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Partitioning of Variance and Estimation of Genetic Parameters for Various Bristle Number Characters of *Drosophila melanogaster*

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Summary. Phenotypic variance for each of several bristle number characters (abdominal, sternopleural, second and third coxal) was partitioned using both hierarchal and diallel designs. Heritabilities and genetic correlations were estimated from parent-offspring regressions and correlations and half-sib correlations.

A high proportion of the genetic variance for abdominal bristle number was due to epistatic and sex-linked gene action, but most of the genetic variance for the other characters was additive autosomal.

The genetic correlations among sternopleural, and second and third coxal bristle numbers were all high, but that between abdominals and sternopleurals was low, while those between abdominals and either second or third coxals were virtually zero. An appreciable proportion of the covariance between abdominal and sternopleural bristle numbers was non-additive genetic.

The diallel method gave more reliable estimates of genetic parameters when non-additive or sex-linked genetic variation was present.

I. Introduction

In terms of present quantitative genetic theory, prediction of short-term selection responses and interpretation of responses actually observed depend on estimates of the components of phenotypic variance and genetic parameters in the base population. Although several methods are available for estimating genetic parameters (LUSH, 1949; FALCONER, 1960), and although many estimates have been reported for various characters in a number of species (*e. g.* SPECTOR, 1956; FALCONER, 1960), few detailed comparisons of estimates from several methods have been carried out.

DAWSON (1965) compared six experimental designs for partitioning phenotypic variance and estimating heritability for developmental rate in *Tribolium*. His comparisons were particularly relevant to the question of estimating genetic parameters in the presence of maternal effects, but ignored sex-linkage and took little account of epistasis. KEARSEY (1965) compared five experimental designs for estimating components of variance and heritability for flowering time in *Papaver*, using approximately the same number of families for each design. His results provide useful comparisons between methods on the basis of information yielded for the same amount of work, but the designs and comparisons used were of more relevance to plant breeding than animal breeding. SCHAFFER and KOJIMA (1963) compared three methods of estimating sex-linked effects for wing length in *Drosophila*. Unfortunately, a completely satisfactory comparison was not obtained as they did not measure progeny of both sexes for the diallel cross.

As a preliminary to various studies of artificial selection for bristle number characters in *Drosophila melanogaster*, phenotypic variances for several bristle characters were partitioned and relevant genetic parameters estimated using a number of different methods on the data of two experiments, one an hierarchal design and the second a diallel design. Both parent-offspring regression and half-sib correlation estimates of heritabilities and genetic correlations were obtained from each experiment. The partitioning of both phenotypic variance and genetic covariance has been carried out, and the designs and methods compared for their ability to either estimate epistatic and sex-linked effects, or provide unbiased heritability estimates when epistatic and sex-linked effects are present.

II. Materials and Methods

The Canberra strain of *Drosophila melanogaster* (LATTER, 1964) was used. This stock originated from more than 100 inseminated females captured at Canberra in the summer of 1959. Subsequently it was maintained as a large laboratory stock (approximately 500 pairs per generation) by Dr. B. D. H. LATTER. Since March, 1964, a sample of this stock has been maintained in our laboratory as a cage population with an average size of 4,000 adults.

The characters scored were the number of bristles on the fourth, fifth and sixth abdominal sternites, the right sternopleural plate, and the right second and third coxa. The means and standard deviations

Table 1. *Means and standard deviations of the characters in Experiment 2*

Character	Mean	Standard deviation
Male		
Fourth abdominal	18.16	1.76
Fifth abdominal	18.54	1.76
Total abdominal (4th + 5th)	36.70	2.86
Sternopleural	9.59	1.08
Second coxal	11.37	0.85
Third coxal	7.72	0.78
Female		
Fourth abdominal	22.45	2.04
Fifth abdominal	22.70	2.12
Total abdominal (4th + 5th)	45.15	3.46
Sternopleural	9.74	1.04
Second coxal	11.39	0.83
Third coxal	7.92	0.81

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of these characters (except for sixth abdominal) from the Experiment 2 data are shown in Table 1. Parents and offspring were cultured under uncrowded conditions on a dead yeast fortified medium (medium F of CLARINGBOLD and BARKER, 1961) in 3 × 1 inch glass vials at 25 ± 0.5 °C and 65–70% relative humidity.

(*i*) *Experiment 1 — hierarchal design*

This design consisted of 62 sires, three dams per sire and 10 male and 10 female offspring per dam. Each of the 62 sires was mated to several females. The parents were scored and the females allowed to lay eggs in separate vials for three days. Random samples of progeny were scored from each of three dam families per sire. Males were scored for sternopleural and fourth and fifth abdominal bristle number, while females were scored for sternopleural and fifth and sixth abdominal bristle number.

