SEX DIMORPHISM AND CANALIZATION IN *DROSOPHILA MELANOGASTER*

By G. L. LEE*† and A. S. FRASER*‡

[*Manuscript received March* 3, 1969]

Summary

Selection to reduce the sex dimorphism of scutellar bristle number associated with the gene *scute* in *D. melanogaster* was successful. Although probit analysis indicated that an increase in canalization had been achieved, the selection line was found to be still quite sensitive to temperature. The selection response was found to have no correlated effect in three other mutant genotypes nor in *scute*$^+$ flies but a slight effect was noted with the gene *Scutoid*.

It is concluded (1) that while selection for increased canalization may reduce sex dimorphism, the reciprocal is not true, and (2) that selection in the presence of mutant genes is unlikely to affect the expression of other mutant genes because of the low polygenic overlap. The concept of polygenic overlap is shown to have application to intergenic situations allowing estimation of the degree of modifier specificity and to intragenic situations where the time of modifier action with respect to translation can be projected.

I. INTRODUCTION

The major bristle systems in *Drosophila melanogaster* show very little variation between the sexes. This is particularly true of the scutellar system. The number of scutellar bristles is strongly canalized at four, with only a small proportion of individuals in unselected populations showing extra or missing scutellar bristles. Fraser (1963) in an extensive study of this character found that the incidence of individuals with deviant number was greater in females than in males, i.e. there was a slight sex dimorphism. The introduction of the *scute* gene results in (1) a decrease in the number of scutellar bristles, (2) a marked increase in the variability of the number of scutellar bristles, and (3) a marked sex dimorphism of scutellar bristle number (Rendel 1959). The use of major genes to expose variation in highly invariable characters had been realized as early as 1946 by Blanc, and Plunkett (1932) explained this greater variability as follows: "processes controlled by wild-type genes usually approach their assymptotes, while those modified by mutant genes may be terminated by the effects of other developmental processes while still very incomplete".

Selection against the induced variability of mutant genes has been successfully practised by Rendel and Sheldon (1960) and Rendel, Sheldon, and Finlay (1965) for number of scutellar bristles in *scute* stocks. Waddington (1960) and Waddington and Robertson (1966) have also successfully reduced the variability of other mutant phenotypes. The criterion of canalization (reduced variability) used by Rendel (1959)

* Genetics Department, University of California, Davis, California, U.S.A.

† Present address: Hawkesbury Agricultural College, Richmond, N.S.W. 2753.

‡ Present address: Department of Biological Sciences, University of Cincinnati, Ohio, U.S.A.

was the width of the particular bristle zones as measured in probits. The selected line was also found to show a relative insensitivity to temperature variation, and a threefold decrease in the sex dimorphism. However, Kindred (1965) found no increase of canalization in a line selected for reduced sensitivity to temperature fluctuation. Druger (1967) independently confirmed this finding, indicating that either the reduced sensitivity to variation of temperature found by Rendel (1959) is fortuitous, or that the relationship between temperature sensitivity and developmental canalization is not simple. A feature of Rendel's results is that the selection was based on a scutellar number of two. Recent reports by this group (Rendel, Sheldon, and Finlay 1966; Rendel, personal communication) state that they have been unable to increase canalization at a scutellar number of 1, nor have they been able to change the constancy of the sex dimorphism of abdominal bristle number despite selection for over 20 generations.

Waddington (1960), using family selection, was able to obtain a decrease of temperature sensitivity in one of four mutant genes, with one other gene showing a slight response. Using disruptive selection, Waddington and Robertson (1966) have repeated this experiment with the gene *Bar*. They claim increased canalization, but in many of their canalization lines the between-sex variability increased. This suggests, as will be seen in our own results, that each sex under certain conditions may have different canalization zones.

It appears that analyses of the genetic basis of changes of variability need to involve all three aspects: changes of the mean number, intrasex variability, and intersex variability. The experiments discussed below were designed to detect the relationship between canalization and sex dimorphism, and to test the theory that canalization systems may have evolved as a consequence of equalizing male and female phenotypes when a considerable amount of the genotypic control of the formation of bristles is located on the sex chromosomes (Wigan 1949).

