GENETIC ANALYSES OF TWO ABDOMINAL BRISTLE SELECTION LINES

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Summary

Chromosome substitution was done among two increased abdominal bristle selection lines representing high and moderate levels of response [designated 20(10%)a and 10(10%)c respectively] and their base population. The resulting stocks were measured in four environments. This made it possible to obtain a minimum interpretation of intrachromosomal differences by using the physiological ploy of separating effects by demonstrating an interaction with environmental treatments.

A minimum of five factors were implicated in selection response in 10(10%)c, and seven in 20(10%)a. The most important finding was that four of the factors were common to the two lines. Further, the seven factors in 20(10%)a were also known to be implicated in response in 40(10%), a still higher line.

This and other evidence suggests that selection does not utilize merely a random sample of the initially segregating loci affecting the character. Possible reasons for non-random utilization of genes were discussed. It is suggested that non-random utilization of genes is likely to have arisen both from gene interaction effects and from different sized effects of genes combined with different initial gene frequencies.

I. INTRODUCTION

Little is known about the selection process. From the assumptions underlying most quantitative genetic theory (viz. that quantitative differences, insofar as they are inherited, depend on the segregation of many genes, each of small effect, which act in a more or less additive fashion) it follows that selection response is expected to involve simultaneous utilization of a random sample of the many available genes of small effect. However, experimental evidence (Payne 1918; Warren 1934; Spickett and Thoday 1966; Costantino, Bell, and Rogler 1967) indicates that genes (or short chromosome segments) of large effect are implicated in selection response in at least some cases.

Until the genes controlling selection response for quantitative characters can be individually identified and their frequencies followed during the selection process, the main avenues for studying the nature of the selection process are comparative genetic analyses of replicate selection lines and genetic analyses of individual lines at different stages of response.

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Most experimental information so far on the nature of the selection process has come from studies of irregular response patterns. Sismandis (1942) showed that early response for scutellar bristles involved chromosome II, while later renewed response involved both chromosomes II and III (i.e. response was at least partly utilizing different genes at different stages). Mather and Harrison (1949) carried out extensive analyses of related abdominal bristle selection lines at various stages of response (shown schematically in Fig. 1). In one line (6) a delayed response was due to chromosome II, while previous response was due to chromosomes *X* and III. In other lines the chromosomes frequently contributed differentially to delayed and earlier response. They argued that the delayed responses were due to recombination. However, Reeve and Robertson (1953) pointed out that irregular response patterns could be due to gene interaction effects. Erway (in Fraser 1967) and Thoday, Gibson, and Spickett (1964) and Spickett and Thoday (1966) found that linkage and interaction effects were both implicated in delayed responses to selection.

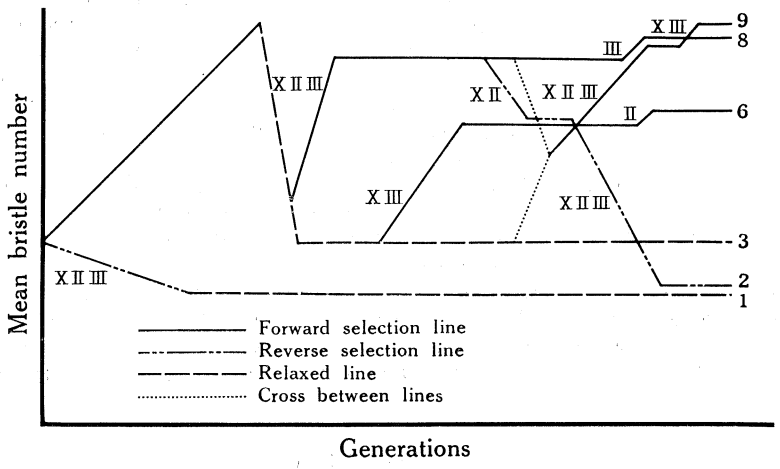

Fig. 1.—Schematic representation of the levels of response and the chromosomes involved in the various responses in the lines of Mather and Harrison (1949).

