THE GENETIC RELATIONSHIP OF TWO QUANTITATIVE CHARACTERS IN *DROSOPHILA MELANOGASTER*.

II. LOCATION OF THE EFFECTS¹

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DAVIES and WORKMAN (1971) described the analysis at the chromosomal level of a set of lines of *Drosophila melanogaster* selected for sternopleural and/or abdominal bristle number. Several lines of evidence suggested that the sets of genes affecting the two characters were largely different. In this paper the location of the "effective regions" of the chromosomes is described. The emphasis was on demonstrating the separability of regions affecting the two characters, and not on extremely precise location or on any possible further subdivision of effective regions.

MATERIALS AND METHODS

The origin of the selected lines and of the standard background stock Oregon WD is described by DAVIES and WORKMAN (1971). The same abbreviated names for the lines are used here. Flies were raised on oatmeal-agar medium in 1/3 pint milk bottles at $25 \pm 1^\circ\text{C}$. The balancer stocks used are described by DAVIES and WORKMAN (1971). The marker stocks used for location were *y ct car* (*y*; yellow, X, 0.0 cM: *ct*; cut, X, 20.0 cM: *car*; carnation, X, 62.5 cM), *dp cn bw* (*dp*; dumpy, II, 13.0 cM: *cn*; cinnabar, II, 57.5 cM: *bw*; brown, II, 104.5 cM), *se cp e* and *ve se cp e* (*ve*; veinlet, III, 0.2 cM; *se*; sepia, III, 26.0 cM: *cp*; clipped, III, 45.3 cM: *e*; ebony, III, 70.7 cM). The *se cp e* and *ve se cp e* stocks were isogenic for bristle number genes. Single chromosomes of each of these genotypes were taken, put onto an Oregon WD background and checked for cytological abnormalities. One was chosen the bristle numbers of which most closely resembled those of Oregon WD. All *se cp e* chromosomes unfortunately had low bristle numbers.

Location analysis: Preliminary analyses showed that the majority of the effects were recessive, so recombinants were always assayed when homozygous. Lethal recombinants in STH III and HH II were assayed heterozygous with a particular chromosome of the other lethal type. Male flies carrying single recombinant chromosomes were obtained from backcrosses to the marker stock concerned. These recombinant chromosomes were maintained by crossing flies carrying them singly to balancer stocks. Test crosses were then used to find which resultant stocks carried recombinant rather than parental marker chromosomes. Homozygous recombinant chromosomes were assayed (20 flies each sex). All analyses were performed on an Oregon WD background which included the Y chromosome.

Analysis of variance was used to determine whether there was a significant heterogeneity among recombinant chromosomes of each phenotype. Where heterogeneity was encountered chromosomes were assigned to groups for bristle number by t-tests and each group tested for homogeneity. The division into groups was almost invariably clearly identical in males and

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females, but was often more sharply defined in one sex than the other. All statistical analyses were therefore carried out on the data from one sex only.

The general principles have been given by THODAY (1961). The number of bristle number classes within each recombinant marker phenotype gives a (minimum) estimate of the number of genes between the marker genes concerned. The number of chromosomes falling into the various classes allows one to estimate the locations of the genes. Recombinants with marker chromosome to the left or to the right of a cross-over point are obtained. The location obtained from the analysis in each case should be the same, as should effects of effective regions unless there is gene interaction. In the more complex analyses, careful consideration of the sizes and bristle numbers of the various bristle number classes within each marker recombinant class usually allows a unique interpretation to be arrived at. In particular, where two characters are being studied, the location of the genes affecting them can be worked out separately, and then the values in both characters of individual recombinants can be checked to see if they correspond to the same crossover position.

Possible technical improvements would be to mark the extreme ends of the chromosomes, thus avoiding ambiguities due to double crossovers, and to use more marker genes per chromosome in order to increase the accuracy of location.

Only loci at which lines differ from a marker stock can be detected. The use of more than one marker stock for a chromosome would only be confusing, so that there is an unavoidable loss of information. The background used in these particular experiments was not that present in the selected line, but DAVIES and WORKMAN (1971) showed that interactions were much less important than direct effects of genes in determining the phenotype. This might not be true of genes affecting other characters. Despite these drawbacks the method proved to be very powerful.