(*ii*) *Experiment 2 — diallel design*

This experiment consisted of 127 two sire × two dam diallel units as described by LERNER (1950). Males 1 and 2 were mated to females 1 and 2 respectively. After two days, the parents were transferred to fresh vials for a further two days. Parents were then removed and male 1 stored with female 2 and male 2 with female 1 for seven days. SHELDON (1963) indicated this time was sufficient to ensure that the female would be fertile to the second male. Each female was then allowed to lay eggs in fresh vials during two consecutive two-day periods. From each of the two vials per sire × dam combination, four pairs of flies were scored for fourth and fifth abdominal, sternopleural, and second and third coxal bristle numbers.

The characters scored in the two experiments differed slightly. In Experiment 1, the fifth and sixth abdominal sternites were scored in females, while in Experiment 2 the fourth and fifth were scored. This is unlikely to be of any consequence since the genetic correlation between abdominal sternites is not significantly different from unity (Table 6). Second and third coxals were scored only in Experiment 2.

In both experiments heritabilities and genetic correlations were estimated from variance and covariance components and from parent-offspring regressions or correlations. Genetic parameters were calculated using the standard formulae of FALCONER (1960) and BECKER (1964). Sire-daughter and dam-son regression estimates of heritability and genetic correlation were corrected for inequality of male and female variances as suggested by CLAYTON, MORRIS and ROBERTSON (1957). Standard errors of genetic correlation from parent-offspring regression and half-sib correlation were estimated by the methods of REEVE (1955) and TALLIS (1959).

Genetic expectations of the components of variance and covariance for the various relationships are given in Table 2. These are taken from BOHIDAR (1964), but have been simplified by ignoring genes with different effects in the two sexes as well as non-additive sex-linked and additive × sex-linked effects.

In Experiment 1 the dam component of variance includes the sire × dam interaction. The diallel design in Experiment 2 enabled this interaction to be estimated. Thus in Experiment 2 the maternal as well as the paternal half-sib estimate was free of dominance effects, being biased only by sex-linked and additive × additive interaction terms. Common environment effects were estimated in the diallel analysis as replicate vials were used for each mating.

Table 2. *Genetic expectations for heritability and variance components estimates*

Estimate	Genetic component*			
	V_A	V_{AA}	V_{As}	V_D
Heritability				
Sire-son	1	1/2		
Sire-daughter	1	1/2	1	
Dam-son	1	1/2	1	
Dam-daughter	1	1/2	1	
Paternal half brother	1	1/4		
Paternal half sister	1	1/4	2	
Maternal half brother	1	1/4	2	
Maternal half sister	1	1/4	1	
Dams within sires for male offspring	1	3/4	2	1
Dams within sires for female offspring	1	3/4	1	1
Variance components				
Sire × Dam component for male offspring		1/8		1/4
Sire × Dam component for female offspring		1/8		1/4

* V_A is the additive autosomal variance,
V_{AA} the additive × additive autosomal variance (*i.e.* 2 locus interactions),
V_{As} the additive sex-linked variance, and
V_D the autosomal dominance variance.

III. Results

(*i*) *Abdominal bristle number*

Analyses of variance, variance components, and the sire and dam component estimates of heritability for the sum of the number of bristles on two abdominal segments are presented in Tables 3 and 4 for Experiments 1 and 2 respectively. Both genetic and phenotypic variances were higher in Experiment 1, but there is no obvious explanation for this. Parent-offspring regression estimates of heritability are given in Table 5.

In Experiment 1, heritability estimates for males were much higher from the dam component than from the sire component. The former contains both more non-additive genetic and sex-linkage variance than the sire component (BOHIDAR, 1964), as well as any common environmental effects. These effects were not separable in the hierarchal analysis. Both sire and dam component estimates for females were much higher than the sire estimate for males. The dam component estimate is again biased by sex-linked and non-additive genetic and common environment effects, while the sire component estimate for females (but not for males) contains sex-linkage.

Parent-offspring regression estimates of heritability in Experiment 1 also varied considerably, but had high sampling errors and did not differ significantly. The average estimate was of similar magnitude to the half-sib estimate for males from the hierarchal analysis. These regression estimates did not vary so widely in Experiment 2, but the average estimate was twice that of the paternal half-sib estimate for males from the diallel analysis.

The between vials effect was significant for females in Experiment 2, but variation due to common en-

vironment contributed only approximately 1.7% and 2.8% of the variance for males and females respectively. The dam component heritability estimate from the hierarchal design includes four times the common environmental variance (FALCONER, 1960), and so could have been biased upward appreciably. The sire × dam interaction was highly significant in the diallel analysis (Experiment 2), the interaction component being nearly half the dam component in each sex. This interaction component contains $^1/_4$ of the dominance variance and $^1/_8$ of the additive × × additive epistasis as well as various portions of other interactions. Depending on the content of this component, it may be taken to indicate that dominance represents 18% of the phenotypic variance, or that additive × additive epistasis represents 36% of the phenotypic variance. The high dam component heritability estimates from the hierarchal analysis (Experiment 1) were not unexpected in the light of this appreciable non-additive genetic component. The high sire component estimate of heritability in females may have been due either to important sex-linked effects or to a higher heritability in females than in males.

