THE GENETIC ARCHITECTURE OF BODY WEIGHT AND EGG HATCHABILITY IN *DROSOPHILA MELANOGASTER*[1]

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THE statistical approach to the genetic architecture of a continuously varying trait may be carried out by analyzing the genotypic variation of the trait in terms of the main and interaction effects of the genes involved. In the terminology of statistical genetics, the main effects are expressed as the additive and dominance effects, and the interactions as the additive × additive, additive × dominance, dominance × dominance and higher order interactions, just as in the analysis of a factorial experiment. The major difficulty of this approach is that the higher order interaction terms in a system of many genes become confusing in their genetic interpretations and often useless in constructing the total blueprint of the genetic architecture for such a system.

When the detailed aspects of linkages are ignored, one of the most useful approaches in this field is that of chromosome assay, especially in Drosophila and, to a lesser extent, wheats (LAW 1966; WEHRHAHN and ALLARD 1965). Drosophila, particularly *D. melanogaster*, has obvious advantages in this respect such as the low chromosome number, the availability of marked, multiple inversion stocks for chromosome manipulation, and the large number of mapped loci for locating polygenic effects. The techniques for chromosome manipulation in Drosophila are adapted from the proposals of MULLER (1936a, b) and most of the pertinent references on this subject are discussed by MOHLER (1965).

Using the method of chromosome manipulation, MATHER and HARRISON (1949) demonstrated that all chromosomes in *melanogaster* differentially affected abdominal chaetal number and showed a gross picture of linkage between polygenes. ROBERTSON (1954) assembled a series of homozygous and heterozygous combinations of chromosomes from selected and control lines to investigate the inheritance of body size. He found both additive and dominance effects together with interactions, these interactions being more pronounced when chromosomes from unrelated strains were combined. KELLER and MITCHELL (1962, 1964) investigated the effects of transferring X chromosomes from each of three inbred lines to an autosomal background from the other two. They showed that domi-

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nance and interactions tended to be more pronounced for fertility than for morphological traits. BREESE and MATHER (1957, 1960) came to the same conclusion in their studies of the third chromosome in which six regions were assayed for their effects on viability and chaeta number.

The objective of the present study was to survey the genetic architectures of two traits, one closely related to fitness and the other not as directly related as the first, using the three major chromosomes of *D. melanogaster* jointly extracted from a wide range of genetic material.

MATERIALS AND METHODS

The four inbred lines used as the base materials were Canton-S (designated by (A), Oregon-R (B), Samarkand (C) and Swedish-B (D). They were chosen both because of their highly inbred nature and because of their different geographic origins. Neglecting the Y and fourth chromosomes, eight true breeding combinations of the three major chromosomes from any pair of the base lines can be synthesized, using techniques involving marked, multiple inversion stocks (e.g., ROBERTSON 1954; COOKE and MATHER 1962). In the present study there were six possible pairs (AB, AC, AD, BC, BD, CD) from the four base lines. All pairs were handled in an identical fashion, using inversion stock *M5*; *CyL*; *MéSb*. The crossing schemes leading to the production of the eight true breeding substitution lines for the AB pair were as outlined in Table 1.

Because of the large number of lines with different chromosomal combinations, it is necessary at this point to explain the notations used to describe them in the text. First, a pair of lines together with all substitution lines derived from them will be described by the genetic title AB, AC, etc. where the capital letters denote the source of each line of the pair. Secondly, each of the eight substitution lines from any pair of base lines will be described by three capital letters denoting the source of the X, 2nd and 3rd chromosomes; e.g., AAA is line A, Canton-S, while ABA is a substitution line containing X and 3rd chromosomes from Canton-S together with a 2nd chromosome from Oregon-R. Thirdly, in progeny derived by crossing such substitution lines, the same notation will be used to describe the homozygous pairs of chromosomes, while an H will denote a chromosomal heterozygote; e.g., the progeny of cross AAA $\times$ ABA will be homozygous for the X and 3rd and heterozygous for the 2nd and hence is labelled AHA. Where a more general form is required, capital letters will be replaced by the symbols 1 and 2 which refer to A and B respectively for the set AB, or to C and D respectively, for the set CD, etc., H still indicating heterozygosity (as in Appendix Tables 1, 2 and 3). Y chromosomes will be denoted by a lower case letter; e.g., $\rightleftharpoons_{c}$ means the Y is from line C.

