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Selection for Increased Abdominal Bristle Number in *Drosophila melanogaster* with Concurrent Irradiation

II. Populations Derived from an Outbred Cage Population

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Summary. Replicate lines, each initially with one hundred pairs of parents selected at 50% intensity, were derived from the Canberra strain. In later generations population size was reduced and selection intensity increased. Three lines were selected without irradiation and five with 1000 *r* *X*-rays per generation for thirty generations. Selection was continued until generation 66. Long-term responses were similar in unirradiated and irradiated lines, and there was evidence that genes with large effects influenced response patterns in both groups of lines.

Investigation of the effects of radiation on selection response in outbred species derives its stimulus from the occurrence of plateaux in populations of domestic and laboratory animal species selected for quantitative characters. Mutagen treatment to induce new variation is one possible method of raising these barriers to selection progress, but experimental irradiation of plateaued populations has given conflicting results. Scossiroli (1954) obtained a very large increase in sternopleural bristle number in one irradiated line derived from a plateaued population of *Drosophila melanogaster*, but little or no response in other irradiated lines. Clayton and Robertson (1964) obtained only small responses in lines plateaued for high and low sternopleural or sternital bristle number. Abplanalp, Lowry, Lerner and Dempster (1964) irradiated chicken sperm for seven generations in a flock derived from a line plateaued for egg number. Subsequent selection response was very similar in the irradiated and control flocks. Roberts (1967), using a line apparently plateaued for six-week body weight in mice, irradiated males with a single dose of 600 *r* *X*-rays and continued selection, but obtained no substantial advance.

In the above experiments radiation treatments were applied to populations which were at apparent plateaux. An alternative approach is to begin irradiation treatments early while response is still occurring; this may increase both the rate of response and the plateau level. Jones (1967) selected for thirty generations for increased abdominal bristle number in a group of lines derived from a non-inbred cage population of *D. melanogaster*, with concurrent irradiation for zero, two, ten or thirty generations. He attributed the often considerable extra response in irradiated lines to mutation occurring at a few loci with large effects on bristle number. The experiment described here extends this work by considering, on a larger scale, the long term effects of concurrent irradiation on selection response in lines with relatively large population size.

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Materials and Methods

Base population: The Canberra wild-type strain of *D. melanogaster* was used (Sheridan, Frankham, Jones, Rathie and Barker 1968). Selection lines were derived from the bulked progeny of a large sample (twelve hundred pairs) taken from a cage population in May, 1965. In work reported by Frankham, Jones and Barker (1968a, b) and Jones, Frankham and Barker (1968), selection lines were initiated by sampling from the same population cage approximately one year earlier and the same quantitative character was subjected to mass selection under a range of population sizes and selection intensities. Their results are compared with the response obtained in the experiment reported here.

Selection programme: Mass selection for increased number of bristles on one abdominal sternite (fifth in females, fourth in males) was carried out in eight selection (*S*) lines: three (*SO*.1, *SO*.2 and *SO*.3) received no radiation treatments and five (*SR*.1, *SR*.2, *SR*.3, *SR*.4 and *SR*.5) received treatments for the first thirty generations. The selection regime was changed several times during the course of the experiment (Table 1). Selection was on a within-culture basis (Hollingdale and Barker 1971).

At generations 10, 20, 30, 40, 53, 57, 61, and 65 relaxed selection sublines were taken from the selection lines. Randomly chosen flies were cultured under crowded conditions, with population size the same as in the selection lines at the corresponding generation (Table 1). No radiation treatments were given. After four generations of relaxation, uncrowded cultures were set up identical to those of the selection lines and forty pairs of progeny were scored for abdominal bristle number in each relaxed selection line to determine the effect of five generations of relaxation on mean bristle number. In sublines commenced at generation 57, bristle number was also scored after two, ten and twenty generations of relaxation.

Until generation 10, unselected control lines were maintained under the same conditions, with the same population size as the selection lines, although mean bristle

numbers for these lines were based on only fifty pairs. Of the unselected (*U*) control lines, three, the *UO* lines, received no radiation and six, the *UR* lines, were irradiated each generation.

Culture conditions: Flies were cultured on a dead yeast fortified medium (medium *F* of Claringbold and Barker 1961) in 5 oz cream bottles, with either twenty pairs (generations 0-23) or five pairs (generations 24–66) of parents per bottle. For twenty pair cultures, parents remained in the culture bottles for one and a half to two days, and for five pair cultures, they remained in the bottles for three days, as these times had given satisfactory results in a preliminary experiment on crowding effects. (There was no detectable effect of crowding on bristle number, although the experiment was not designed to reveal differences of less than one bristle between crowding treatment means.) Cultures were kept at 25 ± 0.5 °C and 65 ± 5% relative humidity in a room lit twelve hours per day (6 a. m. to 6 p.m.).

Irradiation: Prior to mating, adults of both sexes in the irradiated control and selection lines were treated each generation with 1000 *r* *X*-rays (irradiation procedure as in Hollingdale and Barker 1971).

Lethal tests: The selection lines were tested for presence of second and third chromosome lethals at generation 32 (generation 29 in line SR.3). The technique used was similar to that of Brown and Bell (1961). Their tester stock was modified by substituting Canberra wild type chromosomes for chromosomes I and IV. The stock, based on the *Cy* and *Ubx* multiple inversions, was

$$+;\ In\text{-}SM^1,\ al^2\ Cy\ sp^2/dp\ b\ Pm\ ds^{33K};$$
$$Ubx^{130}\ e^s/C\ Sb;\ +\ .$$

In each line seventy-two (eighty in line *SR*.3) initial matings were set up, sampling equal numbers of males and females from the selection line, identified according to source culture and scored for bristle number. In the progeny of the third generation of the test crosses, absence of normal-winged flies indicated that the second chromosome was lethal and absence of flies with normal halteres indicated a lethal third chromosome. Cultures giving few progeny in this generation were retested if necessary. No attempt was made to count the total number of progeny, so that lethality was defined by complete absence of one phenotypic class in a culture with no less than about thirty progeny. Lethal chromosomes were maintained as balanced stocks for later allelism testing. Some semi-lethal chromosomes with very low viability were classed as absolute lethals on the basis of the initial lethal testing, but their semi-lethal nature became apparent during allelism testing, and examination of large numbers of progeny from the balanced stocks usually confirmed the semi-lethality of the chromosomal homozygotes.

Allelism testing of lethal genes: Balanced lethal stocks derived from one selection line were cross-mated to determine if they carried allelic lethal genes. As the aim was to identify high-frequency lethals rather than to give a complete description of all lethals, not all combinations were tested. Generally a subset of the lethal stocks was fully tested, and the allelic groups identified. Suitable crosses were then made to relate these groups to corresponding groups found in other subsets derived from the same selection line. Full details of the combinations tested for chromosomes II and III of the eight selection lines and the results of these crosses were reported by Hollingdale (1969). All chromosome II lethals at appreciable frequencies in different lines were also cross tested.

Results

Analysis of base population: Abdominal bristle number scores for generation 0, prior to any radiation treatments, were used to estimate the base population phenotypic parameters. Means and standard deviations were very similar to those reported by Sheridan *et al.* (1968), although the mean bristle number for females, 22.57 ± 0.05, was a little lower than their estimate. The distribution of bristle scores in both sexes was normal. For one segment abdominal bristle number Sheridan *et al.* (1968) estimated that 10%, 6% and 18% of the phenotypic variance was due to additive autosomal, additive sex-linked and epistatic effects respectively.

