with males when they are in state 0, and they interact with other females when in state 1. In addition, there is one male interaction state: males interact with females when they are in state 0.

As it turns out, we now need three equations, instead of two, to describe the frequencies of individuals in the different states of the mating process. Assuming that males and females are born in state 0, these equations are given by

$$
\begin{aligned}
f_0 + f_1 &= 1 - f_1 \hat{f}_1 \left(1-\hat{Q}\right) \hat{N}\, \tau_{f_1 \times f_1}\left(x, \hat{x}, \rho_x, \hat{\rho}\right), \\
m_0 &= 1 - m_0 \hat{f}_0 \left(1-\hat{Q}\right) \hat{N}\, \tau_{m_0 \times f_0}\left(\hat{x}, y, \rho_y, \hat{\rho}\right), \qquad [66] \\
\alpha(x,\hat{y})\, f_0 \hat{m}_0 \hat{Q} \hat{N} &= \rho_x f_1 + \chi(x,\hat{x})\, f_1 \hat{f}_1 \left(1-\hat{Q}\right) \hat{N},
\end{aligned}
$$

where

$$
\tau_{f_1 \times f_1}\left(x, \hat{x}, \rho, \hat{\rho}\right) = \frac{\chi(x,\hat{x})}{\phi(x)+\rho} \quad \text{and} \quad \tau_{m_0 \times f_0}\left(\hat{x}, y, \rho, \hat{\rho}\right) = \frac{\alpha(\hat{x}, y)}{\mu(y)+\rho} \qquad [67]
$$

represent, respectively, the relative time fraction spent by females on cooperative breeding and the time fraction spent by males on mating. The first and second line in equations [66] look similar to the equations encountered previously (note the term for female-female interactions in the first equation). The third equation relates the relative frequencies f_0 and f_1 to one another. Such additional equations are necessary whenever there are multiple interaction states. They ensure that, at equilibrium, the total rate of transitions leading towards an interaction state equals the total rate of transitions leading away from that state (this so-called detailed balance is automatically ensured when there is only one interaction state).

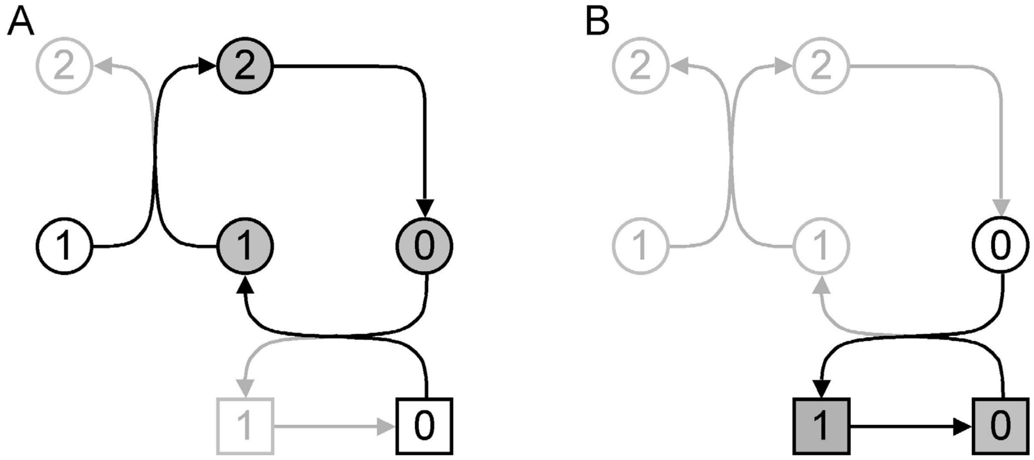


FIGURE 5 – MATING DIAGRAM FOR SPECIES E

(A) Interactions of a mutant female individual. (B) Interactions of a mutant male individual. See the main text for the biological interpretation of the individual states; see also the legend of FIGURE 1.

Although we cannot directly apply our formal results to species E, we can generalize the understanding underlying our main conclusions. Specifically, we can observe from the mating diagram (FIGURE 5) and the mating equations [66] that m_0 is dependent upon $\hat{f}_0$, since male-female interactions are time-consuming. Next, we observe that $\hat{f}_0$ varies with $\hat{m}_0$. Since $\hat{m}_0$ is a function of $\hat{y}$, also m_0 must therefore depend on $\hat{y}$, which implies that selection on the male mating trait is frequency-dependent. The same is true for selection on the female mating trait, since

f_0 is influenced by $\hat{f}_1$, which is a function of $\hat{x}$. We can therefore conclude that adaptive speciation by sexual selection is feasible for species E. In other words, one can choose the functions α, χ, ϕ, and μ in such a way that evolutionary branching of both mating traits can occur.

GENERALIZATIONS

The qualitative conditions for speciation, derived above for basic mating processes, can, in most cases, also be applied to mating processes with multiple interaction states. That is, one can rule out adaptive speciation whenever the mating process does not involve a female-male and a male-female interaction state, or a female-female and a male-male interaction state. Usually the converse also applies, but it is nevertheless possible to construct peculiar mating processes with multiple interaction states including, e.g., a male-male and a female-female interaction state, that do not allow for adaptive speciation by sexual selection. This means that in such more complex models adaptive speciation by sexual selection is ruled out even when it would be possible in corresponding models based on basic mating processes. In the sense that such special cases exist, more complex mating processes are thus even more restrictive than basic mating processes when it comes to speciation by sexual selection.

At any rate, it should be kept in mind that models with multiple interaction states will usually only apply to highly specific cases, since these models assume that the very same set of mating traits is affecting multiple interactions.

DISCUSSION

Intuitively it is easily understood why adaptive speciation through the evolutionary branching of mating traits under sexual selection requires either inter-sexual or intra-sexual selection in both sexes. Adaptive speciation, in general, originates from *frequency-dependent* disruptive selection pressures. To render sexual selection on male mating traits frequency-dependent, the fitness of male mutants has to vary with the mutant mating trait as well as with the resident mating trait. Since sexual selection can result in reproductive isolation only when both male and female mating traits become dimorphic, also female fitness has to depend upon both the mutant and the resident female mating trait. FIGURE 6 illustrates the two possible pathways along which such dependencies on resident mating traits can be realized simultaneously in both sexes. The simplest possible pathway (FIGURE 6A) involves direct interactions in both sexes between mutant and resident individuals of the same sex, corresponding to the intra-sexual selection route. For the other pathway (FIGURE 6B), corresponding to the inter-sexual selection route, frequency-dependent selection is generated indirectly, through interactions between mutant and resident individuals of the opposite sex and through inter-sexual interactions between resident individuals. Although our analytical results were derived for basic mating processes, which are a subset of all the mating processes that can be con-

constructed within our modeling framework, this fundamental explanation readily generalizes to mating processes containing multiple interaction states.

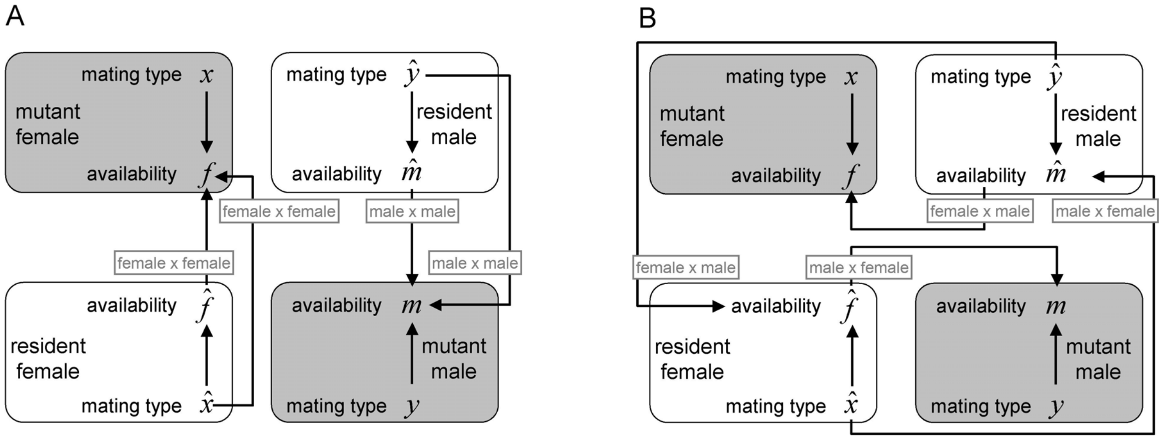


FIGURE 6 – TWO ROUTES TO SPECIATION

Adaptive speciation by sexual selection requires frequency-dependent selection on both mating traits. Frequency dependence can be generated by intra-sexual interactions in both sexes, as in (A), or by inter-sexual selection in both sexes, as in (B). (A) Female-female interactions create a direct and an indirect dependence of mutant female availability (and, hence, fitness) on the resident female mating trait. The direct dependence results from the fact that the rate at which female-female interactions are initiated depends on the mating trait of the resident female with whom the mutant interacts. The indirect dependence results from the effect the resident female mating trait has on the availability of resident females for interactions. A similar reasoning applies to frequency dependence generated by male-male interactions. (B) In the case of inter-sexual selection, female-male and male-female interactions cannot act in parallel (as do male-male and female-female interactions in intra-sexual selection), but must act in sequence, in order to generate frequency-dependence. For example, male availability will only be influenced by resident female availability if there are time-consuming male-female interactions. This, in turn, will only generate frequency-dependent selection if resident female availability is influenced by the resident male mating trait, which requires time-consuming female-male interactions.

THE QUALITATIVE CONDITIONS FOR EVOLUTIONARY BRANCHING APPLIED TO EXISTING MODELS OF SYMPATRIC SPECIATION BY SEXUAL SELECTION

For a given biological system, the derived qualitative conditions [62] and [64] for evolutionary branching through sexual selection can be evaluated directly, without the need for any quantitative analysis, simply by considering the structure of the mating process. The latter is most easily represented in terms of mating diagrams (FIGURES 1-5). If a mating diagram does not show time-consuming intra-sexual interactions in both sexes (requiring a female-female and a male-male interaction state; Appendix A), intra-sexual selection cannot cause joint evolutionary branching of mating traits in both sexes. Similarly, if a mating diagram does not include time-consuming inter-sexual interactions in both sexes (requiring a female-male and a male-female interaction state), the inter-sexual selection route to speciation through evolutionary branching is blocked. On the basis of these two criteria, we can immediately conclude that there is potential for adaptive speciation through inter-sexual selection in the example A-C introduced above (FIGURES 1-3). By contrast, speciation through evolutionary branching is precluded in example D (FIGURE 4), irrespective of all the further biological details that determine the rates of transitions in the mating process.

Surprisingly, the above qualitative criteria also suffice to rule out disruptive frequency-dependent selection as the mechanism responsible for speciation in quite a number of existing models of speciation by sexual selection (TABLE 1). This applies in particular to speciation models based on divergent Fisherian runaway processes (Wu, 1985; Turner & Burrows, 1995; Payne & Krakauer, 1997; Higashi *et al.*, 1999; Takimoto *et al.*, 2000). Classical models of the Fisherian runaway process (Lande, 1981; Kirkpatrick, 1982) assume that the production of offspring is costly for females, but inexpensive for males. Such is the case, for example, when females invest in parental care whereas males contribute only their genes to the offspring. In accordance with the classical Fisherian runaway models, also the speciation models based on Fisherian runaway assume large differences between the potential reproductive rates of males and females. As explained above, this implies that inter-sexual interactions cannot generate the frequency-dependent selection needed for evolutionary branching. In the absence of alternative sources of frequency-dependent selection (such as intra-sexual interactions or ecological processes), speciation in these models must therefore rely on non-adaptive processes.

Indeed, in several of the models listed in TABLE 1, non-adaptive processes are needed to initiate speciation. Specifically, these models do not offer a selective explanation for the origin of variation in female mating preferences. In some of the models, mutation is not included and the existence of variation in female mating preferences is simply presupposed, by assuming different female preference alleles to be present initially (Payne & Krakauer, 1997; Takimoto *et al.* 2000), or by introducing different female preference alleles in the course of the simulation (Turner & Burrows, 1995; Lande *et al.*, 2001). In other models, non-adaptive processes, such as a sudden change of environmental conditions (Higashi *et al.*, 1999), mutations with large phenotypic effects (Turner & Burrows, 1995; Lande *et al.* 2001), genetic drift (Wu, 1995), or mutation combined with weak selection (CHAPTER 3 of this thesis; Gavrilets & Waxman, 2002; Takimoto, 2002) are needed to increase the genetic variation of female mating preferences to the level required for speciation.

At first sight it might seem an irrelevant detail whether speciation is initiated by evolutionary branching or by non-adaptive processes. However, this issue is of some fundamental importance since the initiation of speciation is only one of several obstacles on the road to sympatric speciation. For instance, a population must arrive at a fitness minimum in some way, before it will experience disruptive selection. This is far from trivial, since populations tend to evolve away from a point where selection is disruptive when only non-adaptive processes are responsible for speciation. This problem reveals itself in models as a sensitive dependence on peculiar initial conditions by which the population is assumed to exhibit considerable initial variation in mating traits and to be perched exactly on a fitness minimum (as, e.g., in Higashi *et al.*, 1999; Takimoto *et al.*, 2000).

Another obstacle that must be overcome is competitive exclusion between daughter species. Non-adaptive processes cannot maintain a stable coexistence of daughter species after reproductive isolation has arisen. At best, non-adaptive speciation will therefore result in a transient phase characterized by neutral coexistence of the two daughter species (as also recognized by Turner & Burrows, 1995; Payne & Krakauer, 1997; Takimoto *et al.*, 2000). It has been argued that this need not be problematic, as long as the transient coexistence of the daughter species lasts long enough to allow for a minimal amount of ecological diversification to evolve (Wu, 1985; Turner & Burrows, 1995). However, this argument loses much of its strength for two reasons. First, the prolonged neutral coexistence observed in several models (Payne & Krakauer, 1997; Takimoto *et al.*, 2000) represents, in fact, an artifactual feature of deterministic models, which disappears as soon as only a minimal amount of stochasticity (CHAPTER 4 of this thesis) or more realistic spatial assumptions (De Cara & Dieckmann, in preparation) are incorporated in such models. Second, ecological diversification will only be able to stabilize the coexistence of daughter species if the ecological differences become correlated with the differences in mating traits that have arisen, which requires that a linkage disequilibrium develop between the traits responsible for ecological differentiation and reproductive isolation. Although the latter is often taken for granted, it is in fact far from obvious that such a linkage disequilibrium will always arise since it is counteracted by recombination. Formal models show that such linkage disequilibria can develop when populations are not too large and some symmetry breaking occurs (Dieckmann & Doebeli, 1999) or through pleiotropic ecological effects of the mating traits (Felsenstein, 1981; CHAPTER 3 of this thesis).

The above considerations indicate that adaptive speciation through evolutionary branching provides a more robust mechanism of sympatric speciation than non-adaptive processes. If speciation is initiated by evolutionary branching, both the convergence to a point where disruptive selection acts and the coexistence of daughter species after speciation are automatically ensured by frequency-dependent selection. In such cases, speciation thus proceeds as a fully adaptive process all the way from an initially monomorphic population to stably coexisting daughter species.

THE QUANTITATIVE CONDITIONS FOR SPECIATION AND THE STRUCTURAL STABILITY OF SPECIATION MODELS

If a model cannot give rise to frequency-dependent selection, one might argue that only slight modifications to the structure of the model would suffice to allow for adaptive speciation. For example, we explained how several problematic model features arise from the assumption that males are capable of producing an unlimited number of offspring. If one thus assumed that males lose some amount of time, however small, in their interactions with females, they would no longer be available for mating all of the time: this would introduce a time-consuming male-female interaction, giving rise to frequency-dependent inter-sexual selection in males. Based on the qualitative conditions [62] and [64] for speciation, one might then expect that adaptive speciation would become attainable under these slightly modified assumptions. However, this reasoning overlooks that one still has to check the quantitative conditions [63] and [65] for speciation. Only the latter guarantee that sexual selection is *sufficiently* disruptive, and that the frequency dependence is *sufficiently* strong, such that the evolving population will indeed gradually converge to a point at which it experiences disruptive selection.

A closer inspection of these quantitative conditions for speciation reveals that small modifications of the model structure will generally also have small frequency-dependent fitness effects. Accordingly, sufficiently strong inter-sexual frequency-dependence will only be generated if one is willing to deviate considerably from the typical sex role assumptions made in most current models (e.g., Wu, 1985; Turner & Burrows, 1995; Payne & Krakauer, 1997; Higashi *et al.*, 1999; Takimoto *et al.*, 2000; Takimoto, 2002). Similarly, modifying these models by assuming intra-sexual selection in both sexes, such that condition [62] is satisfied, will not affect the outcome, unless the time fractions spent on intra-sexual interactions are considerable. From this we conclude that existing speciation models are structurally stable, in the sense that small modifications to the structure and assumptions of these models will not qualitatively affect predictions. Consequently, small modifications to the models will not resolve the problematic features discussed above.

ALTERNATIVE MECHANISMS OF ADAPTIVE SPECIATION: LIMITATIONS OF OUR APPROACH

Simultaneous evolutionary branching of mating traits is not the only mechanism by which adaptive speciation might proceed. Several models listed in TABLE 1 (Lande *et al.*, 2001; CHAPTER 3 of this thesis; Gavrilets & Waxman, 2002; Takimoto, 2002) provide examples of what we suggest to refer to as semi-adaptive speciation. In these models, fully adaptive speciation cannot happen, since the qualitative conditions for simultaneous evolutionary branching in both sexes are not satisfied. The initial variation required for speciation can therefore not arise through an adaptive process involving the gradual divergence of a polymorphism through rare mutations with small phenotypic effects and subsequent trait substitutions,

TABLE 1 – MODELS OF ADAPTIVE AND NON-ADAPTIVE SYMPATRIC SPECIATION BY SEXUAL SELECTION

Model	Qualitative conditions satisfied?	Origin of variation	Maintenance of variation	Remarks
Wu (1985)	no	mutation-drift equilibrium	transient coexistence, mutation-drift equilibrium	no cost of female choosiness
Turner & Burrows (1995)	no	mutation with large phenotypic effect	transient coexistence	
Payne & Krakauer (1997)	no	initial conditions	neutral coexistence	loss of neutral coexistence when considering 2D space
Higashi *et al.* (1999), Takimoto *et al.* (2000)	no	initial conditions (external event)	transient coexistence	sensitive dependence on initial conditions
Lande *et al.* (2001)	n.a. (>2 mating traits)	mutations with large phenotypic effects	transient or stable coexistence maintained by frequency-dependent selection	multiple speciation mechanisms involving sex-reversal
CHAPTER 3 of this thesis	no	mutation and weak selection	stable coexistence, frequency-dependent selection	pleiotropic ecological effects of mating traits required
Gavrilets & Waxman (2002)	no	mutation and weak selection	stable coexistence, frequency-dependent selection	single-locus haploid genetics only
Takimoto (2002)	no	mutation and weak selection	stable coexistence, frequency-dependent selection	single-locus haploid genetics only
Almeida & Vistulo de Abreu (2003)	yes	evolutionary branching	stable coexistence, frequency-dependent selection	speciation through intersexual selection
CHAPTER 4 of this thesis	yes	evolutionary branching	stable coexistence, frequency-dependent selection	speciation through intrasexual selection

but must instead be created by non-adaptive processes. In semi-adaptive speciation models, speciation is initiated by, for example, the establishment of a mutant with a phenotype substantially different from that of the resident, or by the accumulation of mutations with small phenotypic effects, which requires weak selection and high mutation rates. Once initial genetic variation has arisen, frequency-dependent selection is generated and can subsequently maintain the stable coexistence of daughter species.

Processes of semi-adaptive speciation illustrate that a lack of potential for evolutionary branching (indicating the lack of frequency-dependent disruptive selection in a monomorphic population) does not imply that frequency-dependent disruptive selection cannot be generated later on during the speciation process in an already polymorphic population. The mathematical reason for this is obvious. The conditions for evolutionary branching are based on the local properties of the invasion fitness function (which implies that they cannot be used to predict the fate of mutant phenotypes that differ considerably from the resident phenotype) and derived under the assumption of mutation-limited evolution (which implies that they cannot be used if mutations can sustain a considerable level of variation in the resident population).

Does this imply that the theory developed in this paper has only a very limited applicability, since, under all practical biological conditions, evolution (at least of single traits) is not mutation-limited and mutations do not necessarily have only small phenotypic effects? We are confident that the answer to this question is negative and that, conversely, the occurrence of semi-adaptive speciation will be restricted to rather specific biological circumstances. As far as the applicability of adaptive dynamics theory is concerned, this opinion is substantiated by mathematical arguments (e.g., Dieckmann & Law, 1996; Metz *et al.*, 1996; Geritz *et al.*, 2002) and individual-based simulations (e.g., Dieckmann *et al.*, 1995; CHAPTER 4 of this thesis), which illustrate that the idealized process of adaptive change through mutations with small effects and subsequent trait substitutions under mutation-limited evolution is a valid and usually rather accurate limit for the evolutionary dynamics in a polymorphic population. Our further claim, concerning the limited biological relevance of semi-adaptive speciation, rests on the observation that mutation can only produce the amount of variation required to trigger speciation when selection (typically, selection on the female mating trait) is very weak, that is, of the order of the mutation rate (CHAPTER 3 of this thesis; Gavrilets & Waxman, 2002; Takimoto, 2002). Although selection on female mating traits has been shown to be very weak in specific natural systems (e.g., female gamete recognition proteins, Swanson & Vacquier, 1998), it is widely recognized that selection on female mating traits cannot be neglected in the context of sexual selection.

The requirement of weak selection will be even more constraining when mating traits are polygenic characters, rather than coded by a single haploid locus (as in Gavrilets & Waxman, 2002; Takimoto, 2002). In that case, recombination would quickly collapse discrete polymorphisms of mating traits, which could initiate speciation much more easily, to unimodal distributions. For this reason, we cannot consider speciation by sequential evolutionary branching as a possible, fully adaptive alternative to speciation by simultaneous evolutionary branching of the mating traits. Sequential evolutionary branching would occur if branching of one of the mating traits were possible only after the other trait has undergone branching and has diversified sufficiently, much like in semi-adaptive speciation, where disruptive frequency-dependent selection is only generated in a sufficiently polymorphic population (examples of models in which sequential evolutionary branching occurs as a result of natural or sexual selection, include, respectively, Doebeli & Dieckmann, 2000; and CHAPTER 4 of this thesis). For the same reasons as explained above for semi-adaptive speciation, the relevance of speciation by sequential evolutionary branching would seem to be limited to cases where disruptive selection is quite strong and where the mating traits are genetically coded by a small number of loci.

Notwithstanding the above, our present study would certainly benefit from further generalizations. Unfortunately, however, there seems to be only limited analytical scope for arriving at such a more general theory. Some progress could presumably be made in dealing with conditions for sequential evolutionary branching or semi-adaptive speciation, at least when one restricts attention to highly idealized genetic systems (see, e.g., CHAPTER 3 & 4 of this thesis). However, a general theoretical framework that is capable of dealing, at the same time, with the global properties of a fitness function and with the dynamical aspects of evolution is not available at present. Apart from this, we also see scope for extending the analysis presented here to mating processes with continuous, rather than discrete, mating states, to multiple mating traits per sex, and to evolutionary characters that are not (or at least not fully) sex-linked in their expression.

THE POTENTIAL FOR SYMPATRIC SPECIATION UNDER NATURAL CONDITIONS

Given the problematic features of non-adaptive models of sympatric speciation, it would seem that the occurrence of non-adaptive speciation might be restricted to rather specific biological circumstances. However, similar limitations apply to adaptive models of speciation by sexual selection, if only for a different reason. Adaptive speciation models certainly improve on the non-adaptive models in terms of the robustness of the speciation process, particularly when it comes to the sensitivity to initial conditions and to the coexistence of daughter species after speciation. However, the increased robustness is associated with more specific requirements for the underlying selective processes.

Speciation by intra-sexual selection will thus be restricted to rather particular biological conditions, since it requires the simultaneous action of multiple selective processes acting on the same set of mating traits. First, male-male competition and female-female competition are needed to generate stable polymorphisms of mating traits. However, due to the widely recognized genetic constraints imposed by recombination, speciation requires more than the evolution of a protected polymorphism: to achieve reproductive isolation, also assortative mating has to evolve. This implies that the considered mating traits do not only have to be involved in intra-sexual interactions, but also in the inter-sexual interactions underlying mate choice . CHAPTER 4 of this thesis, presents an example of robust adaptive speciation by sexual selection based on such assumptions. In general, however, the double requirement highlighted here would seem to severely compromise the general applicability of this route to speciation.

Similarly, speciation by inter-sexual selection requires specific conditions. Most importantly, both males and females have to invest a significant amount of time in the production of offspring, and the interaction between males and females has to be antagonistic. Speciation by inter-sexual selection might therefore be a relevant mechanism for monogamous species with strong sexual conflict (see Almeida & Vistulo de Abreu, 2003, for an example).

As yet, frequency-dependent selection, and the selective restrictions on speciation in general, have only received scant attention in theoretical models of sympatric speciation by sexual selection. Unfortunately, the same is true also for the corresponding empirical studies. The analysis presented here has identified a number of candidate mechanisms (male-male competition, female-female competition, sexual conflict) that can generate the frequency-dependent disruptive selection required for sympatric speciation by sexual selection. This naturally leads to the empirical challenge of evaluating the extent to which these mechanisms contribute to the frequency-dependent selection pressures operating in natural systems. Once this question will have been addressed for a sufficiently broad suit of systems, it will become possible to assess the relevance of sympatric speciation by sexual selection as a mechanism for generating biological diversity.

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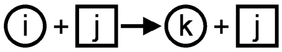
APPENDIX A — INTERACTION STATES

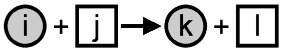
The examples of species A-D, discussed in the main text, illustrate that the complexity of the feedback between mating process, population dynamics and evolution is determined by the types of interaction in a model, and not necessarily by the complexity of a mating process in itself. It is therefore natural to characterize mating systems by the types of interactions (female-male, male-female, female-female, or male-male) they consist of. However, in such a classification care must be exercised not to include interactions in the mating process that do not affect an individual's time budget. For example, the interaction



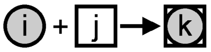
is irrelevant for the time budget of the male, since the male does not change his state.

To exclude irrelevant interactions, we introduce the concept of interaction states. We distinguish female-male, male-female, female-female, and male-male interaction states and use the following definitions (gray-filled symbols are used for indicating the focal individual):

1 – A female state i is a female-male interaction state, if there exists either a female-male interaction in the mating process

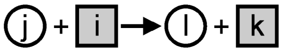


such that $i \neq k$,
or a pair formation reaction

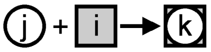


.

2 – A male state i is a male-female interaction state, if there exists either a male-female interaction in the mating process

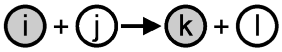


such that $i \neq k$,
or a pair formation reaction



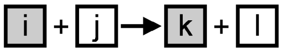
.

3 – A female state i is a female-female interaction state, if there exists a female-female interaction in the mating process



such that $i \neq k$.

4 – A male state i is a male-male interaction state, if there exists a male-male interaction in the mating process



such that $i \neq k$.

Furthermore, we collectively refer to states that are either female-female interaction states, or female-male interaction states, or both, as female interaction states. Similarly, a male interaction state is a state that is a male-female interaction state, a male-male interaction state, or both.

The above definitions allow us to measure the complexity of mating processes in terms of the number of interaction states. For example, the mating processes in the examples of species A-C contain one female-male and one male-female interaction state. By contrast, the mating process of species D contains a female-male interaction state and a male-male interaction state. Note that, in species D, males do interact with females, but this interaction does not affect the male's time budget. Therefore, there is no male-female interaction state in species D.

APPENDIX B — TIME COSTS AND OFFSPRING PRODUCTION RATES FOR SPECIES A-D

SPECIES A

$$\begin{aligned}
&\tau_{f\times m}(x,\hat{y},\rho,\hat{\rho})=\frac{\gamma(x,\hat{y})}{\eta(x,\hat{y})+\rho+\hat{\rho}}\left(1+\frac{\eta(x,\hat{y})}{\varphi(x)+\rho}\right),\\
&\tau_{m\times f}(\hat{x},y,\rho,\hat{\rho})=\frac{\gamma(\hat{x},y)}{\eta(\hat{x},y)+\rho+\hat{\rho}},\\
&\xi(x,y,\rho,\hat{\rho})=\beta(x,y)\frac{\gamma(x,y)\eta(x,y)}{\eta(x,y)+\rho+\hat{\rho}}.
\end{aligned}\tag{B-1}$$

SPECIES B

$$\begin{aligned}
&\tau_{f\times m}(x,\hat{y},\rho,\hat{\rho})=\frac{\alpha(x,\hat{y})}{\varphi_{10}(x)+\rho}+\\
&\frac{\gamma(x,\hat{y})}{\psi(x,\hat{y})+\rho+\hat{\rho}}\left(1+\frac{\psi(x,\hat{y})}{\eta(x,\hat{y})+\rho+\hat{\rho}}\left(1+\frac{\eta(x,\hat{y})}{\varphi_{20}(x)+\varphi_{23}(x)+\rho}\left(1+\frac{\varphi_{23}(x)}{\varphi_{30}(x)+\rho}\right)\right)\right),\\
&\tau_{m\times f}(\hat{x},y,\rho,\hat{\rho})=\frac{\gamma(\hat{x},y)}{\psi(\hat{x},y)+\rho+\hat{\rho}}\left(1+\frac{\psi(\hat{x},y)}{\eta(\hat{x},y)+\rho+\hat{\rho}}\left(1+\frac{\eta(\hat{x},y)}{\mu(y)+\rho}\right)\right),\\
&\xi(x,y,\rho,\hat{\rho})=\left(\frac{\beta_1(x,y)\alpha(x,y)\varphi_{10}(x)}{\varphi_{10}(x)+\rho}+\frac{\beta_2(x,y)\gamma(x,y)\psi(x,y)\eta(x,y)}{(\psi(x,y)+\rho+\hat{\rho})(\eta(x,y)+\rho+\hat{\rho})}\right).
\end{aligned}\tag{B-2}$$

SPECIES C

$$\begin{aligned}
&\tau_{f}(x,\rho,\hat{\rho})=\frac{\rho+\varphi_{02}(x)}{\varphi_{20}(x)},\\
&\tau_{f\times m}(x,\hat{y},\rho,\hat{\rho})=\frac{\gamma(x,\hat{y})}{\eta(x,\hat{y})+\rho+\hat{\rho}}\left(1+\frac{\rho}{\varphi_{20}(x)}+\frac{\eta(x,\hat{y})}{\varphi_{12}(x)+\rho}\left(1+\frac{\rho}{\varphi_{20}(x)}+\frac{\varphi_{12}(x)}{\varphi_{20}(x)}\right)\right),\\
&\tau_{m\times f}(\hat{x},y,\rho,\hat{\rho})=\frac{\gamma(\hat{x},y)}{\eta(\hat{x},y)+\rho+\hat{\rho}},\\
&\xi(x,y,\rho,\hat{\rho})=\beta(x,y)\frac{\gamma(x,y)\eta(x,y)}{\eta(x,y)+\rho+\hat{\rho}}.
\end{aligned}\tag{B-3}$$

SPECIES D

$$\tau_{f\times m}(x,\hat{y},\rho,\hat{\rho})=\frac{\alpha(x,\hat{y})}{\varphi(x)+\rho},$$
$$\tau_{m\times m}(y,\hat{y},\rho,\hat{\rho})=\frac{\nu(y,\hat{y})+\rho}{\mu(y)}, \qquad [B-4]$$
$$\xi(x,y,\rho,\hat{\rho})=\beta(x,y)\alpha(x,y).$$

APPENDIX C — CONDITIONS FOR THE EVOLUTIONARY BRANCHING OF BIVARIATE MATING STRATEGIES

Here, we briefly review some aspects of adaptive dynamics theory concerning the topology of the invasion fitness function in the neighborhood of evolutionary branching points (Metz *et al.*, 1996; Geritz *et al.*, 1998). In models where the invasion fitness is dependent upon a single evolutionary character z, evolutionary branching occurs when monomorphic populations converge to a strategy z^* that is not stable against invasion by alternative types. Such a situation requires that the strategy z^* is an evolutionarily singular strategy, meaning that the fitness gradient $G(\hat{z})$ vanishes at $\hat{z}=z^*$, or

$$G(z^*)=\left.\frac{\partial\lambda(z,\hat{z})}{\partial z}\right|_{z=\hat{z}=z^*}=0. \qquad [C-1]$$

Second, the singular strategy z^* must be attainable by a series of small steps, each consisting of a mutation and trait substitution event. This implies that the fitness gradient must point towards the singular strategy, which requires

$$\left.\frac{\partial G(\hat{z})}{\partial\hat{z}}\right|_{\hat{z}=z^*}<0. \qquad [C-2]$$

Finally, the singular strategy z^* must not be stable against invasion by alternative types. Consequently, the invasion fitness function must attain a minimum with respect to the mutant strategy at $z=z^*$, that is,

$$\left.\frac{\partial^2\lambda(z,\hat{z})}{\partial z^2}\right|_{z=\hat{z}=z^*}>0. \qquad [C-3]$$

Since our model deals with the evolution of a female mating trait x and a male mating trait y, the invasion fitness is dependent on two evolutionary characters. In models with multiple evolutionary characters, the conditions for evolutionary branching are analogous to the conditions [C-1]-[C-3], but we must take into account several additional complexities. For example, the singular strategy can be evolutionarily stable with respect to mutants that differ from the resident in only one of the traits, but evolutionarily unstable with respect to mutants that differ from the resident in multiple traits simultaneously. Similarly, evolution may converge to the singular strategy when mutation remains restricted to any one of the traits, but may, nevertheless, diverge from the singular strategy when all traits are allowed to mutate simultaneously. Finally, unlike in models with a single evolutionary character, convergence stability and evolutionary instability together do not necessarily imply that stable coexistence of multiple resident types is ensured after a polymorphism arises.

Fortunately, we need only worry about the second of these three issues. This is because the expression of the mating traits x and y is sex-limited, implying that mutants will always differ phenotypically in just a single mating trait, even when they carry mutant alleles for both mating traits. As a consequence, we may derive the conditions for the evolutionary stability of the singular strategy, and the conditions for stable coexistence of multiple resident types in a polymorphic population, by considering each mating trait independently from the other mating trait. In other words, we require that each of the two mating traits can undergo evolutionary branching independently from the other mating trait.

Taken together, we arrive at seven conditions for evolutionary branching of the two mating traits. To start with, there must be a singular strategy pair - that is, a pair of mating traits (x^*,y^*) at which the fitness gradients $G_x(\hat{x},\hat{y})$ and $G_y(\hat{x},\hat{y})$ vanish. We therefore require that

$$G_x(x^*,y^*)=\left.\frac{\partial\lambda(x,\hat{y},\hat{x},\hat{y})}{\partial x}\right|_{\substack{x=\hat{x}=x^*\\ \hat{y}=y^*}}=0 \qquad \text{[C-4]}$$

and

$$G_y(x^*,y^*)=\left.\frac{\partial\lambda(\hat{x},y,\hat{x},\hat{y})}{\partial y}\right|_{\substack{\hat{x}=x^*\\ y=\hat{y}=y^*}}=0\,. \qquad \text{[C-5]}$$

Second, evolution must converge towards the singular strategy pair, implying not only that

$$\left.\frac{\partial G_x(\hat{x},\hat{y})}{\partial\hat{x}}\right|_{\substack{\hat{x}=x^*\\ \hat{y}=y^*}}<0 \qquad \text{[C-6]}$$

and

$$\left.\frac{\partial G_y(\hat{x},\hat{y})}{\partial\hat{y}}\right|_{\substack{\hat{x}=x^*\\ \hat{y}=y^*}}<0\,, \qquad \text{[C-7]}$$

but also that

$$\left(\left.\frac{\partial G_x(\hat{x},\hat{y})}{\partial\hat{y}}\right|_{\substack{\hat{x}=x^*\\ \hat{y}=y^*}}+\left.\frac{\partial G_y(\hat{x},\hat{y})}{\partial\hat{x}}\right|_{\substack{\hat{x}=x^*\\ \hat{y}=y^*}}\right)^2<4\left.\frac{\partial G_x(\hat{x},\hat{y})}{\partial\hat{x}}\right|_{\substack{\hat{x}=x^*\\ \hat{y}=y^*}}\left.\frac{\partial G_y(\hat{x},\hat{y})}{\partial\hat{y}}\right|_{\substack{\hat{x}=x^*\\ \hat{y}=y^*}}. \qquad \text{[C-8]}$$

The latter condition, together with [C-6] and [C-7], is necessary to ensure strong convergence stability (Leimar, 2001), i.e., convergence towards the singular strategy pair irrespective of the mutational variance-covariance matrix (see Leimar, 2001, for a more general condition that does not require sex-limited expression of the traits).

Finally, the singular strategy pair should be a fitness minimum with respect to both mutant mating traits, in order to ensure that the singular strategy pair can be invaded by alternative female and male mating trait mutants. Therefore, we require that

$$\left.\frac{\partial^2 \lambda(x,\hat{y},\hat{x},\hat{y})}{\partial x^2}\right|_{\substack{x=\hat{x}=x^* \\ \hat{y}=y^*}} > 0 \qquad [\text{C-9}]$$

and

$$\left.\frac{\partial^2 \lambda(\hat{x},y,\hat{x},\hat{y})}{\partial y^2}\right|_{\substack{\hat{x}=x^* \\ y=\hat{y}=y^*}} > 0\,. \qquad [\text{C-10}]$$

APPENDIX D — DETAILED ANALYSIS OF THE GENERAL FEEDBACK STRUCTURE FOR BASIC MATING PROCESSES

For basic mating processes, the invasion fitness $\lambda = (\rho_x - \hat{\rho}) + (\rho_y - \hat{\rho})$ of mating strategy (x,y) is defined through a system of six equations

$$\begin{aligned}
\rho_x &= \frac{1}{2}\left(f_0 \hat{m}_0 \hat{Q}\left(1-\hat{Q}\right)\hat{N}\frac{\sigma_f(x)}{\sigma_f(\hat{x})} b\left(\hat{B},\hat{N}\right)\xi(x,\hat{y},\rho_x,\hat{\rho}) + \hat{\rho} \right), \\
\rho_y &= \frac{1}{2}\left(\hat{f}_0 m_0 \hat{Q}\left(1-\hat{Q}\right)\hat{N}\frac{\sigma_m(y)}{\sigma_m(\hat{y})} b\left(\hat{B},\hat{N}\right)\xi(\hat{x},y,\rho_y,\hat{\rho}) + \hat{\rho} \right), \\
\hat{\rho}\,\hat{N} &= b\left(\hat{B},\hat{N}\right)\hat{B}\,, \qquad [\text{D-1}] \\
b\left(\hat{B},\hat{N}\right)\hat{B} &= d\left(\hat{B},\hat{N}\right)\hat{N}\,, \\
f &= 1 - f\,\tau_f(x,\rho_x,\hat{\rho}) - f\,\hat{m}\hat{Q}\hat{N}\,\tau_{f\times m}(x,\hat{y},\rho_x,\hat{\rho}) - f\,\hat{f}\left(1-\hat{Q}\right)\hat{N}\,\tau_{f\times f}(x,\hat{x},\rho_x,\hat{\rho})\,, \\
m &= 1 - m\,\tau_m\left(y,\rho_y,\hat{\rho}\right) - m\,\hat{f}\left(1-\hat{Q}\right)\hat{N}\,\tau_{m\times f}\left(\hat{x},y,\rho_y,\hat{\rho}\right) - m\,\hat{m}\hat{Q}\hat{N}\,\tau_{m\times m}\left(y,\hat{y},\rho_y,\hat{\rho}\right).
\end{aligned}$$

In order to facilitate the further analysis, we replace all functions that appear in the general feedback structure by suitably rescaled approximations around the singular strategy. Close to the singular strategy, we have

$$
\begin{aligned}
\frac{f(x,\hat{x},\hat{y})}{f^*} &\approx 1+f_x^*\left(x-x^*\right)+f_{\hat{x}}^*\left(\hat{x}-x^*\right)+f_{\hat{y}}^*\left(\hat{y}-y^*\right)\\
&\quad+\frac{1}{2}\left(f_{xx}^*\left(x-x^*\right)^2+f_{\hat{x}\hat{x}}^*\left(\hat{x}-x^*\right)^2+f_{\hat{y}\hat{y}}^*\left(\hat{y}-y^*\right)^2\right)\\
&\quad+f_{x\hat{x}}^*\left(x-x^*\right)\left(\hat{x}-x^*\right)+f_{x\hat{y}}^*\left(x-x^*\right)\left(\hat{y}-y^*\right)+f_{\hat{x}\hat{y}}^*\left(\hat{x}-x^*\right)\left(\hat{y}-y^*\right),\\
\frac{m(y,\hat{x},\hat{y})}{m^*} &\approx 1+m_y^*\left(y-y^*\right)+m_{\hat{x}}^*\left(\hat{x}-x^*\right)+m_{\hat{y}}^*\left(\hat{y}-y^*\right)\\
&\quad+\frac{1}{2}\left(m_{yy}^*\left(y-y^*\right)^2+m_{\hat{x}\hat{x}}^*\left(\hat{x}-x^*\right)^2+m_{\hat{y}\hat{y}}^*\left(\hat{y}-y^*\right)^2\right)\\
&\quad+m_{y\hat{x}}^*\left(y-y^*\right)\left(\hat{x}-x^*\right)+m_{y\hat{y}}^*\left(y-y^*\right)\left(\hat{y}-y^*\right)+m_{\hat{x}\hat{y}}^*\left(\hat{x}-x^*\right)\left(\hat{y}-y^*\right),
\end{aligned}
\tag{D-2}
$$

where the coefficients f_x^* etc. represent scaled derivatives evaluated at the singular strategy, for example,

$$
f_x^*=\frac{1}{f^*}\frac{\partial f(x,\hat{x},\hat{y})}{\partial x}\bigg|_{\substack{x=\hat{x}=x^*\\ \hat{y}=y^*}} .
\tag{D-3}
$$

Similarly,

$$
\begin{aligned}
\rho_x(x,\hat{x},\hat{y}) &\approx \rho^*+\rho_{x,x}^*\left(x-x^*\right)+\rho_{x,\hat{x}}^*\left(\hat{x}-x^*\right)+\rho_{x,\hat{y}}^*\left(\hat{y}-y^*\right)\\
&\quad+\frac{1}{2}\left(\rho_{x,xx}^*\left(x-x^*\right)^2+\rho_{x,\hat{x}\hat{x}}^*\left(\hat{x}-x^*\right)^2+\rho_{x,\hat{y}\hat{y}}^*\left(\hat{y}-y^*\right)^2\right)\\
&\quad+\rho_{x,x\hat{x}}^*\left(x-x^*\right)\left(\hat{x}-x^*\right)+\rho_{x,x\hat{y}}^*\left(x-x^*\right)\left(\hat{y}-y^*\right)+\rho_{x,\hat{x}\hat{y}}^*\left(\hat{x}-x^*\right)\left(\hat{y}-y^*\right),\\
\rho_y(y,\hat{x},\hat{y}) &\approx \rho^*+\rho_{y,y}^*\left(y-y^*\right)+\rho_{y,\hat{x}}^*\left(\hat{x}-x^*\right)+\rho_{y,\hat{y}}^*\left(\hat{y}-y^*\right)\\
&\quad+\frac{1}{2}\left(\rho_{y,yy}^*\left(y-y^*\right)^2+\rho_{y,\hat{x}\hat{x}}^*\left(\hat{x}-x^*\right)^2+\rho_{y,\hat{y}\hat{y}}^*\left(\hat{y}-y^*\right)^2\right)\\
&\quad+\rho_{y,y\hat{x}}^*\left(y-y^*\right)\left(\hat{x}-x^*\right)+\rho_{y,y\hat{y}}^*\left(y-y^*\right)\left(\hat{y}-y^*\right)+\rho_{y,\hat{x}\hat{y}}^*\left(\hat{x}-x^*\right)\left(\hat{y}-y^*\right)
\end{aligned}
\tag{D-4}
$$

The resident's population size $\hat{N}$ and offspring production rate $\hat{B}$, which influence the resident's per capita birth and death rates b and d, are functions of the resident mating traits only. This time we can truncate the approximation at first order, since the second-order terms turn out to be irrelevant,

$$
\begin{aligned}
\hat{N}(\hat{x},\hat{y}) &\approx N^*+N_{\hat{x}}^*\left(\hat{x}-x^*\right)+N_{\hat{y}}^*\left(\hat{y}-y^*\right),\\
\hat{B}(\hat{x},\hat{y}) &\approx B^*+B_{\hat{x}}^*\left(\hat{x}-x^*\right)+B_{\hat{y}}^*\left(\hat{y}-y^*\right),\\
b\left(\hat{B},\hat{N}\right) &\approx b^*+b_B^*\left(\hat{B}/B^*-1\right)+b_N^*\left(\hat{N}/N^*-1\right),\\
d\left(\hat{B},\hat{N}\right) &\approx d^*+d_B^*\left(\hat{B}/B^*-1\right)+d_N^*\left(\hat{N}/N^*-1\right).
\end{aligned}
\tag{D-5}
$$

Without loss of generality, we choose

$$\begin{aligned}
\sigma_f(x) &\approx \left(1-Q^*\right)\left(1+\sigma_x^*\left(x-x^*\right)+\frac{1}{2}\sigma_{xx}^*\left(x-x^*\right)^2\right), \\
\sigma_m(y) &\approx Q^*\left(1+\sigma_y^*\left(y-y^*\right)+\frac{1}{2}\sigma_{yy}^*\left(y-y^*\right)^2\right).
\end{aligned} \qquad \text{[D-6]}$$

For convenience, we also define

$$\begin{aligned}
Q_{\hat{x}}^* &= \left.\frac{\partial Q(\hat{x},\hat{y})}{\partial \hat{x}}\right|_{\substack{\hat{x}=x^* \\ \hat{y}=y^*}} = -\sigma_x^*\, Q^*\left(1-Q^*\right), \\
Q_{\hat{y}}^* &= \left.\frac{\partial Q(\hat{x},\hat{y})}{\partial \hat{y}}\right|_{\substack{\hat{x}=x^* \\ \hat{y}=y^*}} = \sigma_y^*\, Q^*\left(1-Q^*\right).
\end{aligned} \qquad \text{[D-7]}$$

The functions τ_f, $\tau_{f\times f}$, and $\tau_{f\times m}$ are rescaled and approximated around the singular strategy as follows

$$\begin{aligned}
\tau_f(x,\rho,\hat{\rho}) &\approx \frac{\tau_f^* + \tau_{f,x}^*\left(x-x^*\right)+\tau_{f,\rho}^*\left(\rho-\rho^*\right)+\tau_{f,\hat{\rho}}^*\left(\hat{\rho}-\rho^*\right)+\frac{1}{2}\tau_{f,xx}^*\left(x-x^*\right)^2}{f^*}, \\
\tau_{f\times f}(x,\hat{x},\rho,\hat{\rho}) &\approx \frac{\tau_{ff}^* + \tau_{ff,x}^*\left(x-x^*\right)+\tau_{ff,\hat{x}}^*\left(\hat{x}-x^*\right)+\tau_{ff,\rho}^*\left(\rho-\rho^*\right)+\tau_{ff,\hat{\rho}}^*\left(\hat{\rho}-\rho^*\right)}{N^*\left(1-Q^*\right)f^*f^*} \\
&\quad + \frac{\tau_{ff,xx}^*\left(x-x^*\right)^2 + 2\tau_{ff,x\hat{x}}^*\left(x-x^*\right)\left(\hat{x}-x^*\right)+\tau_{ff,\hat{x}\hat{x}}^*\left(\hat{x}-x^*\right)^2}{2N^*\left(1-Q^*\right)f^*f^*}, \\
\tau_{f\times m}(x,\hat{y},\rho,\hat{\rho}) &\approx \frac{\tau_{fm}^* + \tau_{fm,x}^*\left(x-x^*\right)+\tau_{fm,\hat{y}}^*\left(\hat{y}-y^*\right)+\tau_{fm,\rho}^*\left(\rho-\rho^*\right)+\tau_{fm,\hat{\rho}}^*\left(\hat{\rho}-\rho^*\right)}{N^*Q^*f^*m^*} \\
&\quad + \frac{\tau_{fm,xx}^*\left(x-x^*\right)^2 + 2\tau_{fm,x\hat{y}}^*\left(x-x^*\right)\left(\hat{y}-y^*\right)+\tau_{fm,\hat{y}\hat{y}}^*\left(\hat{y}-y^*\right)^2}{2N^*Q^*f^*m^*}.
\end{aligned} \qquad \text{[D-8]}$$

Note that here we omitted several second-order terms, which, again, turn out to be irrelevant. The functions τ_m, $\tau_{m\times f}$, and $\tau_{m\times m}$ are rescaled and expanded in an analogous way. The function $\xi_{f\times m}$ is rescaled, and approximated around the singular strategy such that

$$\begin{aligned}
&\xi(x,y,\rho,\hat{\rho})\frac{N^*Q^*\left(1-Q^*\right)f^*m^*}{\xi^*} \approx \\
&1+\xi_x^*\left(x-x^*\right)+\xi_y^*\left(y-y^*\right)+\xi_\rho^*\left(\rho-\rho^*\right)+\xi_{\hat{\rho}}^*\left(\hat{\rho}-\rho^*\right) \\
&+\frac{1}{2}\left(\xi_{xx}^*\left(x-x^*\right)^2+2\xi_{xy}^*\left(x-x^*\right)\left(y-y^*\right)+\xi_{yy}^*\left(y-y^*\right)^2\right).
\end{aligned} \qquad \text{[D-9]}$$

Finally, the invasion fitness function is approximated by a standard Taylor expansion around the singular strategy

$$\begin{aligned}\lambda(x,y,\hat{x},\hat{y}) \approx{}& \lambda^* + \lambda_x^*(x-x^*) + \lambda_y^*(y-y^*) + \lambda_{\hat{x}}^*(\hat{x}-x^*) + \lambda_{\hat{y}}^*(\hat{y}-y^*) \\ &+\frac{1}{2}\left(\lambda_{xx}^*(x-x^*)^2 + \lambda_{yy}^*(y-y^*)^2 + \lambda_{\hat{x}\hat{x}}^*(\hat{x}-x^*)^2 + \lambda_{\hat{y}\hat{y}}^*(\hat{y}-y^*)^2\right) \\ &+\lambda_{x\hat{x}}^*(x-x^*)(\hat{x}-x^*) + \lambda_{y\hat{y}}^*(y-y^*)(\hat{y}-y^*) \\ &+\lambda_{xy}^*(x-x^*)(y-y^*) + \lambda_{\hat{x}\hat{y}}^*(\hat{x}-x^*)(\hat{y}-y^*) \\ &+\lambda_{x\hat{y}}^*(x-x^*)(\hat{y}-y^*) + \lambda_{\hat{x}y}^*(\hat{x}-x^*)(y-y^*).\end{aligned} \quad \text{[D-10]}$$

The equations of the general feedback structure [D-1] impose constraints on the coefficients of the approximations [D-2]-[D-9]. For example, by evaluating the equations [D-1] at the singular strategy, we obtain the following set of equations,

$$\begin{aligned}\lambda^* &= 0\,, \\ \rho^* &= b^*\xi^*\,, \\ \rho^*N^* &= b^*B^* = d^*N^*\,, \\ f^* &= 1-\tau_f^*-\tau_{fm}^*-\tau_{ff}^*\,, \\ m^* &= 1-\tau_m^*-\tau_{mf}^*-\tau_{mm}^*\,,\end{aligned} \quad \text{[D-11]}$$

which imply that

$$\begin{aligned}d^* &= \rho^*\,, \\ B^* &= \xi^*N^*\,, \\ b^* &= \frac{\rho^*}{\xi^*}\,.\end{aligned} \quad \text{[D-12]}$$

In the following steps, we take derivates on the right-hand and left-hand sides of equations [D-1] and evaluate the resulting expressions at the singular strategy. When we take derivatives with respect to x, this yields

$$\begin{aligned}\lambda_x^* &= \rho_{x,x}^*\,, \\ \frac{\rho_{x,x}^*}{\rho^*} &= \frac{1}{2}f_x^* + \frac{1}{2}\left(\xi_x^* + \xi_\rho^*\,\rho_{x,x}^*\right) + \frac{1}{2}\sigma_x^*\,, \\ -f_x^* &= \left(\tau_{f,x}^* + \tau_{fm,x}^* + \tau_{ff,x}^*\right) + \rho_{x,x}^*\left(\tau_{f,\rho}^* + \tau_{fm,\rho}^* + \tau_{ff,\rho}^*\right).\end{aligned} \quad \text{[D-13]}$$

Since the fitness gradient vanishes at the singular strategy, see conditions [C-4] and [C-5], we have $\lambda_x^* = 0$. This allows us to solve equations [D-13], yielding

$$\begin{aligned}\rho_{x,x}^* &= 0\,, \\ f_x^* &= \tilde{f}_x^*\,, \\ \xi_x^* &= -\tilde{f}_x^* - \sigma_x^*\,.\end{aligned} \quad \text{[D-14]}$$

Here, and henceforth, we use $\tilde{f}_z^*$ (with z denoting either x, y, $\hat{x}$, $\hat{y}$, xx, yy, ρ, or $\hat{\rho}$) as shorthand notation for $-\tau_{f,z}^* - \tau_{fm,z}^* - \tau_{ff,z}^*$. Similarly ,$\tilde{m}_z^*$ is used as shorthand notation for $-\tau_{m,z}^* - \tau_{mf,z}^* - \tau_{mm,z}^*$.

Applying the same procedure, but this time taking derivatives with respect to y, we find,

$$\begin{aligned} \rho_{y,y}^* &= 0 \,, \\ m_y^* &= \tilde{m}_y^* \,, \\ \xi_y^* &= -\tilde{m}_y^* - \sigma_y^* \,. \end{aligned} \qquad \text{[D-15]}$$

Since $\rho_x(\hat{x},\hat{x},\hat{y}) = \rho_y(\hat{y},\hat{x},\hat{y})$, the above immediately implies that $\rho_{x,\hat{x}}^* = \rho_{y,\hat{x}}^*$ and $\rho_{x,\hat{y}}^* = \rho_{y,\hat{y}}^*$.

Equations for λ_{xx}^*, λ_{xy}^*, and λ_{yy}^*, which are needed to determine whether the singular strategy is resistant against invasion by neighboring strategies, are obtained by taking second-order derivatives on the right-hand and left-hand sides of equations [D-1] and by evaluating the resulting expressions at the singular strategy,

$$\begin{aligned} \lambda_{xx}^* &= \frac{\rho^*}{2-\rho^*\xi_\rho^*}\left(f_{xx}^* + \xi_{xx}^* + \sigma_{xx}^* - 2\left(f_x^*\right)^2 - 2f_x^*\sigma_x^* - 2\left(\sigma_x^*\right)^2\right), \\ f_{xx}^* &= \tilde{f}_{xx}^* + \lambda_{xx}^*\tilde{f}_\rho^* + 2f_x^*\tilde{f}_x^* \,, \\ \lambda_{yy}^* &= \frac{\rho^*}{2-\rho^*\xi_\rho^*}\left(m_{yy}^* + \xi_{yy}^* + \sigma_{yy}^* - 2\left(m_y^*\right)^2 - 2m_y^*\sigma_y^* - 2\left(\sigma_y^*\right)^2\right), \\ m_{yy}^* &= \tilde{m}_{yy}^* + \lambda_{yy}^*\tilde{m}_\rho^* + 2m_y^*\tilde{m}_y^* \,. \end{aligned} \qquad \text{[D-16]}$$

Due to the sex-limited expression of the mating traits, we have $\lambda_{xy} = 0$.
Equations [D-16] can be solved for λ_{xx}^* and λ_{yy}^*, and simplified using equations [D-15]. After this, we obtain

$$\begin{aligned} \lambda_{xx}^* &= \frac{\rho^*}{2-\rho^*\left(\xi_\rho^* + \tilde{f}_\rho^*\right)}\left(f_{xx}^* + \sigma_{xx}^* + \xi_{xx}^* + 2\sigma_x^*\xi_x^*\right), \\ \lambda_{yy}^* &= \frac{\rho^*}{2-\rho^*\left(\xi_\rho^* + \tilde{m}_\rho^*\right)}\left(m_{yy}^* + \sigma_{yy}^* + \xi_{yy}^* + 2\sigma_y^*\xi_y^*\right), \end{aligned} \qquad \text{[D-17]}$$

which corresponds exactly to the first part of equations [42].

The conditions for convergence stability of the singular strategy, see conditions [C-6]-[C-8], can only be evaluated after additional coefficients have been solved. To start with, we take derivatives with respect to $\hat{x}$ and $\hat{y}$ of the expressions in equations [D-1]. Results are rather intricate. The population dynamical equilibrium conditions for the resident, for example, give rise to the following equations,

$$\begin{aligned}\rho^*_{x,\hat{x}} &= d^*_B \frac{B^*_{\hat{x}}}{B^*} + d^*_N \frac{N^*_{\hat{x}}}{N^*} = \xi^* b^*_B \frac{B^*_{\hat{x}}}{B^*} + \xi^* b^*_N \frac{N^*_{\hat{x}}}{N^*} + \rho^* \frac{B^*_{\hat{x}}}{B^*} - \rho^* \frac{N^*_{\hat{x}}}{N^*}\,, \\ \rho^*_{y,\hat{y}} &= d^*_B \frac{B^*_{\hat{y}}}{B^*} + d^*_N \frac{N^*_{\hat{y}}}{N^*} = \xi^* b^*_B \frac{B^*_{\hat{y}}}{B^*} + \xi^* b^*_N \frac{N^*_{\hat{y}}}{N^*} + \rho^* \frac{B^*_{\hat{y}}}{B^*} - \rho^* \frac{N^*_{\hat{y}}}{N^*}\,.\end{aligned} \qquad \text{[D-18]}$$

The equations for ρ_x and ρ_y yield

$$\begin{aligned}\frac{\rho^*_{x,\hat{x}}}{\rho^*}\left(1-\rho^*\left(\xi^*_\rho+\xi^*_{\hat{\rho}}\right)\right) &= f^*_{\hat{x}} + m^*_{\hat{x}} - 2\sigma^*_x\left(1-Q^*\right) + \frac{b^*_B}{b^*}\frac{B^*_{\hat{x}}}{B^*} + \frac{b^*_N}{b^*}\frac{N^*_{\hat{x}}}{N^*} + \frac{N^*_{\hat{x}}}{N^*}\,, \\ \frac{\rho^*_{y,\hat{y}}}{\rho^*}\left(1-\rho^*\left(\xi^*_\rho+\xi^*_{\hat{\rho}}\right)\right) &= f^*_{\hat{y}} + m^*_{\hat{y}} - 2\sigma^*_y Q^* + \frac{b^*_B}{b^*}\frac{B^*_{\hat{y}}}{B^*} + \frac{b^*_N}{b^*}\frac{N^*_{\hat{y}}}{N^*} + \frac{N^*_{\hat{y}}}{N^*}\,.\end{aligned} \qquad \text{[D-19]}$$

Finally, we obtain four equations from the mating process equations,

$$\begin{aligned}-f^*_{\hat{x}}\left(1+\tau^*_{ff}\right) &= \rho^*_{x,\hat{x}}\left(\tau^*_{f,\rho}+\tau^*_{fm,\rho}+\tau^*_{ff,\rho}+\tau^*_{f,\hat{\rho}}+\tau^*_{fm,\hat{\rho}}+\tau^*_{ff,\hat{\rho}}\right) \\ &\quad +\tau^*_{fm}m^*_{\hat{x}}+\tau^*_{ff,\hat{x}}+\tau^*_{ff}f^*_x \\ &\quad +\left(\tau^*_{fm}+\tau^*_{ff}\right)\frac{N^*_{\hat{x}}}{N^*}-\tau^*_{fm}\sigma^*_x\left(1-Q^*\right)+\tau^*_{ff}\sigma^*_x Q^*\,, \\ -m^*_{\hat{x}}\left(1+\tau^*_{mm}\right) &= \rho^*_{x,\hat{x}}\left(\tau^*_{m,\rho}+\tau^*_{mf,\rho}+\tau^*_{mm,\rho}+\tau^*_{m,\hat{\rho}}+\tau^*_{mf,\hat{\rho}}+\tau^*_{mm,\hat{\rho}}\right) \\ &\quad +\tau^*_{mf,\hat{x}}+\tau^*_{mf}\left(f^*_x+f^*_{\hat{x}}\right) \\ &\quad +\left(\tau^*_{mf}+\tau^*_{mm}\right)\frac{N^*_{\hat{x}}}{N^*}+\tau^*_{mf}\sigma^*_x Q^*-\tau^*_{mm}\sigma^*_x\left(1-Q^*\right), \\ -f^*_{\hat{y}}\left(1+\tau^*_{ff}\right) &= \rho^*_{y,\hat{y}}\left(\tau^*_{f,\rho}+\tau^*_{fm,\rho}+\tau^*_{ff,\rho}+\tau^*_{f,\hat{\rho}}+\tau^*_{fm,\hat{\rho}}+\tau^*_{ff,\hat{\rho}}\right) \\ &\quad +\tau^*_{fm,\hat{y}}+\tau^*_{fm}\left(m^*_y+m^*_{\hat{y}}\right) \\ &\quad +\left(\tau^*_{fm}+\tau^*_{ff}\right)\frac{N^*_{\hat{y}}}{N^*}+\tau^*_{fm}\sigma^*_y\left(1-Q^*\right)-\tau^*_{ff}\sigma^*_y Q^*\,, \\ -m^*_{\hat{y}}\left(1+\tau^*_{mm}\right) &= \rho^*_{y,\hat{y}}\left(\tau^*_{m,\rho}+\tau^*_{mf,\rho}+\tau^*_{mm,\rho}+\tau^*_{m,\hat{\rho}}+\tau^*_{mf,\hat{\rho}}+\tau^*_{mm,\hat{\rho}}\right) \\ &\quad +\tau^*_{mf}f^*_{\hat{y}}+\tau^*_{mm,\hat{y}}+\tau^*_{mm}m^*_y \\ &\quad +\left(\tau^*_{mf}+\tau^*_{mm}\right)\frac{N^*_{\hat{y}}}{N^*}-\tau^*_{mf}\sigma^*_y Q^*+\tau^*_{mm}\sigma^*_y\left(1-Q^*\right).\end{aligned} \qquad \text{[D-20]}$$

Equations [D-18]-[D-20] are linear equations in their unknowns, and can therefore be solved for $f^*_{\hat{x}}$, $f^*_{\hat{y}}$, $m^*_{\hat{x}}$, $m^*_{\hat{y}}$, $\rho^*_{x,\hat{x}}$, $\rho^*_{y,\hat{y}}$, b^*_B, b^*_N, $B^*_{\hat{x}}$, and $B^*_{\hat{y}}$.

A similar procedure, involving mixed second-order derivatives with respect to x and $\hat{x}$ or y and $\hat{y}$, can be applied to find $f^*_{x\hat{x}}$, $m^*_{y\hat{y}}$, $\rho^*_{x,x\hat{x}}$, $\rho^*_{y,y\hat{y}}$, $\lambda^*_{x\hat{x}}$, and $\lambda^*_{y\hat{y}}$. In particular, we find

$$\begin{aligned}\lambda^*_{x\hat{x}} = -\tau^*_{ff,x\hat{x}} &- \frac{\left(1+\tau^*_{mm}\right)\left(\tau^*_{ff}\left(\tilde{f}^*_x\right)^2 + \left(\tau^*_{ff,x}+\tau^*_{ff,\hat{x}}\right)\tilde{f}^*_x - \tau^*_{ff,x}\tau^*_{ff,\hat{x}}\right)}{\left(1+\tau^*_{ff}\right)\left(1+\tau^*_{mm}\right)-\tau^*_{fm}\tau^*_{mf}} \\ &+ \frac{\tau^*_{fm}\tau^*_{mf}\left(\tilde{f}^*_x\right)^2 + \left(\tau^*_{mf}\tau^*_{fm,x}+\tau^*_{fm}\tau^*_{mf,\hat{x}}\right)\tilde{f}^*_x - \kappa_1}{\left(1+\tau^*_{ff}\right)\left(1+\tau^*_{mm}\right)-\tau^*_{fm}\tau^*_{mf}} \qquad \text{[D-21]} \\ -\kappa_2 &- \frac{\left(\kappa_3\left(1+\tau^*_{mm}\right)-\kappa_4\tau^*_{fm}\right)\left(\tau^*_{ff,x}-\tilde{f}^*_x\right)+\left(\kappa_4\left(1+\tau^*_{ff}\right)-\kappa_3\tau^*_{mf}\right)\tau^*_{fm,x}}{\left(1+\tau^*_{ff}\right)\left(1+\tau^*_{mm}\right)-\tau^*_{fm}\tau^*_{mf}}.\end{aligned}$$

with

$$\begin{aligned}\kappa_1 &= \tau^*_{fm}\tau^*_{ff,x}\tau^*_{mf,\hat{x}} + \tau^*_{mf}\tau^*_{fm,x}\tau^*_{ff,\hat{x}} - \left(1+\tau^*_{ff}\right)\tau^*_{fm,x}\tau^*_{mf,\hat{x}}\ , \\ \kappa_2 &= \left(\frac{-Q^*_x}{1-Q^*}+\frac{N^*_{\hat{x}}}{N^*}\right)\tilde{f}^*_x + \xi^*_x\left(\xi^*_\rho+\xi^*_{\hat{\rho}}\right)\left(d^*_B\frac{B^*_{\hat{x}}}{B^*}+d^*_N\frac{N^*_{\hat{x}}}{N^*}\right), \\ \kappa_3 &= \frac{-Q^*_x}{1-Q^*}+\frac{N^*_{\hat{x}}}{N^*}+\left(\tilde{f}^*_\rho+\tilde{f}^*_{\hat{\rho}}\right)\left(d^*_B\frac{B^*_{\hat{x}}}{B^*}+d^*_N\frac{N^*_{\hat{x}}}{N^*}\right), \qquad \text{[D-22]} \\ \kappa_4 &= \frac{Q^*_x}{Q^*}+\frac{N^*_{\hat{x}}}{N^*}+\left(\tilde{m}^*_\rho+\tilde{m}^*_{\hat{\rho}}\right)\left(d^*_B\frac{B^*_{\hat{x}}}{B^*}+d^*_N\frac{N^*_{\hat{x}}}{N^*}\right).\end{aligned}$$

As discussed in the main text, the right-hand side of equation [D-21] can be separated into three parts, corresponding to the effects of intra-sexual interactions (first line), to the effects of inter-sexual interactions (second line), and to the ecological effects of the mating traits (third line).

The expression for $\lambda^*_{y\hat{y}}$ shows a similar structure,

$$\begin{aligned}\lambda^*_{y\hat{y}} = -\tau^*_{mm,y\hat{y}} &- \frac{\left(1+\tau^*_{ff}\right)\left(\tau^*_{mm}\left(\tilde{m}^*_y\right)^2 + \left(\tau^*_{mm,y}+\tau^*_{mm,\hat{y}}\right)\tilde{m}^*_y - \tau^*_{mm,y}\tau^*_{mm,\hat{y}}\right)}{\left(1+\tau^*_{ff}\right)\left(1+\tau^*_{mm}\right)-\tau^*_{fm}\tau^*_{mf}} \\ &+ \frac{\tau^*_{fm}\tau^*_{mf}\left(\tilde{m}^*_y\right)^2 + \left(\tau^*_{fm}\tau^*_{mf,y}+\tau^*_{mf}\tau^*_{fm,\hat{y}}\right)\tilde{m}^*_y - \nu_1}{\left(1+\tau^*_{ff}\right)\left(1+\tau^*_{mm}\right)-\tau^*_{fm}\tau^*_{mf}} \qquad \text{[D-23]} \\ -\nu_2 &- \frac{\left(\nu_3\left(1+\tau^*_{ff}\right)-\nu_4\tau^*_{mf}\right)\left(\tau^*_{mm,y}-\tilde{m}^*_y\right)+\left(\nu_4\left(1+\tau^*_{mm}\right)-\nu_3\tau^*_{fm}\right)\tau^*_{mf,y}}{\left(1+\tau^*_{ff}\right)\left(1+\tau^*_{mm}\right)-\tau^*_{fm}\tau^*_{mf}}.\end{aligned}$$

with

$$
\begin{aligned}
\nu_1 &= \tau^*_{mf}\tau^*_{mm,y}\tau^*_{fm,\hat{y}} + \tau^*_{fm}\tau^*_{mf,y}\tau^*_{mm,\hat{y}} - \left(1+\tau^*_{mm}\right)\tau^*_{mf,y}\tau^*_{fm,\hat{y}} \text{ ,}\\
\nu_2 &= \left(\frac{Q^*_y}{Q^*}+\frac{N^*_{\hat{y}}}{N^*}\right)\tilde{m}^*_y + \xi^*_y\left(\xi^*_\rho+\xi^*_{\hat{\rho}}\right)\left(d^*_B\frac{B^*_{\hat{y}}}{B^*}+d^*_N\frac{N^*_{\hat{y}}}{N^*}\right),\\
\nu_3 &= \frac{Q^*_y}{Q^*}+\frac{N^*_{\hat{y}}}{N^*}+\left(\tilde{m}^*_\rho+\tilde{m}^*_{\hat{\rho}}\right)\left(d^*_B\frac{B^*_{\hat{y}}}{B^*}+d^*_N\frac{N^*_{\hat{y}}}{N^*}\right),\\
\nu_4 &= \frac{-Q^*_y}{1-Q^*}+\frac{N^*_{\hat{y}}}{N^*}+\left(\hat{f}^*_\rho+\hat{f}^*_{\hat{\rho}}\right)\left(d^*_B\frac{B^*_{\hat{y}}}{B^*}+d^*_N\frac{N^*_{\hat{y}}}{N^*}\right).
\end{aligned}
\tag{D-24}
$$

The expressions for $\lambda^*_{x\hat{x}}$ and $\lambda^*_{y\hat{y}}$ can be used to evaluate the first and second of the three conditions [C-6]-[C-8] for convergence stability of the singular strategy, since

$$
\begin{aligned}
\left.\frac{\partial G_x(\hat{x},\hat{y})}{\partial \hat{x}}\right|_{\substack{\hat{x}=x^*\\ \hat{y}=y^*}} &= \frac{\partial}{\partial \hat{x}}\left[\left.\frac{\partial \lambda(x,y,\hat{x},\hat{y})}{\partial x}\right|_{\substack{x=\hat{x}\\ x=\hat{y}}}\right]\Bigg|_{\substack{\hat{x}=x^*\\ \hat{y}=y^*}} = \lambda^*_{xx}+\lambda^*_{x\hat{x}} \text{ ,}\\
\left.\frac{\partial G_y(\hat{x},\hat{y})}{\partial \hat{y}}\right|_{\substack{\hat{x}=x^*\\ \hat{y}=y^*}} &= \frac{\partial}{\partial \hat{y}}\left[\left.\frac{\partial \lambda(x,y,\hat{x},\hat{y})}{\partial y}\right|_{\substack{x=\hat{x}\\ x=\hat{y}}}\right]\Bigg|_{\substack{\hat{x}=x^*\\ \hat{y}=y^*}} = \lambda^*_{yy}+\lambda^*_{y\hat{y}} \text{ .}
\end{aligned}
\tag{D-25}
$$

It turns out that the third condition for convergence stability, condition [C-8], can always be satisfied by choosing a suitable value for ξ^*_{xy}. Consequently, this condition is irrelevant for the arguments presented in the main text.

The long-term evolution of multi-locus traits under frequency-dependent disruptive selection

6

G. Sander van Doorn & Ulf Dieckmann

unpublished manuscript

ABSTRACT

Frequency-dependent disruptive selection is widely recognized as an important source of genetic variation. Its evolutionary consequences have been extensively studied using phenotypic approaches, based on quantitative genetics, game theory, or adaptive dynamics. However, the genetic assumptions underlying these approaches are highly idealized and, even worse, predict different consequences of frequency-dependent disruptive selection. Population genetic models, by contrast, enable genotypic approaches but traditionally assume constant fitness values. Only a minority of these models thus addresses frequency-dependent selection, and only a few of these do so in a multi-locus context. An inherent limitation of these studies is that they only investigate the short-term maintenance of genetic variation. Consequently, the long-term evolution of multi-locus characters under frequency-dependent disruptive selection remains poorly understood. We aim to bridge this gap between phenotypic and genotypic models by studying a multi-locus version of Levene's soft selection model. Individual-based simulations and deterministic approximations based on adaptive dynamics theory provide insights into the underlying evolutionary dynamics. Our analysis uncovers a general pattern of polymorphism formation and collapse, likely to apply to a wide variety of genetic systems: after convergence to a fitness minimum and the subsequent establishment of genetic polymorphism at multiple loci, genetic variation becomes increasingly concentrated, until eventually only a single polymorphic locus remains. This evolutionary process combines features observed in quantitative genetics and adaptive dynamics models, and can be explained as a consequence of changes in the selection regime that are inherent to frequency-dependent disruptive selection. Our findings demonstrate that the potential of frequency-dependent disruptive selection to maintain polygenic variation is considerably smaller than naïvely expected.

INTRODUCTION

Frequency-dependent selection plays an important role in the origin and maintenance of genetic variation (Felsenstein, 1976; Hedrick *et al.*, 1997; Slatkin, 1979). Conditions for stable polymorphisms are much relaxed when fitness values are not constant but vary with the frequency of different genotypes present in a population. Protected polymorphism can be established whenever rare genotypes have a selective advantage (Lewontin, 1958). This may even lead to situations in which, at population genetic equilibrium, the heterozygote has a fitness disadvantage (this is the exact opposite of the situation required for stable polymorphisms with constant fitness values). In such a case, the population is caught at a fitness minimum, at which it experiences disruptive selection.

The consequences of such frequency-dependent disruptive selection have most extensively been investigated in the context of quantitative genetics (e.g., Slatkin, 1979; Bulmer, 1980), and in the related frameworks of evolutionary game theory (e.g., Maynard Smith, 1982; Hofbauer & Sigmund, 1998) and adaptive dynamics (e.g., Metz *et al.*, 1996; Geritz *et al.*, 1998; Hofbauer & Sigmund, 1998). Although the insights obtained through these different approaches are similar in some respects (Taylor, 1996a), their predictions for the effects of frequency-dependent disruptive selection are strikingly different. In quantitative genetics (QG) models, the maintenance of genetic variation results from the broadening of continuous phenotype distributions exposed to such selection. In adaptive dynamics (AD) models, frequency-dependent disruptive selection can cause evolutionary branching (Metz *et al.*, 1996; Geritz *et al.*, 1998). Such branching processes characteristically involve the convergence of a monomorphic population to a fitness minimum, followed by the adaptive emergence and further diversification of a discrete polymorphism.

The discordance of these predictions is caused by the different genetic assumptions underlying QG and AD models. QG models are often purely phenomenological, but in those cases where a mechanistic underpinning is given, it is usually assumed that phenotypic characters are influenced by a large number of loci, each of which contributes only marginally to the phenotype. In every generation, the genetic variation present in the parent generation is redistributed among the offspring through recombination and segregation, i.e., as a consequence of sexual reproduction. Since many loci are involved in this process, the distribution of phenotypes in the population is continuous and Gaussian. AD models, in contrast, usually consider asexual reproduction (or single-locus, haploid genetics) and monomorphic populations (see Kisdi & Geritz, 1999; Van Dooren, 1999, for exceptions).

From the viewpoint of population genetics, the assumptions of infinite loci with infinitesimal effects (QG) or of asexual reproduction (AD) are both highly idealized. It is therefore difficult to predict the effect of disruptive frequency-dependent selection for realistic genetic settings. Despite the fact that frequency-dependent selection has been included in the theory of population genetics right from its conception (Fisher, 1930), most of population genetics theory assumes constant fitness values (see, e.g., Clark, 1972; Cockerham *et al.* 1972; Cressman, 1992 for exceptions); such theory cannot be used to predict the consequences of frequency-dependent selection. Especially the evolutionary dynamics of multi-locus characters under frequency-dependent disruptive selection remains elusive. Recently, however, several attempts have been made to bridge the gap between population genetics and phenotypic models of frequency-dependent selection, and particularly the integration of population genetics with evolutionary game theory has received considerable attention (e.g., Cressman, 1992; Hofbauer & Sigmund, 1998).

As a case in point, Bürger (2002ab) presents a population genetic analysis of a model of intraspecific competition that had previously been analyzed within both the QG (e.g., Slatkin, 1979) and the AD framework (e.g., Metz *et al.*, 1996). Bürger focuses on the dynamics and population genetic equilibria of the frequencies of a fixed set of alleles in a multi-locus model with frequency-dependent disruptive selection. He investigates the conditions under which disruptive selection on the phenotypes can be observed, and quantifies the amount of genetic variation that can be maintained. The model exhibits a number of unexpected phenomena, which underscore that the population genetics of frequency-dependent disruptive selection can be surprisingly complex.

An approach from another direction was initiated by Kisdi & Geritz (1999) and Van Dooren (1999), who extended AD models by incorporating diploid genetics and sexual reproduction. Focusing on the simplest interesting example, these authors study the evolution of alleles at a single locus under frequency-dependent disruptive selection. Unlike in standard population genetic models, they explicitly consider mutations with small phenotypic effects; long-term evolution can then proceed as a sequence of substitution steps during which existing alleles are replaced by novel ones created by mutation. As in asexual AD models, frequency-dependent disruptive selection can cause evolutionary branching in diploid sexual populations (this occurs, it is shown, under the same conditions as in asexual models), leading to the establishment of a polymorphism of alleles (Kisdi & Geritz, 1999). As a consequence of the constraints imposed by random mating and segregation, the evolution of dominance-recessivity relations between the alleles is selectively favored (Van Dooren, 1999).

In this paper, we aim to further investigate the long-term consequences of frequency-dependent disruptive selection, by analyzing mutation and allele substitution in a multi-locus model. This approach extends the work of Bürger (2002ab), by allowing for long-term evolution by mutation and allele substitution. At the same time, our work extends the analysis by Kisdi & Geritz (1999), by allowing for multi-locus genetics. We will consider Levene's soft-selection model (Levene, 1953) as a prototypical example of situations generating frequency-dependent disruptive selection. Levene's model is commonly used for studying the maintenance of variation in a heterogeneous environment, it is relatively simple, its population genetics is well known (Roughgarden, 1979), and it has been considered in several related studies (Kisdi & Geritz, 1999; Van Dooren, 1999; Spichtig & Kawecki, 2004).

MODEL DESCRIPTION

ECOLOGICAL ASSUMPTIONS

We consider an organism with discrete, non-overlapping generations in a heterogeneous environment consisting of two habitats. Individuals are distributed at random over the two habitats at the start of each generation. The two habitats differ in ecological conditions such that an individual is more or less adapted to a habitat depending on its ecological strategy z, a one-dimensional quantitative character. Specifically, we assume that an individual's viability in habitat $i=1,2$ is given by

$$v_i(z)=\exp\left(-\tfrac{1}{2}(z-\mu_i)^2/\sigma^2\right), \qquad [1]$$

which implies that the optimal phenotype is μ_1 in the first habitat and μ_2 in the second. The parameter σ is an inverse measure for the intensity of local selection and determines how rapidly viability declines with the difference between an individual's ecological strategy and the locally optimal one. Without loss of generality, we set $\mu_1=-\mu_2=\mu$.

We assume 'soft selection' (Levene, 1953; see also Ravigné et al., 2004): in each generation, a fixed number $f_i\,N$ of randomly chosen adults are recruited from habitat i; throughout, we set $f_1=f_2=\tfrac{1}{2}$. These adults form a single mating pool of population size N, in which mating occurs at random and offspring is produced at the end of every generation.

GENETIC ASSUMPTIONS

The ecological strategy z is encoded by L diploid loci. One or more distinct alleles may occur at every locus. We use the index k to arbitrarily label the different alleles that occur within the population at a specified locus l. Correspondingly, a_{lk} and x_{lk} denote the kth allele at the lth locus and its phenotypic effect (allelic effect), respectively. We initially assume that loci are unlinked and that alleles interact additively at each locus and between loci. Hence, for an individual carrying alleles $a_{lk'}$ and $a_{lk''}$ at the lth locus, the phenotypic effect of this locus is given by $y_l=x_{lk'}+x_{lk''}$, and the individual's ecological strategy is given by

$$z=\sum_{l=1}^{L} y_l\,. \qquad [2]$$

Later in this study we will also consider non-additive interactions within and between loci, as well as genetic linkage between loci.

Unlike previous models (reviewed in Felsenstein, 1976; Hedrick *et al.*, 1976), which were concerned with the short-term evolutionary process of changes in allele frequencies, we do not constrain the set of alleles that may be present in the population. By allowing new alleles to appear through mutation, we can study the long-term evolutionary process of changes in the phenotypic effects of alleles. Mutations occur at rate m per allele per generation and change the phenotypic effect of an allele by an amount drawn from a normal distribution with zero mean and standard deviation σ_m.

INDIVIDUAL-BASED MODEL

TWO SELECTION REGIMES

Our investigations of the individual-based model defined above show that, not unexpectedly, evolutionary outcomes critically depend on the relative magnitude of the parameters μ and σ.

When the optimal strategies in the two habitats are not too different, or when viability selection is weak ($\mu < \sigma$), long-term evolution of the ecological strategy z proceeds towards the generalist strategy $z^* = 0$ (data not shown). Once the population has reached this generalist strategy, no further phenotypic evolution takes place. Mutation-selection balance maintains only a tiny amount of variation in the population. These observations agree with analytical results (Geritz *et al.*, 1998; Kisdi & Geritz, 1999), which predict that the strategy $z^* = 0$ is both convergence stable and evolutionarily stable for $\mu < \sigma$. The former implies that evolution through small phenotypic steps will proceed towards $z^* = 0$, with each step corresponding to the mutation and subsequent substitution of an allele. The latter implies that no allele coding for an alternative phenotype will be able to invade once the phenotype $z^* = 0$ has been established, and, therefore, that the population experiences stabilizing selection at $z^* = 0$.

