A General Approach to Genetic Equilibria with an Uneven Sex Ratio

IGAL MILCHTAICH
School of Mathematics, Raymond and Beverly Sackler
Faculty of Exact Sciences, Tel Aviv University,
Ramat Aviv, 69978 Tel Aviv, Israel

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Abstract

It is shown that in a diploid population at a genetic equilibrium with an uneven sex ratio the distribution of all (inertial) autosomal genes is symmetric between the two sexes. If several allelic genes occur in the population then the sex ratios of the various genotypes uniquely determine the uneven population sex ratio.

The equilibrium is unstable to invasion by new genes which are relatively more frequent among the less numerous sex. A new proof is given to the fact that if genotype sex ratios do not change then, following an invasion by a new allele, a new stable polymorphic equilibrium can only correspond to a sex ratio which is more even than the old one. Application of these general results to a model of offspring sex determination by parents' autosomal genes shows the equilibrium to be unstable to invasion by new genes which tend to increase the production of offspring of the minority sex, as proposed by Fisher. The relevance of the Shaw–Mohler formula in this context is shown to derive from its role in describing the dynamics of autosomal genes in general.

In connection with a model for offspring sex determination by one parent, a new proof is given to the fact that, following an invasion by a new allele, a new stable polymorphic equilibrium can only correspond to a sex ratio which is more even than the old one.

1 Introduction

Fisher (1930) proposed that in populations where the sex ratio is uneven natural selection would favour those parents capable of producing more offspring of the less numerous sex, without affecting their total number of offspring as a result [this is a special case of what is known as the Fisher's principle; see Maynard Smith (1978)].

Suppose, for example, that the proportion M of males is greater than the proportion $F = 1 - M$ of females. The mating prospects for a female are then better than for a male and the number of offspring of a typical male, which is proportional to $1/M$, is therefore less than that of a typical female, which is proportional to $1/F$. The "reproductive

value" of every son produced is therefore smaller than that of every daughter produced. Parents that produce more daughters thus tend to have a greater number of *grand-offspring*.

Analytically, while a typical parent, producing sons and daughters in a ratio of $M : F$, can expect to have a number of grand-offspring proportional to $M \cdot 1/M + F \cdot 1/F = 2$, a parent that produces average number of offspring but with a different ratio $m : f$ of sons to daughters can expect to have a number of grand-offspring proportional to $m \cdot 1/M + f \cdot 1/F$, that is

$$\frac{1}{2}\left[\frac{m}{M} + \frac{f}{F}\right] \tag{1}$$

times the typical number of grand-offspring. Expression (1), which except for the numerical factor is known as the Shaw–Mohler formula [Shaw & Mohler (1953)], is easily seen to be greater than unity when $M > F$ and $f > F$—in agreement with the conclusion of the previous paragraph—and, symmetrically, when $M < F$ and $m > M$.

Fisher concluded that the tendency toward producing more offspring of the less numerous sex would spread in the population. It was, however, pointed out by Hamilton (1967) that Fisher's argument does not apply in situations where, for example, males are the heterogametic sex and the gene for producing more daughters is Y-linked; grand-offspring through daughters are then irrelevant to the transmission of the gene. Indeed, in the situation described the frequency of the gene would decrease in every generation by the factor $m/M < 1$, as the gene is transmitted only from fathers to sons.

In the following such difficulties are avoided by restricting the discussion to autosomal genes. It is further assumed that the frequency by which parents of each sex transmit the gene to their offspring is equal to the relative frequency of the gene in that sex. Genes for which these conditions hold may be named *inertial* genes.

The action of the gene need not be limited to parents only. The concept of offspring sex ratio should thus be replaced by a more general one which serves to describe the correlation existing between the occurrence of the gene and sex in the general context. The *marginal* sex ratio of the gene is such a concept: it is the sex ratio (proportion of males) of the individuals which develop from gene-carrying gametes. It is also the weighted average sex ratio of gene-carrying individuals, double weight is ascribed to homozygotes. The occurrence of the gene is *correlated* with sex if the marginal sex ratio is different from the *general* one, the sex ratio of the whole population.

Without assuming any particular *causal* relation between the occurrence of the gene and sex to exist, it is shown in section 2 that the frequency of the gene increases if its occurrence is correlated with sex in such a way that it is relatively more frequent among the less numerous sex and decreases if it is less frequent there.

Fisher's conclusion (for autosomal genes) is shown in section 3 to follow from this fact.