ABH III STERNOPLEURAL

ABH III ABDOMINAL

FIGURE 1.—Histograms of mean bristle numbers of recombinants between ABH third chromosomes and *ve se cp e*. Only ABH III B abdominal data and ABH III A sternopleural data are given. One rectangle represents one recombinant.

All third chromosomes except those in ABH and one of the STH lethal classes contained *In(3R)Mo*. The breakpoints of this inversion are 93D (about 92 cM) and 98F2-3 (about 106 cM) (LINDSLEY and GRELL 1968) so that it has no direct effect on the *cp-e* interval. However, according to LINDSLEY and GRELL (1968) it reduces crossing over between the centromere and *sr* to 5% and crossing over between *sr* and *ca* to 0.3%, the effect decreasing with increasing distance from the inversion. In these analyses the *cp-e* distance was 3.5 instead of the expected 25.4 cM. The function that would describe the distribution of this effect is unknown, so equal reduction of crossing over throughout the *cp-e* interval has been assumed. Thus the locations in this region are not very meaningful, but estimates of the order, number, effects and separateness of loci in this region still are. Loci will tend to seem to be located further to the right than their true position.

RESULTS

Linkage relations of genes affecting sternopleural and abdominal bristle number in chromosomes important in both direct and correlated responses to selection: The important chromosomes are the STH X chromosome, the ABL second chromosome and the ABH, STH and ABL third chromosomes (DAVIES and WORKMAN 1971). Figures 1-5 show the bristle numbers of homozygous recombinant chromosomes obtained in the location analysis of these chromosomes. Occasional recombinants that were shown to be multiple recombinants have not been included. The detailed process of the subdivision into groups within each recombinant class, and detailed interpretation of the data, are given by DAVIES (1969), but in general the groups and the interpretation are quite clear from the Figures. Table 1 gives the estimated locations and effects of the genes postulated in the interpretation of the data.

There are two important conclusions that can be drawn from these results:

1. All sternopleural genes were separable from all abdominal genes.
2. There was a tendency towards close linkage of genes important in direct responses and genes involved in correlated responses.

It can be seen from Table 1 and Figure 6 that the standard errors of the locations of sternopleural and abdominal genes in the same chromosome often overlap. Such genes are known to be separable because a recombinant or group of recombinants was obtained whose phenotype indicated that it represented a crossover event occurring between the bristle number genes. For example, the separation of the leftmost sternopleural and abdominal genes in the *se-cp* region of STH III is based upon two *++cp e* chromosomes that had the STH III alleles of none of the abdominal genes but had picked up the STH III allele of the leftmost sternopleural gene. In general such a conclusion is based upon the phenotypes of more than one recombinant chromosome. The gene order is clear in most cases from Table 1 and Figure 6, but in the complicated STH and ABL third chromosomes this is not so. The order of genes in the STH third chromosome is *+^{ve}*, ST, *+^{se}*, ST, AB, AB, ST, AB, ST, *+^{cp}*, ST^{high}, lethal, ST^{low}, AB, *+^e*. The order of genes in the ABL third chromosome is *+^{ve}*, ST, *+^{se}*, AB^{high}, ST, AB^{low}, AB^{high}, AB^{high}, ST, *+^{cp}*, AB^{low}, ST, AB^{high}, *+^e*. In two cases (ABH III A 43.7 cM, ABL III "low" 31.9 cM) sternopleural and abdominal genes were not successfully separated, but in each case the equivalent genes in parallel analyses (ABH IIIB, ABL III "high") were separable.

STH X. STERNOPLEURAL STH X. ABDOMINAL

FIGURE 2.—Histograms of mean bristle numbers of recombinants between STH X chromosomes and *y ct car*. One rectangle represents one recombinant.

Many of the genes important in correlated responses are located within 5 cM of a gene important in a direct response; only the sternopleural gene in ABL II and the sternopleural gene around 15.4 cM in ABH III were rather far away. There are several examples of very close linkage, many in STH X and III, but

STH III STERNOLEURAL **STH III ABDOMINAL**

FIGURE 3.—Histograms of mean bristle numbers of recombinants between STH third chromosomes and *ve se cp e*. One rectangle represents one recombinant. Sternopleural bristle number data is for the lower chromosome.

also are in ABH III and one in ABL III as mentioned before. There is no case of pleiotrophy.

