THEORETICAL POPULATION BIOLOGY 31, 339–358 (1987)

The Role of Recombination and Selection in the Modifier Theory of Sex-Ratio Distortion*

SABIN LESSARD

Département de Mathématiques et de Statistique, Université de Montréal, Montréal, Canada H3C 3J7

Received September 12, 1985

The equilibrium configurations for a two-locus multialle model of sex-linked meiotic drive are studied with regard to the recombination fraction: limit cycles can occur in the case of small recombination while stable equilibrium points associated with linkage equilibrium can exist for an intermediate range of recombination values depending on the equilibrium sex ratio, linkage disequilibrium at nearby equilibrium points taking turn with loser linkage. The evolutionary dynamics in two-locus sex-ratio distortion systems is enlightened: while equilibria with a sex ratio closer to $\frac{1}{2}$ are more likely to be stable with respect to perturbations on the frequencies of sex-ratio distorters that are represented at equilibrium, such equilibria are also more vulnerable to the invasion of mutant distorters when there is some degree of linkage with the sex-determining locus. For X-linked multimodifier systems of sex-ratio distortion, differential fertilities and viabilities are incorporated and a maximum principle is suggested.

1. INTRODUCTION

In species where males are heterogametic XY and females homogametic XX, a male can affect its progeny sex ratio by differentially producing the two types of gametes X and Y. Mendelian segregation postulates that X- and Y-gametes are produced in equal numbers at meiosis leading on the average to a one-to-one progeny sex ratio at conception. Meiotic drive in favor of X- or Y-gametes can be viewed as a form of gametic selection at an early stage of the life cycle. Actually it may be difficult to discern meiotic drive per se in producing gametes from gametic selection at a later stage of development because the effects on gene frequencies may be identical.

Sex-ratio distortion can be initiated by sex-linked or autosomal segregation modifiers active in the heterogametic sex. In *Drosophila obscura*

* This research was supported in part by the Direction générale de l'enseignement Supérieure du Québec and the Natural Sciences and Engineering Research Council of Canada, Grant No. A8833.

0040-5809/87 $3.00

and some related species (Gershenson, 1928; Sturtevant and Dobzhansky, 1936) there exists a driving X-chromosome, devoted X_r, such that $X_r Y$-males mainly transmit X_r-gametes to their progeny. This is known as the Sex-Ratio System. In the mosquito *Aedes aegypti*, sex is determined at an autosomal locus, males being heterozygous (Mm) and females homozygous (mm). Moreover there exists a sex-ratio distorter D tightly linked to the sex-determining locus such that the progeny of MD/md males generally contains more males than females (Hickey and Graig, 1966).

Sex-linked meiotic drive modifiers have also been found in the butterfly *Acraea encedon* (Chanter and Owen, 1972) and in the wood lemming *Myopus schisticolor* (Fredga *et al.*, 1976). In this species, the meiotic drive modifier is X-linked and suppresses the male-determining effect of the Y-chromosome leading to heterogametic females having mostly daughters.

Autosomal suppressors of sex-linked meiotic drive distorters have been reported in *D. paramelanica* (Stalker, 1961), *D. affinis* (Novitski, 1974), and in the butterfly *Danaus chrysippus* (Smith, 1975). Autosomal genes directly distorting the progeny sex ratio of the heterogametic sex have been observed in *D. melanogaster* (Bell, 1954; Sandler, 1970) and *D. simulans* (Faulhaber, 1967).

The evolutionary response to X-linked or Y-linked meiotic drive modifiers has been studied in Edwards (1961), Hamilton (1967), Thomson and Feldman (1975), and Curtsinger and Feldman (1980), among others. It has been argued that sex-linked modifiers of progeny sex ratio should increase in frequency under random mating if they promote their own representation in the future generations until genetic fixation is reached. In the long run with the introduction of new mutant modifiers, such an evolutionary process should lead to an all female or an all male population if no other selective forces come into play to balance the tendency (see, e.g., Karlin and Lessard, 1986, Chap. 6). Therefore viability and/or fertility differences, not to mention mating constraints and population structures, have been incorporated to sex-ratio distortion systems to explain the maintenance of polymorphic equilibria in natural populations with sex-linked modifiers. (See Bengtsson (1977) for models incorporating sex determination, sex-ratio distortion, and fertility differences as occurs in the wood lemming.)

Maffi and Jayakar (1981) introduced a two-locus two-allele model of sex-linked meiotic drive with possible applications to *Aedes aegypti* populations in order to investigate the influence of the recombination fraction on the possibility of maintaining polymorphic equilibria in the absence of fitness differences. Numerical simulations partly supported by local stability analyses (see Lessard and Karlin (1982) for a complement of analysis) revealed that limit cycles can occur in the case of tight linkage while multiple stable polymorphic equilibria can coexist when there is sufficient

recombination. On the other hand, Wu (1983) showed that autosomal suppressors of X-linked meiotic drive modifiers as occurs in Drosophila would tend to increase in frequency if no fitness differences are assumed.

In this paper, we first extend Maffi and Jayakar's model by allowing any number of meiotic drive modifiers at a sex-linked locus. We focus on an important class of polymorphic equilibria—those associated with linkage equilibrium—also called symmetric. The exact analytical conditions for internal and external stability of such equilibria in terms of the recombination fraction and the equilibrium population sex ratio are examined. Of particular interest are the threshold values at which there are bifurcations to surrounding limit cycles or nearby isolated non-symmetric equilibrium points. In our description of the equilibrium configurations, special attention is given to the cases of tight linkage and free recombination. We also analyse X-linked modifier systems of sex-ratio distortion in a multiallele setting. In this model, we incorporate the effects of viability, fertility, and virility differences that can be associated with sex-ratio modifiers.

Previous studies on autosomal modifiers of sex-ratio determination (see, e.g., Eshel, 1975; Uyenoyama and Bengtsson, 1979; Eshel and Feldman, 1982; Wu, 1983; Karlin and Lessard, 1983, 1984) have revealed an evolutionary tendency toward on equal representation of males and females in the population. The questions of interest concern the existence and stability of polymorphic equilibria, the fate of mutant modifiers, and the evolutionary properties of the sex ratio. Our main objective in this paper is to describe the evolutionary dynamics of sex-ratio distortion systems when the sex-determination locus and the progeny sex-ratio modifier locus are generally linked in order to highlight the dependence on the recombination fraction. All proofs are relegated to the Appendix.