By contrast, when the difference between the optimal strategies is large, or when viability selection is strong ($\mu > \sigma$), we observe the emergence of a stable phenotypic polymorphism through the process of evolutionary branching (Metz *et al.*, 1996; Geritz *et al.*, 1998). FIGURE 1 shows a simulation for $\mu = 1.5$ and $\sigma = 1.0$. Other parameters are: $N = 1000$, $L = 3$, $\sigma_m^2 = 10^{-3}$ and $m = 10^{-4}$; unless stated otherwise, the same parameter values will be used throughout this paper. As illustrated in the left panel of FIGURE 1, directional evolution first converges towards the generalist strategy $z^* = 0$, where selection turns disruptive. This is because the strategy $z^* = 0$ is convergence stable, but not evolutionarily stable (Geritz *et al.*, 1998; Kisdi & Geritz, 1999). Therefore, alleles coding for alternative phenotypes can invade the generalist population, thus establishing genetic and phenotypic polymorphism (middle and right panel).

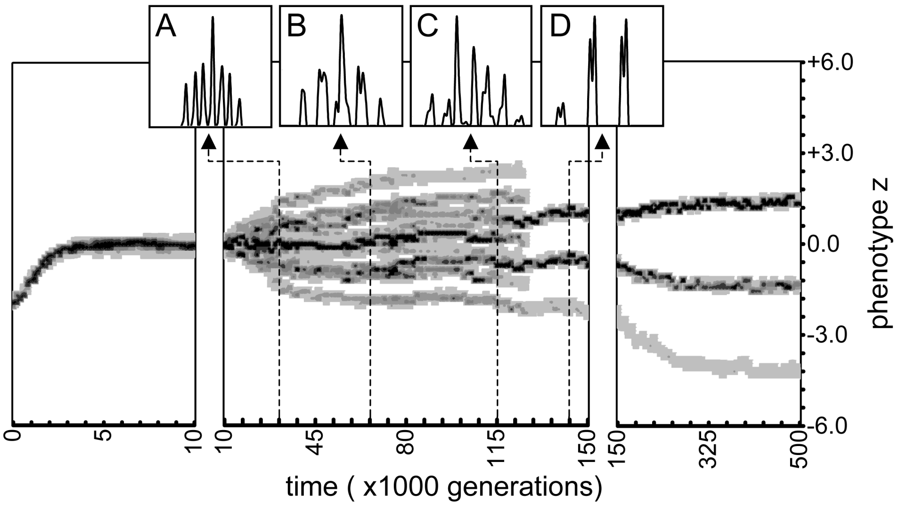


FIGURE 1 – EVOLUTION IN THE INDIVIDUAL-BASED MODEL

The three panels of this figure show the distribution of phenotypes during the rapid convergence to the branching point (left panel), the subsequent phase of diversification at several loci (middle panel), and the final phase of evolution at a single locus (right panel). Small insets A-D show the frequency distribution (frequency is on the vertical axes) of phenotypes (on the horizontal axes) at four moments during the simulation (indicated by dashed lines). Grayscales in the main figure indicate the frequency of phenotypes. At any moment in time, the most common phenotype is shown in black, while less common phenotypes are shown in lighter shades of gray. Note the different scales of the time axis in the three panels.

A GENERAL PATTERN OF POLYMORPHISM FORMATION AND COLLAPSE

We find that establishment of this polymorphism is characterized by a sequence of processes and events:

1 – *Convergence.* During a first phase (FIGURE 1, left panel; generations 0 to 10,000), the evolving population simply converges to the branching point through the gradual adjustment of phenotypic effects, without any significant between-locus or within-locus variation being built up. The first phase thus sets the stage for the establishment of the later polymorphism – by bringing about a regime of frequency-dependent disruptive selection – without yet itself contributing to that process.

2 – *Symmetric divergence.* In a second phase, which commences right after branching (FIGURE 1, middle panel; generations 10,000 to 30,000), the phenotypic differentiation between alleles grows gradually, due to mutations and allelic substitutions. Closer inspection reveals that all loci become polymorphic during this phase. In particular, we observe two equally frequent, distinct classes of alleles with equal but opposite phenotypic effects at each locus. Moreover, the differences between the phenotypic effects of these classes of alleles are roughly equal for all loci. Consequently, the underlying genetic

polymorphism at the L loci gives rise to a total of $2L+1$ phenotypic classes (FIGURE 1, inset A; $L=3$ loci imply 7 such classes).

3 – *Between-locus symmetry breaking.* In a third phase (FIGURE 1, middle panel; generations 30,000 to 125,000), phenotypic variation continues to increase until the distribution of realized phenotypes approximately covers the range from $-\mu$ to μ. However, the symmetry between loci is broken during this phase. At some loci, the alleles continue to diversify, whereas at other loci the differentiation between alleles decreases (FIGURE 1, inset B), until, eventually, only one polymorphic locus remains. This effect was observed to occur independently of the number of loci encoding the ecological strategy and independently of the parameters μ and σ, as long as $\mu > \sigma$. At the remaining polymorphic locus, two classes of alleles give rise to three distinct classes of phenotypes (two homozygotes and a heterozygote; FIGURE 1, inset D).

4 – *Within-locus symmetry breaking.* During the fourth phase (FIGURE 1, generations 125,000+), phenotypic effects and frequencies at the last polymorphic locus become asymmetric. During phase 2, and essentially also during phase 3, the distinct classes of alleles at each particular locus have equal frequencies and opposite but equal effects on the phenotype, such that heterozygotes have phenotypic effects close to zero. During phase 4, this symmetry is lost, such that the heterozygote matches one of the two locally optimal phenotypes, with the other locally optimal phenotypes being matched by one of the homozygotes ($z \approx \mu$ and $z \approx -\mu$; FIGURE 1, right panel). The remaining homozygote expresses a poorly adapted phenotype ($z \approx -3\mu$; FIGURE 1, right panel). This makes it evident that the alleles carried by the latter homozygote have a larger phenotypic effect ($x \approx -3\mu/2$) than the alleles carried by the former homozygote ($x \approx \mu/2$), and that their frequency is lower than $\frac{1}{2}$. In FIGURE 1, this asymmetry primarily grows during phase 4 (inset D), but is already initiated to some slight extent during phase 3 (inset C). Beyond these final adjustments, the population's phenotypic and allelic composition remains stable.

As we will demonstrate below, the four-phase pattern described above is robustly observed in several variations of our basic model. Phases 1 and 4 already occur in single-locus models (Kisdi & Geritz 1999). In this paper we focus on the new patterns resulting from the symmetry breaking between loci during phase 3, and thus on processes that are unique to multi-locus models.

DETERMINISTIC MODEL

DERIVATION OF DETERMINISTIC DYNAMICS

We further investigate the observed loss of polymorphism at all but one locus by analyzing a deterministic approximation of our model. For this purpose we derived deterministic equations for the expected rate of evolutionary change in allelic effects, assuming that mutations are rare and their effects are small. Directional evolution then proceeds by steps involving allelic mutation, invasion, and fixation (Metz *et al.*, 1992, 1996; Dieckmann & Law, 1996; Weissing, 1996; Hofbauer & Sigmund, 1998; Geritz *et al.*, 2002). The outcome of a single step in this process, i.e., whether or not a new mutant allele will be able to invade and substitute an existing resident allele, is determined by the invasion fitness of the mutant allele, i.e., by the rate at which the frequency of the mutant allele increases when it is still rare (Metz *et al.*, 1992, 1996). Mutant alleles with positive invasion fitness have a chance to invade the resident population, and once they have overcome the threat of accidental extinction by demographic stochasticity (Metz *et al.*, 1996; Dieckmann & Law, 1996) they will go to fixation (except under certain special and well-understood circumstances; Geritz *et al.*, 2002). It can be shown that series of such substitution events result in gradual evolutionary change at a rate and in a direction that is related to the gradient of invasion fitness (Dieckmann & Law, 1996).

We followed standard procedures for the derivation of invasion fitness, and the subsequent derivation of dynamical equations for the evolutionary rate of changes in allelic effects (Dieckmann & Law, 1996; Kisdi & Geritz, 1999; details are provided in the APPENDIX).

ILLUSTRATION OF DETERMINISTIC DYNAMICS

A numerical simulation of the resulting model is shown in FIGURE 2. The simulation starts with a population located at the branching point, just after a dimorphism has arisen at two loci. There are two alleles at the first locus, which we will refer to as *A* and *a*, and two alleles at the second locus, henceforth referred to as *B* and *b* (this does not imply that the alleles *A* and *B* are dominant; as before, alleles act additively on the phenotype).

Until about $1.0 \cdot 10^5$ generations, the phenotypic effects of the alleles at both loci diversify rapidly and symmetrically (corresponding to phase 2 as described above), giving rise to five phenotypic classes. The difference between the phenotypic effects of alleles *B* and *b* then diminishes gradually (phase 3), until the allele *B* is lost at about $3.5 \cdot 10^5$ generations, so that only three phenotypic classes remain (which one of the two loci loses its dimorphism depends on arbitrarily small initial asymmetries between them). The difference between the phenotypic effects of alleles *A* and *a* continues to grow throughout phase 3. Finally (phase 4), the alleles at this locus evolve in such a way that one homozygote (*AA*) and the heterozygote (*Aa*) match the optimal phenotypes, whereas the remaining homozygote (*aa*) expresses a sub-optimal phenotype. The frequency of the allele *a* then declines to approximately 0.25. Also the alternative outcome is possible, with the matches provided by *aa* and *Aa* instead (which of these two outcomes will be realized depends on arbitrarily small initial asymmetries between the allelic effects at the remaining dimorphic locus).

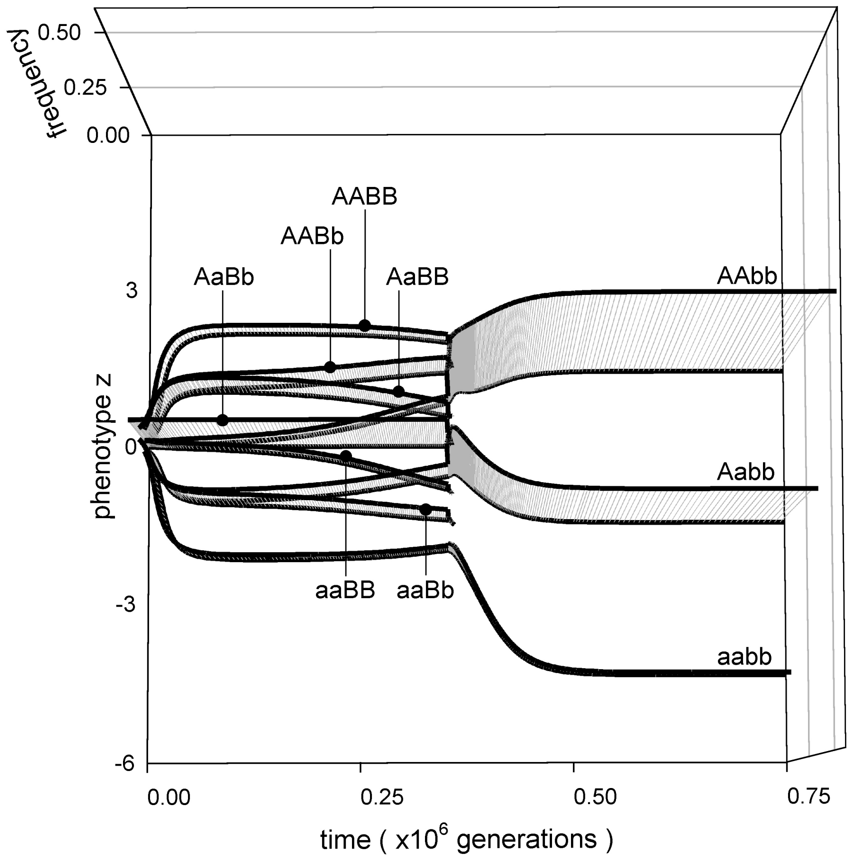


FIGURE 2 – EVOLUTION IN THE DETERMINISTIC MODEL

The deterministic approximation of our model tracks the phenotypic differentiation of alleles at two polymorphic loci. With two alleles at each locus (*A* and *a* at the first, *B* and *b* at the second locus), at most nine different classes of genotypes (indicated by the labels *AABB*,…,*aabb*) are present within the population at any moment in time. Individuals within the same class of genotypes have identical phenotypes. The phenotypes associated with each class of genotypes, and their frequencies, change over time, due to evolutionary change in the phenotypic effects of alleles. The time scale of this process may vary with parameters such as the mutation rate, the mutational variance, and the population size (see the APPENDIX).

Symmetry breaking within and between loci may occur on a faster or slower timescale, depending on initial conditions. Taking into account the expected initial asymmetries between alleles in the individual-based simulations, we find good quantitative agreement between both implementations of our model. Therefore, we use the deterministic model for further investigation.

COMPREHENSIVE ANALYSIS OF DETERMINISTIC DYNAMICS

A comprehensive picture of the evolutionary dynamics of our model can be obtained by focusing on a two-locus diallelic situation (such as illustrated in FIGURE 2) to study the underlying dynamics in allele space. Let us therefore denote the phenotypic effects of alleles *A*, *a*, *B*, and *b* as x_A, x_a, x_B, and x_b, respectively. Without loss of generality, we may define

$$\begin{aligned} x_A &= \bar{x} + \delta_1 + \Delta\,, \quad x_a = \bar{x} - \delta_1 + \Delta\,, \\ x_B &= \bar{x} + \delta_2 - \Delta\,, \quad x_b = \bar{x} - \delta_2 - \Delta\,, \end{aligned} \qquad [3]$$

such that $\bar{x}$ represents the average phenotypic effect of all four alleles, and δ_1 and δ_2 measure the phenotypic differentiation between alleles at the first and second locus, respectively. The quantities $\bar{x}+\Delta$ and $\bar{x}-\Delta$ then represent the average phenotypic effects of the alleles at the first and second locus, respectively. Since alleles interact additively within and between loci, the coefficient Δ has no effect at the phenotypic level, and hence is not subject to selection. This allows us to represent allele space in three dimensions.

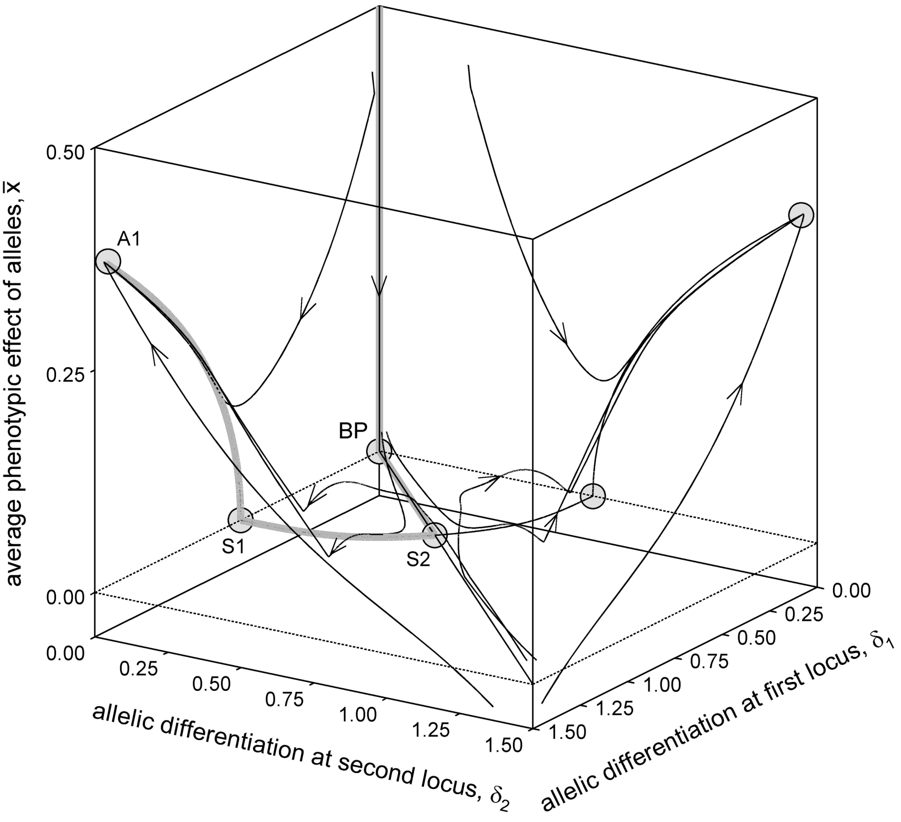


FIGURE 3 – EVOLUTION IN ALLELE SPACE

Simulations of the deterministic approximation of our model, started from various initial conditions, are represented as trajectories in allele space (black lines with arrows). The location of equilibria is indicated by gray circles. The thick gray trajectory highlights how evolution proceeds towards the equilibrium A1 via the equilibria BP, S2, and S1 (for details see the main text). Notice that in this depiction trajectories may intersect with other trajectories, since there exist multiple population genetical equilibria for some combinations of alleles.

FIGURE 3 illustrates the different equilibria we find in allele space. Starting from a population that is monomorphic at both loci ($\delta_1 = \delta_2 = 0$), evolution first converges to the branching point (indicated as BP in FIGURE 3). Any slight degree of dimorphism developing right at the branching point (or, alternatively, having been present initially), takes the population away from this point, towards an equilibrium at which a symmetric allelic dimorphism is established at both loci (equilibrium S2). This equilibrium is not stable, however. Further evolution proceeds towards an equilibrium at which only one locus supports a symmetric allelic dimorphism (equilibrium S1). Since this equilibrium is also not stable, the final phase of evolution involves the transition to an asymmetric allelic dimorphism at a single locus (equilibrium A1).

The sequential approach of an initial condition IC towards the equilibria BP, S2, S1, and A1 in FIGURE 3 can be recognized in the four different phases of the individual-based dynamics shown in FIGURE 1: IC → BP (phase 1), BP → S2 (phase 2), S2 → S1 (phase 3), S1 → A1 (phase 4). The four different phases are the more pronounced the closer trajectories stay to the itinerary IC → BP → S2 → S1 → A1 (see FIGURE 3).

Technically speaking, equilibria like BP, S1, and S2 are called saddle points. Such points are notorious for slowing down dynamics when being approached closely. There are several reasons why such approaches dominate the dynamics of our system:

1 – Due to combinatorial reasons it is unlikely that only a single locus is polymorphic shortly after branching. As long as mutations have small phenotypic effects, one expects the polymorphism to grow initially at the same rate at every locus. Put differently, if the initial phase of phenotypic diversification requires n mutations, then it is much more likely that these mutations are more or less uniformly distributed over loci than that all n mutations occurred at the same locus. As long as n is large relative to the number of loci on which the ecological trait is based, it is therefore probable that the initial asymmetry between loci is small. This confines trajectories ejected from the branching point to the plane $\delta_1 = \delta_2$ (FIGURE 3).

2 – Selection initially tends to decrease the average phenotypic effect of alleles, $\bar{x}$, thus selecting for symmetrical (i.e., equal but opposite) phenotypic effects. This effect is a remnant of the regime of directional selection that drove the monomorphic population towards the branching point: around $\delta_1 = \delta_2 = 0$, selection points towards $\bar{x} = 0$ (FIGURE 3). In conjunction with the first effect, this means that trajectories are ejected from the branching point in the direction $\delta_1 = \delta_2$, $\bar{x} = 0$, i.e., right towards the equilibrium S2.

3 – The closer trajectories pass by S2, the closer they will pass by S1. Since this is a derived effect, the transition from phase 3 to phase 4 will usually be less sharp than that from phase 2 to phase 3 (cf. FIGURE 1).

Populations are thus expected to spend considerable time in the vicinity of the unstable equilibria S2 and S1. This prediction is corroborated by the individual-based simulation shown in FIGURE 1.

ROBUSTNESS WITH RESPECT TO GENETIC ASSUMPTIONS

So far, we have investigated evolution under frequency-dependent disruptive selection in an idealized genetic system, characterized by free recombination and additive interactions within and between loci. In addition, we have assumed that mutations have small phenotypic effects. These simplifying genetic assumptions are habitually made in phenotypic models of evolution, where the details of the underlying genetics are considered to be of secondary importance (see also Weissing, 1996), either because the character under study is likely to be encoded by many loci, or because its genetic basis is unknown. To overcome these limitations, below we investigate the robustness of our results with respect to variations of our genetic assumptions.

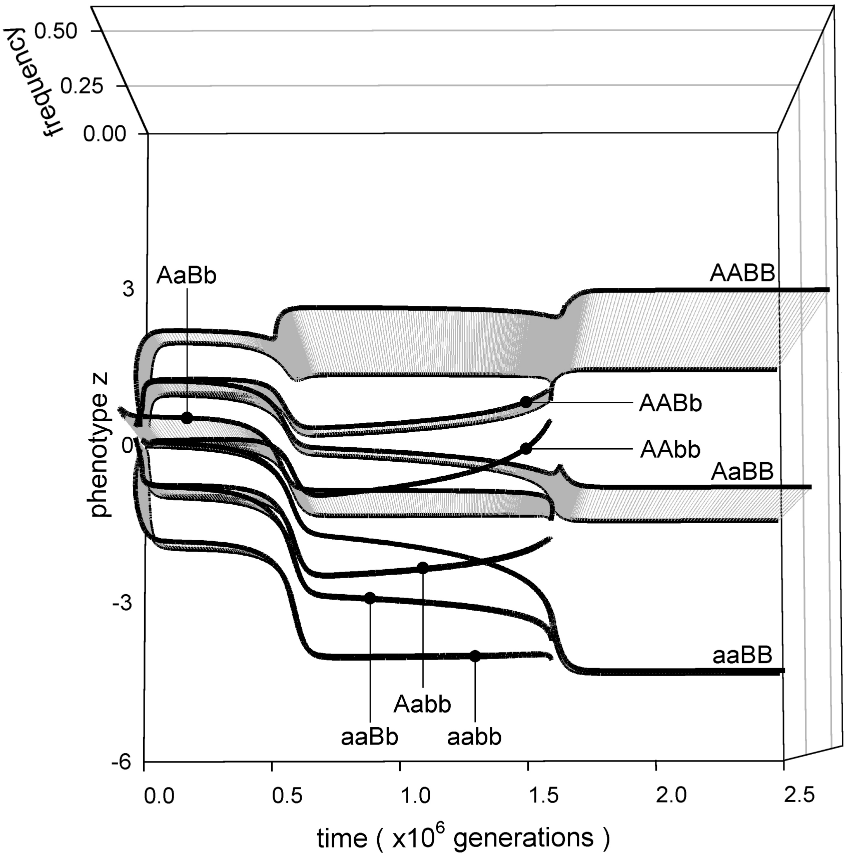


FIGURE 4 – EVOLUTION WITH TIGHT LINKAGE BETWEEN LOCI

Two tightly linked loci ($r = 0.05$), each with two alleles, behave much like a single locus with four 'alleles' (i.e., combinations of alleles). Consequently, differentiation between loci occurs more slowly than differentiation between combinations of alleles.

GENETIC LINKAGE

First, we consider the effects of genetic linkage between loci. FIGURE 4 shows numerical results for our deterministic model with tight linkage between two diallelic loci (the recombination fraction is set to $r = 0.05$).

The two loci initially behave as a single locus with four 'alleles' (given by the haplotypes *AB*, *Ab*, *aB*, and *ab*). Based on the results presented in the preceding sections, we expect that two haplotypes disappear and that the phenotypic effects of the remaining two haplotypes evolve such that one homozygote and the heterozygote express the two locally optimal phenotypes. This is indeed the case. In the first phase of the simulation shown in FIGURE 4 (until about $1.0 \cdot 10^6$ generations), we observe the emergence of a polymorphism of five phenotypic classes, but the frequency of two of the haplotypes (*AB* and *ab*) is much higher than that of the other two haplotypes (*Ab* and *aB*). This can be inferred from the fact that the frequency of the genotypes *AAbb* and *aaBB* is much lower than that of the genotype *AaBb*. After this initial phase, the phenotypic effect of haplotype *ab* becomes strongly negative, allowing the homozygote *AABB* and the heterozygote *AaBb* to express the two locally optimal phenotypes. Due to the tight linkage, asymmetries between the loci evolve more slowly than asymmetries between haplotypes. Eventually however, the polymorphism at one of the loci is lost. In FIGURE 4, the allele *b* disappears shortly after $3.0 \cdot 10^6$ generations.

These results suggest that linkage between loci does affect the relative rates at which asymmetries within and between loci develop, but does not change the partitioning of the evolutionary dynamics into distinguishable phases, the loss of polymorphism at all but one locus, and the final pattern of the evolutionary outcome.

NON-ADDITIVE INTERACTIONS

Second, we consider the effects of non-additive interactions between alleles and between loci. We could relax our assumption of additive genetics by simply imposing fixed, non-additive interactions (e.g., antagonistic or synergistic interactions). We consider this option less than ideal, since it would still constrain the evolutionary process. Instead, we allow for evolutionary change in dominance-recessivity relations and in the weights of individual loci. In this extended approach, the extent to which alleles and loci contribute to the phenotype is flexible and can be shaped by evolution.

Following the modeling framework introduced by Van Dooren (1999), we implemented this flexibility by assuming that an individual's phenotype is determined by the phenotypic effects of the alleles it carries (more precisely, the gene products of the alleles) and by so-called allelic parameters, which determine the extent to which the alleles are expressed, much like regulatory elements in the promotor region of a gene. In addition, we consider modifier loci (e.g., loci coding for transcription factors) that affect the level of expression of all alleles at a given locus. Dominance interactions between alleles derive from the allelic parameters, whereas the weights of individual loci derive from the expression patterns at the modifier loci. An allele's contribution to the phenotype now depends on its weight relative to the weight of the other allele on the same locus, and on the weight of the locus relative to the weights of the other loci. This is illustrated in TABLE 1 for a specific example with two loci. Our approach can easily be extended to allow also for complex epistatic interactions between loci, but, for the sake of conciseness, we refrain from illustrating this here. We allowed both the phenotypic effects of alleles and the allelic parameters to evolve through mutations with small effects. In addition we allowed the weights of loci to evolve through mutations (with small effects) of the alleles at modifier loci (one modifier locus for each ecological trait locus). We assumed free recombination between all loci.

TABLE 1 – INTERACTIONS BETWEEN ALLELES AND BETWEEN LOCI

	locus 1		locus 2	
	allele 1	allele 2	allele 1	allele 2
phenotypic effect	x_{11}	x_{12}	x_{21}	x_{22}
allelic parameter	u_{11}	u_{12}	u_{21}	u_{22}
weight of alleles	$U_{11} = \frac{u_{11}}{u_{11}+u_{12}}$	$U_{12} = \frac{u_{12}}{u_{11}+u_{12}}$	$U_{21} = \frac{u_{21}}{u_{21}+u_{22}}$	$U_{22} = \frac{u_{22}}{u_{21}+u_{22}}$
alleles at modifier locus	w_{11}	w_{12}	w_{21}	w_{22}
weight of loci	$W_1 = \frac{w_{11}+w_{12}}{w_{11}+w_{12}+w_{21}+w_{22}}$		$W_2 = \frac{w_{21}+w_{22}}{w_{11}+w_{12}+w_{21}+w_{22}}$	
phenotype	$z = W_1\left(U_{11}\ x_{11} + U_{12}\ x_{12}\right) + W_2\left(U_{21}\ x_{21} + U_{22}\ x_{22}\right)$			

FIGURE 5 shows numerical results for the extended individual-based model: we again observe rapid convergence to the branching point, followed by a phase of phenotypic diversification. Initially, three out of four loci become polymorphic, but eventually only one polymorphic locus remains. Insets A-C in FIGURE 5 show the relative weights (on the vertical axis) of the four different loci (on the horizontal axis) at three moments during the simulation. Grey bars are used for monomorphic loci; black and white bars are used for polymorphic loci. The subdivision in a white and black part represents the relative weights of the two different alleles that occur on a polymorphic locus. During the initial phase of differentiation (corresponding to phase 2 as described above), the alleles at all polymorphic loci diversify symmetrically (inset A, at $3.0 \cdot 10^4$ generations). There is directional selection to increase the weight of polymorphic loci relative to the weight of the one monomorphic locus (locus 2), while selection on the allelic parameters is still virtually absent. Later on, however, the asymmetries between loci grow (corresponding to phase 3 as described above), until only one polymorphic locus remains (locus 1). During this phase, disruptive selection on the relative weights of polymorphic loci acts alongside selection on allelic effects (which is stabilizing for some loci but diversifying for other loci), such that the locus with the largest differentiation between alleles eventually contributes to the phenotype with the largest relative weight (inset B, at $8.0 \cdot 10^4$ generations). All along, the interaction between alleles at a single locus has remained additive, i.e., the alleles at polymorphic loci have equal relative weights. However, selection for dominance-recessivity interactions between alleles arises as soon as asymmetries evolve between alleles at the remaining polymorphic locus (corresponding to phase 4 as described above). The relative weight of one of the alleles increases, such that, eventually, the phenotype of the (otherwise) maladapted homozygote coincides with the locally optimal phenotype matched by the heterozygote (inset C, at $7.0 \cdot 10^5$ generations).

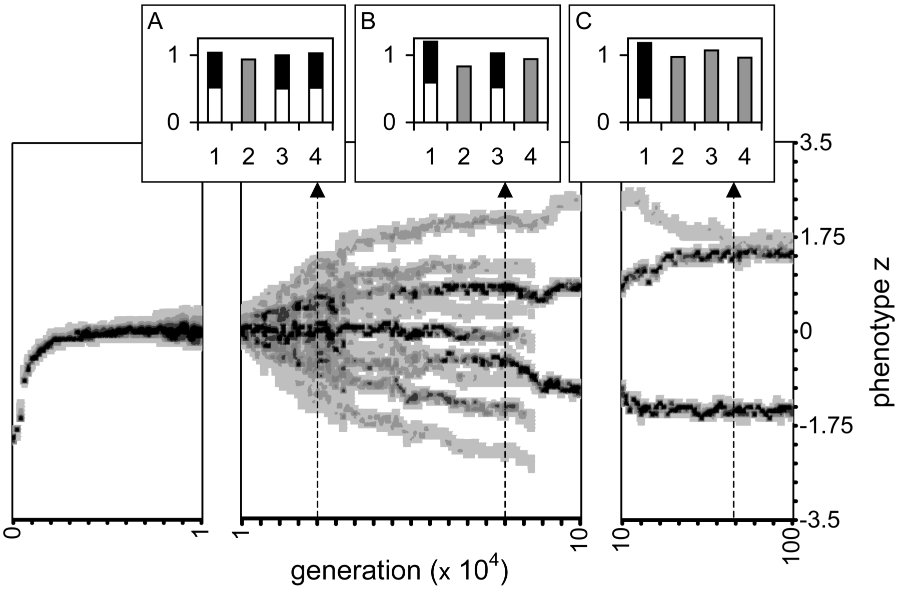


FIGURE 5 – EVOLUTION WITH VARIABLE WEIGHTS FOR ALLELES AND LOCI

Three panels show the distribution of phenotypes in an individual-based simulation as in FIGURE 1. The insets A-C, however, do not show frequency distributions, but the average relative weights of loci and alleles (i.e., the extent to which an allele at a specific locus contributes to the phenotype), at three moments during the simulation (indicated by dashed lines). The height of the bars represents the weight of a locus (in this simulation we kept track of four loci). For polymorphic loci, bars consist of a black and white part, indicating the weights of the different alleles that occur at this locus. Grey bars are used for monomorphic loci. Unlike in FIGURE 1, $L = 4$.

These results show that the evolution of non-additive interactions between alleles and the evolution of loci weights are expected to act alongside the evolution of allelic effects, representing alternative pathways along which the symmetry between and within loci can be broken. The relative contributions of the evolution of allelic effects (the evolution of the gene products), versus the evolution of the weights of alleles and loci (the evolution of gene regulation), will depend on factors like the relative mutation rates of the phenotypic effects, the allelic parameters, and the modifier alleles. All key predictions of our preceding analysis are corroborated even in this extended model. In particular, the characteristic phasing of dynamics from the initial diversification to the final outcome is robustly recovered.

LARGE MUTATIONS

As a third check on the robustness of our results, we explored the effects of large mutational step sizes. FIGURE 6 shows numerical results for our original individual-based model, with all parameters except the mutational variance σ_m^2 and the mutation rate m chosen exactly as in FIGURE 1. In FIGURE 6, the mutational variance σ_m^2 was set 100 times larger than in FIGURE 1, and the mutation rate was set 100 times smaller, such that the expected rate of directional evolution, which scales with $m\sigma_m^2$ (Dieckmann & Law, 1996; see also the APPENDIX), was identical for both simulations.

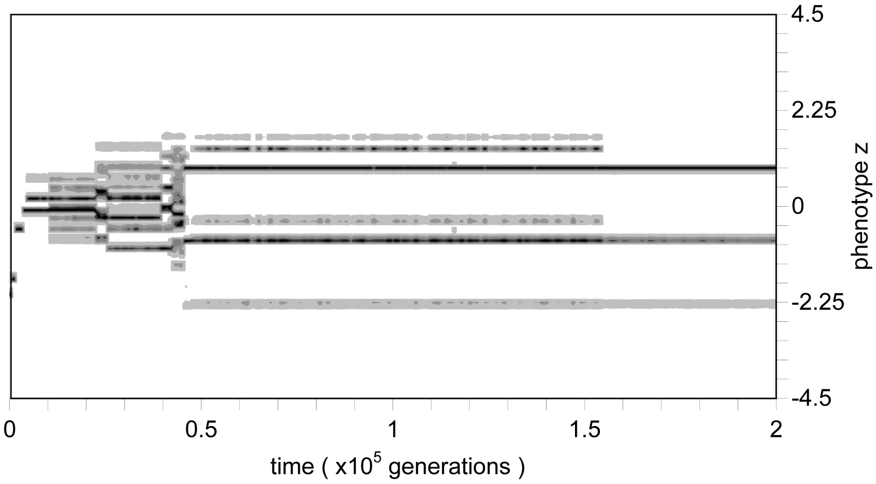


FIGURE 6 – EVOLUTION WITH LARGE MUTATIONAL STEPS

Parameters as in FIGURE 1, except for $\sigma_m^2 = 0.1$ and $m = 10^{-6}$.

These results show that with large mutational steps the whole evolutionary process (of convergence to the branching point, loss of polymorphism on all-but-one locus, and asymmetric differentiation of alleles at the remaining polymorphic locus) is reduced to only a small number of allele substitution events (individual allele substitution events can be recognized as discontinuities in FIGURE 6). Consequently, the stochasticity of the mutation process is much more pronounced, and the variation between replicate simulations is larger. Yet, the average behavior of replicates does not deviate qualitatively from the predictions of our deterministic model. Also the diminished phasing of the evolutionary process is just as predicted, since smaller mutational steps make it easier for the genetic system to track the saddle connections that lie at the heart of the process. FIGURE 6 shows that we can still recognize the different phases discussed before, even when mutation effects are not small, a finding that underscores the robustness of our results. It is clear that the weaker selection and the larger the mutational step size, the more strongly the stochasticity of the mutation process will blur the selection-driven, deterministic component of evolutionary change.

DISCUSSION

Our results show that frequency-dependent disruptive selection is less powerful in maintaining polygenic variation than one would naïvely expect. Frequency-dependent disruptive selection does not lead to the establishment of genetic polymorphism at a large number of loci. Instead, genetic variation is concentrated at a single locus with large phenotypic effect. We observed this outcome in individual-based simulations and in an analytical model, under a range of genetic assumptions, which gives confidence in the robustness of the results. The identified pattern of polymorphism formation and collapse is likely to be widely applicable.

The dynamics observed in our model suggest a conceptual link between the different effects of frequency-dependent disruptive selection observed in quantitative genetics (QG) and adaptive dynamics (AD) models. In the initial phase of diversification, all loci are polymorphic, and the phenotypic differentiation of alleles at each locus is small. Hence, a large number of loci contribute to the genetic variation, and each locus has a small effect on the phenotype. Not surprisingly, the dynamics shortly after evolutionary branching therefore much resembles the maintenance of variation as observed in QG models, where disruptive selection leads to the gradual broadening of a continuous phenotype distribution. Eventually, however, genetic variation becomes concentrated at a single locus, which contributes increasingly strongly to phenotypic variation. In this situation QG methods become inaccurate: we observe the emergence of discrete clusters of phenotypes that create a situation better analyzed by AD methods.

The phenomena of polymorphism formation and collapse observed in our model are a straightforward consequence of the fact that frequency-dependent selection generates a dynamic selection regime. It is a defining feature of frequency dependence that the intensity and direction of selection changes as evolution proceeds, a consequence of the feedback between a population and its environment. In the context of our model, the population first experiences directional selection towards the branching point, then disruptive selection at the branching (leading to diversification at all loci), and subsequently again a type of stabilizing selection (favoring two discrete phenotypes at the patch optima). Selection turns from disruptive to stabilizing as soon as the phenotypic variation in the population has become large enough for the optimal phenotypes in the two patches to occur at appreciable frequencies. At that point, there is no further selection for diversification. Yet, intermediate phenotypes remain at a selective disadvantage. It is an unavoidable consequence of sexual reproduction – at least, as long as individuals mate at random – that such intermediate phenotypes are generated, but, for combinatorial reasons, their frequency is lowest when all genetic variation is concentrated at a single locus. This explains why all loci, except one, eventually become monomorphic. Subsequent evolution, involving symmetry breaking between alleles at the remaining polymorphic locus, increases population mean fitness by further reducing the frequency of maladapted individuals.

Although here we have analyzed only Levene's soft-selection model, we expect that our conclusions apply to a broad class of systems subject to frequency-dependent disruptive selection. Adaptive dynamics theory has revealed the generic shape of fitness landscapes around evolutionary branching points (e.g., Geritz *et al.*, 1997), and adaptive dynamics models have shown that such branching points can be created by a plethora of different ecological mechanisms, including all three fundamental types of ecological interaction (e.g., Doebeli & Dieckmann, 2000). In particular, we expect to observe similar evolutionary phenomena in all cases where the coexistence of an arbitrarily large number of replicators is precluded by a competitive exclusion principle. Such systems must, at some level of diversity, exhibit a transition from disruptive to stabilizing selection favoring the evolution of a discrete, limited set of phenotypes. As in our Levene-type model, where the number of coexisting replicators is bounded by the number of different habitats, this will set an upper limit to the number of loci expected to remain polymorphic in long-term evolution.

In a somewhat different context, Spichtig and Kawecki (2004), who recently also analyzed a multi-locus version of Levene's model, come to a conclusion similar to ours. While their analysis addresses the dynamics and the equilibrium frequencies of a *fixed* set of alleles, other aspects of the two models are similar, allowing for a detailed comparison of results. Also Spichtig and Kawecki (2004) argue that the capacity of soft selection to maintain polygenic variation is smaller than one would expect based on single-locus models. Their conclusion, however, applies to parameter regimes where evolutionary branching does *not* occur since the fitness of intermediate phenotypes is high. Under these conditions, polygenic variation is not maintained, due to the fact that the average phenotype of a polygenic character can be accurately matched with the optimal phenotype without requiring a polymorphism of alleles at individual loci (i.e., with all loci being homozygous, and, hence, with the population being monomorphic). This conclusion does not apply to a single locus, where the realization of an intermediary phenotype typically requires a heterozygous genotype (and, hence, a polymorphic population). Unlike for single-locus characters, the variation of polygenic characters can therefore be low, irrespective of the mean phenotype.

In contrast, our conclusion applies to the maintenance of polygenic variation after evolutionary branching; that is to say, it concerns a complementary parameter regime. In this case, the explanation for the loss of polygenic variation is different and stems from the fact that a single-locus polymorphism allows for a maximal level of phenotypic variation: given a certain degree of overall differentiation between alleles, the phenotypic variance in the population is highest when the polymorphism is concentrated at a single locus. Under conditions that allow for evolutionary branching, a polymorphism of differentiated alleles at a single locus is therefore the most favorable configuration that can be attained within the limits set by sexual reproduction. It allows for the lowest possible frequency of the intermediate phenotypes that are at selective disadvantage in the parameter regime considered here.

Obviously, a single-locus polymorphism will only be favored over polygenic variation when the phenotypic effects of individual alleles are considerable, such that a polymorphism at a single locus can give rise to substantial phenotypic variation. In our model, the phenotypic effects of individual alleles can become arbitrarily large, as a cumulative result of many mutations with small phenotypic effects. In population genetic models, where the set of alleles is kept fixed and mutation is not incorporated, the phenotypic effects of individual alleles are usually limited, such that a polymorphism of alleles at a single locus can only give rise to a modest level of phenotypic variation. In such a situation, we would expect variation to be maintained at multiple loci, since this is the only way to maintain sufficient genetic variation (an expectation confirmed by Bürger, 2002ab; Spichtig & Kawecki, 2004).

This highlights another contrast between our model and existing population genetic analyses of frequency-dependent disruptive selection. The latter consider short-term evolution by investigating allele-frequency changes and the stability properties of population genetical equilibria of a predefined set of alleles. This yields conditions for the short-term maintenance of genetic variation, but does not provide insights about long-term evolution, which occurs though the substitution of the existing alleles by novel, mutant alleles. This process is explicitly considered in our model. Our results therefore primarily concern the long-term maintenance of polygenic variation, an issue that is largely outside the scope of population genetic models (Eshel, 1996).

ACKNOWLEDGEMENTS

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APPENDIX – DERIVATION OF DETERMINISTIC APPROXIMATION

Here we derive a deterministic approximation that captures the dynamics in our individual-based simulation model. To enable this complementary treatment, we assume that (a) mutations occur rarely, such that mutant alleles arise in a resident population that is close to its population genetic equilibrium. Consequently, a mutant allele interacts only with the currently predominant resident alleles, which were successful at ousting previous mutant alleles. We also assume that (b) the population is sufficiently large, such that we may neglect stochasticity in the dynamics of the frequencies of resident alleles, and that (c) changes in the phenotypic effects of alleles caused by mutations are typically small, such that it is meaningful to approximate the long-term dynamics of phenotypic effects deterministically.

The invasion fitness λ specifies the geometric rate of increase of the frequency of a mutant allele while it is rare (e.g., just after it has arisen by mutation; Metz *et al.*, 1992, 1996). When a mutant arises in an otherwise genetically monomorphic resident population, all resident individuals have the same phenotype $\hat{z}$ and all individuals that carry a mutant allele have the same phenotype z. This greatly simplifies the derivation of invasion fitness in our model (see, e.g., Kisdi & Geritz, 1999), which under such conditions is given by

$$\lambda = \tfrac{1}{2}\left[v_1(z)/v_1(\hat{z}) + v_2(z)/v_2(\hat{z})\right]. \tag{4}$$

The first and second term in the square bracket represent, respectively, the frequency of mutant individuals in the first and second habitat after viability selection, and the factor 1/2 simply reflects the assumption that half of the individuals in the mating pool are recruited from either habitat.

When the resident population is polymorphic at one or more loci, the derivation of invasion fitness becomes more complicated, since we then need to keep track of the frequencies of the different resident genotypes. The mutant allele may then also occur in different genetic backgrounds, consisting of different combinations of resident alleles. To keep the analysis tractable, we will restrict ourselves here to the simplest interesting case, by considering a resident population that is polymorphic at two loci ($L = 2$). We denote the alleles at the first locus by A and a, and the alleles at the second locus by B and b (as in the main text, this notation does not imply that the alleles A and B are dominant). The phenotypic effects of the alleles are denoted by x_A, x_a, x_B, and x_b. If $x_A = x_a$ or $x_B = x_b$, the resident population is monomorphic at the corresponding locus. We also consider a mutant allele M, with phenotypic effect x_M, that has arisen through mutation of the allele A at the first locus (other mutant alleles are dealt with analogously).

We choose to describe the dynamics of the resident and mutant allele frequencies in terms of the frequencies of the haploid gametes in which they occur: f_g

denotes the frequency of the gamete g ($g = AB, Ab, aB, ab, MB,$ or Mb) in the parental generation at the moment of reproduction, i.e., after viability selection has occurred. We follow the life cycle of our model to determine its effect on these gamete frequencies:

1 – *Random mating.* Since mating is random, the frequency of offspring carrying the genotype gg', which arises from the combination of gametes g and g', is given by $f_g f_{g'}$.

2 – *Viability selection.* Viability selection changes the genotype frequencies in the offspring generation, such that the frequency $F_{gg'}$ of the genotype gg' after viability selection is, similarly to equation (4),

$$F_{gg'} = f_g f_{g'} \tfrac{1}{2}\left[v_1\left(z_{gg'}\right) \big/ \bar{v}_1 \, / + v_2\left(z_{gg'}\right) \big/ \bar{v}_2 \right], \tag{5}$$

where $z_{gg'}$ denotes the phenotype encoded by the genotype gg' (for example, $z_{ABAb} = 2x_A + x_B + x_b$ and $z_{abMb} = x_a + x_M + 2x_b$) and $\bar{v}_i$ is the average viability in habitat i. While the mutant allele is rare, average viabilities do not depend on the mutant's genotype frequencies,

$$\bar{v}_i = \sum_{g,\,g'=AB,\,Ab,\,aB,\,ab} f_g f_{g'} v_i\left(z_{gg'}\right). \tag{6}$$

3 – *Gamete production.* After viability selection, the next generation is produced through sexual reproduction. The frequencies of the different resident gametes are determined straightforwardly by the resident genotype frequencies after viability selection. For example,

$$f_{AB} = F_{ABAB} + \tfrac{1}{2} \sum_{g=Ab,\,aB} \left(F_{ABg} + F_{gAB}\right) + \tfrac{1}{2}(1-r)\left(F_{ABab} + F_{abAB}\right) + \tfrac{1}{2} r \left(F_{AbaB} + F_{aBAb}\right), \tag{7}$$

where r is the coefficient of recombination between the two loci. The mutant's genotype frequencies do not appear in equation [7], since the frequency of the mutant allele is initially negligible.

Equations [5] to [7] define a recurrence relation for the resident gamete frequencies. This recurrence relation can be iterated until these frequencies converge to a stable equilibrium (reflecting our assumption that resident populations attain their population genetic equilibrium by the time a mutant arises).

For the mutant gamete frequencies we obtain, analogously to equation (7),

$$\begin{aligned} f_{MB} &= \tfrac{1}{2} \sum_{g=AB,\,aB} \left(F_{MBg} + F_{gMB}\right) + \tfrac{1}{2}(1-r) \sum_{g=Ab,\,ab} \left(F_{MBg} + F_{gMB}\right) + \tfrac{1}{2} r \sum_{g=AB,\,aB} \left(F_{Mbg} + F_{gMb}\right), \\ f_{Mb} &= \tfrac{1}{2} \sum_{g=Ab,\,ab} \left(F_{Mbg} + F_{gMb}\right) + \tfrac{1}{2}(1-r) \sum_{g=AB,\,aB} \left(F_{Mbg} + F_{gMb}\right) + \tfrac{1}{2} r \sum_{g=Ab,\,ab} \left(F_{MBg} + F_{gMB}\right). \end{aligned} \tag{8}$$

Here we again used the fact that the mutant allele is rare initially, which allowed us to neglect the frequency of individuals that are homozygous for the mutant allele.

For mutant alleles M that differ only slightly from the resident allele A $\left|x_M - x_A\right|$ is small, and we may use first-order Taylor expansions to approximate the viabilities of phenotypes affected by the mutant allele. For example,

$$v_i\left(z_{MBg}\right)=\exp\left(-\tfrac{1}{2}\left(z_{MBg}-\mu_i\right)^2/\sigma^2\right)\approx\left[1-\left(x_M-x_A\right)\left(z_{ABg}-\mu_i\right)/\sigma^2\right]v_i\left(z_{ABg}\right). \qquad [9]$$

Using these approximations, and after some rearrangement, we find the following recurrence relation for the change of mutant gamete frequencies from one generation to the next,

$$\begin{pmatrix} f_{MB} \\ f_{Mb} \end{pmatrix} \rightarrow \left[\mathbf{F}+\left(x_M-x_A\right)\mathbf{W}\right]\begin{pmatrix} f_{MB} \\ f_{Mb} \end{pmatrix}, \qquad [10]$$

where the matrices $\mathbf{F}$ and $\mathbf{W}$ are defined as

$$\mathbf{F}=\begin{pmatrix} 1-rf_{AB}^{-1}\left(F_{ABAb}+F_{AbaB}\right) & rf_{Ab}^{-1}\left(F_{ABAb}+F_{AbaB}\right) \\ rf_{AB}^{-1}\left(F_{ABAb}+F_{ABab}\right) & 1-rf_{Ab}^{-1}\left(F_{ABAb}+F_{ABab}\right) \end{pmatrix}, \qquad [11]$$

$$\mathbf{W}=\begin{pmatrix} \sum\limits_{g=AB,\,aB} W_g^{AB}+(1-r)\sum\limits_{g=Ab,\,ab} W_g^{AB} & r\sum\limits_{g=AB,\,aB} W_g^{Ab} \\ r\sum\limits_{g=Ab,\,ab} W_g^{AB} & \sum\limits_{g=Ab,\,ab} W_g^{Ab}+(1-r)\sum\limits_{g=AB,\,aB} W_g^{Ab} \end{pmatrix}, \qquad [12]$$

with coefficients

$$W_g^{g'}=-\tfrac{1}{2}f_g\sigma^{-2}\left[\left(z_{gg'}-\mu_1\right)v_1\left(z_{gg'}\right)/\bar{v}_1+\left(z_{gg'}-\mu_2\right)v_2\left(z_{gg'}\right)/\bar{v}_2\right]. \qquad [13]$$

The invasion fitness λ of the mutant allele in the considered polymorphic resident background is now given by the geometric rate of increase of the mutant allele frequency, which equals the dominant eigenvalue of the matrix $\mathbf{F}+\left(x_M-x_A\right)\mathbf{W}$. For small $\left|x_M-x_A\right|$ it can be shown (e.g., Caswell, 1989; Taylor, 1996) that

$$\lambda=1+\left(x_M-x_A\right)\frac{v\,\mathbf{W}\,u}{vu}, \qquad [14]$$

where

$$v=\left(F_{ABAb}+F_{ABab},F_{ABAb}+F_{AbaB}\right) \quad \text{and} \quad u=\begin{pmatrix} f_{AB} \\ f_{Ab} \end{pmatrix} \qquad [15]$$

are the dominant left and right eigenvectors of the matrix $\mathbf{F}$, respectively.

Under suitable assumptions (Dieckmann & Law, 1996; Weissing, 1996; Hofbauer & Sigmund, 1998), the invasion fitness can be used to describe the long-term rate and direction of a series of allelic substitution events. Indeed, using equation (14) and following the derivation scheme employed by Dieckmann & Law (1996), it can be shown that the expected evolutionary rate of change of the phenotypic effect of the currently resident allele A at the first locus satisfies

$$\frac{dx_A}{dt}=\int 2Nm\left(f_{AB}+f_{Ab}\right)\cdot M\left(x_M\,|\,x_A\right)\cdot\alpha\max\left(0,\left(x_M-x_A\right)\frac{v\,\mathbf{W}\,u}{vu}\right)\cdot\left(x_M-x_A\right)dx_M\;, \qquad [16]$$

where t measures evolutionary time in generations. The first factor in the integrand above is the rate at which new mutant alleles arise: the frequency of allele A is given by $f_{AB}+f_{Ab}$, the total number of alleles in a diploid population of size N is $2N$, and m equals the mutation rate per generation. The second factor is the probability density according to which a mutation changes the phenotypic effect at the first locus from x_A to x_M. The third factor is the probability that the mutant allele will successfully invade. This probability is zero when the mutant allele has a geometric rate of increase below that of the resident allele and otherwise is proportional to the fitness advantage s of the mutant allele, as long as s is small. This explains the function $\alpha \max(0,s)$, with α denoting the constant of proportionality, and with $s=(x_M-x_A)(v\,\mathbf{W}\,u)/(vu)$ following from equation [14]. For offspring numbers varying according to a Poisson distribution, we obtain $\alpha = 2$. If the mutant allele succeeds to invade, this causes a change of the resident allele: away from the branching point (and from population dynamical bifurcation points), successful invasion of the mutant allele implies that it will eventually replace the resident allele (Geritz et al., 2002). Successful invasion thus means that the phenotypic effect of the currently resident allele will change by an amount $x_M - x_A$, which explains the integrand's fourth factor.

Collecting all terms that are independent of x_M in front of the integral, and realizing that the integrand above vanishes along half its range since only mutant alleles with either $x_M > x_A$ or $x_M < x_A$ can successfully invade, we can rewrite equation (16) as

$$\frac{dx_A}{dt} = 2Nm\left(f_{AB}+f_{Ab}\right)\alpha\,\frac{v\,\mathbf{W}\,u}{vu}\cdot\tfrac{1}{2}\int\left(x_M-x_A\right)^2 M\left(x_M \mid x_A\right)dx_M\,. \qquad [17]$$

Denoting the mutational variance by σ_m^2, we therefore finally obtain

$$\frac{dx_A}{dt} = N\,m\,\alpha\,\sigma_m^2\left(f_{AB}+f_{Ab}\right)\frac{v\,\mathbf{W}\,u}{v\,u}\,. \qquad [18]$$

Equations for the rate of change in the phenotypic effects of the alleles a, B, and b, are derived analogously.

Part 2

Good Genes

Sexual selection on good genes facilitates sympatric speciation

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Pim Edelaar, G. Sander van Doorn & Franz J. Weissing

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The theoretical feasibility of sympatric speciation – speciation in the absence of spatial isolation – continues to puzzle biologists, despite empirical evidence that it has occurred on numerous occasions (Turelli *et al.*, 2001). Sympatric speciation requires the evolution of assortative mating in the nascent species. It has often been argued that sexual selection could drive this process (Panhuis *et al.*, 2001). Existing models supporting this view are typically based on female preferences for 'arbitrary traits' that diverge by Fisherian runaway selection (Panhuis *et al.*, 2001; Turelli *et al.*, 2001). However, most sexual traits do not seem to be arbitrary, but specifically signal quality ('good-gene' indicators) (Jennions & Petrie, 2000). Here, we propose a novel, less restrictive, and widely applicable mechanism for sympatric speciation that relies crucially on sexual selection on such indicators of good genes.

In a world with only small and large seeds, one could imagine a generalist bird species splitting into a small-billed species feeding on small seeds, and a large-billed species feeding on large seeds. However, the emergence of these two specialist species is prevented by interbreeding, which creates individuals with intermediate bill sizes. For speciation to occur, birds should mate with individuals of similar bill size (Turelli *et al.*, 2001). Suppose that, in a given environment, males with a more adaptive bill size can collect food more efficiently and therefore develop a brighter plumage, such that plumage signals compatibility with the local environment. The evolution of a female preference for such a quality indicator gives a reproductive advantage to locally adapted males. Moreover, it enables females to recognize and avoid maladapted males. Thus, sexual selection on good genes facilitates speciation, since it both enhances ecological disruptive selection for local adaptation and reduces interbreeding.

We illustrate this general idea with an individual-based simulation model (details are provided in the APPENDIX). There are two types of habitat (two seed sizes). The fit of the ecological trait (bill size) to the habitat determines fecundity in females. Both sexes have the tendency to settle in the habitat where their bill size is more adaptive. Mating takes place within these habitats. This induces some assortative mating, yet the population does not split due to the overriding effect of interbreeding (FIGURE 1A). This situation changes when we allow the evolution of a female mating preference for a male ornament (plumage brightness) that signals the match between habitat and ecological trait. Even when costly, female preference spreads in a self-reinforcing manner. By choosing more ornamented (locally adapted) males, females induce disruptive selection on the male ecological trait and, hence, increase male diversity. This, in turn, makes it more profitable to choose a locally adapted male. The mate-choice process thus intensifies ecological disruptive selection. From a certain point onwards, disruptive selection generated by the combination of natural and sexual selection becomes so strong that the species rapidly splits in two (FIGURE 1B-D). A comparable split of the population by natural selection alone would require much stronger selection differentials (APPENDIX). Moreover, sexual selection reduces the frequency of hybridization after

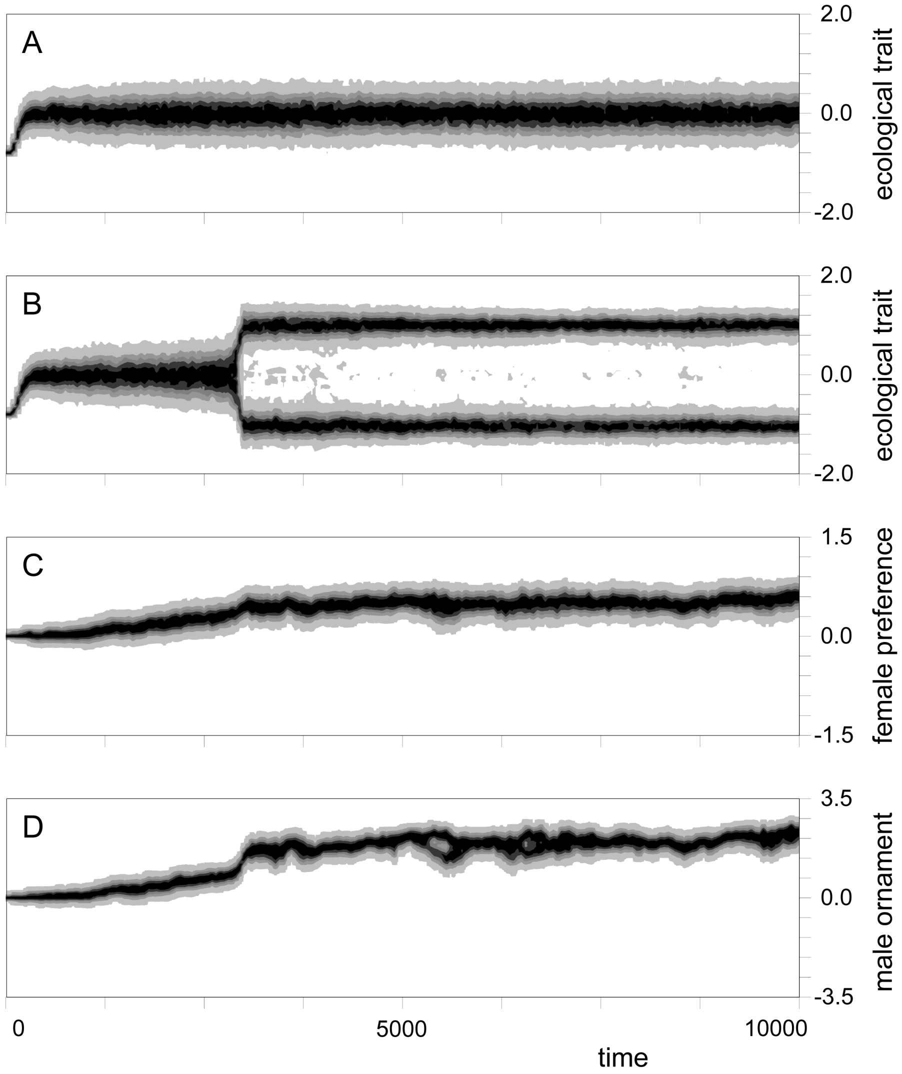

FIGURE 1 – SIMULATION RESULTS

Sexual selection on a trait signaling male quality can cause reproductive isolation between two ecologically specialized populations, when natural selection alone cannot. The two simulations used identical basic parameter values and lasted 10,000 generations (see APPENDIX for details). The grayscale indicates relative frequency in the population, increasing from white to black. Panel A: In the absence of sexual selection, a generalist population evolves despite disruptive natural selection on an ecological trait. Panel B-D (all same simulation): With sexual selection, we see the joint evolution of two reproductively isolated specialist populations (B), female preference (C), and a male ornament (D) signaling adaptedness to a specific habitat.

divergence by one to two orders of magnitude (APPENDIX). Hence, sexual selection has a double effect: it enhances both disruptive selection and assortative mating. The underlying mechanism presented here seems quite general and robust: sexual selection on quality indicators can easily reinforce disruptive natural selection and assortative mating up to the level required for divergence, whenever some degree of assortative mating is initially present.

Our model does not depend on the divergence of mating traits, unlike previous models. Therefore, first, sexual selection does not have to be divergent and sympatric speciation can occur under much less restrictive conditions than in previous models (CHAPTER 4 of this thesis). Second, no genetic associations between mating traits and ecological traits are needed, sidestepping one of the major problems in speciation theory: the disruptive effect of recombination between these traits (Turelli *et al.*, 2001).

There is much empirical evidence for sexual selection on quality indicators (Jennions & Petrie, 2000), yet this process has virtually been ignored in speciation theory (Panhuis *et al.*, 2001; Turelli *et al.*, 2001 but see Lorch *et al.*, 2003). Female preferences for quality indicators will be present in many populations for other reasons (Jennions & Petrie, 2000), but we show that such preferences can even evolve from scratch, because of their self-reinforcing ability to enable ecological specialization and assortative mating. Hence, we expect that comparative and experimental studies will soon confirm the potential of sexual selection on quality indicators to facilitate ecological speciation.

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APPENDIX — MODEL DEFINITION AND SUPPORTING MATERIAL

Our model combines the ecological assumptions of Levene's 'soft selection' model (Levene, 1953) with a revealing-handicap model for the evolution of female preferences (Iwasa *et al.*, 1991). We consider an organism with discrete, non-overlapping generations in a heterogeneous environment consisting of 40 patches, each supporting 40 individuals. Offspring can disperse to any of the patches, where they are exposed to selection. Mating occurs between individuals within the same patch, after which a new generation of dispersers is produced. Parameter values given are those for FIGURE 1.

ECOLOGY

There are two equally frequent patch-types, denoted A and B, which differ in ecological conditions. An individual may be more or less adapted to a certain patch-type depending on its ecological strategy, x, which we assume to be a one-dimensional continuous quantity. The individual's ecological performance, q, in a patch of type i declines with the difference between the individual's ecological strategy x and the optimal strategy μ_i for this patch type:

$$q = q_i(x) = \exp\left(-\frac{1}{2}\left(\frac{x-\mu_i}{\sigma}\right)^2\right). \qquad [1]$$

The parameter σ determines how rapidly q declines with $|x-\mu_i|$. Selection on the ecological strategy turns from stabilizing to disruptive when the ecological difference between the patches, $|\mu_A-\mu_B|$, exceeds 2σ (Geritz *et al.*, 1998). We took $\mu_A = -\mu_B = 1.0$ and $\sigma = 0.75$.

HABITAT CHOICE

Every individual can visit sequentially a number of patches. Its decision to settle in a patch is positively related to its ecological performance q in that patch. Individuals cannot settle in patches that are already full, but all individuals can settle somewhere. Under these assumptions, individuals with the highest ecological performance in one of the habitats visit, on average, fewer patches and are more likely to settle in their preferred patch type, giving them an advantage over less specialized individuals.

NON-RANDOM MATING

We assume a polygynous mating system. Females are choosy and mate only once. Female fecundity is limited by time or energy constraints, and, for simplicity, we take it to be proportional to the female's ecological performance q. A female chooses a male based on her preference p and the size s of the male's ornament: the mating probability is proportional to

$$a = \exp(ps). \qquad [2]$$

Ornament size s is proportional to the male's investment t into the ornament and to the resources available for reproductive investment $R(q)$, which are assumed to increase with q:

$$s = s(t,q) = t \cdot R(q). \qquad [3]$$

Hence, the ornament is a revealing indicator of a male's ecological performance in a given patch. We took $R(q) = 1+3q$.

Male ornament production and female preference are costly: male and female survival until reproductive age are reduced by a factor $\exp(-\beta t^2)$ and $\exp(-\gamma p^2)$, respectively. We took $\beta = 0.5$ and $\gamma = 0.01$.

GENETICS

The characters x, p and t are each separately encoded by $L = 50$ diploid, di-allelic loci. All loci recombine freely, within and between traits. Mutations occur with a frequency $m = 1 \cdot 10^{-4}$ per allele. Mutations have a phenotypic effect of magnitude $\delta x = 0.04$, $\delta p = 0.03$ and $\delta t = 0.07$ for x, p and t, respectively, such that x, for example, may range from –2 to 2.

OBSERVED REPRODUCTIVE ISOLATION

We varied the strength of disruptive natural selection from weak ($\sigma = 0.95$) to strong ($\sigma = 0.05$). With sexual selection, speciation first occurred at $\sigma = 0.80$. Without sexual selection, speciation only occurred for $\sigma < 0.35$. Moreover, for $0.05 < \sigma < 0.35$, the proportion of hybrid individuals was 23 to 58 times higher than in the presence of sexual selection. In fact, the level of hybridization after speciation without sexual selection was always higher than that after speciation with sexual selection for any value of σ.

The evolution of female preferences for multiple indicators of quality

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G. Sander van Doorn & Franz J. Weissing

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ABSTRACT

In a variety of species, females exhibit preferences for multiple male ornaments. Several hypotheses have been proposed to explain this phenomenon. Which, if any, of these hypotheses is the most plausible in general remains largely unresolved based on the available empirical data. Yet, theoretical studies conclude that the evolution of preferences for multiple signals of male quality is unlikely, especially when the use of an additional cue in mate choice strongly increases the overall cost of choice. This would imply that most male courtship characters do not reflect the male's genetic quality, but evolved through Fisherian sexual selection. However, the existing models focus on ornaments that signal overall genetic quality and do not address the possibility that different ornaments provide information about different aspects of quality. Therefore, we develop a model, in which the ornaments act as signals for distinct quality components. When the ornaments provide overlapping information about these quality components, we retrieve the results of earlier models. However, when the ornaments provide independent information, preferences for multiple ornaments may evolve, even when exhibiting multiple preferences is costly. We discuss our results in relation to the *multiple message* and *redundant signal* hypotheses for ornament diversity and identify parallels between Fisherian and good-genes mechanisms for the evolution of multiple ornaments.

INTRODUCTION.

Male courtship displays often comprise multiple sexual signals, involving several behavioral attributes and/or morphological ornaments. What is more, traits like the peacock's tail, which may appear to be a single ornament to the human observer, may in fact convey a multitude of potentially independent signals, encoded by the length of the tail, the number of ocelli, brightness, symmetry, elaboration and so on. Despite the numerous examples of complex male courtship displays in natural systems, the reasons for this complexity remain unclear. In particular, two questions are unresolved. First, to what extent are the different components of the male courtship display subject to female choice? Second, to what extent do those components give information about the condition of the male or about direct or genetic benefits expected by the female?

Depending on the answers to these questions, the components of the male courtship display can be classified as

- *obsolete signals*, i.e., signals for which female preference has been lost, but which are nonetheless maintained, because they are not costly (Møller & Pomiankowski, 1993) or are needed to achieve threshold levels of stimulation (Holland and Rice, 1998);
- signals intended for *multiple receivers*, i.e., signals that are only partly intended for females, the other components of the display being used in male-male competition (Andersson *et al.*, 2002);
- *unreliable signals*, i.e., signals that do not reflect condition, but which are subject to female preferences shaped by Fisher's runaway process (Møller & Pomiankowski, 1993);
- *redundant signals* (or, *back-up signals*), i.e., signals subject to female mating preferences shaped by the handicap process, all indicating the same overall condition of the male (Møller & Pomiankowski, 1993; Johnstone, 1996);
- *multiple messages*, i.e., signals subject to female mating preferences shaped by the handicap process, each reflecting a different aspect of the overall condition of the male (Møller & Pomiankowski, 1993).

Accurate classification thus requires one to determine: which of the signals are used in female choice; which ones are used in other contexts, such as male-male competition; to what extent each of the signals is an indicator of quality; and, finally, to what extent the different signals reflect different aspects of quality. Not surprisingly, these questions have only been partly addressed in most empirical studies, and different empirical studies lend support to each of the various hypotheses (reviewed by Candolin, 2003).

A study on red junglefowl (Ligon *et al.*, 1998), for example, demonstrated that females use only one of the ornaments (comb size) to choose mates, suggesting that the other head and plumage ornaments are either obsolete signals or signals that are used in other contexts. Similarly, a study on mallards (Omland, 1996*ab*) revealed that females choose males predominantly on the basis of bill characteristics, which are believed to reflect male condition accurately, but that they show only weak preferences for feather ornaments. Evidence for the multiple-receiver hypothesis comes from studies on widowbirds (Pryke *et al.*, 2001; Andersson *et al.*, 2002) and scarlet-tufted malachite sunbirds (Evans & Hatchwell, 1992*a,b*), where only one of the feather ornaments appears to be important in female mate choice, while another is used in aggressive interactions between males. In contrast to these examples, where female choice appears to be restricted to a single male trait, studies on yellow-browed leaf warblers (Marchetti, 1998), guppies (Brooks & Couldridge, 1999; Brooks, 2002) and man (in the context of male rather than female mate choice; Grammer *et al.*, 2001) have demonstrated independent preferences for multiple signals, as predicted by the unreliable-signal, redundant-signal and multiple-message hypotheses.

While the above examples focus on the nature of female mate preferences, other studies have looked into the information content of different male signals. Comparative analysis of feather ornaments in birds (Møller, 1993) has been used to support the unreliable-, or obsolete-signal hypothesis, arguing that male secondary sexual characters in species with multiple ornaments show far weaker condition-dependent expression than those in species with only a single ornament (Møller & Pomiankowski, 1993). Other studies, however, show that multiple male signals involved in female mate choice are dependent on overall condition (redundant-signal hypothesis; Candolin, 2003, and references therein) or on different aspects of condition (multiple-message hypothesis; e.g., Møller & Petrie, 2002; Doucet & Montgomerie, 2003).

Based on the empirical data discussed above, it remains unclear which, if any, of the different hypotheses explaining ornament diversity is the most plausible in general. Accurate discrimination between hypotheses is hampered not only by the lack of data. The interpretation of data is also difficult, due to the fact that it is impossible to distinguish between Fisherian and good-genes sexual selection on the basis of observed patterns of condition-dependence (Kokko *et al.*, 2002).

Notwithstanding this, theoretical models (Schluter & Price, 1993; Pomiankowski & Iwasa, 1993; Iwasa & Pomiankowski, 1994) have come to the clear-cut conclusion that the evolution of female preferences for multiple indicators of good genes is less likely than the evolution of multiple preferences by Fisher's runaway process. This is especially true when there are epistatic interactions between the costs of the different female preferences, such that the overall cost of choice is greatly increased when females assess an additional male trait. Put differently, when considering multiple ornaments is costly, females should evolve preferences for the single indicator of quality with the highest reliability, honesty and detectability, and should disregard ornaments that are less reliable, honest or detectable. According to existing theory, the multiple components of male courtship displays must therefore be *obsolete* or *unreliable signals* that do not (or no longer) reflect condition, and are maintained due to low costs or Fisherian runaway selection.

We see reason to re-evaluate this conclusion, since the existing models have limited applicability. The models assume that all ornaments are indicators of general quality or viability, ignoring the possibility that different ornaments reflect different aspects of condition (but see Johnstone, 1996, for an exception). Therefore, most models cannot be used to evaluate the *multiple-message* hypothesis. Moreover, the models do not take into consideration that different ornaments may provide independent estimates of overall condition, and therefore they are not suited to assess the validity of the *redundant-signal* hypothesis.

In this paper, we attempt to arrive at a more general theory for the evolution of female preferences for multiple ornaments. We extend existing dynamical models for the evolution of multiple sexual ornaments by explicitly considering multiple components of viability. As we demonstrate, by means of individual-based simulations and mathematical analysis, female preferences for multiple male ornaments can evolve, even when multiple preferences are costly, as long as the ornaments provide sufficiently independent information about the underlying quality components.

INDIVIDUAL-BASED SIMULATION MODEL

We simulate the evolution of multiple female sexual preferences and male ornaments in a population consisting of N individuals. Every individual carries alleles for two female preferences and two male ornaments. Expression of these alleles is sex-limited. We use p_1 and p_2 to denote the preference values expressed by a female, and t_1 and t_2 to denote the amount of energy or resources invested into ornaments by a male. In addition, individuals carry quality alleles determining two independent quality components, A and B. These represent, for example, tolerance to harmful substances in the environment and resistance against parasites. An individual's phenotypic values for the quality components, denoted as q_A and q_B ($0 \le q_A \le 1$, $0 \le q_B \le 1$), affect male and female viability as well as male attractiveness.

MATE CHOICE

The phenotypic characters t_1 and t_2 are expressed relative to some value optimal for male survival, such that also negative values of t_1 and t_2 are biologically meaningful. For the sake of concreteness, let us suppose that t_1 and t_2 affect the sizes s_1 and s_2 of two male ornaments, again expressed relative to the value optimal for male survival. As we will explain shortly, the realized size of an ornament is determined not only by a male's investment into this ornament, but also by the quality of the male.

Before a female mates, she evaluates the available males based on the size of their ornaments and her own preferences. Females have a higher probability of mating with an 'attractive' male, where 'attractiveness' is quantified by a function

r. In line with earlier models (Pomiankowski & Iwasa, 1993; Iwasa & Pomiankowski, 1994), we assume that

$$r = \exp(p_1 s_1 + p_2 s_2). \qquad [1]$$

For this mate-choice model, females with larger absolute values of p_1 and p_2 have stronger preferences. Positive values of p_1 and p_2 indicate preferences for larger ornaments, negative values of p_1 and p_2 indicate preferences for smaller ornaments, and females with $p_1 = p_2 = 0$ mate at random. Female preferences are assumed to be non-overlapping. That is, when females evolve a single preference (e.g., $p_1 \neq 0$ and $p_2 = 0$), only the corresponding ornament (s_1, and not s_2) affects male attractiveness. A female mates only once. The probability that a given male is allowed to sire her offspring is given by his attractiveness relative to the average attractiveness of other males she encounters.

REVEALING INDICATORS OF QUALITY

The male ornaments are revealing indicators of quality. For all males, the realized size of an ornament is proportional to the investment into that ornament. Yet, low-quality males must invest more to attain the same level of ornament elaboration and, hence, attractiveness. Specifically, we assume that

$$s_i = t_i \, \alpha_i \qquad (i = 1 \text{ or } 2), \qquad [2]$$

where the coefficient α_i reflects how efficiently male investment into ornament i translates into increased mating success. In accordance with the preceding discussion, the coefficient α_i varies with the male's phenotype for the quality components A and B. Unlike other models (Grafen, 1990; Iwasa & Pomiankowski, 1994), we do not consider the possibility that also t_i, the amount of resources invested into the ornament, is different for males with different qualities. Although biologically relevant, such condition-dependent ornament expression is not essential for a revealing handicap mechanism to work (as formally demonstrated by Iwasa *et al.*, 1991). For the sake of simplicity, condition-dependent ornament expression is therefore presently left out of consideration (see also the discussion section).

An ornament is a revealing indicator when the coefficient α_i increases with male quality q_A and/or q_B. To model this dependency, we define four parameters, α_i^{AB}, α_i^{Ab}, α_i^{aB} and α_i^{ab}, for each ornament ($i = 1 \text{ or } 2$), which give the efficiency of ornament production for males with the lowest or highest possible value for each of the quality components. For example, we assume that a male with the highest possible value for quality component A ($q_A = 1$), and the lowest possible value for quality component B ($q_B = 0$), expresses the first ornament with efficiency α_1^{Ab} and the second ornament with efficiency α_2^{Ab}. Similarly, $\alpha_i = \alpha_i^{ab}$ for males that have the lowest possible quality on both quality components ($q_A = q_B = 0$). All else being equal, high-quality males are more attractive than low-quality males, or, at the very least, equally attractive. This implies $\alpha_i^{AB} \geq \alpha_i^{Ab} \geq \alpha_i^{ab}$ and $\alpha_i^{AB} \geq \alpha_i^{aB} \geq \alpha_i^{ab}$.

For males with intermediate qualities ($0 < q_A < 1,\ 0 < q_B < 1$), the efficiency of ornament production is simply given by the weighted average of the efficiencies for extreme males, such that, in general,

$$\alpha_i = q_A q_B \alpha_i^{AB} + q_A (1 - q_B) \alpha_i^{Ab} + (1 - q_A) q_B \alpha_i^{aB} + (1 - q_A)(1 - q_B) \alpha_i^{ab} \ . \qquad [3]$$

For example, a male with intermediate quality for both quality components ($q_A = q_B = 1/2$) will be able to express the second ornament with efficiency $(\alpha_2^{AB} + \alpha_2^{Ab} + \alpha_2^{aB} + \alpha_2^{ab})/4$.

The values of the parameters α_i^k ($k = AB, Ab, aB$ or ab) are determined by a variety of biological factors, such as female perception, the physiology of male ornament production and the ecology of mate choice. All of these factors affect how rapidly male attractiveness increases with increased male investment into the ornament and how easily females can detect quality differences between males based on an ornament. To illustrate this, let us assume that, for a given species and a given ornament the parameters α_i^k are given by

$$\begin{aligned} \alpha_1^{AB} &= 2\,, & \alpha_1^{aB} &= 1\,, \\ \alpha_1^{Ab} &= 1.98\,, & \alpha_1^{ab} &= 0.99\,. \end{aligned} \qquad [4]$$

With equal investment into their first ornament, a male with a high quality for component A will produce an ornament twice the size of that produced by a male with a low quality for the same component. Consequently, even a low level of preference for the first ornament will allow females to discriminate accurately between high- and low-quality males for component A. In contrast, the first ornament provides hardly any information about the male's quality for component B. Two males that differ only in their quality for component B will produce ornaments of roughly the same size, no matter how big the quality difference. Therefore, females will not be able to discriminate between high- and low-quality males for component B on the basis of the first ornament, unless they evolve extremely high levels of preference for this ornament.

As illustrated by the example, ornaments can be reliable indicators for one component of quality, but unreliable indicators for another component of quality. This has important consequences. It is possible to order ornaments with respect to their reliability (i.e., the detectability of quality differences) as long as only one quality component is considered. This is impossible, however, when quality is determined by several independent components, since the most reliable ornament for one component of quality need not be the most reliable indicator for another quality component. In such a case, the conclusion that females will evolve to ignore everything but the most reliable ornament (Schluter & Price, 1993) cannot be applied, since it is impossible to identify a single most reliable ornament. Of course one could attempt to derive alternative ways to order the ornaments, for example based on the fitness effects associated with the ornaments. However, such a classification would be extremely difficult to establish, since it would strongly depend on the frequency distribution of ornaments and preferences in the population.

Moreover, as we will demonstrate in this paper, evolution will not necessarily lead to a mating preference that is solely based on the ornament with the highest associated fitness benefit.

VIABILITY SELECTION

After all females have mated, they produce a new generation of offspring. For simplicity, we assume that generations are discrete and non-overlapping. Viability selection acts at the start of every generation.

Individual survival probabilities vary with the individual's general (i.e., good-genes induced) viability, v, which is taken to depend on the quality components q_A and q_B. Analogously to the procedure followed for the coefficients of attractiveness α_i, we define parameters v_{AB}, v_{Ab}, v_{aB} and v_{ab}, which correspond to the general viabilities of extreme individuals, i.e., individuals with the highest and lowest possible qualities. For individuals with intermediate qualities, v is given by

$$v = q_A\, q_B\, v_{AB} + q_A \left(1-q_B\right) v_{Ab} + \left(1-q_A\right) q_B\, v_{aB} + \left(1-q_A\right)\left(1-q_B\right) v_{ab}\,. \qquad [5]$$

In addition, individual survival probabilities are affected by the costs associated with the expression of preferences (for females) or ornaments (for males). These factors are assumed to interact multiplicatively with general viability. Consequently, male and female survival probabilities, denoted as h_m and h_f, respectively, are given by

$$h_m = v\left(1-c_m\right) \quad \text{and} \quad h_f = v\left(1-c_f\right), \qquad [6]$$

where c_m represents the cost of ornament production and c_f represents the cost of choice.

In line with the assumption that the ornaments are revealing indicators of quality, the cost of expressing an ornament is taken to be independent of a male's quality, and solely determined by the male's investment of resources into the ornament. Consequently, we take

$$c_m = 1 - \exp\left(-\beta_1\, t_1^2 - \beta_2\, t_2^2\right), \qquad [7]$$

such that, in the absence of sexual selection, $t_1 = t_2 = 0$ is the optimal investment into the ornaments. The parameters β_i determine the intensity of stabilizing selection on male investment into the ornament. Note that the costs of expressing the two ornaments interact multiplicatively, implying that the cost of each ornament has an independent effect on fitness.

As mentioned in the introduction, previous studies (Pomiankowski & Iwasa, 1993; Iwasa & Pomiankowski, 1994) indicate that epistatic interactions between the separate costs of choice can qualitatively affect the evolutionary outcome. Therefore, we assume that

$$c_f = 1 - \exp\left(-\gamma_1\, \theta_{12}\, p_1^2 - \gamma_2\, \theta_{21}\, p_2^2\right), \qquad [8]$$

where the constants γ_i determine how rapidly the costs of choice increase with the two preferences, and where the coefficients θ_{ij} modify the cost of choice for ornament i depending on the level of preference for the other ornament. In our simulations, we use

$$\theta_{ij} = \exp\left(\vartheta_i\, p_j^{\,2}\right). \tag{9}$$

When $\vartheta_i > 0$, the cost of evolving a preference for ornament i increases with the level of choosiness for the other ornament. In that case, females that exert mate choice on the basis of two male ornaments face severe costs. When $\vartheta_i < 0$, by contrast, evaluating an ornament becomes less costly with increasing preference for the other ornament. The preferences have independent effects on fitness when $\vartheta_i = 0$. In all cases, random mating ($p_1 = p_2 = 0$) minimizes the cost of choice.

