

Towards a Genetic Theory for the  
Evolution of the Sex Ratio  
II. Haplodiploid and Diploid Models with  
Sibling and Parental Control of the  
Brood Sex Ratio and Brood Size\*

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Population genetic models involving sister, brother, and father control of the brood sex ratio and brood size in both the haplodiploid and diploid cases are constructed and analyzed. The results are interpreted in light of the verbal theories which predict the evolution of the sex ratio to a value which is proportional to the ratio of relatedness of the controlling members of the family to males and to females produced in the brood. In our models, the sex ratio in a certain class of polymorphic equilibria evolves to equal investment in males and females in those cases where the controlling members of the family are symmetrically related to males and females as predicted by the verbal theory. However, the sex ratio in the case of sister control in haplodiploids does not evolve to 1:3, but rather to a value proportional to the ratio of the regression coefficients of additive genotypes. Even so, the predicted sex ratio, which is proportional to 1:3, is in fact an "ESS" in the sense that fixation of a genotype specifying that sex ratio is resistant to the initial increase of all other genotypes.

## 1. INTRODUCTION

Hamilton (1972) and Trivers and Hare (1976) have raised the issue of the relationship between the classical problem of the evolution of the sex ratio (Fisher, 1930, 1958) and the theory of kin selection (Hamilton, 1964a, b). In particular, asymmetries in the measure of genetic relatedness (Hamilton, 1964a, b, 1971a, 1971b, 1972; Crozier, 1970; see also Uyenoyama and

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Feldman, 1981) between the sexes may influence the evolution of the sex ratio. Traditionally, the theories for the evolution of the sex ratio have been phrased in terms of parental fitnesses (e.g., Fisher, 1958, pp. 158–160; Shaw and Mohler, 1953; Bodmer and Edwards, 1960; MacArthur, 1965), i.e., the question posed is as follows: What investment ratio in sons and daughters maximizes parental contribution made through offspring to the third generation? Parental contribution of a genotype to the third generation has been represented as

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

(Shaw and Mohler, 1953), where  $M/F$  is the population sex ratio and  $m/f$  is the brood sex ratio of the genotype in question. The contributions through sons and through daughters are equally weighted as indicated by the factor of  $1/2$ . The reason behind this choice of weighting has traditionally been that “each sex must supply half the ancestry of all future generations” (Fisher, 1958, p. 159), but more recently (Hamilton, 1972; Charnov, 1978) has been rephrased in terms of the symmetry of relatedness in the parent to son and parent to daughter relationships. The issue raised by Hamilton (1972) and Trivers and Hare (1976), however, transfers the focus of discussion to the point of view of offspring, rather than parental, fitness and examines the effect of sibling control of brood sex ratio on the evolution of the sex ratio. The case of sister control of brood sex ratio in haplodiploid organisms has recently been discussed extensively (Hamilton, 1972; Trivers and Hare, 1976; Oster *et al.*, 1977; Oster and Wilson, 1978; Benford, 1978; MacNair, 1978; Charnov, 1978) with particular attention to the asymmetry in relatedness of a sister to her male versus female siblings. From the sister’s point of view, the genetic contribution through sisters is more heavily valued than the contribution through brothers, and it has been argued that the relative weight of the contribution through sisters to that through brothers should reflect the ratio of sister-to-sister relatedness to sister-to-brother relatedness.

Table 1 shows the coefficients of relatedness between various pairs of siblings that have been used in the papers cited above on the evolution of the sex ratio under sister control in haplodiploid organisms (Crozier, 1970; Hamilton, 1971b, p. 62). In the diploid case, the relatedness between siblings is equal to  $1/2$ . Under the assumptions that the entries in Table 1 are the relevant measures of relatedness and further that relatedness reflects the probability of transmission of genes in the controlling sibling through its brothers and sisters, Oster *et al.*, (1977) and Benford (1978) have shown, using mathematical models, that under sister control in the haplodiploid case, the sex ratio at equilibrium will be equal to  $1/4:3/4$ , i.e., the ratio of relatedness between the controlling sister and her sisters and brothers. The

TABLE 1  
Measures of Relatedness Between the  
Controlling Siblings and Brothers and Sisters

		Siblings	
		Sister	Brother
Controlling sibling	Sister	$3/4$	$1/4$
	Brother	$1/2$	$1/2$

1:3 sex ratio predicted by Trivers and Hare (1976) was also found to be an "ESS" by Charnov (1978), who did not make any a priori assumptions about the relevance of the coefficients in Table 1 to evolutionary changes. In contrast, Herbers (1979), using a game theory approach, found that no "unbeatable" sex ratio exists under the assumption of a constant ratio of sister-to-sister relatedness to sister-to-brother relatedness.

The purpose of this paper is to cast the problem of the evolution of the sex ratio in purely genetic terms and to discuss the effect of sibling control in terms of changes in genotypic frequencies. Population genetic models of sex ratio evolution (Nur, 1974; Eshel, 1975; Charnov, 1975; Charlesworth, 1977; Uyenoyama and Bengtsson, 1979) have shown that in the case of control of the sex ratio by either parent in diploids and by the diploid sex in haplodiploids, the results of the population genetic models support the verbal theories. In particular, we showed (Uyenoyama and Bengtson, 1979) by assuming a certain relation between brood sex ratio and brood size, that the deviation between the population sex ratio and the sex ratio predicted by Fisher is minimized at equilibrium. In the present paper, we consider the cases of sister control of brood sex ratio and brood size in both the haplodiploid and diploid cases, brother control in the haplodiploid case, and father control in the haplodiploid case. We have constructed our models only in terms of genotype frequencies and the various measures of relatedness arise after the analysis to provide a heuristic framework within which to view the results. Our analysis lends further support to the view that the regression coefficient (explained below), rather than the entries in Table 1, represents the relevant measure of genetic relatedness. Our results indicate that equal investments between the sexes evolves under sister control in the diploid case under brother control in the haplodiploid case. This result would be expected both on the basis of the entries in Table 1 and on the basis of the regression coefficients shown in Table 2. However, in the case of sister control in haplodiploids, the sex ratio does not evolve to 1:3. If no dominance in the action of the genes controlling brood sex ratio and brood size is assumed,

TABLE 2  
Additive Regression Coefficients between  
Controlling Siblings and Their Brother and Sisters

		Siblings	
		Sister	Brother
Controlling sibling	Sister	$\frac{p_f q_f + p_m q_m - (1/4)f_2}{p_f q_f + p_m q_m}$	$\frac{p_f q_f - (1/4)f_2}{p_f q_f + p_m q_m}$
	Brother	$\frac{p_f q_f - (1/4)f_2}{p_f q_f}$	$\frac{p_f q_f - (1/4)f_2}{p_f q_f}$

then the sex ratio evolves to a value proportional to the ratio of the relevant regression coefficients as predicted by the verbal theory. However, the regression coefficients are not constants but rather frequency-dependent, so the equilibrium sex ratio is not a simple value such as 1:3.