2. A Sex-Linked Two-Locus Multiallele Sex-Ratio Distortion Model

A primary locus with two possible alleles M and m is assumed to be responsible for sex determination: males are Mm while females are mm. A secondary locus allowing alleles $A_1,\ldots, A_n$ governs meiotic drive, namely, the sex ratio in the progeny of males: MA_i/mA_j genotypes segregate gametes carrying the alleles M and m in the proportions s_{ij} and $1-s_{ij}$, respectively. The sex-ratio distortion matrix $S=\|s_{ij}\|$ is assumed to be positive and symmetric, namely $0<s_{ij}=s_{ji}<1$ for all i, j.

A recombination event between the sex-ratio distortion locus and the sex-determination locus occurs with probability r $(0<r\leqslant\frac{1}{2})$ prior to the meiotic drive effects. Hence a typical male MA_i/mA_j transmits the gametes MA_i, MA_j, mA_i, and mA_j with the frequencies $(1-r)s_{ij}$, rs_{ij}, $r(1-s_{ij})$, and

$(1-r)(1-s_{ij})$, respectively, while the gametes mA_i and mA_j are equally represented in the gametic production of a typical female mA_i/mA_j.

Let Z_i be the frequency of mA_i gametes among all m gametes transmitted by males and Y_i be the frequency of MA_i gametes among all M gametes. Moreover, let X_i denote the frequency of mA_i among all gametes produced by females. Assuming random mating (i.e., random union of gametes) and discrete generations in a large population (without sampling drift effects), the recurrence relations over two successive generations are

$$X_i' = \frac{X_i + Z_i}{2} \tag{2.1a}$$

$$Y_i' = \frac{(1-r)\, Y_i \sum_j s_{ij} X_j + r X_i \sum_j s_{ij} Y_j}{\sum_{i,j} s_{ij} X_i Y_j} \tag{2.1b}$$

$$Z_i' = \frac{r Y_i \sum_j (1-s_{ij})\, X_j + (1-r)\, X_i \sum_j (1-s_{ij})\, Y_j}{\sum_{i,j} (1-s_{ij})\, X_i Y_j} \tag{2.1c}$$

for $i = 1,..., n$. In vector notation, the recurrence system (2.1) can be written in the form

$$\mathbf{X}' = \frac{\mathbf{X} + \mathbf{Z}}{2} \tag{2.2a}$$

$$\mathbf{Y}' = \frac{(1-r)\, \mathbf{Y} \circ S\mathbf{X} + r\mathbf{X} \circ S\mathbf{Y}}{\langle \mathbf{X}, S\mathbf{Y} \rangle} \tag{2.2b}$$

$$\mathbf{Z}' = \frac{r\mathbf{Y} \circ (U-S)\, \mathbf{X} + (1-r)\, \mathbf{X} \circ (U-S)\, \mathbf{Y}}{1 - \langle \mathbf{X}, S\mathbf{Y} \rangle} \tag{2.2c}$$

where U is the matrix of all unit entries while $\boldsymbol{X} \circ \boldsymbol{Y} = (X_1 Y_1,..., X_n Y_n)$ denotes the product component by component and $\langle \mathbf{X}, \mathbf{Y} \rangle = \sum_i X_i Y_i$ is the scalar product for two vectors $\mathbf{X} = (X_1,..., X_n)$ and $\mathbf{Y} = (Y_1,..., Y_n)$.

The quantity

$$s = s(\mathbf{X}, \mathbf{Y}) = \langle \mathbf{X}, S\mathbf{Y} \rangle = \sum_{i,j} s_{ij} X_i Y_j \tag{2.3}$$

represents the average segregation ratio in favor of allele M in the population, that is, the proportion of male offspring, the *sex ratio*, in the current generation.

Polymorphic Symmetric Equilibria and Conditions for Their Stability

An important class of equilibria $\{\mathbf{X}^*, \mathbf{Y}^*, \mathbf{Z}^*\}$ of (2.2) has $\mathbf{X}^* = \mathbf{Y}^* = \mathbf{Z}^*$. Such equilibria are said to be *symmetric*. Symmetric

equilibria exhibit linkage equilibrium in the sense that the allelic frequencies at the sex-ratio distortion locus are independent of the sex-determining alleles. In view of (2.2), this occurs if and only if the equilibrium allelic frequency vector $\mathbf{X}^*$ at the sex-ratio distortion locus satisfies the relation

$$\mathbf{X}^* = \frac{\mathbf{X}^* \circ S\mathbf{X}^*}{\langle \mathbf{X}^*, S\mathbf{X}^* \rangle}. \tag{2.4}$$

The associated population sex ratio is $s^* = \langle \mathbf{X}^*, S\mathbf{X}^* \rangle$. We will suppose throughout $s^* \leqslant \frac{1}{2}$. (Otherwise, replace S by $U - S$, i.e., interchange the sexes.)

The assumption S non-singular guarantees that there exists at most one *polymorphic symmetric equilibrium*, i.e., $\mathbf{X}^* = (X_1^*, \ldots, X_n^*)$ exhibiting all positive components and satisfying (2.4). For a polymorphic symmetric equilibrium $\mathbf{X}^*$, the matrix

$$S^* = S(\mathbf{X}^*) = \frac{\mathbf{X}^* \circ S}{s^*}, \tag{2.5}$$

whose ith row is the ith row of S multiplied by X_i^*/s^* for $i = 1, \ldots, n$, is positive. Equation (2.4) says that 1 is an eigenvalue of $S(\mathbf{X}^*)$ with (positive) right eigenvector $\mathbf{X}^*$. The other eigenvalues of $S(\mathbf{X}^*)$, which are real since $S(\mathbf{X}^*)$ is a product of a positive diagonal matrix with a positive symmetric matrix, are all less than 1 in absolute value owing to the Perron–Frobenius theory (see, e.g., Gantmacher, 1959). The stability nature of $\mathbf{X}^*$ can be determined as follows.