Table 3. *Analyses of variance, variance components and estimates of heritability for total abdominal bristle number in Experiment 1*

Source of variation	d. f.	Males		Females	
		Mean square	Variance component	Mean square	Variance component
Between sires	61	58.14**	0.824	99.28**	2.295
Dams within sires	124	33.40**	2.523	30.43**	2.111
Within dams	1674	8.17		9.32	
h^2_S (from sires term)		0.29 ± 0.13		0.67 ± 0.18	
h^2_D (from dams term)		0.88 ± 0.15		0.62 ± 0.11	

** $P < 0.01$

Table 4. *Analyses of variance, variance components and estimates of heritability for total abdominal bristle number in Experiment 2*

Source of variation	d. f.	Males		Females	
		Mean square	Variance component	Mean square	Variance component
Sires	127	14.70**	0.319	30.06**	0.982
Dams	127	21.87**	0.767	32.07**	1.108
Sires × Dams	127	9.60**	0.342	14.34**	0.538
Between vials	508	6.86	0.136	10.04*	0.323
Within vials	3048	6.32		8.74	
h^2_S		0.16 ± 0.07		0.35 + 0.09	
h^2_D		0.40 ± 0.10		0.39 ± 0.09	
Average h^2				0.32	

* $P < 0.05$ ** $P < 0.01$

Table 5. *Parent-offspring regression estimates of heritability for total abdominal bristle number*

Estimate	Experiment 1	Experiment 2
Sire-son	0.22 ± 0.10	0.30 ± 0.04
Dam-son	0.28 ± 0.09	0.35 ± 0.06
Sire-daughter	0.40 ± 0.15	0.34 ± 0.05
Dam-daughter	0.21 ± 0.08	0.42 ± 0.06
Average	0.28	0.35

The importance of genes with different effects in the two sexes can be evaluated from a comparison of the heritability estimates from maternal half-brothers and paternal half-sisters. These have the same expectations if genes have the same effects in the two sexes, assuming maternal effects to be negligible. These estimates were quite similar, so the effects of genes in the two sexes were probably the same.

By solving the equations of the expectations for the different heritability estimates (from Table 2) against the estimates obtained, the size of the genetic components has been estimated. For any estimate, appreciable differences between Experiments 1 and 2 were present but standard errors were larger in Experiment 1. The sire component estimate for males was the lowest of the half-sib estimates in both experiments. These are free of sex-linked effects. Similarly, the sire-son regression estimate was lower than the other regression estimates in Experiment 2, while in Experiment 1 it was lower than all but the daughter-dam regression. This sire-son estimate contains more of the additive × additive autosomal epistasis than half-sib estimates but is free of sex-linkage effects.

It is difficult to partition the variation with much accuracy because of the large sampling errors. Sex-linkage can be estimated from the difference between the sire-son heritability and the other parent-offspring heritability estimates, and from the difference between the sire component heritability for males and other half-sib heritability estimates. About 9% of the variance was sex-linked, as estimated from these differences.

Additive and additive × additive autosomal (epistasis) variance can also be estimated from the various heritability estimates, as well as from the sire × dam interaction component. By substituting the estimate for sex-linked effects, and comparing all the estimates of parent-offspring and half-sib heritability, about 14% of the variance was estimated as additive and 25% epistatic. Most of the sire × dam interaction was apparently due to epistasis, as indicated by the large estimate for this component from the difference between the sire-son heritability and the sire component heritability for males. Consequently dominance effects were probably only minor.

If the genetic correlation between two segments is unity, the ratio of the variance of the difference in bristle numbers (σ^2_d) to the variance of the sum (σ^2_P) may be taken as a measure of developmental error (CLAYTON *et al.*, 1957). Estimates of genetic correlation between abdominal segments are presented in Table 6, and indicate that the genetic correlation can be taken as unity. The variance of the differences between the segments, and the proportion this is of

Table 6. *Genetic correlations between abdominal bristle segments*

Estimate	Males		Females	
	Experiment 1	Experiment 2	Experiment 1	Experiment 2
Analysis of variance and covariance				
Sire component	1.07 ± 0.06	0.92 ± 0.18	0.98 ± 0.04	1.16 ± 0.08
Dam component	0.95 ± 0.06	0.85 ± 0.11	1.02 ± 0.05	1.20 ± 0.08
Parent offspring regression	0.68 ± 0.20	0.95 ± 0.10	1.43 ± 0.37	0.90 ± 0.06
Average			1.02	

Table 7. *Variance of the difference between abdominal bristle segments* (σ_d^2) *and the proportion this is of the variance of their sum* (σ_d^2/σ_P^2)

	Males		Females	
	σ_d^2	σ_d^2/σ_P^2	σ_d^2	σ_d^2/σ_P^2
Experiment 1	3.95	0.34	5.32	0.39
Experiment 2	4.23	0.54	5.14	0.44

Table 8. *Analyses of variance, variance components and estimates of heritability for sternopleural bristle number in Experiment 1*

