GENETIC CORRELATION AND RESPONSE TO SELECTION IN SIMULATED POPULATIONS

Thesis for the Degree of Ph. D.

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ROBERT JACK PARKER

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This is to certify that the

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Lowell D. McGilliard
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GENETIC CORRELATION AND RESPONSE TO SELECTION IN SIMULATED POPULATIONS

By

Robert Jack Parker

AN ABSTRACT OF A THESIS

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ABSTRACT

GENETIC CORRELATION AND RESPONSE TO SELECTION IN SIMULATED POPULATIONS

by Robert Jack Parker

The effects of truncation selection of a primary trait upon the genetic correlation and the correlated response in a secondary trait were examined. Genetic populations and the process of selection were simulated through the use of random numbers generated by a computer.

Selection was made for one of two quantitative traits, and the correlated response in the other trait was measured in each generation. The population was bisexual diploid and the traits were expressed in both sexes. The size of the population of parents was 48 in each generation and mating was random, the number of offspring produced being determined by the level 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.

Three degrees of genetic correlation, three levels of selection, and three levels of environmental variation were simulated. Two models of gene action, an additive model and a model of complete dominance, were considered. In the model of complete dominance, the experiment was carried out

separately for opposite directions of selection.

The genetic correlation was determined by the number of loci which affected both traits and was measured each generation as the product-moment correlation of genotypic values and by two methods utilizing phenotypic covariances between parent and offspring.

In the additive model the genetic correlation, measured as the correlation of genotypic values in each offspring generation, remained consistently near its initial level at all levels of environment when the fraction of offspring saved as parents was as high as one-half. When the fraction of offspring saved became as low as one-fifth, the genetic correlation decreased. A closer examination of the genetic correlation indicated that at low selection intensity the genetic covariance between the traits was maintained. With greater selection intensity, the genetic covariance decreased, but the genetic variances of the traits declined proportionately causing the genetic correlation to be maintained.

Truncation selection caused a decrease in the genetic correlation in those offspring selected to become parents of the next generation. The amount of reduction depended on the heritability of the selected trait rather than on the degree of truncation selection.

Estimates of genetic correlation obtained from phenotypic covariances between parent and offspring fluctuated markedly from the true correlation in the small populations simulated.

The correlated response of the unselected trait to selection of the primary trait agreed closely with response expected from theoretical considerations.

In the model of complete dominance, the change in the genetic correlation when selection was by upper truncation followed essentially the same pattern as in the additive model. When selection was by lower truncation, the behaviour under selection of the genetic correlation conformed to that for the additive model although the decrease in the correlation at high intensity of selection was more rapid. As in the additive model, truncation selection caused a decrease in the genetic correlation in the offspring selected to be parents whether selection was by upper or lower truncation. Estimates of genetic correlation computed from phenotypic covariances between parent and offspring were also poor in the model of complete dominance. The response of the genotypic mean of the unselected trait to selection of the primary trait in opposite directions was quite symmetrical for the first few generations but became distinctly asymmetrical in later generations. At low levels of selection the response was fairly linear but became distinctly curvilinear as the intensity of selection increased.

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The valuable suggestions contributed by Dr. John L. Gill are also gratefully appreciated.

I would like to acknowledge the friendly counsel of my fellow graduate students, Arthur Dayton and William Wunder, who helped make the period of graduate study more enjoyable and rewarding.

I am also grateful for the financial assistance provided by the Department of Dairy in the form of the C. E. Wilson Research Fellowship.

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"There is no more common error
than to assume that, because
prolonged and accurate mathematical
calculations have been made, the
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INTRODUCTION

The improvement of economic traits in livestock depends upon the effective use of genetic variation. Pertinent to this is a knowledge of the relationships among the traits including the genetic and environmental correlations among them. Knowledge of the genetic correlation among traits is necessary to predict the response to selection of traits not directly selected and to combine measurements on different traits in selection indexes to secure maximum improvement. Predictions of this type are valid only to the degree that the estimate of the genetic correlation is valid and to the extent that selection itself does not modify the genetic correlations.

There has not been enough study of genetic correlation and correlated response to selection to allow conclusions to be drawn about their behaviour under selection for questions such as to what extent the correlation can be changed by selection, over how many generations the correlated responses continue, or what is the total correlated response when the limit of selection is reached.

The introduction and rapid development of Monte Carlo methods in recent years has provided a tool for the study of population phenomena in a more detailed manner than has been possible with

either the techniques of mathematical genetics or laboratory studies with biological populations.

The concepts underlying the use of Monte Carlo techniques, the use of some mechanical device to create simulated observations, are not new and may be considered as old as probability and statistics. The Monte Carlo method came into use during the 1940's to identify statistical procedures for obtaining numerical estimates for problems in nuclear physics. With the introduction of high speed computers, interest in the theory and application of Monte Carlo techniques greatly increased.

The applicability of Monte Carlo procedures to quantitative genetics arises from inheritance having a relatively simple probabilistic basis, and Monte Carlo methods involve the simulation of probabilistic mechanisms. Thus, through intelligent simulation of these basic genetic mechanisms, additional insights into their consequences for various situations becomes possible. Yet, it should be stressed that the simulation has to be based on our present theory of biometrical genetics and the results obtained can only be studied in the light of existing theory. The procedures cannot be expected to increase our knowledge of the basic mechanisms themselves. The major contribution may well be to emphasize and clarify points which should have been recognized

previously but which have been overlooked or considered less important than they should have been.

This investigation was to examine the effects of the intensity of selection and the environmental variation upon the behaviour of the genetic correlation and upon the correlated response of traits not selected to selection.

REVIEW OF LITERATURE

A change in other traits not under selection when traits under selection are modified has been observed for some time. The statement by Darwin (1875) indicates that he had noted the importance of correlated variation: "Hence, if man goes on selecting, and thus augmenting, any peculiarity, he will almost certainly modify unintentionally other parts of the structure, owing to the mysterious laws of correlation." In the study of such correlated response the genetic correlation between the traits plays an important role in determining their pattern under selection.

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 it affects. Other possible causes of genetic correlation are usually considered to be minor or transient. For example, according to Lush (1948), linkage can be an important cause only in a population where either the coupling or repulsion phase of the double heterozygote is far more abundant than the other. Such a condition would persist for only a few generations after a cross because in a freely interbreeding population, the coupling

and repulsion phases of the double heterozygote tend rapidly to become equally frequent. For a second example, Lush suggests that an apparent genetic correlation could be caused by different intensities or different directions of selection in non-interbreeding sub-groups of a population. If the whole population were studied as a unit without regard to the sub-groups, the differences between groups could create a genetic correlation in the population, although there would be no genetic correlation within each sub-group considered separately.

The quantitative aspects of genetic correlation were presented by Hazel (1943), who developed a statistical technique to estimate genetic correlation based upon the fundamental formulations of biometrical genetics of Fisher and Wright. The technique of estimation was based on the resemblance between relatives similar to the method used in the estimation of heritability. However, instead of the components of variance of one trait, the components of covariance of the two traits were computed. In general, the more closely the animals are related, the smaller should be the sampling error of the estimate. Sufficient care is needed to avoid correlated environments of the individuals concerned. Estimates of genetic correlations obtained by covariance between relatives have not been precise, however, and are usually subject

to rather large sampling errors.

Reeve (1955) presented a method to estimate the sampling variance of the genetic correlation coefficient between two traits in large samples where the correlation is estimated from the four parent-offspring covariances for the two traits. The variance was expressed in terms of the heritabilities, genetic and phenotypic correlations between the two traits. The variance was the same whether the arithmetic mean or the geometric mean of the covariances involving both characters was used in calculating the genetic correlation.

Robertson (1959) developed a measure of the sampling variance where the genetic correlation is estimated from variance and covariance components for the two traits within and between groups of relatives. He presented formulae for the special case in which the two traits have the same heritability. Since the standard errors of the two heritabilities appear in the formulae, an experiment designed to minimize the sampling variance of an estimate of heritability should also have the optimum structure for the estimation of a genetic correlation. An attempt was made to suggest the form of the more general solution where the two traits have different heritabilities.

Using a different approach, Tallis (1959) presented a general solution which reduced to that presented by Robertson when the

two traits have equal heritabilities. The formula developed by Tallis holds for estimating the sampling variance of a genetic correlation estimated from an analysis of variance and covariance provided the estimate of heritability of neither trait is zero and the number of offspring per sire is constant. A general solution has also been described by Mode and Robinson (1959) for genetic correlations estimated from components of variance in a random model with equal sub-class numbers nested four ways.

Van Vleck and Henderson (1961) presented a procedure for obtaining empirical sampling estimates of genetic correlations obtained from parent-offspring analysis. Sampling variances of these estimates were then compared with the theoretical variances derived by Reeve (1955). They found that for sample sizes of 1,000 or more, the approximate formulae of Reeve for the variance in large samples agreed. For smaller sample sizes (500 or less) the approximations were not close unless the heritabilities of the traits were high. In fact, when the sample size was 100 or less, the approximations were very misleading. Van Vleck and Henderson concluded that for estimating genetic correlations, at least 1,000 sets of observations are needed to obtain reasonable estimates of the sampling variance. Even then the sampling variances may be too large for the estimates to be of use, especially if heritabilities of the traits are low. Heritability

plays a dominant role in determining the sampling variances of estimates of genetic correlation.

Scheinberg (1966) showed the approach suggested by Tallis (1955) could be generalized to estimate the sampling variance of the environmental and phenotypic correlation coefficients as well as that of the genetic correlation coefficient estimated from analysis of variance and covariance. A general formula was developed for the estimated variance of the correlation coefficient from which the sampling variance of any one of the three correlation coefficients could be easily obtained by proper substitution for two sample variables.

These preliminary discussions indicate that most estimates of genetic correlation in economic traits are of doubtful reliability and, moreover, that present methods of estimating the sampling variance of the coefficient are also of questionable value except under special circumstances.

Selection applied to one trait generally results in correlated changes in other traits not under selection. This "correlated response" depends primarily upon the genetic correlation. Yet there has been little research reported on whether the theoretical treatment of correlated response to selection in terms of the genetic correlation is adequate to explain the responses realized in experimental results.

Falconer (1954) reported an experiment with mice in which two-way selection under different environmental conditions was for body weight at six weeks of age in one pair of lines and for tail length in another pair. The test of adequacy of theory came from a comparison of independent estimates from each pair of lines of the genetic correlation between body weight and tail length. Agreement between the estimates was expected to show that the theory upon which the estimation of genetic correlation is based would account fully for the correlated responses observed in the experiment. Falconer found reasonable agreement between the two estimates but concluded that the closeness of the agreement should not be emphasized since the estimates had rather wide fiducial limits.

Reeve and Robertson (1953) selected for wing and thorax length in Drosophila melanogaster and found good agreement between the estimates of the genetic correlation in the base population and the correlated responses obtained when either of the two was selected separately. Their results were based upon fifty generations of selection. The genetic correlation between the two traits was high, however, (0.70), and there is some suggestion, (Clayton et al., 1957), that the magnitude of the genetic correlation affects the accuracy of the predicted response, accidents of genetic sampling

in the correlated trait making the response unpredictable at low levels of genetic correlation.

Clayton et al. (1957), in the third of three papers devoted to an experimental check on quantitative genetic theory, also studied correlated response in Drosophila melanogaster. In their study the genetic correlation between the primary trait (abdominal bristle number) and the secondary trait (sternopleural bristle number) was small although positive (0.05 to 0.10) in the base population. Moderate agreement with predicted correlated response was observed in the early generations while inbreeding was quite low. The correlated response became entirely unpredictable with further selection in later generations. These workers concluded that if the genetic correlation is low, to measure it by correlated response is unwise unless the inbreeding each generation can be kept at a very low level; and that careful experimental design is required to estimate genetic correlations from correlated responses.

Very little is as yet known about the effects of selection on the magnitude of the genetic correlation. Lerner (1958) presented a simple theoretical model suggesting that the genetic correlation between two traits would eventually become negative if selection were applied to both traits simultaneously. Those alleles which

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affect one trait alone or both traits in a plus direction would eventually become fixed under selection for both traits while those alleles having a negative effect on one or both traits would be eliminated. The net result of selection would be to leave segregating only those alleles which have opposite effects on the two traits, thus, resulting in a negative genetic correlation. Lush (1948) makes essentially the same point when discussing the effects of selection on genetic correlation.

While this theory seems sound, there have been few experimental investigations to study the effects of selection on genetic correlations. Friars et al. (1962) reported changes over time in estimates of genetic correlations between traits under simultaneous selection for improvement in poultry. Trends in the magnitude of genetic correlations over years within the same population pointed out the danger of comparing estimates of genetic correlation from one population to another. Negative time trends occurred in sixteen out of the eighteen sets of genetic correlations estimated and six of these were significant. The remaining two sets showed positive but non-significant time trends. Fairly good evidence was thus provided that the genetic correlations were declining over the nine years of this study. The consistency of the negative trends led the authors to conclude that selection rather than progress toward linkage equilibrium was probably the cause. They suggested

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that the additive portion of the genetic covariance could have decreased through selection which would lend support to the theory proposed by Lerner (1958).

While there is limited experimental evidence in the field of quantitative genetics concerning the effect of selection on the genetic correlation, some attention has been paid in other areas to the theoretical consequences of truncation selection of one variable in a bivariate normal distribution upon the correlation coefficient. Aitken (1964) presented a treatment of the problem with reference to testing procedures used in determining admission to educational institutions. One variable was the score obtained in admission tests, which were administered to all individuals in the population to decide on admission or rejection; the other variable was score on achievement test, usually administered at a later date only to those admitted. Clearly the distribution of scores on admission tests had been truncated prior to administering the achievement test since only those scoring high in the former were admitted. Aitken suggested that such truncation will change the marginal distribution of the scores on achievement tests except in the case of independence of the two variables. The correlation between test scores in the truncated portion of the population will differ from that in the original population depending upon the degree of truncation

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exerted. A method was presented to determine the correlation in the underlying population from the correlation observed in the truncated distribution, and values were tabulated for various degrees of truncation and levels of correlation in the truncated portion. While this treatment was applied to a phenomenon somewhat divorced from genetic correlation, similar theoretical arguments would apply to truncated selection for a single trait in biological populations.

Mantel (1966) also discussed the problem from a standpoint similar to that of Aitken, again using the example of the correlation between tests to determine admittance to a school and subsequent performance. It was stressed that the correlation which is actually observed is that within the population of successful candidates rather than within the population of all candidates. Again a method is described whereby the correlation in the general population can be ascertained from the correlation within the restricted population. All that is required is the ratio of the variance of the truncated variable in the restricted portion to that in the unrestricted population. Conversely, the expected correlation in the restricted portion, caused by truncation selection, can be determined if the correlation in the unselected population is known.

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Both Aitken and Mantel indicated that the correlation in the restricted portion will be considerably weaker than that observed in the unrestricted population. If these findings are related to the problem of genetic correlation in animal populations under selection, linear truncation selection of one trait could be expected to cause a decrease in the genetic correlation in the selected portion of the population. However, when this selected group is mated to produce the next generation of individuals, what change can be expected in the genetic correlation when it is estimated in this new generation? There are three correlations involved. Firstly, there is the genetic correlation between the traits in the initial population; secondly, the correlation in the selected group; and finally, the correlation in the new generation of individuals produced by this selected group. This cycle is repeated for each generation of selection. The magnitude of the correlation in any population or sample will depend on the stage of selection and probably also on the type of selection being practised. The nature of this effect that selection is likely to have upon the genetic correlation has not become entirely clear.

Another problem associated with genetic correlation and correlated response to selection is that of "asymmetrical correlated response", discordance of the pattern of correlated response with expectation. For example, the same pattern of

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response in the correlated trait might reasonably be expected when selection is made in opposite directions. Also, the response in one trait on selection for the other should be comparable regardless of which of the correlated traits is selected. These expectations of correlated response have frequently failed to develop in experimental data, however.

Falconer (1960) selected mice for growth rate on high and low planes of nutrition where the same measurements made under two different environments were considered two separate traits. Genetic correlations observed were equal in early generations but were markedly different in later generations. Falconer attributed this asymmetry to changes in the basic parameters due to selection applied.

Asymmetry of genetic correlations also has been observed by Bell and McNary (1963) and by Yamada and Bell (1963) when selection was applied to Tribolium castaneum under two different environments. Siegel (1962) also found asymmetrical response as measured by realized genetic correlation when selection was made for body weight and for breast angle in poultry over four generations. Nordskog and Festing (1962) selected in both high and low directions for body weight and egg weight in poultry and observed asymmetry of the realized genetic correlations between body weight and egg weight when

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either the direction of selection or the trait being selected was considered. Siegel and Nordskog and Festing attributed the asymmetry to differing genetic variances or heritabilities for the two traits.

Clayton et al. (1957), in their paper on correlated response, observed asymmetry in response of sternopleural bristle number in Drosophila melanogaster when selection was made for increased and decreased abdominal bristle number. They found a marked increase in sternopleural bristles in all the high lines but no perceptible change in the low lines when selection was continued over twenty generations. They concluded that genetic drift may play an important part in the correlated response when the genetic correlation is low.

The frequency with which asymmetrical correlated responses have been found does suggest, however, that some mechanism other than genetic sampling is affecting correlated response. Bohren et al. (1966) made a detailed study of asymmetric correlated response to selection using algebraic methods and also using a computer to simulate selection experiments. The results obtained by both methods indicated that asymmetry of correlated response is to be found quite frequently. In fact, the authors suggest that to find symmetry in an experiment might

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be more surprising than asymmetry. The computer was programmed to calculate the change in gene frequency from generation to generation, and from this the expected changes in genetic variances and covariance were calculated as selection proceeded. The procedure was carried out with several models of gene effects and gene frequencies. Probably the most frequent contribution to asymmetry in practice will be from loci contributing negatively to the genetic covariance and having gene frequencies other than 0.5. The authors suggest that accurate prediction of correlated response to selection over many generations is not possible without prior knowledge of the composition and magnitude of the genetic covariance. The validity of existing theory for the prediction of correlated responses is likely to be much poorer than for the prediction of direct responses. Indeed, predictions of correlated response probably should be based on the genetic parameters estimated each generation.

The paper by Bohren et al. is of special interest because it included an investigation of genetic correlation and correlated response utilizing Monte Carlo techniques. There has, of course, been considerable investigation of various aspects of genetic theory by the Monte Carlo method since its introduction to quantitative genetics by Fraser (1957).

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Fraser (1957a) discussed the use of a digital computer to simulate genetic processes and the binary representation of genotypes and the use of logical algebra to allow the identification of the genetic nature of an individual at each locus. The author also explained methods for the determination of phenotypic value and for the simulation of inter-locus interactions, environmental effects, segregation and selection.

In a further paper, Fraser (1957b) reported on the rates of progress under varying intensities of selection and tightness of linkage. Several of the methods discussed in the introductory paper were used to simulate a genetic system of six loci, with provision being made to vary the recombination between loci from $r = 0.5$ to $r = 0.0$, that is, from independent assortment to complete linkage. Linkage was shown to produce no qualitative effect on the rates of advance at values greater than 0.5 per cent recombination. The limitation of the number of loci to less than seven and the lack of provision for dominance relations other than complete dominance were considered to be the major defects of this first program.

Barker (1958a,b) continued the study of simulated genetic systems using basically the same method as described by Fraser (1957a). In the first paper, selection between two autosomal alleles

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at four stages of the life cycle was simulated, and the program was tested by simulating two experiments with Drosophila which had been reported previously in the literature. The first of these, selection between ST and CH chromosomal arrangements on the third chromosome of Drosophila pseudoobscura, provided close agreement between the simulated and experimental results, while the second experiment, selection between wild type and glass in Drosophila melanogaster, did not. Nevertheless, the study did show clearly that selection between two alleles at an autosomal locus was possible with automatic digital computers.

Fraser (1960a) continued the discussion of Monte Carlo methods in a further paper in which he re-emphasized the procedures used and discussed the effects of linkage, dominance, and epistasis. The consideration of epistasis was continued in yet another paper, (Fraser 1960b) where he showed that while selection will lead to fixation at a slow rate in a simple additive genetic system, it will operate in complex epistatic systems to modify the relation of genotype to phenotype, the relationship becoming a sigmoid function. The last paper of the series (Fraser, 1960c) was a direct extension of the previous paper on epistasis and considered the effects of reproductive rate and intensity of selection on genetic structure. Selection against phenotypic extremes can produce a degree of genetic canalization which is more restrictive than that indicated

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These seven papers made a valuable contribution by providing the groundwork for the development of the Monte Carlo method in the field of quantitative genetics. This method furnishes an important tool, readily available to the experimenter. Genetic models can be devised, programmed, and tested in a comparatively short time, permitting the examination of theoretical consequences before experiments with biological organisms are planned. The Sydney series of papers provided the impetus for a number of investigations in the United States during the early 1960's, mostly from the project supported by the National Science Foundation, "Monte Carlo Studies of Genetic Selection," at Iowa State University.

Martin and Cockerham (1960) applied Monte Carlo techniques in a study designed primarily to explore the effects of linkage on the progress of small populations evolving under mass selection. The results indicated that tight linkage can slow down progress from selection when the populations are initially in linkage equilibrium and can result in the fixation of some unfavorable alleles. In some cases less intense selection can lead to more progress.

Baker and Comstock (1961), on the other hand, found that linkage and selection produced genetic means which were just as high as those in populations where selection was practised with no linkage,

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Qureshi (1963) reported a Monte Carlo study to explore the effects of finite population size and linkage on response to selection. All possible combinations of three levels each of population size, linkage, selection intensity, and environmental variance were simulated. The report considered changes in the genotypic mean, genotypic variance, and number of loci fixed for each of thirty generations for additive gene action only. The initial response to selection with no linkage conformed closely to predicted values, and the size of the population affected the rate of response strongly at low intensities of selection. The effects of population size were also appreciable at high intensities of selection when linkage was present, linkage interacting with selection in rate of response when population size was large. The number of generations required to reach the limit generally increased with linkage since the genotypic variance was being conserved and the response was slowed down. In general, delayed response due to moderate linkage was accompanied by a lowering of the total genetic advance. Little evidence was

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In a subsequent report (Qureshi 1964) the investigation of the effects of finite population size and linkage on the response to mass selection was continued for dominance. Two special conditions of dominance were considered, complete dominance of the desired gene and overdominance when the genotypic value of the homozygotes is equal. In complete dominance of desired genes and with initial gene frequency of 0.5, response over generations was negative under tight linkage except when the population size was as large as 64. Intense selection apparently caused a positive response in small populations only under moderate linkage. The fixation of undesirable genes was almost entirely due to population size and linkage. In the case of overdominance, the total response over 30 generations was also apparently due entirely to population size and linkage although the rate of response was evidently affected by intensity of selection. The predicted plateau in the genotypic mean over the 30 generations was observed in overdominance when population size was large and recombinations among loci was high. In general, the effect of selection intensity appeared to be additive to the effects of population size and linkage under both models of gene action. A strong interaction between population size and

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Gill in 1965 presented a series of papers on the effect of population size, linkage, selection intensity, and environmental variation upon genetic change in simulated populations. In addition, nine different models of gene action were considered, three standard non-epistatic models, additive, dominance, and overdominance, together with six different epistatic models. Four levels of environmental variation were simulated. These levels resulted in heritabilities, in the broad sense, of 1.0, 0.75, 0.50, and 0.25 in the initial generation of progeny. Populations were simulated for each of 16 runs associated with each of the nine models of gene action, the content of each parameter set being derived from the orthogonal array of a 1/16 fractional replication of a 4^4 factorial design. The four factors were population size, linkage, selection intensity and environmental variation. Selection was by upper truncation of phenotypes, and each parameter set was continued over 30 non-overlapping generations or until fixation occurred at all loci.

In the first paper of the series (Gill 1965a), the effects of finite population size on advance from selection were considered. The four population sizes simulated were 8, 12, 16, and 32 parents. Under the conditions of complete dominance, the critical size

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of a simulated population with respect to prevention of random extinction of desired alleles was between 16 and 32 individuals, while populations of 30 or more were needed to prevent random loss of alleles when overdominance existed and 1/8 or more of the total population was selected as parents. The effect of population size on the mean was of major importance, relative to the force of selection, only in populations possessing considerable amounts of variation due to dominance effects, their epistatic interactions, or both. In general, the results conformed rather well to existing theory.

In a second paper, Gill (1965b) discussed the results obtained in his simulated populations in comparison with hypothetical progress which could be predicted utilizing the mathematical formulation derived by Griffing (1960). Predictions based on infinite population size, one of Griffing's assumptions, did not conform well with realized response in more realistic populations of restricted size. The futility of predicting for more than a few generations without a re-evaluation of genetic parameters was evident, whether predictions were linear or asymptotic to the selection goal. Random genetic drift, as well as selection, had considerable influence in changing parameter values rather quickly. The author did stress, however, that the rate and magnitude of

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change observed in natural populations may differ from that observed in simulated populations.

Gill (1965c) in the third paper of the series considered the effects of intensity of selection and linkage on the genetic progress of finite populations under each of the nine genetic models. The selection intensities specified were 1/2, 1/4, 1/6, and 1/8 of the progeny populations. These, when combined with the four specified sizes of parent populations, determined progeny population sizes ranging from 16 to 256 in number and corresponded to selected population means which were expected to be 0.8, 1.27, 1.5, and 1.65 standard deviations, respectively, above the mean of the unselected population. To simulate linkage, recombination values of 0.005, 0.05, 0.2, and 0.5 were applied to the adjacent loci on each chromosome, with the probability of crossover being uniform for all adjacent pairs of loci on the same chromosome for a given run. In the populations with complete dominance, complementary factors, and duplicate factors, little or no fixation of undesirable alleles occurred at any level of selection, suggesting that the effect on total response to selection should be small even with selection as intense as 1/6. Selection was effective in advancing the genetic mean in those populations in which the genotype of highest merit was homozygous even in small populations where random drift could be expected to cause fixation of some undesirable

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recessives. In small populations where heterozygous genotypes were optimum, however, selection was evidently rather ineffective against the opposing pressure of random genetic drift.

The different levels of linkage simulated had little effect upon genetic merit, gene frequency, or fixation even in the smallest populations, except during the first few generations of selection. In populations selected for intermediates, linkage disequilibrium in addition to inbreeding appeared to bias estimates of components of genotypic variance - probably the dominance portion - for many generations, selection for the heterozygote evidently maintaining linkage disequilibrium. Under conditions of complete dominance, however, bias in the estimation of components of genotypic variance was considered to be due to inbreeding rather than to linkage disequilibrium.

Young (1966) has also used a high-speed computer to simulate genetic advance in populations under selection. In this study were large populations of 1,000 individuals per generation in each unselected population. Three intensities of selection were used corresponding to selection as parents of the best 80, 50, and 10 per cent of the individuals of each sex from each generation. The trait under selection was controlled by ten loci with two alleles at each locus, the initial gene frequency being 0.5 for each allele

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at each locus and the initial population in linkage equilibrium. In addition, three heritabilities, 0.1, 0.4, and 0.9, measured in the "narrow sense" and three probabilities of recombination, 0.5, 0.2, and 0.05 were simulated, the ten loci forming a single recombination unit. Two models of gene action, additive and complete dominance, were discussed, and each parameter set was under selection for 30 generations. The paper is evidently the first of what will be a series and results from other models of gene action will be presented in later communications.

Under the additive model, agreements between the realized advances and the expected advances predicted from parameters estimated in each generation were in most cases very close. Prediction of genetic advances was slightly less accurate when high selection intensity was applied to lowly heritable traits.

Under the dominance model, predictions were less accurate, overestimating genetic advance when selection pressure was high and underestimating it when selection pressure was low, although agreement was fairly close under low selection pressure. The decline in additive genetic variance was rapid in both models when selection was intense and particularly at high levels of heritability. The effect of linkage on this decline was small although tight linkage tended to accelerate the decline in the additive model during the initial generations but had the opposite effect in later generations.

Linkage apparently had no appreciable effect on genetic advance in these large populations, and no fixation of undesirable alleles was found even at high intensities of selection, again probably due to the large size of the populations simulated.

A genetic model for correlated responses has been described by Bohren et al. (1966) in a paper previously discussed in this review. Expected values of these correlated responses were obtained for each of nine generations of selection. Four different types of loci, A,B,C, and D were considered in the model, gene effects being additive in each case. Locus A affected the first trait only, having no effect on the second, while locus D affected the second trait only. Loci B and C affected both traits, the former making a positive contribution to the covariance, that is, affecting both traits in the same direction; and the latter making a negative contribution to the covariance. The computer was programmed to obtain the expected gene frequency at each locus for each generation. The new gene frequencies were then used to calculate the genetic covariance, the genetic and phenotypic variances, the mean of each trait, and the standardized correlated response for each generation when selection was on either of the two traits. Environmental variance was set equal to the genetic variance in all runs when all gene frequencies were one-half, giving initial heritabilities of both traits close to one-half in every

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case. The entire procedure was carried out for several models of gene effects.

The purpose of the investigation was to study conditions leading to asymmetric correlated responses. Asymmetry resulted when the relative change in gene frequency at the loci contributing positively and negatively to the covariance depended on the trait selected, with the most frequent contribution to asymmetry in practice probably coming from loci contributing negatively to the covariance and having frequencies other than 0.5.

The foregoing review, while indicating that there has been considerable discussion of and interest in genetic correlation, highlights the paucity of reliable information on the nature of the correlation, its behaviour under selection, and the behaviour of the dependent and important correlated response. An examination of the literature on the development of Monte Carlo methods in quantitative genetics research leads to the conclusion that the basic simplicity and applicability of the techniques might provide an approach to the problem of the effects of selection on correlated response. Despite the shortcomings of the simulation method and while the true situation might still remain undiscovered, at least a new avenue might be opened to the problem which could prod other investigators to seek alternative pathways to clarify the situation.

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METHODS AND PROCEDURE

The Experimental Design and Parameters Simulated.

The major objective of this study was to investigate the effects of degree of heritability and of truncation selection of a primary trait upon the behaviour of the genetic correlation and the correlated response in a secondary trait in populations under selection. A completely comprehensive study could embrace all combinations of a large number of different factors, including numerous models of gene action, interaction, and correlation; different levels of environmental variation, genotype-environment correlation and interaction; and various methods and intensities of selection for one or both traits under consideration. The number of traits could also be increased beyond two. Consideration of time and cost, however, quickly limits the size of any such study to an arbitrary number of factors and levels thought to be most important by the investigator. The results obtained in this initial study should, hopefully, prod the researcher in the direction of the potentially most fruitful avenues of inquiry to be explored in later research.

The factors most important for the purposes of the present investigation and the levels allowing for a wide range of effects of each while containing the size of the experiment within reasonable bounds, are given below.