RESULT I. *A polymorphic symmetric equilibrium* $\mathbf{X}^*$ *for the sex-ratio distortion model* (2.2) *with equilibrium population sex-ratio* $s^* = \langle \mathbf{X}^*, S\mathbf{X}^* \rangle$ *less than* $\frac{1}{2}$ *is stable if for every eigenvalue* $\lambda \neq 1$ *of* $S^* = \mathbf{X}^* \circ S/s^*$, *the recombination rate* r *satisfies*

$$\max\{r_1(\lambda), r_2(\lambda)\} < r < \min\{r_*(\lambda), 1/2\} \tag{2.6}$$

where

$$r_*(\lambda) = \begin{cases} \dfrac{\lambda s^*}{1 - 2s^* + 2\lambda s^*} & \text{if} \quad \lambda > 0 \\ 1/2 & \text{if} \quad \lambda < 0 \end{cases} \tag{2.7}$$

$$r_1(\lambda) = \frac{3\lambda^2 s^*}{8 - 3\lambda - (8 - 4\lambda - 6\lambda^2)\, s^*}, \tag{2.8}$$

and $r_2(\lambda)$ *is the root between* 0 *and* $\frac{1}{2}$ *of the quadratic form in* r

$$Q(r) = 1 - b + ac - c^2 \tag{2.9}$$

where

$$\begin{aligned} a &= r(1-\lambda) - 3/2, \\ b &= \frac{r\lambda}{2(1-s^*)}, \\ c &= \frac{(1-2r)(1-s^*+\lambda^2 s^*)}{2(1-s^*)}. \end{aligned} \tag{2.10}$$

The equilibrium $\mathbf{X}^*$ *is unstable if any of the inequalities* (2.6) *is reversed.*

In practice, it suffices to find out the eigenvalues of S^* to apply the criterion (2.6). For example, in the case $n = 2$,

$$\mathbf{X}^* = \left(\frac{s_{22} - s_{12}}{s_{11} - 2s_{12} + s_{22}}, \frac{s_{11} - s_{12}}{s_{11} - 2s_{12} + s_{22}} \right),$$

$$s^* = \frac{s_{11}s_{22} - s_{12}^2}{s_{11} - 2s_{12} + s_{22}},$$

and the only eigenvalue λ of $\mathbf{X}^* \circ S/s^*$ different from 1 is

$$\lambda = \frac{(s_{11} - s_{12})(s_{22} - s_{12})}{s_{11}s_{22} - s_{12}^2}.$$

In general, suppose that a polymorphic symmetric equilibrium $\mathbf{X}^*$ of (2.2) is stable for some intermediate range of recombination values r. According to condition (2.6), the equilibrium will become unstable if r increases sufficiently or decreases beyond some minimum treshold. It can be argued (see Appendix) that there will be a transfer of stability to nearby isolated equilibrium points in the former case while periodic orbits will emerge from $\mathbf{X}^*$ (or will collapse to $\mathbf{X}^*$) by Hopf bifurcations in the latter (see, e.g., Marsden and McCracken, 1976).

Stability of Symmetric Equilibria against Mutant Distorters

Consider a mutant distorter A_{n+1} at the sex-ratio distortion locus. Let $s_{i,n+1}$ be the proportion of M gametes in the gametic pool of either MA_i/mA_{n+1} or MA_{n+1}/mA_i males. Near a polymorphic symmetric equilibrium $\mathbf{X}^* = (X_1^*, \ldots, X_n^*)$ of (2.2), the quantity

$$s_{n+1}^* = \sum_{i=1}^{n} s_{i,n+1} X_i^* \tag{2.11}$$

measures the average marginal sex-ratio distortion in favor of males in the progeny of all males carrying allele A_{n+1}. The fate of the mutant allele depends upon s^*_{n+1}, the population sex-ratio s^*, and the recombination fraction r. We assume throughout $s^*_{n+1} \neq s^*$.

RESULT II. *A polymorphic symmetric equilibrium* $\mathbf{X}^*$ *for the sex-ratio distortion model* (2.2) *with equilibrium population sex ratio* $s^* < \frac{1}{2}$ *will resist the introduction of a mutant distorter* A_{n+1} *if*

$$r(s^* - s^*_{n+1})(1 - 2s^*_{n+1}) > (s^* - s^*_{n+1})^2 \tag{2.12}$$

where r *is the recombination rate and* s^*_{n+1} *is the mutant marginal sex-ratio distortion as defined in* (2.11). *If the inequality* (2.12) *is reversed, i.e.,* $s^*_{n+1} > s^*$ *or otherwise* $r < (s^* - s^*_{n+1})/(1 - s^*_{n+1})$, *the mutant allele* A_{n+1} *will tend to increase in frequency after some generations following its introduction.*

We will refer throughout to condition (2.12) as the condition for *external stability* following the introduction of a new sex-ratio distorter at a symmetric equilibrium while condition (2.6) will define *internal stability* of a symmetric equilibrium within its own allelic system for which all our assumptions will be extended mutatis mutandis. Some limit cases are of particular interest.

Case of Symmetric One-to-One Population Sex-Ratio Equilibria

The stability nature of a symmetric equilibrium $\mathbf{X}^*$ associated with a one-to-one population sex ratio, i.e., $s^* = \langle \mathbf{X}^*, S\mathbf{X}^* \rangle = \frac{1}{2}$, raises an interesting paradox. As a matter of fact, Results I and II extended to this case lead to a rather surprising conclusion.

RESULT III. *At least for* $(3 - \sqrt{3})/4 < r < 1/2$, *a symmetric equilibrium of the sex-ratio distortion model* (2.2) *resulting in a one-to-one population sex ratio is always internally stable and always externally unstable.*

Case of Small Recombination

When the sex-ratio distortion locus and the sex-determination locus are tightly linked, it can be argued that every equilibrium of (2.2) must be symmetric. Moreover, the conditions (2.6) and (2.12) cannot be fulfilled. We conclude that only limit cycles or chaotic behaviors can be observed when r is positive but small.

RESULT IV. *In the case of small recombination, every equilibrium point of the sex-ratio distortion model* (2.2) *is internally as well as externally unstable.*

Case of Free Recombination

With free recombination ($r = \frac{1}{2}$), the conditions (2.6) and (2.12) for stability of a symmetric equilibrium are amenable to simpler forms as stated below.

RESULT V. *Suppose* $r = \frac{1}{2}$ *in the sex-ratio distortion model* (2.2). *A polymorphic symmetric equilibrium* $\mathbf{X}^*$ *with population sex ratio* $s^* < \frac{1}{2}$ *is internally stable if every eigenvalue* $\lambda \neq 1$ *of* $S(\mathbf{X}^*) = \mathbf{X}^* \circ S/s^*$ *is negative. It is externally unstable against a mutant distorter* A_{n+1} *if the average mutant sex-ratio distortion* s^*_{n+1} *of* (2.11) *exceeds* s^*.

(Compare with Karlin and Lessard (1983)).