Uyenoyama and Feldman (1981) have presented arguments supporting the position that in kin selection models the regression coefficient of the additive genotype of the recipient on that of the altruist, rather than the coefficients shown in Table 2, represents the appropriate measure of relatedness. The regression coefficients for the various sibling pairs are given in Table 2. In the diploid case, the relatedness between siblings is equal to

$$\frac{p_f q_f + p_m q_m - (1/4)(f_2 + m_2)}{p_f q_f + p_m q_m},$$

where  $m_2$  is the frequency of the  $Aa$  heterozygote in males and the remaining variables are as defined in Table 2. These regression coefficients were constructed under the following additive scale for male and female genotypes:

	Genotype				
	$AA$	$Aa$	$aa$	$A$	$a$
Value	2	1	0	1	0

It was found by Uyenoyama and Feldman (1981), that in many cases an adaptive function which incorporates both the relevant regression coefficients and the sex ratio after selection proved to be useful in organizing the results. We shall also discuss similar adaptive functions in the present paper. For example, in the case of sister control of the sex ratio, the following

expression for contribution through siblings is a direct representation of the verbal theory:

$$\frac{mb_{S-B}}{M} + \frac{fb_{S-S}}{F},$$

where  $b_{S-B}$  and  $b_{S-S}$  are the additive regressions of brother on sister and sister on sister, respectively. A similar expression is obtained for each genotype and then the adaptive function is formed by averaging these expressions over the various genotypes. There are two properties which a heuristically valuable adaptive function should have: first, the stationary points of the adaptive function should coincide with the interior equilibria of the dynamic model, and second, the local maxima of the adaptive function should coincide with the stable equilibria of the dynamic model. The proposed adaptive functions have those properties in all cases studied except for the case of sister control in haplodiploids. In that case, the adaptive function has those properties only if the additional assumption of no dominance among the alleles controlling the sex ratio is made.

## 2. MODEL CONSTRUCTION

We assume, as in Uyenoyama and Bengtsson (1979), that both brood sex ratio and brood size are determined by genotypes in the controlling relative. We analyze several cases involving control by sisters, brothers, and father in the haplodiploid and diploid cases in order to gain a general impression of the dynamics of sex ratio evolution. The case of control by the mother has been considered for both the diploid and haplodiploid cases by Uyenoyama and Bengtsson (1979).

### 2.1 *Sister Control*

The first case under consideration features control of the brood sex ratio and brood size by sisters. The brood sex ratios and brood sizes which would result if the respective genotypes had complete control of the brood are given in Table 3. In mixed broods, i.e., broods in which more than one sister genotype is present, we assume that resources are divided among the genotypes according to the frequency of each sister genotype, and then each sister genotype raises its sub-brood independently. The total brood size is the average of the sub-brood sizes, weighted by the frequencies of the sister genotypes. The overall frequency of males is an average of the frequency of males among sub-broods, weighted by the sizes of the sub-broods. For example, in a mating of  $Aa \times A$ , half of the sisters will be  $AA$  and half  $Aa$ . The total brood size is  $(1/2)(\sigma_1 + \sigma_2)$  and the overall frequency of males is

TABLE 3  
Brood Sizes and Frequencies of Males in Broods Raised by Sisters

	Genotype of sisters		
	<i>AA</i>	<i>Aa</i>	<i>aa</i>
Frequency of males	$\alpha$	$\beta$	$\gamma$
Brood size	$\sigma_1$	$\sigma_2$	$\sigma_3$

$(\sigma_1\alpha + \sigma_2\beta)/(\sigma_1 + \sigma_2)$ . Brood sex ratios and brood sizes for each parental mating type is shown in Tables 4 and 5 for the haplodiploid and diploid cases. The systems of recursions which are obtained directly from these mating tables are given below:

**Model IA: Haplodiploid, sister control**

$$\begin{aligned}
 T_m p'_m &= f_1(p_m \sigma_1 \alpha + q_m \sigma_2 \beta) + (1/4)f_2[p_m(\sigma_1 \alpha + \sigma_2 \beta) + q_m(\sigma_2 \beta + \sigma_3 \gamma)], \\
 T_m q'_m &= (1/4)f_2[p_m(\sigma_1 \alpha + \sigma_2 \beta) + q_m(\sigma_2 \beta + \sigma_3 \gamma)] + f_3(p_m \sigma_2 \beta + q_m \sigma_3 \gamma), \\
 T_f f'_1 &= p_m\{f_1 \sigma_1(1 - \alpha) + (1/4)f_2[\sigma_1(1 - \alpha) + \sigma_2(1 - \beta)]\}, \\
 T_f f'_2 &= p_m\{(1/4)f_2[\sigma_1(1 - \alpha) + \sigma_2(1 - \beta)] + f_3 \sigma_2(1 - \beta)\} \\
 &\quad + q_m\{f_1 \sigma_2(1 - \beta) + (1/4)f_2[\sigma_2(1 - \beta) + \sigma_3(1 - \gamma)]\}, \\
 T_f f'_3 &= q_m\{(1/4)f_2[\sigma_2(1 - \beta) + \sigma_3(1 - \gamma)] + f_3 \sigma_3(1 - \gamma)\}.
 \end{aligned}$$

**Model IB: Diploid, sister control**

$$\begin{aligned}
 T_m m'_1 &= f_1[m_1 \sigma_1 \alpha + (1/4) m_2(\sigma_1 \alpha + \sigma_2 \beta)] \\
 &\quad + (1/4)f_2[m_1(\sigma_1 \alpha + \sigma_2 \beta) + (1/4) m_2(\sigma_1 \alpha + 2\sigma_2 \beta + \sigma_3 \gamma)], \\
 T_m m'_2 &= f_1[(1/4) m_2(\sigma_1 \alpha + \sigma_2 \beta) + m_3 \sigma_2 \beta] \\
 &\quad + (1/4)f_2[m_1(\sigma_1 \alpha + \sigma_2 \beta) + (1/2) m_2(\sigma_1 \alpha + 2\sigma_2 \beta + \sigma_3 \gamma) \\
 &\quad + m_3(\sigma_2 \beta + \sigma_3 \gamma)] + f_3[m_1 \sigma_2 \beta + (1/4) m_2(\sigma_2 \beta + \sigma_3 \gamma)], \\
 T_m m'_3 &= (1/4)f_2[(1/4) m_2(\sigma_1 \alpha + 2\sigma_2 \beta + \sigma_3 \gamma) + m_3(\sigma_2 \beta + \sigma_3 \gamma)] \\
 &\quad + f_3[(1/4) m_2(\sigma_2 \beta + \sigma_3 \gamma) + m_3 \sigma_3 \gamma], \\
 T_f f'_1 &= f_1\{m_1 \sigma_1(1 - \alpha) + (1/4) m_2[\sigma_1(1 - \alpha) + \sigma_2(1 - \beta)]\} \\
 &\quad + (1/4)f_2\{m_1[\sigma_1(1 - \alpha) + \sigma_2(1 - \beta)] \\
 &\quad + (1/4) m_2[\sigma_1(1\alpha) + 2\sigma_2(1 - \beta) + \sigma_3(1 - \gamma)]\},
 \end{aligned}$$

$$\begin{aligned}
T_f f'_2 &= f_1 \{ (1/4) m_2 [\sigma_1(1-\alpha) + \sigma_2(1-\beta)] + m_3 \sigma_2(1-\beta) \} \\
&\quad + (1/4) f_2 \{ m_1 [\sigma_1(1-\alpha) + \sigma_2(1-\beta)] \\
&\quad + (1/2) m_2 [\sigma_1(1-\alpha) + 2\sigma_2(1-\beta) + \sigma_3(1-\gamma)] \\
&\quad + m_3 [\sigma_2(1-\beta) + \sigma_3(1-\gamma)] \} \\
&\quad + f_3 \{ m_1 \sigma_2(1-\beta) + (1/4) m_2 [\sigma_2(1-\beta) + \sigma_3(1-\gamma)] \}, \\
T_f f'_3 &= (1/4) f_3 \{ (1/4) m_2 [\sigma_1(1-\alpha) + 2\sigma_2(1-\beta) + \sigma_3(1-\gamma)] \\
&\quad + m_3 [\sigma_2(1-\beta) + \sigma_3(1-\gamma)] \} \\
&\quad + f_3 \{ (1/4) m_2 [\sigma_2(1-\beta) + \sigma_3(1-\gamma)] + m_3 \sigma_3(1-\gamma) \}.
\end{aligned}$$