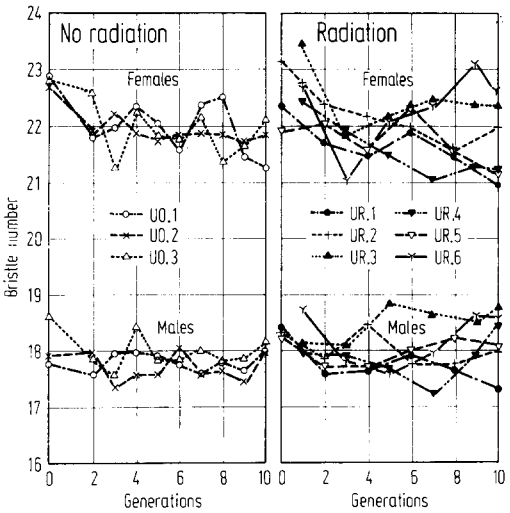

Fig. 1. Mean bristle number for each sex in the unselected control lines. Irradiated control lines (1000 *r* *X*-rays per generation) were scored every second generation

Changes occurring in control lines: Mean bristle numbers for females and males of the unselected lines are shown in Figure 1. The early decrease in bristle number, probably environmental in origin, must be considered as a source of bias in the mean bristle number of the selection lines over the first few generations. A similar but opposite trend in unselected control line means was reported by Frankham *et al.* (1968a); they also concluded that variation in environment was probably the cause. In contrast to results obtained with control lines derived from an inbred line (Hollingdale and Barker 1971), there was no indication that the radiation treatment was itself affecting bristle score. This is in agreement with results of the preliminary experiment on the effects of larval crowding on bristle score in the Canberra strain.

Phenotypic standard deviations for bristle number in individual control lines and average standard deviations (calculated from mean variances) for replicate lines within each radiation treatment were calculated. There was no apparent difference between the mean standard deviations of the unirradiated and irradiated groups. Over the ten generation period the average standard deviation in the irradiated group was 2.04 for females and 1.78 for males as compared with values of 2.00 and 1.73 in the unirradiated group.

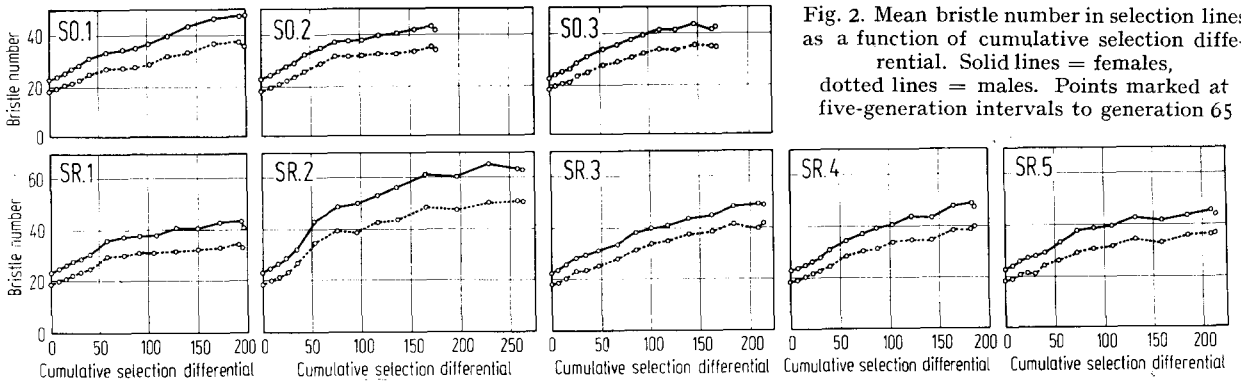

Fig. 2. Mean bristle number in selection lines as a function of cumulative selection differential. Solid lines = females, dotted lines = males. Points marked at five-generation intervals to generation 65

However, comparing the last three generations only, the average standard deviation in the irradiated lines was about 0.2 bristles higher than in the unirradiated lines. Clayton and Robertson (1964) also found a low rate of increase of phenotypic variance in irradiated unselected strains derived from three inbred lines.

Patterns of response in selection lines: Mean bristle number for females and males in the unirradiated and irradiated selection lines are shown in Figure 2 plotted as a function of cumulative selection differential, to compensate for changes in selection intensity. To simplify the graphs, the points were plotted at five generation intervals to generation 65, then the last generation (generation 66). Large generation-to-generation fluctuations in bristle number would make between line comparison difficult if more detail were included.

There was variation between lines in the total selection differential, level of response at generation 66, and the shape of the response curve. Response patterns were similar in females and males. In unirradiated lines, the total selection differential (average for females and males) was a little more than 200 bristles in line *SO*.1, and about 175 bristles in lines *SO*.2 and *SO*.3. Mean bristle numbers for females over the last six generations were 47.5, 42.4 and 41.8 bristles in lines *SO*.1, *SO*.2 and *SO*.3 respectively.

Response in line *SO*.1 occurred in two stages (Figure 2): the initial rate of response was maintained until about generation 30, then there was a ten generation period of slow response followed by a second period with a high rate of response. Response was slow again over the last five generations of selection. There were no mutations with visible effects on the phenotype in this line.

In line *SO*.2, the rate of response became slower after about generation 35 but some response may still have been occurring at the conclusion of the experiment. A second chromosome recessive mutation, *scabrous-like* (Barker and Hollingdale 1970), became frequent in this line. This mutant is not allelic with *scabrous, sca* (Lindsley and Grell 1968).

In *SO*.3 there was very little response in the last 10 to 15 generations. There was some variation in the rate of response earlier in the selection programme, associated with the increase in frequency of a third chromosome recessive mutation, *dark hairy margin* (Barker and Hollingdale 1970). This mutant became fixed in the line at generation 39.

Response patterns of four of the irradiated lines were similar to those of the unirradiated lines. The selection response in the remaining irradiated line, *SR*.2, was much greater than in any other line. This line became homozygous for the second chromosome gene *scabrous*, which has a very large effect on abdominal bristle number. The total applied selection differentials were 195, 215, 185 and 210 bristles for lines *SR*.1, *SR*.3, *SR*.4 and *SR*.5 respectively and 260 bristles for line *SR*.2. Mean bristle numbers for females over the last six generations were 41.6, 48.5, 47.7 and 44.6 bristles for *SR*.1, *SR*.3, *SR*.4 and *SR*.5 respectively and 63.9 bristles for *SR*.2.

In line *SR*.1, response was rapid until generation 30 and then continued at a very slow rate. Line *SR*.3 showed a gradual slowing in rate of response with no abrupt discontinuities in the response pattern. There was also a gradual slowing of the response rate in line *SR*.4 after about generation 30, and in line *SR*.5 after generation 35.