The analysis of this case is complemented by noting that a non-symmetric equilibrium $\{\tilde{\mathbf{X}}, \tilde{\mathbf{Y}}, \tilde{\mathbf{Z}}\}$ of (2.2) with $r = \frac{1}{2}$ must satisfy $\tilde{s} = \langle \tilde{\mathbf{X}}, S\tilde{\mathbf{Y}} \rangle = \frac{1}{2}$ since we have

$$\tilde{s}\tilde{\mathbf{Y}} + (1 - \tilde{s})\,\tilde{\mathbf{Z}} = (\tilde{\mathbf{Y}} + \tilde{\mathbf{X}})/2 = (\tilde{\mathbf{Y}} + \tilde{\mathbf{Z}})/2 \tag{2.13}$$

and $\tilde{\mathbf{Y}} \neq \tilde{\mathbf{Z}}$ by assumption. One-to-one population sex-ratio equilibria are therefore characterized by the equation

$$\tilde{\mathbf{Y}} = \tilde{\mathbf{Y}} \circ S\tilde{\mathbf{X}} + \tilde{\mathbf{X}} \circ S\tilde{\mathbf{Y}} \tag{2.14}$$

and the equality $\tilde{\mathbf{Z}} = \tilde{\mathbf{X}}$. Equation (2.14) arose in previous studies of sex-ratio determination models (Karlin and Lessard, 1983, 1984). It can be interpreted as follows.

Define

$$B(\mathbf{X}) = \operatorname{diag}(S\mathbf{X}) + \operatorname{diag}(\mathbf{X})\, S \tag{2.15}$$

for every frequency vector $\mathbf{X}$ ($\operatorname{diag}(\mathbf{X})$ designates the diagonal matrix with the components of $\mathbf{X}$ on the main diagonal). If the components of $\mathbf{X}$ are all positive, then the matrix $B(\mathbf{X})$ is positive. Its leading eigenvalue, denoted by $\rho(\mathbf{X})$, is real and positive owing to the Perron–Frobenius theory (see, e.g., Gantmacher, 1959). Moreover there exists a unique positive frequency vector $\mathbf{Y} = \mathbf{Y}(\mathbf{X})$ satisfying

$$B(\mathbf{X})\,\mathbf{Y} = \rho(\mathbf{X})\,\mathbf{Y}. \tag{2.16}$$

Comparing Equation (2.16) with (2.14) informs us that the polymorphic one-to-one sex-ratio equilibria are determined by the relation

$$\rho(\tilde{\mathbf{X}}) = 1; \tag{2.17}$$

i.e., they are associated with the level surface corresponding to the value 1

of the spectral functional $\rho(\mathbf{X})$ defined on the set of all positive frequency vectors. Combining this information with Result V allows us to apply the conclusions of Karlin and Lessard (1983, 1984) (see also Lessard, 1986) to the sex-ratio distortion model (2.2).

RESULT VI. *Consider the sex-ratio distortion model* (2.2) *with* $r = \frac{1}{2}$.

(a) *A positive frequency vector* $\mathbf{X}^*$ *with* $s^* = \langle \mathbf{X}^*, S\mathbf{X}^* \rangle < \frac{1}{2}$ *is a polymorphic symmetric equilibrium if and only if it is a critical point of* $\rho(\mathbf{X})$ *defined by* (2.16) *on all positive frequency vectors. It is stable if and only if it corresponds to a local maximum of* $\rho(\mathbf{X})$.

(b) *One-to-one sex-ratio equilibrium surfaces occur if and only if there exist two symmetric equilibria* $\mathbf{X}_\alpha^*$ *and* $\mathbf{X}_\beta^*$ *with equilibrium population sex-ratios* $s_\alpha^* < \frac{1}{2}$ *and* $s_\beta^* > \frac{1}{2}$, *respectively.*

(c) *When an initially stable polymorphic symmetric equilibrium* $\mathbf{X}^*$ *with corresponding* $s^* < \frac{1}{2}$ *becomes unstable following the introduction of a mutant distorter* (*see Result V*), *then in the augmented allelic system, either* (i) *there exists a unique stable symmetric equilibrium that is associated with a population sex ratio closer to* $\frac{1}{2}$ *than* s^*, *or* (ii) $\mathbf{X}^*$ *is enclosed in a one-to-one sex-ratio equilibrium surface.*

(see loc. cit. for more details.)

3. An X-Linked Multiallele Sex-Ratio Distortion Model Incorporating Differential Fertilities and Viabilities

Consider a population where all X_iY genotypes are males and all X_iX_j genotypes females for $i, j = 1,..., n$. An X_iY male produces X_i-carrying gametes with probability s_i and Y-carrying gametes with probability $(1 - s_i)$. Viability and virility are also affected by the modifier and are denoted by b_i and f_i, respectively. We will assume throughout $s_i f_i b_i = \sigma_i > 0$ for $i = 1,..., n$. The viability and fertility of an X_iX_j female are represented by b_{ij} and f_{ij}, respectively. Fertility and virility are assumed to be independent, i.e., the number of offspring produced by a mating $X_iX_j \times X_kY$ is the product $f_{ij} \times f_k$. This is a reasonable assumption at least for *D. pseudoobscura* according to Curtsinger's and Feldman's (1980) observations. The female viability–fertility matrix $V = \|v_{ij}\|$ where $v_{ij} = b_{ij} f_{ij}$ is assumed to be symmetric, positive, and non-singular.

Suppose an infinite population undergoing discrete generations and random mating. Using the notation p_i for the proportion of X_i-chromosomes among all X-chromosomes maternally transmitted to the next generation

and q_i for the corresponding proportion in male parents, the recurrence relations are

$$q_i' = \frac{p_i \sigma_i}{\sum_k p_k \sigma_k}, \qquad i = 1,..., n, \tag{3.1a}$$

$$p_i' = \frac{p_i \sum_j v_{ij} q_j + q_i \sum_j v_{ij} p_j}{2 \sum_{k,l} v_{kl} p_k q_l}, \qquad i = 1,..., n. \tag{3.1b}$$

In terms of the frequency vectors $\mathbf{p} = (p_1,..., p_n)$ and $\mathbf{q} = (q_1,..., q_n)$ and the positive vector $\boldsymbol{\sigma} = (\sigma_1,..., \sigma_n)$, we have

$$\mathbf{q}' = \frac{\mathbf{p} \circ \boldsymbol{\sigma}}{\langle \mathbf{p}, \boldsymbol{\sigma} \rangle}, \tag{3.2a}$$

$$\mathbf{p}' = \frac{\mathbf{p} \circ V\mathbf{q} + \mathbf{q} \circ V\mathbf{p}}{2\langle \mathbf{p}, V\mathbf{q} \rangle}, \tag{3.2b}$$

in the notation of (2.2). A polymorphic equilibrium $\{\mathbf{p}^*, \mathbf{q}^*\}$ of (3.2) represented by $\mathbf{p}^*$ is characterized by the relation

$$(V^{\sigma}\mathbf{p}^*)_i = \langle \mathbf{p}^*, V^{\sigma}\mathbf{p}^* \rangle = w^* \tag{3.3}$$

for every component $i = 1,..., n$ of $V^{\sigma}\mathbf{p}^*$ where

$$V^{\sigma} = \frac{V \circ \boldsymbol{\sigma} + \boldsymbol{\sigma} \circ V}{2} = \left\| v_{ij}\left(\frac{\sigma_i + \sigma_j}{2}\right) \right\|. \tag{3.4}$$