Evidence from comparative genetic analyses of replicate selection lines does not reveal any consistent conclusion. Mather and Harrison (1949), King and Somme (1958), and Scowcroft (1966) have shown that replicate selection lines utilized (at least partly) different genes. Conversely, Thoday, Gibson, and Spickett (1964) and Spickett and Thoday (1966) showed that replicate selection lines at the same level utilized the same genes and higher lines utilized these genes plus extra ones. The former evidence suggests that selection probably utilizes a random sample of the available genes while the latter indicates that selection may utilize genes in a more or less specified (non-random) pattern.

The present experiment was concerned with determining whether selection utilizes a random sample of the available genes. Chromosome substitution was carried out among two selection lines (representing different levels of response) and their base population. The resulting stocks were measured in four environments to obtain a minimum interpretation of intrachromosome differences.

II. Materials and Methods

(a) *Stocks*

(1) Can—the Canberra base population (Sheridan *et al.* 1968) is a large outbred population with mean abdominal bristle numbers on F media at 25°C of approximately 22 in females and 18 in males.

(2) 20(10%)a—a high abdominal bristle number selection line (derived from the Canberra base population) with mean abdominal bristle numbers on F media at 25°C of approximately 43·7 in females and 33·9 in males (Jones, Frankham, and Barker 1968).

(3) 10(10%)c—a high abdominal bristle selection line (derived from the Canberra base population) with mean abdominal bristle numbers on F media at 25°C of approximately 33·6 in females and 27·7 in males (Jones, Frankham, and Barker 1968). These stocks represented high [20(10%)a], medium [10(10%)c], and base population (Can) levels of abdominal bristle number.

(4) LT—this was the marked inversion stock used for chromosome substitution. It had the following constitution:

In (1) sc^{S1L} *sc*^{8R}+*S*, *sc*^{S1} *sc*^{8} *w*^{a} *B*; *In (2 LR) SM1, al*^{2} *Cy cn*^{2} *sp*^{2}/*In (2 LR) bw*^{V1}, *ds*^{33k} *dp*^{ov} *bw*^{V1}; *In (3 LR) Ubx*^{130}, *Ubx*^{130} *e*^{s}/*In (3 LR) C, Sb*; *spa*^{pol}

where Curly (*Cy*), brown-Variegated (*bw*^{V1}, referred to hereafter as *Pm*), Ultrabithorax (*Ubx*), and Stubble (*Sb*) are dominant mutants lethal in homozygotes. These dominant marker genes will be used to refer to the chromosomes they are located on.

(b) *Chromosome Substitution Method*

The eight "homozygous" (refers to origin and not true homozygosity) combinations of *X*, II, and III chromosomes for each pair of lines [viz. Can–10(10%)c, Can–20(10%)a, 20(10%)a–10(10%)c] were produced by the method (similar to that of King and Somme 1958) shown in Table 1. The origin of the fourth and *Y*-chromosomes were both ignored. All *Y*-chromosomes came from the LT stock. The scoring of some *spa*^{pol}/*spa*^{pol} and non-*spa*^{pol} segregants indicated that any effects of chromosome IV were probably small. Briefly the method was to cross 60 line males to 120 virgin LT females. Numerous +/*B* +/*Cy* +/*Ubx* virgin females from this cross were backcrossed to LT males and many virgins of all genotypes collected from the progeny. Crosses between lines were then set up and continued as shown (only the genotypes actually used are included) in Table 1, to produce the "homozygous" stocks. A minimum of 20 samples (except for six stocks) of each chromosome were used in each of the 21 crosses to ensure an adequate sample of selection line chromosomes. Where *B Cy*/+ and *B Cy*/*Pm* had to be distinguished (see crosses 2 and 7 in Table 1) 40 single-pair matings were set up (all other matings were mass matings in bottles) and the distinction made on the presence or absence of *Pm* in the progeny. Difficulties were experienced with sterility and low progeny numbers in these matings. The minimum number of chromosomes contributing to each stock are shown in Table 2, the least being nine for $\frac{\text{Can}}{\text{Can}}\ \frac{\text{Can}}{\text{Can}}\ \frac{\text{20(10\%)a}}{\text{20(10\%)a}}$.

The 27 possible female and 18 possible male genotypes for a particular pair of lines were obtained by using the eight "homozygous" genotypes and producing the "heterozygous" genotypes from appropriate crosses between them.

