CHAPTER 3

SELECTION EXPERIMENT

3.1.1. Introduction: Literature Review

There have been a number of experimental comparisons of undivided and subdivided populations under artificial selection. Most have used models approximating that of Wright (1922); a population subdivided into a number of small lines, with regular selection between and crossing among the lines. A variety of traits in a number of species have been used, but questions arise as to the usefulness of much of the work reported for answering basic questions about the Shifting Balance Theory.

Bowman and Falconer (1960) described the results of inbreeding with and without selection, and of cycles of inbreeding and crossing among inbred lines, on litter size in mice. An initial phase of inbreeding within 10 independent lines showed decreases in litter size to be linearly related to the inbreeding coefficient of litters, with selection within lines failing to retard this decline significantly. The amount of selection applied was however small, and it was concluded that "the failure of selection within lines to reduce the rate of decline is therefore to be attributed to the low intensity of selection and is not necessarily proof that the selection itself was ineffective."

The second phase of this experiment incorporated selection between partially-inbred lines. The first cycle of inbreeding with crossing produced a significant improvement in litter size much faster than selection without inbreeding. Two further cycles were carried out, but neither achieved the same improvement as the first, and in fact the third crossing produced smaller litter sizes than a non-inbred control. This lack of response was attributed to

ineffectiveness of selection, resulting from low repeatability of line means over successive generations, and to inability to sufficiently reduce the level of inbreeding in the second and third crossbred generations.

The authors concluded that cyclical inbreeding and crossing was not a practical proposition, because of the costs of maintaining poor inbred lines, the difficulty in achieving sufficiently low levels of inbreeding in cross-bred generations, and the requirement of a very large number of inbred lines in order to ensure recovery of high levels of heterosis in crossing, particularly in later cycles. It should be pointed out that the character observed, litter size, appeared to have a significant level of dominance variance, with no significant over-dominance or epistasis.

Nassar (1969) reported a comparison of responses to selection for increased scutellar bristles in two laboratory stocks of D. melanogaster, in single large lines, and a number of small lines. The comparison was run over 22 generations of selection, and for a number of generations (differing between stocks) responses in one or more of the small lines was higher than in the large line, and the mean response of the small lines was similar to, or better than, that of the large line. After 10-15 generations, the large line became markedly superior in both stocks, both compared to both individual small lines, and their mean. Further, it was concluded that the results provided no evidence for multiple peak epistasis, since all responses in large and small lines appeared to be due to genes located on the second chromosome.

Given the variability among small line means, and their average superiority over large lines for 10-15 generations, it is unfortunate that this experiment did not include some form of crossing amongst small lines. While the ultimate level of response may not have been increased, the rate of response in the early generations may have been improved. This could rely on no more than selection against lines in which unfavourable alleles were segregating at high

frequencies, due to initial sampling and/or drift from inbreeding. Further results reported by Nassar (1971) suggest that only a single epistatic complex existed in this genetic system, so that no opportunity existed for different small lines to reach different peaks in an adaptive landscape.

Falconer (1971) showed that residual genetic variation existing at a selection limit could be exploited by inbreeding, then crossing amongst selected lines. The study used litter size in mice, and analysis of the genetic architecture of the trait at the limit, and in crosses, suggested that both the limit, and responses to crossing amongst inbred lines at the limit, were due to recessive alleles affecting fertility through loss of eggs and embryos, principally before implantation. In discussion of the results it was pointed out that the selection limit which inbreeding with crossing had overcome was in fact more apparent than real, in that further selection would have eventually removed the recessive alleles limiting response. Further, these results provide no evidence for any selection among interaction systems, before, at, or after the limit. Thus while suggesting a method for improving rates of responses at or near selection limits imposed by segregating recessives, these results offer no support for subdivision with crossing as a more general strategy.

Al-Murrani and Roberts (1974) applied Falconer's (1971) procedure to lines of mice at a selection limit for bodyweight, again apparently caused by segregation of recessive genes of quite large effect. In contrast to Falconer's finding, no appreciable gain from elimination of these recessives was obtained.

Comparison of inbreeding/crossing cycles with simple mass selection, from the start of a selection programme, was carried out by Goodwill (1974). The selected trait was average pupa weight of Tribolium castaneum, and two models of inbreeding/crossing were used: one with crossing every four generations, the other, every eight. The latter produced significantly less response than either mass selection or the 4-generation inbreeding/crossing cycle. Realized

heritability was highest for the mass-selected population, and lowest for the 8-generation inbreeding/crossing cycle, but these differences were not significant. In this study then, inbreeding did not significantly hinder response to selection (i.e. its effectiveness), but did reduce rate of response per generation. Once again, no advantage was observed in a programme of within and between-line selection. It was noted however, that in most generations of this study, there were lines within the subdivided populations superior to the large population for the selected trait, and that these might be usefully expanded to form the basis of new lines. The value of this suggestion would depend on whether such superiority reflected genetic superiority over the large population, and leads to the possibility that a more flexible/dynamic method of exploiting between line differences might be worth consideration.

Madalena and Robertson (1975) compared responses to selection for reduced bristle number in D. melanogaster in single large lines with those obtained in small lines crossed either once to form new composite populations, or in repeated cycles of varying length. None of the subdivided population structures produced as much response as in the single large line, providing further support for Robertson's (1960) conclusion that subdivision will not increase response when gene action is mostly additive.

Similar comparisons between population structure models were reported by Katz and Enfield (1977) and Rathie and Nicholas (1980). Katz and Enfield were selecting for increased pupa weight in T. castaneum, Rathie and Nicholas for increased abdominal bristle number in D. melanogaster, and in neither case was subdivision beneficial. In their conclusions, Rathie and Nicholas pointed out that their results (and those so far reviewed here) in fact throw little light on the validity of Wright's Shifting Balance Theory since there was no suggestion that epistasis was important in the selected trait(s). Further, they pointed out that in fact, "direct evidence on Wright's shifting balance theory

can only be obtained from selection programmes in which epistasis is certain to be present."

The one report of advantage in subdivision in work of this kind to date is that of Katz and Young (1975). They selected for increased adult bodyweight in D. melanogaster in three population structures: a single large line, and two models where the population was subdivided into a number of small lines/ demes. The mode of subdivision was however different from all the studies so far reported, in that there was migration at random, in a cyclic pattern, among demes, at rates of 5% and 10% in the two subdivided treatments. The random nature of the migration ensured that no between-deme selection occurred (i.e. all selection in both subdivided treatments was within-deme), but was aimed at allowing spread of favourable genes/gene complexes throughout the entire array of demes.

Although larger selection differentials were applied to the single large population, significantly greater responses were obtained in both subdivided lines than in the single large line. Consequently realized heritabilities were also higher for the subdivided populations. These effects were consistent over sexes. Thus this study provides evidence supporting subdivision, and it is of interest that this is despite a high level of additive genetic variance in the base population (a heritability estimate of 0.58 ± 0.22), conditions not expected to lead to advantages in subdivision.

The possible explanations suggested for these results were either that in the subdivided systems there had arisen unique interactions within demes followed by fixation of these interactions, or that the extra genetic variance generated between demes by virtue of the inbreeding within them, was utilised at least in part by selection. This extra variation (between-demes) was presumed to become available via the periodic migration among demes.

While both alternatives are feasible, the results and analyses do little to

help choose between them, and do not help to validate the Shifting Balance Theory. When examined in the light of the other studies reported, it is impossible to determine what aspect of this design led to the advantage in subdivision. The trait selected for (adult bodyweight in D. melanogaster) may have been influenced to a greater degree by epistasis than either Drosophila bristle number or Tribolium pupa weight, and the inter-demal migration may have been a more effective means of spreading favourable alleles/gene combinations; or some combination of both factors may have been effective.

A series of papers also concerned with population subdivision but having a somewhat different perspective, are those of Wade and McCauley, investigating group selection using T. castaneum (McCauley and Wade, 1980; Wade, 1976; 1977; 1978; 1982; and Wade and McCauley, 1980). These have all been concerned with the effects of population subdivision on changes in a populational phenotype, population size, and the variance among demes in that character. This trait, together with the fact that no artificial selection on individuals was applied in any of their studies, are two important differences between their work and the methods of other studies reported here. Their various papers have attempted to show that significant genetic differentiation can develop between even quite large demes, and in the presence of migration at rates as high as 25%. Further, they attempted to show that this between-deme genetic variance was available to, and could provide the basis for, successful group selection.

A concept discussed by McCauley and Wade, and further investigated by Slatkin (McCauley and Wade, 1980; Slatkin, 1981) is that of populational heritability. This is analagous to the concept of heritability of the family mean (Falconer, 1981), but differs from it in that it refers to a larger unit of population structure than families, and it can include non-additive variance among sub-populations (whereas the heritability of family means is based on additive variance).

The results from this series of papers may be summarised as follows:

- the effects of inbreeding on population growth rate and productivity decrease as effective deme size increases.
- genetic differentiation between demes is significant even at quite high levels of inter-deme migration.
- the among-deme component of total phenotypic variance, or populational heritability, can reach 30-40% after only c. 5 generations, and is largely independent of local deme size.
- the populational heritability decreases as migration between demes increases.
- group selection between demes can exploit the among-deme variance successfully; a proportional response analogous to the realized heritability being significantly different from zero.

In the summary of his 1982 paper, Wade suggested that the results of these studies indicate that population structure and intergroup selection may play a larger role in the evolution of populations than is generally acknowledged.

One further study that has investigated the effects of population subdivision on response to directional selection, is the simulation work of Madelena and Hill (1972). In this investigation cycles of inbreeding with crossing at intervals were compared with single large populations for various combinations of sub-line sizes and length of crossing cycle. The genetic models used were of additive, or completely dominant genes, and varying recombination frequencies were included.

For completely additive gene action, some short-term gains were achieved over simple mass selection in a large population, but the limit was reduced. The authors develop theory to show, as did Baker and Curnow (1969), that while individual sub-lines may be superior to the large population, the duration of this superiority is likely to be short.

With dominance models results were somewhat different. With recessive alleles present at low frequency, additive variance was increased in small populations, and response in the subdivided models was greater than for single populations. These increased responses were improved if between-line selection was delayed. As with the additive model however, between-line selection depressed the limit as a result of loss of favourable alleles. When recessive alleles were at intermediate or high frequency, subdivision was not of benefit, and delaying between-line selection depressed both short-term responses and the final limits to response.

The last sentence of Madalena and Hill's conclusions is a useful statement about most of the work reviewed here: "we have not investigated epistatic models, for which these line crossing systems were originally proposed by Wright, and some studies with these models could be rewarding."

The general conclusions regarding population subdivision that may be drawn from the papers surveyed here are:

- sub-lining with crossing at intervals, is not very useful except perhaps to hasten removal of rare recessives, either early in the course of selection, or at/near selection limits.
- sub-lining with migration does not appear to fully inhibit genetic differentiation of demes/sub-lines.
- the genetic architecture of the trait chosen appears to be crucial: the only example where subdivision with selection was not disadvantageous involved selection for increased adult bodyweight in D. melanogaster (Katz and Young,1975). As pointed out previously, this study utilised a different method of population subdivision from all the others. The only evidence showing successful between-group selection used a populational characteristic, namely total population size, rather than an individual trait.

It is clear from this brief survey that experimental evaluation of Wright's

recommendations regarding population structure has covered only restricted sections of the field of genetic architectures and population structures possible. In part, this is probably due to Wright's (and Lush's, 1947) recommendations for livestock at least being fairly specific, and in part due to the desirability of modelling traits of practical significance. In the broader evolutionary context, questions arise as to what trait(s) in fact to examine, together with more difficult questions concerning the types of genetic changes with which evolutionary study should be concerned.

3.1.2. Introduction: Outline of the Experiment

The experimental evaluation reported here extends the work in this area while at the same time repeating previous comparisons, in a different population. The sub-lining/crossing regime previously evaluated in a number of separate studies was included, this time with selection for a trait in which subdivision has been reported as favourable, viz. adult bodyweight in *D. melanogaster*. The model of Katz and Young (1975), within-deme selection with random cyclic migration, was included. Finally a new population model was evaluated, which incorprated variable inter-deme migration, both the direction and amount of migration being dependent upon differences in phenotypic means between demes.

The last-mentioned treatment represented an attempt to model all three phases of the Shifting Balance Theory:

- random drift in numerous small demes leading to genetic differentiation amongst them, and to the crossing of saddles in the adaptive landscape in one or more demes.
- within-deme selection in such demes bringing gene frequencies within them to the set particular to the new adaptive peak.

- and finally, excess diffusion from such demes bringing neighbouring demes to control by the same set of gene frequencies.

For reasons of logistic simplicity, no attempt was made in this study to include the effects of isolation by distance in the model; whether demes contributed migrants to others, or received them, was solely determined by their respective phenotypic merits.

A further peculiarity of this "Wrightian" model was that migration between demes was exclusively via females, prior to mating. This method was chosen simply because it was the method employed by Katz and Young (1975), and use of some different method would make comparisons difficult. Of course, many other methods could have been chosen which would have been appropriate to various species and population models: the consequences of the particular model used here, and of alternative models, will also be discussed later.

The inclusion of gene flow from superior to inferior demes was felt to have relevance for both livestock and natural situations. In many species of livestock, individual studs, perceived to be producing genetically superior stock, act as sources for other producers, either stud or commercial. This superiority may not be great, (or even exist) and there may be some divergence in the goals of selection among different studs. This situation is that which Wright views as having led to the diversity of present day breeds of livestock, and to the variation within these breeds. The experimental model also represents a grossly simplified picture of the situation in natural populations: in this case adult bodyweight and not fitness is the trait which determines whether individuals pass on genes to subsequent generations, and migration of individuals into and out of demes is determined by the mean "fitness", or selective merit, of their parent demes.

The last point raises the issue of whether in fact some more direct component of fitness should have been selected for, as in the studies of Wade

and McCauley. However, to facilitate both comparison with the majority of previous work in this area, and to simplify application of individual selection, adult bodyweight of D. melanogaster was chosen.

In conclusion, this study was aimed at extending the scope of earlier comparisons, at the same time linking some previously unconnected researches. In conjunction, a new model, designed to include more of the components of Wright's theory, was investigated.

3.2. Materials and Methods

The basic experimental procedure, common to all treatments, was as follows: Five pairs of adult flies were mass mated in half-pint bottles on F1 yeasted medium (see Chapter 2) for three days, after which time the parents were discarded. All bottles were stored at 25°C and 70% RH. Approximately 8-9 days after start of mating, virgins began emerging and were collected. In all treatments, virgins were as far as possible collected in batches of 50 (25 males and 25 females). Where this was not achieved a further collection(s) was carried out, with the aim of obtaining 25 contemporaneous virgins of each sex, so as to minimise the effects of age differences on weight at scoring. All virgins were stored on yeasted medium, again at 25°C and 70% RH, in groups of 25 in 3x1 inch glass vials. Adult flies were weighed individually using a Mettler HL52 electronic micro-balance. In order to simplify work routines, a two-week cycle of operations was followed, which meant that weighing was always carried out 10.5 days after start of mating. This meant that weighing was not always at 3 complete days after emergence, particularly in the subdivided treatments. Preliminary selection was carried out during weighing, the heaviest 10 flies of each sex being retained from each bottle. Final selection was carried out at mating, which occurred at the end of 14 days after the previous mating. All

flies were therefore 5-6 days old at the start of mating. In all cases, collections took place from 8.00-9.00 am and from 4.00-5.00 pm. The light-dark cycle was 12 hours light, 12 hours dark (from 6.00 am to 6.00 pm light).

In all treatments, the population size was 50 pairs, with these being distributed equally amongst 10 bottles, so that the basic mating unit in all cases was 5 pairs. The proportion selected in all treatments was 20%, with adjustments according to treatment as will be described. The different population structures were simulated by different methods of allocating individuals to the 10 bottles/demes, as follows:

a) Mass Selection in a Single Large Population:- the 50 selected pairs (5 pairs from each bottle) were allocated at random to 10 new bottles. All bottles therefore contributed equally to each generation.

b) Sub-lining with Crossing at Intervals:- at Generation 0, 50 pairs of adult flies were randomly allocated to 10 bottles, 5 pairs to each. Selection was then carried out on a within-bottle basis (i.e. no mixing before allocation to new bottles each generation) for 4 generations. After 4 generations of within-bottle selection, the phenotypic mean for each bottle/sub-line over the previous three generations was calculated (on the basis of individual generation means for each sex, pooled to produce an across-sexes mean by taking logs). The 5 sub-lines having the highest three-generation average were selected, and double the normal numbers of virgins scored and selected from these. The 50 pairs of selected flies were then randomly allocated to 10 bottles, to form 10 new sub-lines. This process can be summarised as follows:

Generation	0	Random Allocation to 10 Bottles
Generations	1,2,3,4	Selection within bottles/sub-lines
Generation	5	Selection within 5 selected sub-lines, selected flies allocated at random to 10 new sub-lines

Generations 6,7,8,9 Selection within bottles/sub-lines

etc.

This treatment is equivalent to Model (iv) of Madalena and Robertson (1975), and to Model CC of Rathie and Nicholas (1980).

c) Sub-lining with Cyclical Random Migration:- as in b), 10 separate sub-lines were initiated by allocation of 5 pairs at random to each of 10 bottles. Thereafter, selection took place within bottles/demes. At each generation, migration between demes was applied by transfering 1 selected virgin female from each bottle/deme to its neighbour in a cyclical pattern. The migrating females were chosen at random from within each group of 5 selected females, and the pattern of transfer was always bottle 1-> bottle 2, 2->3, 3->4,..., 10->1. The migration rate applied was therefore 10%, but with no selection between demes involved. This model is Model B1 of Katz and Young (1975).

d) Sub-lining with Inter-Deme Selection:- 10 demes were initiated at Generation 0 by random allocation of 5 pairs of flies to each 10 bottles. Thereafter selection was applied within-bottles by retaining the heaviest 5/25 flies of each sex. In addition, selection between demes was applied via variable migration, such that demes with higher phenotypic means contributed some selected individuals to lower ranking demes. The exact procedure followed to achieve this was as follows:-

(i) Calculate a pooled mean for each deme correcting for sex and variability within sex:

$$\bar{X}_{ij_p} = \frac{n_{ij}\bar{x}_{ij}\sigma^2_{i\,\sigma\,\varphi} + n_{ij}\bar{x}_{ij}\sigma^2_{i\,\varphi\,\sigma}}{n_{ij}\sigma^2_{i\,\sigma\,\varphi} + n_{ij}\sigma^2_{i\,\varphi\,\sigma}}$$

where $\bar{X}_{ij_p}$ = pooled mean of the j^{th} deme in generation i.

n_{ij} = number of males/females scored in the j^{th} deme.

$\bar{x}_{ij}$ = phenotypic mean for j^{th} deme.

σ_i^2 = phenotypic variance of entire population in generation i.

(ii) Calculate an average pooled mean over three generations (the current and two immediately preceeding generations) for each deme, e.g. in generation t:

$$\bar{x}_{t,j} = \frac{1}{3} \sum_{i=t-3}^{t} x_{i,j}$$

(iii) Calculate the mean and variance for these three-generation meansover the 10 demes.

(iv) Express each deme three-generation mean as a deviation from the overall mean, standardised by the standard deviation amongst them.

(v) Convert this deviation to a migrant pool contribution by multiplying by the value:

$$\text{Conversion factor} = \frac{20}{\sum_{i=1}^{10} |\text{Individual Deme Deviations}|}$$

In effect this scaled the contributions to within the range +10 to -10, and ensured that the total of positive and negative contributions to the migrant pool were both 10 (i.e. +10 and -10). The migrant pool contributions were then rounded to the nearest integer, to give the number of selected parents that were to be "sent to" the migrant pool, in the case of superior demes, and of immigrants received from the migrant pool in the case of inferior demes.

In this treatment, the migrants were virgin selected females, and allocation from the migrant pool to the recipient demes was at random. To help clarify the procedure, the following example is included:

Deme Contribution to Migrant Pool	Number of Females Selected /Retained
-4	Select only the heaviest 1 female from 25 scored; accept 4 female immigrants
0	Select the heaviest 5 females from 25 scored. No migration
+5	Select and retain the heaviest 10 females from those scored. Choose 5 at random, from these 10, to be emigrants.

Thus at every generation, a migrant pool of 10 virgin females was set up, comprising selected individuals from demes superior in mean bodyweight over the previous three generations. The poorer demes then received contributions from this migrant pool according to their degree of inferiority. The overall migration rate was therefore 10%, as in Treatment C, although for individual recipient demes in any one generation, it could be as much as 50% (all female parents immigrants).

Phenotypic means were averaged over three generations as a way of better estimating the mean genetic value of each deme. This model represents an attempt to simulate the three phases of the Shifting Balance Theory:-

1. Drift of local populations due to small deme size (5 pairs), and limited

gene flow between demes.

2. Mass Selection within demes, allowing for local adaptive peaks to be approached.

3. Interdeme Selection, accomplished by gene flow from superior to inferior demes, producing partial replacement of poorer populations by samples from better ones.

e) Control:- a population of 50 pairs was maintained, 5 pairs per bottle as before. At each generation, 5 pairs of individuals were collected at random from each of the 10 bottles. These were then scored and allocated at random to 10 fresh bottles. This constituted an unselected control line.

The initial period over which these treatments were compared was 20 generations of selection (i.e. G_0-G_{21}). Two replicates of each treatment were run, with the two replicates of the entire comparison being run on alternate weeks. Analysis of the results was therefore in terms of blocks rather than replicates (Hurlbert,1984).

At the end of 20 generations, results to that point were reviewed, and it was decided to continue Treatments A, D, and E alone. The reason for this was that in Treatment D, Block 1, a quite unusual genetic effect had appeared and been spread throughout the system of subpopulations, and further investigation was warranted.

At G_{27}, a new comparison amongst the three remaining treatments was initiated. From Treatment A, 50 pairs were sampled at random twice, to set up two new treatments: one, a relaxed selection line (Treatment A_1), and the other using the same procedure as Treatment D (Treatment A_2). The rationale behind these treatments was that responses had slowed considerably in Treatment A, and information about the genetic properties of this treatment at this apparent limit might be obtained from results in these new treatments.

3.3. Results

3.3.1. Selection Differentials

Table 3.1 shows the cumulative selection differentials attained in the selection treatments over 20 generations. In both blocks and sexes, selection differentials were highest in Treatment D, where selection was applied both within and between demes in every generation. No clear differences between the remaining treatments are visible: slightly less selection on males and more selection on females was achieved in Treatment C as compared to Treatment A, suggesting differences between males and females in the effect of subdivision on phenotypic variation.