Such an equilibrium $\mathbf{p}^*$ is unique if it exists in generic cases (namely, when V^{σ} is invertible). Introducing the quantity

$$w_{n+1}^* = \sum_{i=1}^{n} v_{i,n+1}\left(\frac{\sigma_i + \sigma_{n+1}}{2}\right) p_i^* \tag{3.5}$$

and combining together the equations (3.1a) and (3.1b) for a new modifier X_{n+1}, we have the linear approximation

$$p_{n+1}'' \cong p_{n+1}\left(\frac{w_{n+1}^*}{w^*}\right) + (p_{n+1}' - p_{n+1})\left[\frac{\sum_{i=1}^{n} v_{i,n+1}\sigma_i p_i^*}{2w^*}\right] \tag{3.6}$$

near $\mathbf{p}^*$. Therefore the following condition for external stability of $\mathbf{p}^*$ can be ascertained.

RESULT VII. *A polymorphic equilibrium of* (3.2) *is externally stable*

following an increase in the number of modifiers segregating in the population (from n to n+1) if the quantities defined in (3.3) *and* (3.5) *satisfy*

$$w^*_{n+1} < w^*. \tag{3.7}$$

If the inequality (3.7) *is reversed, then* $\mathbf{p}^*$ *is externally unstable.*

In the two-dimensional case (i.e., with two modifiers represented), internal stability can be decided as follows.

RESULT VIII. *A polymorphic equilibrium of* (3.1) *in the case* $n=2$ *exists and is (internally) stable if and only if*

$$v_{12}\left(\frac{\sigma_1+\sigma_2}{2}\right) > \max\{v_{11}\sigma_1, v_{22}\sigma_2\}. \tag{3.8}$$

It exists but is unstable if and only if

$$v_{12}\left(\frac{\sigma_1+\sigma_2}{2}\right) < \min\{v_{11}\sigma_1, v_{22}\sigma_2\}. \tag{3.9}$$

(Compare with Cannings (1967).)

In general, it is surmised (and confirmed by numerical simulations) that a polymorphic equilibrium $\mathbf{p}^*$ of (3.2) is internally stable if and only if the eigenvalues of $\mathbf{p}^* \circ V^\sigma$ different from w^* are all negative as occurs for one-locus multiallele viability regimes with viability matrix V^σ (see, e.g., Karlin, 1978).

When $v_{ij}=1$ for all $i,j=1,\ldots,n$, i.e., without viability and fertility differences in females, polymorphic equilibria are impossible and the fixation state with the highest σ_i is globally stable since the quantity $\sum_{i=1}^n (p_i+q_i/2)\sigma_i$ increases from generation to generation until fixation is reached.

RESULT IX. *The recurrence system* (3.1) *with* $v_{ij}=1$ *for all* $i,j=1,\ldots,n$ *leads in the long run to the fixation state associated with the largest* σ_i.

4. DISCUSSION

The equilibrium structure of sex-linked modifier systems of sex-ratio distortion strongly depends on the recombination fraction: limit cycles for small values, stable symmetric equilibria associated with linkage equilibrium for intermediate values, stable non-symmetric equilibria exhibiting linkage disequilibrium for larger values. We notice a kin resem-

blance with autosomal modifier systems of autosomal segregation (Lessard, 1985; Eshel, 1985; Thomson and Feldman, 1976; Prout *et al.*, 1973). Although the exact threshold values to pass from one equilibrium configuration to another are different, both types of systems possess analogous properties depending on the recombination level.

With free recombination, the sex-ratio distortion model (2.2) is equivalent to an autosomal modifier system of sex determination as studied by Karlin and Lessard (1983, 1984) (see also Eshel and Feldman, 1982; Uyenoyama and Bengtsson, 1979). In this case, it was argued that, from one equilibrium to the next one following the introduction of a new mutant modifier, the population sex ratio should come closer to $\frac{1}{2}$. We have shown that linkage perturbs this evolutionary tendency: although symmetric equilibria with sex ratio closer to $\frac{1}{2}$ are more likely to be (internally) stable, they are also more subject to destabilization by mutant modifiers. With a one-to-one population sex ratio, a symmetric equilibrium is automatically internally stable and externally unstable. This phenomenon may be attributed to the hitchhiking effects of linkage with the sex determination locus that promotes any more polymorphism at the sex-ratio modifier locus and so preserves modifiers that would become extinct otherwise. In such systems, we would still expect a population sex ratio around $\frac{1}{2}$ but kept away from $\frac{1}{2}$ by an increasing number of modifiers. This is another case where evolutionary stability cannot be decided from initial increase properties of mutant modifiers.

X-linked modifier systems of sex-ratio distortion are akin with standard sex-linked viability models as studied by Cannings (1967) and Haldane and Jayakar (1964), among others. Curtsinger and Feldman (1980) studied the problem of protected polymorphism in the case of two modifiers incorporating viability as well as fertility and virility differences . For the general model (3.2) with multiple modifiers, we have shown that the conditions for equilibrium and stability generally correspond to those for one-locus multiallele viability regimes with viability matrix $\|v_{ij}(\sigma_i+\sigma_j)/2\|$ (see, e.g., Karlin, 1972, 1978). Therefore from one equilibrium to the next one following the introduction of a new modifier, we would analogously expect an increase of the mean "fitness" function

$$\sum_{i,j=1}^{n} p_i^* p_j^* v_{ij}\left(\frac{\sigma_i+\sigma_j}{2}\right). \tag{4.1}$$

Thus higher values σ_i would be generally favored unless they are associated with small parameters v_{ij} for $j=1,\ldots,n$. Since $\sigma_i = b_i f_i s_i$ where s_i is the proportion of females in the progeny, a higher representation of females is generally favored although a polymorphism can still be preserved by viability or fertility differences among females.

It is important to stress that female fertility and male virility can be introduced into the model (3.2) because they are assumed to be independent. A general fertility scheme based on mating type would require further analysis. For the two-locus model (2.2), the introduction of viability or fertility selection operating at the modifier locus would certainly be interesting but would lead to a general two-sex two-locus model that is beyond the scope of application of the present analysis.