In all recursions in this paper,  $f_1$ ,  $f_2$ , and  $f_3$  are the frequencies of female genotypes  $AA$ ,  $Aa$ , and  $aa$ , respectively,  $m_1$ ,  $m_2$ , and  $m_3$  are the frequencies of those genotypes in males in the diploid case,  $p_m$  is the frequency of the  $A$  genotype in haplodiploids and is also equal to  $[m_1 + (1/2)m_2]$  in the diploid case,  $q_m = 1 - p_m$ ,  $p_f = [f_1 + (1/2)f_2]$ ,  $q_f = 1 - p_f$ , and  $T_m$  and  $T_f$  are normalizers that ensure  $f'_1 + f'_2 + f'_3 = 1$ ,  $p'_m + q'_m = 1$ , and  $m'_1 + m'_2 + m'_3 = 1$ .

## 2.2 Brother Control

Control of the brood sex ratio and brood size by brother in the diploid case is identical to control by sisters. We will only consider the haplodiploid case of brother control, for which the parameters of the model are given in Table 6. Table 7 shows the mating table for this case. The system of recursions for the case are derived directly from Table 7:

Model II: Haplodiploid, brother control.

$$\begin{aligned}
T_m p'_m &= f_1 \tau_1 \delta + (1/4) f_2 (\tau_1 \delta + \tau_2 \varepsilon), \\
T_m q'_m &= (1/4) f_2 (\tau_1 \delta + \tau_2 \varepsilon) + f_3 \tau_2 \varepsilon, \\
T_f f'_1 &= p_m \{ f_1 \tau_1 (1-\delta) + (1/4) f_2 [\tau_1 (1-\delta) + \tau_2 (1-\varepsilon)] \}, \\
T_f f'_2 &= p_m \{ (1/4) f_2 [\tau_1 (1-\delta) + \tau_2 (1-\varepsilon)] + f_3 \tau_2 (1-\varepsilon) \} \\
&\quad + q_m \{ f_1 \tau_1 (1-\delta) + (1/4) f_2 [\tau_1 (1-\delta) + \tau_2 (1-\varepsilon)] \}, \\
T_f f'_3 &= q_m \{ (1/4) f_2 [\tau_1 (1-\delta) + \tau_2 (1-\varepsilon)] + f_3 \tau_2 (1-\varepsilon) \}.
\end{aligned}$$

## 2.3 Father Control

Control by fathers in the diploid case produces recursions which are identical to those in the case of control by mothers. Because the latter case was studied by Uyenoyama and Bengtsson (1979), we restrict attention to the haplodiploid case of father control of the sex ratio. The parameters of the

TABLE 4  
 Brood Sex Ratios and Brood Sizes Produced by Each  
 Parental Mating Type in the Haplodiploid Case of Sister Control

Parents	Female offspring			Male offspring			Freq. of males	Brood size
	<i>AA</i>	<i>Aa</i>	<i>aa</i>	<i>A</i>	<i>a</i>			
<i>AA</i>	<i>A</i>	1	0	0	1	0	$\alpha$	$\sigma_1$
	<i>a</i>	0	1	0	1	0	$\beta$	$\sigma_2$
<i>Aa</i>	<i>A</i>	1/2	1/2	0	1/2	1/2	$\frac{\sigma_1\alpha + \sigma_2\beta}{\sigma_1 + \sigma_2}$	$(1/2)(\sigma_1 + \sigma_2)$
	<i>a</i>	0	1/2	1/2	1/2	1/2	$\frac{\sigma_2\beta + \sigma_3\gamma}{\sigma_2 + \sigma_3}$	$(1/2)(\sigma_2 + \sigma_3)$
<i>aa</i>	<i>A</i>	0	1	0	0	1	$\beta$	$\sigma_2$
	<i>a</i>	0	0	1	0	1	$\gamma$	$\sigma_3$

TABLE 5  
 Brood Sex Ratios and Brood Sizes Produced by Each  
 Parental Mating Type in the Diploid Case of Sister Control

Parents	Offspring			Freq. of males	Brood size	
	<i>AA</i>	<i>Aa</i>	<i>aa</i>			
<i>AA</i>	<i>AA</i>	1	0	0	$\alpha$	$\sigma_1$
	<i>Aa</i>	1/2	1/2	0	$\frac{\sigma_1\alpha + \sigma_2\beta}{\sigma_1 + \sigma_2}$	$(1/2)(\sigma_1 + \sigma_2)$
	<i>aa</i>	0	1	0	$\beta$	$\sigma_2$
<i>Aa</i>	<i>AA</i>	1/4	1/2	1/4	$\frac{(\sigma_1\alpha + 2\sigma_2\beta + \sigma_3\gamma)}{(\sigma_1 + 2\sigma_2 + \sigma_3)}$	$(\sigma_1 + 2\sigma_2 + \sigma_3)/4$
	<i>aa</i>	0	1/2	1/2	$\frac{\sigma_2\beta + \sigma_3\gamma}{\sigma_2 + \sigma_3}$	$(1/2)(\sigma_2 + \sigma_3)$
<i>aa</i>	<i>aa</i>	0	0	1	$\gamma$	$\sigma_3$

TABLE 6  
Brood Sizes and Frequencies of Males in Broods Raised by Brothers

	Genotype of brothers	
	<i>A</i>	<i>a</i>
Frequency of males	$\delta$	$\varepsilon$
Brood size	$\tau_1$	$\tau_2$

TABLE 7  
Brood Sex Ratios and Brood Sizes Produced by Each  
Parental Mating Type in the Haplodiploid Case of Brother Control

Parents	Female offspring			Male offspring			Freq. of males	Brood size
	<i>AA</i>	<i>Aa</i>	<i>aa</i>	<i>A</i>	<i>a</i>			
<i>AA</i>	<i>A</i>	1	0	0	1	0	$\delta$	$\tau_1$
	<i>a</i>	0	1	0	1	0	$\delta$	$\tau_1$
<i>Aa</i>	<i>A</i>	1/2	1/2	0	1/2	1/2	$\frac{\tau_1 \delta + \tau_2 \varepsilon}{\tau_1 + \tau_2}$	$(1/2)(\tau_1 + \tau_2)$
	<i>a</i>	0	1/2	1/2	1/2	1/2	$\frac{\tau_1 \varepsilon + \tau_2 \delta}{\tau_1 + \tau_2}$	$(1/2)(\tau_1 + \tau_2)$
<i>aa</i>	<i>A</i>	0	1	0	0	1	$\varepsilon$	$\tau_2$
	<i>a</i>	0	0	1	0	1	$\varepsilon$	$\tau_2$

model and the mating table are given in Tables 8 and 9, respectively. The recursion equations below are derived directly from Table 9:

Model III: Haplodiploid, father control.

$$T_m p'_m = p_f,$$

$$T_m q'_m = q_f,$$

$$T_f p'_f = p_f p_m \tau_1 (1 - \delta) + (1/2)[p_m q_f \tau_1 (1 - \delta) + p_f q_m \tau_2 (1 - \varepsilon)],$$

$$T_f q'_f = (1/2)[p_m q_f \tau_1 (1 - \delta) + p_f q_m \tau_2 (1 - \varepsilon)] + q_m q_f \tau_2 (1 - \varepsilon).$$

TABLE 8  
Brood Sizes and Frequencies of Males in Broods Raised by Fathers

	Genotype of father	
	<i>A</i>	<i>a</i>
Frequency of males	$\delta$	$\varepsilon$
Brood size	$\tau_1$	$\tau_2$

TABLE 9  
Brood Sex Ratios and Brood Sizes Produced by Each  
Parental Mating Type in the Haplodiploid Case of Father Control

Parents		Female offspring			Male offspring		Freq. of males	Brood size
		<i>AA</i>	<i>Aa</i>	<i>aa</i>	<i>A</i>	<i>a</i>		
<i>AA</i>	<i>A</i>	1	0	0	1	0	$\delta$	$\tau_1$
	<i>a</i>	0	1	0	1	0	$\varepsilon$	$\tau_2$
<i>Aa</i>	<i>A</i>	1/2	1/2	0	1/2	1/2	$\delta$	$\tau_1$
	<i>a</i>	0	1/2	1/2	1/2	1/2	$\varepsilon$	$\tau_2$
<i>aa</i>	<i>A</i>	0	1	0	0	1	$\delta$	$\tau_1$
	<i>a</i>	0	0	1	0	1	$\varepsilon$	$\tau_2$

### 3. ANALYSIS OF THE MODELS

#### 3.1 Sister Control of Brood Sex Ratio and Brood Size

##### 3.1.1. Haplodiploid case

##### 3.1.1a. Results of the model.

(1) Stability of boundaries. Fixation of the *A* allele is stable to the introduction of *a* if the following condition holds:

$$\frac{3}{4} \frac{[\sigma_1(1-\alpha) - \sigma_2(1-\beta)]}{\sigma_1(1-\alpha)} + \frac{1}{4} \frac{(\sigma_1\alpha - \sigma_2\beta) \sigma_2(1-\beta)}{\sigma_1\alpha\sigma_1(1-\alpha)} > 0. \quad (1)$$

By symmetry, the fixation of the *a* allele is stable to the introduction of *A* if the following holds:

$$\frac{3}{4} \frac{[\sigma_3(1-\gamma) - \sigma_2(1-\beta)]}{\sigma_3(1-\gamma)} + \frac{1}{4} \frac{(\sigma_3\gamma - \sigma_2\beta) \sigma_2(1-\beta)}{\sigma_3\gamma\sigma_3(1-\gamma)} > 0. \quad (2)$$

(2) Interior equilibria. Reduction of the variables at equilibrium to expressions in terms of  $p_m$  alone produces an equilibrium polynomial of the

eighth degree in  $p_m$ . If the following additional assumption is made, then a symmetric root (Eshel, 1975) at which  $\hat{p}_m = \hat{p}_f$  exists.

$$(\sigma_1\alpha - \sigma_2\beta)[\sigma_3(1 - \gamma) - \sigma_2(1 - \beta)] = (\sigma_3\gamma - \sigma_2\beta)[\sigma_1(1 - \alpha) - \sigma_2(1 - \beta)]. \quad (3)$$

The gene frequency  $\hat{p}_m = \hat{p}_f = p^*$  at the symmetric root is given by

$$p^* = \frac{\sigma_3\gamma - \sigma_2\beta}{\sigma_1\alpha + \sigma_3\gamma - 2\sigma_2\beta} = \frac{\sigma_3(1 - \gamma) - \sigma_2(1 - \beta)}{\sigma_1(1 - \alpha) + \sigma_3(1 - \gamma) - 2\sigma_2(1 - \beta)}, \quad (4)$$

which is valid only if either

$$(a) \quad \sigma_2\beta > \sigma_1\alpha, \sigma_3\gamma$$

or

$$(b) \quad \sigma_2\beta < \sigma_1\alpha, \sigma_3\gamma. \quad (5)$$

The female genotypic frequencies at the  $p^*$  equilibrium are given by

$$\begin{aligned} Nf_1^* &= (1/2)(p^*)^2 [\sigma_1(1 - \alpha) + \sigma_2(1 - \beta)], \\ Nf_2^* &= 2p^*q^*\sigma_2(1 - \beta), \\ Nf_3^* &= (1/2)(q^*)^2 [\sigma_2(1 - \beta) + \sigma_3(1 - \gamma)], \end{aligned} \quad (6)$$

where  $N$  ensures that  $f_1^* + f_2^* + f_3^* = 1$ . If  $p^*$  exists and is valid, i.e., if both (3) and (5) hold, then the equilibrium represented by  $\hat{p}_m = \hat{p}_f = p^*$  and (6) is locally stable if (7) holds and is unstable if (7) fails.

$$\begin{aligned} 0 &> \frac{[p^*q^* - (1/8)f_2^*][\sigma_1(1 - \alpha) + \sigma_3(1 - \gamma) - 2\sigma_2(1 - \beta)]}{p^*\sigma_1(1 - \alpha) + q^*\sigma_2(1 - \beta)} \\ &+ (1/2) \frac{[p^*q^* - (1/4)f_2^*](\sigma_1\alpha + \sigma_3\gamma - 2\sigma_2\beta)}{p^*\sigma_1\alpha + q^*\sigma_2\beta} \\ &\times \left[ 1 - \frac{p^*q^*[\sigma_1(1 - \alpha) + \sigma_3(1 - \gamma) - 2\sigma_2(1 - \beta)]}{p^*\sigma_1(1 - \alpha) + q^*\sigma_2(1 - \beta)} \right]. \end{aligned} \quad (7)$$

Under the symmetric root condition (3),  $p^*$  can be factored out of the equilibrium polynomial to produce a sixth degree polynomial in  $p_m$ . We do not know how many of the roots of the sixth degree polynomial can exist simultaneously.

3.1.1b. *Predictions from the adaptive function.* We suggest the following adaptive function:

$$\bar{W} = p^2 \left[ \frac{\sigma_1(1-\alpha) b_{S-S}}{F} + \frac{\sigma_1 \alpha b_{S-B}}{M} \right] + 2pq \left[ \frac{\sigma_2(1-\beta) b_{S-S}}{F} + \frac{\sigma_2 \beta b_{S-B}}{M} \right] + q^2 \left[ \frac{\sigma_3(1-\gamma) b_{S-S}}{F} + \frac{\sigma_3 \gamma b_{S-B}}{M} \right], \quad (8)$$

where  $b_{S-S}$  and  $b_{S-B}$  are the regression coefficients of the additive genotypes of sister on sister and brother on sister, respectively, and  $M/F = T_m/T_f$  is the sex ratio at equilibrium.