II. Material and Methods

The base stocks used in this experiment were two lines selected for extra scutellar bristles and containing $sc^{+}w^{h}$. Erway (personal communication) has shown that these lines (A9 and A18) are homozygous for a gene, termed *x-vert*, which is probably an allele of *pyd* (see Fraser, Erway, and Brenton 1968). Miller (1967) substituted $y^{2}\ sc^{1}\ w^{h}$; *x-vert*. Reciprocal crosses were made of the two stocks; the two progenies were mixed and one generation of random mating allowed to occur. Fifty single-pair matings in which virgin females were used were then established in $\frac{1}{4}$-pint cream jars with standard media. In each generation, 20 flies of each sex per family were scored for scutellar bristle number, and 10 pairs chosen from each of the five families with the lowest sex dimorphism. These were mated at random to form the next generation. Two replicates and one control line were maintained. At the sixth generation of selection, the population size of one of the replicates (R_1) was increased to 100 families although the number of families selected remained at five. Replicate R_2 was discarded at generation 20, and selection was discontinued in R_1 at the 26th generation. This line was then maintained in 10 replicates of sib mating and scored again after 8 generations.

The genes *facet*, *eosin*, *Scutoid*, and *scute*$^{+}$ were continuously backcrossed into R_1 to see if the changes in sex dimorphism of scutellar bristle number were reflected in other mutant genotypes. A number of relaxed selection lines were established during the experiment. One such line, at generation 15, was of the 15 sib-mated lines maintained until generation 25. An experiment to test temperature sensitivity was performed at generation 20 in R_1. Single-pair matings were set up and raised at 18, 24, and 30°C.

III. Results

The generation averages for the two selection lines and the unselected control line are given in Table 1 and summarized in Figures 1 and 2. Probit measures of the width of the different bristle zones are given in Table 2.

Table 1

SCUTELLAR BRISTLE NUMBERS IN SELECTED LINES R_1 AND R_2 AND THE UNSELECTED CONTROL LINE

Generation No.	Mean Scutellar Bristle No.		Sex Dimorphism	Generation No.	Mean Scutellar Bristle No.		Sex Dimorphism
	Females	Males			Females	Males	
	Selected line R_1				Selected line R_2		
0	1·74	0·84	0·90	0	1·65	0·85	0·80
1	1·62	1·10	0·52	1	1·48	0·76	0·72
2	1·59	0·89	0·70	2	1·72	1·00	0·72
3	1·54	0·85	0·69	3	1·75	1·02	0·73
4	1·43	0·83	0·60	4	1·66	1·04	0·62
5	1·71	1·10	0·61	5	1·64	1·07	0·57
6	1·72	1·20	0·52	6	1·66	1·06	0·60
7	1·85	1·35	0·49	7	1·63	1·07	0·56
8	1·88	1·43	0·45	8	1·66	1·15	0·52
9	1·78	1·24	0·54	9	1·38	0·80	0·58
10	1·87	1·43	0·45	10	1·55	0·92	0·63
11	1·95	1·50	0·47	11	1·42	0·66	0·75
12	1·95	1·53	0·42	12	1·39	0·64	0·74
13*a*	2·02	1·70	0·32	13	1·37	0·61	0·75
13*b*	2·05	1·76	0·30	14	1·23	0·57	0·66
14	2·05	1·72	0·33	15			
15	2·12	1·77	0·35	16	1·44	0·73	0·68
16	2·12	1·82	0·30	17	1·17	0·65	0·52
17	2·09	1·83	0·25	18	1·46	0·68	0·76
18	2·07	1·80	0·26	19	1·62	0·96	0·66
19	2·08	1·88	0·21	20	1·45	0·84	0·61
20	2·26	1·93	0·33				
21	2·25	1·92	0·33		Unselected control line		
22				0	1·68	0·85	0·85
23	2·34	1·99	0·35	1	1·80	1·04	0·76
24	2·20	1·98	0·24	3	1·70	0·98	0·72
25	2·28	1·93	0·35	5	1·71	0·87	0·84
				7	1·72	0·90	0·82
				9	1·69	0·75	0·94
				11	1·68	0·85	0·83
				21	1·73	0·79	0·94
				24	1·76	1·01	0·75
				26	1·78	0·81	0·97

Selection for a decrease of the sex dimorphism was effective in the R_1 line up to generation 17, after which the response tended to level off. The probit values for this line show a consistent increase in width of the 2-bristle zone and the line showed little tendency to leave this zone.