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

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

iii. Three levels of selection, 20, 50, and 80 per cent of the offspring each generation.

iv. Three levels of environmental variance, V(E), for X and Y, relative to the expected additive genetic variance in the initial generation of offspring, $V(G_a)$. The levels were chosen in such a way that $h' = \frac{V(G_a)}{V(G_a)+V(E)}$ was equal to 0.1, 0.4, or 0.7. When all of the genetic variance is additive, h' is a measure of heritability in the "narrow" sense. When genetic variance other than additive is present, h' will be greater than heritability in the "narrow" sense.

The four factors, genetic correlation, intensity of selection for X, and environmental variation of X and Y, each at three levels, were considered in all combinations, and each treatment combination or parameter set was replicated. These factors and levels provided 81 treatment combinations in a 3^4 factorial experiment which, when replicated, resulted in 162 parameter sets. The factors

and levels simulated are shown below where b is the fraction of the offspring becoming parents each generation, r_G is the genetic correlation between the two traits in the first generation of offspring, and h'_x and h'_y represent the levels of environmental variation of X and Y, respectively:

		LEVELS	
	0	1	2
FACTOR b	0.80	0.50	0.20
r_G	0.25	0.50	0.75
h'_x	0.10	0.40	0.70
h'_y	0.10	0.40	0.70

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

a) Additive model in which the contributions to the genotypic value were 2, 1, and 0 for the ++, + -, and -- phases, respectively, 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, respectively, at each locus. In this case selection was in both directions, upwards for the dominant allele and downwards for the recessive allele.

These models provided three separate experiments resulting

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in a total of 3 x 162 or 486 parameter sets.

The Structure of the Initial Population.

Specification of the basic structure of the initial population carries with it necessary assumptions which are of sufficient importance to require discussion in some detail.

The population in this study was the bisexual diploid type, and the two quantitative traits X and Y were expressed in both sexes. Since the size of the population was related to the number of parents rather than to the number of offspring produced, the number of parents was held constant each generation for all treatment combinations. The parents were limited to 48 individuals, 24 males and 24 females, and the number of offspring produced by these parents was determined by the selection intensity desired. To provide levels of b, the fraction saved, of 0.80, 0.50, and 0.20 in each generation, 30, 48, and 120 male and female offspring were produced giving 60, 96 or 240 offspring each generation. Selection intensity was equal in the two sexes.

The 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 the possibility of both full-sibs and half-sibs among the offspring in any generation. Sampling without replacement from both male and female parents could have been done and could have allowed for

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an equal number of progeny from each selected parent. But, an equal number of progeny per parent is an idealized situation for natural finite populations and sampling with replacement conforms more closely to the situation in natural populations.

Each parameter set was continued for 30 generations to provide sufficient opportunity to observe a selection limit. The generations were non-overlapping, overlapping generations being an unnecessary additional complexity.

The genetic structure of the base population has to vary with the aims of the investigation being conducted and, to some degree, with the peculiarities of the computer available. The number of loci controlling the genotype, for example, is likely to be limited by the storage capacity of the computer. Clearly the number of loci involved in most quantitative traits in farm animals could not easily be simulated.

The computer system available for this study was the CONTROL DATA 3600, which is a general purpose digital computing system with large storage capacity and exceedingly fast data transmission and which is efficient in solving large scientific problems. Each word in the storage module has a 51 bit structure made up of 48 bits of data and three parity bits, thus allowing for the expedient handling of a 48 bit data word. Magnetic core storage of 32,768 of these 48 bit words is available. For these reasons the number

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of loci affecting each of the two traits was specified as 48, which meant that two 48 bit words could be conveniently used to represent the genotype of each trait, simulating two chromosomes. Thus, four words were required to store the genotype of each individual, and 4 x 48 or 192 words were assigned to the parent population.

No linkage was specified in the genetic structure; all loci were considered to be completely independent and the gene effects equal at all loci. Further restrictions were those of no inter-allelic 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 to allow for changes in gene frequencies in either direction.

As stated previously, two different modes of gene action, additive and complete dominance, were simulated with the mode of gene action the same at all loci for a particular run. For the symmetrical additive model, where the contributions to the genotypic value at each locus were 2, 1, and 0 for the++,+-, and -- phases, respectively, 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 q the gene

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frequency of the plus gene the same at all loci, the expected genotypic mean was 2nq and the expected genotypic variance was 2nq(1-q) where n is the number of loci affecting the trait. In this case, with 48 loci affecting each trait and q equal to 0.5, the expected genotypic mean and variance in the initial generation were 48 and 24 respectively, under the additive scheme.

In the model of complete dominance where the contributions to the genotypic value at each locus were 2, 2, and 0 for the ++, +-, and -- phases, respectively, the genotypic value was $2(n_1+n_2)$ The expected genotypic mean and variance in the initial generation (Kempthorne, 1957) were then 2nq (2-q) and $4n\left[2q(1-q)^3+q^2(1-q)^2\right]$, respectively which resulted in an expected mean and variance in the first generation in the population simulated of 72 and 36, respectively, for each trait. With $q = 0.5$ the genotypic variance was made up of additive genetic variance of 24 and dominance variance of 12.

Levels of environmental variation were simulated relative to the expected additive genetic variance in the first generation of offspring to produce the desired degrees of heritability in the "narrow" sense of 0.1, 0.4, and 0.7 in the additive model. In the model of complete dominance these levels resulted in heritabilities of 0.095, 0.33, and 0.52, respectively. The environmental component was assumed to be

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independent of the genotype and constant over generations. Hence, the phenotypic value of each trait in each individual was determined by adding xc_i to the genotypic value where x was a properly generated normal variate with zero mean and unit variance and c_i was a constant designating the environmental standard deviation required to produce the desired degree of heritability. For the three heritabilities simulated in this investigation, 0.1, 0.4, and 0.7, phenotypic variances of 240, 60, and 34.3 were required. The constants, c_i, required to produce the environmental variances of 216, 36, and 10.3 were, therefore, 14.697, 6.000, and 3.207, respectively. These environmental standard deviations multiplied by a random standard normal deviate and added to the genotypic value resulted in the required phenotypic variance to produce the desired degree of heritability in the initial generation in the additive model. In order to allow study of the effects of the different environmental levels upon the change in the genetic parameters simulated, no attempt was made thereafter to keep heritability constant over the thirty generations.

Simulation of the Genetic Correlation.

Since the genetic correlation was clearly the most important parameter simulated in this study, the method of simulation and

its justification will be discussed in some detail. That the cause of the genetic correlation was attributed solely to pleiotropy should be stressed. The degree of correlation arising from pleiotropic gene action expresses the extent to which the two traits under consideration are influenced by the same genes, and the resulting correlation is the overall effect of all the segregating genes that affect both traits. All of the genes affecting the two traits affected each one in the same direction, thus making a positive covariance. Other systems could have been simulated, some genes affecting one trait in one direction and the other trait in the opposite direction making a negative contribution to the covariance and resulting in a genetic correlation which could vary from -1 to +1. Limitation of the size and scope of the present study prevented the simulation of negative genetic correlations or of zero correlations although these could be interesting parameters for later investigation.

In this investigation the genetic correlation was determined by the number of loci which had an effect on both traits. As 48 loci affected each of the two traits, the number of these 48 which were shared by the two traits determined the degree of pleiotropy and of genetic correlation. To produce genetic correlations of 0.25, 0.50, and 0.75, the number of loci in common was set at 12, 24, and 36, respectively. The remaining loci of the 48 affecting

each trait affected each trait independently. The table below might illustrate the method more clearly:

	Loci A	Loci B	Loci A'
Trait X	+	+	0
Trait Y	0	+	+
No. of Loci	m	n	m'

There were three different types of loci in the genetic system. Those in group A affected trait X only and had no effect on trait Y; those in group A' affected trait Y only and had no effect on trait X; those in group B affected both traits X and Y in the same direction, the magnitude of the effect being the same for both traits. The total number of 48 loci affecting each trait, was made up of n loci which affected both traits plus m or m' loci which affected only trait X or trait Y, respectively. Thus, when the genetic correlation was 0.25, n=12 and m=m'=36; when the genetic correlation was 0.50, n=24 and m=m'=24; and when the genetic correlation was 0.75, n=36 and m=m'=12. Clearly, if the number of loci affecting each trait were not the same, m would not be equal to m'. In this case, however, m was always equal to m'. The genetic correlation was then simulated simply as $\frac{n}{n+m}$. The genotype of trait X, G_X, was determined by the loci in groups A and B, and the genotype of trait Y, G_Y, was

determined by the loci in groups A' and B. In this simulated population, G_X and G_Y were obtained in each generation for each individual. The genetic correlation was measured in each generation as the product - moment correlation between the genotypic values thus:

$$r_G = \frac{\text{cov } G_X G_Y}{\sqrt{V(G_X)\ V(G_Y)}}$$

where r_G is the genetic correlation, cov $G_X G_Y$ is the covariance between the genotypic values, and $V(G_X)$ and $V(G_Y)$ are the variances of the genotypic values.

For the additive case the genetic correlation as measured in this way is simply $\frac{n}{n+m}$, or the ratio of the number of loci which the two traits share to the number of loci affecting each trait, as follows:

$G_X = G_A + G_B$ and $G_Y = G_{A'} + G_B$ since the loci are independent.

$$\therefore\ r_G = \frac{\text{cov}(G_A+G_B)(G_{A'}+G_B)}{\sqrt{V(G_A+G_B)\ V(G_{A'}+G_B)}}$$

$$= \frac{\text{cov}G_AG_{A'} + \text{cov}G_AG_B + \text{cov}G_BG_{A'} + V(G_B)}{\sqrt{\left[V(G_A)+V(G_B)+2\text{cov } G_AG_B\right]\left[V(G_{A'})+V(G_B)+2\text{cov}G_{A'}G_B\right]}}$$

But since all loci are independent and the effects are equal and additive at each locus, all covariances are expected to equal zero.

$$\therefore r_G = \frac{V(G_B)}{\sqrt{\left[V(G_A) + V(G_B)\right]\left[V(G_A') + V(G_B)\right]}}$$

Under the assumptions of the model, these variances can be written in terms of the number of loci, gene frequency (q), and the effect $D = \frac{(++)-(--)}{2}$ at each locus.

$$\therefore r_G = \frac{2nq(1-q)D^2}{\sqrt{\left[2mq(1-q)D^2+2nq(1-q)D^2\right]\left[2m'q(1-q)D^2+2nq(1-q)D^2\right]}}$$

But, gene frequency and D are equal at all loci

$$\therefore r_G = \frac{n}{\sqrt{(n+m)(n+m')}} \quad ;$$

and since $m = m'$ in this case,

$\therefore r_G = \frac{n}{n+m}$ or the ratio of the number of loci affecting both traits to the total number of loci affecting each which was the method of simulating the genetic correlation.

The genetic correlation was also measured 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 allow comparison of the accuracy of the methods. The two methods were:

a) $$\hat{r}_G = \sqrt{\frac{(covPxpPyo).(covPypPxo)}{(covPxpPxo).(covPypPyo)}}$$

b) $\tilde{r}_G = \frac{(\text{covPxpPyo})+(\text{covPypPxo})}{2\sqrt{(\text{covPxpPxo}).(\text{covPypPyo})}}$

where Pxp = phenotypic value of trait X in the parent

Pyp = " " " trait Y " " "

Pxo = " " " trait X " " offspring

Pyo = " " " trait Y " " "

Both of these methods reduce to a measure of $\frac{n}{n+m}$ for the case of equal and additive effects, independence, and equal gene frequency at all loci. This is done as follows for method a) for example:

Let Gxp = genotypic value for trait X in the parent

Gyp = " " " trait Y " " "

Gxo = " " " trait X " " offspring

Gyo = " " " trait Y " " "

and Exp = environmental contribution to trait X in the parent.

Eyp = environmental contribution to trait Y in the parent.

Exo = environmental contribution to trait X in the offspring.

Eyo = environmental contribution to trait Y in the offspring.

$$\hat{r}^2_G = \frac{(\text{cov PxpPyo}).(\text{cov PypPxo})}{(\text{cov PxpPxo}).(\text{cov PypPyo})}$$

$$= \frac{\left[\text{cov}(Gxp+Exp)(Gyo+Eyo)\right]\left[\text{cov}(Gyp+Eyp)(Gxo+Exo)\right]}{\left[\text{cov}(Gxp+Exp)(Gxo+Exo)\right]\left[\text{cov}(Gyp+Eyp)(Gyo+Eyo)\right]}$$

since all covariances between G and E are expected to equal zero.

$$= \frac{(\text{cov } G_{xp}G_{yo})(\text{cov } G_{yp}G_{xo})}{(\text{cov } G_{xp}G_{xo})(\text{cov } G_{yp}G_{yo})}$$

$$= \frac{\left[\text{cov}(G_{Ap}+G_{Bp})(G_{A'o}+G_{Bo})\right]\left[\text{cov}(G_{A'p}+G_{Bp})(G_{Ao}+G_{Bo})\right]}{\left[\text{cov}(G_{Ap}+G_{Bp})(G_{Ao}+G_{Bo})\right]\left[\text{cov}(G_{A'p}+G_{Bp})(G_{A'o}+G_{Bo})\right]}$$

and since all loci are independent

$$= \frac{(\text{cov } G_{Bp}G_{Bo})\ (\text{cov } G_{Bp}G_{Bo})}{(\text{cov } G_{Ap}G_{Ao}+\text{cov } G_{Bp}G_{Bo})(\text{cov } G_{A'p}G_{A'o}+\text{cov } G_{Bp}G_{Bo})}$$

$$= \frac{\left[r_{G_{Bp}G_{Bo}}\sqrt{V(G_{Bp})V(G_{Bo})}\right]\left[r_{G_{Bp}G_{Bo}}\sqrt{V(G_{Bp})V(G_{Bo})}\right]}{\left[r_{G_{Ap}G_{Ao}}\sqrt{V(G_{Ap})V(G_{Ao})}+r_{G_{Bp}G_{Bo}}\sqrt{V(G_{Bp})V(G_{Bo})}\right]\left[r_{G_{A'p}G_{A'o}}\sqrt{V(G_{A'p})V(G_{A'o})}+r_{G_{Bp}G_{Bo}}\sqrt{V(G_{Bp})V(G_{Bo})}\right]}$$

But $r_{G_{Bp}G_{Bo}} = r_{G_{Ap}G_{Ao}} = r_{G_{A'p}G_{A'o}} = 1/2$

and $V(G_{Bp}) = V(G_{Bo})$ and $V(G_{Ap}) = V(G_{Ao}) = V(G_{A'p}) = V(G_{A'o})$

$$\therefore \hat{r}^2_G = \frac{V(G_B)\ V(G_B)}{\left[V(G_A)+V(G_B)\right]\left[V(G_A)+V(G_B)\right]}$$

$$\therefore \hat{r}_G = \frac{V(G_B)}{V(G_A)+\ V(G_B)}$$

which in terms of n, the number of loci, gene frequency and D is equal to

$$\frac{2nq(1-q)D^2}{2nq(1-q)D^2 + 2mq(1-q)D^2}$$

$= \frac{n}{n+m}$ which again measures the genetic correlation as it was simulated due to pleiotropy. The same solution can be

obtained for method b) in which the arithmetic mean of the covariances is used in the numerator rather than the geometric mean, in computing the genetic correlation.

The discussion above has explained the method of simulating the genetic correlation, the justification for this method, and has shown that the usual method of computing the genetic correlation in economic species is also theoretically an adequate measure of pleiotropy.

The Mechanics of Simulation.

In this section the logic of the program developed to simulate the population will be described. A detailed discussion of the structure and genetic properties of the population has already been presented as has a short description of the computer which was available for the study.

A feature common to all types of investigation involving Monte Carlo methods is the use of pseudo-random numbers, which, although truly random only conceptually, have fulfilled as many criteria of randomness as possible. A library program, RANF, was available at Michigan State University for the generation of uniformly distributed pseudo-random numbers. Repeated use of RANF generates a uniformly distributed

sequence of random numbers in either fixed or floating point format. If floating point is used, the numbers range from 0 to less than 1.

The random numbers are produced by the standard multiplicative congruential method. The derivation of the multiplicative method used is of the form

$$X_i + 1 = X_i \lambda \pmod{m}$$

where X is any odd number

$$\lambda = 5^2 \times 15$$

$$m = 2^{47}$$

These parameters have been shown to satisfy the sufficient conditions for a sequence of maximal period. The period for this generator is 2^{45} in the computer which was used. Tests for accuracy, indicated by Rotenberg (1960), were performed and the results agreed very well with the theoretical distribution.

The procedure was modified by Rotenberg (1960) to the form

$X_i + 1 = (2^a + 1)X_i + C$ with $a \geq 2$ and C odd.

In the random number generator available a was set equal to 10 and C equal to 101, making $\lambda = 2^{10} + 1 = 1025$ and $X_i + 1 = 1025X_i + 101$

The method used has passed many tests of randomness including a test of the frequency distribution of the random

numbers, a test of the frequency with which a number of a certain magnitude was followed by a number of another certain magnitude, a test of the frequency distribution of the length of runs of numbers either above or below the mean, serial correlation tests, and others.

When the genotypic value for each individual was determined for each of the two traits, an environmental contribution had to be added to this genotypic value to provide the phenotypic value for each of the traits. The determination of the environmental contribution required the generation of a standard random normal deviate which when multiplied by a constant representing the desired environmental standard deviation, provided the random environmental contribution to each phenotype. Such environmental contributions should have the desired environmental variance to give the desired degree of heritability of the trait. For example, for the additive model, where the expected additive genetic variance is 24 in the first generation of offspring, suppose the heritability of trait X is to be simulated as 0.4 in the first generation. Then, an expected phenotypic variance of 60 is required which means that environmental contributions to the phenotype of each individual for trait X should have a mean of zero and variance of 36. Thus, the environmental contribution is

required to be of the form 6x, where six represents the required environmental standard deviation and $\underline{x}$ is a N(0,1) random deviate.

There are numerous methods available for the generation of random normal deviates and, indeed, generation is not really necessary since tabulated values can be stored in the computer. But, because the generation process was relatively simple, and because a very large number of deviates were required in this study, the deviates were generated as needed.

The general procedure used has been described in detail by Gill (1963). A specified number of uniformly distributed random numbers in the range $-1 \leq r_i < +1$ are generated, added together and the variance coded so that the sum is normally distributed with mean equal to zero and standard deviation equal to one.

In the present investigation twelve random numbers were generated using the same library subroutine as described previously. The random numbers generated were in the range $0 \leq r_i < +1$ and, therefore, had to be coded to produce the desired uniformly distributed random numbers in the range $-1 \leq r_i < +1$. Coding was accomplished by multiplying each generated random number by two and subtracting one,

effectively providing random numbers in the range $-1 \leq r_i < +1$. Twelve of those random numbers were then added together to produce numbers in the range $-12 \leq e_j < +12$. The variance of a uniformly distributed variable is equal to the square of the range divided by 12. Therefore, $V(r_i) = 2^2/12 = 1/3$. Then the variance of $e_j = \sum_{i=1}^{12} r_i$ was equal to 12/3, and standard deviation was 2. But the e_j had to be normally distributed with a standard deviation of 1, which was done by multiplying each e_j by 0.5, providing N(0,1) random deviates. A sample of random deviates was generated and conformed closely to the standard normal distribution.

The mechanics of simulation of the population under selection fell into several quite logical and separate blocks, and the computer program was written in pieces corresponding to each distinct phase of the simulation procedure.

The initial block of the program consisted of instructions which set the numerical constants to be used in the program for that particular parameter set. These constants included the required genetic correlation, specified as the number of genes shared by the two traits; the required level of selection, specified as the number of offspring of each sex to be produced each generation; constants representing the environmental

standard deviation required to produce the desired heritability of each trait; and the replicate number. The constants were then changed in sequence after each parameter run until all combinations of parameter sets had been simulated.

The second part of the program consisted of the generation of the 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. Each word contained 48 bits, B represented the number of bits which contained identical genes in the genotype of each trait, effectively simulating the required genetic correlation. The process of generating the initial parents then proceeded as follows:

A random number was generated and 0.5 subtracted from it. If the number obtained was positive or zero 1 was put in the B^{th} bit of the first word of trait X and of trait Y; if negative, 0 was put in these two locations. This process was repeated for all of B bits of the first word of traits X and Y. 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 of the traits was generated and of these 48 alleles the traits had B alleles

in common. By taking the complement of each of the two arrays, the corresponding alleles of the second chromosome contributing to traits X and Y were generated so that they alternated those of the first chromosome at every locus, producing the desired individual completely heterozygous at every locus for both genotypes with the required B alleles in common. The above procedure was then repeated for 48 individuals alternately of male and female sex.

The third part of the program was concerned with the production of the offspring generation from the parent generation. The number of offspring of each sex to be produced was determined by the desired level of selection to be practised for that parameter run. To provide 20, 50, or 80 per cent of the offspring generation saved and to retain parent population size constant at 48 required the production of 120, 48, or 30 offspring of each sex each generation. When the top 24 individuals of each sex were selected, the required 48 parents were provided for the next generation.

Random numbers again were used to select the two parents to be "mated" to produce each offspring. To select the parents to be mated at random, a random number was generated and multiplied by 24, producing a number in the range 0 to 23.999, which by the addition of 1 gave a number in the range 1 to 24.999.

This number then was truncated to integer value resulting in a number which had the range 1 to 24. The result was the number of the male parent selected to produce the offspring. The same process was followed in the random selection of the female parent.

The next stage involved the production of a random gamete from each parent. To do this, a random number was generated, and depending on the magnitude of the random number, the allele at the B^{th} bit of word number 1 or word number 2 in the genotype of trait X was chosen with equal probability and assigned to the B^{th} bit of word number 1 of both traits X and Y in the offspring. This procedure was followed for all B bits of the male parent in trait X. Since the genotype of trait Y in the first B bits was identical to that of trait X for the first B bits, these alleles could be ignored in trait Y in the parent. The remaining (48 - B) bits in trait X were then assigned in the same random manner to the (48 - B) bits of trait X in word number 1 in the offspring, and then the (48 - B) bits in trait Y were assigned to the (48 - B) bits of trait Y in word number 1 in the offspring. Thus, the offspring genotype was now completed for word number 1 in both trait X and trait Y with the first B alleles identical. In other words, the contribution of the male gamete was completed. The same

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procedure was followed for the female parent, effectively allocating the alleles in word number 2 of both traits and producing the genotypes of the two traits in the offspring, again with the desired genetic correlation between them.

The whole process was then repeated until the required number of offspring of each sex had been produced.

The fourth stage in the program involved genotypic evaluation. The purpose was to calculate the numerical value of the genotype for each trait in each individual offspring, depending upon the model of gene action being simulated. For the additive model the genotype could be evaluated simply by summing the 1's or 0's over all 48 loci for each of the two traits, X and Y.

In the complete dominance model, however, the genotypes had to be evaluated by gene pairs at each locus, the genotypic value of the individual for each trait consisting of the sum of the 48 allelic-pair values. In the model the ++and +- phases each had a value of two while the -- phase had a value of zero. Thus, the method used to evaluate the genotype was to determine the number of ++and +- phases in the 48 loci affecting each trait. To do this logical algebra was used. For example, consider the simple 4-locus genotype $\frac{1100}{1010}$. A logical "OR", which determines the loci having one or both alleles equal to 1,

gives the result $\frac{1100}{1010} = 1110$ and thus identifies the loci either homozygous for the dominant allele or heterozygous. The number of such loci could then be multiplied by two to give the numerical genotypic value for that trait for the complete dominance model. For the simple 4-locus example above, the genotypic value would be 3 x 2 or 6.

Following the genotypic evaluation of each trait, depending upon the model, an environmental contribution was added to the genotypic value to provide the phenotypic value for each trait. In this part of the program, standard random normal deviates, generated in the manner described previously, were multiplied by a constant representing the desired environmental standard deviation of the trait to provide the environmental contribution to the phenotype. This environmental contribution was then added to the genotype to give the phenotypic value for each trait. The model is illustrated below:

$$G_X + E_X = P_X$$

$$G_Y + E_Y = P_Y$$

where G_X and G_Y are the genotypic values of the two traits.

E_X and E_Y are the environmental contributions to each trait.

P_X and P_Y are the phenotypic values of the two traits. Thus, at this stage in the simulation procedure, the48 locus

genotype of each trait, together with G_X, G_Y, E_X, E_Y, P_X, and P_Y were determined for each of the offspring. These values provided all the information required to calculate the desired statistics for output for each offspring generation.

First, the gene frequency was determined for each trait by simply summing the number of plus alleles in each genotype and dividing by the total number of genes, which was 96 times the number of offspring produced. Then, the genotypic mean, variance, and standard deviation, the environmental variance, and the phenotypic mean, variance and standard deviation were calculated for each of the two traits. Following this the genotypic, environmental, and phenotypic covariance, and finally, the product-moment correlation between trait X and trait Y were calculated, to determine the genetic, environmental, and phenotypic correlation between the two traits. In addition, the heritability of each trait measured as $\frac{V(G_X)}{V(P_X)}$ and $\frac{V(G_Y)}{V(P_Y)}$ was calculated. The final output statistics in each generation of offspring were the two estimates of the genetic correlation measured from parent-offspring covariances as described earlier in this section.

The next subroutine was concerned with the selection of the parents of the next generation. As already stated, the process in this study involved the upper or lower truncation selection

based on individual phenotype of trait X, of 24 males and females to provide the 48 parents of the next generation. Selection was accomplished by ranking each sex on the phenotype for trait X, either from high to low or from low to high depending upon the direction of selection desired, and then the 24 winning individuals were retained and relocated as the parents of the next generation. In the additive model, only the best 24 individuals of each sex were selected each generation while in the complete dominance model two separate experiments were carried out, the best 24 individuals of each sex being chosen in one and the worst 24 in the other each generation.

Following selection and relocation of the selected group, the statistics which were calculated in the unselected offspring generation were again calculated, with the exception of the two estimates of genetic correlation measured from parent-offspring covariances. In addition both primary and secondary selection differentials were measured as the difference between the phenotypic mean of the selected group and the unselected group for traits X and Y, respectively.

Also calculated in the selected group was the number of loci fixed in either the plus or minus phase of the genotype. The determination of fixation of loci was done for both the pleiotropic loci alone and for all loci for each of the two traits.

Hence, there were eight measures of fixation of loci calculated, these being:

i. Loci fixed 0 in the pleiotropic section of the genotype of X and of Y.

ii. Loci fixed 0 in the whole 48-locus genotype of X and of Y.

iii. Loci fixed 1 in the pleiotropic section of the genotype of X and of Y.

iv. Loci fixed 1 in the whole 48-locus genotype of X and of Y.

To determine the number of loci fixed in each case logical algebra was again used. A logical "OR", described previously, when calculated over all 48 individuals, indicated the number of loci fixed 0 while the logical "AND" or logical product determined the loci fixed 1. For example, the logical "AND" or logical product of the simple genotype $\frac{1100}{1010}$ is 1000; that is, when both alleles are 1, this equals 1 and any other combination equals 0. When the logical "AND" was determined over all 48 individuals, the number of loci fixed 1 could be found. The logical "OR" of the above genotype is 1110 and the complement taken over all 48 individuals indicated the number of loci fixed 0.

After all statistics had been calculated, the next generation of offspring was produced from the 48 selected parents and the cycle repeated until 30 generations of offspring had been produced for that particular parameter set. The parameter

run was then replicated for that parameter set. After replication one of the constants was changed, and the parameter runs were continued for all treatment combinations.

The computer time for the simulation procedure was approximately 225 minutes to complete each model of gene action. Thus, each of the 162 parameter runs required an average of 83 seconds, an average of 2.8 seconds to simulate each generation.

RESULTS AND DISCUSSION

The Additive Model

In the additive model the contributions to the genotypic value were 2, 1, and 0, respectively, for the ++, + -, and -- genotypes at each locus. Three levels each of initial genetic correlation, intensity of selection, and environmental variation of X and Y provided 81 treatment combinations which when replicated resulted in 162 parameter sets. Variations between replicates were small and were not of sufficient interest to justify a detailed presentation of the results for each replicate. Results for all statistics presented graphically were averaged over the two replicates.

Statistics were calculated for each of the 30 generations of selection in a given parameter set. However, to reduce the large amount of data to a manageable quantity, only the results for every fifth generation are presented. The expected genotypic means, environmental and genotypic variances of traits X and Y, and the expected genetic covariance and correlation between the two traits in the first generation of offspring were:

i. Expected genotypic means were 48 for X and Y

ii. Expected genotypic variances were 24 for X and Y

iii. Expected environmental variances for each trait were

10.3, 36, and 216 for $h^2 = 0.7$, 0.4 and 0.1, respectively.

iv. Expected genotypic covariances were 6, 12, and 18 for initial expected genetic correlations of 0.25, 0.50, and 0.75, respectively.

The results in the first generation of simulated offspring showed close agreement with the expected values in almost every case.

The Effect of Selection on the Genetic Correlation.

In figures 1.1, 1.2, and 1.3 the change in the genetic correlation measured as the product-moment correlation of genic values is presented. The solid line represents the genetic correlation measured in the unselected offspring generation while the broken line indicates the correlation measured in the truncated part of the offspring generation, or in 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 whole offspring generation with the selection intensity. The correlation includes 60 individuals when $b = 0.8$, 96 individuals when $b = 0.5$, and 240 individuals when $b = 0.2$.

Since the environmental variance of trait Y, the unselected

trait, had no effect on the genetic correlation measured from genic values, r_G was averaged over the three levels of environmental variance of Y and, as stated before, over the two replicates. Thus, each point on the graphs represents the average of six estimates of genetic correlation.

The distribution of the correlation coefficient, estimated from small samples, is known to be far from normal when ρ is other than zero. Thus, rather than an arithmetic average of r, each r was transformed to z, which does approximate the normal distribution at all levels of ρ . For small values of r, z is nearly equal to r, but as r approaches unity, z increases without limit. The value of z corresponding to a given r was obtained from those tabulated by Fisher (1958). These z values were then averaged and the resulting mean z reconverted to r.

