COMPUTER SIMULATION OF DIRECTIONAL SELECTION IN LARGE POPULATIONS.
II. THE ADDITIVE × ADDITIVE AND MIXED MODELS

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THE change of the additive genetic variance (σ_A^2) under selection and the ability of the estimate of heritability in the narrow sense (LUSH 1940) (h^2) to predict genetic gain are important considerations in formulating breeding plans and understanding selection experiments. YOUNG (1966) has discussed the value of prediction and the decay of additive genetic variance when the character is controlled by additive genes (the "A model") and genes with some dominance (the "D model"). He assumed control by ten loci, and simulated selective breeding by computer.

In the same series of investigations further models were used, the results of which are reported here. One was epistatic (the "E"), in which the genetic value of each pair of loci was assumed to be determined by the product of their respective additive values (A × A interaction). In the A, D and E models the ten loci of each individual were assumed to show a uniform gene action, either all additive, all dominance, or all epistatic. In addition to these, four slightly more complex genetic situations were investigated, in each of which, a fraction of the ten loci was assumed to show one effect (e.g. additive or dominance) and the remaining fraction a different effect (e.g. epistatic). These will be referred to as mixed models, and a detailed description of each will be given later.

FRASER (1960) was the first to use an epistatic model in computer simulation of selection though his results do not bear on the problem studied here.

GRIFFING (1960) reported the theoretical consequence of directional selection with a character controlled by genes showing A × A epistasis. A most interesting finding was that decline of the population mean was an expected consequence of relaxation of selection, without recourse to natural selection. GRIFFING also presented an approximation for predicting selection gains in a large population, with A × A epistasis. GILL (1965a) simulated genetic advance under truncation selection for some small populations (8 to 32 individuals). The trait under selection was assumed to be determined by 40 loci and four genetic models were used. He found that under an A × A conditional epistatic model predictions of gains from GRIFFING's formula were in most cases overestimates, and concluded that random genetic drift plus changes in genetic variance had been responsible for the disagreement.

In a different report, GILL (1965b) again simulated gains by selection in small

populations (8 to 32 individuals), with nine other genetic models. The rates of advance differed widely, depending on the model assumed. In general, larger populations attained higher means at the end of 30 generations of selection, indicating that the smaller the population the greater the loss of favourable alleles, while genetic advance was faster with the conditional A × A than with the additive model.

The A × A model used here differs from GILL's, while the mixed models have not previously been analysed. Again, as in the first paper of the series, the predictive ability of heritability (h^2) and changes under selection in the additive genetic variance ($\sigma_A{}^2$) were the main problems under consideration.

The parameters and the computer programme: The parameters used in the present work were the same as in the previous paper. Briefly these were: (1) The size of the unselected population in each generation was 1000; (2) The character under selection was assumed to be controlled by ten loci with two alleles at each locus; (3) The initial gene frequency for each allele was set at 0.5; (4) Three selection intensities (I) were used. These intensities ($I = 10\%$, 50% and 80%) refer to the proportions of individuals saved for breeding; (5) The character under selection was modified by a normally distributed random factor and three levels of initial heritability ($h^2 = 0.1$, 0.4 and 0.9) were assumed; (6) Three recombination probabilities ($r = 0.05$, 0.2 and 0.5) between adjacent loci were used and these were assumed to be constant throughout 30 generations of selection.

The computer programme used was essentially the same as before. The operations again mimicked a population under random mating with truncation selection. Each population started with a fixed combination of I, r, h^2 and was selected for 30 generations under each genetic model. There was a slight technical difference in the programme when the epistatic model was used; genetic values corresponding to gene doses of each pair of loci were read into genetic value storages. Apart from this, the programme remained unchanged. Additive genetic variances were again calculated by the regression method and the nonadditive variances were obtained by differences. Further details of the computer programme and the parameters used have been described in the first paper of this series (YOUNG 1966).

The Additive × Additive (E) Model

The genetic value of a pair of loci was assumed to be the product of their respective values. In particular, assuming the additive values of *AA*, *Aa* and *aa* to be $2\sqrt{2}$, $\sqrt{2}$ and 0, then for two adjacent loci the genetic values for the different genotypes were:

	AA	*Aa*	*aa*
BB	8	4	0
Bb	4	2	0
bb	0	0	0

The present model differed from GILL's (1965b) conditional model in that only a single peak (*AABB*) of genetic value was assumed. The ten loci were assumed to form five adjacent interacting units, with no interaction between nonadjacent

loci. Thus loci 1 and 2, 3 and 4, were assumed to form two units, and no interaction was assumed for loci 1 and 3 or 2 and 4. The programme therefore simulated only a fraction of all possible 2-factor A × A interactions. This may be partly compensated by the higher scale (e.g., $AABB = 8$, $AaBB = 4$, etc.) used, compared with A(2, 1, 0) and D(2, 2, 0). The genetic value of an individual was calculated by summing the genetic values of the five pairs of interacting loci.

Replicated runs. Examination of the results of the repeated runs under identical parameters, but different random sequences, showed that the agreement between runs for E were as good as those for A and D. Under high selection pressure (high heritability and selection intensity) the agreements were excellent, while under low selection pressure the runs agreed not quite as well. Tightness of linkage did not appear to effect the results. In Figure 1A and B two extreme cases are shown as illustration. It may be concluded that the effect of genetic drift was unimportant in this analysis.

(A) $I = 80\%$, $r = 0.5$. (B) $I = 10\%$, $r = 0.5$.