Selection was ineffective in the R_2 line. A possible explanation for this lack of response is that there are three zones of low sex dimorphism at 0, 2, and 4 bristles, and R_2 between the 0- and 2-bristle zones became stranded as a result of selection of families from both zones, i.e. families with means close to 2 and families with means close to 0 were mated *inter-se* and the resulting population always had an intermediate mean. Table 3 shows the average means of selected R_2 families in a sample of generations. A cut-off point of 1·30 was chosen because the greater probit width of the 2-bristle zone makes this point a more accurate divider of the 0 and 2 zones than the arithmetic mean of 1.

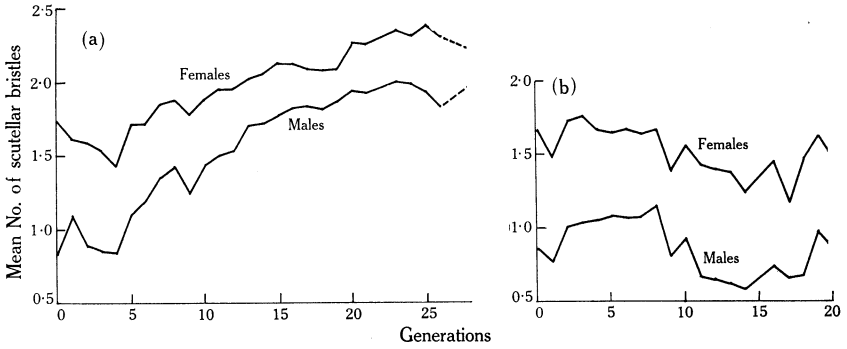

Fig. 1.—Selection response of the replicate lines R_1 (*a*) and R_2 (*b*).

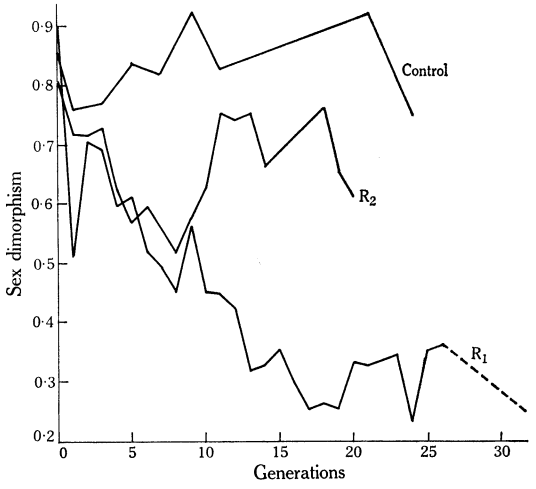

Fig. 2.—Sex dimorphism of the scutellar bristle number in the replicate lines and the unselected control line.

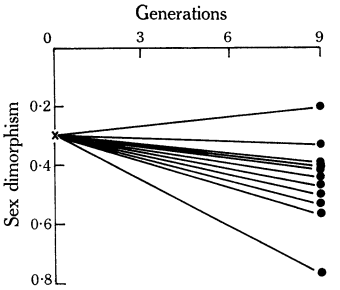

Fig. 3.—Change in sex dimorphism in sib-mated relaxed selection lines (see text).

Mass-mated relaxed selection lines regressed back to the control sex-dimorphism values. This did not occur by regression of the mean number of scutellar bristles along the same path as the response to directed selection. Instead the mean number of bristles in females tended to increase whilst the bristle number in males either remained constant or regressed only slightly (see Table 4).

