Heredity (1982), **49** (2), 199–209 0018-067X/82/05210199$02.00

AN ATTEMPT TO DETECT GENETIC VARIATION IN SEX RATIO IN *DROSOPHILA MELANOGASTER*

M. A. TORO* and B. CHARLESWORTH

School of Biological Sciences, University of Sussex, Brighton BN1 9QG, U.K.

Received 29.iii.82

SUMMARY

Two experiments were carried out in order to test for the existence of genetic variability in sex ratio in an outbred population of *Drosophila melanogaster*. In the first, the existence of heterogeneity in progeny sex ratio among dams and among sires was tested. No significant heterogeneity was detected. The heterogeneity variance component among sires was estimated to be only $4{\cdot}4 \times 10^{-6}$. In the second experiment, artificial family selection for increased and decreased proportions of males was practised for nine generations in each of two replicate lines. Selection was successful for decreased proportions, but this was shown to be due to the presence of sex-linked recessive lethals. There was no evidence for an increase in the proportion of males in the lines selected for increased proportions. The realised heritability of sex ratio was estimated as $-0{\cdot}0053$, with an upper bound of $0{\cdot}0033$. It is concluded that genetic variation in sex ratio is effectively absent in this population. If this result were general it would cast doubt on the relevance of adaptative theories of primary sex ratio as far as diploid organisms are concerned.

1. INTRODUCTION

FISHER (1930) analysed the action of natural selection on the sex ratio. He showed that if there is an excess of females in the population, a parent who produces only sons will, on average, have more grandchildren than one who produces only daughters, or a mixture of sons and daughters. Therefore, genes tending to restore the sex ratio towards unity will spread; the same is true if there is an excess of females. Since then, Fisher's ideas have been extended to situations where there is local mate competition (Hamilton, 1967; Taylor and Bulmer, 1980), and where there is variation in offspring quality as a result of the mother's reproductive condition (Trivers and Willard, 1973; Charnov, 1979; Bull, 1981).

All these arguments have assumed that the sex ratio is an evolutionary variable under genetic control, usually by genes expressed in the parents. It is thus of importance to know if genetic variance for sex ratio exists because if this is not the case, the arguments about adaptive sex ratio would not be relevant. Major genes that can alter the sex ratio through meiotic drive are known in *Drosophila* (Sturtevant and Dobzhansky, 1936; Stalker, 1961; Jungen, 1968) and in the mosquito *Aedes aegypti* (Hickey and Craig, 1966). Such cases suggest that it might also be reasonable to expect genetic variation in sex ratio due to the segregation of minor genes affecting primary sex ratio.

* Present address (to which reprint requests should be sent): Instituto Nacional de Investigaciones Agrarias, Departmento de Genética, Carretera de La Coruña Km. 7, Madrid-35, Spain.

In haplo-diploid species, where the female can control the progeny sex ratio by determining whether or not an egg is fertilised, there is good evidence for sex ratio variation. For example, sex ratios in parasitoid species of Hymenoptera display patterns of variation between species that have clear selective significance (Hamilton, 1967; Waage, 1982) and there is also evidence of sex ratio variation within species (Werren *et al.*, 1981). In contrast, there is little convincing evidence of genetic variation in primary sex ratio in diploid organisms (reviews by Maynard Smith, 1978; Williams, 1979; Hohenboken, 1981). In man, the large data sets analysed by Edwards (1962, 1970) do not suggest the existence of a genetic component to variance in sex ratio. In domestic cattle, Bar-Anan and Robertson (1975) and Skjervold and James (1979) report small but significant birth sex ratio variation between the progenies of different bulls, but it is not clear whether this is due to variation in the primary sex ratio or to differences in sex-specific prenatal mortality rates. In poultry, Foster and McSherry (1980) concluded that there was no significant genetic variation in the sex ratio at hatching, on the basis of a study of between-family variation.