2 Analysis of Gene Frequencies

The total frequency Z of a gene A in a diploid population is a weighted average of the frequency X of A in the male population and the frequency Y of A in the female

population:

$$Z = M\,X + F\,Y. \tag{2}$$

Furthermore,

$$M\,X = m\,Z \quad \text{and} \quad F\,Y = f\,Z, \tag{3}$$

where m is the proportion of the male phenotype of A and $f = 1 - m$ is the proportion of the female phenotype of A in the population under consideration. m is also the probability that the individual which develop from an A-carrying gamete would be a male; it is thus the marginal sex ratio of A.

Assuming A to be inertial, X and Y are also the frequencies by which male and female parents, respectively, transmit A to their offspring. The frequency Z' of A among the offspring is then

$$Z' = \frac{1}{2}\,X + \frac{1}{2}\,Y \tag{4}$$

$$= \frac{Z}{2}\left[\frac{m}{M} + \frac{f}{F}\right]. \tag{5}$$

Since the factor on the right-hand side of (5) is similar to the Shaw–Mohler formula—and is referred to as such in the following—we may deduce at once that $Z' > Z$ if $m < M$ and females are the less numerous sex or if $m > M$ and males are the less numerous sex. Thus the frequency of A increases if the relevant inequality between m and M holds and decreases if the converse inequality holds. At an *uneven equilibrium*, an equilibrium with an uneven sex ratio, the marginal sex ratio m of A is therefore necessarily equal to the general sex ratio M. Hence [by (3)], an uneven equilibrium is *symmetric*: $X = Y = Z$.

Thus: *For a diploid population to be in an uneven genetic equilibrium it is necessary that the occurrence of every inertial gene be uncorrelated with sex.*

If the frequency of A is low and the population is otherwise in a genetic equilibrium and if the marginal sex ratio—which in this case is practically the sex ratio of the heterozygotes—is only little affected by changes in the frequency of A, then the Shaw–Mohler formula is nearly constant.

Hence: *An uneven equilibrium is evolutionary unstable: if males predominate then it is unstable to invasion by inertial genes whose marginal sex ratio is lower than the general sex ratio, and if females predominate—to invasion by inertial genes whose marginal sex ratio is higher than the general one.*

Since this evolutionary instability is common to all uneven genetic equilibria, regardless of the particular mechanism which determine the sex ratio, it may, to some degree, account for the paucity of heavily biased sex ratios in dioecious diploid populations.

3 Autosomal Genes Affecting Offspring Sex Ratio

Consider an inertial gene that acts only on parents and affects only the sex ratio of the offspring produced. The fate of the individual gene-carrying gamete is assumed not to be affected by the occurrence of the gene. Then the chance of an individual developing

from a gene-carrying gamete to be a male, say, is equal to the proportion of males in the progeny of the parent who produced the gamete. Hence, the marginal sex ratio of the gene is in the present situation the same as the offspring sex ratio it induces in parents. In particular, a gene that tends to increase the production of offspring of one sex is more common among that sex, as indicated by Nur (1974).

Therefore it follows from the argument of section 2 that the gene would spread in the population if it tends to increase the production of offspring of the less numerous sex. This is essentially Fisher's conclusion. The models studied by Shaw (1958) demonstrate this result.

Furthermore, by (5), the rate with which the frequency of the gene increases is characterized by the Shaw–Mohler formula. In the present situation the interpretations of m and f in (5) coincide with those in (1). Thus, the latter expression, which in the introduction is shown to be related to selection arguments, is shown here to derive from a general genetical principle [eqn (5)] governing the dynamics of inertial genes in a population with an uneven sex ratio. Another aspect of the relationship between (1) and the underlying genetics is described by Uyenoyama & Bengtsson (1979).

4 Multiple Alleles

The sex ratio of the homozygotes may be different from that of the heterozygotes. Generalizing, consider a single locus with an arbitrary number of inertial alleles $A_1, A_2, A_3, \ldots$ having non-vanishing frequencies $Z_1, Z_2, Z_3, \ldots$ in a population at an uneven equilibrium. Let m_{ij} be the sex ratio of genotype A_iA_j in that population. Random mating between males and females is assumed.