Predictive value of h^2. The present results showed that the prediction of long term genetic advance, assuming constant heritability, again proved to be of limited

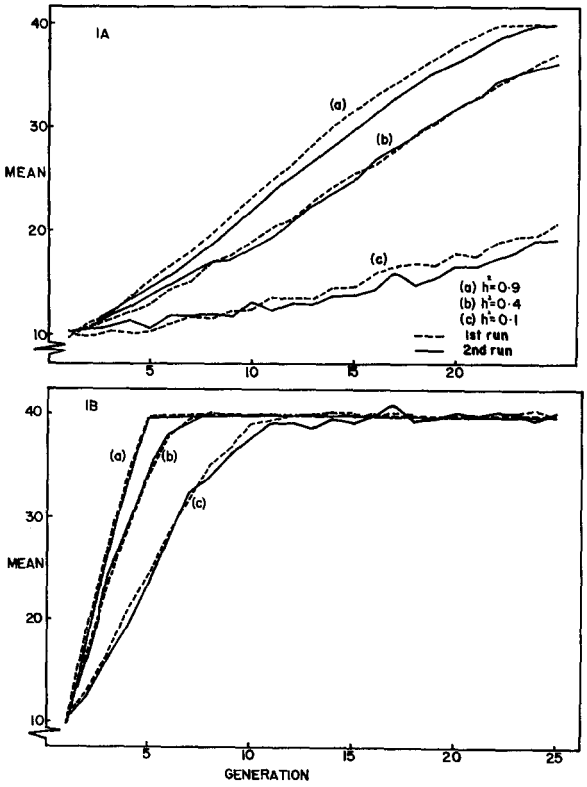

FIGURE 1.—Additive × additive model: Replicate runs of six populations with the same recombination probability (r) and different initial heritability (h^2) under different intensities of selection (I).

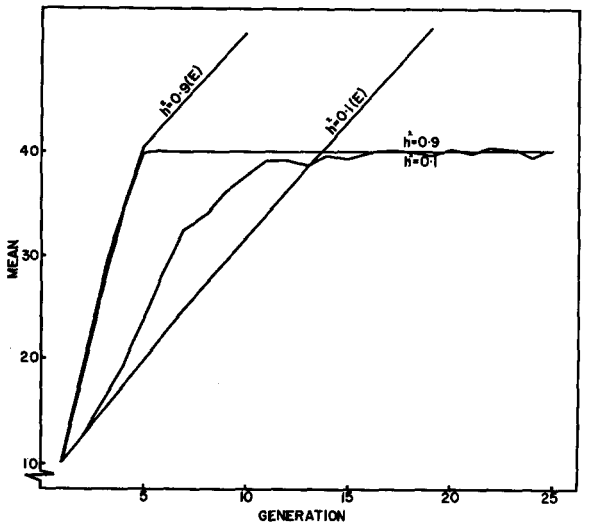

FIGURE 2.—Additive × additive model: Comparisons between realised and expected genetic advance with $I = 10\%$, $r = 0.5$, and two levels of h^2 (0.9 and 0.1), h^2 being assumed constant throughout.

value. Figure 2 illustrates this for two extreme situations when $h^2 = 0.9$ and $h^2 = 0.1$. Under high selection pressure the constant h^2 predicts gains reasonably well for 3 or 4 generations, but under low pressure the predictions were inaccurate after 1 or 2 generations.

Predictions of advance based on h^2 values calculated in each generation were also made, comparisons between predicted and realised gains being shown in Figures 3A, B, C and D. When selection intensity was high ($I = 0.10$), agreement was good for the 4 to 5 initial generations of selection for all levels of h^2 and r, but thereafter predictions tended to give underestimates (detailed data not shown). In later generations predicted gains reached a plateau at values below the realised figures. Similar results were obtained when $I = 50\%$ (Figures 3A, B). The agreement in this case was again fairly close, and a lower h^2 did not lead to greater discrepancy. Again, however, the predictions tended to give underestimates. When selection intensity was low (Figures 3C, D) agreement was only good when h^2 was high or intermediate. When h^2 and selection intensity were both low, agreement was poor, and in these circumstances predictions were in excess of the actual gains over 30 generations of selection. Under moderate or high selection pressure the populations all reached the maximum expected value, indicating that there was no loss of favourable alleles during selection.

Changes in additive genetic variances. A characteristic of the present results was the increase in σ_A^2 for several generations of selection in all populations. (Figures 4A, B, C). The reason for this has been investigated and will be discussed in a later section of this paper. Under high selection pressure (Figure 4A) σ_A^2 showed a small increase for two generations, followed by a rapid decrease; the nonadditive genetic variance declined from the start and both types of variance