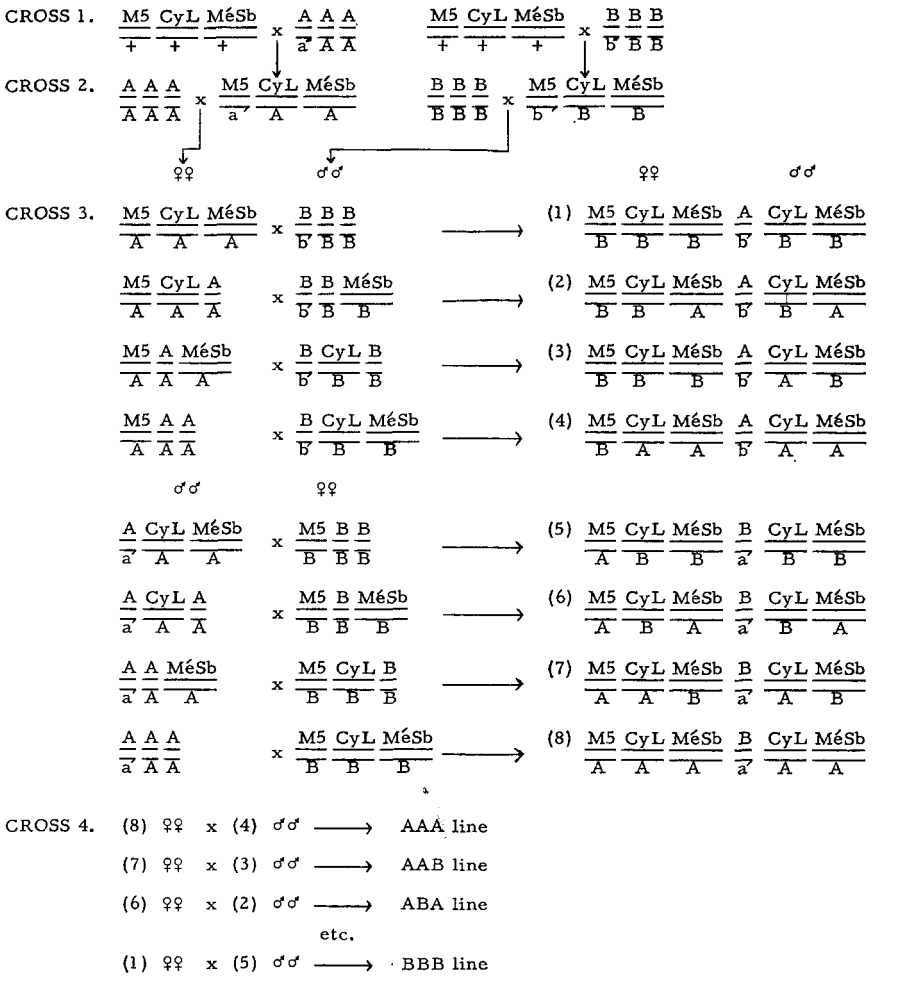
Now returning to Table 1, it can be seen that the first three crosses produce flies of various inversion heterozygotes, crosses among which will allow the extraction of the eight substitution lines. All that remains is to cross flies with homologous wild chromosomes from the same source (cross 4): e.g., (8) ♀♀ $\times$ (4) ♂♂ produce AAA offspring; (7) ♀♀ $\times$ (3) ♂♂ produce AAB offspring, etc.

As ROBERTSON (1954) and others have pointed out, the efficiency of inversions to suppress crossovers is decreased markedly when triple heterozygotes are used, and the present scheme was adopted to minimize any effective recombination that might result. Since the original lines were reextracted in three independent sets (e.g., A was reextracted in AB, AC, and AD), the extracted lines could be compared with one another and with the original inbreds to test for such effects. In fact, whatever hidden recombination might have occurred was not large enough to produce any significant differences between the replicate extractions or between the extracted lines and the original base lines, suggesting that the technique was probably adequate.

It should be noted that all the substitution lines extracted by this technique have their X and Y chromosomes from different base lines. To obtain the comparable lines in which the X and Y are from the same source, lines with identical autosomes but different X chromosomes were crossed, and the male progeny backcrossed to the female line. It was thus possible to maintain

TABLE 1

Crossing scheme employed in production of chromosome substitution lines



two sets of each group of eight lines, one set having their X and Y from different sources (labelled by *) and the other with their X and Y from the same source (no label).

By crossing these eight substitution lines, in the manner outlined in Table 2, it was possible to obtain all the possible 27 female and 18 male homozygous and heterozygous chromosome combinations. This scheme was designed so that each line was used with about equal frequency as both male and female parents.