Figure 1.1 shows the change in the genetic correlation at each of the three levels of selection when the environmental variance was large ($h'^2_x = 0.1$). The most noticeable point brought out by this graph was the remarkable consistency of the genetic correlation in the whole offspring generation over the 30 generations of selection at all three levels of selection and at all three levels of genetic correlation in the initial generation. The estimates of genetic correlation when the

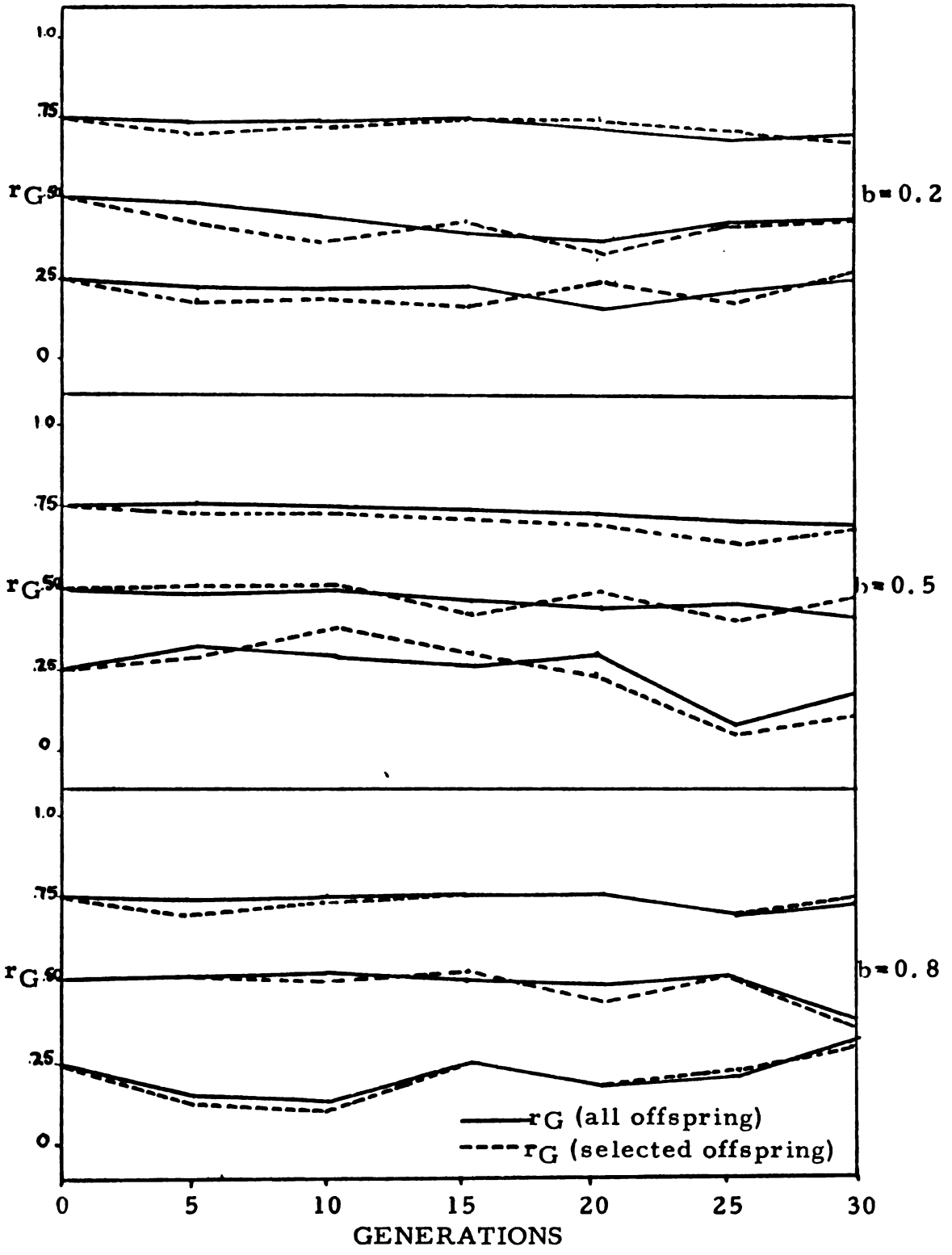

Figure 1.1 Change in genetic correlations at three levels of selection when $h_x^2 = 0.1$ (additive model).

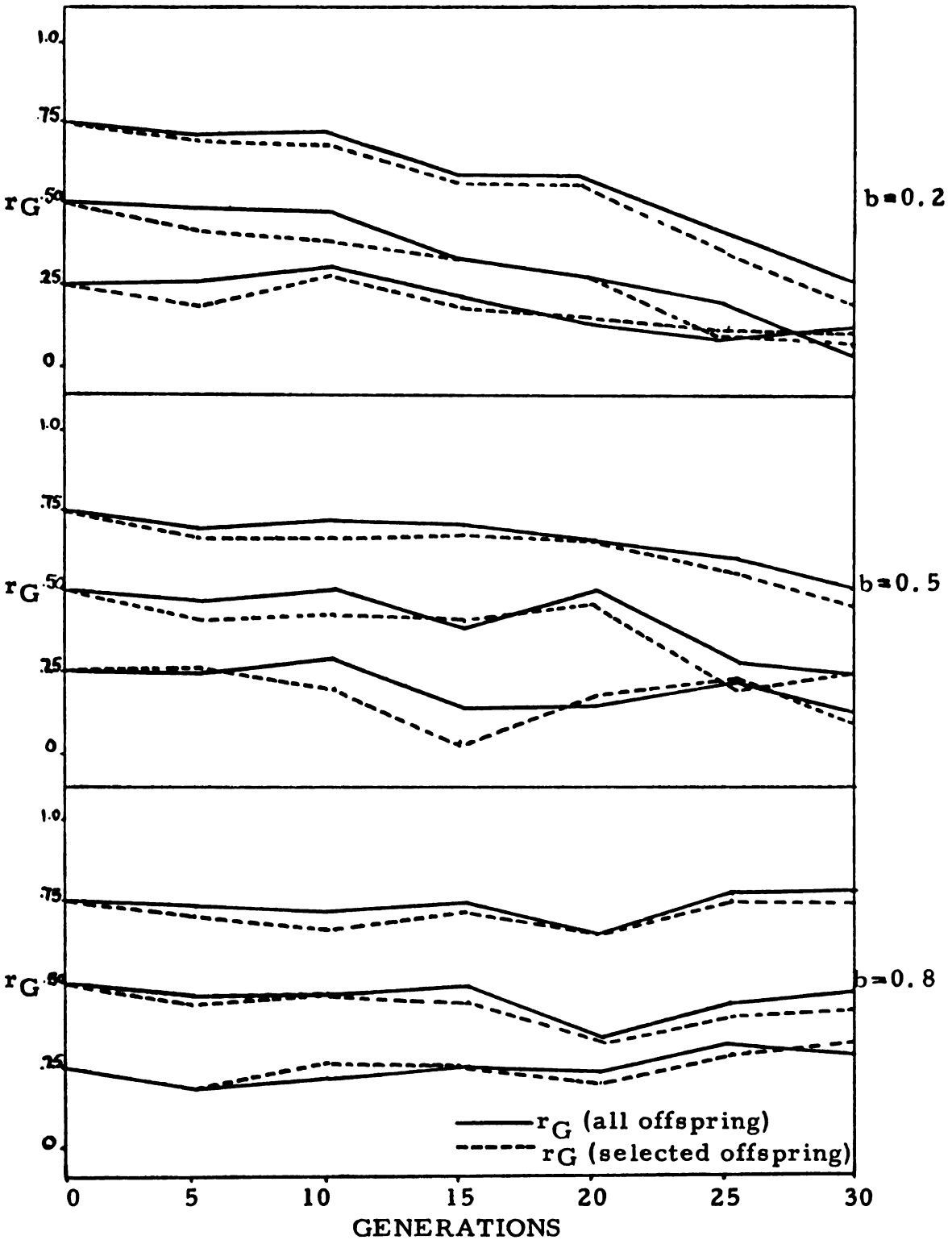

Figure 1.2 Change in genetic correlations at three levels of selection when $h^2_x = 0.4$ (additive model).

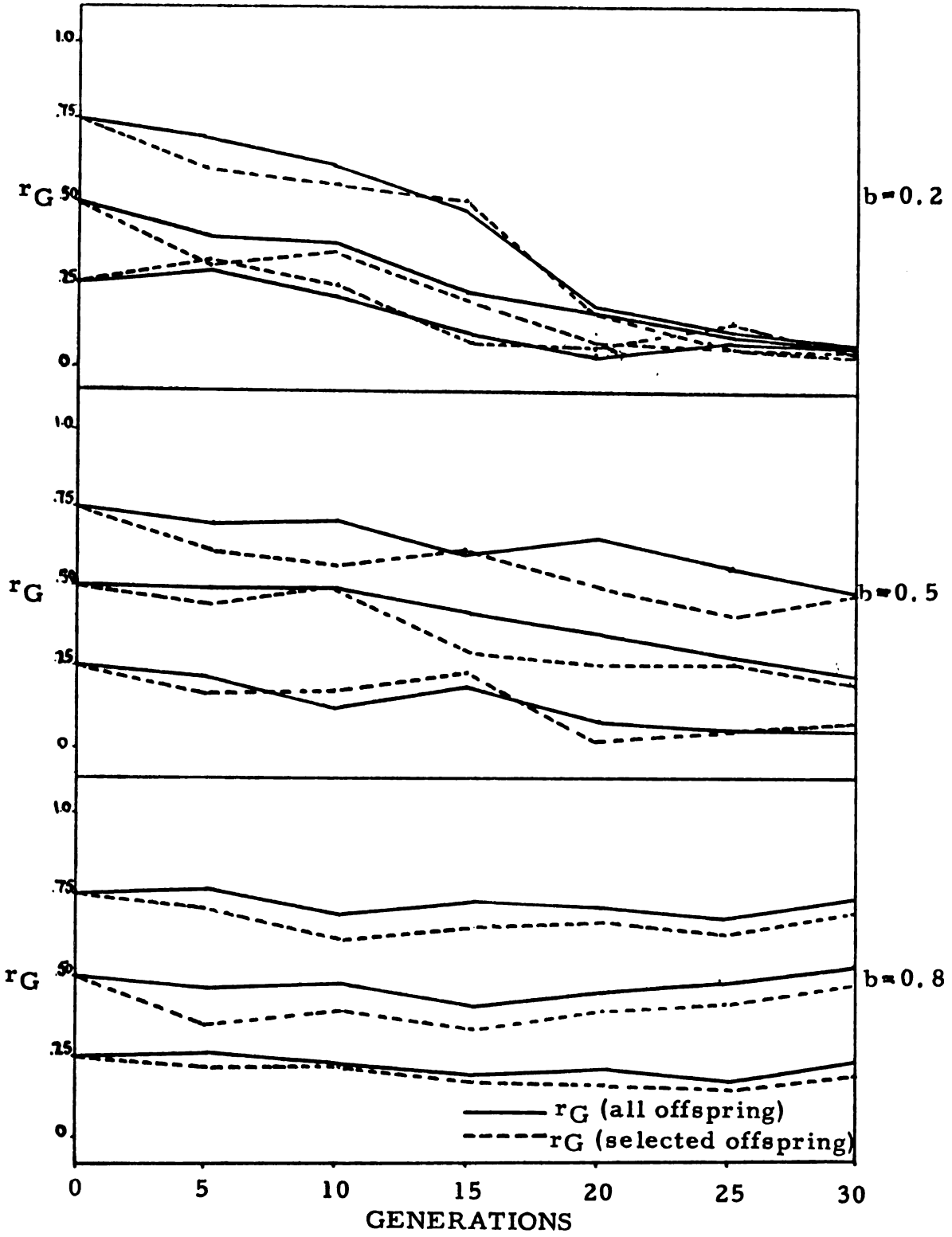

Figure 1.3 Change in genetic correlations at three levels of selection when $h_x^2 = 0.7$ (additive model).

initial correlation was low ($r_G = 0.25$) 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 ($h'^2_x = 0.4$), the genetic correlation again remained near the level in the first generation at all levels of correlation when $b = 0.8$. At intermediate selection ($b = 0.5$), however, the first indication of a decrease in the correlation over the 30 generations was noticeable especially when initial $r_G = 0.75$. The trend was not so obvious at low correlation. At high selection intensity ($b = 0.2$) the decreasing trend in the genetic correlation became even more distinct when initial $r_G = 0.75$ or 0.5. The correlation did remain high for some considerable time, however. For example, at the 20th generation of selection the genetic correlation, originally 0.75, was still almost 0.65. Again when the initial genetic correlation was low ($r_G = 0.25$), the change in the genetic correlation was more erratic and the trend not nearly so clear.

In Figure 1.3, where heritability was high, the genetic correlation again remained at its initial level when $b = 0.8$. The tendency for the correlation to decrease again became noticeable only after the 15th generation of selection. It was only when the selection intensity was high ($b = 0.2$) and

environmental variance was low ($h'_x = 0.7$) that a rapid decline occurred in the genetic correlation. This decline was noticeable at all three levels of genetic correlation but did not become extremely rapid until after the 15th generation of selection and did not reach zero until the 30th generation.

In general, the most remarkable observation from the nine graphs is the consistency of the genetic correlation at all three levels of environmental variance and when the fraction of offspring saved as parents was as high as one-half. It was only when the fraction of offspring saved became as low as one fifth ($b = 0.2$) that the genetic correlation was considerably affected, and then the effect only became large when heritability of the selected trait was high ($h'_x = 0.7$). When selection level was low ($b = 0.8$), there was apparently little change in the genetic correlation over 30 generations of selection regardless of the heritability of the selected trait. There was clearly an interaction between level of selection and level of heritability, a rapid decrease in the genetic correlation requiring the combination of both high level of selection and high heritability. These results indicate that the levels of selection practised in animal species would not have much effect upon the magnitude of the genetic correlation unless heritability was very high. The theoretical model presented by Lerner (1958) in which he

suggested that selection would result in the genetic correlation declining to zero, does not hold over the 30 generations of selection practised here unless both selection intensity and level of heritability are high.

The correlated response of trait Y to selection of trait X is dependent on the genetic covariance between the traits. Falconer (1960) shows that the correlated response can be estimated as $h_x h_y r_G \sqrt{V(P_y)}$ which reduces to $\frac{\text{cov } G_x G_y}{\sqrt{V(P_x)}}$, showing that the correlated response is a function of the genetic covariance. The genetic correlation measured as $\frac{\text{cov } G_x G_y}{\sqrt{V(G_x)\ V(G_y)}}$ could remain at a high level even though the genetic covariance was decreasing if the genetic variances of the two traits were decreasing proportionately.

To examine the change in the genetic correlation more closely, the components of the correlation, the genetic covariance and the genetic variances of the two traits, are graphed individually. These are presented in Figures 2.1 to 2.9. The solid line again indicates the change in the genetic correlation measured in the unselected offspring each generation while the broken lines indicate the covariance and the two genetic variances.

At low selection intensity and low heritability (Figure 2.1), the genetic covariance was maintained quite well over the 30

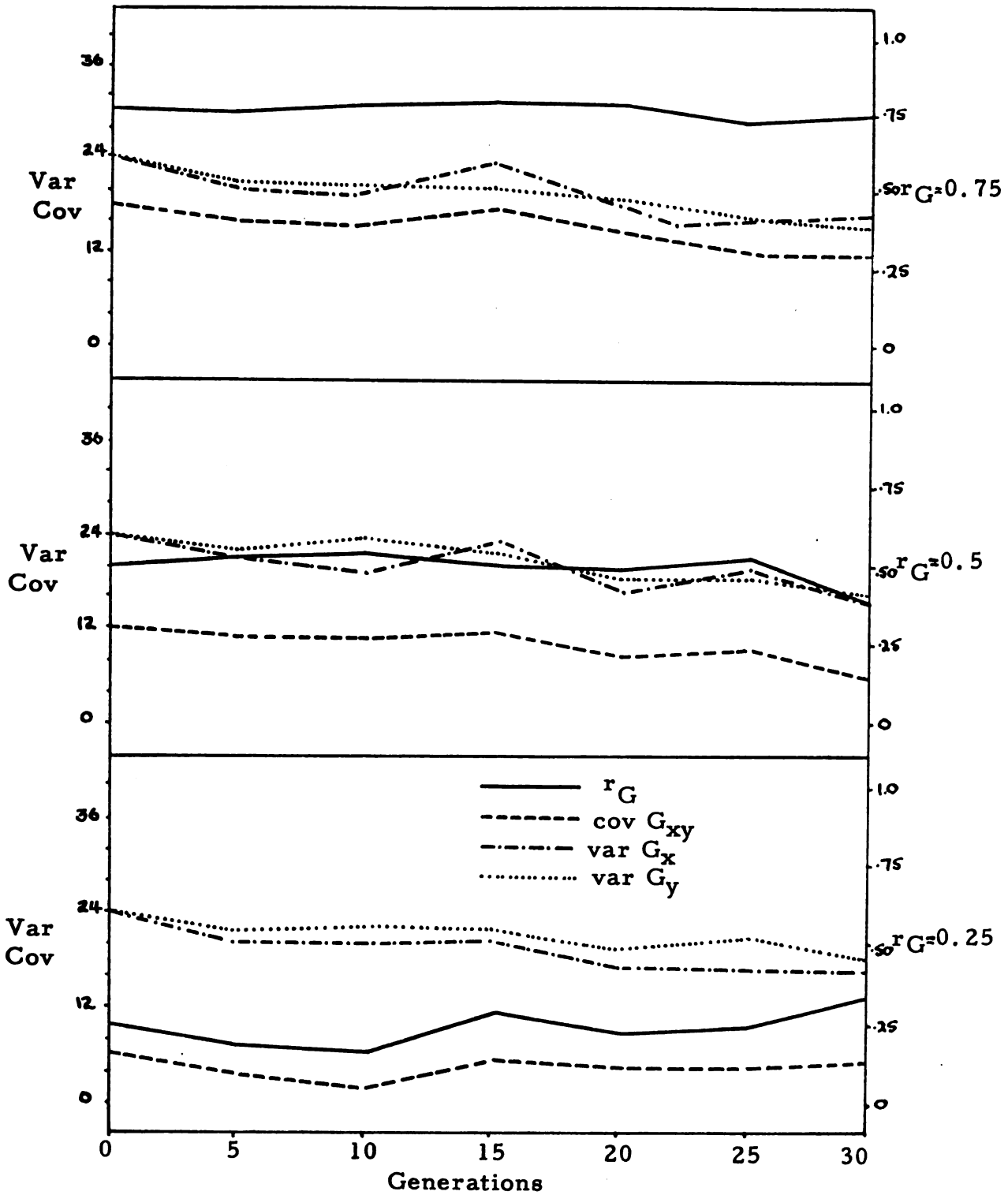

Figure 2.1 The relationship between the genotypic variances, covariance, and genetic correlation at three levels of correlation when b=0.8 and h_x^2 = 0.1 (additive model).

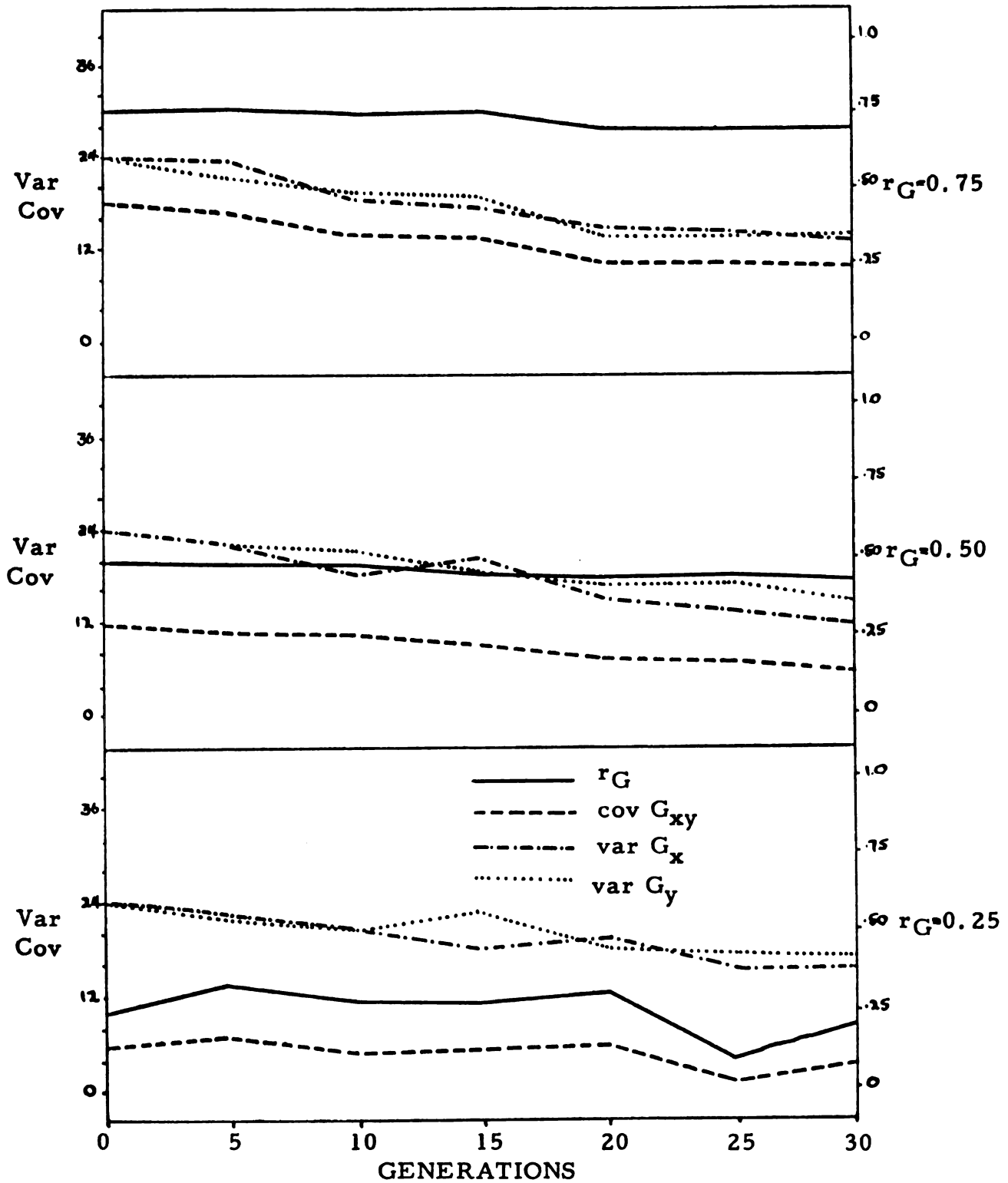

Figure 2.2 The relationship between the genotypic variances, covariance, and genetic correlation at three levels of correlation when b=0.5 and h^2_x = 0.1 (additive model).

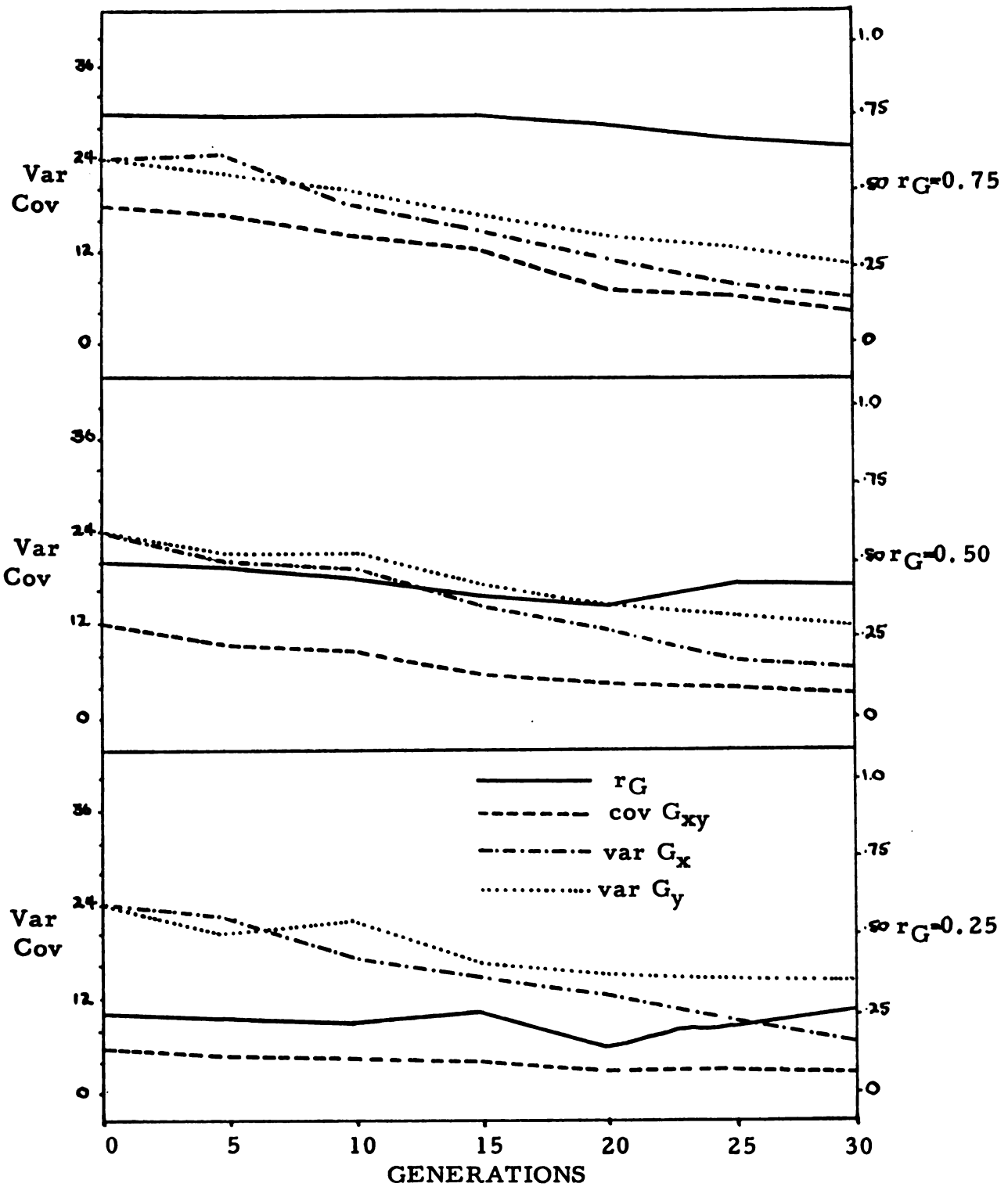

Figure 2.3 The relationship between the genotypic variances, covariance, and genetic correlation at three levels of correlation when b=0.2 and h'_x=0.1 (additive model).

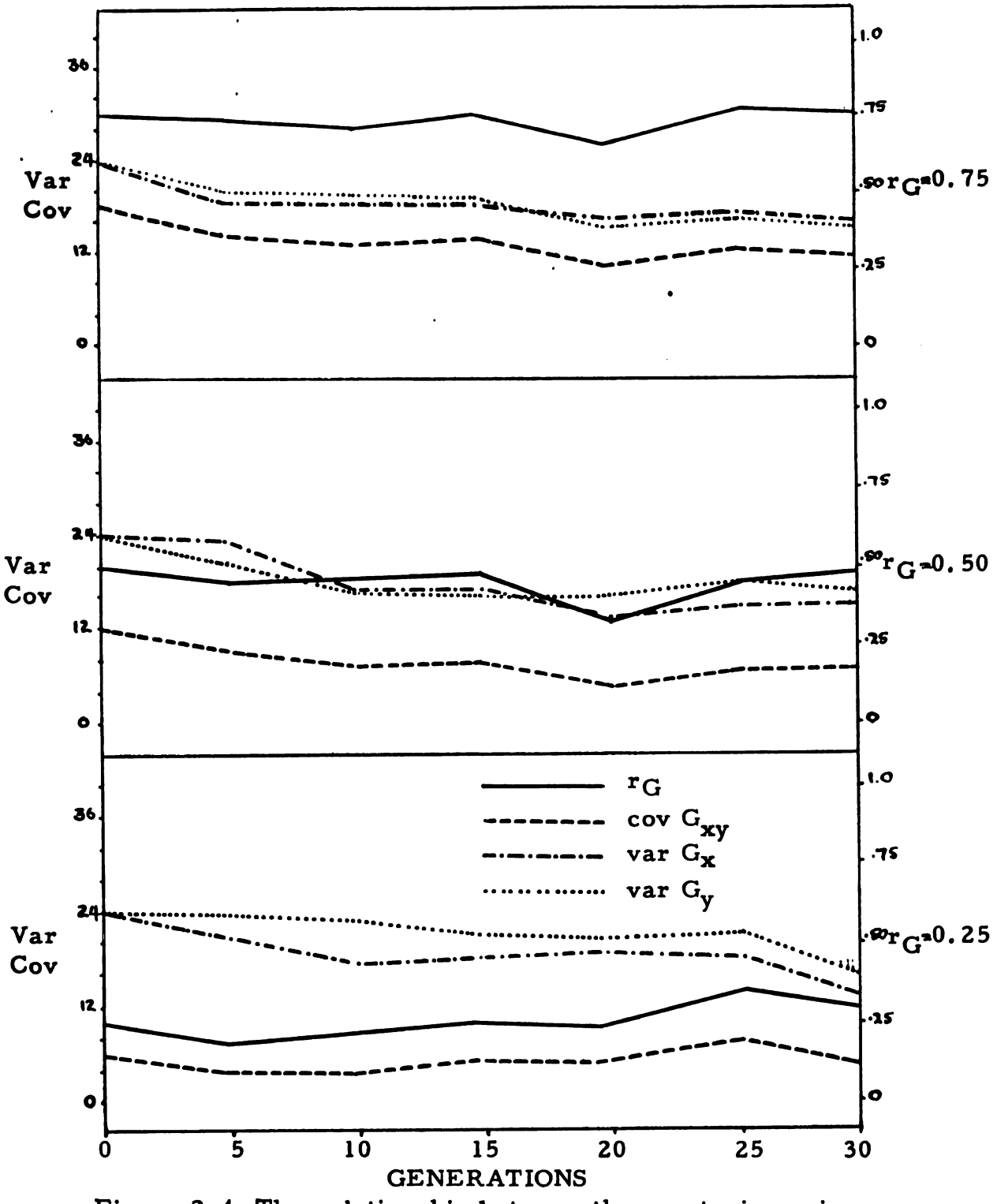

Figure 2.4 The relationship between the genotypic variances, covariance, and genetic correlation at three levels of correlation when b=0.8 and h^2_x = 0.4 (additive model).

