QUANTITATIVE GENETICS OF *DROSOPHILA MELANOGASTER*. I. SEXUAL DIMORPHISM IN GENETIC PARAMETERS FOR WING TRAITS

DAVID E. COWLEY,*[,1] WILLIAM R. ATCHLEY[†,1] AND J. J. RUTLEDGE*

**Department of Meat and Animal Sciences and †Department of Genetics, University of Wisconsin, Madison, Wisconsin 53706*

Manuscript received October 17, 1985
Revised copy accepted July 12, 1986

ABSTRACT

Sexual dimorphism in genetic parameters is examined for wing dimensions of *Drosophila melanogaster*. Data are fit to a quantitative genetic model where phenotypic variance is a linear function of additive genetic autosomal variance (common to both sexes), additive genetic *X*-linked variances distinct for each sex, variance due to common rearing environment of families, residual environmental variance, random error variance due to replication, and variance due to measurement error and developmental asymmetry (left *vs.* right sides). Polygenic dosage compensation and its effect on genetic variances and covariances between sexes is discussed. Variance estimates for wing length and other wing dimensions highly correlated with length support the hypothesis that the Drosophila system of dosage compensation will cause male *X*-linked genetic variance to be substantially larger than female *X*-linked variance. Results for various wing dimensions differ, suggesting that the level of dosage compensation may differ for different traits. Genetic correlations between sexes for the same trait are presented. Total additive genetic correlations are near unity for most wing traits; this indicates that selection in the same direction in both sexes would have a minor effect on changing the magnitude of difference between sexes. Additive *X*-linked correlations suggest some genotype × sex interactions for *X*-linked effects.

SEX dimorphism in animals usually involves differences in size, color and various body adornments. In *Drosophila melanogaster*, not only are females larger than males for most body dimensions but also the sexes differ in pigmentation, the number of visible abdominal segments, structure of the genitalia, presence of sex combs, shape of various body parts, behavior and numerous other features. Stimulated by discussions about sexual selection and related topics, many biologists have examined the complexity in expression of sexual differences (*e.g.*, ATCHLEY 1971).

In many instances, biologists have been satisfied simply with demonstrating that sex differences exist in the phenotypic means of traits. However, sex dimorphism in genetic parameters underlying morphological and physiological

[1] Present address: Department of Genetics, North Carolina State University, Raleigh, North Carolina 27695-7614.

traits may have a far-reaching impact in an evolutionary sense. When sex differences occur in narrow-sense heritability (h^2) and when the additive genetic correlation between sexes (for the same trait) is less than unity, sex dimorphism may evolve if there is selection. Under these conditions, both direct and indirect responses to selection may differ between sexes in spite of equivalent selection intensities (GRIFFING 1965). Sex dimorphism might also arise indirectly if genetic covariance structure between traits differs by sex. Thus, selection could cause the difference between sexes to evolve in a second correlated trait if correlated response differs by sex. Obviously, elucidation of the underlying factors involved in expression of sex differences in variance-covariance structure is critical to understanding the evolutionary significance of this phenomenon.

The simplest sex difference in variance-covariance structure is a scaling relationship. In this instance, one sex is larger than the other, and the variance scales with the trait mean. Sex differences in variance arising from a simple scaling relationship can be minimized when the data are suitably transformed to a common scale (WRIGHT 1968; FALCONER 1981). In an evolutionary sense, scaling is not expected to contribute to the evolution of sexual dimorphism, insofar as the scaling relationship influences both the additive genetic and phenotypic variances equally in both sexes.

Variance-covariance structure may differ between sexes from genotype by sex interaction where the same autosomal genes are expressed differentially in the hormonal environment of each sex (ROBERTSON 1959; EISEN and LEGATES 1966). A genotype × sex interaction should result in a genetic correlation between males and females that is less than unity.

A more complex arrangement results when some loci influencing expression of a trait occur on the *X* chromosome. A specific type of genotype by sex interaction is dosage compensation of the *X* chromosome. Dosage compensation in Drosophila differs from the mammalian system in that both *X* chromosomes in females function and jointly produce the same amount of gene product as the single *X* chromosome does in males. Concentration of various *X*-linked enzymes suggest that an *X* chromosome is twice as active in males as in females (BAKER and BELOTE 1983, and references therein).

The level of dosage compensation in the Drosophila system will complicate expression of polygenic variability. For example, given a single *X*-linked locus with two alleles, no dominance in females and complete dosage compensation in males, the *X*-linked variance for males is twice the *X*-linked variance for females. Conversely, if there is no dosage compensation in males, the *X*-linked variance for females is twice that in males (JAMES 1973). Since the *X* chromosome in Drosophila is about 20% of the haploid genome, clarifying the mechanism for dosage compensation is of considerable importance in the development of a realistic genetic model to identify unique sources of additive genetic variance for each sex.

Previous quantitative genetic studies of sex dimorphism have been of two primary types: (1) estimates of variance components for single traits, *e.g.*, bristle number or wing length in Drosophila (SCHAFFER and KOJIMA 1963; SHERIDAN

et al. 1968; FRANKHAM 1977) or body weight in mice (EISEN and LEGATES 1966); (2) single trait selection studies to modify the magnitude of sex dimorphism (FRANKHAM 1968a,b; EISEN and HANRAHAN 1972; BIRD and SCHAFFER 1972).

In this paper the following questions are addressed: (1) Is *X*-linked variance a substantial portion of additive genetic variance for wing traits of *Drosophila melanogaster*? (2) Is *X*-linked variance for males greater than *X*-linked variance for females? (3) Do different wing dimensions have additive genetic variances composed of similar proportions of *X*-linked and autosomal variances? (4) Do the relative proportions of autosomal and *X*-linked variances suggest that gene effects are randomly distributed between the autosomes and the *X* chromosome?

The wing was chosen because it is known to exhibit sex dimorphism in overall size, and the *X* chromosome has been shown to have a substantial effect on wing length (BIRD and SCHAFFER 1972). The wing has been the subject of numerous genetic and developmental analyses and is therefore developmentally well known. The wing is a single developmental entity that is derived from a single imaginal disc that is subdivided into anterior and posterior compartments (BRYANT 1978). Finally, wings in Drosophila are used not only for locomotion and dispersal but also for mating displays. Because of this dual function, wings may be subject to both natural and sexual selection.