Two characters were scored, live body weight and egg hatchability. For the body weight measurements the crosses were made as shown in Table 2, and eggs were collected from the fertilized females by a technique described by KOJIMA and KELLEHER (1963). Fifty eggs from each cross were transferred to shell vials containing cornmeal medium, previously inoculated with yeast. In order to reduce the magnitude of errors involved in live body weight measurements, every attempt was made to keep the environmental conditions at the time of weighing

TABLE 2

Matings between isogenic substitution lines

Females	Males							
	AAA	AAB	ABA	ABB	BAA	BAB	BBA	BBB
AAA	AAA	.....	.....	.....	HAA (A)	HAH (A)	HHA (A)	.....
AAB	AAH	AAB	.....	AHB	.....	.....	.....	.....
ABA	AHA	.....	ABA	.....	.....	.....	HBA (A)	.....
ABB	AHH	.....	ABH	ABB	HHH (A)	.....	.....	.....
BAA	.....	.....	.....	.....	BAA	BAH	.....	BHH
BAB	.....	HAB (B)	.....	.....	.....	BAB	.....	BHB
BBA	.....	.....	.....	.....	BHA	.....	BBA	BBH
BBB	.....	HHB (B)	HBH (B)	HBB (B)	.....	.....	.....	BBB

Letters A and B in parentheses denote the sources of the Y chromosome, while A and B without parentheses denote chromosomal homozygotes of the respective origin. Letter H stands for a chromosomal heterozygote.

consistent over all crosses. Flies were weighed as soon as possible after emergence, and records kept of the average weight (in mg) of the first ten males and females to emerge. The experimental material was incubated at 25°C, and replicated in two blocks. Since as far as the female progeny were concerned the * and non-* sets were identical, there were in effect two replicates per block. All 324 crosses [27 genotypes × 2 sets (* and non-*) × 6 pair combinations] in each block were raised simultaneously and were completely randomized so that comparisons may be allowed both within and between pair combinations. The two blocks were separated in time by a period of two weeks.

Hatchability data were obtained by allowing flies to lay eggs on paper caps covered with blackened agar and yeast (the same technique as used for collecting eggs for the body weight experiment), and then removing all but 100 of these eggs per cap. The caps, with eggs, were placed face down over a milk bottle containing water to a depth of one inch, and the eggs left to hatch, the water being used to maintain a constant high humidity for hatching. The number of eggs that had hatched after 36 hours in the bottle was recorded. The time span of 36 hours was considered to be sufficient for all viable eggs to hatch. The experimental design and temperature condition were the same as those used for the body weight tests.

Two sets of hatchability measurements were made. First, the crosses described in Table 2 were made and the hatchability of the resulting eggs scored—hatchability P. Secondly, the hatchability of eggs, laid by the progeny of these crosses, were scored—hatchability PP. These two sets were used to compare the relative effect of egg and maternal genotypes on hatchability.

RESULTS

Body weight: The female weights of 27 genotypes for each base line pair averaged over the four "replicates" (the * and non-* in each of two blocks) are listed in Appendix Table 1. For the male data, the "Y chromosome effects" were determined by the comparison of the * set with the non-* set in each combination

of the base lines. In only one combination (BC) was there any "Y effect," and thus the male data were summarized as if there was no "Y effect" for any combination. Appendix Table 2 contains the male weights of 18 genotypes for each base line combination averaged over the four "replicates."

The analysis of the data was quite straightforward, sexes and pair combinations being treated separately. Having established significant differences between the 27 female (18 male) genotypes, by testing against the variation due to the replicate × genotype interaction, the sum of squares for genotypes was partitioned into the individual 1 df components. These components were set up to test the additive and dominance effects, and the first and second order interchromosomal interaction effects in the conventional factorial model of gene effects. The two main effects of a given chromosome, the additive and dominance effects, were computed by the use of the additive (differences between the two homozygotes) and dominance (difference between twice the heterozygote and the sum of the two homozygotes) contrasts among the marginal values of the three genotypes of

TABLE 3

Distribution of significant chromosome effects and interactions (body weight)