GENETICS

Individuals are diploid. The male traits t_1 and t_2, as well as the female preferences p_1 and p_2, are each separately determined by a single locus. We assume a continuum of alleles at these loci; that is, the phenotypic effect of each allele is a continuous quantity. Male trait alleles interact additively to determine the phenotypic trait value, as do female preference alleles to determine the preference value. Mutations at trait and preference loci occur with probability m per allele per generation, and are modeled by altering the phenotypic effect of an allele by a number drawn from a normal distribution with narrow width σ_m.

Each of the two quality components q_A and q_B is separately encoded by L di-allelic loci. At every quality locus there may either be a high-quality or a low-quality allele. The quality alleles interact additively; that is, every low-quality allele decreases the individual's quality by an amount $1/(2L)$. Hence, the phenotypic qualities q_A and q_B simply represent the fraction of high-quality alleles at loci for quality component A and B, respectively. Variation at the quality loci is maintained through biased deleterious mutations, which occur with probability μ per allele per generation. Beneficial mutations, converting a low-quality allele into a high-quality allele occur with probability ν per allele per generation, where $\nu \ll \mu$.

All genes are unlinked and are transmitted according to normal Mendelian genetics.

INDIVIDUAL-BASED SIMULATION RESULTS

We start by investigating a situation in which both ornaments are equally revealing indicators for both quality components. In this case, the two quality components jointly influence the size of each of the male ornaments and, crucially, they do so in the same way for both ornaments. In essence, this makes the distinction between the two quality components arbitrary, and, therefore, we would expect the same evolutionary outcome as in a model where only a single quality component is considered.

The individual-based simulations confirm this expectation. When the two ornaments completely overlap in the information they provide about the two quality components, the evolutionary outcome is mainly determined by the joint cost of choice. If the joint cost of choice is low ($\vartheta_i < 0$), such that preference for an ornament becomes less costly as a female exhibits stronger preferences for the other ornament, preferences for multiple ornaments readily evolve (FIGURE 1A). However, when the joint cost of choice is high ($\vartheta_i > 0$), such that the separate costs of choice combine in a super-multiplicative manner and females face severe costs when they consider multiple ornaments, preference for only one of the ornaments evolves (FIGURE 1B).

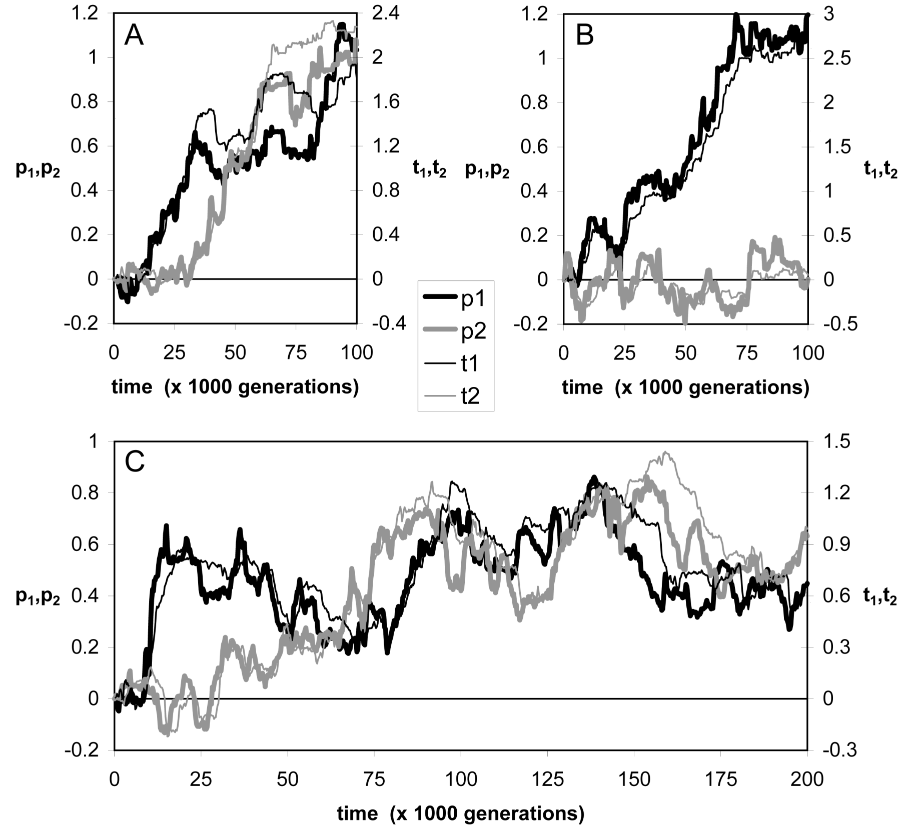

FIGURE 1 – INDIVIDUAL-BASED SIMULATIONS

In panels (A) and (B), the ornaments provide overlapping information about the quality components. Multiple preferences evolve in (A) but not in (B), due to the fact that the joint cost of choice is low in (A) but high in (B). In panel (C), the joint cost of choice is high, as in (B), but the ornaments now provide independent information about the quality components. Despite the high joint cost of choice, multiple preferences evolve. See TABLE 1 for parameter values.

These results are not surprising. When a single ornament provides full information about all components of quality, a female that exerts a preference for a second ornament will not obtain additional information about the male's genetic quality. Compared with a female that expresses only a single preference, she pays a different price to obtain the same information. Whether or not multiple ornaments evolve will therefore mainly depend on whether or not the costs of multiple preferences are lower than the costs of choice in case of a single preference. This will only be the case if the joint cost of choice is low.

TABLE 1 – PARAMETER VALUES USED IN THE SIMULATIONS

figure	α_1^{AB}	α_1^{Ab}	α_1^{aB}	α_1^{ab}	α_2^{AB}	α_2^{Ab}	α_2^{aB}	α_2^{ab}	ϑ_1	ϑ_2
1A[a]	3.0	1.5	1.5	0.5	3.0	1.5	1.5	0.5	-0.25	-0.25
1B	3.0	1.5	1.5	0.5	3.0	1.5	1.5	0.5	0.25	0.25
1C	2.0	2.0	0.5	0.5	2.0	0.5	2.0	0.5	0.25	0.25
2A[b]	2.0	2.0	2.0	-[c]	3.0	3.0	3.0	-	1.0	1.0
2B	2.0	2.0	2.0	-	3.5	3.0	3.0	-	1.0	1.0
2C	3.0	2.0	2.0	-	4.5	3.0	3.0	-	1.0	1.0
2D	4.0	2.0	2.0	-	4.5	3.0	3.0	-	1.0	1.0
2E	4.0	3.0	2.0	-	4.5	1.5	4.0	-	1.0	1.0
2F	3.2	3.2	0.0	-	2.0	0.8	0.2	-	1.0	1.0

a) Other parameters in FIGURE 1 were $N = 1000$, $\beta_1 = \beta_2 = 0.5$, $\gamma_1 = \gamma_2 = 0.01$, $v_{AB} = 1.0$, $v_{Ab} = v_{aB} = 0.5$, $v_{ab} = 0.0$, $m = 0.001$, $\sigma_m = 0.05$, $L = 10$, $\mu = 0.01$, $\nu = 0.0001$.

b) Other parameters in FIGURE 2 were $\mu_A = 0.06$, $\mu_B = 0.05$, $\beta_1 = 0.4$, $\beta_2 = 0.6$, $\gamma_1 = 0.07$, $\gamma_2 = 0.04$, $v_{AB} = 1.0$, $v_{Ab} = 0.7$, $v_{aB} = 0.8$.

c) The parameters α_i^{ab} are irrelevant for the analytical model.

The preceding cost-benefit analysis becomes less straightforward when the two ornaments provide different information about the two quality components. For example, let us suppose that the first ornament provides information about the first quality component but not about the second quality component. Similarly, the second ornament provides information about the second quality component, but not about the first. Let us again compare two females, the first one exhibiting preferences for a single ornament, the second exhibiting preferences for both ornaments. It is clear that the two females not only pay different costs, but also obtain different benefits. The first female obtains information about one of the quality components only. Hence, she runs the risk of selecting a mate that has low quality on the quality component she neglects to evaluate. The second female, on the other hand, obtains full information about the male's quality. She will always be able to select a high-quality mate.

Of course, it remains to be shown that the benefits of exhibiting multiple preferences can also outweigh the costs, even when the joint cost of choice is high. In FIGURE 1C, we simulated the evolution of preferences for two ornaments that, as in our example, provided non-overlapping quality information, with a high joint cost of choice, exactly as in FIGURE 1B. Multiple female preferences evolve, illustrating that, for the parameters used in this simulation, the benefit of exhibiting multiple preferences outweighs the high joint cost of choice.

MATHEMATICAL ANALYSIS

Due to the stochasticity observed in simulations, it is difficult to characterize thoroughly the behavior of the model for a wide range of parameter conditions. Therefore, we approximate the dynamics of our stochastic, individual-based model by deterministic equations. The latter can be analyzed mathematically, yielding further insights into the evolution of multiple female preferences for indicators of independent quality components. To enable this complementary treatment, we make a number of simplifying assumptions.

We concentrate on a genetically simplified version of our simulation model. We assume that individuals are haploid, and that each of the two quality components is determined by a single locus with two alleles (A and a for the first quality component; B and b for the second). The alleles A and B confer high quality for their respective quality components; alleles a and b confer low quality.

The female preferences and the male ornaments may be based on any arbitrary number of loci. However, we do assume that the genes coding for preferences and ornaments mutate only rarely. This allows us to focus on a population with negligible variation for female preference and for male investment into ornaments. Specifically, we assume that almost all individuals carry 'resident' alleles coding for the resident phenotype, whilst a tiny fraction of the individuals carries a mutant allele at one of its preference or ornament loci.

DYNAMICS OF GENOTYPE FREQUENCIES AT QUALITY LOCI

Under these simplifications, we need to keep track of four quality genotype frequencies for both resident and mutant individuals. As derived in APPENDIX A, the dynamics of these genotype frequencies can be described by a system of recurrence equations, given by

$$\begin{aligned}\vec{z}_{n+1} &= \mathbf{M}\hat{\mathbf{T}}(\vec{z}_n)\vec{z}_n\,,\\ \vec{\varepsilon}_{n+1} &= \mathbf{M}\mathbf{T}(\vec{z}_n)\vec{\varepsilon}_n\,.\end{aligned} \qquad [10]$$

Here, $\vec{z}_n$ and $\vec{\varepsilon}_n$ represent vectors containing the genotype frequencies in generation n at the viability loci for the resident and the mutant, respectively. The matrix $\mathbf{M}$ incorporates the effects of biased mutations of the quality genotypes. The matrices $\hat{\mathbf{T}}(\vec{z}_n)$ and $\mathbf{T}(\vec{z}_n)$ are transition matrices that incorporate the effects of viability selection and non-random mating, exactly as described for the individual-based simulation model. The elements of $\hat{\mathbf{T}}(\vec{z}_n)$ and $\mathbf{T}(\vec{z}_n)$ vary with the quality genotype frequencies of the resident, since these determine the availability of mating partners. In addition, the matrix elements vary with female preferences and male investment into ornaments. The mutant transition matrix $\mathbf{T}(\vec{z}_n)$ depends on the mutant phenotype, but also on the resident phenotype, since mutants interact with resident individuals during mate choice. The resident transition matrix $\hat{\mathbf{T}}(\vec{z}_n)$ depends only on the resident phenotype, since resident individuals interact predominantly with other resident individuals.

The system of equations [10] is non-linear in the resident genotype frequencies, but linear in the mutant genotype frequencies. Given that the resident genotype frequencies at the quality loci attain a stable equilibrium distribution $\vec{z}^*$ after some time, we may apply standard stability analysis to find the long-term growth rate of the mutant population. The latter is determined by the dominant eigenvalue λ of the matrix $\mathbf{MT}(\vec{z}^*)$. When $|\lambda|>1$, the mutant density will increase until, in most cases, the mutant has replaced the former resident. When $|\lambda|<1$, the mutant will disappear after some time. Hence, λ can be used as a measure for the mutant's invasion fitness (Metz *et al.*, 1996).

EVOLUTIONARY DYNAMICS

Under suitable assumptions (Weissing, 1996), evolution can be described as a sequence of mutation and trait-substitution events. The invasion fitness may be used not only to predict the outcome of a single step in this sequence, but also to predict the direction and rate of the process as a whole. In other words, from the mutant's invasion-fitness function, we may derive equations describing the evolutionary dynamics of the phenotypic characters of our model. Starting from equation [10], this procedure is outlined in detail in APPENDIX B. Here, we only give the end result, consisting of two equations for the evolutionary rate of change in male investment into the ornament and two equations for the evolutionary rate of change in female preference. The former are as follows:

$$\frac{dt_i}{d\tau}=\frac{1}{2}\frac{\partial \ln\left(\left(1-c_m\right)r_{AB}\right)}{\partial t_i} \qquad \left(i=1 \text{ or } 2\right), \tag{11}$$

where τ represents evolutionary time. Male ornament sizes will change until a stable equilibrium is reached. At equilibrium $dt_i/d\tau=0$, which, in view of [11], implies that the equilibrium amount of resources invested into the ornaments maximizes the product of male survival (represented by $1-c_m$) and attractiveness for the most viable males (represented by r_{AB}; cf. equation [1]-[3]). The attractiveness of low-quality males (r_{Ab}, r_{aB} and r_{ab}) does not enter into the equations for male investment into ornaments, since low-quality males occur at low frequencies only.

The equations for the evolutionary rate of change in female preference are given by

$$\frac{dp_i}{d\tau} = \frac{1}{2}\frac{\partial \ln\left(1-c_f\right)}{\partial p_i} + \mu_A w_A \frac{\partial \ln\left(R_A\right)}{\partial p_i} + \mu_B w_B \frac{\partial \ln\left(R_B\right)}{\partial p_i}. \qquad [12]$$

In these equations, μ_A and μ_B denote the rate of deleterious mutations for quality components A and B, respectively. The coefficients w_A and w_B denote the reproductive values of males carrying a deleterious mutation at one of the quality loci. The functions R_A and R_B, which vary with the preferences present in the resident population, represent the attractiveness of high-quality males relative to the attractiveness of males carrying a deleterious mutation, i.e.,

$$R_A = \frac{r_{AB}}{r_{aB}}, \quad R_B = \frac{r_{AB}}{r_{Ab}}. \qquad [13]$$

According to equation [12], female choosiness evolves to a level at which the costs and benefits of choice are balanced. When there is no variation among males (no mutation bias, $\mu_A = \mu_B = 0$), female preference will evolve to maximize $1-c_f$, that is, the cost of choice will be minimized. When mutation bias creates variation among males, the female preferences will evolve away from their respective viability-selection optima to a point where $\partial \ln\left(1-c_f\right)/\partial p_i$ is negative, provided that $\partial \ln\left(R_A\right)/\partial p_i$ and $\partial \ln\left(R_B\right)/\partial p_i$ are sufficiently larger than zero.

The terms $\partial \ln\left(R_k\right)/\partial p_i$ ($k = A$ or B) have an interesting biological interpretation. For females that mate randomly, low-quality males are, by definition, as attractive as high-quality males. To females that exhibit stronger preferences, however, high-quality males will appear increasingly attractive. Therefore, R_A and R_B are monotonically increasing functions of p_1 and p_2. Ornaments may differ in the rates at which R_A and R_B increase with female preference. Exactly these differences are quantified by the terms $\partial \ln\left(R_k\right)/\partial p_i$, which represent the rate at which the relative attractiveness of high-quality males (for quality component k) increases with female preference for ornament i. If $\partial \ln\left(R_k\right)/\partial p_i$ is only slightly larger than zero, the relative attractiveness of high-quality males increases only slowly with preference, and females must evolve high levels of choosiness before they can accurately distinguish high-quality from low-quality males. In this situation, we say that the 'information content' of ornament i is low for quality component k, meaning that the ornament i is a poor revealing indicator for quality component k. On the other hand, when $\partial \ln\left(R_k\right)/\partial p_i$ is large, even low levels of choosiness will allow females to select males with high quality for quality component k. In this case, we say that the ornament i has a high information content for quality component k.

It is a direct consequence of [12] that a female preference p_i will converge on its viability selection optimum (maximal $1-c_f$) when the corresponding ornament is not a revealing indicator for at least one component of quality (i.e., when $\partial \ln\left(R_A\right)/\partial p_i = \partial \ln\left(R_B\right)/\partial p_i = 0$).

A SPECIFIC EXAMPLE

For the sake of concreteness, let us now choose, as in the individual based simulation model,

$$\begin{aligned} r_k &= \exp\left(\alpha_1^k\, p_1\, t_1 + \alpha_2^k\, p_2\, t_2\right), \\ c_m &= 1 - \exp\left(-\beta_1\, t_1^2 - \beta_2\, t_2^2\right), \\ c_f &= 1 - \exp\left(-\gamma_1\, \theta_{12}\, p_1^2 - \gamma_2\, \theta_{21}\, p_2^2\right), \end{aligned} \qquad [14]$$

with $k = ab, aB, Ab$ or AB. For simplicity, we consider only non-negative values of preference and male investment into ornaments from here on, i.e., $p_1 \geq 0$, $p_2 \geq 0$, $t_1 \geq 0$, $t_2 \geq 0$. As in the individual-based simulations, the costs of exhibiting a preference for one of the ornaments may depend on the level of choosiness for the other ornament. Therefore, the baseline costs of choice, γ_i, are modified by the coefficients for the joint cost of choice, θ_{12} and θ_{21}, which are allowed to vary with p_2 and p_1, respectively (as in equation [9]).

Equation [11] now reduces to a simple equation, with equilibrium solutions (denoted by tildes)

$$\tilde{t}_i = \frac{1}{2}\frac{\alpha_i^{AB}}{\beta_i}\tilde{p}_i. \qquad [15]$$

Substituting these equilibrium solutions into [12] eventually yields two equations, from which the equilibrium values for the female preferences can be solved (APPENDIX C). With $i = 1$ or 2, $j = 1$ or 2 and $j \neq i$, these equations are given by

$$\tilde{p}_i\, \gamma_i\, \theta_{ij} + \vartheta_j\, \gamma_j\, \tilde{p}_i\, \tilde{p}_j^2\, \theta_{ji} = \mu_A\, \tilde{w}_A\, \kappa_i^A\, \tilde{p}_i + \mu_B\, \tilde{w}_B\, \kappa_i^B\, \tilde{p}_i\,. \qquad [16]$$

The left-hand side of this equation represents the costs of choice, the right-hand side the benefits of choice. As indicated by the tilde, the reproductive values $\tilde{w}_A$ and $\tilde{w}_B$ are functions of the equilibrium preference values. The constants κ_i^k ($k = A$ or B) derive from the terms $\partial \ln\left(R_k\right)/\partial p_i$. They measure the information content of the ornaments, i.e., the amount of information that ornament i gives about quality component k. To be exact,

$$\kappa_i^A = \frac{\alpha_i^{AB}\left(\alpha_i^{AB} - \alpha_i^{aB}\right)}{2\beta_i}, \quad \kappa_i^B = \frac{\alpha_i^{AB}\left(\alpha_i^{AB} - \alpha_i^{Ab}\right)}{2\beta_i}. \qquad [17]$$

EQUILIBRIA AND THEIR STABILITY

The solutions of the equilibrium conditions [15] and [16] fall into three categories. We always find a trivial equilibrium ($\tilde{p}_1 = \tilde{p}_2 = \tilde{t}_1 = \tilde{t}_2 = 0$) at which females mate at random. In addition, we may find boundary equilibria, at which females exhibit a preference for just one of the ornaments, and internal equilibria, at which females exhibit preferences for multiple ornaments.

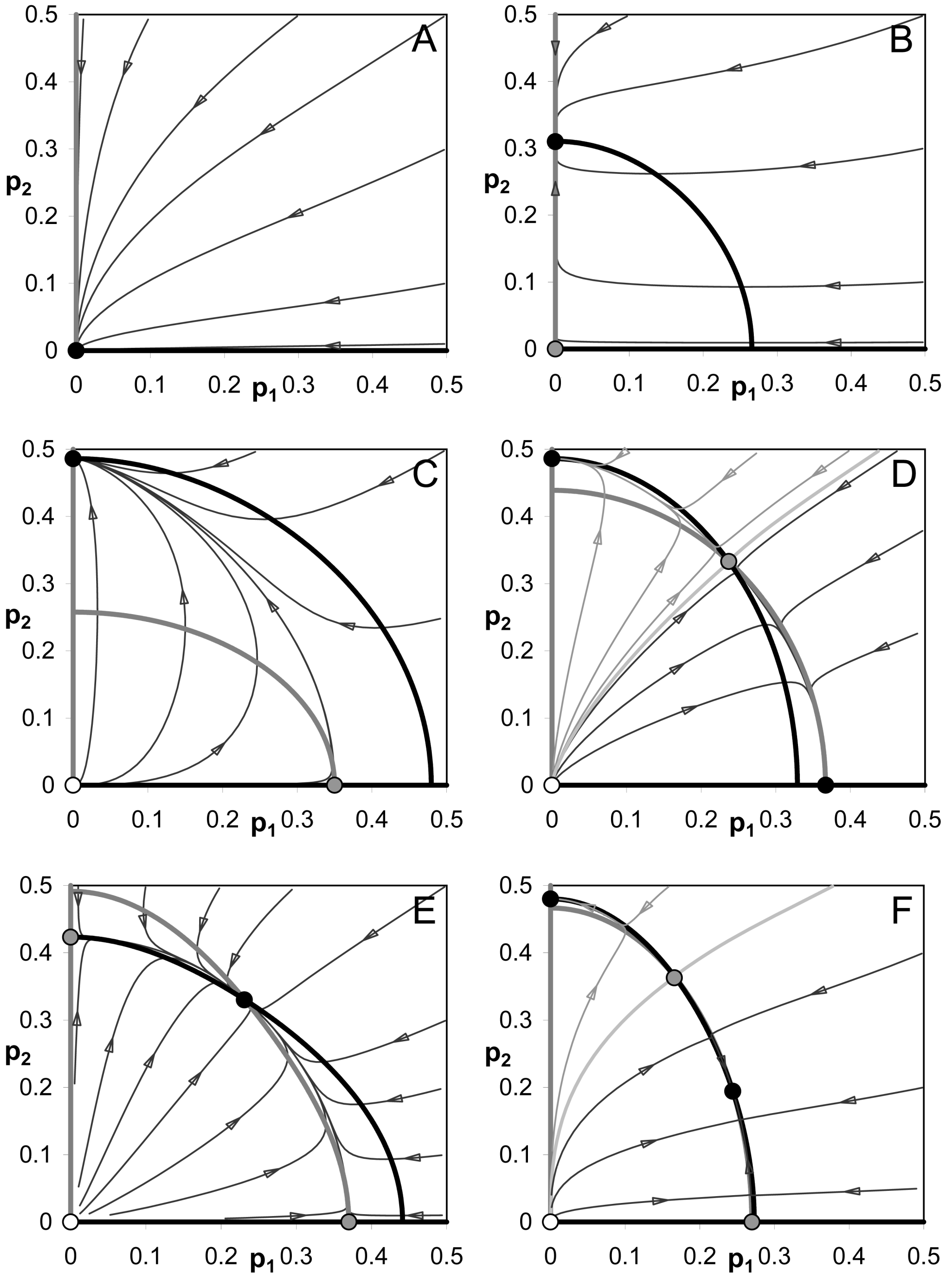

FIGURE 2 – Qualitatively different outcomes of the model

The panels (A)-(F) represent projections of the four-dimensional trait space of our model, in which information about the male characters is not shown. Each phase portrait shows projected trajectories, computed for the full four-dimensional system (lines with arrows); null-clines, computed using a quasi-steady-state assumption for the male characters (thick gray lines: p_1-isocline; thick black lines: p_2-isocline); and equilibria (open circles: unstable nodes; gray-filled circles: saddle points; black-filled circles: stable nodes). If there exist multiple stable equilibria (D, F), a thick light-gray line indicates the boundary between the basins of attraction of the equilibria. See TABLE 1 for parameter values.

The trivial equilibrium is stable when the information content of both ornaments is low (APPENDIX C), such that the benefits of mate choice (the right-hand side of [16]) do not outweigh the costs of choice (the left-hand side of [16]). A representative phase portrait for this case is shown in FIGURE 2A. FIGURE 2 depicts projections of the four-dimensional phase space of our model, in which information about the dynamics of the male characters t_1 and t_2 is not shown. These projections, however, provide nearly complete information about the dynamics, since the male characters evolve on a timescale much faster than that on which female preferences change (provided that the mutation biases and the costs of choice are small). Consequently, t_1 and t_2 are always close to a quasi-equilibrium value, defined by [15].

When the information content of the ornaments is sufficiently high (APPENDIX C), the trivial equilibrium loses its stability and the system may evolve to a boundary equilibrium, at which females exert a preference for one ornament only. We find one (FIGURE 2B) or two (FIGURE 2C-E) boundary equilibria, depending on whether the benefits of choice outweigh the costs for only one ornament or for both ornaments. In FIGURE 2C, female preferences always evolve to the same boundary equilibrium, as one would expect for the situation in which one of the ornaments is a superior indicator of quality (Schluter & Price, 1993). Indeed, for the parameters used in FIGURE 2C, the second ornament has a higher information content for both quality components.

When both ornaments provide comparable net benefits (as in FIGURE 2D-E), we find an internal equilibrium. It is already known that multiple preferences can be stable when the joint cost of choice is low ($\vartheta_i < 0$), even when the two ornaments provide completely overlapping information about quality (FIGURE 1A; Iwasa & Pomiankowski, 1994). Therefore, we assume, from here on, that the joint cost of choice is high ($\vartheta_i > 0$). Under these conditions, multiple preferences are unstable if the ornaments provide overlapping information about the two quality components (FIGURE 2D, FIGURE 1B; Iwasa & Pomiankowski, 1994). However, as soon as the ornaments provide sufficiently independent information about the two quality components, the internal equilibrium becomes stable and multiple preferences can evolve (FIGURE 2E, FIGURE 1C). If an internal equilibrium exists, it is unique in most cases. For a narrow range of parameters, however, we may find a phase portrait like that in FIGURE 2F, with multiple internal equilibria. Multiple internal equilibria occur when one ornament (ornament 1 in FIGURE 2F) is a highly reliable indicator for one quality component, but a poor indicator for the other quality component, whereas the other ornament (ornament 2 in FIGURE 2F) is a mediocre indicator for both quality components. Depending on the initial conditions, the population will then either end up in a boundary equilibrium, where females assess only the mediocre indicator for both quality components, or in an internal equilibrium, where females assess both ornaments.

Clearly, the existence of an internal equilibrium, its stability and the stability of the two boundary equilibria, depend on the costs and benefits of choice, particularly on the joint cost of choice and the degree of overlap in the information content of the two ornaments (APPENDIX C). To investigate further the conditions under which multiple preferences are stable, we systematically varied the information contents of the two ornaments, thereby changing the benefits of choice for the two ornaments as well as the extent to which they provide independent information about quality. This analysis revealed that multiple preferences are stable for a wide range of parameter conditions (APPENDIX D). Even when the ornaments provide considerably different benefits of choice, females may not only evolve preferences for the superior ornament (as predicted by Schluter & Price, 1993), but also for the inferior ornament, as long as the ornaments provide sufficiently independent information. This conclusion does not only hold for the specific model studied in this paper. Under quite weak and general assumptions it can be shown that our conclusions apply to more complex mate-choice scenarios (APPENDIX E).

DISCUSSION

Our analysis demonstrates that the scope for the evolution of multiple female preferences is wider than previously reported. Female preferences for multiple indicators of quality may evolve not only when the joint cost of choice is low, but also when the different ornaments provide information about different components of quality. In addition to these possibilities (see APPENDIX E), multiple preferences can evolve when increasing preferences yield diminishing returns in terms of the benefits of choice, as would be the case when the costs of ornament production rise sharply (Johnstone, 1996). We therefore conclude that there is no *a priori* reason to rule out the *multiple-message* hypothesis as a suitable hypothesis for the evolution of female preferences for multiple male ornaments.

REDUNDANT SIGNALS AND MULTIPLE MESSAGES

Throughout this paper, we have interpreted the two quality characters A and B of our model as independent components of an individual's quality. Under this interpretation, ornaments that reveal non-overlapping information about the quality characters convey 'multiple messages', as proposed by the *multiple-message* hypothesis. At the same time, however, the two quality characters A and B jointly determine an individual's general viability, and they might therefore be interpreted as independent estimates of an individual's overall genetic quality, as proposed by the *redundant-signal* (or *back-up-signal*) hypothesis.

This illustrates that it is almost impossible to distinguish between a *multiple-message* and a *redundant-signal* mechanism. Indeed, in the original formulation of the two hypotheses (Møller & Pomiankowski, 1993), the distinction between these hypotheses is blurred and it depends on the level of description whether ornaments should be considered as multiple messages or as redundant signals. In fact, one can only distinguish between the two hypotheses if one is able to demonstrate that different females weigh the information provided by the different ornaments in a different manner (this would be indicative of a *multiple-message* mechanism). On the basis of the current model, which does not address this level of complexity, we may therefore extend our conclusions to encompass the *redundant-signal* hypothesis; that is to say, multiple preferences can evolve - even when the joint of choice is high - when the ornaments provide independent estimates of an individual's overall genetic quality.

This conclusion may have implications for the interpretation of empirical results. First, the extensive evidence for the *multiple-message* hypothesis could as well be interpreted as evidence in support of the *redundant-signal* hypothesis, for which evidence is scarce (Candolin, 2003). This is because it is usually impossible to distinguish between the case in which the viability components defined by the human observer are functionally relevant for the choosing female (*multiple-message* hypothesis), and the case in which they merely serve as correlates of some underlying quantity the female wishes to estimate (*redundant-signal* hypothesis).

Second, the *redundant-signal* hypothesis has predominantly been tested by investigating whether different components of the male courtship display are positively correlated with one another (Candolin, 2003). The presence of such a positive correlation among male courtship traits is then interpreted as supporting the *redundant-signal* hypothesis, whereas the absence of such a correlation, or even a negative correlation, is interpreted as evidence against the *redundant-signal* hypothesis. Our results indicate that multiple ornaments can evolve as redundant signals, but only when the ornaments are independent indicators of quality. Under a *redundant-signal* mechanism, we would therefore expect the expression of any individual male courtship trait to be correlated with overall quality. However, we would expect a weak correlation, or no correlation at all among the different traits. We would not expect to find strong positive correlations among traits, since this would imply that the male indicator traits do not provide independent estimates of overall quality. This suggests that testing the redundant signal hypothesis based on the pattern of correlations among male courtship traits, as discussed above, may be problematic.

HANDICAPS VERSUS FISHERIAN TRAITS

In their papers on the evolution of multiple ornaments, Iwasa and Pomiankowski (Pomiankowski & Iwasa, 1993; Iwasa & Pomiankowski, 1994) argue that sexual traits in species with multiple ornaments evolved predominantly through Fisherian runaway sexual selection, and not via the handicap process. This idea is based on their conclusion that multiple female preferences can evolve for Fisherian traits but not for handicaps (assuming that the joint cost of choice is high). Given the fact that the distinction between Fisherian and handicap models of sexual selection is merely conceptually useful (Kokko *et al.*, 2002), this conclusion is surprising.

Iwasa and Pomiankowski's conclusion derives from a comparison between a Fisherian (Pomiankowski & Iwasa, 1993), and a handicap model (Iwasa & Pomiankowski, 1994) for the evolution of multiple preferences. Together with the difference in the mechanism of sexual selection (Fisherian versus handicap sexual selection), these models differ also in another aspect: in the Fisherian model, two male characters are subject to independent biased-mutation processes, but in the handicap model, a single biased-mutation process, acting on general viability, causes variation in the expression of both male ornaments. Although it is biologically reasonable to suppose that mutation acts differently on Fisherian traits versus handicaps, it is desirable, from a mathematical point of view, to decouple the assumptions on the mechanism of sexual selection from the assumptions on the mutation process. This allows one to determine whether the different outcome of the two models is explained by the mere fact that one is a Fisherian model and the other a handicap model or whether perhaps the different assumptions on the mutation process are responsible for the observed difference in the outcome of the models.

In this paper, we have demonstrated that female preferences can evolve for multiple handicaps if the male ornaments provide independent information about different components of quality. In this scenario, expression of the male ornaments is subject to independent biased-mutation processes - exactly as in the Fisherian runaway model of Pomiankowski & Iwasa (1993) - due to the fact that the quality components underlying those ornaments are separately and independently affected by mutation bias. Along the same lines, an appropriate Fisherian counterpart of the handicap model of Iwasa and Pomiankowski (1994) would model a situation in which both male characters are affected by the same biased-mutation process. Such a situation is, in fact, approximated by a special case of our model, namely that where the two ornaments provide completely overlapping information about quality. In this case, only a single preference can evolve (FIGURE 1B, 2C-D). This result also holds in the special case where an individual's viability is independent of its quality components (i.e., when $v_{AB} = v_{Ab} = v_{aB} = v_{ab}$). Under these conditions, the quality components A and B can be reinterpreted as sets of genes that merely modify the expression of the male ornaments. Our model then becomes a Fisherian model, in which females only benefit from being choosy through a sexy-son mechanism. Since the two ornaments are equally affected by the two sets of modifier genes, the distinction between these two sets of genes is arbitrary, implying that the variation in the expression of male ornaments is essentially created by a single biased-mutation process. Crucially, one would expect that only a single preference would evolve in this Fisherian model, exactly as in the handicap model of Iwasa and Pomiankowski (1994), which also considers only a single biased-mutation process.

We conclude that ornament diversity is predominantly determined by the number of independent components of variation about which the ornaments provide information. Whether or not these components of variation correlate with fitness components other than attractiveness (as assumed in good-genes models) seems not to affect the outcome in a qualitative fashion (cf. Kokko *et al.*, 2002).

However, these conclusions do not eliminate the possibility that, as suggested by Pomiankowski & Iwasa (1993; Iwasa & Pomiankowski, 1994), sexual traits in species with multiple ornaments have evolved predominantly through Fisherian runaway sexual selection, and not via the handicap process. For example, sexual traits that are only weakly associated with quality traits (i.e., Fisherian traits) may be more likely to experience independent biased mutations than traits that are strongly associated with quality traits (handicap traits). Such an effect would produce the pattern as proposed by Iwasa and Pomiankowski, not due to a fundamental difference between Fisherian and handicap traits, but due to a correlated difference in the genetic architecture underlying Fisherian and handicap traits.

METHODOLOGICAL REMARKS

Our analytical model is a hybrid model, consisting of a population-genetical model for the genotype frequencies at the quality loci and an adaptive-dynamics model for the female preferences and male sexual characters. An important advantage of this approach is that it allows us to express directly the fitness gradients in terms of parameters of the model, without the need to keep track of genetic variances and covariances, as in quantitative-genetic models (e.g., Iwasa & Pomiankowski, 1994). Although it is possible to estimate the values of genetic (co)variances as functions of model parameters (Pomiankowski & Iwasa, 1993, using theory developed by Barton & Turelli, 1991), this procedure is quite complex and involves restrictive assumptions.

The adaptive-dynamics method describes evolution as a mutation-limited process. This assumption allowed us to derive fitness functions relatively easily, but at the same time forced us to assume that there is negligible genetic variation in the resident population for the characters under consideration. This assumption is clearly problematic for the quality components, since the handicap principle relies on the continual presence of genetic variation in quality. Therefore, we used a simple explicit genetic model to keep track of the frequencies of quality genotypes. For the female preferences and male sexual characters, the assumption of mutation-limited evolution has the important consequence that we cannot address the effects of genetic covariances between preferences, between the male sexual characters and between preferences and male characters. The latter covariances are instrumental in driving Fisher's runaway process of sexual selection (Fisher, 1930).

In view of the above, we made an effort to check our analytical results against individual-based computer simulations, which do not rely on the assumptions needed in analytical phenotypic models of evolution. Despite the restrictions imposed by our method of analysis, we found good qualitative agreement between the simulations and our analytical results. We found no differences in the number of equilibria or their stability properties, but we did observe small quantitative differences in the exact location of equilibrium points or transient dynamics. Apart from this, it is reassuring to find, for the special case in which the ornaments provide overlapping information about genetic quality, that the results of our model fit with previous models (Schluter & Price, 1993; Iwasa & Pomiankowski, 1994), realizing, of course, that the existing models rely on similarly restrictive assumptions (e.g., assumptions of weak-selection; Taylor, 1996*a*).

CONDITION-DEPENDENT TRAIT EXPRESSION

In this study, we have treated the information content of an ornament as a fixed parameter of the model. This approach excludes the possibility that males adjust their investment into ornaments depending on their quality, in order to realize an optimal pattern of condition-dependent ornament expression (Grafen, 1990). An obvious next step is to extend our model to allow for this.

Analysis of such an extended model (CHAPTER 9 of this thesis) reveals that condition-dependent trait expression does not lead to results that are qualitatively different from the results of the present paper, unless females adjust their preferences more rapidly than males can fine-tune their trait expression pattern. In this case, females may be continually forced to direct their preferences to novel ornaments, since evolution in males will tend to lower slowly the information content of any ornament on which female preference is acting. This process of sexual conflict over the information of content of ornaments may lead to the continual change of sexual preferences and male ornament expression.

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APPENDIX A — DYNAMICS OF QUALITY GENOTYPE FREQUENCIES

In order to facilitate further analysis, we approximate the dynamics of our stochastic individual-based simulations by a deterministic model that describes the change of quality genotype frequencies from one generation to the next. This procedure is feasible only for a genetically simplified version of our simulation model.

Our aim is to investigate a mutant's potential to invade in a resident population. We assume that almost all individuals carry 'resident' alleles coding for the phenotype $\left(\hat{p}_1, \hat{p}_2, \hat{t}_1, \hat{t}_2\right)$ and that a tiny fraction of the individuals carries a mutant allele at one of its preference or ornament size loci. Since mutations occur only rarely, the mutant phenotype $\left(p_1, p_2, t_1, t_2\right)$ will typically differ from the resident phenotype in only one of its components.

We assume that individuals are haploid, and that each of the two quality components is determined by a single locus with two alleles (A and a for the first quality component; B and b for the second). The alleles A and B confer high quality for their respective quality components; alleles a and b confer low quality. Under these simplifying assumptions, we need to keep track of four quality genotypes frequencies only. For the resident, these will be denoted z_{AB}, z_{Ab}, z_{aB} and z_{ab}. For the mutant, they are denoted ε_{AB}, ε_{Ab}, ε_{aB} and ε_{ab}. The mutant is rare initially, such that

$$\varepsilon_{AB}+\varepsilon_{Ab}+\varepsilon_{aB}+\varepsilon_{ab} \quad \ll \quad z_{AB}+z_{Ab}+z_{aB}+z_{ab}\approx 1\,. \qquad \text{[A-1]}$$

The quality genotype frequencies change due to viability selection, non-random mating and mutation. Below, we discuss these three processes in more detail.

VIABILITY SELECTION

Viability selection acts differently on males and females, since the expression of preferences and ornaments is sex-limited. Therefore, we must calculate the quality genotype frequencies after viability selection separately for males and females. For resident and mutant males, we apply standard replicator equations, yielding

$$\begin{aligned} z'_{m,k} &= z_k\,\frac{h_{m,k}\left(\hat{t}_1,\hat{t}_2\right)}{\overline{h}_m\left(\hat{t}_1,\hat{t}_2\right)}\,, \\ \varepsilon'_{m,k} &= \varepsilon_k\,\frac{h_{m,k}\left(t_1,t_2\right)}{\overline{h}_m\left(\hat{t}_1,\hat{t}_2\right)}\,, \end{aligned} \qquad \text{[A-2]}$$

where $k=ab, aB, Ab$ or AB and $z'_{m,k}$ and $\varepsilon'_{m,k}$ denote the quality genotype frequencies in resident and mutant males after viability selection. The function $h_{m,k}\left(t_1,t_2\right)$ represents the viability of a male with quality genotype k and ornament sizes t_1 and t_2. As in our individual-based simulation model (cf. equation [6]), we take

$$h_{m,k}\left(t_1,t_2\right)=\left(1-c_m\left(t_1,t_2\right)\right)v_k\,, \qquad \text{[A-3]}$$

corresponding to the assumption that the viability cost, $c_m\left(t_1,t_2\right)$, associated with the expression of the ornaments is equal for all males irrespective of their quality genotype.

The viabilities v_k associated with the four possible quality genotypes differ such that $v_{AB}\geq v_{Ab}\geq v_{ab}$ and $v_{AB}\geq v_{aB}\geq v_{ab}$. The population remains variable for the alleles at the quality loci due to deleterious mutations.

The average male fitness, $\overline{h}_m\left(\hat{t}_1,\hat{t}_2\right)$, is independent of the mutant male phenotype, since the density of mutant males can be neglected with respect to the density of resident males. Using [A-3], we find that

$$\overline{h}_m\left(\hat{t}_1,\hat{t}_2\right)=\left(1-c_m\left(\hat{t}_1,\hat{t}_2\right)\right)\overline{v}\,, \qquad \text{[A-4]}$$

with $\overline{v}$ denoting the average general viability.

We may now simplify equation [A-2], yielding

$$z'_{m,k}=\frac{v_k}{\overline{v}}z_k \quad , \quad \varepsilon'_{m,k}=\frac{v_k}{\overline{v}}\,\frac{1-c_m\left(t_1,t_2\right)}{1-c_m\left(\hat{t}_1,\hat{t}_2\right)}\varepsilon_k\,. \qquad \text{[A-5]}$$

A similar procedure yields the quality genotype frequencies in resident and mutant females, $z'_{f,k}$ and $\varepsilon'_{f,k}$,

$$z'_{f,k}=\frac{v_k}{\bar{v}}z_k \quad , \quad \varepsilon'_{f,k}=\frac{v_k}{\bar{v}}\,\frac{1-c_f\left(p_1,p_2\right)}{1-c_f\left(\hat{p}_1,\hat{p}_2\right)}\varepsilon_k \,. \qquad [\text{A-6}]$$

where $c_f\left(p_1,p_2\right)$ gives the viability cost associated with the expression of the female mating preferences.

NON-RANDOM MATING

Let $M_k\left(p_1,p_2,t_1,t_2,\hat{t}_1,\hat{t}_2\right)$ denote the (per-capita) probability that a female, with preferences p_1 and p_2, will mate with a male with quality genotype k and ornament sizes t_1 and t_2. Note that this probability also depends on the resident male strategies $\hat{t}_1$ and $\hat{t}_2$, since our focal male must compete for mating opportunities with other males. If we assume, as in the individual-based simulation model, that females mate only once and that mating probabilities are proportional to male attractiveness, then

$$M_k\left(p_1,p_2,t_1,t_2,\hat{t}_1,\hat{t}_2\right)=\frac{r_k\left(p_1,p_2,t_1,t_2\right)}{\bar{r}\left(p_1,p_2,\hat{t}_1,\hat{t}_2\right)}\,, \qquad [\text{A-7}]$$

where, in line with [1]-[3], $r_k\left(p_1,p_2,t_1,t_2\right)$ measures a male's attractiveness to a particular type of female. Male attractiveness depends on male quality, in line with the assumption that the male ornaments are revealing indicators of quality. All else being equal, males of lower quality are less attractive. The coefficient $\bar{r}\left(p_1,p_2,\hat{t}_1,\hat{t}_2\right)$ represents the average attractiveness of the males encountered by the female, and is defined as

$$\bar{r}\left(p_1,p_2,\hat{t}_1,\hat{t}_2\right)=\sum_{k'=ab,aB,Ab,AB} z'_{m,k'}\; r_{k'}\left(p_1,p_2,\hat{t}_1,\hat{t}_2\right). \qquad [\text{A-8}]$$

To find the frequency of the quality genotype k after mating, we first compute the frequency of mating events between a female with quality genotype k' and a male with quality genotype k''. Next, we sum over all possible combinations of male and female quality genotypes and weigh them according to the probability $Q^k_{k'\times k''}$ that the genotype k is generated through Mendelian segregation from the parental genotypes k' and k''. For the resident, this yields the following quality genotype frequencies after mating (denoted z''_k)

$$z''_k=\sum_{k'=ab,aB,Ab,AB} z'_{f,k'} \sum_{k''=ab,aB,Ab,AB} z'_{m,k''}\; Q^k_{k'\times k''}\; M_{k''}\left(\hat{p}_1,\hat{p}_2,\hat{t}_1,\hat{t}_2,\hat{t}_1,\hat{t}_2\right). \qquad [\text{A-9}]$$

In calculating the quality genotype frequencies after mating for the mutant (denoted ε''_k), we must take care to distinguish two cases. When a mutant individual mates with a resident individual, the mutant can be either the female or the male in the mating pair. Therefore,

$$\begin{aligned}\varepsilon''_k=&\frac{1}{2}\sum_{k'=ab,aB,Ab,AB} z'_{f,k'} \sum_{k''=ab,aB,Ab,AB} \varepsilon'_{m,k''}\; Q^k_{k'\times k''}\; M_{k''}\left(\hat{p}_1,\hat{p}_2,t_1,t_2,\hat{t}_1,\hat{t}_2\right)+\\ &\frac{1}{2}\sum_{k'=ab,aB,Ab,AB} \varepsilon'_{f,k'} \sum_{k''=ab,aB,Ab,AB} z'_{m,k''}\; Q^k_{k'\times k''}\; M_{k''}\left(p_1,p_2,\hat{t}_1,\hat{t}_2,\hat{t}_1,\hat{t}_2\right).\end{aligned} \qquad [\text{A-10}]$$

The factor $1/2$ in front of both terms reflects the fact that only half of the offspring will inherit the mutant allele from its mutant parent.

We assume that the two quality loci recombine freely. Thus,

$$Q^{k}_{k' \times k''} = \frac{\delta_A(k,k') + \delta_A(k,k'')}{2} \cdot \frac{\delta_B(k,k') + \delta_B(k,k'')}{2} , \tag{A-11}$$

where $\delta_A(k,k') = 1$ if the two alleles for quality component A of the quality genotypes k and k' are identical and zero otherwise (with an analogous definition for δ_B).

In what follows, it will be convenient to represent equations [A-9] and [A-10] in terms of matrix equations. Let us therefore define vectors $\vec{z}$, $\vec{z}'$, $\vec{\varepsilon}$ and so on, which contain as elements the corresponding quality genotype frequencies. For example,

$$\begin{aligned} \vec{z} &= (z_{ab}, z_{aB}, z_{Ab}, z_{AB})^T , \\ \vec{\varepsilon} &= (\varepsilon_{ab}, \varepsilon_{aB}, \varepsilon_{Ab}, \varepsilon_{AB})^T . \end{aligned} \tag{A-12}$$

We may then write

$$\begin{aligned} \vec{\varepsilon}'' &= \mathbf{T}(\vec{z})\, \vec{\varepsilon} , \\ \vec{z}'' &= \hat{\mathbf{T}}(\vec{z})\, \vec{z} , \end{aligned} \tag{A-13}$$

where the elements of the transition matrices $\mathbf{T}(\vec{z})$ and $\hat{\mathbf{T}}(\vec{z})$ can be found by combining equations [A-5] to [A-10]. The matrix element $\mathbf{T}_{kk'}(\vec{z})$ represents the frequency at which a mutant individual with quality genotype k' transmits its mutant genes to offspring with genotype k (not yet taking into account the occurrence of deleterious mutations). It is given by

$$\begin{aligned} \mathbf{T}_{kk'}(\vec{z}) = {} & \frac{1}{2} \frac{v_{k'}}{\bar{v}} \frac{1 - c_m(t_1,t_2)}{1 - c_m(\hat{t}_1,\hat{t}_2)} \frac{r_{k'}(\hat{p}_1,\hat{p}_2,t_1,t_2)}{\bar{r}(\hat{p}_1,\hat{p}_2,\hat{t}_1,\hat{t}_2)} \sum_{k''=ab,aB,Ab,AB} Q^{k}_{k' \times k''} \frac{v_{k''} z_{k''}}{\bar{v}} + \\ & \frac{1}{2} \frac{v_{k'}}{\bar{v}} \frac{1 - c_f(p_1,p_2)}{1 - c_f(\hat{p}_1,\hat{p}_2)} \sum_{k''=ab,aB,Ab,AB} Q^{k}_{k' \times k''} \frac{v_{k''} z_{k''}}{\bar{v}} \frac{r_{k''}(p_1,p_2,\hat{t}_1,\hat{t}_2)}{\bar{r}(p_1,p_2,\hat{t}_1,\hat{t}_2)} . \end{aligned} \tag{A-14}$$

When this expression is evaluated for a mutant that is phenotypically equivalent to the resident, we obtain the elements of the resident transition matrix. In other words,

$$\hat{\mathbf{T}}(\vec{z}) = \mathbf{T}(\vec{z})\big|^{\wedge} , \tag{A-15}$$

where the notation $x\big|^{\wedge}$ is introduced to denote that the expression x should be evaluated for a mutant that is phenotypically equivalent to the resident. In other words,

$$x\big|^{\wedge} = x\big|_{p_1=\hat{p}_1,\, p_2=\hat{p}_2,\, t_1=\hat{t}_1,\, t_2=\hat{t}_2} . \tag{A-16}$$

Deleterious mutations

Deleterious mutations at the two quality loci occur at rate μ_A and μ_B, respectively. The quality genotype frequencies in the next generation can easily be computed from the frequencies after mating. In matrix notation,

$$\begin{aligned} \vec{z}_{n+1} &= \mathbf{M}\,\vec{z}''_n\,, \\ \vec{\varepsilon}_{n+1} &= \mathbf{M}\,\vec{\varepsilon}''_n\,, \end{aligned} \qquad \text{[A-17]}$$

where, the index n represents the generation number. Up to first order in μ_A and μ_B,

$$\mathbf{M} = \begin{pmatrix} 1 & \mu_B & \mu_a & 0 \\ 0 & 1-\mu_B & 0 & \mu_A \\ 0 & 0 & 1-\mu_A & \mu_B \\ 0 & 0 & 0 & 1-\mu_A-\mu_B \end{pmatrix}. \qquad \text{[A-18]}$$

Combining [A-13] and [A-17], we finally arrive at a dynamical model for the quality genotype frequencies of mutant and resident,

$$\begin{aligned} \vec{z}_{n+1} &= \mathbf{M}\hat{\mathbf{T}}(\vec{z}_n)\vec{z}_n\,, \\ \vec{\varepsilon}_{n+1} &= \mathbf{M}\mathbf{T}(\vec{z}_n)\vec{\varepsilon}_n\,. \end{aligned} \qquad \text{[A-19]}$$

APPENDIX B — Invasion fitness and fitness gradients

The fate of the mutant can be determined by iterating the recurrence equations [A-19]. If the mutant density increases, the mutant will be able to invade; if it decreases, the mutant will disappear after some time. Alternatively, we may attempt to derive, from equation [A-19], the mutant's invasion-fitness function. This function, denoted as λ, measures the mutant's geometrical growth rate when rare and, as such, directly predicts whether or not the mutant will be able to invade in the resident population. Moreover, when evolution can be described as a series of mutation and trait-substitution events, the invasion-fitness function can be used to derive equations for the evolutionary dynamics of preferences and ornaments (Hofbauer & Sigmund, 1998).

Resident equilibrium

We study the invasion by a mutant of a resident population that has attained a stable distribution of quality genotype frequencies. This stable frequency distribution, $\vec{z}^*$, can be found by solving the equation

$$\vec{z}^* = \mathbf{M}\hat{\mathbf{T}}(\vec{z}^*)\vec{z}^*\,, \qquad \text{[B-1]}$$

which yields, up to first order in μ_A and μ_B,

$$\vec{z}^* = \left(0,\ \frac{\mu_A}{1-\dfrac{1+\hat{R}_A}{2\,V_A\,\hat{R}_A}},\ \frac{\mu_B}{1-\dfrac{1+\hat{R}_B}{2\,V_B\,\hat{R}_B}},\ 1-\frac{\mu_A}{1-\dfrac{1+\hat{R}_A}{2\,V_A\,\hat{R}_A}}-\frac{\mu_B}{1-\dfrac{1+\hat{R}_B}{2\,V_B\,\hat{R}_B}} \right)^T . \qquad \text{[B-2]}$$

Here, $V_A = v_{AB}/v_{aB}$ and $V_B = v_{AB}/v_{Ab}$ represent the viability of high-quality individuals relative to individuals carrying a deleterious mutation at one of the quality loci. The coefficients $\hat{R}_A$ and $\hat{R}_B$ represent the relative attractiveness of high-quality males, again relative to males that carry a deleterious mutation at a quality locus. In general,

$$R_A(p_1,p_2,t_1,t_2) = \frac{r_{AB}(p_1,p_2,t_1,t_2)}{r_{aB}(p_1,p_2,t_1,t_2)},\quad R_B(p_1,p_2,t_1,t_2) = \frac{r_{AB}(p_1,p_2,t_1,t_2)}{r_{Ab}(p_1,p_2,t_1,t_2)}, \qquad \text{[B-3]}$$

and, specifically for the resident, $\hat{R}_A = R_A(\hat{p}_1,\hat{p}_2,\hat{t}_1,\hat{t}_2)$ and $\hat{R}_B = R_B(\hat{p}_1,\hat{p}_2,\hat{t}_1,\hat{t}_2)$. Note that, when the ornaments do not reveal quality, such that $\hat{R}_A = \hat{R}_B = 1$ and mating is random, equation [B-2] simplifies to the equilibrium frequency distribution under standard mutation-selection balance (Crow & Kimura, 1970).

INVASION FITNESS AND FITNESS GRADIENTS

We may now proceed to compute the invasion-fitness function λ. The invasion fitness is defined as the geometric rate of increase of the mutant population density when the mutant is still rare. Since the invasion of the mutant in the resident population is described by the recurrence equation

$$\vec{\varepsilon}_{n+1} = \mathbf{MT}(\vec{z}^*)\,\vec{\varepsilon}_n\ , \qquad \text{[B-4]}$$

the invasion fitness can be found as the dominant eigenvalue of the matrix $\mathbf{MT}(\vec{z}^*)$.

Although it is possible to compute the invasion fitness for arbitrary combinations of mutant and resident phenotypes, we refrain from doing so, since the resulting expression is too complicated to give any insights. Instead, we focus on mutants that differ only slightly from the resident. For such mutants, the fitness difference between the mutant and the resident is proportional to the gradient of the invasion-fitness function, in the direction in which the mutant differs from the resident. Moreover, under suitable assumptions (Weissing, 1996; Hofbauer & Sigmund, 1998), the direction and size of this fitness gradient determines the direction and rate of evolution of the female preferences and male ornament sizes. In mathematical terms,

$$\begin{aligned} \frac{d\hat{p}_i}{d\tau} &= \lambda_{p_i}\ , \\ \frac{d\hat{t}_i}{d\tau} &= \lambda_{t_i}\ , \end{aligned} \qquad \text{[B-5]}$$

where τ represents evolutionary time, and the λ_x denote derivatives of the invasion-fitness function. For example,

$$\lambda_{p_1} = \frac{\partial \lambda}{\partial p_1}\Bigg|^{\wedge} . \qquad \text{[B-6]}$$

Underlying equation [B-5] is the assumption that the mutation rates of the female preference and male ornament genes are equal, and that a mutation in one of the characters does not have pleiotropic effects on another character.

According to a standard result (e.g., Caswell, 1989; Taylor, 1996b), the selection gradients can also be written as

$$\lambda_{p_i} = \frac{\partial \lambda}{\partial p_i}\Bigg|^{\wedge} = \frac{1}{\vec{w}\,\vec{u}}\quad \vec{w}\,\mathbf{M}\frac{\partial \mathbf{T}\left(\vec{z}^{*}\right)}{\partial p_i}\Bigg|^{\wedge}\vec{u} . \qquad \text{[B-7]}$$

Here, $\vec{w}$ and $\vec{u}$ denote, respectively, the left and right dominant eigenvectors of the matrix $\mathbf{M}\hat{\mathbf{T}}\left(\vec{z}^{*}\right)$. From equation [B-1], it follows that $\vec{u} = \vec{z}^{*}$. The left eigenvector $\vec{w}$ contains the reproductive values w_{ab}, w_{aB}, w_{Ab} and w_{AB} of the different quality genotypes. They can be solved from the equation $\vec{w}\,\mathbf{M}\hat{\mathbf{T}}\left(\vec{z}^{*}\right) = \vec{w}$, yielding, up to the lowest order in μ_A and μ_B,

$$w_{AB} = 1 , \quad w_{aB} = \frac{1+\hat{R}_A}{\hat{R}_A\left(4\,V_A - 1\right) - 1} , \quad w_{Ab} = \frac{1+\hat{R}_B}{\hat{R}_B\left(4\,V_B - 1\right) - 1} . \qquad \text{[B-8]}$$

We omit the reproductive value of the lowest quality class, since it turns out to be irrelevant.

After some simplification, we obtain, from equation [A-14] and [B-7], expressions for the selection gradients. For the male ornaments, we find, up to the lowest order in μ_A and μ_B,

$$\lambda_{t_i} = \frac{1}{2}\,\frac{\dfrac{\partial}{\partial t_i}\Big(\big(1 - c_m(t_1, t_2)\big)\, r_{AB}\big(\hat{p}_1, \hat{p}_2, t_1, t_2\big)\Big)\Bigg|^{\wedge}}{\Big(1 - c_m\big(\hat{t}_1, \hat{t}_2\big)\Big)\, r_{AB}\big(\hat{p}_1, \hat{p}_2, \hat{t}_1, \hat{t}_2\big)} = \frac{1}{2}\,\frac{\partial \ln\big(\left(1 - c_m\right) r_{AB}\big)}{\partial t_i}\Bigg|^{\wedge} , \qquad \text{[B-9]}$$

where $i = 1$ or 2. This expression indicates that males maximize the product of survival and attractiveness for the highest quality class. Hence, the evolution of the male ornaments is not at all affected by the reduced attractiveness of males carrying a deleterious mutation at one of the quality loci. This is because the less viable males occur only at a low frequency, of the same order as the mutation rate.

Selection on female preferences is only weak, at least as long as we assume that the costs of female choice are only small (i.e., of the same order of magnitude as the mutation rates μ_A and μ_B, as in other good-genes models). In this case, we find, up to first order in μ_A and μ_B,

$$\lambda_{p_i} = \frac{\left.\frac{\partial}{\partial p_i}\left(1-c_f(p_1,p_2)\right)\right|^{\wedge}}{2\left(1-c_f(\hat{p}_1,\hat{p}_2)\right)} + \mu_A \frac{\left.\frac{\partial}{\partial p_i} R_A\left(p_1,p_2,\hat{t}_1,\hat{t}_2\right)\right|^{\wedge}}{\hat{R}_A\left(\hat{R}_A\left(4\,V_A-1\right)-1\right)} + \mu_B \frac{\left.\frac{\partial}{\partial p_i} R_B\left(p_1,p_2,\hat{t}_1,\hat{t}_2\right)\right|^{\wedge}}{\hat{R}_B\left(\hat{R}_B\left(4\,V_B-1\right)-1\right)} \quad \text{[B-10]}$$

$$= \frac{1}{2}\left.\frac{\partial \ln\left(1-c_f\right)}{\partial p_i}\right|^{\wedge} + \mu_A\hat{\varpi}_A \left.\frac{\partial \ln\left(R_A\right)}{\partial p_i}\right|^{\wedge} + \mu_B\hat{\varpi}_B \left.\frac{\partial \ln\left(R_B\right)}{\partial p_i}\right|^{\wedge},$$

where the coefficients $\hat{\varpi}_A$ and $\hat{\varpi}_B$ represent the reproductive values of males carrying a deleterious mutation at one of their quality loci. To be exact,

$$\hat{\varpi}_A = \frac{1}{\hat{R}_A\left(4\,V_A-1\right)-1} \quad , \quad \hat{\varpi}_B = \frac{1}{\hat{R}_B\left(4\,V_B-1\right)-1}. \quad \text{[B-11]}$$

The equations [B-5], [B-9] and [B-10] give rise to equations [11] and [12] in the main text (since this did not result in ambiguities, we chose to omit the hats that were previously used to distinguish resident traits and coefficients from mutant traits and coefficients).

APPENDIX C — EQUILIBRIA AND THEIR STABILITY

In the individual-based simulations, we chose the functions r_k ($k = ab, aB, Ab$ or AB), c_m and c_f as follows:

$$\begin{aligned} r_k\left(p_1,p_2,t_1,t_2\right) &= \exp\left(\alpha_1^k\, p_1\, t_1 + \alpha_2^k p_2 t_2\right), \\ c_m\left(t_1,t_2\right) &= 1-\exp\left(-\beta_1\, t_1^2 - \beta_2\, t_2^2\right), \\ c_f\left(p_1,p_2\right) &= 1-\exp\left(-\gamma_1\,\theta_{12}\, p_1^2 - \gamma_2\,\theta_{21}\, p_2^2\right). \end{aligned} \quad \text{[C-1]}$$

The joint-cost-of-choice coefficients θ_{12} and θ_{21}, are allowed to vary with p_2 and p_1, respectively, as follows:

$$\theta_{ij}\left(p_j\right) = \exp\left(\vartheta_i p_j^2\right). \quad \text{[C-2]}$$

For this specific example model, the equilibrium condition $\lambda_{t_i} = 0$ now reduces to a simple equation, with solutions (denoted by tildes)

$$\tilde{t}_i = \frac{1}{2}\frac{\alpha_i^{AB}}{\beta_i}\tilde{p}_i\,. \quad \text{[C-3]}$$

Substituting these equilibrium solutions into $\lambda_{p_i} = 0$ eventually yields an equation from which the equilibrium values for the female preferences can be solved:

$$\gamma_i\,\tilde{p}_i\,\theta_{ij}\left(\tilde{p}_j\right) + \vartheta_j\,\gamma_j\,\tilde{p}_i\,\tilde{p}_j^2\,\theta_{ji}\left(\tilde{p}_i\right) = \mu_A\,\tilde{\varpi}_A\,\kappa_i^A\,\tilde{p}_i + \mu_B\,\tilde{\varpi}_B\,\kappa_i^B\,\tilde{p}_i\,, \quad \text{[C-4]}$$

where the reproductive values $\tilde{\varpi}_A$ and $\tilde{\varpi}_B$ are given by

$$\tilde{\varpi}_A = \frac{1}{\exp\left(\kappa_1^A\,\tilde{p}_1^2 + \kappa_2^A\,\tilde{p}_2^2\right)\left(4\,V_A-1\right)-1}, \quad \tilde{\varpi}_B = \frac{1}{\exp\left(\kappa_1^B\,\tilde{p}_1^2 + \kappa_2^B\,\tilde{p}_2^2\right)\left(4\,V_B-1\right)-1}, \quad \text{[C-5]}$$

and where the constants κ_i^k ($k = A$ or B) measure the information content of the ornaments, that is, they represent the amount of information that ornament i gives about quality component k. To be exact,

$$\kappa_i^k = -\frac{1}{\tilde{p}_i} \frac{\partial \ln\left(R_k\right)}{\partial p_i} \right|^{\sim}, \qquad \text{[C-6]}$$

implying that

$$\kappa_i^A = \frac{\alpha_i^{AB}\left(\alpha_i^{AB} - \alpha_i^{aB}\right)}{2\beta_i}, \quad \kappa_i^B = \frac{\alpha_i^{AB}\left(\alpha_i^{AB} - \alpha_i^{Ab}\right)}{2\beta_i}. \qquad \text{[C-7]}$$

TRIVIAL AND BOUNDARY EQUILIBRIA

It is easy to see from [C-3] and [C-4] that a trivial equilibrium exists at $\tilde{p}_1 = \tilde{p}_2 = \tilde{t}_1 = \tilde{t}_2 = 0$. At this equilibrium, females show preferences for neither one of the ornaments. The dynamical stability of the equilibrium is determined by the Jacobian matrix of the dynamical system [B-5]. The equilibrium is stable (i.e., evolution will converge towards the equilibrium) when

$$\gamma_i > \frac{\mu_A\, \kappa_i^A}{4\, V_A - 2} + \frac{\mu_B\, \kappa_i^B}{4\, V_B - 2} \quad \text{for } i = 1 \text{ and } i = 2\,; \qquad \text{[C-8]}$$

that is, when the costs of mate choice outweigh the benefits for every ornament independently. Stability criterion [C-8] and the other stability criteria that follow are derived under the assumption that the cost of choice coefficients, γ_i, and the mutation biases, μ_A and μ_B, are small relative to the other parameters, such that the timescale on which female preferences evolve is much slower than that on which the male characters evolve.

When condition [C-8] is violated, the trivial equilibrium is unstable and there exist one or two boundary equilibria at which females exhibit a preference for one of the ornaments only. For example, when condition [C-8] is violated for the first ornament, female preference for the first ornament, p_1, can increase until it reaches an equilibrium value $\tilde{p}_1 > 0$, which satisfies

$$\gamma_1 = \frac{\mu_A\, \kappa_1^A}{\left(4\, V_A - 1\right)\exp\left(\kappa_1^A\, \tilde{p}_1^2\right) - 1} + \frac{\mu_B\, \kappa_1^B}{\left(4\, V_B - 1\right)\exp\left(\kappa_1^B\, \tilde{p}_1^2\right) - 1}. \qquad \text{[C-9]}$$

At this equilibrium, $\tilde{t}_1 > 0$ is given by [C-3] and $\tilde{p}_2 = \tilde{t}_2 = 0$. The equilibrium is stable when

$$\gamma_2 + \tilde{p}_1^2\left(\gamma_1\, \vartheta_1 + \gamma_2\, \vartheta_2\right) > \frac{\mu_A\, \kappa_2^A}{\left(4\, V_A - 1\right)\exp\left(\kappa_1^A\, \tilde{p}_1^2\right) - 1} + \frac{\mu_B\, \kappa_2^B}{\left(4\, V_B - 1\right)\exp\left(\kappa_1^B\, \tilde{p}_1^2\right) - 1}, \qquad \text{[C-10]}$$

where we have assumed that the ϑ_i are small.

In a similar manner, there exists a boundary equilibrium with $\tilde{p}_2 > 0$, $\tilde{t}_2 > 0$ and $\tilde{p}_1 = \tilde{t}_1 = 0$ when condition [C-8] is violated for the second ornament. At this equilibrium, females exhibit a preference for the second ornament only. The equilibrium value $\tilde{p}_2$ can be solved from

$$\gamma_2 = \frac{\mu_A\,\kappa_2^A}{(4\,V_A - 1)\exp\left(\kappa_2^A\,\tilde{p}_2^2\right) - 1} + \frac{\mu_B\,\kappa_2^B}{(4\,V_B - 1)\exp\left(\kappa_2^B\,\tilde{p}_2^2\right) - 1}, \qquad \text{[C-11]}$$

and the equilibrium is stable when

$$\gamma_1 + \tilde{p}_2^2\left(\gamma_1\,\vartheta_1 + \gamma_2\,\vartheta_2\right) > \frac{\mu_A\,\kappa_1^A}{(4\,V_A - 1)\exp\left(\kappa_2^a\,\tilde{p}_2^2\right) - 1} + \frac{\mu_B\,\kappa_1^B}{(4\,V_B - 1)\exp\left(\kappa_2^B\,\tilde{p}_2^2\right) - 1}. \qquad \text{[C-12]}$$

The benefits of choice decrease with increasing preference, whereas the costs of choice increase with increasing preference. Consequently, female preferences cannot evolve to arbitrarily large values. This implies that there must exist a stable internal attractor if the trivial equilibrium and both boundary equilibria are unstable.

From the conditions for the existence and stability of the boundary equilibria, it is clearly impossible for both boundary equilibria to be unstable if the joint costs of choice are high and the ornaments provide overlapping information about the quality components.

To illustrate this, let us assume that the ornaments provide completely overlapping information about the quality components. In this situation, we can find a constant k such that

$$\kappa_2^A = k\,\kappa_1^A\,, \quad \kappa_2^B = k\,\kappa_1^B\,. \qquad \text{[C-13]}$$

For $k < 1$, the first ornament has a higher information content for both quality components; for $k > 1$, the second ornament has the highest information content. Combining [C-9], [C-10] and [C-13], we find that the first boundary equilibrium, at which females exhibit preference for the first ornament only, is unstable when

$$\gamma_2 + \tilde{p}_1^2\left(\gamma_1\,\vartheta_1 + \gamma_2\,\vartheta_2\right) < k\,\gamma_1\,. \qquad \text{[C-14]}$$

This becomes more likely when k is large, i.e., when the second ornament has a higher information content than the first, or when $\gamma_1 > \gamma_2$, i.e., when preference for the first ornament is more costly than preference for the second ornament.

By combining [C-11] to [C-13], we find that the other boundary equilibrium is unstable when

$$\gamma_1 + \tilde{p}_2^2\left(\gamma_1\,\vartheta_1 + \gamma_2\,\vartheta_2\right) < \frac{1}{k}\gamma_2\,. \qquad \text{[C-15]}$$

After rearranging [C-14] and [C-15], we find that both boundary equilibria are unstable when

$$k\,\gamma_1 + k\,\tilde{p}_2^2\left(\gamma_1\,\vartheta_1 + \gamma_2\,\vartheta_2\right) < \gamma_2 < k\,\gamma_1 - \tilde{p}_1^2\left(\gamma_1\,\vartheta_1 + \gamma_2\,\vartheta_2\right), \qquad \text{[C-16]}$$

which can only be true when $\gamma_1\,\vartheta_1 + \gamma_2\,\vartheta_2 < 0$; in other words, when the joint cost of choice is low.

INTERNAL EQUILIBRIA

Internal equilibria correspond to solutions of the following equation

$$\gamma_i\,\theta_{ij}\left(\tilde{p}_j\right) + \vartheta_j\,\gamma_j\,\tilde{p}_j^2\,\theta_{ji}\left(\tilde{p}_i\right) = \mu_A\,\tilde{w}_A\,\kappa_i^A + \mu_B\,\tilde{w}_B\,\kappa_i^B\,. \qquad \text{[C-17]}$$

Standard stability analysis reveals that internal equilibria are either stable nodes or saddle points, depending on the sign of $\det(\mathbf{J})$, the determinant of the Jacobian matrix of the dynamical system [B-5]. We find

$$\det(\mathbf{J}) = 4\tilde{p}_1^2\,\tilde{p}_2^2\Big(g_A\,g_B\left(\kappa_1^A\,\kappa_2^B-\kappa_1^B\,\kappa_2^A\right)^2 \\ -2\left(g_A\,\kappa_1^A\,\kappa_2^A+g_B\,\kappa_1^B\,\kappa_2^B\right)\left(\gamma_1\,\vartheta_1+\gamma_2\,\vartheta_2\right)-\left(\gamma_1\,\vartheta_1+\gamma_2\,\vartheta_2\right)^2\Big). \qquad \text{[C-18]}$$

where the positive coefficients g_k ($k=A$ or B) are given by

$$g_k=\mu_k\tilde{w}_k\left(1+\tilde{w}_k\right). \qquad \text{[C-19]}$$

From [C-18], it follows that multiple preferences can only be stable if

$$\left(\kappa_1^A\kappa_2^B-\kappa_1^B\kappa_2^A\right)^2>2\frac{g_A\,\kappa_1^A\,\kappa_2^A+g_B\,\kappa_1^B\,\kappa_2^B}{g_A\,g_B}\left(\gamma_1\,\vartheta_1+\gamma_2\,\vartheta_2\right)+\left(\gamma_1\,\vartheta_1+\gamma_2\,\vartheta_2\right)^2. \qquad \text{[C-20]}$$

The left-hand side of this inequality is related to ω, the measure for ornament independence used in APPENDIX D. To be precise,

$$\omega=\frac{4\,\beta_1\,\beta_2}{\left(\alpha_1^{AB}\alpha_2^{AB}\right)^2}\left(\kappa_1^A\,\kappa_2^B-\kappa_1^B\,\kappa_2^A\right). \qquad \text{[C-21]}$$

If the ornaments do not provide independent information about the underlying quality components ($\omega=0$), condition [C-20] reduces to

$$2\frac{g_A\,\kappa_1^A\,\kappa_2^A+g_B\,\kappa_1^B\,\kappa_2^B}{g_A\,g_B}\left(\gamma_1\,\vartheta_1+\gamma_2\,\vartheta_2\right)+\left(\gamma_1\,\vartheta_1+\gamma_2\,\vartheta_2\right)^2<0, \qquad \text{[C-22]}$$

which can only be true when the joint cost of choice is low, i.e., $\gamma_1\,\vartheta_1+\gamma_2\,\vartheta_2<0$.

When the joint cost of choice is high, condition [C-20] can be satisfied when the ornaments provide independent quality information and have high information contents. Hence, even when the joint cost of choice is high, preferences for multiple ornaments may evolve.

Note that, when $\gamma_1\vartheta_1+\gamma_2\vartheta_2<2\left(g_A\,\kappa_1^A\,\kappa_2^A+g_B\,\kappa_1^B\,\kappa_2^B\right)/\left(g_A\,g_B\right)$, i.e., when the cost of choice is very low, the equilibrium loses its stability. We do not consider this to be realistic, however, since it corresponds to the implausible situation that the viability benefits of exhibiting multiple preferences outweigh the costs of choice for the independent ornaments. Exhibiting multiple preferences then confers a viability advantage and preferences evolve to arbitrarily large values.

APPENDIX D — MODEL OUTCOME FOR DIFFERENT PARAMETER CONDITIONS

The dependence of the outcome of the model on key-parameters, such as the information content of the ornaments and the degree of overlap in the information content, is summarized in FIGURE 3. This figure applies to a situation where the joint cost of choice is high. Each point in FIGURE 3 represents a combination of parameter values. The surfaces in the figure delineate regions within which the evolutionary outcome of the model (i.e., the different possibilities given in FIGURE 2) does not change qualitatively.

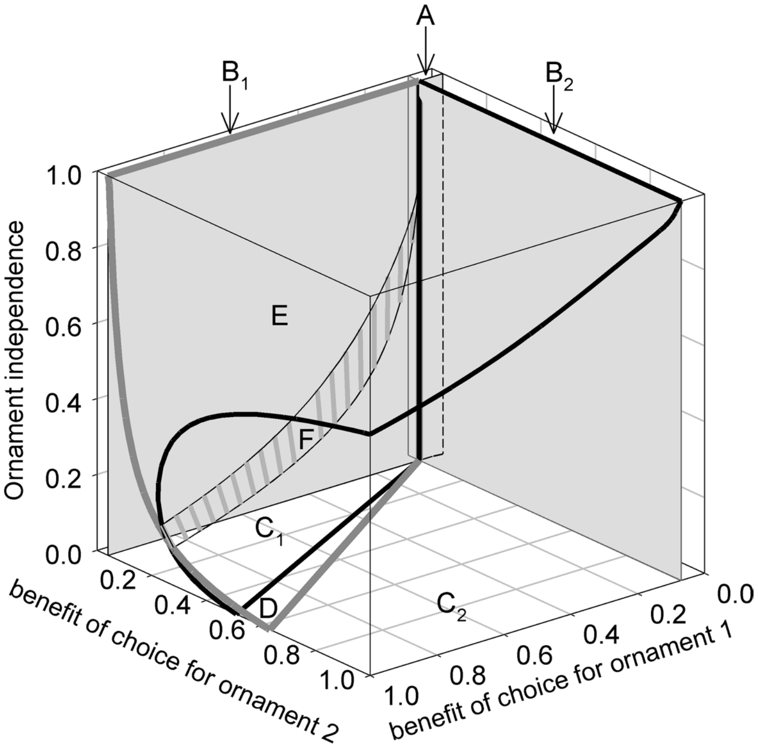

FIGURE 3 – BIFURCATION DIAGRAM

Parameter space can be subdivided into regions, within which the behavior of the model does not change qualitatively. The letters A-F, used to label the different regions, correspond to the labels of the panels in FIGURE 2, such that, for example, the outcome of the model in region A is represented by phase portrait A in FIGURE 2. For regions B and C, we use indices 1 and 2 to distinguish the two qualitatively different configurations that may arise when only one of the boundary equilibria is stable. For example, in regions B$_2$ and C$_2$, preference can evolve for ornament 2 but not for ornament 1, exactly as in FIGURE 2B & 2C, respectively. In region B$_1$ and C$_1$, preference can evolve for ornament 1 only (as in FIGURE 2B & 2C, when the roles of p_1 and p_2 are reversed). Bifurcations of the p_1 and p_2 boundary equilibria occur on the surfaces with gray and black boundary lines, respectively. Bifurcations of the internal equilibrium occur on the hatched surface. Along the axes, we varied the α_i^k. To ensure that every point in the figure corresponds to a unique combination of parameters, we imposed the parameter constraints $\alpha_1^{aB} = 0$, $\alpha_1^{Ab} = \alpha_1^{AB}$, $\alpha_2^{aB} + \alpha_2^{Ab} = \alpha_2^{AB}$. Other parameters are as in FIGURE 2 (see TABLE 1).

Along the horizontal axes, we change the benefits of choice for the two ornaments (the respective right-hand sides of [16]), by varying the information-content constants κ_i^A and κ_i^B ($i = 1 \text{ or } 2$). Along the vertical axis, we vary ornament independence, i.e., the extent to which the ornaments provide independent information about quality. We define ornament independence ω as

$$\omega = \rho\left(\kappa_1^a \, \kappa_2^b - \kappa_1^b \, \kappa_2^a\right), \qquad \text{[D-1]}$$

where the scaling factor ρ is chosen such that ω varies between –1 and 1 (APPENDIX C). When both ornaments are equally revealing indicators for both quality components, $\omega = 0$. Contrastingly, the ornaments are completely independent ($\omega = 1$) when ornament 1 provides information about quality component A only, whereas ornament 2 provides information about quality component B only.

An internal equilibrium exists only when female preference can evolve for both ornaments independently (APPENDIX C). Therefore, we concentrate on the region in parameter-space in which both boundary equilibria exist. In FIGURE 3, this region is delimited by the gray surfaces. These demarcate, for each of the ornaments, threshold levels for the benefit of choice above which the benefits outweigh the costs of choice for a single preference (the threshold levels are simply the values of the parameters γ_i). Correspondingly, the evolutionary outcome of the model will be as in FIGURE 2B for parameter combinations that lie behind one of the gray surfaces in FIGURE 3, and as in FIGURE 2A for parameter combinations that lie behind both gray surfaces.

Let us focus, for the moment, on the floor plane of FIGURE 3. On this floor plane, the ornaments provide completely overlapping information about quality, as in FIGURE 1A-B and FIGURE 2A-D. Depending on the benefits of choice for the two ornaments, we may find three qualitatively different evolutionary outcomes. When the benefit of choice for one of the ornaments is large relative to that for the other ornament, females will always evolve preferences for the former (as in FIGURE 2C). If the benefits of choice outweigh the costs of choice by approximately equal amounts for both ornaments, females will also always evolve preferences for a single ornament only. However, the evolutionary outcome will depend on the initial conditions, as in FIGURE 2D. The higher the joint cost of choice, the more likely it is that both boundary equilibria are simultaneously stable, giving rise to a situation where the evolutionary outcome is dependent on initial conditions.

The three evolutionary outcomes represented by the phase portraits 2C and 2D occur in regions of parameter space that are delimited by two boundary surfaces. These are represented in FIGURE 3 by transparent surfaces, bounded by thick gray and black lines. Mathematically, the boundary surfaces represent the parameter combinations at which the internal equilibrium arises or disappears through a bifurcation involving also one of the boundary equilibria. Crucially, when we no longer restrict ourselves to the floor plane, but allow the ornaments to provide increasingly independent information about the quality components, we will always cross one of the bifurcation surfaces. By doing so, we enter a region in parameter-space where both boundary equilibria are unstable and the internal equilibrium is stable (as in FIGURE 2E). Multiple internal equilibria (as in FIGURE 2F) exist for parameter combinations in the hatched, flattened region.

APPENDIX E — STABILITY CRITERIA FOR MULTIPLE PREFERENCES IN A GENERAL MODEL

In this appendix, we aim to demonstrate that the conclusions reached for our specific example are also valid in a more general context. To this end, we will analyze a general mate-choice model and investigate the conditions under which female preferences for multiple ornaments are stable.

We start from the general expressions for the gradients of the invasion-fitness function that we derived earlier (cf. [B-9],[B-10]),

$$\lambda_{t_i}=\frac{1}{2}\frac{\partial\ln\left(\left(1-c_m(t_1,t_2)\right)r_{AB}(p_1,p_2,t_1,t_2)\right)}{\partial t_i},$$
$$\lambda_{p_i}=\frac{1}{2}\frac{\partial\ln\left(1-c_f(p_1,p_2)\right)}{\partial p_i}+\mu_A\hat{w}_A\frac{\partial\ln\left(R_A(p_1,p_2,t_1,t_2)\right)}{\partial p_i}+\mu_B\hat{w}_B\frac{\partial\ln\left(R_B(p_1,p_2,t_1,t_2)\right)}{\partial p_i}. \quad \text{[E-1]}$$

The first of these two expressions indicates that the male characters t_1 and t_2 evolve to maximize the product of male survival and male attractiveness. We make no assumptions regarding the functions c_m and r_{AB}, other than that there always exists an optimum value to which the male characters evolve. Moreover, the functions c_m and r_{AB} should be such that this optimum value is unique. Under these assumptions, there exist functions $T_i^{\mathrm{opt}}(p_i)$, which give the optimal investment into ornament i as a function of female preference for that ornament (assuming that female preferences are non-overlapping).

Without loss of generality, we may measure preferences in such a way that a randomly mating female has the phenotype $p_i=0$ (i). In the absence of sensory bias, $p_i=0$ minimizes the cost of choice, for any given level of preference for the other ornament (ii). The male characters are measured such that $t_i=0$ corresponds to the optimal strategy when females mate randomly (i.e., when the male characters evolve in response to viability selection only) (iii). It is reasonable to suppose that females cannot estimate a male's quality from an ornament that is not exaggerated (i.e., when $t_i=0$) (iv).

