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Evolutionary Principles for General Frequency-dependent Two-phenotype Models in Sexual Populations

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The evolutionary dynamics in general two-sex two-phenotype frequency-dependent selection models are studied with respect to underlying multi-allele one-locus genetic systems. Two classes of equilibria come into play: genotypic equilibria, with equilibrium allelic frequencies independent of the phenotype, and phenotypic equilibria, which are characterized by equal mean phenotypic fitnesses. The exact conditions for genotypic equilibria to exist and be stable and for phenotypic equilibria to exist and be evolutionarily attractive are examined. Using adequate definitions of mean fitnesses in general contexts of frequency-dependent selection in dioecious populations, we show that two phenotypes, when they can coexist in the population, tend to balance their fitnesses as far as is allowed by the genetic system as more alleles responsible for phenotype determination are introduced into the population.

An intuitive basis for frequency-dependent selection thinking can be found in Fisher's book *The Genetical Theory of Natural Selection*, first published in 1930:

> "A Batesian mimic . . . will receive less protection the more numerous it is in comparison with its model; a dimorphic Batesian mimic will therefore adjust the numbers of its two forms if these are dependent upon a single Mendelian factor, until they receive equal protection; any increase in the numbers of one form at the expense of the other would diminish the advantage of the former and increase that of the latter, thus producing a selective action tending to restore the original proportion". (p. 185, revised edition, 1958).

According to Fisher's rationale, if two or more phenotypes are segregating in a population, these phenotypes are expected to equalize their average fitnesses at equilibrium. This general equilibration principle was more recently proposed by Lloyd (1977) and Slatkin (1979). However, if we take into account underlying genetic structures, the feasibility of stable equilibria with equal phenotypic fitnesses is not guaranteed. General conditions for fitness equilibration necessarily to occur at equilibrium, with special references to heterostylous plants, were given in Heuch (1979) and generalized further in Taylor (1984). This is the case, for instance, if it is possible to order the phenotypic classes corresponding to a partition of genotypes into phenotypes and find an allele in each class that is not represented in the succeeding classes at equilibrium. Theoretically, it still remains to prove for each particular genetic system considered that an equilibrium actually exists and is stable.

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Another theoretical approach of great interest consists of looking at the evolution toward equal phenotypic fitnesses. Given a specific genetic system responsible for phenotype expression, what are the long term effects of the introduction of mutant alleles into the system? Such a problem was solved in Lessard (1984) for general frequency-dependent two-phenotype models in monoecious random mating populations. Assuming a probabilistic phenotype-determination system based on the genotype at a single locus, global convergence in the long run (via genetic mutations if necessary) to either equal phenotypic fitnesses or fixation of only one phenotype was proved. The exact conditions for equilibration to occur were also pointed out.

In the case of dioecious populations, different phenotypes may have different fitness values in males and females. In order to measure the mean phenotypic fitnesses in the whole population, we must resort to the notion of reproductive value:

> "If we consider the aggregate of an entire generation of... offspring it is clear that the total reproductive value of the males in this group is exactly equal to the total value of all the females, because each sex must supply half the ancestry of all future generations of the species." (Fisher, 1958, p. 159).

Therefore, in sexual populations, the evolution of phenotypes subject to sex-differentiated frequency-dependent selection is entangled with the evolution of the sex ratio. Although the notion of reproductive value suggests a definition of fitness in sexual populations based on the sex ratio, such a fitness measure is intrinsically frequency-dependent due to sex differences and cannot generally be treated like standard fitnesses in monoecious populations. (See Lessard (1984) for general two-sex haploid models which are shown to be formally equivalent to frequency-dependent selection models for monoecious diploid populations.) For pure sex ratio evolution models, we refer to Karlin & Lessard (1983, 1984). In these models, the phenotype coincides with sex. Optimality properties of one-to-one population sex ratios were shown to characterize a wide class of models with random mating.

The purpose of this paper is to unify two kinds of frequency-dependent selection: frequency-dependent selection due to interactions between individuals and affecting viability from conception to maturity, and frequency-dependent selection arising from sex differences and sexual reproduction. For this purpose, we examine general two-sex two-phenotype models, including a fertility model based on the phenotypes of the parents. For all models considered, we assume that the phenotype is probabilistically determined at a single autosomal locus in a dioecious diploid population and we study the evolutionary patterns from the equilibrium configurations and stability properties.

Frequency-Dependent Fertility Model Based On Two Phenotypes

Consider two phenotypic classes, C_1 and C_2, segregating in a sexually reproducing population, such that the reproductive success (i.e. fertility) of a mating pair depends upon the phenotypic composition of the pair and possibly also on the phenotypic frequencies in the whole population. Such hypotheses are particularly relevant for species in which there is parental and/or social care of offspring.

Introducing the quantities

$$p_i = \sum_{j=1}^{n} p_{ij} \quad \text{and} \quad z_i = \sum_{j=1}^{n} p_{ij} v_{ij}$$

which represent the absolute frequencies of allele A_i in all adults of a given generation and in adults that are of phenotype C_1, respectively, the recurrence system of equations (1) reduces to

$$p_i' = \frac{z_i Q(w) + p_i R(w)}{wQ(w) + R(w)}, \quad \text{for } i = 1, \ldots, n, \tag{2a}$$

$$z_i' = \frac{z_i\{(V\mathbf{z})_i(\alpha - 2\mu + \delta) + (V\mathbf{p})_i(\mu - \delta)\} + p_i\{(V\mathbf{z})_i(\mu - \delta) + (V\mathbf{p})_i\delta\}}{wQ(w) + R(w)}$$

$$\text{for } i = 1, \ldots, n, \tag{2b}$$

where

$$Q(w) = w\alpha + (1-2w)\mu - (1-w)\delta,$$

$$R(w) = w\mu + (1-w)\delta,$$

$$(V\mathbf{z})_i = \sum_{j=1}^{n} v_{ij} z_j \quad \text{and} \quad (V\mathbf{p})_i = \sum_{j=1}^{n} v_{ij} p_j,$$

with $\mathbf{p} = (p_1, \ldots, p_n)$, $\mathbf{z} = (z_1, \ldots, z_n)$ and $w = \sum_{i=1}^{n} z_i$.