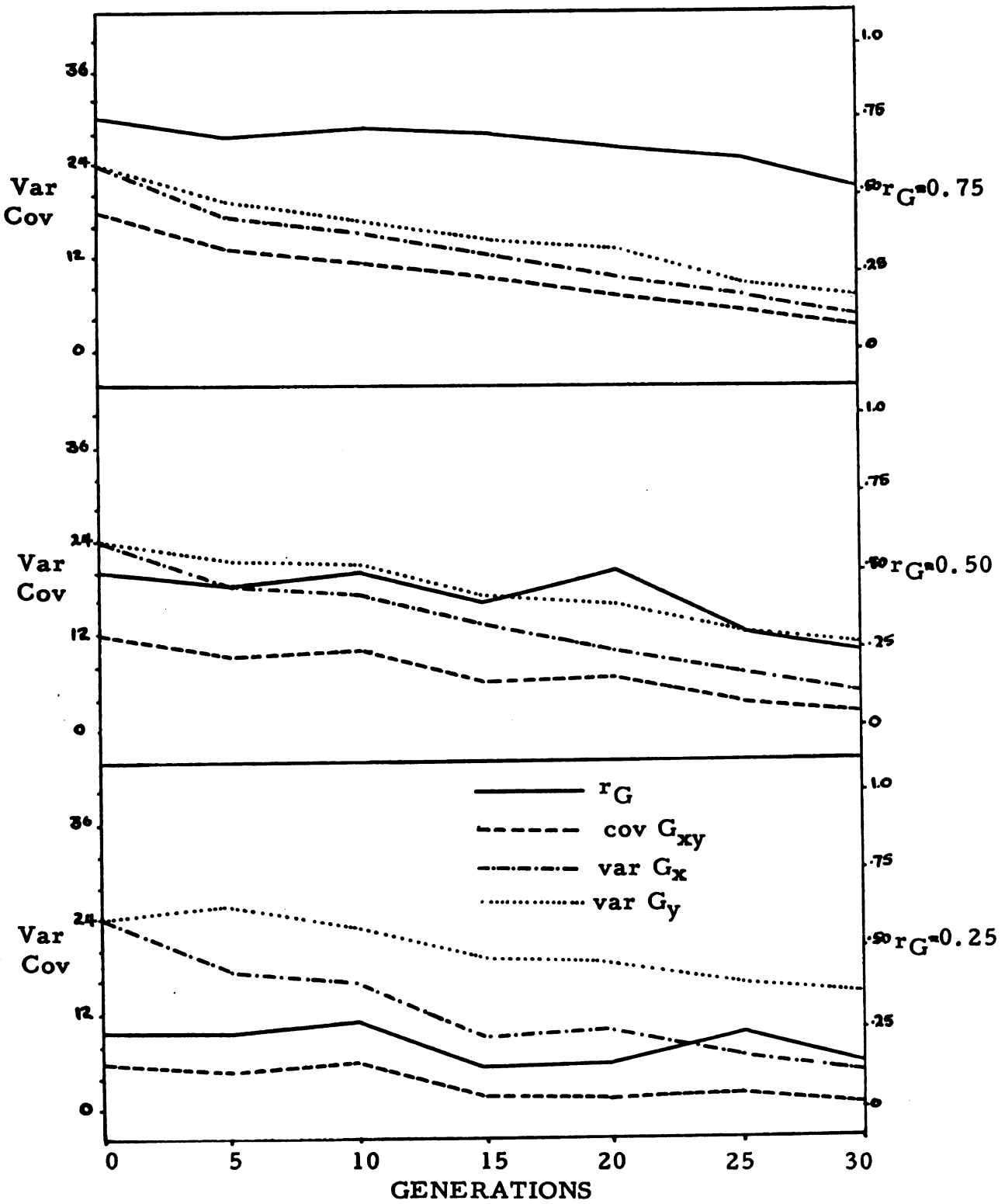

Figure 2.5 The relationship between the genotypic variances, covariance, and genetic correlation at three levels of correlation when b = 0.5 and h^2_x = 0.4 (additive model).

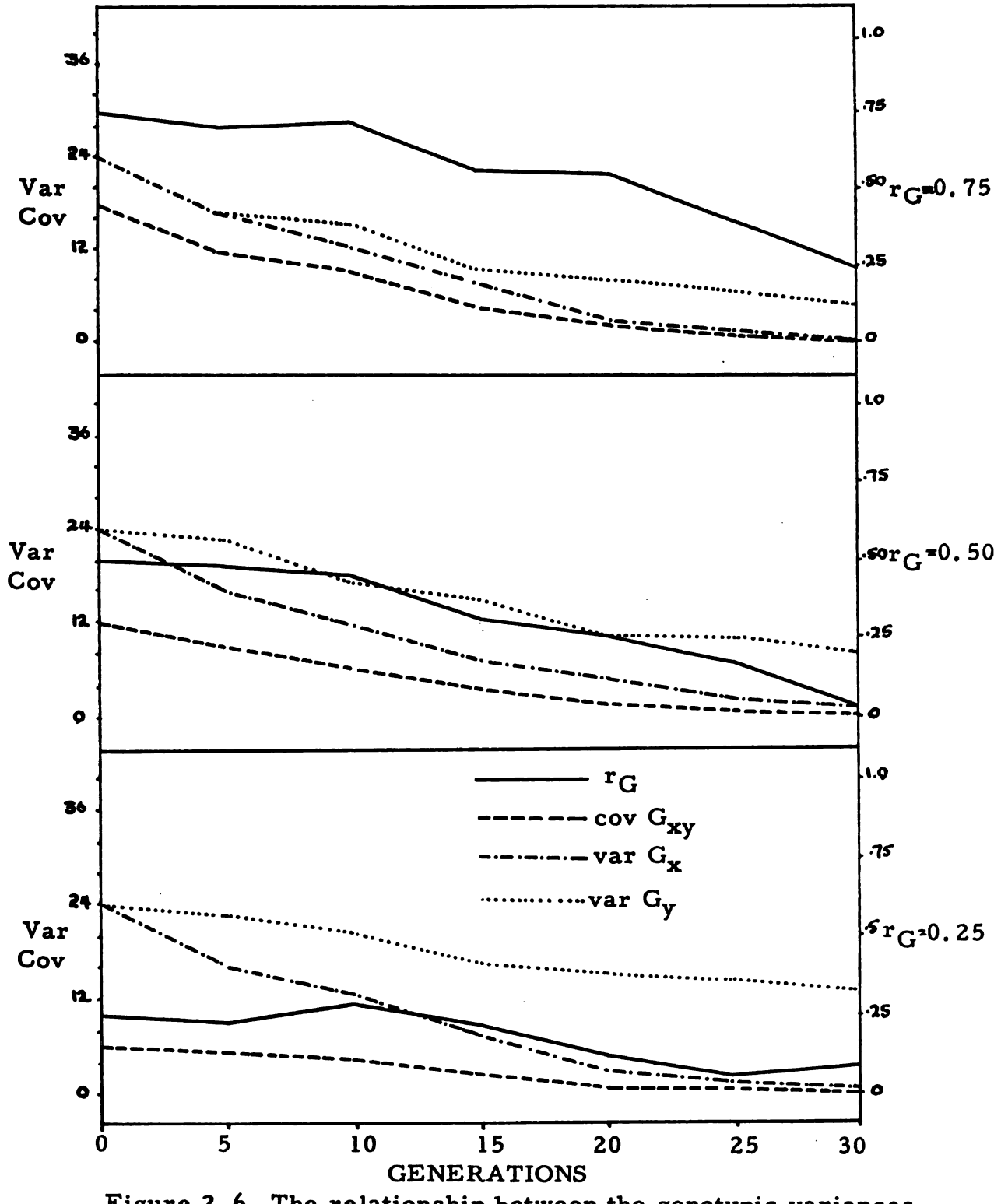

Figure 2.6 The relationship between the genotypic variances, covariance, and genetic correlation at three levels of correlation when b=0.2 and h^2_x = 0.4 (additive model).

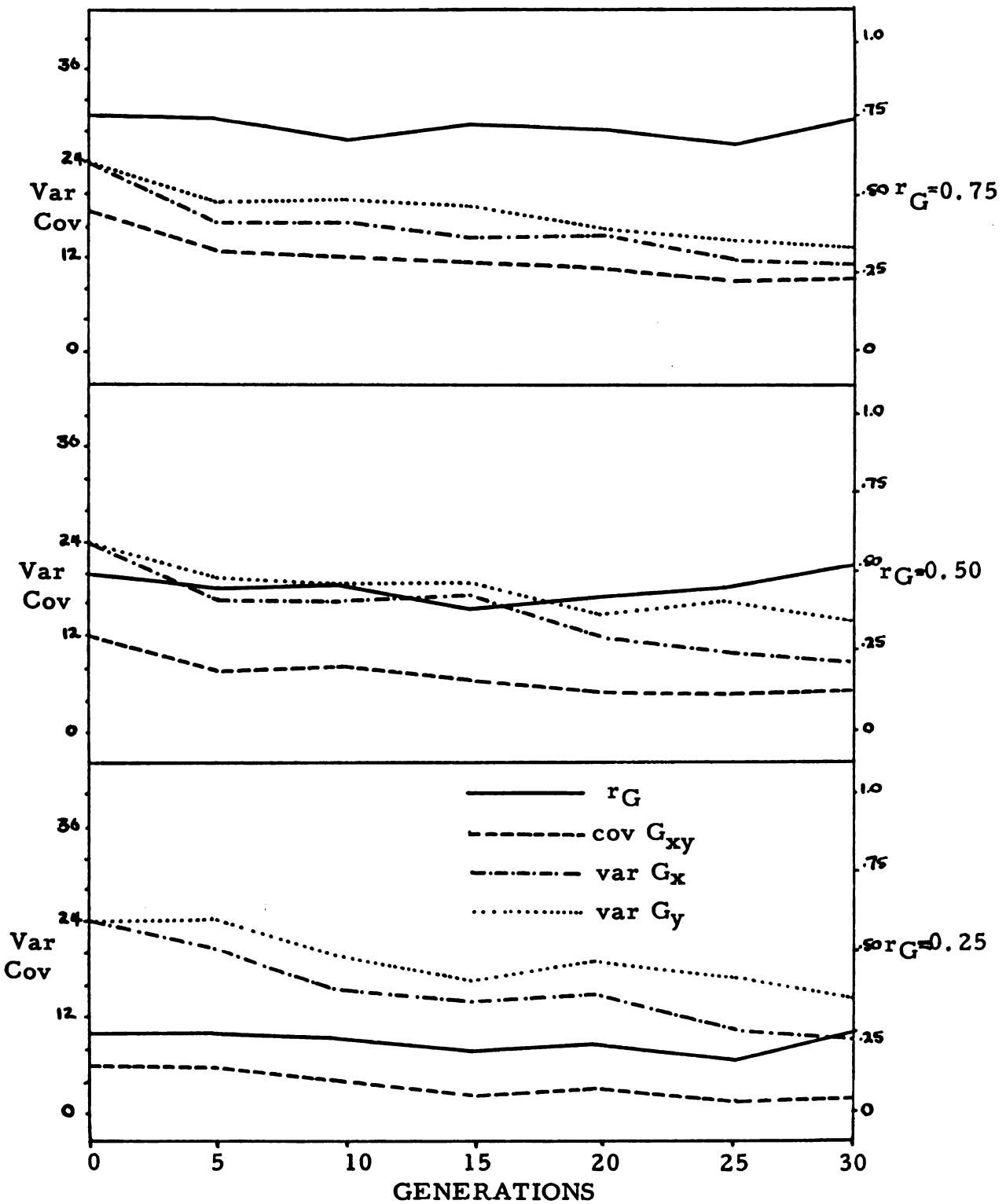

Figure 2.7 The relationship between the genotypic variances, covariance, and genetic correlation at three levels of correlation when b=0.8 and h^2_x=0.7 (additive model).

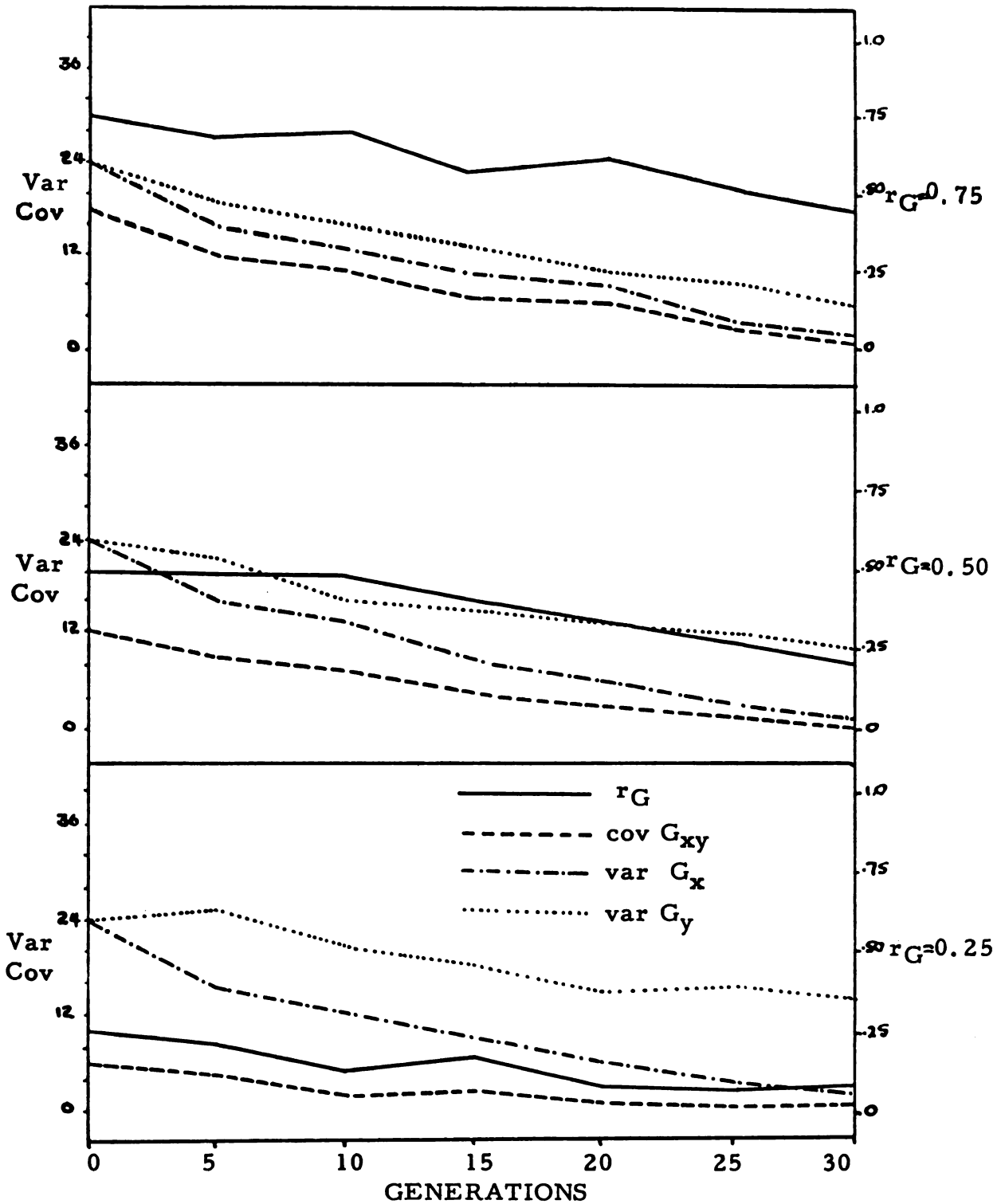

Figure 2.8 The relationship between the genotypic variances, covariance, and genetic correlation at three levels of correlation when b=0.5 and h^2_x = 0.7 (additive model).

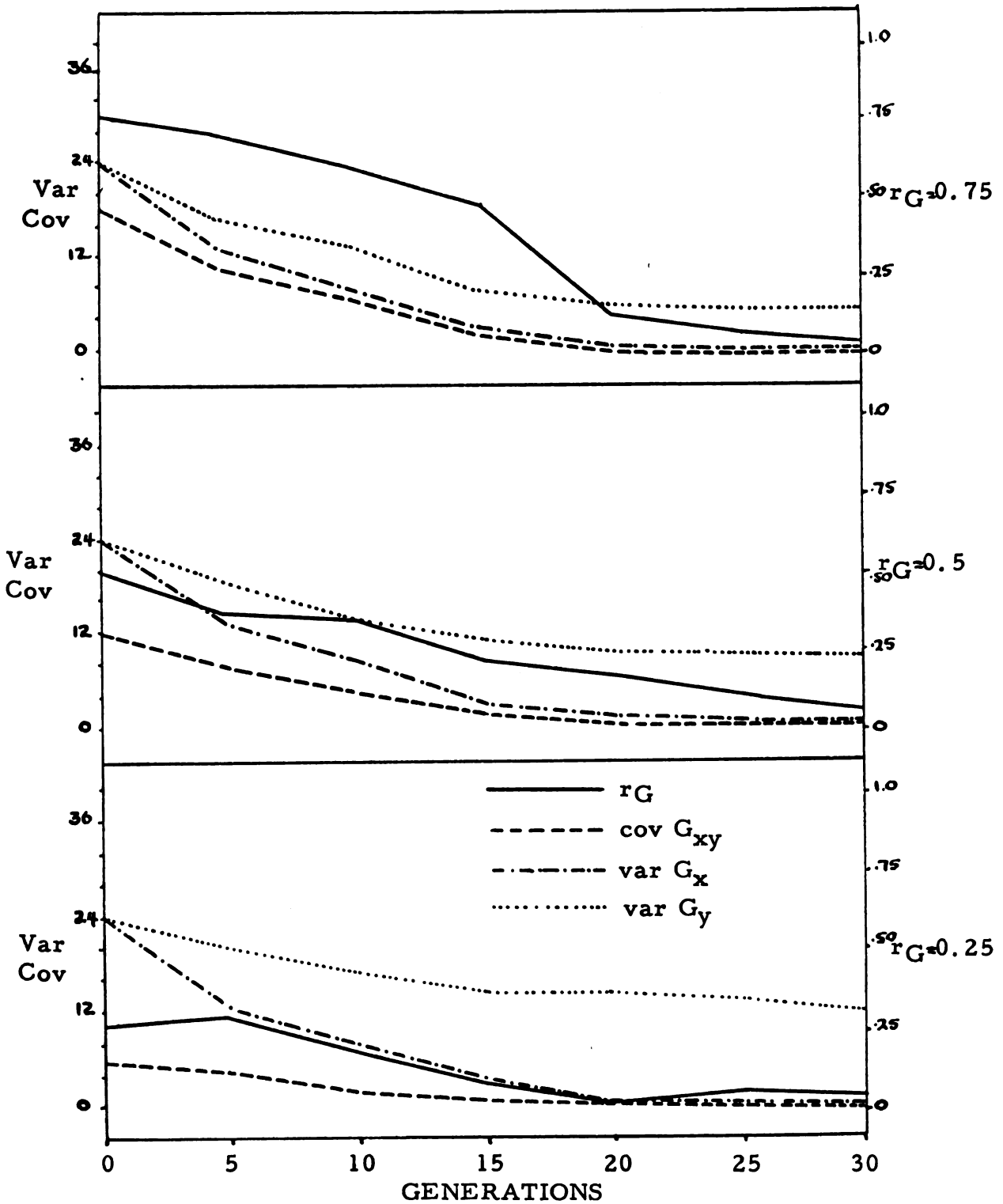

Figure 2.9 The relationship between the genotypic variances, covariance, and genetic correlation at three levels of correlation when b=0.2 and h_x^2=0.7 (additive model).

generations of selection, and the genetic correlation remained near its initial level in every case. The genetic covariance was also conserved at the remaining two levels of environmental variance when b = 0.8 (Figures 2.4, 2.7). Thus, when level of selection was low, the genetic covariance remained quite high over all levels of environment, the greatest decrease being at low environmental variance (h'_x = 0.7) when genetic covariance was about halved over the 30 generations of selection. (Figure 2.7)

With increasing level of selection there was a greater decrease in the covariance. At (b = 0.5), for example, the covariance clearly decreased at all levels of heritability while the correlation remained high and only decreased noticeably at h'_x = 0.7 (Figures 2.2, 2.5, 2.8). For initial r_G of 0.75 in Figure 2.2 the genetic correlation was still 0.7 at the 30th generation while the covariance had dropped from 18 to almost 9 or by about 50 per cent. The genetic variances of the two traits had also declined in proportion to the covariance causing the correlation to remain high.

When the selection intensity was high (b = 0.2), the genetic covariance quickly declined at all levels of environmental variance (Figures 2.3, 2.6, 2.9). In Figure 2.6 when initial r_G was 0.75, the genetic covariance had already dropped from

18 to about 2 by the 20th generation while the genetic correlation had only decreased from 0.75 to about 0.55. In general, at low levels of selection both the genetic covariance and variance were maintained over the 30 generations of selection. At higher levels of selection, however, there was a distinct downward trend in the genetic covariance; but because of an accompanied decrease in the denominator of the correlation coefficient, 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. This sudden decrease can be clearly seen in Figure 2.9 where the genetic correlation, initially at 0.75, was still close to 0.50 at the 15th generation despite a rapid decrease in the genetic covariance. By the 20th generation, however, the genetic correlation had dropped almost to 0.1 at a time when the decrease in the genetic covariance was leveling out.

A reduced correlation could, in fact, be due to an increase in genetic variance rather than a decrease in genetic covariance. An example can be seen in Figure 2.1 when the initial genetic correlation was 0.5; there was a small decrease in the genetic correlation between the 10th and 15th generations. Yet, the

genetic covariance increased somewhat in the same period, the reduction in the genetic correlation being due to an increase in the genetic variance of the selected trait. Thus, the genetic correlation does not necessarily provide a reliable estimate of the genetic covariance.

The Genetic Correlation in the Truncated Distribution.

The effect of linear truncation of one variable on the marginal distribution of a correlated variable has been discussed previously. In general, the conclusion was that the correlation observed within the sample of individuals selected as parents will be lower than that observed within the population of all offspring. The theoretical treatment of this problem by Aitken (1964) and by Mantel (1966) has already been reviewed and was concerned solely with the phenotypic correlation between the variables. Whether the same effect would hold for the genetic correlation has been examined by measuring the genetic correlation each generation in those offspring selected to be parents of the next generation. The phenotypic correlation is a function of both the genetic correlation and heritability and also of any environmental correlation between the traits. Thus a reduction in the phenotypic correlation between the variables might not necessarily mean a reduction in the genetic

correlation.

Figures 1.1, 1.2, and 1.3 show the genetic correlation both in the complete offspring generation (solid line) and in those offspring selected as parents or in the truncated distribution (broken line). Clearly, truncation has caused some decrease in the genetic correlation. This decrease is apparently a function of heritability rather than of degree of truncation selection. When the environmental variance was high relative to the genetic variance ($h'_x = 0.1$), there was some tendency for the genetic correlation in the selected groups to be lower. The difference, however, was quite small and not consistent. Again at $h'_x = 0.4$ (Figure 1.2) the difference between the two correlations did become larger, but neither level of selection nor initial degree of genetic correlation had any appreciable effect although the difference did seem rather more consistent when initial r_G was 0.75 than otherwise. When the environmental variance was high relative to genetic variance (Figure 1.3), the effect of truncation selection on the genetic correlation became considerably greater. There was a more consistent reduction in the genetic correlation, and the reduction was greater. Again there was little effect of level of selection on this decrease. The results shown in Figure 1.3 correspond most closely with those expected in the phenotypic correlation

since the expectation of the phenotypic correlation approaches the genetic correlation when heritabilities are high.

The effect of truncation selection of one variable is to reduce the observed genetic correlation between it and another variable and should be kept in mind in selection practice. The amount of this reduction depends markedly on the heritability of the selected trait rather than on the intensity of selection practised. A more detailed examination of this problem, including the effect of selection on the environmental and phenotypic correlations and on the heritabilities as well as on the genetic correlation, should be carried out to clarify the effect of selection on all of these parameters and on their interrelationships. The statistics required to examine these relationships were available, but a detailed investigation was considered beyond the scope of the present study.

The Estimates of Genetic Correlation from Phenotypic Covariances between Parent and Offspring.

In addition to measuring the genetic correlation from the product-moment correlation of genic values, two separate estimates were obtained from the covariances between phenotypes of parent and offspring. The two methods were:

a) $$\hat{r}_G = \sqrt{\frac{(covPxpPyo).(covPypPxo)}{(covPxpPxo).(covPypPyo)}}$$

b) $$\tilde{r}_G = \frac{(covPxpPyo)+(covPypPxo)}{2\sqrt{(covPxpPxo).(covPypPyo)}}$$

where Pxp = phenotypic value of trait X in the parent

Pyp = " " " " trait Y " " "

Pxo = " " " " trait X in the offspring

Pyo = " " " " trait Y " " "

Since the number of observations upon which the genetic correlation is estimated is known to have considerable effect on the precision of the estimate, it should be emphasized at the outset that the sample size available here was small and varied with the level of selection practised. The number of observations on which the estimates were made was 60, 96, and 240 when the level of selection b was 0.8, 0.5 and 0.2, respectively.

Results of both methods were extremely erratic and were almost impossible to interpret. Extreme selection of parents could be expected to bias the correlation, and extreme selection occurred here when the number of observations was largest. When level of selection was low, the number of observations was small resulting, in both cases, in unreliable estimates.

Lush (1948) has suggested that where sampling errors are a major concern, as when the volume of data is small, it may be better to use an arithmetic mean of the two covariances in the numerator rather than the geometric mean. Thus, although both methods of estimation gave completely unintelligible results, a few examples of results when the arithmetic mean of the two covariances in the numerator was used are presented.

The results are in Table 1 and are the deviations of the genetic correlations measured by parent-offspring covariances from the product-moment correlations of genic values. Only two levels of selection were considered, $b = 0.8$ and $b = 0.2$, in an attempt to detect any difference due to number of observations. In addition, five different combinations of environmental variances are presented to examine as wide a range of these effects as practicable. Sample estimates are given for the first replicate for generations 2 through 6 and then for every fifth generation thereafter. To distinguish any pattern in the results is futile. Most estimates fluctuate markedly and apparently randomly from the true correlation. In fact, it is rather rare to find an estimate within ± 0.20 of the expected correlation. In general, however, there is some tendency for the correlation to be considerably underestimated. Of the 300 deviates shown in Table 1, 179 were

TABLE 1. Deviation of the genetic correlation by parent-offspring covariances from the product-moment correlation of genotypic values (Additive Model)

				GENERATION									
r_G	b	h'_x	h'_y	2	3	4	5	6	10	15	20	25	30
.25	0.8	0.1	0.1	-0.01	-0.79	0.24	-1.19	1.06	0.71	0.21	0.50	0.09	-7.51
"	"	0.1	0.7	-0.04	0.04	-1.34	1.24	0.53	4.82	0.45	-0.01	0.65	-0.92
"	"	0.4	0.4	-0.53	-0.70	-1.15	-0.30	-0.80	-0.90	1.93	0.01	-0.64	0.30
"	"	0.7	0.1	1.68	0.43	1.13	-0.74	-0.55	-1.64	-0.23	-0.03	-1.38	0.19
"	"	0.7	0.7	-0.37	-0.59	0.20	0.16	-2.32	1.08	0.17	1.90	1.02	-1.63
"	0.2	0.1	0.1	-4.61	-3.76	-0.88	0.22	-0.34	1.97	0.18	-0.42	-0.27	-4.20
"	"	0.1	0.7	1.35	-1.89	-2.16	-0.11	0.52	-0.33	-0.73	-0.14	-9.33	-0.86
"	"	0.4	0.4	-1.57	-2.41	-0.91	-0.42	-0.48	-1.44	-0.25	-0.72	1.42	0.64
"	"	0.7	0.1	0.89	0.15	-0.80	0.96	-0.96	-0.65	-0.32	-1.18	0.44	4.72
"	"	0.7	0.7	-1.54	-0.26	0.07	-0.53	-1.85	-0.38	1.72	0.26	1.43	0.43
.50	0.8	0.1	0.1	-0.74	-0.17	-0.34	-1.57	-2.61	0.47	-0.24	0.23	-0.13	0.41
"	"	0.1	0.7	-0.06	0.37	0.03	0.04	-2.28	0.51	-1.37	-1.13	0.02	0.04
"	"	0.4	0.4	5.34	-3.45	-0.03	-1.29	-0.44	-1.25	-0.68	0.15	0.43	1.47
"	"	0.7	0.1	-0.53	0.36	0.57	0.09	-1.03	-1.19	-2.62	0.29	-0.77	-2.65
"	"	0.7	0.7	0.28	0.70	-0.12	-0.63	0.09	1.31	0.45	-0.25	-0.28	0.78
"	0.2	0.1	0.1	-0.18	-1.30	-5.34	0.13	-3.01	-0.02	-1.03	-1.33	-0.07	-2.06
"	"	0.1	0.7	-1.12	2.34	0.06	-1.29	-0.96	-4.23	0.70	-1.46	-0.33	3.39
"	"	0.4	0.4	0.16	-5.82	-1.93	-4.67	1.70	0.96	-1.25	-0.32	2.29	-1.45
"	"	0.7	0.1	-2.41	0.21	-1.58	-2.55	-0.49	-1.59	0.04	-1.77	-0.01	2.32
"	"	0.7	0.7	-1.03	0.02	0.23	-5.19	-5.66	-3.65	0.93	3.67	2.57	4.55

TABLE 1. (continued)

				GENERATION									
rG	b	h'_x	h'_y	2	3	4	5	6	10	15	20	25	30
.75	0.8	0.1	0.1	1.24	-1.53	0.21	1.19	-0.31	1.16	-0.78	0.43	-0.81	-1.16
"	"	0.1	0.7	2.48	0.75	0.44	-3.65	2.94	-0.22	-0.54	-2.47	0.72	-0.03
"	"	0.4	0.4	1.87	0.18	-1.77	-0.59	0.10	-0.67	-0.46	-0.90	-0.40	3.56
"	"	0.7	0.1	-2.83	-2.13	-3.71	9.67	-0.15	-1.74	-0.63	-0.93	-0.70	0.47
"	"	0.7	0.7	-2.74	-0.99	-0.52	-4.00	0.08	1.66	1.33	-0.97	-0.15	-0.94
"	0.2	0.1	0.1	2.57	5.79	-6.00	-1.74	-2.39	0.32	1.10	-0.30	-0.89	-0.46
"	"	0.1	0.7	-1.88	-2.45	-0.44	1.42	-1.32	0.53	1.49	-0.72	-0.83	2.13
"	"	0.4	0.4	5.04	-0.69	-0.74	-2.14	-1.40	-1.01	-0.56	0.43	-2.93	-0.95
"	"	0.7	0.1	-0.83	-1.24	-1.02	0.42	4.80	-6.04	-1.89	-7.36	2.19	-3.06
"	"	0.7	0.7	-1.45	1.15	-2.32	-2.07	0.21	0.33	0.38	-1.14	2.13	1.36

negative, underestimating the true correlation. The overall average deviation was - 0.33. When the level of selection was low (b = 0.8) and thus, the number of observations small, the average deviation was - 0.18; while at b = 0.2 and number of observations 240, the average deviation was - 0.59. The general tendency for the true correlation to be underestimated increased with intensity of selection. However, the complexity and magnitude of the bias and of the 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 a population of the size simulated in this study and when intensity of selection is extreme.