These general considerations have useful implications. First, because of (iii), we may write

$$T_i^{\mathrm{opt}}(p_i)=p_i\,\mathrm{T}_i(p_i), \quad \text{[E-2]}$$

such that

$$T_i^{\mathrm{opt}}(0)=0. \quad \text{[E-3]}$$

The function T_i in [E-2] determines how strongly male investment into ornament i changes in response to a change in female preference i.

Second, (ii) implies that we may write

$$\ln\left(1-c_f(p_1,p_2)\right)=-p_1^2\,\Gamma_1(p_1)-p_2^2\,\Gamma_2(p_2)-p_1^2\,p_2^2\,\Theta(p_1,p_2), \quad \text{[E-4]}$$

such that

$$\left.\frac{\partial \ln\left(1-c_f(p_1,p_2)\right)}{\partial p_i}\right|_{p_i=0}=0\,. \qquad \text{[E-5]}$$

The functions Γ_i in [E-4] determine how the local baseline costs of choice vary with p_i, and Θ gives the local strength of the epistatic interaction between the costs of choice.

Third, (i) and (iv), together with the assumption that the preferences are non-overlapping, allow us to write

$$\begin{aligned}\ln\left(R_A(p_1,p_2,t_1,t_2)\right)&=p_1\,t_1\,A_1(p_1,t_1)+p_2\,t_2\,A_2(p_2,t_2)\,,\\ \ln\left(R_B(p_1,p_2,t_1,t_2)\right)&=p_1\,t_1\,B_1(p_1,t_1)+p_2\,t_2\,B_2(p_2,t_2)\,,\end{aligned} \qquad \text{[E-6]}$$

such that

$$R_k(0,0,t_1,t_2)=R_k(0,p_2,t_1,0)=R_k(p_1,0,0,t_2)=R_k(p_1,p_2,0,0)=1\,. \qquad \text{[E-7]}$$

The latter condition simply ensures that mating is random with respect to quality when females exhibit no preferences or when males do not produce an elaborate ornament. The functions A_i and B_i in [E-6] determine the local information content of the ornaments.

To facilitate further analysis, we assume that the male characters evolve on a fast timescale, relative to that on which female preferences evolve. This assumption is valid when the cost of choice and the mutation bias are small relative to the other parameters, as is normally assumed in models of sexual selection. If the male characters evolve on a fast timescale, they are always close to their optimum values. Consequently, we may assume that

$$t_i=p_i\,\mathrm{T}_i(p_i)\,. \qquad \text{[E-8]}$$

We now substitute [E-4] and [E-6] into the expression for the fitness gradients λ_{p_1} and λ_{p_2}, evaluate the derivatives, and finally substitute in [E-8], which yields

$$\begin{aligned}\lambda_{p_i}=&-p_i\left(\Gamma_i(p_i)+\frac{p_i}{2}\frac{\partial\Gamma_i(p_i)}{\partial p_i}+p_j^2\,\Theta(p_1,p_2)+\frac{p_i\,p_j^2}{2}\frac{\partial\Theta(p_1,p_2)}{\partial p_i}\right)\\ &+\mu_A\hat{w}_A p_i\left(\mathrm{T}_i(p_i)\,A_i\left(p_i,p_i\,\mathrm{T}_i(p_i)\right)+p_i\,\mathrm{T}_i(p_i)\left.\frac{\partial A_i(p_i,t_i)}{\partial p_i}\right|_{t_i=p_i\,\mathrm{T}_i(p_i)}\right)\\ &+\mu_B\hat{w}_B p_i\left(\mathrm{T}_i(p_i)\,B_i\left(p_i,p_i\,\mathrm{T}_i(p_i)\right)+p_i\,\mathrm{T}_i(p_i)\left.\frac{\partial B_i(p_i,t_i)}{\partial p_i}\right|_{t_i=p_i\,\mathrm{T}_i(p_i)}\right).\end{aligned} \qquad \text{[E-9]}$$

In the specific example model discussed earlier, the functions T_i, Γ_i, A_i and B_i were constant. The function Γ_i, for example, evaluated to the constant value γ_i, and the expression $\mathrm{T}_i(p_i)\,A_i\left(p_i,p_i\,\mathrm{T}_i(p_i)\right)$ evaluated to the information-content constant κ_i^A.

Let us now suppose that there exists an equilibrium $\tilde{p}_1>0$, $\tilde{t}_1>0$, $\tilde{p}_2>0$, $\tilde{t}_2>0$, at which females exhibit multiple preferences and males express multiple ornaments. Around this equilibrium, we approximate the functions T_i, Γ_i, Θ, A_i and B_i as follows

$$\begin{aligned}
\mathrm{T}_i(p_i) &\approx \tau_i + \tau_{ii}(p_i - \tilde{p}_i),\\
\Gamma_i(p_i) &\approx \frac{1}{2}\gamma_i + \frac{1}{6}\gamma_{ii}(p_i - \tilde{p}_i),\\
A_i(p_i, t_i) &\approx a_i + a_{ii}(p_i - \tilde{p}_i) + a'_{ii}(t_i - \tilde{t}_i),\\
B_i(p_i, t_i) &\approx b_i + b_{ii}(p_i - \tilde{p}_i) + b'_{ii}(t_i - \tilde{t}_i),\\
\Theta(p_1, p_2) &\approx \frac{1}{4}\vartheta + \frac{1}{12}\vartheta_1(p_1 - \tilde{p}_1) + \frac{1}{12}\vartheta_2(p_2 - \tilde{p}_2),
\end{aligned} \qquad \text{[E-10]}$$

where $i = 1 \text{ or } 2$, $j = 1 \text{ or } 2$ and $j \neq i$.

Since the reproductive values $\hat{w}_A$ and $\hat{w}_B$ are functions of R_A and R_B, respectively (see equation [B-11]), we may approximate them close to the equilibrium as follows:

$$\begin{aligned}
\hat{w}_A &\approx w_A + \upsilon_A\Big(\big(p_1 t_1 A_1(p_1, t_1) - \tilde{p}_1 \tilde{t}_1 A_1(\tilde{p}_1, \tilde{t}_1)\big) + \big(p_2 t_2 A_2(p_2, t_2) - \tilde{p}_2 \tilde{t}_2 A_2(\tilde{p}_2, \tilde{t}_2)\big)\Big),\\
\hat{w}_B &\approx w_B + \upsilon_B\Big(\big(p_1 t_1 B_1(p_1, t_1) - \tilde{p}_1 \tilde{t}_1 B_1(\tilde{p}_1, \tilde{t}_1)\big) + \big(p_2 t_2 B_2(p_2, t_2) - \tilde{p}_2 \tilde{t}_2 B_2(\tilde{p}_2, \tilde{t}_2)\big)\Big).
\end{aligned} \qquad \text{[E-11]}$$

We now linearize λ_{p_i} as follows:

$$\begin{aligned}
\lambda_{p_i} = -\frac{\tilde{p}_i}{2}&\left(\left(\frac{\gamma_{ii}}{2} + \tilde{p}_j^2 \frac{\vartheta_i}{4}\right)(p_i - \tilde{p}_i) + \left(\tilde{p}_j \frac{\vartheta_i \tilde{p}_i + \vartheta_j \tilde{p}_j}{6} + \vartheta \tilde{p}_j\right)(p_j - \tilde{p}_j)\right)\\
&+\mu_A w_A \tilde{p}_i\left(\tau_{ii} a_i + \tau_i a_{ii} + (a_{ii} + \tau_i a'_{ii})(\tau_i + \tilde{p}_i \tau_{ii})\right)(p_i - \tilde{p}_i)\\
&\mu_A \upsilon_A \tilde{p}_i^2 \tau_i (a_i + \tilde{p}_i a_{ii})\left(\tau_i (a_i + \tilde{p}_i a_{ii}) + (\tau_i + \tilde{p}_i \tau_{ii})(a_i + \tilde{p}_i \tau_i a'_{ii})\right)(p_i - \tilde{p}_i)\\
&\mu_A \upsilon_A \tilde{p}_i \tilde{p}_j \tau_i (a_i + \tilde{p}_i a_{ii})\left(\tau_j (a_j + \tilde{p}_j a_{jj}) + (\tau_j + \tilde{p}_j \tau_{jj})(a_j + \tilde{p}_j \tau_j a'_{jj})\right)(p_j - \tilde{p}_j)\\
&+\mu_B w_B \tilde{p}_i\left(\tau_{ii} b_i + \tau_i b_{ii} + (b_{ii} + \tau_i b'_{ii})(\tau_i + \tilde{p}_i \tau_{ii})\right)(p_i - \tilde{p}_i)\\
&+\mu_B \upsilon_B \tilde{p}_i^2 \tau_i (b_i + \tilde{p}_i b_{ii})\left(\tau_i (b_i + \tilde{p}_i b_{ii}) + (\tau_i + \tilde{p}_i \tau_{ii})(b_i + \tilde{p}_i \tau_i b'_{ii})\right)(p_i - \tilde{p}_i)\\
&\mu_B \upsilon_B \tilde{p}_i \tilde{p}_j \tau_i (b_i + \tilde{p}_i b_{ii})\left(\tau_j (b_j + \tilde{p}_j b_{jj}) + (\tau_j + \tilde{p}_j \tau_{jj})(b_j + \tilde{p}_j \tau_j b'_{jj})\right)(p_j - \tilde{p}_j),
\end{aligned} \qquad \text{[E-12]}$$

where we have made use of the fact that the fitness gradients vanish when evaluated at the equilibrium.

In matrix notation, the linearized system [E-12] can be represented as

$$\begin{pmatrix} \lambda_{p_1} \\ \lambda_{p_2} \end{pmatrix} = \mathbf{J}\begin{pmatrix} p_1 - \tilde{p}_1 \\ p_2 - \tilde{p}_2 \end{pmatrix}. \qquad \text{[E-13]}$$

We choose to decompose the Jacobian matrix $\mathbf{J}$ into separate matrices, representing the effects of the cost and benefits of choice. To be precise,

$$\mathbf{J} = -\mathbf{C} + \mathbf{B} + \mathbf{E}. \qquad \text{[E-14]}$$

Here,

$$\mathbf{C} = \frac{\tilde{p}_1\tilde{p}_2}{2}\begin{pmatrix} 0 & \vartheta \\ \vartheta & 0 \end{pmatrix}, \qquad \text{[E-15]}$$

incorporates the effect of the joint costs of choice. The matrix $\mathbf{B}$ incorporates the benefits associated with exhibiting multiple preferences. It can be further decomposed as

$$\mathbf{B} = 2\mathbf{P}\,\mathbf{T}\,\mathbf{K}\mathbf{G}\mathbf{K}^{\mathrm{T}}\,\mathbf{T}\,\mathbf{P}, \qquad \text{[E-16]}$$

where,

$$\mathbf{P} = \begin{pmatrix} \tilde{p}_1 & 0 \\ 0 & \tilde{p}_2 \end{pmatrix}, \quad \mathbf{T} = \begin{pmatrix} \tau_1 & 0 \\ 0 & \tau_2 \end{pmatrix}, \quad \mathbf{K} = \begin{pmatrix} a_1 & b_1 \\ a_2 & b_2 \end{pmatrix}, \quad \mathbf{G} = \begin{pmatrix} \mu_A \upsilon_A & 0 \\ 0 & \mu_B \upsilon_B \end{pmatrix}. \qquad \text{[E-17]}$$

The matrix $\mathbf{E}$, finally, incorporates higher-order effects, such as changes in the baseline costs of choice (as determined by the γ_{ii}), changes in the joint cost of choice (as determined by the ϑ_i), changes in the rate at which male investment into ornaments responds to a change in female choosiness (as determined by the τ_{ii}) and changes in the overlap in the information conveyed by the ornaments (as determined by the a_{ii}, a'_{ii}, b_{ii}, b'_{ii}). Ignoring the interactions between such higher order effects,

$$\mathbf{E} = \mathbf{E_C} + \mathbf{E_G^A} + \mathbf{E_G^B} + 2\mathbf{P}^2\mathbf{T}\mathbf{E_K}\mathbf{G}\mathbf{K}^{\mathrm{T}}\mathbf{T}\mathbf{P} + \mathbf{P}\mathbf{T}\mathbf{K}\mathbf{G}\left(\mathbf{T}\mathbf{E_K} + \mathbf{T}^2\mathbf{E}'_{\mathbf{K}} + \mathbf{E_T}\mathbf{K}\right)^{\mathrm{T}}\mathbf{P}^2, \qquad \text{[E-18]}$$

where

$$\mathbf{E_C} = \begin{pmatrix} -\frac{\tilde{p}_1}{4}\left(\gamma_{11} + \frac{\vartheta_1\tilde{p}_2^2}{2}\right) & -\frac{\tilde{p}_1\tilde{p}_2}{12}\left(\tilde{p}_1\vartheta_1 + \tilde{p}_2\vartheta_2\right) \\ -\frac{\tilde{p}_1\tilde{p}_2}{12}\left(\tilde{p}_1\vartheta_1 + \tilde{p}_2\vartheta_2\right) & -\frac{\tilde{p}_2}{4}\left(\gamma_{22} + \frac{\vartheta_2\tilde{p}_1^2}{2}\right) \end{pmatrix},$$

$$\mathbf{E_G^A} = \begin{pmatrix} \mu_A w_A \tilde{p}_1\left(\tau_{11}a_1 + \tau_1 a_{11} + \left(a_{11} + \tau_1 a'_{11}\right)\left(\tau_1 + \tilde{p}_1\tau_{11}\right)\right), & 0 \\ 0, & \mu_A w_A \tilde{p}_2\left(\tau_{22}a_2 + \tau_2 a_{22} + \left(a_{22} + \tau_2 a'_{22}\right)\left(\tau_2 + \tilde{p}_2\tau_{22}\right)\right) \end{pmatrix}, \qquad \text{[E-19]}$$

$$\mathbf{E_G^B} = \begin{pmatrix} \mu_B w_B \tilde{p}_1\left(\tau_{11}b_1 + \tau_1 b_1 + \left(b_{11} + \tau_1 b'_{11}\right)\left(\tau_1 + \tilde{p}_1\tau_{11}\right)\right), & 0 \\ 0, & \mu_B w_B \tilde{p}_2\left(\tau_{22}b_2 + \tau_2 b_2 + \left(b_{22} + \tau_2 b'_{22}\right)\left(\tau_2 + \tilde{p}_2\tau_{22}\right)\right) \end{pmatrix},$$

$$\mathbf{E_K} = \begin{pmatrix} a_{11} & b_{11} \\ a_{22} & b_{22} \end{pmatrix}, \quad \mathbf{E}'_{\mathbf{K}} = \begin{pmatrix} a'_{11} & b'_{11} \\ a'_{22} & b'_{22} \end{pmatrix}, \quad \mathbf{E_T} = \begin{pmatrix} \tau_{11} & 0 \\ 0 & \tau_{22} \end{pmatrix}.$$

If we assume that the higher-order effects are small, we find, after some manipulation

$$\begin{aligned} \det(\mathbf{J}) &= \det(\mathbf{C}) + \det(\mathbf{K}) + 2[\mathbf{C}]_{1,2}\,[\mathbf{K}]_{1,2} \\ &= \tilde{p}_1^2\tilde{p}_2^2\left(-\frac{\vartheta^2}{4} + 4\tau_1^2\,\tau_2^2\,g_A\,g_B\left(a_1b_2 - a_2b_1\right)^2 - 2\vartheta\tau_1\tau_2\left(g_A\,a_1\,a_2 + g_B\,b_1\,b_2\right)\right), \qquad \text{[E-20]} \\ \mathrm{tr}(\mathbf{J}) &= \mathrm{tr}(\mathbf{C}) + \mathrm{tr}(\mathbf{K}) \\ &= -2\,g_A\left(\tilde{p}_1^2\tau_1^2a_1^2 + \tilde{p}_2^2\tau_2^2a_2^2\right) - 2\,g_B\left(\tilde{p}_1^2\tau_1^2b_1^2 + \tilde{p}_2^2\tau_2^2b_2^2\right). \end{aligned}$$

where $g_A = -\mu_A \upsilon_A$ and $g_B = -\mu_B \upsilon_B$ are positive constants. Using [E-11], [E-6] and [B-11], it follows that

$$g_k = -\mu_k \upsilon_k = -\mu_k \frac{dw_k}{d(\ln(R_k))} = -\mu_k R_k \frac{dw_k}{dR_k} = \mu_k w_k (1+w_k) . \qquad \text{[E-21]}$$

Multiple preferences are stable when $\det(\mathbf{J}) > 0$ and $\mathrm{tr}(\mathbf{J}) < 0$. From [E-20] it is easy to see that the latter condition is always satisfied, implying that the equilibrium is either a stable node or a saddle point. The former condition can only be satisfied when $\vartheta < 0$; that is, when the joint cost of choice is low, or when $a_1 b_2 - a_2 b_1 \neq 0$ (i.e., when the ornaments provide non-overlapping information about quality). Note that, when the cost of choice is very low ($\vartheta < -8\tau_1\tau_2(g_A a_1 a_2 + g_B b_1 b_2)$), the equilibrium loses its stability. We do not consider this possibility to be realistic, however, since it corresponds to the implausible situation that the viability benefits of exhibiting multiple preferences outweigh the cost of choice for the independent preferences. Exhibiting multiple preferences then confers a viability advantage and preferences evolve to arbitrarily large values.

As it turns out, the information content parameters κ_i^k, used in the specific example model, and the joint cost of choice can be expressed in terms of parameters of the general model, as follows:

$$\begin{gathered} \kappa_1^A = \tau_1 a_1 , \quad \kappa_1^B = \tau_1 b_1 , \\ \kappa_2^A = \tau_2 a_2 , \quad \kappa_2^B = \tau_2 b_2 , \\ \vartheta_1 \gamma_1 + \vartheta_2 \gamma_2 = \vartheta/4 , \end{gathered} \qquad \text{[E-22]}$$

implying that [E-20] is equivalent to [C-18], the stability criterion for the internal equilibrium of the specific example model. From this, we may conclude that the results of our specific example model are generally applicable as long as higher-order effects do not play a crucial role.

If higher-order effects do contribute significantly to the selection gradients, we cannot neglect the matrix $\mathbf{E}$. In that case, multiple preferences can be stable, even when the joint cost of choice is high and the ornaments provide overlapping information about quality; for example, when the baseline costs of choice increase rapidly ($\gamma_{ii} > 0$), when the overlap in the information content of the ornaments decreases rapidly ($a_{ii} \neq 0$, $b_{ii} \neq 0$, $a'_{ii} \neq 0$, $b'_{ii} \neq 0$), or when increasing levels of preference are met with diminishing investment by males into their ornaments ($\tau_{ii} < 0$) (cf. Johnstone, 1996).

Sexual conflict and the evolution of female preferences for indicators of male quality

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G. Sander van Doorn & Franz J. Weissing

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ABSTRACT

Several seemingly general insights offered by theory on the evolution of mating preferences for good-gene indicators rely on the assumption that female preference and male ornament expression eventually converge on stable equilibrium levels. Examples of such results are the conclusion that no stable preference can evolve for ornaments that are pure epistatic indicators, or the conclusion that females should disregard all ornaments except the one that provides the most reliable information about genetic quality. We show that the assumption of equilibrium dynamics is problematic and characterize conditions under which female preference and male condition-dependent signaling continue to evolve without ever attaining equilibrium values. Such continual evolution is driven by the joint action of the handicap process and a sexual conflict over the information content of signals used in mate choice. Although the existence of this conflict has long been acknowledged, its consequences have never been formally investigated. By means of a standard model for good-genes sexual selection we demonstrate that several seemingly general conclusions of sexual selection theory, including those mentioned above, do no longer hold if the system does not converge to an equilibrium. In addition, our results provide a mechanism for the apparent frequent loss of sexually selected traits and they offer an alternative explanation for the evolution of preferences for multiple ornaments.

INTRODUCTION

Why do females so often show mating preferences for elaborate male ornaments that evidently reduce the male's viability? This question touches upon one of the central issues in sexual selection theory and it has led to the development of several theories for the evolution of female mating preferences (Andersson, 1994). One widely held view (reviewed in Maynard Smith, 1991) is that female choice based on costly male traits is adaptive, since the male ornaments, on which female preference acts, serve as indicators of 'good genes'.

A male ornament is an indicator of good genes when its level of expression correlates with the genetic quality of the male. From the female's point of view, it is obvious why it pays to choose males based on such an ornament: stronger female preferences for the male ornament translate into a higher probability that the female's offspring will inherit the good genes carried by the father. From the male's perspective, however, the function of the ornament as a quality signal is only of secondary importance. For males, the ornament predominantly functions to attract females and to increase mating success. Hence, there exists a conflict between the sexes over the information content of signals that are used to provide information about the male's genetic quality. Whereas female interests are served by ornaments that reliably signal the genetic quality of the male, it is in the interest of males to undermine the association between the good genes and the expression of the ornament as far as possible, such that males that do not carry the good genes are also able to attract females.

In view of this conflict, it has long been realized that the signaling function of the ornament must be induced by genetic or physiological processes that cannot easily be modified without large costs (potential mechanisms are discussed in, e.g., Clutton-Brock, 1982; Hamilton & Zuk, 1982; Møller, 1990; Andersson, 1994). In such a case, the male ornament handicaps male survival in such a way that cheating (i.e., expressing the ornament without carrying the good genes) is costly (Zahavi, 1975). To capture the essence of this idea, early theoretical models of the handicap process (e.g., Maynard Smith, 1985; Pomiankowski, 1987; Andersson, 1994, and references therein) assume that the genetic and physiological architecture of the ornament dictates a fixed pattern of condition-dependent ornament expression.

The early models successfully provide proof of principle that costly female preference may evolve for male traits that act as signals of genetic quality. However, by imposing a fixed relation between quality and ornament expression, these models completely eliminate the sexual conflict over the information content of the ornament. By doing so, they do not address the issue of the maintenance of condition-dependent signaling in a situation where males can adapt their investment into the ornament in relation to their own genetic quality.

As an important next step, Grafen (1990), Iwasa *et al.* (1991) and Iwasa & Pomiankowski (1994) therefore investigated the evolution of optimal patterns of condition-dependent investment into the ornament. Two conclusions can be drawn from their analyses. First, an optimal pattern of condition-dependent investment maximizes the fitness of males, irrespective of their quality, and not that of females. In particular, low-quality males will increase their investment into the ornament as long as this increases their reproductive success. This makes it more difficult for females to distinguish high-quality from low-quality males. Hence, the expression of the ornament by low-quality males reduces the information content of the ornament and the fitness of females. Second, condition-dependent signaling will only be maintained when the ornament is either a 'condition-dependent' or a 'revealing' handicap. By definition (e.g., Iwasa *et al.*, 1991), an ornament is a condition-dependent indicator, when it is more costly to produce for low-quality males; an ornament is a revealing indicator, if its state reflects the condition of the male, such that the attractiveness of low-quality males is lower than that of high-quality males, even if they invest equally into the ornament. In both cases, the optimal level of investment is lower for low-quality males, either because the cost of ornament expression increases more rapidly for such males (condition-dependent handicap), or because the benefit in terms of mating success increases more slowly (revealing handicap).

Like many other models of sexual selection, the analyses of Grafen (1990), Iwasa *et al.* (1991) and Iwasa & Pomiankowski (1994) focus on the evolutionary equilibria of the male condition-dependent signaling strategy, rather than on evolutionary rates and dynamics. This restriction is potentially problematic, for two reasons. First, it is well established an evolutionarily stable strategy (ESS, *sensu* Maynard Smith & Price, 1973) need not be attained in the course of evolution. In fact, evolution may lead away from an ESS (Abrams *et al.*, 1993). Second, the attainability of evolutionary equilibria may depend not only on selective factors but also on genetic details, such as the magnitude of genetic variances and covariances or the structure of the mutation process (Matessi & Di Pasquale, 1996). It has already been demonstrated that the latter problem is relevant for sexual selection theory: in the context of Fisherian sexual selection, a change of genetic variances and covariances may destabilize evolutionary equilibria, leading to a continual change of mating preferences (Iwasa & Pomiankowski, 1995).

Also for good-genes sexual selection, it has been demonstrated that the actual evolutionary outcome can be different than one would expect from equilibrium considerations. First, the processes that destabilize Fisherian sexual selection can also operate in good-genes models, when the good-genes mechanism acts only weakly (Iwasa & Pomiankowski, 1999). Second, the good-genes mechanism itself may also lead to instabilities (Houle & Kondrashov, 2002), especially when one takes into account that the sexual conflict over the information content of ornaments generates a potentially destabilizing feedback between the strategies of the two sexes: evolution of the female preference affects the benefit of expressing the ornament, and it has an impact on the evolution of the male signaling strategy. At the same time, evolution of male condition-dependent signaling tends to decrease the value of the ornament as an indicator of quality, and it reduces the optimal level of female choosiness. In fact, it is well known that sexual conflict often results in an endless arms race between the strategies of the sexes (Gavrilets, 2000).

Wiens (2001) mentions the feedback between female preference and male signaling as a possible explanation for the apparent frequent turnover and loss of sexually selected traits, arguing that female preference for an ornament (and, hence, the corresponding ornament itself) will disappear as soon as males manage to express their ornaments regardless of their genetic quality. In addition, Hill (1994) suggests sexual conflict in the evolution of quality indicators to be responsible for the elaboration of male signals from their initial rudimentary form to larger or more complex manifestations of the ornament. According to this idea, females are forced to assess additional and novel ornament features in order to still be able to estimate the quality of the male whenever the information content of male advertisement decreases. This may result in an evolutionary race, where a correlation between male ornament expression and male genetic quality can only be maintained through the establishment of increasingly complex female mating preferences and increasingly elaborate male ornaments (Hill, 1994).

AN OVERVIEW OF THE MODEL AND SOME METHODOLOGICAL REMARKS

To investigate the joint evolutionary dynamics of female preference and male condition-dependent signaling, we develop and analyze a formal model. The model allows us to study the combined action of good-genes sexual selection and sexual conflict over the information content of an ornament. The biological assumptions of our model are comparable to those made in a previous model of the handicap process with flexible male condition-dependent signaling (Iwasa & Pomiankowski, 1994). As in this previous model, we study the evolution of four quantitative traits: genetic quality, female mating preference for an ornament and investment into the ornament by high- and low-quality males. The genetic quality of an individual affects its viability. Moreover, it influences the costs of ornamentation and/or male attractiveness: the male ornament is a condition-dependent or revealing handicap. Female fitness depends on genetic quality and the cost of choice. Male fitness depends on genetic quality, the cost of ornament production and male mating success. The latter is determined - through mate choice - by the preferences of females and the realized size of the ornament.

We deviate from existing models in one aspect. Instead of using a quantitative-genetics framework (reviewed in Mead & Arnold, 2004), we use adaptive dynamics theory (Metz *et al.*, 1992; Dieckmann & Law, 1996; Hofbauer & Sigmund, 1998; Geritz *et al.*, 1998) to analyze the evolutionary dynamics. This allows us to express directly the fitness gradients in terms of parameters of the model, without the need to keep track of genetic variances and covariances, as is necessary in quantitative genetic models. Although it is possible to estimate equilibrium values of the genetic variances and covariances as functions of the model parameters (Pomiankowski & Iwasa, 1993, using theory developed by Barton & Turelli, 1991), this procedure is quite complex and involves restrictive assumptions. What is more, information about equilibrium values is probably not sufficient in our case. Since we are particularly interested in the evolutionary dynamics, we would need to keep track of the dynamics of variances and covariances. Again, this is possible but quite complicated (Barton and Turelli, 1991).

The adaptive dynamics method describes evolution as a mutation-limited process. New mutant alleles arise only rarely, such that the genetic variation in female preference and male condition-dependent signaling is typically small. Therefore, we consider only the interactions between mutant individuals and the predominant type in the resident population. Depending on the invasion fitness (Metz *et al.*, 1992) of the mutant allele, it may either disappear or replace the resident allele. Long-term evolution takes place as a sequence of such mutation and allele-substitution events.

The assumption of mutation-limited evolution simplifies the analysis of the model but, when applied to genetic quality, it does not allow for the presence of variation in this character. Since the handicap process relies on such variation, we explicitly model deleterious mutations in genetic quality. For the female preference and male sexual characters, the assumption of mutation limited-evolution has the consequence that we cannot address the effects of genetic covariances between preferences and male characters. Such covariances are instrumental in driving Fisher's runaway process of sexual selection (Fisher, 1930). Despite this restriction, we feel confident that our model captures the essence of evolution in polymorphic populations with small genetic variation. This conviction is based on the fact that the adaptive dynamics and quantitative genetics approach often yield similar results (Taylor, 1996a), on previous comparisons between a comparable mutation-limited model of good-genes sexual selection and individual-based computer simulations (CHAPTER 8 of this thesis), and on the results of Barton & Turelli (1991), who demonstrated that indirect selection forces on female preference, generated through covariance with male characters, are so weak that they are unlikely to qualitatively affect the evolution of female preference.

THE MODEL

GENETIC QUALITY

We initially assume that an individual's genetic quality can be represented as a one-dimensional quantity. Genetic quality is determined by an arbitrary number of diploid loci with two alleles at each locus (denoted **A** and **a**). The allele **A** confers a higher quality; the allele **a** confers a lower quality. Variation at the quality loci is maintained through biased deleterious mutations, which convert **A** alleles into **a** alleles. Deleterious mutations occur with probability μ per genome per generation.

To keep the model analytically tractable, we assume that the rate of beneficial mutations, which convert a low-quality to a high-quality allele can be neglected relative to the rate of deleterious mutations, and that μ is small itself, such that we may neglect the frequency of individuals that carry more than one low-quality allele. Under these simplifying assumptions, we need to keep track of the frequencies of two different types of individuals: individuals that carry only high-quality alleles (phenotype A) and individuals that carry a deleterious mutation at one of their quality loci (phenotype a). Throughout this paper, the index k will be used to refer to these quality phenotypes (i.e., $k = A$ or a).

VIABILITY SELECTION

We assume that generations are discrete and non-overlapping. Viability selection acts at the start of every generation. Individual survival probabilities vary with the individual's general (i.e., good-genes induced) viability, which is determined by the individual's genetic quality. The general viabilities associated with the different quality phenotypes are defined by the parameters v_k, where $v_A > v_a$.

In addition, individual survival probabilities are affected by the costs associated with the expression of preferences (for females) or ornaments (for males). These factors are assumed to interact multiplicatively with general viability. For females with quality phenotype k, the survival probability until the moment of reproduction is given by h_k^f, where

$$h_k^f = v_k \left(1 - c^f(p)\right). \qquad [1]$$

The cost of choice, $c^f(p)$, varies with female preference p. Females with larger absolute values of p exert stronger mating preferences (see below). Females with $p = 0$ mate at random. We assume that random mating minimizes the costs of choice and, specifically, that

$$c^f(p) = 1 - \exp\left(-\gamma\, p^2\right), \qquad [2]$$

where the parameter γ determines how rapidly the cost of choice increases with the strength of preference.

For males with quality phenotype k, the survival probability is given by h_k^m, where

$$h_k^m = v_k \left(1 - c_k^m(t_k)\right). \qquad [3]$$

The function c_k^m represents the cost of ornament production and it varies with t_k, the male's investment of resources into the ornament. As implied by the index k, male investment into the ornament is condition dependent, i.e., males may invest more or less resources into the ornament, depending on their genetic quality. In addition, we allow for the possibility that the male's genetic quality directly influences the cost of ornament production, as is commonly assumed in condition-dependent handicap models. Specifically, the ornament may be more costly to produce for low-quality males, such that, even when all types of males would invest equally t units of resources into their ornament, $c_a(t) > c_A(t)$. Hence, genetic quality may influence the cost of ornament production both directly and indirectly, through a condition-dependent handicap mechanism and through condition-dependent investment into the ornament.

The phenotypic characters t_k are expressed relative to some value optimal for male survival (implying that also negative values of t_k are biologically meaningful), such that $t_k = 0$ is the optimal investment into the ornament when only viability selection is considered. The cost of ornament production increases for males that deviate from the viability selection optimum according to

$$c_k^m(t_k) = 1 - \exp\left(-\beta_k\, t_k^2\right), \qquad [4]$$

where the parameters β_k determine the intensity of stabilizing selection on the investment of resources into the ornament. In general, $\beta_a \geq \beta_A$, and, when the ornament is a condition-dependent handicap, $\beta_a > \beta_A$.

MATE CHOICE

For the sake of concreteness, let us suppose that females evaluate males based on the realized size, $S_k(t_k)$, of their ornament. Ornament size is again expressed relative to the value optimal for male survival. Males that invest more into their ornament will produce a larger ornament. Hence, $S_k(t_k)$ is an increasing function of t_k. In addition to this, we allow for the possibility that the realized size of an ornament is directly affected by the quality of the male, as is commonly assumed in revealing-handicap models.

Before a female mates, she evaluates the available males based on the size of their ornaments and her own preferences. Females have a higher probability of mating with an 'attractive' male, where attractiveness is quantified by a function r_k. The attractiveness of a male with quality phenotype k is proportional to

$$r_k(p, t_k) = \exp\left(p\, S_k(t_k)\right). \qquad [5]$$

In this mate-choice model, also known as the 'psychophysical' or 'open-ended' model of mate preference (Lande, 1981), positive values of p indicate preferences for larger ornaments, negative values of p indicate preferences for smaller ornaments, and females with $p = 0$ mate at random. A female mates only once. The probability that a given male is allowed to father her offspring is given by the male's attractiveness relative to the average attractiveness of the other males present in the population.

For all males, the realized size of an ornament is proportional to the investment into that ornament. Yet, if the ornament is a revealing indicator of quality, low-quality males must invest more to attain the same level of ornament elaboration and, hence, attractiveness. Therefore, we assume that

$$S_k(t_k) = t_k\,\alpha_k. \tag{6}$$

The parameters α_k reflect how efficiently male investment into the ornament translates into larger (more attractive) ornament for a male with quality phenotype k. All else being equal, high-quality males are more attractive than low-quality males, or, at the very least, equally attractive. Hence, $\alpha_A \geq \alpha_a$.

A SPECIFIC EXAMPLE

The values of the parameters α_k and β_k are determined by a variety of biological factors, such as female perception, the physiology of male ornament production and the ecological setting of mate choice. From the female's point of view, these parameters determine, directly and indirectly, how easily a female can discriminate between high- and low-quality males. From the male's point of view, they determine how easily a male can produce an elaborate ornament without actually carrying high-quality alleles. To illustrate this, let us compare two different species, where, for specific ornaments, the parameters α_k and β_k are given by

species 1: $\alpha_A = 2 \quad \alpha_a = 1 \quad \beta_A = 1 \quad \beta_a = 5$
species 2: $\alpha_A = 2 \quad \alpha_a = 1.99 \quad \beta_A = 1 \quad \beta_a = 1.01$

With equal investment into their ornament, high-quality males of species 1 will produce an ornament twice the size of that produced by low-quality males (this is because $\alpha_A = 2\,\alpha_a$ in species 1). Consequently, even a low level of preference will allow females of species 1 to discriminate accurately between high- and low-quality males, provided, of course, that low-quality males do not invest at least twice as much into their ornament (such that $t_a > 2\,t_A$). The latter is, however, quite unlikely to evolve, since doubling the investment into the ornament would drastically reduce the viability of low-quality males. In fact, the marginal cost of ornament production is much higher for low-quality males ($\beta_a = 5\,\beta_A$ in species 1) such that these males would already pay a markedly higher cost of ornament production even if they invested an equal amount of resources into their ornament as high-quality males.

The situation is markedly different in species 2. With equal investment into their ornament, two males that differ only in their quality will produce ornaments of roughly the same size (this is because $\alpha_A \approx \alpha_a$). Therefore, the females of species 2 will not be able to discriminate between high- and low-quality males, unless they evolve extremely high levels of preference, or unless low-quality males invest fewer resources into their ornaments. There is, however, no reason why the latter should be so: when low-quality males invest more resources into their ornament, they enjoy the same increase of attractiveness as high-quality males without incurring much higher costs (because $\beta_a \approx \beta_A$). Hence, one would expect low-quality males to invest the same amount of resources into their ornament as high quality males, such that, eventually, $t_a \approx t_A$.

INFORMATION CONTENT

The example illustrates that ornaments can differ with respect to their information content. For some ornaments a low level of preference is sufficient to accurately distinguish high-quality from low-quality males (we will say that such ornaments have a high information content), for other ornaments females have to evolve high levels of preference to do so (such ornaments have a low information content). The information content of an ornament is determined by two factors. First, it depends on the constraints on male ornament expression that are imposed by the parameters α_k and β_k. Second, it depends on the pattern of condition-dependent ornament expression. The latter is determined by the current distribution of the male strategic variables t_k.

The parameters α_k and β_k capture constraints on male condition-dependent signaling that arise from the genetic and physiological architecture of the male ornament. They determine what one could call the 'intrinsic' information content of the ornament. For example, when $\alpha_A > \alpha_a$ and $\beta_A = \beta_a$, the ornament intrinsically provides information about genetic quality through a revealing handicap mechanism; when $\alpha_A = \alpha_a$ and $\beta_A < \beta_a$, it does so through a condition-dependent handicap mechanism; and finally, when $\alpha_A > \alpha_a$ and $\beta_A < \beta_a$ the ornament is a revealing- and a condition-dependent handicap at the same time. In contrast, the ornament does not intrinsically provide information about genetic quality when $\alpha_A = \alpha_a$ and $\beta_A = \beta_a$ (in this case, the ornament is a pure epistatic indicator; Maynard Smith, 1991). We emphasize that our use of the terms revealing handicap, condition-dependent handicap and epistatic indicator always refers to the intrinsic information content of the ornament, and not necessarily to its realized information content.

Indeed, in all of these cases, the 'realized' information content of the ornament could be different from its intrinsic information content, due to the condition-dependent signaling strategy of males. For example, if the ornament is a pure epistatic indicator, it may still convey information about genetic quality if, for some reason, low-quality males invest less into the ornament than high-quality males. In this case, the realized information content is larger than the intrinsic information content. Similarly, if the ornament is a revealing handicap, it may still provide no information whatsoever about quality. Suppose, for example that low-quality males invest more into the ornament than high-quality males, such that they compensate exactly for their reduced attractiveness. In this case, the realized information content is obviously smaller than the intrinsic information content of the ornament.

It is clear that further evolution will take place whenever there is a discrepancy between the realized and the intrinsic information content of the ornament. For example, if the realized information content is higher than the intrinsic information content of the ornament, low-quality males could easily increase their mating success by evolving stronger expression of the ornament. In the opposite case, low-quality males experience relatively high costs of signaling and they are expected to reduce their investment into the ornament. Intuitively, one would expect the realized information content of an ornament to agree with its intrinsic information content in evolutionary equilibrium. Indeed, in the following section, we demonstrate how this intuition can be formalized. This does not imply, however, that such an equilibrium will ever be attained in the course of evolution. This issue will be addressed in a subsequent section of the paper, which deals with evolutionary dynamics and the stability of evolutionary equilibria.

EVOLUTIONARY EQUILIBRIA

We study the long-term evolution of the female mating preference and the pattern of condition- dependent ornament expression in males by considering evolution as a sequence of mutation and allele-substitution events. Specifically, we assume that the genes coding for the female preference and investment into the ornament by males mutate only rarely. This allows us to focus on a population with negligible variation for these traits. We then consider an initially rare mutant allele that causes individuals to differ slightly in one of their phenotypic components from the resident individuals, and we investigate whether this mutant allele will be able to increase in frequency or not. Mathematically, the potential for the mutant allele to spread is determined by the mutant's invasion fitness λ, i.e., by the long-term geometric rate of increase of the mutant allele frequency in the context set by the current resident alleles (Metz *et al.*, 1992). The mutant will spread if its geometric rate of increase is positive, i.e., when its invasion fitness is larger than that of the resident allele.

The fitness difference between mutant and resident alleles is approximated by the gradient of the invasion fitness function. Hence, this gradient determines the direction of evolutionary change. If the selection gradient is positive, subsequent mutation and trait substitution events will lead to an increase of the trait under consideration (and vice versa, if it is negative).

GENERAL PROPERTIES OF EVOLUTIONARY EQUILIBRIA

A derivation of the invasion fitness function and its gradients with respect to the different evolutionary characters is given in APPENDIX A. Only the end result of this derivation is given here. The selection gradients with respect to the male characters, denoted λ_{t_k}, are given by

$$\lambda_{t_k} = \frac{\partial \lambda}{\partial t_k} = b_k \frac{\partial \ln\left(r_k(p,t_k)\left(1-c_k^m(t_k)\right)\right)}{\partial t_k}, \qquad [7]$$

where the coefficients b_k are strictly positive (see APPENDIX A).

Under suitable assumptions, evolution proceeds in a direction correlated with the direction of the selection gradient (Dieckmann & Law, 1996; Hofbauer & Sigmund, 1998). Potential endpoints of evolution are therefore points where the selection gradient vanishes (i.e., points where $\lambda_{t_k} = 0$). In view of [7], male condition-dependent investment into the ornament will therefore evolve to maximize $r_k(p,t_k)\left(1-c_m^k(t_k)\right)$, the product of male attractiveness and male survival for each male quality phenotype. Under quite general assumptions on the functions r_k and c_k^m, this implies that, in equilibrium, low-quality males will invest less resources into the ornament, because the cost of ornamentation increases more rapidly for such males (condition-dependent handicap), because attractiveness increases less rapidly (revealing handicap), or because both of these mechanisms act at the same time.

A similar intuitive interpretation can be given for λ_p, the selection gradient with respect to female preference. In APPENDIX A, we derive that

$$\lambda_p = \frac{\partial \lambda}{\partial p} = \frac{1}{2}\frac{\partial \ln\left(1-c^f(p)\right)}{\partial p} + \frac{1}{2}\mu\, w \frac{\partial \ln\left(R(p,t_A,t_a)\right)}{\partial p}, \qquad [8]$$

where w is the reproductive value of males that carry a low-quality allele, and where the function R measures the attractiveness of high-quality males relative to that of males carrying a deleterious mutation, i.e.,

$$R(p,t_A,t_a) = \frac{r_A(p,t_A)}{r_a(p,t_a)}. \qquad [9]$$

According to equation [8], female choosiness evolves to a level at which the costs and benefits of choice are balanced. When there is no variation among males (no mutation bias, $\mu = 0$), female preference will evolve to maximize $1-c^f(p)$, implying that the cost of choice is minimized. When mutation bias creates variation among males, female preference will evolve away from its viability-selection optimum to a point where $\partial \ln\left(1-c^f(p)\right)/\partial p$ is negative, provided that $\partial \ln(R)/\partial p$ is sufficiently larger than zero (for the moment, we suppose, without loss of generality, that p, t_A and t_a are positive).

The latter term has an interesting biological interpretation. For females that mate at random, low-quality males are, by definition, as attractive as high-quality males. To females that exhibit stronger preferences, however, high-quality males will appear increasingly attractive, because such males invest more into their ornament (this is to be expected when the ornament is a condition-dependent handicap), or because the ornament is a revealing indicator. Therefore, R is a monotonically increasing function of p. The term $\partial \ln(R)/\partial p$ represents the rate at which the relative attractiveness of high-quality males increases with female preference and it reflects the detectability of quality differences. It is related to the realized information content of the ornament, which we define as the detectability of quality differences relative to the level of choosiness, i.e., as $1/p \cdot \partial \ln(R)/\partial p$.

If $\partial \ln(R)/\partial p$ is only slightly larger than zero, the relative attractiveness of high-quality males increases only slowly with preference, even when female preference is already high. Therefore, females must evolve high levels of choosiness before they can accurately distinguish high-quality from low-quality males. In this situation, the realized information content of the ornament is low, meaning that the ornament is a poor indicator for genetic quality. On the other hand, when $\partial \ln(R)/\partial p$ is large, even low levels of choosiness will allow females to select high-quality males. In this case, the ornament has a high realized information content.

It is a direct consequence of [8] that the female preference p will converge on its viability selection optimum (maximal $1-c^f$) when the ornament does not provide information about quality (i.e., when $\partial \ln(R)/\partial p = 0$).

EVOLUTIONARY EQUILIBRIA FOR OUR SPECIFIC MODEL

After these general considerations, we now return to the specific model defined earlier. Hence, we substitute specific functions for r_k, c_k^m and c^f, as defined in equations [2] and [4]-[6]. This immediately yields simple expressions for the equilibrium investment of resources, t_A^* and t_a^*, into the ornament by males:

$$t_k^* = p^* \frac{\alpha_k}{2\beta_k}. \qquad [10]$$

Depending on whether the ornament is a revealing indicator or a condition-dependent handicap, either α_k is smaller or β_k is larger for low-quality males. For both types of handicaps and at equilibrium, low-quality males will therefore invest less into their ornaments.

The equilibrium female preference, p^*, cannot be obtained in a simple explicit form. From equation [8], it follows that,

$$-\frac{\partial \ln\left(1-c^f\left(p^*\right)\right)}{\partial p^*} = \mu\, w^* \frac{\partial \ln\left(R\left(p^*, t_A^*, t_a^*\right)\right)}{\partial p^*}, \qquad [11]$$

which implies that,

$$2\gamma\, p^* = \mu\, w^*\, p^* \left(\frac{1}{p^*} \frac{\partial \ln\left(R\left(p^*, t_A^*, t_a^*\right)\right)}{\partial p^*} \right). \qquad [12]$$

As indicated by the asterisk, the reproductive value w^* is a function of p^* (see APPENDIX A). The factor between brackets on the right-hand side is the realized information content of the ornament in equilibrium. When it is zero, there can be no stable costly mate preference (in this case, the only equilibrium solution is $p^* = 0$).

We now use equations [9] and [10], to find that the realized information content of the ornament in equilibrium equals

$$\frac{1}{p^*} \frac{\partial \ln\left(R\left(p^*, t_A^*, t_a^*\right)\right)}{\partial p^*} = \frac{1}{p^*}\left(\alpha_A\, t_A^* - \alpha_a\, t_a^*\right) = \frac{\alpha_A^2}{2\beta_A} - \frac{\alpha_a^2}{2\beta_a}. \qquad [13]$$

We interpret the constant on the right-hand side as the intrinsic information content of the ornament, since it only depends on the inherent characteristics of the ornament that are represented by the parameters α_k and β_k, and not on the male signaling strategy. Equation [13] corroborates the intuition that the realized information content of an ornament (on the left-hand side) should equal its intrinsic information content (on the right-hand side) in evolutionary equilibrium. When the ornament is a revealing handicap ($\alpha_A > \alpha_a$), a condition-dependent handicap ($\beta_A < \beta_a$), or both at the same time, the intrinsic information content of the ornament is strictly positive. Contrastingly, when $\alpha_A = \alpha_a$ and $\beta_A = \beta_a$, the ornament is a pure epistatic indicator, and its intrinsic information content is zero.

From [10] and [12], it is easy to see that there always exists a trivial equilibrium $p^* = t_k^* = 0$ at which females mate at random. In addition there exists a pair of non-trivial equilibria ($p^* = \pm\tilde{p}$, $\tilde{p} > 0$), where females choose males with costly ornaments, when

$$2\gamma < \mu \frac{\frac{\alpha_A^2}{2\beta_A} - \frac{\alpha_a^2}{2\beta_a}}{2\frac{v_A}{v_a} - 1}, \qquad [14]$$

that is, when the intensity of sexual selection for increased choosiness outweighs the intensity of stabilizing viability selection on female preference.

EVOLUTIONARY DYNAMICS AND THE STABILITY OF EQUILIBRIA

In the previous section we identified evolutionary equilibria by searching for points at which the selection gradients vanish. To determine whether evolution will actually proceed towards these equilibria, we must determine the stability properties of the equilibria. This requires us to be more specific about the actual evolutionary dynamics of the female preference and the male investment pattern.

The evolutionary dynamics of traits is determined not only by the direction of the selection gradient but also by genetic factors. In quantitative genetics models, for example, these genetic factors are incorporated by a genetic variance-covariance matrix, which determines how easily selection can lead to an evolutionary response in a specific phenotypic direction. In adaptive dynamics models, the genetic variance-covariance matrix does not play a role, since evolution is assumed to be mutation-limited. Instead, adaptive dynamics models have to deal with genetic constraints imposed by the mutation process. For example, evolution may preferentially proceed in certain phenotypic directions due to the fact that mutations are more likely to result in phenotypic deviations in these directions. Alternatively, selection on one trait may lead to a correlated response in another due to the fact that mutations of the one trait have pleiotropic effects on the other.

Indeed, following Dieckmann & Law (1996), the evolutionary rate of change in the female preference p and the male investment strategy t_k is given by

$$\frac{d}{d\tau}\begin{pmatrix} p \\ t_A \\ t_a \end{pmatrix} = \rho\, \mathbf{G} \begin{pmatrix} \lambda_p \\ \lambda_{t_A} \\ \lambda_{t_a} \end{pmatrix}, \qquad [15]$$

where the matrix $\mathbf{G}$ is the mutational variance-covariance matrix, which contains as elements the mutational variances and covariances of the different traits. The proportionality constant ρ scales the rate of evolution with the rate at which beneficial mutations arise (ρ depends on the population size and the mutation rate; Dieckmann & Law, 1996). Finally, τ represents evolutionary time. A unit of evolutionary time roughly corresponds to the generation time if mutation on average creates a single new mutant phenotype per generation in the entire population; it is of the order of one tenth of the generation time, when mutation creates on average ten new mutant phenotypes per generation, and so on.

MUTATIONS WITH PLEIOTROPIC EFFECTS

In view of [15], the attainability, or convergence stability, of equilibria depends on the direction of selection in the vicinity of the equilibrium. An equilibrium is bound to be convergence stable when the selection gradients point towards the equilibrium from whatever direction it is approached (Leimar, 2001). However, if this is not the case, convergence stability may also be qualitatively affected by the underlying genetics (Matessi & Di Pasquale, 1996). This implies that an equilibrium may only be attained if the mutational variance-covariance matrix satisfies certain conditions.

As illustrated in FIGURE 1, this is especially relevant for our model. The figure shows two numerical simulations of the dynamical system [15] (APPENDIX B, equation [B-1]) that differ only with respect to the mutational variance-covariance matrix $\mathbf{G}$. For the sake of concreteness, we supposed that the female preference p and the male investment strategies t_k are encoded by a large number of genes, and that a fraction f of the male investment genes is expressed irrespective of the genetic quality of the male. Hence, a mutation in one of these genes has pleiotropic effects on the characters t_A and t_a simultaneously. These pleiotropic effects enter the matrix $\mathbf{G}$ as mutational covariances (APPENDIX B). By varying f we can smoothly vary between two extreme scenarios. Male investment into the ornament is completely independent of genetic quality if $f = 1$. In this case, all male investment genes are expressed irrespective of male quality, such that all males invest equally into the ornament. Conversely, the male characters t_A and t_a evolve completely independently if $f = 0$. In this case, there are no genes that are expressed in both high- and low-quality males, such that mutations always affect the phenotype of either high- or low-quality males, but not both at the same time.

If the overlap between the male investment genes is large enough, evolution converges on a stable non-trivial equilibrium, as in the upper panel of FIGURE 1, where $f = 0.4$. In equilibrium, a stable level of female preference is attained, and also the male investment pattern reaches a stable state where $t_a = 4/5\, t_A$ (this is because $\alpha_a = 4/5\, \alpha_A$ and $\beta_a = \beta_A$; cf. equation [10]). However, when we decrease the overlap between the male ornament genes, the equilibrium loses its stability (FIGURE 1, lower panel; $f = 0.3$). Instead of attaining stable equilibrium values, the female preference and the investment into the ornament by males now continue to fluctuate, and eventually approach a stable limit cycle. This limit cycle is projected in FIGURE 2 (black curve), together with some illustrative trajectories (gray) that connect the different equilibria to one another and to the limit cycle. In this figure, p and t_A are both represented on the horizontal axis. This is possible, since t_A is always close to its quasi-equilibrium value (equation [10]), and, hence, proportional to p (see also FIGURE 1, where the lines representing p and t_A are virtually overlapping).

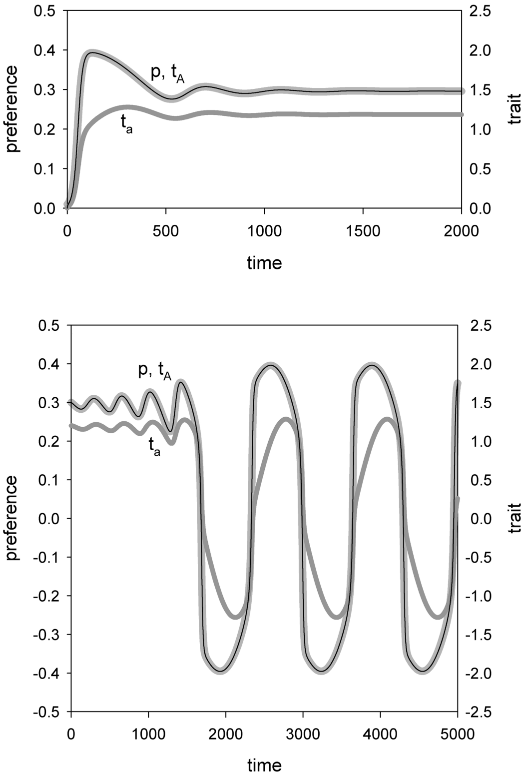


FIGURE 1 – GENETIC DETAILS AFFECT THE STABILITY OF EQUILIBRIA

Upper panel: female preference and male investment into the ornament reach a stable equilibrium where females are choosy and where males develop costly ornamentation. The equilibrium loses its stability when the pleiotropic effects of mutations in male investment genes are decreased. The simulation shown in the lower panel starts from initial conditions close to the equilibrium. Female preference and male investment levels diverge from the equilibrium until they eventually attain a stable limit cycle. Parameters are $\alpha_A = 5.0$, $\alpha_a = 4.0$, $\beta_A = \beta_a = 0.5$, $\gamma = 0.05$, $\mu = 0.05$, $v_A = 1.0$, $v_a = 0.5$.

The continual evolution of the female mating preference and the male condition-dependent investment pattern along the limit cycle is driven by a sexual conflict over the information content of the ornament. Such a conflict can arise when there is a potentially large discrepancy between the realized and intrinsic information contents of the ornament. Indeed, in the simulations shown in FIGURE 1 & 2, we assumed that the ornament was a revealing indicator with a low intrinsic information content. This does not necessarily imply, however, that females can never use the ornament to gain accurate information about genetic quality. Imagine, for example, that female preference is weak and that, for some reason $t_a < t_A$, as is the case at point A in FIGURE 2. In this situation, females benefit from evolving preference, since females can very easily discriminate between high- and low-quality males based on the ornament. This is not so much because the ornament intrinsically provides a lot of information about quality, but rather because of the pattern of condition-dependent investment that is currently adopted by the male population. In other words, although the intrinsic information content of the ornament is low, its realized information content is not.

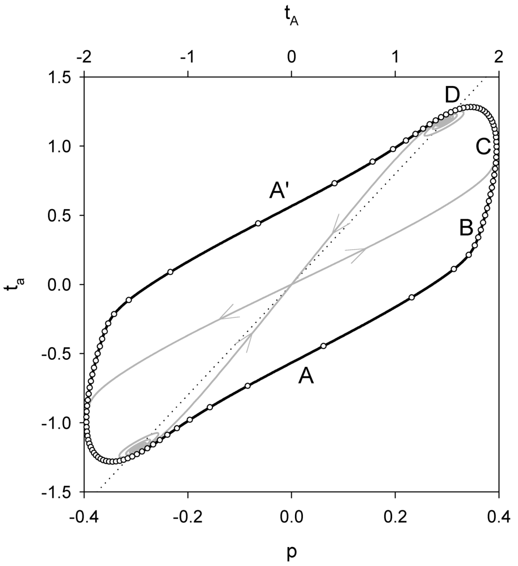


FIGURE 2 – CONTINUAL EVOLUTION ALONG A LIMIT CYCLE

Sexual conflict over the information content of the ornament drives the female preference and the male investment strategy along a limit cycle (black curve). White circles are plotted at regular time intervals, to give an indication of the rate of evolution along the cycle. Gray trajectories connect the trivial and non-trivial equilibria to one another and to the limit cycle. The dotted line marks the optimal investment of resources into the ornament by low-quality males, for any given level of female preference. Parameters are as in FIGURE 1.

In view of the high realized information content of the ornament, it is not surprising that females rapidly evolve preference for the ornament up to the level where the benefit of choice is balanced by the cost (point B in FIGURE 2). Along with the increase of female choosiness, males increase their investment into the ornament, as one would predict based on equation [10]. Since the ornament is an indicator that reveals only limited information about quality ($\alpha_a = 4/5\,\alpha_A$ and $\beta_a = \beta_A$), one would expect this to lead to a situation where $t_a = 4/5\,t_A$, as in the upper panel of FIGURE 1. Although this situation is eventually attained (point D in FIGURE 2; the dotted line is to indicate all points where $t_a = 4/5\,t_A$), it is only approached slowly. Apparently, the male character t_a evolves at a very low rate. This has an important consequence: for a long time the ornament continues to function as an indicator with a high realized information content, even though its intrinsic information content is low.

There are two reasons for the low rate of evolution of the male character t_a. First, t_a is expressed only in low-quality males. The frequency of such males is of the order of the mutation rate μ, such that t_a is hardly exposed to selection. Second, selection on the other male characters only weakly affects t_a (through pleiotropic mutations), since the overlap between male investment genes is low ($f = 0.3$ in FIGURE 2).

Even though the male character t_a only slowly approaches its optimal value, low-quality males will inevitably catch up with the high-quality males. The realized information content of the ornament will therefore eventually decrease to match the intrinsic information content (point D in FIGURE 2). Initially (between point B and C in FIGURE 2), females respond to the gradual erosion of the realized information content of the ornament by evolving stronger preferences. By doing so, females can continue to distinguish between high- and low-quality males, even though the difference between these males has become smaller (cf. Hill, 1994). Eventually, this strategy turns out to be fruitless. Evolving stronger preferences yields higher costs of choice, whereas it cannot prevent that the benefit of choice is gradually eroded further. In the end, females have evolved strong preferences for an ornament that now only provides rather limited information about genetic quality. The costs of choice are no longer balanced by the benefits, and the female preference collapses (point C in FIGURE 2).

The decrease of female preference is immediately tracked by a reduction of the investment by high-quality males. Low-quality males again respond much more slowly. Beyond point D in FIGURE 2, low-quality males invest even more resources into their ornament than high-quality males. This provides an additional selection pressure for the females to lower their preference for the ornament, until it is lost completely (point A′ in FIGURE 2). From this point onwards, the whole process repeats itself in the other direction.

AN EXPLORATION OF DIFFERENT PARAMETER CONDITIONS

Due to the large number of parameters, it is hard to characterize the behavior of the model under a wide range of conditions. We therefore reduced the number of parameters by rescaling the model (APPENDIX B). By doing so, we were able to identify dimensionless combinations of parameter that govern the model's behavior.

In FIGURE 3A, we systematically varied two of these dimensionless parameter combinations. Along the vertical axis, we vary c, the relative cost of choice; along the horizontal axis, we vary V, the typical rate of evolution of the male characters relative to that of the female preference.

These dimensionless parameters are defined as follows:

$$c = \frac{4\gamma\,\beta_A}{\mu\alpha_A^2}, \quad V = \frac{\mathrm{var}(t_A)}{\mathrm{var}(p)}\frac{4\beta_A^2}{\alpha_A^2}. \qquad [16]$$

The relative cost of choice c, measures the intensity of viability selection on female preference, which is quantified by 2γ, relative to that of sexual selection, which is quantified by $\mu\alpha_A^2/(2\beta_A)$. The coefficients $\mathrm{var}(t_A)$ and $\mathrm{var}(p)$ that appear in the definition of V are the mutational variances of the male character t_A and the female preference, respectively. When $\mathrm{var}(t_A) \gg \mathrm{var}(p)$, such that V is large, comparable intensities of selection on t_A and p lead to a much larger phenotypic change in the male character per generation and, hence, to a larger rate of evolution of this character. Contrastingly, when V is small, mutation is more likely to create variation in female preference, such that the evolutionary rate of change of p is larger than that of t_A, provided of course, that both characters experience selection of comparable intensity.

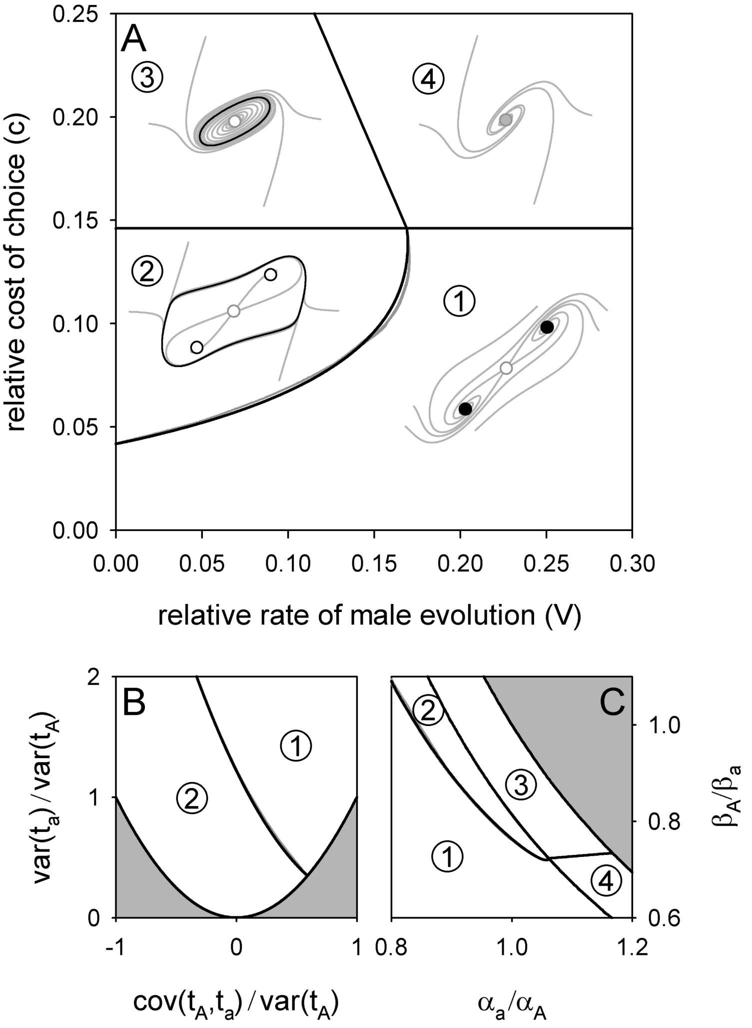


FIGURE 3 – MODEL BEHAVIOR UNDER DIFFERENT PARAMETER CONDITIONS

The dynamics of the model can be of four types (see the main text), which each occur within a specific region (1 to 4) of parameter space. In panel A to C, we vary different combinations of parameters. Black lines demarcate points where the number or stability of equilibria changes through local bifurcations; gray lines (often very close to local bifurcation lines) demarcate points where limit cycles appear or disappear through global bifurcations. Gray areas extend over biologically unrealistic parameter combinations. The dynamics within each region is characterized by a representative phase portrait (as in FIGURE 2). In these phase portraits, black lines indicate stable limit cycles and circles mark equilibrium points (gray is used for the trivial equilibrium; black for the non-trivial equilibrium; filled circles indicate stable equilibria; open symbols indicate unstable equilibria). Except when a parameter combination was varied, we used the values $\alpha_a/\alpha_A = \beta_A/\beta_a = 0.9$, $v_a/v_A = 0.7$, $c = 0.1$, $V = 0.15$, $\mu = 0.01$, $\mathrm{var}(t_a)/\mathrm{var}(t_A) = 1.0$, $\mathrm{cov}(t_A,t_a)/\mathrm{var}(t_A) = 0.1$.

Roughly speaking, the dynamics of the model can be of four qualitatively different types. As illustrated in FIGURE 3A, all of these types may occur when V and c are varied. The figure is subdivided into four large regions (labeled as region 1 to 4), and the dynamics of the model within a region is represented by the schematic phase portraits (cf. FIGURE 2) shown within that region. The regions are separated by boundary lines, which correspond to combinations of V and c where a qualitative change occurs in the stability or the number of equilibria (local bifurcation lines; black), or where limit cycles appear or disappear through global bifurcations (gray lines).

In region 1 of FIGURE 3A, where V is large and c is small, the dynamics is as in FIGURE 1A; that is, evolution converges on a stable nontrivial equilibrium where females are choosy and where males develop a costly ornament. The equilibrium is destabilized when the relative rate of male evolution V is decreased. We then enter region 2, where the female preference and the male investment strategy evolve continuously along a limit cycle, as in FIGURE 1B and FIGURE 2. The mechanism responsible for the appearance of oscillations is a classical one: by decreasing the relative rate of male evolution, we lower the rate at which males respond to changes in the female preference. This effectively introduces a delay in the feedback between the evolution of female preference and male condition-dependent investment (it is well known from engineering that systems with delayed feedbacks are prone to exhibit oscillatory behavior).

Changes in the relative cost of choice predominantly influence the number of equilibria. FIGURE 3A illustrates that the nontrivial equilibria (i.e., the equilibria at which females exert costly mate preference) disappear as soon as the relative cost of choice becomes larger than a certain threshold value (cf. equation [14]). Hence, we find convergence to the trivial equilibrium (i.e., the equilibrium where females mate at random and where males do not express a costly ornament) in region 4, and oscillations around the trivial equilibrium in region 3. The model's behavior in region 3 illustrates again that predictions based on equilibrium considerations can be misleading: although the cost of choice is high relative to the intrinsic information content of the ornament, and although one would therefore not expect females to evolve mating preferences, it is obvious that the cost of choice is not minimized in region 3. Due to the continual change of mating preferences, the realized information content of the ornament is above its intrinsic information content for most of the time. As a consequence, the average benefit of choice is larger than one would expect from equilibrium considerations. This also explains why female choosiness can evolve when the relative cost of choice are higher than the threshold level determined by equation [14].

The same four types of dynamical behavior are found when other parameters are varied (the labeling of the regions in FIGURES 3B & C corresponds to the labeling used in FIGURE 3A). In FIGURE 3B, for example, we vary the relative genetic covariance between the male characters (on the horizontal axis) and their mutational variances relative to one another (on the vertical axis). The gray background extends over biologically unrealistic parameter combinations (APPENDIX B). From FIGURE 3B, we conclude that continual evolution of the female preference and the male investment pattern is likely when the male characters evolve independently ($\mathrm{cov}(t_A, t_a) = 0$) or when mutations typically affect the male characters in opposite phenotypic directions ($\mathrm{cov}(t_A, t_a) < 0$). This agrees with the results shown in FIGURE 1, where a decrease of the mutational covariance between male characters (the parameter f) gave rise to oscillatory dynamics. Likewise, continual evolution is more likely when the mutational variance of the character t_a is low, such that this character can evolve only slowly. Again, this will destabilize the evolutionary feedback through introducing a longer delay in the evolutionary response by low-quality males.

FIGURE 3C, finally, features the same configuration of regions and bifurcation lines as FIGURE 3A. In FIGURE 3C, we vary the intrinsic information content of the ornament. Along the horizontal axis, we vary the relative coefficients of attractiveness ($\alpha_a/\alpha_A < 1$ for a revealing handicap). Along the vertical axis, we vary the relative coefficients for the cost of ornamentation ($\beta_A/\beta_a < 1$ for a condition-dependent handicap). The gray region extends over irrelevant parameter combinations. At the border of the gray region, the ornament is a pure epistatic indicator that does not intrinsically provide information about quality. The intrinsic information content of the ornament increases as one moves downward or towards the left in the plot. As we emphasized before, it is the potential discrepancy between the realized and intrinsic information content of the ornament that is responsible for the observed evolutionary cycling of the female preference and the male investment strategy. This discrepancy can only be large when the intrinsic information content of the ornament is low. Otherwise, deviations from the optimal male investment pattern would be too costly to persist over a long time. This agrees with FIGURE 3C, where continual evolution of female preferences (regions 2 and 3) is restricted to the area in parameter space where the intrinsic information content of the ornament is low. In the remaining areas, equilibrium analysis correctly predicts the outcome of evolution. In addition, we can draw the conclusion that both revealing- and condition-dependent models may exhibit oscillatory behavior.

MULTIPLE VIABILITY COMPONENTS, MULTIPLE ORNAMENTS

In this section, we investigate two extensions of our model, in order to illustrate some of the potential consequences of sexual conflict over the information content of signals in more complex settings.

MULTIPLE VIABILITY COMPONENTS

Our first extension concerns our assumption that genetic quality can be represented as a one-dimensional quantity. Although this is a standard assumption, made in many models of sexual selection on good genes (for exceptions, see Johnstone, 1996; CHAPTER 8 of this thesis), it is usually the case that genetic quality has several independent aspects (e.g. Doucet & Montgomerie, 2003).

To explore the consequences of this complication, we extend our model by introducing a second set of quality genes that influence genetic quality. To be precise, we assume that genetic quality is determined by two distinct quality components (instead of one), denoted A and B. These quality components represent, for example, tolerance to harmful substances in the environment and resistance against parasites. Each of the quality components is determined by many loci with two alleles (**A** and **a** for the first quality component; **B** and **b** for the second). As in the original model, we assume that deleterious mutations maintain variation in both quality components, but that they are sufficiently rare, such that we may neglect the frequency of individuals that carry more than one deleterious mutation. Under these assumptions, we must keep track of the frequencies of three types of individuals: high-quality individuals that carry no deleterious mutations (*AB* individuals), low-quality individuals that carry an **a** allele (*aB* individuals) and low-quality individuals that carry a **b** allele (*Ab* individuals). Males can vary their investment into the ornament depending on their quality for each of the quality components. The levels of investment by the three types of males are denoted t_{AB}, t_{aB} and t_{Ab}.

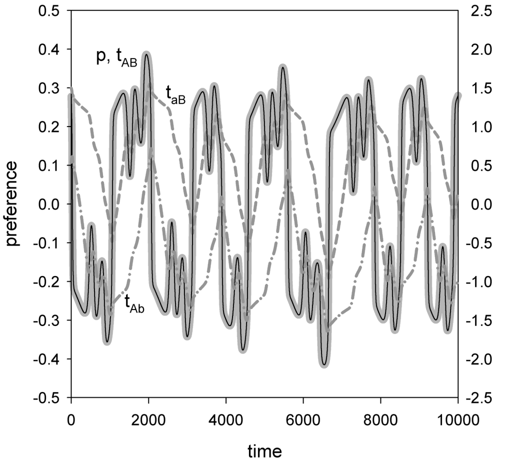


FIGURE 4 – COMPLEX DYNAMICS OF AN INDICATOR FOR TWO DISTINCT ASPECTS OF QUALITY

The advantage of evolving a preference varies with the investment into the ornament by low-quality males (dashed line: males with low quality for the first component; dot-dashed line: males with low-quality for the second component). For each quality component separately, the benefit of choice decreases when the realized information content of the ornament drops. For the ornament as a whole, the information content varies in a complex manner, due to the fact that it serves as an indicator for two quality components at the same time. Much like the chaotic movement of a double pendulum, this results in an unpredictable dynamics of the female preference. Parameters are $\mu_A = \mu_B = 0.025$, $\alpha_{AB} = \alpha_{aB} = \alpha_{Ab} = 5.0$, $\beta_{AB} = \beta_{aB} = \beta_{Ab} = 0.5$, $\gamma = 0.05$, $v_{AB} = 1.0$, $v_{aB} = v_{Ab} = 0.5$. Mutations in a male investment character had no pleiotropic effects on other characters.

A derivation of the invasion fitness and the selection gradients for this extended model follows a procedure similar to the one given in Appendix A for our original model (details are available from the authors). The main difference between the extended and the original model is that the selection gradient with respect to the female preference now contains two separate contributions to the benefit of choice, one for each quality component. To be exact (cf. equation [8]),

$$\lambda_p = \frac{1}{2}\frac{\partial \ln\left(1-c^f(p)\right)}{\partial p} + \frac{1}{2}\mu_A w_A \frac{\partial \ln\left(R_A(p,t_{AB},t_{aB})\right)}{\partial p} + \frac{1}{2}\mu_B w_B \frac{\partial \ln\left(R_B(p,t_{AB},t_{Ab})\right)}{\partial p}, \quad [17]$$

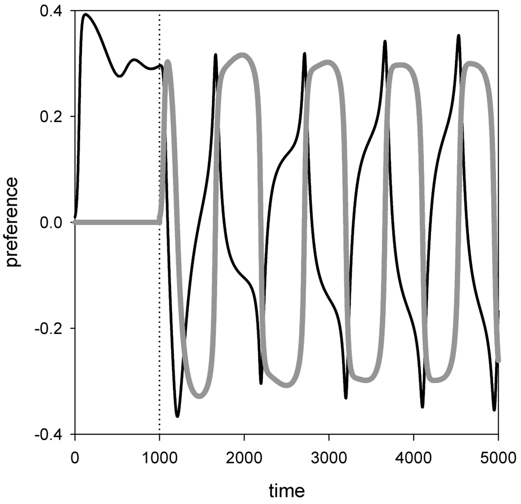
where μ_K denotes the rate of deleterious mutations in quality component K (K=A or B), where w_K is the reproductive value of a males with a low quality for component K, and where R_K denotes the attractiveness of a high-quality male relative to that of a male with a low quality for component K.

In a model with multiple quality components, it is possible that the ornament is a signal with a high intrinsic information content for one component of quality, and a low intrinsic information content for another. In such a situation, there is a potentially large discrepancy between the intrinsic and the realized information content of the ornament with respect to one of the quality components. As in the original model, this may lead to continual evolution of female mating preferences along a limit cycle.

Even more complex behavior emerges when the ornament has a low intrinsic information content for both quality components. The realized information content of the ornament can then differ from its intrinsic information content in both aspects of quality. Much like the motion of a double pendulum, this may result in a fundamentally unpredictable dynamics of the female preference and the male investment strategy. An example of this is given in FIGURE 4, where we show the evolution of female preference for a pure epistatic indicator and the male investment strategy in a model where male quality is determined by two independent quality components.

MULTIPLE ORNAMENTS

The second extension of our model deals with the evolution of multiple preferences and ornaments. Male courtship displays often comprise multiple sexual signals, involving several behavioral attributes and/or morphological ornaments. The reasons for this complexity are poorly understood. Theoretical models indicate that the evolution of multiple preferences is not straightforward. At first sight, females are expected to evolve preference for the single indicator of quality with the highest reliability, honesty and detectability (Schluter & Price, 1993; Iwasa & Pomiankowski, 1994), and they should disregard ornaments that are less reliable, honest or detectable. This is especially true when there are epistatic interactions between the costs of the different female preferences, such that the overall cost of choice is greatly increased when females assess an additional male trait (Iwasa & Pomiankowski, 1994). In CHAPTER 8 of this thesis, we investigated one solution to this problem and demonstrated that preferences for multiple ornaments can evolve, even when exhibiting multiple preferences is costly, as long as the ornaments provide information about different aspects of genetic quality, or when they provide sufficiently independent estimates of overall genetic quality. In the present paper, we provide a second solution to the problem: the non-equilibrium dynamics of preferences and male investment may lead to the establishment of multiple preferences even in situations where this is impossible in evolutionary equilibrium.



The figure shows the evolution of two female preferences in a species with two male ornaments. In the first 1000 time units, female preference for the first ornament (black line) reaches a stable equilibrium level (exactly as in FIGURE 1, upper panel). After 1000 time units we introduce a small level of preference for a second ornament. Although the second ornament has a lower intrinsic information content, female preference for the second ornament (gray line) can increase. Both female preferences remain in the population. Again, the system eventually attains a stable limit cycle. Parameters are as in FIGURE 1, upper panel. The additional parameters for the second ornament (see APPENDIX B) are given by $\alpha'_A = 7.0$, $\alpha'_a = 6.0$, $\beta'_A = \beta'_a = 0.5$, $\gamma' = 0.08$.

FIGURE 5 – PREFERENCES FOR TWO TRULY REDUNDANT SIGNALS

An example of such a situation is given in FIGURE 5. The figure shows the evolution of two female preferences for two male ornaments that both act as signals of genetic quality (see APPENDIX B for details). As in our original model, genetic quality was assumed to be a one-dimensional quantity. Hence the ornaments did not act as signals for distinct quality components, nor did they provide independent estimates of genetic quality. What is more, the costs of choice were taken to combine in a super-multiplicative manner, such that the use of a second preference greatly increased the cost of mate choice. Based on existing theory (Schluter & Price, 1993; Iwasa & Pomiankowski, 1994; CHAPTER 8 of this thesis), we would therefore expect the evolution of at most a single female preference. Yet, FIGURE 5 clearly demonstrates that this expectation is wrong.

In the simulation shown in FIGURE 5, the parameters of the first ornament are identical to those of the ornament in FIGURE 1. For the first 1000 time units, the simulation therefore behaves exactly as the simulation shown in the upper panel of FIGURE 1: female preference for the first ornament (black line) and male investment into that ornament (not shown) approach a stable equilibrium level. After 1000 time units, we slightly perturbed the system by introducing a small preference for the second ornament (gray line). The second ornament is an ornament with a low intrinsic information content. Based on existing theory, we would therefore not expect female preference for the second ornament to increase. Yet, females evolve preferences for the second ornament even though it is an inferior indicator of quality and even though using multiple preferences is costly.

The reason for the apparent contradiction between existing theory and the simulation in FIGURE 5 is that the former assumes that evolution will lead to a stable equilibrium. In this paper, we have demonstrated that this need not be the case. FIGURE 5 illustrates that the potential occurrence of non-equilibrium dynamics is not merely interesting from a mathematical point of view; it also undermines the predictions of existing theory. In the context of multiple ornaments, it is clear that non-equilibrium dynamics allows for the dynamical maintenance of more ornaments and female preferences than would be expected from equilibrium considerations.

MULTIPLE ORNAMENTS AND MULTIPLE VIABILITY COMPONENTS

The latter can be illustrated even more convincingly when the two extended versions of our original model are combined and generalized. This yields a model where genetic quality is determined by several distinct quality components, and where female mate choice is based on an arbitrary number of ornaments and preferences.

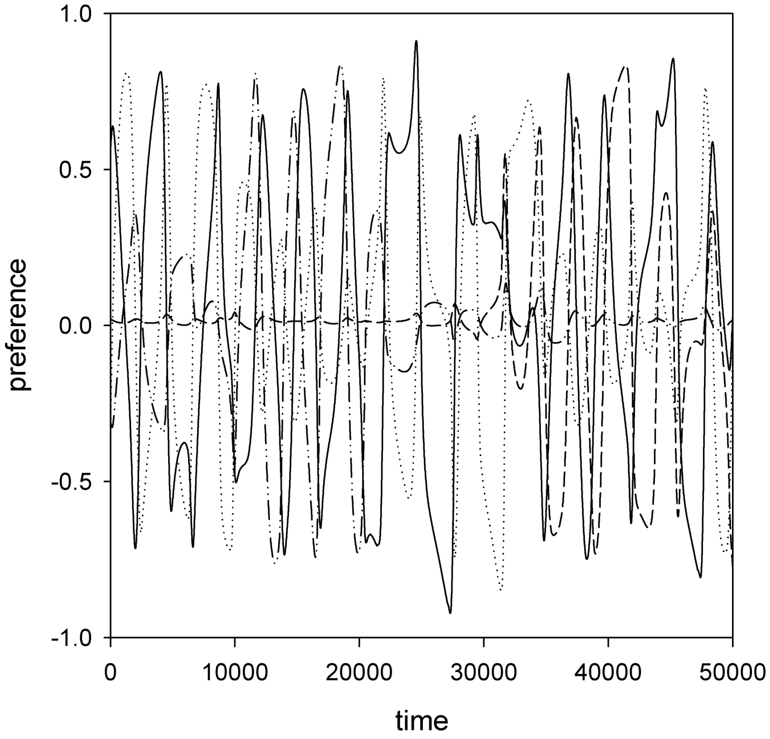


FIGURE 6 – PREFERENCES FOR MANY ORNAMENTS

An example of the evolution of preferences for four ornaments that act as indicators for two quality components (A and B). For all ornaments, $\beta_{AB} = \beta_{aB} = \beta_{Ab} = 0.1$, $\alpha_{AB} = 1.0$, $\gamma = 0.01$. The joint cost of choice was high (see APPENDIX B). The figure shows a representative replicate of a large number of simulations where the parameters α_{aB} and α_{Ab} were randomly chosen for each ornament, in such a way that $\alpha_{aB} + \alpha_{Ab} = 1.5$, $\alpha_{aB} < 1.0$ and $\alpha_{Ab} < 1.0$. Other parameters were taken as $v_{AB} = 1.0$, $v_{aB} = v_{Ab} = 0.8$, $\mu_A = \mu_B = 0.02$.

This combined model allows for the maintenance of many ornaments and preferences even when the number of quality components is low. We observed the joint evolution of up to twenty ornaments and preferences in a model with just two quality components, a result that bears analogy to the dynamical coexistence of a large number of competing species on just a few resources (Huisman & Weissing, 1999). For the sake of clarity, we provide a less dramatic example of a simulation in FIGURE 6. In this simulation we observe the complex dynamics of four preferences for four different male ornaments that act as signals for two quality components. At any moment in time, females assess several, but not all of the ornaments, even though the cost of using multiple preferences is high. On a longer timescale, there can be switches in the set of preferences used by females.

To assess the likelihood of the maintenance of many preferences, we ran simulations with a large number ornaments. In each simulation, we chose random parameters for the ornaments, but ensured that the intrinsic information content of the ornaments was comparable for all ornaments. Non-equilibrium dynamics of multiple preferences was observed in many of these simulations, but was most easily attained in situations where the intrinsic information content of the ornaments was low.