The equations (2a) yield two classes of equilibria: (I) $Q(w) = 0$, and (II) $z_i = p_i w$ for $i = 1, \ldots, n$. In the latter case, the companion equations (2b) lead to the complementary condition (II') $p_i w = p_i (V\mathbf{p})_i$ for $i = 1, \ldots, n$, which characterizes the so-called *genotypic equilibria* entirely determined by the matrix V and the equilibrium allelic frequencies $p_1, \ldots, p_n$. The former class defines the *phenotypic equilibria* that depend on the phenotype frequencies but may also involve supplementary genetic constraints. The existence of phenotypic equilibria is not immediate and has not been demonstrated in general for (2a, b). (See, e.g. Karlin (1968, pp. 256–267) and Boorman & Levitt (1980, pp. 72–74) for some special cases based on two allleles with dominance of one of the alleles.) However the local stability properties of the genotypic equilibria are analytically tractable and can suggest the global dynamics.

Consider a *polymorphic genotypic equilibrium* $\mathbf{p}^* = (p_1^*, \ldots, p_n^*)$, namely

$$p_i^* = \frac{(V^{-1}\mathbf{1})_i}{\sum_{k=1}^{n} (V^{-1}\mathbf{1})_k} > 0, \quad \text{for } i = 1, \ldots, n, \tag{3}$$

where $(V^{-1}\mathbf{1})_i$ is the ith component of the inverse matrix of V times the unit vector $\mathbf{1} = (1, \ldots, 1)$. Note that if a polymorphic genotypic equilibrium $\mathbf{p}^*$ exists, then the corresponding equilibrium frequency w^* for the phenotype C_1 is such that

$$w^* = \sum_{i=1}^{n} v_{ij} p_i^*, \quad \text{for } j = 1, \ldots, n. \tag{4}$$

Therefore every gene chosen at random belongs to a C_1 individual with probability

w^* and the allelic frequencies are the same in the phenotypic classes C_1 and C_2. A local stability analysis yields the following result

> A polymorphic genotypic equilibrium $\mathbf{p}^*$ is stable if and only if the matrix $\|v_{ij}-v_{in}-v_{nj}+v_{nn}\|_{i,j=1}^{n-1}$ is negative definite in the case $Q(w^*)>0$ or positive definite in the case $Q(w^*)<0$. (See Appendix A for proofs). (5)

The condition (5) gives the *internal stability condition* of any genotypic equilibrium if we consider only the alleles that are represented at equilibrium.

Suppose that a mutant (i.e. not previously represented) allele A_{n+1} is introduced into the population. Defining the quantity

$$w_{n+1}^* = \sum_{i=1}^{n} v_{i,n+1} p_i^*, \tag{6}$$

the frequency p_{n+1} of A_{n+1} near the equilibrium state $\mathbf{p}^*$ and to a first order (linear) approximation satisfies the recurrence relation

$$p'_{n+1} \cong \left\{ \frac{w_{n+1}^* Q(w^*) + R(w^*)}{w^* Q(w^*) + R(w^*)} \right\} p_{n+1}. \tag{7}$$

Therefore the frequency of A_{n+1} will increase near $\mathbf{p}^*$ accordingly as

$$w_{n+1}^* > w^* \text{ if } Q(w^*)>0, \quad \text{or} \quad w_{n+1}^* < w^* \text{ if } Q(w^*)<0. \tag{8}$$

The condition (8) is the *external instability condition* for a genotypic equilibrium. It says that a mutant allele destabilizes a genotypic equilibrium where Q is positive if the marginal mutant frequency of C_1 exceeds the equilibrium frequency of C_1, or vice versa where Q is negative. Note that the mean fitnesses of the phenotypes C_1 and C_2 in the fertility model (2) are

$$F_1(w) = w\alpha + (1-w)\mu, \quad \text{and} \quad F_2(w) = w\mu + (1-w)\delta, \quad \text{respectively,} \tag{9}$$

so that $Q(w) = F_1(w) - F_2(w)$. Therefore, the function Q is positive or negative accordingly as the mean fitness of C_1 exceeds or not the mean fitness of C_2.

Frequency-dependent Two-phenotype Two-sex Model

Let the matrices $V = \|v_{ij}\|_{i,j=1}^{n}$ and $U - V = \|1 - v_{ij}\|_{i,j=1}^{n}$ give the probabilities that the genotypic compositions $\{A_iA_j\}_{i,j=1}^{n}$ lead to the expression of the two possible phenotypes C_1 and C_2, respectively, in an infinite population. The phenotype-determination matrices V and $U-V$ are assumed to be independent of sex. However, the phenotypes themselves are subject to sex-differentiated frequency-dependent selection.