All the sternopleural genes that were located in the analysis of ABL III (Figures 5 and 6, Table 1) increase sternopleural bristle number relative to *ve se cp e*. They are clearly not part of the initial positively correlated response, but may perhaps be regarded as having been involved in a secondary correlated response, associated with the increase in frequency of the uniquely important abdominal gene at 31.8 cM. The bristle numbers of *ve se cp e* were unfortunately three bristles below those of Oregon WD and Athens base, so that possibly some negative effects might have been obscured.

Analysis of the remaining lines: The results of the analysis of the other lines are presented in Table 2 and Figure 6, together with results for STH II abdominal and ABL II sternopleural bristle number. The locations of the first gene in the γ -*ct* region of the HH X chromosome and of the gene in the *dp-cn* region of the HL second chromosome are tentative. These effects are small and it was difficult to assign chromosomes clearly to groups because of environmental variance. One chromosome, the LH third, produced no results capable of clear interpretation and the data are not presented.

With two exceptions, all genes affecting sternopleural bristle number were successfully separated from genes affecting abdominal bristle number. An effect on abdominal and an effect on sternopleural bristle number were both located to 4.2 ± 1.8 cM on the LL third chromosome, but the number of recombinants

ABL II STERNOPLEURAL ABL II ABDOMINAL

FIGURE 4.—Histograms of mean bristle numbers of recombinants between ABL second chromosomes and *dp cn bw*. One rectangle represents one recombinant.

made was not very large (26). A lethal gene in the LL second chromosome was inseparable from a two bristle effect on abdominal and a small effect on sternopleural bristle number. It was also closely linked to another gene affecting sternopleural bristle number. Seventy-two recombinants were made in this region, which is, however, very long (44.5 cM). It is quite likely that these are cases of the closest linkage detected rather than of pleiotropy. Table 2 shows that there

ABL III STERNOPLURAL **ABL III ABDOMINAL**

FIGURE 5.—Histograms of mean bristle numbers of recombinants between ABL third chromosomes and *ve se cp e*. One rectangle (sternopleural) or one square (abdominal) represents one recombinant. Sternopleural data is for the highest chromosome III class.

were a number of other cases of fairly close linkage between genes affecting the two characters.

Further points from the location analyses: 1. There were a number of clear cases of intrachromosomal gene interaction, since the effect of certain genes was different between classes of the two reciprocal recombinant types. Gene interaction affecting sternopleural bristle number was found in the lower HH X chromosome, the HH third chromosome and the STH X chromosome. There were very strong interactions affecting abdominal bristle number in the ABL third chromosome, including complete epistasis of a low effect over several high effects.

2. The low abdominal effects of the ABL and LL third chromosome were attributable to the effects of one gene, which located in a very similar position in the third chromosomes from the two lines. The effect of this gene is sufficient to account for all the response to selection of these lines. The ABL III allele, at least, reduced viability considerably when homozygous. The accelerated re-