Table 3.1: Cumulative Selection Differentials (mg)

Treatment	Block 1		Block 2	
	Male	Female	Male	Female
A	2.0715	3.5564	1.8513	3.2991
B	1.8309	3.5260	1.8326	3.4700
C	1.8459	3.5758	1.8070	3.4223
D	2.1243	3.8442	1.9803	3.5054

When the cumulative selection differentials are expressed in units of the mean phenotypic standard deviations of the respective treatments (Table 3.2), it appears that selection applied in the subdivided treatments was generally less than that applied in Treatment A, and that amongst the subdivided treatments, more selection, in units of standard deviations, was applied in Treatments B and D than in Treatment C.

Table 3.2: Cumulative Selection Differentials, Expressed in Units of Standard Deviations

Treatment	Block 1		Block 2	
	Male	Female	Male	Female
A	23.57	23.23	23.92	23.23
B	21.93	21.55	22.08	21.55
C	20.72	20.37	20.84	20.88
D	21.03	23.16	22.74	21.72

This effect can also be seen when selection differentials are expressed as a proportion of that expected from selection theory. The expected total selection differential for 20 generations of selection is:

$$C.S.D. = 20 \times i \times \sigma_p$$

where C.S.D. = Cumulative Selection Differential

i = 1.345 (sampling 5 from 25 individuals, Becker (1984))

σ_p = population standard deviation, phenotypic.

In calculating these values, estimates of within-deme variance were used, since this is the variation available for within-deme selection. These results are presented in Table 3.3, which shows that the total amount of selection applied in the subdivided populations was a smaller proportion of that expected than for treatment A. Thus, while more phenotypic variation was present, selection did not utilise it as fully. Katz and Young (1975) reported lower cumulative selection differentials in subdivided vs. undivided lines, but no information was presented on levels of variation in the respective treatments. Hammond (1973) found no differences in the ratio of actual to expected selection differentials between different population sizes.

Table 3.3: Proportion of Expected Selection Differential Realised (%)

Treatment	Block 1		Block 2		
	Male	Female	Male	Female	Mean
A	81.63	94.60	88.70	93.79	87.18
B	51.66	78.56	72.42	80.42	70.77
C	63.85	58.75	75.15	80.07	70.96
D	71.29	87.90	72.57	77.56	77.33

The observation in this study, namely increased variation available for selection but reduced amounts of selection applied, in the subdivided treatments, suggests that departures from normality occurred in the small, partially inbred demes of the subdivided treatments. This hypothesis was tested by calculating skewness and kurtosis for all bottles/subpopulations of the four selected treatments in each of four generations, G_5, G_{10}, G_{15} and G_{17}. The results are presented in Table 3.4. While they do not represent an exhaustive analysis of the situation, there was a tendency for all treatments to show skewing of subpopulation distributions to the left (lighter bodyweights), particularly Treatment B, and for all treatments to show some degree of leptokurtosis. This tendency was more marked in the subdivided treatments. If these sample averages are an accurate guide, then much of the increased phenotypic variance present in the demes of the subdivided treatments was due to an extended tail at the lower end of the phenotypic distribution, and thus unavailable to selection for increased bodyweight (the effect on the phenotypic distribution of a major gene/gene combination arising in Treatment D, block 1, will be discussed in Chapter 4 of this thesis).

Table 3.4a: Average Skewness, over Subpopulations (q_1)

Treatment	Block 1		Block 2	
	Male	Female	Male	Female
A	-0.2004	-0.1333	-0.0675	-0.1360
B	-0.4315	-0.4893	-1.0231	-0.5240
C	-0.2223	0.0949	-0.0533	-0.1200
D	-0.0972	0.1499	-0.1470	-0.0937

Table 3.4b: Average Kurtosis, over Subpopulations (q_2)

Treatment	Block 1		Block 2		Mean
	Male	Female	Male	Female	
A	0.4147	0.4364	0.3801	0.4020	0.408
B	5.7906	0.5919	3.4838	0.5177	2.596*
C	1.0382	3.4964	0.5372	0.5269	1.400
D	0.9632	1.6512	0.3149	0.4555	0.846

S_{g2} = 0.887 *P<0.05

Three factors may contribute to increased phenotypic variance in small inbred lines:

- linkage disquilibrium
- increases in environmental variance
- changes in frequency of rare recessive (and dominant) alleles (Falconer, 1981). The increased phenotypic variance in the subdivided selection treatments seems likely to be due to changes in frequency of rare recessives, since the skewness and kurtosis statistics indicate largertails at the lower end of the distribution. Such distributions would have apparently more variation available to selection, whilst in fact variation in the direction of increased weight might be reduced: this would lead to increased discrepancies between expected and realised selection differentials.

The total selection differential applied to each treatment may be

partitioned in the case of Treatments B and D into between-deme and within-deme components. For these the between-deme selection differentials may be estimated using a method analogous to that used for selection among individuals, the mean difference between the selected demes and the overall mean in the generation at which selection takes place. In treatment D, no between-deme selection was applied on males, but some of the response in male bodyweight will however be due to correlated responses to inter-deme selection via females. The partitioned selection differentials for Treatments B and D are presented in Table 3.5.

The results for Treatment B show that the amount of between-deme selection declined over the three rounds of sub-lining/selection, and that the total amount of between-deme selection represented only c. 3% of all selection applied. Thus even if this selection were 100% effective, the gain due to sub-lining was very small. In contrast, between-deme selection in Treatment D accounted for c. 10% of the total amount of selection applied. Of the two treatments which exploited between-deme selection, Treatment D appears to have been more effective. Madalena and Hill (1972) compared different intensities of between-line selection using simulation, and found that increasing the intensity of between-line selection increased responses in the short-term (up to 5 generations), but beyond that point structures using lower intensities of between-line selection had superior responses.

Table 3.5: Partitioning of Selection Differentials, Treatments B and D (mg)

Treatment	Block 1		Block 2	
	Male	Female	Male	Female
B: Between-Deme selection at:				
G4	0.0337	0.0462	0.0382	0.0674
G9	0.0103	0.0132	0.0186	0.0479
G14	0.0075	0.0262	0.0176	0.0300
B: Total Between-Deme	0.0483	0.0856	0.0641	0.1452
Total Within and Between-Deme	1.8309	3.5260	1.8326	3.4700
Between-deme as % of Total	2.638	2.427	3.496	4.186
D: Between Deme	-	0.3896	-	0.3527
Total Between- and Within-Deme	2.1243	3.8442	1.9803	3.5054
Between-Deme as % of Total		10.135		10.062

3.3.2. Responses to Selection

Absolute changes in bodyweight are presented in Table 3.6. The main point of interest is the increase in female bodyweight in the unselected control. This is most likely to have been due to a positive correlation between bodyweight and fecundity, with the result that natural selection in the laboratory would favour increased bodyweight. Such a positive correlation has been reported elsewhere (Martin and Bell, 1960; Misra, 1970). The lack of any significant increase in male bodyweight in the unselected control suggests only a weak genetic correlation between male and female bodyweight; this is in agreement with the base population estimates for this study (see Table 2.6).

Responses corrected for changes in the control line are presented in Table

3.7. The first point to note is that, with the exception of Treatment C, responses were larger in Block 1 than Block 2. Unfortunately, no test of significance for blocks is possible in this design; it must be assumed that some combination of initial sampling, accumulated environmental effects, and sampling during the course of the experiment, account for the differences between blocks. The effect can be seen also in the unselected control (Table 3.6).

Table 3.6: Absolute Responses to Selection (mg)

Treatment	Block 1		Block 2	
	Male	Female	Male	Female
A	0.3560	0.7922	0.2715	0.7065
B	0.2238	0.6068	0.1315	0.4186
C	0.1732	0.4222	0.1641	0.4842
D	0.3585	0.5768	0.2573	0.4517
E	0.0204	0.1156	-0.0193	0.0833

Table 3.7: Corrected Responses to Selection (mg)

Treatment	Block 1		Block 2	
	Male	Female	Male	Female
A	0.3356	0.6766	0.2908	0.6232
B	0.2034	0.4912	0.1508	0.3353
C	0.1498	0.3066	0.1807	0.4009
D	0.3381	0.4612	0.2766	0.3684

The responses in all treatments are shown plotted against generation number, in Figures 3.1-3.4. (All figures relating to this Chapter are presented together at the end of the Chapter, pp. 93 - 107). The total responses, and the responses vs. time, show that for both blocks and sexes, responses were greatest for Treatment A (the mass-selected, single large population). For male bodyweight, Treatment D showed very similar responses to Treatment A in both blocks, and Treatments B and C showed 50-60% of response in A, although their

order was reversed in the two blocks. For female bodyweight, Treatment A again showed greatest response, while subdivided treatments achieved c. 50-65% of response in A. Further, the ranking on response was different for the two blocks for the subdivided treatments.

The graphs of corrected response against generation number (Figs. 3.5-3.8) show that during the first 7 generations, male bodyweight differences between the treatments were not great, but after that point Treatments A and D were clearly superior to B and C. Treatments B and C appear to have plateaued by approximately G_{16} in both blocks, and in Block 2, treatments A and D were not clearly responding after G_{18}. Distinctions between treatments over time in female bodyweight were less clear, although Treatment A was superior to the subdivided treatments over most of the course of selection. Evidence for plateauing is less clear than for male bodyweight, although in block 2, the subdivided treatments showed reduced response rates after c. G_{14}.

Tables 3.8-3.10 (following pages) show the model for analysis of variance of response, the AOV itself and the comparison of treatment means for response. As mentioned previously, the design does not allow a test of significance for blocks: an approximate F-test can be carried out if the assumption is made that most of the Treatments x Blocks x Generations variance is due to residual error (i.e. the interaction component itself is minimal). This gives F values for blocks of 16 and 41 for males and females respectively, both highly significant on 1 and 80 df. For both male and female responses, there was a significant treatment effect, ($P<0.001$ for males and $P<0.05$ for females).

Table 3.10 shows the ranking of mean responses (over blocks) for male and female bodyweight. Treatment A showed the greatest response for male bodyweight, Treatment D showed significantly less response than A, but significantly more than treatments B and C, which were themselves significantly different from the control. For female bodyweight, treatment A showed the greatest responses, then

treatments B, C, and D, which were significantly different from the control.

Table 3.8: Model for Analysis of Variance for Response to Selection

Design	Source	n	Random/Fixed
	Blocks	2	Random
	Treatments	5	Fixed
	Generations (within treatments)	21	Fixed

Source	df	Expected Mean Square
Constant	1	
Blocks	1	$\sigma^2_E + 2\sigma^2_B$
Treatments	4	$\sigma^2_E + 10\sigma^2_{T.B} + 5\sigma^2_T$
Treat. x Blocks	4	$\sigma^2_E + 10\sigma^2_{T.B}$
Generations	20	$\sigma^2_E + 42\sigma^2_{G.B} + 20\sigma^2_G$
Gens. x Blocks	20	$\sigma^2_E + 42\sigma^2_{G.B}$
Treats. x Gens.	80	$\sigma^2_E + 210\sigma^2_{T.G.B} + 42\sigma_{2T.G}$
Treats. x Blocks x Generations	80	$\sigma^2_E + 210\sigma^2_{T.G.B}$

NB: Tests of Significance

1. Treatments vs. Treatments x Blocks
2. Generations vs. Generations x Blocks
3. Treatments x Generations vs. Treatments x Blocks x Generations.

Table 3.9: Analysis of Variance for Response, Generations 0-21

Source	df	Male		Female	
		MS	F	MS	F
Constant	1	1.93511		12.46506	
Blocks	1	0.05735		0.09845	
Treatments	4	0.24951	24.51***	0.62927	9.72*
Bls. x Trt.	4	0.01018		0.06471	
Gen.	20	0.04486	30.94***	0.19937	16.77**
Bl. x Gens.	20	0.00145		0.01189	
Trts. x Gens.	80	0.00353	10.38***	0.01132	4.70***
Trts. x Bls.x Gens.	80	0.0034		0.00241	

Table 3.10: Comparison of Treatment Means for Response, Generations 0-21

Treatment	Male	Female
A	0.16923 a	0.38838 a
B	0.08628 c	0.26838 b
C	0.08888 c	0.23993 b
D	0.15907 b	0.28166 b
E	-0.02349 d	0.04981 c

NB: Means with same subscript not significantly different, $P<0.05$ (Duncan's Multiple Range Test)

3.3.3. Realised Heritabilities

Realised heritabilities were calculated as the regression of cumulative corrected response to selection on cumulative selection differential, within sexes (see Appendix C), and their standard errors according to Hill (1972). Table 3.11 shows these realised heritabilities, and Table 3.12 the treatment means for the two sexes. As with the selection responses, realised heritabilities were lower in Block 2 than Block 1. This suggests that genetic sampling differences existed between the two blocks in addition to any environmental differences.

Table 3.11: Realised Heritabilities and Standard Errors

Treatment	Block 1		Block 2	
	Male	Female	Male	Female
A	0.188 (0.020)	0.178 (0.027)	0.136 (0.017)	0.156 (0.026)
B	0.133 (0.019)	0.114 (0.028)	0.099 (0.017)	0.104 (0.028)
C	0.111 (0.019)	0.077 (0.029)	0.096 (0.018)	0.080 (0.028)
D	0.168 (0.022)	0.128 (0.028)	0.145 (0.019)	0.121 (0.028)

Table 3.12: Treatment Means for Realised Heritability

Treatment	Male	Female
A	0.162 a	0.167 a
B	0.116 a	0.109 bc
C	0.104 b	0.079 c
D	0.157 a	0.125 b

NB: Means with same subscript not significantly different $P<0.05$ (Duncan's Multiple Range Test).

As compared with the base population estimates, the realised heritabilities were considerably lower for males and slightly lower for females. Further, the realized heritabilities were lower in the subdivided populations than in Treatment A and for these treatments, were also lower in females than males. However, the ranking of the Treatments was the same in both sexes.

Figures 3.9-3.12 are plots of cumulative corrected response against cumulative selection differential. For male responses, they show quite a clear separation between Treatments A and D on the one hand, and Treatments B and C on the other. For Treatments A and D, the plots appear linear for most of their range, but there is again evidence for plateauing in Treatments B and C. The separation between Treatment A and the remaining three treatments is clear in the plots for female response against selection differential. As with response

vs generation number, there is not such a strong suggestion of plateauing in female response vs selection differential as in males.

3.3.4. Changes in Phenotypic Variance

Table 3.13 shows the overall mean population standard deviations for each treatment over generations 1 to 21. In both blocks and sexes, the amount of phenotypic variation was least in the unselected control, Treatment A the next lowest, and the three subdivided treatments had the highest levels of variation. The standard deviations for the subdivided treatments were approximately 20% greater than for the control in females, while for males, they were approx. 10% greater than the lowest value (with the exception of Treatment D, block 1, which will be discussed later).

Table 3.14 shows regression coefficients of population phenotypic standard deviation on generation number. These were calculated in order to see whether any of the selection treatments produced significant increases or decreases in phenotypic variance. The only treatments showing negative trends to any extent were A and E; however in neither case were the regression coefficients significantly different from zero.

Table 3.13: Mean Population Standard Deviation (mg)

Treatment	Block 1		Block 2	
	Male	Female	Male	Female
A	0.0879	0.1531	0.0774	0.1420
B	0.0835	0.1636	0.0830	0.1610
C	0.0891	0.1755	0.0867	0.1639
D	0.1010	0.1660	0.0871	0.1614
E	0.0825	0.1447	0.0761	0.1358

Table 3.14: Regression Coefficients of Population Standard Deviation vs Generation No.(mg/gen)

Treatment	Block 1			Block 2		
	Male	Female		Male	Female	
A	-0.00080	0.00180	*	-0.00045	0.00097	
B	0.00071	0.00240	*	0.00019	0.00139	
C	0.00006	0.00010		0.00019	0.00005	
D	0.00030	0.00310	***	0.00065	0.00318	**
E	-0.00046	-0.00130		-0.00096	0.00007	

* Regression Coefficient Signif. Different from zero $P<0.05$,

** $P<0.01$,

*** $P<0.001$

Significant positive regression coefficients were obtained for female standard deviation, Block 1, Treatments A and B ($P<0.05$), and for female standard deviation, Treatment D, both blocks ($P<0.001$ and $P<0.01$ in Blocks 1 and 2 respectively). Male standard deviation increased in Treatments B, C and D in both blocks, D showing the greatest change, but none of the regression coefficients were significantly different from zero. Figures 3.13-3.16 show the changes in phenotypic standard deviation with time in both blocks and sexes. There was considerable variation within all treatments, with large fluctuations between generations. There was also a suggestion of curvilinearity, with variances slightly higher at the beginning and end of the period of selection. Such a pattern is consistent with initial reductions in additive genetic variance as a result of selection (Falconer, 1981; Bulmer, 1971) and a later slight increase due to scale effects, changes in environmental variance as the proportion of homozygous loci increases, or an increase in genetic variance resulting from frequencies of the rare favourable alleles being moved from initially low, to intermediate levels by selection.

The regressions of phenotypic standard deviation against generation number

were tested for curvilinearity by comparing goodness-of-fit under the linear model with that achieved using a quadratic model. Quadratic models explained significantly more variation for:

- Treatment A, males, both blocks
- Treatment B, both sexes and blocks
- Treatment C, males, block 1
- Treatment D, all cases except block 2 males
- Treatment E, no cases.

These results provide some support for the suggestion of a reduction in phenotypic standard deviation after the early generations, with later increases for the reasons suggested.

3.3.5. Partitioning of Phenotypic Variance

The observed phenotypic variation may be partitioned into between and within bottle (deme) components:

$$\sigma^2_P = \frac{d}{d+1}\sigma^2_{PB} + \frac{nd}{nd+1}\sigma^2_{PW}$$

where σ^2_P = total phenotypic variance

σ^2_{PB} = phenotypic variance between bottles

σ^2_{PW} = phenotypic variance within bottles

d = no. of bottles/demes, and n = individuals scored per bottle/deme.

These observational components could be further partitioned into genetic and environmental components:

$$\sigma^2_{PB} = \sigma^2_{GB} + \sigma^2_{EB}$$

where σ^2_{GB} = Genetic Variance among bottles/Demes

σ^2_{EB} = Environmental Variance among Bottles.

and $\sigma^2_{PW} = \sigma^2_{GW} + \sigma^2_{EW}$

and σ^2_{GW} = Genetic Variance within Bottles/Demes

σ^2_{EW} = Environmental Variance within Bottles.

Because differing degrees of isolation of demes (bottles) were imposed in the four selection treatments, different proportions of between- and within-deme variance might be expected. By making a simple assumption, estimates of the proportion of between-deme variance that was genetic can be obtained. Genetic differentiation amongst subpopulations is essential for any form of group selection to be effective, and estimates of the degree of differentiation are essential for both prediction and interpretation.

The partitioning of phenotypic variance via analysis of variance procedures may be extended to provide information about genetic differentiation if the assumption is made that in Treatment A, where allocation of selected individuals to bottles was random in every generation, there were no significant genetic differences between bottles over several generations, and thus observed between-bottle variance comprised solely environmental variance. This is unlikely to hold for single generations, but over the entire course of selection, genetic differences in each generation should disappear.

Table 3.15 presents the components of phenotypic variance across generations, and the intra-class correlation, averaged over blocks, for each treatment in both blocks. The model fitted was:

$$y_{ijkl} = \mu + b_i + g_{ij} + d_{ijk} + e_{ijkl}$$

where y_{ijkl} = bodyweight of l^{th} individual in the k^{th} deme in generation j in block i

μ = mean bodyweight

b_i = effect of the i^{th} block

g_{ij} = effect of j^{th} generation in block i

d_{ijk} = k^{th} deme in the j^{th} generation in block i.

The results show that both between- and within-deme components of variance were slightly higher in the subdivided treatments. In males, the intra-class correlations were also higher in the subdivided treatments, but in females, there was less variation in r, with only Treatment C differing notably from the undivided Treatment A. These treatment differences were not significant for either variance components or for the intra-class correlation in either males or females. However, the pattern of increases in variance components was in agreement with expectations from theory. Subdivision to any degree into sub-lines will produce genetic differentiation amongst the sub-lines, due to the combined effects of sampling and inbreeding. This was observed in this experiment; for both males and females, the between-bottle variance increased with subdivision. These increases were much greater in proportion to Treatment A in males than in females. (This difference remains after expressing all variations as coefficients of variation, and so is not due to scale effects). This might be expected if similar levels of genetic differentiation were produced in males and females, but male bodyweight had a higher true heritability, as the base population parameters indicate. Possible reasons for the increases in within-deme variance were discussed in Section 3.3.1. As with between-deme variance, the increases in within-deme variance compared with Treatment A, were greater in males than females, and these differences remained after scale effects were removed. This may also be due to higher heritability of male than female bodyweight.

Table 3.15: Variance Components and-Intra-class Correlations, Across Generations

Treatment	Male			Female		
	σ^2_B	σ^2_W	r_I	σ^2_B	σ^2_W	r_I
A	0.00144	0.00643	0.15937 (0.023)	0.00572	0.02038	0.20705 (0.026)
B	0.00323	0.01181	0.19413 (0.025)	0.00766	0.02635	0.19180 (0.025)
C	0.00351	0.00977	0.23142 (0.027)	0.00859	0.02732	0.25530 (0.025)
D	0.00273	0.01136	0.20106 (0.024)	0.00679	0.02595	0.20268 (0.026)

The partitioning of phenotypic variance gives a simple description of the population subdivision in the experimental treatments. Using the assumptions previously mentioned, this description can be extended to include genetic differences between bottles/demes. This provides a parameter describing between-deme variation in a manner analogous to the description of between individual effects provided by the individual heritability. By using the between-bottle variance for treatment A as an estimate of between bottle environmental variance, the between-deme heritability is given by:

$$h^2_{DEME} = (\sigma^2_{Pi} - \sigma^2_{PA})/\sigma^2_{Pi}$$

where h^2_{DEME} = Between-Deme heritability for treatment i

σ^2_{Pi} = Between-Deme Phenotypic Variance component for treatment i

σ^2_{PA} = Between-Deme Phenotypic Variance component for treatment A.