		AB		AC		AD		BC		BD		CD	
Effect	Chromosome	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂
additive (a)	X	+	+	+	..	+	+	+	+	+	+	..	..
	2	+	+	..	..	+	+	+	+	+	+	..	..
	3	..	..	+	+	+	+	..	..	..	..	..	..
dominance (d)	X	..	—	..	—	..	—	..	—	..	—	..	—
	2	..	..	..	..	..	..	..	..	..	+	..	..
	3	..	..	..	..	..	..	..	..	..	+	..	..
a × a	X×2	..	..	..	..	..	..	..	..	..	..	..	..
	X×3	..	..	..	..	..	..	..	..	..	..	+	..
	2×3	..	..	..	..	+	..	..	..	..	..	+	..
d × d	X×2	..	—	..	—	..	—	..	—	..	—	..	—
	X×3	..	—	..	—	..	—	..	—	..	—	..	—
	2×3	..	..	..	..	..	..	..	..	..	..	..	..
a × d	X×2	..	..	..	..	..	..	..	..	..	..	..	..
	2×X	..	—	..	—	..	—	..	—	..	—	..	—
	X×3	..	..	..	..	..	..	..	..	..	..	..	..
	3×X	..	—	..	—	..	—	..	—	..	—	..	—
	2×3	..	..	..	..	..	..	..	..	..	..	..	..
	3×2	..	..	..	..	..	..	..	..	..	..	..	..
a × a × a	X×2×3	..	..	..	..	..	..	..	..	..	..	..	..
a × a × d	X×2×3	..	..	..	..	..	..	..	..	..	..	..	..
a × d × a	X×2×3	..	..	..	..	..	..	..	..	..	..	..	..
a × d × d	X×2×3	..	..	..	..	..	..	..	..	..	..	..	..
d × a × a	X×2×3	..	—	..	—	..	—	..	—	..	—	..	—
d × a × d	X×2×3	+	—	..	—	..	—	..	—	..	—	..	—
d × d × a	X×2×3	..	—	..	—	..	—	..	—	..	—	..	—
d × d × d	X×2×3	..	—	..	—	..	—	..	—	..	—	..	—

* A + stands for a case with $P < 0.01$.
† A — indicates the fact that dominance or dominance interactions of the X chromosome cannot occur in the male.
‡ Blanks are the cases with nonsignificant effects.

that chromosome. The interaction contrasts were obtained by the cross-products of the coefficients of the appropriate main effect contrasts. The results are given in Table 3, in which statistically significant components are marked by a +.

Several points of interest emerge from this analysis (Table 3). It is immediately apparent that the additive effects make the most significant contribution, while dominance and interchromosomal interactions are relatively rare. Concentrating on the additive effects, it can be seen that the male and female data agree well. All chromosomes have additive effects on body weight, but more significant effects are found for the X and 2nd than for the 3rd. Table 4 shows the results of variance ratio tests on the additive, dominance, additive × additive, and dominance × dominance interactions, after pooling the different chromosome effects in Table 3. This confirms the preponderance of the additive effects, but suggests that interactions do exist between chromosomes.

In the above analysis there is one additive difference for each chromosome in the six sets (A—B; A—C; A—D; B—C; B—D; C—D). Since there are only four basic genomes for each chromosome, the six additive differences could, in the absence of interchromosomal interaction, be explained using a model involving only three parameters. Thus if p = A—B; q = B—C and r = C—D then the six additive differences can be equated to various combinations of p, q and r as follows: A—B $= p$; A—C $= p + q$; A—D $= p + q + r$; B—C $= q$; B—D $= q + r$; C—D $= r$. Least squares solutions were obtained for p, q and r for each chromosome and sex, and the agreement of the observed differences with their expected values was tested. Since there were four replicated observations on each additive difference, the replicate variances were available to be used as weights in the estimation. For the female data, these variances were expected to be homogeneous over chromosomes and sets, so that unweighted estimates were obtained and the model tested

TABLE 4

Significance levels for the main effects and principal interactions for all sets (body weight)

	AB	AC	AD	BC	BD	CD
additive (a)						
♀	<0.001	<0.001	<0.001	<0.001	<0.001	N.S.
♂	<0.001	0.01–0.001	<0.001	<0.001	<0.001	N.S.
dominance (d)						
♀	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.
♂	N.S.	N.S.	0.02–0.01	N.S.	<0.001	N.S.
a × a						
♀	N.S.	N.S.	N.S.	N.S.	N.S.	0.01–0.001
♂	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.
d × d						
♀	0.02–0.01	N.S.	N.S.	N.S.	N.S.	N.S.
♂	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.
Other interactions						
♀	0.01–0.001	N.S.	N.S.	N.S.	N.S.	N.S.
♂	N.S.	N.S.	N.S.	0.02–0.01	<0.001	N.S.

N.S.—Not significant.

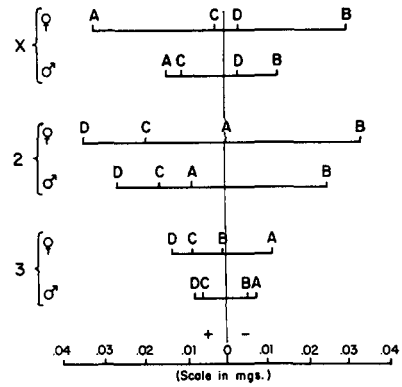


FIGURE 1.—Relative contributions of each genome in deviations from the mean effect of each chromosome (Body weight).

by variance ratio. These variances for the males were, however, heterogeneous; weighted estimates of p, q and r were obtained and the model tested by a χ^2 test. The estimates of p, q, r and the tests of the model are set out in Table 5. These differences expressed graphically are also shown in Figure 1. Several interesting points emerge from a consideration of these estimates. First, the magnitudes of the chromosome effects in the males are less than those for the females. Since the male is hemizygous for the X chromosome, the additive effect for this chromosome might be expected to be half of that in the female, and is, in fact slightly less than half. This does not explain the autosomal differences, so there is then a sex difference independent of the X chromosome. The order of the four genomes, on the other hand, is exactly the same for male and female, this order differing with the chromosome. It would seem that genomes C and D are fairly similar on each chromosome. Genome A has the largest effect on body weight on the X chromosome while D and C have the larger effect on the 2nd and 3rd.