TABLE 1

Results of location analysis of chromosomes important in correlated responses

Line, chromosome, character	Location of gene (centimorgans)	Effect of gene* (1) (2)		Line, chromosome, character	Location of gene (centimorgans)	Effect of gene* (1) (2)	
ABH III B†	35.7 ± 1.6	+2	+1.6	STH X	0.83 ± 0.82	—1.0	—0.8
abdominal	43.7 ± 0.9	+2.1	+1.4	sternopleural	15.8 ± 1.7	+2.0	—0.8
	48.5 ± 1.5	+1.2	+1.2		22.8 ± 1.35	+2.0	+4.6
	59.6 ± 3.4	+1.0	+1.6		38.1 ± 2.7	+1.2	+1.2
	68.3 ± 1.3	+0.9	+1.1		63.0 ± 0.45	+1.0	+0.8
ABH III B	15.25 ± 2.6	+2.7	+2.1	STH X	15.0 ± 1.8	+2.0	+2.7
sternopleural	32.4 ± 1.5	+1.7	+1.9	abdominal			
	43.7 ± 0.9	+2.0	+2.2	STH III low‡	18.8 ± 2.5	+1.4	+1.6
ABH III A†	35.6 ± 2	+2.1	+1.8	sternopleural	27.3 ± 0.7	+1.7	+1.5
abdominal	43.7 ± 1.1	+1.1	+1.1		39.2 ± 1.35	+1.7	+1.9
	60.5 ± 2.8	+0.9	+0.9		44.4 ± 0.6	+1.1	+1.0
ABH III A	15.45 ± 2.7	+2.5	+2.3		59.4 ± 2	+6.4	+5.7
sternopleural	30.8 ± 1.7	+5.9	+5.8		65.4 ± 2	—1.7	—1.4
	42.1 ± 1.47	+2.5	+2.2	STH III high	22.8 ± 2.1	+1.3	—
ABL III	28.3 ± 0.7	+1.8	+6.3	sternopleural	28 ± 1.1	+1.4	+1.1
abdominal	31.75 ± 0.9	—4.1	—19.5		40 ± 1.6	+1.7	+2.0
	35.3 ± 1.3	0	+1.2		43.3 ± 1.1	+0.9	+1.1
	38.8 ± 1.2	0	+1.6		63.3 ± 2.4	+6.6	+6.4
	50.5 ± 1.5	—4.7	+1.5	STH III	28.2 ± 0.9	+1.4	+1.6
	69.6 ± 1.0	0	+3.0	abdominal	34.8 ± 1.45	+1.5	+1.5
ABL III‡	3.6 ± 2.3	+1.6	+2.1		42.2 ± 1.1	+1.6	+1.9
“high”	32.6 ± 1.4	+1.6	+1.7		66.5 ± 1.6	+1.4	+1.1
sternopleural	44 ± 0.7	+1.9	+2.0	ABL II “low”	47.4 ± 4	—1.2	—1.3
	65.2 ± 2.0	+2.7	+2.5	abdominal	94 ± 3.5	—3.2	—3.0
ABL III “low”	31.9 ± 1.4	+1.7	+1.5	ABL II “high”	49.8 ± 3.1	—1.0	—0.9
sternopleural	65.6 ± 2.3	+2.1	+2.7	abdominal	96.2 ± 4.35	—1.1	—1.3
				ABL II	23.7 ± 3.5	—1.9	—1.5
				sternopleural			

* Effects of genes: (1) is the effect of a gene (number of bristles) when the part of the chromosome to the left of it is from the selection line. (2) is the effect of a gene when the part of the chromosome to the right of it is from the selection line. The means of effects in the two sexes are given.

† ABH III A refers to the group of chromosomes with high sternopleural and low abdominal effect, ABH III B to the group of chromosomes with high abdominal and intermediate sternopleural effect.

‡ High and low refer to sternopleural in STH and to abdominal in ABL.

sponses in these lines (DAVIES and WORKMAN 1971) were probably due to the delayed increase in frequency of this gene of very large effect. Such a delayed response could occur if the gene was initially at a low frequency (LATTER 1965a,b; 1966). In other lines, all of the LH high abdominal effect was attributable to a single gene on the X chromosome, and genes with sternopleural effects greater than five bristles were found in the third chromosomes of ABH and STH.

3. Bristle number differences between samples of the same chromosome type from the same line were successfully explained in a number of cases; ABH III, HH X and LL II sternopleural and abdominal, ABL II abdominal, STH III and ABL III sternopleural (Tables 1 and 2).

FIGURE 6.—Summary of locations of genes affecting sternopleural and abdominal bristle number. Those marked with an asterisk have a negative effect with respect to the standard bristle marker stock. The previous locations marked with solid lines are taken from WOLSTENHOLME and THODAY (1963), THODAY, GIBSON and SPICKETT (1964) and SPICKETT and THODAY (1966). The previous locations marked with dotted lines represent less exact results from the work of WIGAN (1949), BREESE and MATHER (1957) and LOUW (1968).

4. Many of the genes located in the various lines map in similar positions and have similar effects (Figure 6), suggesting that different lines responded to identical selection pressures by utilizing variability at the same loci. Figure 6 shows considerable clustering about certain map positions, but the allelism of effects in different lines was not established experimentally.