(c) *Experimental Methods*

The environments used were two temperatures (20 and 25°C) and two media—dead yeast fortified (medium F of Claringbold and Barker 1961), and ordinary (Ord) unfortified medium. For each female genotype two replicate bottles, each with five pairs of parents, were set up in each environment and 10 flies scored for abdominal bristle number from each bottle, and for each male genotype three replicates each of 10 flies in each environment were scored for abdominal bristle number. Replicates for female genotypes with "heterozygous" *X*-chromosomes were produced from reciprocal crosses between appropriate stocks, and males in these provided the extra male replicate.

This experiment consisted of three factorial experiments (i.e. one for each pair of lines) for each sex, each as follows:

Females—$3X \times 3II \times 3III$ genotypes $\times$ two temperatures $\times$ two media with two replicates and 10 individuals scored per replicate.

Males—$2X \times 3II \times 3III$ genotypes $\times$ two temperatures $\times$ two media with three replicates and 10 individuals scored per replicate.

TABLE 1

CHROMOSOME SUBSTITUTION METHOD

"a" is a chromosome from line a and "b" a chromosome from line b, the subscripts 1, 2, and 3 refer to X, II, or III chromosome respectively, and }×{ indicates all combinations

$\frac{B}{B}\ \frac{Cy}{Pm}\ \frac{Ubx}{Sb}$ ♀ × $\frac{a_1}{\ }\ \frac{a_2}{a_2}\ \frac{a_3}{a_3}$ ♂ | $\frac{B}{B}\ \frac{Cy}{Pm}\ \frac{Ubx}{Sb}$ ♀ × $\frac{b_1}{\ }\ \frac{b_2}{b_2}\ \frac{b_3}{b_3}$ ♂

↓ | ↓

Generation 1

$\frac{B}{a_1}\ \frac{Cy}{a_2}\ \frac{Ubx}{a_3}$ ♀ × $\frac{B}{\ }\ \frac{Cy}{Pm}\ \frac{Ubx}{Sb}$ ♂ | $\frac{b_1}{B}\ \frac{b_2}{Cy}\ \frac{b_3}{Ubx}$ ♀ × $\frac{B}{\ }\ \frac{Cy}{Pm}\ \frac{Ubx}{Sb}$ ♂

↓ | ↓

Generation 2

$$\left.\begin{matrix} X \\ B/B \\ a_1/B \\ a_1 \\ B \end{matrix}\right\} \times \left\{\begin{matrix} \text{II} \\ Cy/Pm \\ Cy/a_2 \\ Pm/a_2 \end{matrix}\right\} \times \left\{\begin{matrix} \text{III} \\ Ubx/Sb \\ Ubx/a_3 \\ Sb/a_3 \end{matrix}\right. \qquad \left.\begin{matrix} X \\ B/B \\ b_1/B \\ b_1 \\ B \end{matrix}\right\} \times \left\{\begin{matrix} \text{II} \\ Cy/Pm \\ Cy/b_2 \\ Pm/b_2 \end{matrix}\right\} \times \left\{\begin{matrix} \text{III} \\ Ubx/Sb \\ Ubx/b_3 \\ Sb/b_3 \end{matrix}\right.$$

Crosses

(1) $\frac{a_1}{B}\ \frac{a_2}{Cy}\ \frac{a_3}{Ubx}$ ♀ × $\frac{a_1}{\ }\ \frac{a_2}{Cy}\ \frac{a_3}{Ubx}$ ♂ → $\frac{a_1}{a_1}\ \frac{a_2}{a_2}\ \frac{a_3}{a_3}$

(2) $\frac{a_1}{B}\ \frac{a_2}{Cy}\ \frac{Ubx}{Sb}$ ♀ × $\frac{B}{\ }\ \frac{Cy}{Pm}\ \frac{b_3}{Ubx}$ ♂ → $\frac{a_1}{B}\ \frac{a_2}{Cy}\ \frac{b_3}{Ubx}$ ♀ × $\frac{a_1}{\ }\ \frac{a_2}{Cy}\ \frac{b_3}{Ubx}$ ♂ → $\frac{a_1}{a_1}\ \frac{a_2}{a_2}\ \frac{b_3}{b_3}$