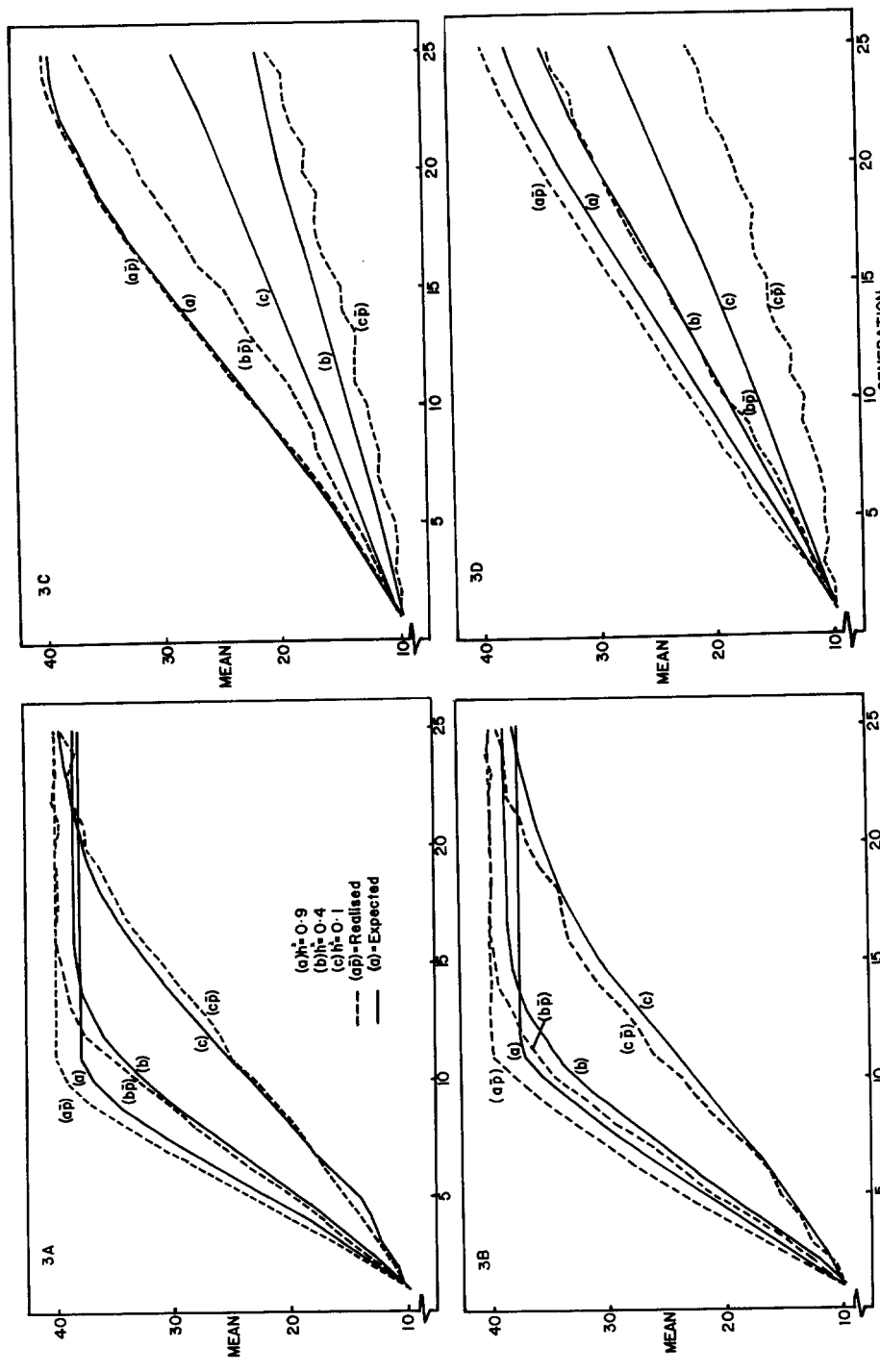

FIGURE 3.—Additive × additive model: Comparisons between realised and expected genetic advance under two selection intensities (I) for population with different initial heritabilities (h^2) and recombination probabilities (r), but with h^2 recalculated each generation.
(A) $I = 50\%, r = 0.5$. (B) $I = 50\%, r = 0.05$. (C) $I = 80\%, r = 0.5$. (D) $I = 80\%, r = 0.05$.

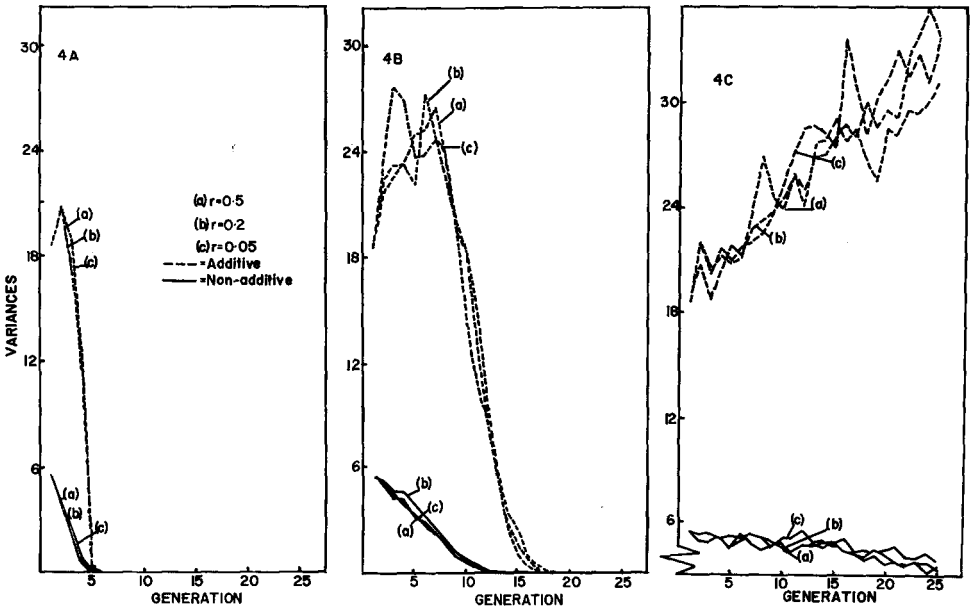

FIGURE 4.—Additive × additive model: Changes in the additive and nonadditive genetic variances under different selection intensities (I) for populations with different initial heritabilities (h^2) and recombination probabilities (r).

(A) $I = 10\%$, $h^2 = 0.9$. (B) $I = 50\%$, $h^2 = 0.4$. (C) $I = 80\%$, $h^2 = 0.1$.

vanished after 4 generations. Under intermediate selection pressure (Figure 4B) the additive genetic variance increased for 7 to 8 generations then showed a rapid decline and was gone by 17 generations; the nonadditive variance declined from the start and was gone by 12 generations. Under low selection pressure (Figure 4C) the additive genetic variance increased rapidly even up to 25 generations; the nonadditive variance showed a general slow decline, but did not entirely disappear.

An interesting feature of the changes in genetic variance was that when selection pressures were mild, tight linkage tended to inhibit the increase in σ_A^2 (Figure 4B, line C). This may be the result of the initial gene frequencies assumed for each pair of loci. As will be discussed later, when two interacting loci are equal in gene frequency the additive genetic variance is at a local minimum, hence tight linkage would tend to prevent the attainment of optimal combinations of frequencies for higher σ_A^2. The effect of linkage on changes in variance was not apparent when selection pressure was high (Figure 4A).