Note that there are qualitative differences in the dynamics uncovered for the case of small recombination (Sect. 2) and the case of absolute linkage (Sect. 3). Our intuition from the latter case and the optimality principle (4.1) for stable equilibrium points does not apply to the former case where limit cycles can occur. Actually the existence of periodic orbits can be inferred by Hopf bifurcations in the vicinity of symmetric equilibria as the recombination fraction diminishes. However, the stability of such an orbit (i.e., the existence of a limit cycle) cannot be inferred from a local linear analysis near the equilibrium where it occurs. For the equilibrium can become unstable in such a way that either an unstable orbit collapses to it or a stable orbit emerges from it. For the model (2.2), the latter hypothesis is more likely since there are no stable equilibrium points (polymorphic or not) with small recombination (Result IV) and stable orbits were actually observed in Maffi's and Jayakar's (1981) simulations.

It is also important to be aware that non-symmetric equilibria can exist and be stable for the model (2.2) as the recombination rate increases and, as a matter of fact, such equilibria have proved to be optimal in the sense of Result IV in the case of free recombination.

Finally, we have assumed that MA_i/mA_j and MA_j/mA_i genotypes segregate M- and m-carrying gametes in the same relative proportions. It would be interesting to relax this assumption of symmetry.

APPENDIX

Proof of Result I. Writing $\mathbf{X} = \mathbf{X}^* + \xi$, $\mathbf{Y} = \mathbf{X}^* + \eta$, and $\mathbf{Z} = \mathbf{X}^* + \chi$ where $\mathbf{X}^*$ is a positive frequency vector satisfying $S^*\mathbf{X}^* = \mathbf{X}^* \circ S\mathbf{X}^*/s^* = \mathbf{X}^*$ with $s^* \leqslant \frac{1}{2}$, transformation (2.2) for the variables $\{\xi, \eta, \chi\}$ whose components are small and add to zero is linearly approximated by the coefficient matrix

$$\begin{bmatrix} I/2 & 0 & I/2 \\ rI + (1-r)\,S^* & (1-r)\,I + rS^* & 0 \\ (1-r)\,I - \dfrac{rs^*}{1-s^*}\,S^* & rI - \dfrac{(1-r)\,s^*}{1-s^*}\,S^* & 0 \end{bmatrix}. \tag{A1}$$

The relevant eigenvalues are those of the 3×3 matrix

$$\begin{bmatrix} 1/2 & 0 & 1/2 \\ r+(1-r)\lambda & (1-r)+r\lambda & 0 \\ (1-r)-\dfrac{rs^*}{1-s^*}\lambda & r-\dfrac{(1-r)s^*}{1-s^*}\lambda & 0 \end{bmatrix} \tag{A2}$$

where λ is any eigenvalue of S^* different from 1, actually less than 1 in absolute value. The characteristic polynomial to consider is in the form

$$g(\mu) = \mu^3 + a\mu^2 + b\mu + c \tag{A3}$$

where

$$a = r(1-\lambda) - 3/2,$$

$$b = \frac{r\lambda}{2(1-s^*)},$$

$$c = \frac{(1-2r)(1-s^*+\lambda^2 s^*)}{2(1-s^*)}.$$

Using the linear fractional transformation (see, e.g., Ahlfors, 1966)

$$\mu = \frac{\zeta+1}{\zeta-1}, \tag{A4}$$

the roots of $g(\mu)$ located in the unit circle correspond to the roots with negative real parts of the polynomial

$$f(\zeta) = \zeta^3 + A\zeta^2 + B\zeta + C \tag{A5}$$

where

$$A = \frac{3+a-b-3c}{1+a+b+c},$$

$$B = \frac{3-a-b+3c}{1+a+b+c},$$

$$C = \frac{1-a+b-c}{1+a+b+c}.$$

The Routh–Hurwitz criterion (see, e.g., Gantmacher, 1959) informs us that all zeros of f have negative real parts if and only if

$$A > 0, \qquad C > 0, \qquad \text{and} \qquad AB - C > 0. \tag{A6}$$

It can be checked that $C>0$ if and only if $g(1)>0$ where

$$g(1)=1+a+b+c$$
$$=\frac{\lambda}{2(1-s^*)}[\lambda s^*-(1-2s^*+2\lambda s^*)r]. \tag{A7}$$

Actually this condition ensures through a detailed analysis that every *real* root of g cannot exceed 1 in absolute value. Moreover, in this case, $A>0$ only if

$$3+a-b-3c=\frac{1}{2(1-s^*)}[r(8-3\lambda-8s^*+4\lambda s^*+6\lambda^2 s^*)-3\lambda^2 s^*]>0. \tag{A8}$$

Finally, a necessary and sufficient condition to ensure $AB-C>0$ is

$$Q(r)=1-b+ac-c^2>0. \tag{A9}$$

Since $Q(0)<0$ and $Q(1/2)>0$, the quadratic form $Q(r)$ has one and only one root from minus to plus between 0 and $\frac{1}{2}$. This completes the proof of Result I.

Observe that if $C>0$ but $A<0$ or $AB-C<0$, then there exists a complex conjugate pair of eigenvalues outside the unit circle. When these eigenvalues cross the unit circle, there is a Hopf bifurcation and therefore existence of a periodic orbit. On the other hand, when the value of C crosses 0, there is a bifurcation to nearby equilibrium points (see, e.g., Marsden and McCracken, 1976).

Proof of Result II. The linear approximation of transformation (2.1) for the $(n+1)$th components $\{X_{n+1},\ Y_{n+1},\ Z_{n+1}\}$ near a symmetric equilibrium $\mathbf{X}^*$ involving only the first n components is given by the non-negative coefficient matrix

$$\begin{bmatrix} 1/2 & 0 & 1/2 \\ \dfrac{rs^*_{n+1}}{s^*} & \dfrac{(1-r)\,s^*_{n+1}}{s^*} & 0 \\ \dfrac{(1-r)(1-s^*_{n+1})}{(1-s^*)} & \dfrac{r(1-s^*_{n+1})}{(1-s^*)} & 0 \end{bmatrix} \tag{A10}$$

where

$$s^*=\sum_{i,j=1}^{n} s_{ij}X_i^*X_j^*,$$

$$s^*_{n+1}=\sum_{i=1}^{n} s_{i,n+1}X_i^*.$$

Assume $s^* \leqslant \frac{1}{2}$. The eigenvalues governing external stability are the roots of

$$h(\gamma) = \gamma^3 - \left[\frac{s^* + 2(1-r)\, s^*_{n+1}}{2s^*}\right]\gamma^2 - \left[\frac{(1-r)(s^* - s^*_{n+1})}{2s^*(1-s^*)}\right]\gamma + \left[\frac{(1-2r)\, s^*_{n+1}(1-s^*_{n+1})}{2s^*(1-s^*)}\right]. \quad \text{(A11)}$$