(3) $\frac{B}{\ }\ \frac{a_2}{Pm}\ \frac{a_3}{Ubx}$ ♂ × $\frac{b_1}{B}\ \frac{Cy}{Pm}\ \frac{Ubx}{Sb}$ ♀ → $\frac{b_1}{B}\ \frac{a_2}{Cy}\ \frac{a_3}{Ubx}$ ♀ × $\frac{b_1}{\ }\ \frac{a_2}{Cy}\ \frac{a_3}{Ubx}$ ♂ → $\frac{b_1}{b_1}\ \frac{a_2}{a_2}\ \frac{a_3}{a_3}$

(4) $\frac{a_1}{B}\ \frac{Cy}{Pm}\ \frac{a_3}{Ubx}$ ♀ × $\frac{B}{\ }\ \frac{b_2}{Pm}\ \frac{Ubx}{Sb}$ ♂ → $\frac{a_1}{B}\ \frac{b_2}{Cy}\ \frac{a_3}{Ubx}$ ♀ × $\frac{a_1}{\ }\ \frac{b_2}{Cy}\ \frac{a_3}{Ubx}$ ♂ → $\frac{a_1}{a_1}\ \frac{b_2}{b_2}\ \frac{a_3}{a_3}$

(5) $\frac{a_1}{B}\ \frac{Cy}{Pm}\ \frac{Ubx}{Sb}$ ♀ × $\frac{B}{\ }\ \frac{b_2}{Pm}\ \frac{b_3}{Ubx}$ ♂ → $\frac{a_1}{B}\ \frac{b_2}{Cy}\ \frac{b_3}{Ubx}$ ♀ × $\frac{a_1}{\ }\ \frac{b_2}{Cy}\ \frac{b_3}{Ubx}$ ♂ → $\frac{a_1}{a_1}\ \frac{b_2}{b_2}\ \frac{b_3}{b_3}$

(6) $\frac{B}{\ }\ \frac{a_2}{Pm}\ \frac{Ubx}{Sb}$ ♂ × $\frac{b_1}{B}\ \frac{Cy}{Pm}\ \frac{b_3}{Ubx}$ ♀ → $\frac{b_1}{B}\ \frac{a_2}{Cy}\ \frac{b_3}{Ubx}$ ♀ × $\frac{b_1}{\ }\ \frac{a_2}{Cy}\ \frac{b_3}{Ubx}$ ♂ → $\frac{b_1}{b_1}\ \frac{a_2}{a_2}\ \frac{b_3}{b_3}$

(7) $\frac{B}{\ }\ \frac{Cy}{Pm}\ \frac{a_3}{Ubx}$ ♂ × $\frac{b_1}{B}\ \frac{b_2}{Cy}\ \frac{Ubx}{Sb}$ ♀ → $\frac{b_1}{B}\ \frac{b_2}{Cy}\ \frac{a_3}{Ubx}$ ♀ × $\frac{b_1}{\ }\ \frac{b_2}{Cy}\ \frac{a_3}{Ubx}$ ♂ → $\frac{b_1}{b_1}\ \frac{b_2}{b_2}\ \frac{a_3}{a_3}$

(8) $\frac{b_1}{B}\ \frac{b_2}{Cy}\ \frac{b_3}{Ubx}$ ♀ × $\frac{b_1}{\ }\ \frac{b_2}{Cy}\ \frac{b_3}{Ubx}$ ♂ → $\frac{b_1}{b_1}\ \frac{b_2}{b_2}\ \frac{b_3}{b_3}$

III. RESULTS

Recombination effects during construction of the stocks can be gauged by comparing means for 10(10%)c and 20(10%)a with their reconstructed stocks. Male and female means for the reconstructed stocks [21·2 and 18·2, 33·3 and 26·7,

and 41·9 and 31·8, for Can, 10(10%)c, and 20(10%)a, respectively, on F media at 25°C] were at least 94% of the original line means. As the decreases in mean bristle number in the reconstructed stocks were quite small recombination and contributions from chromosome IV were probably only of minor importance in the chromosome substitution.

