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Genetic Correlation and Response to Selection in Simulated Populations

I. Additive Model[1]

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Summary. Effects of truncation selection of a primary trait upon genetic correlation with a secondary trait were examined over 30 generations in genetic populations simulated by computer. Populations were 24 males and 24 females mated randomly with replacement; number of offspring was determined by intensity of selection. Each trait was controlled by 48 loci segregating independently, effects were equal at every locus, and gene frequency was arbitrarily set at 0.5 at each locus in the initial generation. All combinations of three genetic correlations, three intensities of selection, and three environmental variances were simulated. Gene action was additive. Genetic correlation was set by number of loci which affected both traits and was measured each generation as the product-moment correlation of genotypic values and estimated by two methods of combining phenotypic covariances between parent and offspring.

Genetic correlations in each offspring generation remained consistently near initial correlations for all environmental variances when fraction of offspring saved as parents was as large as one-half. When the fraction of offspring saved was as small as one-fifth, genetic correlations decreased but most rapidly with heritability high and after the 15th generation of selection. Truncation selection caused genetic correlation to decrease in those offspring selected to become parents of the next generation. Amount of reduction depended on heritability of the selected trait rather than on degree of truncation selection. Estimates of genetic correlation from phenotypic covariances between parent and offspring fluctuated markedly from real correlations in the small populations simulated.

Knowledge of genetic correlation among traits is necessary to predict response to selection of traits not directly selected and to combine measurements on different traits in selection indexes. There has not been enough study of genetic correlation and correlated response to selection to conclude about their behaviour under selection for questions such as to what extent correlations can be changed by selection, over how many generations correlated responses continue, or what is the total correlated response when the limit of selection is reached. This investigation was to examine how intensity of selection and environmental variation affect behaviour of genetic correlation.

The most important underlying cause of genetic correlation appears to be pleiotropy, a gene affects two or more traits; the segregating gene causes simultaneous variation in the traits. Quantitative aspects of genetic correlation were presented by HAZEL (1943), who developed a statistical technique to estimate genetic correlation from resemblance between relatives. REEVE (1955), ROBERTSON (1959), TALLIS (1959), VAN VLECK and HENDERSON (1961), and SCHEINBERG (1966) have considered methods to estimate the large sampling variance, but these usually apply under special circumstances. BROWN (1969) examined empirically sampling distributions of genetic correlation.

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Little is known about effects of selection on genetic correlation. LERNER (1958) presented a simple theoretical model suggesting that a genetic correlation between two traits would eventually become negative if selection were applied to both traits simultaneously. Only those alleles with opposite effects on the two traits would be left segregating. FRIARS *et al.* (1962) reported declining genetic correlations between traits under simultaneous selection for improvement in poultry over nine years. The authors suggested the additive portion of the genetic covariance may have decreased through selection.

Methods and Procedure

Experimental Design and Parameters Simulated

The major objective was to investigate how heritability and truncation selection of a primary trait affect behaviour of genetic correlation. The simplicity and applicability to quantitative genetics encouraged observing this by simulation. Factors and amounts allowing for a range of effects of each while containing the size of the experiment within reasonable bounds were:

1. Two traits X and Y with selection by upper or lower truncation on the phenotype of the individual for X alone. Y was not selected, but correlated response was observed.

2. Three degrees of genetic correlation, 0.25, 0.50, and 0.75, between X and Y in the initial generation of offspring.

3. Three intensities of selection, 80, 50, and 20 per cent of the offspring saved for breeding each generation.

4. Three environmental variances, $V(E)$, for X and Y relative to the expected additive genetic variance, $V(G_a)$, in the initial generation of offspring. Amounts were chosen such that $h^2 = V(G_a)/[V(G_a) + V(E)]$, heritability in the narrow sense, was equal to 0.1, 0.4, or 0.7.

Three levels of each of the four factors — genetic correlation, intensity of selection for X, and environmental variation of X and of Y — were considered in all combinations, and each combination or parameter set was replicated. Each replicate of these factors and levels provided 81 treatment combinations in a 3^4 factorial experiment.