Figures 4A, B and C represent only a fraction of the results, but they are typical examples and there seems little point in presenting extensive figures in great detail.

Mixed Models

As mentioned previously, mixed models of A + D, A + E, D + E and A + D + E were also used. Owing to the enormous amount of data available, it is uninter-

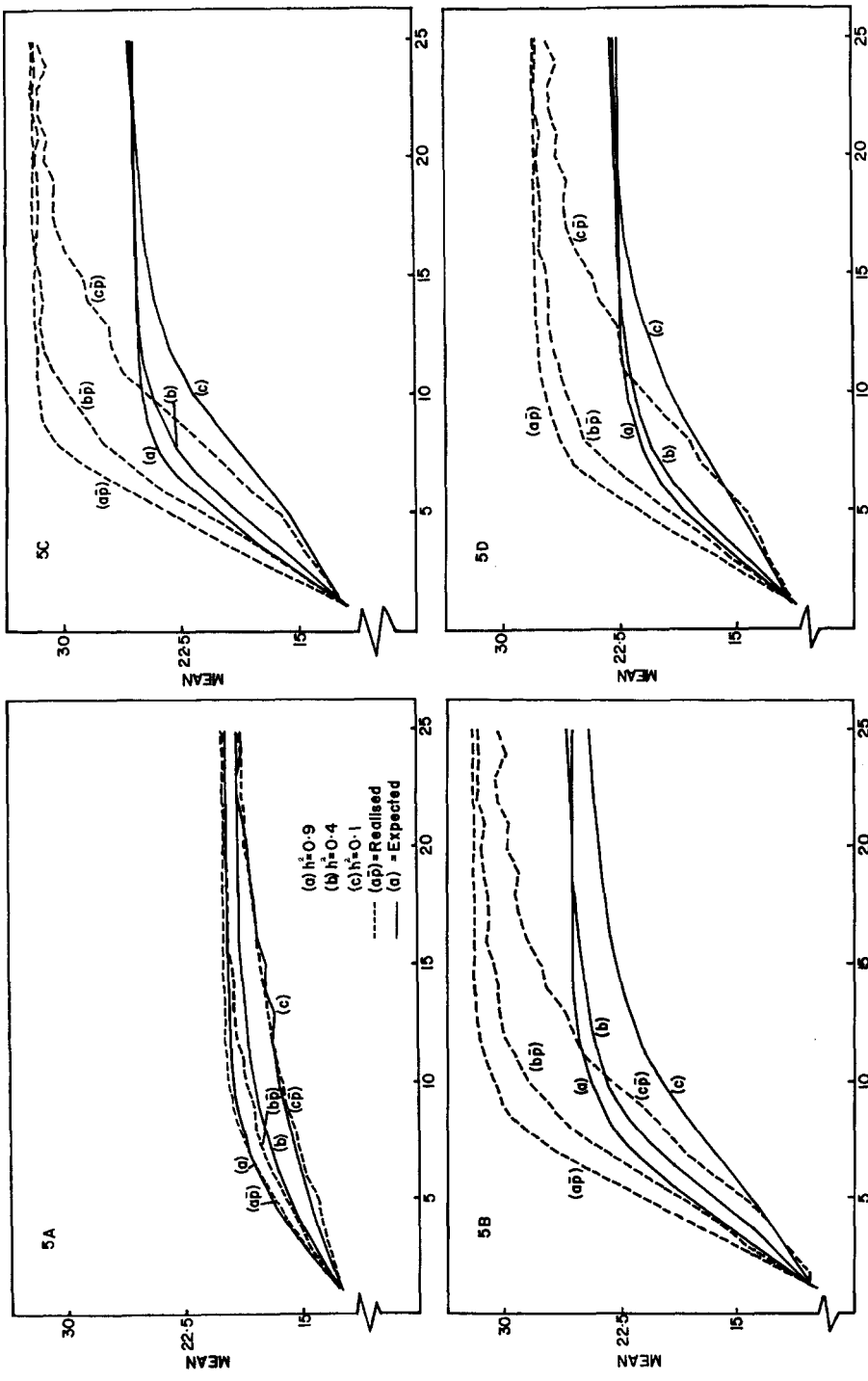

FIGURE 5.—Mixed models: Comparisons between realised and expected genetic advance in four mixed models, with $I = 50\%$, $r = 0.5$. (A) A+D model. (B) A+E model. (C) D+E model. (D) A+D+E model.

esting to present detailed descriptions of results obtained under different sets of circumstances. In this section, therefore, results will be very briefly discussed, while a summary for all models will be given in DISCUSSION.

The A + D model. Five additive and five dominance loci were assumed. The genetic values assumed for A and D were given in the first paper. Agreement between expected and realised gains was close, even under low selection intensity and low heritability. Figure 5A shows a typical set of results when $I = 0.50$ and $r = 0.50$. The agreement obtained under the present assumption was as good as for the additive model, and there was again no evidence of any loss of favourable alleles nor of any appreciable effect of linkage on genetic progress. The curves for the decay of the additive genetic variance were of the constantly decreasing type and were again mainly influenced by both h^2 and I. The nonadditive variance in most cases persisted in the population for many generations. A typical set of results is shown in Figure 6A for $I = 50\%$ and $h^2 = 0.4$ with different linkage values.

The A + E model. Four additive and three pairs of A × A loci were assumed. A most striking feature of the results was consistent under-estimation by the predicted gains when selection pressures were high or intermediate (Figure 5B), but when selection pressures were low the situation was reversed (data not shown here). Discrepancies between the predicted and the realised gains were often appreciable; under high selection pressure the mean error of the prediction amounted to something like 25% of the total advance, while under low selection pressure the mean errors were about 5% of the range. Disagreement was evident even in the early generations of selection, particularly when selection pressure was high or intermediate. The cumulative effect of the lower predicted gains resulted in large differences in the realised and predicted plateaus after the exhaustion of genetic variances.