These are all of magnitude less than 1 if and only if $h(1) > 0$ where

$$h(1) = \frac{1}{2s^*(1-s^*)}\,[r(s^* - s^*_{n+1})(1 - 2s^*_{n+1}) - (s^* - s^*_{n+1})^2], \quad \text{(A12)}$$

owing to the Perron–Frobenius theory which guarantees that the largest eigenvalue in magnitude is actually positive (see, e.g., Gantmacher, 1959). Note that $h(1) > 0$ implies $s^*_{n+1} < s^*$ under the condition $s^* \leqslant \frac{1}{2}$. In such a case,

$$h'(1) = \frac{(s^* - s^*_{n+1})[4(1-s^*) - (1-r)] + 4r(1-s^*)\, s^*_{n+1}}{2s^*(1-s^*)} > 0,$$

and then there cannot exist a positive root of h greater than 1. The proof of Result II is complete.

Proof of Result III. It suffices to set $s^* = \frac{1}{2}$ in Results I and II and recall that every eigenvalue $\lambda \neq 1$ of S^* is actually less than 1 in absolute value. Therefore, the polynomial $Q(r)$ in (2.9) satisfies

$$\begin{aligned} Q(r) &\geqslant 1 - r - \frac{3(1-2r)}{2} - (1-2r)^2 \\ &= -\frac{3}{2} + 6r - 4r^2 \\ &= -4\left[r - \frac{3-\sqrt{3}}{4}\right]\left[r - \frac{3+\sqrt{3}}{4}\right], \end{aligned} \quad \text{(A13)}$$

and $Q(r)$ is positive between $(3-\sqrt{3})/4$ and $1/2$. The rest of the proof is straightforward.

Proof of Result VII. Defining $X = p'_{n+1}/p_{n+1}$, the recurrence equation (3.6) yields

$$X' = a + \frac{b}{X}$$

where

$$a=\frac{\sum_{i=1}^{n} v_{i,n+1}\sigma_i p_i^*}{2w^*},$$

$$b=\frac{\sum_{i=1}^{n} v_{i,n+1}\sigma_{n+1} p_i^*}{2w^*}.$$

From any positive starting position, the iterates of X converge to the positive root of x^2-ax-b, i.e., $(a+\sqrt{a^2+4b})/2$, which is less than 1 if and only if $a+b=w_{n+1}^*/w^*<1$.

Proof of Result VIII. Defining

$$X=q_1, \qquad \text{and} \qquad Y=\frac{p_1\sigma_1}{p_1\sigma_1+p_2\sigma_2}, \tag{A14}$$

the recurrence system (3.1) for the case $n=2$ can be written in the form

$$X'=Y,$$

$$Y'=\frac{[v_{11}\sigma_1-v_{12}(\sigma_1+\sigma_2)/2]\,XY+v_{12}\sigma_1 X/2+v_{12}\sigma_2 Y/2}{\left(\begin{array}{c}[v_{11}\sigma_1-v_{12}(\sigma_1+\sigma_2)+v_{22}\sigma_2]\,XY+[v_{12}\sigma_1-v_{22}\sigma_2]\,X\\ +[v_{12}\sigma_2-v_{22}\sigma_2]\,Y+v_{22}\sigma_2\end{array}\right)}$$

$$=v(X, Y). \tag{A15}$$

At equilibrium, we must have

$$X^*=Y^*=\frac{v_{22}\sigma_2-v_{12}(\sigma_1+\sigma_2)/2}{v_{22}\sigma_2-v_{12}(\sigma_1+\sigma_2)+v_{11}\sigma_1}. \tag{A16}$$

Suppose $0<X^*<1$. Writing $X=X^*+\xi$ and $Y=X^*+\eta$, the linear terms for the transformation relating the quantities $\{\xi, \eta\}$ are described by the coefficient matrix

$$\begin{bmatrix} 0 & 1 \\ v_X(X^*, X^*) & v_Y(X^*, X^*) \end{bmatrix} \tag{A17}$$

where v_X and v_Y denote the partial derivatives of $v(X, Y)$ with respect to X and Y, respectively. It can be checked that $v_X(X^*, X^*)$ and $v_Y(X^*, X^*)$ are both positive. Therefore the eigenvalues of the coefficient matrix at hand are all less than 1 in magnitude if and only if its characteristic polynomial evaluated at 1 is positive, i.e.,

$$1>v_X(X^*, X^*)+v_Y(X^*, X^*)=\frac{d}{dX}v(X, X)|_{X=X^*}. \tag{A18}$$

This will be the case if and only if

$$2X^*[v_{11}\sigma_1 - v_{12}(\sigma_1+\sigma_2)/2] < 0. \tag{A19}$$

With the condition $0 < X^* < 1$, this is equivalent to

$$v_{12}\left(\frac{\sigma_1+\sigma_2}{2}\right) > \max\{v_{11}\sigma_1, v_{22}\sigma_2\}. \tag{A20}$$

Otherwise, we must have

$$v_{12}\left(\frac{\sigma_1+\sigma_2}{2}\right) < \min\{v_{11}\sigma_1, v_{22}\sigma_2\} \tag{A21}$$

and the polymorphic equilibrium X^* is unstable.

Proof of Result IX. With $v_{ij} = 1$ for all $i, j = 1,..., n$ in (3.1), we have

$$\begin{aligned}\sum_{i=1}^{n}(p_i' + q_i'/2)\,\sigma_i &= \sum_{i=1}^{n}\left(\frac{p_i+q_i}{2}\right)\sigma_i + \left[\sum_{i=1}^{n} p_i\sigma_i^2\right] \Big/ \left(2\left[\sum_{i=1}^{n} p_k\sigma_k\right]\right) \\ &\geqslant \sum_{i=1}^{n}\left(\frac{p_i+q_i}{2}\right)\sigma_i + \frac{1}{2}\sum_{i=1}^{n} p_i\sigma_i \\ &= \sum_{i=1}^{n}(p_i + q_i/2)\,\sigma_i \end{aligned} \tag{A22}$$

with equality only at a fixation state (i.e., $p_i = 1$ for some i).

Acknowledgments

This research started from a discussion with Professor Ilan Eshel. I thank Dr. Marcy Uyenoyama and an anonymous referee for judicious comments.