TABLE 2

NUMBER OF SAMPLES OF EACH CHROMOSOME CONTRIBUTING TO EACH "HOMOZYGOUS" GENOTYPE

Genotype			No. of Samples of Each Chromosome Contributing
X	II	III	
20(10%)a / 20(10%)a	20(10%)a / 20(10%)a	Can / Can	16
Can / Can	Can / Can	10(10%)c / 10(10%)c	12
20(10%)a / 20(10%)a	20(10%)a / 20(10%)a	10(10%)c / 10(10%)c	17
10(10%)c / 10(10%)c	10(10%)c / 10(10%)c	20(10%)a / 20(10%)a	11
10(10%)c / 10(10%)c	10(10%)c / 10(10%)c	Can / Can	10
Can / Can	Can / Can	20(10%)a / 20(10%)a	9
Remainder			20 or more

Female and male analyses for the three genotype pairs are shown in Tables 3 and 4 respectively. There was heterogeneity in the error terms but as the analysis of variance is robust (Cochran 1947), this is unlikely to affect the interpretation. This heterogeneity probably arose from the different efficiencies of the three marked inversion chromosomes in preventing recombination (MacIntyre and Wright 1966), as well as the different effects of the various chromosomes being manipulated. There were significant differences between the three lines in all three chromosomes, the difference between 20(10%)a and 10(10%)c *X*-chromosomes only being significant in females. Genotype mean and main effect temperature and media mean abdominal bristle numbers for females and males have been recorded as Appendices 1 and 2.*

A common ploy in physiology is to show that an effect is due to more than one process by demonstrating an interaction with an imposed treatment. Using this approach a minimum interpretation of intrachromosome differences can be obtained, i.e. a chromosome × environment interaction indicates that chromosomes from

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two lines differ by at least two genes. However, care must be taken to distinguish between "true" interactions and interactions caused by scale effects. The male data were analysed on both normal and log scales to determine which scale was more appropriate. Conclusions were the same for both scales but the chromosome × environment interactions present on the untransformed scale were non-significant on the log scale for the Can–10(10%)c and Can–20(10%)a [but not for 10(10%)c–20(10%)a] analyses. As comparisons among analyses indicated that at least two gene differences were present on each of the chromosomes involved in the above-mentioned chromosome × environment interactions (i.e. that they were probably

TABLE 3

ANALYSES OF VARIANCE OF FEMALE ABDOMINAL BRISTLE NUMBER FOR THE THREE GENOTYPE PAIRS

Source of Variation	D.F.	Can–10(10%)c Mean Square	D.F.	Can–20(10%)a Mean Square	D.F.	10(10%)c–20(10%)a Mean Square
X	2	72·00**	2	39·45**	2	13·36**
II	2	393·45**	2	1051·32**	2	26·07**
X × II	4	1·91	4	2·79	4	2·72
III	2	227·26**	2	1329·13**	2	904·86**
X × III	4	7·68**	4	0·65	4	3·62
II × III	4	2·61	4	0·28	4	4·52
X × II × III	8	0·87	8	4·12**	8	1·19
Temperature (*T*)	1	91·36**	1	170·67**	1	279·71**
III × *T*					2	9·24*
X × III × *T*					4	5·17*
Media (*M*)	1	612·06**	1	590·00**	1	844·91**
X × *M*			2	5·67*		
II × *M*			2	17·06**		
III × *M*	2	5·60*	2	5·75*	2	26·26**
T × *M*	1	34·24*			1	30·23**
Bulked non-significant interactions	76	1·28	73	1·28	70	2·23
Between replicates (error)	108	1·26	108	1·50	108	2·08

* $P < 0{\cdot}05$. ** $P < 0{\cdot}01$.

"true" interactions) the untransformed scale was considered the most appropriate and results were presented on that scale. Means for each of the cases in which there were significant first-order chromosome × environment interactions have been recorded as Appendices 3 and 4.* The difference between Can and 20(10%)a *X*-chromosomes was due to at least two factors (*X* × media interaction in females) while the Can–10(10%)c and 10(10%)c–20(10%)a *X*-chromosome differences may be single gene differences. However, analyses of sex-dimorphism ratio (Frankham 1968) indicated that 10(10%)c and 20(10%)a differed from Can by the same two *X*-chromosome factors. Thus, the simplest interpretation of the *X*-chromosome differences is that 10(10%)c differs from Can by two factors (A, B), 20(10%)a from Can by three factors (A, B, C), and 20(10%)a from 10(10%)c by a single factor (C). In chromosome II, 10(10%)c differed from Can by a single factor D, 20(10%)a from

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Can by two (D, E) factors (II × media interactions in both sexes), and 20(10%)a from 10(10%)c by a single factor (E). Differences among the lines in chromosome III were all due to at least two genes [III × media interactions in females and