The experiment was conducted separately for each of these two models of gene action:

a) Additive model in which contributions to the genotypic value were 2, 1, and 0 for ++, +−, and −− phases at each locus. Selection was for the desirable allele.

b) Model of complete dominance in which the contributions to the genotypic value were 2, 2, and 0 for the ++, +−, and −− phases at each locus. Selection was in both directions, upward for the dominant allele and downward for the recessive allele.

Results for the additive model only are presented in this paper; results for the model of complete dominance are in a separate communication.

Structure of population

Populations were bisexual, diploid, and X and Y were traits of both sexes. Size of population was related to number of parents rather than to number of offspring by limiting the number of parents each generation per treatment combination to 48, 24 males and 24 females. Selection intensity determined the number of offspring produced by these parents. To make the fraction saved (b) 0.8, 0.5, and 0.2 in each generation, 30, 48, and 120 males and females were produced giving 60, 96, and 240 offspring. Selection intensity was equal in the two sexes.

Selected parents were mated at random by sampling with replacement, and each mating produced one offspring, the sex of which was specified alternately. This procedure allowed for both full sibs and half sibs among the offspring in any generation. Each parameter set was continued for 30 generations to provide opportunity to observe a selection limit. Generations were nonoverlapping.

Each word in the storage module of the Control Data 3600 had a 51 bit structure allowing for expedient handling of a 48 bit data word with three parity bits. Magnetic core storage of 32,768 of these 48 bit words was available. For these reasons the number of loci affecting each of the two traits was 48 which meant that two 48 bit words could conveniently represent the genotype of each trait. Thus, four words were required to store the genotype of each individual.

No linkage was in the genetic structure, all loci were completely independent, and gene effects were equal at all loci. Further restrictions were no interallelic gene interactions and no interaction between genotype and environment. Gene frequency at each locus was arbitrarily set at 0.5 in the initial generation by simulating complete heterozygosity at each locus in the base population. For the additive model the genotypic value for each trait was $2n_1 + n_2$ where n_1 is the number of ++ phases and n_2 the number of +− phases in the genotype. With independent assortment and frequency of the + gene (q) the same at all loci, the expected genotypic mean was $2nq$ and expected genotypic variance was $2nq(1-q)$ where n is the number of loci affecting the trait. With 48 loci affecting each trait and $q = 0.5$, the expected genotypic mean and variance in the initial generation were 48 and 24.

In the first generation of offspring, environmental variances of 216, 36, and 10.3 were simulated relative to the expected additive genetic variance for heritabilities of 0.1, 0.4, and 0.7. The environmental component was independent of genotype and constant over generations. To allow study of effects of different environmental variances upon change in genetic parameters simulated, no attempt was made to restrict change in heritability over the 30 generations.

Simulation of genetic correlation

Genetic correlation was attributed solely to pleiotropy and expressed the extent to which the two traits under consideration were influenced by the same segregating genes. All genes affecting the two traits affected each one in the same direction, thus making a positive covariance. As 48 loci affected each of the two traits, the number of these 48 which were shared by the two traits determined genetic correlation. To produce genetic correlations of 0.25, 0.50, and 0.75, numbers of loci in common were set at 12, 24, and 36. Remaining loci of the 48 affected each trait independently. Genetic correlation was measured in each generation as the product-moment correlation of genotypic values $r_G = CovGxGy/[V(Gx) \cdot V(Gy)]^{1/2}$ where r_G is genetic correlation, $CovGxGy$ is covariance between genotypic values, and $V(Gx)$ and $V(Gy)$ are variances of genotypic values. Genetic correlation was also estimated each generation by the method proposed by HAZEL (1943) utilizing covariances between phenotypes of parent and offspring. Two variations of HAZEL's method were used to compare accuracy of methods. The two methods were:

a) $r_G = [(CovPxpPyo)\,(CovPypPxo)/(CovPxpPxo)\,(CovPypPyo)]^{1/2}$

b) $r_G = (CovPxpPyo + CovPypPxo)/$
$2\,[(CovPxpPxo)\,(CovPypPyo)]^{1/2}$

where Pxp and Pxo are phenotypic values of trait X in parent and offspring, and Pyp and Pyo are phenotypic values of trait Y in parent, offspring.