In the results of the additive model presented here, no account was taken of estimates in which the two covariances in either the numerator or denominator were of unlike sign because no geometric mean is possible from the root of a negative number. The cases where this occurred were identified in the two experiments under the model of complete dominance and will be discussed later.

Correlated Response to Selection.

The results of primary interest were the changes in the

genotypic mean of trait X at all combinations of selection intensity and of environmental variation and the correlated change in the genotypic mean of Y at three levels of genetic correlation between the two traits. For the additive model the expected genotypic mean for the first generation of offspring was 48 for both traits X and Y. The changes can be seen best by the behaviour of the graphs of genotypic means plotted against the generation number for each combination of conditions. The graphs are presented in Figures 3.1, 3.2 and 3.3. The solid line represents the response in trait X to direct selection while the broken lines indicate the correlated response of trait Y to selection for X at three degrees of genetic correlation between X and Y. Again the agreement between replicates was close so that repeated runs were averaged. Also, since the correlated response is independent of the heritability of the correlated trait, the means in the correlated trait were averaged over the three levels of environment. Each point on the graph for trait Y represents the average of six observations. The response in trait X, directly selected, is independent of both the heritability of the correlated trait and of the degree of genetic correlation between the two traits, and each point in the graph for trait X represents

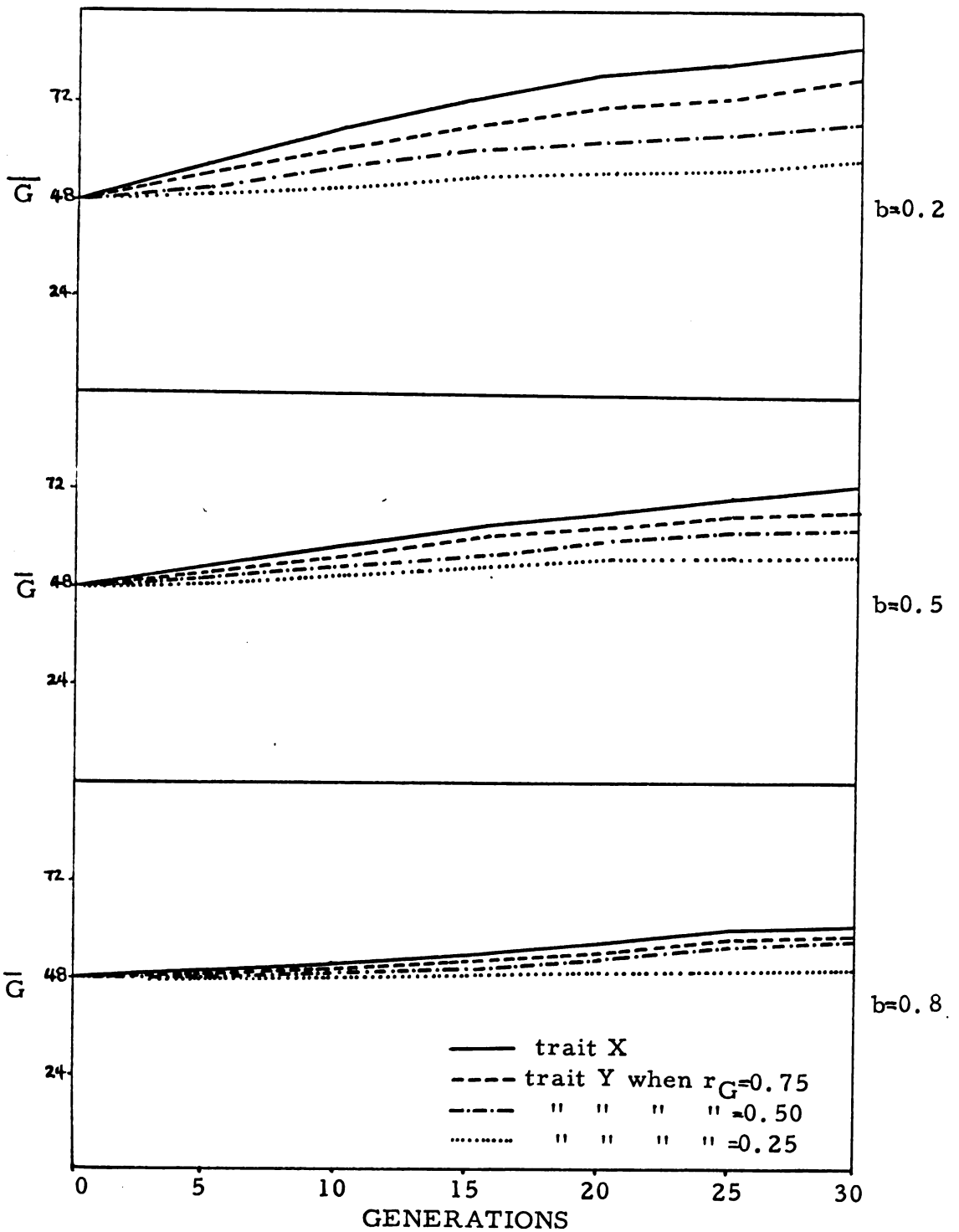

Figure 3.1 Mean genetic progress at three levels of selection when $h^2_x = 0.1$ (additive model).

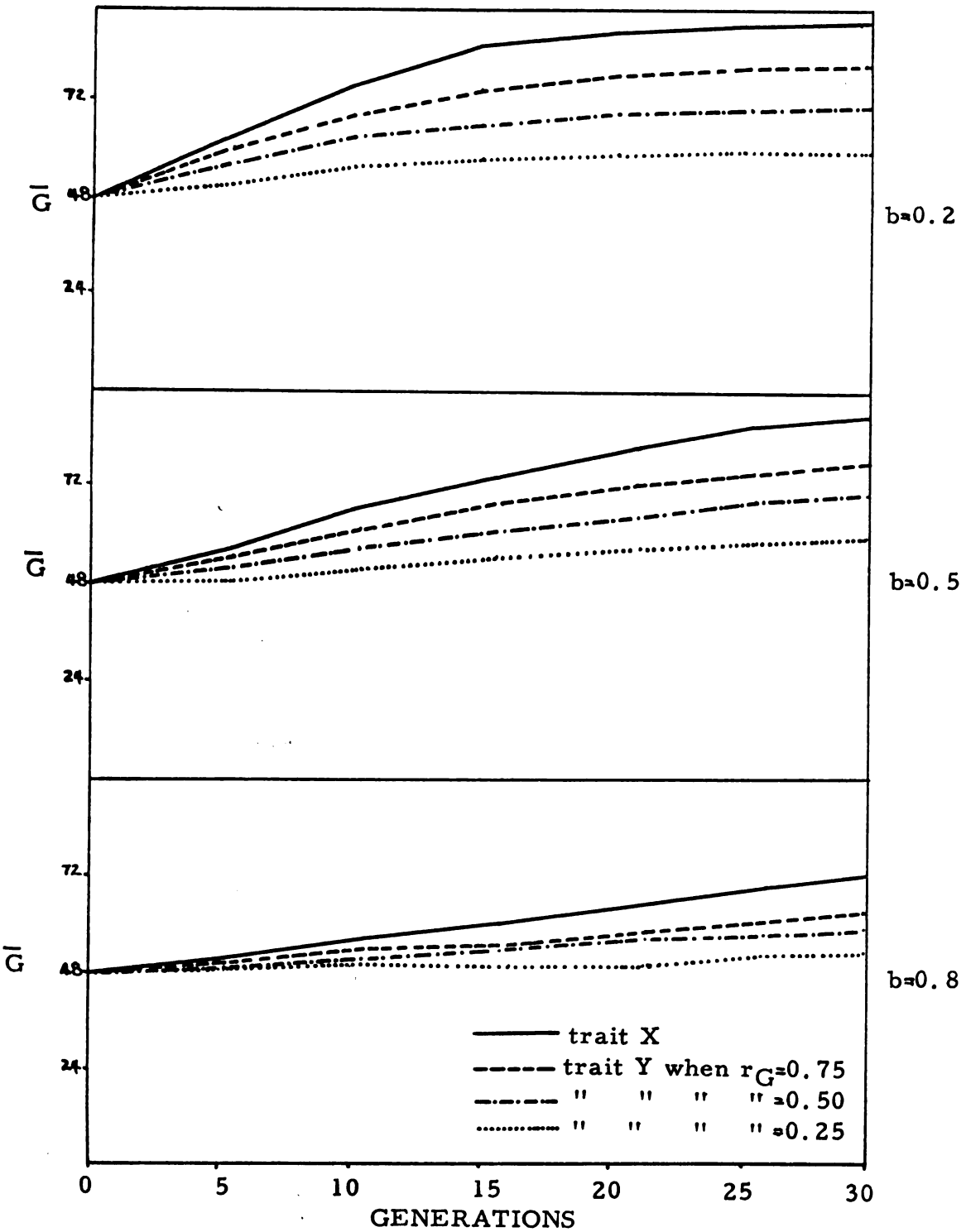

Figure 3.2 Mean genetic progress at three levels of selection when $h^2_x = 0.4$ (additive model).

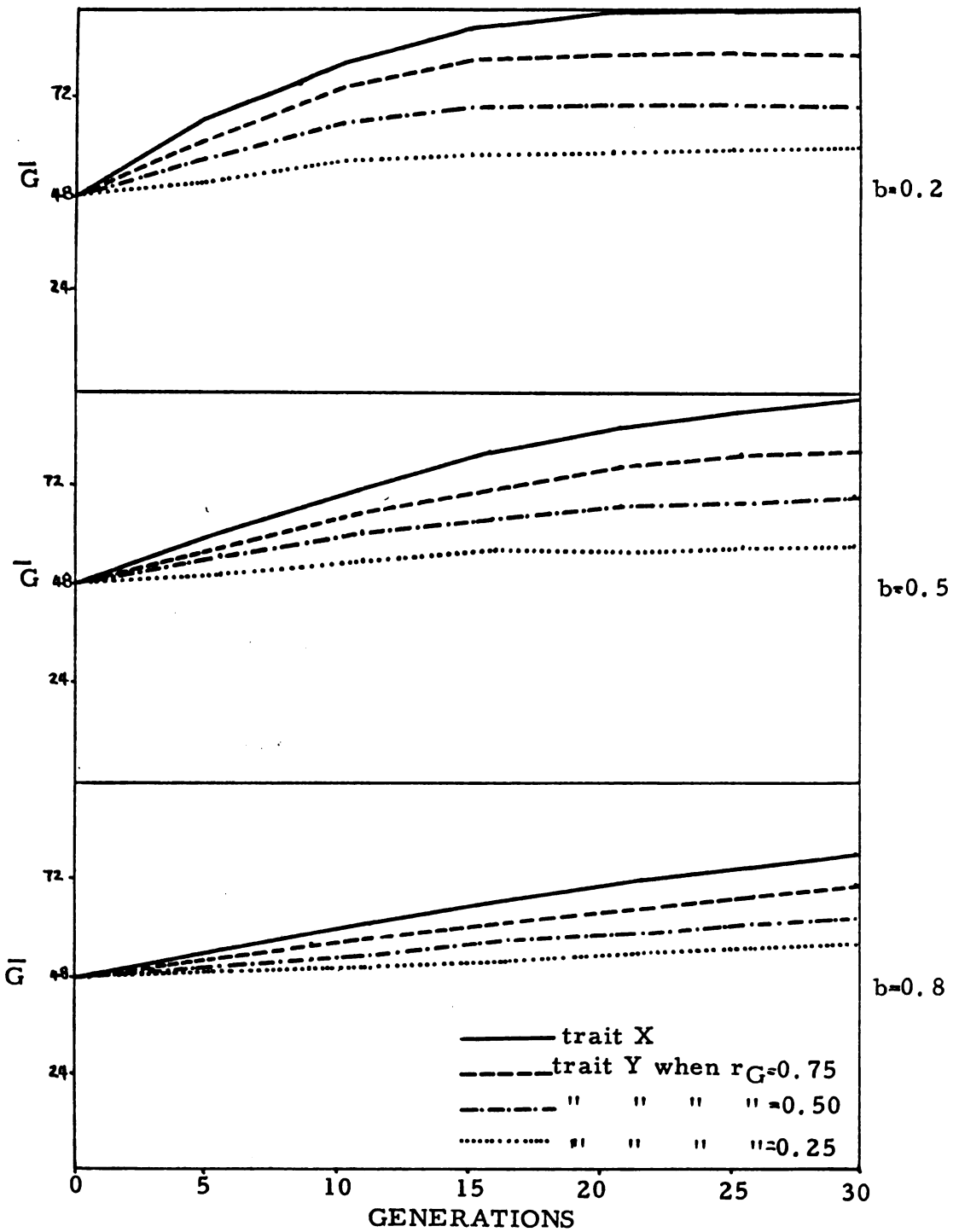

Figure 3.3 Mean genetic progress at three levels of selection when $h^2_x = 0.7$ (additive model).

the average of 18 observations. Again, as in the presentation of the genetic correlations, only every fifth generation has been plotted.

Under the assumption of normal distribution of phenotypic values, the corresponding means of the offspring selected to be parents in terms of standard deviation (z/b) for the truncation selection of 0.8, 0.5 and 0.2 are 0.35, 0.80 and 1.40, respectively. The numbers of progeny produced to simulate the above intensities of selection were 60, 96, and 240. The direct response to selection of trait X is given by $\sqrt{V(P_x)}h_x^2\ z/b$ where $\sqrt{V(P_x)}$ is the phenotypic standard deviation of X and h_x^2 is the heritability. The above formula reduces to $\frac{z}{b}\ \frac{V(G_x)}{\sqrt{V(P_x)}}$ where $V(G_x)$ is the genic variance of the selected trait. The correlated response of trait Y to selection for trait X is given by $h_x h_y r_{G_x G_y} \sqrt{V(P_y)}\ z/b$ which reduces to $\frac{z}{b}\ \frac{\mathrm{cov}G_x G_y}{\sqrt{V(P_x)}}$.

Thus the direct response to selection $= \frac{z}{b}\ \frac{V(G_x)}{\sqrt{V(P_x)}}$

The correlated response to selection $= \frac{z}{b}\ \frac{\mathrm{cov}G_x G_y}{\sqrt{V(P_x)}}$

Therefore, the ratio of the correlated response of Y to selection for X to the direct response of X is simply $\frac{\mathrm{cov}G_x G_y}{V(G_x)}$. Now, as previously shown, the genotype of X is made up of contributions of loci A which affect X alone and of loci B which affect X and

also affect Y equally in the same direction. The genotype of Y is made up of contributions of loci A' which affect Y alone and of loci B.

Thus, cov $G_x G_y$ = cov $(G_A+G_B)(G_{A'}+G_B)$

= $V(G_B)$ since all other covariances are expected to equal zero.

Therefore, the ratio of the correlated response to the direct response is simply $\frac{V(G_B)}{V(G_X)}$ so that the observed correlated response of Y to selection for X should be in direct proportion to the response of X, depending on the number of loci shared by the two traits.

The results shown graphically in Figures 3.1, 3.2 and 3.3 indicate that the expected correlated response occurred in almost every case. Perhaps the only exception was in the case of low heritability and low selection intensity (bottom graph of Figure 3.1). There the expected response in Y when the genetic correlation was low was not observed. This lack of response could be attributed to random sampling in the correlated trait. When the genetic correlation is low, 36 of the 48 loci affecting trait Y are under no selection pressure and could have considerable random effect on the genotypic mean especially when the number of observations is small as is the case when $b = 0.8$.

When either level of selection was low or heritability was low or both, the response to selection was linear over all 30 generations for trait X and for trait Y at all three levels of genetic correlation. As expected, the response increased as heritability increased or as selection intensity increased. At intermediate or low environmental variance (Figures 3.2, 3.3) the response became distinctly curvilinear to the selection goal, especially in trait X. Only when heritability was high and selection intensity high (Figure 3.3 top graph) was the selection goal for X reached and then at the 30th generation. The results agree very well with expected response, although random fluctuations could be important in the correlated trait when the heritability and genetic correlation are both low.

The selection goal or expected maximum advance from selection is different depending on the type selection practised. With direct selection the goal is 96 for all cases. With indirect selection the goal is 60, 72, or 84 when the genetic correlation between the traits is 0.25, 0.50 and 0.75, respectively. For example, in selecting directly for X, the genotypic mean can be moved from its initial value of 48 to 96 at which time all loci would be homozygous for the plus allele. When the genetic correlation was 0.50 the response in Y would have a limit of 72; that is, when the 24 loci shared with X were homozygous for the plus

allele contributing 48 units to the genotypic mean, and the remaining 24 loci contribute only 24 units on the average, the same as in the initial generation, since these 24 loci were under no selection pressure. Thus the selection goal for the correlated trait would be 72.

In order to illustrate better the agreement of the results with those expected from theoretical considerations, the responses in X and Y at the 15th and 30th generations of selection have been presented in a different way in Table 2. The response has been determined as the per cent progress toward the selection goal. Since the selection goal differs depending on whether the trait was selected directly, and, if not, on the genetic correlation, this method provides a better basis of comparison of the different conditions. The formula used was:

$$R_{15} = \frac{\bar{G}_{15} - \bar{G}_0}{L_{30} - \bar{G}_0} \times 100 \text{ or } R_{30} = \frac{\bar{G}_{30} - \bar{G}_0}{L_{30} - \bar{G}_0} \times 100$$

where R_{15} or R_{30} is the per cent progress toward the selection goal at the 15th or 30th generation of selection. $\bar{G}15$ or $\bar{G}_{30}$ is the observed genotypic mean at the 15th or 30th generation.

TABLE 2. The response in trait X and correlated response in Y at three levels of genetic correlation, measured as per cent of selection goal achieved at the 15th and 30th generation. (Additive model).

Level of Selection b	Level of Environment h'_x	Generation	Response to Selection as per cent of Selection Goal			
			TraitX	TraitY r_G=0.25	TraitY r_G=0.50	TraitY r_G=0.75
0.8	0.1	15	13.1	5.0	17.5	14.9
		30	25.6	18.3	34.4	27.7
	0.4	15	25.4	13.9	22.2	21.3
		30	48.5	41.1	43.6	41.9
	0.7	15	34.6	34.2	32.8	33.0
		30	62.9	68.1	62.1	59.1
0.5	0.1	15	30.0	35.3	32.0	31.2
		30	54.2	66.4	62.5	50.6
	0.4	15	53.9	52.3	55.1	52.5
		30	86.0	92.3	90.8	85.8
	0.7	15	66.9	65.0	66.9	63.5
		30	96.5	82.3	94.9	94.0
0.2	0.1	15	53.1	49.8	52.2	53.3
		30	82.7	85.0	81.5	81.9
	0.4	15	80.2	93.1	84.5	79.4
		30	98.8	117.5	98.9	97.3
	0.7	15	92.9	96.9	92.4	95.3
		30	100.0	106.9	96.3	100.0

$\bar{G}_0$ is the initial genotypic mean and is equal to 48 in every case.

L_{30} is the selection goal or limit.

The Table substantiates what has already been observed in the graphs. When the level of selection was other than 0.8 or heritability was other than 0.1, there was very close agreement between the per cent response in X and that in Y when the genetic correlation was 0.5 or 0.75. This agreement was close at both the 15th and 30 generations of selection. When level of selection was low and the number of observations small, random sampling was apparently having a greater effect, especially when the genetic correlation was 0.25 or 0.50. The agreement was much less close for all combinations when the genetic correlation was low (0.25), the correlated trait sometimes exceeding the response of the selected trait and sometimes showing less response. Also, it was only when the genetic correlation was 0.25 that the response in Y actually exceeded the selection goal at the 30th generation. The discrepancies observed at low correlation or low level of selection could be attributed in every case to random sampling.

The correlated response to selection behaved almost entirely as expected from theoretical considerations. The amount of response in the unselected trait was directly proportional to

that in the selected trait and apparently depended entirely on the genetic covariance between the traits. Only when the genetic covariance was low or when level of selection was low did chance have any noticeable effect on the correlated response.

The Model of Complete Dominance

In the complete dominance model the contributions to the genotypic value were 2, 2, and 0, respectively, for the ++, + -, and -- genotypes at each locus. The three levels each of initial genetic correlation, intensity of selection, and environmental variation of X and Y again provided 81 replicated treatment combinations. In addition, selection was carried out in two directions, upwards for the dominant allele and downwards for the recessive allele, which resulted in two separate experiments each one made up of 162 parameter sets.

The results obtained for the model of complete dominance have been averaged over replicates and over the levels of environment of the correlated trait as was done in the additive model. The statistics calculated were also the same as in the additive model. The only difference was in the mode of gene action and, in one experiment, in the direction of truncation selection. The different mode of gene action resulted in a different contribution of the heterozygote to the genotypic value

and changed the expected values of the genotypic mean, variances, and covariance, for the two traits. There was no change in the expected genetic correlation. For the complete dominance model, the expected genotypic means, genotypic and environmental variances of traits X and Y, and the expected genotypic covariance and genetic correlation between the two traits in the first generation of offspring were:

i. Expected genotypic mean was 72 for X and Y.

ii. Expected genotypic variance was 36 for both X and Y

iii. Expected environmental variance for each trait was 10.3, 36, and 216 for $h' = 0.7$, 0.4, and 0.1, respectively, the same as in the additive model. However, the heritability of the two traits, measured in the "narrow" sense would be less in the complete dominance model.

iv. Expected genotypic covariances were 9, 18, and 27 for initial expected genetic correlations of 0.25, 0.50, and 0.75, respectively.

Again the results obtained in the first generation of offspring showed close agreement with the expected values.

The Effect of Selection on the Genetic Correlation.

Figures 4.1, 4.2, 4.3 show the change in the genetic correlation over 30 generations of selection by upper truncation

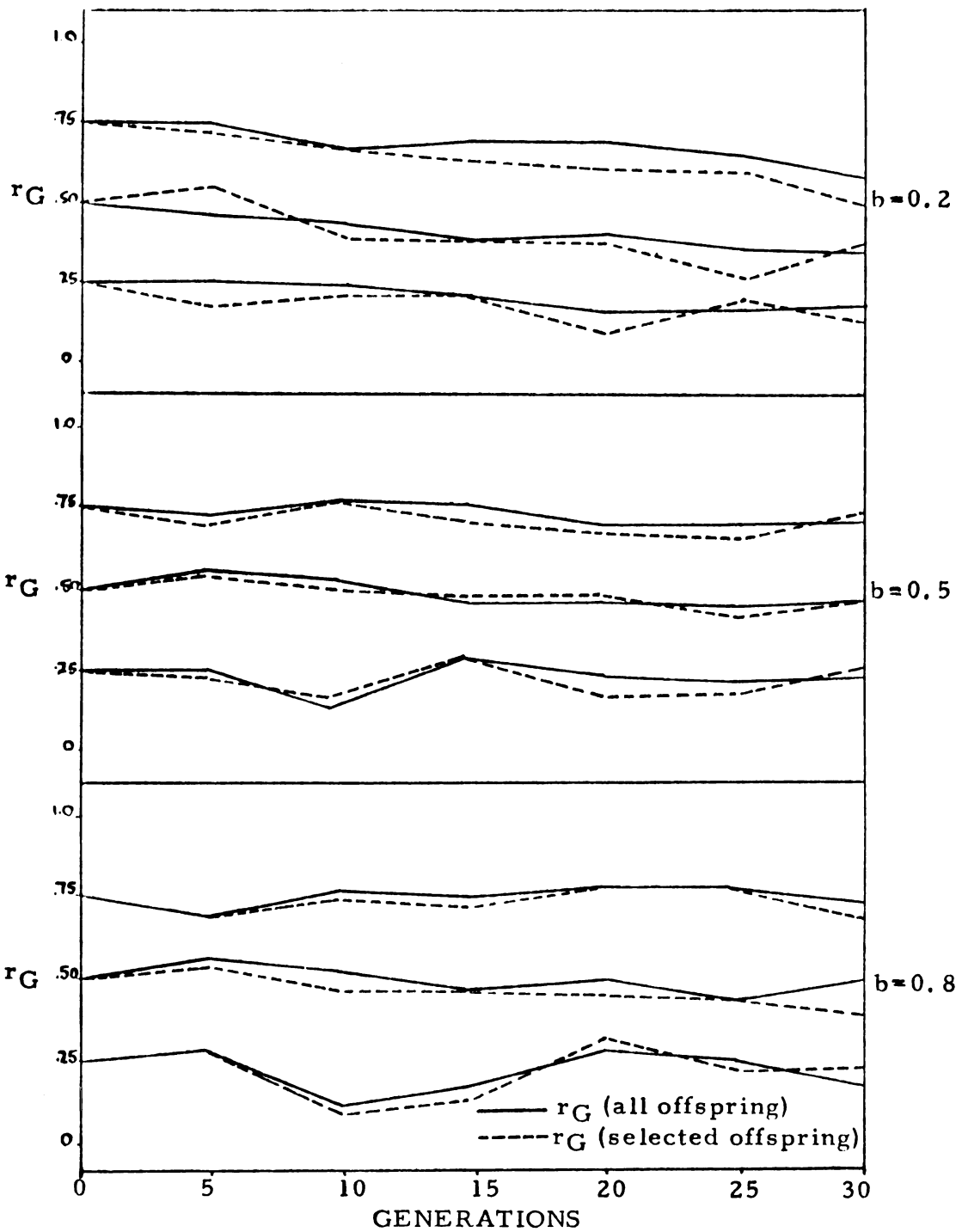

Figure 4.1 Change in genetic correlations at three levels of selection when $h^2_x = 0.1$ (complete dominance). Selection by upper truncation.

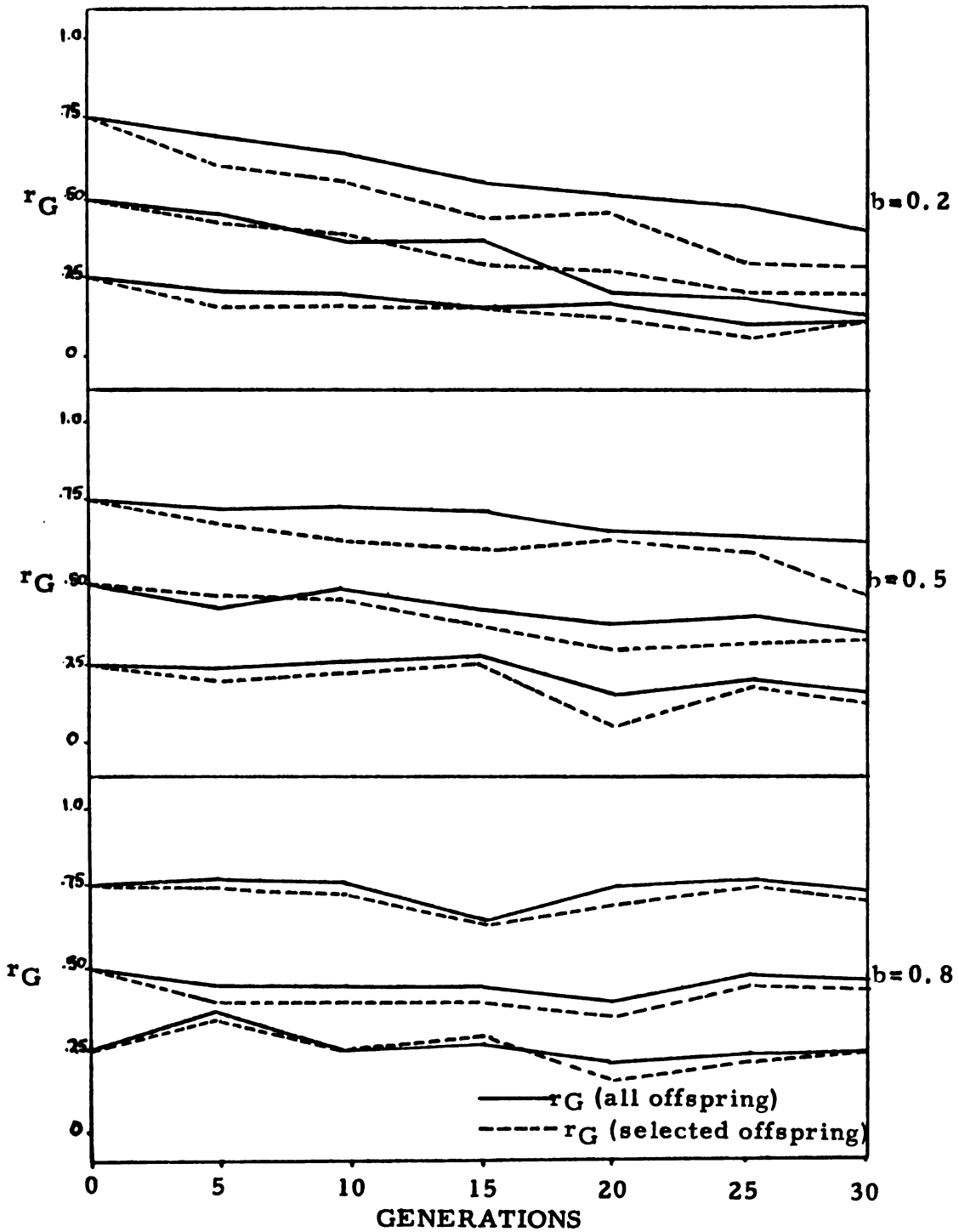

Figure 4.2 Change in genetic correlations at three levels of selection when $h^2_x = 0.4$ (complete dominance). Selection by upper truncation.