(1) Boundary stability. The adaptive function would predict stability of the fixation of the  $A$  allele if the following condition holds:

$$\frac{b_{S-S}[\sigma_1(1-\alpha) - \sigma_2(1-\beta)]}{\sigma_1(1-\alpha)} + \frac{b_{S-B}(\sigma_1\alpha - \sigma_2\beta)}{\sigma_1\alpha} > 0. \quad (9)$$

The values of the regression coefficients  $b_{S-S}$  and  $b_{S-B}$  must be found as limits near the fixation state. These limiting values are

$$b_{S-S} = \frac{(1/2) \lambda \sigma_1 \alpha}{\lambda \sigma_1 \alpha + (\sigma_1 \alpha + \sigma_2 \beta)(1/2)}, \quad (10)$$

$$b_{S-B} = \frac{(1/2) [\lambda \sigma_1 \alpha + (\sigma_1 \alpha + \sigma_2 \beta)]}{\lambda \sigma_1 \alpha + (1/2)(\sigma_1 \alpha + \sigma_2 \beta)},$$

where  $\lambda$  is equal to the leading eigenvalue of the stability matrix near the fixation, or the larger root of the following quadratic:

$$\lambda^2 - \frac{\lambda(1/4)[\sigma_1(1-\alpha) + \sigma_2(1-\beta)]}{\sigma_1(1-\alpha)} - \frac{(1/4)\sigma_2(1-\beta)(\sigma_1\alpha + \sigma_2\beta)}{\sigma_1(1-\alpha)\sigma_1\alpha} = 0. \quad (11)$$

It can be shown that (9) holds if (1) holds and (9) fails if (1) fails. The stability condition predicted by the adaptive function, then, agrees with the stability condition derived directly from the dynamic model. Note that if 3/4 and 1/4 were substituted in (9) for  $b_{S-S}$  and  $b_{S-B}$ , respectively, then (9) would not in general agree with (1).

(2) Interior equilibria. For the adaptive function in (8) to be heuristically valuable, we also require that the interior equilibria correspond with the stationary points of (8) and that the stable interior equilibria be found at the local maxima of (8). Partial differentiation of (8) with respect to  $p$  produces:

$$\frac{\partial \bar{W}}{\partial p} = 2 \left\{ \frac{\{p[\sigma_1(1-\alpha) - \sigma_2(1-\beta)] - q[\sigma_3(1-\gamma) - \sigma_2(1-\beta)]\} b_{S \rightarrow S}}{F} + \frac{\{p(\sigma_1\alpha - \sigma_2\beta) - q(\sigma_3\gamma - \sigma_2\beta)\} b_{S \rightarrow B}}{M} \right\} = 0. \quad (12)$$

If the symmetric root condition (3) holds, then (12) predicts equilibria at

$$(a) \quad p = p^*$$

or (13)

(b)

$$\frac{\{\sigma_1(1-\alpha) + \sigma_3(1-\gamma) - 2\sigma_2(1-\beta)\} b_{S \rightarrow S}}{F} + \frac{(\sigma_1\alpha + \sigma_3\gamma - 2\sigma_2\beta) b_{S \rightarrow B}}{M} = 0.$$

The first equilibrium in (13) is an equilibrium point of the model. The adaptive function would predict the local stability of  $p^*$  if the second partial derivative with respect to  $p$  at  $p^*$  is negative, i.e., if

$$0 > \frac{\{\sigma_1(1-\alpha) + \sigma_3(1-\gamma) - 2\sigma_2(1-\beta)\} b_{S \rightarrow S}^*}{F^*} + \frac{(\sigma_1\alpha + \sigma_3\gamma - 2\sigma_2\beta) b_{S \rightarrow B}^*}{M^*}, \quad (14)$$

where  $b_{S \rightarrow S}^*$  and  $b_{S \rightarrow B}^*$  are the regressions at the  $p^*$  equilibrium,

$$b_{S \rightarrow S}^* = \frac{[p^*q^* - (1/8)f_2^*]}{p^*q^*},$$

$$b_{S \rightarrow B}^* = \frac{[p^*q^* - (1/4)f_2^*]}{p^*q^*},$$

and the sex ratio at the  $p^*$  equilibrium is

$$\frac{M^*}{F^*} = \frac{T_m^*}{T_f^*} = \frac{p^*\sigma_1\alpha + q^*\sigma_2\beta}{p^*\sigma_1(1-\alpha) + q^*\sigma_2(1-\beta)}.$$

Comparison of (7) and (14) indicates that the stability condition predicted by the adaptive function coincides with the stability condition derived from the model only if no dominance is assumed, i.e.,

$$\sigma_1(1-\alpha) + \sigma_3(1-\gamma) - 2\sigma_2(1-\beta) = \sigma_1\alpha + \sigma_3\gamma - 2\sigma_2\beta = 0. \quad (15)$$

Of course, if (15) is satisfied,  $p^*$  is not valid.

The second equilibrium class in (13) predicted by the adaptive function

specifies a particular sex ratio. The sex ratio corresponding to the roots of the sixth degree equilibrium polynomial derived from the model under the symmetric roots condition (3) agrees with the sex ratio predicted in (13) only if (15), the no dominance condition, holds.

### 3.1.2. Diploid case

#### 3.1.2a. Results of the model.

(1) Stability of boundaries. Fixation of the  $A$  allele is resistant to the initial increase of the  $a$  allele if

$$\frac{[\sigma_1(1-\alpha) - \sigma_2(1-\beta)]}{\sigma_1(1-\alpha)} + \frac{(\sigma_1\alpha - \sigma_2\beta)}{\sigma_1\alpha} > 0. \quad (16)$$

By symmetry, the  $A$  allele fails to increase when rare if

$$\frac{[\sigma_3(1-\gamma) - \sigma_2(1-\beta)]}{\sigma_3(1-\gamma)} + \frac{(\sigma_3\gamma - \sigma_2\beta)}{\sigma_3\gamma} > 0. \quad (17)$$

(2) Interior equilibria. Reduction of the equilibrium conditions to a polynomial in  $p_m$  alone has proven to be extremely difficult in this case. If (3), the symmetric root condition, holds, then  $\hat{p}_m = \hat{p}_f = p^*$ , where  $p^*$  is given by (4) is an equilibrium of this model as well. Under the assumption in (3), the remaining interior equilibria are determined as the roots of a sixth degree polynomial in  $p_m$ . Again, the maximum number of valid roots of this polynomial that can exist simultaneously is unknown.