References

Ahlfors, L. V. 1966. "Complex Analysis," 2nd ed., McGraw–Hill, New York.

Bell, A. E. 1954. A gene in *Drosophila melanogaster* that produces all male progeny, *Genetics* **39**, 958–959.

Bengtsson, B. O. 1977. Evolution of the sex ratio in the wood lemming, *Myopus schisticolor*, *in* "Measuring Selection in Natural Populations" (F. B. Christiansen and T. M. Fenchel, Eds.), pp. 333–343, Springer-Verlag, New York.

Cannings, C. 1967. Equilibrium, convergence and stability at a sex-linked locus under natural selection, *Genetics* **56**, 613–618.

Chanter, A. O., and Owen, D. F. 1972. The inheritance and population genetics of sex ratio in the butterfly *Acraea encedon*, *J. Zool.* **166**, 363–383.

CURTSINGER, J. W. AND FELDMAN, M. W. 1980. Experimental and theoretical analysis of the "sex-ratio" polymorphism in *Drosophila pseudoobscura*, *Genetics* **94**, 445–466.

EDWARDS, A. W. F. 1961. The population genetics of "sex-ratio" in *Drosophila pseudoobscura*, *Heredity* **16**, 291–304.

ESHEL, I. 1975. Selection on sex ratio and the evolution of sex determination, *Heredity* **34**, 351–361.

ESHEL, I. 1985. Evolutionary genetic stability of Mendelian segregation and the role of free recombination in the chromosomal system, *Amer. Nat.* **125**, 412–420.

ESHEL, I., AND FELDMAN, M. W. 1982. On evolutionary genetic stability of the sex ratio. *Theor. Pop. Biol.* **21**, 430–439.

FAULHABER, S. H. 1967. An abnormal sex ratio in *Drosophila simulans*, *Genetics* **56**, 189–213.

FREDGA, K., GROPP, A., WINKING, H., AND FRANK, F. 1976. Fertile *XX* and *XY* type females in the wood lemming *Myopus schisticolor*, *Nature* (*London*) **261**, 225–227.

GANTMACHER, F. R. 1959. "The Theory of Matrices," Vol. 2, Chelsea, New York.

GERSHENSON, S. 1928. A new sex-ratio abnormality in *Drosophila obscura*, *Genetics* **13**, 488–507.

HALDANE, J. B. S., AND JAYAKAR, S. D. 1964. Equilibria under natural selection at a sex-linked locus, *J. Genet.* **59**, 29–36.

HAMILTON, W. D. 1967. Extraordinary sex ratios. *Science* **156**, 477–488.

HICKEY, W. A., AND GRAIG, G. B. 1966. Genetic distortion of sex ratio in a mosquito, *Aedes aegypti*, *Genetics* **53**, 1177–1196.

KARLIN, S. 1972. Some mathematical models of population genetics, *Amer. Math. Monthly* **79**, 699–739.

KARLIN, S. 1978. Theoretical aspects of multilocus selection balance, I, *in* "Studies in Mathematical Biology," Part II, "Populations and Communities" (S. A. Levin, Ed.), MAA Studies in Mathematics, Vol. 16, pp. 503–587.

KARLIN, S., AND LESSARD, S. 1983. On the optimal sex ratio, *Proc. Natl. Acad. Sci. USA* **80**, 5931–5935.

KARLIN, S., AND LESSARD, S. 1984. On the optimal sex ratio: A stability analysis based on a characterization for one-locus multiallele viability models, *J. Math. Biol.* **20**, 15–38.

KARLIN, S., AND LESSARD, S. 1986. "Theoretical Studies on Sex Ratio Evolution," Princeton Univ. Press, Princeton, NJ.

LESSARD, S. 1985. The role of recombination in the modifier theory of autosomal segregation distortion, *Theor. Pop. Biol.* **28**, 133–149.

LESSARD, S. 1986. Evolutionary principles for general frequency-dependent two-phenotype models in sexual populations, *J. Theor. Biol.* **119**, 329–344.

LESSARD, S., AND KARLIN, S. 1982. A criterion for stability–instability at fixation states involving an eigenvalue one with applications in population genetics, *Theor. Pop. Biol.* **22**, 108–126.

MAFFI, G., AND JAYAKAR, S. D. 1981. A two-locus model for polymorphism for sex-linked meiotic drive modifiers with possible applications to *Aedes aegypti*, *Theor. Pop. Biol.* **19**, 19–36.

MARSDEN, J. E., AND MCCRACKEN, M. 1976. "The Hopf Bifurcation and Its Applications," Applied Math. Sciences, Vol. 19, Springer-Verlag, New York.

NOVITSKI, E. 1947. Genetic analysis of an anomalous sex ratio condition in *Drosophila affinis*, *Genetics* **32**, 526–534.

PROUT, T., BUNDGAARD, J., AND BRYANT, S. 1973. Population genetics of modifiers of meiotic drive. I. The solution of a special case and some general implications, *Theor. Pop. Biol.* **4**, 446–465.

SANDLER, L. 1970. The regulation of sex-chromosome heterochromatic activity by an autosomal gene in *Drosophila melanogaster*, *Genetics* **64**, 481–493.

SMITH, D. A. S. 1975. All-female broods in the polymorphic butterfly *Danaus chrysippus* and their ecological significance, *Heredity* **34**, 363–371.

STALKER, H. D. 1961. The genetic systems modifying meiotic drive in *Drosophila paramelanica*, *Genetics* **46**, 177–202.

STURTEVANT, A. H., AND DOBZHANSKY, T. 1936. Geographical distribution and cytology of "sex ratio" in *Drosophila pseudoobsura* and related species, *Genetics* **21**, 473–490.

THOMSON, G. J., AND FELDMAN, M. W. 1975. Population genetics of modifiers of meiotic drive. IV. On the evolution of sex-ratio distortion, *Theor. Pop. Biol.* **8**, 202–211.

THOMSON, G. J., AND FELDMAN, M. W. 1976. Population genetics of modifiers of meiotic drive. III. Equilibrium analysis of a general model for the genetic control of segregation distortion, *Theor. Pop. Biol.* **10**, 10–25.

UYENOYAMA, M. K., AND BENGTSSON, B. O. 1979. Towards a genetic theory for the evolution of the sex ratio, *Genetics* **93**, 721–736.

WU, CHUNG-I 1983. The fate of autosomal modifiers of the sex-ratio trait in *Drosophila* and other sex-linked meiotic drive systems, *Theor. Pop. Biol.* **24**, 107–120.

Printed in Belgium