Since at an uneven equilibrium the occurrence of the various alleles is uncorrelated with sex the genotypes are in Hardy–Weinberg proportions. Therefore the marginal sex ratio of the allele A_i is

$$m_i = \sum_j m_{ij} Z_j. \tag{6}$$

At an uneven equilibrium the marginal sex ratios of all the alleles are equal to the general sex ratio, i.e.

$$m_i = M, \quad i = 1, 2, \ldots. \tag{7}$$

These equalities imply the inequalities

$$\min_i m_i(\zeta) \leq M \leq \max_i m_i(\zeta), \tag{8}$$

where $m_i(\zeta)$ corresponds to an arbitrary frequencies vector $\zeta = (\zeta_1, \zeta_2, \ldots)$ ($\sum_i \zeta_i = 1$) which is substituted for $Z = (Z_1, Z_2, \ldots)$ in (6). For by $m_{ij} = m_{ji}$,

$$\min_i \sum_j m_{ij} \zeta_j \leq \sum_i Z_i \sum_j m_{ij} \zeta_j = \sum_i \zeta_i \sum_j m_{ij} Z_j = M,$$

which proves the left-hand inequality of (8). The other inequality is similarly proven. Note that these two weak inequalities can only simultaneously be equalities.

Formula (8) shows, in particular, that M is *unique*: only one uneven equilibrium sex ratio is consistent with the given genotype sex ratios. For if the frequencies vector

ζ corresponds to some other uneven equilibrium sex ratio $M(\zeta)$, then applying (7) to that equilibrium shows the left-hand side and the right-hand side of (8) to be equal to $M(\zeta)$.

If the matrix $\|m_{ij}\|$ is regular, then the equilibrium allele frequencies are unique too.

These uniqueness properties critically depend on the assumption that the equilibria considered are *polymorphic*, i.e. the corresponding allele frequencies are different from zero.

4.1 INVASION BY A NEW INERTIAL ALLELE

Suppose that males are the less numerous sex. This assumption causes no real loss of generality since the roles of the two sexes are interchangeable. Then, by section 2, the equilibrium is unstable to invasion by a new low-frequency allele A_0 if $m_0 > M$, where m_0 is given by (6). This condition for instability was found by Eshel & Feldman (1982). Note that the validity of (6) for $i = 0$ follows from the fact that the presence of $A_1, A_2, \ldots$ is uncorrelated with sex. A formal proof would follow the line of thought of eqns (10–14) below.

Suppose that a new uneven polymorphic equilibrium is ultimately established, involving all the alleles $A_0, A_1, A_2, \ldots$. If the sex ratios of the various genotypes are *rigid* then applying (8) to the old frequencies $\zeta_0 = 0, \zeta_1 = Z_1, \zeta_2 = Z_2, \ldots$ and to the new sex ratio $M = M_{\text{new}}$ yields the inequalities

$$M_{\text{old}} < M_{\text{new}} < m_0. \tag{9}$$

Thus, if $\|m_{ij}\|$ does not change then the new sex ratio is intermediate between the old one and the initial marginal sex ratio of the invading allele. Karlin & Lessard (1986) proved that, under certain conditions, a new *stable* polymorphic equilibrium can in fact only correspond to a sex ratio which is intermediate between the old one and the one-to-one sex ratio. This may be interpreted as an evolutionary tendency towards an even sex ratio. An alternative proof is given in Appendix A.

5 Offspring Sex Ratio Determined by Mother's Autosomal Genes

As a special case of the situation considered in section 3 consider a model in which the alleles $A_1, A_2, A_3, \ldots$ of an autosomal locus affect only one of the two parents, say the female. $\underline{m}_{ij}$ is the proportion of males in the progeny of an A_iA_j mother.

Let Y'_{ij} be the frequency of genotype A_iA_j among female offspring when $i = j$ and half the frequency when $i \neq j$. If random mating is assumed then

$$Y'_{ij} = (X_i Q_j + Q_i X_j)/2, \tag{10}$$

where X_i is the frequency of A_i in the male population and Q_i is the frequency by which mothers transmit A_i to their daughters. The frequency of A_i among female offspring is

$$Y'_i = (X_i + Q_i)/2. \tag{11}$$

If the occurrence of A_j is uncorrelated with sex ($X_j = Y_j = Z_j$) and its frequency does not change over the generations then by (11):

$$Y'_j = X_j = Q_j. \tag{12}$$

It follows from (10–12) that the various genotypes of females are in Hardy–Weinberg proportions:

$$Y'_{ij} = Y'_i Y'_j. \tag{13}$$

Hence, the proportion of males in the progeny of a female parent which is induced by A_i is

$$\underline{m}_i = \sum_j \underline{m}_{ij} Y_j = \sum_j \underline{m}_{ij} Z_j. \tag{14}$$

By section 3, a necessary condition for an uneven equilibrium is that this proportion be equal to the general sex ratio:

$$\underline{m}_i = M. \tag{15}$$

Formally, these equations are similar to (6) and (7). Hence, in particular, the equilibrium uneven sex ratio, if it exists, is uniquely determined by $\left\|\underline{m}_{ij}\right\|$. Note that $\underline{m}_{ij}$ is in general different from the corresponding genotype sex ratio m_{ij}: Since a formula analogous to (13) holds for male genotypes, and since $X_j = Y_j$, each genotype has the same relative frequency in males and in females. Thus, the sex ratios of all the genotypes are equal to M and eqn (7) holds trivially. Thus it is only $\left\|\underline{m}_{ij}\right\|$ which determine the uneven equilibrium sex ratio in the present model.