Let $F_i = F_i(w)$ be the expectation for a female of phenotype C_i of participating in reproduction ($i=1,2$) if w is the frequency of C_1 in the population and therefore $1-w$ is the frequency of C_2. The corresponding selective functions for a male are given by $M_i = M_i(w)$ for $i=1,2$. Assume random mating and a 1:1 progeny sex ratio at conception. If the frequencies of the ordered genotypes $\{A_iA_j\}_{i,j=1}^{n}$ are

denoted by $\{p_{ij}\}_{i,j=1}^n$ in the female parental population and $\{q_{ij}\}_{i,j=1}^n$ in the male parental population, with corresponding allelic frequencies $\{p_i\}_{i=1}^n$ and $\{q_i\}_{i=1}^n$, respectively, the recurrence equations over two successive non-overlapping generations take the form

$$\begin{aligned} 2Fp'_{ij} &= (p_iq_j + p_jq_i)[v_{ij}F_1 + (1-v_{ij})F_2], \\ 2Mq'_{ij} &= (p_iq_j + p_jq_i)[v_{ij}M_1 + (1-v_{ij})M_2], \end{aligned} \tag{10}$$

where

$$\begin{aligned} F = F(w) &= wF_1 + (1-w)F_2, \\ M = M(w) &= wM_1 + (1-w)M_2, \end{aligned}$$

with $w = w(\mathbf{p}, \mathbf{q}) = \sum_{i,j=1}^n v_{ij}p_iq_j$. In terms of the allelic frequencies $p_i = \sum_{j=1}^n p_{ij}$ and $q_i = \sum_{j=1}^n q_{ij}$ for $i = 1, \ldots, n$, we have

$$\begin{aligned} 2Fp'_i &= p_i[V\mathbf{q})_i(F_1 - F_2) + F_2] + q_i[(V\mathbf{p})_i(F_1 - F_2) + F_2], \\ 2Mq'_i &= p_i[(V\mathbf{q})_i(M_1 - M_2) + M_2] + q_i[(V\mathbf{p})_i(M_1 - M_2) + M_2], \end{aligned} \tag{11}$$

where $\mathbf{p} = (p_1, \ldots, p_n)$, $\mathbf{q} = (q_1, \ldots, q_n)$, $(V\mathbf{p})_i = \sum_{j=1}^n v_{ij}p_j$ and $(V\mathbf{q})_i = \sum_{j=1}^n v_{ij}q_j$. In particular, these equations entail the relation

$$\frac{2Fp'_i - (p_i + q_i)F_2}{F_1 - F_2} = \frac{2Mq'_i - (p_i + q_i)M_2}{M_1 - M_2}. \tag{12}$$

Hence at equilibrium, we must have for every $i = 1, \ldots, n$

$$p_i[(2F - F_2)(M_1 - M_2) + M_2(F_1 - F_2)] = q_i[(2M - M_2)(F_1 - F_2) + F_2(M_1 - M_2)],$$

or equivalently, after easy algebraic manipulations,

$$p_iQ(w) = q_iQ(w) \tag{13}$$

where

$$Q(w) = \frac{F_1(w) - F_2(w)}{2F(w)} + \frac{M_1(w) - M_2(w)}{2M(w)}.$$

Therefore an equilibrium $\{\mathbf{p}, \mathbf{q}\}$ for (11) must satisfy either (I) $Q(w) = 0$ where $w = w(\mathbf{p}, \mathbf{q}) = \sum_{i,j=1}^n v_{ij}p_iq_j$, or (II) $p_i = q_i$ for all i, and then $p_iw = p_i(V\mathbf{p})_i$ for $i = 1, \ldots, n$. In our terminology, the equilibrium is either phenotypic or genotypic.

Note that the condition $Q(w) = 0$ is not sufficient for equilibrium in (11) and a deeper analysis is required. The occurrence of phenotypic equilibria can be checked directly for some two-allele cases (see, e.g. O'Donald (1980) for a special case with dominance and St Lawrence & O'Donald (1981) for a homozygote-heterozygote scheme). For the multiallele case, we rely on the following general characterization.

> If $Q(\tilde{w}) = 0$, then there exists a non-genotypic equilibrium $\{\tilde{\mathbf{p}}, \tilde{\mathbf{q}}\}$ for (11) with $w(\tilde{\mathbf{p}}, \tilde{\mathbf{q}}) = \tilde{w}$ if and only if there exist two genotypic equilibria $\{\mathbf{p}^*, \mathbf{p}^*\}$ and $\{\mathbf{p}^{**}, \mathbf{p}^{**}\}$ such that $w(\mathbf{p}^*, \mathbf{p}^*) < \tilde{w} < w(\mathbf{p}^{**}, \mathbf{p}^{**})$. Actually there exist a whole surface of phenotypic equilibria corresponding to $\tilde{w}$ and separating $\mathbf{p}^*$ from $\mathbf{p}^{**}$. (See Appendix B for more details).

On the other hand, the exact conditions for the existence and stability of genotypic equilibria can be readily obtained. These conditions follow the scheme of the previous model. Namely, a polymorphic genotypic equilibrium $\{\mathbf{p}^*, \mathbf{p}^*\}$ for (11) exists if and only if $\mathbf{p}^*$ satisfies (3); the equilibrium is stable if the internal stability condition (5) with the function Q of (13) and $w^* = w(\mathbf{p}^*, \mathbf{p}^*)$ holds; it is destabilized by a mutant allele according to the external instability condition (8) (see Appendix C).

For the two-sex two-phenotype model (11), a measure of the mean fitness of C_i is

$$\bar{F}_i(w) = \frac{F_i(w)}{2F(w)} + \frac{M_i(w)}{2M(w)}, \quad \text{for } i = 1, 2, \tag{15}$$

so that $Q(w) = \bar{F}_1(w) - \bar{F}_2(w)$ analogously to the previous model.