Changes in the additive and nonadditive variances (Figure 6B) were again functions of h^2 and I, and both variances showed the characteristic rise for a few generations, with subsequent decline. The reason for this will be discussed later.

The D + E model. Four dominance loci and three pairs of A × A loci were assumed. The results are similar to those for the A + E model. Figures 5C and 6C are typical results. Under high selection pressure the mean error of prediction for each population was even higher than that for the A + E model (see Table 1), but under low pressure the predictions were more accurate. The pictures of decay in genetic variance were again similar to those for the A + E model, except that the nonadditive variance survived much longer. The similarity of the results between the A + E and D + E models was no doubt due to the inclusion of the A × A loci, as the scales used for these were higher than those for the A and D models.

The A + D + E model. Three additive, three dominance and two pairs of A × A loci were assumed. The influence of the E loci was still evident (Figures 5D, 6D). Predictions of genetic gains in most cases were again underestimates, and the discrepancies were more serious when selection pressures were intermediate or high. The additive and the non-additive genetic variances vanished quickly under

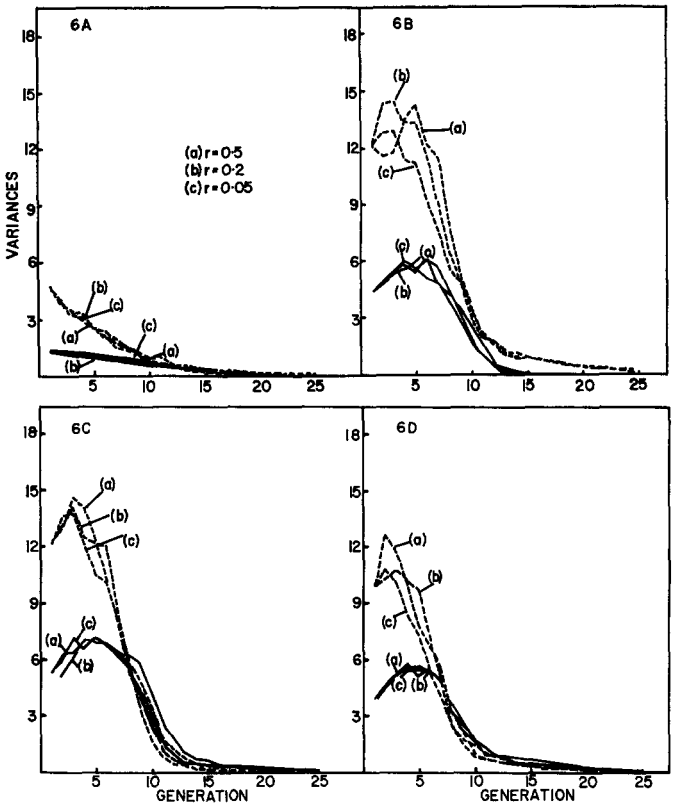

FIGURE 6.—Mixed models: Changes in the additive and nonadditive genetic variances with $l = 50\%$, $h^2 = 0.4$.
(A) A+D model. (B) A+E model. (C) D+E model. (D) A+D+E model.

extremely high selection pressure, but under low pressure both variances showed first a slight increase and then a very gradual reduction of σ_A^2 but a gradual increase in the nonadditive variance.

Comparison Between Models

It seems worthwhile to point out some general characteristics of the results for different models. With all genes additive, predictions of genetic gains were always accurate, but with genes showing dominance predictions were less accurate when selection pressures were high. With genes showing the A × A interaction, genetic gains were always underestimated when selection pressures were high or intermediate. The predictive ability of h^2 using the mixed models varied with the models concerned; with the A + D model the predictions were almost as accurate as those for A, while predictions tended to be inaccurate in any model involving epistatic loci. The results of the predictions for all seven models are summarised in Table 1. The mean differences for the first 12 generations were calculated as the predicted minus the realised gain, so that a negative sign indicates that the

TABLE 1

Mean differences between estimated and realised genetic advances (Calculated as estimated minus realised)