Source of variation	d. f.	Males		Females	
		Mean square	Variance component	Mean square	Variance component
Between sires	61	3.894**	0.0550	4.461**	0.0800
Dams within sires	124	2.198**	0.1073	2.061**	0.1168
Within dams	1674	1.125		0.893	
h_S^2		0.17 ± 0.08			0.29 ± 0.10
h_D^2		0.33 ± 0.09			0.43 ± 0.10
Average h^2				0.31	

** $P < 0.01$

Table 9. *Analyses of variance, variance components, and estimates of heritability for sternopleural bristle number in Experiment 2*

Source of variation	d. f.	Males		Females	
		Mean square	Variance component	Mean square	Variance component
Sires	127	1.8674**	0.0537	1.7517**	0.0378
Dams	127	2.2266**	0.0762	1.7763**	0.0394
Sires × Dams	127	1.0081	−0.0030	1.1464	0.0250
Between vials	508	1.0317	0.0028	0.9461	−0.0057
Within vials	3048	1.0206		0.9689	
h_S^2		0.19 ± 0.06			0.14 ± 0.06
h_D^2		0.27 ± 0.07			0.15 ± 0.06
Average h^2				0.19	

** $P < 0.01$

the variance of their sum are shown in Table 7. This proportion was quite high in both experiments. However, if the correlation between developmental errors of the two segments were negative, our estimate for developmental error would be too high, while that for true environmental would be too small.

The partitioning of variation for two segments can be summarised thus:

Additive autosomal	0.12−0.16
Additive sex-linked	0.08−0.10
Additive × additive autosomal	0.20−0.30
True environmental	0.00−0.05
Developmental error	0.34−0.54

If the two segments are taken as repeated measurements of the same character, the heritability of the sum is $\frac{n}{1+(n-1)r_P}$ times that for each segment. In this case $n=2$, and $r_P=0.36$ (see section (*v*)), so that approximately 10%, 6% and 18% of the phenotypic variance for one abdominal segment was due to additive autosomal, sex-linkage and epistatic effects respectively.

(*ii*) *Sternopleural bristle number*

The analyses of variance, variance components, and sire and dam component estimates of heritability for sternopleural bristle number are presented in Tables 8 and 9 for Experiments 1 and 2 respectively, and parent-offspring regression estimates in Table 10.

In Experiment 1, the sire component estimate of heritability for males was considerably less than the other estimates. This indicated that sex-linkage, non-additive genetic variance or common environment could be present. However, neither sire × dam interaction nor between vials mean squares were significantly greater than the within vials mean square in Experiment 2. The sire × dam interaction component of variance was about half of each of the sire and dam components in females and was effectively zero in males. Further, the half-sib estimates in Experiment 2 offer little indication of sex linkage. The parent-offspring estimates in both experiments gave little indication of sex-linkage and no estimates differed significantly from the mean. Non-additive genetic variance was apparently less important than for abdominal bristle number and sex-linkage was not indicated. Most of the genetic variation was therefore additive autosomal.

(*iii*) *Second and third coxal bristle number*

The analyses of variance, variance components, and sire and dam component estimates of heritability for

Table 10. *Parent-offspring regression estimates of heritability for sternopleural bristle number*

Estimate	Experiment 1	Experiment 2
Sire-son	0.16 ± 0.09	0.22 ± 0.04
Dam-son	0.18 ± 0.08	0.17 ± 0.04
Sire-daughter	0.18 ± 0.13	0.12 ± 0.04
Dam-daughter	0.26 ± 0.08	0.20 ± 0.04
Average	0.19	0.18

the number of bristles on the second and on the third coxa are shown in Tables 11 and 12 respectively, and parent-offspring regression estimates in Table 13.

For second coxals, parent-offspring heritability estimates (average 0.16) were slightly lower than the half-sib estimates (average 0.23), but no clear evidence was present for either sex-linkage or non-additive effects, and most of the genetic variation was apparently additive.

No significant differences were present between heritability estimates for third coxals. There was no consistent evidence for sex-linkage or non-additive effects and again most of the genetic variation was apparently additive.

Table 11. *Analyses of variance, variance components, and estimates of heritability for second coxal bristle number*

Source of variation	d. f.	Males		Females	
		Mean square	Variance component	Mean square	Variance component
Sires	127	1.354**	0.0418	1.142**	0.0388
Dams	127	1.389**	0.0440	1.179**	0.0411
Sires × Dams	127	0.686	0.0050	0.522	−0.0116
Between vials	508	0.646	0.0013	0.614	−0.0044
Within vials	3048	0.641		0.632	
h^2_S		0.23 ± 0.07		0.22 ± 0.06	
h^2_D		0.24 ± 0.07		0.23 ± 0.06	
Average h^2		0.23			

** $P < 0.01$

Table 12. *Analyses of variance, variance components, and estimates of heritability for third coxal bristle number*

Source of variation	d. f.	Males		Females	
		Mean square	Variance component	Mean square	Variance component
Sires	127	0.715**	0.0162	1.255**	0.0422
Dams	127	0.999**	0.0302	0.951**	0.0232
Sires × Dams	127	0.516	−0.0094	0.580	−0.0036
Between vials	508	0.591	0.0084	0.609	0.0034
Within vials	3048	0.557		0.595	
h^2_S		0.11 ± 0.05		0.26 ± 0.07	
h^2_D		0.20 ± 0.06		0.14 ± 0.05	
Average h^2		0.18			

** $P < 0.01$

(iv) Genetic correlations

Covariance analyses between characters were carried out in a similar way to the variance analyses. Genetic correlations between traits were estimated from the appropriate components of covariance and variance. The half-sib estimates of genetic correlation between the traits are given in Table 14 and the parent-offspring estimates in Table 15.