TABLE 2

PROBIT WIDTHS OF THE DIFFERENT BRISTLE ZONES IN SELECTED LINES AND THE UNSELECTED CONTROL LINE

Generation No.	Male Mean ($\bar{x}$)	Width of Bristle Zones in Males			Female Mean ($\bar{x}$)	Width of Bristle Zones in Females		
		1	2	3		1	2	3
				Selected line R_1				
0	0·84	1·0	2·32		1·74	1·20	2·03	1·07
1	1·10	1·18			1·62	1·01	2·39	
2	0·89	1·06	2·20		1·59	1·05	2·39	
3	0·85	0·97	1·85		1·54	0·99	2·27	
4	0·84	1·07	1·70		1·43	1·14	2·82	
5	1·10	1·06	2·18		1·70	1·17	2·66	
6	1·20	1·02	2·17		1·72	0·86	2·49	0·81
7	1·35	1·16	2·18	0·77	1·85	1·00	2·91	1·14
8	1·43	1·01	2·44		1·88	0·99	2·66	1·15
9	1·24	1·01	2·26		1·78	0·92	2·71	0·83
10	1·43	1·06	2·47		1·87	1·04	2·86	
11	1·50	1·08	2·24		1·94	1·06	2·65	
12	1·53	1·12	2·71		1·95	1·06	2·65	
13	1·70	1·20	2·78		2·02	1·35	3·23	1·33
14	1·76	1·25	2·59		2·05	1·24	3·13	1·34
15	1·72	1·18	2·50		2·05	0·93	3·21	
16	1·77	1·16	2·77		2·12	0·92	3·18	
				Selected line R_2				
1	0·85				1·65	0·99	2·01	1·31
2	0·76	1·14			1·48	1·20	2·67	
3	1·00	1·07	2·00		1·72	1·14	2·29	1·15
4	1·02	1·20	2·01		1·75			
5	1·04	1·13	1·83		1·66	1·19	2·29	
6	1·07	1·21	2·11		1·64	1·19	2·19	1·02
7	1·06	1·14	2·10		1·66	1·01	2·43	
8	1·07	1·21	2·07		1·63	1·25	2·32	
9	1·15	1·23	2·24		1·66	1·14	2·33	1·01
10	0·80	1·26			1·38	1·39	2·86	
11	0·92	1·07	1·95		1·55	1·23	2·41	
12	0·66	1·27	1·71		1·39	1·09	2·38	
13	0·64	1·17			1·39	1·09	2·38	
14	0·61	1·20			1·37	1·22	2·72	
15	0·56	1·15	1·63		1·23	1·26	2·51	
				Unselected control line				
1	—	1·16			—	1·21	1·89	
3	—	1·25			—	1·56	2·37	
5	0·87	1·08			1·71	1·01	2·61	
7	0·90	1·17	1·94		1·72	1·14	2·78	0·82
9	0·75	1·08			1·69	1·15	2·82	
11	0·85	1·09			1·68	0·99	2·88	

Sib-mated relaxed selection lines showed only a partial regression of sex dimorphism to control-line values (see Fig. 3). As a comparison, a set of unselected lines with similar values of the bristle means to those in the sib-mated relaxed selection lines were scored, and the results are given in Table 5.

It would appear that while natural selection is not opposing the increase in bristle number it is opposing the decrease in sex dimorphism. The result of this is that when selection is relaxed the lines return toward the original sex dimorphism while the combined bristle mean of the sexes changes very little.

TABLE 3

MEAN SCUTELLAR BRISTLE NUMBERS OF THE FIVE SELECTED FAMILIES IN SEVEN TYPICAL R_2 FAMILIES ILLUSTRATING THE TENDENCY TO BIMODALITY

Generation No.	Average No. in Selected Families	
	High (toward 2-bristle zone)	Low (toward 0-bristle zone)
4	1·55, 1·45, 1·40	0·90, 0·90
7	1·80	1·20, 1·15, 1·10, 0·90
9	1·70, 1·55, 1·50	1·10, 1·05
13	1·45, 1·35	1·20, 1·20, 1·10
15	1·75, 1·55, 1·45, 1·40	1·20
17	1·6, 1·55	1·20, 1·20, 1·20

TABLE 4

FINAL MEAN SCUTELLAR BRISTLE NUMBERS OF THE MASS-MATED RELAXED SELECTION LINES

R_1 Generation No.	Means after Five Generations of Relaxed Selection		
	Females	Males	Sex Dimorphism
8	1·50	1·09	0·41
10	1·42	0·90	0·52
14	1·56	0·94	0·62
16	1·91	1·38	0·53
20	1·93	1·47	0·46
22	2·01	1·46	0·55

The genes *white-eosin* and *facet* when backcrossed into R_1 and the control showed identical expression in both lines with no alteration of their normal sex dimorphism. *Scutoid* is a second-chromosome dominant which acts as a homozygous lethal. *Scutoid* has a similar effect to *scute*[1] and the two genes interact to completely remove all scutellar bristles. However, by backcrossing *Scutoid* into the *scute*[+] backcross (described below) it was possible to evaluate the effects of selection on this gene. It was found that a considerable reduction in the sex dimorphism associated

with this gene had been achieved. The mean scutellar bristle numbers in two independent experiments scored after backcrossing for 12 generations are presented in the following tabulation:

Expt. No.	Genotype	Female Mean	Male Mean	Sex Dimorphism
1	Selection line R_1	0·61	0·50	+0·11
	Control	0·78	0·53	+0·25
2	Selection line R_2	0·60	0·85	−0·25
	Control	0·74	0·63	+0·11

In the first experiment only whole bristles were scored but, unlike *scute*, *Scutoid* has a tendency to produce bristle vestiges and empty sockets as well as removing bristles completely. Since such vestiges suggest a residual bristle-forming activity at these sites, these were scored as whole bristles in the second experiment. This resulted in a marked increase in the male means, showing that this effect was primarily present in males.

TABLE 5

RANGE OF SEX DIMORPHISM SHOWN BY *scute*[1] LINES WITH COMPARABLE FEMALE MEAN SCUTELLAR BRISTLE NUMBERS

Experiment	Female Mean	Male Mean	Sex Dimorphism
Rendel 1959	2·40	1·90	0·50
	2·40	1·70	0·70
	2·26	1·27	0·99
	2·63	1·67	0·96
	2·52	1·68	0·84
Payne 1920	2·40	1·53	0·87
Miller 1967	2·42	2·40	0·38
	2·36	2·19	0·17
	2·44	1·87	0·57
Druger 1967	2·50	0·99	1·51
	2·35	1·50	0·85
	2·40	1·20	1·20
From Figure 4	2·40	1·43	0 97

The effect of selection at the *scute* level upon expression at the *scute*$^+$ level was negligible, as shown in the following tabulation, which records the mean scutellar bristle numbers of the *sc*$^+$ backcross line:

No. of Backcross Generations	Female Mean	Male Mean	Sex Dimorphism
0	6·59	5·76	0·83
7	5·35	4·93	0·43
14	5·60	5·02	0·56
21	6·36	5·54	0·82

This is in general agreement with Rendel (1959) and Fraser (1966) who found that extremely intense selection at the *scute* level is required before any correlated response is noted at the *scute*$^+$ level.

Although the probit values calculated for R_1 (Table 2) indicate that there has been an increase in canalization, the critical test is to see whether the line is now less sensitive to temperature. Quite the opposite was found to be the case. At temperatures of 18, 24, and 30°C the sex dimorphism in the control line was 1·04, 0·77, and 0·70 respectively, whilst that in selection line R_1 was 0·78, 0·33, and 0·56 respectively. Thus, line R_1 was much more sensitive to high temperature than the control line and about equally sensitive to low temperature. The means are given in Figure 4 and the response of the sex dimorphism can be gauged from the distance

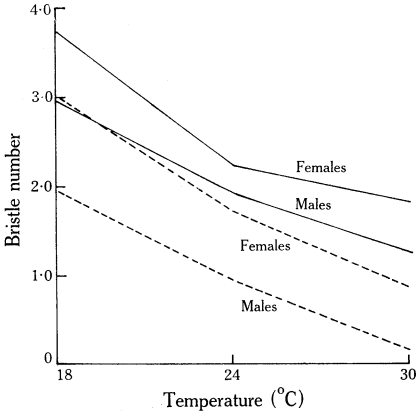

Fig. 4.—Response to temperature of R (———) and control lines (– – –).

between the male and female response lines. The sex dimorphism is lower in R_1 at all three temperatures, indicating a conservation of selection response, but there is no evidence at all of canalization of bristle number. The sex dimorphism was responsive to temperature only in line R_1.

IV. Conclusions and Discussion

We have shown that sex dimorphism of the *scute* gene is amenable to selection but only under certain conditions. These require the avoidance of artificial adaptive peaks created by selection in zones flanked on either side by zones of low sex dimorphism. While this type of disruptive selection may reduce the variability of the population, it will usually increase the between-sex variability.