MATERIALS AND METHODS

Flies used in this study were obtained from seven randomly mating populations of approximately 2000 individuals each. These populations were initiated in 1975 from large samples taken from the University of Wisconsin Arboretum. To facilitate laboratory work, progeny were reared from matings grouped into 14 experiments (or replicates).

For each experiment, approximately 50 randomly chosen males were each mated to four randomly chosen females in a paternal half-sib design. Within each half-sib family, two full-sib families were chosen for subsequent analyses. Progeny were randomly chosen from these two families in the following manner. Two progeny of each sex were taken from one family, and one individual of each sex was chosen from the remaining family (Bainbridge Nested Design; BAINBRIDGE 1963). A total of 494 half-sib families, each composed of three individuals of each sex, were used in this study.

Both wings were removed from each fly and were permanently mounted on a microscope slide. Thirteen measurements were recorded on each wing, using an ocular micrometer on a dissection microscope. Both wings were measured to estimate variance due to measurement error and developmental asymmetry between the left and right sides.

Landmarks on the wing are designated A through N, and distances between reference points are recorded as AE, GI, GL, MN, CL, AC, CE, CI, EI, EL, IL, FD, and HB (Figure 1). Results for these traits are subsequently grouped into wing length, width, distance between wing vein ends and distances between crossveins. The anterior-posterior compartment boundary of the wing lies just anterior and approximately parallel to the fourth longitudinal vein (distance AE) (BRYANT 1978).

Scaling: Since comparison of *X*-linked variance estimates between sexes can be biased by scale effects (unlike heritability and genetic correlation, which are scale-free), males and females were first scaled to have the same mean by the method of FALCONER and KING (1953). Then, a linear regression of standard deviation on trait mean using all

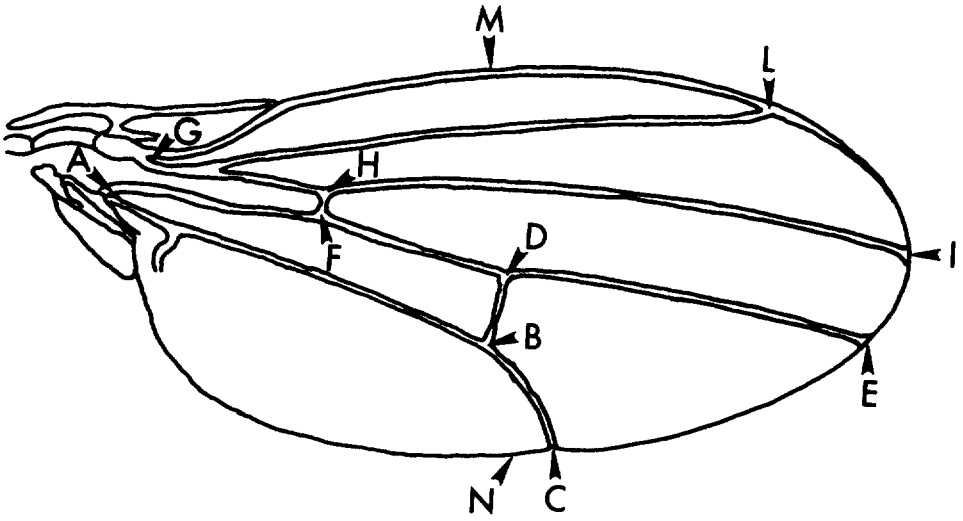

FIGURE 1.—Representative wing of *Drosophila melanogaster* with landmarks used in the present study. Distances between two points were recorded as AC, AE, FD, GI, GL, CE, CI, CL, EI, EL, IL, HB and MN.

13 wing dimensions was calculated separately for each sex. Intercept and slope were not significantly different between sexes, so a pooled regression of standard deviation on mean was calculated. The relation between standard deviation and mean was decoupled by transforming the scaled data according to WRIGHT (1968), where the transformed data is $x' = \log(x + k)$, and k is the ratio of intercept to slope for the pooled regression of standard deviation on mean. For these analyses $k = 468$.

A logarithmic transformation of the data seemed biologically reasonable since cell multiplication in the wing imaginal disc of Drosophila is exponential through the third larval instar (MARTIN 1982). However, it was determined that a simple logarithmic transformation was inappropriate because the regression of standard deviation on mean for log-transformed data had a negative slope that was significantly different from zero.

Genetic model: It is assumed that the phenotypic (total) variance for the ith sex ($i = m$ for males, f for females) is

$$\sigma^2_{Pi} = \sigma^2_{Expi} + \sigma^2_{Aa} + \sigma^2_{Axi} + \sigma^2_C + \sigma^2_e + \sigma^2_{Ni}, \quad (1)$$

where σ^2_{Expi} is variance due to experiments for the ith sex; σ^2_{Aa} is additive genetic autosomal variance; σ^2_{Axi} is additive genetic X-linked variance for the ith sex; σ^2_C is variance due to common rearing environment of full-sib families; σ^2_e is residual environmental variance; and σ^2_{Ni} is variance due to measurement error and developmental asymmetry.

In Drosophila, the X chromosome is large and comprises about 20% of the genome. The male is the heterogametic sex (XY) and sex determination is of the XO form (BAKER and BELOTE 1983). The large X chromosome may be an important source of additive genetic variance that clearly could differ between sexes depending on the level of dosage compensation. Therefore, separate X-linked additive genetic parameters are assumed for each sex. The Y chromosome of Drosophila is largely heterochromatic, and it has not been shown to contribute substantially to polygenically determined traits. Variance due to the Y chromosome is assumed to be zero.

Common environment and residual environment variances (σ^2_C and σ^2_e) and the autosomal variance (σ^2_{Aa}) are each assumed equal between sexes. The model excludes autosomal dominance, X-linked dominance in females, and epistatic variances. If dominance and epistasis are important sources of variance, σ^2_C and σ^2_e will be contaminated

with fractions of nonadditive and epistatic variances, and this will bias the estimate of σ^2_{Axm}. If autosomal variance is not equal between sexes, the estimate of σ^2_{Axf} will be biased.