Mechanics of simulation

The first of several logical and separate blocks in simulation prescribed numerical constants for that particular parameter set. These constants included genetic correlation, number of genes shared by the two traits; intensity of selection, number of offspring of each sex to be produced each generation; environmental standard deviations required to produce the desired heritability of each trait; and number of replicate. Constants were changed after each parameter run until all combinations of parameter sets had been simulated.

The second part of the program generated 48 initial parents, 24 males and 24 females. To do this, four words of memory were assigned to each individual, the first two words representing the genotype of the individual for trait X and the second two words representing the genotype for trait Y. Of the 48 bits per word, B represented the number of bits which contained identical genes in the genotype of each trait, effectively simulating the required genetic correlation. For the first B bits of the first word of both traits X and Y, the same 1 or 0 was allocated with equal probability. For the remaining (48-B) bits 1 or 0 was allocated with equal probability independently for trait X and for trait Y. In this way, an array of 48 alleles affecting each trait was generated and the traits had B alleles of these in common. Corresponding alleles of the second word of each of the traits alternated those of the first word at every locus producing the individual completely heterozygous at every locus for both genotypes still with B alleles in common. This procedure was then repeated for 48 individuals, alternately male and female.

The third part of the program was concerned with producing the offspring generation from parent generation. Either 120, 48, or 30 offspring of each sex were produced each generation to provide 20, 50, or 80 percent of the offspring generation saved to maintain parent population size at 48.

The fourth stage in the program evaluated genotypes simply by summing 1's and 0's over all 48 loci for each trait X and Y.

Following genotypic evaluation an environmental contribution was added to the genotypic value to provide the phenotypic value for each trait. This environmental deviate was formed by the product of a constant for the desired environmental standard deviation and a standard normal deviate generated as described by GILL (1965). Thus, at this stage the 48 locus genotype together with genotypic and phenotypic values for each trait described each offspring. From these, each offspring generation could be characterized by gene frequency, genotypic and phenotypic mean and variance for each of the two traits; genotypic, environmental, and phenotypic covariances and correlations between traits; and heritability of each trait measured as the ratio of genotypic to phenotypic variance.

Parents of the next generation were selected by ranking offspring from high to low on phenotype of trait X and then retaining the top 24 individuals of each sex. After selection the same statistics were calculated for the selected group as for the unselected offspring with the exception of the two estimates of genetic correlation from parent-offspring covariances. After all statistics had been calculated, the next generation of offspring was produced from the 48 selected parents by random mating with replacement, and the cycle was repeated until 30 generations of offspring had been produced for that particular parameter set. The run was then replicated for that parameter set. After replication, one of the constants was changed, and parameter runs were continued for all 81 treatment combinations.

Results and Discussion

Variations between replicates were too small to justify detailed presentation of each replicate. All statistics presented graphically were averaged over two replicates. Statistics were calculated for each of 30 generations of selection in a given parameter set; however, only results for every fifth generation are presented.

Effect of selection on genetic correlation

In figures 1.1, 1.2, and 1.3 changes in genetic correlation measured as product-moment correlation of genotypic values are presented for the unselected offspring and for those offspring selected to be the parents of the next generation. Thus, the number of individuals upon which r_G is measured is always 48 in the selected group but varies in the unselected offspring with intensity of selection. The correlation includes 60 individuals when $b = 0.8$, 96 individuals when $b = 0.5$, and 240 individuals when $b = 0.2$.

Since environmental variance of trait Y, the unselected trait, had no effect on genetic correlation, r_G was averaged over the three amounts of environmental variance of Y and, as stated before, over two replicates. Thus, each point on the graphs represents six genetic correlations, each first transformed to z with the tables of FISHER (1958) and the resulting mean z reconverted to r_G.