This estimator relies on the assumptions that a) all permanent differences

between bottles in treatment A are environmental and any genetic differences are random and have an insignificant effect.

b) the magnitude of environmental differences between bottles is similar in all treatments. This is perhaps questionable, since where significant inbreeding levels are reached in the demes of the subdivided treatments, there may be fitness-related density differences. If so, the estimates obtained will exaggerate the degree of genetic differentiation.

Between-deme heritabilities estimated as described above, are presented in Table 3.16. For both males and females, the greatest amount of variance between demes was generated in Treatment C, with Treatment D generating the smallest amount of between-deme variance across generations.

The estimated between-deme heritabilities, within sexes, were fairly similar, particularly in males. Interestingly, the estimated values were considerably higher than the realised heritabilities for individual selection, and very similar to the base population estimates.

Table 3.16: Estimates of Between-Deme Genetic Variance and Between-Deme Heritability, Subdivided Treatments

Treatment	Male			Female		
	σ^2_{BG}	σ^2_{BP}	h^2_D	σ^2_{BG}	σ^2_{BP}	h^2_D
B	0.00179	0.00323	0.554	0.00194	0.00766	0.253
C	0.00207	0.00351	0.590	0.00287	0.00859	0.334
D	0.00129	0.00273	0.473	0.00107	0.00679	0.157

Two other measures of subpopulation differentiation may be estimated for this data set. Jackson and James (1970) proposed the use of:

$$\emptyset n = \sigma_{GB} / \sigma_{PW}$$

where σ_{BG} = genetic between-stud standard

deviation

σ_{PW} = phenotypic within-stud standard deviation

in determining whether to select from one or many sheep studs. Using the same assumptions as before to estimate σ_{GB}, this statistic was calculated; (Table 3.17,). This measures the between-deme variation relative to the phenotypic variation within demes, while ϕ itself expresses the ratio of between/within genetic standard deviation. This has also been calculated, using the base population estimates for male and female heritability. For bodyweight ϕh was higher for males than female, and in both sexes was highest for Treatment C, then treatment B, with Treatment D showing the lowest degree of differentiation amongst the subdivided treatments. ϕ itself showed the same ranking amongst treatments, but its value for males and females was very similar.

Table 3.17: James' ϕh, Subdivided Treatments, Across Generations

Treatment	Male		Female	
	ϕh	ϕ	ϕh	ϕ
B	0.389	0.550	0.271	0.542
C	0.460	0.651	0.324	0.648
D	0.337	0.476	0.203	0.406

Slatkin (1981) introduced the concept of populational heritability, and showed that it could be estimated in two ways: firstly, by the fraction of total variance that is between-lines as discussed above, and secondly, by the correlation between means of individual subpopulations and those derived from them. The square of the latter is the coefficient of determination (Sokal and Rohlf,1981). Both this correlation and its square were calculated for all treatments. However the square is only presented for the comparisons within

Treatment B, as all the estimates of r had a similar sampling error, and taking squares biassed upwards the r^2 values especially for Treatment A, which had a mean r value close to zero. The results are presented in Tables 3.18 and 3.19. Table 3.18 shows the values of the correlation and its square for treatment B, in those generations used to select amongst sub-lines in this treatment.

There was no clear tendency for the coefficient of determination to either increase or decrease between the first and second generation of each pair of means. It might be expected that the correlation between deme means in successive generations would increase as the demes became more inbred (as the genetic variation within each deme is reduced, the variance due to genetic sampling will be reduced). Obviously, the degree of homozygosity within demes did not reach sufficiently high levels for the correlation to reach significant levels in this case.

Table 3.19 shows the average correlation coefficients for each treatment. The overall pattern amongst treatments for both statistics was similar to that seen with other methods of describing the phenotypic variance: the subdivided treatments were more differentiated than treatment A, and of the subdivided treatments, treatment C exhibited the greatest degree of between-deme differentiation.

Table 3.18: Coefficients of Determination, Successive Generations, Treatment B

Generations	Block 1		Block 2	
	Male	Female	Male	Female
2-3	0.377	0.084	0.139	0.138
3-4	0.025	0.091	0.421	0.130
7-8	0.410	0.012	0.102	0.358
8-9	0.033	0.000	0.010	0.236
12-13	0.417	0.041	0.019	0.004
13-14	0.128	0.152	0.336	0.238
$\bar{r}^2$	0.232	0.063	0.172	0.184

Table 3.19: Between-Generation Correlations of Deme Means, Averaged Across Generations (Standard Error)

Treatment	Block 1	Block 2
A	0.0797 (0.309)	-0.0009 (0.318)
B	0.1737 (0.397)*	0.2257 (0.504)**
C	0.3031 (0.341)***	0.3939 (0.324) ***
D	0.2332 (0.461)**	0.2371 (0.388)**

* P<0.05, ** P<0.01, *** P<0.001

3.4. Further Selection

As was previously mentioned, results were reviewed at the end of 20 generations of selection, and it was decided at that point to continue the experiment in Treatments A, D and the control. The reasons for this were two-fold; firstly, to see if genetic variance for bodyweight remained in the mass-selected treatments, secondly, and more importantly, to follow further an extreme variant which arose in Treatment D, block 1 at G_{13}. The variant was first noticed in deme 4 of that treatment and thereafter spread throughout the remaining demes. Demes containing this presumed genetic effect at moderate to high frequency showed a characteristic phenotypic distribution in both males and females: a sharp peak slightly below the overall mean, and a series of fairly well-defined peaks to the heavy side of the distribution. As will be discussed later, this pattern was maintained even at what were presumably quite high frequencies of the gene(s).

The experimental procedures followed in this stage of selection were identical to those of the first stage. Thus the comparison between Treatments A, D and the control was continued. The selection experiment stopped altogether at G_{34}, or after 33 generations of selection.

At G_{27}, two new lines were initiated from Treatment A. Responses appeared to have ceased by that stage, so in order to examine the genetic architecture of that treatment at that stage, a relaxed selection line and a line subdivided, and including within-and between-deme selection (i.e. using the model of Treatment D), were established from Treatment A. This comparison, between an apparently plateaued line, and relaxed and subdivided lines derived from it, was essentiatlly the same as that reported by Falconer (1977), although the mode of subdivision with selection was different.

3.4.1. Selection Differentials

Table 3.20 shows the selection differentials applied in the period G_{21}-G_{34}. There were no significant differences in the amounts of selection applied, between treatments A and D in block 2, over this period. In block 1, however, selection differentials were almost twice as great in Treatment D as in Treatment A. This reflects the effect on phenotypic variances of the gene(s) mentioned previously: distributions in both sexes in this treatment became highly skewed to the heavy end of the distribution. As in the initial period of selection, selection differentials for Treatment A block 2 were only c. 90% of those attained in block 1,treatment A.

Table 3.20: Cumulative Selection Differentials, G_{21}-G_{34} (mg)

Treatment	Block 1		Block 2	
	Male	Female	Male	Female
A	1.5785	2.8638	1.4223	2.6011
D	3.5191	5.2742	1.4732	2.5210

3.4.2. Responses to Selection

Tables 3.21 and 3.22 present the absolute and corrected responses to selection for treatments A, D and control over the period G_{21}-G_{34}. There was a slight upward trend in the unselected control in block 1, and a downward trend in unselected female bodyweight, block 2. Comparison with Table 3.7 shows that there was as much increase in unselected bodyweight in the block 1 control as had occurred in the previous 21 generations, while in block 2 the increase over the first 20 generations in unselected female weight was matched by the decrease over the next 13 generations.

Table 3.21: Absolute Responses to Selection, G_{21}-G_{34} (mg)

Treatment	Block 1		Block 2	
	Male	Female	Male	Female
A	0.2229	0.3341	0.1008	0.2881
D	0.4216	0.5558	0.1437	0.3050
E	0.0526	0.1280	-0.0100	-0.1004

Responses to selection continued over this period, in both blocks and both treatments. In block 2, responses were slightly greater for Treatment D than treatment A, while in block 1, responses in both males and females in treatment D, were almost double those observed in Treatment A. Whereas responses over this period for Treatment A were considerably less than in the first 20 generations (30-60%), as much response was obtained in Treatment D, block 1, over the period G_{21}-G_{34}, as in the first 20 generations. This again reflects the effects of the gene(s) mentioned previously.

Table 3.22: Corrected Responses to Selection, G_{21}-G_{34} (mg)

Treatment	Block 1		Block 2	
	Male	Female	Male	Female
A	0.1703	0.2061	0.1108	0.3885
D	0.3690	0.4278	0.1537	0.4054

The pattern of responses over this period is shown in Figures 3.17-3.24. Those of absolute responses include treatments A_1 and A_2; A_1 a subdivided and A_2 a relaxed treatment. From Figure 3.17 it can be seen that Treatment D, block 1, surpassed Treatment A in male bodyweight after G_{23}, and was always heavier than A after that point. Female bodyweight in treatment D, block 1, fluctuated quite markedly over this period but never surpassed Treatment A. In block 2, treatment D was superior to A in males throughout the period, but female bodyweights were slightly lighter than in A. This pattern is still apparent in the responses corrected for changes in the control and for differences in original weight, although the differences between male bodyweight for treatments A and D in block 2 become very small. The most important feature of responses over this period was treatment D becoming heavier than A for male bodyweight, in block 1, at generation 23.

3.4.3. Realised Heritabilities

Realised heritabilities for Treatment A and D over the period G_{21}-G_{34} are presented in Table 3.23. In block 2, there was little difference between A and D for either males or females; selection was slightly more effective in Treatment D than A. In block 1 however, although much more response was obtained in Treatment D, proportionately more selection was applied, so that realised heritabilities were significantly lower than in treatment A.

Table 3.23: Realised Heritabilities, G_{21}-G_{34} (Standard Errors)

Treatment	Block 1		Block 2	
	Male	Female	Male	Female
A	0.126 (0.002)	0.107 (0.004)	0.112 (0.002)	0.124 (0.003)
D	0.082 (0.004)	0.047 (0.006)	0.120 (0.002)	0.142 (0.004)

For both treatments the realised heritabilities over this period were lower than in the first phase of selection, with the reduction being more marked in block 1. All selection differentials, responses and realised heritabilities were higher in block 1 over the first 20 generations of selection. This suggests that utilisation of genetic variance was initially more complete in all treatments of block 1, but that in the later period of selection, utilisation proceeded at similar rates in both blocks.

The marked reduction in female realised heritability in Treatment D, block 1 (from 0.128 to 0.047), with no concomitant reduction in the same treatment, block 2, suggests that the type of genetic change occurring in this treatment was markedly different in the two blocks. The pattern of responses over this period in the two blocks was quite different for treatment D, providing further suggestion of different types of genetic change occurring.

3.4.4. Relaxed and Subdivided Lines: Selection Differentials

Table 3.24 shows the selection differentials applied over the period G_{27}-G_{32}, in the two initial experimental treatments, and the subdivided treatment, A_1, set up from treatment A at G_{27}. The methods of subdivision and of within- and between-deme selection in A1 were identical to those of treatment D. The amounts of selection applied in this treatment were similar to treatment A over this period.

Table 3.24: Cumulative Selection Differentials, G_{27}-G_{32}

Treatment	Block 1		Block 2	
	Male	Female	Male	Female
A	0.6140	1.0785	0.5462	1.1546
A_1	0.6225	1.0959	0.4973	1.0579
D	1.3166	1.9331	0.5609	1.1387

3.4.5. Relaxed and Subdivided Lines: Responses

Tables 3.25 and 3.26 show absolute and corrected responses over the period G_{27}-G_{32}, with their standard errors. Treatment A_2 was a relaxed line founded as a sample of from Treatment A: procedures for A_2 were identical to those of E, the control treatment begun at G_0.

Table 3.25: Absolute Responses to Selection, G_{27}-G_{32} (mg)

Treatment	Block 1		Block 2	
	Male	Female	Male	Female
A	0.0593	0.1242	0.0461	0.0017
A_1	0.0762	0.0320	0.0190	-0.0615
A_2	-0.0522	0.0648	-0.0358	0.1230
D	0.0570	0.1300	0.0401	0.0159
E	-0.0564	-0.0630	-0.0178	-0.1588

Absolute changes over this period suggest a negative environmental trend, since both male and female bodyweight declined in treatments A_2 and E. Female bodyweight in block 2, A_2, was a marked exception to this general observation; increasing as much over this period, without any selection, as either of selected treatments. This suggests that drift or genotype-by-environment interaction were significant over this period.

Table 3.26: Corrected Responses, G_{27}-G_{32} (mg)

Treatment	Block 1		Block 2	
	Male	Female	Male	Female
A	0.1157	0.1872	0.0639	0.1605
A_1	0.1326	0.0950	0.0368	0.0973
A_2	0.0042	-0.0018	-0.0180	0.2818
D	0.1134	0.1930	0.0579	0.1747

Treatments A, A_1, and D all showed increases over this period. Overall, responses in Treatments A and D were similar in both blocks, with those of Treatment A_1 being approximately half as much. Male response in block 1 was an exception to this; response in A_1 was slightly greater than in A or D.

With the exception of female bodyweight in block 2, there were no significant changes in bodyweight in treatment A_2, the relaxed line. This suggests that, over this limited period, and allowing for the observed drift, gene frequencies in Treatment A were stable or nearly so at the time of sampling. Had natural selection been limiting responses at this point, a downward trend in bodyweight would have been expected (and at a greater rate than for the unselected control line, E). The fact that selection was still effective in treatment A at this point suggests that residual genetic variation was present, but there was little or no "homeostatic selection" returning bodyweight to lower levels. This observation is supported by the observation that bodyweight did not change in Treatment A for a large number of generations after G_{34}, when it was maintained without selection, at a population size of 50 pairs. This suggests that additive variation for bodyweight remained in this population, due to genes at intermediate frequencies with no natural selection acting upon them.

3.4.6. Relaxed and Subdivided Lines: Realised Heritabilities

Table 3.27 lists realised heritabilities, with their standard errors, for the period G_{27}-G_{32}. The comparison amongst treatments A, A_1 and D for realized heritability is similar to that for corrected response. With the exception of male bodyweight in block 1, the effectiveness of the selection applied in treatment A_1 was lower than in A or D. Thus, in contrast with the results of Falconer (1971), subdivision with selection did not produce extra response as compared with a mass selected line in this study. As will be more fully discussed later, this comparison is not direct, since the traits involved, the time-scale, and the mode of subdivision with selection were all different. The immediate conclusion from this study is that subdivision with selection did not quickly uncover and utilise non-additive variation, if any were present, in a line in which responses to mass selection were slowing.

Table 3.27: Realised Heritabilities, G_{27}-G_{32}

Treatment	Block 1		Block 2	
	Male	Female	Male	Female
A	0.151 (0.003)	0.046 (0.004)	0.131 (0.003)	0.040 (0.004)
A_1	0.193 (0.003)	0.040 (0.004)	0.097 (0.003)	0.027 (0.004)
D	0.023 (0.005)	-0.040 (0.007)	0.140 (0.003)	0.097 (0.004)

3.5.1. Generations 0 to 34: Selection Differentials

The remaining comparison within the selection experiment is that between treatments A and D over the entire 33 generations of selection. Total amounts of selection applied over this period are presented in Table 3.28. In block 2, the amounts of selection applied to the two treatments were very similar over the period, slightly more selection being applied in Treatment D (approximately 4% more). Slightly more selection was applied in block 1 than block 2 for treatment A, this again must be attributed to the effects of sampling.

Table 3.28: Cumulative Selection Differentials, G_0-G_{34} (mg)

Treatment	Block 1		Block 2	
	Male	Female	Male	Female
A	3.6500	6.4202	3.2736	6.1575
D	5.6434	9.1164	3.4535	6.3652

3.5.2. Generations 0 to 34: Responses to Selection

Absolute reponses to selection over the entire period of selection are presented in Table 3.29, and Figures 3.25-3.28. There was a significant increase in unselected bodyweight, male and female, in block 1, but no significant change in either in block 2. The corrected responses (Table 3.30, and Figures 3.29-3.32) present a conflicting picture. In block 2, treatment D achieved slightly more response in males, but less response in females. In block 1 there was little difference between A and D for female bodyweight, but more response in male bodyweight in treatment D.

Table 3.29: Absolute Responses, G_0-G_{34} (mg)

Treatment	Block 1		Block 2	
	Male	Female	Male	Female
A	0.5789	1.1263	0.3723	0.9946
D	0.7801	1.1326	0.4010	0.7567
E	0.0730	0.2436	-0.0293	-0.0171

When the plots of long-term response (Figures 3.29-3.32) are examined, it is clear that extra response in Treatment D, block 1, males, really only became apparent after about generation 23. While female response in the same treatment was similar to that of Treatment A after this point, it fluctuated quite violently in the later period of selection. Both these observations will be

discussed further in the light of the postulated major gene(s).

Table 3.30: Corrected Responses, G_0-G_{34} (mg)

Treatment	Block 1		Block 2	
	Male	Female	Male	Female
A	0.5059	0.8827	0.4016	1.0117
D	0.7071	0.8890	0.4303	0.7738

3.5.3. Generations 0 to 34: Realised Heritabilities

Realised heritabilities for treatments A and D over the period G_0 to G_{34}, are presented in Table 3.31. In all cases except males, block 2, selection was more accurate in treatment A than treatment D. The estimates for both males and females for treatment A were quite consistent, from 11.2% to 13.7%. In treatment D, female realised heritability was slightly lower than that of males in both blocks.

Table 3.31: Realised Heritabilities, G_0-G_{34} (Standard Errors)

Treatment	Block 1		Block 2	
	Male	Female	Male	Female
A	0.124 (0.002)	0.127 (0.003)	0.112 (0.002)	0.137 (0.003)
D	0.106 (0.003)	0.073 (0.004)	0.120 (0.002)	0.108 (0.003)

Two features of these results (and those of the four treatments of the initial comparison) when compared with the base population parameters are:

- firstly, that both male and female realised heritabilities are considerably lower than the base population estimates, and

- secondly, the difference between males and females in the base population estimates of heritability is not reflected in the realized estimates:

over both the first 20 and entire 33 generations of selection, the male estimates are only slightly higher than those for females.

3.6. Discussion

The primary objective of this experiment was to compare responses to artificial selection in a number of population structures, two of which (B and C) had not previously been compared in the one study, and a third (D) which contained new features. Discussion of the results will therefore begin with comparisons within the scope of this study, followed by examination of their wider relevance, and will initially deal with the first 20 generations of selection.

Amongst the treatments previously studied (A, B, and C), there were only small differences in the amounts of selection applied. It appeared that extra between-deme variance generated by sub-lining was not utilised effectively by the procedures of Treatment B. This probably reflects low intensity of selection between lines at each crossing - the proportion selected was 5 out of 10 - and different results would be expected if selection were more intense. No inter-deme selection was applied in Treatment C, so it is not surprising that the selection applied was similar to that of Treatment A. In both blocks, selection differentials in both sexes were highest for treatment D. This reflects imposition of both within- and between-deme selection every generation. These conclusions are supported by the partitioning of selection differentials, showing that close to 10% of the total selection differential applied to Treatment D was due to selection between demes. In none of the studies reported previously was there any mention of the amount of selection applied between demes. Obviously, the level will be related to the intensity of selection among sub-lines (in this study, 5 out of 10). Madalena and Hill (1972) varied the intensity of selection among sub-lines, but did not report actual amounts of

selection. The observed selection differentials in this study suggest that any genetic differences between sub-lines are utilised to a greater extent by the model used in Treatment D than the sub-lining/crossing model.

The only other point to observe about the realised selection differentials is that they were lower than expected on the basis of the intensity of selection and average phenotypic parameters for the experimental populations. Realised selection differentials are commonly smaller than expected (Falconer, 1981), but it is of interest that there were differences between treatments in the ratio of realised to expected selection differentials in this study. This appears to be due to differences in deviations from normality in the units (bottles/demes) of each treatment within which selection was applied. The ratio was highest for treatment A, in which deviations from normality were smallest, and lowest for treatments B and C, where deviations were highest. Treatment D achieved a proportion of expected selection differential midway between those of treatments B and C on the one hand, and that of treatment A on the other, and on average, distributions in individual demes for this treatment were more normal than those of B and C, but less so than in treatment A.

Katz and Young (1975) reported significantly lower selection differentials for their treatment B1 (analogous to the present treatment C) as compared with the mass selected line. No such reduction was observed in this study. The only difference between methods of the two studies was in number of sub-populations (and therefore total population size): Katz and Young used 6 demes each comprising 5 pairs of parents, as compared with 10 demes each of 5 pairs of parents in this study. The smaller effective population size may have resulted in a reduction in phenotypic variance; unfortunately no data were presented on these parameters.

The observed selection differentials lead to the conclusion that population subdivision had little effect on the amount of selection applied. Depending on

the amount and method of selection between population sub-units, the total selection differential could be apportioned differently. Also, it appears that where sub-units of population are small, base population parameters may overestimate realised selection differentials unless corrections are made for observed deviations from normality.

Given that total amounts of selection applied were similar in all treatments, but that the composition in terms of within- and between-deme selection varied, comparison of responses may throw some light on the relative effectiveness of these two modes of selection.

The responses obtained provide no evidence for any advantage in subdivision for either males or females. Treatment D produced very similar responses in males to those obtained in Treatment A, but when taken over blocks, response in Treatment D was significantly less than in Treatment A. Treatment D showed significantly greater responses than either B or C. Similarly for female bodyweight, responses were greatest in Treatment A, with no significant differences among the remaining three treatments.

These results agree with previous reports, with the exception of Katz and Young (1975). They provide no evidence for any advantage in subdivision for traits estimated to have reasonable levels of additive genetic variation. There is no support from these results for the findings of Katz and Young, that subdivision with regular, random but cyclic migration between demes produced greater responses than simple mass selection. Considering that base population estimates of genetic parameters, phenotypic means and variances were all similar (although variances in the selection lines appeared smaller in Katz and Young's report), this difference is not easily explained. The stock used in their study was a four-way cross that had been maintained at population size of 40 pairs for 25 generations. That used in this study had only been in the laboratory for a short time and was derived from a sample of approximately 50 wild-caught females

(see Section 2.1). Apart from the estimate of heritability in the base population, no further information was provided on the genetic make-up of the stock used by Katz and Young. The explanations proposed in their paper for the superiority of both subdivided treatments, namely the existence and fixation (in the subdivided populations) of favourable epistatic complexes, and the increase in total genetic variance in the subdivided treatments due to inbreeding within demes, do not help explain the discrepancy between the two sets of results. Of the two, the fixation of epistatic systems in their study, and absence of, or failure to fix, such systems, in this, seems more reasonable, but unfortunately cannot be tested in any way. The apparent lack of any replication in their study reduces the usefulness and explanation of their result. In the apparent absence of major non-additive variance (but noting the results of Treatment D, block 1), the results obtained in the present study suggest that the Katz and Young model for population subdivision has little to offer.