It is of considerable interest to examine the covariance between sexes for the same trait. By randomly pairing males with females within full-sib families, the phenotypic covariance between sexes is

$$\sigma_{P(m,f)} = \sigma_{Exp(m,f)} + \sigma_{Aa(m,f)}/2 + \sigma_{Ax(m,f)}/2 + \sigma^2_C + \sigma_{e(m,f)} + \sigma_{N(m,f)}. \quad (2)$$

Statistical analyses: The completely random model used in the five-level nested analysis of variance is

$$Y_{ijklm} = \mu + Exp_i + S_{j(i)} + D_{k(ij)} + W_{l(ijk)} + N_{m(ijkl)}, \quad (3)$$

where μ = population mean; Exp_i = the effect due to the ith replicate experiment; $S_{j(i)}$ = effect of the jth sire nested in the ith replicate experiment; $D_{k(ij)}$ = the kth dam nested in the jth sire in the ith replicate; $W_{l(ijk)}$ = the lth progeny (fly) nested in the kth dam in the jth sire in the ith replicate.

The phenotypic variance (1) can be expressed for each sex as

$$\sigma^2_P = \sigma^2_{Exp} + \sigma^2_S + \sigma^2_D + \sigma^2_W + \sigma^2_N. \quad (4)$$

Covariance of half-sibs is given by σ^2_S, whereas σ^2_D is the covariance of full-sibs nested in half-sib families. Covariance between individuals within full-sib families is given by σ^2_W. The variance between replicate experiments (σ^2_{Exp}) is assumed to be random sampling variance, and σ^2_N is a combination of measurement error and developmental instability between left and right wings. Expectations (BOHIDAR 1964; JAMES 1973) of these design variance components in terms of the assumed genetic model are given in the APPENDIX.

Four times the intraclass correlation of half-sibs is usually given as an estimate of narrow-sense heritability (h^2). However, if X-linked variance is an important part of total additive genetic variance, the intraclass correlation of half-sibs will underestimate heritability for males and overestimate heritability for females (Table A2, APPENDIX). Therefore, heritability calculated in this manner is useful in assessing the importance of X-linked variance.

Mean squares from the nested analysis of variance were equated to their expectations in terms of causal components; the resulting system of equations was solved by least squares, and estimators of the unknown causal components were obtained. Additional details of the statistical analyses are outlined in the APPENDIX.

Correlations between sexes for the same trait: Like heritability estimates, the half-sib and full-sib correlations may be biased estimates of the additive genetic correlation between sexes for the same trait. Therefore, correlations calculated from design components are included for comparison with correlations calculated from estimated causal components. Comparison in this manner offers additional evidence regarding the presence of X-linked effects for various wing traits.

The half-sib covariance component between sexes divided by the half-sib variance component for males may be considered a genetic regression coefficient (b_{Aa}) for female autosomal effects on male autosomal effects. This is a qualitative assessment, because it is not possible to test statistically for a significant deviation from a value of 1.0 that is expected if autosomal effects are the same in both males and females.

Genomic distribution of polygenic variance: One might hypothesize that the relative proportions of autosomal and X-linked variance reflect the proportion of the total genome occupied by the autosomes and the X chromosome. If gene effects are small and randomly distributed throughout the genome, then the amount of X-linked additive genetic variance should be approximately equal to the proportion of the haploid genome occupied by the X chromosome.

There are at least two methods of estimating the relative size of chromosomes, *i.e.*, the number of map units and the number of bands on the polytene salivary gland chromosomes. There are 282 map units, of which 66 are on the X chromosome,

TABLE 1

Mean ($\bar{Y}$), phenotypic standard deviation(s) and coefficient of variation (*CV*) for 13 wing traits in *Drosophila*

Trait	Males			Females		
	$\bar{Y}$	s	CV	$\bar{Y}$	s	CV
Wing length						
AE	1822.91	53.53	2.94	2095.17	57.59	2.75
GI	1810.91	54.68	3.02	2071.94	59.26	2.86
GL	1466.39	52.53	3.58	1692.28	57.41	3.39
Wing width						
MN	903.28	28.74	3.18	1012.61	30.28	2.99
CL	933.62	29.68	3.18	1040.86	31.68	3.04
Between vein ends						
AC	1218.15	42.10	3.46	1396.92	43.74	3.13
CE	778.87	26.92	3.46	894.48	30.59	3.42
CI	936.10	31.26	3.33	1058.56	34.97	3.30
EI	205.23	12.17	5.93	216.30	13.92	6.44
EL	586.31	21.43	3.66	631.02	23.42	3.71
IL	488.15	22.75	4.66	527.65	25.81	4.89
Crossveins						
FD	409.28	26.04	6.36	464.02	28.78	6.20
HB	474.21	26.66	5.62	542.99	30.13	5.55

Data are in microns. The sample size is 1482 individuals (2964 wings) of each sex. The means differed between sexes at $P < 0.001$ for all traits.

suggesting that the *X* comprises 23% of the total genome. There are 5162 bands on the salivary gland chromosomes, of which 1024 are on the *X* (LINDSLEY and GRELL 1967). Thus, 20% of the bands are on the *X* chromosome. If genes controlling a given wing trait are randomly distributed throughout the genome and have small equal effects, then 20–23% of the total additive genetic variance in females would be *X*-linked.

Since the *X* chromosome in male Drosophila is dosage compensated, the percentage of the haploid genome occupied by the *X* chromosome (*M*) may be larger than that for the female genome (*F*) by a factor (k) proportional to the level of dosage compensation

$$M = 100kF/(kF + 100 - F). \quad (5)$$

Assuming complete dosage compensation, then $k = 2$ and the expected proportion of additive genetic *X*-linked variance for males is 33–38%. With no dosage compensation, $k = 1$ and the expected proportion is the same as that for females.

RESULTS

Means and phenotypic standard deviations for the 13 wing traits before scaling and transformation are given in Table 1. Highly significant mean differences between sexes ($P < 0.001$) exist for all traits. Females are about 15% larger than males for wing length (traits AE, GI and GL) and are about 12% larger in wing width (MN) and CL). Three distances at the tip of the wing in the anterior compartment (EI, EL and IL) average 7% larger in females. The remaining distances between vein ends (AC, CE and CI) and the distances between crossveins (FD and HB) average 14% larger in females.