DISCUSSION

Several seemingly general insights offered by theory on the evolution of mating preferences for good-gene indicators rely on the assumption that female preference and male ornament expression eventually converge on stable equilibrium levels. An example of such a general insight is the conclusion that no stable exaggeration of costly female preference is possible when the ornament is a pure epistatic indicator (Iwasa *et al.*, 1991). Another example is the conclusion that females should evolve preferences for the single indicator of quality with the highest reliability, honesty and detectability and should disregard ornaments that are inferior in these aspects (Schluter & Price, 1993); at least, unless using an additional cue in mate choice only weakly increases the cost of choice (Iwasa & Pomiankowski, 1994) or unless the ornaments provide independent information about distinct aspects of quality (CHAPTER 8 of this thesis).

Our results provide proof of principle that the joint evolution of female preference and male condition-dependent signaling need not attain stable equilibrium levels. In fact, the interplay between sexual selection for good genes and sexual conflict over the information content of an ornament can lead to continual evolution of the female preferences and the male signaling strategy. We demonstrated that existing theory cannot be trusted when such non-equilibrium dynamics occurs. This is made clear in FIGURE 4, which, contrary to the naïve expectation, shows the long-term persistence of a costly female preference for an ornament that is a pure epistatic indicator in evolutionary equilibrium. Similarly, FIGURE 5 shows the joint evolution of preferences for two ornaments, in a situation where existing theory predicts that females should use only a single preference. In addition to illustrating the inherent limitations of a theory based on the assumption of equilibrium dynamics, these examples demonstrate that the interpretation of empirical data may change when the potential occurrence of non-equilibrium dynamics is taken into account.

The oscillations of mate preference and male ornament expression observed in our model superficially resemble those observed in Fisherian models of sexual selection (Iwasa & Pomiankowski, 1995). Although the mechanism underlying the oscillations is quite different in the two types of models, both the Fisherian and the good genes model illustrate the importance of genetic constraints on the evolutionary dynamics of secondary sexual characters. More generally, both these models show that a purely phenotypic approach to the modeling of sexual selection, without the appreciation of genetic details, is inappropriate.

Apart from its theoretical consequences, the continual evolution observed in our model is interesting in its own right. As illustrated by FIGURE 6, the evolution of multiple ornaments and preferences can lead to expression patterns of sexual characters that are highly variable and dynamic on the phylogenetic time scale. Therefore, the dynamics observed in our model may offer a mechanistic explanation for the apparent frequent loss and turnover of sexually selected traits within species (Wiens, 2001). In addition, the model provides an alternative mechanism for divergence of sexually selected traits between allopatric populations (Schluter & Price, 1993). This may be of relevance in the context of speciation (see the AFTERTHOUGHTS to this chapter). Third, the model captures some aspects of the ideas originally developed by Hill (1994), who suggests that sexual conflict in the evolution of quality indicators is responsible for an evolutionary arms race, in which females are continuously forced to direct their attention to additional and novel ornament features. And finally, as already indicated, our results provide an alternative explanation for the evolution of multiple mate preferences (see Candolin, 2004, for a review of current explanations).

WHEN SHOULD WE EXPECT NON-EQUILIBRIUM DYNAMICS OF PREFERENCES AND INDICATOR TRAITS?

The question whether or not the continual evolution of preferences and good-gene indicators is a biologically relevant evolutionary phenomenon ultimately remains an empirical one. Based on our analysis, we can however give a number of conditions under which sexual conflict over the information content of ornaments is most likely to affect the evolutionary dynamics.

First, males should be able to change their investment into the ornament according to their genetic quality. Instabilities leading to continual evolution are more likely when the investment strategies of high- and low-quality males can evolve independently, or when the genetic architecture of the ornament tends to enlarge the differences between high- and low-quality males (due to negative pleiotropic interactions between male investment genes, for example; FIGURE 3B). Second, discrepancies between the realized and intrinsic information content of the ornament should not be too costly. This requires that the ornament is a poor indicator of quality and that the intrinsic information content of the ornament is low (FIGURE 3C). In this case, females cannot easily detect low-quality males. Also, males that invest less than the optimal value do not suffer disproportionately from reduced attractiveness. Consequently, there can be a discrepancy between the realized and the intrinsic content of the ornament, without this resulting in strong selection on males to realize their optimal level of investment. Third, discrepancies between the realized and intrinsic information content of the ornament should persist for sufficient lengths of time, such that female preference can reach a high value before males attain their optimal investment strategy. This requires that the rate of male evolution is low relative to the rate of female evolution. Whether or not this will actually be the case depends on genetic details (specifically, on the mutational variances of the traits) and on the intensity of selection on the male and female sexual characters.

DIRECTIONS FOR FUTURE RESEARCH

The results of the present paper identify potentially important limitations of the existing theory of sexual selection on indicator traits. We expect that these limitations are relevant for the interpretation of empirical data concerning the evolution of multiple preferences and ornaments, the genetic basis of male indicator traits and the genetic and physiological constraints responsible for condition-dependent signaling, to give a few examples.

Directly demonstrating non-equilibrium dynamics of secondary sexual characters, as observed in our model, in a natural population will be difficult, however. This is because the probability of actually observing a qualitative change in the female preference and male ornamentation is quite low: such changes are expected to occur rapidly whereas the evolution in periods between qualitative changes proceeds only slowly, due to the fact that selection on male condition-dependent signaling is weak (FIGURE 1 & 2). To make matters worse, even if we were able to measure levels of female preference and male investment for prolonged periods of time, it need not be the case that the fluctuations in female preference and male signaling can easily be detected. There are disturbing examples of systems in which hardly observable non-equilibrium dynamics already causes substantial deviations from the predictions based on equilibrium considerations (e.g., Huisman & Weissing, 1999). A direct demonstration of the consequences of sexual conflict over the information content of ornaments is perhaps most feasible in a laboratory setting (cf. Rice, 1996). Complementary indirect evidence might be obtained from phylogenetic studies and from comparisons between allopatric populations of the same species.

Further theoretical work is needed to investigate the consequences of sexual conflict under less idealized genetic assumptions. It will be particularly interesting to study the phenomena described in this paper in genetically explicit individual-based simulation models, or in a quantitative genetics framework. This would offer a check of our hybrid modeling approach, and it would provide some insight into the limitations associated with the different methods available to study phenotypic evolution.

APPENDIX A — Invasion fitness and selection gradients

Our aim is to investigate a mutant's potential to invade in a resident population. We assume that almost all individuals carry 'resident' alleles coding for the phenotype $\left(\hat{p},\hat{t}_A,\hat{t}_a\right)$ and that a tiny fraction of the individuals carries a mutant allele at one of its preference or ornament expression loci. Since mutations occur only rarely, the mutant phenotype $\left(p,t_A,t_a\right)$ will typically differ from the resident phenotype in only one of its components.

Under our simplifying assumptions with respect to the genetic basis of the quality, we need to keep track of two quality phenotype frequencies only. For the resident, these will be denoted z_A, the frequency of individuals that carry no deleterious mutations, and z_a, the frequency of individuals that carry a single deleterious mutation. For the mutant, these quantities are denoted ε_A and ε_a. The mutant is rare initially, such that

$$\varepsilon_A+\varepsilon_a \quad \ll \quad z_A+z_a \quad \approx \quad 1. \qquad \text{[A-1]}$$

The quality phenotype frequencies change due to viability selection, non-random mating and mutation. Below, we discuss these three processes in more detail.

Viability selection

Viability selection acts differently on males and females, since the expression of preferences and ornaments is sex-limited. Therefore, we must calculate the quality phenotype frequencies after viability selection separately for males and females. For resident and mutant males, we apply standard replicator equations (Crow & Kimura, 1970), yielding

$$\begin{aligned} z'_{m,k} &= z_k \frac{h_k^m\left(\hat{t}_k\right)}{\bar{h}_m\left(\hat{t}_A,\hat{t}_a\right)}, \\ \varepsilon'_{m,k} &= \varepsilon_k \frac{h_k^m\left(t_k\right)}{\bar{h}_m\left(\hat{t}_A,\hat{t}_a\right)}, \end{aligned} \qquad \text{[A-2]}$$

where $z'_{m,k}$ and $\varepsilon'_{m,k}$ denote the quality phenotype frequencies in resident and mutant males after viability selection. The function $h_m^k\left(t_k\right)$, which represents the viability of a male with quality phenotype k, is defined in equation [1] in the main text.

Since the density of mutant males can be neglected with respect to the density of resident males, the average male fitness, $\overline{h}_m\left(\hat{t}_A,\hat{t}_a\right)$, is independent of the mutant male phenotype. It is given by

$$\overline{h}_m\left(\hat{t}_A,\hat{t}_a\right)=\sum_{k=A,a} z_k\, v_k\left(1-c_k^m\left(\hat{t}_k\right)\right). \tag{A-3}$$

The quality phenotype frequencies in resident and mutant females, $z'_{f,k}$ and $\varepsilon'_{f,k}$, can be given in a somewhat simpler form as

$$z'_{f,k}=\frac{v_k}{\overline{v}}z_k \quad , \quad \varepsilon'_{f,k}=\frac{v_k}{\overline{v}}\frac{1-c^f(p)}{1-c^f(\hat{p})}\varepsilon_k . \tag{A-4}$$

where

$$\overline{v}=\sum_{k=A,a} z_k\, v_k \ , \tag{A-5}$$

represents the average general viability.

NON-RANDOM MATING

Let $M_k\left(p,t_k,\hat{t}_A,\hat{t}_a\right)$ denote the (per-capita) probability that a female with preference p will mate with a male with quality phenotype k that invests t_k units of resource into his ornament. Note that this probability also depends on the resident male strategies $\hat{t}_A$ and $\hat{t}_a$, since our focal male must compete for mating opportunities with other males. We assume that females mate only once and that mating probabilities are proportional to male attractiveness. Then,

$$M_k\left(p,t_k,\hat{t}_A,\hat{t}_a\right)=\frac{r_k(p,t_k)}{\overline{r}\left(p,\hat{t}_A,\hat{t}_a\right)} , \tag{A-6}$$

where, in line with [5], $r_k\left(p,t_k\right)$ measures a male's attractiveness to a particular type of female. The coefficient $\overline{r}\left(p,\hat{t}_A,\hat{t}_a\right)$ represents the average attractiveness of the males encountered by the female, and is defined as

$$\overline{r}\left(p,\hat{t}_A,\hat{t}_a\right)=\sum_{k=A,a} z'_{m,k}\, r_k\left(p,\hat{t}_k\right). \tag{A-7}$$

To find the frequency of the quality phenotype k after mating, we first compute the frequency of mating events between a female with quality phenotype k' and a male with quality phenotype k''. Next, we sum over all possible combinations (k',k'') of male and female quality phenotypes (when deleterious mutations are rare, we may neglect mating between two low-quality individuals) and weigh them according to the probability $Q^k_{k'\times k''}$ that the phenotype k is generated through Mendelian segregation from the parental phenotypes k' and k''. The weighing factors $Q^k_{k'\times k''}$ are given by

$$Q^k_{k'\times k''}=\frac{\delta(k,k')+\delta(k,k'')}{2} , \tag{A-8}$$

where $\delta(A,A)=1$ and $\delta(A,a)=\delta(a,A)=0$.

For the resident, this procedure yields the following quality phenotype frequencies after mating (denoted z''_k)

$$z''_k = \sum_{(k',k'')=(A,A),(A,a),(a,A)} z'_{f,k'}\, z'_{m,k''}\, Q^k_{k'\times k''}\, M_{k''}\left(\hat{p},\hat{t}_{k''},\hat{t}_A,\hat{t}_a\right). \qquad \text{[A-9]}$$

In calculating the quality phenotype frequencies after mating for the mutant (denoted ε''_k), we have to distinguish two cases. When a mutant individual mates with a resident individual, the mutant can be either the female or the male in the mating pair. Therefore,

$$\begin{aligned}\varepsilon''_k = &\frac{1}{2}\sum_{(k',k'')=(A,A),(A,a),(a,A)} z'_{f,k'}\, \varepsilon'_{m,k''}\, Q^k_{k'\times k''}\, M_{k''}\left(\hat{p},t_{k''},\hat{t}_A,\hat{t}_a\right)+\\ &\frac{1}{2}\sum_{(k',k'')=(A,A),(A,a),(a,A)} \varepsilon'_{f,k'}\, z'_{m,k''}\, Q^k_{k'\times k''}\, M_{k''}\left(p,\hat{t}_{k''},\hat{t}_A,\hat{t}_a\right).\end{aligned} \qquad \text{[A-10]}$$

The factor $1/2$ in front of both terms reflects the fact that only half of the offspring will inherit the mutant allele from its mutant parent.

In what follows, it will be convenient to represent equations [A-9] and [A-10] in terms of matrix equations. Let us therefore define vectors $\vec{z}$, $\vec{z}'$, $\vec{\varepsilon}$ and so on, which contain as elements the corresponding quality phenotype frequencies. For example,

$$\vec{z} = \begin{pmatrix} z_A \\ z_a \end{pmatrix}, \quad \vec{\varepsilon} = \begin{pmatrix} \varepsilon_A \\ \varepsilon_a \end{pmatrix}. \qquad \text{[A-11]}$$

We may then write

$$\begin{aligned}\vec{\varepsilon}'' &= \mathbf{T}(\vec{z})\,\vec{\varepsilon}\,,\\ \vec{z}'' &= \hat{\mathbf{T}}(\vec{z})\,\vec{z}\,,\end{aligned} \qquad \text{[A-12]}$$

where the elements of the transition matrices $\mathbf{T}(\vec{z})$ and $\hat{\mathbf{T}}(\vec{z})$ can be found by combining equations [A-2] to [A-10]. The matrix element $\mathbf{T}_{kk'}(\vec{z})$ represents the frequency at which a mutant individual with quality phenotype k' transmits its mutant genes to offspring with phenotype k (not yet taking into account the occurrence of deleterious mutations). It is given by

$$\begin{aligned}\mathbf{T}_{kk'}(\vec{z}) = &\frac{1}{2}\,\frac{h^m_{k'}(t_{k'})}{\bar{h}_m\left(\hat{t}_A,\hat{t}_a\right)}\,\frac{r_{k'}(\hat{p},t_{k'})}{\bar{r}\left(\hat{p},\hat{t}_A,\hat{t}_a\right)} \sum_{\substack{k''=A,a\\(k',k'')\neq(a,a)}} Q^k_{k'\times k''}\,\frac{v_{k''}z_{k''}}{\bar{v}}+\\ &\frac{1}{2}\,\frac{v_{k'}}{\bar{v}}\,\frac{1-c^f(p)}{1-c^f(\hat{p})} \sum_{\substack{k''=A,a\\(k',k'')\neq(a,a)}} Q^k_{k'\times k''}\,\frac{h^m_{k''}\left(\hat{t}_{k''}\right)z_{k''}}{\bar{h}_m\left(\hat{t}_A,\hat{t}_a\right)}\,\frac{r_{k''}\left(p,\hat{t}_{k''}\right)}{\bar{r}\left(\hat{p},\hat{t}_A,\hat{t}_a\right)}.\end{aligned} \qquad \text{[A-13]}$$

When this expression is evaluated for a mutant that is phenotypically equivalent to the resident, we obtain the elements of the resident transition matrix. In other words,

$$\hat{\mathbf{T}}(\vec{z}) = \mathbf{T}(\vec{z})\big|^{\hat{}}\,, \qquad \text{[A-14]}$$

where the notation $x\Big|^{\wedge}$ is introduced to denote that the expression x should be evaluated for a mutant that is phenotypically equivalent to the resident. In other words,

$$x\Big|^{\wedge} = x\big|_{p=\hat{p},\, t_k=\hat{t}_k} \,. \qquad \text{[A-15]}$$

DELETERIOUS MUTATIONS

Deleterious mutations at loci coding genetic quality occur at rate μ per generation per genome. The quality phenotype frequencies in the next generation can easily be computed from the frequencies after mating. In matrix notation,

$$\begin{aligned} \vec{z}_{n+1} &= \mathbf{M}\,\vec{z}_n'' \,, \\ \vec{\varepsilon}_{n+1} &= \mathbf{M}\,\vec{\varepsilon}_n'' \,, \end{aligned} \qquad \text{[A-16]}$$

where, the index n represents the generation number. Up to first order in μ,

$$\mathbf{M} = \begin{pmatrix} 1-\mu & 0 \\ \mu & 1 \end{pmatrix}. \qquad \text{[A-17]}$$

Combining [A-12] and [A-16], we finally arrive at a dynamical model for the quality phenotype frequencies of mutant and resident,

$$\begin{aligned} \vec{z}_{n+1} &= \mathbf{M}\hat{\mathbf{T}}(\vec{z}_n)\vec{z}_n \,, \\ \vec{\varepsilon}_{n+1} &= \mathbf{M}\mathbf{T}(\vec{z}_n)\vec{\varepsilon}_n \,. \end{aligned} \qquad \text{[A-18]}$$

The fate of the mutant can be determined by iterating the recurrence equations [A-18]. If the mutant density increases, the mutant will be able to invade; if it decreases, the mutant will disappear after some time. Alternatively, we may attempt to derive, from equation [A-18], the mutant's invasion-fitness function. This function, denoted as λ, measures the mutant's geometrical growth rate when rare and, as such, directly predicts whether or not the mutant will be able to invade in the resident population. Moreover, using results of Dieckmann & Law (1996) the invasion-fitness function can be used to derive equations for the evolutionary dynamics of the female preference and the male condition-dependent investment strategy into the ornament.

RESIDENT EQUILIBRIUM

We study the invasion by a mutant of a resident population that has attained a stable distribution of quality phenotype frequencies. This stable frequency distribution, $\vec{z}^*$, can be found by solving the equation

$$\vec{z}^* = \mathbf{M}\hat{\mathbf{T}}\big(\vec{z}^*\big)\vec{z}^* \,, \qquad \text{[A-19]}$$

which yields, up to first order in μ,

$$\vec{z}^* = \begin{pmatrix} 1-\mu/\hat{s} \\ \mu/\hat{s} \end{pmatrix}, \qquad \text{[A-20]}$$

where

$$\hat{s} = 1 - \frac{1}{2}\frac{v_a}{v_A}\left(\frac{1}{\hat{R}} + 1\right). \qquad \text{[A-21]}$$

The coefficient $\hat{s}$ is an aggregate selection coefficient that combines the viability disadvantage and the mate-choice disadvantage ($1/\hat{R}$; in males only) of the low-quality phenotype *a*. Indeed, using the notion of this aggregate selection coefficient, equation [A-20] can be interpreted as the equilibrium frequency distribution under mutation-selection balance (Crow & Kimura, 1970).

The coefficient $\hat{R}$ in [A-21] represents the attractiveness of high-quality males relative to that of males carrying a deleterious mutation at a quality locus. In general,

$$R(p, t_A, t_a) = \frac{r_A(p, t_A)}{r_a(p, t_a)}, \qquad \text{[A-22]}$$

and, specifically for the resident, $\hat{R} = R(\hat{p}, \hat{t}_A, \hat{t}_a)$.

INVASION FITNESS FUNCTION

We may now proceed to compute the invasion-fitness function λ. The invasion fitness is defined as the geometric rate of increase of the mutant population density when the mutant is still rare. Since the invasion of the mutant in the resident population is described by the recurrence equation

$$\vec{\varepsilon}_{n+1} = \mathbf{MT}(\vec{z}^*)\,\vec{\varepsilon}_n, \qquad \text{[A-23]}$$

the invasion fitness can be found as the dominant eigenvalue of the matrix $\mathbf{MT}(\vec{z}^*)$.

Although it is possible to compute the invasion fitness for arbitrary combinations of mutant and resident phenotypes, we refrain from doing so, since the resulting expression is too complicated to give any insights. Instead, we focus on mutants that differ only slightly from the resident. For such mutants, the fitness difference between the mutant and the resident is proportional to the gradient of the invasion-fitness function, in the direction in which the mutant differs from the resident. For example, the fitness difference between the resident and a mutant that differs slightly in its female preference is related to the selection gradient λ_p, where,

$$\lambda_p = \left.\frac{\partial \lambda}{\partial p}\right|^{\wedge}. \qquad \text{[A-24]}$$

According to a standard result (e.g., Caswell, 1989; Taylor, 1996b), the latter can also be written as

$$\lambda_p = \left.\frac{\partial \lambda}{\partial p}\right|^{\wedge} = \frac{1}{\vec{w}\,\vec{u}}\quad \vec{w}\,\mathbf{M}\left.\frac{\partial \mathbf{T}(\vec{z}^*)}{\partial p}\right|^{\wedge}\vec{u}. \qquad \text{[A-25]}$$

Here, $\vec{w}$ and $\vec{u}$ denote, respectively, the dominant left and right eigenvectors of the matrix $\mathbf{M}\hat{\mathbf{T}}(\vec{z}^*)$. From equation [A-19], it follows that $\vec{u} = \vec{z}^*$. The left eigenvector $\vec{w}$ contains the reproductive values w_A and w_a of the different quality phenotypes. They can be solved from the equation $\vec{w}\,\mathbf{M}\hat{\mathbf{T}}(\vec{z}^*)=\vec{w}$, yielding, up to the lowest order in μ (the first order terms turn out to be irrelevant),

$$w_A=1\,,\quad w_a=\frac{1+\hat{R}}{\hat{R}\left(4\dfrac{v_A}{v_a}-1\right)-1}\,. \qquad \text{[A-26]}$$

After some simplification, we obtain, from equation [A-13] and [A-25], expressions for the selection gradients. For the male ornaments, we find, up to the first order in μ,

$$\begin{aligned}\lambda_{t_A}&=\frac{1}{2}\left.\frac{\partial\ln\left(r_A(\hat{p},t_A)\left(1-c_A^m(t_A)\right)\right)}{\partial t_A}\right|^{\wedge}\left(1-\hat{w}\,z_a^*\right),\\ \lambda_{t_a}&=\frac{1}{2}\left.\frac{\partial\ln\left(r_a(\hat{p},t_a)\left(1-c_a^m(t_a)\right)\right)}{\partial t_a}\right|^{\wedge}\hat{w}\,z_a^*\,,\end{aligned} \qquad \text{[A-27]}$$

which are represented in a simplified form in equation [7] in the main text (where hats were omitted, since this did not result in ambiguities). The coefficient $\hat{w}$ represents the reproductive value of males carrying a deleterious mutation at one of their quality loci. To be exact,

$$\hat{w}=\frac{2}{\hat{R}\left(4\dfrac{v_A}{v_a}-1\right)-1}\,. \qquad \text{[A-28]}$$

Selection on female preferences is only weak, at least as long as we assume that the costs of female choice are only small (i.e., of the same order of magnitude as the mutation rate μ, as in other good-genes models). In this case, we find, up to first order in μ,

$$\lambda_p=\frac{1}{2}\left.\frac{\partial\ln\left(1-c^f(p)\right)}{\partial p}\right|^{\wedge}+\frac{1}{2}\mu\,\hat{w}\left.\frac{\partial\ln\left(R(p,\hat{t}_A,\hat{t}_a)\right)}{\partial p}\right|^{\wedge}, \qquad \text{[A-29]}$$

which corresponds to equation [8] in the main text.

APPENDIX B — METHODS FOR THE FIGURES

Where APPENDIX A deals with a general mate-choice model, we now turn our attention to the specific model (as defined by equation [2] & [4]-[6] in the main text).

NUMERICAL SIMULATIONS (METHODS FOR FIGURE 1 & 2)

We start from the dynamical system

$$\frac{d}{d\tau}\begin{pmatrix}\hat{p}\\ \hat{t}_A\\ \hat{t}_a\end{pmatrix}=\frac{N\,m}{2}\mathbf{G}\begin{pmatrix}\lambda_p\\ \lambda_{t_A}\\ \lambda_{t_a}\end{pmatrix}, \qquad \text{[B-1]}$$

where τ represents evolutionary time and where the proportionality constant $Nm/2$ scales the rate of evolution of the characters with the rate at which beneficial mutations arise (N is the population size and the constant m is the rate of mutations in p, t_A and t_a; Dieckmann & Law, 1996). The matrix $\mathbf{G}$ is the mutational variance-covariance matrix, given by

$$\mathbf{G} = \begin{pmatrix} \mathrm{var}(p) & 0 & 0 \\ 0 & \mathrm{var}(t_A) & \mathrm{cov}(t_A, t_a) \\ 0 & \mathrm{cov}(t_A, t_a) & \mathrm{var}(t_a) \end{pmatrix}. \qquad \text{[B-2]}$$

The parameters $\mathrm{var}(p)$, $\mathrm{var}(t_A)$ and $\mathrm{var}(t_a)$ are the mutational variances; $\sqrt{\mathrm{var}(p)}$, for example corresponds to the average phenotypic effect of a single mutation on female preference. The mutational covariance $\mathrm{cov}(t_A, t_a)$ measures the strength of pleiotropic interactions between the male characters t_A and t_a. We assume that there is no mutational covariance between the female preference and the male characters.

In the numerical simulations represented in FIGURE 1 & 2, we took $\mathrm{var}(p) = \mathrm{var}(t_A) = \mathrm{var}(t_a) = 1$, and $\mathrm{cov}(t_A, t_a) = f$, where f is interpreted as the fraction of male investment genes that is expressed irrespective of the male's genetic quality (this is merely one of many alternative interpretations of f). Male ornament expression is completely independent of genetic quality if $f = 1$. Conversely, the male characters t_A and t_a evolve completely independently if $f = 0$.

SIMPLIFICATION OF THE MODEL (METHODS FOR FIGURE 3)

To facilitate the characterization of the model's behavior for different parameter combinations (FIGURE 3), we simplify the model by rescaling. This procedure reduces the effective number of parameters, by identifying combinations of parameters that govern the model's behavior.

We start by introducing new variables $\tilde{p}$, $\tilde{t}_A$, $\tilde{t}_a$ and $\tilde{\tau}$, given by

$$\begin{gathered} \tilde{p} = \hat{p}\,\frac{\alpha_A}{2\sqrt{\beta_A}}, \quad \tilde{\tau} = \tau\,\frac{\alpha_A^2\,\mathrm{var}(p) N m \mu}{8\,\beta_A}, \\ \tilde{t}_A = \hat{t}_A \sqrt{\beta_A}, \quad \tilde{t}_a = \hat{t}_a\,\frac{\alpha_A\,\beta_a}{\alpha_a\,\sqrt{\beta_A}}. \end{gathered} \qquad \text{[B-3]}$$

We substitute these new variables into [B-1] and obtain, after simplification,

$$\frac{d}{d\tilde{\tau}} \begin{pmatrix} \tilde{p} \\ \tilde{t}_A \\ \tilde{t}_a \end{pmatrix} = \begin{pmatrix} 1 & 0 & 0 \\ 0 & V & V\eta\alpha \\ 0 & \dfrac{V\eta}{\alpha\beta} & \dfrac{V\phi}{\beta} \end{pmatrix} \begin{pmatrix} -c\tilde{p} + \left(\tilde{t}_A - \alpha^2\beta\tilde{t}_a\right)\tilde{w} \\ \left(\tilde{p} - \tilde{t}_A\right)\left(1/\mu - \tilde{w}\tilde{z}_a^*\right) \\ \left(\tilde{p} - \tilde{t}_a\right)\tilde{w}\tilde{z}_a^* \end{pmatrix}, \qquad \text{[B-4]}$$

where the two components on the right-hand side represent, from left to right, the rescaled mutational variance-covariance matrix, and the rescaled selection gradient vector. From the latter, we can see immediately that, in equilibrium, $\tilde{p} = \tilde{t}_A = \tilde{t}_a$.

The rescaled frequency, $\tilde{z}_a^*$ ($\kappa = A$ or B), and reproductive values, $\tilde{w}$, of low-quality males are given by

$$\tilde{z}_a^* = \frac{1}{1 - \frac{v}{2}\left(1 + \exp\left(\alpha^2 \beta\, \tilde{t}_a \left(2\tilde{p} - \tilde{t}_a\right) - \tilde{t}_A\left(2\tilde{p} - \tilde{t}_A\right)\right)\right)},$$
$$\tilde{w} = \frac{1}{\left(4/v - 1\right)\exp\left(\tilde{t}_A\left(2\tilde{p} - \tilde{t}_A\right) - \alpha^2\beta\, \tilde{t}_a\left(2\tilde{p} - \tilde{t}_a\right)\right) - 1}. \qquad \text{[B-5]}$$

The new parameters that appear in [B-4] and [B-5], are dimensionless combinations of the parameters of the original model. For example, the dimensionless parameter V is given by

$$V = \frac{\mathrm{var}(t_A)}{\mathrm{var}(p)} \frac{4\beta_A^2}{\alpha_A^2}, \qquad \text{[B-6]}$$

and it measures the rate of evolution of the male characters relative to that of the female preference. The dimensionless parameter c represents the intensity of viability selection on female preference (through the cost of choice) relative to that of sexual selection. It is given by

$$c = \frac{4\gamma\, \beta_A}{\alpha_A^2\, \mu}. \qquad \text{[B-7]}$$

Particularly interesting are the dimensionless parameters $\alpha = \alpha_a / \alpha_A$ and $\beta = \beta_A / \beta_a$, which are related to the intrinsic information content of the ornament. In fact, the intrinsic information content I is given by

$$I = \frac{\alpha_A^2}{2\beta_A}\left(1 - \alpha^2 \beta\right). \qquad \text{[B-8]}$$

When $\alpha^2\beta = 1$, low-quality males can express the ornament without extra costs and without being detected by females as low-quality males (in this case, the ornament is a pure epistatic indicator). By contrast, when $\alpha^2\beta = 0$, it is impossible for low-quality males to reach the same level of attractiveness as high-quality males without incurring large costs. In biologically relevant cases, the intrinsic information content of the ornament is positive, which implies that $\alpha^2 < 1/\beta$ (the gray region in FIGURE 3C extends over parameter combinations that do not satisfy this constraint).

The remaining dimensionless parameters are given by

$$\phi = \frac{\mathrm{var}(t_a)}{\mathrm{var}(t_A)}, \quad \eta = \frac{\mathrm{cov}(t_A, t_a)}{\mathrm{var}(t_A)} \quad \text{and} \quad v = \frac{v_a}{v_A}. \qquad \text{[B-9]}$$

In biologically relevant cases, the eigenvalues of the mutational variance-covariance matrix are positive. This imposes the constraint $\phi > \eta^2$ (the gray region in FIGURE 3B extends over parameter combinations where this inequality is violated).

MULTIPLE ORNAMENTS (METHODS FOR FIGURE 5 & 6)

In FIGURE 5, we modeled the evolution of two female preferences p and p' and two male ornaments that both act as signals of genetic quality. The amount of resources invested into these ornaments is determined by the strategic variables t_k and t'_k. As in our original model, we again assume that genetic quality can be represented as a one-dimensional quantity. We furthermore assumed that the ornaments have independent effects on mate choice. Female preference for one male ornament does not extend to the other. Analogously to our original model, the attractiveness of males was taken to be proportional to

$$r_k(p,p',t_k,t'_k)=\exp(\alpha_k\, p\, t_k+\alpha'_k\, p'\, t'_k)\,. \qquad \text{[B-10]}$$

Also the costs of ornamentation for males were assumed to interact multiplicatively, i.e.,

$$c_k^m(t_k,t'_k)=\exp(-\beta_k t_k^2-\beta'_k t_k'^2)\,. \qquad \text{[B-11]}$$

As a worst-case scenario, the costs of choice were taken to combine in a super-multiplicative manner, such that the use of a second preference greatly increases the cost of mate choice. In particular, we took

$$c^f(p,p')=\exp(-\gamma\,\theta(p')p^2-\gamma'\theta(p)p'^2)\,, \qquad \text{[B-12]}$$

with $\theta(p)=\exp(1/4p^2)$. Again, the derivation of the invasion fitness function follows a procedure similar to the one given above for our original model (details are available from the authors).

For the simulation shown in FIGURE 6, we generalized equations [B-10]-[B-12] in the straightforward way to an arbitrary number of preferences and ornaments (see also CHAPTER 8 of this thesis).

Afterthoughts on chapter 8 & 9

Multiple ornaments and speciation

Models of speciation through sexual selection have, up till now, mainly focused on divergent Fisherian runaway processes as the mechanism responsible for speciation (Turner & Burrows, 1995; Payne & Krakauer, 1997; Higashi *et al.*, 1999). Fisherian self-reinforcing sexual selection is interesting in the context of speciation since it may generate strong prezygotic reproductive isolation through the rapid evolution of female mating preferences for exaggerated male secondary sexual traits. What is more, female preferences may be directed towards arbitrary male traits, which need not function as signals for genetic quality, as in good genes models. In other words, depending on random initial biases, the Fisherian runaway process may unfold in any arbitrary direction. As a consequence, reproductive isolation will rapidly build up in allopatry, between geographically separated populations (Lande, 1981), or in sympatry, when divergent runaway processes occur simultaneously within a single population (Higashi *et al.*, 1999).

With their emphasis on Fisherian self-reinforcing sexual selection, the theoretical models deviate markedly from the majority of empirical studies in the field of sexual selection. The latter usually consider female mate choice to be adaptive in the sense that it acts upon male secondary sexual traits that function as indicators of genetic quality other than mere attractiveness (Jennions & Petrie, 2000). In such a case, female preference will not be directed towards arbitrary male traits, but only towards male traits that reliably signal high quality. One would therefore predict that the outcome of sexual selection for good genes is less arbitrary than the outcome of Fisherian self-reinforcing selection acting on its own. This makes it less evident that sexual selection will lead to divergence between allopatric populations, let alone that it will be able to promote divergent evolution under sympatric conditions.

In this 'afterthoughts' section, we investigate two alternative potential solutions to this problem. In the first part of this section, we extend the model of CHAPTER 8, to investigate the potential for the divergence of mating preferences for multiple habitat-specific ornaments. In the second part, we focus on the model of CHAPTER 9, and ask whether the continual coevolution of female mating preferences and male signaling strategies might perhaps facilitate speciation.

PREFERENCES FOR MULTIPLE HABITAT-SPECIFIC ORNAMENTS

Theoretical models for the evolution of mate preferences for multiple sexual ornaments indicate that females are more likely to evolve preferences for those ornaments that provide the most accurate, honest and reliable information about quality. Hence, isolated populations are expected to diverge in female preferences whenever the optimal choice strategy differs between the two populations (Schluter & Price, 1993). Such would be the case when, for example, the populations experience different environmental conditions which affect the accuracy, honesty and reliability of the information given by respective male sexual signals.

To test whether this idea might also work for populations that are not completely geographically isolated, we extended the model developed in CHAPTER 8 of this thesis, in order to model female mate choice in a population faced with a spatially heterogeneous environment. The population occurs in 40 patches, half of which belong to habitat 1, the other half belong to habitat 2. Each patch supports a fixed number of 40 individuals. Within each patch, females choose males based on their preference for two different ornaments, which provide information about two quality components, exactly as in CHAPTER 8 of this thesis. Deleterious mutations are only expressed in one of the two habitats: individuals with low quality for the first quality component have a lower viability in the first habitat, but not in the second. Similarly, individuals with low quality for the second quality component have a lower viability in the second habitat, but not in the first. We think of a situation where the two quality components confer immunity to two different pathogens that both occur in one of the two habitats but not in the other. The two male ornaments are revealing indicators of quality only in their respective selective habitats; that is, ornament 1 is a revealing indicator for the first quality component in habitat 1, but a pure epistatic indicator in habitat 2. Similarly, ornament 2 is a revealing indicator for the second quality component in habitat 2, but a pure epistatic indicator in habitat 1.

If individuals would only be faced with habitat 1, one would expect the population to evolve preference for the first ornament only (and analogously for habitat 2 and the second ornament). Indeed, we observe divergence of mating preferences according to this expectation if there is no migration between the subpopulations that live in the two different habitats (allopatric case, data not shown). At the other extreme (i.e., when the population is randomly redistributed over the patches every generation), we observe the evolution of preferences for both ornaments even when the joint cost of choice is high (sympatric case, data not shown), a result that is in accordance with the conclusions of CHAPTER 8 of this thesis. For a narrow range of intermediate migration rates, we observe an evolutionary dynamics that combines the features of the allopatric and sympatric case (FIGURE 1).

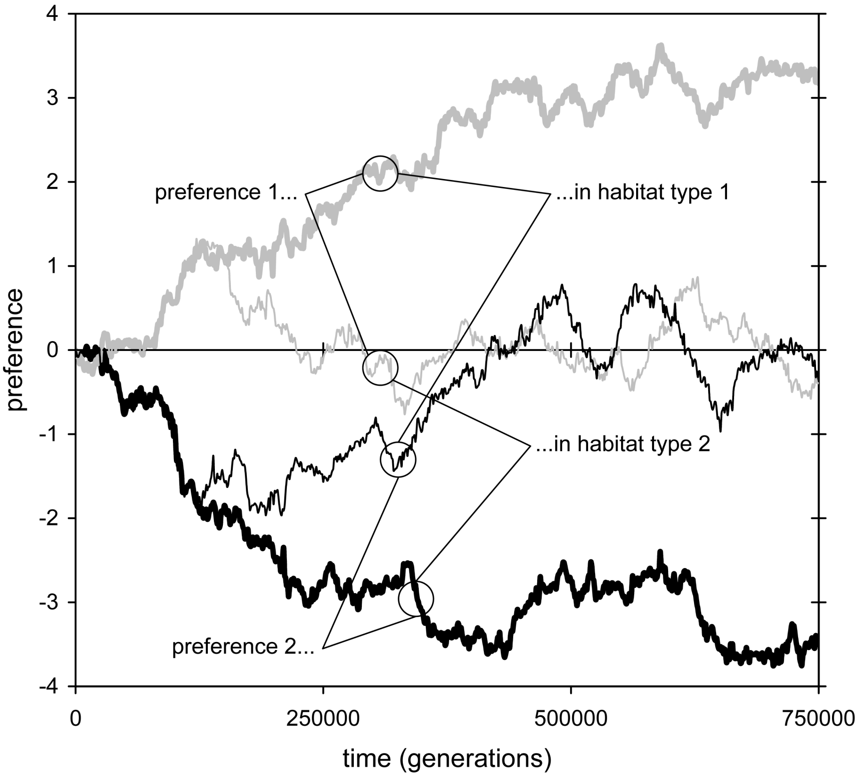


FIGURE 1 – SPECIATION THROUGH THE DIVERGENCE OF MATING PREFERENCES FOR TWO ORNAMENTS

The mate choice model developed in CHAPTER 8 of this thesis was implemented for a population in a heterogeneous environment consisting of two habitats. Ornament 1 is a revealing indicator for the first quality component in habitat 1 only, since deleterious mutations of the first quality component are only expressed in the first habitat. Ornament 2 is a revealing indicator in habitat 2 only, since deleterious mutations of the second quality component are only expressed in the second habitat. After the initial evolution of mating preferences for both ornaments in both habitats, the mating preferences diverge, giving rise to reproductive isolation between the subpopulation in the two habitats. In this simulation, both quality components were based on 50 diploid loci; every deleterious mutation decreased viability by a factor 0.99. Every generation, 1% of the population migrated between patches. This is for all $\alpha_k = 4.0$, except $\alpha_{aB} = \alpha_{ab} = 1.0$ in habitat 1 and $\alpha_{Ab} = \alpha_{ab} = 1.0$ in habitat 2. Other parameters: $\beta_1 = \beta_2 = 1.0$, $\gamma_1 = \gamma_2 = 0.01$, $\vartheta_1 = \vartheta_2 = 0.05$, $L = 5$, $\mu = 0.001$, $\nu = 1.0 \cdot 10^{-5}$, $m = 0.01$, $\sigma_m = 0.04$.

In the initial phase of the simulation (up to generation 125,000), females evolve preferences for both ornaments. Later on, the mating preferences of females that occur in different habitat types diverge, until, eventually, females only assess the ornament that provides information about the quality component that is relevant for the habitat the female lives in. As a result of the divergence of mating preferences, males from one habitat have a low mating success in the other habitat. The level of gene flow between the two habitats therefore decreases to low levels, which facilitates the further genetic divergence of the two sub-populations in the different habitats through the accumulation of mutations that are deleterious in the other habitat.

A similar type of dynamics can be observed in an analytical version of the model (FIGURE 2). The mathematical analysis reveals that evolution may converge on different types of equilibria. As illustrated in FIGURE 2, there exist two stable boundary equilibria, where females assess only a single ornament. In addition, there is an internal equilibrium at which females assess both ornaments. The basin of attraction of the internal equilibrium extends nearly to the boundary equilibria, indicating that minor stochastic fluctuations could easily induce the population to shift from a boundary equilibrium to the internal equilibrium.

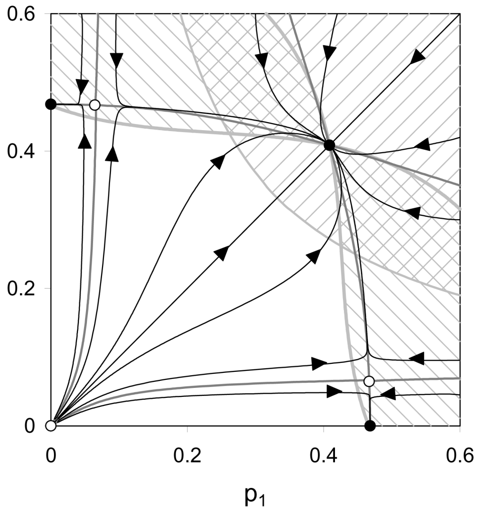


FIGURE 2 – MATHEMATICAL ANALYSIS

An analytical version of the individual-based simulation model represented in FIGURE 1, reveals the location of evolutionary equilibria in the model. Preferences for the first and second ornament are shown on the horizontal and vertical axes, respectively. Apart from an internal equilibrium, where females assess both ornaments, there are two boundary equilibria. The internal equilibrium is convergence stable, but not evolutionary stable; the invasion of mutants may lead to the establishment of protected dimorphism (see main text). We would therefore expect further evolution and diversification of mating preferences after convergence to the internal equilibrium. This is for 1% migration per generation. Other parameters are: $\beta_1 = \beta_2 = 0.5$, $\gamma_1 = \gamma_2 = 0.02$, $\vartheta_1 = \vartheta_2 = 0.05$, $\mu_A = \mu_B = 0.05$. All $\alpha_k = 2.5$ and $v_k = 1.0$, except $\alpha_{aB} = 0.5$ and $v_{aB} = 0.9$ in habitat 1 and $\alpha_{Ab} = 0.5$ and $v_{Ab} = 0.9$ in habitat 2

As soon as the population has converged on the internal equilibrium (cf. FIGURE 1, generation 0-125,000), further evolution will take place. This is because the internal equilibrium is not stable against invasion by mutants with other combinations of preferences. The mutants that are able to increase in numbers in a resident population at the internal equilibrium, lie within the first hatched area (hatched with gray lines from lower left to upper right). Successful invasion by one such mutant may lead to the establishment of a protected dimorphism. Indeed, this is possible for all combinations of preferences $(p_1, p_2) = (x, y)$ and $(p_1, p_2) = (y, x)$ that lie within the second hatched area (hatched with gray lines from upper left to lower right). Crucially, the area designating potential mutants and the area of protected dimorphism partially overlap (cross-hatched area), indicating that the internal equilibrium is an evolutionary branching point. Based on this fact, we would expect the establishment of a dimorphism and subsequent diversification of mating preferences after convergence on the internal equilibrium. This expectation is in line with the dynamics observed in the individual-based simulation (FIGURE 1, generation 125,000-750,000).

Although the diversification of mating preferences through evolutionary branching observed in the model is mathematically interesting, the model unfortunately has only limited practical relevance, since speciation is only observed for very low migration rates. Intuitively it is clear why this is so: Selection for the diversification of mating preferences is only weak (at least, as long as the cost of choice is low). Hence, already limited migration between the habitats is sufficient to homogenize the gene pools of the two subpopulations. Due to the restrictive conditions under which speciation occurs, we therefore preliminarily conclude that female mate choice for multiple habitat-specific ornaments is not likely to result in speciation, except under (nearly) allopatric conditions.

DIVERSIFICATION OF MATING PREFERENCES THROUGH SEXUAL CONFLICT

A different way in which good-genes sexual selection could contribute to speciation is suggested by the results of CHAPTER 9 of this thesis. In this chapter, we observed continuous coevolution of female preferences and male signaling strategies driven by the interplay between the handicap process and sexual conflict over the information content of signals used in mate choice. It is immediately clear that such dynamical evolutionary behavior could easily lead to the divergence of secondary sexual traits between populations that are geographically separated. To test whether the same is possible between populations that exchange migrants, we ran individual-based simulations of the model developed in CHAPTER 9 in a meta-population context (FIGURE 3).

We simulate a meta-population consisting of 11 patches, each supporting a constant number of 100 individuals. Female mate choice is based on 7 ornaments, which provide information about two genetic quality components (cf. CHAPTER 9, FIGURE 6; parameters are given in TABLE 1). The joint cost of choice is high. Migrants disperse to a common migrant pool and from there to a random patch in the meta-population (island-migration model).

There is no other force than random drift to promote the divergence of preferences between subpopulations. Yet, given the fact that switches in preference can occur very rapidly in this model (CHAPTER 9, FIGURE 1), one could imagine the repeated changes of female preference to cause (temporary) reproductive isolation between subpopulations. This idea, however, is not supported by the simulations, which demonstrate that already very low migration rates (1‰ per generation in FIGURE 3) suffice to homogenize the meta-population with respect to the preferences used by females to choose males.

Differentiation between subpopulations only arises when diversification of mating preferences is selectively advantageous. Such could, for instance, be the case when the meta-population experiences disruptive ecological selection, and when interbreeding between individuals from different patches is disadvantageous. In the simulation of FIGURE 3 (after 25,000 generations) we therefore modeled the evolution of an ecological trait under disruptive selection that affected the viability of individuals. The ecological trait was coded by a single diploid locus, with two alleles *A* and *a*. We assume that the patches of the meta-population are situated along an ecological gradient; the viabilities of the three ecological genotypes in the different patches are given in TABLE 2.

With ecological disruptive selection, we observe the establishment of different sets of preferences, effectively subdividing the meta-population into two reproductively isolated groups, according to the ecological characteristics of the patches (FIGURE 3 after 25,000 generations). The divergence of mating preferences locks onto the ecological differences between patches, such that patch 1-5 become reproductively isolated from patch 7-11 (patch 6 may belong to either one of these groups). Ecological disruptive selection on its own cannot give rise to a reduction of gene flow between ecologically dissimilar patches.

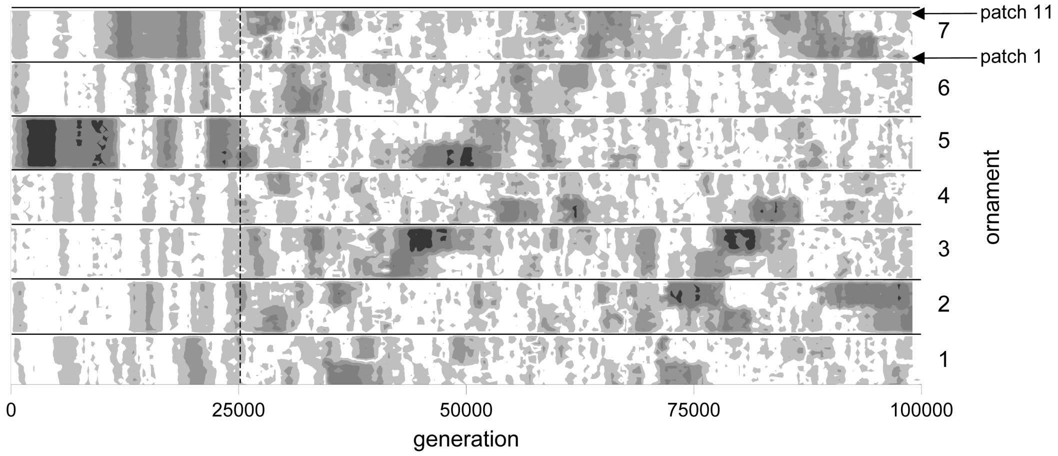


FIGURE 3 – CONTINUAL EVOLUTION OF PREFERENCES IN A META-POPULATION

The gray-scale coloring indicates the level of female preference for seven different ornaments in a meta-population. For each ornament, we show the level of choosiness in patch 1 to 11 (only indicated for the last ornament) of the meta-population. Before generation 25,000, there is hardly any differentiation between the patches: migration is very efficient in homogenizing preferences over the meta-population. After generation 25,000, we included disruptive selection on an ecological trait. Interbreeding between individuals from different patches is now disadvantageous. This favors the diversification of preferences in the meta-population, leading to reproductive isolation between the subpopulation in patch 1-5 and that in patch 7-11.

Yet, as before, reproductive isolation evolves only under restrictive conditions: hybridization must be strongly disfavored, requiring strong ecological disruptive selection, and the migration rate must be very low, in order to prevent the homogenization of mating preferences through gene flow. We therefore conclude that also this model in its current form does not provide a satisfactory general example of speciation through good-genes sexual selection.

TABLE 1 – SIMULATION PARAMETERS[a]

ornament	α_{AB}	α_{Ab}	α_{aB}	α_{ab}
1	5.0	4.94	1.74	1.0
2	5.0	4.86	2.07	1.0
3	5.0	4.74	2.42	1.0
4	5.0	4.58	2.79	1.0
5	5.0	3.75	3.88	1.0
6	5.0	1.98	4.88	1.0
7	5.0	1.19	4.99	1.0

a) Other parameters: $\beta = 0.5$, $\delta = 0.04$, $\vartheta = 2.0$, $\sigma_m = 0.05$, $\mu = 0.05$, $\nu = 0.0005$.

TABLE 2 – VIABILITIES OF ECOTYPES

patch	genotype		
	AA	*Aa*	*aa*
1	1.0	0.125	0.0
2	0.9	0.125	0.1
3	0.8	0.125	0.2
4	0.7	0.125	0.3
5	0.6	0.125	0.4
6	0.5	0.125	0.5
7	0.4	0.125	0.6
8	0.3	0.125	0.7
9	0.2	0.125	0.8
10	0.1	0.125	0.9
11	0.0	0.125	1.0

Part 3

Male-male Competition

The evolution of social dominance I: two-player models

10

G. Sander van Doorn, Geerten M. Hengeveld & Franz J. Weissing

Behaviour 140 (2003), p. 1305-1332

ABSTRACT

A difference in dominance rank is an often-used cue to resolve conflicts between two animals without escalated fights. At the group level, adherence to a dominance convention efficiently reduces the costs associated with conflicts, but from an individual's point of view, it is difficult to explain why a low ranking individual should accept its subordinate status. This is especially true if, as suggested by several authors, dominance not necessarily reflects differences in fighting ability but rather results from arbitrary historical asymmetries. According to this idea, rank differentiation emerges from behavioral strategies, referred to as winner and loser effects, in which winners of previous conflicts are more likely to win the current conflict, whereas the losers of previous conflicts are less likely to do so. In order to investigate whether dominance, based on such winner and loser effects, can be evolutionarily stable, we analyze a game theoretical model. The model focuses on an extreme case in which there are no differences in fighting ability between individuals at all. The only asymmetries that may arise between individuals are generated by the outcome of previous conflicts. By means of numerical analysis, we find alternative evolutionarily stable strategies, which all utilize these asymmetries for conventional conflict resolution. One class of these strategies is based on winner and loser effects, thus generating evolutionarily stable dominance relations even in the absence of differences in resource holding potential.

INTRODUCTION

An efficient way to reduce the disadvantages associated with group living is to decide the inevitable conflicts of interests between group members by means of ritualized conflict resolution strategies (Maynard Smith, 1982), rather than by physical fighting. Such strategies make use of some asymmetry between individuals other than the outcome of an escalated fight to resolve conflicts (Maynard Smith & Parker, 1976; Hammerstein, 1981). An often-used asymmetry is a difference in dominance rank (Schjelderup-Ebbe, 1922).

Although the establishment of dominance relations takes away the need to engage in costly fights in every small quarrel, this 'solution' has merely moved the problem to another level. Instead of having to resolve many small conflicts over resources, individuals now have to resolve one large conflict, that is, they must decide on the issue which of them will obtain the dominant status.

This decision could be based on some underlying asymmetry between individuals (Landau, 1951a), such as a difference in size or another aspect of their resource holding potential (RHP). When dominance status is always determined by the outcome of escalated fights, and when the largest individuals always win these fights, these individuals might automatically become dominant. Theoretical considerations indicate, however, that such *direct* determination of dominance by RHP asymmetries cannot account for the ordered structure of social hierarchies observed empirically (e.g. Schjelderup-Ebbe, 1922), unless the RHP asymmetries between individuals are unrealistically large (Landau, 1951a; Mesterton-Gibbons & Dugatkin, 1995).

Yet, when there are relatively small RHP differences between individuals, social ranks could still be *indirectly* determined by RHP asymmetries. This requires that individuals can accurately observe the small RHP differences and, most importantly, that all individuals adhere to a convention, such as 'the larger one is allowed to be dominant' (Parker, 1974; Maynard Smith & Parker, 1976; Hammerstein, 1981). Now, rank differentiation between large and small individuals does not arise because the smaller individual is unable to win, but rather because the smaller individual submits to a convention that assigns it the subordinate status. Such conventions can be used even when the size differences between individuals are only small, or when size differences do not accurately reflect actual RHP differences (Hammerstein, 1981). However, since the asymmetries between individuals are usually based on fixed individual characteristics, this mechanism cannot explain a number of experimental results. For example, experiments using cockroaches (Dugatkin *et al.*, 1994) and cichlid fish (Chase *et al.*, 2002) have shown that repeatedly reconstituting groups of individuals may result in completely different dominance hierarchies, indicating that dominance status need not at all be determined by fixed asymmetries between individuals.

Indeed, several authors have argued that dominance may be based on random historical or social asymmetries generated by the interactions between individuals (Landau, 1951b; Hogeweg & Hesper, 1983; Bonabeau *et al.*, 1996; but see Bonabeau *et al.*, 1999). For example, individuals that have previously won conflicts may have a higher tendency to behave aggressively in the current conflict, while losers in previous conflicts may act more submissively. These two effects – in the literature known as winner and loser effects (Chase *et al.*, 1994), respectively – result in a positive feedback between past performance and future probabilities of winning, from which rank differentiation may emerge (Hogeweg & Hesper, 1983). Winner and loser effects based on various proximate mechanisms have been described for a variety of species in the literature (see Chase *et al.*, 1994, and references therein). Moreover, theoretical models have shown that winner and loser effects give rise to rank differentiation within pairs of individuals and, as an emergent property at the group level, a stable linear dominance hierarchy, even without underlying asymmetries between individuals (Landau 1951b; Hogeweg & Hesper, 1983; Hemelrijk, 1999).

At the very least, these lines of evidence suggest that dominance is not completely determined by individual characteristics such as RHP asymmetries. Yet, the implication that dominance is – to some extent – based on random historical asymmetries leads one to ask why the subordinate individual should accept its subordinate status. Why should a low-ranking individual not fight, when its status was assigned arbitrarily and when, consequently, its chances of winning are fair? Winning would entitle it to the long-term benefits of the dominant status, and it would seem that these benefits should certainly outweigh the costs of a few extra fights, especially when one takes into consideration that dominance relations can persist for very long times.

These considerations indicate that the evolutionary stability (Maynard Smith & Price, 1973) of dominance based on winner and loser effects is far from obvious. One can rightfully ask whether winner and loser effects can evolve when the outcome of a previous interaction is arbitrary, that is, when the conflict outcomes hold no information about underlying RHP asymmetries. One can also question whether winner and loser effects may function as evolutionarily stable mechanisms to obtain information about hidden RHP asymmetries. To be sure, the history of winning and losing fights provides indirect information about differences in fighting ability. It is, however, not at all clear how large these asymmetries have to be in order to support evolutionarily stable winner and loser effects.

In this paper, we attempt to approach these questions by means of game-theoretical methods. We will develop an idealized model of repeated animal conflicts, and analyze the conditions under which a dominance convention based on winner and loser effects may evolve. For the sake of our argument, we concentrate initially on an extreme case in which there are no RHP differences at all between individuals. This allows us to investigate whether winner and loser effects can exist without such asymmetries. Moreover, we focus our attention in this paper on rank differentiation within pairs of individuals. Therefore, we restrict ourselves to the analysis of a two-player model. In CHAPTER 11 of this thesis, we extend our model to more than two players, allowing us to investigate the formation of social hierarchies.

MODEL DESCRIPTION

Game theory is well suited to analyze evolutionary problems where the success of a given strategy depends strongly on the strategies of other players, as in the case of conflict resolution strategies (Maynard Smith, 1982). However, the game-theoretical approach has a severe limitation in that it allows for the analysis of highly idealized models only. Models that attempt to give right to the biological complexity often run into the problem of delimiting the strategy space in such a way that mathematical analysis is still tractable.

In our case, the same problem occurs, since conflict resolution strategies may depend in a complicated way on the outcome of past conflicts (not only between the focal individual and its opponent, but also between the focal and other opponents, or between the opponent and the other individuals in the group). Moreover, individuals may behave differently depending on whether previous conflicts were resolved by means of ritualized or escalated fights. To cope with this problem, we will develop a number of idealized models, rather than a single more realistic model. This allows for an exploration of the biological complexities in several directions, without losing the tractability of the individual models.

Our first abstraction is that we describe conflicts between two individuals as a (slightly modified) Hawk-Dove game (Maynard Smith, 1982). That is to say, we assume that, whenever a conflict over some resource arises, individuals independently choose between two actions, designated Hawk and Dove. An individual playing Hawk is willing to escalate the conflict if the opponent does not retreat. An individual playing Dove retreats if the opponent escalates. Hence, if a Hawk meets a Dove the conflict is resolved without an escalated fight, and the Hawk obtains the resource. We will interpret Hawk-Dove interactions as 'dominance interactions'. If two Doves meet, the conflict is resolved without aggression, and either one of the players obtains the resource with equal probability, or they equally split the resource (we consider these two cases separately). Finally, if two Hawks meet, the conflict escalates, until one of them is injured and the other gets the resource. For the moment, we restrict ourselves to the special case where both players have an equal probability of winning the escalated conflict. This assumption allows us to investigate whether dominance, based on winner and loser effects, can be evolutionarily stable without any underlying RHP asymmetries. According to these rules, the payoffs for a focal individual are as follows

opponent focal	Hawk		Dove	
Hawk	$V-D$	if the focal wins	$V-D$	
	$-C-D$	if the focal loses		
Dove	0		$V/2$	if the resource is equally divided
			V	if the focal obtains the resource
			0	if the opponent obtains the resource

The parameter V denotes the value of the resource, and C denotes the cost of losing an escalated fight. Note that our payoffs differ slightly from those of the classical Hawk-Dove game: we included a small cost D associated with playing Hawk. In the case that two players play a single round of this modified Hawk-Dove game, the evolutionarily stable probability of playing Hawk, denoted p, can be calculated as

$$p = \frac{V-2D}{C}. \qquad [1]$$

For $D = 0$ one recovers the well-known mixed evolutionarily stable strategy (ESS) of the classical Hawk-Dove game (Maynard Smith, 1982).

We assume that individuals have a large number of conflicts. To be precise, the probability that two individuals will not interact again is taken to be $1/T$, such that, on average, individuals interact T times. Our next idealization is to assume that individuals can only remember the outcome of the preceding conflict and have no information about earlier conflicts. This restricts the strategy space to a class of simple memory-dependent strategies. In order to delimit the strategy space further, one needs to make assumptions about the information that is actually available to individuals.

In this paper, it is assumed that, whenever an individual meets an opponent, the individuals will use information about the previous conflict with this particular opponent only. So, individuals do not take into consideration any information that relates to either their own or their opponent's relation with other individuals. The effect of the use of such social information in conflict resolution strategies is discussed in CHAPTER 11 of this thesis. The restrictions imposed in the present paper allow us to describe all interactions between individuals in a group (whatever its size) as repeated pair wise conflicts between 'isolated' pairs of players. Under these assumptions, an individual may find itself in six different situations: it may have won or lost the previous conflict after an escalated fight, it may have won or lost after a dominance interaction or, it may have obtained the resource or not after an interaction without aggression. A conflict resolution strategy $\vec{p}$ is now defined as a vector of six strategic parameters

$$\vec{p} = \left(p_{hhw} \quad p_{hhl} \quad p_{hd} \quad p_{dh} \quad p_{ddw} \quad p_{ddl} \right), \tag*{[2]}$$

which prescribe the probability to play Hawk when an individual finds itself in either one of these six situations. The first two letters of the subscripts denote the actions of the focal and its opponent, respectively, in the previous conflict (h for Hawk, d for Dove). The third letter of the subscript, where necessary, denotes the outcome of the previous conflict (w for 'win', i.e. when the resource was obtained, l for 'lose' otherwise). The six strategic parameters are allowed to vary between δ and $1-\delta$, where δ is a small number (see the APPENDIX). This 'trembling hand' approach (Selten, 1975) is used to introduce occasional errors in decision-making, preventing the evolution of equilibrium solutions that are sensitive to such errors.

TABLE 1 – THE DIFFERENT INFORMATION LEVELS OF THE TWO-PLAYER MODEL

model variant 1: undividable resource
one of the players obtains the resource after a Dove-Dove interaction

level	interpretation	constraints
NONE	Use no information	$p_{hhw} = p_{hhl} = p_{hd} = p_{dh} = p_{ddw} = p_{ddl}$
DVD	Discriminate cases in which the resource was won (victories) from cases in which it was not (defeats)	$p_{hhw} = p_{hd} = p_{ddw}$ $p_{hhl} = p_{dh} = p_{ddl}$
DVD-HH	As level DVD, but, in addition, remember whether the previous conflict was resolved by Hawk-Hawk interaction or otherwise	$p_{hd} = p_{ddw}$ $p_{dh} = p_{ddl}$
DVD-DD	As level DVD, but, in addition, remember whether the previous conflict was resolved by Dove-Dove interaction or otherwise	$p_{hhw} = p_{hd}$ $p_{hhl} = p_{dh}$
ALL	Use all available information	none

model variant 2: dividable resource
the resource is split equally after a Dove-Dove interaction

level	interpretation	constraints
NONE	Use no information	$p_{hhw} = p_{hhl} = p_{hd} = p_{dh} = p_{ddw} = p_{ddl}$
DVD	Discriminate victories from defeats	$p_{hhw} = p_{hd}$, $p_{hhl} = p_{dh}$ $p_{ddw} = p_{ddl}$
ALL	Use all available information otherwise	$p_{ddw} = p_{ddl}$

INFORMATION LEVELS

The fact that we distinguish between the strategic variables p_{ddw} and p_{ddl} implies that an individual is assumed to be able to discriminate between Dove-Dove interactions in which it obtained the resource and Dove-Dove interactions in which it did not. This presupposes that the resource is not equally divided when both players play Dove. Otherwise, it would be impossible to designate a winner and a loser of the interaction. Nevertheless, we may still account for the case that the resource is equally divided, without resulting in any asymmetry between individuals, by imposing the constraint $p_{ddw} = p_{ddl}$. In fact, one may consider other constraints too, reflecting for instance limited mental capacities. These constraints result in what we will refer to as models of different 'information levels'.

At the lowest information level (which we will refer to as information level NONE), individuals use no information whatsoever regarding the outcome of the previous conflict. At level NONE, individuals may only vary their overall tendency to play Hawk, as implied by the constraint $p_{hhw} = p_{hhd} = p_{hd} = p_{dh} = p_{ddw} = p_{ddl}$. At the highest information level, (level ALL) no restrictions on the strategic parameters are imposed (with the exception of $p_{ddw} = p_{ddl}$ for the scenario where the resource is divided), corresponding to the case where individuals are able to distinguish all possible conflict outcomes and may behave differently in each of the six possible conflict situations. Intermediate information levels correspond to situations where individuals may only distinguish victories (cases in which the resource was obtained) from defeats (cases in which the resource was not obtained) (level DVD), or situations where individuals discriminate between victories and defeats and, in addition, behave differently after escalated fights (level DVD-HH) or Dove-Dove interactions (level DVD-DD). TABLE 1 shows the restrictions imposed on the strategic parameters for each of these information levels.

EQUILIBRIA OF THE MODEL

The model was analyzed by a combination of analytical and numerical techniques (see the APPENDIX for details). We first calculated the expected payoff, $W(\vec{q},\vec{p})$, of a rare mutant playing strategy $\vec{q}$ in a resident population where all individuals play strategy $\vec{p}$. From the payoff function, one obtains fitness gradients, which, under suitable assumptions (Hofbauer & Sigmund, 1998, chapter 9: adaptive dynamics), determine the direction and rate of evolution of the strategy $\vec{p}$.

The results presented throughout this paper, were obtained by simulations for one particular choice of parameters ($V = 0.3, C = 1, D = 0.025, T = 100$). Under these parameter conditions, the evolutionarily stable probability of playing Hawk for a single round of the Hawk-Dove game is given by $p = 0.25$ (equation [1]). We checked other parameter conditions and found qualitatively similar results, at least as long as individuals have a large number of interactions ($T \gg 1$) and as long as fighting is costly and the cost of playing hawk is low relative to the value of the resource ($0 < D < V < C$).

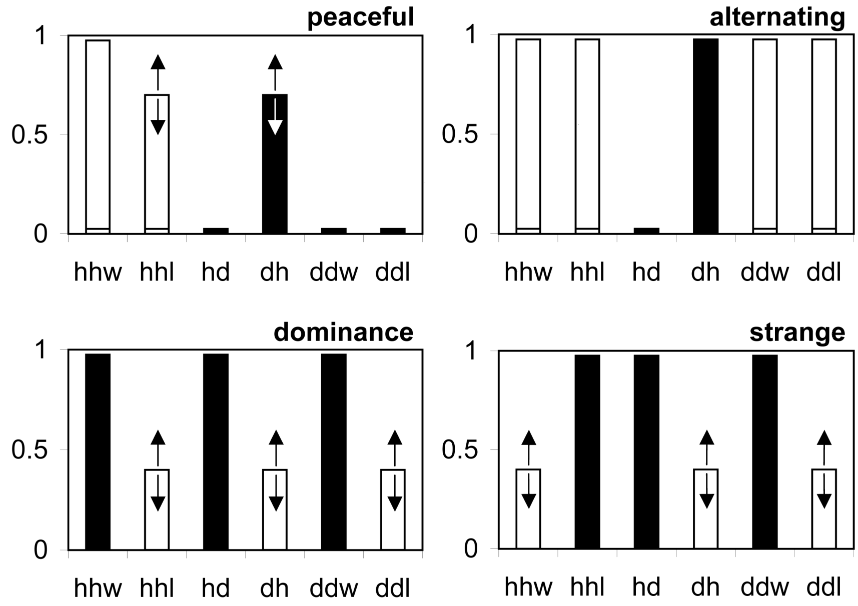


FIGURE 1 – CHARACTERISTICS OF THE DIFFERENT TYPES OF EQUILIBRIA

Except for the mixed equilibrium, which is described in the text, the four plots show for every category of equilibria the probabilities of playing Hawk in each of the six different situations in which an individual may find itself. Each equilibrium category has distinguishing features, which are indicated by black bars. The other strategic parameters are of lesser importance (indicated by white bars). Strategic parameters may vary between different equilibria belonging to the same category (as indicated by arrows, or by showing alternative high/low values). Upper left: Peaceful strategies satisfy two conditions. Individuals do not fight after Dove-Dove (*ddw* or *ddl*) or Hawk-Dove (*hd*) interactions and individuals do not always fight after a Dove-Hawk (*dh*) interaction. The different peaceful equilibria differ in their behavior after Hawk-Hawk (*hhw* or *hhl*) interactions, which, however, hardly ever occur. Upper right: In alternating equilibria, all strategic parameters approach pure strategies. Most importantly the strategic parameters p_{hd} and p_{dh} approach 0 and 1, respectively. Consequently, individuals alternate between two roles. Lower left: Dominance equilibria are characterized by the property that individuals that won the previous conflict will always play Hawk (black bars). Lower right: Strange equilibria are similar to dominance equilibria, except that the loser of an escalated fight, rather than the winner, continues to play Hawk.

In order to determine what kind of strategies evolve in the model, we ran a large number of simulations using the fitness gradient approach explained in the APPENDIX. Each simulation was started from random initial conditions and continued until convergence to an equilibrium. For most information levels (TABLE 1), multiple stable equilibria exist. The equilibria can be classified into five clearly distinguishable categories (FIGURE 1): mixed (M), dominance (D), alternating (A), peaceful (P) and strange (S). Most categories contain a number of different equilibria, which all share the defining properties of the particular class of equilibria.

TABLE 2 – EQUILIBRIA OF THE TWO-PLAYER MODEL

type[a]	label[b]	level[c]					values of strategic parameters[d]					
		NONE	DVD	DVD-HH	DVD-DD	ALL	p_{hhw}	p_{hhl}	p_{hd}	p_{dh}	p_{ddw}	p_{ddl}
M	M1	+	-	-	-	-	0.25	0.25	0.25	0.25	0.25	0.25
D	D1	-	+	-	-	-	1	0.24	1	0.24	1	0.24
	D2	-	+	-	+	-	1	0.25	1	0.25	1	1
	D3	-	-	+	-	-	1	0	1	0.32	1	0.32
	D4	-	-	-	-	+	1	0.86	1	0	1	1
A	A1	-	+	+	+	+	0	1	0	1	0	1
	A2	-	-	+	-	+	1	0	0	1	0	1
	A3	-	-	-	+	+	0	1	0	1	1	0
	A4	-	-	-	-	+	1	0	0	1	1	0
P	P1	-	+	-	+	-	0	0.74	0	0.74	0	0
	P2	-	-	-	-	+	0	1	0	0.73	0	0
	P3	-	-	-	-	+	1	0	0	0.73	0	0
S	S1	-	-	+	-	-	0	1	1	0.32	1	0.32
	S2	-	-	-	-	+	0.86	1	1	0	1	1

a) Equilibria were classified into five categories: M (mixed), D (dominance), A (alternating), P (peaceful) and S (strange).

b) Label used in FIGURE 2 & 3 and text.

c) See TABLE 1 for a description of the information levels. A '+' is to indicate that an equilibrium occurs in an information level, a '-' is to indicate that it does not.

d) 400 simulations were started for every level from random initial conditions and continued until convergence to an equilibrium. The values of the strategic parameters were kept between 0.025 and 0.975 (see the APPENDIX). For convenience, the values 0 and 1 are used to represent these extreme values. Parameters were: $V = 0.3$, $C = 1.0$, $D = 0.025$, $T = 100$.

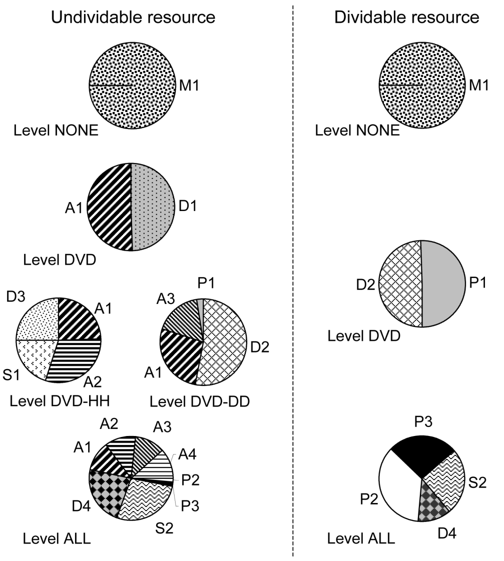


FIGURE 2 – EQUILIBRIA OF THE TWO-PLAYER MODEL

For every information level, 400 simulations were run, starting from random initial conditions, until convergence to an equilibrium. The pie diagrams show the different equilibria that were found at the given information level as well as the proportion of simulations in which they were reached by evolution. As explained in FIGURE 1, unique labels (M, D, A, P or S, shown next to the pie slices) could be assigned to every equilibrium. These labels can also be found in TABLE 2, which shows the values of the six strategic parameters for every equilibrium. Parameters as in TABLE 2.

Below we will describe the defining properties of the equilibrium categories. An overview is presented in FIGURE 2 and TABLE 2.

The simplest equilibrium type is the 'mixed' equilibrium. It occurs only when individuals have no memory and use no information whatsoever about the previous conflict (information level NONE). In this case, the game is identical to a simple Hawk-Dove game. Indeed, for the mixed equilibrium of information level NONE, the values of all strategic parameters are identical and equal to the mixed ESS of a single Hawk-Dove game (equation [1]).

The 'dominance' equilibria are characterized by a winner and loser effect: the winner of the previous fight is willing to escalate, whereas the loser is less likely to do so, which results in a positive correlation between the outcomes of past and future conflicts. This can most easily be seen from a time series of decisions that we generated for these strategies. For example, a typical time series of the decisions of two players playing the dominance strategy D3 (TABLE 2) against each other would be

$$\begin{array}{l} \text{player 1: } \\ \text{player 2: } \end{array} \begin{matrix} d \\ d \end{matrix} \begin{pmatrix} d \\ h \end{pmatrix}_3 \begin{matrix} h \\ h \end{matrix} \begin{pmatrix} d \\ h \end{pmatrix}_3 \begin{matrix} h \\ h \end{matrix} \begin{pmatrix} h \\ d \end{pmatrix}_8 \begin{matrix} h \\ h \end{matrix} \begin{pmatrix} d \\ h \end{pmatrix}_4 \begin{matrix} h \\ h \end{matrix} \ldots,$$

where the brackets are used to abbreviate repeated (series of) decisions, h is shorthand for the decision 'play Hawk' and d for 'play Dove'. We assume that all players play 'Dove' in their first interaction with a new opponent. As this time series shows, there are periods in which one individual plays Hawk in every interaction and the other individual plays Dove. During these periods, the individual that plays Hawk can be interpreted to dominate the other individual, resulting in an unequal division of the resource between the players. A period of dominance asymmetry ends with an escalated fight, after which the dominance relation between the players may either be reversed or not.

Another dominance strategy, D4, is characterized by prolonged series of escalated conflicts followed by a long lasting dominance relation:

$$\begin{array}{l}\text{player 1: } d\\ \text{player 2: } d\end{array}\begin{pmatrix}h\\h\end{pmatrix}_7\begin{pmatrix}h\\d\end{pmatrix}_{24}\begin{pmatrix}h\\h\end{pmatrix}_5\begin{pmatrix}d\\h\end{pmatrix}_{19}\cdots$$

'Alternating' equilibria are conventions in which the resources are equally divided over the players. In these equilibria, individuals alternate between an aggressive and a subordinate role. As a result, the players take turns in claiming the resource, and both obtain an equal share:

$$\begin{array}{l}\text{player 1: } d\\ \text{player 2: } d\end{array}\begin{pmatrix}hd\\dh\end{pmatrix}_{11}\begin{array}{c}hd\\dd\end{array}\begin{pmatrix}hd\\dh\end{pmatrix}_{16}\cdots$$

The different alternating strategies differ only in the resolution of Hawk-Hawk and Dove-Dove situations, which occur with a low probability only (when one of the player has made a mistake). Because of the low frequency of escalated fights, mean population fitness is higher for alternating strategies than for dominance strategies.

Moreover, individuals may also equally divide the resources without aggression at all (i.e. without playing Hawk). This results in a 'peaceful' strategy for which mean population fitness is even higher than for alternating strategies:

$$\begin{array}{l}\text{player 1: }\\ \text{player 2: }\end{array}\begin{pmatrix}d\\d\end{pmatrix}_{19}\begin{pmatrix}hd\\dh\end{pmatrix}_3\begin{pmatrix}d\\d\end{pmatrix}_{25}\begin{pmatrix}hd\\dh\end{pmatrix}_4\begin{array}{c}h\\d\end{array}\begin{pmatrix}d\\d\end{pmatrix}_{54}.$$

Peaceful strategies evolve when the resource is equally split after a Dove-Dove interaction but they also have a small range of attraction when this is not the case. This indicates that the peaceful strategies are sensitive to deviations from an exactly symmetric resolution of Dove-Dove interactions. The difference in mean population fitness between peaceful and alternating strategies is caused by the small cost of playing Hawk, as determined by the parameter D. When there is no cost associated with playing Hawk ($D = 0$), peaceful strategies can be invaded and replaced by alternating strategies.

The final category of equilibria comprises of 'strange' strategies. They are the paradoxical counterparts of dominance strategies, where the loser instead of the winner of escalated fights is allowed the dominant status. Although this class of equilibria does not seem to make sense from a biological point of view, similar solutions have been found in other game theoretical models. They occur because any asymmetry between individuals (in our case, the outcome of escalated fights) can be used to settle a conflict in both a common sense (D strategy) and a paradoxical manner (S strategy) (Maynard Smith, 1982). Both common sense and paradoxical solutions are evolutionarily stable. However, paradoxical solutions will only evolve from a situation where the asymmetry is ignored, when the asymmetry between individuals is completely payoff-irrelevant (i.e., irrelevant with respect to the expected costs and benefits of the current conflict) (Hammerstein, 1981). For our model, this means that S strategies will no longer evolve from biologically reasonable initial conditions as soon as RHP asymmetries between individuals are included (see below).

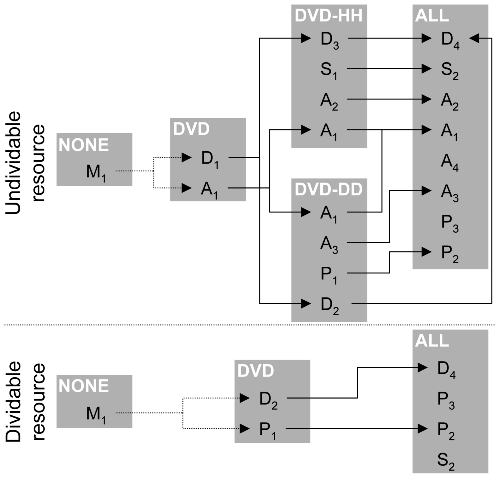


FIGURE 3 – TRANSITIONS BETWEEN INFORMATION LEVELS

In the course of evolution, species may shift from lower to higher information levels (gray rectangles), as increasingly detailed information is processed. To investigate the effect of a transition between two information levels (say a transition from level x to level y), we ran 200 simulations with initial conditions slightly perturbed from an equilibrium of information level x, until convergence to an equilibrium of level y. In most cases, all simulations started from a particular equilibrium converged to a single equilibrium at the higher information level (as indicated by the solid arrows). Only from the mixed equilibrium of level NONE two equilibria can be reached with about equal probability at the higher information level DVD (dashed arrows). Parameters as in TABLE 2.

EVOLUTIONARY PATHWAYS

It is likely that conflict resolution strategies tend to become more complex during the course of evolution, as selection sequentially removes some or all of the constraints imposed in our models. For example, the ability to remember whether the previous conflict was won or lost could evolve first (corresponding to a transition from level NONE to level DVD). This could then be followed by a further elaboration of mental abilities such that escalated fights are discriminated from other interactions (transition to level DVD-HH) and finally that all aspects of the previous interaction are taken into account (level ALL). In fact, evolution could proceed along a number of possible pathways in the following manner:

$$\begin{tikzcd} \text{dividable resource:} & \text{NONE} \arrow[r] & \text{DVD} \arrow[r] & \text{ALL} & \\ & & & \text{DVD-HH} \arrow[dr] & \\ \text{undividable resource:} & \text{NONE} \arrow[r] & \text{DVD} \arrow[ur] \arrow[dr] & & \text{ALL} \\ & & & \text{DVD-DD} \arrow[ur] & \end{tikzcd}$$

We simulated the different routes along which strategies can become more complex (FIGURE 3). After leaving the mixed strategy equilibrium of information level NONE, the equilibrium strategies attained in level DVD, remain in the same category (dominance, alternating, peaceful or strange) during further transitions to higher information levels. If changes occur, they are quantitative only (e.g. a population playing strategy D_1 at level DVD evolves to strategy D_3 at level DVD-HH), meaning that the type of strategy is always preserved during transitions between information levels.

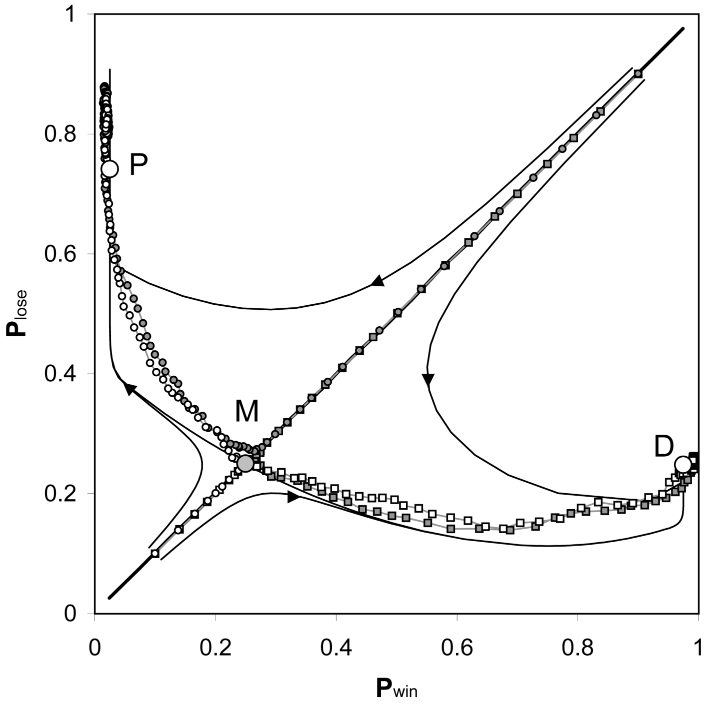


FIGURE 4 – THE DICHOTOMY BETWEEN DOMINANCE STRATEGIES AND OTHER CONVENTIONS

In the dividable resource scenario, there are three strategic variables at information level DVD (defined in the text as p_{win}, p_{lose} and p_{equal}). We show here a two-dimensional projection of the strategy space, since p_{equal} does not influence the dynamics. We studied the evolution of conflict resolution strategies after a transition from level NONE to level DVD, by means of individual based simulations (details are given in the APPENDIX). The figure shows four different simulations, started from two initial conditions: the simulations denoted by white circles and squares were started from the initial condition $p_{\text{win}} = p_{\text{lose}} = p_{\text{equal}} = 0.1$, the simulations denoted by gray symbols were started at $p_{\text{win}} = p_{\text{lose}} = p_{\text{equal}} = 0.9$. In the first 10,000 generations of the simulations, strategic parameters were constrained to information level NONE. Evolution then proceeds along the diagonal towards the mixed strategy equilibrium of level NONE (big gray circle). Data is plotted every 500 generations. After 10,000 generations, the constraint on the strategic parameters was removed, corresponding to a transition to information level DVD. The individual based simulations then diverged, with equal probability, to either the dominance equilibrium (lower right, gray and white squares) or the peaceful equilibrium (upper left, gray and white circles). The mixed strategy equilibrium is exactly on the boundary line (thick black line) between the domains of attraction of the stable equilibria of information level DVD (big white circles). The thin black lines with arrows represent deterministic evolutionary trajectories of the model at information level DVD. Parameters as in TABLE 2.