Maternal Inheritance Version of the General Two-sex Two-phenotype Model

Suppose that an A_iA_j mother is of phenotype C_1 with probability v_{ij} and C_2 with probability $1 - v_{ij}$ $(i, j = 1, \ldots, n)$. Let w be the frequency of C_1 among all mothers of a given generation. It is assumed that a female offspring from a pure C_i-mother has a fitness $F_i(w)$ while the corresponding fitness function for a male offspring is $M_i(w)$ $(i = 1, 2)$. The frequencies p_{ij} and q_{ij} of the ordered genotype A_iA_j $(i, j = 1, \ldots, n)$ in the female and male populations, respectively, are related over two successive generations with random mating by the recurrence equations

$$\begin{aligned} 2Fp'_{ij} &= \sum_{k,l=1}^{n} p_{ik}q_{jl}[v_{ik}F_1 + (1 - v_{ik})F_2] + \sum_{k,l=1}^{n} p_{jl}q_{ik}[v_{jl}F_1 + (1 - v_{jl})F_2] \\ 2Mq'_{ij} &= \sum_{k,l=1}^{n} p_{ik}q_{jl}[v_{ik}M_1 + (1 - v_{ik})M_2] + \sum_{k,l=1}^{n} p_{jl}q_{ik}[v_{jl}M_1 + (1 - v_{jl})M_2] \end{aligned} \tag{16}$$

where $F = wF_1 + (1 - w)F_2$ and $M = wM_1 + (1 - w)M_2$ with $w = \sum_{i,k=1}^{n} v_{ik}p_{ik}$. If we denote by p_i and q_i the frequencies of allele A_i $(i = 1, \ldots, n)$ in females and males, respectively, and define $z_i = \sum_{k=1}^{n} v_{ik}p_{ik}$ for $i = 1, \ldots, n$, then the recurrence system can be written into the form

$$\begin{aligned} 2Fp'_i &= z_i(F_1 - F_2) + p_iF_2 + q_iF, \\ 2Mq'_i &= z_i(M_1 - M_2) + p_iM_2 + q_iM, \\ 2Fz'_i &= [z_i(F_1 - F_2) + p_iF_2](V\mathbf{q})_i + q_i[(V\mathbf{z})_i(F_1 - F_2)] + (V\mathbf{p})_iF_2. \end{aligned} \tag{19}$$

In particular, for every $i = 1, \ldots, n$

$$p'_i + q'_i = z_i\left\{\frac{F_1 - F_2}{2F} + \frac{M_1 - M_2}{2M}\right\} + p_i\left\{\frac{F_2}{2F} + \frac{M_2}{2M}\right\} + q_i,$$

so that, at equilibrium, we must have

$$\begin{aligned} z_i\left\{\frac{F_1 - F_2}{2F} + \frac{M_1 - M_2}{2M}\right\} &= p_i\left\{1 - \frac{F_2}{2F} - \frac{M_2}{2M}\right\} \\ &= p_iw\left\{\frac{F_1 - F_2}{2F} + \frac{M_1 - M_2}{2M}\right\}. \end{aligned} \tag{20}$$

The equilibria of (19) satisfying $z_i^* = p_i^* w^*$ for $i = 1, \ldots, n$ have $q_i^* = p_i^* = p_i^*(V\mathbf{p}^*)_i / w^*$ for $i = 1, \ldots, n$. These equilibria are genotypic in our terminology, with the same allelic frequencies in males and females. It can be checked that a polymorphic genotypic equilibrium with $Q(w^*) > 0$ where $Q = (F_1 - F_2)/2F + (M_1 - M_2)/2M$ is internally stable if the matrix $\|v_{ij} - v_{in} - v_{nj} + v_{nn}\|_{i,j=1}^{n-1}$ is negative definite and externally unstable following the introduction of a mutant allele A_{n+1} if $\sum_{i=1}^{n} v_{i,n+1} p_i^* > w^*$. For the case $Q(w^*) < 0$, it suffices to replace the quantities v_{ij} by $\|1 - v_{ij}\|$ in the above conditions (see Appendix D for technical details).

On the other hand, the non-genotypic equilibria $\{\tilde{\mathbf{p}}, \tilde{\mathbf{q}}, \tilde{\mathbf{z}}\}$ for (19) must satisfy $Q(\tilde{w}) = 0$ where $\tilde{w} = \sum_{i=1}^{n} \tilde{z}_i$. It can be argued as in Appendix B that such phenotypic equilibria exist if and only if there exist two genotypic equilibria with associated w^* and w^{**}, respectively, such that $w^* < \tilde{w} < w^{**}$. Therefore the equilibrium configurations are similar to the configurations of the previous two-sex two-phenotype model (11).

Discussion

In a previous paper (Lessard, 1984), we considered frequency-dependent two-phenotype selection models in monoecious random mating populations: two phenotypes C_1 and C_2 whose frequencies in the population are w and $1 - w$, respectively, have fitnesses assumed to be given by general functions $F_1(w)$ and $F_2(w)$, respectively, that can take into account interactions between individuals from conception to maturity and any other form of frequency-dependent selection based on phenotype of offspring but independent of mating. In this case, if the phenotype is probabilistically determined at a single multiallelic locus, the phenotypic equilibria correspondong to the zeros of $Q(w) = F_1(w) - F_2(w)$ with negative slope and the phenotypic fixation states $w = 0$ and $w = 1$ such that $Q(0) < 0$ and $Q(1) > 0$, respectively, are evolutionarily attractive. An *evolutionarily attractive state* w^* with domain of attraction D_{w^*} is defined as a phenotypic equilibrium such that a population in a phenotypic state w in D_{w^*} will evolve to w^*, with the introduction of mutant alleles if necessary. Once reached, an evolutionarily attractive state is stable as a population phenotypic equilibrium against any mutation within the genetic system under consideration.