5.1 INVASION BY A NEW INERTIAL ALLELE

Let A_0 be a new low-frequency allele invading the population. In deriving (14) no assumptions are made regarding the distribution of the allele A_i. It therefore holds for $i = 0$ as well. By section 3, A_0 would spread in a population where males are the less numerous sex if it tends to increase the production of sons—i.e. if $\underline{m}_0 > M$, where $\underline{m}_0$ is given by (14). This condition was found by Eshel & Feldman (1982). In a population where females are the less numerous sex A_0 would spread if it tends to increase the production of females.

As in section 4, it follows from inequalities similar to (8) that if a new uneven polymorphic equilibrium is ultimately established than the new sex ratio is intermediate between the old one and $\underline{m}_0$. Moreover, as with the model of rigid genotype sex ratios a new stable uneven sex ratio is necessarily intermediate between the old one and the one-to-one sex ratio; see Karlin & Lessard (1986) and Appendix B.

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APPENDICES

For the model of rigid genotype sex ratios, and for the model of offspring sex determination by mother's autosomal genes the following is shown: *An uneven polymorphic equilibrium with sex ratio $M > \frac{1}{2}$ is unstable if there exists another uneven equilibrium with a lower sex ratio.*

Interchanging the two sexes immediately yields the following corollary: *An uneven polymorphic equilibrium with sex ratio $M < \frac{1}{2}$ is unstable if there exists another uneven equilibrium with a higher sex ratio.*

As shown in section 4, the latter equilibrium is necessarily *not* polymorphic.

APPENDIX A

Proof for the Model of Rigid Genotype Sex Ratios

By (5) and (7), at an uneven polymorphic equilibrium

$$dZ_i' = dZ_i + \frac{1}{2}\left[\frac{1}{M} - \frac{1}{F}\right] Z_i \, dm_i, \tag{A.1}$$

where dZ_i ($\sum_i dZ_i = 0$) are small deviations from the equilibrium values Z_i of the allele frequencies and dm_i are the corresponding deviations from M of the marginal sex ratios. It is shown below that $dM = 0$.

It *formally* follows from (6) that

$$dm_i = \sum_j m_{ij}\, dZ_j, \tag{A.2}$$

but, as (6) refers only to equilibrium, this equality has to be verified.

The frequency of genotype A_iA_j among the offspring of the present generation is

$$Z'_{ij} = (X_i Y_j + Y_i X_j)/2 \tag{A.3}$$

if $i = j$, and twice that value if $i \neq j$. Hence by (4) and the symmetric nature of uneven equilibria:

$$\begin{aligned} dZ'_{ij} &= (X_i\, dY_j + Y_i\, dX_j + Y_j\, dX_i + X_j\, dY_i)/2 \\ &= Z_i\, dZ'_j + Z_j\, dZ'_i = d\left(Z'_i\, Z'_j\right). \end{aligned} \tag{A.4}$$

By (A.4) and the equality $m_i Z_i = \sum_j m_{ij} Z_{ij}$:

$$m_i\, dZ_i + Z_i\, dm_i = \sum_j m_{ij}\,(Z_i\, dZ_j + Z_j\, dZ_i),$$

$$M\, dZ_i + Z_i\, dm_i = Z_i \sum_j m_{ij}\, dZ_j + M\, dZ_i,$$

and thus (A.2) is verified. By (A.4), (6) and (7), $dM = \sum_{i,j} m_{ij}\, dZ_{ij}$ is equal to 0.

Since for $M > \frac{1}{2}$ the factor $[1/M - 1/F]$ is negative, it remains to show that the matrix $\left\|Z_i m_{ij}\right\|$ admits a negative characteristic value $\lambda < 0$. For if $\varepsilon_1, \varepsilon_2, \ldots$ are the components of a corresponding characteristic vector then by definition $\sum_j Z_i m_{ij} \varepsilon_j = \lambda \varepsilon_i$, hence $M \sum_j \varepsilon_j = \sum_{i,j} Z_i m_{ij} \varepsilon_j = \lambda \sum_i \varepsilon_i$, and therefore $\sum_i \varepsilon_i = 0$ and choosing $dZ_i = \varepsilon_i$ yields [by (A.1) and (A.2)]:

$$dZ'_i = dZ_i \left(1 + \frac{\lambda}{2}\left[\frac{1}{M} - \frac{1}{F}\right]\right). \tag{A.5}$$