Corresponding to  $\hat{p}_m = \hat{p}_f = p^*$ , there exists a single valid equilibrium point when (5) holds with

$$Nm_1^* = (1/2)p^*[p^*(\sigma_1\alpha + \sigma_2\beta) - (1/4)f_2^*(\sigma_1\alpha - \sigma_3\gamma)].$$

$$Nm_2^* = 2p^*q^*[\sigma_2\beta + (1/4)f_2^*(\sigma_1\alpha + \sigma_3\gamma - 2\sigma_2\beta)],$$

$$Nm_3^* = (1/2)q^*[q^*(\sigma_2\beta + \sigma_3\gamma) + (1/4)f_2^*(\sigma_1\alpha - \sigma_3\gamma)],$$

where  $N$  ensures that  $m_1^* + m_2^* + m_3^* = 1$  and  $f_2^*$  is given as the single root lying in the ranges  $(0, 2p^*)$  and  $(0, 2q^*)$  of the following quadratic:

$$\begin{aligned} & (1/8)(\sigma_1\alpha + \sigma_3\gamma - 2\sigma_2\beta)^2 T_f^*(f_2^*)^2 + f_2(\sigma_1\alpha + \sigma_3\gamma - 2\sigma_2\beta) \\ & \times (1/2)\{T_f^*\sigma_2\beta + \sigma_2\beta\sigma_2(1-\beta) + (1/2)p^*q^*\sigma_2(1-\beta)(\sigma_1\alpha + \sigma_3\gamma - 2\sigma_2\beta) \\ & + (1/2)p^*q^*\sigma_2\beta[\sigma_1(1-\alpha) + \sigma_3(1-\gamma) - 2\sigma_2(1-\beta)]\} \\ & - 2p^*q^*(\sigma_1\alpha + \sigma_3\gamma - 2\sigma_2\beta) \\ & \times \{\sigma_2\beta\sigma_2(1-\beta) + (1/2)p^*q^*\sigma_2(1-\beta)(\sigma_1\alpha + \sigma_3\gamma - 2\sigma_2\beta) \\ & + (1/2)p^*q^*\sigma_2\beta[\sigma_1(1-\alpha) + \sigma_3(1-\gamma) - 2\sigma_2(1-\beta)]\} = 0. \end{aligned}$$

The values of  $f_1$  and  $f_3$  at this equilibrium are given by

$$Lf_1^* = (1/2)p^*[\sigma_1(1-\alpha) + \sigma_2(1-\beta)] - (1/4)f_2^*[\sigma_1(1-\alpha) - \sigma_3(1-\gamma)],$$

$$Lf_3^* = (1/2)q^*\{q^*[\sigma_2(1-\beta) + \sigma_3(1-\gamma)] + (1/4)f_2^*[\sigma_1(1-\alpha) - \sigma_3(1-\gamma)]\},$$

where  $L$  is equal to

$$\sigma_2(1-\beta) + (1/2)p^*q^*[\sigma_1(1-\alpha) + \sigma_3(1-\gamma) - 2\sigma_2(1-\beta)]$$

$$+ (1/8)f_2^*[\sigma_1(1-\alpha) + \sigma_3(1-\gamma) - 2\sigma_2(1-\beta)].$$

This equilibrium point corresponding to  $p^*$  is locally stable if the following condition holds:

$$0 > \frac{(\sigma_1\alpha + \sigma_3\gamma - 2\sigma_2\beta)}{T_m^*} + \frac{[\sigma_1(1-\alpha) + \sigma_3(1-\gamma) - 2\sigma_2(1-\beta)]}{T_f^*}. \quad (18)$$

3.1.2b. *Predictions from the adaptive function.* We propose the adaptive function in (8). In the diploid case, the regressions  $b_{S-S}$  and  $b_{S-B}$  are always identical.

(1) *Boundary stability.* The adaptive function would predict stability of the fixation of the  $A$  allele if (9) holds. Because  $b_{S-S}$  and  $b_{S-B}$  are positive, (9) reduces to (16). The adaptive function, then, correctly predicts the stability condition derived directly from the model.

(2) *Interior equilibria.* Partial differentiation of  $\bar{W}$  produces the prediction that the two classes of equilibria in (13) exist under the symmetric root condition (3).

As in the haplodiploid case,  $\hat{p} = p^*$  does indeed correspond to the symmetric equilibrium of the model. The adaptive function predicts stability of the  $p^*$  equilibrium if (14) holds. Again, cancellation in (14) of  $b_{S-S}^*$  and  $b_{S-B}^*$ , which are both equal to a positive quantity, produces (18). Unlike the haplodiploid case, the adaptive function in this model precisely predicts the correct stability condition without additional assumptions such as no dominance.

Finally, the sex ratio at the equilibrium determined as the valid roots of the sixth degree equilibrium polynomial obtained from the model when (3) holds, is exactly equal to the sex ratio predicted by the adaptive function in (13b). Again, no additional assumptions such as no dominance are required for agreement between the sex ratio predicted by the adaptive function and the actual sex ratio calculated at equilibrium from the model.

### 3.2 Brother Control of Brood Sex Ratio and Brood Size

#### 3.2a Results of the model.

(1) Stability of boundaries. Fixation of the  $A$  allele is stable to the introduction of the  $a$  allele if

$$\frac{\tau_1(1-\delta) - \tau_2(1-\varepsilon)}{\tau_1(1-\delta)} + \frac{\tau_1\delta - \tau_2\varepsilon}{\tau_1\delta} > 0. \quad (19)$$

By symmetry, the fixation of the  $a$  allele is stable if

$$\frac{\tau_2(1-\varepsilon) - \tau_1(1-\delta)}{\tau_2(1-\varepsilon)} + \frac{\tau_2\varepsilon - \tau_1\delta}{\tau_2\varepsilon} > 0. \quad (20)$$

It is impossible for (19) and (20) to both hold simultaneously.

(2) Interior equilibria. The interior equilibrium corresponds to the single valid root in (0.1) of the following quadratic:

$$\begin{aligned} & -p_m q_m (\tau_1 \delta - \tau_2 \varepsilon)^2 \{ \tau_1 (1 - \delta) - \tau_2 (1 - \varepsilon) \} \\ & + (1/2) p_m (\tau_1 \delta + \tau_2 \varepsilon) \{ \tau_1 (1 - \delta) (\tau_1 \delta - \tau_2 \varepsilon) + \tau_1 \delta [ \tau_1 (1 - \delta) - \tau_2 (1 - \varepsilon) ] \} \\ & + (1/2) q_m (\tau_1 \delta + \tau_2 \varepsilon) \{ \tau_2 (1 - \varepsilon) (\tau_1 \delta - \tau_2 \varepsilon) + \tau_2 \varepsilon [ \tau_1 (1 - \delta) - \tau_2 (1 - \varepsilon) ] \} = 0. \end{aligned} \quad (21)$$

The root comes into existence only if both (19) and (20) fail. Genotypic frequencies in females are determined by  $\hat{p}$ , the valid root of (21), as follows:

$$\begin{aligned} N\hat{f}_1 &= \hat{p}_m^2 [ (\tau_1 \delta p_m + \tau_2 \varepsilon q_m) - (1/2) (\tau_1 \delta - \tau_2 \varepsilon) ], \\ N\hat{f}_2 &= 2\hat{p}_m \hat{q}_m (p_m \tau_1 \delta + q_m \tau_2 \varepsilon), \\ N\hat{f}_3 &= \hat{q}_m^2 [ (\tau_1 \delta p_m + \tau_2 \varepsilon q_m) + (1/2) (\tau_1 \delta - \tau_2 \varepsilon) ], \end{aligned} \quad (22)$$

where  $N$  ensures that  $\hat{f}_1 + \hat{f}_2 + \hat{f}_3 = 1$ . Because of the close relationship between the condition for existence of the  $\hat{p}$  equilibrium and the stability conditions (19) and (20) of the boundaries, it is likely that the interior equilibrium  $\hat{p}$  is stable if both boundaries are unstable, but the local stability of  $\hat{p}$  has not been investigated.