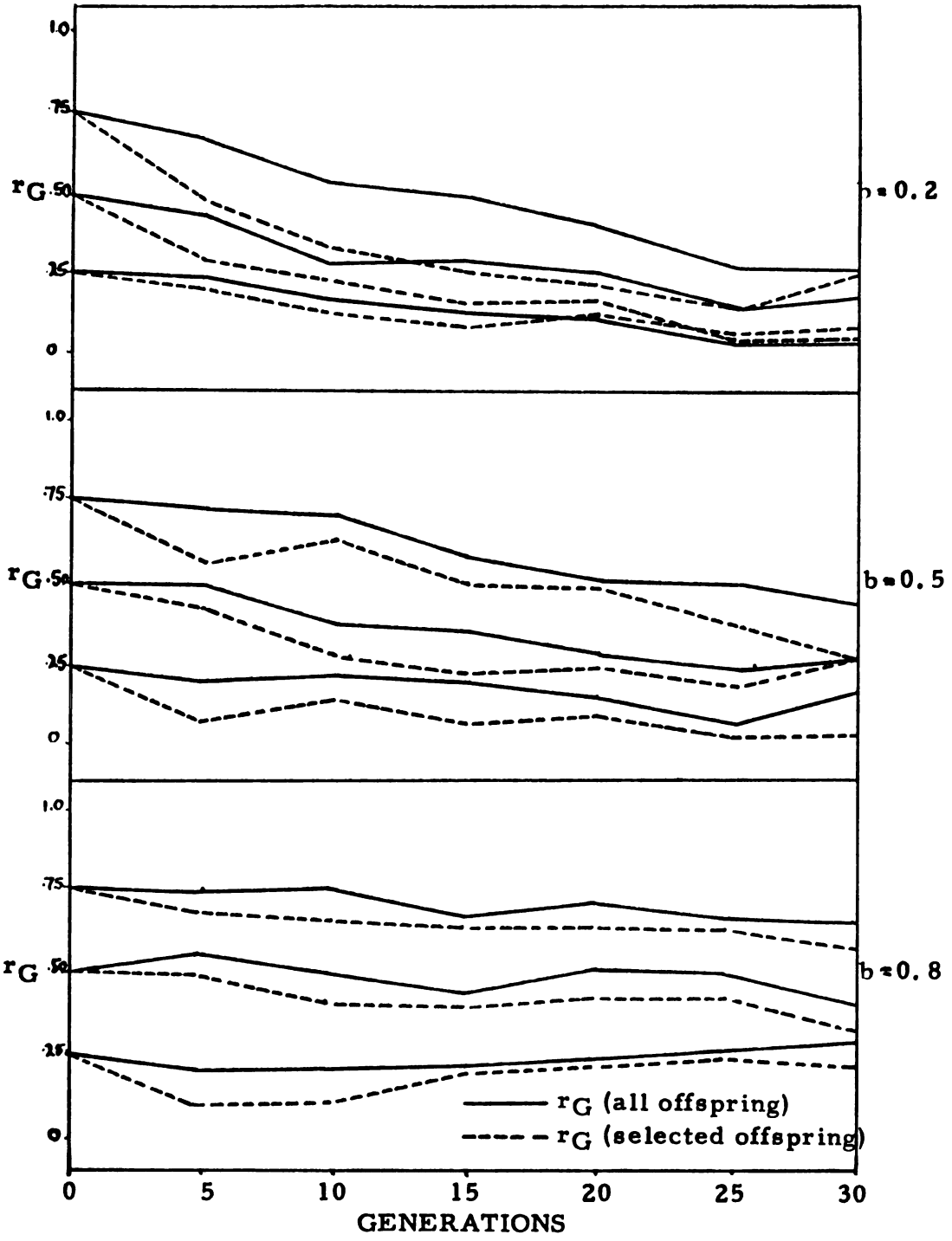

Figure 4.3 Change in genetic correlations at three levels of selection when $h^2_x = 0.7$ (complete dominance). Selection by upper truncation.

of phenotype. Again, as in the additive scheme, the correlation was measured as the product-moment correlation of genic values, the solid line indicating the correlation in the unselected offspring and the broken line representing the correlation in the group of offspring selected to be parents. The values of r were again transformed to z before averaging and then converted to r.

The results followed essentially the same pattern as those in the additive model. The estimate of the genetic correlation remained high when level of selection was low or environmental variance was high, with the estimates tending to fluctuate more at a correlation of 0.25 than otherwise. At high selection intensity ($b = 0.2$) the genetic correlation tended to decline over the 30 generations. However, the amount of the decline was not as great as in the additive model. At high selection intensity and low environmental variance, for example, (Figure 4.3, top graph), the genetic correlation although showing a steady decline, did not become zero by the 30th generation, nor was the sudden drop evident after the 15th generation as in the additive scheme.

All nine graphs again exhibit a consistency in the genetic correlation except perhaps when selection intensity was high and environmental variation was low. Selection must be intense

before having an appreciable effect on the genetic correlation. Most traits in animals are likely to be controlled by much more complex genetic systems than any one used here, and in more complex systems the genetic correlation should be even less affected by selection. To conclude that the genetic correlation need not be estimated as frequently as has been thought is not necessarily valid, however.

The change in the genetic correlation by selection was again studied more closely by graphing the genotypic covariance, and variances of the two traits. These graphs are presented in Figures 5.1 to 5.9

When both level of selection and heritability were low (Figure 5.1), the genotypic covariance and variances were all maintained over the 30 generations of selection. With increased selection intensity, however, the genotypic covariance and variance of the selected trait decreased more rapidly. This decay in the covariance and variance of the selected trait became quite rapid and distinctly curvilinear at high level of selection ($b = 0.2$, Figures 5.6, 5.9). The decrease began immediately, and probably the greatest decrease came in the first generations. There was quite a distinct drop between the first and the fifth generation, and then the curve began to level out through the 30th generation. This result agrees with that

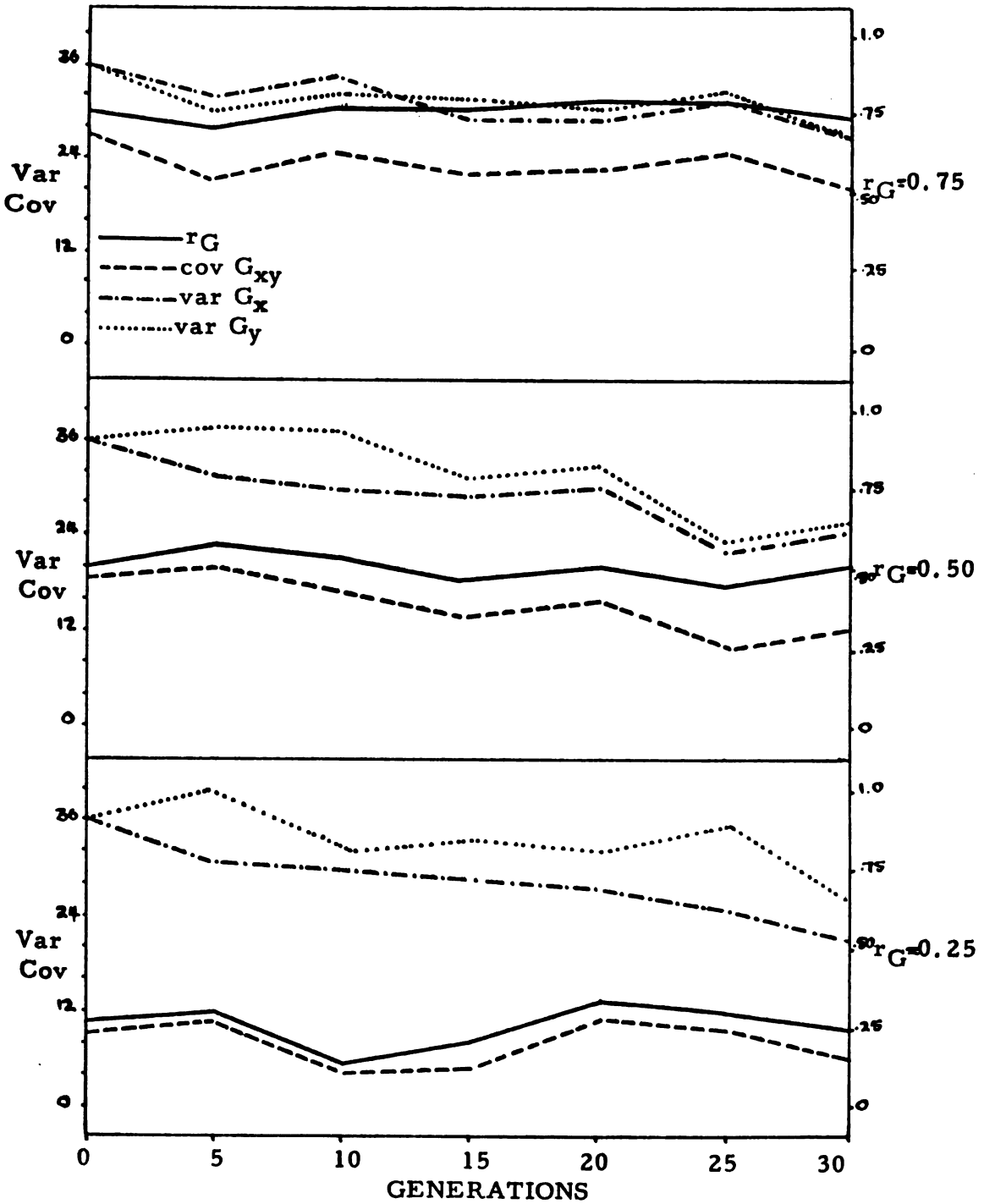

Figure 5.1 The relationship between the genotypic variances, covariance, and genetic correlation at three levels of correlation when b = 0.8 and h^2_x = 0.1 (complete dominance). Selection by upper truncation.

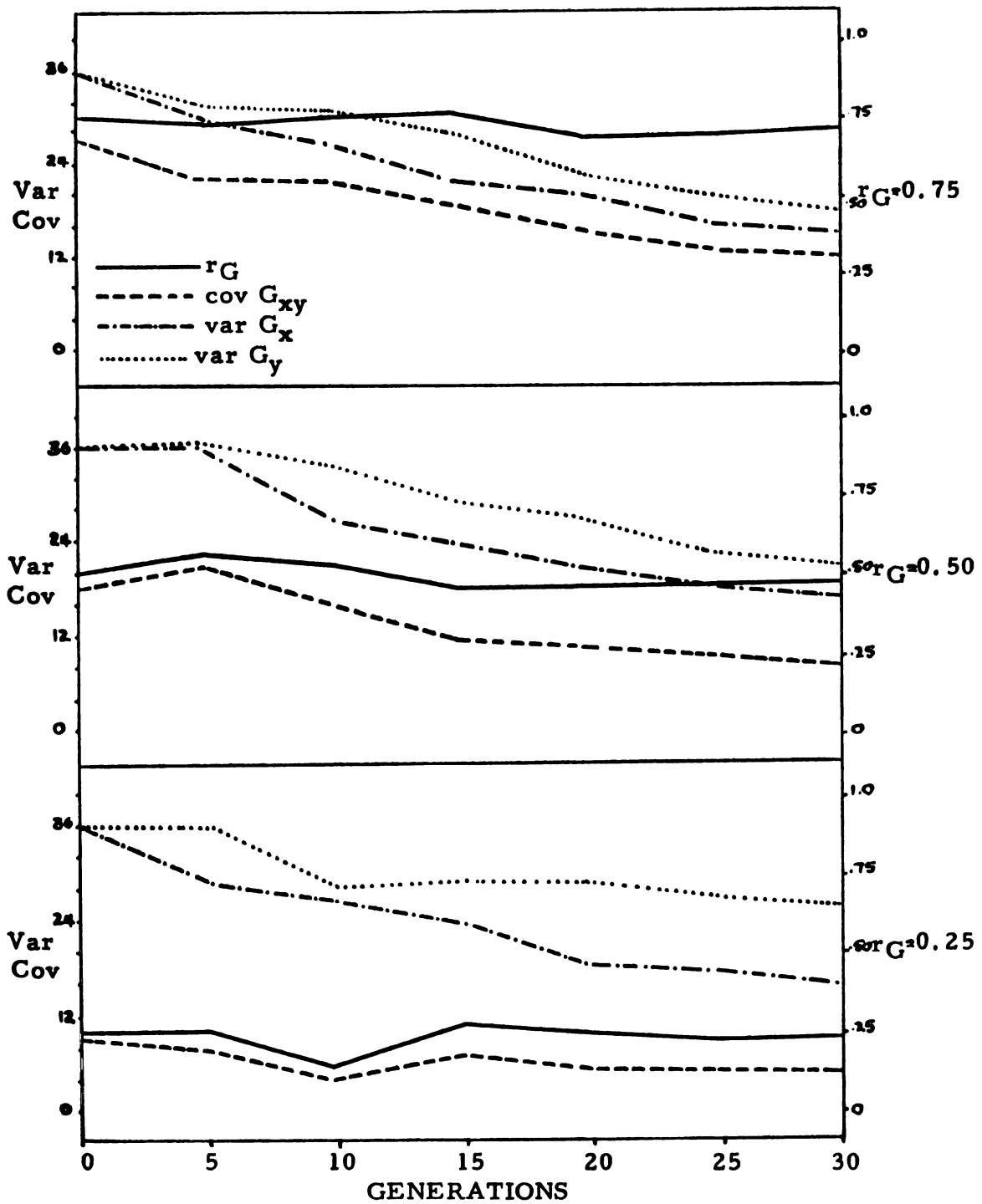

Figure 5.2 The relationship between the genotypic variances, covariance, and genetic correlation at three levels of correlation when b=0.5 and h_x^2 = 0.1 (complete dominance). Selection by upper truncation.

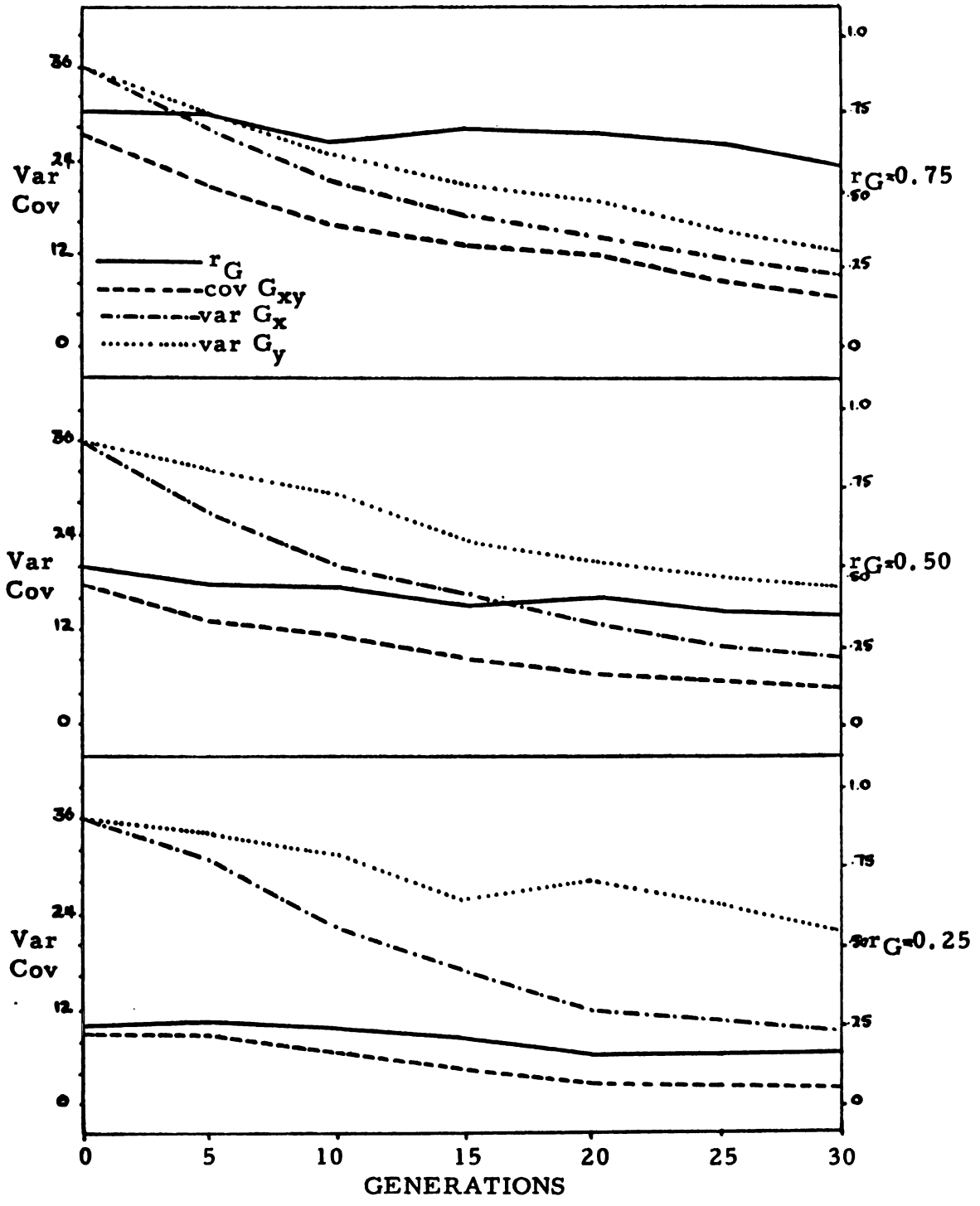

Figure 5.3 The relationship between the genotypic variances, covariance, and genetic correlation at three levels of correlation when b = 0.2 and h^2_x = 0.1 (complete dominance). Selection by upper truncation.

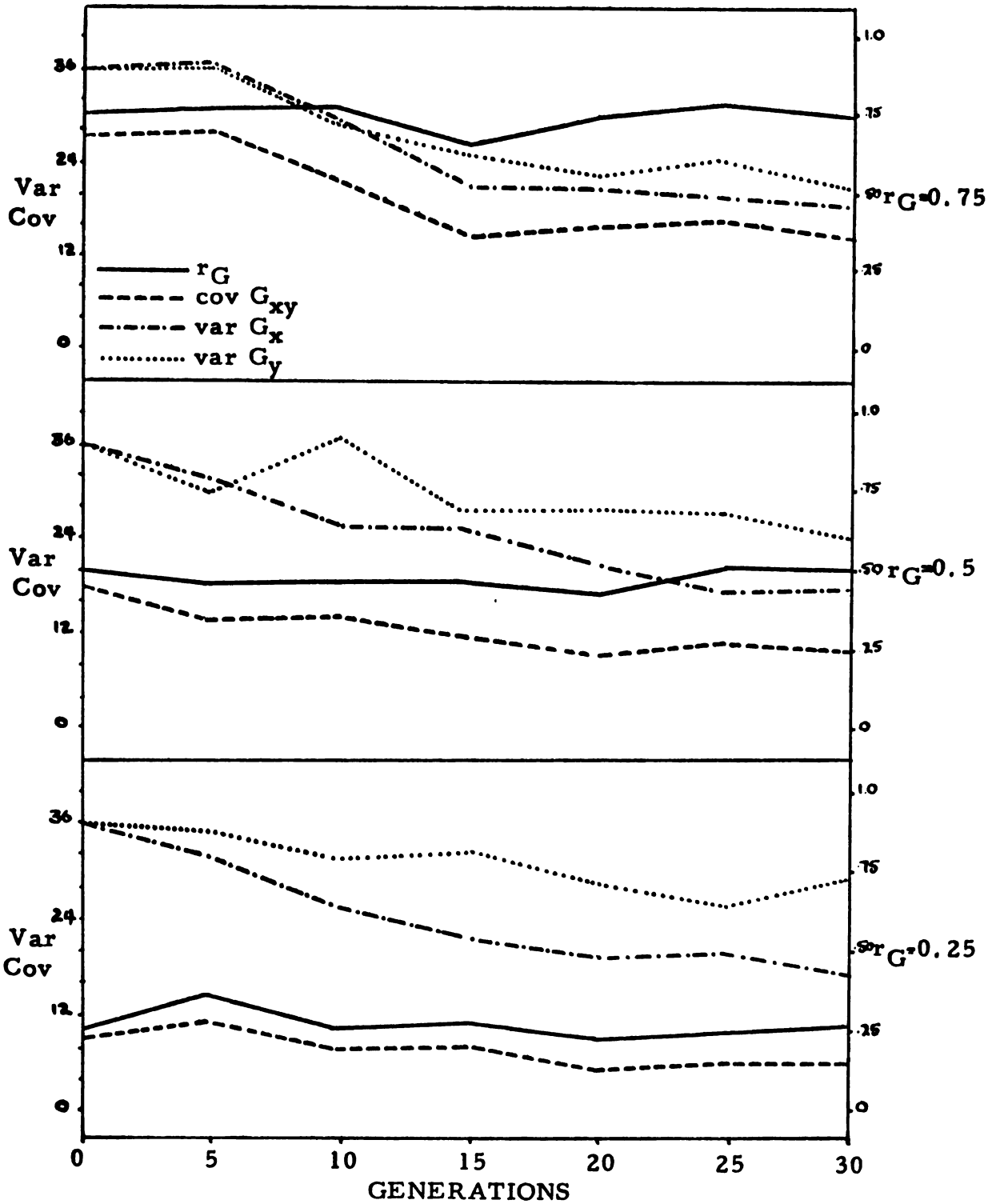

Figure 5.4 The relationship between the genotypic variances, covariance, and genetic correlation at three levels of correlation when b=0.8 and h'_x = 0.4 (complete dominance). Selection by upper truncation.

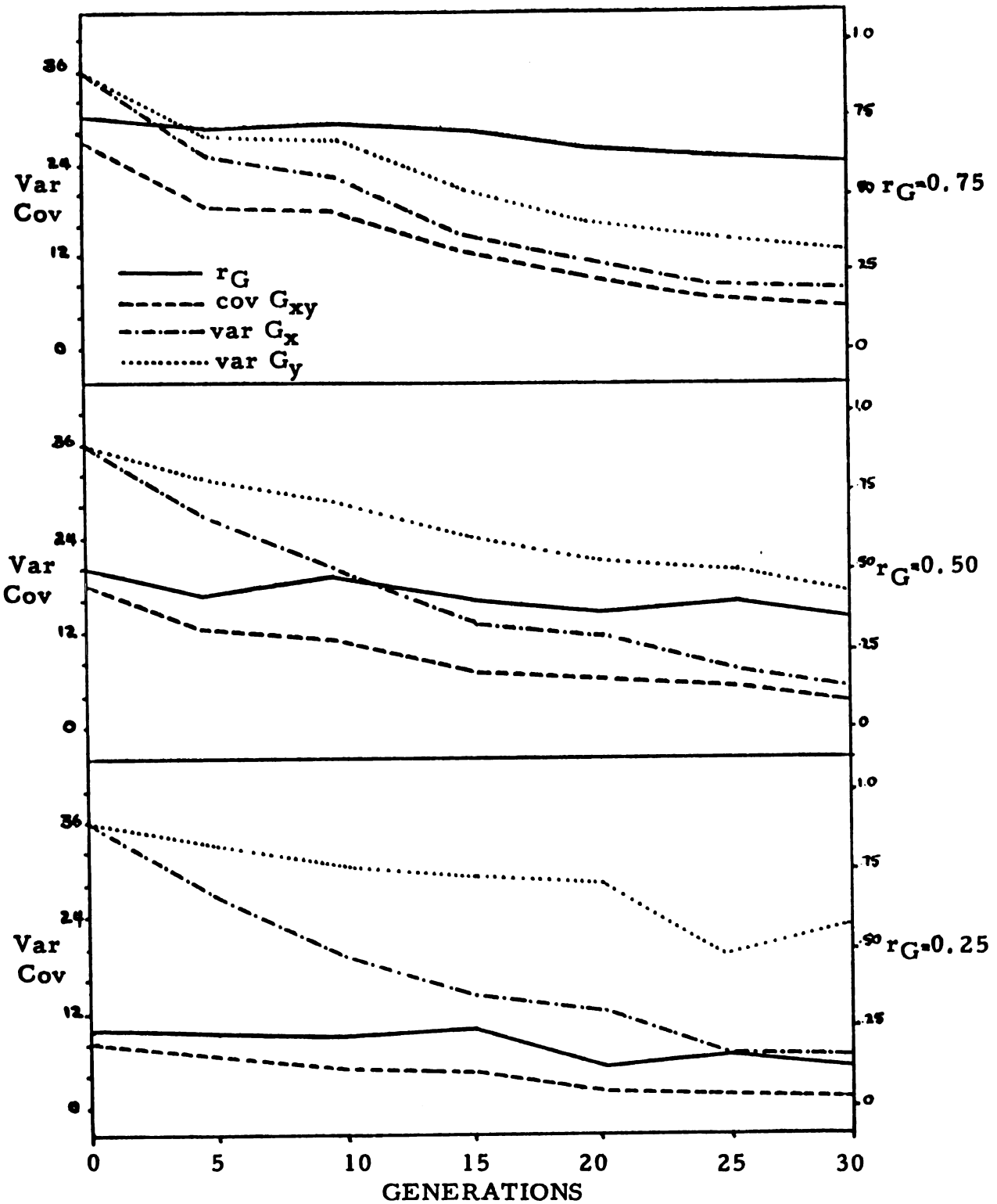

Figure 5.5 The relationship between the genotypic variances, covariance, and genetic correlation at three levels of correlation when b = 0.5 and h^2_x = 0.4 (complete dominance). Selection by upper truncation.

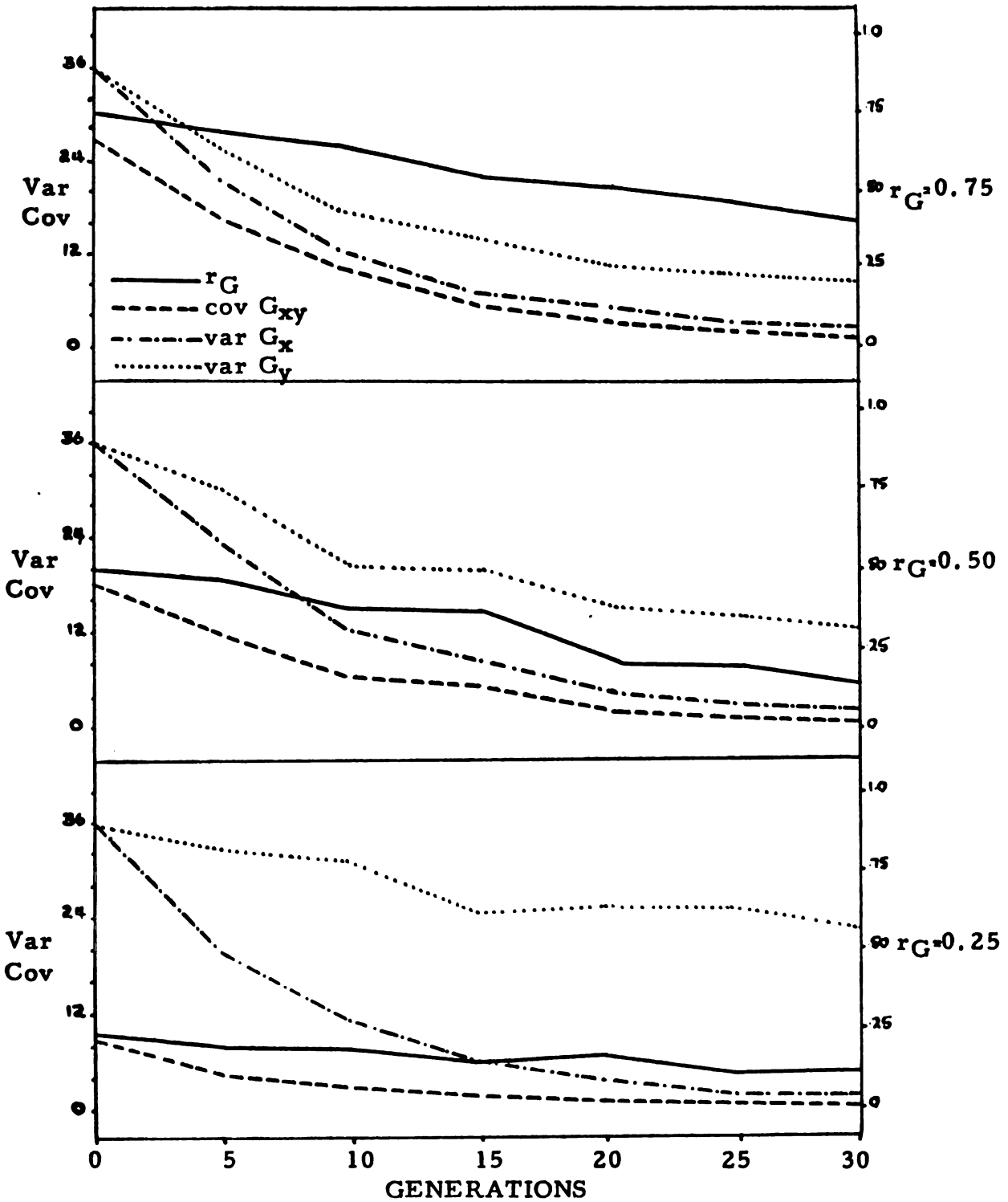

Figure 5.6 The relationship between the genotypic variances, covariance, and genetic correlation at three levels of correlation when b=0.2 and h'_x = 0.4 (complete dominance). Selection by upper truncation.

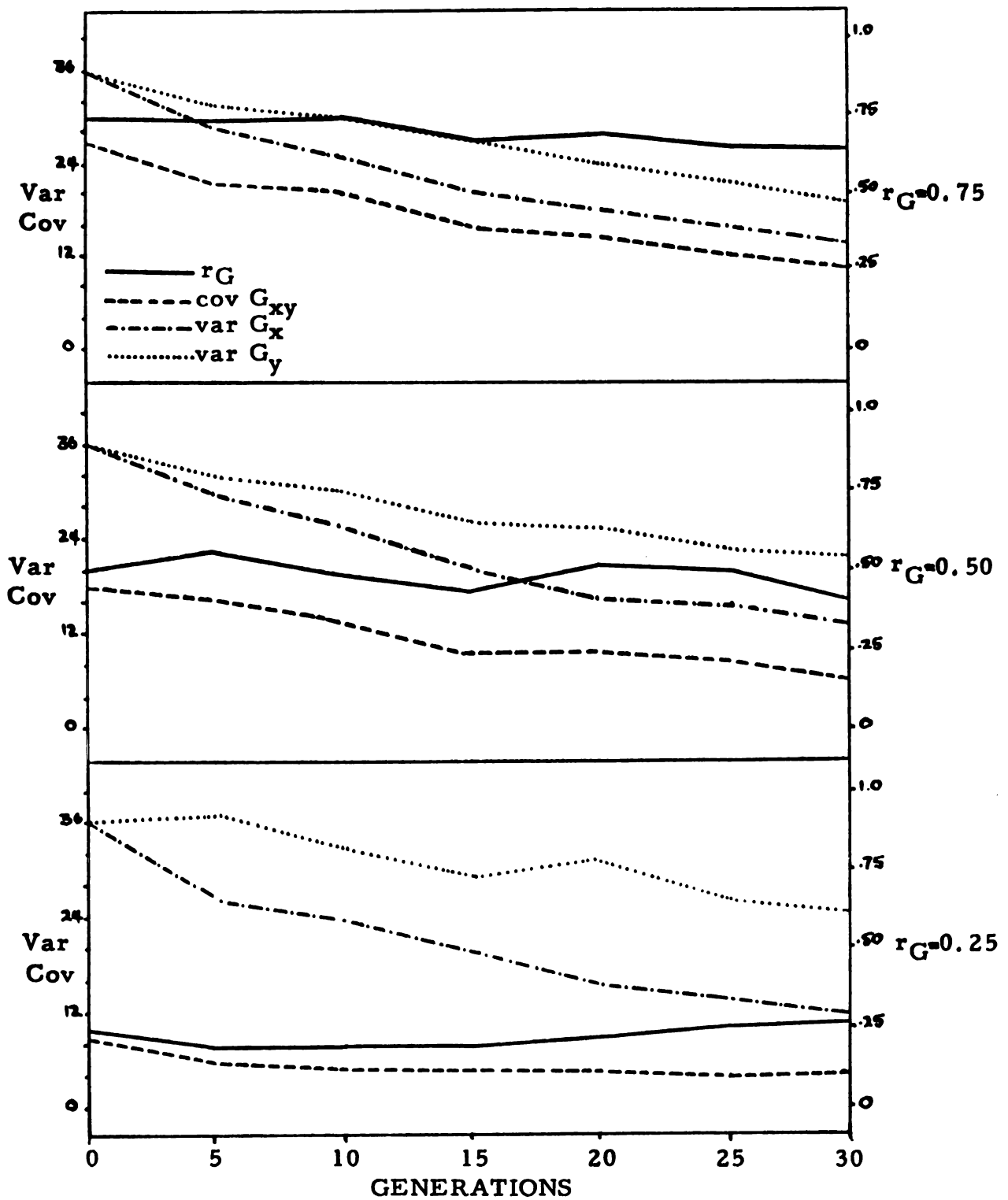

Figure 5.7 The relationship between the genotypic variances, covariance, and genetic correlation at three levels of correlation when b=0.8 and h_x^1 = 0.7 (complete dominance). Selection by upper truncation.