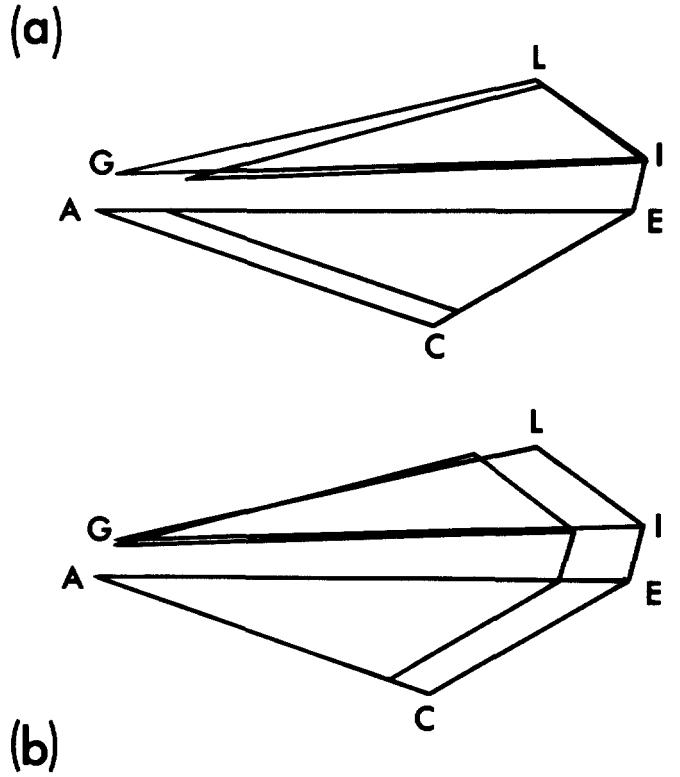

FIGURE 2.—Male and female means (n = 1482 for each sex) superimposed for traits AE, GI, GL, AC, CE, CI, CL, EI, EL and IL. The fourth longitudinal wing vein (AE) approximately coincided with the anterior-posterior compartment boundary and is used as a common reference line. Point E is the common point between sexes in (a), and point A is the common endpoint in (b).

In Figure 2, male and female means for ten traits (AE, GI, GL, AC, CE, CI, CL, EI, EL and IL) are superimposed, assuming the fourth longitudinal wing vein (AE) as a reference line. The anterior-posterior compartment boundary lies just anterior and approximately parallel to distance AE. This permits a visual comparison within compartments of differences in wing dimensions between sexes. The positioning of the longitudinal veins is virtually identical in both sexes, even though size differs significantly. In general, the female wing is simply a scaled-up version of the male wing, *i.e.*, the sexes differ in size but not in pattern formation within the wing.

Table 2 gives phenotypic variance and repeatability (R) for each wing trait after scaling and log-transformation. Repeatability measures the magnitude of the variance between individuals relative to the total variance. In the context of these data, the amount by which R deviates from unity is the proportion of variance attributable to measurement error and developmental asymmetry. For females, R ranges from 0.74 (for trait EI) to 0.94 (for trait GI). In males, R varies from 0.69 (for EI) to 0.93 (for traits AE and GI). Average repeatability

TABLE 2

Phenotypic variance (σ_P^2) for log-transformed, scaled data and repeatability (R) between left and right wings

Trait	Males		Females	
	σ_P^2	R	σ_P^2	R
Wing length				
AE	1.061 ± 0.028	0.93 ± 0.004	0.934 ± 0.024	0.93 ± 0.004
GI	1.116 ± 0.029	0.93 ± 0.004	1.007 ± 0.026	0.94 ± 0.003
GL	1.451 ± 0.038	0.91 ± 0.004	1.303 ± 0.034	0.91 ± 0.004
Wing width				
MN	0.866 ± 0.023	0.90 ± 0.005	0.770 ± 0.020	0.88 ± 0.006
CL	0.880 ± 0.023	0.89 ± 0.005	0.810 ± 0.021	0.89 ± 0.005
Between vein ends				
AC	1.228 ± 0.032	0.90 ± 0.005	1.012 ± 0.026	0.89 ± 0.005
CE	0.930 ± 0.024	0.85 ± 0.007	0.916 ± 0.024	0.88 ± 0.006
CI	0.973 ± 0.025	0.90 ± 0.005	0.961 ± 0.025	0.91 ± 0.004
EI	0.639 ± 0.017	0.69 ± 0.014	0.754 ± 0.019	0.74 ± 0.012
EL	0.809 ± 0.021	0.81 ± 0.009	0.837 ± 0.022	0.81 ± 0.009
IL	1.118 ± 0.029	0.79 ± 0.010	1.234 ± 0.032	0.79 ± 0.010
Crossveins				
FD	1.798 ± 0.047	0.80 ± 0.009	1.708 ± 0.044	0.76 ± 0.011
HB	1.634 ± 0.042	0.80 ± 0.009	1.593 ± 0.041	0.79 ± 0.010

by character grouping is (male, female): wing length 0.92, 0.93; wing width 0.90, 0.89; distances between vein ends 0.82, 0.84; crossvein traits 0.80, 0.78.

The product-moment correlation across traits for repeatability of a trait and the trait mean is 0.88 for males and 0.90 for females, indicating that the greater the distance between landmarks, the more accurately the trait is measured. This is to be expected when an ocular micrometer is used to measure small distances.

Design (observational) variance components from the nested analysis of variance are expressed in Table 3 as a percentage of phenotypic variance. The sire variance component (σ_S^2) for all wing traits is substantial, indicating the presence of additive genetic variance. The percentages for σ_S^2 are larger for females than for males for wing length traits AE, GI and GL and also for traits AC, CE, CI, EI, EL and HB. The inference consistent with the genetic model is that X-linked variance causes σ_S^2 for females to be larger than for males. This is because the covariance of half-sisters includes one-half of the X-linked variance, whereas the covariance of half-brothers includes only one-quarter of the autosomal variance (Table A2, APPENDIX).