Ambiguous results have also been obtained by artificial selection experiments, in which families with high and low sex ratios have been selectively bred. King (1918) practised such selection on the Norwegian rat, and found a response to selection in both directions. Weir (1953) obtained opposite changes in sex ratio in lines of mice selected for increased and decreased blood pH. These correlated responses seem to have been due to a chance association between genes affecting pH and sex ratio, since replicate experiments did not yield the same result (Weir and Clark, 1955). Lavie and Beiles (1981) found evidence for a decrease in sex ratio (proportion of males) in lines of *Tribolium castaneum* selected for low sex ratio, but no evidence for a significant upward response in lines selected for high sex ratio. The response in the low lines could be due to the presence of sex linked lethals, and a similar effect was reported by Falconer (1954) in *Drosophila melanogaster*. Falconer failed to obtain a response to selection for an increase in sex ratio in this *Drosophila* material, nor was there any response in his experiments on mice. But his *Drosophila* experiments used a population originating from a single wild-caught female, and hence might have lost some genetic variability. The relatively small numbers that can be used in selection experiments on mammals limits the sensitivity of this kind of test of genetic variability.

It therefore seemed worth re-examining the question of the existence of genetic variation in sex ratio in *D. melanogaster*, using a population with a genetic base similar to that of a wild population, and scoring a sufficiently large number of individuals to overcome the large binomial sampling component of sex ratio variance. Two experiments will be described here. The first investigated the genetic component of sex ratio variance using comparisons of families, and the second involved artificial family selection for increased and decreased sex ratios.

2. Materials and methods

(i) *Source of the experimental population (IV population)*

The experimental population of *Drosophila melanogaster* (IV) used in the two experiments reported here was founded from a cross of 21 isofemale

lines homokaryotypic for the standard gene arrangements of the major chromosomes; the stock was derived in late 1976 from a stock founded by 200 flies of each sex, collected by P. T. Ives in 1975 near South Amherst, Massachusetts (U.S.A.). The South Amherst population is among the best known natural population and has been studied continuously for 40 years (Ives, 1970).

(ii) *Culture methods*

The population has been maintained since its foundation in ten one-third pint milk bottles with Lewis *Drosophila* medium. Each bottle of a new generation was derived from a pair of bottles of the preceding generation, ensuring substantial gene flow between bottles. The total adult population never fell below the number of the original sample and was usually into the thousands. All handling was performed at room temperature using CO_2 anaesthesia. The flies were kept at $25 \pm 1{\cdot}5$°C under a 12 hours light-dark cycle.

The culture methods attempted to retain the genetic variation of the population. Electrophoretic studies have shown a normal level of variability in the few enzyme loci studied: phosphoglucomutase, alcohol dehydrodrogenase and α-glycerophosphate dehydrogenase.

(iii) *Experimental Procedure*

Experiment 1 (*Genetic variance estimation*)

The matings were set up by placing each of 22 males with 10 females in a vial for 72 hours, after which each female was placed in a separate vial. Twenty-four hours later all females were transferred to new vials and discarded after a further 24 hours. The progeny emerging in each vial were removed and counted daily until cultures were exhausted. The whole experiment was repeated five times at two-weekly intervals.

Experiment 2 (*Selection experiment*)

For the foundation stock, 80 virgin females and 80 males were collected from the IV stock and 80 single-pair matings set up. The females were allowed to lay eggs at 25°C for four days. They were then transferred to fresh vials for a second laying period. This second set of vials were kept at 18°C for five or six days and then transferred to the 25°C room. This was done to prolong the period between egg and pupa from 9 to 14–16 days.

The progeny from the first set of vials were removed and counted daily for one week, and then the sex ratio in each family was calculated. Fifty randomly chosen families were assigned to the selection line and ten to the control line. The ten families from the selection line with the highest sex ratio constituted generation 1 of the upward selection line *M* (the line selected to increase the proportion of males) and the ten families with the lowest sex ratio made up the downward selection line *F* (the line selected for an increased proportion of females).