However, despite the apparent consistency in this picture, the simple additive model does not completely explain the observed results, since in four out of the six tests shown in Table 5 there is a significant discrepancy between observed and expected. This discrepancy must be due to some significant interactions occurring within and between chromosomes, and it would be of interest to determine which types of interactions might be responsible. This is not completely obvious from an inspection of Table 3. A more direct way of determining this point is to look at the signs of the observed interaction effects. In the case of female body weight, the procedure is straightforward. For dominance, or the additive $\times$ additive and dominance $\times$ dominance interactions there are three orthogonal contrasts (see Table 3) for each of the six sets, giving rise to 18 different contrasts per type of interaction. If there is no trend in these interactions to increase or decrease body weight, then the positive value contrasts are expected to occur as often as the negative value contrasts within the bounds of sampling error. Any significant departure from the 1:1 ratio of + to − contrasts would indicate the presence of directional interactions. Part (a) of Table 6 shows the numbers of + and −

TABLE 5

Least squares estimates of the additive effects, and tests for the additive model (body weight)—see text

	Chromosome		
	X	2	3
♀♀ data			
p	0.0612	0.0328	—0.0129
q	—0.0316	—0.0525	—0.0075
r	0.0053	—0.0140	—0.0028
$V.R._{(3/54)}$	4.25	8.97	1.30
P	0.05–0.01	<0.001	>0.20
♂♂ data			
p	0.0270	0.0328	—0.0022
q	—0.0235	—0.0407	—0.0105
r	0.0134	—0.0097	—0.0011
$\chi^2_{(3)}$	2.25	13.68	16.14
P	0.7–0.5	0.01–0.001	<0.001

contrasts observed for the dominance, additive × additive and dominance × dominance interactions, and χ^2 tests for the numbers of + and − signs. There is no evidence for directional dominance or dominance × dominance interaction, but the additive × additive interactions mostly decrease body weight. For the higher order interactions (not given in Table 6) no direction was observed. However, it is important to remember that the genetic effects and interactions considered here are those between chromosomes and not of individual loci. For example, an additive × additive interaction within a chromosome is included in the additive effect of that chromosome.

For body weight, then, it appears that the major differences between the homologous chromosomes are due to simple additive effects—but significant additive × additive interactions, though less important, do exist between chromosomes, and these tend to decrease body weight.

Hatchability: A comparison of the two separate hatchability experiments (P and PP) demonstrated that this character is almost entirely dependent on the

TABLE 6

Sign test for directional dominance and interactions

Effect	+	−	χ^2	P
(a) Female body weight				
dominance	11	7	0.9	not significant
additive × additive	4	14	5.6	0.02–0.01
dominance × dominance	7	11	0.9	not significant
(b) Hatchability				
dominance	18	0	18.0	<0.001
additive × additive	11	7	0.9	not significant
dominance × dominance	4	14	5.6	0.02–0.01

maternal genotype. In the first experiment (P) where the eggs were of the required genotype, nearly all genetic differences could be traced back to the mother's genotype. Since all the mothers were isogenic, these data can give no information on dominance, and will only detect additive type differences and thus will not be considered further in this paper. Instead, attention will be concentrated on the second experiment (PP) in which mothers were of the 27 required genotypes.

The preliminary analysis was identical to that used for female body weight. The percent hatchability for all genotypes averaged over the four replicates is shown in Appendix Table 3, while Tables 7 and 8 show the significant genetic components of variance. Two points must be made in connection with these analyses. First, the error variance was fairly large, partly due to the relatively crude technique for assessing hatchability. Although more refined techniques are available, the author's laboratory was not equipped to handle an experiment of the present size (6 sets × 27 genotypes × 4 replications = 648 determinations) in a short interval of time in a refined manner. Secondly, for obtaining homo-

TABLE 7

Distribution of significant chromosome effects and interactions (hatchability)