The general pattern of response conformed to the expectation that response rate would decline with continued selection, *i.e.* there was an asymptotic approach to the selection limit. An exponential response curve of this type was predicted by Robertson (1960) for limits caused by loss of additive genetic variance and by James (1962) for limits caused by decline in fitness. Some exceptions to this pattern of declining realized heritability have been mentioned. There was another exception: response rates in the irradiated lines (except *SR*.2) were higher in the period of selection at 20% intensity (Table 1) than in the immediately preceding period, whereas in the unirradiated lines the rates were similar in the two periods or lower in the later period. These high response rates in the irradiated lines could be due to selection of newly induced mutations which had not been effectively utilized by the previous period of

Table 1. *History of each replicate selection line*

Generation number	Number of pairs scored	Percentage selected	Population size (number of pairs of selected parents)	Number of cultures	Expected standardized selection differentials[+]
0–10	200	50.0	100	5	0.78
11–23	160	50.0	80	4	0.78
24–31	100	20.0	20	4	1.35
32–45	60	33.3	20	4	1.03
46–66	100	20.0	20	4	1.35

[+] From Becker (1964).

selection at 50% intensity. The rate of increase in frequency of a selected allele depends on its frequency, and is low when the gene frequency is close to zero (or unity, Falconer 1954, 1960). Irradiated lines at generation 24 (when the selection pressure was increased and the population size simultaneously reduced) probably contained a reservoir of induced mutations still at low frequencies. These mutations, plus others affecting bristle number induced by the continuing radiation treatments, would be increased in frequency more rapidly under the higher selection pressure. By generation 24 in unirradiated lines only those genes with small effects on bristle number would be still at low frequencies, as most initially rare genes present in original samples from the base population would have reached moderate frequencies earlier in the selection programme, and recent recombinants or spontaneous mutations would not be sufficiently numerous to have much effect.

The response pattern in line *SR*.2 showed a marked acceleration in the rate of response from generation 15 to generation 25, when *scabrous* was increasing in frequency. This gene was first definitely detected as the homozygote at generation 15 and reached fixation in the population at generation 24. After generation 25, response continued to be rapid for some time but then became slower. Mean bristle number at this time was over 60 bristles in females and close to 50 bristles in males. Difficulty in scoring such high numbers may have affected the accuracy of selection to some extent, but experimental error in individual bristle counts would have been only a small proportion of the phenotypic standard deviation.

The initial rates of response for all lines were underestimated since unselected control line means declined over the first few generations. Estimates of realized heritability free from this source of bias were calculated using the response to selection measured as the deviation from the average of control lines in each generation. These estimates, using the average response in females and males, are presented in Table 2 and are comparable to estimates given by Frankham *et al.* (1968a) for other selection lines from the same base population.

Table 2. *Realized heritability (%) for generations 0 to 10 in each line (averaged over sexes)*

	Line	Realized heritability
Unirradiated lines:	SO.1	22.7 ± 1.9
	SO.2	24.0 ± 1.4
	SO.3	20.7 ± 1.4
	Average	22.5
Irradiated lines:	SR.1	22.6 ± 2.0
	SR.2	23.2 ± 1.2
	SR.3	24.7 ± 1.2
	SR.4	17.5 ± 1.4
	SR.5	22.9 ± 1.9
	Average	22.2

Selection differentials: Average selection differentials applied to the lines for each period in the selection programme and the expected selection differentials are presented in Table 3. Expected selection differentials were calculated from the average phenotypic standard deviation in the base population ($s = 1.955$) and the standardized selection differentials (Table 1). Selection differentials were slightly less than expected in the initial period (generations 0 to 10), and slightly greater in most lines in the second period of 50% selection, but lines *SR*.2 (segregating for *scabrous*) and *SR*.3 had considerably larger selection differentials during this second period. In later periods, selection differentials were larger than expected in all lines, owing to increased phenotypic variances.

Changes in variance: Phenotypic variance increased in all lines. With the exception of line *SO*.1 after generation 50, variances were higher in irradiated lines than in the unirradiated. The phenotypic standard deviation and the coefficient of variation

Table 3. *Average selection differentials in the unirradiated (SO) and irradiated (SR) selection lines, for uniform selection regime periods (Table 1)*

Generations	Average selection differentials								
	Expected	*SO*.1	*SO*.2	*SO*.3	*SR*.1	*SR*.2	*SR*.3	*SR*.4	*SR*.5
0–10	1.52	1.48	1.51	1.44	1.45	1.53	1.51	1.42	1.49
11–23	1.52	1.62	1.58	1.52	1.62	2.47	2.07	1.60	1.53
24–31	2.64	3.49	3.21	3.41	3.45	4.45	4.03	3.44	3.10
32–45	2.02	2.85	2.78	2.69	3.22	3.95	3.24	3.04	3.54
46–66	2.64	4.86	3.60	3.52	4.30	6.06	4.66	3.94	5.10

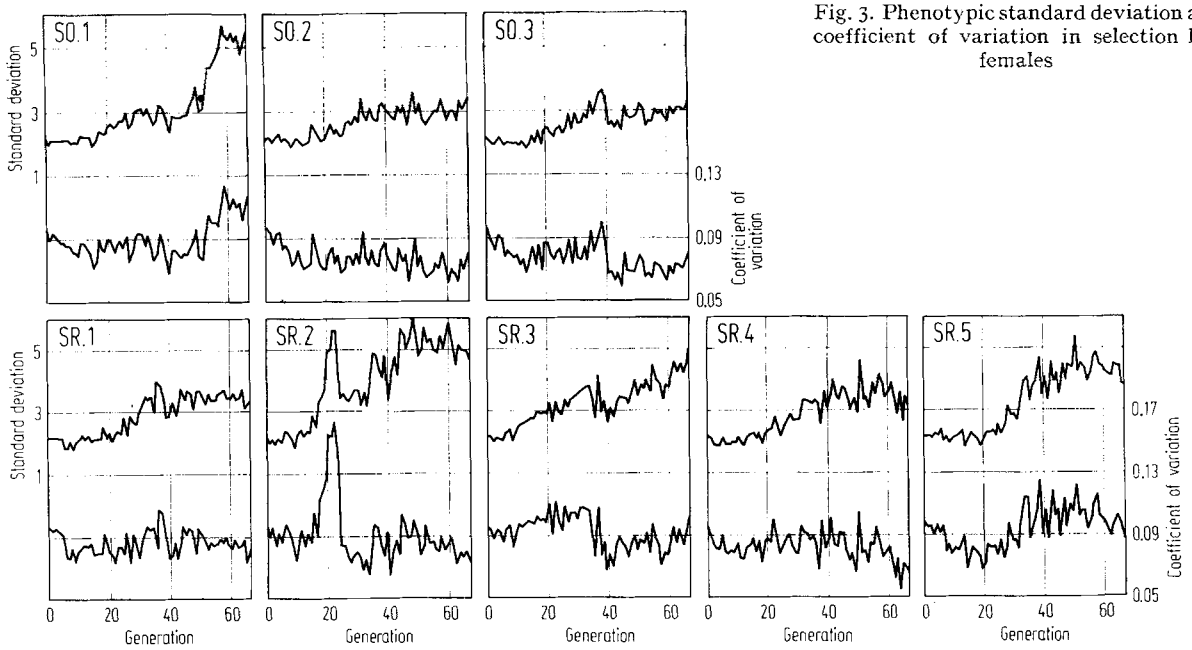

Fig. 3. Phenotypic standard deviation and coefficient of variation in selection line females

(equivalent to the standard deviation on a logarithmic scale, Wright 1952, Lewontin 1966) in females of each line are shown in Figure 3 for every generation. The phenotypic standard deviation in males was generally lower and the coefficient of variation higher, but patterns of change were similar to those for females. Comparing standard deviations and coefficients of variation, a part of the variance increase could be attributed to scale effects, since coefficients of variation did not generally increase as the mean increased in later generations. However, a log transformation of the data would not have resulted in a constant variance. The coefficients of variation showed different patterns of change in each line and these could be related to changes in rate of response to selection and, in some cases, to effects of individual genes, *e.g.* the abrupt decrease in coefficient of variation in line *SO*.3 occurred when *dark hairy margin* was fixed at generation 39, and the peak about generation 20 in line *SR*.2 was caused by segregation of *scabrous* homozygotes at intermediate frequencies.