3.2b Predictions from the adaptive function. We propose the following adaptive function which is constructed in a manner entirely analogous to (8).

$$\bar{W} = p \left[ \frac{\tau_1(1-\delta) b_{B-S}}{F} + \frac{\tau_1 \delta b_{B-B}}{M} \right] + q \left[ \frac{\tau_2(1-\varepsilon) b_{B-S}}{F} + \frac{\tau_2 \varepsilon b_{B-B}}{M} \right], \quad (23)$$

where  $b_{B-S}$  and  $b_{B-B}$  are the regressions of brother on sister and brother on brother, respectively. In this case,  $b_{B-S} = b_{B-B}$  and both are positive.

(1) Boundary stability. The adaptive function predicts stability of the fixation of the  $A$  allele if (19) holds and stability of the fixation of the  $a$  allele if (20) holds.

(2) Interior equilibria. Partial differentiation of (23) with respect to  $p$  predicts a single equilibrium class in which the sex ratio is equal to

$$\frac{M}{F} = \frac{-(\tau_1 \delta - \tau_2 \varepsilon)}{[\tau_1(1 - \delta) - \tau_2(1 - \varepsilon)]}. \quad (24)$$

It can be shown that at the equilibrium defined by (21) and (22), the sex ratio is equal to (24).

### 3.3 *Father Control of Brood Sex Ratio and Brood Size*

#### 3.3a. *Results of the model.*

(1) Stability of boundaries. Allele  $a$  cannot increase when rare if

$$\tau_1(1 - \delta) - \tau_2(1 - \varepsilon) > 0 \quad (25)$$

At the other boundary, allele  $A$  cannot increase when rare if the inequality in (25) is reversed.

(2) Interior equilibria. No interior equilibria exist in this case.

3.3b *Predictions from the adaptive function.* We propose the following adaptive function:

$$\bar{W} = p \left[ \frac{\tau_1(1 - \delta) b_{F \rightarrow D}}{F} + \frac{\tau_1 \delta b_{F \rightarrow S}}{M} \right] + q \left[ \frac{\tau_2(1 - \varepsilon) b_{F \rightarrow D}}{F} + \frac{\tau_2 \varepsilon b_{F \rightarrow S}}{M} \right], \quad (26)$$

where  $b_{F \rightarrow D}$  and  $b_{F \rightarrow S}$  are the additive regressions of father on daughter and father on son, respectively. In the haplodiploid case,  $b_{F \rightarrow S} = 0$  and  $b_{F \rightarrow D} = 1$ , so the adaptive function reduces to

$$\bar{W} = \frac{[p\tau_1(1 - \delta) + q\tau_2(1 - \varepsilon)]}{F}. \quad (26')$$

(1) Boundary stability. The adaptive function in (26') predicts stability of the fixation of  $A$  when (25) holds and stability of the fixation of  $a$  when (25) fails.

(2) Interior equilibria. In agreement with the model, the adaptive function predicts no interior equilibria.

#### 4. DISCUSSION

We have analyzed four genetic models of sex ratio evolution in which various members of the immediate family exercise control over the brood sex ratio and brood size. In our models, the dependence of brood sex ratio and brood size on the genotypic composition of certain members of the family represents control by those members. We regard the sex ratio to which the population evolves in the various cases as the "preferred" sex ratio from the point of view of the controlling family members. Our models, then, provide a test of the logical consistency of the verbal models of sex ratio evolution. The central question under discussion concerns the effect of asymmetries in genetic relatedness between the sexes on evolution of the sex ratio in haplodiploid and diploid organisms. In particular, we have tested the prediction of Trivers and Hare (1976) that a 1:3 sex ratio evolves under sister control in the haplodiploid case. We review our results in the following four sections: (1) construction of the models, (2) boundary behavior, (3) structure of the interior equilibria, and (4) on the validity of the verbal predictions from kin selection theory.

##### 4.1 *Construction of the Models*

As in our previous paper (Uyenoyama and Bengtsson, 1979), we assume that brood size is not necessarily independent on the brood sex ratio. In particular, if the sexes require different amounts of expenditure (Fisher, 1958, pp. 158–160; Bodmer and Edwards, 1960), then brood size will be determined by brood sex ratio and the expenditure needed to procedure a female relative to that needed to produce a male. To each genotype among the controlling members of a family, we assign a brood sex ratio and a brood size which would describe the brood produced if that genotype were in complete control of the brood. If more than one genotype exists among the controlling members in a single family, as is the case under sibling (sister or brother) control, then we assume that resources are allocated to each genotype in proportion to the frequency of that genotype among the controlling members of the family and that genotype then raises its sub-brood independently of other genotypes. The overall brood sex ratio will be an average of the sub-brood sex ratios, weighted by sub-brood size as shown in Tables 3 through 9. The construction described here differs from that of Charnov (1978) who assumed that the overall brood sex ratio is an average of the sub-brood sex ratios, weighted by the frequency of the controlling genotypes, rather than by sub-brood size.

Our Model III of father control of the sex ratio represents a special case of the model of Thomson and Feldman (1975) for the "SR" system which involves segregation distortion of the sex chromosomes and is similar to the model of Bengtsson (1977) for sex-linked sex reversion genes. In agreement

with both of the papers cited above, the condition for increase of a gene which affects both the fertility and sex ratio in broods produced by males is

$$\tau_1(1 - \varepsilon) - \tau_2(1 - \varepsilon) > 0,$$

i.e., that the produce of the fertility and frequency of females for the rare genotype be greater than that of the common genotype. Further, Thomson and Feldman (1975) showed that no interior equilibria are possible under sex ratio and fertility differences alone and that viability differences in females must exist for a stable polymorphism to arise. In agreement with their result, we find that no polymorphic equilibria exist in this case.

#### 4.2 *Boundary Behavior*

Stability conditions derived from the dynamic models were compared to expectations formed on the basis of a family of adaptive functions. The adaptive functions were averages over genotypes of quantities of the form

$$\frac{mb_{C \rightarrow M}}{M} + \frac{fb_{C \rightarrow F}}{F}, \quad (27)$$

where  $M/F$  is the population sex ratio,  $m/f$  is the brood sex ratio,  $b_{C \rightarrow M}$  is a measure of relatedness between the controlling members and the males produced in the brood, and  $b_{C \rightarrow F}$  is the relatedness between the controlling members and the females produced in the brood. Comparison of the actual stability conditions derived from the model with the conditions suggested by the adaptive functions provides a test of the definition of the relatedness in Table 2 (see Uyenoyama and Feldman, 1981). The relatedness for the father-to-son and father-to-daughter cases are 0 and 1, respectively, under both definitions. Further, the brother-to-sister relatedness is equal to the brother-to-sister relatedness under both definitions, although the measures are frequency-dependent in the case of the regressions. The same is true for sister-to-sibling relatedness in the diploid case. The only model studied here which provides a test of the appropriateness of the two definitions is the case of sister control in haplodiploids. In that case, the adaptive function correctly predicts the boundary stability condition if the appropriate regression coefficients rather than the coefficients in Table 1 are used as the measures of relatedness. Similarly, Uyenoyama and Feldman (1981) found that adoption of the regression coefficient, rather than the values in Table 1, as the measure of relatedness removes all apparent departures from Hamilton's rule in the case of additive fitnesses that were noted by Cavalli-Sforza and Feldman (1978).