This implies that the transition from information level NONE, where no information is used, to level DVD, where individuals discriminate between different conflict outcomes in the simplest possible way, determines the outcome of long-term evolution. Further analysis of these transitions is facilitated by the fact that the strategies at information levels NONE and DVD contain only a small number of independent variables, allowing the application of qualitative and graphical methods.

FIGURE 4 shows a projection of the strategy space corresponding to information level DVD of the model. The data in FIGURE 4 are based on the dividable resource scenario, but the following results are also valid for the undividable resource scenario. On the axes are two strategic variables: $p_{\text{win}}\ (=p_{hhw}=p_{hd})$ and $p_{\text{lose}}\ (=p_{hhl}=p_{dh})$. Information about the third strategic variable, $p_{\text{equal}}\ (=p_{ddw}=p_{ddl})$, is lost in this projection, but, as it turns out, this variable does not influence the dynamics qualitatively with respect to the aspects we wish to discuss here. The strategy space corresponding to information level DVD contains the strategies of information level NONE as a subset. Strategies at information level NONE are characterized by $p_{\text{win}}=p_{\text{lose}}=p_{\text{equal}}$ and, therefore, evolution at information level NONE is constrained to the diagonal. As FIGURE 2 showed, there is only one evolutionary equilibrium at level NONE. For our choice of parameters, the values of the three strategic variables at this equilibrium are $p_{\text{win}}=p_{\text{lose}}=p_{\text{equal}}=0.25$. Indeed, FIGURE 4 shows that individual based simulations (see the APPENDIX) converge to this equilibrium along the diagonal from different initial conditions, as long as individual strategies are constrained to information level NONE. As soon as evolution away from the diagonal is allowed, corresponding to a transition from level NONE to level DVD, the simulations diverge and approach either the dominance equilibrium of level DVD in the lower right corner of the strategy space, or the peaceful equilibrium in the upper left corner. In the undividable resource scenario, the latter equilibrium is an alternating equilibrium.

Closer inspection reveals that there are three evolutionary equilibria at information level DVD. Two of them are stable (the dominance and peaceful equilibrium) and the third one is unstable. The unstable equilibrium coincides exactly with the mixed equilibrium of information level NONE (equilibrium M_1 of TABLE 2). This equilibrium is stable with respect to movement along the diagonal, hence the convergence to this equilibrium at level NONE, but unstable in the direction perpendicular to the diagonal. The diagonal serves as the boundary line (separatrix) between the regions of attraction of the two stable equilibria. Specifically, the mixed equilibrium is exactly on this boundary line. This explains why both peaceful and dominance equilibrium can be reached from the mixed equilibrium M_1 with about equal probability during the transition between information levels NONE and DVD.

A phase space analysis of the model for undividable resource shows a very similar topology of equilibrium points.

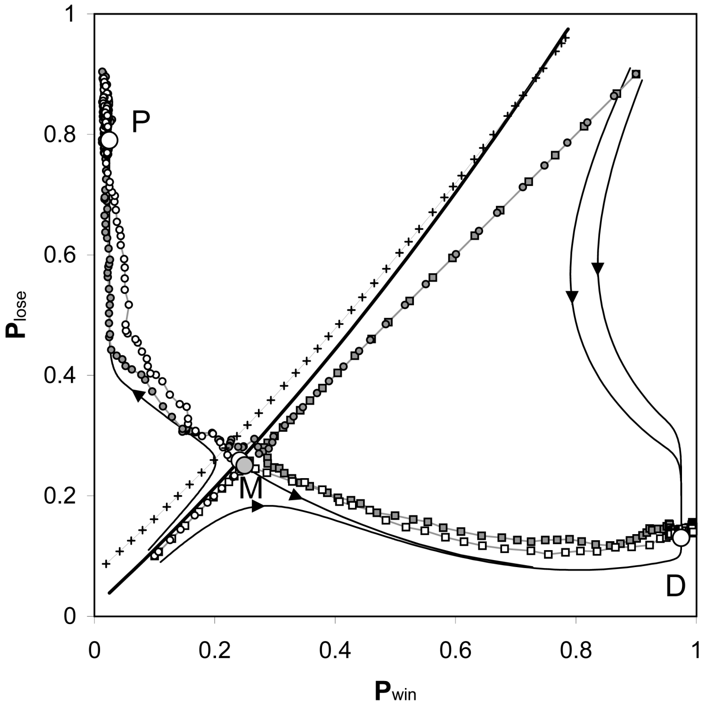


FIGURE 5 – EFFECTS OF RHP DIFFERENCES AND ERRORS IN INDIVIDUAL RECOGNITION

As in FIGURE 4, we performed individual based simulations corresponding to a transition between information levels NONE and DVD (dividable resource scenario), this time including hidden RHP differences between individuals as explained in the text. Due to these RHP asymmetries, the boundary line or separatrix (thick black line) between the domains of attraction of the equilibria of information level DVD (big white circles) has shifted slightly away from the diagonal. Therefore, the mixed equilibrium of information level NONE (big gray circle) no longer coincides with the unstable equilibrium of level DVD. Formally, this implies that further evolution from the equilibrium of information level NONE will always converge to the dominance equilibrium, as shown by the deterministic trajectory (thin black lines with arrows) leading from the mixed equilibrium (M) to the dominance equilibrium (D). The individual based simulations, however, show that, due to stochastic effects, convergence to the peaceful equilibrium is still possible, although the probability of reaching this equilibrium is now smaller than 50% (for this level of RHP asymmetry approximately 20% of the simulations converged to the peaceful equilibrium). When individuals not only differ in RHP, but also make occasional errors in individual recognition, the separatrix will shift even further away from the diagonal: the gray line with crosses represents the separatrix when individuals make errors in 50% of their interactions.

ASYMMETRIES IN RESOURCE HOLDING POTENTIAL

We have deliberately made the strong assumption that there are no observable or hidden RHP differences between the players. This allowed us to demonstrate that evolutionarily stable dominance relations based on winner and loser effects are possible without such underlying asymmetries. We are, of course, aware of the fact that in many biological systems RHP asymmetries are present. In order to check to what extent our conclusions are affected by the presence of RHP asymmetries, we analyzed an extended version of our model. In this model, we assumed that there are two RHP classes, representing individuals with high and low fighting ability. If two individuals, belonging to the same RHP class, engage in a Hawk-Hawk interaction, each of them has an equal probability of winning, as before. However, when two individuals belonging to different RHP classes engage in an escalated fight, the individual with the low fighting ability has a considerably lower probability of winning (for the sake of concreteness, we will assume it wins only one out of eight fights).

In order to study the effects of RHP asymmetries, we again considered the transition between information level NONE and DVD. As shown in FIGURE 5, RHP asymmetries result in one important qualitative change. The boundary line between the domains of attraction of the dominance and peaceful equilibrium has shifted slightly. Because of this, the mixed equilibrium M_1 no longer coincides exactly with the unstable equilibrium of level DVD. In fact, the equilibrium M_1 is now in the domain of attraction of the dominance equilibrium, and formally speaking, evolution from the mixed equilibrium M_1 will now always lead to the dominance equilibrium. However, even with this large RHP asymmetry, the mixed equilibrium is still very close to the separatrix. Therefore, the peaceful equilibrium can still be reached if the evolutionary dynamics is subject to small perturbations. Indeed, individual based simulations, which naturally include such small perturbations, show that the peaceful equilibrium is still attained, be it with a lower probability than the dominance equilibrium.

At higher information levels, RHP differences influenced the precise location of the equilibria (particularly for the dominance and strange equilibria), as well as the probability with which equilibria were reached from random initial conditions. However, RHP differences did not affect the transitions between higher information levels along the evolutionary pathways as shown in FIGURE 3.

THE EFFECT OF ERRORS IN INDIVIDUAL RECOGNITION

Another assumption that we made is that individuals have complete information about their situation. In other words, players always accurately remember the outcome of the previous conflict with their opponent. In order to check the robustness of our model with respect to this assumption, we again ran simulations for the transition from level NONE to level DVD, this time varying the quality of individual memory. That is to say, players made occasional errors in that they sometimes based their decision on a previous conflict with an arbitrary other opponent. These errors in individual recognition did not influence the results as long as there were no RHP asymmetries included. This is explained by the fact that the quality of individual memory is irrelevant when no information about the outcome of the previous conflict is used. The fitness of mutant strategies $p_{\text{win}} \approx p_{\text{lose}} \approx p_{\text{equal}}$ that are close to the strategies of information level NONE is therefore not at all affected by the quality of individual memory. Consequently, the boundary separating the domains of attraction of the dominance and peaceful equilibrium will remain on the diagonal and the unstable equilibrium of level DVD will continue to coincide with the mixed equilibrium of level NONE, as in FIGURE 4, regardless of the likelihood of errors in individual recognition.

However, when hidden RHP differences are included, errors in individual recognition enlarge the effect of the presence of RHP differences (FIGURE 5). The separatrix shifts farther away from the diagonal, and, consequently, the unstable equilibrium of level DVD moves farther away from the mixed equilibrium of level NONE. Still, the effects are not large and the behavior of the model with RHP differences and errors in individual recognition is comparable to the original model, which assumed complete information and absence of RHP asymmetries.

The interaction effect between RHP differences and errors in individual recognition results from the fact that the RHP differences between individuals generate a positive correlation between the outcomes of interactions between opponents and through time. There will be individuals that consistently win a large fraction of their escalated conflicts (those with high RHP) and others that consistently lose a large fraction of their conflicts (those with low RHP). If the population plays a peaceful or alternating strategy, mistakes in individual recognition will often lead to unintended escalated fights. In a population playing the dominance strategy, however, mistakes will be less likely to have unintended effects. For example, an individual may mistake its current opponent for another opponent it encountered previously. However, due to the correlation between the outcomes of the previous interactions with these two opponents, the mistake will often not have an effect at all, since the intended behavior for the one opponent is equal to the intended behavior for the other opponent. In short, if the population plays the dominance strategy, individuals can generalize to some extent from the outcome of previous interactions with arbitrary opponents. This makes individuals playing the dominance strategy less vulnerable to mistakes in individual recognition than individuals playing the alternating or peaceful strategy. This explains why the interaction between RHP differences and quality of individual recognition favors evolution of a dominance strategy.

DISCUSSION

Our results show that the historical asymmetry generated by the outcome of a previous interaction can be used as the deciding asymmetry in a conflict resolution strategy, even when this asymmetry is completely arbitrary and holds no information about differences in RHP. This conclusion is in line with well-established results from game theory, which state that any asymmetry, regardless of whether or not it accompanies a difference in RHP or expected payoff between the players, must be used for conventional conflict settlement. In other words, any strategy that ignores the asymmetry cannot be an ESS. This result was first shown by Maynard Smith & Parker (1976) and later established rigorously by Selten (1980, 1983, 1988). They also demonstrated two aspects of ESSs based on asymmetries that are particularly worth mentioning here.

First, the asymmetry can be used in different ways to resolve the conflict (Maynard Smith and Parker, 1976; Hammerstein, 1981). In our model, the asymmetry generated by the outcome of the previous conflict can be used such that the winner of a previous conflict escalates and the loser retreats, but also the opposite is possible. Hence, we find dominance and alternating strategies, which are counterparts with respect to the way in which the asymmetry after a Hawk-Dove or Dove-Hawk interaction is used. Moreover, we find different alternating strategies, which are a set of mutually opposite conventions with respect to the behavior after Hawk-Hawk or Dove-Dove interactions, and 'strange' strategies, which are the counterparts of dominance strategies.

The second aspect is that all ESSs that are based on an asymmetry must be pure strategies (Selten, 1980; Van Damme, 1991). For our model, this would imply that an individual should either always escalate or always retreat whenever it observes an asymmetry. Whether an individual can observe the asymmetry or not depends on the information level. This property of ESSs in asymmetric conflicts explains why the alternating strategies are pure strategies and why, in general, the evolved equilibrium strategies at higher levels, where more asymmetries are taken into account, contain more components that are pure (compare for example the strategies P_1 and P_2, or D_2 and D_4). Yet, many of the strategies that evolved in our model (particularly the dominance strategies) seem to be forbidden by Selten's theorem, since they still contain mixed strategic components. We suggest two possible (co-dependent) explanations for this apparent contradiction. First, it strongly depends on the set of alternative strategies whether a given strategy may be considered evolutionarily stable or not. While Selten's theorem presupposes a large strategy set, we have strongly delimited the space of possible options. In particular, we excluded strategies with perfect recall, which are crucial for Selten's argument (Van Damme, 1991). Second, Selten's theorem relies on the assumption that the payoff function $W(\vec{q},\vec{p})$ is linear in both the mutant and the resident strategy (Weissing, 1996). In our case, it is easy to see that repeated interactions between a pair of players leads to a payoff function that is highly nonlinear in both of its components. Such nonlinearities result in a decoupling of the properties of evolutionary stability (i.e., stability against invasion by rare mutants) and convergence stability (i.e., being approachable by a series of gene substitution events), which coincide in games with a linear fitness function (Geritz *et al.,*1998). As argued by Bulmer (1994, appendix 8.1), Selten's result only applies to situations where evolutionary stability implies convergence stability, which may not be the case in our model.

Our results regarding the evolutionary pathways along which conflict resolution strategies may change show that evolved strategies obeying an asymmetry are robust against changes in the amount or detail of the available information. That is to say, the type of strategy will not change when a higher information level is reached. Moreover, there will be selection to use increasingly detailed information in populations that play a dominance strategy. The important dichotomy between dominance and other strategies occurs already at low information levels, implying that already very simple strategies allow for a dominance convention.

We checked the robustness of our results with respect to two assumptions. First, hidden RHP asymmetries did not change our results dramatically. This is in line with the conclusions of Hammerstein (1981), who noted that models with payoff-irrelevant asymmetries are valid limiting cases of models with payoff-relevant asymmetries. Our conclusions are also confirmed by a more recent study (Crowley, 2001) on the evolution of memory dependent strategies in a repeated Hawk-Dove game with observable RHP differences. Second, our results were not affected by occasional errors in individual recognition, unless the lower quality of individual memory was accompanied by RHP asymmetries between individuals. In that case, errors in individual recognition tend to favor the evolution of a dominance strategy, since dominance conventions are less vulnerable to errors in individual recognition than alternating or peaceful strategies. This may be one of the reasons why we often find dominance strategies and not alternating strategies in nature: when a dominance hierarchy has formed, mistakes in individual recognition and recollection of previous events do not have large effects, since the outcomes of interactions are positively correlated between opponents and through time.

The two-player approach taken in the present paper has the important limitation that we cannot investigate the important issue of hierarchy formation. The current approach excludes the use of social information other than the information that can be extracted from previous interactions with the current opponent. Hierarchies, or even correlations between the outcomes of interactions with different opponents, can therefore only arise due to strong RHP asymmetries. However, when individuals base their behavior in a conflict on experiences with other group members than their current opponent, genuinely social hierarchies may arise as a result of bystander effects or transitive inference. A proper investigation of this issue requires a model in which conflict resolution strategies are made dependent on social relations other than the relation between the player and its opponent.

CHAPTER 11 of this thesis will focus on the importance of such 'third-party' effects on the evolution of social conventions.

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APPENDIX — METHODS FOR MATHEMATICAL ANALYSIS AND INDIVIDUAL BASED SIMULATIONS

THE PAYOFF FUNCTION

In order to derive equations governing the evolutionary dynamics of the conflict resolution strategies, we must first compute, for every conflict between two individuals, the probability that an individual finds itself in each of the six possible individual states, corresponding to the six possible outcomes of a single Hawk-Dove interaction. This can be done, if we know the strategy of each of the two players and the outcome of the previous conflict, since this determines the behavior of each player in the current conflict.

We will label the six possible outcomes of a conflict with the numbers 1-6, as follows

conflict type	outcome for focal individual	label
Hawk-Hawk	victory	1
	defeat	2
Hawk-Dove	victory	3
	defeat	4
Dove-Dove	victory	5
	defeat	6

Let us suppose that the first player, which we will arbitrarily regard as the focal individual, has strategy $\vec{p}=\left(p_{hhw} \quad p_{hhl} \quad p_{hd} \quad p_{dh} \quad p_{ddw} \quad p_{ddl}\right)$ and that the second player (the opponent) has strategy $\vec{q}=\left(q_{hhw} \quad q_{hhl} \quad q_{hd} \quad q_{dh} \quad q_{ddw} \quad q_{ddl}\right)$. By way of example, let us further suppose that the previous conflict between these players ended as an escalated fight, which was won by the second player (hence the conflict ended with outcome 2).

The outcome of the previous conflict uniquely determines the probabilities with which both players will play 'Hawk' in the current conflict. In our example, player 1 will play 'Hawk' in the current conflict with probability p_{hhl} and player 2 will do so with probability q_{hhw}. It is now easy to calculate the likelihood of each of the six possible outcomes of the current conflict. For example, the probability that the current conflict will be resolved by a dominance interaction in which player 1 claims the resource and player 2 retreats (outcome 3) is given by the transition probability $m_{3,2}$, where

$$m_{3,2} = p_{hhl}\left(1 - q_{hhw}\right). \qquad [3]$$

Similarly, we may compute other transition probabilities m_{ij} $(i, j = 1\ldots6)$, which give the probability that the current conflict will end with outcome i, given that the previous conflict ended with outcome j. The 36 transition probabilities, m_{ij}, can be ordered in a transition matrix $\mathbf{M}(\vec{p},\vec{q})$, such that m_{ij} is the element at the i-th row and j-th column of $\mathbf{M}(\vec{p},\vec{q})$. The column vectors of $\mathbf{M}(\vec{p},\vec{q})$ each correspond to one of the six possible outcomes of the *previous* conflict and their elements represent the likelihood of each of the six outcomes of the *current* conflict.

Next, we define vectors $\vec{u}_n(\vec{p},\vec{q})$, which contain the probabilities that the focal individual finds itself in each of the six possible individual states corresponding to the six outcomes of the n-th conflict between the two players. It is easy to see that

$$\vec{u}_n(\vec{p},\vec{q}) = \mathbf{M}(\vec{p},\vec{q})\,\vec{u}_{n-1}(\vec{p},\vec{q}). \qquad [4]$$

We assume that the players both play 'Dove' in their first conflict. Hence,

$$\vec{u}_1(\vec{p},\vec{q}) = \begin{pmatrix} 0 & 0 & 0 & 0 & \frac{1}{2} & \frac{1}{2} \end{pmatrix}^{\mathrm{T}}. \qquad [5]$$

Equations [4] and [5] together uniquely determine the sequence $\vec{u}_1(\vec{p},\vec{q}), \vec{u}_2(\vec{p},\vec{q}), \vec{u}_3(\vec{p},\vec{q}),\ldots$ from which the average expected payoff, $W(\vec{p},\vec{q})$, of an individual playing strategy $\vec{p}$ against an opponent playing strategy $\vec{q}$ can be calculated.

In order to find $W(\vec{p},\vec{q})$, we must first calculate the expected payoff $w_n(\vec{p},\vec{q})$ to the focal player (i.e., player 1) in the n-th conflict. The payoffs associated with the six possible conflict outcomes are represented in a payoff vector $\vec{v}$ as follows

$$\vec{v} = \begin{pmatrix} V-D & -C-D & V-D & 0 & V & 0 \end{pmatrix}. \qquad [6]$$

The expected payoff $w_n(\vec{p},\vec{q})$ to the focal player in the n-th conflict quantity can now be obtained by weighing the payoffs associated with every possible conflict outcome with the probability of occurrence of that outcome in the n-th conflict. In other words,

$$w_n(\vec{p},\vec{q}) = \sum_{i=1\ldots6} (\vec{v})_i \left(\vec{u}_n(\vec{p},\vec{q})\right)_i = \vec{v}\,\vec{u}_n(\vec{p},\vec{q}). \qquad [7]$$

Under the assumption that individuals interact T times on average, the average expected payoff $W(\vec{p},\vec{q})$ can now be calculated as

$$W(\vec{p},\vec{q}) = \frac{1}{T}\sum_{n=1}^{\infty}\left(1 - 1/T\right)^{n-1} w_n(\vec{p},\vec{q}). \qquad [8]$$

The factor $\left(1 - 1/T\right)^{n-1}$ is necessary to weigh the expected payoff of the n-th conflict with the probability that this conflict will actually occur.

EVOLUTIONARY DYNAMICS

We are interested in finding strategies that are (a) stable against invasion by alternative strategies, and (b) attainable by evolution, more specifically, by a series of small phenotypic steps, each corresponding to a mutation and subsequent trait substitution. Such strategies must at least be best replies to themselves (Weissing, 1996). A strategy $\vec{p}$ is a best reply to itself when there are no alternative mutant strategies $\vec{q}$ that would outperform an individual playing the resident strategy $\vec{p}$ against its opponents (which also play the strategy $\vec{p}$). That is to say, a strategy $\vec{p}$ is a best reply to itself when

$$\text{for all alternative strategies } \vec{q}: \quad W(\vec{q},\vec{p}) \leq W(\vec{p},\vec{p}). \qquad [9]$$

If condition [9] holds, the strategy $\vec{p}$ is called a Nash equilibrium strategy. Unfortunately, not all Nash equilibria are evolutionarily stable. That is, Nash equilibria need not be stable against invasion by alternative strategies (problems may arise when there are alternative best replies $\vec{r}$ such that $\vec{r} \neq \vec{p}$ and $W(\vec{r},\vec{p}) = W(\vec{p},\vec{p})$). Moreover, condition [9] gives no information regarding the attainability, or convergence stability, of the strategy $\vec{p}$. Convergence stability ensures that a population, which is slightly perturbed away from the strategy $\vec{p}$, will evolve back to the strategy $\vec{p}$. Formally, we would need to check higher order conditions to ensure the evolutionary and convergence stability of the strategy $\vec{p}$ (Eshel, 1983, Geritz *et al.*, 1998). Here, we refrained from doing so. Instead, we took a different approach and derived a dynamical equation for the evolutionary rate of change of the strategy $\vec{p}$. Evolutionarily stable strategies correspond to equilibria of this equation, and the stability of these equilibria will give information about the convergence stability properties of the corresponding strategy.

Let us suppose that the strategy $\vec{q}$ is a rare mutant strategy that has arisen in an otherwise monomorphic resident population, in which all individuals play the strategy $\vec{p}$. The probability that the mutant invades this resident population is determined by the difference between the average expected payoff of the mutant, $W(\vec{q},\vec{p})$, and the average expected payoff of the resident, $W(\vec{p},\vec{p})$. Note that the initial rareness of the mutant is responsible for the fact that invasion of the mutant is independent of the performance of the resident against mutant opponents (measured by $W(\vec{p},\vec{q})$) and performance of the mutant against mutant opponents (measured by $W(\vec{q},\vec{q})$). If mutations occur in small steps, the resident and mutant strategies will only be slightly different from each other. In that case, $\|\vec{p}-\vec{q}\|$ is small, which allows us to approximate the fitness difference between mutant and resident as

$$W(\vec{q},\vec{p}) - W(\vec{p},\vec{p}) \approx (\vec{p}-\vec{q}) \cdot \left.\frac{\partial W(\vec{q},\vec{p})}{\partial \vec{q}}\right|_{\vec{q}=\vec{p}} . \qquad [10]$$

In other words, the fitness difference between mutant and resident is proportional to the local fitness gradient. Therefore, the fitness gradient determines the probability of invasion, which is directly related to the rate of evolution of the strategy $\vec{p}$. In other words,

$$\frac{\partial\vec{p}}{\partial t} = \kappa\,\mathbf{G}\left.\frac{\partial W(\vec{q},\vec{p})}{\partial\vec{q}}\right|_{\vec{q}=\vec{p}} \qquad [11]$$

(Hofbauer & Sigmund, 1998, Chapter 9). In this equation, the rate constant κ is determined by the population size and the rate of mutations. The matrix $\mathbf{G}$ is a mutational variance-covariance matrix. Evolutionarily stable strategies $\vec{p}$ are also equilibrium solutions of equation [11]. Moreover, stable equilibria of equation [11] correspond to convergence stable strategies, usually, but not necessarily (Geritz, 1998), continuously stable strategies (that is, convergence stable and evolutionarily stable strategies, Eshel, 1983).

We used the mutational variance-covariance matrix $\mathbf{G}$ to implement the constraints corresponding to the different information levels. For example, we used the 6×6 identity matrix as the mutational variance–covariance matrix for information level ALL (that is, all six traits were assumed to evolve independently). Similarly, we used the following matrix as mutational variance-covariance matrix at level DVD (dividable resource scenario),

$$\mathbf{G}^{(\text{DVD, dividable resource})} = \begin{pmatrix} 1 & 0 & 1 & 0 & 0 & 0 \\ 0 & 1 & 0 & 1 & 0 & 0 \\ 1 & 0 & 1 & 0 & 0 & 0 \\ 0 & 1 & 0 & 1 & 0 & 0 \\ 0 & 0 & 0 & 0 & 1 & 1 \\ 0 & 0 & 0 & 0 & 1 & 1 \end{pmatrix}, \qquad [12]$$

corresponding to the assumption that evolution of the strategic parameters is controlled by the constraints $p_{hhw} = p_{hd}$ (hence $\mathbf{G}_{1,1} = \mathbf{G}_{3,3} = \mathbf{G}_{1,3} = \mathbf{G}_{3,1}$), $p_{hhl} = p_{dh}$ ($\mathbf{G}_{2,2} = \mathbf{G}_{4,4} = \mathbf{G}_{2,4} = \mathbf{G}_{4,2}$) and $p_{ddw} = p_{ddl}$ ($\mathbf{G}_{5,5} = \mathbf{G}_{6,6} = \mathbf{G}_{5,6} = \mathbf{G}_{6,5}$).

In order to solve equation [11], we used a standard algorithm for the numerical integration of ordinary differential equations. The elements of the local fitness gradient were computed numerically using equations [4]-[8] by calculating the fitness of the six mutant strategies that differed slightly from the resident in one of the strategic variables. The strategic parameters were allowed to vary between δ and $1-\delta$, where δ is a small number (throughout this paper, we used $\delta = 0.025$). This 'trembling hand' approach (Selten, 1975) was used in order to exclude equilibrium strategies that are sensitive to occasional errors in decision-making.

INDIVIDUAL BASED SIMULATIONS

In the individual based simulations, we traced the evolution of a population of 100 individuals. Each of these had repeated Hawk-Dove interactions as described under 'model description'. The total payoff gained in interactions throughout its lifetime determined the number of offspring produced by an individual. The three strategic parameters of an individual's conflict resolution strategy were each determined by a diploid locus. We assumed normal Mendelian inheritance, free recombination between loci and additive interactions between alleles. Mutations, altering the phenotypic effect of an allele slightly (by 1%) occurred at a low frequency (1% per allele per generation).

The evolution of social dominance II: Multi-player models

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G. Sander van Doorn, Geerten M. Hengeveld & Franz J. Weissing

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ABSTRACT

The social hierarchies observed in natural systems often show a high degree of transitivity. Transitive hierarchies do not only require rank differentiation within pairs of individuals but also a higher level ordering of relations within the group. Several authors have suggested that the formation of linear hierarchies at the group level is an emergent property of individual behavioral rules, referred to as winner and loser effects. Winner and loser effects occur if winners of previous conflicts are more likely to escalate the current conflict, whereas the losers of previous conflicts are less likely to do so. According to this idea, an individual's position in a hierarchy may not necessarily reflect its fighting ability, but may rather result from arbitrary historical asymmetries, in particular the history of victories and defeats. However, if this is the case, it is difficult to explain from an evolutionary perspective why a low ranking individual should accept its subordinate status. Here we present a game theoretical model to investigate whether winner and loser effects giving rise to transitive hierarchies can evolve and under which conditions they are evolutionarily stable. The main version of the model focuses on an extreme case in which there are no intrinsic differences in fighting ability between individuals. The only asymmetries that may arise between individuals are generated by the outcome of previous conflicts. We show that, at evolutionary equilibrium, these asymmetries can be utilized for conventional conflict resolution. Several evolutionarily stable strategies are based on winner and loser effects and these strategies give rise to transitive hierarchies.

INTRODUCTION

In CHAPTER 10 of this thesis, we investigated the evolution of conflict resolution strategies in repeated conflicts between isolated pairs of individuals. We demonstrated that asymmetries generated by the outcome of previous interactions can be used for the resolution of future conflicts, even when the outcome of previous conflicts is not related to differences in resource holding potential (RHP, Parker, 1974). In particular, evolution may lead to behavioral strategies known as winner and loser effects, (Chase *et al.*, 1994), which, once established, cannot be replaced by alternative strategies.

Winner and loser effects generate a positive feedback between past performance and future probabilities of winning, which will automatically result in rank differentiation within isolated pairs of players. However, rank differentiation within pair wise relations will not automatically lead to a social hierarchy. When dominance within a pair is arbitrarily determined by winner and loser effects, it need not necessarily be true that, if individual A is dominant over B, and B is dominant over C, that then also A is dominant over C. In other words, the resulting social structure would most likely be intransitive, which is in contrast to the (almost) linear, transitive, social hierarchy that is observed in many biological systems (a classical example being the pecking order in a group of chickens, Schjelderup-Ebbe, 1922).

Apparently, it is not arbitrarily determined which of the two individuals in a pair becomes dominant. A linear hierarchy will arise if this decision depends not only on the history of interactions within the pairs, but also on relations with other individuals than the current opponent. Such a dependency could arise if winner and loser effects do not only act within pairs, but also between pairs. In other words, the individuals in a group would not only have to behave dominantly when they encounter an individual from which they previously won, as before, but also when they are dominant over many other group members. Similarly, individuals would not only have to act subordinately when they encounter individuals from which they previously lost, but also when they are subordinate to many other group members. These behavioral rules will have the effect that an individual has a higher probability of becoming dominant if it is already dominant in its relations with other individuals. Indeed, theoretical models have shown that between-pair winner and loser effects (or bystander effects) can give rise to stable linear dominance hierarchies (Landau, 1951b; Hogeweg & Hesper, 1983; Bonabeau *et al.*, 1996). Moreover, experimental studies (Chase, 1982; Chase *et al.*, 1994, and references therein) have demonstrated between-pair winner and loser effects based on various proximate mechanisms (e.g. mediated by hormones influencing aggressiveness Oliviera *et al.*, 2001).

There are alternative explanations for the transitivity of hierarchies in natural systems. A certain degree of transitivity could be induced by RHP-asymmetries between individuals. For example, if an individual has a very high RHP relative to its competitors, then this individual is likely to become the highest-ranking individual, since it will win most of its escalated conflicts. However, the conclusion that the transitivity of social hierarchies in natural systems is caused by underlying RHP asymmetries in this direct way seems implausible. In fact, the probability of finding a linear hierarchy in a group of modest size, where rank differences are determined directly by RHP asymmetries, is negligibly small even for high levels of RHP asymmetries (Landau, 1951a; Mesterton-Gibbons & Dugatkin, 1995; but see Appleby, 1983). Alternatively, social conventions based on RHP assessment (Maynard Smith & Parker, 1976; Hammerstein, 1981), could lead to transitive social hierarchies. For example, if individuals adhere to the convention that larger individuals are always allowed to win, then this will result in a transitive hierarchy in which ranks are directly related to size. However, the idea that dominance rank is completely determined by individual attributes such as RHP cannot explain experimental results (in cockroach, Dugatkin *et al.*, 1994, and cichlid fish, Chase *et al.*, 2002) showing that repeatedly reconstituting groups of individuals may result in completely different dominance hierarchies.

Without completely denying the importance of RHP asymmetries, the idea that social dominance emerges from winner and loser effects within and between pairs of individuals, immediately implies that dominance status is assigned, at least to some extent, arbitrarily. This is quite puzzling, at least from the perspective of the subordinate individual. If dominance rank is assigned arbitrarily, why should a subordinate individual accept its unfortunate position?

A possible answer to this question is that it is simply too costly for a low ranking individual to break the conflict resolution convention that (arbitrarily) assigned it to its subordinate status. A subordinate individual wanting to break the social convention would have to behave aggressively, despite its subordinate status. At the same time, its opponent will still treat it as a subordinate, and, hence, will behave aggressively as well. Consequently, a low ranking individual would have to go through many escalated and costly fights in order to ascend the social hierarchy.

In CHAPTER 10 of this thesis, we showed that this argument does indeed apply to within-pair winner and loser effects. In the present study, we investigate whether the evolutionary validity of this argument extends to between-pair winner and loser effects. To this end, we construct a game-theoretical model, in which the evolution of social conflict resolution strategies can be studied. Specifically, we are interested in the question whether between-pair winner and loser effects can evolve, and, once they have evolved, whether they are stable against invasion by alternative strategies.

MODEL DESCRIPTION AND ANALYSIS

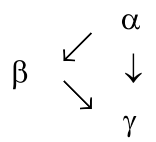
As in CHAPTER 10 of this thesis, we model conflicts between two individuals by a slightly modified Hawk-Dove game (Maynard Smith, 1982). For simplicity, we focus on that version of the model where not only Hawk-Hawk, but also Dove-Dove interactions create an asymmetry: when two 'Doves' meet they do not divide the resource, but either one of them obtains the resource with equal probability. Hence, the payoffs for a focal individual are given by

opponent focal	Hawk		Dove	
Hawk	$V-D$	if the focal wins	$V-D$	
	$-C-D$	if the focal loses		
Dove	0		V	if the focal obtains the resource
			0	if the opponent obtains the resource

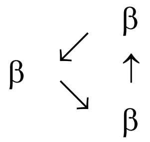
The parameter V denotes the value of the resource, C denotes the cost of losing an escalated fight, and there is a small cost D associated with playing Hawk. For the sake of our argument, we deliberately restrict ourselves for the moment to the special case where all individuals have an equal probability of winning escalated conflicts. This represents a worst-case scenario in which there are no underlying RHP asymmetries between individuals.

We assume that individuals participate in a large number of conflicts. On average, individuals interact T times. Individuals can only remember the outcome of the preceding conflict and have no information about earlier conflicts. In CHAPTER 10 of this thesis, we studied in detail how different asymmetries generated by the outcome of the previous conflict between an individual and its opponent may influence the course of actions in the current conflict. This aspect of conflict resolution is kept as simple as possible in the current paper: with respect to the outcome of the previous conflict, individuals are assumed to remember only whether they obtained the resource or not. Instead, the current model explores a different dimension of biological complexity. Individuals may now base their behavior not only on the preceding conflict between them and their current opponent, but also on their previous interaction with another individual (allowing for an effect of overall social rank on behavior) or on the previous interaction of their opponent with another individual (allowing for bystander effects, Chase, 1982). To keep things as simple as possible, let us first focus on a group of three individuals only (hence, we refer to this model as the three-player model). Individuals remember whether they obtained the resource or not in the previous conflict with both of their group members. An individual may therefore be in three states, which can be interpreted as different social ranks: it may have won from both other group members (we refer to such an individual as the α-individual), it may have won one conflict, but may have lost the other (β-individual), or it may have lost both conflicts (γ-individual).

Since the relation between the individuals in a pair is always asymmetric (by assumption), there can only be two social configurations within a group of three players. The first one is a transitive hierarchy, where one player is an α-individual, another is a β-individual and the third player is a γ-individual. The second possible social configuration is an intransitive hierarchy. Intransitive hierarchies occur when the first player won its previous conflict with the second player, the second player won its previous conflict with the third player, and the third player won its previous conflict with the first player. In such a case, all three players are in the same individual state (all three are β-individuals), but this does not mean that there are no asymmetric relations within pairs of players. The two possible social configurations are schematically shown below, with arrows pointing towards the loser of the previous conflict.



transitive



intransitive

A transition from a transitive to an intransitive social configuration occurs when the α-individual loses a conflict from the γ-individual. Similarly, an intransitive social configuration transforms into a transitive one when an individual wins a conflict with the opponent from which it previously lost.

In a transitive hierarchy, individuals can find themselves in six different conflict situations, depending on their own rank and on the rank of their opponent. In an intransitive hierarchy, all conflicts are between β-individuals, but there are nevertheless two different conflict situations: a player could encounter either the individual from which it previously won or the individual from which it previously lost. In total, there are therefore eight different conflict situations. Consequently, a conflict resolution strategy $\vec{p}$ consists of eight parameters, each prescribing the probability of playing Hawk for an individual that finds itself in the corresponding conflict situation:

$$\vec{p} = \begin{pmatrix} p_{\alpha\beta} & p_{\beta\alpha} & p_{\alpha\gamma} & p_{\gamma\alpha} & p_{\beta\gamma} & p_{\gamma\beta} & p_{\beta^+\beta^-} & p_{\beta^-\beta^+} \end{pmatrix}. \qquad [1]$$

The first letter of the subscripts indicates the state of the focal individual and the second letter the state of its opponent, where '+' ('-') is used, when necessary, to denote the β-individual that won (lost) the previous conflict.

We have supposed that individuals can recognize each other individually, or at least can accurately remember the outcome of conflicts with each of their group members. We deliberately made this assumption, since errors in individual recognition would automatically generate between-pair winner and loser effects if within-pair winner and loser effects have evolved. In other words, we will assume, at least initially, that individual recognition is perfect in order to be to be able to study the evolution of within-pair and between-pair winner and loser effects independently. Later, we will check the robustness of the results with respect to errors in individual recognition.

We consider a number of variants of the model that differ in the assumptions regarding the cognitive abilities of the players. We assume that individuals belonging to species with low cognitive abilities are unable to process all available social information, which translates into constraints on the strategic parameters. For example, if individuals remember the outcome of their previous interaction with all group members, but not the outcome of their opponent's previous interactions with other group members, then they cannot distinguish all conflict situations. For instance, the conflict situations

$$\begin{tikzcd}
& \text{opponent} \arrow[dl] \arrow[dd] & & & \text{opponent} \arrow[dl] \\
\text{focal} \arrow[dr] & & \text{and} & \text{focal} \arrow[dr] & \\
& \text{bystander} & & & \text{bystander} \arrow[uu]
\end{tikzcd}$$

would be indistinguishable for the focal individual. The fact that individuals cannot discriminate between two conflict situations implies that their behavior must be the same in both situations. Therefore, we must impose a constraint on the strategic parameters ($p_{\beta\alpha} = p_{\beta^-\beta^+}$ for this example).

TABLE 1 – THE DIFFERENT INFORMATION LEVELS OF THE THREE-PLAYER MODEL

level	constraints	interpretation
1	$p_{\alpha\beta} = p_{\beta\alpha} = p_{\alpha\gamma} = p_{\gamma\alpha} =$ $p_{\beta\gamma} = p_{\gamma\beta} = p_{\beta^+\beta^-} = p_{\beta^-\beta^+}$	No information about any previous conflict is used
2	$p_{\alpha\beta} = p_{\alpha\gamma} = p_{\beta\gamma} = p_{\beta^+\beta^-}$ $p_{\beta\alpha} = p_{\gamma\alpha} = p_{\gamma\beta} = p_{\beta^-\beta^+}$	Only the outcome of the previous conflict between the focal and its opponent is used
3^a	$p_{\alpha\beta} = p_{\alpha\gamma}$, $p_{\gamma\alpha} = p_{\gamma\beta}$ $p_{\beta\alpha} = p_{\beta\gamma} = p_{\beta^+\beta^-} = p_{\beta^-\beta^+}$	Individuals base their decision on their own rank
3^b	$p_{\beta\alpha} = p_{\gamma\alpha}$, $p_{\alpha\gamma} = p_{\beta\gamma}$ $p_{\alpha\beta} = p_{\gamma\beta} = p_{\beta^+\beta^-} = p_{\beta^-\beta^+}$	Individuals base their decision on their opponent's rank
4^a	$p_{\alpha\beta} = p_{\alpha\gamma}$, $p_{\beta\gamma} = p_{\beta^+\beta^-}$ $p_{\beta\alpha} = p_{\beta^-\beta^+}$, $p_{\gamma\alpha} = p_{\gamma\beta}$	Individuals disregard their opponent's relation with the bystander
4^b	$p_{\beta\alpha} = p_{\gamma\alpha}$, $p_{\alpha\beta} = p_{\beta^+\beta^-}$ $p_{\gamma\beta} = p_{\beta^-\beta^+}$, $p_{\alpha\gamma} = p_{\beta\gamma}$	Individuals disregard their own relation with the bystander
5	none	All available social information is used

Different assumptions regarding the complexity of the information used by individuals result in seven model variants characterized by different 'information levels' (TABLE 1). The seven variants of the model allow us to investigate how social information, besides the information obtained from previous interactions with the current opponent, may influence conflict resolution strategies. In particular, we are interested in the question whether conflict resolution strategies may evolve that give rise to between-pair winner and loser effects.

As in CHAPTER 10 of this thesis, the evolution of the system was investigated by means of an adaptive dynamics approach (see the APPENDIX for details).

EQUILIBRIA OF THE MODEL

For each information level, we ran a large number of simulations from random initial conditions until convergence to an equilibrium. At most levels, multiple stable equilibria exist. The equilibria can be classified into five categories (TABLE 2). The first category (hereafter labeled by 'M', for 'mixed') contains the mixed strategy equilibrium, in which no social information is used. Equilibrium strategies belonging to the second category (labeled by 'D', for 'dominance') are characterized by winner and loser effects. They lead to more or less stable transitive hierarchies. The third category (labeled by 'A', for 'alternating') contains equilibrium strategies that result in very unstable hierarchies, in which individuals continuously switch their social positions. The fourth category (labeled by 'T', for 'triangular') consists of equilibrium strategies that lead to stable intransitive social configurations. The final category contains hybrid strategies, which combine features of dominance, alternating and triangular strategies.

The distinguishing properties of dominance, alternating and triangular equilibria are further explained in FIGURE 1. An overview of all the equilibria that were found in the simulations is presented in FIGURE 2. In order to illustrate the social dynamics corresponding to the different equilibrium types, we generated time series of the decisions and ranks of the three players. These will be discussed below.

The simplest equilibrium type is the mixed equilibrium. This occurs only at information level 1, where no information about previous conflicts is used. Consequently, the game is equivalent to a simple Hawk-Dove game, and all strategic parameters evolve towards the evolutionarily stable probability of playing Hawk

$$p = \frac{V - 2D}{C}. \qquad [2]$$

Since no social information is used, the time series of ranks (upper rows) and decisions (lower rows) of the three players shows no obvious structure. It is given here to allow for a comparison with the time series for dominance, alternating and triangular strategies.

player 1:	β	γ	β	γ	γ	β	α	β	γ	β	β	α	β	γ	β	β	γ	...
player 2:	β	α	α	β	α	β	β	β	α	β	γ	γ	γ	β	β	α	α	...
player 3:	β	β	β	α	β	β	γ	β	β	β	α	β	α	α	β	γ	β	...
player 1:	0 d	d	d d	0	h	0 h	0 d d d d	d	h	0	d	d	d 0 h d d	d	0 0	d	h	...
player 2:	d d	0	0 d	d	d	d 0	h d 0 d 0	d	d	d	0	0	d d h 0 d	0	d d	0	h	...
player 3:	d 0	d	d 0	d	0	d h	d 0 h 0 d	0	0	d	d	h	0 h 0 h 0	d	d d	h	0	...

[3]

Time series [3] shows the social ranks of the three players and their decisions in a series of pair wise conflicts. The decision 'play Hawk' is denoted by '*h*' and the decision 'play Dove' by '*d*'. We assume that all players start as β-individuals in their first interaction. Dashed lines indicate transitions between different hierarchies and a '0' indicates the player that does not take part in the conflict (i.e., the bystander).

The dominance equilibria are characterized by the emergence of a more or less stable dominance hierarchy where the probability of playing Hawk increases with higher social rank. This is illustrated by the following typical time series corresponding to the dominance strategy D4 (see TABLE 2) of level 5 (all available information is used):

player 1:	β	γ	β	α	β	γ	⋯
player 2:	β	α	α	β	α	α	⋯
player 3:	β	β	γ	γ	γ	β	⋯
player 1:	d	0 d 0 h	0 h d h d 0 h 0 h	h 0 h 0 h	h h 0 h h	0 d d d 0	⋯
player 2:	h	h h h 0	h 0 h 0 h h 0 h h	0 h 0 h h	0 0 h h 0	h 0 h 0 h	⋯
player 3:	0	h 0 d h	d d 0 d 0 d d d 0	d d d d 0	d d d 0 h	d h 0 h h	⋯

[4]

As can be seen from time series [4] and from TABLE 2, the α-individual always plays Hawk. The β-individual always plays Hawk against the γ-individual, and usually Dove against the α-individual. Finally, the γ-individual plays Hawk with low probability when playing against the β-individual and Dove otherwise. This results in social dominance relations that may persist for quite some time.

Social stability is lacking when an alternating strategy has evolved. For example, in a population playing strategy A1, the social configuration changes after every conflict

player 1:	β	β	γ	γ	β	β	γ	γ	γ	β	β	β	γ	γ	β	α	β	β	α	α	β	γ	β	γ	β	β	α	β	γ	⋯
player 2:	β	γ	β	α	β	γ	β	α	β	β	α	β	β	α	α	β	β	γ	γ	β	α	α	β	α	α	β	γ	γ	β	⋯
player 3:	β	α	α	β	β	α	α	β	α	β	γ	β	α	β	γ	γ	β	α	β	γ	γ	β	β	β	γ	β	β	α	α	⋯
player 1:	0	d	0	h	0	d	0	0	h	0	0	d	0	h	h	d	0	h	0	d	d	h	d	h	0	h	d	d	h	⋯
player 2:	d	h	h	d	d	h	h	d	0	h	d	0	h	0	d	0	d	0	h	h	0	d	h	0	d	d	0	h	0	⋯
player 3:	h	0	d	0	h	0	d	h	d	d	h	h	d	d	0	h	h	d	d	0	h	0	0	d	h	0	h	0	d	⋯

[5]

Without any constraints, there are five possible alternating strategies (A1 and A4-A7). All of them occur with approximately equal frequency in level 5, where individuals use all available social information (FIGURE 2).

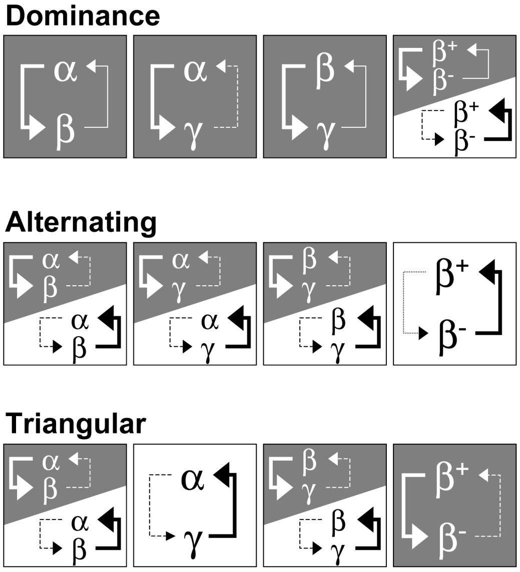


FIGURE 1 – CHARACTERISTICS OF THE DIFFERENT TYPES OF EQUILIBRIA

Within the framework of our three-player model, conflicts are possible between α- and β-individuals, between α- and γ-individuals, between β- and γ-individuals, and, finally, between two β-individuals in case of an intransitive hierarchy. For dominance (D), alternating (A) and triangular (T) equilibrium strategies (explained in the text), these four conflict types are represented by four squares. The stability of the corresponding social relations is indicated by the scale-scale coloring of the square: white indicates that the social relation changes after every interaction (unstable relation), scale indicates that the social relation will have a low probability of changing after an interaction (stable social relation). The relation between α- and β-individual, for example, is quite stable in a dominance convention. This is because the α-individual always plays Hawk (indicated by the thick arrow), whereas the β-individual plays Hawk far less often (indicated by the thin arrow). Very unstable social relations, as, for example, the relation between α- and γ-individual in a triangular strategy, occur when the individual that previously won, never plays Hawk (dashed arrow), whereas it's opponent always plays Hawk, leading to a reversal of the social relation. The stability of the social relations in a dominance hierarchy is a common feature of dominance strategies that distinguishes them from the alternating strategies: in alternating strategies, at least one, but usually more than one relation in the transitive social configuration is unstable. There are several alternating strategies, each corresponding to a different possible combination of stable and unstable relations (see TABLE 2). For example, and as indicated by the two alternative representations of the corresponding square, the relation between α- and β-individual may either be stable or unstable in alternating strategies. Triangular strategies are characterized by the fact that the relations in an intransitive hierarchy are stable, whereas the relation between α- and γ-individual is unstable.

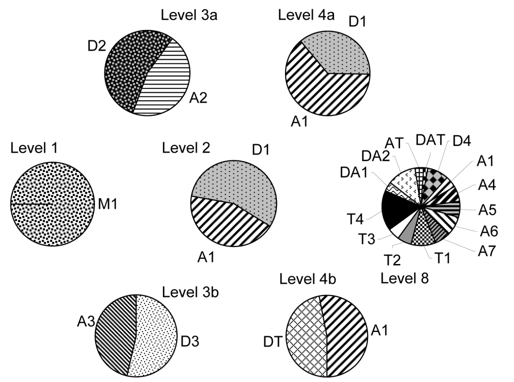


FIGURE 2 – EQUILIBRIA OF THE THREE-PLAYER MODEL

For every information level, 200 simulations were run, starting from random initial conditions, until convergence to an equilibrium. The pie diagrams show the different equilibria that were found at an information level as well as the proportion of simulations in which they were reached by evolution. As explained in the text, unique labels indicating the strategy type (M, D, A, T, or combinations for hybrid types) were assigned to every equilibrium. These labels can also be found in TABLE 2, which shows the values of the eight strategic parameters for every equilibrium. The simulation parameters were chosen as follows: $V = 0.3$, $C = 1.0$, $D = 0.025$, $T = 50$.

The triangular strategies are similar to the alternating strategies in the sense that they are also pure strategies. They exist only in level 5 (all available information). Triangular strategies occur when (a) the γ-individual always plays 'hawk' against the α-individual, which results in a triangular social configuration, and (b) the social relations in a triangular configuration are stable. There are four possible configurations, which, effectively, do not differ (differences only arise when a player makes a mistake). A typical time series is shown below.

$$\begin{array}{l} \text{player 1:} \\ \text{player 2:} \\ \text{player 3:} \\ \hline \text{player 1:} \\ \text{player 2:} \\ \text{player 3:} \end{array} \begin{pmatrix} & \beta & \\ & \beta & \\ & \beta & \\ \hline h & 0 & d \\ d & , h \text{ or } & 0 \\ 0 & d & h \end{pmatrix}_{28} \begin{array}{c:c:c:} \beta & \beta & \gamma \\ \beta & \gamma & \beta \\ \beta & \alpha & \alpha \\ \hline 0 & d & h \\ d & h & 0 \\ d & 0 & d \end{array} \begin{pmatrix} & \beta & \\ & \beta & \\ & \beta & \\ \hline d & 0 & h \\ h & , d \text{ or } & 0 \\ 0 & h & d \end{pmatrix}_{16} \begin{array}{c} \beta \\ \beta \\ \beta \\ \hline h \\ 0 \\ h \end{array} \begin{pmatrix} & \beta & \\ & \beta & \\ & \beta & \\ \hline d & 0 & h \\ h & , d \text{ or } & 0 \\ 0 & h & d \end{pmatrix}_{37} \begin{array}{c:c:c:l} \beta & \gamma & \gamma & \ldots \\ \beta & \beta & \alpha & \ldots \\ \beta & \alpha & \beta & \ldots \\ \hline d & 0 & h & \ldots \\ 0 & h & 0 & \ldots \\ d & d & d & \ldots \end{array} \qquad [6]$$

The brackets are used to abbreviate a repeated series of interactions.

Apart from the equilibria belonging to these categories, a number of equilibrium strategies are hybrids of the different types. For example, a time series corresponding to the strategy DT shows prolonged periods of a triangular social configuration, as in [6], alternating with periods of linear dominance hierarchies as in [4]. Switches between these qualitatively different types of social dynamics occur with low probability, that is, when individuals make a mistake. Other interesting hybrid strategies are DA_1 and DA_2, where the rank differences between α- and γ-individual and β- and γ-individual are stable, but α- and β-individuals alternate ranks (DA_1), or where the rank differences between α- and β-individual and α- and γ-individual are stable, but β- and γ-individuals alternate ranks (DA_2).

TABLE 2 – EQUILIBRIA OF THE THREE-PLAYER MODEL

type[b]	label[d]	level	values of strategic parameters[a, c]							
			$p_{\alpha\beta}$	$p_{\beta\alpha}$	$p_{\alpha\gamma}$	$p_{\gamma\alpha}$	$p_{\beta\gamma}$	$p_{\gamma\beta}$	$p_{\beta^+\beta^-}$	$p_{\beta^-\beta^+}$
M	M_1	1	**0.25**	**0.25**	**0.25**	**0.25**	**0.25**	**0.25**	**0.25**	**0.25**
D	D_1	2,4^a	1	0.22	1	0.22	1	0.22	1	0.22
	D_2	3^a	1	0.46	1	0	0.46	0	0.46	0.46
	D_3	3^b	1	0.33	1	0.33	1	1	1	1
	D_4	5	**1**	**0.42**	**1**	**0**	**1**	**0.22**	**0.22**	**1**
A	A_1	2, 4^a, 4^b, 5	**0**	**1**	**0**	**1**	**0**	**1**	**0**	**1**
	A_2	3^a	0	0	0	1	0	1	0	0
	A_3	3^b	0	1	0	1	0	0	0	0
	A_4	5	1	0	0	1	0	1	0	1
	A_5	5	0	1	0	1	1	0	0	1
	A_6	5	1	0	0	1	1	0	0	1
	A_7	5	0	1	1	0	0	1	0	1
T	T_1	5	**1**	**0**	**0**	**1**	**1**	**0**	**1**	**0**
	T_2	5	1	0	0	1	0	1	1	0
	T_3	5	0	1	0	1	1	0	1	0
	T_4	5	0	1	0	1	0	1	1	0
hybrid	DA_1	5	0	1	1	0	1	0.65	0	1
	DA_2	5	1	0.34	1	0	0	1	0	1
	DT	4^b, 5	1	0.31	1	0.31	1	0	1	0
	AT	5	0	1	1	0	0	1	1	0
	DAT	5	1	0.22	1	0.17	0	1	1	0

a) 200 simulations were started for every level from random initial conditions and continued until convergence to an equilibrium. The values of the strategic parameters were kept between 0.025 and 0.975 (see the APPENDIX). For convenience, the values 0 and 1 are used to represent these extreme values. Parameters were: $V = 0.3$, $C = 1.0$, $D = 0.025$, $T = 50$.

b) Equilibria were classified into five categories: M (mixed), D (dominance), A (alternating), T (triangular) and hybrids of these types.

c) Boldface indicates the equilibria that were used to construct the example time series shown in the text.

d) Label used in FIGURE 2 & 3 and text.

EVOLUTIONARY PATHWAYS

Up to here, we have separately analyzed the variants of the model for individuals with different cognitive abilities (corresponding to the different information levels). However, it is likely that the complexity of information used in conflict resolution strategies will change in the course of evolution. When selection removes the constraints imposed in the different information levels, and cognitive abilities increase, strategies shift from one information level to another. For example, the ability to remember the outcome of the previous conflict with an opponent could evolve first (corresponding to a transition from level 1, where no information is used, to level 2, where decisions are based only on the previous interaction with the current opponent). This could then be followed by a further elaboration of mental abilities, such that the information from relations with other individuals (level 4^a) and finally the full complexity of social relations within the group (level 5) is taken into account when deciding on the choice of action in a conflict. A second possible pathway would proceed from level 1 (no information is used) via level 3^a (decisions are based on one's own rank) and level 4^a (decisions are based on one's own rank and on the relation with the current opponent) to level 5 (all information is used). Two final, biologically less likely, pathways proceed from level 1 (no information) via level 2 (decisions are based on the relation with the current opponent) or 3^b (decisions are based on opponent's rank) to level 4^b (decisions are based on opponent's rank and on the relation with the current opponent) and finally to level 5 (all information is used).

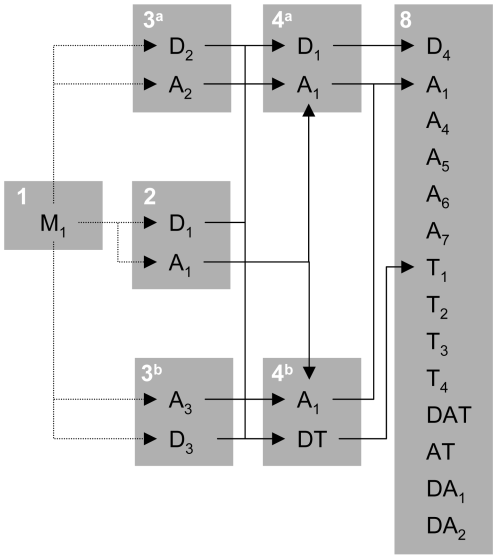


FIGURE 3 – TRANSITIONS BETWEEN INFORMATION LEVELS

In the course of evolution, species may shift from lower to higher information levels, as increasingly detailed information is processed. To investigate the effect of a transition between two information levels (say a transition from level x to level y), we ran 200 simulations with initial conditions slightly perturbed from an equilibrium of information level x, until convergence to an equilibrium of level y. In most cases, all simulations starting from a particular equilibrium converged to a single equilibrium at the higher information level. This is indicated in the figure by the solid arrows between equilibria at the different information levels (scale rectangles). However, in the transitions from the mixed equilibrium of level 1 (where no information is used) to higher information levels, multiple alternative equilibria can be reached (as indicated by dashed arrows). Parameters as in FIGURE 2.

In order to investigate the evolution of conflict resolution strategies along these evolutionary pathways, we simulated the following transitions between information levels (FIGURE 3):

$$\begin{tikzcd}
 & \text{level } 3^a \arrow[r] & \text{level } 4^a \arrow[dr] & \\
\text{level } 1 \arrow[ur] \arrow[r] \arrow[dr] & \text{level } 2 \arrow[ur] \arrow[dr] & & \text{level } 5\,. \\
 & \text{level } 3^b \arrow[r] & \text{level } 4^b \arrow[ur] &
\end{tikzcd}$$

In most cases, simulations started close from an equilibrium in a lower level converge to a unique equilibrium of the same type in the next level. This result shows that most strategies are robust against changes in the amount or detail of social information that is used to base decisions on. There are two exceptions to this rule. First, simulations do not converge to a unique equilibrium, but converge with equal probability to two different equilibria in the transitions from level 1 (no information is used) to higher levels. Second, there is a change of equilibrium type ($D_1/D_3 \rightarrow DT \rightarrow T_1$, see FIGURE 3) along the pathways via level 4^b (decisions are based on opponent's rank and on the relation with the current opponent). Along these pathways, individuals base their behavior on their opponent's rank before using information about their own rank, which, on the proximate level, does not seem very likely.

If individuals use information about their own social rank before they use information about their opponent's rank (pathways along levels 2, 3^a and 4^a), there is a dichotomy between dominance and alternating strategies. This dichotomy occurs already in the first transition along the pathway, implying that already very simple strategies allow for dominance conventions. Moreover, these considerations suggest that, although there are many different equilibria at information level 5 (all information is used), only two of these equilibria (D_4 and A_1) seem relevant as possible outcomes of long term biological evolution.

ASYMMETRIES IN RESOURCE HOLDING POTENTIAL

The equilibria of level 2 (decisions are based on the relation with the current opponent), 3^a (decisions are based on one's own rank) and 3^b (decisions are based on opponent's rank) are reached with equal probability from the mixed strategy ESS of level 1 (no information is used). This is because the mixed equilibrium of level 1 is exactly located at the boundary separating the initial conditions from which the alternative equilibria of levels 2, 3^a and 3^b are reached (shown for the transition from level 1 to level 3^a in FIGURE 4). This feature disappears as soon as there are RHP asymmetries between the players (FIGURE 5). Due to these RHP asymmetries, the boundary plane between the domains of attraction of the dominance and alternating equilibrium shifts slightly. The mixed equilibrium of level 1 is no longer on the border between the two domains of attraction, but in the interior of the domain

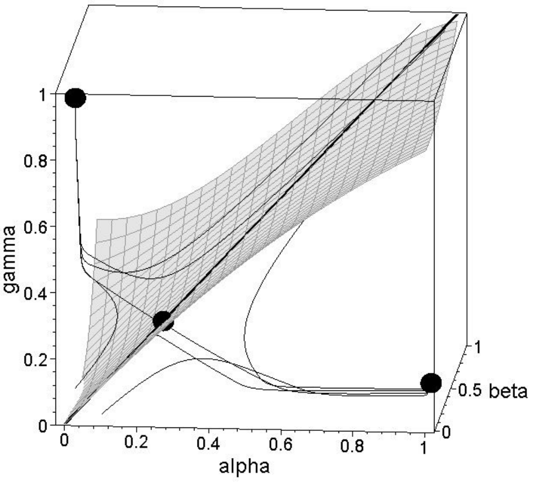


FIGURE 4 – THE DICHOTOMY BETWEEN DOMINANCE STRATEGIES AND OTHER CONVENTIONS

At information level 3^a, individuals base their decision on their own rank. Consequently, there are three strategic variables (the probability of playing Hawk when in rank α, β and γ, respectively), which are represented by the three axes of the plot. At information level 1, individuals use no information, and only the overall tendency of playing Hawk can change through evolution. Therefore, if constrained to information level 1, evolution will proceed along the thick black diagonal towards the mixed strategy equilibrium of level 1 (equilibria are represented by black spheres). However, the mixed strategy equilibrium is unstable with respect to movement away from the diagonal, so after a transition to level 3^a (where individuals behave differently depending on their own rank), evolution converges to either the dominance (lower right) or alternating equilibrium (upper left). Since the mixed equilibrium of level 1 is exactly located on the plane separating the domains of attraction of the stable equilibria of information level 3^a, both equilibria are attained with equal probability. The thin black lines represent deterministic evolutionary trajectories of the model at information level 3^a. Parameters as in FIGURE 2.

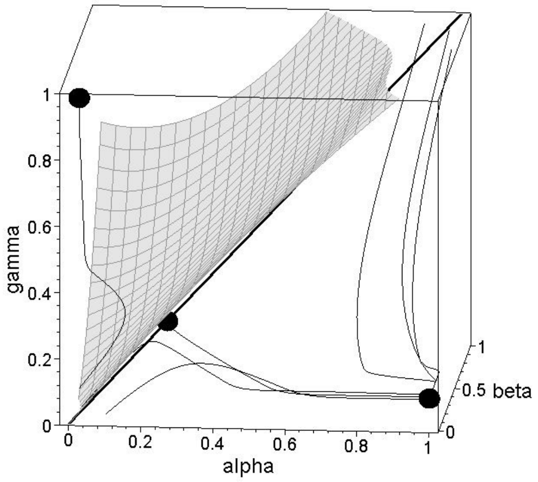


FIGURE 5 – EFFECTS OF DIFFERENCES IN RESOURCE HOLDING POTENTIAL

This figure is identical to FIGURE 4, except for the fact that this figure is based on an extended version of our model in which hidden RHP asymmetries between individuals were included. To be precise, we assumed that there were two equally frequent RHP classes, representing strong and weak individuals. In an escalated conflict between a strong and a weak individual, the strong individual had a high probability of winning the conflict (87.5%). Due to these RHP asymmetries, the boundary plane between the domains of attraction of the stable equilibria of information level 3^a (decisions based on own rank) has shifted slightly, such that the mixed equilibrium of information level 1 (no information is used) is now in the interior of the domain of attraction of the dominance equilibrium. Formally, this implies that further evolution from the equilibrium of information level 1 will always converge to the dominance equilibrium, as shown by the deterministic trajectory leading from the mixed equilibrium to the dominance equilibrium.

of attraction of the dominance equilibrium. In our deterministic model, the alternating equilibrium can now no longer be reached from the mixed equilibrium of level 1. However, even with large RHP asymmetries (as in FIGURE 5), the mixed equilibrium of level 1 is still very close to the border between the two domains of attraction. Due to stochastic fluctuations, which are likely to be present under natural conditions, the alternating equilibrium may therefore still be attainable in practice.

INTERACTIONS IN LARGER GROUPS AND THE EFFECTS OF ERRORS IN INDIVIDUAL RECOGNITION

Up to here, we have restricted ourselves to a fixed group size of three individuals and assumed that players had complete information about the outcome of previous conflicts. We refrain from relaxing these assumptions within our deterministic model framework, but instead, we use stochastic individual based simulations to extend our model to arbitrary group size and to check the validity of our results with respect to the assumption that the players can accurately remember the outcome of previous conflicts between all players in the group.

There are different ways in which the three-player model can be generalized to arbitrary group sizes. We choose an option that deviates as little as possible from the original model. We assume that an individual bases its decision in a conflict on (1) its relation with its current opponent, (2) its relation with *one* bystander, which is randomly selected from the other group members, and (3) the relation between its opponent and a (potentially different) bystander, which is also randomly selected from the other group members. Individuals can now find themselves in eight qualitatively different social situations, which can be interpreted exactly as in the original three-player model. The different information levels of the three-player model can similarly be translated directly to the model with arbitrary group size. We varied the amount of social information that could be accurately remembered by an individual by changing the likelihood of errors in individual recognition. Individuals could either mistake their opponent or the bystander for an arbitrary other individual in the group.

FIGURE 6 shows the results of two individual based simulations, which differed only in the seed used to initialize the random number generator. In these simulations, groups consisted of five individuals, and errors in individual recognition were quite frequent (50% of the cases). As can be seen from FIGURE 6, there are two different evolutionary equilibria. The equilibrium that is attained in the simulation represented in the upper panel is a dominance equilibrium: the α-individual almost always plays Hawk, the β-individual usually plays Hawk against the γ-individual, but hardly ever against the α-individual, and the γ-individual never plays Hawk. The simulation shown in the lower panel of FIGURE 6 converges to an equilibrium that corresponds to an alternating equilibrium of the three-player model: this time the γ-individual is the most likely to play Hawk, leading to unstable social configurations.

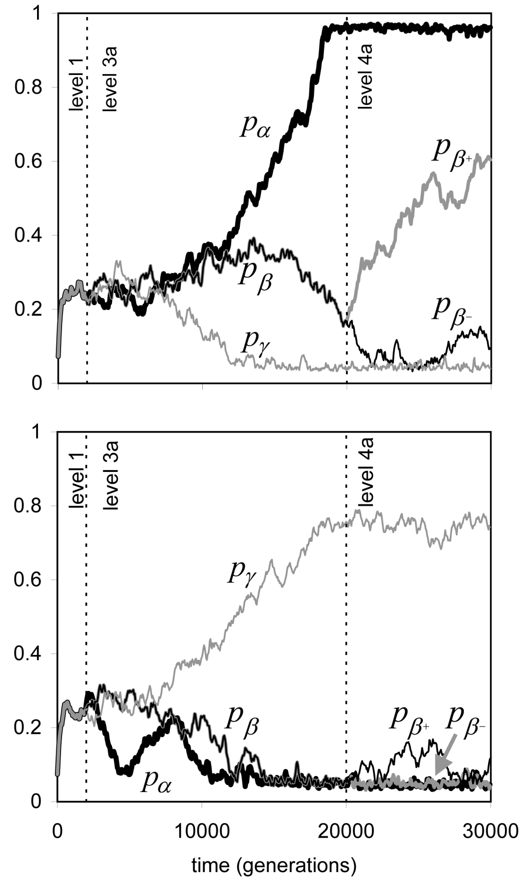


FIGURE 6 – INDIVIDUAL BASED SIMULATIONS

Individual based simulations (see the APPENDIX for details) were used to extend the model to arbitrary group sizes and to vary our assumptions on the quality of individual recognition. We simulated a population of 50 groups, each consisting of 5 individuals. Errors in individual recognition occurred frequently (individuals made mistakes in 50% of the cases). Transitions to higher information levels occurred at generation 2000 and 20000. The two panels of the figure show the values of the strategic parameters in two replicate simulations, differing only in the seed used to initialize the random number generator. In the upper panel, evolution leads to a dominance strategy, in which higher-ranking individuals behave more aggressively. In the lower panel, the outcome of evolution is an alternating strategy, in which the lowest ranking individuals are most aggressive. The labels shown in the plots denote groups of constrained strategic parameters: $p_\alpha \triangleq p_{\alpha\beta} = p_{\alpha\gamma}$, $p_\beta \triangleq p_{\beta\alpha} = p_{\beta\gamma} = p_{\beta^+\beta^-} = p_{\beta^-\beta^+}$, $p_\gamma \triangleq p_{\gamma\alpha} = p_{\gamma\beta}$, $p_{\beta^+} \triangleq p_{\beta\gamma} = p_{\beta^+\beta^-}$, $p_{\beta^-} \triangleq p_{\beta\alpha} = p_{\beta^-\beta^+}$. Payoffs and other parameters were as in FIGURE 2.

These results, together with the other simulations we performed, indicate that the presence of multiple qualitatively different conflict resolution conventions is a robust phenomenon. For all combinations of group size (4,5,8,10) and probabilities of errors in individual recognition (0%, 25%, 50%, 100%) tested, we found dominance and alternating equilibria. The analogues of triangular strategies, in which all individuals have exactly the same social rank, never evolved. This is because the maintenance of such maximally intransitive hierarchies in larger groups requires complete social information. In our simulation model, complete social information is unattainable by definition, since the relation with only one of the bystanders is considered in a conflict decision. Replicates of the simulations shown in FIGURE 6 moreover indicate that the alternative conventions are reached with equal probability from the mixed equilibrium of level 1 (8 out of 20 replicates converged to the dominance convention). Inclusion of hidden RHP-differences between individuals (as in FIGURE 5) biases the outcome towards convergence to the dominance convention (18 out of 20 replicates, data not shown).

DISCUSSION

The outcome of conflicts between individuals in a social group automatically generates historical asymmetries between individuals. Such asymmetries may either pertain to previous conflicts between a focal individual and its opponent, or to previous conflicts with other group members. For example, in a social group in which a linear hierarchy has been established, there are at least two qualitatively different asymmetries between the highest- and lowest-ranking individual. First, there is a direct asymmetry: the highest-ranking individual is obviously dominant over the lowest-ranking one. Second, there is an indirect asymmetry: the highest-ranking individual is dominant over other group members, which, in their turn, are dominant over the lowest-ranking individual. Our results show that both these direct and indirect asymmetries can be used as cues for conventional conflict resolution, leading to within- and between-pair winner and loser effects, respectively. Within-pair winner and loser effects lead to rank differentiation within pairs of individuals, between-pair winner and loser effects lead to the ordering of social relation within the group into a transitive hierarchy. Winner and loser effects may evolve even when the historical asymmetries, generated by the outcomes of previous conflicts between individuals, hold no information about differences in resource holding potential.

Apart from strategies that give rise to transitive dominance hierarchies, our analysis reveals that there are other possible evolutionarily stable conflict resolution strategies. Contrary to the dominance strategies, which are characterized by within- and/or between-pair winner and loser effects, these alternating strategies are comparable to the paradoxical strategies described by Maynard Smith (1982), in the sense that the loser rather than the winner of previous fights is most likely to escalate. This leads to a constantly changing, egalitarian social configuration. In addition to the alternating strategies, evolution may lead to strategies that lock onto an intransitive social configuration that gives equal payoff to all group members (triangular strategies). The occurrence of both alternating and triangular strategies, next to the 'common-sense' dominance strategies is in accordance with game theoretical results, which state that any asymmetry between players (a) must be used for conventional conflict settlement, and (b) can be used in both a paradoxical and common-sense way (Maynard Smith & Parker, 1976; Hammerstein, 1981; Selten, 1980; see also the discussion of CHAPTER 10 of this thesis.

The triangular strategies do not seem relevant within the context of biological evolution, since they are sensitive to errors in individual recognition and can only evolve when individuals have access to complete social information. The alternating strategies, however, are robust against errors in individual recognition. They can evolve even when individuals have access to only limited social information. In line with the results of previous game theoretical models, paradoxical strategies (alternating and triangular) are less likely to evolve than the common-sense strategies (dominance) if there are underlying RHP asymmetries (Hammerstein, 1981).

However, the bias towards evolution of dominance strategies is small, even when the underlying RHP asymmetries are large. The latter finding may change considerably as soon as individuals base their decisions in conflicts not on a single previous conflict (as we assumed for simplicity) but on a large number of previous interactions. The same may be true when the probabilities of interaction between individuals are not fixed (as assumed in our model) but modified by spatial self-structuring (Hemelrijk, 2000).

Together with CHAPTER 10 of this thesis, the present study provides a proof of principle that evolutionarily stable dominance relationships need not necessarily reflect intrinsic differences between individuals, such as RHP asymmetries, but that they may result from arbitrary historical asymmetries. The acknowledgement of the potential of social conventions, which could, in principle, be based on quite arbitrary asymmetries, can help to understand several aspects of social dominance that are difficult to explain with an approach focusing only on intrinsic differences between individuals. Yet, for a full understanding of social dominance and social hierarchy formation these two approaches should not be opposed to one another but combined. In this, and the preceding chapter of this thesis, we made only a small step towards this end. Certainly more work is needed to fully integrate social dominance conventions relating to intrinsic differences between individuals, such as direct assessment of the opponent's RHP, and the conventions relating to arbitrary asymmetries, such as winner and loser effects.

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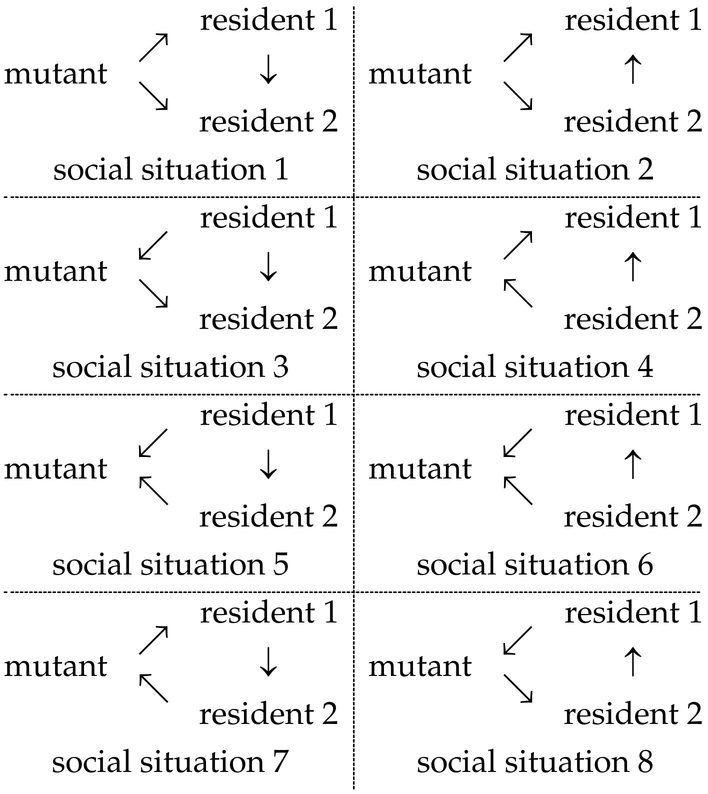
APPENDIX — METHODS FOR MATHEMATICAL ANALYSIS AND INDIVIDUAL BASED SIMULATIONS

THE PAYOFF FUNCTION

The average expected payoff $W(\vec{q},\vec{p})$ of a rare mutant playing strategy $\vec{q}$ against resident individuals that play strategy $\vec{p}$ can be derived from the transition probabilities between the different situations in which the mutant individual can find itself. Every mutant individual is in a group with two resident individuals. From here on, we will arbitrarily label the resident individuals as 'resident 1' and 'resident 2', and we will define the eight possible social situations as follows



with the arrows pointing towards the loser of the previous fight.

We may now compute the transition matrix $\mathbf{M}(\vec{q},\vec{p})$, the elements ($m_{i,j}$) of which give the transition probability from social situation j to social situation i ($i,j=1\ldots8$). For example, a transition from social situation 1 to social situation 3 occurs when there is a conflict between mutant and resident 1 (which occurs with probability $1/3$) and when the mutant loses this conflict. The latter may occur with probability $1/2$ after both mutant and resident 1 play Hawk, or after the mutant plays Dove and resident 1 plays Hawk, or with probability $1/2$ after both mutant and resident play Dove. In social situation 1, the mutant is the α-individual, whereas its opponent is the β-individual. Therefore, the mutant plays Hawk with probability $q_{\alpha\beta}$, and resident 1 plays Hawk with probability $p_{\beta\alpha}$. Consequently, we find

$$m_{3,1}=\frac{1}{3}\left(\frac{1}{2}p_{\beta\alpha}q_{\alpha\beta}+p_{\beta\alpha}\left(1-q_{\alpha\beta}\right)+\frac{1}{2}\left(1-p_{\beta\alpha}\right)\left(1-q_{\alpha\beta}\right)\right). \qquad [7]$$

In the same way, the transition probability from social situation 1 to social situation 2 is given by

$$m_{2,1}=\frac{1}{3}\left(\frac{1}{2}p_{\gamma\beta}p_{\beta\gamma}+p_{\gamma\beta}\left(1-p_{\beta\gamma}\right)+\frac{1}{2}\left(1-p_{\gamma\beta}\right)\left(1-p_{\beta\gamma}\right)\right). \qquad [8]$$

Note that this transition probability is independent of the mutant strategy, since a transition from social situation 1 to 2 occurs only after a conflict between the two resident individuals.

After having computed the other elements of $\mathbf{M}(\vec{q},\vec{p})$ in a similar way, we also need to calculate the expected costs $\vec{c}(\vec{q},\vec{p})$ and benefits $\vec{b}(\vec{q},\vec{p})$ (to the mutant) associated with every social situation. For example, in social situation 1, the expected benefit from the previous conflict is $2/3\,V$. This is because the mutant was involved in the previous conflict with probability $2/3$ (it was a mere bystander in $1/3$ of the cases). However, if the mutant was involved in the last conflict, it certainly obtained the resource (corresponding to a benefit V). This follows from the fact that the mutant is the α-individual in social situation 1 and, hence, gained the resource in its last conflict with both other group members. In the other social situations the benefits to the mutant are as follows

$$\vec{b}(\vec{q},\vec{p})=\begin{pmatrix}\frac{2}{3}V & \frac{2}{3}V & \frac{1}{3}V & \frac{1}{3}V & 0 & 0 & \frac{1}{3}V & \frac{1}{3}V\end{pmatrix}. \qquad [9]$$

The expected costs $\vec{c}(\vec{q},\vec{p})$ in a given social situation pertain to the expected costs of the *next* conflict. The next conflict may occur between the mutant and resident 1, between the mutant and resident 2, or between the two residents. In the latter cases, the mutant incurs no costs. In the former two cases, there are costs if the mutant plays Hawk and when the conflict escalates and the mutant loses. To be precise, the expected cost $\overline{c}(q,p)$ to a mutant that plays Hawk with probability q in a conflict with another individual playing Hawk with probability p is $\overline{c}(q,p)=1/2\,pqC+qD$. Averaging over all possible conflicts that may occur in a social situation, we find

$$\vec{c}(\vec{q},\vec{p})=\frac{1}{3}\begin{pmatrix}\overline{c}(q_{\alpha\beta},p_{\beta\alpha})+\overline{c}(q_{\alpha\gamma},p_{\gamma\alpha})\\ \overline{c}(q_{\alpha\beta},p_{\beta\alpha})+\overline{c}(q_{\alpha\gamma},p_{\gamma\alpha})\\ \overline{c}(q_{\beta\alpha},p_{\alpha\beta})+\overline{c}(q_{\beta\gamma},p_{\gamma\beta})\\ \overline{c}(q_{\beta\alpha},p_{\alpha\beta})+\overline{c}(q_{\beta\gamma},p_{\gamma\beta})\\ \overline{c}(q_{\gamma\alpha},p_{\alpha\gamma})+\overline{c}(q_{\gamma\beta},p_{\beta\gamma})\\ \overline{c}(q_{\gamma\alpha},p_{\alpha\gamma})+\overline{c}(q_{\gamma\beta},p_{\beta\gamma})\\ \overline{c}(q_{\beta^+\beta^-},p_{\beta^-\beta^+})+\overline{c}(q_{\beta^-\beta^+},p_{\beta^+\beta^-})\\ \overline{c}(q_{\beta^+\beta^-},p_{\beta^-\beta^+})+\overline{c}(q_{\beta^-\beta^+},p_{\beta^+\beta^-})\end{pmatrix}^{\mathrm{T}}. \qquad [10]$$

Next, we define vectors $\vec{u}_n(\vec{q},\vec{p})$, which contain the probabilities that the mutant finds itself in each of the eight possible social situations in the n-th conflict. The vectors $\vec{u}_n(\vec{q},\vec{p})$ satisfy

$$\vec{u}_n(\vec{q},\vec{p})=\mathbf{M}(\vec{q},\vec{p})\,\vec{u}_{n-1}(\vec{q},\vec{p}). \qquad [11]$$

We assume that the players start in an intransitive hierarchy. Hence,

$$\vec{u}_0(\vec{q},\vec{p})=\begin{pmatrix}0 & 0 & 0 & 0 & 0 & 0 & \frac{1}{2} & \frac{1}{2}\end{pmatrix}^{\mathrm{T}}. \qquad [12]$$

Equations [11] and [12] together uniquely determine the sequence $\vec{u}_0(\vec{q},\vec{p})$, $\vec{u}_1(\vec{q},\vec{p})$, $\vec{u}_2(\vec{q},\vec{p}),\ldots$ which determines the average expected payoff, $W(\vec{q},\vec{p})$, of an individual playing strategy $\vec{q}$ against opponents playing strategy $\vec{p}$.