Changes in fitness: No fitness components were measured, but general productivity of the selection lines was observed. Decline in productivity did not prevent selection being made at full intensity, except in line *SR*.4. In this line insufficient progeny were obtained from some cultures in some generations after generation 51. Productivity was improved on relaxation of selection or after periods of less intense selection, *e.g.* at generation 45 (at the end of the period of 33.3% selection intensity) it would have been possible to select at 10% intensity in all lines.

Relaxation of selection: The change in mean bristle number from the level of the selection line at the commencement of relaxation is shown in Figure 4 for females after five generations of relaxation. (The relaxed subline derived from line *SR*.1 at generation 10 was accidentally lost.) Bristle number in males was proportionately similar. Bristle number in sublines from the unirradiated lines regressed little on relaxation, except in sublines derived from *SO*.1 after generation 50, when there was a high level of phenotypic variance during the second period of response in this selection line. In general the decline

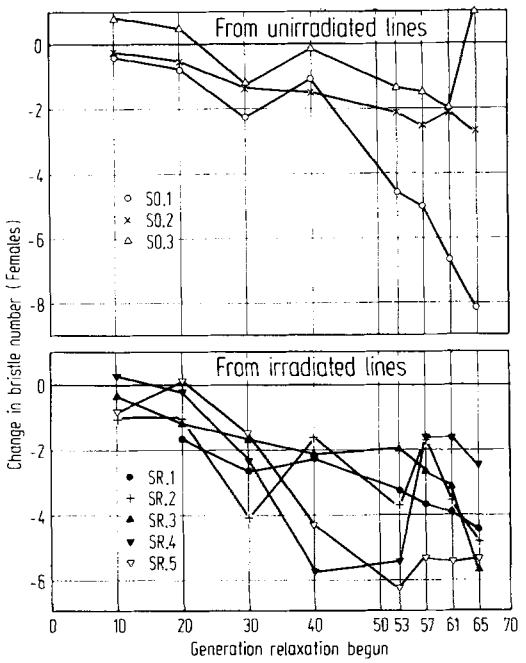

Fig. 4. The effect of five generations of relaxation on female mean bristle number, measured as the change in bristle number from the level of the selection line at the commencement of relaxation

in mean bristle number after five generations of relaxation was greater in later generations than early in the selection programme. This was true for both unirradiated and irradiated lines and differed from the results of Frankham *et al.* (1968b) who found that most of their lines "fell only slightly on relaxation and this decline became less in later generations". However, some of their lines carrying particular lethals at high frequencies did show marked regression on relaxation of selection, similar to results in Figure 4. One exception here was line *SR*.4, where the effect of relaxation was less marked in later generations. Bristle number declined by more than five bristles in sublines relaxed at generations 40 and 53, but only by about two bristles in later generations. However, as shown in Figure 5 for sublines relaxed at generation 57, the mean bristle number continued to decline rapidly over a longer period in *SR*.4 than in the other lines. *SR*.4 had reduced productivity in later generations and the subline taken from it at generation 57 was not very fit even after five generations of relaxation.

There was no evidence of strong natural selection continuing to force bristle number back to the level of the base population. All sublines were well above the initial population level after twenty generations of relaxation (Figure 5) and, except in *SR*.4, the mean regressed slowly over the last ten generations, although in *SO*.1, *SR*.1 and *SR*.5 the initial decline had been rapid.

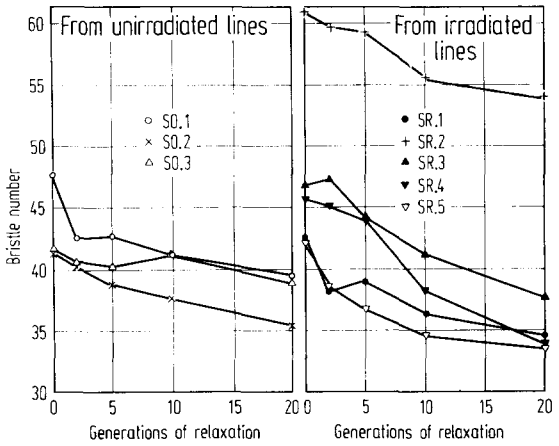

Fig. 5. The effect of relaxation of selection on mean bristle number of females in sublines taken from the selection lines at generation 57

Lethal tests: Results of the lethal tests are summarized in Table 4. Visible mutations present in lines *SO*.2, *SO*.3 and *SR*.2 were located at the same time. The immediate effects of radiation were not important in these results — the irradiated lines received *X*-ray treatments for only the first thirty generations of the selection programme, so that *SR*.3 was the only line irradiated immediately prior to lethal testing, *i.e.* the parents of the *SR*.3 flies used in the first cross of the lethal test had been treated with *X*-rays. The frequency of lethals was high in lines with no history of irradiation, confirming that recently induced lethal mutations were not necessarily responsible for the high observed frequencies in treated lines. Particular lethal genes with a selective advantage as heterozygotes through an effect on abdominal bristle number (pleiotropy or linkage) could be maintained at high frequencies. If all selected parents were heterozygous for a particular lethal, the gene frequency in the progeny would be 33.3%. If two lethal genes were closely linked in repulsion, a balanced system could develop, with each lethal gene approaching a frequency of 50%. Such a system could be eventually disrupted by recombination, unless maintained by some special mechanism — such as selective disadvantage of the coupling phase recombinants. The actual prevalence of particular lethals at high frequencies was determined by allelism testing.

Table 4. *Results of lethal tests carried out at generation 29 in line SR.3 and generation 32 in all other lines*

Line	Mean bristle number at time of test		Number of genomes tested	Percent lethal[+]		Comments
	Females	Males		II	III	
SO.1	34.3	27.2	68	44.1	51.5	
SO.2	35.9	29.3	70	27.1	31.4	*scabrous-like* on II
SO.3	34.1	28.2	62	50.0	24.2	*dark hairy margin* on III
SR.1	36.1	29.3	63	41.3	96.8	
SR.2	48.5	39.3	65	38.5	78.5	*scabrous* on II
SR.3	35.0	28.2	65	84.6	81.5	
SR.4	36.3	28.5	60	53.3	95.0	
SR.5	33.6	27.1	68	67.6	70.6	

[+] Classified as absolute lethals at initial lethal testing.

In Table 5 the lethals (and other mutants) are classified according to their frequencies in the lines. The frequency classes were arbitrarily chosen to indicate lethals present at low (<10%), intermediate, and high (25% to 35%) frequencies. The partial allelism testing procedure used underestimated the frequency of particular lethals, but the bias diminished as the frequency increased, and was not important for lethals present at intermediate to high frequencies. The high frequency class contained any recessive lethals present in all or most selected parents. The higher frequency classes contained lethals in balanced systems and non-lethal mutants which were approaching fixation (*dark hairy margin*) or fixed (*scabrous*). All lines contained particular lethals with frequencies greater than 10%, but the radiation lines, as a group, had more of these than the unirradiated lines.