In this paper, we have studied three different models involving two phenotypes in dioecious populations in order to test the robustness of our previous results on frequency-dependent selection and suggest some general principles on fitness calculation and evolutionary dynamics in sexual populations. In dealing with phenotypic selection models with underlying genetic systems, there are three problems of particular interest: (1) Existence and characterization of phenotypic equilibria associated with equal mean phenotypic fitnesses; (2) Local stability conditions of genotypic (non-phenotypic) equilibria arising from the underlying genetic structure and conditions for the initial increase in frequency of mutant alleles near such equilibria; (3) Global convergence to evolutionarily attractive states via allelic mutation within the underlying genetic system. While global dynamical properties

seem difficult to prove for models with general fertility schemes and/or sex-differentiated frequency-dependent selection, the local stability conditions at the non-phenotypic equilibria can suggest the evolutionary tendencies.

For the fertility model (2) in which the fitness of an offspring depends on the phenotypes of its parents and possibly also the frequencies of the phenotypes in the parental generation in the large, the existence of phenotypic equilibria in the general case is still an open problem. Nevertheless the condition (5) for internal stability with the condition (8) for external instability of a genotypic equilibrium implies (see, e.g. Kingman, 1961) that a non-phenotypic equilibrium where Q is positive (i.e. where the mean fitness of phenotype C_1 exceeds the mean fitness of phenotype C_2 with the formulas given in (9)) is stable if and only if the frequency of C_1 is locally maximized at equilibrium. Symmetrically, in the case where Q is negative, a non-phenotypic equilibrium is stable if and only if it corresponds to a local maximum for the frequency of C_2 with respect to the underlying allelic frequencies. Therefore, near a non-phenotypic equilibrium, the dynamical genetic system locally favors an increase in the frequency of C_1 as long as C_1 has a larger fitness and similarly for C_2. This property suggests that the zeros from plus to minus of $Q(w) = F_1(w) - F_2(w)$, where w is the frequency of C_1 and $1-w$ the frequency of C_2 are evolutionary attractive at least in a weak sense. The same is true for $w=0$ if $Q(0)<0$ and $w=1$ if $Q(1)>0$.

Note that the fertility model (2) is general enough to include cases of kin selection, specifically those with parent-to-offspring fitness transfers as considered in Cavalli-Sforza & Feldman (1978). Other cases with sib-to-sib interactions as studied in Uyenoyama *et al.* (1980) are beyond the scope of application of our present analysis.

For the two-sex two-phenotype model (11) and the corresponding maternal inheritance version (19), the function Q takes the form

$$Q = \frac{F_1 - F_2}{2F} + \frac{M_1 - M_2}{2M} \tag{21}$$

where F_i and M_i represent the mean fitnesses of C_i ($i=1,2$) in the female and male populations, respectively, whose mean fitnesses are F and M, respectively. ($M:F$ is the sex ratio at maturity if we allume a 1 : 1 sex ratio at conception.) This expression for Q has an intuitive interpretation. In the model (11), if w_i is the frequency of C_i offspring ($i=1,2$), then the proportion of C_i adult females will be w_iF_i/F and the proportion of C_i adult males w_iM_i/M. Under random mating, the frequency of C_i parents for the next generation will be

$$w_i\left(\frac{F_i}{2F} + \frac{M_i}{2M}\right), \qquad i=1,2. \tag{22}$$

Since w_i is the probability that an offspring chosen at random is of phenotype C_i, the quantity in brackets in (22) is proportional to the probability of a C_i offspring of participation in reproduction ($i=1,2$). This quantity corresponds to the well-known Shaw–Mohler formula (Shaw & Mohler, 1953). Observe that Q in (21) is simply the difference between these quantities for C_1 and C_2.

In the case $F_1 = 1$, $F_2 = 0$, $M_1 = 0$, $M_2 = 1$, the phenotype coincides with sex (all females being of phenotype C_1 and all males of phenotype C_2) and

$$Q = \frac{1}{w} - \frac{1}{1-w} = \frac{1-2w}{w(1-w)} \tag{23}$$

where w is the proportion of females in the population. Such models in a multi-allele context were introduced in Eshel & Feldman (1982) in order to study the evolution of the sex ratio under genetic mutation. The existence conditions and the evolutionary properties of one-to-one population sex ratio equilibria corresponding to $w = 1/2$ were given in Karlin & Lessard (1983, 1984). Maternal inheritance versions were also considered in this case and led to similar conclusions.

Our results in the case of general fitness functions extend our previous studies on sex ratio evolution. Particularly interesting is the necessary and sufficient condition (14) for the existence of phenotypic equilibria: a phenotypic equilibrium corresponding to a zero w^* of Q exists if and only if there exist genotypic equilibria on both sides of w^*. Moreover a phenotypic equilibrium generally corresponds to a surface of equilibrium allelic frequencies and those that are potentially evolutionary attractive, apart from the fixation states, corresponds to the zeros from plus to minus of Q. The analysis for the model (19) with maternal inheritance is more recondite but leads to the same conclusions.

Models of sexual selection are subsumed in our general formulation (11). Pure sexual selection arising from female preferences between two types of males in competition as recently studied in Raper (1982) in a general multi-allele framework corresponds to the case $F_1 = F_2$ with no selective differentials in females. See, e.g. Karlin & Raper (1979), Eshel (1979), Charlesworth & Charlesworth (1981) for examples of frequency-dependent fitnesses in males as consequences of sexual selection. Some models taking also into account fitness differences in females were considered in the framework of two allele one-locus genetic systems in O'Donald (1980) and St Lawrence & O'Donald (1981). Our general conclusions apply to all these models.

Models for the evolution of altruistic traits in populations subdivided into small groups have also led to general fitness functions (see, e.g. Matessi & Jayakar, 1976; Cohen & Eshel, 1976; Karlin & Matessi, 1983). Our frequency-dependent selection model (11) is relevant to such cases even with sex-differentiated fitnesses.