The responses obtained in Treatment B, using a regime of sub-lining/ crossing similar to that recommended by Wright and Lush, and previously investigated in several studies, are in agreement with the general conclusion that such sub-lining and crossing is unlikely to be of any advantage where appreciable levels of additive genetic variance are available. However, greater short-term responses may have been achieved had selection among sub-lines been more intense, but the expectation is that the limit to response would have been reduced. As it was, response in Treatment B in this study appeared to be slowing after c. 15 generations of selection, so the limit may have been reduced by even the mild between-line selection applied.

Responses in Treatment D, at least for male bodyweight, were closer to Treatment A, than treatments B and C. The simplest explanation for this is that, at least for males, the level of migration was sufficient to overcome any effects of subdivision on genetic variance, i.e. to ensure that the system of

demes could be treated as a single population. (This agrees with the theoretical expectations, that on average one migrant every other generation is sufficient to preserve homogeneity of the population (Wright, 1977; and earlier)). In practical terms, this supports findings in livestock species which suggest that genetic differences between herds within livestock breeds are insignificant (Robertson and Asker, 1951; Short and Carter, 1956). The results for female bodyweight, where all the subdivided populations showed similar responses, and all were significantly lower than Treatment A, suggest that inbreeding may have contributed to a decline in response. If, as the base population estimates suggest, some of the total variance in female bodyweight was due to genes showing overdominance, inbreeding would reduce selection response when compared to less inbred populations. (There was also a significant proportion of total variance that was non-additive or common environmental for males, but there the level of additive variance was twice that of females, and would presumably support responses to selection for longer).

The similarities among selection differentials mean that the patterns observed for response carry-over with little alteration to the realized heritabilities. Selection was most efficient in Treatment A, and least efficient in Treatment C. For male bodyweight, selection in Treatment D was almost as effective as that in treatment A, but significantly less efficient in females.

The realized heritabilities for all treatments were very similar for males and females. This contrasts with both the base population estimates (where that of males was twice that of females), and the results of Katz and Young, where male realized heritability estimates were approximately half those for females. This suggests that the weaknesses discussed concerning the base population estimations were valid, and that linkage disequilibrium, effects of sampling, of selection, and inbreeding depression during the course of selection, all contributed to the realized heritability being lower than the estimated. In

particular the close agreement between male and female realized heritabilities suggests that the design used to estimate the base population parameters may have underestimated the additive component of total variance for female bodyweight, or seriously overestimated that component in males. Other reports in the literature suggest realized heritabilities for adult bodyweight of D. melanogaster of approximately 20% (Martin and Bell, 1960; Frahm and Kojima, 1966), which agree quite well with the result for Treatment A.

There were slight, but generally non-significant increases in population standard deviation over the period of selection in Treatments B and C. The control, and males in Treatment A, showed small non-significant decreases in phenotypic variance, whilst phenotypic variance in female bodyweight in Treatment A increased slightly. Larger increases in both male and female phenotypic variance were observed in Treatment D in both blocks, the increases in females being significant. In block 1, increases in variance in Treatment D may be attributed to the appearance and spread of the major gene(s), but the increases seen in block 2 for this treatment suggest that more between-deme variance may be generated and maintained in this population structure than any of the others. Over the period of selection of this study, this extra variation did not appear to be readily available to selection, at least as applied here.

Several methods of describing the partitioning of total phenotypic variance among- and within-demes (cf. bottles, sub-lines) were used. In general, they produce similar comparisons among treatments. The ranking of treatments in terms of proportion of total phenotypic variance that is among demes/sub-lines, from highest to lowest, was C>B>D>A. Such descriptions of the partitioning of phenotypic variance in experimental populations are rare, but an understanding of this partitioning, and how it relates to underlying genetic differentiation, is essential for studies involving any form of selection amongst sub-units of population. Slatkin (1981) developed formulae for estimation of the

"populational heritability", and James (1966) described methods for selection from one or several populations. Both studies used solely additive genetic models, and neither included any environmental variance between demes/sub-units.

James (1966) and Jackson and James (1970) discussed use of the criterion:

$$r\,\phi\,h > \frac{h^2 \log N}{1.25 + \log N}$$

N = number of populations sampled

h^2 = within-population heritability

ϕ = ratio of between- to within -population genetic standard deviations

and r = correlation between the assessment of population mean breeding value and the true value,

to determine whether to select from one or several populations in choosing foundation stock for breeding programmes. Using $h^2 = 0.2$, and the estimates of ϕh in Table 3.17, the criterion was met for all three subdivided treatments provided r is greater than from 0.2 (for largest values of ϕh) to 0.4 (for smallest ϕh). Estimates of r^2 (= Between-Deme Heritability) were presented in Table 3.16, and as shown in Table 3.32, in only one case (female, Treatment D) was the observed r value smaller than that required to meet the criterion. This suggests that the three models of population subdivision all produced substantial between-deme variation, and were the aim to establish a new breeding population, selection amongst the subpopulations would at least improve short-

term response. This is of no direct significance in practice, except to the extent that Treatment D is a model of livestock populations: if it is a valid model (i.e. the amount of genetic differentiation is of the same order as for quantitative traits in livestock), then some selection amongst populations may be beneficial. This agrees with the conclusions of Jackson and James, regarding short-term responses to selection.

Table 3.32: Observed r Values, and r Values Required for Selection among Sub-populations

Treatment	Male			Female		
	Øh	Req'd r	Obs. r	Øh	Req'd r	Obs. r
B	0.389	0.229	0.74	0.271	0.328	0.50
C	0.460	0.193	0.77	0.324	0.274	0.58
D	0.337	0.264	0.69	0.203	0.438	0.40

The agreement between methods of describing the partitioning of phenotypic variance was better in terms of ranking than of the actual values. The coefficients of determination were generally lower than the corresponding intra-class correlations and Øh values. This may reflect the fact that the coefficients of determination were based on a very small (n=10) number of pairs of means at each generation. Differences between treatments were more obvious in r than r^2, and for all treatments there were many instances of negative correlations between deme means in successive generations.

Treatment C showed the highest level of genetic differentiation between-demes; paradoxically, conditions were best for inter-deme selection in this structure, yet the absence of any inter-deme selection (and resultant increased effective gene flow) was a major contributing factor in producing these conditions. The observation of significant genetic differentiation in this treatment, in which there was persistent migration between demes, agrees with

the findings of Wade (1982), who observed significant levels of genetic differentiation among demes with even quite high levels of gene flow (up to 25% every generation).

This investigation of genetic differentiation in the four selection treatments together with the results obtained from selection, suggests that methods of combining between- and within-deme selection must involve compromises between the forces that increase genetic differentiation and thus provide suitable conditions for successful between-deme selection, and those that favour response to within-deme selection, viz. maintenance of appreciable genetic variation within demes by regular gene flow. It does not appear that models of subdivision previously employed (i.e. B and C) consistently achieve this compromise. In particular, treatment B models, at least for predominantly additive genetic action, do not offer evidence of any benefit. Reasonable amounts of between-deme variation were generated, but the method of selection employed did not utilise this efficiently. The results of Madalena and Hill (1972) suggest that more efficient use of this variation results in, at best, short-term gains only. Treatment C generated the largest amounts of between-deme variation, but this was not utilised in this model except indirectly via random gene flow. For this model to be more effective than mass selection would seem to require the existence of gene complexes having large selective advantage, that could be spread among demes by the random migration.

Treatment D represents an attempt to reach a compromise between the requirements for successful within- and between-deme selection. Of the three subdivided treatments, it was closest to Treatment A in both responses to selection and measures of genetic differentiation. There is no clear evidence from this experiment, however, that the model employed in Treatment D did any more than reduce the effects of subdivision (via inbreeding) sufficiently to allow responses to be almost as great as those in Treatment A, in the absence of

subdivision.

It is in the light of the overall pattern of results over the first 20 generations of selection that the results in Treatment D, block 1, are of special interest. Although not apparent in the overall responses until later generations, the effect appeared in that treatment at generation 13, and resulted in immediate increases in phenotypic variance. The behaviour of this effect suggested the possibility of its involving a gene combination and its spread throughout the set of demes in Treatment D (which will be fully discussed in Chapter 4) was in accord with the diffusion patterns suggested by Wright and others for favourable epistatic complexes. The appearance and spread of the effect encouraged continuing the selection; this enabled further observation of the behaviour of the effect, and also some investigation of the genetic architecture of treatment A after more than 25 generations of selection.

In both blocks, responses over the later phase of selection were greater in Treatment D than treatment A. Whilst in block 1 this can be attributed to the large effect segregating, no such cause was apparent in block 2, and therefore it appears that more genetic variation remained available in treatment D than treatment A in this block. The difference between treatments in response was greater for male bodyweight, although when selection differentials were accounted for, the differences between A and D were reduced. In block 2 it appears that treatments A and D were utilising existing genetic variation with similar efficiencies. Segregation of the large effect, presumably at moderate to high frequencies, in Treatment D, block 1, led to both selection differentials and responses during the second phase of the experiment being much greater in treatment D than A. When expressed as realised heritability however, the efficiency of selection was much lower in treatment D. Whilst not conclusive, this suggests that the genetic mechanisms involved in this effect were something other than the simple additive model.

The treatments initiated at G_{27} were designed to investigate the type and extent of genetic variation remaining in the mass selected populations after a long period of selection (viz. 26 generations). Changes upon relaxation were only very small (line A_2) over 5 generations. This suggests that natural selection was not inhibiting further responses at this stage, at least in the mass-selected lines. Although reversed selection was not carried out, the observation of continued responses over the later period of selection, albeit at a slower rate, is evidence of additive genetic variance available. No extra response was obtained over the period G_{27}-G_{32} by subdividing using the Treatment D model. Given the continuing response in Treatment A, this suggests that if any non-additive variation existed in the A populations at this stage, or if there were deleterious recessives segregating, there was sufficient additive variation to mask any such effects. Realised heritabilities in treatment A_1 were mostly slightly lower than those of treatment A. This could be explained by a reduction in additive variance within demes in A_1 reducing the responses to selection.

From the results over the period G_{27}-G_{32} it appears that selection responses could have been maintained for some time longer in this population. Robertson (1960) showed that with additive genes, the theoretical maximum response that can be expected is given by:

$$R_{max} = 2N_e \; i \; h^2 \; \sigma_p$$

If this is evaluated for males and females of this population using the base population estimates, the respective maxima are:

R_{max}, Male = 1.345 mg

R_{max}, Female = 1.513 mg.

These are more than double the observed total corrected responses over 33 generations of selection. If the value of Ne is reduced from the maximum possible in line with reports in the literature (see Falconer, 1981), to c. 70% of census value, and realised heritability is used instead of the base

population estimate, these values for maximum response become:

Rmax, Male = 0.45 mg Rmax, Female = 0.95 mg.

Excluding the values for Treatment D, block 1, where the assumption of additivity is dubious, the responses over 33 generations of selection were similar to these adjusted estimates of the limit. Obviously, the value used for heritability in this expression affects the limit profoundly. In this case the base population estimate of heritability in males was much larger than the effective value over the period of selection, and therefore, the expected male limit is far higher than that achieved.

The simplest explanation of responses slowing over the later period of selection is that the genetic variance had been reduced. From the limited investigation of the situation in the mass selection line at this stage, it does not appear reasonable to postulate any strong natural selection inimical to increases in bodyweight, or overdominance/dominance to any significant extent at that stage.

The immediate conclusion from this selection experiment is that subdividing populations for increased selection response when moderate amounts of additive variance remain available, is not recommended. At best, a procedure aimed at combining individual and deme merit (treatment D), will under such circumstances, reduce the negative effects of subdivision arising from inbreeding and drift. In many instances, this may be the principal effect of such gene flow as does occur between sub-populations of livestock and in the broader sense of maintaining additive genetic variation for many characters, in populations of wild species.

The significance of the Shifting Balance Theory, and studies derived from it, lies however in the capacity for detection and spread of favourable epistatic systems. In this light, the results discussed to this point appear to be of little direct consequence, and the results for Treatment D, block 1,

assume importance. The following chapter describes these results in greater detail, and the further efforts made to understand the effect underlying those results.