Effect	Chromosome	AB	AC	AD	BC	BD	CD
additive (a)	X	..	..	..	..	..	..
	2	..	..	..	+	..	..
	3	..	+*	..	..	..	..
dominance (d)	X	..	..	..	+	..	..
	2	+	+	..	+	+	..
	3	..	+	+	+	+	+
a × a	X×2	..	..	..	..	..	..
	X×3	..	..	..	..	..	+
	2×3	+	..	..	..	+	..
d × d	X×2	..	..	..	..	..	..
	X×3	..	..	..	..	..	..
	2×3	..	..	..	..	+	..
a × d	X×2	..	..	..	..	..	..
	2×X	..	+	..	+	..	..
	X×3	..	..	..	..	..	..
	3×X	..	..	..	..	..	..
	2×3	..	..	..	..	..	..
	3×2	..	..	..	..	..	..
a × a × a	X×2×3	..	+	..	..	..	..
a × a × d	X×2×3	..	..	..	..	..	..
a × d × a	X×2×3	..	..	..	..	..	..
a × d × d	X×2×3	..	..	..	..	..	..
d × a × a	X×2×3	..	..	..	..	..	..
d × a × d	X×2×3	..	..	..	..	..	..
d × d × a	X×2×3	..	..	..	..	..	..
d × d × d	X×2×3	..	..	..	..	..	..

* A + stands for a case with $P < 0.01$.
† Blanks are the cases with nonsignificant effects.

TABLE 8

Significance levels for the main effects and principal interactions for all sets (hatchability)

	AB	AC	AD	BC	BD	CD
additive (a)	N.S.*	0.01–0.001	N.S.	<0.001	N.S.	N.S.
dominance (d)	<0.001	<0.001	0.001	<0.001	<0.001	0.001
a × a	0.01–0.001	0.01–0.001	N.S.	N.S.	<0.001	0.01
d × d	N.S.	N.S.	N.S.	N.S.	0.01–0.001	N.S.
Interaction	N.S.	0.01–0.001	N.S.	N.S.	N.S.	N.S.

* N.S.—Not significant.

geneous error variance these data should have been transformed into angles. However, it was felt that a change of scale would inevitably lead to a different idea of the genetic system involved, and since most percentages observed were in the 90's, the percentage scale was retained.

Inspection of Tables 7 and 8 shows the great preponderance of significant dominance effects for this trait and these effects are mostly in the direction of high hatchability as can be seen in part (b) of Table 6. It is apparent that all three major chromosomes contribute to hatchability, but that the 2nd and 3rd chromosomes seem to command more variability than the X. The relative insignificance of the additive effects stands out in comparison to the case of body weight. The additive effects are definitely less exhibited compared to the dominance effects in this trait. Thus, it can be stated that there is a positive and strong evidence of heterosis among the chromosomes tested.

These tables, particularly Table 8, show the presence of significant interchromosomal interaction. The sign test in part (b) of Table 6 shows that the dominance × dominance interactions are mostly in the opposing direction to the dominance effects. Such a situation of dominance type interactions mimics a duplicate gene action.

DISCUSSION

It is apparent that the two characters studied have quite different genetic architectures. Body weight is governed mainly by chromosomes with additive effects and additive × additive interactions, while hatchability exhibits pronounced dominance accompanied by the dominance × dominance interactions resembling duplicate gene action.

It has been argued that one can infer from the genetic architecture of a trait to the type of selection to which the trait has been exposed in the past (MATHER 1953, 1966; JINKS 1955). It is postulated that directional selection on a quantitative trait will result in the trait manifesting directional dominance and duplicate-type gene interaction, such a system suited for the maintenance of a uniformly high fitness in a population. Stabilizing selection will result in either little dominance or, if present, ambi-directional dominance, with the interactions also weak or ambi-directional. The present results lend support to the now large

TABLE 9

Genetic architecture of various characters in Drosophila. A ?-mark stands for "unknown"

Character	Dominance		Interaction		Reference
	Presence	Directional	Presence	Type	
Viability	Yes	Yes	Yes	duplicate	BREESE and MATHER 1960
Egg production (*melanogaster*)	Yes	Yes	?	?	KELLER and MITCHELL 1964
Egg production (*pseudoobscura*)	Yes	?	Yes	?	KOJIMA and KELLEHER 1962 RICHARDSON and KOJIMA 1965
Egg hatchability	Yes	Yes	Yes	duplicate	Present paper
Fecundity	Yes	Yes	?	?	KELLER and MITCHELL 1964
Egg-pupal survival	Yes	Yes	?	?	KELLER and MITCHELL 1964
Development time	Yes	Yes	?	?	KELLER and MITCHELL 1964
Yield of progeny	Yes	Yes	Yes	duplicate	BARNES 1966
Thorax length	Weak	No	Weak	?	KELLER and MITCHELL 1962
Wing length	No	. .	No	. .	KELLER and MITCHELL 1962
Body weight	No	. .	Weak	?	Present paper
Body weight (*pseudoobscura*)	Weak	No	Weak	?	FRAHM and KOJIMA 1966 KOJIMA and OHTA (unpub.)
Abdominal chaetae	Weak	No	Weak	?	KELLER and MITCHELL 1962
Abdominal chaetae	Weak	No	Weak	?	BREESE and MATHER 1957
Sternopleural chaetae	Weak	No	Weak	?	BREESE and MATHER 1957
Sternopleural chaetae	Weak	No	No	. .	HILL 1964