Figure 1.1 shows changes in genetic correlation at each intensity of selection when environmental variance was large ($h_x^2 = 0.1$). Most noticeable is the consistency of the genetic correlation in the unselected offspring over the 30 generations of selection at all three intensities of selection and at all three

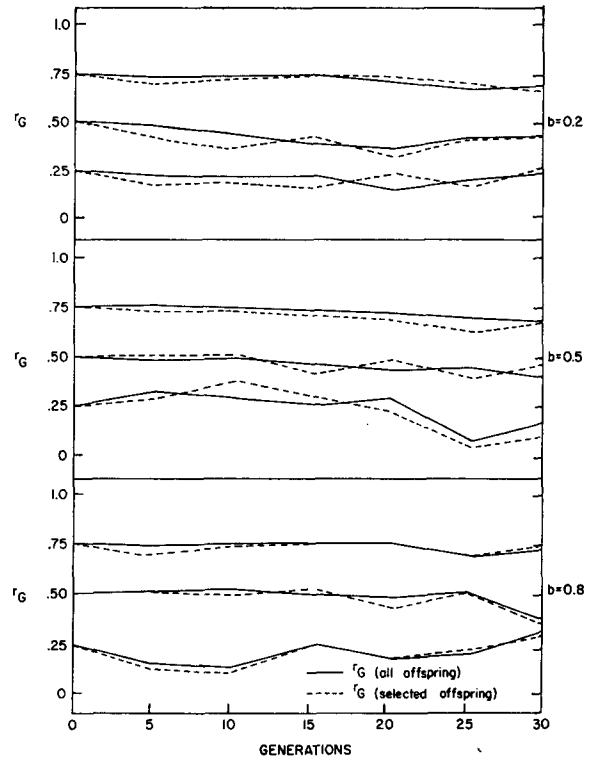

Fig. 1.1. Change in genetic correlation at three intensities of selection when $h_x^2 = 0.1$ (additive model)

genetic correlations of the initial generation. When initial genetic correlation was low ($r_G = 0.25$), genetic correlations were more erratic than at the two higher levels of correlation but did remain close to 0.25 over all 30 generations.

In Figure 1.2, intermediate environmental variance ($h_x^2 = 0.4$), genetic correlations again remained near those in the first generation when $b = 0.8$. Intermediate selection ($b = 0.5$), however, decreased correlation over the 30 generations. The trend was not so obvious at low correlation. At high selection intensity ($b = 0.2$) the trend of decreasing genetic correlation became even clearer when initial $r_G = 0.75$ or 0.5. The correlation did remain high for some time, however. For example, at the 20th generation of selection the genetic correlation, originally 0.75, was still almost 0.65. Again when initial genetic correlation was low ($r_G = 0.25$), change in the genetic correlation was more erratic and the trend not nearly so clear.

In Figure 1.3, where heritability was high, genetic correlation again remained at its initial level when $b = 0.8$. Anew the tendency for the correlation to decrease became distinct only after the 15th generation of selection. It was only when selection was intense ($b = 0.2$) and environmental variance was low ($h_x^2 = 0.7$) that genetic correlation declined rapidly. This decline occurred at all three genetic correlations but did not become extremely rapid until after the