FIG. 3.1: ABSOLUTE RESPONSE VS. GENER. NO.,MALE,BLOCK 1

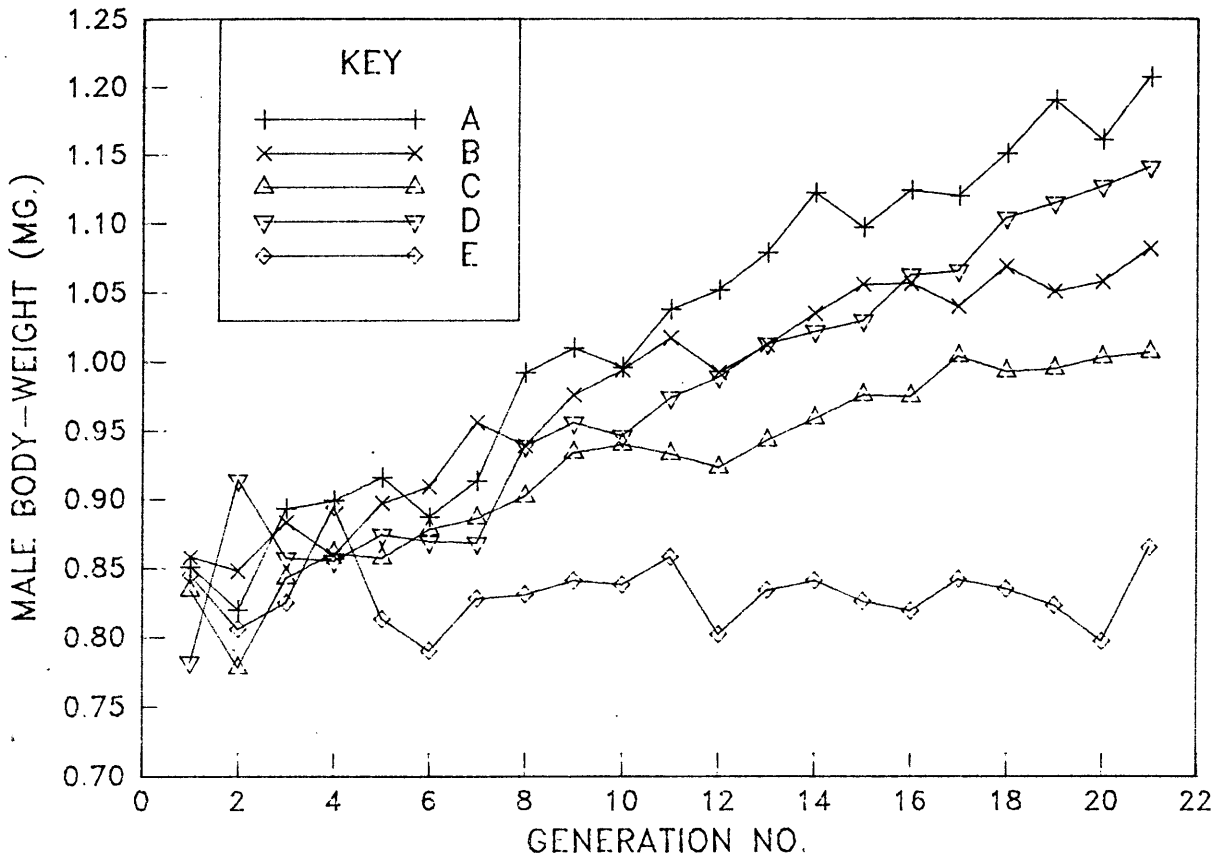

FIG. 3.2: ABSOLUTE RESPONSE VS. GENER. NO.,FEMALE,BLOCK 1

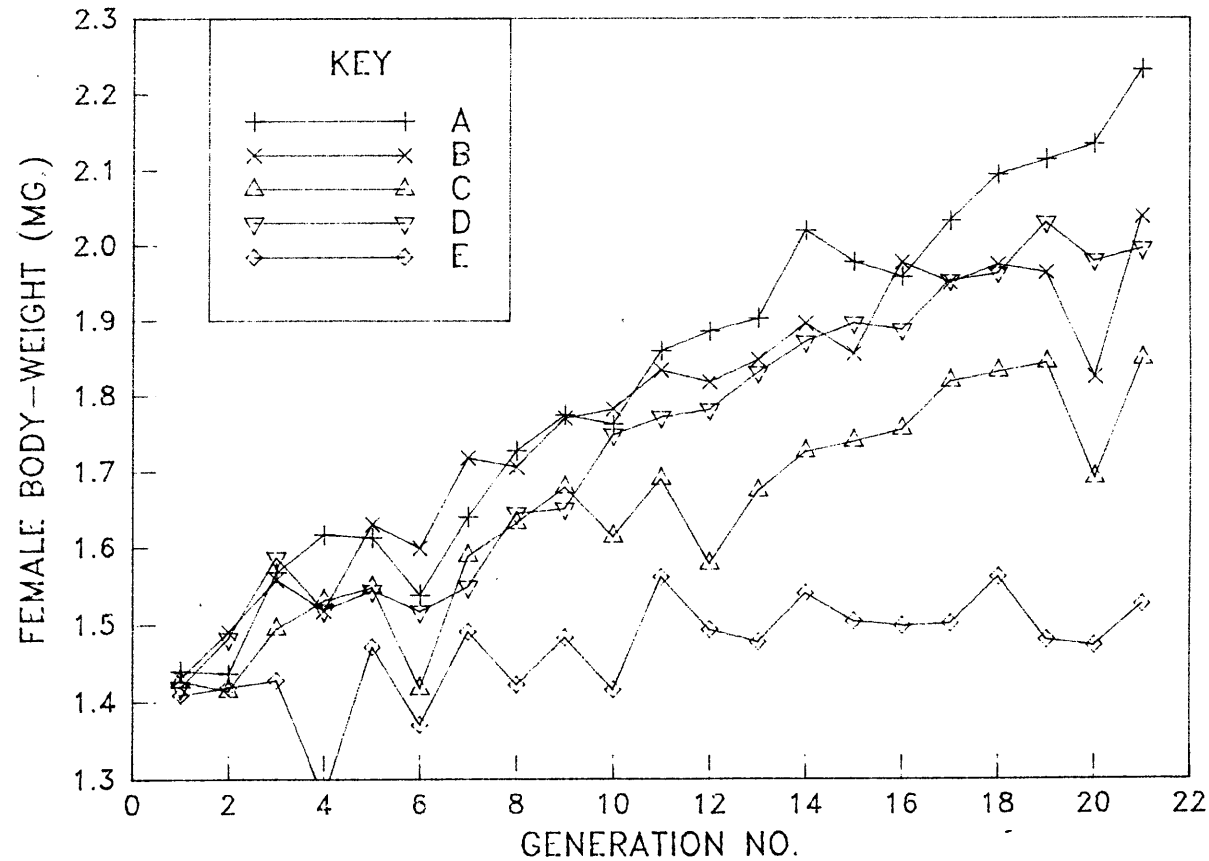

FIG. 3.3: ABSOLUTE RESPONSE VS. GENER. NO.,MALE,BLOCK 2

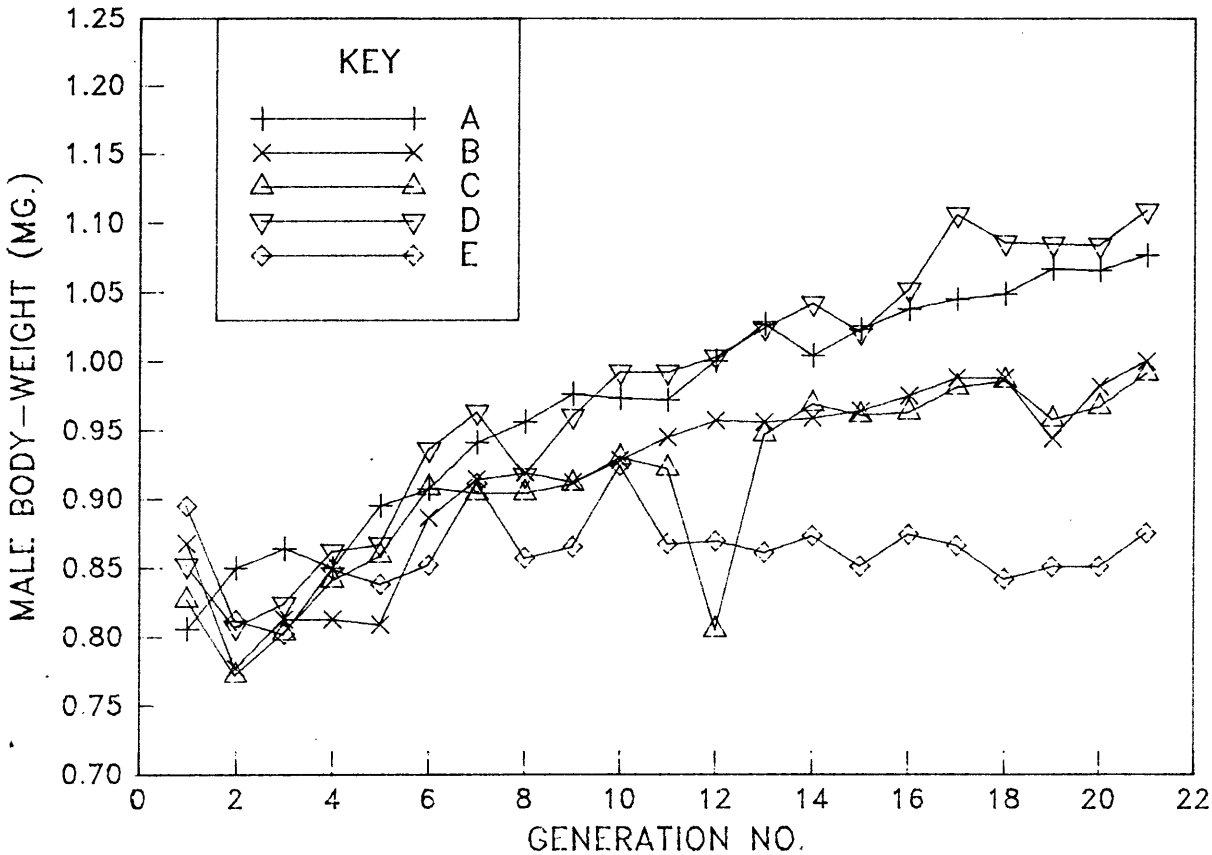

FIG. 3.4: ABSOLUTE RESPONSE VS. GENER. NO.,FEMALE,BLOCK 2

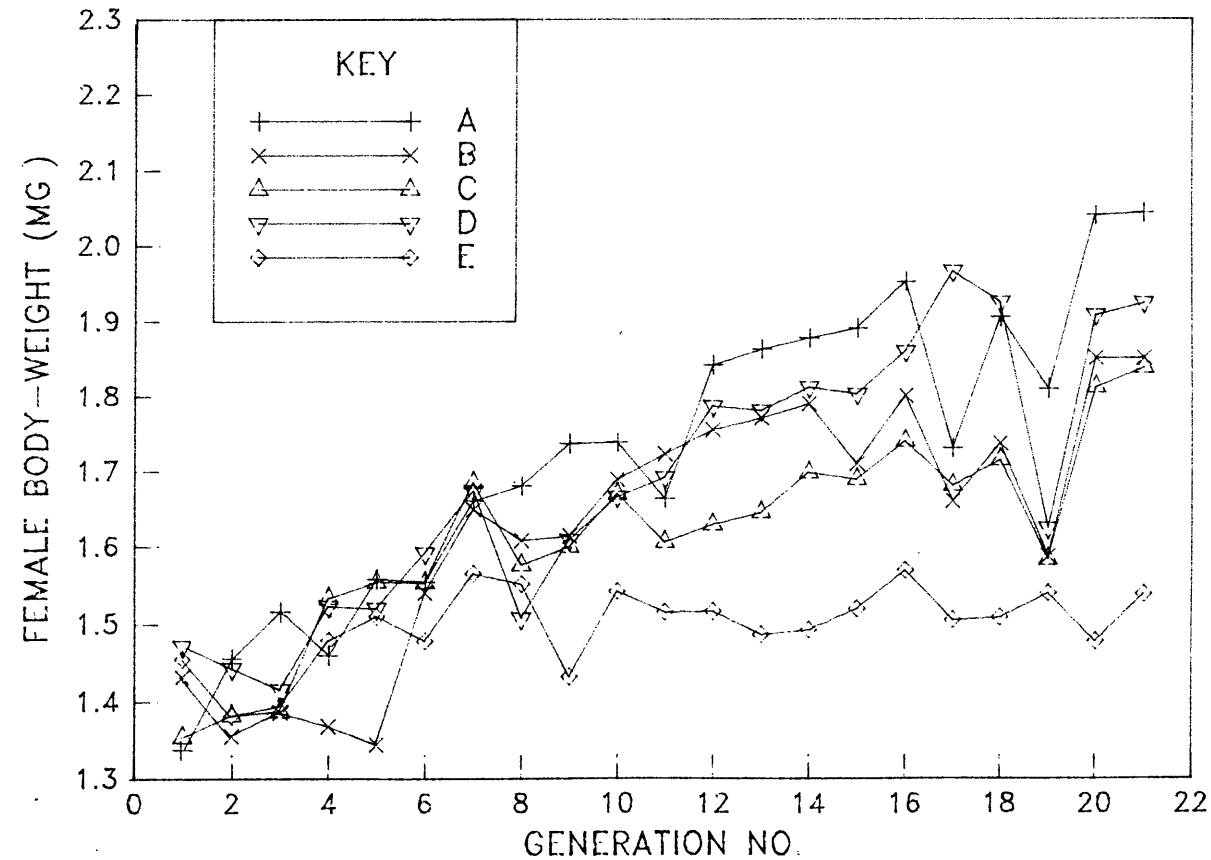

FIG. 3.5: CUM. RESP.(CORR.) VS. GENER. NO.,MALE,BLOCK 1

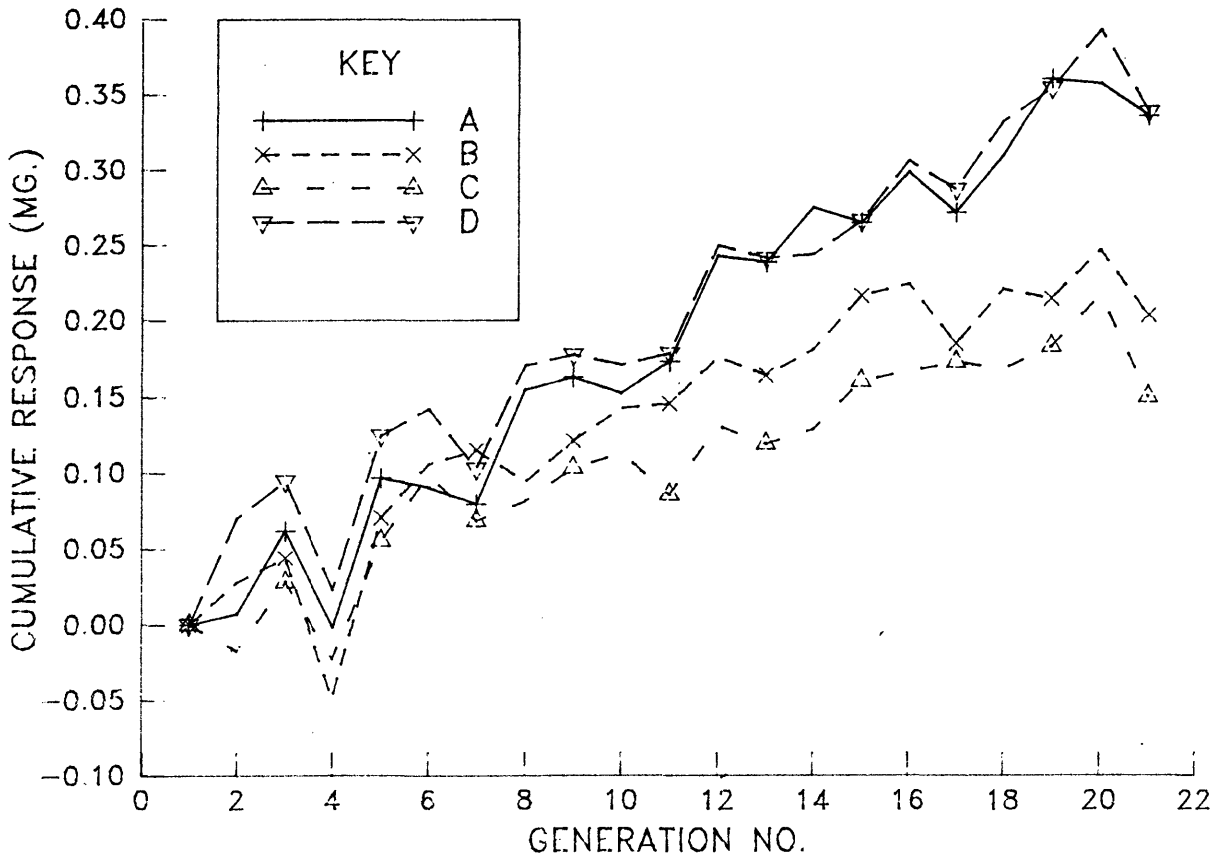

FIG. 3.6 CUM. RESP.(CORR.) VS. GENER. NO.,MALE,BLOCK 2

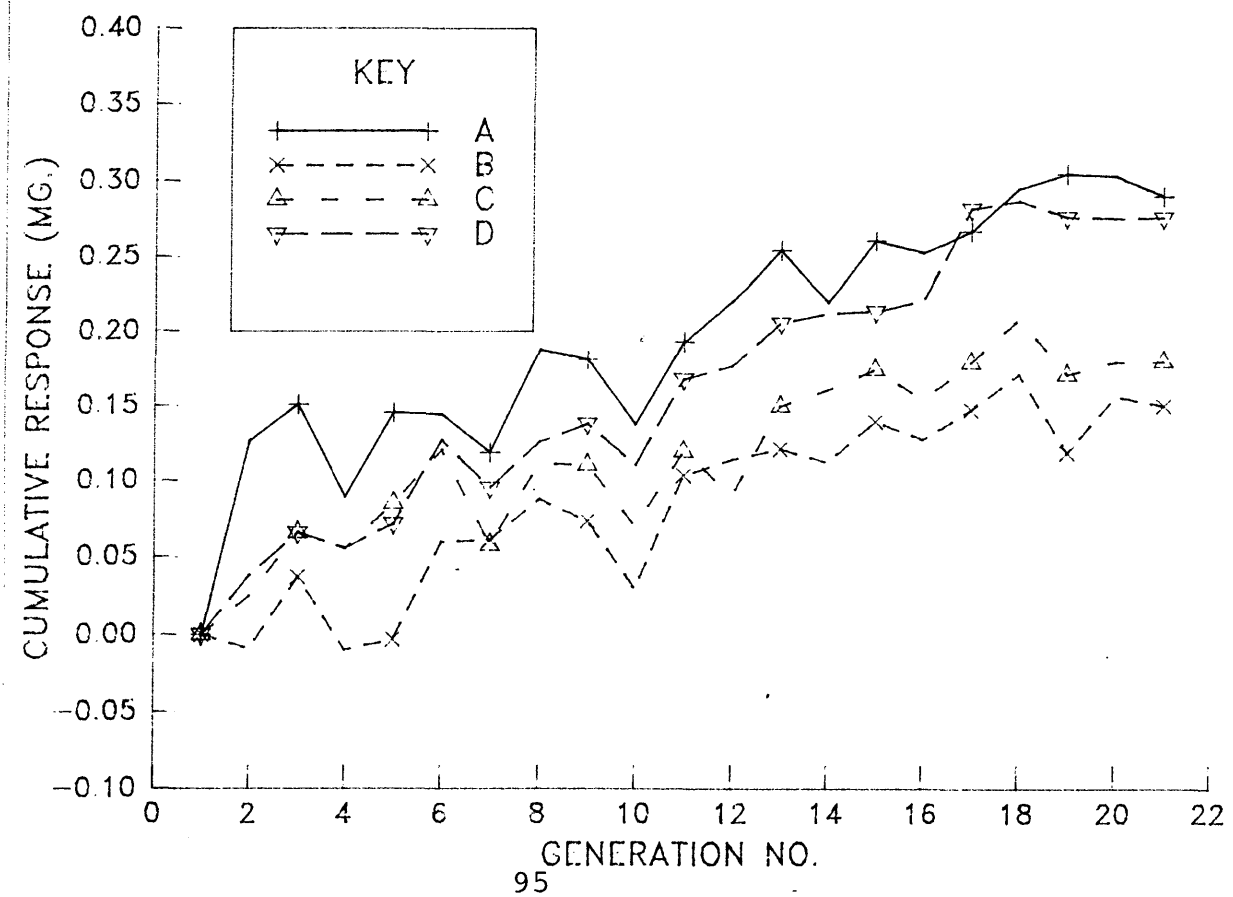

FIG. 3.7: CUM. RESP.(CORR.) VS. GENER. NO.,FEMALE,BLOCK 1

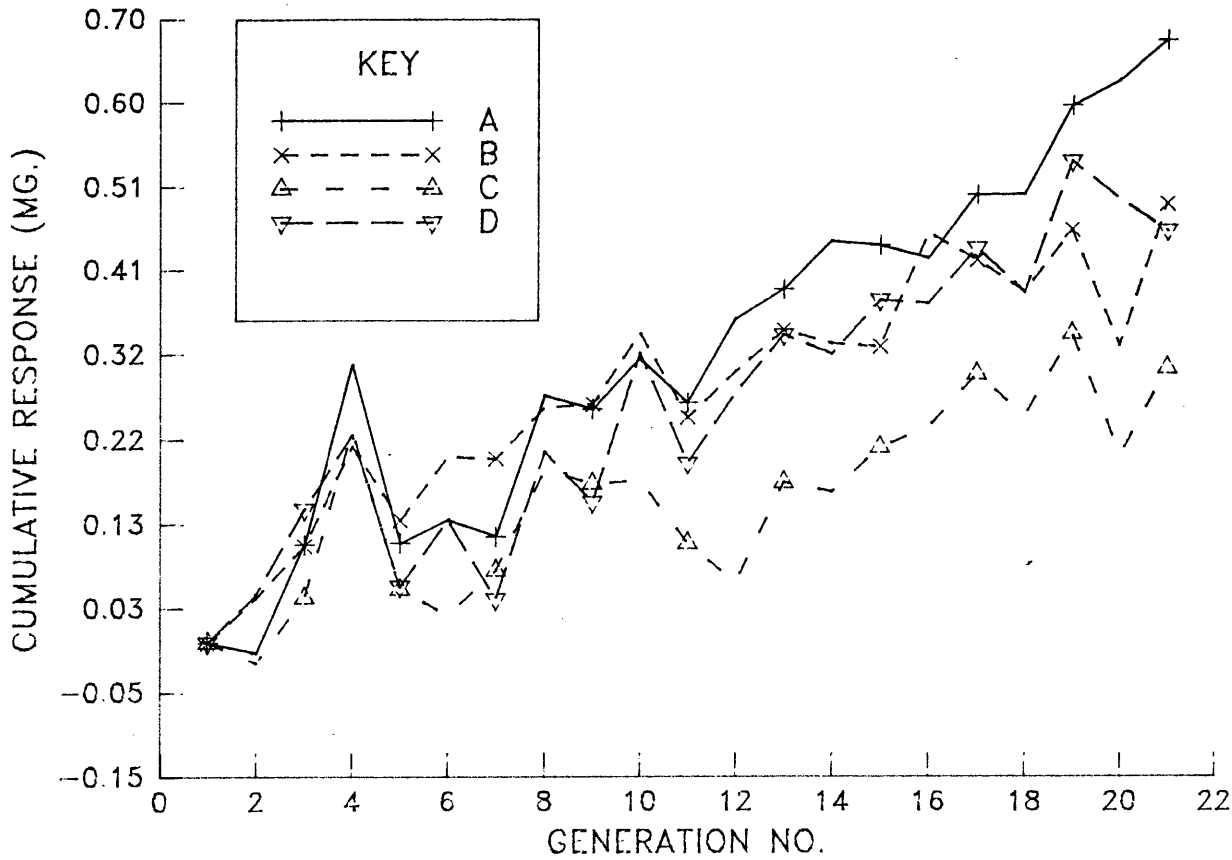

FIG. 3.8. CUM. RESP.(CORR.) VS. GENER. NO.,FEMALE,BLOCK 2

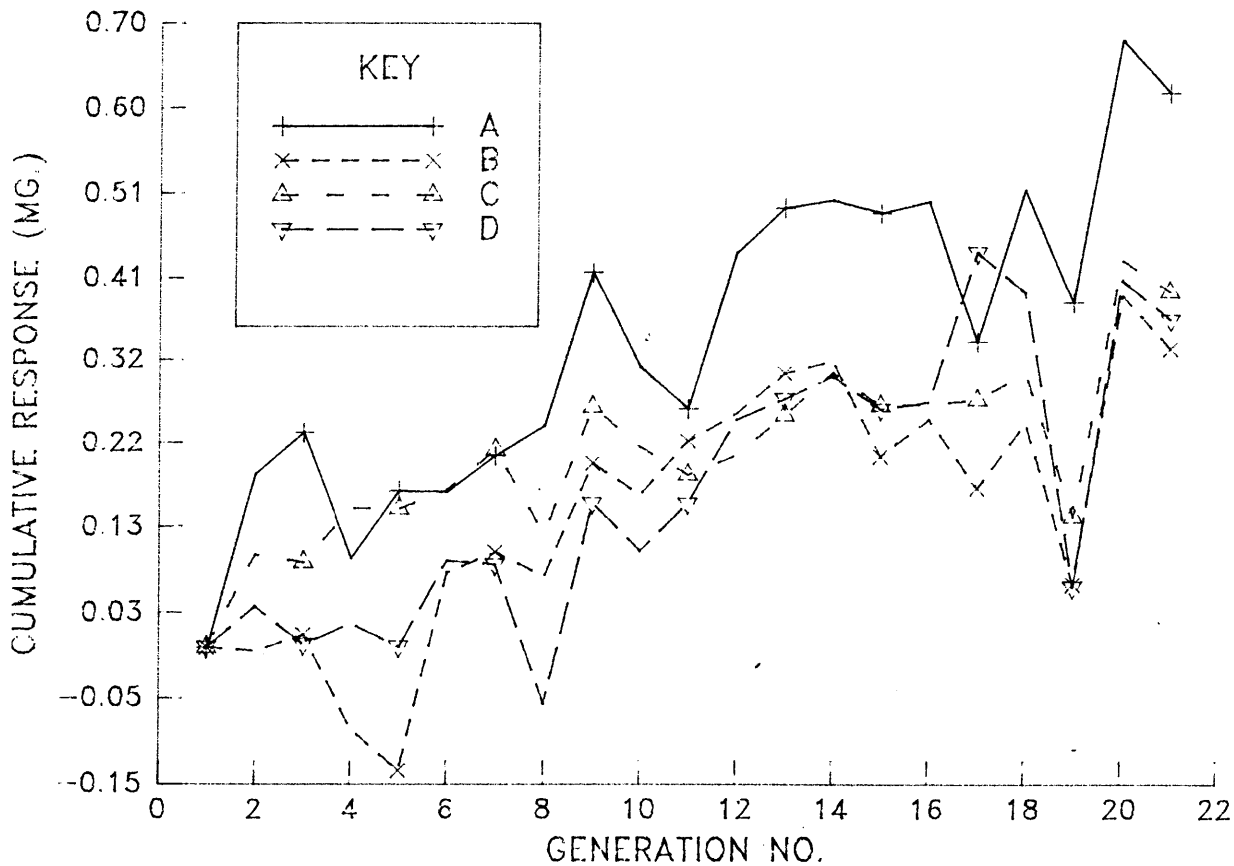