5. CLAYTON and ROBERTSON (1957) and FRANKHAM, JONES and BARKER (1968b) found lethal genes that seemed to have important effects in some lines. In these lines all lethal genes except the one in some LL second chromosomes were shown to be separable from all effects on either character. All effects were separable from *In(3R)Mo* in all lines, though some were effectively closely linked because of the crossover suppression due to the inversion. One of the STH III lethals was separable from *In(3R)Mo*, so that the STH third chromosome was not a stable balanced lethal system. In the HH second chromosome one lethal was associated with and inseparable from *In(21)t*, and the other lethal was in the chromosome region covered by it, so that this was a stable balanced lethal system. A gene or genes within *In(21)t* increased sternopleural bristle number, but all abdominal effects were separable from the inversion. On the

TABLE 2

Location analysis of other chromosomes

Line, chromosome, character	Location of gene (centimorgans)	Effect of gene* (1) (2)		Line, chromosome, character	Location of gene (centimorgans)	Effect of gene* (1) (2)	
HH X "high"	32.6 $\pm$ 3.2	+2.8	+2.8	LL II "low"	27.0 $\pm$ 3.5	-1.9	-1.3
sternopleural	52.2 $\pm$ 3	+1.0	+1.2	sternopleural			
HH X "inter- mediate"	18.9 $\pm$ 1.1	-0.1	-1.3	LL II "high"	26.2 $\pm$ 2.4	-2.0	-2.0
sternopleural	36.0 $\pm$ 2.8	+1.2	+2.7	sternopleural	32.2 $\pm$ 2.6	+2.0	+1.2
HH X "high"	53.7 $\pm$ 2.8	+1.8	+1.0		31.6 $\pm$ 2.6	0	+0.8
abdominal	13.0 $\pm$ 2.1	+1.7	+0.9	LL II "low"	94.1 $\pm$ 3.8	-2.0	-2.1
	36.5 $\pm$ 3.5	+2.8	+1.6	abdominal			
	61.3 $\pm$ 1.2	+1.1	+1.3	LL II "lower"	31.6 $\pm$ 2.6	-1.3	-1.6
HH X "inter- mediate"	12.1 $\pm$ 1.7	+1.9	+2.0	abdominal (not separated from lethal)			
abdominal	61.7 $\pm$ 0.8	+1.2	+0.8		92.1 $\pm$ 4.8	-2.4	-1.9
HH II	27.8 $\pm$ 3.8	+1.1	+0.9	LL III	4.2 $\pm$ 1.8	+2.0	+2.2
sternopleural	65.5 $\pm$ 2.6	+1.2	+1.2	sternopleural	26.5 $\pm$ 0.4	-2.3	-2.4
HH II	48.6 $\pm$ 3.3	+2.1	+2.3		34.2 $\pm$ 1.1	+0.9	+1.1
abdominal				LL III	4.2 $\pm$ 1.8	+3.2	+3.4
HH III	5.9 $\pm$ 2.1	?	+1.6	abdominal	28.5 $\pm$ 0.8	+6.2	+5.5
sternopleural	8.8 $\pm$ 2.3	?	+1.4		32.3 $\pm$ 1.1	-11.5	-13.3
	21.2 $\pm$ 1.9	?	+1.1		36.8 $\pm$ 1.6	0	+2.7
	28.0 $\pm$ 0.8	+1.0	+1.0	STH II	48.6 $\pm$ 3	+2.1	+1.8
	34.7 $\pm$ 1.3	+1.1	+1.2	abdominal			
	44.3 $\pm$ 0.6	+0.9	+1.2	ABH II	28.5 $\pm$ 3.2	+1.1	+1.0
	57.1 $\pm$ 3.3	+1.6	+1.2	sternopleural	67.1 $\pm$ 3	+1.1	+1.3
	67.3 $\pm$ 2.2	+4.0	+0.9	LH X	12.7 $\pm$ 1.8	+2.9	+3.1
HH III	7.8 $\pm$ 2.3	?	+1.3	sternopleural			
abdominal	16.4 $\pm$ 2.4	?	+2.3	HL II	46.4 $\pm$ 4.3	-0.8	-0.8
	65.9 $\pm$ 2.5	+2.7	+2.7	abdominal	95.1 $\pm$ 4.2	-1.5	-1.5
				HL III	22.5 $\pm$ 1.9	+3.6	+3.0
				sternopleural	28.4 $\pm$ 1.2	+2.2	+2.4
					44.2 $\pm$ 0.8	+2.3	+2.3
					66.5 $\pm$ 2.2	+2.8	+3.3

* Effects of genes: (1) is the effect of a gene (number of bristles) when the part of the chromosome to the left of it is from the selection line. (2) is the effect of a gene when the part of the chromosome to the right of it is from the selection line. The means of effects in the two sexes are given.

whole the role of the lethals and inversions in the responses to selection must have been small and largely indirect.