In *SO*.1, the two intermediate chromosome III lethals were at frequencies close to 10% and one of

Table 5. *Number of second and third chromosome lethals in various frequency ranges in each selection line; semi-lethals (S) and mutants (M) with morphological effects are also noted*

Line	Frequency range (%)									
	5.0–9.9		10.0–24.9		25.0–34.9		35.0–54.9		> 55.0	
	II	III	II	III	II	III	II	III	II	III
SO.1		2 + 1 *S*	3	2						
SO.2	1	1 *SM*†		2	1 *SM*+					
SO.3		1 *SM*†	1	1	1					1 *M*+
SR.1	1	1	1 + 1 *SM*	3		1				
SR.2	3	1 *SM*†	1	3 + 1 *S*		1			1 *M*+	
SR.3	2 + 1 *M*	2	3	1	1	2				
SR.4	1	1 + 1 *SM*	3 + 1 *SM*	2 + 1 *S*				2		
SR.5	1	1	4 + 1 *S*	4						

† Allelic.
+ *SM* in *SO*.2 is *scabrous-like*.
+ *M* in *SO*.3 is *dark hairy margin*.
+ *M* in *SR*.2 is *scabrous*.

the chromosome II lethals (IIa) had a frequency of about 12%. Line *SO*.2 contained the second chromosome semi-lethal mutant *scabrous-like* at high frequency and two chromosome III lethals at intermediate frequencies. Line *SO*.3 had the third chromosome mutant *dark hairy margin* segregating in it at generation 32. This mutant was fixed in the line at generation 39. This line also had one chromosome III lethal in the intermediate range and two chromosome II lethals, one high and one intermediate in frequency. These two chromosome II lethals and the mutants *scabrous-like* and *dark hairy margin* were studied in more detail (Hollingdale 1971). Lethals at appreciable frequencies in these three unirradiated lines were presumably derived from the base population (or arose by spontaneous mutation). The presence of the same lethal, *SO*.1 II_A (frequency c. 12%) ≡ *SR*.3 II_A (frequency c. 24%), in two lines and a semi-lethal mutant with fine, short bristles at low frequency in three lines is evidence that the base population had been the source of some of these genes. The Canberra cage population carried such deleterious alleles at very low frequencies, as Frankham *et al.* (1968b) showed that there were no lethals in the base population at a frequency of greater than 2% but 9.5% of second chromosomes and 14.5% of third chromosomes carried lethal genes.

Chromosome III lethals were predominant in *SR*.1, one at high frequency, three at intermediate frequencies. There were also a second chromosome lethal and a semi-lethal mutant at intermediate frequencies. The second chromosome mutant *scabrous* was fixed in *SR*.2. This gene had a large effect on bristle number (Hollingdale 1971) so that while it was segregating in the line selection would have been primarily for this one gene, and chance associations of other second chromosome genes with *scabrous* could have been important. However, there was one second chromosome lethal at intermediate frequency (about 12%). One chromosome III lethal was present at a high frequency and there were three lethals and a semi-lethal at intermediate frequencies, all about 11% or 12%. *SR*.3 had two chromosome II lethals at 24% and 25% respectively and two chromosome III lethals at high frequencies as well as two second chromosome lethals and one third chromosome lethal at the lower end of the intermediate frequency range. *SR*.4 apparently had developed a third chromosome balanced lethal system, as lethals III_A and III_B each occurred at a frequency of 40% and none of the sample chromosomes carried both of them. There were other lethals and semi-lethals in the intermediate frequency range, one lethal on chromosome III at a frequency of 18% and the others (four on chromosome II, two on chromosome III) at frequencies from 10% to 13%. *SR*.5 had no high frequency lethals but several at intermediate frequencies on both chromosomes. The highest frequency of a specific lethal in this line was 18%.

Discussion

The short-term (generations 0 to 10) estimate of realized heritability, 22.5% in unirradiated selection lines with one hundred pairs of parents, extends the results of Frankham *et al.* (1968a). They found that realized heritability increased with increase in population size; mean values were 14.6%, 16.2% and 18.3% for population sizes of ten, twenty and forty pairs of parents respectively. Also, for units with the same number scored, the realized heritability increased as selection intensity decreased. For two hundred pairs scored, estimates for 10%, 20% and 50% selection intensity were 13.4%, 15.8% (Frankham *et al.*) and 22.5% (this paper). This emphasizes the importance of selecting in large populations, not only to obtain higher levels of total response in the long term (Robertson 1960), but also for a faster rate of improvement in early generations, since average selection differentials seem to be little affected by population size. However, the short-term effect of population size may be less extreme in other organisms with larger numbers of chromosomes and recombination in both sexes.

Latter (1964), selecting on total bristle number on two sternites at 50% intensity in replicate lines with fifty pairs of parents, calculated a realized heritability of 29.1% on the basis of a single sternite. This value, for the same Canberra strain, is considerably higher than values obtained with a larger population size in the present experiment. However, culture conditions were different in the two experiments, and this seems the likely cause of the discrepancy.

Levels of response in the selection lines, excluding line *SR*.2 (*scabrous*), were well below the limits now known for this character in Canberra derived lines (Jones *et al.* 1968, Frankham *et al.* 1968b). Response levels higher than those of the unirradiated lines can be realized at least in some special circumstances involving selection in large populations at high intensity, or crossing of selected lines, or the influence of a major mutant (*scabrous*).

The occurrence of *scabrous* in line *SR*.2 made it difficult to assess the final level of response in the irradiated lines. However, as *scabrous* alleles had previously been found in two unirradiated lines derived from the Canberra base population (Jones *et al.* 1968, Rathie and Barker 1968), the *scabrous* allele in *SR*.2 may not have been a result of the *X*-radiation treatment. The remaining four irradiated lines had final mean bristle numbers below the high level of line *SR*.2 or the highest line of Jones *et al.* (1968).

Comparing final levels of response of the unirradiated and irradiated lines, and excluding line *SR*.2, the irradiated line with the lowest mean, *SR*.1, was similar to two of the unirradiated lines, *SO*.2 and *SO*.3, while the remaining irradiated lines, *SR*.3, *SR*.4 and *SR*.5, were fairly similar to each other and to the highest unirradiated line, *SO*.1. This failure of the radiation treatment to bring about any worthwhile increase in the final level attained by selection needs to be considered in the light of what is known of the genetics of abdominal bristle number. The bristle number traits of *D. melanogaster* have been perhaps the most extensively studied of any metric traits, and the Canberra population is becoming well characterized in this respect, through the work of Latter (1963, 1964), Jones (1967), Frankham *et al.* (1968b), Jones *et al.* (1968), Rathie and Barker (1968), Sheridan *et al.* (1968), Sheridan (1969), Hammond and James (1970) and Robertson (1969), involving the abdominal, scutellar, second coxal, and sternopleural bristle systems.