For wing width (MN and CL) and traits IL and FD, the percentages for σ_S^2 are similar between sexes. This may indicate that X-linked variance is a minor portion of phenotypic variance for these traits. An alternative explanation is

TABLE 3

Design (observational) variance components as a percentage of phenotypic variance for log-transformed, scaled data

Trait	Sex	σ^2_{Exp}	σ^2_S	σ^2_D	σ^2_W	σ^2_N
Wing length						
AE	M	4	7	32	50	7
	F	7	14	31	41	7
GI	M	4	7	32	50	7
	F	7	13	29	44	7
GL	M	10	7	28	46	9
	F	10	11	29	41	9
Wing width						
MN	M	8	13	24	45	10
	F	12	15	26	35	12
CL	M	6	9	28	46	11
	F	10	10	29	40	11
Between vein ends						
AC	M	7	11	27	45	10
	F	6	18	27	38	11
CE	M	4	8	29	44	15
	F	11	13	24	39	13
CI	M	4	9	31	46	10
	F	12	13	26	40	9
EI	M	6	10	28	25	31
	F	9	14	24	27	26
EL	M	6	11	23	41	19
	F	13	15	24	30	18
IL	M	14	17	12	36	21
	F	17	17	19	26	21
Crossveins						
FD	M	9	14	22	35	20
	F	10	14	22	30	24
HB	M	10	11	27	33	19
	F	13	17	19	30	21

that there is *X*-linked variance and that the autosomal variance is not equal between sexes.

For all wing traits except trait IL, the covariance of full-sibs σ^2_D is a much greater percentage of phenotypic variance than the covariance of half-sibs. For males this indicates that some combination of common environmental variance, nonadditive genetic variance and *X*-linked variance contributes to phenotypic variance. For females it indicates the presence of some combination of common environmental effects and nonadditive genetic effects.

In Table 4, estimates of causal variance components (equation 1) are given as a percentage of phenotypic variance. The percentages for σ^2_{Exp} and σ^2_N are the same as given in Table 3 and are not included in Table 4. Several features of these results are noteworthy. First, estimates of causal variance components

TABLE 4

Iterated weighted least squares (WLS) estimates of causal variance components as a percentage of phenotypic variance

Trait	Sex	Causal Components			
		σ^2_{Aa}	σ^2_{Ax}	σ^2_C	σ^2_e
Wing length					
AE	M	23	30	15	22
	F	26	17	17	25
GI	M	23	25	16	25
	F	25	15	18	28
GL	M	25	18	16	23
	F	27	11	17	26
Wing width					
MN	M	42	23	7	10
	F	47	10	8	11
CL	M	30	15	16	22
	F	33	5	17	24
Between vein ends					
AC	M	38	26	8	11
	F	46	14	10	13
CE	M	34	16	13	20
	F	34	9	13	20
CI	M	35	15	15	21
	F	36	7	15	21
EI	M	42	0	19	8
	F	36	7	16	7
EL	M	36	20	9	11
	F	35	14	9	11
IL	M	59	9	0	0
	F	54	11	0	0
Crossveins					
FD	M	52	11	6	2
	F	55	2	6	2
HB	M	46	14	7	4
	F	48	8	7	4

are very uniform for the three wing-length traits (AE, GI and GL). Second, the percentage of *X*-linked variance in males is nearly double the percentage of *X*-linked variance of females for traits AE, GI, GL, MN, CL, AC, CE, CI and HB. This is consistent with the hypothesis that the mechanism of dosage compensation in Drosophila will cause male *X*-linked variance to be twice the *X*-linked variance of females.

Several different estimates of narrow-sense heritability (h^2) are given by sex, with approximate standard errors, in Table 5. The heritability estimates derived from half-sib intraclass correlations (subscript *HS*) are strongly sexually dimorphic for all traits except CL, IL and FD. For wing length, h^2_{HS} averages

TABLE 5

Narrow-sense heritability of wing traits

Trait	Males		Females		Difference
	h^2_{HS}	h^2_{LS}	h^2_{HS}	h^2_{LS}	h^2_{HS}
Wing length					
AE	29 ± 8	52 ± 11	57 ± 7	43 ± 12	4 ± 5
GI	26 ± 8	47 ± 6	53 ± 8	40 ± 7	7 ± 6
GL	28 ± 8	43 ± 7	46 ± 8	38 ± 7	5 ± 5
Wing width					
MN	50 ± 8	66 ± 14	62 ± 7	57 ± 15	7 ± 6
CL	35 ± 8	45 ± 9	39 ± 8	38 ± 9	5 ± 5
Between vein ends					
AC	44 ± 8	64 ± 10	70 ± 6	60 ± 12	4 ± 5
CE	34 ± 8	49 ± 1	53 ± 8	44 ± 1	9 ± 6
CI	35 ± 8	50 ± 1	50 ± 8	43 ± 1	4 ± 5
EI	39 ± 8	37 ± 10	54 ± 8	43 ± 8	6 ± 6
EL	44 ± 8	56 ± 14	59 ± 7	49 ± 14	6 ± 6
IL	66 ± 7	68 ± 18	70 ± 6	65 ± 16	12 ± 6
Crossveins					
FD	56 ± 8	63 ± 7	56 ± 8	57 ± 7	5 ± 5
HB	43 ± 8	60 ± 7	67 ± 6	56 ± 6	4 ± 5

h^2_{HS} estimated from the intraclass correlation of half-sibs; h^2_{LS} calculated from iterated WLS estimates of causal variance components; heritability of the difference between sexes is twice the intraclass correlation of half-sibs.

28% for males and 52% for females. As noted previously, when there is *X*-linked variance h^2_{HS} underestimates heritability for males and overestimates heritability for females, and *X*-linked variance is a likely cause of the sexually dimorphic values for h^2_{HS}.

For comparison, heritability estimated from least squares estimates of causal variance components (subscript *LS*) are also given in Table 5. These estimates of heritability differ from h^2_{HS} for most of the wing traits in that the sexual dimorphism is reversed, *i.e.*, h^2_{LS} is larger for males. This is to be expected if males and females have common autosomal variance but males have larger *X*-linked variance.

Table 5 also gives an approximate estimate of heritability for the difference between sexes, which is calculated as two times the intraclass correlation of half-sibs (see APPENDIX). These values suggest that the heritability of sexual dimorphism is low but that some autosomal additive genetic variance is present for the difference between sexes.