The purpose of the second set of vials was to provide virgin flies for the next generation. Five males and five females were collected from each

vial corresponding to those families that had been selected. The matings were of random pairs, but brother-sister pairs were avoided in order to minimise the inbreeding. This cycle was repeated for nine generations for *M*, *F* and Control lines. The whole experiment was repeated after a two weeks interval.

In generation nine in replicate 1, and eight in replicate 2, a mistake in food preparation was made (lack of yeast in the medium) that caused an increase in the mortality of the flies. After generation nine, a final evaluation of the sex ratio was made in each line. Two males and two virgin females were collected from each of the families and 100 single-pair matings were set up and allowed to lay eggs for 24 hours, then transferred to fresh vials for a second laying period of 24 hours, and so on for ten laying periods, after which they were discarded.

3. Results

(i) *Experiment 1. Estimation of the genetic variance*

The data have been classified according to four main factors: replicate, sire family, dam family, and egg laying period. A χ^2 heterogeneity analysis was carried out and the results are given in table 1. The overall sex ratio was $0{\cdot}4971 \pm 0{\cdot}0015$, based on a sample size of 107,789. The χ^2 value for the deviation from 0·50 was 3·64 with 1 degree of freedom. This value is not significant. There was no heterogeneity between replicates or between laying periods; the extremely good fit for the last factor is probably due to chance, but indicates that the two values correspond to two different measurements of the same character.

The next analysis considered heterogeneity in sex ratio between dams within sires. The heterogeneity χ^2 was computed as 844·11 with 854 degrees of freedom and it is clearly not significant ($P < 0{\cdot}50$). Data from dams were therefore pooled for each sire and the resulting heterogeneity χ^2 between sire families was 80·14 with 104 degree of freedom ($P > 0{\cdot}50$). In spite of the lack of heterogeneity, the component of variance (σ_s^2) between sires in the proportion of males was estimated. There are three methods available for this estimation: the χ^2 method (Robertson and Lerner, 1949), the simplified maximum likelihood method (Robertson, 1951), and the method of Bar-Anan and Robertson (1975). The results of the three methods are given in table 2. Two methods gave similar estimates of σ_s^2; $4 \pm 3 \times 10^{-6}$ approximately. However, the χ^2 method gave a negative estimate of σ_s^2.

The variance between the sires in the "real" sex ratio has also been estimated, treating the sex ratio from the two laying periods as independent

TABLE 1

Heterogenetiy χ^2 analysis of sex ratio

Source of variation	df	χ^2	Probability
Deviation from 0·50	1	3·64	$0{\cdot}10 > P > 0{\cdot}05$
Between replicates	4	2·91	$P > 0{\cdot}50$
Between egg laying periods	1	0·01	$P > 0{\cdot}50$
Between sires within replicates	104	80·14	$P > 0{\cdot}50$
Between dams within sires	854	844·11	$P > 0{\cdot}50$
Between vials within dams	805	765·09	$P > 0{\cdot}50$

TABLE 2

Estimates of the heterogeneity variance between sires

Method	σ_s^2	$V(\sigma_s^2)$
χ^2	$-12{\cdot}75\times10^{-6}$	—
Simplified maximum likelihood	$4{\cdot}45\times10^{-6}$	$1{\cdot}01\times10^{-9}$
Bar-Anan and Robertson	$4{\cdot}23\times10^{-6}$	$1{\cdot}07\times10^{-9}$

measures and estimating the covariance between the two, the problem of weighting being solved using the Bar-Anan and Robertson method. The final estimate of the covariance between the proportion of males in the two groups of offspring was $1{\cdot}32\pm4{\cdot}75\times10^{-5}$.

(ii) *Experiment 2. Artificial selection for sex ratio*

The mean of the sex ratio character and its standard error in the two replicates of the foundation stock are shown in table 3. The overall sex ratio is $0{\cdot}5004\pm0{\cdot}0035$, based on a sample size of 19,965. There was no heterogeneity between replicates ($\chi^2=0{\cdot}626, P>0{\cdot}25$) or between families ($\chi^2=155{\cdot}93$, $P>0{\cdot}25$). The heterogeneity variance between families in the proportion of males (σ_p^2) has also been estimated using the three methods described above: χ^2, simplified maximum likelihood, and Bar-Anan and Robertson's method. The results are give in table 4. The three methods yield similar estimates of σ_p^2, all of them being not significantly different from zero.