In natural populations characters such as abdominal and sternopleural bristle number are probably subject to stabilizing selection for an intermediate optimum, though just why this is so remains unclear (Reeve and Robertson 1954, Mather 1966, Kearsey and Barnes 1970). As characters with no direct and obvious relation to fitness they are expected to have relatively high heritabilities (compared to "fitness" characters subject to directional selection in natural populations) and to show little dominance. According to Fisher's theory of dominance (Fisher 1958), genes determining abdominal bristle number are expected to show dominance in either direction so that linked complexes may appear to be entirely lacking in dominance. In the Canberra base population Sheridan *et al.* (1968) found dominance effects to be "probably only minor" for abdominal bristle number. There was, however, a sizable additive x additive interaction component, which would influence the selection response in the early generations (Griffing 1960).

Directional selection imposed on such systems has resulted in patterns of response which, in the short term, are generally regular and predictable from the variance component analyses of the initial populations, though variation between replicate lines has often been large, *e.g.* Clayton, Morris and Robertson (1957), Sheldon (1963), Latter (1964) and Frankham *et al.* (1968a). In the long term, the theory of limits to selection as developed by Robertson (1960) and James (1962), predicts an asymptotic approach of the mean of the selected trait to an expected limit which is a function of the effective population size and the selection intensity. However, in practice long-term response patterns have been more irregular, with discontinuities or "waves of response" a common feature, *e.g.* Clayton and Robertson (1957), Thoday, Gibson and Spickett (1964), Fraser, Scowcroft, Nassar, Angeles and Bravo (1965), and Jones *et al.* (1968).

Fraser and Hansche (1964) considered several models proposed to account for such irregularities in response patterns. Fraser's (1957) computer simulation results showed that, where linkage was close (approximately 0.5%) and the population size small, the disruption of linked complexes of polygenes (= minor loci) could account for discontinuities in the response pattern. There are alternative hypotheses which can also explain the same phenomenon. As listed by Fraser and Hansche these are: "(1) a number of minor loci, each at intermediate frequency, with a few major, recessive loci, each at low frequency, [and] (2) a number of minor loci, each at intermediate frequency, which when shifted by selection, act as modifiers of a few normally minor loci, transforming these into major loci". Their simulation results showed that the first of these, the rare "major recessive" hypothesis, was as satisfactory as the "linked complex" hypothesis in producing complex response patterns similar to those obtained in their scutellar bristle selection lines. The "evolved major gene" hypothesis proposed by Reeve and Robertson (1953), was supported by results of Fraser (1963) on the "*Bare*" phenotype in *Drosophila simulans*. Miller and Fraser (1968) found that a major third chromosome mutant, *x-vert*, affecting scutellar bristle number, interacted in a complex manner with systems of modifiers, and the interaction was dependent on the

genotype, *i.e.* presence of *scute* or its wild type allele. Fraser (1967) suggested that *x-vert* was also an "evolved major gene" with low penetrance in unselected backgrounds.

In actual selection lines, irregular response patterns may be due to combinations of these three models, although in certain cases, as noted by Mather in the discussion of Fraser and Hansche's paper, the rare major recessive hypothesis is not relevant because of the composition of the initial populations, *e.g.* base populations derived from the progeny of a cross between two inbred lines. Detailed analyses of sternopleural bristle number selection lines, including location of polygenes by Thoday's (1961) method, have been carried out by Thoday, Gibson and Spickett (1964) and Spickett and Thoday (1966). Similar accelerated responses occurring at different times in lines with common ancestry were found to be probably due to recombination occurring between two third chromosome loci. The analysis also showed that these genes had approximately equal effects on bristle number and did not interact with each other. However, one of the genes interacted strongly with a second chromosome located polygene and together these three genes accounted for 80% of the response of one of the lines.

Scowcroft (1966) examined accelerated responses in a group of three related lines, selected for increased scutellar bristle number, and found considerable differences in the behaviour of the lines. In one case he suggested that an effective factor resulting from recombination within a polygenic system required a modified genetic background for full expression. In another line, in which the plateau was opposed by natural selection, he postulated that a high bristle gene, lethal when homozygous, or a sex-linked male sterile gene associated with a high bristle factor (through pleiotropy or linkage) could be possible mechanisms maintaining genetic variability at the plateau. He also suggested that small duplications, which occur at a frequency much higher than the spontaneous mutation rate, could explain accelerated responses.

Results such as those of Thoday *et al.* (1964) led Robertson (1966) to the conclusion that "the number of important loci controlling the genetic variability in quantitative variation is rather less than . . .previously thought", and that less than ten loci probably would account for perhaps 80% of the difference between his extreme selected bristle lines. Spickett and Thoday (1966) pointed out that "locatable polygenes are a non-random sample comprising the most extremely effective genes or linked complexes of a continuous spectrum" including genes of vanishingly small effect, and that their analysis underestimated the number of loci affecting the quantitative trait. Genes directly affecting the character would not be detected if they were present in the standard as well as the selected population and genes with no direct effect on the character could nevertheless influence its variance through fitness interactions. In fact Breese and Mather (1960) found that linkage of polygenic complexes governing viability and abdominal bristle number was an important factor in the loss of fitness that has been commonly found as a correlated response in lines selected over long periods (Mather and Harrison 1949, Clayton and Robertson 1957, Rathie and Barker 1968).

The genetic basis of bristle characters in the Canberra population appears to be similarly complex. Latter (1963), analysing the effects on fitness of short-term selection for abdominal bristle number, found that "most of the individual [bristle] genes have virtually no effects on reproductive fitness". This conforms to the generally held view that abdominal bristle number is not closely related to fitness. Rathie and Barker (1968) found that fitness declined more under continuous than under intermittent selection, although some replicates in each selection treatment showed little or no loss of reproductive fitness. On the basis of these results, they suggested that linked complexes including genes deleterious to fitness and those affecting abdominal bristle number are important in the Canberra population. Frankham *et al.* (1968b) found that in some lines lethals with apparently little effect on bristle number reached high frequencies during selection, possibly because of linkage to abdominal bristle number genes. They suggested a combination of large gene effects, linkage, and gene interaction as the cause of irregularities in the responses of their lines. The genetic dissimilarity between lines (as shown by several criteria), the results of crosses between high selected lines, and the retention of genetic variation over long periods (Jones *et al.* 1968) indicated that initially rare genes of large effect or genes with effects dependent on the genetic background are important in this population. Hammond and James (1970) in an analysis of the moments of the distribution of family means in the unselected Canberra population, found no evidence of rare genes with large effects on abdominal bristle number.

Frankham *et al.* (1968b) considered that their results in the Canberra population, as well as similar results in other populations, suggest that many loci are potentially capable of influencing a quantitative character but that only a few genes determine most of the response in any particular line. Spickett and Thoday (1966) came to a similar conclusion concerning the "Dronfield" wild stock: any one selection experiment "exploited only a limited number of the chaeta-number genes segregating in the 'wild' stock". This restricted exploitation occurs partly because the majority of laboratory selection experiments use fairly small population sizes and thus the limits to selection are artifacts of the intensity of selection and the population size rather than basic properties of the initial population (Robertson 1966). Where

initially rare genes are important very large effective population sizes may be required to reduce replicate variation in response patterns. One of our colleagues, Mr. K. Hammond, is currently investigating replicate variability in greater detail.