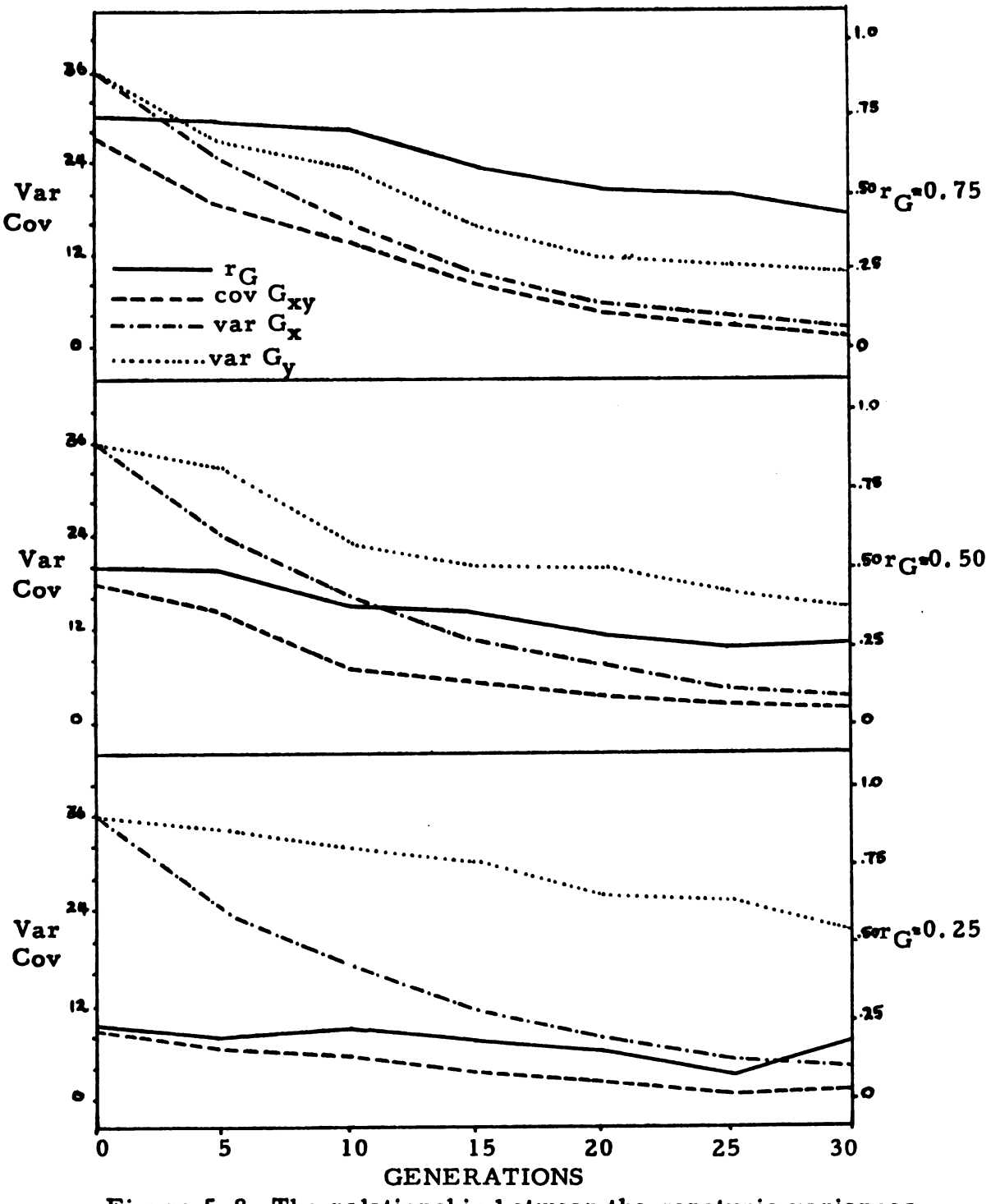

Figure 5.8 The relationship between the genotypic variances, covariance, and genetic correlation at three levels of correlation when b = 0.5 and h^2_x = 0.7 (complete dominance). Selection by upper truncation.

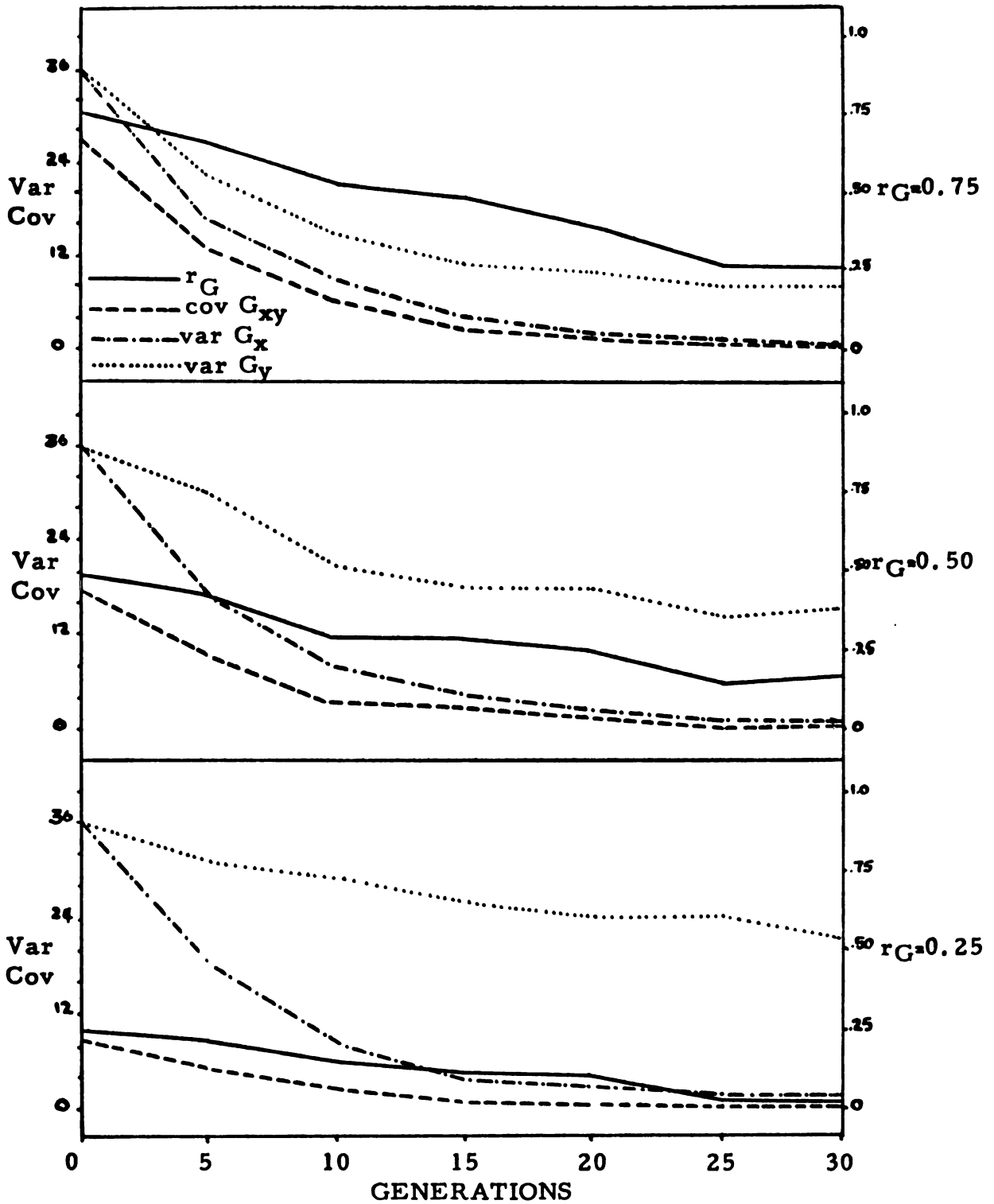

Figure 5.9 The relationship between the genotypic variances, covariance, and genetic correlation at three levels of correlation when b = 0.2 and h^2_x = 0.7 (complete dominance). Selection by upper truncation.

expected from selection for a dominant allele, the change in gene frequency by selection becoming more difficult as the frequency of the recessive gene becomes less. For example, in Figure 5.9, top graph, the genetic variance had decreased from 36 to about 6 by the 15th generation of selection, yet did not reach zero in any case over the last 15 generations of selection. During this time dominance of the favoured gene was actually a hindrance in changing gene frequency because of the abundance of the favoured gene in the population. Essentially the same conditions are acting to maintain the genetic correlation as in the additive model.

As previously noted, a further experiment was conducted under the complete dominance model. Rather than to select upwards for the dominant allele, selection was by lower truncation for the recessive allele. All other assumptions and conditions were identical to those for the first experiment. Figures 6.1, 6.2, and 6.3 show the change in the genetic correlation over 30 generations of selection. The behaviour of the genetic correlation measured in the unselected offspring (solid line) conformed closely in most cases to that already observed for the additive model and in the complete dominance model when selection was by upper truncation. When selection level was high, however, the decrease in the genetic correlation

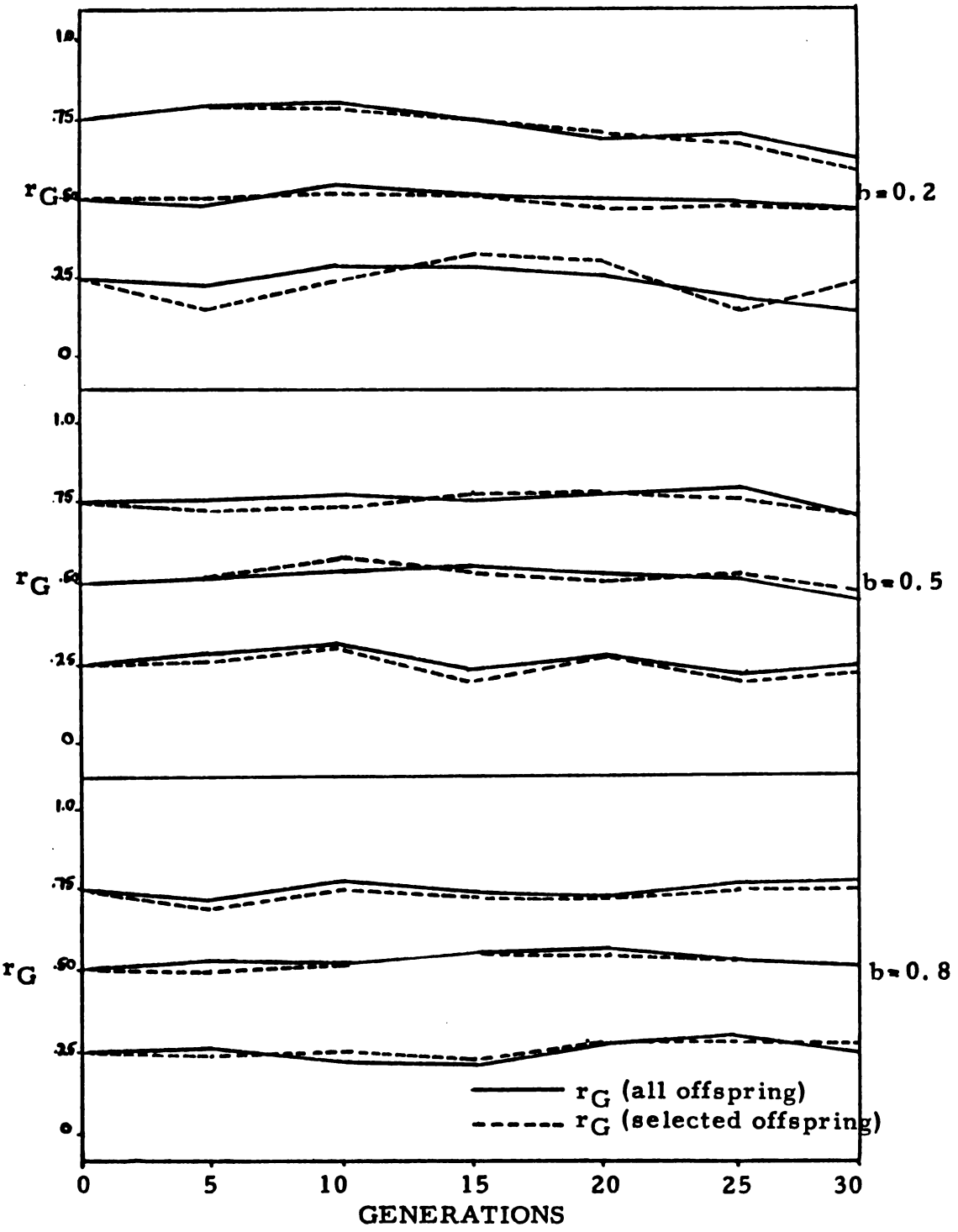

Figure 6.1 Change in genetic correlations at three levels of selection when $h^2_x = 0.1$ (complete dominance). Selection by lower truncation.

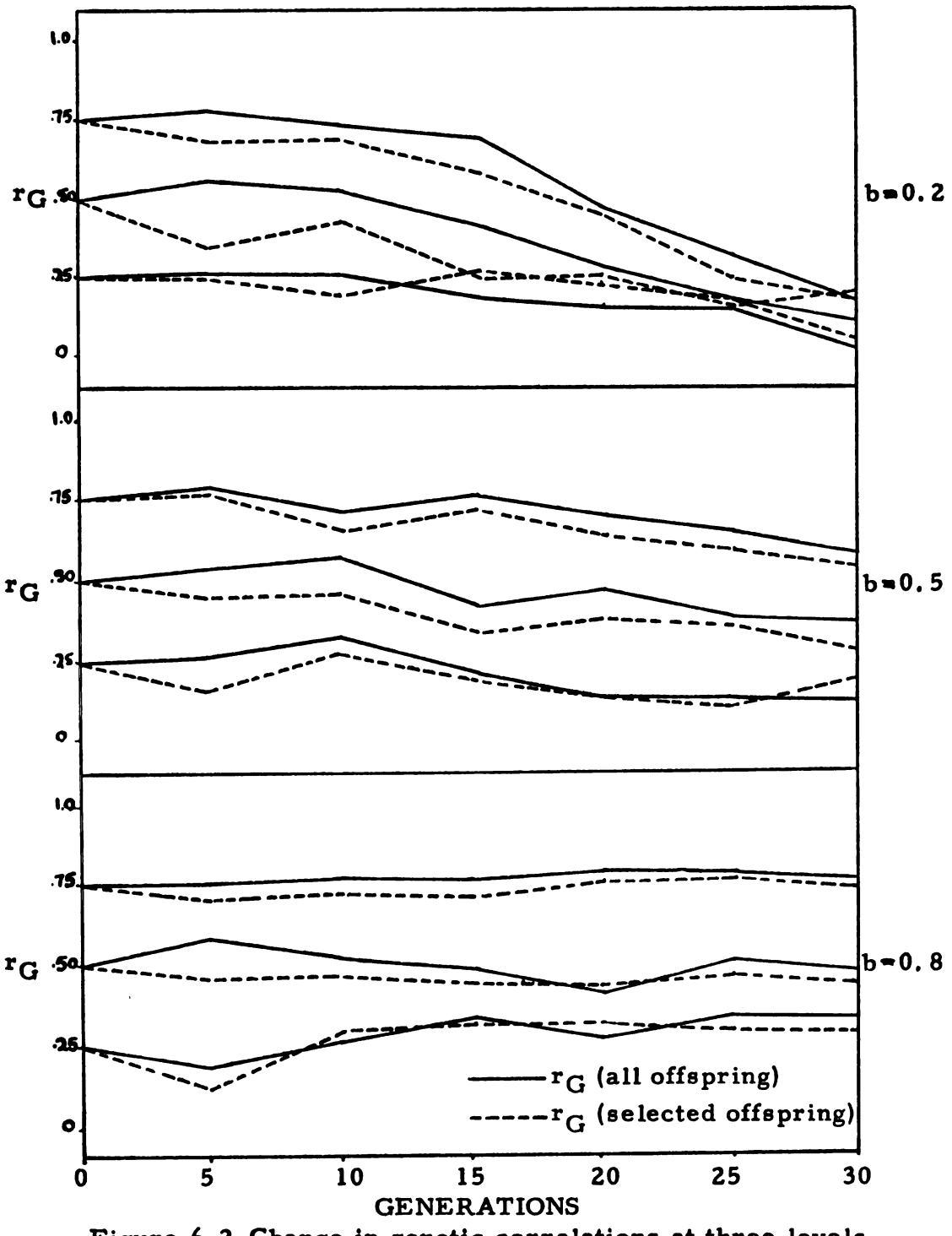

Figure 6.2 Change in genetic correlations at three levels of selection when $h^2_x = 0.4$ (complete dominance). Selection by lower truncation.

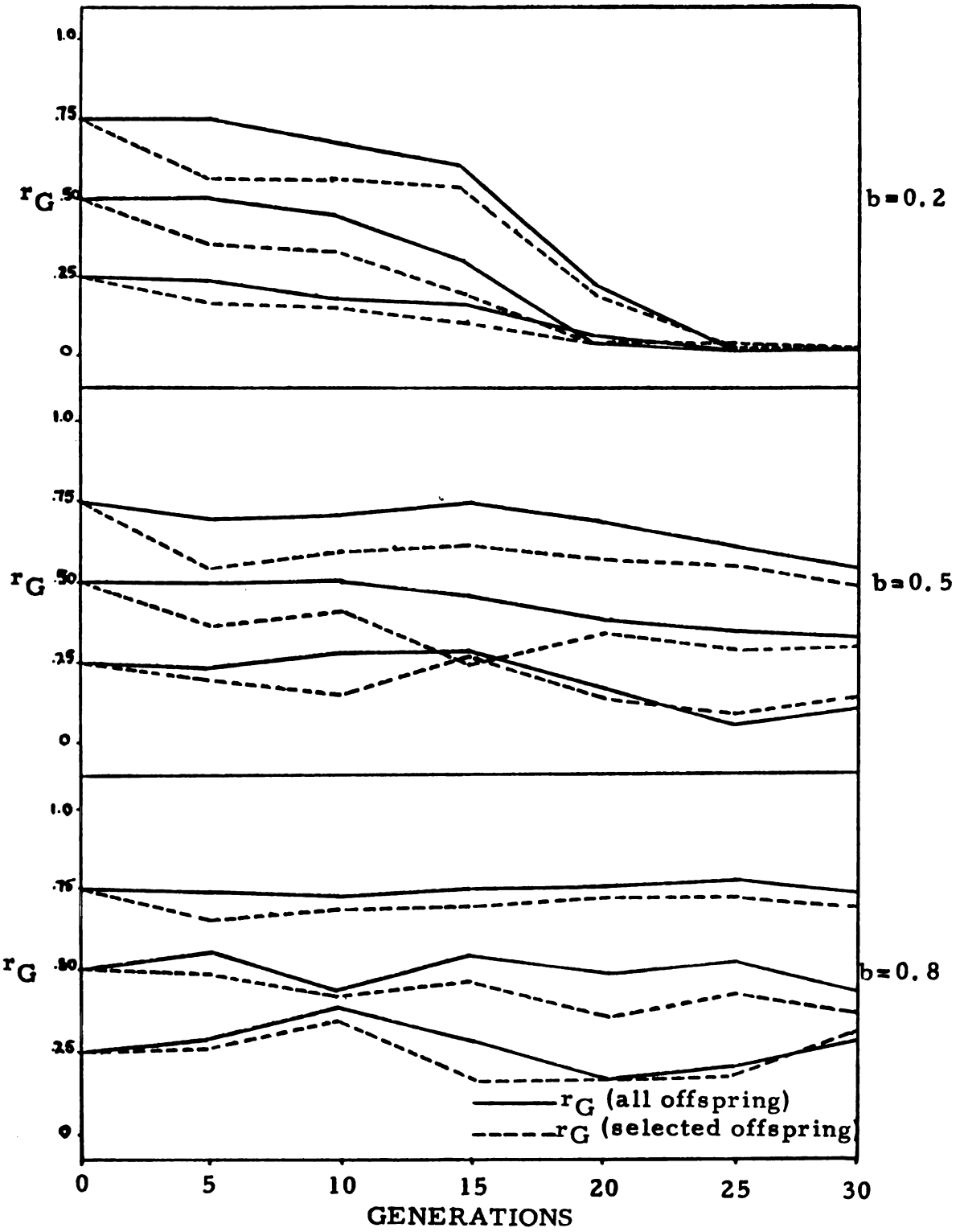

Figure 6.3 Change in genetic correlations at three levels of selection when $h^2_x = 0.7$ (complete dominance). Selection by lower truncation.

was more rapid and reached zero by the 25th generation of selection when heritability was high (Figure 6.3).

Figures 7.1 to 7.9 present the genotypic covariance along with the genotypic variances of the two traits. When level of selection was low ($b = 0.8$), the genotypic covariance and variances of the two traits were maintained at near the initial level especially when environmental variance was high (Figure 7.1) As level of selection increased to $b = 0.2$, the decrease in the genetic covariance became quite rapid regardless of the level of environment, although the decay was more extreme when environmental variance was small (Figure 7.9). The covariance and variance were maintained at a fairly high level for the first five or, at most, first ten generations, and then the decrease became very rapid and curvilinear through generation 20 after which they levelled out again through the 30th generation.

The shape of the curves showing the decay in the genotypic covariance and variance of the selected trait were quite different for selection by lower truncation from selection by upper truncation. When selection was by upper truncation, a rapid decrease occurred in the early generations of selection while lower truncation selection did not change the magnitude of the genetic variance or covariance greatly until after the 5th generation, and then the change became quite rapid. These observations

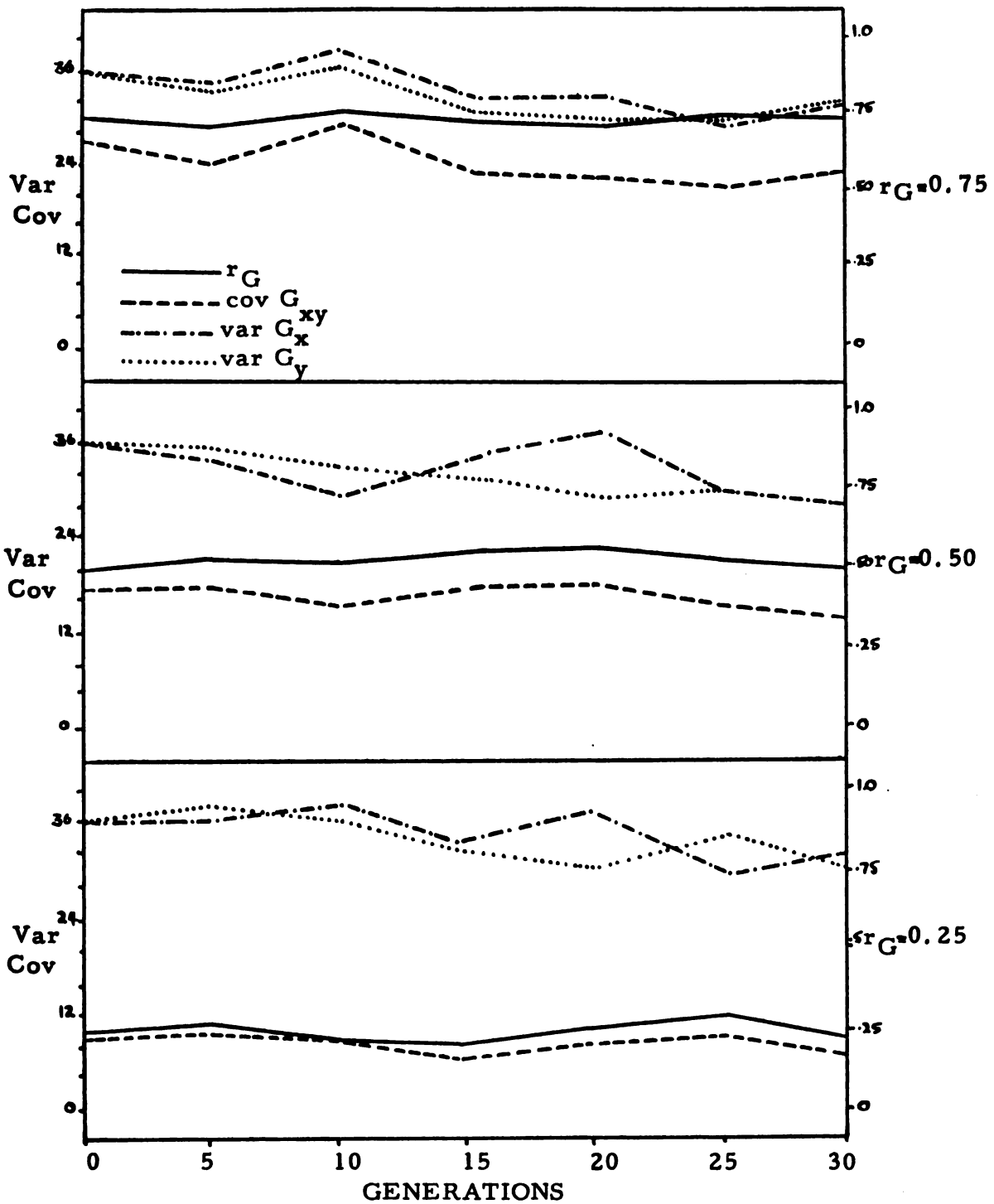

Figure 7.1 The relationship between the genotypic variances, covariance, and genetic correlation at three levels of correlation when b=0.8 and h^2_x = 0.1 (complete dominance). Selection by lower truncation.

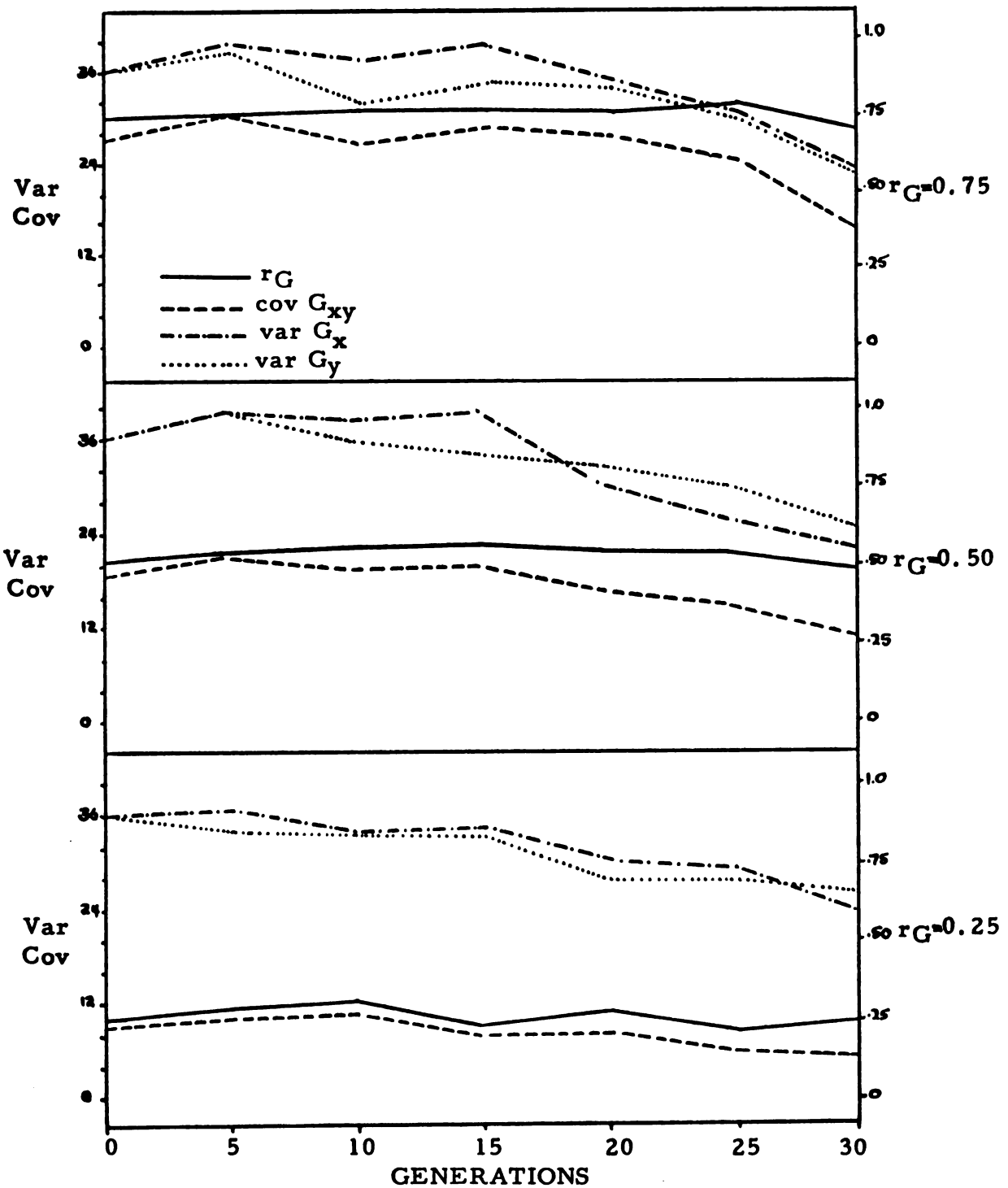

Figure 7.2 The relationship between the genotypic variances, covariance, and genetic correlation at three levels of correlation when b=0.5 and h'_x = 0.1 (complete dominance). Selection by lower truncation.