In Experiment 1, the average genetic correlation between abdominals (this refers to total abdominals

Table 13. *Parent-offspring regression estimates of heritability for second and third coxal bristle number*

Estimate	Second coxal	Third coxal
Sire-son	0.13 ± 0.04	0.19 ± 0.04
Dam-son	0.14 ± 0.04	0.17 ± 0.04
Sire-daughter	0.12 ± 0.04	0.20 ± 0.05
Dam-daughter	0.25 ± 0.04	0.11 ± 0.04
Average	0.16	0.17

Table 14. *Half-sib correlation estimates of genetic correlation between the characters*

	Experiment 1	Experiment 2		
	Sternopleural	Sternopleural	Second coxal	Third coxal
Sire component — males				
Abdominal	0.20 ± 0.20	0.27 ± 0.20	0.04 ± 0.20	−0.46 ± 0.25
Sternopleural			0.62 ± 0.16	0.57 ± 0.23
Second coxal				0.59 ± 0.21
Dam component — males				
Abdominal	0.28 ± 0.13	−0.12 ± 0.15	0.03 ± 0.16	0.00 ± 0.16
Sternopleural			0.58 ± 0.17	0.67 ± 0.16
Second coxal				0.41 ± 0.17
Sire component — females				
Abdominal	0.39 ± 0.14	0.07 ± 0.19	0.16 ± 0.16	0.09 ± 0.16
Sternopleural			0.62 ± 0.18	0.27 ± 0.20
Second coxal				0.81 ± 0.14
Dam component — females				
Abdominal	0.26 ± 0.14	0.36 ± 0.17	−0.19 ± 0.16	0.31 ± 0.18
Sternopleural			0.68 ± 0.17	0.72 ± 0.21
Second coxal				0.40 ± 0.20
Average estimates				
Abdominal	0.28	0.15	0.01	−0.02
Sternopleural			0.63	0.56
Second coxal				0.55

Table 15. *Parent-offspring estimates of genetic correlations between the characters*

	Experiment 1	Experiment 2		
	Sternopleural	Sternopleural	Second coxal	Third coxal
Sire-son				
Abdominal	0.16 ± 0.28	0.28 ± 0.13	0.05 ± 0.16	0.36 ± 0.13
Sternopleural			0.62 ± 0.17	0.41 ± 0.15
Second coxal				0.75 ± 0.18
Dam-son				
Abdominal	0.58 ± 0.21	0.14 ± 0.13	0.14 ± 0.15	0.02 ± 0.14
Sternopleural			0.53 ± 0.17	0.61 ± 0.17
Second coxal				0.62 ± 0.18
Sire-daughter				
Abdominal	0.43 ± 0.21	0.16 ± 0.15	0.29 ± 0.16	0.15 ± 0.13
Sternopleural			1.08 ± 0.23	0.21 ± 0.18
Second coxal				0.57 ± 0.18
Dam-daughter				
Abdominal	0.47 ± 0.20	0.40 ± 0.11	0.02 ± 0.11	0.21 ± 0.15
Sternopleural			0.42 ± 0.13	0.36 ± 0.19
Second coxal				0.77 ± 0.18
Average estimates				
Abdominal	0.41	0.24	−0.09	0.18
Sternopleural			0.66	0.40
Second coxal				0.68

unless otherwise stated) and sternopleurals was 0.24 in males and 0.33 in females from the half-sib estimates and 0.37 and 0.45 from the parent-offspring estimates. Estimates in Experiment 2 ranged from −0.12 to 0.36 from the half-sib correlation, and from 0.14 to 0.40 from the parent-offspring correlation. Standard errors for these estimates were large, and as REEVE (1955) pointed out, it is doubtful if they can be used to test the significance of differences between estimates as the sampling distribution of the genetic correlation is skewed. Further, VAN VLECK and HENDERSON (1961) indicated that caution must be used in interpreting estimates of genetic correlation when heritabilities are low, or the coefficient of variation of the heritabilities exceeds 20%. Heritabilities here were of the order of 0.20 with standard errors of 0.05 or slightly larger. In view of the wide variation between heritability estimates for abdominal bristle number, the wide fluctuations between estimates of genetic correlation of this trait with others are not surprising. The average of all the estimates of genetic correlation between abdominals and sternopleurals was 0.35 in Experiment 1 and 0.19 in Experiment 2, so the true value probably lies in this range.