FIG. 3.9: CUM. RESP.(CORR.) VS. CUM. SEL. DIFF.,MALE,BLOCK 1

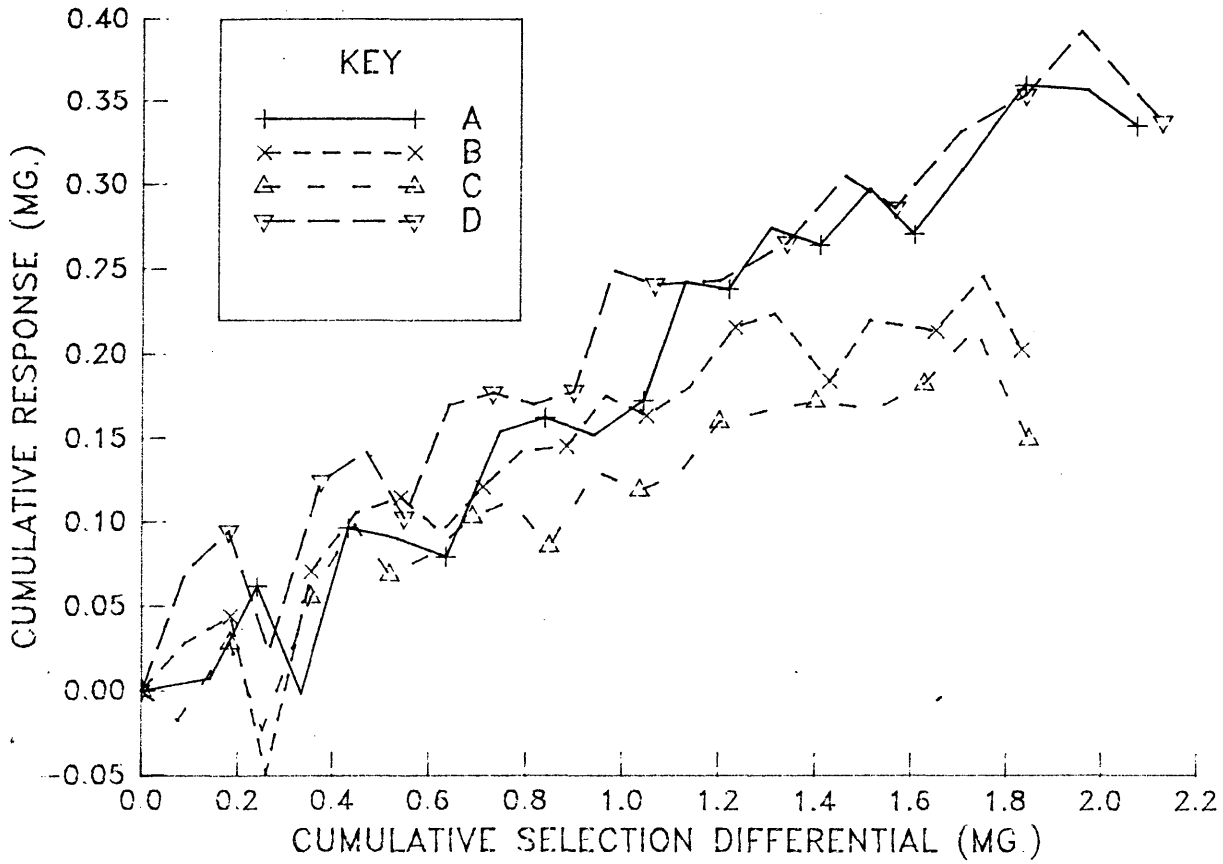

FIG. 3.10. CUM RESP.(CORR.) VS. CUM. SEL. DIFF.,MALE,BLOCK 2

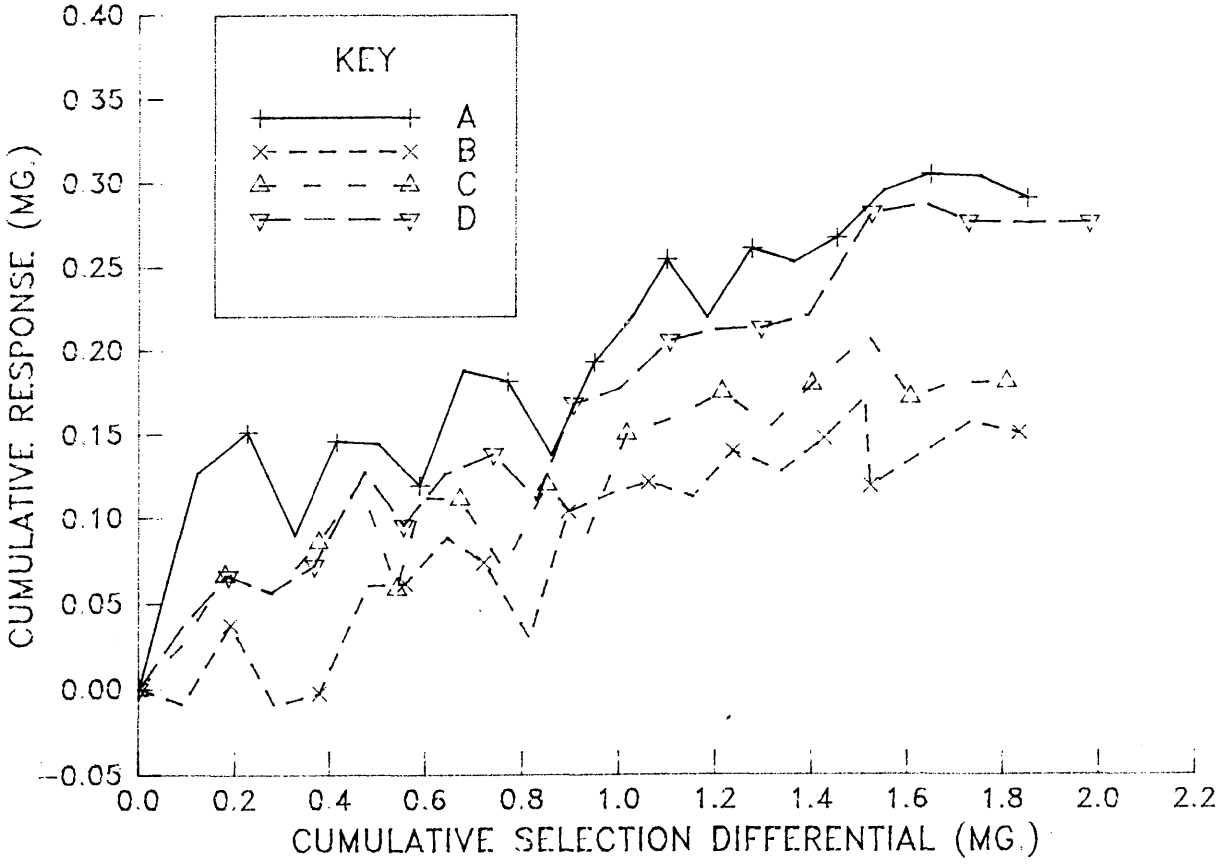

FIG. 3.11: CUM. RESP.(CORR.) VS. CUM. SEL. DIFF.,FEMALE,BLOCK 1

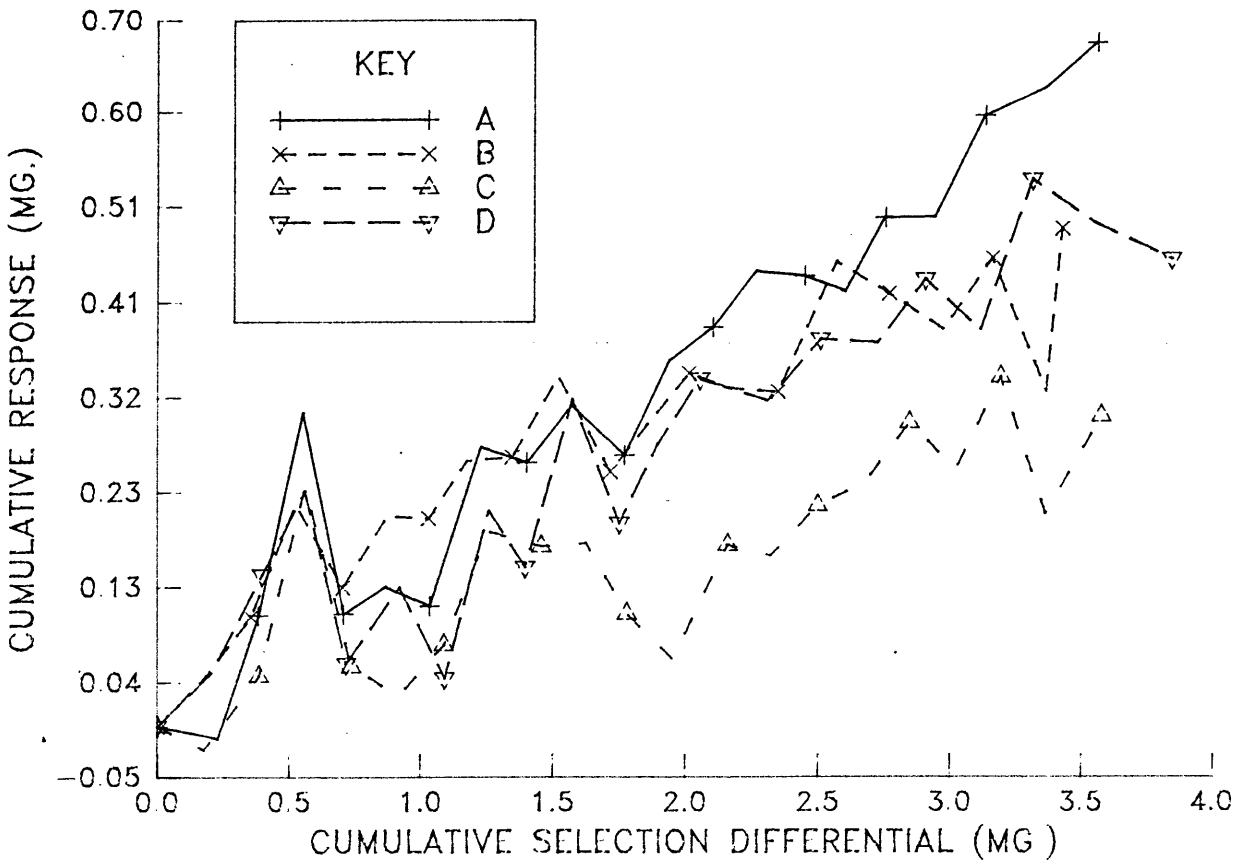

FIG. 3.12: CUM. RESP.(CORR.) VS. CUM. SEL. DIFF.,FEMALE,BLOCK 2

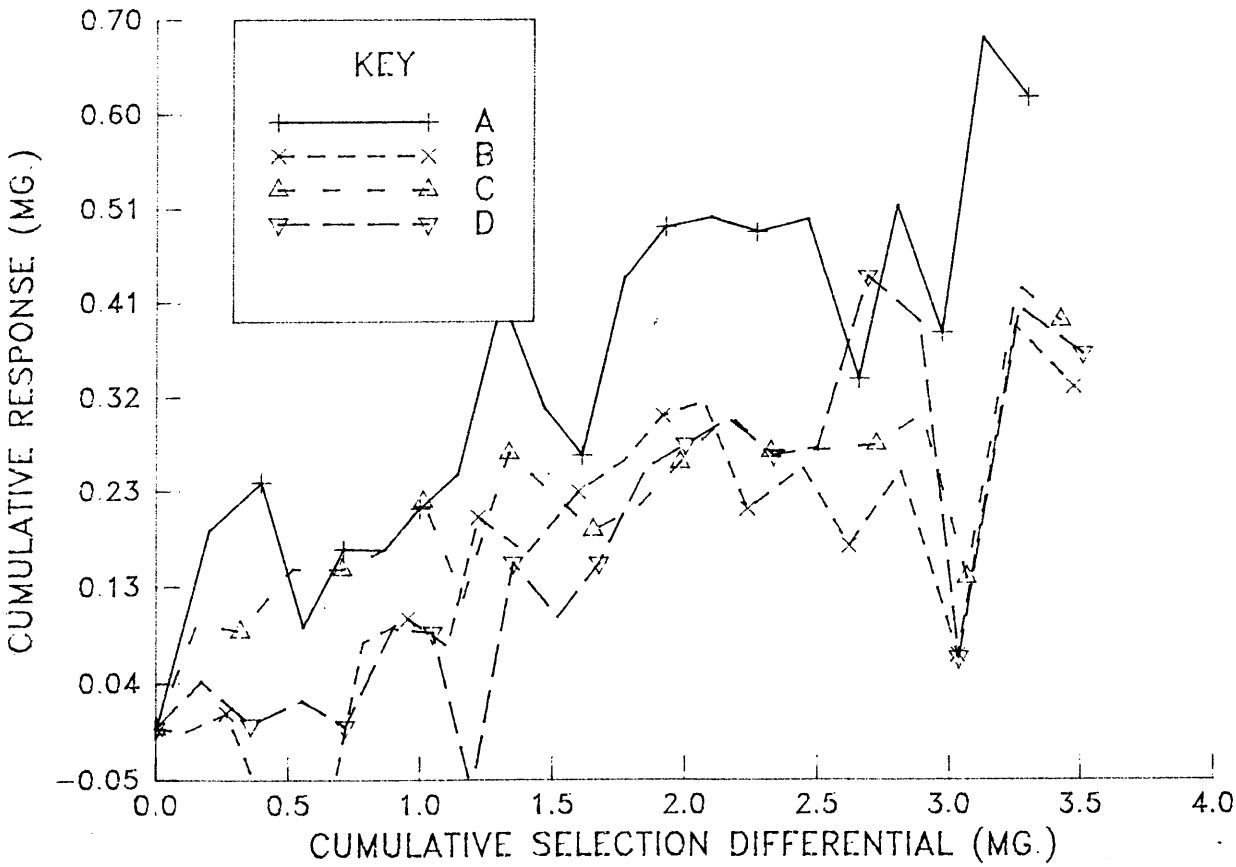

FIG. 3.13: PHENOTYPIC STD. DEV. VS. GENER. NO.,MALE,BLOCK 1

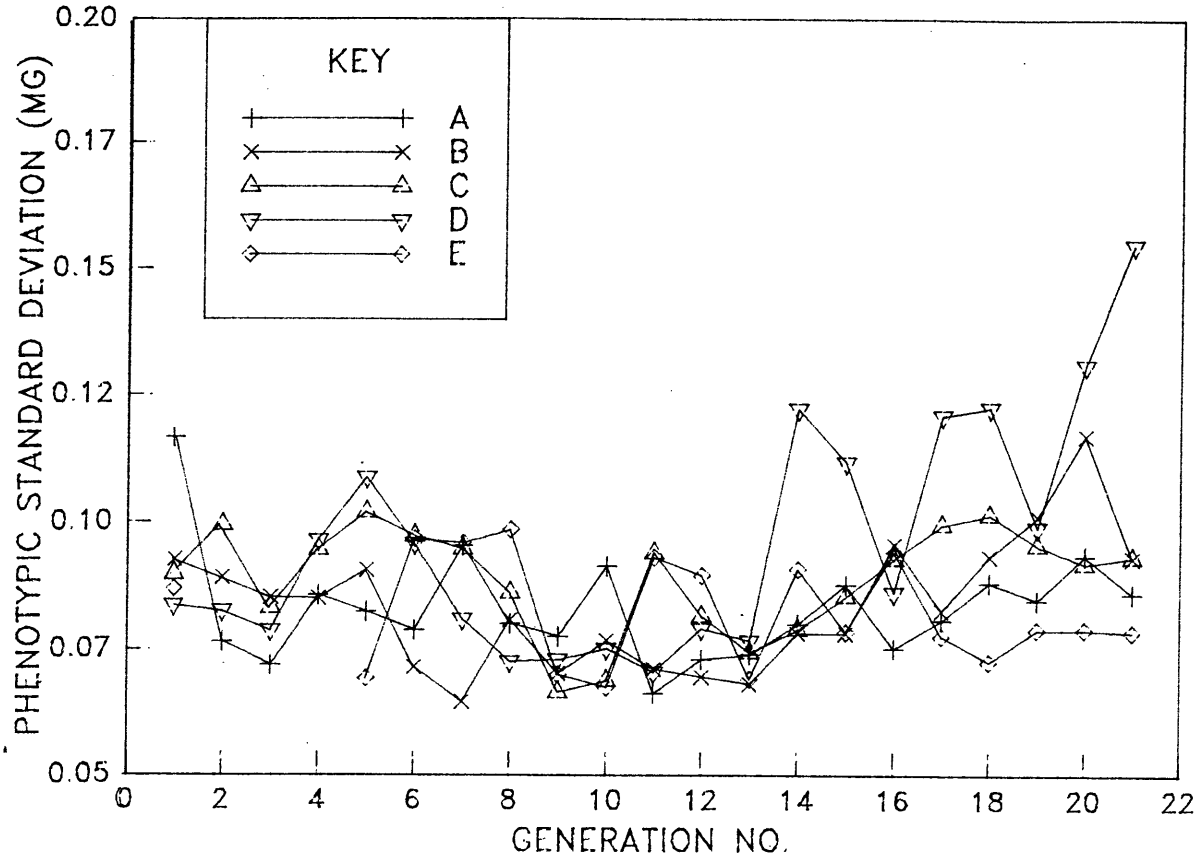

FIG. 3.14: PHENOTYPIC STD. DEV. VS. GENER. NO.,MALE,BLOCK 2

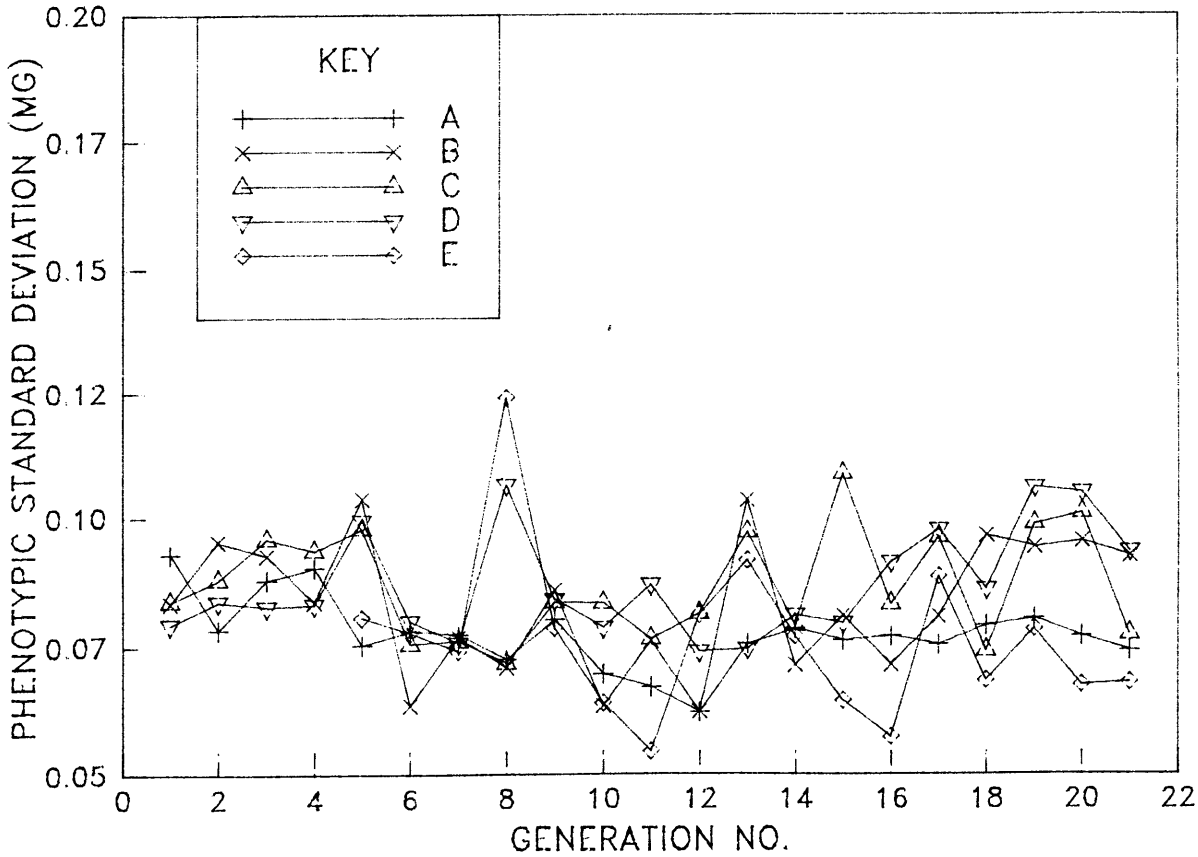

FIG. 3.15: PHENOTYPIC STD. DEV. VS. GENER. NO.,FEMALE,BLOCK 1

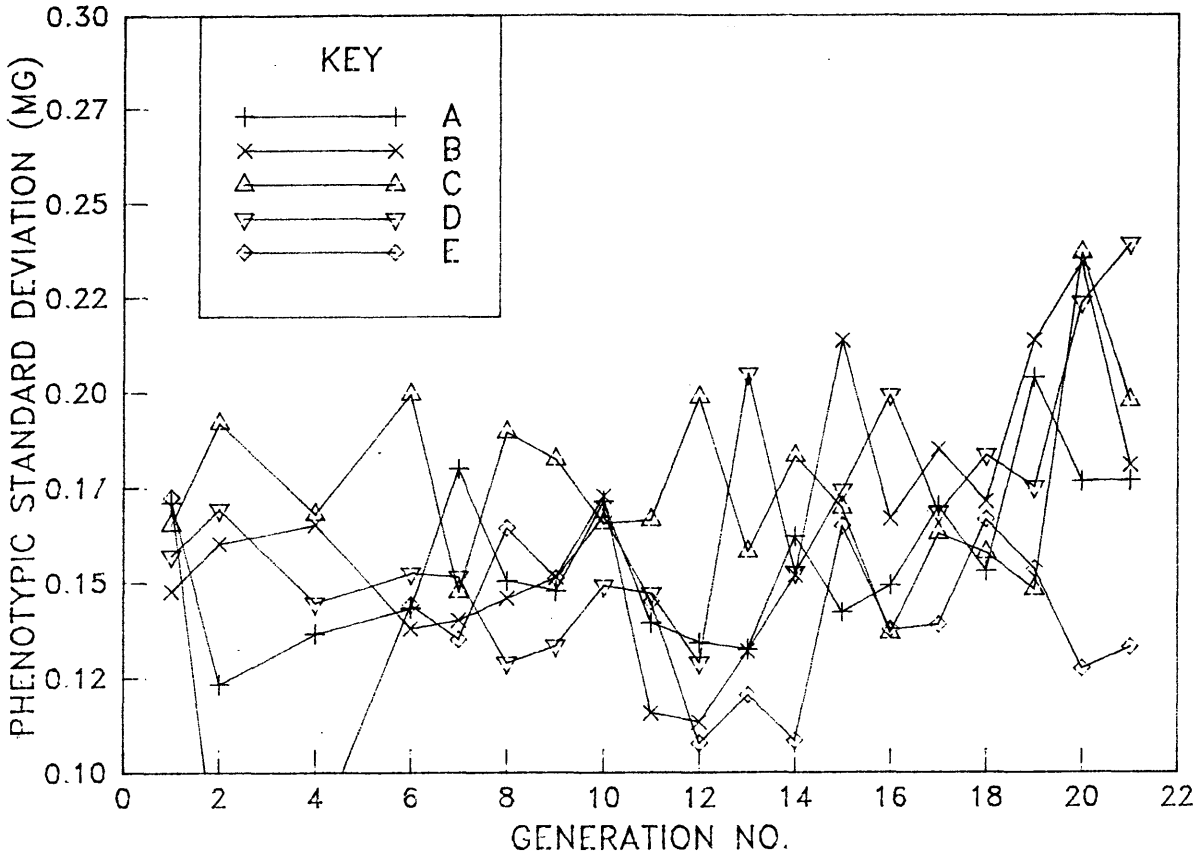