TABLE 3

Parameters of the foundation stock

Line	No. of progenies	No. of flies		Sex ratio
		Males	Females	
Replicate 1	79	5887	5822	$0{\cdot}5028\pm0{\cdot}0046$
Replicate 2	66	4104	4152	$0{\cdot}4970\pm0{\cdot}0055$
Total	145	9991	9974	$0{\cdot}5004\pm0{\cdot}0035$

TABLE 4

Estimates of the heterogeneity variance between families in the foundation stock

Method	$\sigma_p^2(\times10^{-4})$	$V(\sigma_p^2)(\times10^{-8})$
χ^2	1·559	—
Simplified maximum likelihood	1·682	4·452
Bar-Anan & Robertson	1·616	5·346

The results of selection are shown in tables 5 and 6. For each replicate, line and generation, the overall sex ratio and the standard error (assuming binomial sampling) have been calculated. The first striking result is the large sex ratio changes from downward selection, with remarkably constant values for the sex ratio in the upward selection. The most probable explanation for the response from downward selection is that it is due to sex-linked lethals. This was demonstrated by appropriate crosses with the multiple-inverted X-chromosome balancer FM7, which carries the markers y^{31d} *sc dm* w^aB. It is not known if there are one or several sex-linked lethals and whether they were already present in the base population or arose by

TABLE 5

Sex ratio values during selection (Replicate 1)

Generation	Downward selection (F)	Upward selection (M)	Control
0	0·5001±0·0058	0·5001±0·0058	0·4933±0·0130
1	0·4048±0·0058	0·5091±0·0062	0·4900±0·0116
2	0·4992±0·0059	0·5106±0·0054	0·5103±0·0113
3	0·4880±0·0066	0·5005±0·0062	0·5064±0·0130
4	0·4869±0·0070	0·5078±0·0067	0·5032±0·0120
5	0·4339±0·0075	0·5051±0·0063	0·4941±0·0135
6	0·4803±0·0072	0·4898±0·0070	0·4710±0·0162
7	0·4771±0·0066	0·5082±0·0076	0·5042±0·0077
8	0·5034±0·0085	0·5126±0·0069	0·5224±0·0083
9	0·4792±0·0098	0·4948±0·0138	0·4962±0·0153

TABLE 6

Sex ratio values during selection (Replicate 2)

Generation	Downward selection (F)	Upward selection (M)	Control
0	0·4974±0·0063	0·4974±0·0063	0·4988±0·0143
1	0·4567±0·0053	0·5089±0·0059	0·5196±0·0125
2	0·4899±0·0073	0·5070±0·0069	0·4901±0·0138
3	0·4916±0·0056	0·4964±0·0076	0·4997±0·0113
4	0·5000±0·0060	0·5163±0·0065	0·5229±0·0128
5	0·4925±0·0068	0·5006±0·0070	0·4764±0·0149
6	0·4761±0·0077	0·5120±0·0071	0·5124±0·0129
7	0·4884±0·0068	0·5009±0·0067	0·5024±0·0068
8	0·4821±0·0090	0·5014±0·0080	0·4625±0·0107
9	0·4545±0·0066	0·5106±0·0071	0·5092±0·0062

mutation during the selective process: the latter is possible, since the rate of mutation to sex linked lethals is 0·2 per cent per generation (Falconer, 1954). The analysis of these lines will not be discussed further.

In order to detect any sex ratio change in the male lines, the regression of sex ratio on generation has been calculated for each replicate, as shown in table 7. The regression coefficients are very small and the standard errors are mostly greater than the actual values of the estimates. As has been explained in section 2, a more accurate estimation of the sex ratio was carried out on each line at the end of the selection experiment. The final results, pooled over vials and over laying periods, are given in table 8.