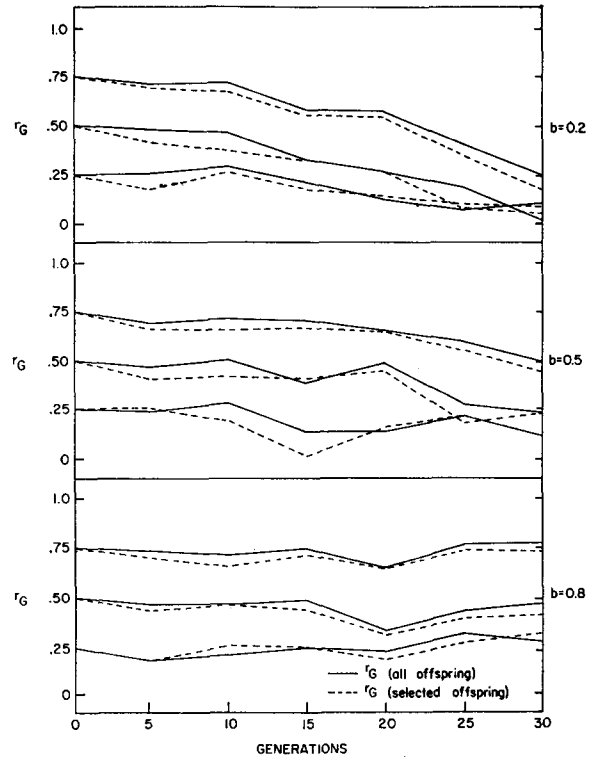

Fig. 1.2. Change in genetic correlation at three intensities of selection when $h_x^2 = 0.4$ (additive model)

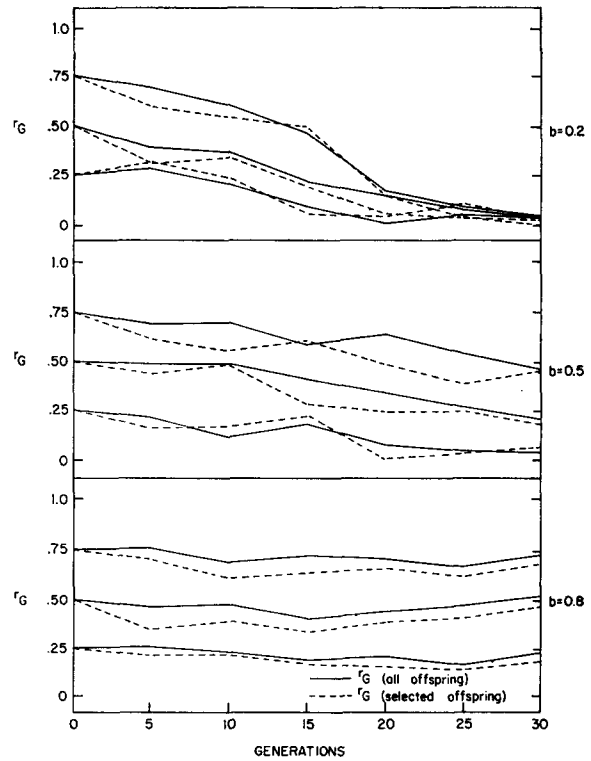

Fig. 1.3. Change in genetic correlation at three intensities of selection when $h_x^2 = 0.7$ (additive model)

15th generation of selection and did not reach zero until the 30th generation.

In general, most remarkable in the nine graphs is the consistency of the genetic correlation at all three environmental variances and when the fraction of offspring saved as parents was as high as one-half. Only when the fraction of offspring saved became as low as one-fifth ($b = 0.2$) was genetic correlation considerably affected, and then the effect became large only when heritability of the selected trait was high ($h_x^2 = 0.7$). This was clearly an interaction between selection and heritability, a rapid decrease in genetic correlation requiring both intense selection and high heritability. These results indicate that usual selection in animal species would have little effect upon genetic correlation unless heritability were very high.

Genetic correlation could remain high although genetic covariance between traits was decreasing if genetic variances of the two traits were decreasing proportionately. Examination of these components showed, in general, for mild selection initial genetic covariance and variances were maintained over the 30 generations of selection. With more intense selection, however, there was a distinct downward trend in genetic covariance; but because of an accompanied decrease in the genetic variance of the selected trait, the genetic correlation remained quite near its initial level. Only when both selection intensity and heritability were high was the correlation coefficient decreased markedly, and this decrease mostly came suddenly after the 15th generation of selection despite earlier rapid decreases in *CovGxGy* and *V(Gx)* and slower regular and stable changes in all three components during later generations (Fig. 2).