FIG. 3.16. PHENOTYPIC STD. DEV. VS. GENER. NO.,FEMALE,BLOCK 2

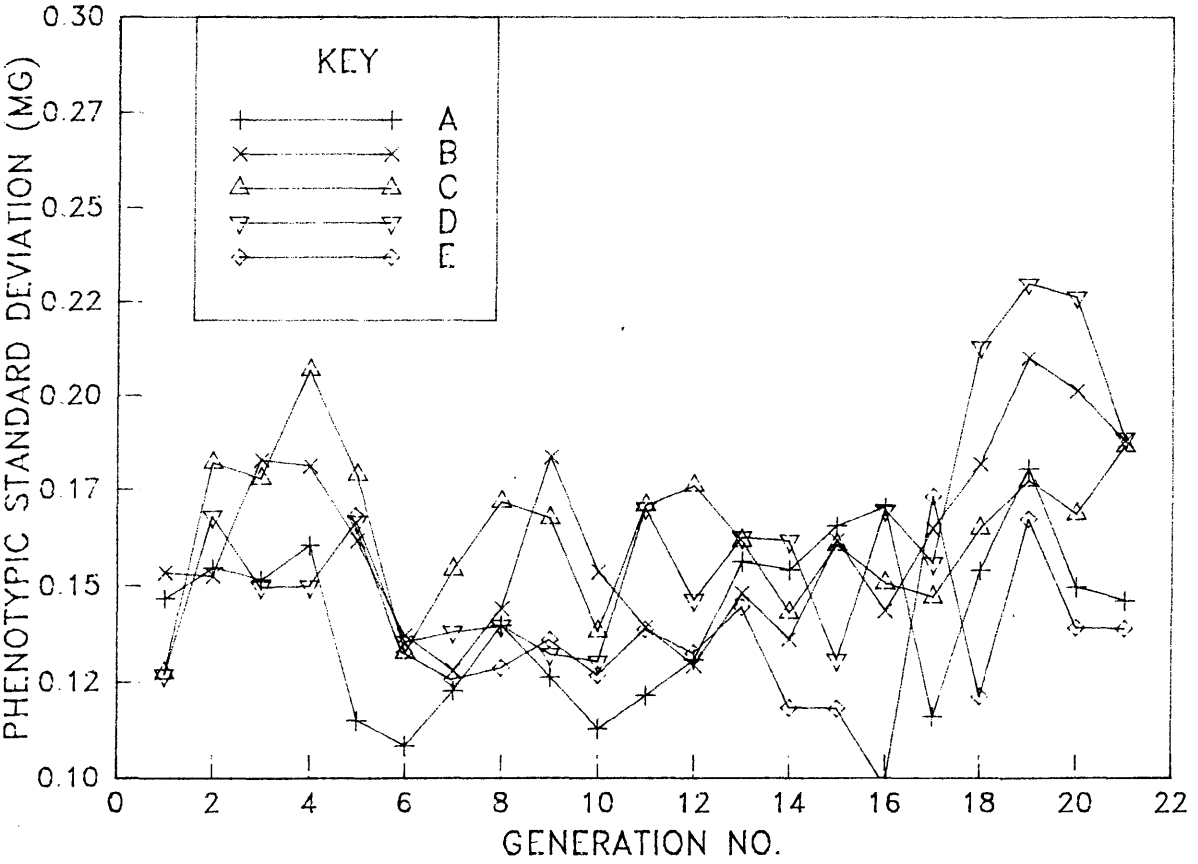

FIG. 3.17: ABSOLUTE RESPONSE VS. GENER. NO.,G21-G34,MALE,BLOCK 1

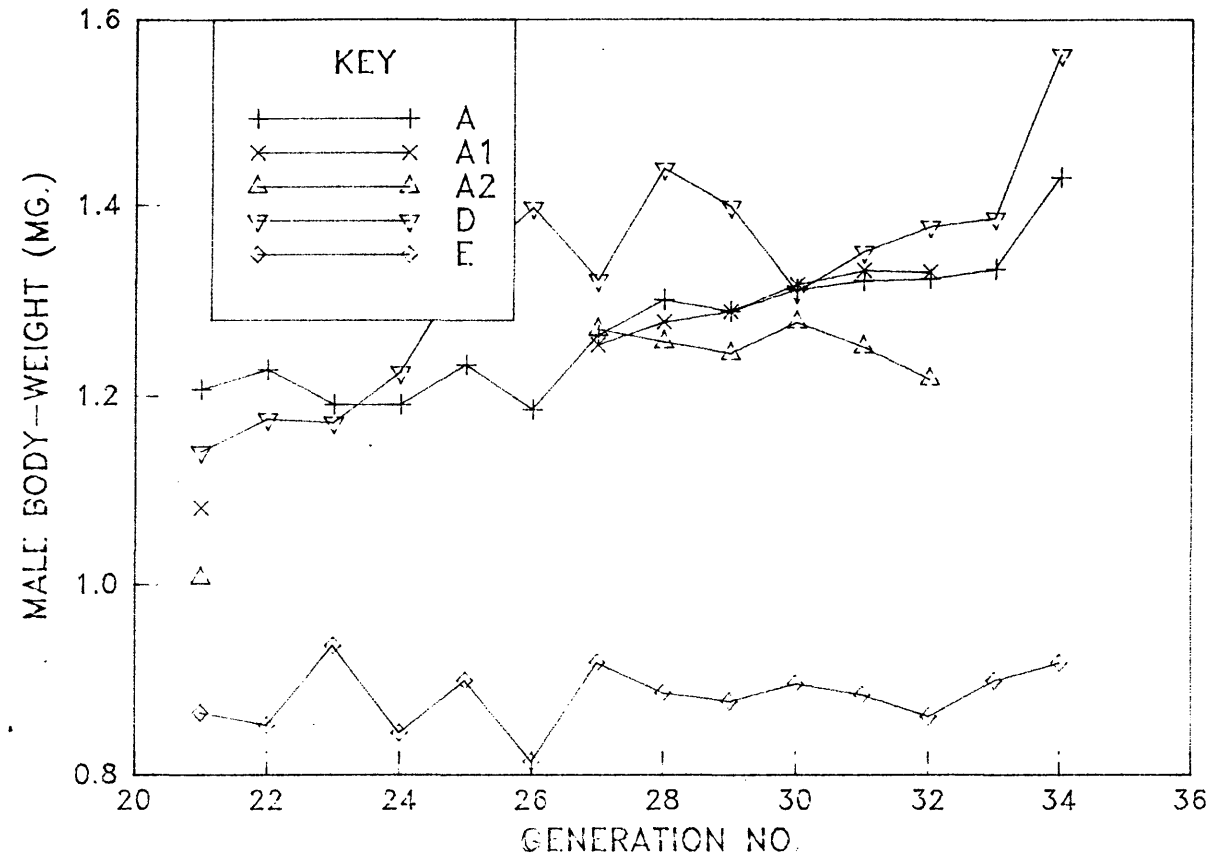

FIG. 3.18. ABSOLUTE RESPONSE VS. GENER. NO.,G21-G34,FEMALE,BLOCK 1

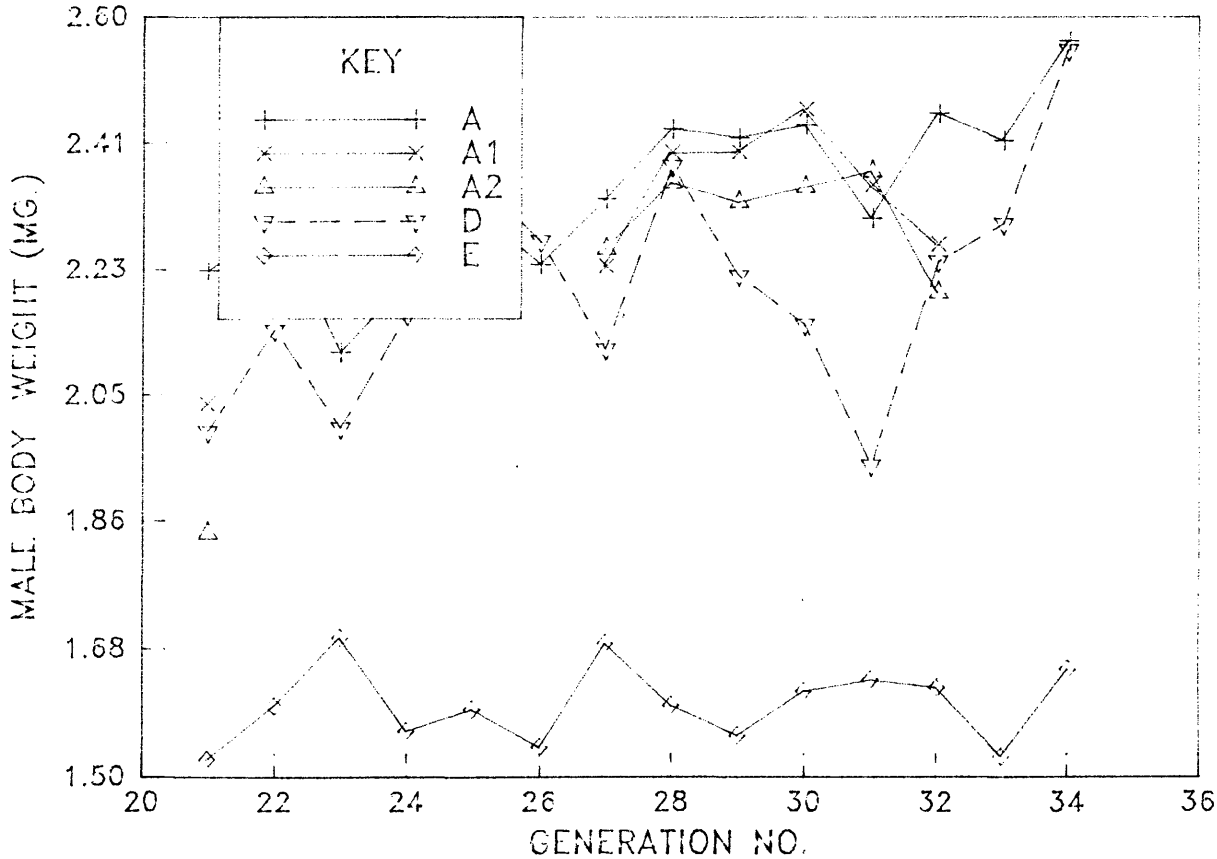

FIG. 3.19: ABSOLUTE RESPONSE VS. GENER. NO.,G21–G34,MALE,BLOCK 2

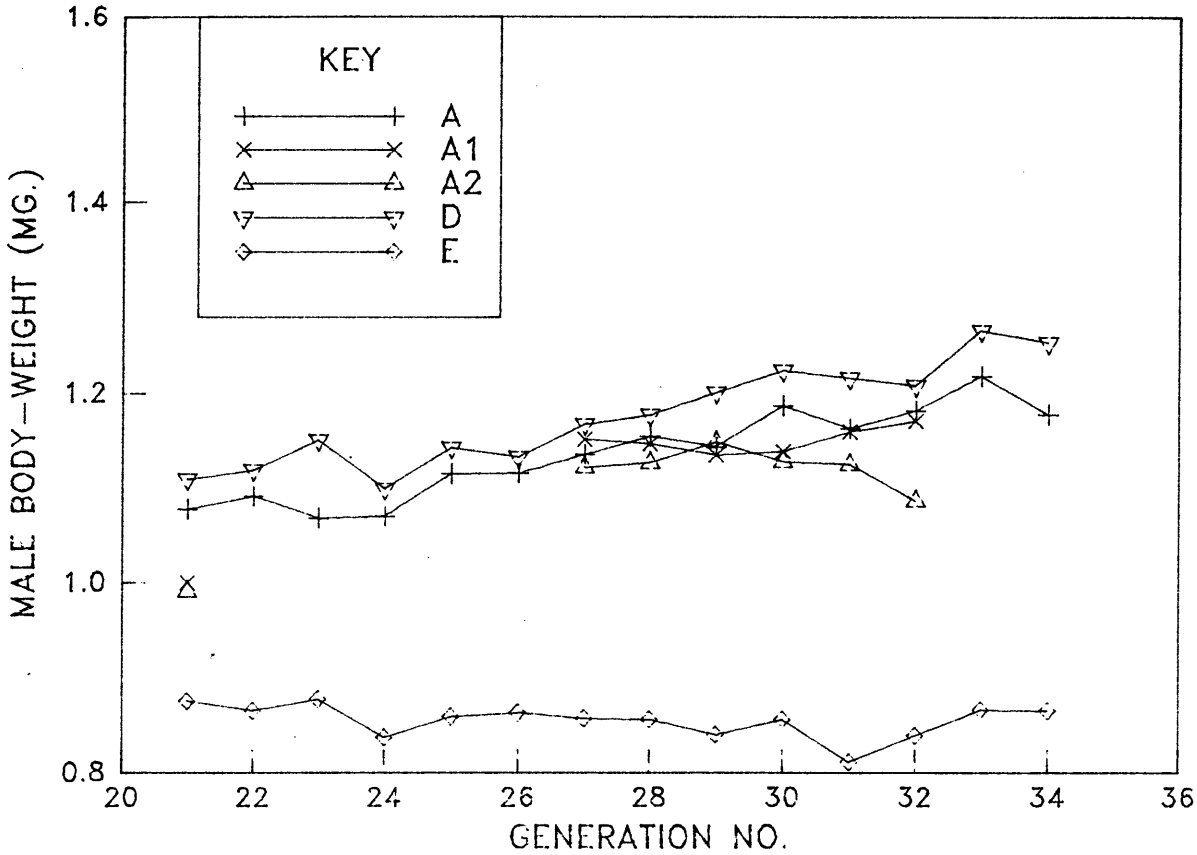

FIG. 3.20: ABSOLUTE RESPONSE VS. GENER. NO.,G21–G34.FEMALE,BLOCK 2

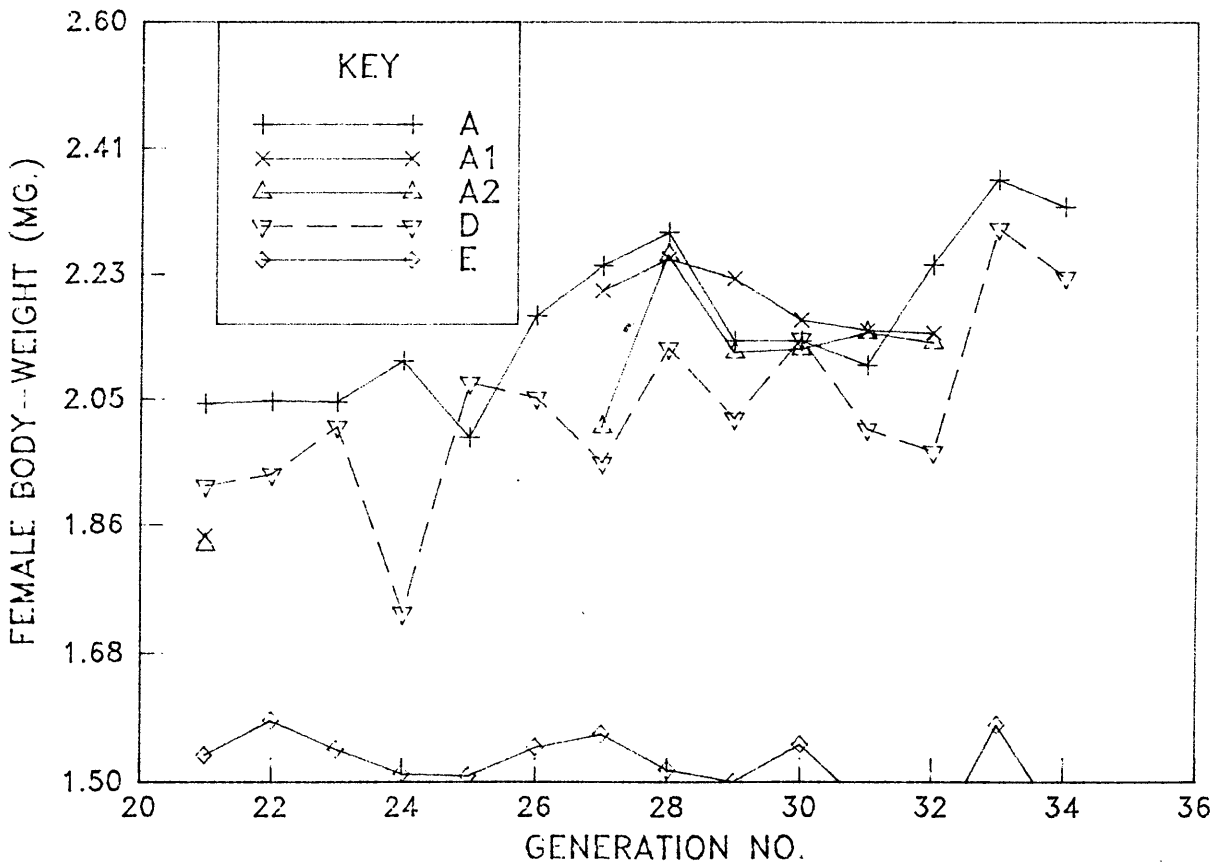

FIG. 3.21: CUM. RESP. (CORR.) VS. GENER. NO.,G21–G34,MALE,BLOCK 1

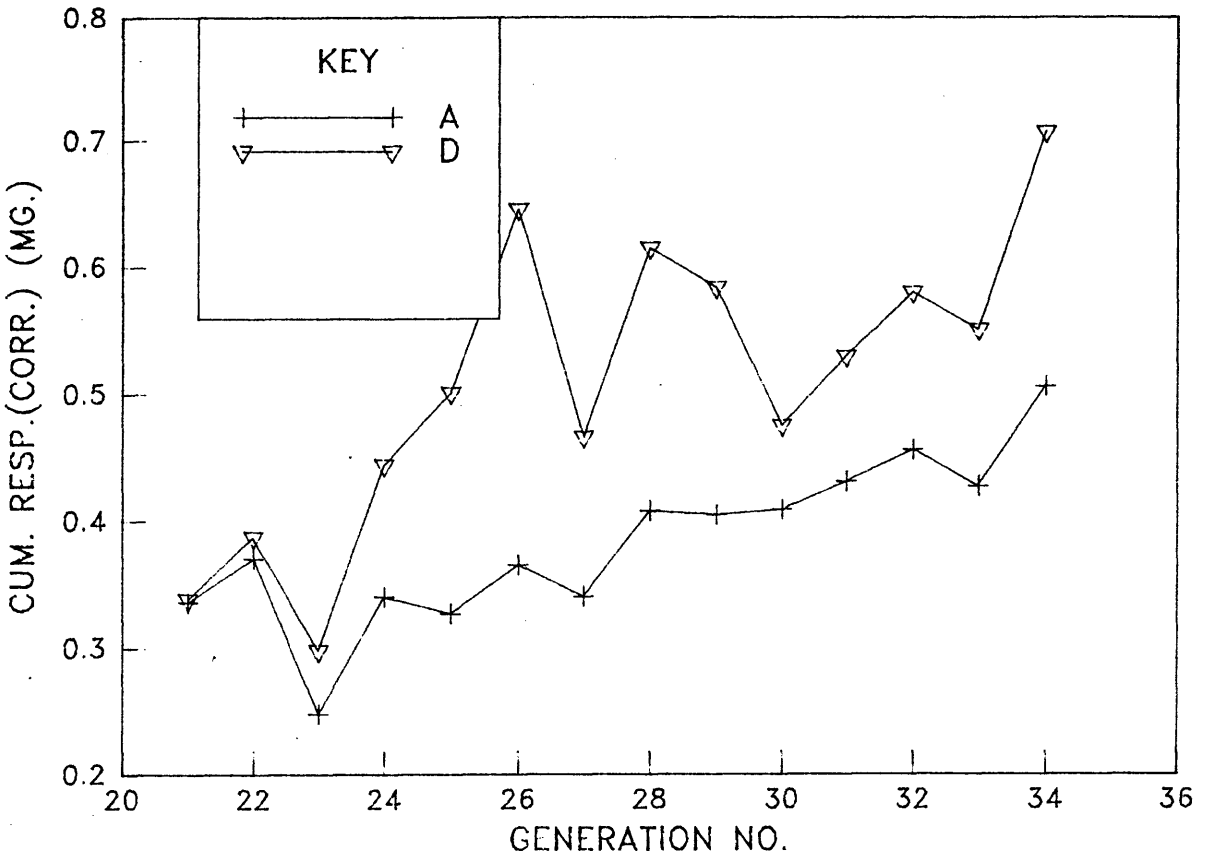

FIG. 3.22: CUM. RESP. (CORR.) VS. GENER. NO.,G21–G34,FEMALE,BLOCK 1

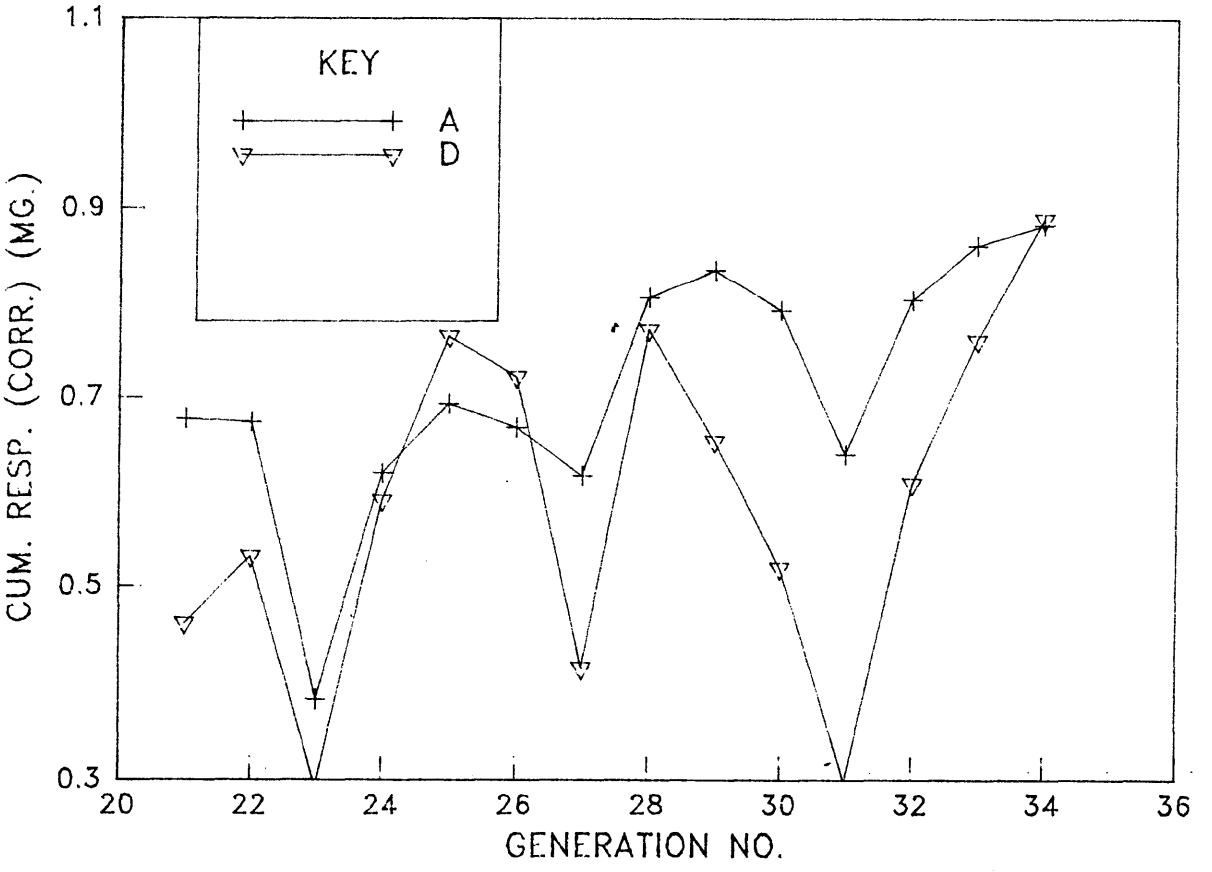

FIG. 3.23: CUM. RESP. (CORR.) VS. GENER. NO.,G21–G34,MALE,BLOCK 2