In abdominal bristle number selection lines derived from the Canberra population, Jones (1967) found that those which received *X*-radiation treatments generally gave greater responses than their unirradiated controls. Phenotypic variances in the irradiated lines were much higher than in the controls. This extra response and increased variance could be accounted for by "mutations at only five or six loci with effects of half a standard deviation". In his control lines (and in most of the selection lines of Jones *et al.* 1968) there was little change in the phenotypic variance from the initial level. The exceptions were all lines in which lethals at medium to high frequency were detected. An initially rare gene with a large effect on bristle number causes a large increase in the additive genetic component of the variance as it is increased in frequency by selection (Latter 1965). If rare 'large' genes are themselves lethal when homozygous, high variances persist as the genes continue to segregate, but if detected lethals reach high frequency because of linkage in coupling to high bristle genes, then variances may later decline if recombination allows such high bristle genes to become fixed (and the lethals to be eliminated). Both these types of pattern of change in phenotypic variance were found by Jones *et al.* (1968).

Phenotypic variance increased in all our selection lines, although variances were generally higher in the irradiated lines than in the unirradiated. Considering also results of lethal tests and the effects of relaxation of selection, this indicated that initially rare genes with large effects were important in all selected lines, but such genes were of negligible importance in the five unirradiated selection lines of Jones (1967). As population size in each of our lines was high (initial samples from the base population were twenty times as large as those used by Jones, two hundred pairs as compared with ten pairs) there was a high probability that some rare genes derived from the base population would influence the pattern of response to selection. This, then, must account for the apparent conflict of results. If the selection programme gives a good chance of concentrating rare genes from a large outbred base population, and if the cumulative effects of these genes on fitness prevent any one line from using more than a few of the possible array of 'large' genes, then induced mutation will possibly increase the size of this array of initially rare 'large' genes, but will not produce spectacular increases in response. However, where lines originally do not include rare 'large' genes, then radiation treatment has a chance of causing mutation at one of the loci where alleles of large effect are possible, and so in these circumstances irradiation can bring about an appreciable increase in the level of response.

The same argument applies to the discrepancy between our results and those of Kitagawa (1967) and Scossiroli and Scossiroli (1959). In both these sets of experiments appreciably greater selection responses were obtained with irradiation, but the base populations were derived from hybrids between two inbred or isogenic strains, so that the unirradiated lines initially could not have contained rare 'large' genes.

Why have induced mutations with large effect been so uncommon in selection programmes using inbred base populations? Why, for example, was there no evidence of genes with large effects in the irradiated lines derived from inbred line N5 (Hollingdale and Barker 1971)? This may be because the expression of such genes is dependent on the rest of the genotype. The background genotype of a previously selected population could enhance the expression of mutants affecting the selected trait, and the genetic variation available in an outbred base population could provide a more suitable combination of modifiers than the single combination present in the genotype of an inbred line. Mutant expression is dependent on the genetic background — see for example the work of Gaul, Grunewaldt and Hesemann (1968) on the expression of macro-mutations in barley.

Mutations with large effects on the phenotype are more likely to be deleterious than ones with only small effects (Fisher 1958). Loss of fitness in lines showing large increases in response following radiation treatments is therefore likely to be a common problem. The possibility of utilizing induced mutations with individually small effects requires further consideration. Such induced mutations can cause significant increases in variance, without adversely affecting fitness, if the irradiated populations can be screened to remove all major mutants (Gregory 1968). 'Small' mutations were apparently responsible for the slight extra response in irradiated lines derived from inbred line N5, and they may account for the higher variance in the irradiated Canberra lines.

Gregory's screening technique represents a somewhat different approach to that advocated by Scossiroli (1968). The latter's suggestion that irradiated populations should not be subjected to immediate artificial selection was based on the assumption that useful induced mutations are often deleterious because of linkage rather than strict pleiotropy. Delaying selection would make available a wider array of combinations of induced mutations with the genetic background of the irradiated population. Either approach is more suitable for use with mutation breeding of plant species, where large populations may be handled after a single radiation treatment. Plants are able to withstand much higher doses of radiation than animal species and their greater reproductive potential and, in some species, selffertiliz-

ing breeding system, makes mutation breeding a practical proposition for crop improvement.

The potential usefulness of mutagenesis as a means of raising plateau levels in animal populations must be acknowledged to be limited. The work with *Drosophila* has shown that selection limits are likely to be artifacts of the selection regime, so that simple crossing of selected lines is likely to be better than radiation as a source of variation amenable to selection.