In order to find $W(\vec{q},\vec{p})$, we must first calculate the expected payoff $w_n(\vec{q},\vec{p})$ to the mutant in the n-th conflict. This quantity is given by

$$w_n(\vec{q},\vec{p}) = \vec{b}(\vec{q},\vec{p})\,\vec{u}_n(\vec{q},\vec{p}) - \vec{c}(\vec{q},\vec{p})\,\vec{u}_{n-1}(\vec{q},\vec{p}). \qquad [13]$$

The first term in equation [13] measures the expected benefit to the mutant in the n-th conflict. The second term is the expected cost to the mutant incurred in reaching the current social situation from the $(n-1)$-th conflict.

Under the assumption that every pair of individuals interact T times on average, the average expected payoff $W(\vec{q},\vec{p})$ can now be calculated as

$$W(\vec{q},\vec{p}) = \frac{1}{3T}\sum_{n=1}^{\infty}\left(1-\frac{1}{3T}\right)^{n-1} w_n(\vec{q},\vec{p}). \qquad [14]$$

The factor $\left(1-1/(3T)\right)^{n-1}$ is necessary to weigh the expected payoff of the n-th conflict with the probability that this conflict will actually occur.

EVOLUTIONARY DYNAMICS

Under the assumption that evolution proceeds in small steps at a rate and in the direction determined by the magnitude and sign of the selection gradient (Hofbauer & Sigmund, 1998, chapter 9), the evolution of the strategy $\vec{p}$ can be described by

$$\frac{\partial\vec{p}}{\partial t} = \kappa\,\mathbf{G}\left.\frac{\partial W(\vec{q},\vec{p})}{\partial\vec{q}}\right|_{\vec{q}=\vec{p}}. \qquad [15]$$

In this equation, the rate constant κ depends on the population size and the rate of mutations. The matrix $\mathbf{G}$ is a mutational variance-covariance matrix, which we used to implement the constraints corresponding to the different information levels, as explained in CHAPTER 10 of this thesis. We imposed that all strategic parameters are within the range $[\delta, 1-\delta]$, in order to exclude evolution towards equilibrium strategies that are sensitive to occasional errors in decision-making ('trembling hand' approach, Selten, 1975). Throughout this paper, we took $\delta = 0.025$.

INDIVIDUAL-BASED SIMULATIONS

In the individual-based simulations, individuals were distributed at the start of every generation (generations were discrete) into N groups, each consisting of G individuals. Individuals then engaged in repeated Hawk-Dove interactions with other individuals from their group. On average, every pair of individuals interacted T times. At the end of every generation, individuals from all groups were collected in one big mating pool. Offspring was generated by sexual reproduction, and the number of offspring produced by an individual was proportional to the total payoff gained in interactions throughout its lifetime. We furthermore assumed that the strategic parameters of an individual's conflict resolution strategy were each determined by a diploid locus. We assumed normal Mendelian inheritance, free recombination between loci and additive interactions between alleles. Mutations, altering the phenotypic effect of an allele slightly (by 1%) occurred at a low frequency (1% per allele per generation).

Dutch Part

Hoofdstukken in het Nederlands

NATUURLIJKE SELECTIE ONDER DE LOEP

Natuurlijke selectie is één van de centrale begrippen in de evolutie-theorie. Desondanks is er verwarring over wat natuurlijke selectie nu eigenlijk selecteert. Zijn dat eigenschappen die goed zijn voor de soort, voor de groep, voor het individu of voor iets anders misschien? De onduidelijk onstaat daaruit dat natuurlijke selectie niet één enkel proces is, maar bestaat uit allerlei deel-processen die met elkaar interacteren en ook nog eens op verschillende niveaus blijken te werken. Hoog tijd dus om eens in te zoomen op de complexiteit van natuurlijke selectie...

Zappend langs de verschillende kanalen van mijn TV kom ik terecht bij een natuur-documentaire. Ik zie beelden van de Afrikaanse savanne met voorbijtrekkende kuddes zebra's. Een commentaarstem attendeert mij op het feit dat een leeuwin de jacht heeft geopend op één van de zebra's: een oude, zieke zielepoot. Dat doet ze, zo hoor ik terwijl ik de leeuwin haar eerste hap van de inmiddels gedode zebra zie verorberen, om de zebra-populatie gezond te houden. De commentaarstem legt uit: "De leeuwen doden de oude en zwakke zebra's het eerst. Hierdoor verdwijnen slechte genetische eigenschappen uit de zebra populatie. Dat komt niet alleen de zebra's maar uiteindelijk ook de leeuwen ten goede, want zo verzekeren de leeuwen zich van gezonde en voedzame prooidieren. De natuur zit mooi in elkaar !".

De rest van de natuurfilm heb ik maar bekeken met het geluid uit. Ook voor wie geen kaas heeft gegeten van evolutiebiologie is het hopelijk duidelijk dat het commentaar bij de documentaire berust op onzin met een wetenschappelijk sausje. Natuurlijk kiest de leeuwin de oude en zwakke dieren. Maar dat doet ze niet omdat ze daardoor het natuurlijk evenwicht bewaart, maar omdat de oude en zwakke zebra's nu eenmaal veel makkelijker te vangen zijn. Ze zou precies hetzelfde doen als het voor de zebra-populatie om de een of andere reden nadelig was de oude en zwakke dieren te doden.

Ook al is het vaak makkelijk de fout aan te wijzen in redeneringen die eigenschappen en gedrag van organismen verklaren vanuit 'wat goed is voor het natuurlijk evenwicht' of 'goed voor het overleven van de soort', toch zijn dit soort redeneringen hardnekkig aanwezig in het biologisch gedachtengoed van het grote publiek. Dit is niet zo vreemd, want het idee van selectie op ecosysteem- of soortniveau past goed bij het aantrekkelijke beeld van een harmonieuze natuur waarin individuen samenwerken ten behoeve van het grotere geheel. Het idee dat eigenschappen van organismen verklaard worden vanuit 'wat goed is voor het individu zelf', met andere woorden dat evolutie selecteert op individueel niveau, schetst een veel minder aanlokkelijk beeld van de natuur: een beeld van competitie en egoïsme in plaats van samenwerking en opofferingsgezindheid.

12

Lastige problemen binnen de evolutie theorie

G. Sander van Doorn

HET ECB

Dit hoofdstuk bestaat uit een aantal korte artikelen die geschreven zijn in samenwerking met het Expertise Centrum Biologie (ECB). Het ECB is een initiatief van het Nederlands Instituut voor Biologie, gericht op het bevorderen van het contact tussen wetenschap en maatschappij. Met het oog daarop publiceert het ECB via www.kennislink.nl vrij toegankelijke artikelen voor scholieren en andere geïnteresseerden. Mijn bijdrage aan het ECB bestaat uit vijf artikelen die elk afzonderlijk een openstaand probleem in de evolutietheorie behandelen. Deze artikelen hebben direct of indirect met mijn eigen onderzoek te maken, en ze geven daardoor ook een overzicht van mijn promotieonderzoek.

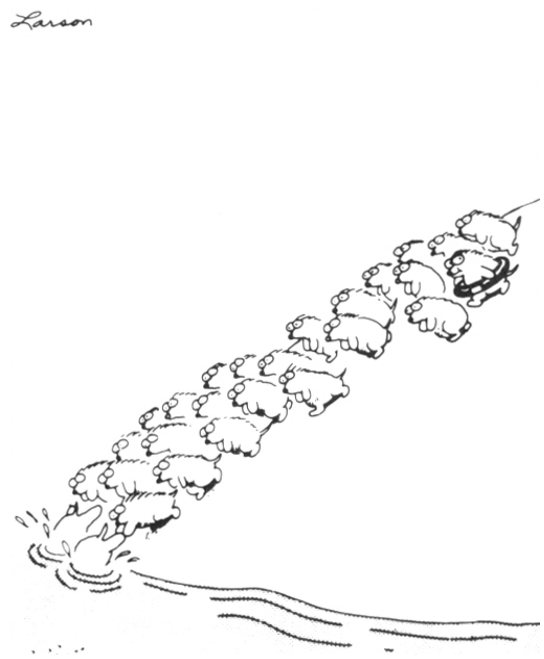
HET PROBLEEM VAN *GROEP-SELECTIE*

Waarom kan ik zo stellig het commentaar uit de natuur-documentaire als onzin bestempelen? Wat is er eigenlijk mis met die verklaring, of, wat is er beter aan een verklaring die redeneert vanuit optimaliteit op individueel niveau? Ook binnen de biologie wordt nog af en toe gebakkeleid over deze vragen. Tegenwoordig zijn de meeste evolutiebiologen overtuigd van het feit dat selectie vooral begrepen moet worden op individueel niveau. Maar nog niet zo lang geleden verklaarde men allerlei gedrag van dieren vanuit wat goed leek voor het overleven van de soort, zoals de prooikeuze van de leeuwin uit de natuurserie werd verklaard als een manier waarop het natuurlijk evenwicht blijft bewaard. Om nog een ander voorbeeld te geven, het gedrag van stekelbaarsmannetjes, die soms een deel van de eieren in hun nest opeten, werd verklaard als een manier om te voorkomen dat de populatie te hard groeit, waardoor sterke concurrentie om voedsel zou onstaan.

In 1962 publiceerde V. C. Wynne-Edwards een invloedrijk boek (Wynne-Edwards, 1962) waarin het idee van selectie voor eigenschappen die goed zijn voor de soort, werd uitgewerkt. Hij stelde in zijn boek dat veel aspecten van diergedrag erop gericht zijn de populatiegrootte beperkt te houden, zodat er geen voedseltekort ontstaat. Alleen soorten met dit soort groeps-regulerende mechanismen zouden levensvatbaar zijn op de lange termijn; soorten zonder regulatie mechanismen zouden al snel hun voedselbronnen uitputten en vervolgens uitsterven. Wynne-Edwards verondersteld een sterke competitie tussen populaties. Populaties waarin de individuen zich opofferen ten gunste van de populatie als geheel zijn in het voordeel ten opzichte van populaties waarin individuen zichzelf niet opofferen. Met andere woorden: selectie werkt op basis van verschillen tussen groepen (vandaar dat dit proces *groep-selectie* wordt genoemd).

Wat Wynne-Edwards zich onvoldoend realiseerde is dat selectie niet alleen kan werken op verschillen tussen groepen individuen, maar ook op verschillen binnen een groep individuen. Vier jaar na het verschijnen van het boek van Wynne-Edwards publiceerde G. C. Williams een boek (Williams, 1966) waarin hij uiteenzette dat juist die verschillen tussen individuen een doorslaggevende rol spelen. Met andere woorden, evolutie vindt voornamelijk plaats door selectie op het niveau van het individu en niet door groep-selectie. Williams' argument gaat als volgt. Stel je een populatie voor waarin de individuen bereid zijn zichzelf op te offeren als dat de populatie als geheel ten goede komt. Denk hierbij bijvoorbeeld aan een populatie lemmingen, waarvan de individuen zichzelf bij voedseltekort opofferen door zich in zee te werpen. Volgens de groep-selectie theorie zouden genetische eigenschappen die ten grondslag liggen aan zulk zelfmoord gedrag geselecteerd worden, omdat groepen met dit regulatie mechanisme een hogere overlevingskans hebben. Stel je nu voor dat er binnen een populatie genetische variatie bestaat tussen de lemmingen. Sommige lemmingen zijn sterk geneigd zich op te offeren, anderen zijn dat minder (zie de cartoon van Gary Larson, FIGUUR 1).

Welk van deze twee types lemmingen heeft de hoogste kans zijn genetisch materiaal door te geven aan de volgende generatie? Het antwoord op deze vraag staat haaks op de groep-selectionistische voorspelling dat bereidheid tot zelfmoord geselecteerd zal worden.



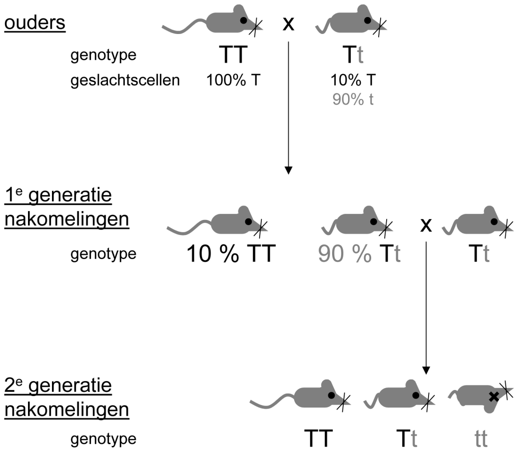
FIGUUR 1 – OPOFFERINGSGEZINDHEID VERSUS ZELFZUCHTIG GEDRAG

Deze cartoon van Gary Larson verbeeldt de zwakke plek van groep-selectionistische argumenten, namelijk, het selectieve voordeel dat zelfzuchtige mutanten hebben ten opzichte van hun opofferingsgezinde soortgenoten.

De lemmingen uit het voorbeeld maken meer algemeen duidelijk dat de opofferingsgezindheid, die we op grond van groep-selectie argumenten verwachten, vaak niet de uitkomst van evolutie zal zijn. De reden daarvoor is dat selectie op het niveau van het individu 'bedriegers' begunstigt, die wel de lusten van de opofferingsgezindheid genieten maar de lasten ontlopen. Met name in gevallen waarin de belangen van het individu conflicteren met het groepsbelang kunnen er dan ook grote verschillen ontstaan tussen de werkelijke uitkomst van evolutie en de uitkomst die we op grond van groep-selectionistische argumenten zouden verwachten.

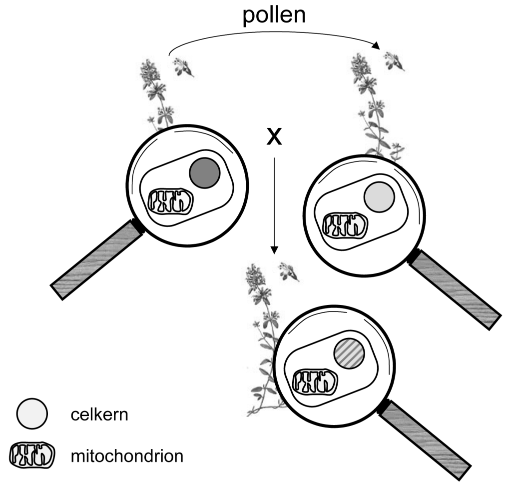
VERSCHILLENDE NIVEAUS VAN SELECTIE

Merk op dat de kern van Williams' argument niet is dat groep-selectie niet bestaat, maar dat selectie op groep niveau meestal veel zwakker zal zijn dan selectie op niveau van het individu. Er kunnen eigenschappen ontstaan die nog zo goed zijn voor de groep, als het de individuen binnen de groep lukt voordeel te behalen ten opzichte van hun groepsgenoten, dan zullen ze dat niet nalaten. Zelfs niet als dat ten koste gaat van de groep als geheel. Op dezelfde manier is het niet onmogelijk dat er selectie bestaat op nog hogere niveaus dan dat van de groep, zoals op soorts- of ecosysteem niveau (denk aan het commentaar bij de natuurserie) maar die selectie zal op haar beurt nog zwakker zijn.



FIGUUR 2 – HET *T*-COMPLEX VAN DE HUISMUIS

In natuurlijke huismuispopulaties komt het zogenaamde *t*-complex voor. Het *t*-complex bestaat uit een aantal nauw gekoppelde genen die een manier gevonden hebben om de eerlijke verdeling van de chromosomen tijdens de meiose te ontduiken. Individuen die een *t*-allel bezitten, zijn makkelijk van wildtype individuen te onderscheiden. Door de interactie met een ander gen hebben *t*-dragers een kortere staart. Als een wildtype vrouwtje (*TT*) paart met een mannetje dat een *t*-allel draagt, dan draagt een aanzienlijk groter deel van hun nakomeling dan de verwachte 50% het *t*-allel. Dat komt waarschijnlijk doordat zaadcellen die het *t*-allel dragen de wildtype zaadcellen uit de weg ruimen. Het *t*-allel heeft op gen-niveau dus een duidelijk voordeel ten opzichte van het wildtype allel *T*. Op individueel niveau is dat zeker niet het geval. Zodra het *t*-allel in frequentie toeneemt, ontstaan er met grotere waarschijnlijkheid paartjes waarvan beide partners het *t*-allel dragen. Die paartjes produceren zoons die homozygoot zijn voor het *t*-allel. In veel gevallen zijn die individuen steriel of niet levensvatbaar.



FIGUUR 3 – MANNELIJKE STERILITEIT IN ECHTE TIJM (*THYMUS VULGARIS*)

Als twee planten kruisen is het DNA in de celkern van de nakomelingen voor de helft afkomstig van de vader (de plant die het pollen produceerde) en voor de andere helft afkomstig van de moeder (de plant waarop het pollen door bestuiving terecht kwam). Voor allerlei componenten van het cytoplasma geldt dit niet. Dat is niet zonder betekenis, want verschillende celorganellen, zoals de mitochondriën en de chloroplasten bevatten ook DNA. Zoals in de meerderheid van soorten zijn in Echte tijm (*Thymus vulgaris*) de mitochondriën afkomstig van de moeder. Voor een gen dat zich bevindt in een mitochondrion is de productie van pollen daarom niet zo interessant. Sterker nog, het zou in het voordeel van zo'n gen zijn als de pollenproductie zou stoppen, en de energie die daaraan werd besteed geïnvesteerd zou worden in de productie van zaadbeginsels. Meer zaadbeginsels betekenen voor een mitochondrion immers een hogere kans op representatie in de volgende generatie. Inderdaad is in Echte Tijm een mitochondriale factor ontstaan die mannelijke steriliteit veroorzaakt: dragers van deze eigenschap produceren geen pollen, maar wel zaadbeginsels. Dat is gunstig voor het mitochondrion, maar slecht voor het individu want de afname in pollenproductie wordt niet gecompenseerd door de toename van de productie van vrouwelijke gameten. Dat is waarschijnlijk ook de reden waarom er op het nucleaire DNA suppressor-genen zijn geëvolueerd die de effecten van de mitochondriale muiterij onderdukken.

Zoals soorten bestaan uit populaties en populaties uit individuen, zo bestaan individuen uit cellen, en binnen cellen bevinden zich chromosomen waarop zich genen bevinden. Je zou je terecht kunnen afvragen of er dan ook selectie op celniveau, chromosoom-niveau en gen-niveau is. En zo ja, verwachten we dan niet dat de selectie op die niveaus weer veel sterker zal zijn dan selectie op individueel niveau?

Dat selectie op lagere niveaus dan het individuele wel degelijk bestaat en bovendien potentieel sterk is, wordt duidelijk aan de hand van een groot aantal voorbeelden. Ik noem er hier twee: het *t*-complex van de huismuis (FIGUUR 2) en mannelijke steriliteit in Echte Tijm (*Thymus vulgaris*) (FIGUUR 3). Deze twee voorbeelden laten zien dat selectie op lager gelegen niveaus de neiging zal hebben de integriteit en samenwerking op hogere niveaus te verstoren. Het is dan ook maar goed dat onder normale omstandigheden selectie op niveau van het individu het wint van selectie op de onderliggende niveaus.

Ietwat abstracter kunnen we stellen dat natuurlijke selectie werkzaam is op alle organisatie niveaus binnen de levende natuur, van het moleculair genetische niveau tot het soortsniveau, en met wat goede wil zelfs tot het ecologische niveau. Welk van die verschillende niveaus het belangrijkst is in de praktijk wordt bepaald door door drie factoren:

Genetische variatie. Natuurlijke selectie kan alleen werken als er verschillen zijn tussen de eenheden waarop selectie werkt (dat kunnen bijvoorbeeld allelen, cellen, individuen of populaties zijn) binnen de verschillende niveaus.

Verschillen in voortplantingssucces. Niet alleen moeten de eenheden verschillen, die verschillen moeten zich ook kunnen vertalen in verschillen in voortplantingssucces.

Erfelijkheid. Er moet een relatie bestaan tussen de genetische eigenschappen van de ouders en die van de nakomelingen.

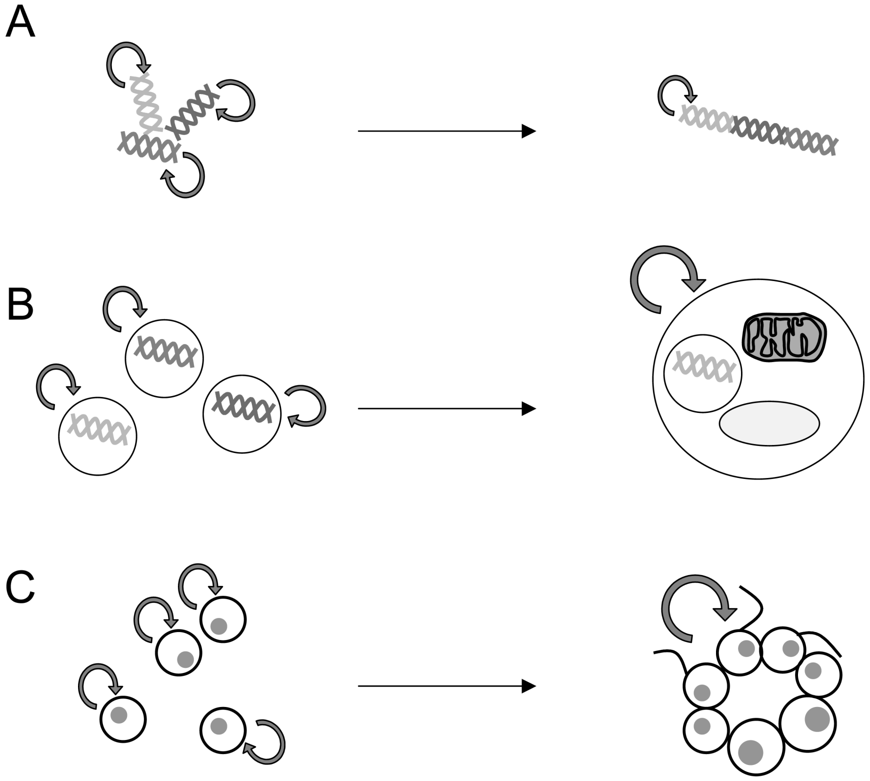
We kunnen nu begrijpen waarom onder normale omstandigheden in meercellige organismen de samenwerking tussen de cellen niet verstoord wordt door selectie op niveaus lager dan het individuele niveau: de cellen van het organisme zijn genetisch identiek (er is geen genetische variatie tussen de cellen), de cellen kunnen zich bovendien niet onafhankelijk voortplanten (de celdeling wordt van bovenaf gereguleerd) en bij de voortplanting van het individu worden de chromosomen tijdens de meiose eerlijk verdeeld over de geslachtscellen. Andersom kunnen we nu ook begrijpen hoe conflicten tussen selectie op niveau van het individu en selectie op lager gelegen niveaus de werking van het meercellig organisme kunnen verstoren (zie ook de voorbeelden muis en tijm): verschillende soorten kanker ontstaan door mutaties (genetische variatie!) als gevolg waarvan de kankercellen zich onttrekken aan de van bovenaf opgelegde controle van de celdeling (er ontstaan verschillen in voortplantingssucces). Het *t*-complex van de huismuis wordt niet weggeselecteerd doordat het *t*-allel de eerlijke verdeling van chromosomen tijdens de meiose ontduikt (wat opnieuw een verschil in voortplantingssucces mogelijk maakt).

Dat selectie op hogere niveaus dan het individuele niveau meestal verwaarloosbaar is, volgt uit het feit dat er gewoonlijk meer genetische variatie is binnen groepen dan tussen groepen (vooral als de groepen groot zijn). Anders gezegd, als de groepen groot zijn, vind je een groot deel van de genetische verschillen tussen individuen uit verschillende groepen ook al tussen individuen afkomstig uit dezelfde groep. Even zo belangrijk is dat de erfelijkheid op het niveau van groepen laag is: doordat de individuen onafhankelijk van elkaar reproduceren en af en toe migreren tussen groepen, is er niet een duidelijke relatie tussen de genetische opmaak van groepen in opeenvolgende generaties. Andersom zal groep-selectie een grote invloed hebben in situaties waarin individuen zich niet onafhankelijk van elkaar voortplanten, nauwelijks migreren tussen groepen en nauw verwant zijn binnen de groep (denk hierbij bijvoorbeeld aan kolonies van sociale insecten).

BELANGRIJKE STAPPEN TIJDENS DE EVOLUTIE VAN HET LEVEN OP AARDE

Met het inruilen van groep-selectionistische argumenten ten gunste van verklaringen gebaseerd op selectie op het niveau van het individu, is de aandacht van de biologie in veel vakgebieden verlegd. Het recentere inzicht dat selectie op meerdere niveaus tegelijk werkt en dat er tegengestelde belangen tussen de niveaus van selectie kunnen bestaan, maakt het makkelijker allerlei conflicten te begrijpen. Zoals het conflict van het mitochondriaal DNA in tijm en een individueel tijmplantje of het conflict in een kolonie sociale insecten tussen de werksters (die zich niet voortplanten) en de koningin. De keerzijde van de medaille is dat het veel moeilijker is geworden te begrijpen waarom genen, cellen of individuen met elkaar samenwerken en tijdens die samenwerking hun eigen kans op voortplanting voorbij laten gaan ten gunste van het belang van de groep.

Het probleem is des te schrijnender omdat juist de belangrijkste kwalitatieve stappen in de evolutie van het leven op aarde gekenmerkt worden door een overgang van onafhankelijk replicerende eenheden naar een samenwerkend groter geheel (Maynard-Smith & Szathmáry, 1995). Hoewel er nog veel onduidelijk is over de vroegste evolutie zijn er wel duidelijke ideeen en hypothesen over de eerste belangrijke stappen in de evolutie van het leven. Als voorbeeld zijn in FIGUUR 4 drie belangrijke stappen schematisch weergegeven. Bij al de belangrijke evolutionaire stappen zien we dat eenheden die zich voorheen onafhankelijk van elkaar voortplantten, de controle over hun eigen reproductie opgeven en zich voortaan in dienst stellen van de groep. De voordelen daarvan zijn duidelijk: nauwe samenwerking maakt een verdeling van taken mogelijk waardoor complexere problemen kunnen worden opgelost. Het probleem zit ’m alleen daar in dat taakverdeling al samenwerking vereist. Aanvankelijk is het voordeel van samenwerken nog klein omdat er nog nauwelijks taakverdeling is. Bovendien is de selectie op het niveau van de onafhankelijke eenheden nog sterk, omdat er nog geen controle mechanismen zijn en er nog veel variatie bestaat tussen de eenheden. Hoe voorkom je in zo'n geval dat er 'valsspelers' ontstaan die wel de vruchten van de samenwerking plukken maar daar zelf niet aan bijdragen?



FIGUUR 4 – DRIE BELANGRIJKE STAPPEN IN DE EVOLUTIE VAN HET LEVEN OP AARDE

Er is nog veel onduidelijk over prebiotische evolutie. Veel biologen achten het waarschijnlijk dat de oorsprong van het leven ligt bij vrij replicerende RNA moleculen. De aaneenschakeling van die onafhankelijke moleculen tot één chromosoom (A) dat zich als één geheel vermenigvuldigt (kromme pijl) was een belangrijke stap die ingewikkelder stofwisselingsprocessen mogelijk maakte. Een ander voorbeeld van zo'n kwalitatieve stap is het ontstaan van de eukaryote cel (B) met zijn gespecialiseerde cel-organellen (celkern, mitochondriën en chloroplasten). Algemeen wordt aangenomen dat de eukaryote cel ontstaan is als symbiose tussen aanvankelijk onafhankelijke prokaryote cellen. Een derde overgang die de deur opende naar een nieuw niveau van complexiteit is die van ééncelligheid naar meercelligheid (C). Meercelligheid maakt cel differentiatie en specialisatie mogelijk. Andere belangrijke stappen in de evolutie die hier niet staan weergegeven, zijn de overgang van asexuele naar sexuele voortplanting en van solitaire naar sociale leefwijzen.

Oplossingen voor dit probleem zijn niet makkelijk te geven. Het sleutel-idee in de meeste verklaringen is dat populaties steeds worden opgedeeld in kleine groepjes waartussen geen of nauwelijks uitwisseling bestaat. In het kader van pre-biotische evolutie, bijvoorbeeld, wordt hierbij gedacht aan binding van moleculen aan het geladen oppervlak van mineralen, waarbij door ruimtelijke patroonvorming afgescheiden deelpopulaties kunnen ontstaan, of aan spontaan vormende blaasjes (de voorlopers van cellen) waarin een klein aantal RNA moleculen zich repliceert. Door deze processen neemt de uitwisseling van 'individuen' (RNA moleculen, genen) tussen groepen (chromosomen, primitieve cellen) af. Daardoor onstaat een hoge verwantschapsgraad tussen de individuen binnen de groep en relatief veel variatie tussen de groepen. Zoals hierboven uitgelegd, verzwakt dit alles de invloed van individuele selectie en vergroot het de invloed van groep-selectie. Immers, als je als RNA molekuul opgesloten zit in een cel vanwaaruit je niet kunt ontsnappen, is het minder aantrekkelijk wel te profiteren van het werk van ander moleculen maar niet zelf mee te helpen. Als je dat namelijk zou doen, dan zou je je binnen de kortste keren in een cel bevinden waarbinnen geen enkel RNA molekuul zich nog inzet voor de cel (alle andere molekulen zijn namelijk, op den duur, kopieën van jezelf).

Hoewel het dus, ook in theoretische modellen, mogelijk lijkt selectie op individueel niveau te overwinnen door selectie op groepsniveau, is dat zeker niet makkelijk. De omstandigheden waaronder de groep-selectie sterk genoeg wordt om de selectie op lagere niveaus te overwinnen, lijken vaak onwaarschijnlijk. Dit laatste hoeft natuurlijk geen probleem te zijn, gezien de lange tijd die het leven op aarde heeft gehad om zich te ontwikkelen. Toch houd ik persoonlijk het gevoel dat juist de belangrijke evolutionaire overgangen naar hogere organisatieniveaus nog nauwelijks begrepen worden. De terechte verwerping van groep-selectionistische argumenten binnen de gedrags-biologie en het belang dat aan groep-selectie wordt toegekend als het gaat om cruciale stappen in de evolutie van het leven op aarde verhouden zich op gespannen voet met elkaar. Voor sommigen zal die verhouding hoogstens paradoxaal zijn, voor anderen ronduit tegenstrijdig: misschien zit de natuur uiteindelijk toch mooier in elkaar dan we dachten.

SURVIVAL OF THE FITTEST?

Als er iets blijft hangen bij iemand die voor het eerst hoort van Darwin's evolutietheorie, dan is het waarschijnlijk het idee van 'survival of the fittest'[1]*. Dit korte zinnetje vat samen dat door natuurlijke selectie op de lange duur alleen die eigenschappen worden doorgegeven aan de volgende generatie die een voordeel opleveren in de strijd om het bestaan. Alleen de sterkste overleeft, en dat verklaart waarom soorten in de loop van de tijd steeds veranderen. Oude kenmerken worden vervangen door nieuwe, beter aangepaste eigenschappen. Moeilijker alleen is dit idee te rijmen met het feit dat binnen soorten veel variatie bestaat. Zijn al die varianten dan even sterk, of even fit, zoals biologen zeggen? Dat zou wel heel toevallig zijn...*

Biologische voorbeelden van soorten waarbinnen duidelijke variatie in een bepaald kenmerk bestaan, zijn niet moeilijk te vinden. We kunnen dicht bij huis beginnen. Tussen verschillende mensen bijvoorbeeld bestaat duidelijk waarneembare variatie in huidskleur, haarkleur en oogkleur. Iets soortgelijks geldt in extreme mate voor onze huisdieren. Het verschil tussen een teckel en een Sint Bernhard hond is enorm, maar toch behoren ze beide tot dezelfde soort. Datzelfde gaat op voor vlaamse reuzen en dwerg-konijntjes, en ook voor perzische kat en europese korthaar. Maar niet alleen soorten die hebben blootgestaan aan menselijke fok- of veredelingsprogramma's zijn toonbeeld van hoge diversiteit. Ook sommige in het wild levende soorten vertonen opvallend veel variatie.

Een fascinerend voorbeeld daarvan vinden we bij de side-blotched lizard, een hagedissensoort die voorkomt in het zuidwesten van de Verenigde Staten en het noorden van Mexico. Binnen de mannetjes zijn drie variëteiten te onderscheiden[2] die van elkaar verschillen in de kleur van hun keel, en in gedrag. De mannetjes met een oranje keel zijn groot en agressief. Zij monopoliseren meerdere vrouwtjes, die ze fanatiek verdedigen tegen andere mannetjes. Mannetjes met een blauwe keel hebben een andere strategie: zij beperken hun aandacht tot een enkel vrouwtje dat ze zorgvuldig bewaken. Mannetjes met een gele keel, tot slot, doen zich voor als vrouwtje, en slagen er op die manier in af en toe stiekem te paren met de vrouwtjes in het harem van een oranje mannetje.

EVOLUTIE ALS HET BEKLIMMEN VAN EEN TOP IN HET 'FITNESS LANDSCHAP'

Zo makkelijk het is (meer) voorbeelden op te noemen van soorten waarbinnen aanzienlijke variatie in eigenschappen bestaat, zo lastig lijkt het een evolutionaire verklaring te geven voor al die variatie. Want evolutie treedt op door natuurlijke selectie, waarbij individuen die slecht zijn aangepast het veld moeten ruimen voor

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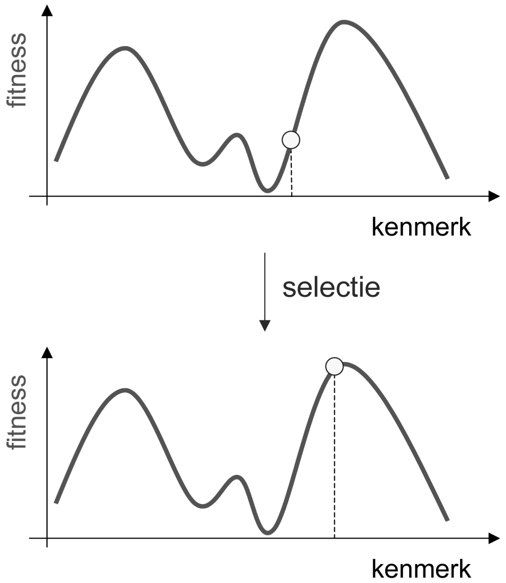
[1] De term "survival of the fittest" is niet door Darwin zelf bedacht maar door Herbert Spencer, een 19^e eeuwse britse filosoof en socioloog.

[2] zie voor een foto http://www.biology.ucsc.edu/~barrylab/lizardland/lizard_heads.jpg

individuen die beter zijn toegerust in de strijd om het bestaan. Dat alles zou toch uiteindelijk moeten leiden tot 'survival of the fittest', het overleven van de best aangepaste? Nu dan, het zou wel heel erg toevallig zijn als alle varianten die we binnen één soort zien allemaal het best aangepast zijn. Er kan er maar één de beste zijn!

Voor wie het probleem nog niet zo ziet, kan de volgende metafoor verhelderend zijn. Stel je voor dat we de kenmerken van een populatie individuen weergeven langs één of meerdere assen en aan elk kenmerk, of combinatie van kenmerken, een fitness waarde toekennen. Als we dat doen, ontstaat een plaatje dat 'het fitnesslandschap' wordt genoemd (FIGUUR 5). De kenmerken die op dit moment in een populatie aanwezig zijn, bepalen waar de populatie zich op dit moment in het landschap bevindt. Door mutaties kunnen nieuwe eigenschappen ontstaan en kenmerken veranderen, met andere woorden, mutaties zorgen ervoor dat de populatie zich door het fitnesslandschap kan bewegen. Die beweging is niet willekeurig van richting, want natuurlijke selectie zorgt ervoor dat alleen die mutaties behouden blijven die de fitness verhogen. Daardoor zal de populatie in de loop van de tijd steeds hoger opklimmen in het fitnesslandschap totdat een top wordt bereikt.

Het wordt nu meteen duidelijk waarom we op het eerste gezicht helemaal niet verwachten dat er variatie binnen een populatie kan bestaan, want vanuit vrijwel elk punt op het fitness landschap kun je maar op één manier omhoog lopen naar slechts één enkele top. En bovendien, als het al zou voorkomen dat er variatie is en sommige individuen in de populatie zich op een andere top bevinden dan de rest van de populatie, dan nog zullen de verschillende toppen waarop de populatie zich bevindt, meestal ongelijk van hoogte zijn, waardoor slechts één enkel kenmerk zal overblijven en de variatie zal verdwijnen.



FIGUUR 5 – HET FITNESS LANDSCHAP

Als we aannemen dat we aan elk kenmerk of combinatie van kenmerken een fitness waarde kunnen toekennen en bovendien de kenmerken kunnen rangschikken langs één (zoals hierboven) of meerdere assen, dan kunnen we een 'fitness landschap' tekenen. Een populatie (lichtgrijze stip) die zich op een bepaalde plek in het fitness landschap bevindt, kan door mutaties door het fitness landschap bewegen. Selectie zorgt ervoor dat alleen mutaties met hogere fitness behouden blijven. Daardoor beweegt de populatie steeds omhoog in het fitness landschap, totdat een top wordt bereikt.

ZONDER VARIATIE GEEN EVOLUTIE

Het probleem van het ontstaan en behouden blijven van variatie is een kwestie die evolutiebiologen al lang bezig heeft gehouden. Niet voor niks, overigens, want variatie is essentieel voor evolutie. Zonder variatie is er geen 'materiaal' waarop selectie kan werken. Ditzelfde komt tot uitdrukking in een fundamentele stelling, die als basis dient voor veel wiskundige modellen van evolutie:

DE MATE VAN EVOLUTIONAIRE VERANDERING VAN EEN KENMERK
=
DE HOEVEELHEID VARIATIE IN HET KENMERK
×
DE STERKTE VAN SELECTIE OP HET KENMERK

Met andere woorden: zonder variatie geen evolutie!

Niet alleen is variatie nodig voor evolutie van eigenschappen binnen soorten. Op grotere schaal is het proces van soortvorming natuurlijk ook afhankelijk van variatie die aanvankelijk binnen één soort is ontstaan. Hoe kunnen anders twee soorten ontstaan uit één enkele voorouder soort, als daarbinnen geen variatie bestaat en alle individuen identiek zijn?

OPLOSSINGEN

Het zal duidelijk zijn dat, gezien het belang van het probleem, evolutie biologen wel met antwoorden moesten komen op de vraag hoe variatie binnen een populatie ontstaat en hoe die variatie behouden blijft. Antwoorden zijn er inderdaad gekomen, en wel maarliefst vier verschillende:

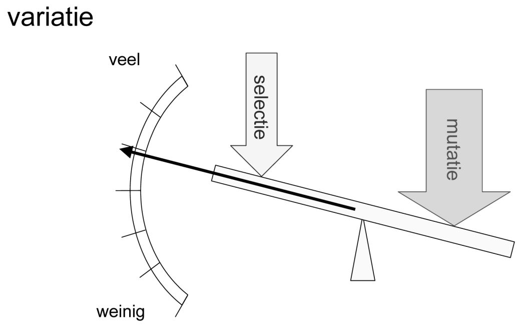
1 – Variatie onstaat door mutaties
2 – Variatie ontstaat door variabele omstandigheden in tijd en ruimte
3 – Variatie ontstaat door genetische beperkingen op de evolutionaire mogelijkheden
4 – Variatie ontstaat door frequentie-afhankelijke selectie

OPLOSSING 1 — DE MUTATIE-SELECTIE BALANS

De eerste oplossing -dat variatie ontstaat door mutaties- is briljant in z'n eenvoud. Natuurlijke selectie is immers niet het enige proces is dat de hoeveelheid variatie binnen een soort bepaalt. Terwijl natuurlijke selectie de variatie verkleint, doordat varianten met een lage fitness worden weggewerkt ('survival of the fittest'), ontstaan er natuurlijk nieuwe varianten door mutaties. De hoeveelheid variatie binnen de soort wordt dan ook bepaald door een evenwicht (de zogenoemde mutatie-selectie balans, FIGUUR 6) tussen de snelheid waarmee selectie inferieure typen verwijdert, en de snelheid waarmee mutatie nieuwe typen doet ontstaan.

FIGUUR 6 – DE MUTATIE-SELECTIE BALANS

Twee processen bepalen hoeveel variatie in een soort kan blijven bestaan. Selectie vermindert variatie doordat inferieure types weggeselecteerd worden. Door mutatie ontstaan daarentegen nieuwe types, waardoor de variatie toeneemt. Uiteindelijk zal een evenwicht worden bereikt tussen deze processen.

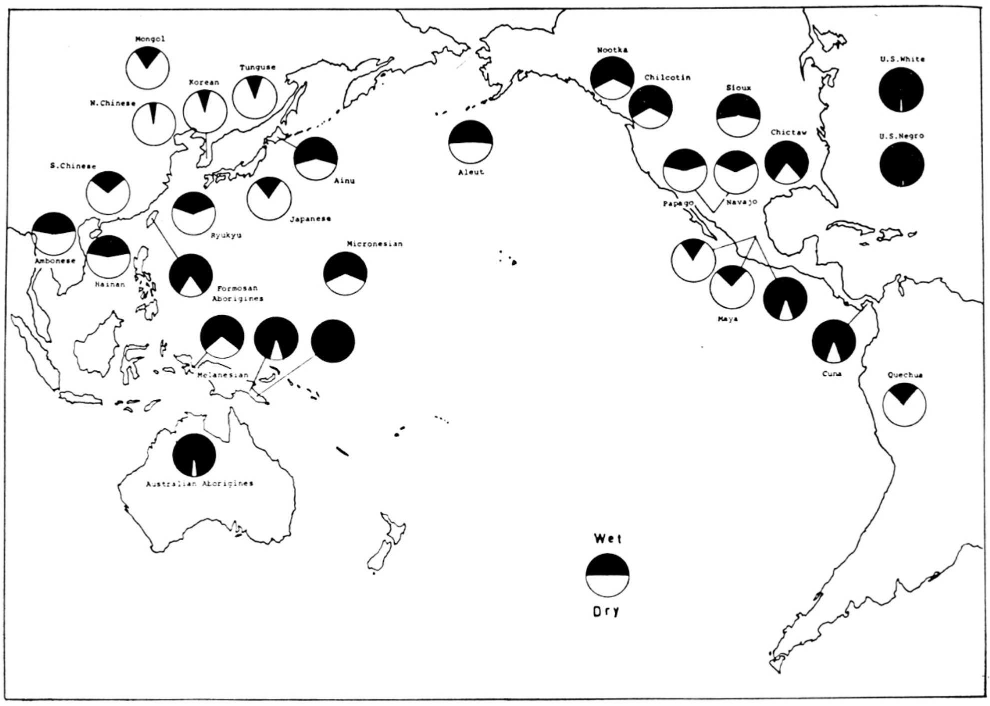


De snelheid waarmee mutaties optreden is in het algemeen slechts zeer klein. Dat betekent dat ook de snelheid waarmee selectie varianten uit de populatie verwijdert heel klein moet zijn, wil er iets te merken zijn van de variatie die door mutatie is ontstaan. Dat wil zeggen, mutaties kunnen alleen voor merkbare variatie zorgen als de selectie op het kenmerk waarin we geïnteresseerd zijn slechts zwak is. Voor veel eigenschappen, zoals de verschillen tussen de verschillende hondenrassen, of de verschillende strategieen van de side-blotched lizards, lijkt het vergezocht te veronderstellen dat selectie op die eigenschappen heel zwak is, maar voor verschillende kenmerken op genetisch niveau is die aanname niet zo gek. Zo bestaan binnen populaties vaak verschillende vormen van bepaalde enzymen die worden gecodeerd door verschillende varianten van een gen. Bij fruitvliegjes vinden we bijvoorbeeld een snelle en een langzame variant van het enzym ADH, dat een belangrijke functie vervuld bij de afbraak van alcohol. Bij de mens vinden we verschillende bloedgroepen (o.a. A/B/O en rhesus factor). Selectie op deze eigenschappen is uiterst zwak, en daardoor kan de polymorfie binnen een populatie blijven bestaan.

OPLOSSING 2 — VARIABELE OMSTANDIGHEDEN IN TIJD EN RUIMTE

Eigenschappen die gunstig zijn op één bepaalde plek, of in één bepaalde periode van het jaar, hoeven dat nog niet te zijn op een andere plek of in een andere periode. Wie in een land woont waar de zon altijd schijnt, doet er goed aan een donkere huidskleur te ontwikkelen, om zo de schadelijke effecten van de overvloedige zonnestraling te beperken. Wie daarentegen woont op een plek waar het altijd regent, kan de investering van het aanmaken van pigment maar beter achterwege laten. Dit bespaart niet alleen de fysiologische kosten, maar is ook minder beperkend voor de synthese van vitamine D in de huid onder invloed van zonlicht.

Het samenspel van migratie en verschillende selectie-optima op verschillende plekken kan, binnen en tussen populaties, voor variatie zorgen. Bij mensen is dit overduidelijk als het gaat om eigenschappen als huidskleur, maar ditzelfde mechanisme speelt ook een rol bij een groot aantal minder opvallende aspecten waarin mensen verschillen, zoals bijvoorbeeld de afbraaksnelheid van alcohol, resistentie tegen bepaalde bacteriën, of de samenstelling van het oorsmeer (FIGUUR 7).



FIGUUR 7 – OORSMEER VARIATIE BIJ DE MENS

Tussen populaties in Oost-Azië, Australië en Noord- en Zuid-Amerika bestaan verschillen in de samenstelling van het oorsmeer. Oorsmeer komt voor in twee varianten: nat (zwart) en droog (wit). De frequentie waarin nat oorsmeer voorkomt binnen populaties neemt toe voor populaties in gebieden waar het klimaat vochtig en warm is. Nat oorsmeer verkleint de kans op oor infecties onder deze omstandigheden. Onder droge en koude omstandigheden, daarentegen, is droog oorsmeer beter. Bron: Omoto (1973).

OPLOSSING 3 — GENETISCHE BEPERKINGEN OP DE EVOLUTIONAIRE MOGELIJKHEDEN

Dat genetische beperkingen variatie in stand kunnen houden wordt het best duidelijk aan de hand van het voorbeeld van de ziekte sikkelcel-anemie. Deze ziekte komt in veel tropische landen in hoge frequentie voor. Sikkelcel-anemie is erfelijk en wordt veroorzaakt door een mutatie in het gen dat codeert voor hemoglobine, het eiwit dat het zuurstoftransport in het bloed verzorgt. De ziekte is ernstig en leidt vaak tot de dood. Sikkelcel-anemie ontstaat bij mensen die twee defecte kopieën van het hemoglobine-gen bezitten. Individuen die één normale kopie en één defecte kopie van het gen bezitten, zijn dragers van de aandoening, maar worden zelf niet ziek. Vanwege de serieuze consequenties van de ziekte, zou je verwachten dat de frequentie van het afwijkende hemoglobine allel binnen de korste keren door selectie teruggebracht zal worden tot lage waarden, maar dat blijkt niet het geval: in sommige afrikaanse populatie is de frequentie van het allel hoger dan 20%.

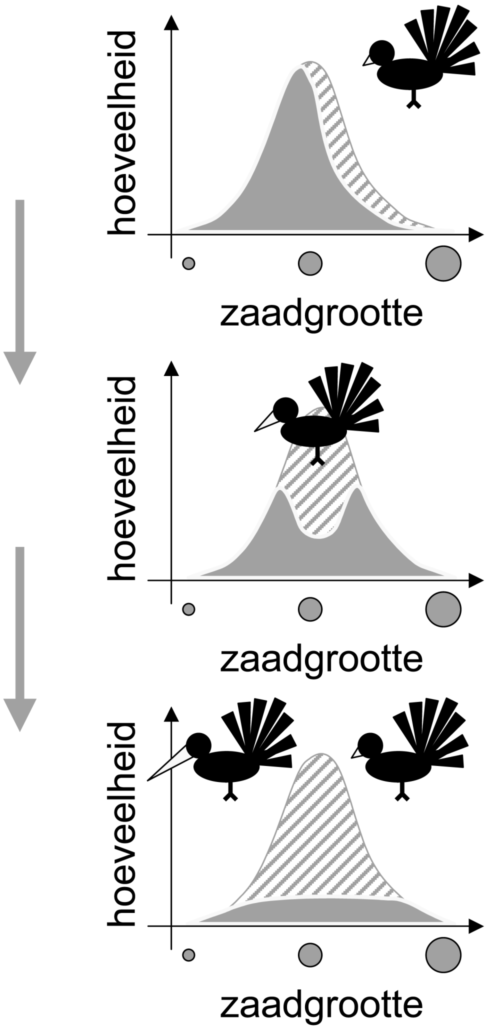
De opzienbarend hoge frequentie van het allel dat sikkelcel-anemie veroorzaakt, bleef onverklaard totdat onderzoekers ontdekten dat sikkelcel-anemie vooral voorkomt in landen waar ook de ziekte malaria voorkomt. Nader onderzoek wees uit dat dragers van de ziekte sikkelcel-anemie, juist vanwege hun afwijkende hemoglobine, minder zwaar te lijden hadden onder malaria infecties. Dit leidt ertoe dat, in populaties waar malaria voorkomt, dragers van de ziekte sikkelcel-anemie de hoogste fitness hebben, want zij leiden niet aan sikkelcel-anemie, zoals de individuen die twee afwijkende hemoglobine allelen dragen, en zijn minder vatbaar voor malaria dan de individuen die twee normale kopieën van het hemoglobine gen dragen. Juist omdat de heterozygoten (de dragers van sikkelcel-anemie) de hoogste fitness hebben blijft in de populatie variatie bestaan. Voor heterozygoten zijn twee verschillende allelen nodig. Dit heeft als bij-effect dat er ook homozygoten met lagere fitness zullen ontstaan.

Het voorbeeld van sikkelcel-anemie laat zien dat variatie kan ontstaan door genetische beperkingen op de evolutionaire mogelijkheden. In dit geval wordt de genetische beperking veroorzaakt doordat er nooit een populatie kan ontstaan met alleen maar heterozygoten, zelfs niet als die een veel hogere fitness hebben. Immers, als heterozygoten met elkaar paren ontstaan er vanzelf weer homozygoten.

OPLOSSING 4 — FREQUENTIE-AFHANKELIJKE SELECTIE

De traditionele manier van denken over evolutie en natuurlijke selectie gaat uit van het idee dat kenmerken een vast fitnessvoordeel of -nadeel opleveren. Ikzelf heb die aanname ook gemaakt, toen ik een fitness landschap tekende (FIGUUR 5). De aanname van vaste fitnesswaardes voor eigenschappen maakt het een stuk makkelijker selectie goed te beschrijven, maar dat wil natuurlijk nog niet zeggen dat die aanname ook altijd klopt. Sterker nog, het is eigenlijk heel makkelijk om voorbeelden te verzinnen van eigenschappen waarvan het fitness voordeel helemaal niet vast is, maar verandert. Hiermee doel ik niet op veranderingen in ruimte of tijd, zoals eerder behandeld, maar op veranderingen van de fitness waarde van een eigenschap in relatie tot de frequentie van die eigenschap in de populatie.

Laten we als voorbeeld nog eens kijken naar de side-blotched lizard. Mannetjes met een oranje keel gedragen zich agressief en zijn meestal in staat het vrouwtje van een mannetje met een blauwe keel af te pakken. Toch leggen de mannetjes met een oranje keel het af tegen de blauwe mannetjes als er veel mannetjes met gele keel zijn. Gele mannetjes zijn in staat stiekem te paren met de vrouwtjes van het oranje mannetje, maar niet met het vrouwtje van een blauw mannetje, want die bewaakt zijn vrouwtje te goed. Het succes van elk van de drie verschillende strategieën hangt dus sterk af van de strategie van de andere mannetjes in de populatie: oranje is in het voordeel als er veel blauwe mannetjes zijn en weinig gele, blauw is in het voordeel als er veel gele mannetjes zijn en weinig oranje, en geel is in het voordeel als er veel oranje mannetjes zijn en weinig blauwe.



FIGUUR 8 – HET ONTSTAAN VAN VARIATIE DOOR FREQUENTIE-AFHANKELIJKE SELECTIE

In de habitat van een vogelsoort komen veel verschillende zaden voor, van uiterst kleine tot grote zaden. Het spectrum van aanwezige zaden wordt in bovenstaande plaatjes weergeven door de gearceerde verdeling. Merk op dat zaden van gemiddelde grootte het vaakst voorkomen. De vogels die van de zaden eten kunnen niet alle zaden eten, maar moeten zich specialiseren op een deel van de zaden. Specialisatie treedt op door aanpassingen van de snavelvorm: korte snavels zijn geschikt voor het kraken van de grote zaden, lange snavels zijn juist geschikt voor het efficient verzamelen van de kleinste zaden. Als de vogels aanvankelijk korte snavels hebben (bovenste plaatje), dan worden alleen de grote zaden opgegeten. Een groot deel van de zaden (grijze verdeling) blijft dan over. Omdat veel meer zaden van gemiddelde grootte beschikbaar zijn, zal door evolutie de snavel van de vogels geleidelijk aan groter worden, totdat die precies is afgestemd op de meest voorkomende zaden (middelste plaatje). Nu is de concurrentie om zaden van gemiddelde grootte het hoogst, en blijven de kleinste en grootste zaden onopgegeten achter. Dit maakt het interessant concurrentie te ontlopen en juist te specialiseren op de grote of de kleine zaden. Deze optie werkt alleen als de populatie 'evolutionair vertakt' in vogels met grote en kleine snavel (onderste plaatje). Zonder het opsplitsen zouden we immers opnieuw belanden in de situatie van het bovenste plaatje.

Als de fitness van een eigenschap afhangt van de frequentie van andere eigenschappen in de populatie, dan kunnen we niet langer een vaste fitness toekennen aan zo'n eigenschap, en wordt de metafoor van het fitnesslandschap misleidend. Bij frequentie-afhankelijke selectie kunnen we beter denken aan een fitness-'zeeschap', d.w.z. een steeds veranderende relatie tussen eigenschappen en fitness. De continue verandering wordt veroorzaakt doordat enerzijds evolutie leidt tot veranderende eigenschappen in de populatie en anderzijds de veranderende samenstelling van de populatie leidt tot veranderingen in de sterkte en richting van selectie. Deze interactie kan tot vreemde resultaten leiden. Eén van de mogelijke uitkomsten is dat de populatie steeds omhoog beweegt in het fitness-zeeschap, maar uiteindelijk toch in een fitness dal terecht komt (dit scenario wordt aan de hand van een voorbeeld uitgelegd in FIGUUR 8). Deze tegen-intuitieve mogelijkheid is interessant als het gaat om het ontstaan van variatie, want in veel gevallen kan de populatie alleen maar uit het dal ontsnappen door variabel te worden. Via dit proces, dat 'evolutionair vertakken' wordt genoemd, kan frequentie-afhankelijke selectie niet alleen variatie binnen de soort behouden, maar die zelfs doen ontstaan. Dit laatste, zo suggereren verschillende onderzoekers, is een belangrijke eerste stap naar het ontstaan van nieuwe soorten.

EVOLUTIE OP HET HOOGSTE NIVEAU: HET ONTSTAAN VAN NIEUWE SOORTEN

Een verkorte versie van dit artikel werd gepubliceerd samen met
Gaby van Caulil in Bionieuws 6 (29 maart 2002).

Hoe ontstaan nieuwe soorten? Deze vraag, die al generaties biologen bezig hield, zorgt ook nu nog voor wetenschappelijke opschudding. De strijd tussen twee concurrende theorieën, die al jaren geleden leek beslecht, lijkt opnieuw op te laaien. Naast de oude argumenten, vinden we opvallend nieuw 'wapentuig' in de discussie: experimenten en observaties die de traditioneel gevestigde opvatting onderuit halen en nieuwe theoretische ontwikkelingen die de oplossing zouden kunnen geven voor een aantal oude problemen.

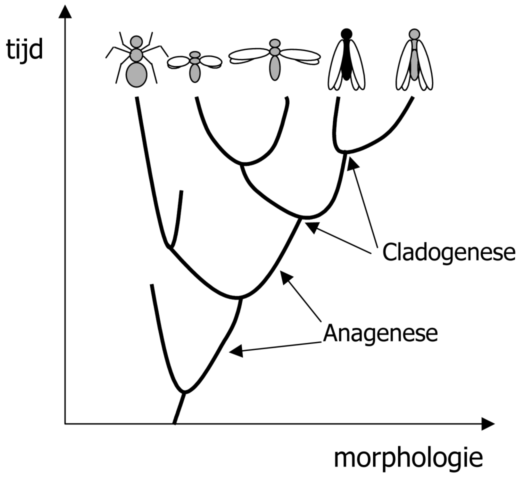
Wie er even over nadenkt, valt het op hoeveel verschillende soorten organismen op deze aarde voorkomen. De enorme diversiteit van het leven maakt het tot een wonderlijk en ingewikkeld schouwspel. De verwondering kan alleen maar toenemen als we ons realiseren dat het leven niet zo is begonnen zoals het vandaag is –dat blijkt uit de fossielen-, en ook niet zo zal blijven –alleen al vanwege de snel veranderende menselijke invloeden op de natuur.

De diversiteit van de natuur is continu aan verandering onderhevig. Waar komen al die soorten vandaan en hoe zijn ze ontstaan? Wie voor het antwoord op deze vraag te rade gaat bij Charles Darwin, de beroemde grondlegger van de evolutietheorie en schrijver van het boek 'On the Origin of Species' ('Over het Onstaan van Soorten'), komt bedrogen uit. In tegenstelling tot wat de titel van zijn boek doet vermoeden, schrijft Darwin nauwelijks iets over het onstaan van soorten (soortvorming), zeker niet in vergelijking met de aandacht die hij besteedt aan de veranderingen die door natuurlijke selectie kunnen optreden binnen een soort (FIGUUR 9). Het is niet zo dat Darwin simpelweg nooit heeft nagedacht over soortvorming. Waarschijnlijker is dat hij zijn eigen ideeën over 'het ultieme mysterie' (zoals hij het zelf omschreef) te onduidelijk vond.

Opmerkelijk genoeg blijft tot op de dag van vandaag onduidelijkheid bestaan over de manier waarop nieuwe biologische soorten ontstaan. Het wetenschappelijk debat over soortvorming heeft al een lange geschiedenis, maar lijkt de laatste jaren in een stroomversnelling te raken. Sommige wetenschappers wagen het zelfs te spreken van een fundamentele verschuiving in de manier waarop over soortvorming wordt nagedacht. In dit artikel zal ik proberen dieper in te gaan op de nieuwe ideeën over soortvorming die tot zoveel wetenschappelijke opschudding leiden. Maar eerst zal ik een kort overzicht geven van de geschiedenis van het soortvormingsdebat.

FIGUUR 9 – ANAGENESE EN CLADOGENESE

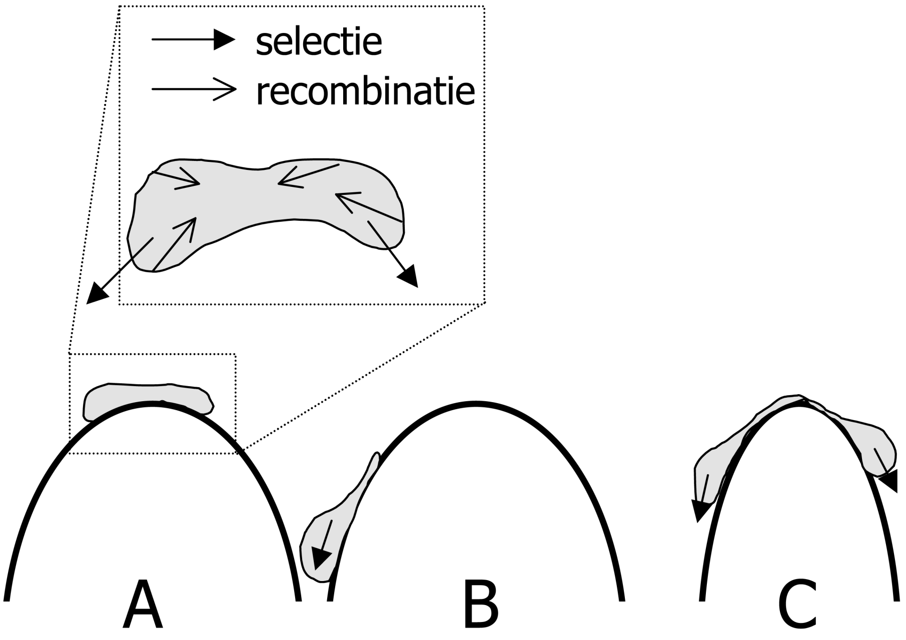
Biologen geven de evolutionaire verhoudingen tussen verschillende soorten vaak weer d.m.v. een evolutionaire boom. De takken van de boom symboliseren dan de geleidelijke evolutionaire verandering van bijvoorbeeld morphologie die optreedt binnen een soort. Dit proces wordt anagenese genoemd, en dit is het voornaamste onderwerp van Darwin's werk. Nieuwe soorten ontstaan op punten waar de boom vertakt. Dit proces, dat cladogenese, of -in gewoon Nederlands- soortvorming, wordt genoemd, is fundamenteel verschillend van anagenese.



TERUG IN DE TIJD

De bioloog Ernst Mayr heeft als één van de grondleggers van de moderne evolutie theorie, waarin Darwin’s theorie van evolutie door natuurlijke selectie en de erfelijkheidsleer (die teruggaat op het werk van Gregor Mendel) zijn samengesmeed, een grote invloed gehad op de theorie omtrent soortvorming. Ernst Mayr verzamelde in Nieuw Guinea zoveel mogelijk verschillende soorten paradijsvogels, en bij deze klus viel het hem op dat verschillende soorten vaak van elkaar gescheiden zijn door een geografische barrière, zoals bijvoorbeeld een bergketen, of de zee tussen twee eilanden. Deze waarneming vormde de basis van zijn theorie van soortvorming. Mayr stelde voor dat nieuwe soorten kunnen ontstaan wanneer een populatie door een geografische barrière wordt opgesplitst in twee geïsoleerde delen. Elk van deze twee delen gaat vervolgens zijn eigen evolutionaire weg. Als de geografische isolatie maar lang genoeg in stand blijft, resulteert dit in twee soorten die, zelfs als de geografische scheiding verdwijnt, niet meer met elkaar zullen kruisen. Dit is kortweg wat bekend staat als het allopatrisch model voor soortvorming.

Het allopatrisch model van soortvorming wordt in de meeste tekstboeken scherp afgezet tegen een concurrerend model: dat van sympatrische soortvorming. In dit model wordt soortvorming allereerst gezien als het resultaat van disruptieve selectie. Dit type selectie treedt op wanneer extreme varianten van een eigenschap het beter doen dan intermediaire varianten, zoals bijvoorbeeld bij een insectensoort die op twee verschillende gastheerplanten voorkomt: omdat elke gastheerplant specifieke aanpassingen vereist, doet een individu dat zich specialiseert het beter dan een individu dat zich niet specialiseert op één van de gastheersoorten.



FIGUUR 10 – DE THEORETISCHE PROBLEMEN VAN SYMPATRISCHE SOORTVORMING

De moeilijkheden van sympatrische soortvorming worden het best duidelijk aan de hand van de volgende analogie: stel je een biologische populatie onder disruptieve selectie voor als een grote waterdruppel die op de top van een heuveltje ligt (A). De plek die de druppel heeft symboliseert de (morphologische) kenmerken van de populatie. Onder invloed van de zwaartekracht zal de druppel van het heuveltje afrollen (B). In biologische zin betekent dit dat de kenmerken van de populatie onder invloed van selectie veranderen. Hoewel de druppel aan twee zijden van het heuveltje af kan rollen, zal de druppel in het algemeen niet opsplitsen (er treedt geen soortvorming op). Dit komt omdat de druppel water bij elkaar gehouden wordt door de oppervlaktespanning. In biologische populaties is een soortgelijke kracht aan het werk, die de kenmerken van individuen in de populatie bij elkaar houdt: dit is de recombinatie van kenmerken die elke generatie opnieuw plaatsvindt bij de voortplanting (close up). Aan de hand van dit voorbeeld wordt duidelijk dat sympatrische soortvorming alleen kan optreden wanneer disruptive selectie sterk genoeg is, en wanneer de cohesie van de populatie vanwege sexuele voortplanting kan worden overwonnen (C). Een tweede probleem wordt duidelijk als we ons afvragen hoe de druppel water terecht is gekomen op de top van de heuvel: onder invloed van de zwaartekracht (selectie) zal de druppel (de populatie) immers alleen maar wegbewegen van de top (het punt waar selectie disruptief is).

In de wetenschappelijke discussie is veel aandacht besteed aan de vraag of soorten nu voornamelijk door allopatrische of sympatrische soortvorming zijn ontstaan. Het antwoord op deze vraag heeft zo z'n consequenties voor de manier waarop we tegen evolutie aankijken. Immers, iemand die een sterk voorstander is van allopatrische soortvorming zou zeggen dat evolutie op het niveau van soorten gedreven wordt door toevallige externe gebeurtenissen (zoals het ontstaan van gletsjers en bergketens), terwijl de aanhanger van sympatrische soortvorming er van uit gaat dat soortvorming optreedt door biologische processen binnen de populatie die, in principe, voorspelbaar zijn.

Tot voor zo'n tien jaar terug was de algemene mening in deze discussie dat de overgrote meerderheid van soorten is ontstaan door allopatrische soortvorming, en dat sympatrische soortvorming alleen onder zeer specifieke ecologische omstandigheden mogelijk is. Deze mening was gebaseerd op waarnemingen uit de natuur en gegevens uit experimenten (empirische gegevens) maar ook op theoretische modellen.

Voor allopatrische soortvorming is door Mayr en andere biologen veel empirische evidentie verzameld, maar gevallen van overduidelijke sympatrische soortvorming bleken veel moeilijker te vinden. Een belangrijke uitzondering hierop vormt het werk van Guy Bush en anderen, die door hun studies aan het appelvliegje Rhagoletis hebben laten zien dat sympatrische soortvorming door specialisatie op verschillende gastheerrassen (zoals in het voorbeeld hierboven) mogelijk is. Desondanks lieten labstudies met het fruitvliegje Drosophila zien dat disruptieve selectie zonder geografische isolatie vrijwel nooit leidt tot soortvorming. Theoretische modellen, tot slot, maakten duidelijk dat sympatrische soortvorming in principe wel mogelijk is, maar zeker niet onder algemene omstandigheden (FIGUUR 10).

MOLECULAIRE PROBLEMEN

De wetenschappelijke eensgezindheid met betrekking tot het meest aannemelijk model van soortvorming is blijven bestaan totdat de ontwikkeling van de moleculaire biologie het mogelijk maakte evolutionaire stambomen op grond van DNA-sequenties te reconstrueren. De methode werkt als volgt: binnen een groep verwante soorten wordt bij elke soort de DNA sequentie van een bepaald stukje van het erfelijk materiaal bepaald. Door nu de DNA sequenties te vergelijken kunnen onderzoekers iets te weten komen over de volgorde waarin soorten uit elkaar zijn ontstaan en hoe lang geleden soortvorming heeft plaatsgevonden.

In een aantal gevallen hebben deze analyses onverwachte resulaten opgeleverd. Neem bijvoorbeeld het geval van de cichliden in de grote meren van Oost-Afrika. Oorspronkelijk werd altijd gedacht dat de enorme variëtiet aan cichlidesoorten is ontstaan door herhaaldelijke fluctuaties van het waterniveau in de meren. Door een daling van de waterspiegel kan een meer namelijk opgesplitst raken in een aantal kleinere meertjes, waardoor de verschillende vispopulaties van elkaar geïsoleerd raken en kunnen evolueren tot verschillende soorten. Als de waterspiegel na lange tijd weer stijgt, kunnen de nieuw gevormde soorten zich opnieuw verspreiden binnen het grote meer. Dit scenario leidt tot de voorspelling dat soorten het nauwst verwant zijn met vergelijkbare soorten uit een ander meer, en minder verwant met soorten in hetzelfde meer.

Fascinerend genoeg wijst de evolutionaire stamboom op grond van moleculaire gegevens precies in de tegenovergestelde richting: alle soorten binnen een meer, en dat kunnen er honderden zijn (!), stammen af van slechts enkele voorouder soorten. Voor een meer als Lake Victoria, dat zo'n 12.000 jaar geleden nog volledig droog stond, betekent dat dat honderden soorten cichliden in korte tijd (12.000 jaar!) binnen het meer zijn ontstaan.

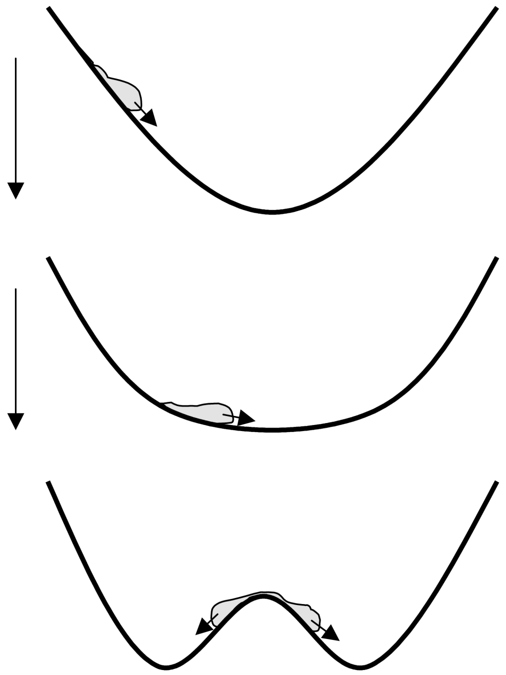
Soortgelijke gegevens werden gevonden voor een groep van enkele cichlidensoorten in een klein kratermeer in Kameroen: het meer is zo klein, en van een zodanige vorm, dat de aanwezigheid van geografische barrières in dit meer uitgesloten kan worden. Op grond van deze en andere gegevens zijn biologen voorzichtiger geworden in hun stellige afwijzing van sympatrische soortvorming. Veel biologen menen nu dat sympatrische soortvorming mogelijk is, de belangrijke vraag is alleen welke omstandigheden daarvoor nodig zijn.

NIEUWE MODELLEN

Ook op het gebied van de theorievorming hebben deze verassende resulaten geleid tot een hernieuwde belangstelling voor sympatrische soortvorming. Immers, de moleculaire gegevens hebben niets kunnen veranderen aan de theoretische bezwaren die aan het licht kwamen in de eerdere generatie modellen voor sympatrische soortvorming (FIGUUR 10). Het is natuurlijk belangrijk een passend antwoord op deze bezwaren te vinden, omdat discrepanties tussen modellen en de praktijk vaak aangeven dat we belangrijke aspecten van een probleem nog niet begrijpen.

Belangrijke vooruitgang is geboekt op twee gebieden. Het eerste betreft het algemeen probleem dat optreedt zodra selectie disruptief is (een voorwaarde voor sympatrische soortvorming): als extreme kenmerken het beter doen dan gemiddelde dan is het veel waarschijnlijker dat een populatie opschuift naar één van de extremen dan dat die populatie splitst in twee delen (zie ook FIGUUR 10). Het probleem kan alleen worden opgelost als een populatie door natuurlijke selectie zelf naar het punt gedreven wordt waar splitsing door disruptieve selectie mogelijk is.

Deze tegen-intuitieve combinatie van selectiekrachten lijkt onwaarschijnlijk, maar blijkt toch mogelijk te zijn, weliswaar onder bepaalde aannames en altijd in situaties waarbij de selectiekrachten op individuen afhankelijk zijn van de strategie van de rest van de populatie (FIGUUR 11). Dit is in veel ecologische scenarios het geval. Neem bijvoorbeeld vogels die concurreren om voedsel. Laten we aannemen dat de vorm van de snavel bepaalt welk type voedsel –hier zaden van verschillende groottes– een vogel het meest efficient kan verzamelen. Vanuit een enkel individu bekeken zal de optimale snavel vorm uiteraard afhankelijk zijn van de hoeveelheid zaden die van de verschillende groottes aanwezig is, maar natuurlijk ook van de snavel vorm van de andere individuen. Dit laatste bepaalt immers met hoeveel concurrenten onze vogel zijn kostje moet delen. Deze interactie tussen de competitie strategieën van individuen maakt selectie op de vorm van de snavel frequentie afhankelijk, d.w.z. de sterkte en richting van selectie hangen af van de competitie strategieën van de rest van de populatie.



FIGUUR 11 – FREQUENTIE AFHANKELIJKE SELECTIE

In het meest eenvoudige geval hangen de selectie krachten op een populatie niet af van de toestand van de populatie. In dat geval leidt evolutie altijd weg van het punt waar soortvorming kan plaatsvinden. Wil sympatrische soortvorming optreden, dan moet de populatie door selectie in de richting van het punt van disruptieve selectie worden geduwd. Dit kan alleen als selectie 'frequentie afhankelijk' is, d.w.z. de sterkte en richting van selectie hangt af van de toestand van de populatie. In de waterdruppel analogie van figuur 10 betekent dit dat de vorm van de heuvel verandert als de druppel van plaats verandert.

Het tweede probleem dat in de aandacht staat, is het ontstaan van reproductieve isolatie: voor soortvorming is niet alleen disruptieve selectie nodig maar ook dat individuen van de soorten-in-wording na verloop van tijd niet langer met elkaar paren. De klassieke soortvormingsmodellen toonden al aan dat reproductieve isolatie niet uit het niets ontstaat, zeker niet in het algemene geval waar er niet een directe selectiekracht op werkt. Een oplossing zou kunnen zijn dat sexuele selectie zorgt voor extreme partner voorkeuren bij de vrouwtjes en extreme secondaire geslachtskenmerken bij de mannetjes, en dat die voorkeuren en kenmerken verschillend worden tussen de beide soorten in wording. Op die manier kiezen vrouwtjes alleen mannetjes binnen hun eigen soort en dat voorkomt kruisingen tussen individuen van verschillende soorten.

Al lang is bekend dat dit soort partnervoorkeuren bij vrouwtjes, samen met extreme ornamenten bij de mannetje, snel kunnen evolueren (dit is bijvoorbeeld voorgesteld als de verklaring van de enorme staart van de mannetjespauw en het belang dat de vrouwtjespauw aan die staart hecht bij de partnerkeuze), maar nieuw is het idee dat tegelijkertijd binnen één enkele populatie verschillende voorkeuren en kenmerken zouden kunnen ontstaan. Hoewel de eerste theoretische modellen erop wijzen dat dit inderdaad tot de mogelijkheden behoort, is het wat mij betreft nog een open vraag of sexuele selectie door vrouwelijke partnerkeuze onder algemene omstandigheden tot sympatrische soortvorming kan leiden. Dit laat niet weg dat allerlei observaties erop wijzen dat sexuele selectie wel degelijk een belangrijke rol speelt bij soortvorming: vaak kunnen nauw verwante soorten het best worden onderscheiden op grond van hun secondaire geslachtskenmerken!

Conclusie

Solide empirisch bewijs voor sympatrische soortvorming is nog steeds moeilijk te verkrijgen en ook de theoretische problemen zijn nog niet volledig opgelost. Toch zijn de nieuwe resulaten en de snelle ontwikkelingen in het soortvormings onderzoek fascinerend. In snel tempo worden nieuwe stukjes van de puzzel zichtbaar. Het beeld dat ontstaat laat in ieder geval meer ruimte voor sympatrische soortvorming, daar is vrijwel iedereen het over eens. De meningen verschillen nog sterk over de vraag hoe belangrijk en algemeen sympatrische soortvorming is, en nu we weten dat soorten ook kunnen ontstaan zonder geografische isolatie verschuiven de onderzoeksvragen subtiel in de richting van vragen als: “Waarom vinden we niet een aparte soort kever op elke struik die we tegenkomen?”, of “Waarom vindt sympatrische soortvorming niet heel vaak plaats?”.

Een beroemde bioloog zou het idee van sympatrische soortvorming eens vergeleken hebben met de mazelen: “Iedereen krijgt het, maar iedereen geneest er ook weer van”. De tijd zal leren of hij ditmaal gelijk krijgt, maar voorlopig lijkt het erop dat de huidige epidemie een chronisch karakter draagt.

WEG MET DE MAN!

In tegenstelling tot wat zangers en dichters ons doen geloven, is niet de vrouw maar juist de man een waar mysterie. Althans, als je het evolutiebiologen zou vragen. Het evolutionair nut van vrouwen is meteen duidelijk, maar wat hebbben we aan mannen? Mannen leveren in de meeste soorten slechts een beperkte bijdrage aan de productie van nakomelingen, maar verbruiken wel de helft van de energie en bouwstoffen beschikbaar voor de voortplanting. Zouden we niet veel beter af zijn als we ons zonder mannen, dat wil zeggen, ongeslachtelijk zouden voortplanten? Op zoek naar de voordelen van geslachtelijke voortplanting.

Mensen doen het, guppies doen het, fruitvliegen doen het. Regenwormen doen het ook, net als tuinbonen, golden-retrievers en champignons. Ik heb het over geslachtelijke voortplanting, de voortplantingswijze waarbij nakomelingen ontstaan uit de versmelting van het genetisch materiaal van hun ouders. Belangrijk detail: één van die ouders is vrouw, de andere is man. Er bestaan zoveel soorten die zich geslachtelijk voortplanten, dat je je nauwelijks kunt voorstellen dat het ook anders zou kunnen. Laat staan dat je je zou afvragen wat eigenlijk de voordelen zijn van geslachtelijke voortplanting. Toch is dat een vraag die evolutiebiologen zichzelf hebben gesteld. Met onverwacht resultaat, want het onderzoek naar het evolutionair nut van geslachtelijke voortplanting heeft, naast een aantal onduidelijke voordelen, bovenal een overduidelijk nadeel van geslachtelijke voortplanting aan het licht gebracht. Dat nadeel is de man.

GESLACHTELIJKE VERSUS *ON*GESLACHTELIJKE VOORTPLANTING

Het antwoord op de vraag naar het evolutionair voordeel van geslachtelijke voortplanting hangt natuurlijk sterk af van het alternatief waarmee we geslachtelijke voortplanting vergelijken. Wie in de natuur op zoek gaat naar alternatieve voortplantingswijzen, vindt al snel soorten die zich niet alleen geslachtelijk maar ook ongeslachtelijk kunnen voortplanten. Allerlei planten, bijvoorbeeld, kunnen zich niet alleen -door middel van bestuiving- geslachtelijk voortplanten, maar ook ongeslachtelijk, met behulp van uitlopers (aardbei) of wortelstokken (gember), door afsplitsing van bollen (ui), of door productie van zaden zonder bestuiving (paardebloem). Ook dieren zoals poliepen, watervlooien en bladluizen wisselen geslachtelijke en ongeslachtelijke voortplantingsfasen af. Een aantal hogere organismen is zelfs volledig afhankelijk van ongeslachtelijke voortplanting en heeft het vermogen tot geslachtelijke voortplanting verloren.

Ook bacteriën planten zich uitsluitend ongeslachtelijk voort, door zich simpelweg in tweeën te delen. Daaruit kunnen we afleiden dat ongeslachtelijke voortplanting de oudste voortplantingswijze is. Geslachtelijke voortplanting moet echter al vroeg in de evolutie zijn ontstaan, want de grote meerderheid van hogere organismen plant zich geslachtelijk voort. De uitzonderingen op die regel laten zien dat ongeslachtelijke voortplanting verschillende malen opnieuw is ontstaan in voorheen geslachtelijk voortplantende soorten. Opvallend genoeg heeft die overgang van geslachtelijke naar ongeslachtelijke voortplanting zich in verreweg de meeste gevallen recent voltrokken. Met andere woorden, op een paar uitzonderingen na bestaan er geen hogere organismen die vroeg in de evolutie zijn ontstaan en zich al die tijd ongeslachtelijk hebben voortgeplant.

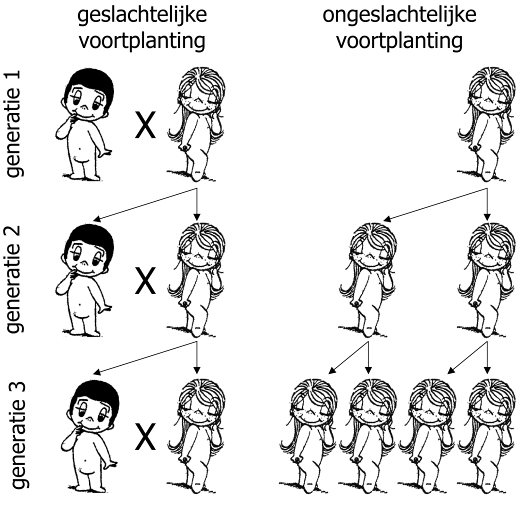
Het verklaren van deze waarnemingen is niet makkelijk. Het lijkt aannemelijk dat geslachtelijke voortplanting een bepaald voordeel heeft boven ongeslachtelijke voortplanting. Geslachtelijke voortplanting is immers ooit ontstaan uit ongeslachtelijke voortplanting. Bovendien lijkt het ontbreken van evolutionair oude soorten met ongeslachtelijke voortplanting er op te wijzen dat ongeslachtelijk voortplantende organismen het op de lange duur niet redden. Daartegenover staat dat ongeslachtelijke voortplanting herhaaldelijk is ontstaan in hogere organismen die zich voorheen geslachtelijk voortplantten. Dit laatste leidt eerder tot de omgekeerde conclusie, namelijk dat juist ongeslachtelijke voortplanting voordelen heeft boven geslachtelijke voortplanting.