	Selection intensity and recombination probability																	
	$I=80\%$						$I=50\%$						$I=20\%$					
	$r=0.50$		$r=0.20$		$r=0.05$		$r=0.50$		$r=0.20$		$r=0.05$		$r=0.50$		$r=0.20$		$r=0.05$	
	Mean Diff.	Percent Mean/ Range	Mean Diff.	Percent Mean/ Range	Mean Diff.	Percent Mean/ Range	Mean Diff.	Percent Mean/ Range	Mean Diff.	Percent Mean/ Range	Mean Diff.	Percent Mean/ Range	Mean Diff.	Percent Mean/ Range	Mean Diff.	Percent Mean/ Range	Mean Diff.	Percent Mean/ Range
Initial $h2=0.9$																		
A	0.05	0.54	0.01	0.12	0.01	0.12	0.09	0.89	0.04	0.40	—0.12	—1.22	0.19	1.87	0.22	2.17	0.16	1.59
D	—0.06	—1.30	0.18	3.68	—0.14	—2.78	0.21	4.14	0.29	5.80	0.27	5.40	1.67	33.36	1.70	34.10	1.65	32.96
E	0.01	0.03	—0.24	—0.80	—0.84	—2.82	—1.58	—5.26	—1.60	—5.35	—2.01	—6.71	—3.32	—11.05	—3.54	—11.82	—3.84	—12.79
A+E	—1.12	—5.07	—1.72	—7.84	—1.94	—8.80	—4.35	—19.76	—4.74	—21.54	—5.20	—23.63	—5.20	—23.66	—6.09	—27.68	—5.64	—25.62
D+E	—1.90	—9.48	—1.96	—9.81	—2.24	—11.19	—4.68	—23.42	—4.64	—23.21	—5.38	—26.89	—5.73	—28.66	—5.67	—28.35	—5.98	—29.92
A+D	0.10	1.32	0.10	1.36	—0.14	—1.89	—0.06	0.83	—0.20	—2.69	0.17	2.32	0.19	2.52	0.11	1.48	0.20	2.64
A+D+E	—1.92	—11.62	—2.10	—12.73	—2.15	—13.03	—3.89	—23.56	—3.98	—24.11	—4.38	—26.52	—5.07	—30.74	—4.63	—28.05	—4.55	—27.59
Initial $h2=0.4$																		
A	—0.05	—0.52	—0.16	—1.62	—0.02	—0.18	0.04	—0.43	—0.10	—0.98	—0.20	—2.01	0.40	4.03	0.01	0.07	0.12	1.18
D	—0.24	—4.76	—0.17	—3.34	—0.31	—6.20	0.08	1.62	0.14	2.90	—0.01	—0.20	1.24	24.80	0.77	15.44	0.68	13.52
E	—0.34	—11.15	—0.25	—8.82	0.14	0.47	—0.75	—2.50	—0.81	—2.71	—1.20	—4.01	—2.33	—7.77	—3.23	—10.76	—2.72	—9.06
A+E	—0.91	—4.12	—1.65	—7.48	—0.95	—4.30	—3.59	—16.33	—3.82	—17.37	—4.53	—20.57	—5.20	—23.63	—5.86	—26.63	—5.33	—24.21
D+E	—1.04	—5.20	—1.30	—6.51	—0.97	—4.87	—3.80	—18.98	—3.89	—19.46	—3.94	—19.71	—5.11	—25.54	—5.06	—25.30	—4.93	—24.66
A+D	—0.04	—0.56	—0.02	—0.20	—0.20	—2.72	—0.41	—5.48	—0.20	—2.73	—0.16	—2.16	0.31	4.19	—0.04	—0.49	0.20	2.73
A+D+E	—1.00	—6.07	—1.09	—6.62	—0.92	—5.59	—3.06	—18.53	—3.48	—21.07	—3.51	—21.28	—4.34	—26.28	—4.13	—25.02	—4.88	—29.60
Initial $h2=0.1$																		
A	—0.66	6.56	—0.06	—0.57	0.41	4.12	0.22	2.16	0.02	0.25	—0.06	—0.61	0.56	5.59	0.20	2.02	—0.28	—2.82
D	—0.07	—1.46	—0.18	—3.56	—0.04	—0.78	0.35	7.04	—0.07	—1.44	—0.01	—0.16	1.00	20.04	0.60	11.98	0.74	14.76
E	3.16	10.53	2.70	9.01	2.62	8.75	0.55	1.84	—0.58	—1.94	—0.76	—2.55	—1.69	—5.64	—1.21	—4.93	—1.93	—6.42
A+E	1.95	8.88	0.94	4.25	1.11	5.03	—1.94	—8.83	—1.28	—5.84	—1.96	—8.90	—3.32	—15.08	—4.23	—19.21	—3.72	—16.90
D+E	0.50	2.52	0.61	3.04	0.37	1.87	—1.84	—9.20	—1.37	—6.86	—1.67	—8.36	—3.53	—17.64	—4.81	—24.05	—2.97	—14.84
A+D	—0.06	—0.87	0.11	—1.44	—0.13	—1.77	0.23	3.09	0.12	1.57	0.05	0.68	0.32	4.31	0.42	5.64	0.43	5.69
A+D+E	0.62	3.79	0.08	0.46	0.62	3.79	—0.75	—4.54	—1.74	—10.54	—1.76	—10.65	—2.27	—13.78	—3.29	—19.93	—2.61	—15.83

* A=under the additive model; D=under the dominance model; E=under the epistatic model; A+E, D+E, A+D, A+D+E= mixed models.

predicted gain gave an under-estimate. Also, different models have different ranges of genetic advance; for example, under the A model the mean increased from 10 to 20 units, while under the E model the mean started at 10 units and reached a plateau at 40 units. To facilitate comparisons between models, mean differences in each model were also expressed as percentages of the total range of advance. The variances of the difference for the first 12 generations are shown in Table 2. From Tables 1 and 2 it is evident that predictions with E were more erratic than with A or D, although under high selection pressure of means of errors of prediction for D were higher. It is interesting to note that means and variances of errors of prediction were almost always higher under mixed models involving epistasis. In such models, although only a portion of the total number of loci were assumed to be interacting, predictions of gains were much more erratic than with pure epistasis.

Among results obtained for mixed models which assumed two types of gene effects (A + D, A + E, and D + E), predictions assuming the D + E effects were in general less accurate. When some of the loci in the D + E model were replaced by additive loci (A + D + E model) there was in general an increase in the size (on a percentage basis) of errors of prediction. This is unexpected as it seems