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Literature cited

1. Abplanalp, H., Lowry, D., Lerner, I. M., Dempster, E. R.: Selection for egg number with X-ray-induced variation. Genetics **50**, 1083–1100 (1964). — 2. Barker, J. S. F., Hollingdale, B.: Report of new mutants in *Drosophila melanogaster*. Drosophila Inf. Serv. **45**, 39 (1970). — 3. Becker, W. A.: Manual of Procedures in Quantitative Genetics. Pullman: Washington State University 1964. — 4. Breese, E. L., Mather, K.: The organisation of polygenic activity within a chromosome in *Drosophila*. II. Viability. Heredity, Lond. **14**, 375–399 (1960). — 5. Brown, W. P., Bell, A. E.: Genetic analysis of a "plateaued" population of *Drosophila melanogaster*. Genetics **46**, 407–425 (1961). — 6. Claringbold, P. J., Barker, J. S. F.: The estimation of relative fitness of *Drosophila* populations. J. Theoret. Biol. **1**, 190–203 (1961). — 7. Clayton, G. A., Morris, J. A., Robertson, A.: An experimental check on quantitative genetical theory. I. Short-term responses to selection. J. Genet. **55**, 131–151 (1957). — 8. Clayton, G. A., Robertson, A.: An experimental check on quantitative genetical theory. II. The long-term effects of selection. J. Genet. **55**, 152–170 (1957). — 9. Clayton, G., Robertson, A.: The effects of X-rays on quantitative characters. Genet. Res. **5**, 410 to 422 (1964). — 10. Falconer, D. S.: Asymmetrical response in selection experiments. Symposium on Genetics of Population Structure, I.U.B.S. Publ., Ser. B, **15**, 16–41 (1954). — 11. Falconer, D. S.: Introduction to Quantitative Genetics. Edinburgh: Oliver and Boyd 1960. — 12. Fisher, R. A.: The Genetical Theory of Natural Selection, 2nd ed. New York: Dover 1958. — 13. Frankham, R., Jones, L. P., Barker, J. S. F.: The effects of population size and selection intensity in selection for a quantitative character in *Drosophila*. I. Short-term response to selection. Genet. Res. **12**, 237–248 (1968a). — 14. Frankham, R., Jones, L. P., Barker, J. S. F.: The effects of population size and selection intensity in selection for a quantitative character in *Drosophila*. III. Analyses of the lines. Genet. Res. **12**, 267–283 (1968b). — 15. Fraser, A.: Variation of scutellar bristles in *Drosophila*. I. Genetic leakage. Genetics **48**, 497–514 (1963). — 16. Fraser, A.: Variation of scutellar bristles in *Drosophila*. XV. Systems of modifiers. Genetics **57**, 919–934 (1967). — 17. Fraser, A. S.: Simulation of genetic systems by automatic digital computers. II. Effects of linkage on rates of advance under selection. Aust. J. biol. Sci. **10**, 492–499 (1957). — 18. Fraser, A. S., Hansche, P. E.: Simulation of genetic systems. Major and minor loci. "Genetics Today", Proc. XI Int. Congr. Genet. **3**, 507–516 (1964). — 19. Fraser, A. S., Scowcroft, W., Nassar, R., Angeles, H., Bravo, G.: Variation of scutellar bristles in *Drosophila*. IV. Effects of selection. Aust. J. biol. Sci. **18**, 619–641 (1965). — 20. Gaul, H., Grunewaldt, J., Hesemann, C. U.: Variation of character expression of barley mutants in a changed genetic background. Mutations in Plant Breeding II, pp. 77–95, Vienna: I.A.E.A. 1968. — 21. Gregory, W. C. (ed.): A radiation breeding experiment with peanuts. Radiat. Bot. **8**, 81–147 (1968). — 22. Griffing, B.: Accommodation of linkage in mass selection theory. Aust. J. biol. Sci. **13**, 501–526 (1960). — 23. Hammond, K., James, J. W.: Genes of large effect and the shape of the distribution of a quantitative character. Aust. J. biol. Sci. **23**, 867–876 (1970). — 24. Hollingdale, B.: Irradiation and selection in *Drosophila melanogaster*. Ph. D. Thesis, University of Sydney (1969). — 25. Hollingdale, B.: Analyses of some genes from abdominal bristle number selection lines in *Drosophila melanogaster*. Theoret. Appl. Genet. **41** (1971). — 26. Hollingdale, B., Barker, J. S. F.: Selection for increased abdominal bristle number in *Drosophila melanogaster* with concurrent irradiation. I. Populations derived from an inbred line. Theoret. Appl. Genet. **41**, 208 – 215 (1971). — 27. James, J. W.: Conflict between directional and centripetal selection. Heredity, Lond. **17**, 487–499 (1962). — 28. Jones, L.P.: Effects of X-rays on response to selection for a quantitative character of *Drosophila melanogaster*. Genet. Res. **9**, 221–231 (1967). — 29. Jones, L. P., Frankham, R., Barker J. S. F.: The effects of population size and selection intensity in selection for a quantitative character in *Drosophila*. II. Long-term response to selection. Genet. Res. **12**, 249–266 (1968). — 30. Kearsey, M. J., Barnes, B. W.: Variation for metrical characters in *Drosophila* populations. II. Natural selection. Heredity, Lond. **25**, 11–21 (1970). — 31. Kitagawa, O.: The effects of X-ray irradiation on selection response in *Drosophila melanogaster*. Jap. J. Genet. **42**, 121–137 (1967). — 32. Latter, B. D. H.: Genetic homeostasis and the theory of canalization. In: Statistical Genetics and Plant Breeding. N.A.S.-N.R.C. Publ. **982**, 455–467 (1963). — 33. Latter, B. D. H.: Selection for a threshold character in *Drosophila*. I. An analysis of the phenotypic variance on the underlying scale. Genet. Res. **5**, 198–210 (1964) . — 34. Latter, B. D. H.: The response to artificial selection due to autosomal genes of large effect. I. Changes in gene frequency at an additive locus. Aust. J. biol. Sci. **18**, 585–598 (1965). — 35. Lewontin, R. C.: On the measurement of relative variability. Syst. Zool. **15**, 141–142 (1966). — 36. Lindsley, D. L., Grell, E. H.: Genetic Variations of *Drosophila melanogaster*. Carnegie Inst. Wash. Publ. **627** (1968). — 37. Mather, K.: Variability and selection. Proc. R. Soc., B, **164**, 328–340 (1966). — 38. Mather, K., Harrison, B. J.: The manifold effect of selection. I. and II. Heredity, Lond. **3**, 1–52 and 131–162 (1949). — 39. Miller, D. H., Fraser, A. S.: Variation of scutellar bristles in *Drosophila*. XIII. Effects of *scute* alleles. Aust. J. biol. Sci. **21**, 61–74 (1968). — 40. Rathie, K. A., Barker J. S. F.: Effectiveness of regular cycles of intermittent artificial selection for a quantitative character in *Drosophila melanogaster*. Aust. J. biol. Sci. **21**, 1187–1213 (1968). — 41. Reeve, E. C. R., Robertson, F. W.: Studies on quantitative inheritance. II. Analysis of a strain of *D. melanogaster* selected for long wings. J. Genet. **51**, 276–316 (1953). — 42. Reeve, E. C. R., Robertson, F. W.: Studies in quantitative inheritance. VI. Sternite chaeta number in *Drosophila*: a metameric quantitative character. Z. indukt. Abstamm- u. VererbLehre **86**, 269–288 (1954). — 43. Roberts, R. C.: The limits to artificial selection for body weight in the mouse. IV. Sources of new genetic variance — irradiation and outcrossing. Genet. Res. **9**, 87–98 (1967). — 44. Robertson, A.: A theory of limits in artificial selection. Proc. R. Soc., B **153**, 234–249 (1960). — 45. Robertson, A.: Artificial selection in plants and animals. Proc. R. Soc., B **164**, 341–349 (1966). — 46. Robertson, D. E.: Selection in small populations. Ph. D. Thesis, University of New South Wales (1969). — 47.

Scossiroli, R. E.: Effectiveness of artificial selection under irradiation of plateaued populations of *Drosophila melanogaster*. Symposium on Genetics of Population Structure, I.U.B.S. Publ., Ser. B **15**, 42–66 (1954). – 48. Scossiroli, R. E.: Selection experiments in a population of *Triticum durum* irradiated with X-rays. Mutations in Plant Breeding II, pp. 205–217. Vienna: I.A.E.A. 1968. – 49. Scossiroli, R. E., Scossiroli, S.: On the relative role of mutation and recombination in responses to selection for polygenic traits in irradiated populations of *D. melanogaster*. Int. J. Radiat. Biol. **1**, 61–69 (1959). – 50. Scowcroft. W. R.: Variation of scutellar bristles in *Drosophila*. X. Selection limits. Aust. J. biol. Sci., **19**, 807–820 (1966). – 51. Sheldon, B. L.: Studies in artificial selection of quantitative characters. I. Selection for abdominal bristles in *Drosophila melanogaster*. Aust. J. biol. Sci. **16**, 490–515 (1963). – 52. Sheridan, A. K.: The effect of simultaneous selection for correlated quantitative characters on selection responses and genetic parameters. Ph. D. Thesis, University of Sydney (1969). – 53. Sheridan, A. K., Frankham, R., Jones, L. P., Rathie, K. A., Barker, J. S. F.: Partitioning of variance and estimation of genetic parameters for various bristle number characters of *Drosophila melanogaster*. Theoret. Appl. Genet. **38**, 179–187 (1968). – 54. Spickett, S. G., Thoday, J. M.: Regular responses to selection. 3. Interaction between located polygenes. Genet. Res. **7**, 96–121 (1966). – 55. Thoday, J. M.: Location of polygenes. Nature, Lond. **191**, 368–370 (1961). – 56. Thoday, J. M., Gibson, J. B., Spickett, S. G.: Regular responses to selection. 2. Recombination and accelerated response. Genet. Res. **5**, 1–19 (1964). – 57. Wright, S.: The genetics of quantitative variability. In: Quantitative Inheritance, edited by E.C.R. Reeve and C. Waddington, pp. 5–41, London: H.M.S.O. 1952.

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