collection of evidence confirming these theories. Some examples based on Drosophila experiments are shown in Table 9.

Perhaps the weakest link between the theory and its evidence as it exists at present, is the lack of knowledge of proved cases of stabilizing selection. One feels reasonably certain in assuming that characters such as viability, hatchability, fecundity and so forth must be under directional selection in Drosophila species. However, litter size in pigs (cited by DARLINGTON and MATHER 1949) and clutch size in swifts (PERRINS 1964) have intermediate optimum values since parents may not be capable of coping with too large a family. Chaetae number in Drosophila has been assumed to be under stabilizing selection (MATHER 1960), and BARNES (1966) has shown that flies of intermediate chaeta number yield most progeny. PARSONS and KAUL (1965) have shown the similar case for mating performance. Birth weights in man (KARN and PENROSE 1952) and in rats (JINKS and BROADHURST 1963) appear to be traits with intermediate optima, as does flowering time of *Nicotiana rustica* (JINKS 1954) and of Papaver (LAWRENCE 1965). All these characters conform to the expected genetic architecture, i.e., weak ambi-directional dominance and little detectable interactions. However, it is still felt that there is too little critical experimental evidence on stabilizing selection.

Based upon whatever evidence is available now, characters which are known to undergo directional or stabilizing selection seem to agree with the expected

genetic architecture. Thus, one might infer from the architecture of body weight found in this study that this too is principally exposed to stabilizing selection.

The experimental results on genetic architecture of a trait are then evidence for the evolution of dominance. But this link between the theory and evidences still poses a few problems that need to be resolved before the completion of the link. The first concerns the scale by which a trait is measured. It is well known that a change in the scale of measurement often alters the picture of interactions considerably. The proposed theory assumes that the genotypes are measured in the same scale as that in which selection is acting. However, one is usually ignorant of what this natural scale is, and normally uses the simplest, most conventional or most convenient scale for data analyses. One must then assume that the observed genetic effects are so gross that the dominance relationships between homozygotes and heterozygotes are maintained approximately whatever "reasonable" scale is used. This assumption holds true for such cases as the natural scale being multiplicative and the measured scale being additive with relatively small differences among genotypic values.

The second question is the problem of overdominance. The existence of overdominance of a locus controlling fitness is indicated with such evidence as the repeated observations of polymorphisms for enzyme systems in man, Drosophila and other organisms. However, the theories related to the evolution and mechanisms of overdominance are not yet clearly established.

It is to be hoped that data will become available to allow a more critical assessment of these ideas.

SUMMARY

Chromosome assay techniques have been used to investigate the genetic architecture of live body weight and egg hatchability, using chromosomes from four divergent inbred Drosophila stocks. Body weight was found to be governed mainly by additive variations, while egg hatchability showed marked heterosis (due to directional dominance) and duplicate type chromosomal interactions. These findings were discussed in terms of the types of selection to which each trait may have been exposed in the past.

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APPENDIX TABLE 1

Female body weight in mg, obtained by averaging the four replicates

			AB	AC	AD	BC	BD	CD
1	1	1	1.225	1.227	1.215	1.078	1.091	1.272
1	1	H	1.355	1.311	1.262	1.161	1.125	1.249
1	1	2	1.283	1.322	1.407	1.206	1.062	1.245
1	H	1	1.228	1.275	1.287	1.150	1.182	1.259
1	H	H	1.350	1.314	1.250	1.174	1.186	1.282
1	H	2	1.189	1.294	1.320	1.175	1.172	1.286
1	2	1	1.278	1.315	1.458	1.211	1.275	1.090
1	2	H	1.206	1.352	1.344	1.189	1.331	1.276
1	2	2	1.221	1.365	1.409	1.232	1.307	1.292
H	1	1	1.301	1.274	1.195	1.148	1.166	1.266
H	1	H	1.225	1.294	1.243	1.156	1.120	1.266
H	1	2	1.328	1.319	1.310	1.193	1.168	1.249
H	H	1	1.151	1.227	1.223	1.188	1.191	1.266
H	H	H	1.279	1.272	1.275	1.236	1.199	1.289
H	H	2	1.200	1.309	1.359	1.195	1.251	1.312
H	2	1	1.179	1.220	1.287	1.299	1.345	1.225
H	2	H	1.245	1.309	1.325	1.240	1.279	1.254
H	2	2	1.192	1.337	1.362	1.295	1.275	1.273
2	1	1	1.171	1.225	1.172	1.188	1.175	1.297
2	1	H	1.178	1.265	1.178	1.272	1.171	1.284
2	1	2	1.205	1.280	1.278	1.211	1.173	1.278
2	H	1	1.147	1.244	1.287	1.340	1.205	1.306
2	H	H	1.159	1.271	1.239	1.283	1.248	1.245
2	H	2	1.168	1.261	1.191	1.241	1.234	1.274
2	2	1	1.138	1.235	1.287	1.230	1.299	1.242
2	2	H	1.136	1.245	1.287	1.324	1.359	1.281
2	2	2	1.151	1.267	1.285	1.269	1.323	1.224