Genetic correlations between abdominal and either second or third coxals were low and varied widely. The average estimate for abdominals — second coxals was 0.01 from the half-sib, and −0.09 from the parent-offspring correlation, while the corresponding estimates for abdominals — third coxals were −0.02 and 0.18 respectively.

Sternopleurals, second and third coxals were highly correlated genetically with each other. The averages of all estimates were 0.48, 0.65 and 0.62 for sternopleurals — third coxals, sternopleurals — second coxals, and second-third coxals respectively.

MODE and ROBINSON (1959) showed that genetic covariance can be partitioned in a manner analogous to genetic variance, and the genetic expectations of variance and covariance components were similar. The use of the diallel analysis in Experiment 2 enabled us to partition genetic covariance by equating expected and observed estimates. Male and female components were frequently of a different scale and these were corrected by equating estimates of similar expectation in the two sexes (dam component for males and sire component for females). The partitioning of genetic covariance for the various character combinations is shown in Table 16. Estimates are not presented for abdominals with either second or third coxals as these correlations were small and inconsistent. Standard errors of the covariance components were estimated in a similar way to those for variance components (TALLIS, 1959), and were all of large magnitude (approximately one quarter of the value of the component). The estimates for partitioning of genetic covariance therefore must be treated as approximate only.

The sire × dam interaction component may be taken as estimating $^1/_4$ of the dominance, or $^1/_8$ of the epistasis. As epistasis was the major component of this interaction for abdominals, it has been taken as estimating $^1/_8$ of the epistasis here. These estimates then represent a maximum statistical estimate of the importance of epistasis in the genetic covariance.

Important epistatic effects were evident for fourth-fifth abdominals and abdominals — sternopleurals, and to a lesser extent for second coxal — third coxal. In addition, the first two combinations showed sex-linked effects. The negative sex-linked component for abdominals — sternopleurals could correspond with the resource distributing genes of RENDEL (1963). The genetic correlation between abdominal segments was unity, so the partitioning of genetic variance for one abdominal segment should be similar to the partition of genetic covariance between segments. For a single abdominal segment approximately 29%, 18% and 53% of the genetic variance was additive autosomal, additive sex-linked, and epistatic, respectively, while the corresponding proportions of genetic co-

Table 16. *Partitioning of genetic covariance for the character combinations* (%)

Component	Fourth-fifth abdominal	Abdominal-sternopleural	Sternopleural -second coxal	Sternopleural -third coxal	Second coxal -third coxal
V_A	18	7	83	100	68
V_{As}	9	−20	0	0	0
V_{AA}	73	73	−17	0	32

variance were 18%, 9%, and 73%. Except for the higher epistatic component from the genetic covariance, these estimates are similar, suggesting that the partitioning of genetic covariance gives at least some idea of the importance of the various genetic components.

Table 17. *Phenotypic correlations between the characters*

	Sternopleural		Second coxal	Third coxal
	Experiment 1	Experiment 2		
Males				
Abdominal	0.13	0.07	0.03	0.04
Sternopleural			0.11	0.07
Second coxal				0.10
Females				
Abdominal	0.14	0.13	0.03	0.03
Sternopleural			0.15	0.09
Second coxal				0.11

(v) Phenotypic correlations

The phenotypic correlations between the characters are shown in Table 17. These all had standard errors of approximately 0.02. There was a low positive correlation between all systems. Between abdominal segments the phenotypic correlation was 0.47 in Experiment 1 and 0.36 in Experiment 2. From the formula, $r_P = h_x \cdot h_y \cdot r_G + e_x \cdot e_y \cdot r_E$ (FALCONER, 1960, where r_P, r_G, and r_E are the phenotypic, genetic and environmental correlations respectively, h^2 the heritability, $e^2 = 1 - h^2$, and the subscripts x and y refer to two different characters) it is possible to estimate the environmental correlation between the systems. These were all quite small but have not been included since they were subject to large errors.

(vi) Distributions of the characters

The distributions of the characters were studied to determine whether they were likely to be reponsible for the discrepancies between estimates of genetic parameters. The frequency distributions for female abdominal, sternopleural, second and third coxal bristle numbers are shown in Table 18. An indication of any asymmetry in the distributions can be obtained by measuring the probit width for each bristle class (RENDEL, 1963), and these values also are given in Table 18.

There was no evidence of asymmetry for abdominals, the mean class width being 0.3 standard deviations. RENDEL (1963) also found no evidence of asymmetry for abdominals, above 12 bristles per segment.

Asymmetrical distributions were evident for the other bristle systems, with longer tails present in the upward direction. For instance, the width of bristle classes for sternopleurals declines from 1.130 probits for the 9 bristle class to 0.616 for the 13 class. It should also be noted that the number of classes for sternopleural and coxal bristle numbers are quite small. Analyses of variance which assume a continuous normal distribution must be applied with caution to discontinuous variables with so few classes (YULE and KENDALL, 1950).