The hypothesis that the genes affecting wing traits are randomly distributed throughout the genome may be considered by examining the proportion σ^2_{Ax}/σ^2_A (Table 6). As previously noted, if gene effects are small and randomly distributed and dosage compensation is complete in males, then 33–38% of

TABLE 6

Additive *X*-linked variance divided by total additive variance and the relative magnitude of male *X*-linked variance to female *X*-linked variance

Trait	σ^2_{Ax}/σ^2_A		$\sigma^2_{Axm}/\sigma^2_{Axf}$
	Males	Females	
Wing length			
AE	57	40	1.94
GI	52	37	1.82
GL	42	28	1.85
Wing width			
MN	36	17	2.74
CL	33	13	3.42
Between vein ends			
AC	41	23	2.28
CE	31	22	1.67
CI	30	17	2.12
EI	0	16	0
EL	35	29	1.36
IL	13	17	0.71
Crossveins			
FD	17	3	5.93
HB	23	15	1.66

the total additive genetic variance in males and 20–23% in females will be *X*-linked variance.

For wing length (AE, GI and GL), *X*-linked variance is 50% of total additive genetic variance for males and 35% for females. For wing width, the corresponding percentages are 35% for males and 15% for females. The percentages for traits AC, CE, CI and EL are 34% for males and 23% for females. The remaining traits, EI, IL, FD and HB, average 13% for both sexes.

Thus it appears that for wing width (MN and CL) and traits AC, CE, CI and EL that polygenic effects are randomly distributed between the autosomes and the *X* chromosome. In contrast, wing length appears to have a greater percentage of *X*-linked variance than expected if polygenic effects are randomly distributed. For traits EI, IL, FD and HB the percentages are much less than expected for randomly distributed gene effect. These results are, of course, predicated on gene effects being small and equal.

The ratio of male to female *X*-linked variance provides an estimate of the level of dosage compensation in males (Table 6). In a polygenic sense, dosage compensation seems to be complete, or nearly so, for all wing traits except CL, EI, EL, IL and FD.

Genetic correlations between sexes for each trait are given in Table 7. The half-sib correlations (subscript *HS*) show low to moderate deviation from unity, whereas the full-sib correlations (*FS*) are close to 1.0 for most traits. If there is *X*-linked variance for a trait in females, then r_{HS} will underestimate the

TABLE 7

Estimates of the genetic correlation between males and females for the same trait

Trait	r_{HS}	r_{FS}	b_{Aa}	r_A	r_{Ax}
Wing length					
AE	0.92	0.93	1.20	0.93	0.32
GI	0.82	0.99	1.10	0.97	0.62
GL	0.87	1.00	1.05	0.99	0.61
Wing width					
MN	0.86	0.94	0.90	0.91	0.51
CL	0.83	0.98	0.83	0.91	0.84
Between vein ends					
AC	0.93	0.92	1.07	0.93	0.32
CE	0.77	0.96	0.96	0.88	0.68
CI	0.89	0.86	1.06	0.81	0.01
EI	0.87	0.79	1.11	0.68	[a]
EL	0.86	0.96	1.01	0.94	0.35
IL	0.79	1.19	0.85	1.00	1.29
Crossveins					
FD	0.89	0.80	0.87	0.81	−0.34
HB	0.95	0.91	1.16	0.93	0.25

b = genetic regression coefficient of female on male autosomal effects; design (observational) components were used to obtain half-sib (*HS*) and full-sib (*FS*) correlations and b; iterated WLS estimates of causal components were used to obtain total additive (A) and X-linked (Ax) correlations. Subscript Aa denotes additive autosomal.

[a] Could not be calculated.

autosomal genetic correlation. Similarly, r_{FS} may include nongenetic common environmental effects in addition to epistatic effects and, thus, may overestimate the total additive genetic correlations.

For comparison, total additive genetic correlations between sexes (r_A) are included in Table 7. Except for traits CI, EI, FD and HB, values of r_A are greater than or equal to the half-sib correlations r_{HS}. This supports the hypothesis that there is *X*-linked variance influencing wing traits of Drosophila.

Correlations between males and females for *X*-linked effects, r_{Ax}, are in general much less than unity. This may indicate genotype × sex interaction for *X*-linked loci. A genotype × sex interaction cannot be more specifically elucidated with these results, because it is impossible with these data to separate differences in dosage compensation from effects of loci that are sex-limited.

DISCUSSION

It is clear from the results that male and female variances for wing dimensions differ, and this difference persists after scaling the data so that males and females have the same mean. An important point to be made is that estimates of heritability based on half-sib intraclass correlations may be inappropriate for predicting response to selection when there is significant variance attributable to the *X* chromosome. However, it is also important to note that this conclusion is predicated on the mechanism of dosage compensation of the *X* chromosome

in Drosophila and on the *X* chromosome being a major portion of the entire genome. In mammals for instance, when dosage compensation of the *X* chromosome is through chromosome inactivation in the female and the *X* chromosome is relatively small, one would expect *X*-linked variance to be a minor portion of genetic variation, and the mechanism of dosage compensation would not contribute to sexually dimorphic variances.

The least squares estimates of *X*-linked variances for each sex support the hypothesis that dosage compensation in males will inflate the *X*-linked variance for that sex. The ratio of male to female *X*-linked variance supports the hypothesis that dosage compensation in a polygenic sense is complete or nearly so for most wing traits examined. The exceptions to this qualitative judgment include traits for which repeatability was also low, and the results for those traits are subject to greater error in interpretation.

Estimates of narrow-sense heritability estimated from the intraclass correlations of half-sibs differ significantly between sexes for many wing traits. These values lead to divergent predicted responses to selection and imply that selection in the same direction in both sexes would increase the magnitude of sexual dimorphism. When data are fit to a genetic model including *X*-linked variance parameters, the heritabilities (h^2_{LS}) are closer in magnitude between sexes with male heritability higher than heritability for females, and the sex differences in h^2_{LS} are probably not significant.