DISCUSSION

The firm conclusions from the results of these experiments are:

- The sets of genes affecting sternopleural and abdominal bristle number are distinct.
- Gene linkage is the main basis of the genetic relationship of these characters.
- These linkages were important in the correlated responses to selection.

The reliability of the analyses is evidenced by chromosomes from the same bristle number group showing the same gene locations and effects, and their resolution by the successful explanation of the bristle number heterogeneity of certain chromosomes in terms of known gene effects. Since only genes whose line alleles differed from the marker stock allele could be detected, some loci important in the responses to selection may have been missed. Nevertheless, a large proportion of the information obtained must be relevant to the responses, particularly where genes have large effects. The separability of abdominal and sternopleural genes is clear, but of course only applies to those loci that were segregating in the base population.

The genes involved in correlated responses tended to be closely linked to genes involved in direct responses, and there were no cases of pleiotropy. Linkages themselves are not sufficient for correlated responses to occur. If the linkage relations of "+" and "-" alleles of genes affecting these characters were random, then correlated responses would only occur erratically by sampling error. In this case if correlated responses occurred, they should be positively or negatively correlated with equal frequency. If, on the other hand, linkage disequilibrium for alleles of these genes were present in the population, correlated responses would occur repeatedly and predominantly in one direction relative to the direct response. Close linkages would enhance correlated responses in both situations. All correlated responses in the lines described have occurred in the same direction as the direct response. CLAYTON *et al.* (1957c) found positively correlated sternopleural responses in high abdominal lines, but little or no correlated response in low abdominal lines. JONES, FRANKHAM and SHERIDAN (1969) found positively correlated sternopleural responses in twenty-six high abdominal lines and positively correlated abdominal responses in fifteen out of sixteen lines selected for high or low sternopleural bristle number. There is clearly a strong tendency for selection on one of these characters to lead to a correlated response in the same direction in the other character. These data, and the nonrandomness of linkages of sternopleural and abdominal genes, make it probable that linkage disequilibrium of alleles of closely linked genes was the basis of the correlated responses.

Linkage disequilibrium could have been generated during the selection process by sampling and selection pressure, but a significant proportion of all correlated responses occurred in the first generation of selection, so that linkage disequilibrium must have existed in the "Athens base" laboratory population. This could have been because of sampling effects during the establishment of this population from the progeny of wild flies. Alternatively linkage disequilibria for these genes might exist in the wild population.

The results of JONES *et al.* (1969) and CLAYTON *et al.* (1957c) suggest that linkage disequilibrium for these genes may be a general phenomenon in wild populations of *Drosophila melanogaster*. If so, the selective force that caused the linkage disequilibrium could have caused the correlated responses. There could be interaction at the level of fitness between the abdominal and sternopleural genotypes of flies, such that individuals with similar phenotypic values

for the two characters had high fitnesses. Several weak arguments can be made against this; a) the selective pressure causing linkage disequilibrium might not be present under laboratory conditions, b) linkage disequilibrium might have arisen by chance in the history of the lines rather than by natural selection, c) the strong tendency towards close linkage of genes involved in direct and correlated responses indicates that the linkages played an important role in the correlated responses, whereas on the above model correlated responses should occur whether there is linkage or not.

The correlated responses in these lines probably arose through the action of artificial selection on a population with pre-existing linkage disequilibrium of alleles of closely linked abdominal and sternopleural genes. There was also a small component due to sampling error, and perhaps due to the natural selective basis of the linkage disequilibrium.

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SUMMARY

Genes were located which were responsible for the effects on abdominal and sternopleural bristle number of chromosomes extracted from three lines of *Drosophila melanogaster* selected for abdominal or sternopleural bristle number and showing a correlated response in the other character. It was shown, to the limits of resolution of the analysis method, that all genes affecting sternopleural bristle number were separable from all genes affecting abdominal bristle number. There was no case of pleiotropy. Genes involved in correlated responses were often closely linked to genes involved in direct responses. Location of the effects in other lines which had been selected for both characters also showed that the genes affecting the two characters were largely separable. In only two cases was separation not achieved. Correlated responses probably occurred by the action of artificial selection on a population with preexisting linkage disequilibrium for alleles of sternopleural and abdominal genes, the correlated responses being enhanced by close linkages. There was also a component due to sampling error. An alternative not ruled out is that there is strong interaction at the fitness level between these characters, so that natural selection could have partially or completely caused the correlated responses.

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