### 4.3 Structure of the Interior Equilibria

In addition to prediction of boundary stability conditions, a useful adaptive function should predict the identity of interior equilibria and the stability conditions of those equilibria. The adaptive functions suggested here have these properties in a special case of our models in which two additional assumptions are made. The additional assumptions, (3) and (15), are that a symmetric equilibrium ( $\hat{p}_m = \hat{p}_f$ ) exist and that no dominance in the effect on brood sex ratio and brood size exist. The significance of these assumptions are discussed in turn.

First, as indicated before, the adaptive function (shown below for the case of sister control) involves an average across genotypes of the expression in (27):

$$\begin{aligned} \bar{W} = & f_1 \left[ \frac{\sigma_1 \alpha b_{S-B}}{M} + \frac{\sigma_1 (1 - \alpha) b_{S-S}}{F} \right] + f_2 \left[ \frac{\sigma_2 \beta b_{S-B}}{M} + \frac{\sigma_2 (1 - \beta) b_{S-S}}{F} \right] \\ & + f_3 \left[ \frac{\sigma_3 \gamma b_{S-B}}{M} + \frac{\sigma_3 (1 - \gamma) b_{S-S}}{F} \right]. \end{aligned}$$

In our models, we have substituted Hardy-Weinberg frequencies for  $f_1, f_2,$  and  $f_3,$  but for the moment we can view  $\bar{W}$  as a general average over expressions for contribution. By identical reasoning as in Uyenoyama and Bengtsson (1979) it can be shown by the use of Lagrange's method of undetermined multipliers (Courant, 1937, p. 190 *et seq.*) that a maximum of  $\bar{W}$  does not exist unless the symmetric root condition (3) holds. Further, we showed (Uyenoyama and Bengtsson, 1979) that (3) is in fact satisfied in a number of cases including the case of "constant total expenditure" (Spieth, 1974). Under the assumptions that expenditure determines brood size and that the total expenditure is constant across genotypes, the following relationship between brood size and brood sex ratio must hold (see Uyenoyama and Bengtsson, 1979 for further discussion):

$$\begin{aligned} \sigma_1 [\alpha + (1 - \alpha) \phi] &= \sigma_2 [\beta + (1 - \beta) \phi] = \sigma_3 [\gamma + (1 - \gamma) \phi] \\ \tau_1 [\delta + (1 - \delta) \phi] &= \tau_2 [\varepsilon + (1 - \varepsilon) \phi], \end{aligned} \quad (28)$$

where  $\phi$  is the expenditure required to raise a female relative to the required to raise a male. If the assumption in (28) is made then the symmetric root condition (3) is satisfied.

Second, the assumption of no dominance (15) was necessary in the case of sister control in haplodiploids in order that the suggested adaptive function have the desirable properties described above. Similarly, Uyenoyama and Feldman (1981) found that in the cases of sister-to-brother and sister-to-sib altruism in both haplodiploids and diploids, the adaptive functions predicted

the results only if no dominance is assumed in the  $h_i$  parameters which represent the propensity of the various genotypes to perform altruism. In that paper, they noted that the departures of the results from the predictions could be due either to incorrect formulation of some aspect of the adaptive function (e.g., definition of the relatedness measure, the differentiation procedure, etc.) or to limitations of verbal kin selection theory itself. We tend to favor the latter interpretation, although the significance of the departures remains unexplained. In the present paper, the case of sister control in diploids did not require the additional assumption of no dominance although the results for the case of sister control in haplodiploids still depart from predictions unless (15) holds.

#### 4.4 On the Validity of the Verbal Predictions of Kin Selection Theory

Under the assumptions, then, that both (3) and (15) hold, the adaptive functions predict the sex ratios shown in Table 10 at the asymmetric equilibria. Also shown are the values of the equilibrium sex ratios in the Fisher case represented by (28). The results for the case of mother control are from Uyenoyama and Bengtsson (1979). All results conform to the prediction from verbal kin selection theory that the sex ratio should evolve to a value proportional to the ratio of the relatedness measures between the

TABLE 10  
Population Sex Ratio Attained at the Asymmetric Equilibria

Model	Population sex ratio	Value in Fisher case
Sister control		
Haplodiploid	$\frac{-(\sigma_1\alpha + \sigma_3\gamma - 2\sigma_2\beta)b_{S \rightarrow B}}{[\sigma_1(1 - \alpha) + \sigma_3(1 - \gamma) - 2\sigma_2(1 - \beta)] b_{S \rightarrow S}}$	$\frac{\phi b_{S \rightarrow B}}{b_{S \rightarrow S}}$
Diploid	$\frac{-(\sigma_1\alpha + \sigma_3\gamma - 2\sigma_2\beta)}{[\sigma_1(1 - \alpha) + \sigma_3(1 - \gamma) - 2\sigma_2(1 - \beta)]}$	$\phi$
Brother control		
Haplodiploid	$\frac{-(\tau_1\delta - \tau_2\varepsilon)}{[\tau_1(1 - \delta) - \tau_2(1 - \varepsilon)]}$	$\phi$
Father control		
Haplodiploid	—	—
Mother control		
Haplodiploid or diploid	$\frac{-(\sigma_1\alpha + \sigma_3\gamma - 2\sigma_2\beta)}{[\sigma_1(1 - \alpha) + \sigma_3(1 - \gamma) - 2\sigma_2(1 - \beta)]}$	$\phi$

controlling members of the family and the males and females produced in the brood. However, because the frequency-dependent regression coefficient, rather than the constants in Table 1 appear to represent the appropriate measures of relatedness, the equilibrium sex ratio in the haplodiploid case is not proportional to 1:3. Depending on the particular values of the brood frequencies of males  $\alpha$ ,  $\beta$ , and  $\gamma$  in our model, the equilibrium sex ratio may vary considerably although it is proportional, in the no dominance case, to  $b_{S-B}/b_{S-S}$ . Even if it were the case that the theory discussed here could be applied directly to natural populations, no single sex ratio such as 1:3 would be expected to obtain in all populations (cf. Trivers and Hare, 1976). Note that deviations in the sex ratio from 1:3 would be expected even in the absence of evidence of worker-queen conflict.

Although the sex ratio at equilibrium does not in general converge to  $\phi/3$  in the case of sister control in haplodiploids, that sex ratio nevertheless represents an "ESS" (Maynard Smith and Price, 1973; Maynard Smith, 1974) in the sense that if the population is fixed for a genotype which produces that sex ratio in its broods, then no other genotype can increase when rare. This result agrees with that of Charnov (1978), who used different methods to reach this conclusion. The condition for local stability of the fixation of the  $A$  allele is given by (1), which reduces to the following in the Fisher case represented by (28):

$$(\beta - \alpha)\{3\alpha[\beta + (1 - \beta)\phi] - (1 - \beta)\phi[\alpha + (1 - \alpha)\phi]\} > 0. \quad (29)$$

It can be shown that the only value for  $\alpha$  at which (29) is always positive regardless of the value of  $\beta$  is

$$\frac{\alpha}{1 - \alpha} = \frac{1}{3}\phi.$$

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