The values of h^2_{LS} for wing length are in good agreement with realized heritability reported for previous selection studies on wing length of Drosophila. REEVE and ROBERTSON (1953) reported heritability of wing length (trait AE in this paper), estimated by regression of offspring on mid-parent size, to be about 0.2–0.3, whereas realized heritability was about 0.5. ROBERTSON (1960) reported realized heritabilities for selection on large and small cell size in the wing. Realized heritabilities were 0.58 for large cell size and 0.44 for small cell size. Realized heritability of wing area was reported to be 0.56 (ROBERTSON 1962).

Total additive genetic correlations between sexes for the same trait are high for most traits and probably do not deviate significantly from unity. This indicates that correlated response in one sex to selection on the other sex would be reciprocally equal, assuming equal selection intensities in each sex. Thus, any divergence in total response to selection would be due to real differences in the narrow sense heritability.

BIRD and SCHAFFER (1972) carried out a selection experiment to change the magnitude of sex dimorphism in wing length in Drosophila. They found that selection to decrease sex dimorphism decreased female wing length, whereas selection to increase sex dimorphism decreased male wing length. These authors interpreted these results as selection acting on the level of dosage compensation in males, since chromosomal substitutions showed a significant effect on sex dimorphism in this trait was attributable to the *X* chromosome.

The *X*-linked correlations between sexes for the same trait do not yield definitive information regarding dosage compensation and other genotype × sex interactions. If male and female genic effects are the same sign (*e.g.*, pos-

itive), then a single *X*-linked locus with two alleles in the Drosophila system has a correlation between male and female genic effects of 0.71, regardless of the assumed level of dosage compensation. The covariance between sexes for an *X*-linked locus is (JAMES 1973)

$$\sigma_{Ax(m,f)} = (\sigma_{Axm}\sigma_{Axf})/\sqrt{2}, \quad (6)$$

and this result holds for all genic values as long as they are the same sign for each sex. JAMES further states that the result holds even if there is *X*-linked dominance in females. However, the relation is not necessarily true if there are more than two alleles or if epistasis occurs between loci. The condition required for the relation to be true for multiple alleles and loci is that the ratio of female to male genic effects must be constant for all alleles and loci (JAMES 1973).

If approximately equal ratios of female to male genic effects are assumed for all alleles and loci, and no epistasis, then the maximum *X*-linked genetic correlation should be near 0.71 (*i.e.*, $1/\sqrt{2}$), and the value of the *X*-linked correlation should be unaffected by the level of dosage compensation.

These results concern the possible further evolution of sexual dimorphism if there is selection, but perhaps a more interesting biological question these data cannot address is: How does sexual dimorphism arise in a highly integrated functional unit, such as the insect wing? Initially, the imaginal disc of the putative Drosophila wing is composed of approximately 38 cells (MADHAVAN and SCHNEIDERMAN 1977). Mitosis in wing disc cells begins 15–17 hr after hatching and persists into the pupal instar. Whether sex differences occur in these initial components of early development is unknown. However, it is possible that differences between sexes could occur in the number of cells initially incorporated into the wing disc, the time of onset and termination of mitosis, or the rate of cell division. Although larger as adults, females emerge as adult flies about 4 hr earlier than males (BAINBRIDGE and BOWNES 1981), suggesting a differential rate of development.

We are grateful to WILLIAM ENGELS for providing the flies used in this experiment and to WENDY BENZ for her technical assistance on the project. We have benefited from theoretical discussions with JAMES F. CROW and EUGENE EISEN. BRUCE RISKA, SCOT NEWMAN and MARGARET DENTINE provided helpful comments on various drafts of this manuscript. Two anonymous reviewers also provided comments that were very helpful. This research was supported by the College of Agriculture and Life Sciences, University of Wisconson-Madison; the National Science Foundation (DEB-7906058); and a Hatch grant (2783) to W.R.A.

LITERATURE CITED

ATCHLEY, W. R., 1971 Components of sexual dimorphism in *Chironomus* larvae (Diptera: Chironomidae). Am. Nat. **105:** 455–466.

BAINBRIDGE, T. R., 1963 Staggered, nested designs for estimating variance components. pp. 93–103. ASQC Annual Conference Transactions.

BAINBRIDGE, S. P. and M. BOWNES, 1981 Staging the metamorphosis of *Drosophila melanogaster*. J. Embryol. Exp. Morphol. **66:** 57–80.

BAKER, B. S. and J. M. BELOTE, 1983 Sex determination and dosage compensation in *Drosophila melanogaster*. Annu. Rev. Genet. **17:** 345–393.

BIRD, M. A. and H. E. SCHAFFER, 1972 A study of the genetic basis of sexual dimorphism for wing length in *Drosophila melanogaster*. Genetics **72:** 475–487.

BOHIDAR, N. R., 1964 Derivation and estimation of variance and covariance components associated with covariance between relatives under sex-linked transmission. Biometrics **20:** 505–521.

BRYANT, P., 1978 Pattern formation in imaginal discs. pp. 230–336. In *The Genetics and Biology of Drosophila*, Vol. 2C, Edited by M. ASHBURNER and T. R. F. WRIGHT. Academic Press, New York.

DRAPER, N. R. and N. M. SMITH, 1981 Applied regression analysis. John Wiley & Sons, New York.

EISEN, E. J. and J. P. HANRAHAN, 1972 Selection for sexual dimorphism in body weight of mice. Aust. J. Biol. Sci. **25:** 1015–1024.

EISEN, E. J. and J. E. LEGATES, 1966 Genotype-sex interaction and the genetic correlation between the sexes for body weight in *Mus musculus*. Genetics **54:** 611–623.

FALCONER, D. S., 1981 Introduction to quantitative genetics. Longman, New York.

FALCONER, D. S. and J. W. B. KING, 1953 A study of selection limits in the mouse. J. Genet. **51:** 561–581.

FRANKHAM, R., 1968a Sex and selection for a quantitative character in Drosophila. I. Single-sex selection. Aust. J. Biol. Sci. **21:** 1215–1223.

FRANKHAM, R., 1968b Sex and selection for a quantitative character in Drosophila. II. The sex dimorphism. Aust. J. Biol. Sci. **21:** 1225–1237.

FRANKHAM, R., 1977 The nature of quantitative genetic variation in Drosophila. III. Mechanism of dosage compensation for sex-linked abdominal bristle polygenes. Genetics **85:** 185–191.