TABLE 4

ANALYSES OF VARIANCE OF MALE ABDOMINAL BRISTLE NUMBER FOR THE THREE GENOTYPE PAIRS

Source of Variation	D.F.	Can–10(10%)c Mean Square	D.F.	Can–20(10%)a Mean Square	D.F.	10(10%)c–20(10%)a Mean Square
X	1	158·96**	1	91·13**	1	1·76
II	2	167·56**	2	467·35**	2	11·39**
X × II	2	4·21**	2	1·72	2	0·62
III	2	222·23**	2	967·34**	2	470·33**
X × III	2	2·87*	2	7·86**	2	2·50
II × III	4	3·26**	4	3·90*	4	1·50
X × II × III	4	1·48	4	1·71	4	0·94
Temperature (*T*)	1	27·95**	1	36·26**	1	104·59**
III × *T*	2	3·16*	2	3·71*		
X × III × *T*					2	5·65*
X × II × III × *T*	4	2·16*				
Media (*M*)	1	390·16**	1	376·31**	1	497·17**
II × *M*			2	3·62*		
III × *M*					2	17·76**
T × *M*	1	12·56**				
Bulked non-significant interactions	45	0·72	48	1·28	48	1·59
Between replicates (error)	144	0·82	144	1·18	144	1·56

* $P < 0·05$. ** $P < 0·01$.

III × temperature interactions in males for both Can–10(10%)c and Can–20(10%)a; and for 10(10%)c–20(10%)a III × media interactions in both sexes, a III × temperature interaction in females, and *X* × III × temperature interactions in both sexes].

TABLE 5

SIMPLEST INTERPRETATION OF DIFFERENCES AMONG Can, 10(10%)c, AND 20(10%)a

Line Comparison	Factors Differentiating the Lines		
	X	II	III
Can–10(10%)c	A, B	D	F, G
Can–20(10%)a	A, B, C	D, E	F, H
10(10%)c–20(10%)a	C	E	G, H

The most likely interpretation is that 10(10%)c differs from Can by two factors (F, G), 20(10%)a from Can by two factors (F, H), and 20(10%)a from 10(10%)c by two factors (G, H). Thus, differences among the three lines were due to a minimum of eight factors as shown in Table 5.

Comparisons of the average effects of the various chromosomes give an indication of the effect of the factors. These estimates are biased if chromosome-interaction effects are present as the chromosomes are compared in partly different genetic backgrounds. However, chromosome-interaction effects were not large in comparison to the main effects so the biases should not be serious. The average effect of factors A and B combined was 2·00 and 1·72 bristles in females and males, respectively (they cannot be separated as they do not appear alone), C 0·85 and 0·18, D 4·60 and 3·03, E 1·21 and 0·79. The effects of factors F, G, and H could not be separated but F plus G had effects of 3·55 and 3·51, F plus H 8·59 and 7·33, and the substitution of H for G effects of 7·08 and 5·09. It seems that factors D and H and to a lesser degree E have relatively large effects.

An indication of interactions between these factors can be obtained from the analyses of variance. There were significant $X \times$ III interactions in both sexes, and significant $X \times$ II, II $\times$ III, and $X \times$ II $\times$ III $\times$ temperature interactions in males between Can and 10(10%)c. Between Can and 20(10%)a there was an $X \times$ II $\times$ III interaction in females and $X \times$ III and II $\times$ III interactions in males, while between 10(10%)c and 20(10%)a there was a significant $X \times$ III $\times$ temperature interaction in both sexes. The chromosome interactions were not simply scale effects as a log transformation of the male data removed none of the interactions in males. The analyses of sex-dimorphism ratio (Frankham 1968) also revealed interactions between some of the chromosomes.

IV. Discussion

The simplest interpretation of these results is that five and seven factors, with effects varying from small to large and between which there were some interactions, were responsible for selection response in the two lines. However, the factors within chromosomes were only separated because of their different physiological effects so this experiment does not establish whether the factors were single genes or multiple genes. Spickett's (1963) evidence that each of the genes he investigated had a different physiological effect indicates that these factors may have been single genes. The results are consistent with the suggestion that few genes are mainly responsible for selection response (Thoday 1967) but do not provide critical support for it.