Our definition of an evolutionary attractive equilibrium in the context of frequency-dependent selection is linked to the notion of ESS. In Lessard (1984), we showed that the evolutionary attractive states in monoecious random mating populations correspond to ESS's as originally defined in Maynard Smith & Price (1973) by comparing payoff functions. The exact evolutionary properties of phenotypic equilibria corresponding or not to ESS's are generally more difficult to establish for dioecious populations with underlying genetic systems. This paper is an attempt to understand the evolutionary dynamics in such populations in a general context of sex-differentiated frequency-dependent selection.

In Maynard Smith (1982) an ESS is defined at the phenotypic level as a strategy that is better than any other once adopted by all members of a population. In a

APPENDIX A

Let $\mathbf{p}^* = (p_1^*, \ldots, p_n^*)$ be a polymorphic genotypic equilibrium of (2) with $w^* = (V\mathbf{p}^*)_i$ for $i = 1, \ldots, n$. Writing $\mathbf{p} = \mathbf{p}^* + \boldsymbol{\xi}$, $\mathbf{y} = \mathbf{z}/w = \mathbf{p}^* + \boldsymbol{\eta}$, and $w = w^* + \gamma$ where $\boldsymbol{\xi}$, $\boldsymbol{\eta}$, and γ are small perturbations, we have over two successive generations

$$\begin{bmatrix} \boldsymbol{\xi}' \\ \boldsymbol{\eta}' \\ \gamma' \end{bmatrix} = \frac{1}{[w^*Q(w^*) + R(w^*)]} \begin{bmatrix} R(w^*)I & w^*Q(w^*)I & 0 \\ R(w^*)[I + V(\mathbf{p}^*)] & w^*Q(w^*)[I + V(\mathbf{p}^*)] & 0 \\ 0 & 0 & 0 \end{bmatrix} \begin{bmatrix} \boldsymbol{\xi} \\ \boldsymbol{\eta} \\ \gamma \end{bmatrix}$$

$$+ \text{higher order terms} \tag{A1}$$

where $V(\mathbf{p}^*) = D_{\mathbf{p}^*}V/w^*$. ($D_{\mathbf{p}^*}$ denotes the diagonal matrix with the components of $\mathbf{p}^*$ on the main diagonal.) Apart from 0, the eigenvalues for the linear terms in (A1) are in the form

$$1 + \left\{ \frac{w^*Q(w^*)}{w^*Q(w^*) + R(w^*)} \right\} \lambda \tag{A2}$$

where λ is an eigenvalue for $V(\mathbf{p}^*)$. The corresponding right eigenvectors are in the form $(a\boldsymbol{\xi}; b\boldsymbol{\xi}; 0)$ where $V(\mathbf{p}^*)\boldsymbol{\xi} = \lambda\boldsymbol{\xi}$. Only the cases $\boldsymbol{\xi} = (\xi_1, \ldots, \xi_n)$ with $\sum_{i=1}^n \xi_i = 0$ are relevant in (A1) since the components of $\mathbf{p}^* + \boldsymbol{\xi}$ always sum up to 1. This eliminates the eigenvalue $\lambda = 1$ with associated right eigenvector $\mathbf{p}^*$. The other eigenvalues $\lambda_1, \ldots, \lambda_{n-1}$ of $V(\mathbf{p}^*)$ are real (since $V(\mathbf{p}^*)$ is the product of a symmetric matrix with a diagonal matrix) and strictly less than 1 in absolute value (owing to the Perron-Frobenius theory for positive matrices). Moreover these eigenvalues have corresponding right eigenvectors $\boldsymbol{\xi}^{(1)}, \ldots, \boldsymbol{\xi}^{(n-1)}$ whose components sum up to 0 (because they must be perpendicular to the left eigenvector associated with $\lambda = 1$ which is $\mathbf{1} = (1, \ldots, 1)$). We refer to Gantmacher (1959) for a review of matrix theory.

Therefore in the case $Q(w^*) > 0$, the relevant eigenvalues in (A1) are all less than 1 in absolute value if and only if the eigenvalues $\lambda_1, \ldots, \lambda_{n-1}$ of $V(\mathbf{p}^*)$ are all negative, or equivalently,

$$\begin{aligned} \sum_{i,j=1}^{n-1} (v_{ij} - v_{in} - v_{nj} + v_{nn})\xi_i^{(k)}\xi_j^{(k)} &= \sum_{i,j=1}^{n} v_{ij}\xi_i^{(k)}\xi_j^{(k)} \\ &= w^* \sum_{i=1}^{n} \frac{\xi_i^{(k)}}{p_i^*} (V(\mathbf{p}^*)\boldsymbol{\xi}^{(k)})_i \\ &= w^* \lambda_k \sum_{i=1}^{n} \frac{(\xi_i^{(k)})^2}{p_i^*} \\ &< 0 \end{aligned} \tag{A3}$$

for the corresponding right eigenvectors $\boldsymbol{\xi}^{(k)} = (\xi_1^{(k)}, \ldots, \xi_n^{(k)})$ with $\xi_n^{(k)} = -\xi_{n-1}^{(k)} - \ldots - \xi_1^{(k)}$ for $k = 1, \ldots, n-1$. These $(n-1)$ vectors being independent, the matrix $\| v_{ij} - v_{in} - v_{nj} + v_{nn} \|_{i,j=1}^{n-1}$ is actually negative definite everywhere if (A3) holds.

In the case $Q(w^*) < 0$, we get by symmetry the stability condition $\|(1 - v_{ij}) - (1 - v_{in}) - (1 - v_{nj}) + (1 - v_{nn})\|_{i,j=1}^{n-1}$ negative definite, i.e. $\| v_{ij} - v_{in} - v_{nj} + v_{nn} \|_{i,j+1}^{n-1}$ positive definite.