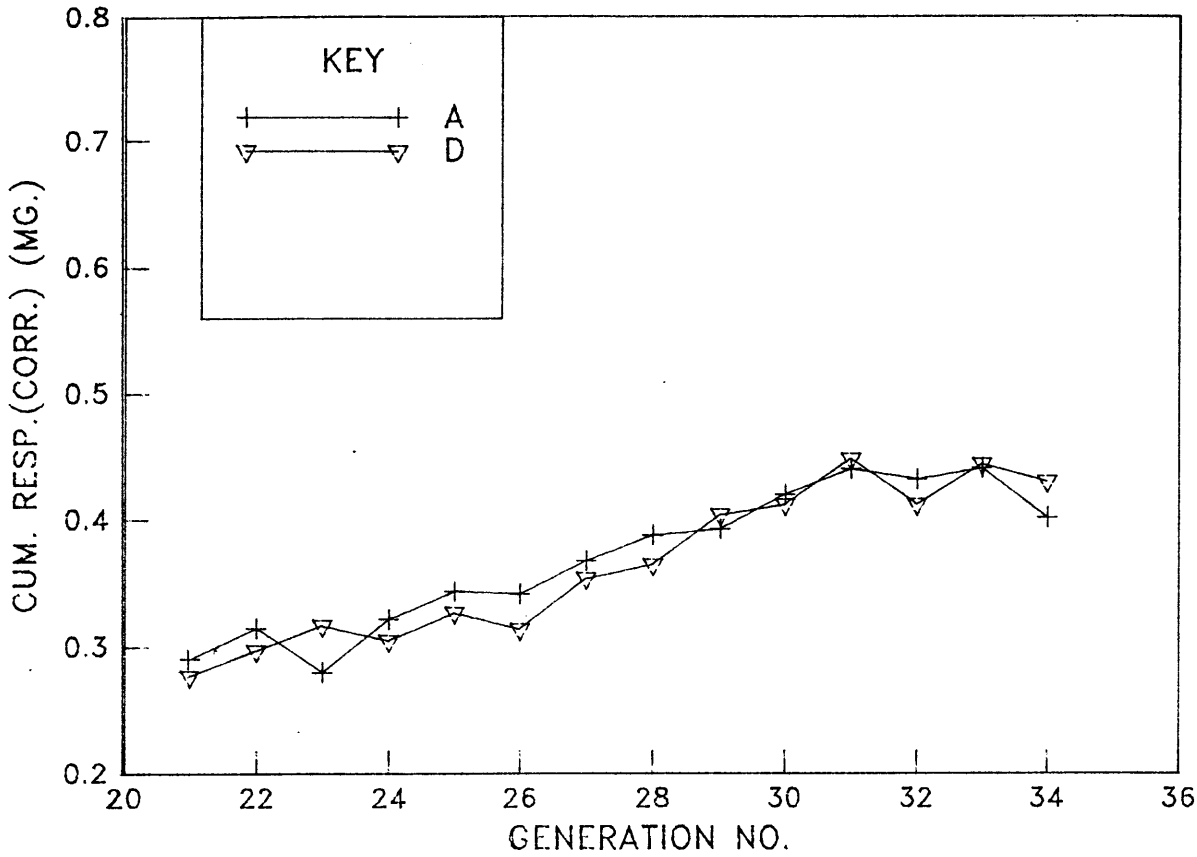

FIG. 3.24: CUM. RESP. (CORR.) VS. GENER. NO.,G21–G34,FEMALE,BLOCK 2

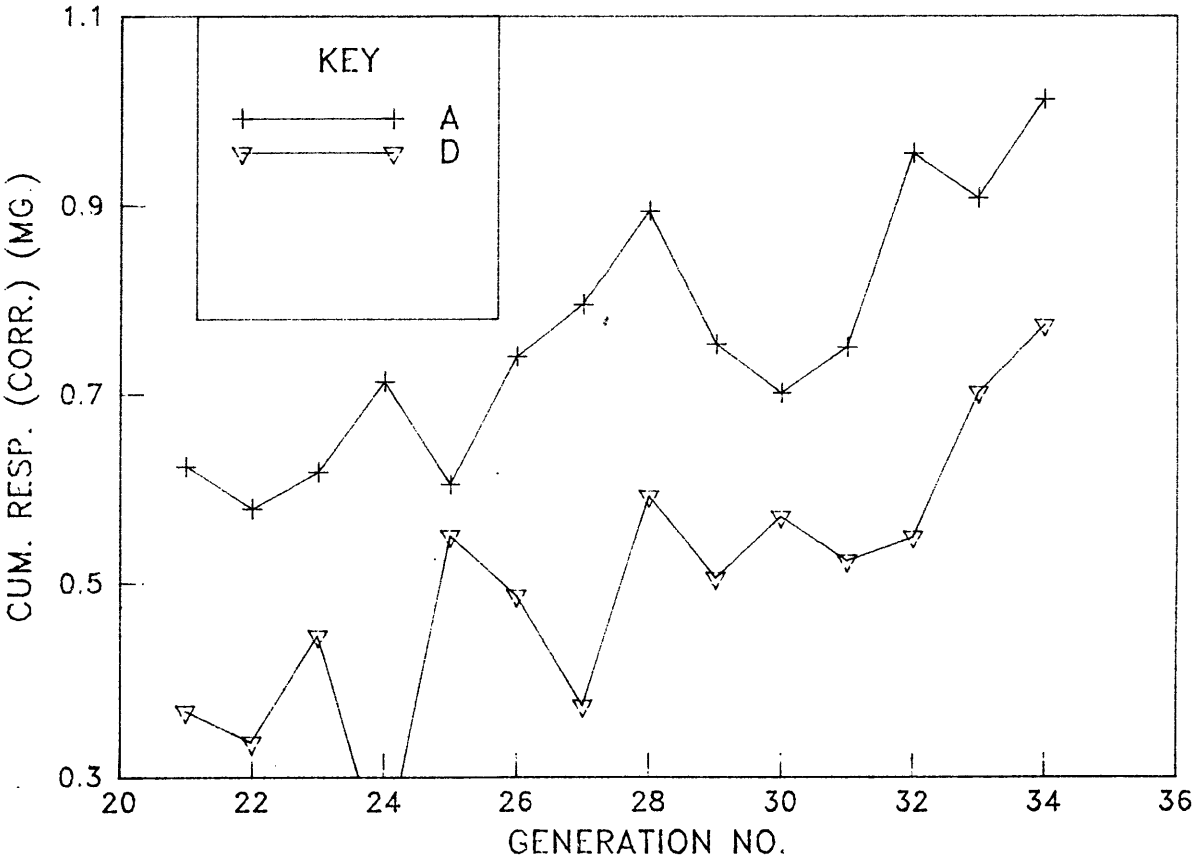

FIG. 3.25: ABSOLUTE RESPONSE VS. GENER. NO.,G0–G34,MALE,BLOCK 1

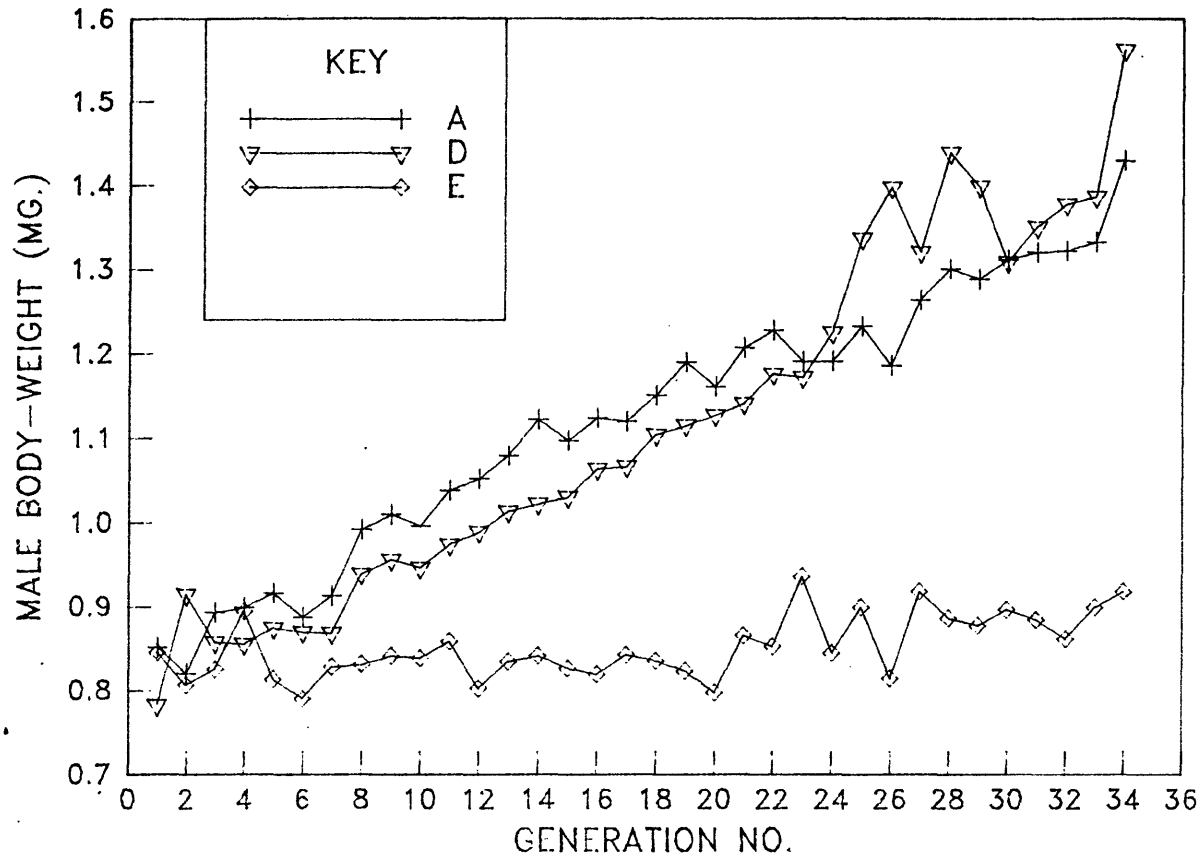

FIG. 3.26: ABSOLUTE RESPONSE VS. GENER. NO.,G0–G34,FEMALE,BLOCK1

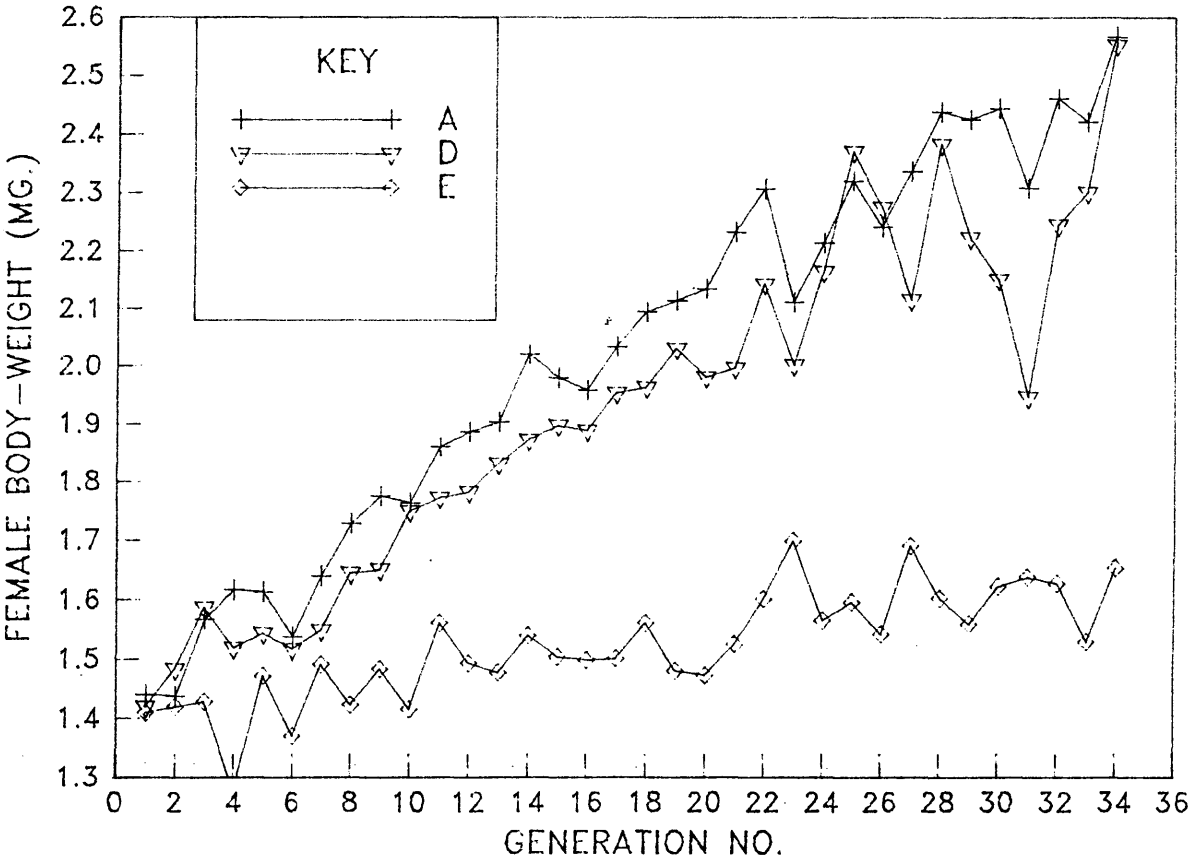

FIG. 3.27: ABSOLUTE RESPONSE VS. GENER. NO.,G0–G34,MALE,BLOCK 2

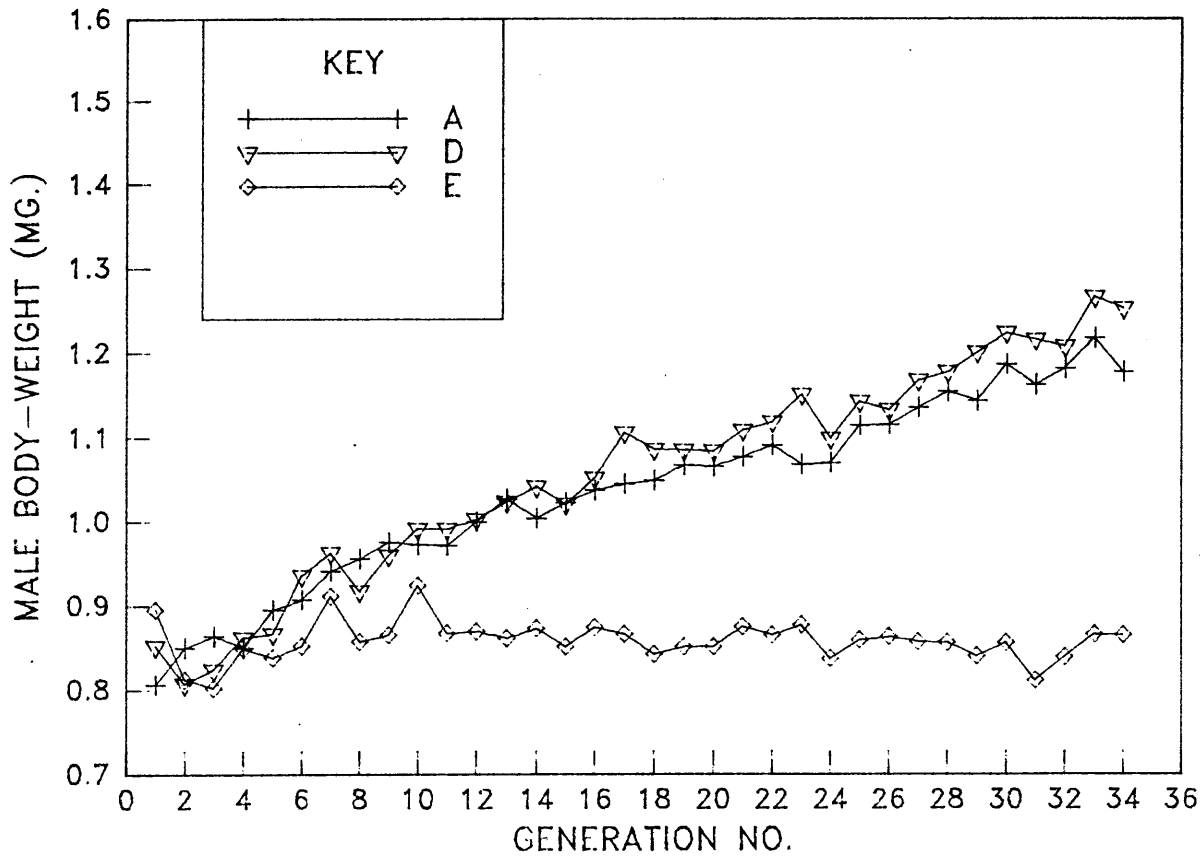

FIG. 3.28: ABSOLUTE RESPONSE VS. GENER. NO.,G0 -G34,FEMALE,BLOCK 2

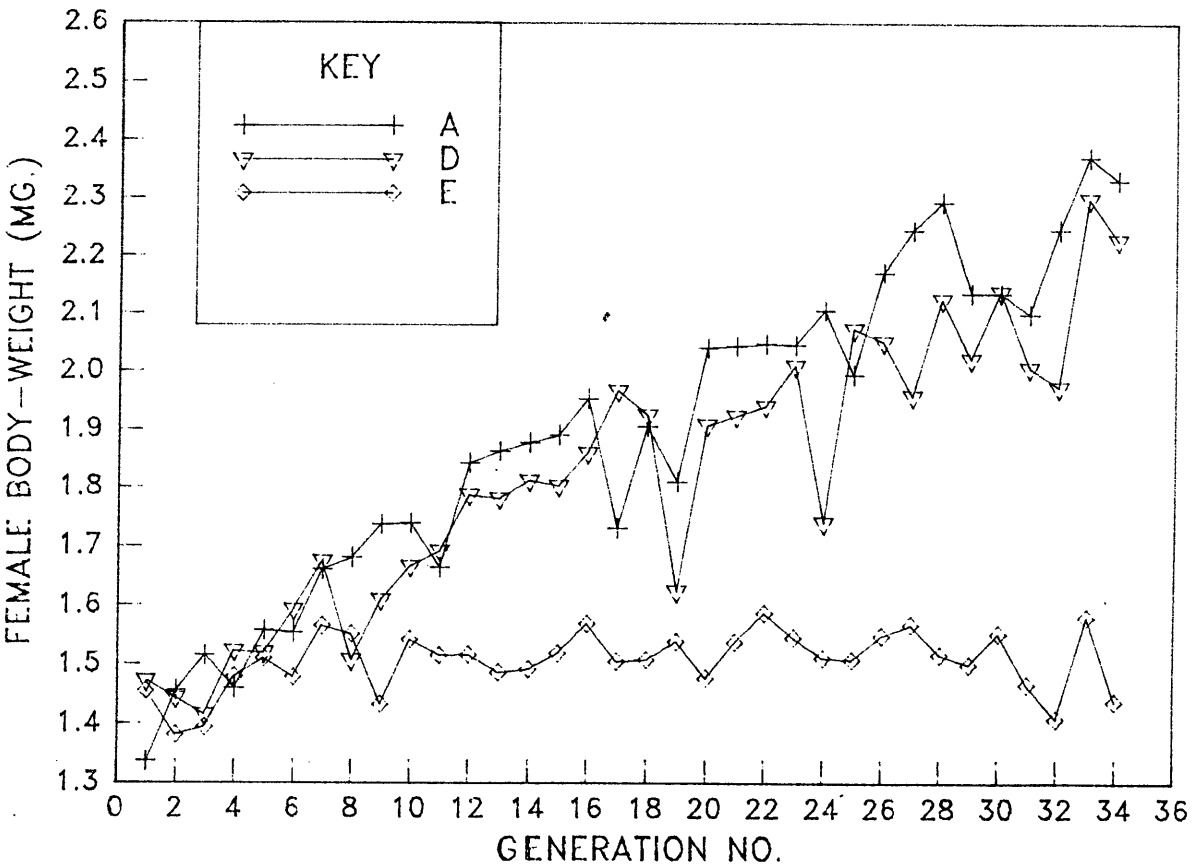

FIG. 3.29: CUM. RESP. (CORR.) VS. GENER. NO.,G0–G34,MALE,BLOCK 1

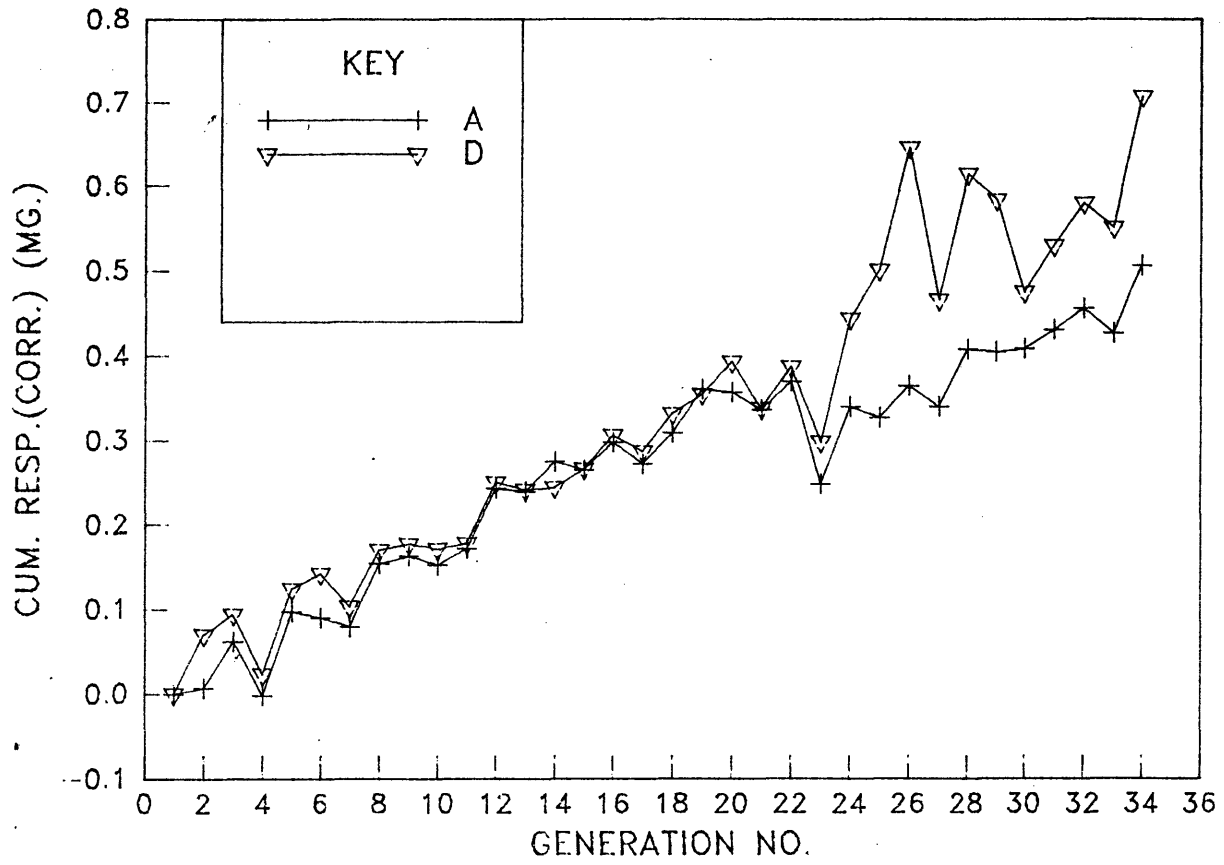

FIG. 3.30: CUM. RESP. (CORR.) VS. GENER. NO.,G0–G34,FEMALE,BLOCK 1