The most important finding was that four of the five factors responsible for selection response in 10(10%)c also contributed to response in 20(10%)a. Further, Frankham, Jones, and Barker (1968) obtained evidence that the factors for response in 20(10%)a were also contributing to response in 40(10%) (the highest selection line of Jones, Frankham, and Barker 1968) while 10(10%)b differed from these lines to an unknown degree. Thus, these results are similar to those of Thoday, Gibson, and Spickett (1964) and Spickett and Thoday (1966) in that both indicate that selection lines with similar origins and at the same level of response utilize most of the same genes, while higher lines change at most of the same loci as lower ones, plus at extra loci. Sturtevant (1918) also found that identical genes were utilized in two of his selection lines from a similar origin while other lines were different to an unknown degree. Sismandis (1942) found that response in four selection lines originating from progeny of the same female could be separated into two outcomes and that

the two lines having each outcome were apparently identical (the interrelation between the two outcomes was not determined). Mather and Harrison (1949), King and Somme (1958), Scowcroft (1966), and Frankham, Jones, and Barker (1968) have found that selection response in replicate lines was due to at least partly different genes, the degree of similarity not being determined. However, more detailed analyses in the latter two cases revealed important genetic effects common to a number of lines. Fraser (1967) pointed out that the major portion of the differences among the lines of Fraser *et al.* (1965) (including those analysed by Scowcroft 1966) was due to the presence or absence of a single gene. Frankham, Jones, and Barker (1968) were able to show that response in their highest line involved the same genes utilized in a line showing lesser response, plus extra genes. Thus, the presence of genetic differences between selection lines gives little or no indications as to whether they have genes in common. The poor selection response McBride (1965) obtained in crosses between replicate selection lines (not necessarily plateaued) from single samples of the same base population indicates that they utilized most of the same genes, while crosses between lines from different samples gave zero or only moderate response as compared with the higher parent, indicating that they probably utilized many common genes. Milkman (1965) showed that three of five crossveinless strains, which originated by selection from separate inseminated females captured at the same location, were identical and the other two were closely related. Roberts (1967) has also suggested that much of the response in different mouse selection lines (with some overlap in their base populations) was due to the same genes and that they differed, mainly at linked loci. All this evidence suggests that the genes utilized during selection are not simply a random sample of the initially segregating loci affecting expression of the character.

There are two main ways (not mutually exclusive) in which replicate selection lines would be expected to utilize many of the same genes. The first involves non-random utilization of genes only within the limits set by a range of gene effects, a range of gene frequencies, and advantageous or disadvantageous linkages, i.e. the probability that a particular gene will contribute to selection response is increased with increasing size of effect, increased with higher base population gene frequencies, and decreased by linkage to genes with disadvantageous effects on the character. Genes with large effects and at intermediate to high gene frequencies in the base population are expected to contribute to response in all selection lines, genes with intermediate effects and intermediate frequencies are expected to contribute to response in most lines, genes with small effects and intermediate frequencies or large effects and low frequencies are expected to contribute to response in some lines, and so on. The second involves non-random utilization of genes because of gene interaction effects.

A number of lines of evidence suggest that gene-interaction effects are involved (but not necessarily the complete explanation). There were significant interactions between chromosomes in the present analysis. Cavalli and Maccacaro (1952) found that positive interactions and sequential utilization of genes were involved in selection response. Two of the genes located by Spickett and Thoday (1966) were not utilized until they were involved in a favourable positive interaction. Further, the high degree of use of the same genes in replicate lines with higher lines utilizing extra

genes, as found by Milkman (1965), Thoday, Gibson, and Spickett (1964), and Spickett and Thoday (1966), combined with the results presented here requires a rather specific set of circumstances with the first explanation and indicates that the first explanation alone is not adequate. The different lethal genes present in our different lines (Frankham, Jones, and Barker 1968) indicate that gene interactions alone are not a sufficient explanation.

The available evidence indicates that the effects considered under both explanations are all involved in the non-random utilization of genes during selection. In any case present quantitative genetic models need to be critically examined as accumulating evidence points to the invalidity of assumptions (especially those concerning size of gene effects, number of genes, and importance of gene interactions) underlying them. As Wright (1959) and Dickerson (1963) have pointed out there is need for models in quantitative genetics based on generalizations at the physiological and biochemical level.

V. Acknowledgments

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