GRIFFING, B., 1965 Influence of sex on selection. I. Contribution of sex-linked genes. Aust. J. Biol. Sci. **18:** 1157–1170.

JAMES, J. W., 1973 Covariance between relatives due to sex-linked genes. Biometrics **29:** 584–588.

LINDSLEY, D. L. and E. H. GRELL, 1967 Genetic variations of *Drosophila melanogaster*. Carnegie Inst. Wash. Publ. 627.

MADHAVAN, M. V. and H. A. SCHNEIDERMAN, 1977 Histological analysis of growth of imaginal discs and histoblast nests during the larval development of *Drosophila melanogaster*. Wilhelm Roux' Arch. Dev. Biol. **183:** 269–305.

MARTIN, P. F., 1982 Direct determination of the growth rate of *Drosophila* imaginal discs. J. Exp. Zool. **222:** 92–102.

REEVE, E. C. R. and F. W. ROBERTSON, 1953 Studies in quantitative inheritance. II. Analysis of a strain of *Drosophila melanogaster* selected for long wings. J. Genet. **51:** 276–316.

ROBERTSON, A., 1959 The sampling variance of the genetic correlation coefficient. Biometrics **15:** 469–485.

ROBERTSON, F. W., 1960 Studies in quantitative inheritance. XII. Cell size and number in relation to genetic and environmental variation of body size in *Drosophila*. Genetics **44:** 869–896.

ROBERTSON, F. W., 1962 Changing the relative size of the body parts of *Drosophila* by selection. Genet. Res. **3:** 169–180.

SCHAFFER, H. E. and K. KOJIMA, 1963 Sex-linked inheritance for a quantitative trait in *Drosophila pseudoobscura*. Genetics **48:** 775–786.

SHERIDAN, A. K., R. FRANKHAM, L. P. JONES, K. A. RATHIE and J. S. F. BARKER, 1968 Partitioning of variance and estimation of genetic parameters for various bristle characters of *Drosophila melanogaster*. Theor. Appl. Genet. **38:** 179–187.

WRIGHT, S., 1968 Evolution and the genetics of populations. In: *Genetic and Biometric Foundations*, Vol. 1. The University of Chicago Press, Chicago.

Communicating editor: B. S. WEIR

APPENDIX

The least squares solution for causal variance and covariance components is

$$\mathbf{b} = (\mathbf{X}'\mathbf{X})^{-1}\mathbf{X}\mathbf{y}, \tag{A.1}$$

where **b** is the vector of causal variance components, **X** is a design matrix with rows that express the expectations of mean squares and mean products in terms of causal components, and **y** is a vector of mean squares.

In solving for **b**, the expectations in **X** excluded σ^2_N and $\sigma_{Nm_jf'}$ the design variance and covariance components between left and right wings. The mean squares and mean products in **y** were "corrected" by subtracting $\hat{\sigma}^2_N$ from mean squares and $\hat{\sigma}^2_{Nm_jf}$ from mean products. Nine design components were used to obtain solutions for eight causal components. The design components used were sire, dam and within dams for each sex individually and the same three components for the covariance between sexes.

The initial least squares estimates are refined by iterative weighted least squares. The weighed least squares solution is

$$\mathbf{b} = (\mathbf{X}'\mathbf{V}^{-1}\mathbf{X})^{-1}\mathbf{X}'\mathbf{V}^{-1}\mathbf{y}, \tag{A.2}$$

where **b**, **X** and **y** are described above, and **V** is a diagonal matrix expressing the variance of the mean squares and mean products in **y**, *i.e.*,

$$V(MS) = 2[E(MS)]^2/\text{d.f.} \tag{A.3}$$

The values in **V** were obtained by using the estimated vector **b**. Values of **b** stabilized after two to four iterations.

Standard errors of the causal variance and covariance components were taken as the square root of the appropriate diagonal element of

$$V(\mathbf{b}) = s^2(\mathbf{X}'\mathbf{V}^{-1}\mathbf{X})^{-1}, \tag{A.4}$$

where

$$s^2 = (Y - \hat{Y})^2 \text{ with one degree of freedom.} \tag{A.5}$$

The estimated $\hat{Y}$ values followed DRAPER and SMITH (1981).

The difference between sexes was evaluated by randomly pairing full brothers with full sisters and obtaining the difference between them. These data were then transformed identically to the data for sexes individually. The estimate of heritability for the difference between sexes was obtained by multiplying the intraclass correlation between sexes by two. Expectations for the difference between sexes (Table A2) were derived by the formula for the variance of a difference.

TABLE A1

Expectations of mean squares of the Bainbridge nested ANOVA for paternal half-sib mating design

Source	d.f.	Expected mean squares
Replicates	13	$\sigma^2_N + 2\ \sigma^2_W + 10/3\ \sigma^2_D + 6\ \sigma^2_S + 210.6\ \sigma^2_{Exp}$
Sires/replicates	480	$\sigma^2_N + 2\ \sigma^2_W + 10/3\ \sigma^2_D + 6\ \sigma^2_S$
Dams/sires	494	$\sigma^2_N + 2\ \sigma^2_W + 8/3\ \sigma^2_D$
Flies/dams	494	$\sigma^2_N + 2\ \sigma^2_W$
Wings/flies	1481	σ^2_N

All effects are assumed random.

TABLE A2

Expectations of design (observational) variance components in terms of causal variance components

	Causal components							
	$\sigma^2_{\lambda a}$	$\sigma^2_{\lambda xm}$	$\sigma^2_{\lambda xf}$	σ^2_c	σ^2_e	$\sigma_{\lambda amf}$	$\sigma_{\lambda xmf}$	σ_{emf}
Males								
σ^2_S	1/4							
σ^2_D	1/4	1/2		1				
σ^2_W	1/2	1/2			1			
Females								
σ^2_S	1/4		1/2					
σ^2_D	1/4		1/4	1				
σ^2_W	1/2		1/4		1			
Difference between sexes								
σ^2_S	1/2		1/2			−1/2		
σ^2_D	1/2	1/2	1/4			−1/2	−1	
σ^2_W	1	1/2	1/4		2			−2
Cov(male, female)								
σ_S						1/4		
σ_D				1		1/4	1/2	
σ_W								1