APPENDIX TABLE 2

Male body weight in mg, obtained by averaging the four replicates

			AB	AC	AD	BC	BD	CD
1	1	1	0.908	0.920	0.899	0.795	0.806	0.941
1	1	H	0.986	0.958	0.978	0.846	0.818	0.945
1	1	2	0.936	0.980	0.974	0.807	0.808	0.916
1	H	1	0.927	0.936	0.958	0.833	0.863	1.010
1	H	H	0.947	0.980	0.982	0.889	0.928	0.962
1	H	2	0.906	0.944	0.998	0.852	0.862	0.968
1	2	1	0.897	0.930	0.946	0.883	0.904	0.950
1	2	H	0.904	1.003	0.976	0.890	0.950	0.961
1	2	2	0.860	1.000	1.000	0.922	0.920	0.968
2	1	1	0.870	0.922	0.856	0.875	0.846	0.958
2	1	H	0.902	0.957	0.929	0.902	0.880	0.917
2	1	2	0.909	0.968	0.894	0.857	0.816	0.942
2	H	1	0.848	0.962	0.947	0.984	0.926	0.922
2	H	H	0.856	0.971	0.948	0.940	0.902	0.957
2	H	2	0.864	0.950	0.925	0.914	0.857	0.893
2	2	1	0.858	0.956	0.947	0.952	0.937	0.890
2	2	H	0.865	0.940	0.967	0.966	0.992	0.937
2	2	2	0.842	0.963	0.977	0.986	0.950	0.963

APPENDIX TABLE 3

Hatchability (percent), obtained by averaging the four replicates

			AB	AC	AD	BC	BD	CD
1	1	1	82.25	91.25	78.00	95.75	95.55	93.25
1	1	H	93.25	96.25	93.67	97.50	98.75	96.25
1	1	2	93.00	90.50	88.00	97.00	94.25	86.00
1	H	1	95.50	95.50	85.67	98.25	98.50	95.00
1	H	H	97.00	98.00	96.00	97.50	95.75	99.75
1	H	2	96.25	97.00	85.33	98.00	97.75	81.00
1	2	1	86.25	91.75	61.67	92.00	93.00	93.50
1	2	H	96.25	95.00	98.33	94.25	98.25	92.50
1	2	2	86.00	95.75	83.00	91.25	97.00	93.00
H	1	1	95.25	92.00	94.33	96.50	93.00	94.25
H	1	H	98.25	97.25	90.67	99.00	99.25	95.75
H	1	2	97.25	97.75	88.67	96.25	91.75	92.50
H	H	1	96.75	95.75	76.00	98.75	97.25	97.50
H	H	H	99.00	97.50	97.67	98.75	98.50	98.25
H	H	2	97.25	95.25	86.33	99.25	98.00	96.00
H	2	1	93.50	92.00	91.00	93.00	92.75	90.00
H	2	H	92.00	93.00	95.33	99.00	99.25	96.75
H	2	2	91.25	89.00	93.67	98.25	98.50	95.50
2	1	1	87.00	77.25	69.67	94.25	94.75	85.00
2	1	H	95.25	94.00	90.33	97.50	98.25	98.25
2	1	2	96.00	97.50	71.67	96.50	93.50	89.75
2	H	1	90.50	94.00	87.67	96.75	96.75	96.50
2	H	H	97.50	97.75	87.00	98.75	98.00	98.50
2	H	2	91.50	93.75	87.67	96.75	97.25	98.25
2	2	1	96.50	91.00	83.00	83.75	90.50	87.75
2	2	H	97.00	96.00	95.33	96.50	97.25	97.00
2	2	2	86.75	95.50	83.00	92.75	97.00	91.00