The evidence from the *scute*$^+$ and mutant substitution lines indicate that the induction of selectable variation by mutant genes is not a sufficient condition to allow gains from such selection to be realized in non-mutant genotypes. The fact that polygenic variation associated with one gene has little effect in the presence of other alleles of that gene or when other loci of similar effect are present has been known for some time (Haskell 1943; Cocks 1954), although there have been some successful attempts (Fraser and Kindred 1962) in which major genes were used to expose usable genetic variation. It would seem wiser to first attempt to expose underlying variation by environmental stress (see Waddington 1961) before attempting to use major genes because of the possibility of low polygenic overlap.

The degree of polygenic overlap between non-allelic genes could be considered as a reflection of the importance of generalized modifying genes affecting the character

under consideration. A low overlap would therefore suggest that locus-specific modifiers are more critical. Such specific modifiers may well be analogous to gene control systems as described in bacteria. Kindred (1967) found that the modifiers affecting *Tabby*, a vibrissan mutant in the mouse, are effective in the *crinkled* mutant genotype, but not in the presence of the mutant *Ragged*. She favoured the hypothesis that *Ragged* affects a metabolic pathway separate from that affected by *Tabby* and *crinkled*. Considerations stemming from data on polygenic overlap should lead to critical conclusions concerning the relative importance of major genes and their control mechanisms in quantitative inheritance. For instance, we might expect that modifiers acting before translation would affect mutant genes and their wild-type alleles similarly, whilst post-translational modifiers would affect each differently. The finding in this paper and others (cited above) of low polygenic overlap between a mutant gene and its wild-type allele would then be indicative of the major role in this system being played by post-translational (but locus or gene product specific) modifiers. However, despite this projection such questions must, for the moment, remain largely unanswered. In the meantime, experiments are being conducted in which a suppressor gene is being used, which presumably acts at translation to further dissect such situations in *Drosophila*.

The concept of canalization involves the suppression of reactions to environmental stress. Consequently, a critical test of a change of canalization is to determine whether any change has occurred in sensitivity to environmental stress. Rendel and Sheldon (1960) have exposed cultures to different temperatures and thus shown that increased canalization has a correlated effect: a decreased response to temperature variation. Rendel and Sheldon (1960) state that whatever is responsible for the sex differences seen in *scute* flies appears to have far less effect after selection for low variance. Our results show that the reverse does not hold. It was possible, by selection, to decrease sex dimorphism in order to effect a reduction of the variability of bristle number at a single temperature, but under conditions of temperature stress there was a release of variation. There is strong evidence for the absence of a mammalian-type dosage compensation mechanism in *Drosophila* (Muller and Kaplan 1966; Young 1966; Lee 1968). Muller and Kaplan (1966) invoke sex-limited and sex-linked modifying genes, but if these exist for the gene *scute*^{1} our selection experiment has failed to uncover them. Furthermore, Parsons (1960) found no difference in homeostatic ability between the sexes which would be expected if sex-linked genes contributed to stability. The finding of Fraser and Green (1964) that two doses of *scute*^{1} in the male causes considerable overcompensation is evidence that some compensatory mechanism is operative, and this, together with the high developmental component of variance reported for scutellar bristles (Latter 1963, 1964), supports a developmental interpretation of dosage compensation in *Drosophila* (see Lee 1968). Harrison (1953) observed a reversal of sex dimorphism in a disruptive selection experiment involving chaetae number. This was attributed to an exchange between the X and Y chromosomes such that the resulting Y chromosome made a positive contribution to bristle number, although the possibility that sex-limited genes were responsible was not entirely discounted.

A number of phenodeviants occurred repeatedly in the latter generations of our experiment indicating that normal development had been disrupted. Such

developmental instabilities are further evidence of failure to achieve a generalized canalization by concentrating only upon sex dimorphism. Canalization can now be seen as an extremely complex state largely independent of such things as temperature fluctuation, genetic pattern, and sex dimorphism, although affecting all of them.

V. References

Blanc, R. (1946).—Dominigenes of the vestigial series in *Drosophila melanogaster*. *Genetics, Princeton* **31**, 395–420.

Cocks, B. (1954).—Polygenic systems controlling the expression of the major mutant genes which affect chaetae number in *D. melanogaster*. *Heredity, Lond.* **8**, 13–43.

Druger, M. (1967).—Selection and the effect of temperature on scutellar bristle number in *Drosophila*. *Genetics, Princeton* **56**, 39–47.

Fraser, A. S. (1963).—Variation of scutellar bristles in *Drosophila*. I. Genetic leakage. *Genetics, Princeton* **48**, 497–514.