APPENDIX B

We first observe that

$$Q+\frac{F_1-F_2}{2F}+\frac{M_1-M_2}{2M}=0 \tag{B1}$$

if and only if

$$\frac{F_1}{2F}+\frac{M_1}{2M}=\frac{F_2}{2F}+\frac{M_2}{2M}=1 \tag{B2}$$

where $F=wF_1+(1-w)F_2$ and $M=wM_1+(1-w)M_2$ such that in all circumstances

$$w\left(\frac{F_1}{2F}+\frac{M_1}{2M}\right)+(1-w)\left(\frac{F_2}{2F}+\frac{M_2}{2M}\right)=1.$$

Under the condition $Q=0$, the recurrence equations (11) yield

$$p_i'+q_i'=p_i+q_i \quad \text{for } i=1,\ldots,n \tag{B3}$$

and then there is equilibrium if and only if

$$p_i=p_i(H\mathbf{q})_i+q_i(H\mathbf{p})_i \quad \text{for } i=1,\ldots,n \tag{B4}$$

where

$$H=\frac{(F_1-F_2)V+F_2U}{2F}$$

with the notation U for the matrix that has all unit entries. Note that the entries of H are all between 0 and 1 owing to (B2). Henceforth the functions F_1, F_2, and F are evaluated at a zero $\tilde{w}$ of Q and the equilibrium condition (B4) is associated with the condition $w(\mathbf{p},\mathbf{q})=\tilde{w}$. It is also assumed that $F_1<F_2$ at $\tilde{w}$ (relabel the phenotypes if necessary).

The system of equations (B4) is in a form that was studied in Karlin and Lessard (1984). In matrix notation, we have

$$\mathbf{p}=B_H(\mathbf{q})\mathbf{p} \quad \text{with } B_H(\mathbf{q})=D_{H\mathbf{q}}+D_{\mathbf{q}}H \tag{B5}$$

($D_{\mathbf{q}}$ and $D_{H\mathbf{q}}$ are the diagonal matrices with $\mathbf{q}$ and $H\mathbf{q}$, respectively, on the main diagonal.) In such a case, the Perron-Frohenius theory for non-negative matrices informs us that $B_H(\mathbf{q})$ has 1 as its principal eigenvalue (the largest eigenvalue in magnitude) with $\mathbf{p}$ as a principal right eigenvector. Writing $\rho_H(\mathbf{q})$ for the principal eigenvalue of $B_H(\mathbf{q})$ for every frequency vector $\mathbf{q}$ (i.e. a vector whose components are all non-negative and sum up to 1), the equilibrium relation (B4) is characterized by the equation

$$\rho_H(\mathbf{q})=1. \tag{B6}$$

On the other hand, it can be shown (see Karlin & Lessard, 1984) that for every

genotypic equilibrium $\mathbf{p}^* = (p_1^*, \ldots, p_n^*)$ for H (or equivalently for V) we have

$$\rho_H(\mathbf{p}^*) = \max\left\{2\sum_{i=1}^{n} p_i^*(H\mathbf{p}^*)_i \text{ and } (H\mathbf{p}^*)_k \text{ for all } k \text{ such that } p_k^* = 0\right\},$$

$$= 2\sum_{i=1}^{n} p_i^*(H\mathbf{p}^*)_i \quad \text{if } \rho_H(\mathbf{p}^*) > 1, \tag{B7}$$

$$= \frac{(F_1 - F_2)w(\mathbf{p}^*, \mathbf{p}^*) + F_2}{(F_1 - F_2)\tilde{w} + F_2} \quad \text{if } w(\mathbf{p}^*, \mathbf{p}^*) < \tilde{w}.$$

(Note that $(H\mathbf{p}^*)_k < 1$ for all k since the entries of H are between 0 and 1 while $F_1 < F_2$ by assumption.) Moreover we have also the property that every local maximum (and in particular the global maximum) of $\rho_H(\mathbf{q})$ with respect to frequency vectors corresponds to a genotypic equilibrium.

Similarly, (B5) and (B6) are equivalent to

$$\mathbf{q} = B_{U-H}(\mathbf{p})\mathbf{q} \quad \text{and} \quad \rho_{U-H}(\mathbf{p}) = 1 \tag{B8}$$

respectively, and every genotypic equilibrium $\mathbf{p}^{**}$ (in particular the equilibrium corresponding to the global maximum of $\rho_{U-H}(\mathbf{p})$) with $\rho_{U-H}(\mathbf{p}^{**}) > 1$ is such that $w(\mathbf{p}^{**}, \mathbf{p}^{**}) > \tilde{w}$, and vice versa.

We are now ready to conclude. If a non-genotypic equilibrium associated with w exists, then the function $\rho_H(\mathbf{q})$ and $\rho_{U-H}(\mathbf{p})$ take the value 1 at some frequency vectors $\mathbf{p}$ and $\mathbf{q}$, and their global maxima over all frequency vectors exceed 1. The genotypic equilibria corresponding to these maxima, $\mathbf{p}^*$ and $\mathbf{p}^{**}$, respectively, satisfy

$$w(\mathbf{p}^*, \mathbf{p}^*) < \tilde{w} < w(\mathbf{p}^{**}, \mathbf{p}^{**}). \tag{B9}$$

Conversely, if two genotypic equilibria $\mathbf{p}^*$ and $\mathbf{p}^{**}$ satisfy (B9), then (B7) yields

$$\rho_H(\mathbf{p}^*) > 1 > \rho_H(\mathbf{p}^{**})$$

and the continuous function $\rho_H(\mathbf{q})$ takes the value 1 corresponding to $\tilde{w}$ somewhere on every continuous path of frequency vectors joining $\mathbf{p}^*$ and $\mathbf{p}^{**}$.