There is no significant difference between the male and the control lines ($\chi^2 = 2{\cdot}63$ for replicate 1, $\chi^2 = 0{\cdot}05$ for replicate 2, $\chi^2 = 1{\cdot}54$ for the two replicates pooled). It is interesting to note that the control lines have a higher sex ratio (see later).

TABLE 7

Regression coefficients (b) *of sex ratio on generation for the upward selection and control lines*

Replicate 1		Replicate 2	
Line	$b(\times 10^{-4})$	Line	$b(\times 10^{-4})$
Upward selection	−1·79±7·70	Upward selection	4·58±7·46
Control	1·79±1·25	Control	−5·23±11·26

TABLE 8

Sex ratios in the final evaluation of the upward selection and control lines

Line	No. of progenies	No. of flies Males	Females	Sex ratio
Replicate 1				
Upward selection	75	16,558	16,592	0·4995 ± 0·0027
Control	84	16,842	16,456	0·5058 ± 0·0027
Replicate 2				
Upward selection	96	20,323	20,394	0·4991 ± 0·0025
Control	102	21,197	21,202	0·4999 ± 0·0024
Replicate 1 + 2				
Upward selection	171	36,881	36,986	0·4993 ± 0·0018
Control	186	38,039	37,658	0·5025 ± 0·0018

TABLE 9

Goodness of fit to a 1:1 sex ratio in the upward selection and control lines

Line	χ^2	Probability
Replicate 1		
Upward selection	0·03	$P > 0·50$
Control	4·47	$0·05 > P > 0·025$
Replicate 2		
Upward selection	0·12	$P > 0·50$
Control	0·00	$P > 0·50$
Replicate 1 + Replicate 2		
Upward selection	0·15	$P > 0·50$
Control	1·92	$0·25 > P > 0·10$

Table 9 shows the χ^2 values for the deviations from a 0·50 sex ratio. For the male lines, the values are clearly not significant. The control lines gave a significant value for replicate 1 but the significance disappears when the two replicates are pooled.

A heterogeneity χ^2 test has also been performed to investigate the possible existence of heterogeneity between families. The χ^2 values and their probabilities are given in table 10. The data have been combined over vials and over laying periods. In all the lines, the heterogeneity between families is significant, except for the male line in replicate 2. The heterogeneity variances have been estimated and the results are given in table 11. The three methods yield similar estimates of σ_p^2 but not of $V(\sigma_p^2)$.

TABLE 10

χ^2 tests for heterogeneity between families in the upward selection and control lines

Line	df	χ^2	Probability
Replicate 1			
Upward selection	79	103·74	$P < 0·05$
Control	83	126·48	$P < 0·05$
Replicate 2			
Upward selection	95	109·91	$0·10 < P < 0·25$
Control	101	163·03	$P < 0·05$

TABLE 11

Estimates of the heterogeneity variance between families in the upward and control lines

Line and Method	$\sigma_p^2(\times 10^{-4})$	$V(\sigma_p^2)(\times 10^{-8})$
Replicate 1		
Upward selection		
χ^2	2·19	—
ML	1·24	0·73
BA & R	1·33	1·24
Control		
χ^2	3·32	—
ML	2·70	0·84
BA & R	3·00	1·97
Replicate 2		
Upward selection		
χ^2	0·87	—
ML	0·63	0·65
BA & R	0·67	0·82
Control		
χ^2	3·78	—
ML	1·96	1·49
BA & R	2·72	1·47

4. DISCUSSION

The search for genetic variability for sex ratio in *Drosophila melanogaster* has yielded negative results in the present study. The main aim of Experiment 1 was to identify sources of heterogeneity other than that due to binomial sampling, and which could be attributed to genetic variance in sex ratio. The χ^2 analysis of table 1 has shown, however, that differences between vials and between families were as expected from binomial sampling. Two methods have been used to estimate the heterogeneity variance in sire families. The first uses the distribution of overall sex ratio for individual sires, and gave an estimate of about $4.4 \pm 32 \times 10^{-6}$. The second estimates the covariance between the two measurements of the sex ratio in the offspring of the sire families and gave a value of $13 \pm 47 \times 10^{-6}$. Neither estimate was significant. Indeed, the close agreement with expectation from binomial sampling was surprising and implied that environmental factors affecting sex ratio were virtually absent.