Few classes and asymmetry would be expected to lower the accuracy of estimates of genetic parameters, so that the actual sampling variances of the heritabilities were probably larger than those we have estimated (methods as given by BECKER, 1964).

(vii) Correlation between the same character in the two sexes

Genetic correlations between the same character in the two sexes were estimated for each character from half-sib correlations and are shown in Table 19. The genetic correlations were quite high for second and third coxals. Estimates for sternopleurals were also high with the exception of the sire component estimate from Experiment 2 which was relatively low.

Table 18. *Frequency distributions, and probit widths of the bristle classes for total abdominal, sternopleural, second and third coxal bristle numbers in females*

Total abdominal			Sternopleural			Second coxal			Third coxal		
Class	Frequency	Probit width	Class	Frequency	Probit width	Class	Frequency	Probit width	Class	Frequency	Probit width
33	1		7	13		9	11		5	1	1.190
34	3		8	302	1.352	10	419	1.159	6	43	
35	11	0.400	9	1094	1.130	11	1379	1.254	7	993	1.767
36	10	0.181	10	1160	0.968	12	1224	1.334	8	1526	1.264
37	24	0.255	11	544	0.866	13	216	1.134	9	601	1.081
38	43	0.262	12	120	0.664	14	12	0.511	10	92	0.943
39	64	0.241	13	26	0.616	15	3		11	8	
40	116	0.234	14	4	0.462						
41	207	0.330	15	0							
42	261	0.303	16	1							
43	325	0.300									
44	342	0.276									
45	363	0.280									
46	359	0.285									
47	312	0.277									
48	279	0.299									
49	197	0.279									
50	130	0.256									
51	95	0.280									
52	52	0.242									
53	38	0.310									
54	22	0.407									
55	6	0.289									
56	4										

Estimates of genetic correlation between the sexes for abdominals were considerably less than unity in both Experiments. EISEN and LEGATES (1966) used the heritability of the difference between males and females as a convenient measure of an incomplete genetic correlation between the sexes. In Experiment 2, both sire and dam mean squares were significant in the analysis of the difference between males and females. The heritabilities of the difference between sexes from the sire and dam components were 0.11 and 0.16 respectively. This suggests that the effects of the genes may be different in the two sexes. However, the presence of sex-linkage also lowers the genetic correlation between sexes. The sire component of covariance between males and females is free of sex-linkage (BOHIDAR, 1964), while the sire component of variance for females includes sex-linkage. The expectation for the sire component estimate, when genes have the same effect in the two sexes is as follows:

$$r_G = \frac{V_A + 1/4\ V_{AA}}{\sqrt{(V_A + 1/4\ V_{AA})\ (V_A + 1/4\ V_{AA} + 2\ V_{As})}}$$

Similarly for the dam component estimate, the denominator contains more sex-linkage than the numerator for either the hierarchal or diallel analysis, the expectations being as follows (again assuming gene effects the same in both sexes):

(*i*) hierarchal analysis —

$$r_G = \frac{V_A + 3/4\ V_{AA} + V_D + V_{As}}{\sqrt{(V_A + 3/4\ V_{AA} + V_D + V_{As})\ (V_A + 3/4\ V_{AA} + V_D + 2\ V_{As})}}$$

(*ii*) diallel analysis —

$$r_G = \frac{V_A + 1/4\ V_{AA} + V_{As}}{\sqrt{(V_A + 1/4\ V_{AA} + V_{As})\ (V_A + 1/4\ V_{AA} + 2\ V_{As})}}$$

For our estimates of V_A, V_{As}, V_{AA}, V_D (14, 9, 25, 0%), the genetic correlations expected were approximately 0.73 from the sire component in each experiment, 0.91 from the dam component in Experiment 1 and 0.87 from the dam component in Experiment 2. The bias due to sex-linkage can be removed by multiplying the values of the correlations in Table 19 by the reciprocals of the correlations expected from the above equations. After correcting for these biases the average estimate for abdominal bristle number becomes 0.78.

Thus the bias due to sex-linkage accounts for much of the reduction in genetic correlation but the estimate is still considerably less than unity. Selection for difference in bristle number between the two sexes then should be effective, as was found by FRANKHAM (1967).

Table 19. *Genetic correlations between the same character in the two sexes*

	Abdominal	Sternopleural	Second coxal	Third coxal
Sire component				
Experiment 1	0.41 ± 0.15	0.80 ± 0.14		
Experiment 2	0.75 ± 0.15	0.49 ± 0.21	0.76 ± 0.15	0.91 ± 0.20
Dam component				
Experiment 1	0.73 ± 0.09	0.86 ± 0.14		
Experiment 2	0.65 ± 0.10	0.97 ± 0.16	0.92 ± 0.13	0.89 ± 0.19

IV. Discussion

A feature of our results was the poor agreement between heritability estimates, particularly for abdominal bristle number. This contrasts with CLAYTON *et al.* (1957) who found excellent agreement between parent-offspring regression, full-sib and half-sib correlation estimates for abdominals. The large variation between our estimates appears to have been caused by a combination of sex-linkage and sampling. For the other characters sampling seems to have been the major cause of the variation between estimates. It is possible that the restricted number of classes and asymmetrical distributions also contributed to the variation between estimates for these characters. However, skewness and a severely restricted number of classes were not present for abdominals, but this character showed most variation between estimates.