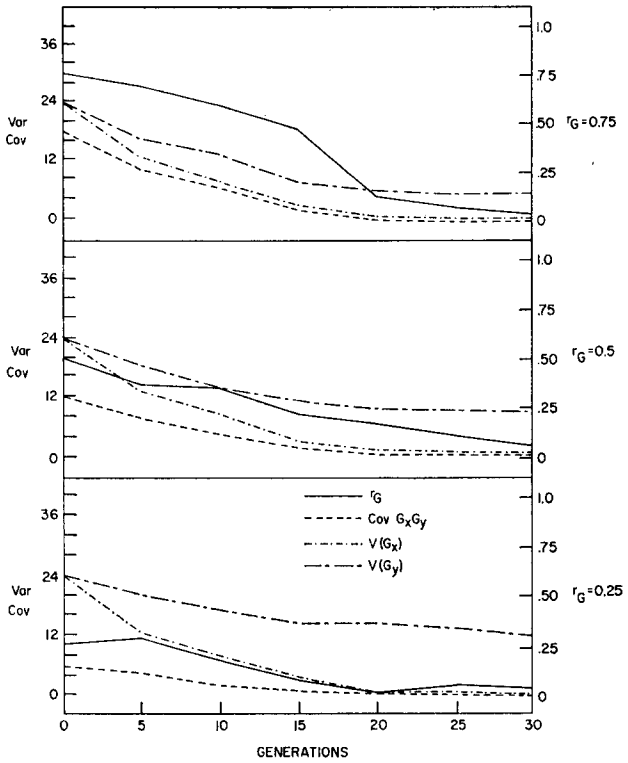

Fig. 2. Relationships between genotypic variances, covariance, and genetic correlation for three initial genetic correlations when $b = 0.2$ and $h_x^2 = 0.7$ (additive model)

Genetic correlation in the truncated distribution

Effect of linear truncation of one variable on the marginal distribution of a correlated variable has been discussed by AITKIN (1964) and by MANTEL (1966). In general, the conclusion was that phenotypic correlation observed within the sample of selected individuals will be lower than that observed within the unselected population. Whether the same would hold for genetic correlation was examined by measuring genetic correlation each generation in those offspring selected to be parents of the next generation. Phenotypic correlation is a function of genetic correlation, heritability, and also of any environmental correlation between traits. Thus, a reduction in phenotypic correlation between variables might not necessarily mean a reduction in genetic correlation.

Clearly truncation decreased genetic correlations from those of the unselected in Figures 1.1, 1.2, and 1.3. This decrease is apparently a function of heritability rather than of degree of truncation selection. Genetic correlations in the selected offspring became increasingly and more consistently less as environmental variance decreased relative to genotypic variance. There was little effect of amount of selection on this decrease. The results in Figure 1.3 correspond most closely with those expected for phenotypic correlation since expectation of phenotypic correlation approaches genetic correlation when heritabilities are high.

Genetic correlations from phenotypic covariances between parent and offspring

Genetic correlations estimated by two methods of combining covariances between phenotypes of parent and offspring were compared with product-moment correlations of genotypic values. Since number of observations upon which genetic correlation is estimated affects considerably precision of the estimate, that sample sizes were small and varied with amount of selection should be emphasized.

Results of both methods were extremely erratic and were almost impossible to interpret. Intense selection of parents could be expected to bias correlation, and extreme selection occurred when number of observations was largest. When selection was mild, number of observations was small, resulting in both cases in unreliable estimates. Most estimates fluctuated markedly from the true correlation. In fact, rare was the estimate within ± 0.2 of the correlation

of genotypic values. BROWN (1969) concluded that little information is available to estimate genetic correlation when pairs of parent-offspring are less than 200. In general, however, the correlation tended to be considerably underestimated, a tendency which increased with selection intensity. The complexity and magnitude of bias and sampling errors prevented any attempt to examine them further. Suffice it to say that it is unwise to estimate genetic correlations from parent-offspring covariances in an intensely selected population of sizes simulated in this study.

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