The results of the selection experiment are also very clear. Nine generations of selection have been unable to increase the sex ratio. Selection was very effective in increasing the number of females, but the presence of sex linked lethals was shown to be responsible for this. There are two relevant lines of evidence. Firstly, the coefficients of regression on generation of the sex ratios from upward selection are not significantly different from zero. Secondly, in the final evaluation of the selection response the sex ratio in the upward selection lines was $0{\cdot}4993 \pm 0{\cdot}0018$, less than the sex ratio of the control lines, $0{\cdot}5025 \pm 0{\cdot}0018$. The ratio of total response to total selection differential is $-0{\cdot}0032/0{\cdot}606 = -0{\cdot}0053$. This is the simplest estimator of the realised heritability of sex ratio. Falconer (1954) has suggested another method for obtaining an upper bound for the heritability: the observed total response is $-0{\cdot}0032$ and twice the standard error of this difference is 0·002 so that the maximum admissible heritability at

the 95 per cent level is 0·0033. When all the data from each generation are pooled, the sex ratios in the male and control lines were found to be $0{\cdot}5029 \pm 0{\cdot}0012$ and $0{\cdot}5024 \pm 0{\cdot}0014$ respectively. These are remarkably similar, although a slight excess of males seems to exist.

The heterogeneity between families was found to be significant in the final evaluation of the *M* and control lines. Because no heterogeneity was found in the foundation stock or in Experiment 1, this heterogeneity might have been brought about during the period of selection, probably as a collateral effect of inbreeding. Despite the fact that brother-sister mating was avoided, there has been a decrease in the number of flies emerging per vial, as table 12 shows for the first seven generations. Campos Rosado and Robertson (1966) have argued that with inbreeding there are fluctuations in gene frequency about their equilibrium values. These fluctuations will decrease the mean fitness of the population by increasing the frequencies of homozygotes. While at autosomal loci this decrease will affect both sexes, at sex-linked loci it will affect only the homogametic sex. Consequently, the frequency of the homogametic sex is expected, on average, to be reduced. Thus, inbreeding can provide an explanation not only for the heterogeneity between families, but also for the slight excess of males in both male and control lines. An alternative explanation is simply to assume that for inbred flies, the difference between male and female adult mortality (due, for example, to crowded conditions) has been magnified. Probably both genetical and enviromental factors play a role in this heterogeneity.

TABLE 12

Number of flies per vial by generation

Generation	Upward selection	Control
0	136·55	135·65
1	135·69	146·21
2	139·53	131·70
3	107·78	142·41
4	116·54	142·41
5	112·89	146·12
6	101·85	110·03
7	99·17	103·97

Whereas theoretical considerations of the action of natural selection of the adaptive control of progeny sex ratio have advanced rapidly in the last few years, little progress has been made in establishing empirical evidence in favour of the existence of such adaptation, except for haplo-diploid organisms (Maynard Smith, 1978, 1980; Williams, 1979). There are basically two difficulties in testing Fisher's ideas and its extensions: (a) it is very difficult to get reliable estimates of the primary sex ratio, especially in higher vertebrates, and (b) even if distorted values of the sex ratio exist, non-adaptive hypotheses can be proposed such as differences in mortality according to sex, or special physiological properties of the reproductive system.

From the present study it seems clear that multi-factorial genetic variance of sex ratio in *Drosophila melanogaster* is effectively absent. Furthermore, this evidence raises doubts about theories of adaptive sex ratio in

all diploid organism and suggests that this character differs from most metrical characters in its underlying genetic control. It supports the hypothesis that the sex ratio, being a simple consequence of Mendelian segregation, is not susceptible to evolutionary forces, and that the sex ratio problem is only an example of a simpler problem namely, the stability of the Mendelian mechanism.

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