The average heritability estimate for single segment abdominals was of a similar magnitude to those for the other bristle characters. LATTER (1964) suggested that the heritabilities for one segment abdominals, sternopleurals, and scutellars were the same. His values were higher than ours although he used the same population, but maintained on a different medium.

Phenotypic variation for total abdominal bristle number on two segments has been partitioned in a similar manner by REEVE and ROBERTSON (1954) and CLAYTON *et al.* (1957), and they also found that approximately 50% of the total variation was genetic, while there was a high developmental error component, and very little common environmental variation. However, SHELDON (1963) obtained a higher common environmental component, smaller developmental error and a smaller proportion of genetic variation. Our population differed from those of all these workers in that it showed a large epistatic variance component, a moderate sex-linked component and only a moderate additive genetic component. SHELDON (1963) did find some evidence for sex linkage in his population but CLAYTON *et al.* (1957) did not report any such evidence. For sternopleurals and second and third coxals most of the genetic variation appeared to be additive.

Prediction of response from sex-linked genes is more complex than that from autosomal genes. GRIFFING (1965) showed that, if the effects of the genes are the same in both sexes, the response in females will equal that expected from autosomal genes, while that in the males will be only one half this amount. The average response of the two sexes will be $^3/_4$ of that expected if the variance were autosomal. Therefore, expected responses could be estimated using the proportion of additive autosomal variance plus $^3/_4$ of the sex-linked additive variance as heritability. This expected heritability for total abdominal bristle number then would be 0.21 and that for one abdominal segment 0.15.

GRIFFING (1960) showed that additive × additive genetic variance would also be expected to contribute to the response, but the magnitude of its contribution declines rapidly with each additional generation of selection.

The large differences between estimates of genetic correlation were not surprising as the theoretical

sampling variances of the estimates were high. Consequently, only a rough idea of the magnitude of the genetic correlations was obtained. ROBERTSON (1957, 1962) has suggested that an appreciable portion of the genetic covariances between body size and fecundity, and between thorax and wing length in *Drosophila*, was non-additive. The use of the diallel design in Experiment 2 enabled us to partition genetic covariance. Appreciable non-additive components were present for fourth-fifth abdominals and abdominals — sternopleurals, but not for the other character combinations. Wider use of this technique in similar studies on the nature of genetic correlation may be justified.

BEILHARZ (1963) discussed some of the problems of detecting sex-linked effects, while SCHAFFER and KOJIMA (1963) found that none of their methods were completely satisfactory for estimating them. However, they did not obtain data on both sexes in their diallel cross. We obtained data on both sexes in the diallel analysis and this provided a fairly satisfactory method for estimating sex-linked effects. A satisfactory estimate of sex-linked effects was also obtained from comparisons between the heritabilities estimated from the regressions of both male and female offspring on both sire and dam. The hierarchal analysis was not satisfactory for estimating sex-linkage as epistasis was present, but the parent-offspring regression estimate of sex-linkage was unaffected by this.

The diallel proved to be the only satisfactory method for estimating epistasis. The hierarchal analysis gave indications that it was present but these were somewhat unclear because of sex-linkage. Where possible, the use of the diallel analysis is advisable for estimating genetic parameters and partitioning variation, especially when epistasis, sex-linkage, or maternal effects are present. This method can be easily combined with parent-offspring regressions by measuring the parents in the diallel.

Zusammenfassung

Für eine Anzahl verschiedener Borstenzahl-Charaktere (abdominales, sternopleurales, 2. und 3. coxales Segment) wurde die phänotypische Varianz unter Verwendung hierarchischer und dialleler Versuchsanlagen unterteilt. Anhand von Elter-Nachkommen-Regressionen und -Korrelationen und von Halbgeschwister-Korrelationen wurden Heritabilitäten und genetische Korrelation geschätzt.

Ein hoher Anteil der genetischen Varianz für die Zahl abdominaler Borsten wurde durch epistatische Effekte und die Wirkung geschlechtsgekoppelter Gene bedingt. Bei den anderen Charakteren war der größte Anteil der genetischen Varianz additiv autosomal.

Die genetische Korrelation zwischen der Zahl der Borsten sternopleural und 2. und 3. Segment coxal war durchweg hoch, zwischen abdominal und sternopleural niedrig und zwischen abdominal und sowohl 2. und 3. coxal praktisch gleich null.

Ein bemerkenswerter Anteil der Covarianz zwischen der Zahl abdominaler und sternopleuraler Borsten war nicht-additiv genetisch.

Die Diallel-Methode ergab zuverlässigere Schätzungen der genetischen Parameter, wenn nicht-additive oder geschlechtsgebundene genetische Variation vorlag.

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