Since $\left\|Z_i m_{ij}\right\|$ is similar to the symmetric matrix $\left\|\sqrt{Z_i}\, m_{ij} \sqrt{Z_j}\right\|$, and the two matrices hence share the same set of characteristic values, it suffices to show that the numerical range of the quadratic form $\Phi(\xi_1, \xi_2, \ldots) = \sum_{i,j} m_{ij}\, \xi_i\, \xi_j$ contains negative numbers. Indeed, by applying (7) to both equilibria we find that $\Phi(Z_1 - \zeta_1, Z_2 - \zeta_2, Z_3 - \zeta_3, \ldots) = \bar{M} - M < 0$, where $\zeta_1, \zeta_2, \ldots$ are the allele frequencies which correspond to the uneven equilibrium with sex ratio $\bar{M} < M$.

Note that, since it is implied by (7) that the spectral radius of $\left\|Z_i m_{ij}\right\|$ is M (Gantmacher, 1959), $|\lambda| \leq M$. Moreover, since $-\lambda$ is a characteristic value [with characteristic vector $(\varepsilon_1, \varepsilon_2, \ldots)$] of the matrix $\left\|Z_i f_{ij}\right\|$, $|\lambda| \leq F$. It hence follows from (A.5) that

$$\left|dZ'_i\right| < \frac{3}{2}\left|dZ_i\right|, \tag{A.6}$$

which puts an upper bound to the rate of change of Z_i near uneven polymorphic equilibria.

APPENDIX B

Proof for the Model of Offspring Sex Determination by Mother's Autosomal Genes

The proof is similar to that for the first model. Equations (A.3) and (4) should be replaced by (10) and (11), respectively, m_{ij} should be replaced by $\underline{m}_{ij}$ and, where appropriate, frequencies in the whole population should be replaced by frequencies in the female population.

The only difficulty is that the analogue of eqn (A.2),

$$d\underline{m}_i = \sum_j \underline{m}_{ij}\, dY_j, \tag{B.1}$$

does not directly leads to an equation similar to (A.5). However, choosing $dY_1, dY_2, \ldots$ to be the components of a characteristic vector of the matrix $\left\| Z_i \underline{m}_{ij} \right\|$ with characteristic value $\lambda < 0$ does lead, on the next generation, to the equality

$$Z_i\, dm'_i = \underline{\lambda}\, dZ'_i, \tag{B.2}$$

and hence, on the following generation, to the formula [derived from (A.1)]

$$dZ''_i = dZ'_i \left(1 + \frac{\underline{\lambda}}{2} \left[\frac{1}{M} - \frac{1}{F} \right] \right), \tag{B.3}$$

if the equalities

$$2\, dm'_i = d\underline{m}_i \tag{B.4}$$

and

$$2\underline{\lambda}\, dZ'_i = \lambda\, dY_i \tag{B.5}$$

hold for some negative constant $\underline{\lambda}$.

Equation (B.4) is derived from the equality $F\,(X_i + Q_i) = f'_i\,(X_i + Y_i)$, which follows from (3), (4) and (11), and the identity $F\,Q_i = \underline{f}_i\, Y_i$ (where $\underline{f}_i = 1 - \underline{m}_i$): differentiating at equilibrium and canceling identical terms yields $Z_i\, d\underline{f}_i = 2Z_i\, df'_i$, hence (B.4).

Equation (B.5) is satisfied on the first generation if dX_i is properly chosen, according to (4). Since, by (3) and (B.2),

$$F\, dY'_i = f'_i\, dZ'_i + Z'_i\, df'_i = F\, dZ'_i - \underline{\lambda}\, dZ'_i, \tag{B.6}$$

the validity of (B.5) on the second generation, and hence on all subsequent generations, is implied by (B.3) and (B.6) if $\underline{\lambda}$ is chosen to be the unique negative root of the quadratic equation

$$2 + \underline{\lambda} \left(\frac{1}{M} - \frac{1}{F} \right) = \lambda \left(\frac{1}{\underline{\lambda}} - \frac{1}{F} \right). \tag{B.7}$$

Note that, since $|\lambda| \leq F < M$, $\underline{\lambda}$ satisfies the approximation $\lambda < \underline{\lambda} < \lambda/2$. Hence, by comparison of (B.3) with (A.5), the allele frequencies, with initial conditions as indicated above, diverge more slowly from their unstable equilibrium values than in the corresponding case with rigid genotype sex ratios.