APPENDIX C

Writing $\mathbf{p} = \mathbf{p}^* + \boldsymbol{\xi}$ and $\mathbf{q} = \mathbf{p}^* + \boldsymbol{\eta}$ in (11) where $\mathbf{p}^*$ is a polymorphic genotypic equilibrium, we have over two successive generations

$$\begin{bmatrix} \boldsymbol{\xi}' \\ \boldsymbol{\eta}' \end{bmatrix} = \begin{bmatrix} B_{H^*}(\mathbf{p}^*) & B_{H^*}(\mathbf{p}^*) \\ B_{G^*}(\mathbf{p}^*) & B_{G^*}(\mathbf{p}^*) \end{bmatrix} \begin{bmatrix} \boldsymbol{\xi} \\ \boldsymbol{\eta} \end{bmatrix} + \text{higher order terms}, \tag{C1}$$

where

$$B_{H^*}(\mathbf{p}^*) = I/2 + D_{\mathbf{p}^*}H^*$$

$$B_{G^*}(\mathbf{p}^*) = I/2 + D_{\mathbf{p}^*}G^*$$

$$H^* = \frac{(F_1^* - F_2^*)V + F_2^*U}{2F^*}$$

$$G^* = \frac{(M_1^* - M_2^*)V + M_2^*U}{2M^*}$$

with "*" meaning an evaluation at $\mathbf{p}^*$ or $w^* = w(\mathbf{p}^*, \mathbf{p}^*)$.

The relevant eigenvalues for internal stability of $\mathbf{p}^*$ are those of $B_{H^*}(\mathbf{p}^*) + B_{G^*}(\mathbf{p}^*) = B_{H^*+G^*}(\mathbf{p}^*)$ with right eigenvectors whose components sum up to 0. These eigenvalues are in the form

$$1 + w^*Q^*\lambda$$

where

$$Q^* = \frac{F_1^* - F_2^*}{2F^*} + \frac{M_1^* - M_2^*}{2M^*}$$

and λ is an eigenvalue for $V(\mathbf{p}^*) = D_{\mathbf{p}^*}V/w^*$. The rest of the analysis for internal stability follows from Appendix A.

For external instability, it suffices to notice that the frequencies p_{n+1} and q_{n+1} of a mutant allele A_{n+1} near $\mathbf{p}^*$ satisfy

$$p'_{n+1} + q'_{n+1} = (p_{n+1} + q_{n+1})[1 + (w^*_{n+1} - w^*)Q^*] + \text{higher order terms},$$

where w^*_{n+1} is defined in (6).

APPENDIX D

Defining the variables $\boldsymbol{\xi} = \mathbf{p} - \mathbf{p}^*$, $\boldsymbol{\zeta} = \mathbf{q} - \mathbf{p}^*$, $\boldsymbol{\eta} = \mathbf{z}/w - \mathbf{p}^*$ and $\gamma = w - w^*$ where $\mathbf{p}^*$ with associated w^* is a polymorphic genotypic equilibrium of (19), the linear terms for the transformation of these variables over two successive generations form the matrix

$$\begin{bmatrix} \frac{F_2^*}{2F^*}I & \frac{1}{2}I & \frac{w^*(F_1^* - F_2^*)}{2F^*}I & 0 \\ \frac{M_2^*}{2M^*}I & \frac{1}{2}I & \frac{w^*(M_1^* - M_2^*)}{2M^*}I & 0 \\ \frac{F_2^*}{2F^*}\{I + V(\mathbf{p}^*)\} & \frac{1}{2}\{I + V(\mathbf{p}^*)\} & \frac{w^*(F_1^* - F_2^*)}{2F^*}\{I + V(\mathbf{p}^*)\} & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \tag{D1}$$

where "*" indicates an evaluation at the genotypic equilibrium $\mathbf{p}^*$ and $V(\mathbf{p}^*) = D_{\mathbf{p}^*}V/w^*$. Trying eigenvectors in the form $(a\boldsymbol{\xi}; b\boldsymbol{\xi}; c\boldsymbol{\xi}; 0)$ where $\boldsymbol{\xi}$ is an eigenvector

for $V(\mathbf{p}^*)$, the relevant eigenvalues correspond to the eigenvalues of

$$\begin{bmatrix} \frac{F_2^*}{2F^*} & \frac{1}{2} & \frac{w^*(F_1^*-F_2^*)}{2F^*} \\ \frac{M_2^*}{2M^*} & \frac{1}{2} & \frac{w^*(M_1^*-M_2^*)}{2M^*} \\ \frac{F_2^*}{2F^*}(1+\lambda) & \frac{1}{2}(1+\lambda) & \frac{w^*(F_1^*-F_2^*)}{2F^*}(1+\lambda) \end{bmatrix} \tag{D2}$$

where λ is an eigenvalue of $V(\mathbf{p}^*)$ different from 1. Apart from 0, the relevant eigenvalues are in the form

$$\frac{1}{2}\left\{1+\frac{\lambda w^*(F_1^*-F_2^*)}{2F^*} \pm \sqrt{\left[1-\frac{\lambda w^*(F_1^*-F_2^*)}{2F^*}\right]^2+2\lambda Q^*}\right\} \tag{D3}$$

These quantities are less than 1 in magnitude if and only if $\lambda Q^* < 0$ for every possible λ.

For a mutant allele A_{n+1}, the relevant eigenvalues are those of (D2) with λ replaced by $(w_{n+1}^*/w^* - 1)$ where w_{n+1}^* is the quantity defined in (6). Therefore the condition for external instability is $(w_{n+1}^* - w^*)Q^* > 0$.