Fraser, A. S. (1966).—Variation of scutellar bristles in *Drosophila*. XII. Selection in *scute* lines. *Aust. J. biol. Sci.* **19**, 147–54.

Fraser, A. S., Erway, L., and Brenton, W. (1968).—Variation of scutellar bristles in *Drosophila*. XIV. Effects of temperature and crowding. *Aust. J. biol. Sci.* **21**, 61–74.

Fraser, A. S., and Green, M. M. (1964).—Variation of scutellar bristles in *Drosophila*. III. Sex dimorphism. *Genetics, Princeton* **50**, 357–62.

Fraser, A. S., and Kindred, B. M. (1962).—Selection for an invariant character, vibrissa number, in the house mouse. III. Correlated response. *Aust. J. biol. Sci.* **15**, 188–206.

Harrison, B. J. (1953).—Reversal of a secondary sex character by selection. *Heredity, Lond.* **7**, 153–64.

Haskell, G. M. L. (1943).—The polygenes affecting the manifestation of *scute* in *Drosophila melanogaster*. *J. Genet.* **45**, 269–76.

Kindred, B. (1965).—Selection for temperature sensitivity in *scute Drosophila*. *Genetics, Princeton* **52**, 723–8.

Kindred, B. (1967).—Selection for an invariant character, vibrissa number, in the house mouse. V. Selection on *non-Tabby* segregants from *Tabby* selection lines. *Genetics, Princeton* **55**, 365–73.

Latter, B. D. H. (1963).—Genetic homeostasis and the theory of canalisation. In "Statistical Genetics and Plant Breeding". NAS–NRC Publ. No. 982, pp. 455–62.

Latter, B. D. H. (1964).—Selection for a threshold character in *Drosophila*. I. An analysis of the phenotypic variance on the underlying scale. *Genet. Res.* **5**, 198–210.

Lee, G. L. (1968).—Dosage compensation as a developmental phenomenon. *Genet. Res.* **11**, 115–18.

Miller, D. H. (1967).—The effect of substitution at the *scute* locus on the expression of responses to selection. M.Sc. Thesis, University of California, Davis.

Muller, H. J., and Kaplan, W. D. (1966).—The dosage compensation of *Drosophila* and mammals as showing the accuracy of the normal type. *Genet. Res.* **8**, 41–59.

Parsons, P. A. (1960).—Homeostasis. *Nature, Lond.* **186**, 411.

Payne, F. (1920).—Selection for high and low bristle number in the mutant strain "*reduced*". *Genetics, Princeton* **5**, 501–26.

Plunkett, C. R. (1932).—Temperature as a tool of research in phenogenetics: methods and results. Proc. 6th Int. Congr. Genet., Ithaca, N.Y. Vol. 2, pp. 158–60.

Rendel, J. M. (1959).—Canalization of the *scute* phenotype of *Drosophila*. *Evolution* **13**, 425–39.

Rendel, J. M., and Sheldon, B. L. (1960).—Selection for canalization of the *scute* phenotype in *Drosophila melanogaster*. *Aust. J. biol. Sci.* **13**, 36–47.

Rendel, J. M., Sheldon, B. L., and Finlay, D. E. (1966).—Selection for canalization of the *scute* phenotype. II. *Am. Nat.* **100**, 13–32.

Rendel, J. M., Sheldon, B. L., and Finlay, D. E. (1966).—Selection genetics—*Drosophila*. A. Rep. Anim. Res. Lab. CSIRO, Aust., 1965–66. pp. 2–3.

WADDINGTON, C. H. (1960).—Experiments on canalizing selections. *Genet. Res.* **1**, 140–50.

WADDINGTON, C. H. (1961).—Genetic assimilation. *Adv. Genet.* **10**, 257–92.

WADDINGTON, C. H., and ROBERTSON, E. (1966).—Selection for developmental canalization. *Genet. Res.* **7**, 303–12.

WIGAN, L. G. (1949).—The distribution of polygenic activity on the *X*-chromosome of *Drosophila melanogaster*. *Heredity, Lond.* **3**, 53–66.

YOUNG, W. J. (1966).—*X*-linked electrophoretic variation in 6-phosphogluconate dehydrogenase. *J. Hered.* **57**, 58–60.