HET TWEEVOUDIG NADEEL VAN GESLACHTELIJKE VOORTPLANTING

Wat kunnen we doen om een beter beeld krijgen van de voordelen en nadelen van geslachtelijke voortplanting? Eén mogelijkheid is op zoek te gaan naar twee soorten die in alle opzichten gelijk zijn, behalve in hun voortplantingswijze. We kunnen dan onderzoeken welke van de twee soorten het uiteindelijk zal winnen als ze competitie van elkaar ondervinden. Interessante kandidaten voor zo'n onderzoek zijn de renhagedissen van het geslacht *Cnemidophorus*. *Cnemidophorus* renhagedissen komen voor in het westelijk deel van Noord-Amerika. Bijna alle soorten in het geslacht planten zich geslachtelijk voort, maar er zitten ook enkele soorten tussen die zich ongeslachtelijk voortplanten. *Cnemidophorus uniparens* (uniparens betekent één-ouder) is zo'n aseksuele (of ongeslachtelijk voortplantende) renhagedis. Alle *Cnemidophorus uniparens* individuen zijn vrouwtjes, die zonder tussenkomst van mannetjes in staat zijn eieren te leggen. Uit die eieren onstaat vervolgens een nieuwe generatie vrouwtjes.

Als *Cnemidophorus uniparens* individuen onder dezelfde omstandigheden worden opgekweekt als individuen van een verwante seksuele (of geslachtelijk voortplantende) *Cnemidophorus* soort, zien we dat vrouwtjes van beide soorten een vergelijkbaar aantal eieren leggen. Van de eieren van de aseksuele soort groeit 100% uit tot vrouwtjes. Voor de seksuele soort groeit 50% uit tot vrouwtjes en de andere 50% groeit uit tot mannetje. Dat laatste is een belangrijk verschil, want de mannetjes van de seksuele soort leggen natuurlijk geen eieren voor de volgende generatie nakomelingen. Kortom, slechts de helft van de nakomelingen van een seksueel vrouwtje helpt mee met het leggen van eieren voor de volgende generatie. De ander (mannelijke) helft doet niks, behalve vrouwtjes bevruchten dan. Als we een generatie verder zijn, zullen er dus, vergeleken met het aantal seksuele individuen, twee keer zoveel aseksuele individuen zijn (zie ook FIGUUR 12). Immers, alle aseksuele individuen kunnen meehelpen met het leggen van eieren voor de volgende generatie. Kortom, de seksuele soort groeit twee keer zo traag als de aseksuele soort, omdat de seksuele soort 50% van de energie die beschikbaar is voor de voortplanting verspilt aan de produktie van mannetjes.

Bij geslachtelijke voortplanting wordt de helft van de beschikbare energie en bouwstoffen geïnvesteerd in de produktie van mannen. Die mannen doen vervolgens niet mee bij het produceren van de volgende generatie. Om een geslachtelijk voortplantende populatie in stand te houden, moet een vrouwtje daarom minstens twee nakomelingen produceren. Onder diezelfde omstandigheden groeit een populatie waarvan de individuen zich ongeslachtelijk voortplanten. Als een aseksueel vrouwtje gemiddeld twee nakomelingen produceert, verdubbelt de populatieomvang zich elke generatie. Dit komt omdat elke nakomeling ook zelf kan bijdragen aan de groei van de populatie.



FIGUUR 12 – HET TWEEVOUDIG NADEEL VAN GESLACHTELIJKE VOORTPLANTING

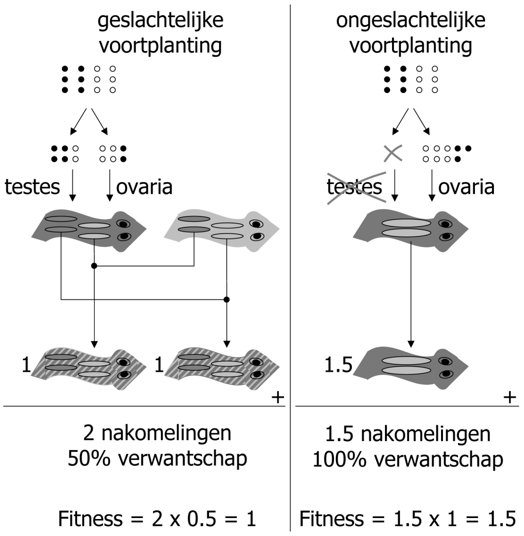
De kosten van de productie van mannetjes staan bekend als 'het tweevoudig nadeel van geslachtelijke voortplanting'. Het tweevoudig nadeel van geslachtelijke voortplanting brengt tot op de dag van vandaag evolutiebiologen in verlegenheid. Hoe kunnen we verklaren dat geslachtelijke voortplanting in zoveel soorten voorkomt, als geslachtelijke voortplanting het zo overduidelijk lijkt af te leggen tegen ongeslachtelijke voortplanting? Afgezien van dit wetenschappelijk probleem is het tweevoudig nadeel van geslachtelijke voortplanting natuurlijk voer voor feministen van de harde lijn en een aanslag op het ego van de gemiddelde (=mannelijke) evolutiebioloog.

Wat de onderliggende motivatie ook moge zijn, een indrukwekkend gezelschap wetenschappers heeft zich gestort op de vraag of geslachtelijke voortplanting misschien voordelen heeft die kunnen opwegen tegen het tweevoudig nadeel. Het resultaat van deze inspanningen is een aantal argumenten dat laat zien dat het nadeel van geslachtelijke voortplanting soms minder groot is dan tweevoudig, en een groot aantal theorieën dat laat zien dat geslachtelijke voortplanting onder bepaalde omstandigheden inderdaad voordelen heeft.

WAAROM HET MISSCHIEN SOMS MEEVALT MET HET TWEEVOUDIG NADEEL…

Verschillende biologen hebben erop gewezen dat het argument van het tweevoudig nadeel van geslachtelijke voortplanting berust op belangrijke onderliggende aannames die zeker niet algemeen opgaan. Zo wordt bijvoorbeeld verondersteld dat de productie van mannetjes ten koste gaat van de produktie van vrouwtjes. In het geval van de renhagedissen, bijvoorbeeld, kan een vrouwtje maar een beperkt aantal eieren leggen. Voor elk ei waaruit een mannetje ontstaat, moet ze een dochter inleveren.

Toch zijn er ook gevallen te bedenken waarin de productie van zoons niet noodzakelijkerwijs ten koste gaat van de productie van dochters. Denk bijvoorbeeld aan hermaphrodieten. Hermaphrodieten zijn tweeslachtige organismen, zoals veel planten, regenwormen of platwormen. Hermaphrodieten produceren natuurlijk geen zoons of dochters (ze zijn immers tweeslachtig), maar ze moeten wel beslissen hoeveel energie ze investeren in de productie van mannelijke en vrouwelijke geslachtscellen en -organen.



FIGUUR 13 – MINDER DAN TWEEVOUDIG NADEEL

Geslachtelijk voortplantende platwormen investeren de helft van de beschikbare bouwstoffen in mannelijke functie (testes) en de andere helft in vrouwelijke functie (ovaria). Ongeslachtelijk voortplantende platwormen hoeven niets in mannelijke functie te investeren. Dit hoeft niet te betekenen dat ze daardoor twee maal zo grote ovaria kunnen vormen. Neem bijvoorbeeld aan dat eiwitten (gesymboliseerd door zwarte rondjes) en vetten (witte rondjes) nodig zijn om de geslachtsorganen aan te leggen. Voor de testes zijn deze bouwstoffen nodig in de verhouding 2:1, voor de ovaria in de verhouding 1:2. In dit getalvoorbeeld kan een askesuele platworm slechts anderhalf keer zo grote ovaria aanleggen als een seksuele platworm. De seksuele platworm produceert twee nakomelingen (één uit eigen eieren en één door bevruchting van een ei van een andere seksuele platworm), die elk voor de helft meetellen, omdat maar de helft van het DNA aan de nakomelingen is doorgegeven. De aseksuele worm produceert gemiddeld anderhalf maal zo veel eieren als een seksuele platworm en geen sperma. Gemiddeld produceert de aseksuele worm dus anderhalve nakomeling uit eigen eieren, met 100% identiek genetisch materiaal. Een vergelijking van de fitness van de seksuele en aseksuele platworm laat nu niet een tweevoudig nadeel zien, maar een 'anderhalf-voudig' nadeel voor geslachtelijke voortplanting.

Ook voor hermaphrodieten is er een nadeel van geslachtelijke voortplanting. Een populatie aseksuele hermaphrodieten waarvan de individuen nakomelingen produceren uit onbevruchte eieren, kan immers harder groeien dan een seksuele populatie. De aseksuele hermaphrodieten hoeven immers niets te investeren in hun mannelijke functie en kunnen daardoor meer eieren produceren. Toch is het heel goed denkbaar dat een afname van investering in mannelijke functie zich niet terugbetaalt in een *even grote* toename van de investering in vrouwelijke functie. Dat kan bijvoorbeeld veroorzaakt worden doordat verschillende grondstoffen nodig zijn bij de produktie van mannelijke en vrouwelijke geslachtscellen en –organen (FIGUUR 13). In zo'n geval is de groeisnelheid van de aseksuele populatie dan ook minder dan twee keer zo groot als die van de seksuele populatie. Met andere woorden, als de investering in mannelijke functie (zoons) niet heel direct ten koste gaat van de investering in vrouwelijke functie (dochters) is het nadeel van geslachtelijke voortplanting is minder dan tweevoudig.

Een ander uitgangspunt dat we kunnen bekritiseren is de aanname dat mannetjes geen enkel positief effect hebben op het aantal nakomelingen dat een vrouwtje produceert. Die aanname klopt niet voor soorten waar het mannetje bijdraagt aan de ouderlijke zorg (denk aan stekelbaarsjes, zeepaardjes en mensen, bijvoorbeeld). Dankzij de hulp van het mannetje kan een vrouwtje in een geslachtelijk voortplantende soort meer nakomelingen grootbrengen dan een ongeslachtelijk voortplantend vrouwtje, dat het zonder mannetje moet doen. Een deel van de energie die is verspild aan mannetjes wordt daarom terugverdiend door de bijdrage van het mannetje aan ouderlijke zorg. Ook in soorten met mannelijk ouderlijke zorg is het nadeel van geslachtelijke voortplanting daarom minder dan tweevoudig.

DE SLEUTEL TOT DE VOORDELEN VAN GESLACHTELIJKE VOORTPLANTING

Bij het vergelijken van geslachtelijke en ongeslachtelijke voortplanting hebben we tot nu één belangrijk verschil tussen deze twee voortplantingswijzen onbesproken gelaten. Anders dan bij ongeslachtelijke voortplanting, vindt bij geslachtelijke voortplanting een reductie deling (meiose) plaats. Bij de meiose wordt het aantal chromosomen in de cel gehalveerd. Dit is nodig omdat een nakomeling ontstaat uit de versmelting van twee geslachtscellen: één van de man en één van de vrouw. Zonder meiose zou een nakomeling twee keer zoveel chromosomen bezitten als zijn ouders, en dat kan op de lange duur niet goed gaan...

Door de meiose dragen beide ouders 50% van hun genetisch materiaal over aan de nakomeling (dit is het tweevoudig nadeel van geslachtelijke voortplanting op genetisch niveau!). Om de meiose eerlijk te laten verlopen, worden de chromosomen willekeurig verdeeld, zodat alle chromosomen evenveel kans hebben terecht te komen in de volgende generatie. Dit heeft een belangrijk bij-effect. Door de willekeur van meiose is het resultaat van elke reductie deling anders. Niet alleen de chromosomenparen worden onafhankelijk van elkaar over de dochtercellen verdeeld, ook binnen chromosomen paren vindt recombinatie van genen plaats door crossing-over. Het resultaat van al dit gehussel met genen is dat geen enkele nakomeling gelijk aan een andere zal zijn. Met andere woorden, geslachtelijke voortplanting levert nakomelingen op die onderling genetisch verschillend zijn. Ongeslachtelijke voortplanting, daarentegen, levert genetisch identieke nakomelingen op (klonen).

Is dit verschil in variatie onder nakomelingen misschien de sleutel tot voordelen die het tweevoudig nadeel van geslachtelijke voortplanting kunnen compenseren? Die vraag kunnen we pas beantwoorden als we weten wat de evolutionaire voordelen zijn van het genereren van variatie door recombinatie, zoals dat tijdens geslachtelijke voortplanting gebeurt.

DE EFFECTEN VAN RECOMBINATIE

Recombinatie zorgt voor het ontstaan van nieuwe gen combinaties. Dat kan uiterst voordelig zijn als een soort zich moet aanpassen aan veranderende milieuomstandigheden en als daarvoor meerdere mutaties nodig zijn. Bij aseksuele organismen moeten de noodzakelijke mutaties in elke lijn afzonderlijk plaatsvinden, en daardoor kan het erg lang duren voordat een lijn zich heeft aangepast. In een seksuele populatie, daarentegen, kunnen de mutaties van verschillende individuen via geslachtelijke voortplanting snel gecombineerd worden tot een optimaal aangepast genotype.

De keerzijde van de medaille is natuurlijk dat recombinatie ook goed op elkaar afgestemde gencombinaties verwoest. Dit is een groot nadeel als de milieu omstandigheden constant zijn. In een aseksuele populatie zullen alle nakomelingen het genotype van hun ouder overerven. We mogen aannemen dat dit genotype ook het optimale genotype is als de milieuomstandigheden al lange tijd niet veranderd zijn. In een seksuele populatie ontstaan daarentegen steeds varianten van het meest optimale genotype. Die varianten hebben per definitie steeds een lagere fitness.

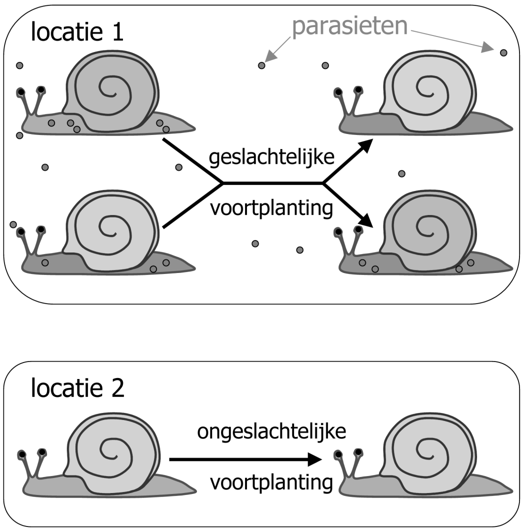
Geslachtelijke voortplanting zorgt natuurlijk niet alleen voor de recombinatie van voordelige mutaties, maar ook voor de recombinatie van schadelijke mutaties. Een positief effect daarvan is dat recombinatie nakomelingen kan opleveren die minder schadelijke mutaties hebben dan hun ouders. Zoiets is in aseksuele populaties onmogelijk. Als in een aseksuele populatie door toeval een mutatie gefixeerd raakt, is er geen weg terug, met als gevolg dat aseksuele populaties in de loop van de tijd steeds meer schadelijke mutaties ophopen. Dit proces treedt niet op in seksuele populaties, dankzij recombinatie.

Recombinatie van schadelijke mutaties heeft nog een tweede effect, maar daarvan hangt het opnieuw van de omstandigheden af of het effect positief danwel negatief is. Door recombinatie ontstaat variatie in het aantal schadelijke mutaties per individu. Het kan zo zijn dat de schadelijke mutaties daardoor efficienter door natuurlijke selectie uit de populatie kunnen worden verwijderd, waardoor de fitness van de populatie toeneemt. Daarvoor is het wel nodig dat de negatieve effecten van een extra mutatie groter worden naarmate een individu al meer schadelijke mutaties bij zich draagt. In dat geval, zijn de fitnesskosten per mutatie het hoogst voor individuen met veel mutaties. Zulke individuen zijn er niet alleen in absolute, maar ook in relatieve zin bijzonder slecht aan toe, en zij zullen met hoge waarschijnlijk weggeselecteerd worden. Daardoor neemt de hoeveelheid mutaties in de populatie af. Het tegenovergestelde gebeurt wanneer de negatieve effecten van een extra mutatie afnemen naarmate een individu al meer schadelijke mutaties bij zich draagt. In dat geval worden juist de individuen met weinig mutaties relatief sneller wegggeselecteerd.

KOSTEN EN BATEN

Klaarblijkelijk is het niet zo makkelijk te zeggen hoe het evolutionair kosten-baten plaatje van geslachtelijke voortplanting eruitziet. Afhankelijk van de milieuomstandigheden en de precieze manier waarop schadelijke mutaties elkaar beïnvloeden heeft geslachtelijke voortplanting voordelen dan wel nadelen ten opzichte van ongeslachtelijke voortplanting. Geslachtelijke voortplanting heeft de beste kansen wanneer de milieuomstandigheden steeds veranderen en wanneer schadelijke mutaties elkaars effecten versterken. Zijn dit realistische omstandigheden, of zijn de milieuomstandigheden veel vaker constant en hebben nadelige mutaties gezamenlijk minder effect dan hun afzonderlijke effecten opgeteld? Het antwoord op die belangrijke vragen moet uiteindelijk door slimme waarnemingen en experimenten uit de natuur komen.

Veel slakken kunnen zich geslachtelijk en ongeslachtelijk voortplanten. Vergelijkend onderzoek aan nieuw-zeelandse slakken populaties heeft aangetoond dat de frekwentie van geslachtelijke voortplanting hoger is naarmate er meer parasieten in het milieu voorkomen. Geslachtelijke voortplanting zorgt op plaatsen met veel parasieten (locatie 1) voor variatie, en dat maakt snellere aanpassingen in de evolutionaire wapenwedloop met de parasiet mogelijk. Op plaatsen zonder parasieten (locatie 2) zijn die voortdurende aanpassingen niet noodzakelijk, en is juist ongeslachtelijke voortplanting optimaal.



FIGUUR 14 – ZIEKTEVERWEKKERS EN GESLACHTELIJKE VOORTPLANTING

Experimentele evolutie met behulp van micro-organismen is een veelbelovende techniek, die het mogelijk maakt uit te vinden hoe mutaties elkaars schadelijke effecten beïnvloeden. De eerste experimenten in die richting zijn inmiddels afgerond, maar het is op dit moment nog te vroeg voor algemene conclusie (als die er al zijn). Voor wat betreft de veranderlijkheid van de milieuomstandigheden richt de aandacht zich momenteel vooral op ziekteverwekkers als oorzaak van continue verandering. Ziekteverwekkers evolueren in hoog tempo nieuwe strategieen waarmee ze de verdedigingsmechanismen van hun gastheer ondermijnen. Daardoor is voortdurende verandering van die verdedigingsmechanismen noodzakelijk. Ziekteverwekkers en hun gastheren zijn verwikkeld in een niet ophoudende evolutionaire wapenwedloop, waarin geslachtelijke voortplanting een voorsprong kan opleveren. Ondersteuning voor dit idee komt bijvoorbeeld uit overzicht studies die een verband aantonen tussen de frequentie van geslachtelijke voortplanting en de grootte van het gevaar van ziekteverwekkers (FIGUUR 14).

Ook al zou uiteindelijk blijken dat de omstandigheden optimaal zijn geweest voor de evolutie van geslachtelijke voortplanting, het zal de vraag blijven of de voordelen van geslachtelijke voortplanting opwegen tegen de aanzienlijke reductie in efficiëntie die gepaard gaat met het produceren van mannen. Het tweevoudig nadeel van geslachtelijke voortplanting lijkt een onoverwinnelijke barrière voor elk afzonderlijk voordeel van geslachtelijke voortplanting dat we kunnen bedenken. Daar komt nog eens bij dat veel voordelen van geslachtelijke voortplanting pas op de lange termijn zichtbaar worden, terwijl het tweevoudig nadeel direct voelbaar is. Daarom zijn de meeste biologen het erover eens dat we het antwoord moeten zoeken in de combinatie van verschillende voordelen, in de hoop dat die voordelen elkaar wellicht kunnen versterken. Het zijn mooie ideeën, maar hoe het in de praktijk zou moeten werken is nog onbekend. Kortom, het lijkt erop dat het mysterie van de man voorlopig onopgelost blijft.

De evolutie van macho’s en nerds

In veel soorten wordt dominantie gebruikt om conflicten op te lossen. Hoe worden verschillen in dominantie bepaald? Berusten ze simpelweg op krachtsverschillen of veeleer op (onbewuste) afspraken binnen een populatie? Een theoretisch model laat zien dat door evolutie inderdaad afspraken kunnen ontstaan waardoor winnaars blijven winnen en verliezers steeds verliezen, zelfs wanneer iedereen gelijke kans heeft conflicten te winnen.

Allerlei dieren leven in groepen. Dat brengt voordelen met zich mee, zoals een betere bescherming tegen roofdieren, en de mogelijkheid tot samenwerken bij het jagen of het grootbrengen van de jongen. Ondanks deze voordelen zijn de belangen van groepsgenoten ook vaak tegengesteld. Bijvoorbeeld wanneer alle mannetjes in de groep azen op hetzelfde paringsbereide vrouwtje, of op dezelfde sappige vrucht. Dat kan natuurlijk niet anders dan problemen geven...

De vaak tegengestelde belangen van groepsgenoten ontaarden regelmatig in conflicten, die, in een sociale groep, niet zo gemakkelijk als bij solitair levende soorten kunnen worden opgelost door elkaar te ontwijken. Conflicten kunnen natuurlijk worden uitgevochten, maar dat is niet altijd een goede oplossing. De agressieveren onder ons weten het uit eigen ervaring, maar ook wie wel eens in een natuurfilm het gevecht tussen twee zee-olifanten of edelherten tijdens de paringstijd heeft gezien, weet dat vechten riskant is. Er is een grote kans op (mogelijk fatale) verwondingen, met de bijbehorende negatieve uitwerking op de fitness van het vechtende individu. ‘Fitness’ is biologisch jargon voor ‘de bijdrage van erfelijk materiaal aan volgende generaties’ (dit wordt vaak ruwweg bepaald door het aantal nakomelingen dat een individu tijdens zijn leven produceert).

Omdat hoge kosten zijn verbonden aan het aangaan van felle gevechten, is het niet verwonderlijk dat allerlei sociaal levende soorten alternatieve strategieën hebben ontwikkeld om conflicten op te lossen. Vaak komt het niet zover dat een conflict ontaardt in een fel gevecht, maar valt de beslissing al in een eerder stadium, waarin de twee tegenstanders ogenschijnlijk nog niets anders hebben gedaan dan dreigen. Als alternatief voor het gevecht wordt de uitkomst van het conflict nu niet direct bepaald door de krachtsverhoudingen maar door een andere asymmetrie tussen beide tegenstanders. Dat kan een verschil in lichaamsgrootte zijn of het onderscheid tussen de bezitter van een territorium en de indringer, maar –en dat zien we verassend vaak– zo’n asymmetrie kan ook berusten op een veel minder tastbaar onderscheid tussen individuen, namelijk een verschil in sociale rang.

DOMINANTIE

Sociale rangordes (dominantie hierarchieën) kom je tegen in uiteenlopende soorten, van kippen tot wolven. Bij het observeren van zo'n soort is meestal relatief makkelijk dominante individuen ('macho's') en onderdanige individuen ('nerds') te onderscheiden. Iedereen met middelbare school ervaring weet dat een macho bij conflicten met een nerd systematisch voordeel heeft, en het conflict vaak wint zonder dat hij hoeft te vechten. Ook wie een klein toom kippen observeert zal ontdekken dat er tussen elk paar van individuen een verschil in sociale rang bestaat: één van beide is dominant over de ander. Bovendien blijkt één individu dominant over alle andere individuen, een tweede individu is dominant over alle anderen behalve over de nummer één, enzovoort, zodat er een duidelijke sociale rangorde is te onderscheiden (bij kippen heel toepasselijk de pikorde genoemd).

Het geweldloos oplossen van conflicten met behulp van rang asymmetrieën vereist natuurlijk dat alle individuen in de populatie het eens zijn over de vraag wie dominant is over wie. Bovendien moet iedereen zich ook daadwerkelijk aan de 'afspraak' houden dat degene die dominant is het conflict wint. Dit vereiste van eensgezindheid leidt tot interessante vragen. Is het eigenlijk wel te verwachten dat iedereen zich aan dezelfde afspraak wil en blijft houden? In het bijzonder, waarom zou een individu onder aan de rangorde zich houden aan een afspraak die voor hem nadelig uitpakt? Waarom komen de nerds niet in opstand?

Gek genoeg is er lange tijd geen aandacht geweest voor deze vraag. Er is weliswaar informatie over de werking van bijvoorbeeld stress-hormonen en hoe die agressief gedrag kunnen beinvloeden, maar deze kennis kan alleen verklaren *hoe* dominante en onderdanige individuen zich gedragen, en niet *waarom* ze zich zo gedragen. Die *waarom*-vraag is eigenlijk een evolutionaire vraag. Als we kunnen uitleggen dat evolutie selecteert voor een conflict-oplossings-strategie waarbij gebruik wordt gemaakt van sociale rang verschillen, dan hebben we –voor biologen tenminste– antwoord gegeven op de vraag waarom dominantie bestaat.

EEN MODEL VOOR CONFLICTEN

Om de evolutie van sociale dominatie nader te bestuderen nemen we onze toevlucht tot theoretische modellen. Een theoretische benadering is indirect en als onderzoeker weet je nooit helemaal zeker of het model dat je bestudeert iets over de werkelijkheid zegt. Toch worden modellen veel gebruikt in de evolutiebiologie, omdat evolutie simpelweg te langzaam verloopt om via experimenten of (gedrags)observatie interessante evolutionaire vragen te kunnen beantwoorden. Een ander belangrijk voordeel van theoretische modellen is dat je als onderzoeker de aannames van het model kunt varieren en zo van allerlei relatief vage ideeën kunt testen of ze kloppen. Bij dit gebruik van theoretische modellen is het vaak ongewenst een model te maken dat zo exact mogelijk overeenkomt met de werkelijkheid, omdat het model dan veel te ingewikkeld wordt. Het doel is een model te maken dat zo simpel mogelijk is, maar toch nog steeds alle essentiele componenten bevat om de onderzoeksvraag te beantwoorden.

Ons model voor de evolutie van dominantie word ook zo'n minimaal model. We beginnen bij het beschrijven van een conflict tussen twee individuen ('spelers'), die allebei aanspraak maken op, bijvoorbeeld, een sappige vrucht. Laten we simpelweg aannemen dat in dit soort conflicten beide spelers kunnen kiezen uit twee mogelijke acties: dreigen (afgekort: D) en het conflict ontwijken (afgekort: O). We gaan er verder vanuit dat beide individuen tegelijkertijd kiezen zonder te weten wat de tegenstander kiest. Er zijn dan vier mogelijke conflict-situaties (TABEL 1).

TABEL 1 – CONFLICTSITUATIES EN UITKOMSTEN

conflict situatie	keuzes		oplossing van het conflict	wie krijgt de vrucht?	fitnesspunten	
	speler 1	speler 2			speler 1	speler 2
1	D	D	'vechten'	speler 1	55	-205
				speler 2	-205	55
2	D	O	speler 2 geeft op	speler 1	55	0
3	O	D	speler 1 geeft op	speler 2	0	55
4	O	O	'loten'	speler1	60	0
				speler 2	0	60
			'eerlijk delen'	eerlijk delen	30	30

Als beide spelers dreigen, dan escaleert het conflict tot een gevecht dat net zo lang doorgaat totdat één van de spelers gewond of uitgeput raakt en moet opgeven. Als één van de spelers dreigt en de ander ontwijkt, dan is de oplossing van het conflict simpel: de dreigende speler krijgt de vrucht. Als, tenslotte, beide spelers het conflict ontwijken dan wordt het conflict vreedzaam opgelost. In dat geval zijn er twee mogelijkheden waarvan we hier alleen de eerste zullen bekijken: de beide spelers 'loten' wie de vrucht krijgt, waarbij elk van beide gelijke kans heeft, of de vrucht wordt netjes in twee gelijke delen verdeeld en elke speler krijgt de helft.

Nu moeten we natuurlijk gaan nadenken over de consequenties die de verschillende uitkomsten van het conflict hebben op de fitness van de vechtende individuen. Het lijkt voor de hand liggend dat het bemachtigen van de vrucht een positief effect op de fitness heeft, omdat de voedingswaarde van de vrucht gebruikt kan worden om nakomelingen te produceren, terwijl verwondingen een negatief effect hebben, omdat een gewond individu tijd nodig heeft om te herstellen, waarin hij zich niet kan voortplanten. Om het model zo eenvoudig mogelijk te houden, nemen we aan dat we deze positieve en negatieve effecten simpelweg tegen elkaar kunnen afwegen, en daarom drukken we ze beide uit in fitness punten: in het voorbeeld van TABEL 1 hebben we bijvoorbeeld de waarde van de vrucht op 60 fitness punten gesteld, en de kosten van verwondingen op 200 fitness punten. Verder nemen we aan dat dreigen ook wat kost, door verhoogde stress in agressieve toestand. In ons getalvoorbeeld kost dreigen 5 punten.

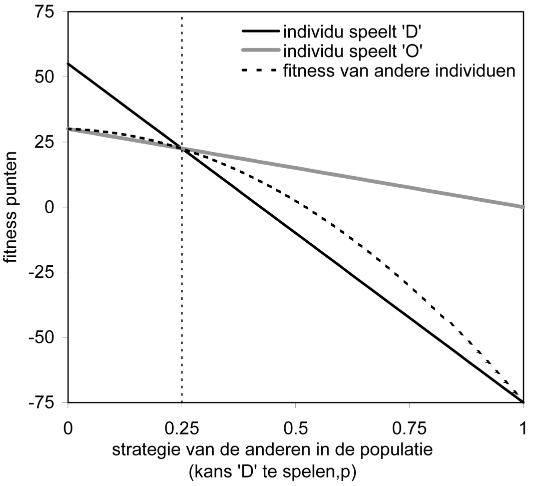
SPIEREN OF AFSPRAKEN?

Het laatste aspect van het model dat nog een verduidelijking behoeft is wat er gebeurd als beide spelers dreigen. In zo'n geval ontstaat een gevecht, waarbij één van beiden wint, maar het kan best zo zijn dat de ene speler een veel grotere kans op winst heeft dan de ander, omdat hij veel sterker is. Dat kan natuurlijk een belangrijk effect hebben. Sommige onderzoekers menen zelfs dat hierin de verklaring van dominantie ligt: een nerd vecht niet omdat hij door het krachtsverschil tussen hem en de macho simpelweg toch nooit zou kunnen winnen. Eigenlijk wordt dominantie dan volledig bepaald door de onderlinge krachtsverhoudingen. Dit idee is alleen wel tegen het zere been van andere onderzoekers, die menen dat dominantie veel onafhankelijker is van krachtsverschillen en eigenlijk berust op min of meer willekeurige afspraken tussen individuen. Zo'n -mogelijk onbewuste (!)- afspraak zou kunnen zijn: 'we vechten één keer, en degene die wint mag dominant zijn, en de verliezer wordt het onderdanige individu'.

Er zijn bewijzen gevonden voor dit soort afspraken. In verschillende soorten, waaronder vissen, apen, kreeften en vogels, is er inderdaad een 'winnaar-' of 'verliezer-effect', waarbij de winnaar van een conflicten een hogere kans heeft ook volgende conflicten te winnen en/of de verliezer een hogere kans heeft de volgende keer opnieuw te verliezen. Bovendien is in theoretische modellen aangetoond dat 'winnaar-' en 'verliezer-' effecten, veel beter dan onderliggende krachtsverschillen, de pikordes kunnen verklaren die we bij, bijvoorbeeld, kippen zien. Er is alleen één probleem: zoals we al eerder opmerkten is het nog allerminst duidelijk hoe dit soort afspraken in de evolutie kan (blijven) bestaan. Juist omdat we in deze vraag geinteresseerd zijn, zullen we aannemen dat er volstrekt geen krachtsverschillen tussen individuen bestaan, m.a.w. als er een gevecht plaatsvindt, hebben beide spelers gelijke kans te winnen. Door deze aanname weten we zeker dat eventuele dominantie strategieën die in onze simulaties evolueren zeker niet verklaard kunnen worden door onderliggende krachtsverschillen, maar wel moeten berusten op afspraken.

DE OPTIMALE STRATEGIE

Individuen die meer fitness punten hebben verdiend, zullen meer nakomelingen voortbrengen, en daarom zal evolutie uiteindelijk leiden tot een optimale strategie die zoveel mogelijk fitness punten oplevert. Kan een dominantie-strategie zo'n optimale strategie zijn? Voordat we die vraag kunnen beantwoorden, moet eerst duidelijk zijn wat we precies bedoelen met een 'optimale' strategie. Dat is niet zo makkelijk te zeggen, want de optimale beslissing in een conflict hangt af van wat je tegenstander doet. Als je zeker zou weten dat je tegenstander 'D' kiest, dan is het slim 'O' te kiezen. Zo krijg je weliswaar geen punten, maar dat is altijd nog beter dan wanneer je ook 'D' had gespeeld: in dat geval krijg je soms 55 punten, maar even vaak moet je 205 punten inleveren (zie TABEL 1). Gemiddeld ga je er dan 75 punten op achteruit! Daartegenover staat dat, wanneer je zeker zou weten dat je tegenstander 'O' speelt, je beter 'D' kunt spelen: in dat geval weet je zeker dat je de vrucht krijgt, terwijl je moet delen als je ook 'O' zou spelen.



FIGUUR 15 – EEN EVOLUTIONAIR STABIELE STRATEGIE.

In de tekst rekenden we al voor hoeveel fitness-punten een individu ontvangt als hij speelt tegen tegenstanders uit een populatie waarin iedereen altijd 'D' speelt, of altijd 'O' speelt. Ook voor populaties waarin alle individuen 'D' spelen met een wilekeurige kans tussen deze twee extremen (uitgezet op de x-as) kunnen we makkelijk uitrekenen hoeveel fitness-punten een afwijkend individu kan verwachten als hij zich niet zou houden aan de populatie strategie maar altijd 'D' speelt (zwarte lijn), of altijd 'O' speelt (grijze lijn). Daarnaast rekenden we uit hoeveel fitness punten de niet afwijkende individuen in de populatie verdienen (dikke stippellijn). Links van de dunne stippellijn is de fitness van agressievere individuen hoger dan de fitness van individuen die niet afwijken van de populatie strategie. We verwachten daarom dat in de loop van evolutie strategieën zullen onstaan waarmee individuen vaker 'D' spelen, waardoor de populatie naar rechts zal schuiven over de x-as. Rechts van de stippellijn aangekomen zijn individuen die minder vaak 'D' spelen in het voordeel. Daarom zal de populatie weer naar links opschuiven. Alleen exact op de dunne stippellijn loont het niet af te wijken van de anderen in de populatie. Dus, voor onze keuze van de parameters is de evolutionair stabiele strategie: in 25% van de gevallen dreigen en in 75% van de gevallen ontwijken.

Uit deze argumenten blijkt al dat altijd dreigen geen optimale strategie is, in die zin dat, als iedereen in de populatie altijd 'D' speelt, het loont van die strategie af te wijken (datzelfde geldt voor de strategie altijd ontwijken). Een interessante vraag is nu of er misschien strategieën te vinden zijn waar het voor niemand loont van de strategie af te wijken die de rest van de populatie speelt. Zulke strategieën zijn 'optimaal' in het licht van evolutie, omdat ze het eindpunt van evolutie zijn: zolang het nog loont af te wijken van de rest van de populatie, zullen individuele strategieën, en dus de gemiddelde strategie in de populatie, nog veranderen. Pas als het voor niemand meer loont van strategie te veranderen, is een evolutionair evenwicht bereikt. Dit soort evolutionaire evenwichts strategieën is zo belangrijk dat ze een eigen naam hebben gekregen (evolutionair stabiele strategieën). Bovendien is er een hele tak van de wiskundige biologie (de biologische speltheorie) ontwikkeld om evolutionair stabiele strategieën op te sporen en te onderzoeken.

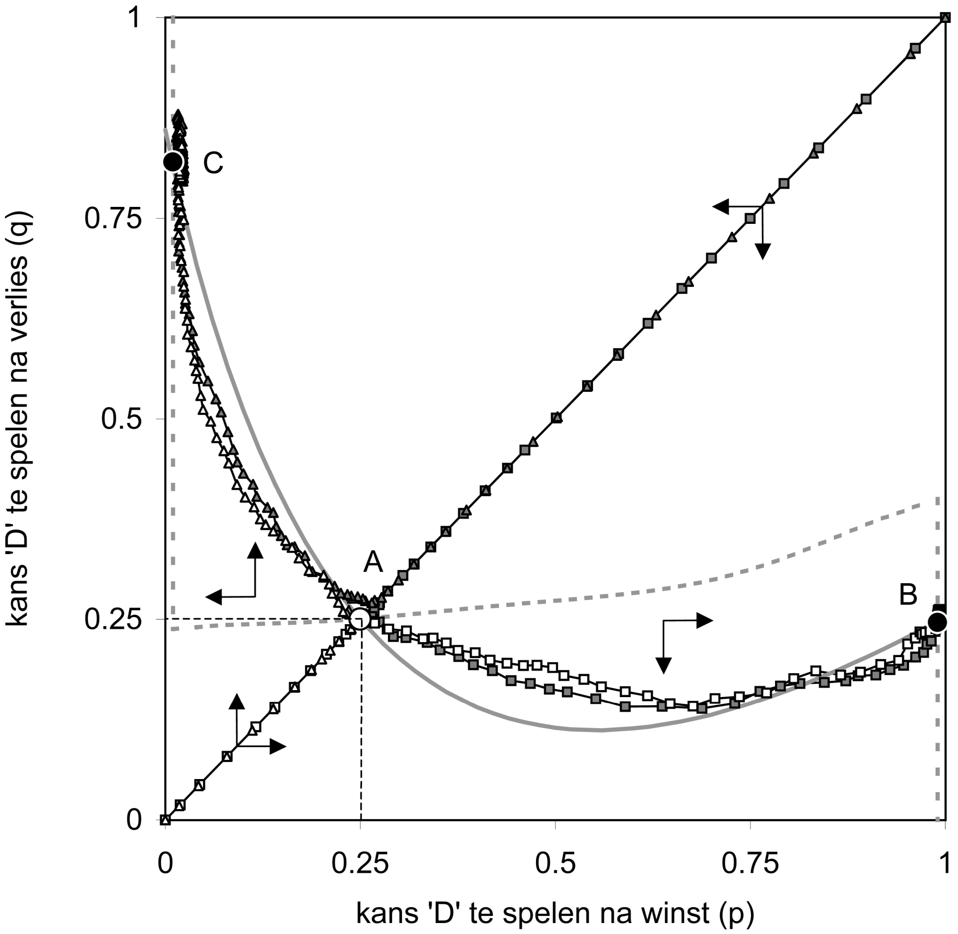
Bestaat er zo'n evolutionair stabiele strategie in ons model? Als we de mogelijke strategieën beperken tot heel simpele strategieën, waarbij individuen bij elk conflict dreigen met kans p, en het conflict ontwijken met kans $1-p$, kan zo'n strategie eenvoudig grafisch worden gevonden (FIGUUR 15).

In interessantere gevallen, waarbij individuen hun gedrag afhankelijk maken van de uitkomst van conflicten in het verleden, kunnen we de evolutionair stabiele strategieën uitrekenen met behulp van computer simulaties. De meest eenvoudige manier waarop individuen hun gedrag kunnen laten bepalen door de uitkomst van voorgaande conflicten is door hun gedrag afhankelijk te maken van de uitslag van het laatste conflict dat ze met hun huidige tegenstander hadden. Dat kan door in het huidige conflict 'D' te spelen met kans p als ze de vorige keer wonnen en met kans q als ze de vorige keer verloren.

Zoals FIGUUR 16 laat zien, zijn er twee belangrijke evolutionair stabiele oplossingen binnen deze categorie van strategieën. In een populatie waarin strategie B van FIGUUR 16 is geëvolueerd blijven winnaars van conflicten zich agressief gedragen in volgende conflicten, terwijl verliezers van voorgaande conflicten bij het volgend conflict het gevecht zullen ontwijken. Daardoor blijven winnaars winnen en verliezers verliezen. Elke serie van conflicten tussen twee individuen zal dus onherroepelijk ontaarden in een macho-nerd relatie. Dit is anders in een populatie waarin strategie C van FIGUUR 16 is geëvolueerd. Daar zijn het juist de verliezers van het voorgaande gevecht die gaan dreigen, en de winnaars die zich koest houdt. Als gevolg daarvan wisselen individuen elkaar af in hun rol als winnaar of verliezer, waardoor een eerlijke verdeling van fitnesspunten over de spelers ontstaat.

WAAROM DE NERD ZICH SCHIKKEN MOET

Terug nu naar onze oorspronkelijk vraagstelling. Waarom schikt een onderdanig individu zich in zijn ongunstige positie? Onze theoretische analyse heeft laten zien dat afspraken zoals het winnaar- en verliezer-effect evolutionair stabiele strategieën zijn, zelfs als winst of verlies helemaal niets zegt over krachtsverschillen tussen individuen. De evolutionaire stabiliteit impliceert dat het niet loont (zelfs niet voor iemand onder aan de rangorde) van de afpraak af te wijken. De situatie van een nerd is vergelijkbaar met die van een Nederlandse automobilist die een auto bestuurt in Groot-Brittanië. Ook al zou hij liever aan de



FIGUUR 16 – EVOLUTIE VAN EEN WINNAAR- EN VERLIEZER-EFFECT

Als de kans 'D' te spelen na verlies (q) in evolutie niet zou veranderen, dan zouden we, net als in FIGUUR 15, opnieuw de evolutionair stabiele waardes van de kans 'D' te spelen na winst (p) kunnen bepalen. Deze evolutionair stabiele p-waardes bij vaste q zijn weergegeven door de grijze stippellijn. Omgekeerd kunnen we ook de evolutionair stabiele q bij vaste p uitrekenen (grijze lijn). De snijpunten van deze twee lijnen zijn de evolutionair stabiele strategieën (cirkels, A-C) voor het geval zowel p als q kan evolueren. De lijnen met driehoekjes en vierkantjes geven vier computer simulaties weer die gestart zijn vanuit $(0,0)$ (wit) of $(1,1)$ (grijs). In het begin van de computer simulatie hebben we opgelegd dat individuen hetzelfde reageren na winst en verlies, dus $p = q$. We zijn dan in dezelfde situatie als in FIGUUR 15, en evolueren langs de diagonaal naar de evolutionair stabiele strategie A, bij $(0.25, 0.25)$. Dit is dezelfde evolutionair stabiele strategie als die van FIGUUR 15. Het loont niet van de populatie strategie af te wijken als de populatie zich precies in A bevindt. Dit verandert zodra de populatie zelfs maar een klein stukje van A verwijdert raakt. Als dat gebeurt, en p en q zijn beide vrij te evolueren, dan evolueert de populatie weg van A (pijltjes geven de richting van evolutie aan). In de helft van de gevallen eindigt evolutie in evolutionair stabiele strategie B (vierkantjes), in de andere helft van de gevallen in C (driehoekjes). In B blijven winnaars vechten, en verliezers spelen vaak 'O'. Daardoor heeft de winnaar een grote kans het volgende conflict opnieuw te winnen. Er is in B dus sprake van een winnaar- verliezer-effect.

goede kant van de weg rijden (macho zijn), hij doet er beter aan zich te schikken in de afspraak zoals die nu eenmaal in Groot-Brittanië geldt. Het nadeel dat hij ondervindt doordat hij gedwongen is aan de verkeerde kant van de weg te rijden, is kleiner dan de schade die hij als spookrijder zou oplopen.

Deze analogie maakt het ook meteen duidelijk waarom je een conflict-oplossingsstrategie die gebaseerd is op dominantie niet zomaar kan vervangen door een andere afspraak, zoals strategie C van FIGUUR 16. Deze strategie is eerlijker (iedereen krijgt evenveel) en ook beter (in de populatie als geheel wordt nooit gevochten!) dan de dominatie strategie B van FIGUUR 16, maar desondanks kan deze strategie de dominatie strategie niet verdringen als niet iedereen tegelijk bereid is van strategie B naar C te wisselen. Het omgekeerde is trouwens ook waar, en dat brengt ons op de vraag waarom we in de natuur geen soorten vinden die de ‘eerlijk delen’ strategie C spelen. Wie ideeën heeft, mag het zeggen, want die vraag is nog open. Mijn voorlopig idee, gesteund door computersimulaties, is dat de eerlijk delen strategie veel gevoeliger is voor vergissingen en dat deze daarom niet zo makkelijk evolueert.

Verschillende toespelingen, waaronder de titel van dit stuk, suggereren dat deze resultaten ook iets zeggen over asymmetrische machtsrelaties bij mensen. Ik ben niet bang deze suggestie te wekken, omdat de mens een sociale soort is waarin evolutionaire selectiekrachten werken die niet principieel verschillend lijken van de selectiekrachten die werken in andere sociaal levende diersoorten (waarop dit soort modellen binnen de biologie van toepassing worden geacht). Natuurlijk moeten we heel voorzichtig zijn met het al te direct vertalen van de resultaten van simpele modellen naar de ingewikkelde werkelijkheid, vooral als het gaat om ons verfijnde sociaal gedrag. Desalniettemin kunnen deze resultaten bij macho’s wellicht wat bescheidenheid teweegbrengen (ze hebben gewoon mazzel gehad), en bij nerds wat trots (ze zijn in ieder geval niet te slap om te winnen).

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In het oog, uit het hart –

sympatrische soortvorming door seksuele selectie

Nederlandse Samenvatting — Dutch Summary

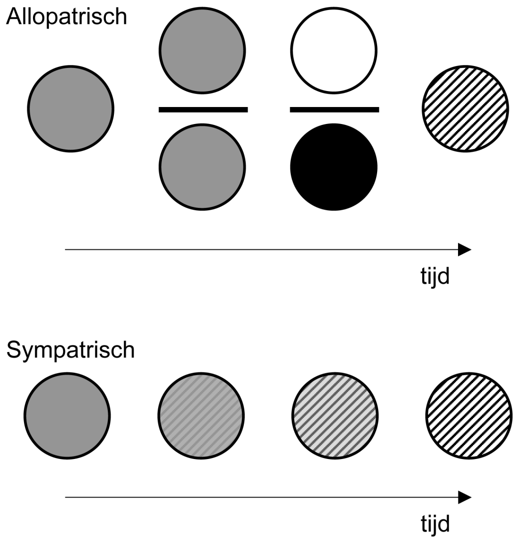
De aandacht van dit proefschrift richt zich niet op de takken van de evolutionaire boom, maar op de vertakkingspunten. Het proces dat daar optreedt wordt kortweg *soortvorming* genoemd. Soortvorming is de opsplitsing van een bestaande soort in twee nieuwe dochtersoorten. Anders dan men zou wensen voor zo'n fundamenteel evolutionair proces, wordt soortvorming nog niet goed begrepen.

SYMPATRISCHE SOORTVORMING

De meest gangbare theorie voor soortvorming is die van *allopatrische* soortvorming. Aanhangers van deze theorie menen dat soortvorming alleen kan optreden in populaties die ruimtelijk van elkaar zijn gescheiden. Denk bijvoorbeeld aan populaties stekelbaarzen in twee verschillende meren, of aan populaties regenwormen op twee verschillende eilanden. 'Allopatrisch' (uit het Grieks) kan vrij vertaald worden als 'in verschillende leefgebieden'. Allopatrische soortvorming wordt in gang gezet door invloeden van buitenaf (zoals de vorming van een gletsjer of de verandering van de loop van een rivier) die ervoor zorgen dat populaties ruimtelijk van elkaar gescheiden raken. Pas later, als de ruimtelijke scheiding gedurende langere tijd in stand blijft, zullen de populaties, alleen al door toevallige mutaties, ook erfelijk van elkaar gaan verschillen (FIGUUR 2).

FIGUUR 2 – TWEE THEORIEËN VOOR SOORTVORMING

Bij allopatrische soortvorming ontstaan erfelijke verschillen (weergegeven als verschillende grijstinten) nadat een populatie (gesymboliseerd door een cirkel) in twee delen is gesplitst door een geografische barrière. Als de ruimtelijke scheiding tussen de populaties lang genoeg in stand blijft, ontstaan twee nieuwe soorten, die naast elkaar blijven voortbestaan als de geografische barrière weer verdwijnt. Sympatrische soortvorming, daarentegen treedt op binnen een enkele populatie, waarin door selectie geleidelijk erfelijke variatie ontstaat.



Niet alle biologen zijn even gelukkig met de allopatrische theorie van soortvorming. Vooral het feit dat allopatrische soortvorming sterk afhankelijk is van onvoorspelbare gebeurtenissen (zoals de vorming van een gletsjer) en toevalsprocessen stuit sommigen tegen de borst. Immers, deze afhankelijkheid maakt evolutie tot een onvoorspelbaar proces dat in belangrijke mate gestuurd wordt door invloeden van buitenaf. Bovendien verloopt allopatrische soortvorming erg langzaam. Populaties moeten lange tijd ruimtelijk gescheiden blijven voordat ze door willekeurige mutaties zo sterk van elkaar zijn gaan verschillen dat ze bij secundair contact niet meer zullen kruisen.

Als alternatief stellen tegenstanders van de allopatrische theorie daarom voor dat soortvorming primair een gevolg is van selectie, en dat ruimtelijke scheiding helemaal niet nodig is. Soortvorming wordt daarmee een van binnenuit gedreven proces, en dat maakt evolutie in beginsel voorspelbaar. Dit idee vormt de kern van de theorie van *sympatrische* soortvorming ('sympatrisch' betekent vrij vertaald 'in hetzelfde leefgebied') (FIGUUR 2). Aanhangers van de sympatrische theorie hebben voor een aantal specifieke biologische situaties aannemelijk gemaakt dat sympatrische soortvorming mogelijk is. Desondanks werd, tot voor kort, sympatrische soortvorming door de meerderheid van biologen verworpen als een aannemelijke algemene verklaring voor soortvorming. Die afwijzing was grotendeels gebaseerd op wiskundige modellen die formeel leken aan te tonen dat sympatrische soortvorming verre van vanzelfsprekend was.

De waardering voor de theorie van sympatrische soortvorming is de laatste jaren sterk toegenomen. Nieuwe gegevens, beschikbaar gekomen dankzij de ontwikkeling van moleculair genetische technieken, zijn daarvoor mede verantwoordelijk. Op grond van die gegevens blijkt in een aantal gevallen de sympatrische theorie van soortvorming beter in staat de evolutionaire relaties tussen soorten te verklaren dan de allopatrische theorie. Een fascinerend voorbeeld daarvan vinden we in kleine Afrikaanse kratermeertjes. Sommige van die meertjes bezitten een opmerkelijke soortenrijkdom aan cichliden (tropische vissen). Elk van de soorten heeft zich gespecialiseerd op een verschillende ecologische rol in het meer. Oorspronkelijk werd gedacht dat deze soorten afkomstig waren uit naburige riviersystemen en dus niet direct verwant zouden zijn. Op grond van de moleculair genetische gegevens blijkt echter dat de soorten in een meertje zeer nauw verwant zijn, en hoogst waarschijnlijk in het kratermeertje zelf uit elkaar zijn ontstaan. Het onderzochte kratermeertje is alleen zo klein dat het nauwelijks is voor te stellen dat daarin ruimtelijke scheiding van populaties mogelijk is. Dit moet wel betekenen dat de diversiteit aan cichliden in het kratermeertje door sympatrische soortvorming is ontstaan.

Deze en andere bevindingen hebben een nieuwe impuls gegeven aan het theoretisch onderzoek naar sympatrische soortvorming. Ook dit proefschrift draagt daaraan bij. Ik laat zien dat de nieuwe generatie wiskundige modellen eigenlijk deeloplossingen aanlevert voor de belangrijkste theoretische bezwaren tegen sympatrische soortvorming. In tegenstelling tot de gangbare benadering, waarin verschillende modellen tegen elkaar worden afgezet als elkaar uitsluitende alternatieven, kies ik ervoor de verschillende deeloplossingen met elkaar te combineren. Mijn proefschrift richt zich daarom op het samenspel van *frequentie-afhankelijke selectie* en *seksuele selectie* – twee sleutelbegrippen die in de afzonderlijke deeloplossingen centraal staan, en die ik in de loop van deze samenvatting gedetailleerd zal bespreken. Met behulp van wiskundige methoden laat ik zien dat deze factoren in combinatie soortvorming door selectie mogelijk maken. Hoewel dit aantoont dat de theoretische bezwaren tegen sympatrische soortvorming overkomelijk zijn, wil dit nog niet zeggen dat sympatrische soortvorming ook vaak zal optreden. Integendeel, het lijkt erop dat het juiste samenspel tussen frequentieafhankelijke en seksuele selectie slechts onder specifieke omstandigheden wordt gerealiseerd.

DISRUPTIEVE SELECTIE

Soorten verschillen van elkaar in één of meerdere kenmerken, en soortvorming gaat altijd gepaard met het ontstaan van variatie in erfelijke eigenschappen. Deze erfelijke variatie kan ontstaan doordat een populatie selectiekrachten in tegenovergestelde richtingen ondervindt (zogenoemde *disruptieve* selectie). Sterke disruptieve selectie kan een populatie in twee stukken uiteen scheuren, maar daarvoor is het wel nodig dat disruptieve selectie langdurig op de populatie inwerkt.

Hier ligt een probleem, want een populatie onder invloed van disruptieve selectie is in veel opzichten vergelijkbaar met een bal op een top van een heuvel. Als de bal precies op de top ligt kan hij nog aan twee verschillende kanten naar beneden rollen, maar zodra de bal ook maar iets bij de top vandaan beweegt, is het duidelijk aan welk van beide zijden hij zal wegrollen. Evenzo kan ook een populatie eenvoudig aan de invloed van disruptieve selectie ontsnappen, zonder op te splitsen, door ofwel in de ene richting, ofwel in de andere richting te evolueren.

Hoe kunnen we voorkomen dat een populatie op deze manier aan de invloed van disruptieve selectie ontsnapt? Het antwoord op deze vraag vinden we in zogeheten *ecologische modellen van soortvorming*, die veel aandacht hebben gegeven aan het probleem van disruptieve selectie. De oplossing die zij aandragen laat zich in twee woorden samenvatten: *frequentieafhankelijke selectie*. Selectie is frequentieafhankelijk, wanneer de fitness die bij een bepaalde eigenschap hoort, niet constant is, maar afhankelijk van de eigenschappen van andere individuen in de populatie (zoals bij gezelschapsspelletjes waarbij het succes van een strategie mede bepaald wordt door de strategie van de tegenstanders). Als gevolg daarvan veranderen de richting en de kracht van selectie bij elke evolutionaire aanpassing die de populatie ondergaat. Frequentieafhankelijke selectie kan een populatie, telkens als die dreigt te ontsnappen aan de invloed van disruptieve selectie, terugbrengen in de toestand waarin disruptieve selectie werkzaam is – een veelbelovende situatie als het gaat om soortvorming. Een populatie in die toestand is vergelijkbaar met een bal die langdurig in evenwicht wordt gehouden op het topje van een vinger: telkens als de bal dreigt weg te rollen, wordt, door snel met de vinger te bewegen, het krachtenspel dat op de bal inwerkt aangepast, waardoor het evenwicht kan worden hersteld.

REPRODUCTIEVE ISOLATIE

Als een populatie door frequentieafhankelijke selectie gevangen wordt gehouden onder de invloed van disruptieve selectie is aan een belangrijke voorwaarde voor soortvorming voldaan. Echter, soortvorming is dan nog niet gegarandeerd. Een sterke kracht werkt het opsplitsen van de populatie tegen. Een populatie hangt namelijk niet als los zand aan elkaar maar vormt een sterk samenhangend geheel.

Die innerlijke samenhang is een gevolg van geslachtelijke voortplanting. Elke generatie ontstaan nieuwe combinaties van erfelijke eigenschappen door de vermenging van het erfelijk materiaal van de ouders in hun nakomelingen. Deze vermenging is zeer efficiënt in het laten samenvloeien van soorten. Zozeer zelfs, dat de afwezigheid van uitwisseling van erfelijke informatie gebruikt wordt als belangrijk criterium bij de indeling van organismen in verschillende soorten. Alle populaties die direct of indirect via geslachtelijke voortplanting erfelijke informatie uitwisselen behoren volgens dit criterium tot dezelfde soort. Een soort is dan ook altijd *reproductief geïsoleerd* van andere soorten, d.w.z., tussen verschillende soorten vindt geen uitwisseling van erfelijke informatie plaats. Dit houdt in dat soortvorming alleen mogelijk is wanneer, tijdens het opsplitsen van de populatie door disruptieve selectie, ook meteen reproductieve isolatie ontstaat.

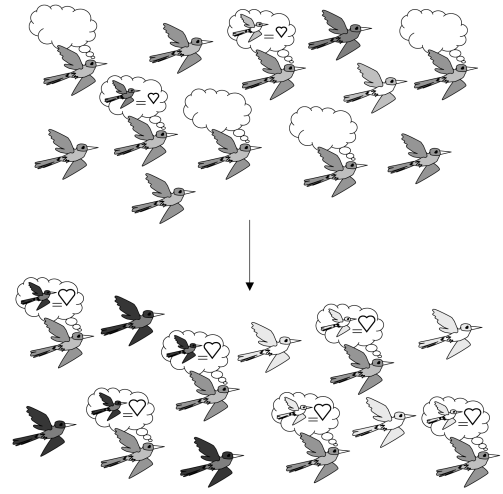
De ecologische modellen voor soortvorming wijden nauwelijks aandacht aan de mechanismen die ten grondslag liggen aan de evolutie van reproductieve isolatie. Dit is een onderwerp dat veeleer centraal staat in zogeheten seksuele selectie modellen voor soortvorming. Deze modellen richten zich op reproductieve isolatie als gevolg van selectieve partnerkeuze. Selectieve partnerkeuze komt voor in een groot aantal soorten. In de meeste gevallen zijn het de vrouwtjes die kieskeurig zijn in het uitzoeken van een partner. Nauw verwante soorten verschillen vaak opmerkelijk sterk in eigenschappen die een rol spelen bij selectieve partnerkeuze, en de soortenrijkdom is vaak hoog in groepen van soorten waarin selectieve partnerkeuze een belangrijke rol speelt. Veel biologen zien dit als aanwijzing dat selectieve partnerkeuze en soortvorming oorzakelijk samenhangen. *Seksuele selectie*, het tweede sleutelbegrip dat in dit proefschrift centraal staat, wordt bij uitstek verantwoordelijk gehouden voor de evolutie van selectieve partnerkeuze. Het lijkt dan ook voor de hand te liggen dat seksuele selectie een grote invloed heeft op soortvorming.

SEKSUELE SELECTIE

Seksuele selectie leidt, net als natuurlijke selectie, tot betere aanpassing van organismen aan de leefomstandigheden waarin ze zich bevinden. Bij seksuele selectie gaat het daarbij om aanpassingen die het individu succesvoller maken in de competitie om partners. In veel gevallen gaan die aanpassingen gepaard met een verlaging van de overlevingskans. Met andere woorden, seksuele en natuurlijke selectie werken elkaar vaak tegen.

Dit is ook het geval bij selectieve partnerkeuze. Als vrouwtjes bepaalde voorkeuren ontwikkelen (bijvoorbeeld, een voorkeur voor een gekleurd verenkleed) ontstaat een sterke seksuele-selectiedruk op de mannetjes om aan die voorkeur te voldoen. Zolang het seksuele-selectievoordeel groot genoeg is zullen de mannetjes daarom een kleurig verenkleed te ontwikkelen, zelfs als dit zou leiden tot een lagere overlevingskans. Evenzo is het voor een vrouwtje voordelig kieskeurig te zijn, zelfs als ze daardoor langer naar een geschikte partner moet zoeken (met alle risico's van dien). Een kieskeurig vrouwtje paart namelijk met een aantrekkelijke man. Hij zal zijn genen doorgeven aan zijn zoons, die daardoor waarschijnlijk ook aantrekkelijk zullen zijn, en een hoog voortplantingssucces zullen genieten. Een vrouwtje profiteert dus indirect van haar kieskeurigheid dankzij het hogere voortplantingssucces van haar zoons.

Onder de juiste omstandigheden kan dit proces zichzelf versterken, waardoor binnen zeer korte tijd bij de vrouwtjes sterke paringsvoorkeuren ontstaan en bij de mannetjes daarop aangepaste secundaire geslachtskenmerken (zoals een kleurig verenkleed). Dit proces verloopt als een mode gril, waarbij een bepaald kledingstuk populair wordt omdat iedereen het draagt, en niet zozeer omdat de kwaliteit of de functionaliteit van het kledingstuk zoveel beter is dan die van de alternatieven. Anders gezegd, seksuele selectie kan leiden tot selectieve partnerkeuze op grond van willekeurige kenmerken. Het is, bijvoorbeeld, evengoed mogelijk dat paringsvoorkeuren ontstaan voor een licht verenkleed, dan dat ze ontstaan voor een donker, of een gestreept verenkleed. Deze constatering is interessant als het gaat om soortvorming. Immers, als seksuele selectie kan leiden tot de snelle evolutie van verschillende partnervoorkeuren is het misschien ook wel mogelijk dat verschillende partnervoorkeuren op hetzelfde moment binnen één populatie ontstaan.



FIGUUR 3 – SOORTVORMING DOOR SELECTIEVE PARTNERKEUZE

Als er variatie in paringsvoorkeuren bestaat, kan seksuele selectie in verschillende richtingen tegelijk verlopen. In dit voorbeeld hebben aanvankelijk de meeste vrouwtjes geen paringsvoorkeur. Enkele vrouwtjes hebben echter een voorkeur voor lichtere of donkerdere mannetjes. In de loop van de tijd ontstaan door seksuele selectie tegelijkertijd steeds sterkere paringsvoorkeuren voor lichte en donkere mannetjes. Binnen de populatie zijn nu twee groepen ontstaan die niet langer onderling kruisen.

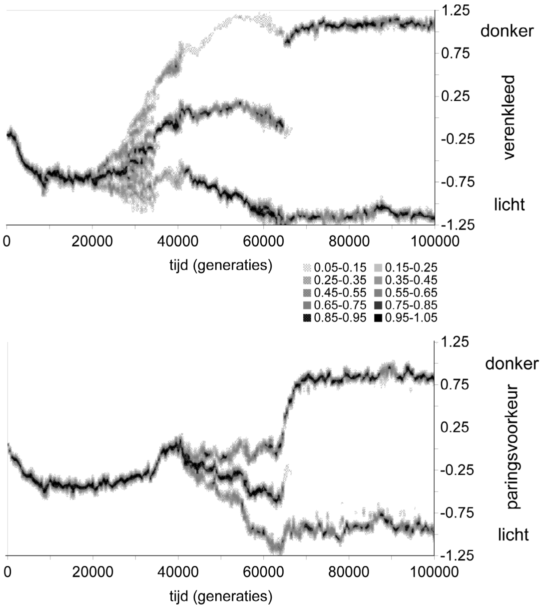
Seksuele selectie modellen voor soortvorming hebben kortgeleden aangetoond dat deze mogelijkheid inderdaad bestaat. De modellen gaan uit van een populatie waarbinnen variatie bestaat in de paringsvoorkeuren van vrouwtjes. Sommige vrouwtjes hebben een voorkeur voor een licht verenkleed, andere voor een donker verenkleed (FIGUUR 3). De variatie in paringsvoorkeuren genereert disruptieve seksuele selectie op de mannetjes, die alleen maar toeneemt in kracht als de paringsvoorkeuren van de vrouwtjes sterker worden. Door het zelfversterkende proces van seksuele selectie dat aan de evolutie van paringsvoorkeuren ten grondslag ligt, kan de populatie snel opsplitsen in twee delen die door selectieve partnerkeuze reproductief van elkaar zijn geïsoleerd. Uiteindelijk bestaat het ene deel van de populatie uit mannetjes met een lichte kleur en vrouwtjes met een voorkeur voor een licht verenkleed, en het andere deel uit mannetjes met een donkere kleur en vrouwtjes met een voorkeur voor een donker verenkleed (FIGUUR 3).

Vanwege de sterke reproductieve isolatie die binnen één populatie kan ontstaan door uiteenlopende paringsvoorkeuren, is gesuggereerd dat het zelfversterkende proces van seksuele selectie op paringsvoorkeuren gemakkelijk kan leiden tot sympatrische soortvorming. Ten onrechte, want een nauwkeuriger bestudering van de modellen laat zien dat de twee 'dochtersoorten' die door seksuele selectie ontstaan niet stabiel met elkaar kunnen samenleven. Concurrentie tussen beide soorten zorgt ervoor dat al heel snel één van de soorten verdwijnt. Dit gebeurt sneller naarmate de variatie van paringsvoorkeuren in de populatie kleiner is. Zonder aanzienlijke variatie van paringsvoorkeuren wordt het proces van soortvorming niet eens opgestart. De populatie ontsnapt dan als het ware aan disruptieve seksuele selectie door meteen ofwel uitsluitend paringsvoorkeur voor een licht verenkleed, ofwel uitsluitend paringsvoorkeur voor een donker verenkleed te ontwikkelen. Deze uitkomst wordt verklaard door een probleem dat we al eerder tegenkwamen: een populatie kan vaak eenvoudigweg van de invloed van disruptieve selectie ontsnappen zonder op te splitsen.

CONCLUSIES VAN DIT PROEFSCHRIFT

Op grond van bovenstaande overwegingen is maar één slotsom mogelijk. Seksuele selectie modellen van sympatrische soortvorming geven een verklaring voor de evolutie van reproductieve isolatie, maar zijn onvolledig door een gebrek aan frequentieafhankelijke selectie. Immers, frequentieafhankelijke selectie kan voorkomen dat een populatie zonder op te splitsen ontsnapt aan de invloed van disruptieve selectie. Frequentieafhankelijke selectie alleen is echter onvoldoende voor soortvorming, omdat soortvorming ook de evolutie van reproductieve isolatie vereist. Dit onderwerp ontvangt juist weer weinig aandacht in de ecologische modellen van soortvorming. Ik concludeer dan ook dat sympatrische soortvorming een samenspel vereist van seksuele en frequentieafhankelijke selectie, dat alleen kan worden gerealiseerd als de ecologische en seksuele selectie aspecten van soortvorming met elkaar zijn verweven. Deze conclusie gaat in tegen de gebruikelijke benadering, waarin ecologische en seksuele selectie modellen worden afgeschilderd als elkaar uitsluitende alternatieven.

Het samenspel van frequentieafhankelijke en seksuele selectie wordt onderzocht in de hoofdstukken 2 t/m 5 van dit proefschrift. In hoofdstukken 2 en 3 combineer ik ecologische en seksuele selectie modellen van soortvorming op de meest eenvoudige manier, door een model te ontwikkelen waarin zowel seksuele selectie als frequentieafhankelijke natuurlijke selectie een rol spelen. HOOFDSTUK 2 richt zich daarbij op een specifiek voorbeeld en HOOFDSTUK 3 op een meer algemene analyse. Het model laat zien hoe seksuele en frequentieafhankelijke selectie samen kunnen zorgen voor reproductieve isolatie en stabiele samenleving van de dochtersoorten na soortvorming. Het gecombineerde model biedt echter nog geen volledige oplossing voor soortvorming door selectie. De populatie kan namelijk pas opsplitsen zodra voldoende variatie in paringsvoorkeuren bestaat. Selectie alleen blijkt niet in staat die variatie in paringsvoorkeuren te veroorzaken; het model is daarvoor afhankelijk van de variatie die door mutatie in de populatie ontstaat.



FIGUUR 4 – SYMPATRISCHE SOORTVORMING DOOR SEKSUELE SELECTIE

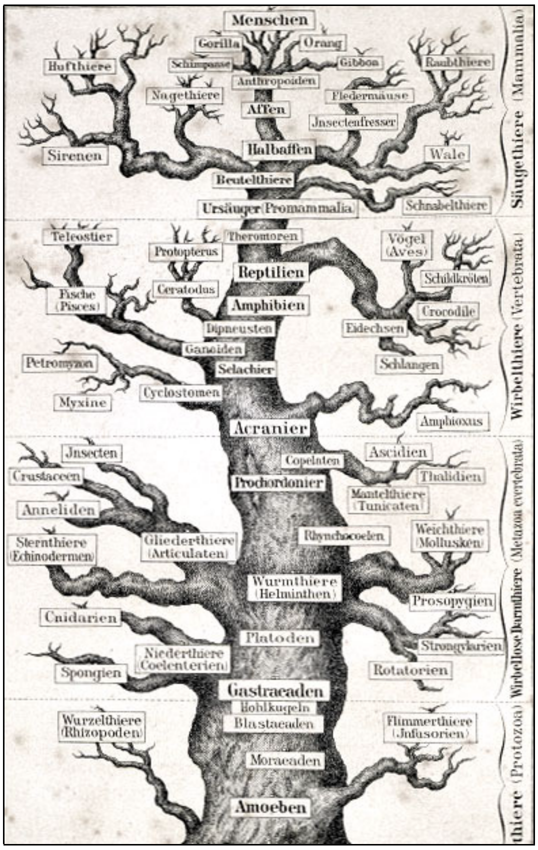
Deze simulatie uit HOOFDSTUK 4 van dit proefschrift laat zien dat seksuele selectie kan leiden tot sympatrische soortvorming. In de loop van de tijd ontstaan twee verschillende paringsvoorkeuren bij de vrouwtjes (onderste deelfiguur) en daarop aangepaste kenmerken bij de mannetjes (bovenste deelfiguur). Voor dit resultaat moest het standaard model voor selectieve partnerkeuze aanzienlijk worden uitgebreid: in deze simulatie is er sterke competitie tussen vrouwtjes om beschikbare mannetjes en sterke competitie tussen de mannetjes om toegang tot de vrouwtjes.

In de hoofdstukken 4 en 5 van dit proefschrift combineer ik niet de modellen zelf, maar de inzichten van ecologische en seksuele selectie modellen. In deze hoofdstukken gaat het om de vraag in hoeverre seksuele selectie zelf kan leiden tot frequentieafhankelijkheid. HOOFDSTUK 4 maakt duidelijk dat standaard seksuele selectie modellen niet tot frequentieafhankelijke selectie kunnen leiden. Pas als het standaardmodel aanzienlijk wordt uitgebreid, kunnen aan alle voorwaarden voor soortvorming door selectie worden voldaan (FIGUUR 4). De resultaten van hoofdstuk 4 worden bevestigd door een algemene analyse in HOOFDSTUK 5. Dit hoofdstuk leidt tot de slotsom dat frequentieafhankelijke seksuele selectie kan ontstaan in slechts twee gevallen. Het eerste geval vereist dat zowel mannetjes als vrouwtjes een belangrijk deel van hun tijd investeren in de productie van nakomelingen en daarbij tegengestelde belangen hebben. Het tweede geval vereist competitie tussen mannetjes onderling om toegang tot de vrouwtjes, maar ook competitie tussen vrouwtjes onderling om toegang tot de mannetjes.

DE EVOLUTIONAIRE STAMBOOM

Het aantal verschillende soorten organismen dat op aarde leeft, wordt geschat op ca. 10-100 miljoen. Volgens evolutiebiologen zijn al die verschillende soorten ontstaan door het splitsen van reeds bestaande soorten. Overeenkomstig dit idee wordt het leven op aarde gerangschikt volgens een evolutionaire stamboom (FIGUUR 1), die aangeeft welke soort, op welk moment, uit welke vooroudersoort is ontstaan.

Evolutionaire stambomen bestaan uit takken en vertakkingspunten. Het proces dat optreedt langs de takken van de boom wordt sinds Darwin, de grondlegger van de evolutietheorie, en Mendel, de grondlegger van de erfelijkheidsleer, goed begrepen: de erfelijke eigenschappen van soorten veranderen geleidelijk door *mutatie*, waarbij nieuwe varianten ontstaan door willekeurige veranderingen in het erfelijk materiaal, en *selectie*, waarbij individuen die slecht zijn aangepast het veld moeten ruimen voor individuen die beter zijn toegerust in de strijd om het bestaan. Mutatie en selectie leiden samen tot aanpassing van organismen aan de leefomstandigheden waarin deze zich bevinden.



FIGUUR 1 – EEN EVOLUTIONAIRE STAMBOOM

Deze evolutionaire stamboom is gemaakt door Ernst Häckel (Häckel, 1874), die als een van de eersten organismen volgens een boom rangschikte. Häckel baseerde zijn stamboom op uiterlijke kenmerken en stelde zich evolutie voor als een proces dat tot steeds ingewikkelder levensvormen leidt. Om die reden plaatste hij de mens in het uiterste topje van de boom. Tegenwoordig worden evolutionaire stambomen bepaald op basis van moleculair genetische gegevens die verkregen worden uit DNA-analyse. In een moderne stamboom staat de lengte van de takken voor de hoeveelheid genetische verschillen. De mate van verwantschap tussen twee willekeurige soorten A en B groter is naarmate de kortste route tussen A en B langs de boom korter is. Aan de uiteinden van de takken staan de nu levende soorten, dieper in de boom verscholen vinden we hun gemeenschappelijke voorouders. De moderne, exacte methode van het vaststellen van evolutionaire stambomen heeft ondertussen aangetoond dat Häckel's stamboom onjuist is.

De hoofdstukken 6 t/m 11 van dit proefschrift behandelen niet soortvorming zelf, maar verschillende deelprocessen die van belang zijn om soortvorming beter te begrijpen. HOOFDSTUK 6, bijvoorbeeld, richt zich op de vraag hoe precies de erfelijke variatie toeneemt in een populatie onder invloed van frequentie-afhankelijke disruptieve selectie. De HOOFDSTUKKEN 7 T/M 9 onderzoeken alternatieve seksuele selectie mechanismen die ook leiden tot selectieve partnerkeuze. De paringsvoorkeuren die daarin een rol spelen zijn niet gericht op willekeurige kenmerken, maar op eigenschappen die vrouwtjes in staat stellen een partner te kiezen met 'goede genen', d.w.z, met een hoge kwaliteit. Deze hoofdstukken dragen bij aan een beter begrip van seksuele selectie voor 'goede genen', en suggereren daarnaast verschillende mogelijkheden waarop dit type seksuele selectie kan bijdragen aan soortvorming, bijvoorbeeld door het versterken van bestaande disruptieve selectie. De HOOFDSTUKKEN 10 EN 11, tenslotte, richten zich op de evolutie van sociale dominantieverhoudingen. De resultaten van dit hoofdstuk zijn van belang voor het beter begrijpen van competitie tussen mannetjes om toegang tot partners, een factor die ook voor soortvorming van belang is, zoals we in de hoofdstukken 4 en 5 hebben gezien.

In dit proefschrift laat ik zien dat het samenspel van frequentieafhankelijke en seksuele selectie ook daadwerkelijk soortvorming door selectie mogelijk maakt. Dit toont aan dat de theoretische bezwaren tegen sympatrische soortvorming overkomelijk zijn, en dat soortvorming, onder de juiste omstandigheden, begrepen kan worden als een voorspelbaar proces. In welke mate deze conclusie consequenties heeft voor het algemene beeld dat we van soortvorming hebben, hangt in belangrijke mate af van het voorbehoud 'onder de juiste omstandigheden'. Het voor sympatrische soortvorming noodzakelijke samenspel tussen frequentieafhankelijke- en seksuele selectie lijkt alleen te worden gerealiseerd onder bijzondere biologische omstandigheden. De overwegingen in dit proefschrift doen vermoeden dat competitie tussen individuen van dezelfde sekse en tegenstrijdige belangen bij de productie van nakomelingen daaraan een essentiële bijdrage leveren. Deze, en andere voor soortvorming noodzakelijke deelprocessen, worden niet alleen theoretisch slechts ten dele begrepen (zie de hoofdstukken 6 t/m 11), ze zijn ook nog nauwelijks in hun relatie tot soortvorming onderzocht in natuurlijke populaties. Een definitief oordeel over de waarschijnlijkheid van sympatrische soortvorming is daarom pas mogelijk nadat meer gegevens van natuurlijke populaties beschikbaar zijn gekomen. Voorlopig lijkt echter de conclusie gerechtvaardigd dat sympatrische soortvorming slechts kan optreden onder uitzonderlijke omstandigheden.

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Author Affiliations and Addresses

ULF DIECKMANN

- — Adaptive Dynamics Network, International Institute for Applied Systems Analysis, Schlossplatz 1, 2361 Laxenburg, Austria.
- — Section Theoretical Biology, Institute of Biology, Leiden University, Kaiserstraat 63, 2311 GP Leiden, The Netherlands.

PIM EDELAAR

- — Theoretical Biology Group, Center for Ecological and Evolutionary Studies, University of Groningen, Kerklaan 30, 9751 NN Haren, The Netherlands.
- — Department of Biology, MSC 3AF, New Mexico State University, Las Cruces, NM 88003, USA.
- — Department of Zoology, University of British Colombia, Vancouver, BC V6T 1Z4, Canada.

PIETERNELLA C. LUTTIKHUIZEN

- — Evolutionary Genetics Group, Center for Ecological and Evolutionary Studies, University of Groningen, Kerklaan 30, 9751 NN Haren, The Netherlands.
- — Netherlands Institute for Sea Research (NIOZ), P.O. Box 59, 1790 AB Den Burg, Texel, The Netherlands.
- — Current address: Institute for Biodiversity and Ecosystem Dynamics, University of Amsterdam, P.O. Box 94062, 1090 GB Amsterdam, the Netherlands.

GEERTEN M. HENGEVELD

- — Theoretical Biology Group, Center for Ecological and Evolutionary Studies, University of Groningen, Kerklaan 30, 9751 NN Haren, The Netherlands.
- — Current address: Resource Ecology Group, Center for Ecosystem Studies, Wageningen University, Bornsesteeg 69, Building 119, 9708 PD Wageningen, the Netherlands.

G. SANDER VAN DOORN

- — Theoretical Biology Group, Center for Ecological and Evolutionary Studies, University of Groningen, Kerklaan 30, 9751 NN Haren, The Netherlands.

FRANZ J. WEISSING

- — Theoretical Biology Group, Center for Ecological and Evolutionary Studies, University of Groningen, Kerklaan 30, 9751 NN Haren, The Netherlands.

O Lord, how great is the number of your works! In
wisdom you have made them all; the earth is full
of the things you have made.
If you send out your spirit, they are created; you
make new the face of the earth.

— PSALMS 140: 24, 30 —

The rainbow, to which the cover of this book makes reference, connects the sky to the earth. According to the Bible, God established the rainbow as a symbol of his promise of mercy to every living creature on the earth after the Deluge. I know how rainbows arise, but this does not withhold me from feeling grateful every time I see one. In the same way, my work as an evolutionary biologist, which has resulted in this thesis, does not withhold me to be a Christian. In fact, I have never entirely understood why so many people lose their admiration once they remotely understand how something works. In fact, the only way for me to make sense of the world is to believe in God, who created the earth and preserved it ever since; I can come to him thanks to his son Jesus, and can learn to know him personally through his Holy Spirit. No scientific insight can take the place of this belief, simply because science has only a limited scope.

In my activities as an evolutionary biologist, I have made an effort to abide by the general principles of good scientific practice; I have considered the controversies arising from this attitude as my personal problems and not as scientific ones. Yet, objective science is a myth. Therefore, I think it is a good thing that scientists are more open about their personal beliefs. This also includes those that have turned science itself into their personal religion.

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Stellingen behorende bij het proefschrift

SEXUAL SELECTION AND SYMPATRIC SPECIATION

van Sander van Doorn

1 – De biologische soort is net zo min een fundamentele eenheid van biodiversiteit als het geslacht, de familie of de orde.

— contra, bijvoorbeeld, Harrison (1998), in Howard & Berlocher (eds.), Endless Forms.

2 – Selectieve partnerkeuze speelt een ondergeschikte rol bij sympatrische soortvorming door seksuele selectie.

— dit proefschrift, contra o.a. Higashi et al.(1999), Nature 402, p. 523-526.

3 – Op grond van wiskundige modellen kan geen van de hypothesen voor de evolutie van complexe paringsvoorkeuren gebaseerd op meerdere indicatoren van genetische kwaliteit, worden uitgesloten.

— dit proefschrift, contra o.a. Iwasa & Pomiankowski (1994), Evolution 48, p. 853-867.

4 – Groep-selectie is het belangrijkste verklarende principe bij cruciale kwalitatieve stappen[1] in de evolutie van het leven.

— [1] Maynard Smith & Szathmáry (1995), The Major Transitions in Evolution.

5 – Op basis van alleen een wiskundige theorie voor selectie, en zonder een wiskundige theorie voor constraints, komen evolutionaire redeneringen niet verder dan 'adaptive story telling'.

6 – De algemene uitkomst van DNA-microarray experimenten (nl. dat de expressie van een groot aantal genen verandert bij elke verandering in het milieu[1]) staat op gespannen voet met de verwachting dat de reductionistische methode met succes kan worden toegepast in de levenswetenschappen.

—[1] bijvoorbeeld, DeRisi et. al (1997), Science 278, p. 680-686.

7 – Theoretische modellen kunnen altijd worden genegeerd. Is het niet omdat de aannames te simpel zijn, dan is het wel omdat de analyse te ingewikkeld is.

8 – Nu het lekenpraatje is afgeschaft[1], is de promotieplechtigheid tot een onbegrijpelijk theaterstuk verworden.

—[1] UK 22

9 – Metallic lak en naveltruitjes doen vermoeden dat zowel vrouwen als mannen hun partner kiezen op basis van 'revealing handicaps'.

10 – Data maken niet gelukkig.

— contra Bio-informatici