TABLE 2

Variance of differences between estimated and realised genetic advances

	I=80%			I=50%			I=10%		
	r=0.50	r=0.20	r=0.05	r=0.50	r=0.20	r=0.05	r=0.50	r=0.20	r=0.05
Initial $h^2 = 0.9$									
A	0.001	0.002	0.006	0.003	0.004	0.008	0.049	0.016	0.041
D	0.002	0.009	0.006	0.011	0.018	0.017	0.296	0.429	0.304
E	0.055	0.057	0.225	0.401	0.181	0.348	0.245	0.299	0.488
A+E	0.892	1.140	1.803	4.863	5.906	6.385	1.468	2.100	1.921
D+E	1.687	1.395	1.918	4.800	5.007	7.311	1.679	1.952	2.147
A+D	0.004	0.004	0.009	0.039	0.050	0.045	0.066	0.055	0.012
A+D+E	1.819	1.902	1.961	3.219	4.031	3.787	1.190	0.955	0.895
Initial $h^2 = 0.4$									
A	0.023	0.004	0.016	0.011	0.007	0.015	0.131	0.016	0.142
D	0.017	0.012	0.018	0.027	0.015	0.013	0.310	0.192	0.135
E	3.161	0.028	0.107	0.170	0.031	0.161	0.570	0.359	0.486
A+E	0.161	0.744	0.370	2.887	9.256	5.484	1.141	1.802	1.922
D+E	0.272	0.512	0.362	3.958	5.029	4.837	1.863	1.831	1.272
A+D	0.024	0.008	0.013	0.044	0.025	0.024	0.034	0.009	0.053
A+D+E	0.738	0.569	0.508	2.548	3.105	3.084	0.787	0.769	0.851
Initial $h^2 = 0.1$									
A	0.038	0.061	0.047	0.020	0.036	0.033	0.042	0.090	0.088
D	0.070	0.030	0.046	0.040	0.036	0.038	0.084	0.152	0.181
E	1.958	2.815	1.747	0.175	0.127	0.469	0.918	0.398	0.220
A+E	0.555	0.591	0.172	2.825	0.712	2.269	3.766	1.964	1.755
D+E	0.049	0.225	0.140	1.892	0.873	2.265	3.669	2.550	1.438
A+D	0.050	0.034	0.030	0.025	0.048	0.036	0.051	0.088	0.066
A+D+E	0.074	0.145	0.095	1.001	1.598	1.973	2.322	1.547	0.814

reasonable to expect an increase in the precision of prediction when some additive loci were included in the D + E model. In view of the above evidence it may be concluded that the prediction of genetic advance by the value of h^2 becomes less accurate as the genetic model, involving some epistasis, becomes more complex.

Half lives and full lives of σ_A^2 under selection for all models have also been calculated. The half life of σ_A^2 for E was appreciably longer than for A and D under the same selection pressures. This is expected, as with E there was always an increase in σ_A^2 following initial generations of selection, while with A and D σ_A^2 could only decrease.

However, even with E the half-life of σ_A^2 could be as low as 4 to 5 generations, if the selection pressure was high. Under low or medium selection pressure the half-life could be longer than 30 generations. Results for the mixed models were much influenced by the presence or absence of epistatic loci. Thus with A + D the lengths of half-life were about intermediate between those for A and D. The inclusion of any epistatic loci always led to a longer half-life of σ_A^2 in all mixed models. Table 4 presents results for A + D + E; data for other mixed models are not presented.

From the results presented in Tables 1 to 4 it can be seen that tightness of linkage has no marked effect on the precision of prediction of genetic gain nor on the rate of decay in σ_A^2. There was a suggestion, however, that when h^2 and selection intensity were both low, tight linkage tended to increase the half-life of σ_A^2 slightly.

DISCUSSION

The increase in σ_A^2 under selection with the A × A model indicated that the maximum value of σ_A^2 occured at a gene frequency other than that assumed at the beginning of selection ($q = 0.5$ for all loci). That this is so may be seen from the following consideration.

TABLE 3

Half-life and full-life of the additive genetic variance in different populations under the additive × additive model

	I=80%			I=50%			I=10%		
	r=0.50	r=0.20	r=0.05	r=0.50	r=0.20	r=0.05	r=0.50	r=0.20	r=0.05
Initial $h^2 = 0.9$									
H	19.5	18.5	20.6	7.8	8.0	8.2	3.3	3.2	3.2
F	24	23	26	10	11	11	5	4	5
Initial $h^2 = 0.4$									
H	23.9	24.5	27.1	10.9	11.1	10.7	4.8	4.3	4.7
F	>30	>30	>30	17	17	19	9	9	9
Initial $h^2 = 0.1$									
H	>30	>30	>30	19.1	18.5	20.0	8.6	9.0	8.5
F	..	..	..	>30	>30	>30	16	17	17

* Half-life (H) = number of generations of selection required to reduce σ^2_A *to* one half of its initial value.
Full-life (F) = number of generations of selection required to reduce σ^2_A to zero.

TABLE 4

Half life and full life of the additive genetic variance in different populations under the additive + Dominance + Epistatic model

	I=80%			I=50%			I=10%		
	r=0.50	r=0.20	r=0.05	r=0.50	r=0.20	r=0.05	r=0.50	r=0.20	r=0.05
Initial $h^2 = 0.9$									
H	10.7	10.9	7.6	4.2	4.2	3.6	1.4	1.6	1.5
F	>30	29	>30	14	14	19	12	12	17
Initial $h^2 = 0.4$									
H	14.8	14.9	16.0	6.2	5.9	5.3	2.5	2.7	2.4
F	>30	>30	>30	>30	27	>30	22	22	18
Initial $h^2 = 0.1$									
H	28.9	27.5	>30	12.2	11.1	11.4	5.1	5.3	4.8
F	>30	>30	. .	>30	>30	>30	>30	>30	>30

Consider two loci X and Y with two alternative alleles in each locus, A and a for locus X, B and b for locus Y. The frequency of A is p, and of a is q $(p + q = 1)$, the frequency of B being r and of b, t $(r + t = 1)$. If loci X and Y were showing the A × A interaction, with genetic values of $AABB = 8$, $AaBB = 4$ etc. as used in the present study, it can be shown that when X and Y are not linked, the additive genetic variance of the population is

$$\sigma_A^2 = \frac{32\, p^2 r^2 (q+t)^2}{pq + rt} .$$

The stationary value of σ_A^2 for values of p and r can be investigated by calculating $\partial \sigma_A^2/\partial p = 0$ and $\partial \sigma_A^2/\partial r = 0$ and solving the two simultaneous equations. The results of partial differentiations were

$$2(pq + rt)(1 - 2p + t) - p(q + t)(1 - 2p) = 0 \qquad (1)$$
$$2(rt + pq)(1 - 2r + q) - r(t + q)(1 - 2r) = 0 \qquad (2)$$

The equations have a solution when $p = r$. When $p = r$

$$\sigma_A^2 = 64\, p^3 q,$$

which turns out to be an equation for minimum values of σ_A^2 for various p values. From among the minimum values the maximum is reached when $p = 0.75$. In this case each pair of loci will contribute 6.75 units to the additive genetic variance and the total σ_A^2 at the maximum-minimum value will be 33.75. Since gene frequencies for each pair of loci were set at $p = r = 0.5$ at the beginning of selection, it is therefore not surprising that there was an increase in σ_A^2 after a few generations of selection in each population.

A point worthy of note is that when selection presure was high, the increase in σ_A^2 under selection was less than when pressure was low; under high pressure the peaks of σ_A^2 reached levels much lower than the maximum-minimum value of 33.75, while under low pressure the peaks of σ_A^2 often exceeded this value. The results seem intuitively reasonable as high selection pressure would tend to push

the frequencies of the favoured genes at a greater speed towards fixation, so that there was insufficient time available for the formation of an optimal combination of gene frequencies for higher σ_A^2. This situation would be further enhanced if the gene frequencies for the loci were set at the minimum condition of $p = r$, as in the present study. Conversely at low selection pressure, relatively more time was available for the formation of favourable combinations of gene frequencies by crossing over.

The increase in σ_A^2 in the initial generations of selection for A × A, compared with the consistent decrease in the same variance for A and D, plus the nonlinear changes in σ_A^2 with selection shown in this study, support two obvious conclusions which have been much discussed but for which there has been little previous data: (1) There is no reason to expect similar changes in genetic parameters for any two traits of the same organism, under similar selection pressure. (2) It is not surprising to find a difference in rate of change in genetic parameters and a difference in rate of gain for the same character in two populations under similar selection pressure.

If we accept the above as reasonable then the desirability of estimating genetic parameters as often as possible during selection cannot be denied.

The results also show that h^2 is a relatively poor predictor of genetic gain when genes are epistatic or when some of the genes involved show epistatic interaction, even though the epistatic effect assumed here was a relatively simple one. One could imagine under more complex situations, such as the double peaked conditional A × A used by GILL (1965a) or with A × D or D × D models, the predictive ability of h^2 might be even poorer. It is well established in quantitative genetics that when h^2 is low or when mass selection produces no appreciable advance, then greater genetic gain might be obtained by using techniques such as family selection, crossing of inbred lines and so on. In view of the present conclusions from the A × A and mixed models, it seems worthwhile to propose that when accurate estimates of h^2 have failed to predict gains adequately then it may also be worthwhile to consider breeding methods for the exploitation of the non-additive genetic variations for greater rate of gain, without waiting for a plateau to be reached.

With the present population size of 1000 individuals, genetic drift was found to be unimportant in the results of simulated selection under various genetic models. For the same population tightness of linkage, at least for the levels of recombination probabilities assumed, had no effect on selection limits and had a negligible effect on the rate of change in genetic variances. These findings are in contrast to the results of earlier studies by FRASER (1957), MARTIN and COCKERHAM (1960) and GILL (1965) in small populations of 20 to 40 individuals. In designing selection experiments, it would be valuable to have some knowledge of the minimum size of population in which both linkage and drift would be expected to have only negligible effects on selection results. Further investigation in this area, using the high speed computer, seems to be warranted.

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SUMMARY

Genetic advances under truncation selection were simulated. It was assumed that the character under selection was controlled by ten loci, the size of each unselected population being 1000 individuals per generation. Different levels of selection intensities, initial heritabilities and recombination probabilities were incorporated in seven genetic models.—In an earlier paper the results for the additive (A) and dominance (D) models were reported. In this paper the results obtained assuming an additive × additive epistatic (E) model are discussed together with those for the A + D, A + E, D + E and A + D + E mixed models.—With the E model, predictions of genetic gains were underestimated when selection pressures were high or intermediate. The predictive ability of h^2 when mixed models were used varied with the model under consideration: for the A + D model predictions were almost as accurate as for the A model, but were erratic for any mixed models involving epistasis. Predictions tended to be more inaccurate as mixed models, involving some epistasis, became more complex.—With the E model, as well as with mixed models involving some epistasis, the additive genetic variance under the present conditions always increased after initial generations of selection. This is in contrast to results obtained for the A and D models under identical conditions. A direct consequence of this was the longer half and full lives of the additive genetic variance calculated for models involving epistatic loci.—Tightness of linkage had no appreciable effects on the predictive ability of h^2, the ultimate genetic advance and the decay of genetic variances.

LITERATURE CITED

FRASER, A. S., 1957 Simulation of genetic systems by automatic digital computers. I. Introduction. II. Effects of linkage on rates of advance under selection. Australian J. Biol. Sci. **10**: 484–491, 492–499. —— 1960 Simulation of genetic systems by automatic digital computers. VI. Epistasis. Australian J. Biol. Sci. **13**: 150–162.

GILL, J. L., 1965a A Monte Carlo evaluation of predicted selection response. Australian J. Biol. Sci. **18**: 999–1007. —— 1965b Effects of finite size on selection advance in simulated genetic populations. Australian J. Biol. Sci. **18**: 599–617.

GRIFFING, B., 1960 Theoretical consequences of truncation selection based on the individual phenotype. Australian J. Biol. Sci. **13**: 307–343.

LUSH, J. L., 1940 Intra-sire correlations and regressions of offspring on dam as a method of estimating heritability of characters. Am. Soc. Anim. Prod. Proc. 1940, 293–301.

MARTIN, F. G., JR., and C. C. COCKERHAM, 1960 High speed selection studies pp. 35–45. *Biometrical Genetics*. Edited by O. KEMPTHORNE. Pergamon Press, London.

YOUNG, S. S. Y., 1966 Computer simulation of directional selection in large populations. I. The programme, the additive and the dominance models. Genetics **53**: 189–205.