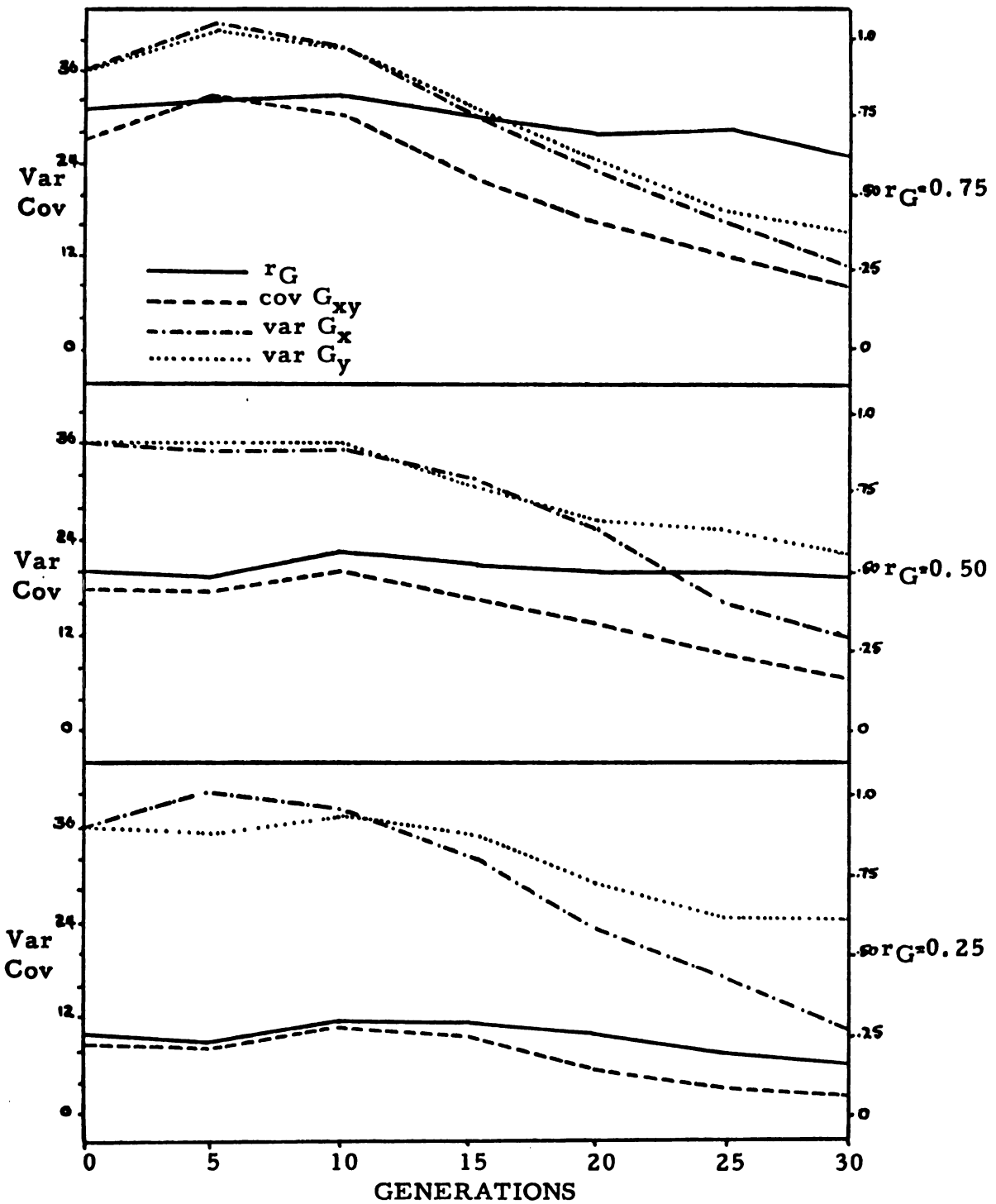

Figure 7.3 The relationship between the genotypic variances, covariance, and genetic correlation at three levels of correlation when b=0.2 and h^2_x = 0.1 (complete dominance). Selection by lower truncation.

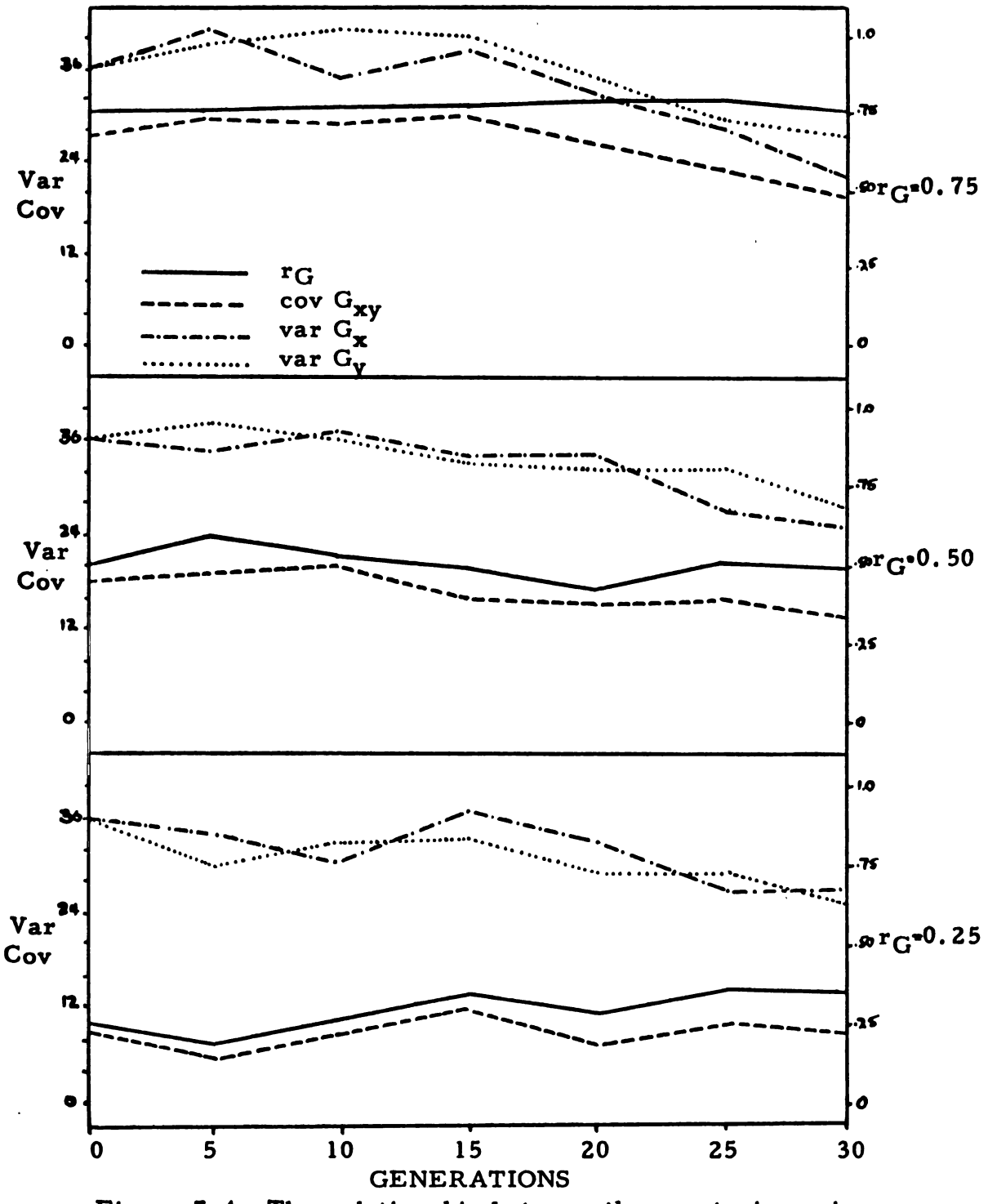

Figure 7.4 The relationship between the genotypic variances, covariance, and genetic correlation at three levels of correlation when b=0.8 and h^1_x = 0.4 (complete dominance). Selection by lower truncation.

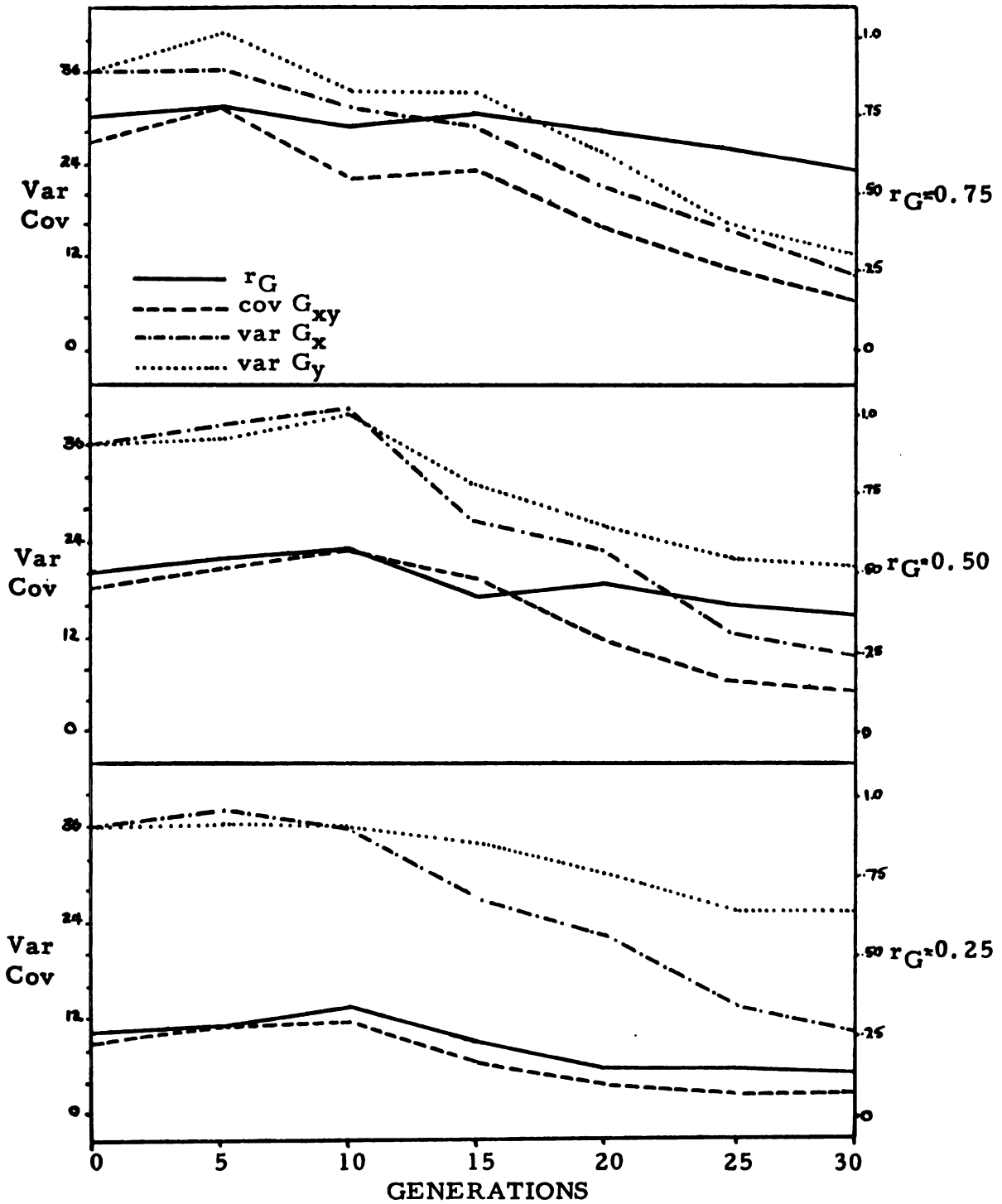

Figure 7.5 The relationship between the genotypic variances, covariance, and genetic correlation at three levels of correlation when b= 0.5 and h^2_x = 0.4 (complete dominance). Selection by lower truncation.

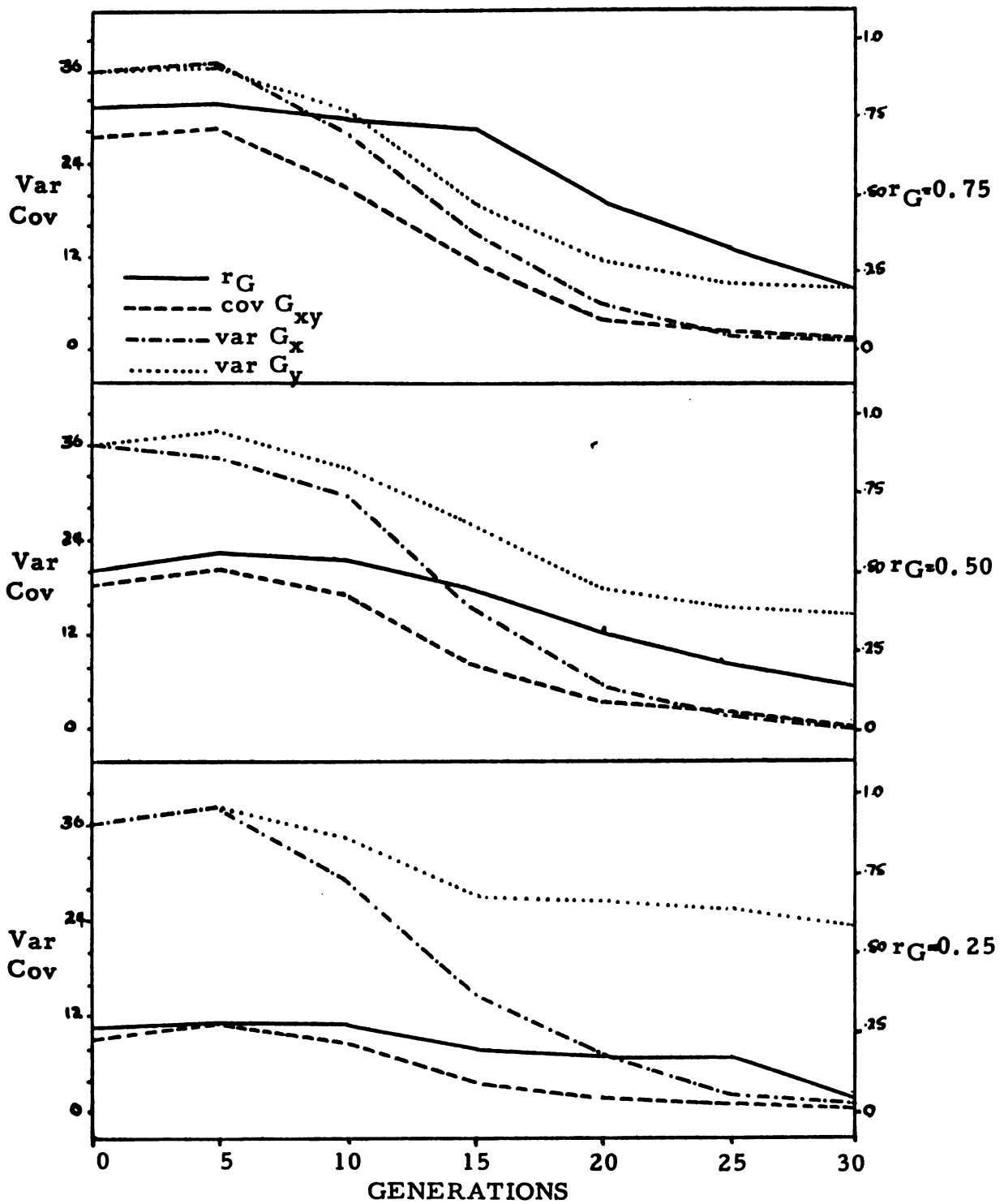

Figure 7.6 The relationship between the genotypic variances, covariance, and genetic correlation at three levels of correlation when b=0.2 and h^2_x = 0.4 (complete dominance). Selection by lower truncation.

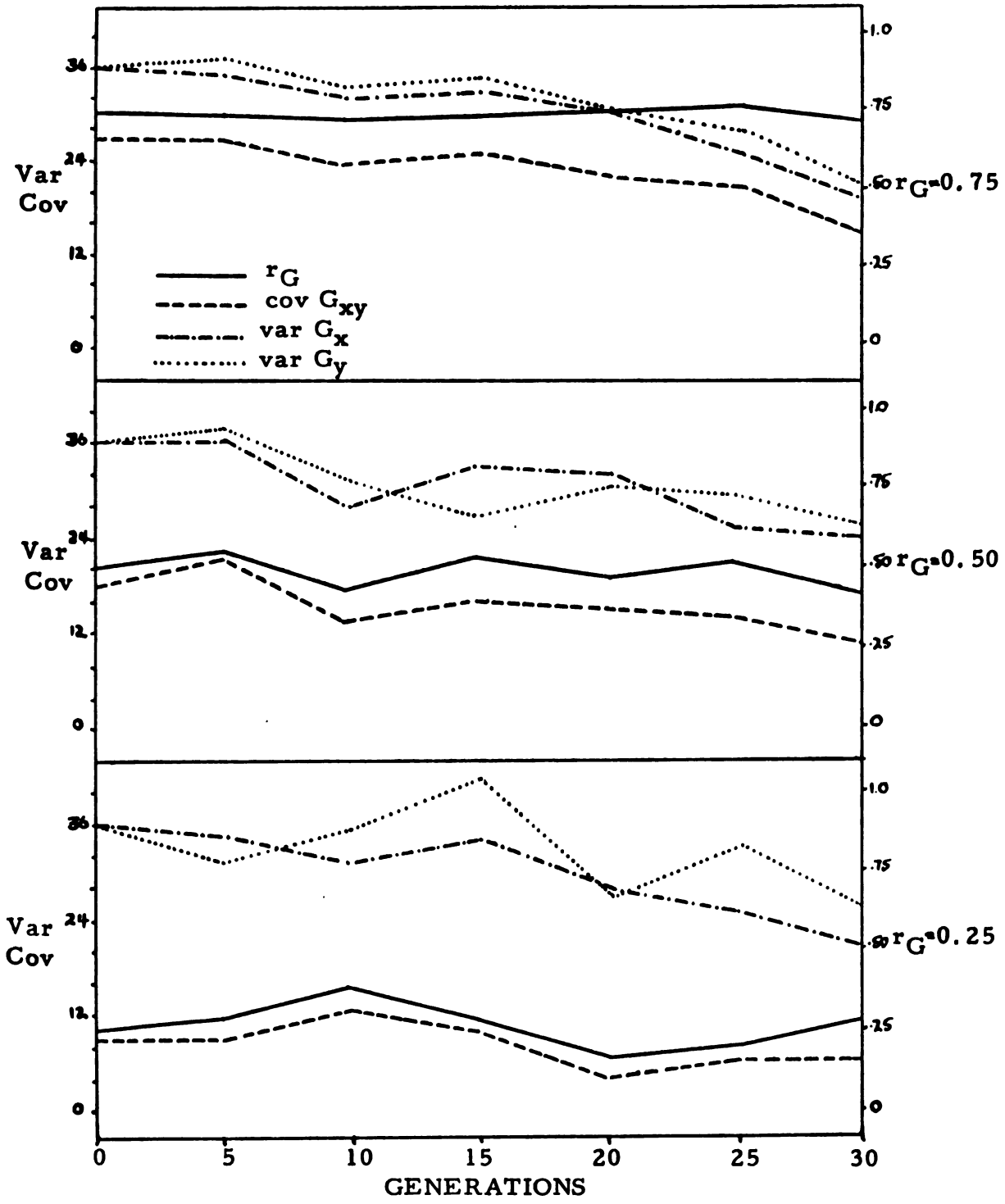

Figure 7.7 The relationship between the genotypic variances, covariance, and genetic correlation at three levels of correlation when b=0.8 and h^2_x = 0.7 (complete dominance). Selection by lower truncation.

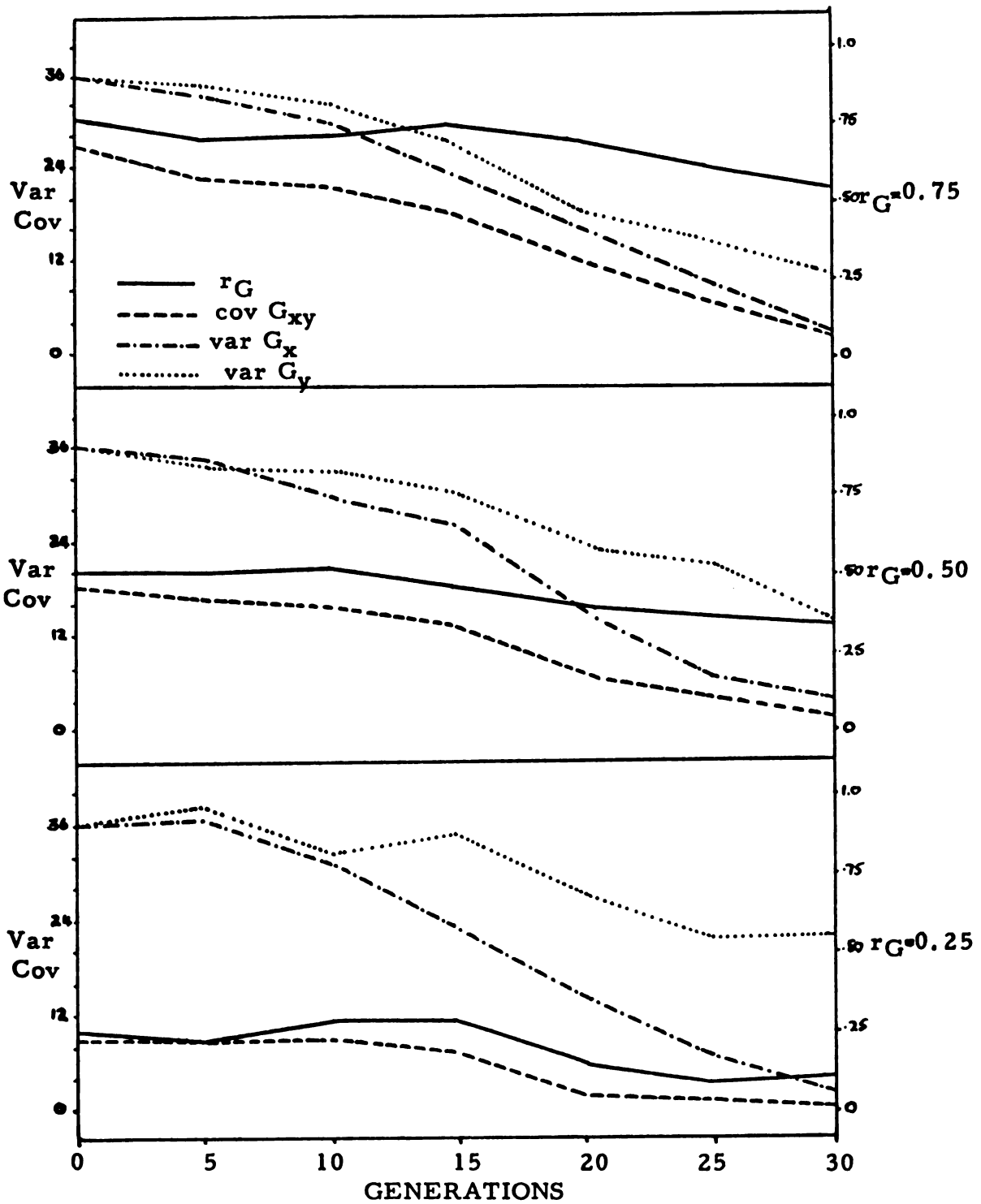

Figure 7.8 The relationship between the genotypic variances, covariance, and genetic correlation at three levels of correlation when b = 0.5 and h^2_x = 0.7 (complete dominance). Selection by lower truncation.

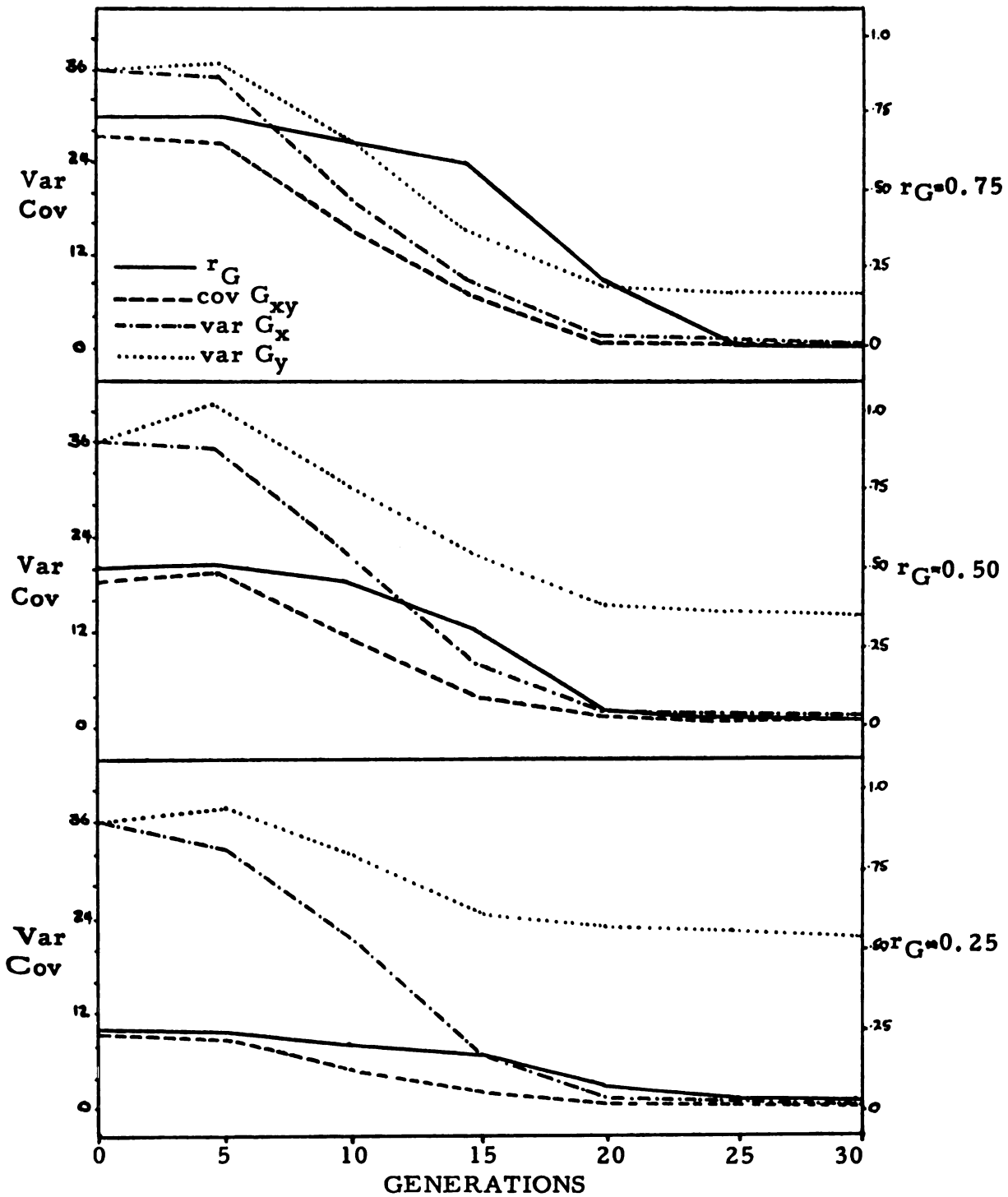

Figure 7.9 The relationship between the genotypic variances, covariance, and genetic correlation at three levels of correlation when b=0.2 and h'^2_x=0.7 (complete dominance). Selection by lower truncation.

follow quite logically from the theory of the rate of change in gene frequency under selection when dominance exists. These circumstances have been discussed in detail by Lush (1945).

An interesting observation in the results, especially in the dominance model, was the close agreement between the change in the genotypic covariance and the change in the genotypic variance of trait Y, the correlated trait. A distinctly similar pattern of response can be noted in every treatment combination (see, for example, Figures 7.6 and 7.9). This similarity of response to selection is expected, however, since each is simply measuring the reduction in the genotypic variance in the loci of type B, those loci which affect both traits in the same direction. That the genotypic covariance is a measure of the genotypic variance of the common loci has been shown previously; all other covariances are expected to be zero. Also, the only loci affecting trait Y which are under selection pressure are those same loci which are shared. Thus, the decrease in the genotypic covariance and in the genetic variance of Y are both a function of the change in gene frequency at the pleiotropic loci.

The Genetic Correlation in the Truncated Distribution.

The effect of linear truncation of X on the genetic correlation

again has been graphed for both experiments in the complete dominance model. The broken lines in Figures 4.1 to 4.3 and in Figures 6.1 to 6.3 represent the genetic correlation in the group selected to be parents for upwards and downwards selection, respectively.

As in the additive model, truncation selection caused some decrease in the genetic correlation. When selection was by upper truncation, the amount of reduction in the genetic correlation was a function of both level of heritability and selection. The amount of decrease seemed greater than was observed in the additive model. The magnitude of the initial genetic correlation was also affecting the amount of decrease, a larger and more consistent reduction resulting when the initial correlation was 0.75 than when 0.25. When level of selection was high ($b = 0.2$) and environmental variance was low (Figure 4.3), the amount of decrease became very large when the initial genetic correlation was 0.75. In fact, the genetic correlation in the selected group was generally about 0.2 less than the correlation in the whole offspring generation. Again, however, when intensity of selection and heritability were both low, the reduction in the genetic correlation was fairly small, which can be expected when 80 per cent of the offspring are selected to be parents.

When selection was by lower truncation (Figures 6.1 to 6.3) similar results were obtained, the amount of reduction increasing as heritability increased. As observed in the additive model, level of environmental variance was more important than level of selection in producing a decrease in the correlation in the selected group.

The Estimates of Genetic Correlation Obtained from Phenotypic Covariances between Parent and Offspring.

In the model of complete dominance two separate estimates of the genetic correlation were obtained from the covariances between parent and offspring phenotypes. However, because of the erratic results obtained in the additive model, the estimate for the complete dominance model using the geometric mean of the two covariances in both numerator and denominator was rejected if the two covariances in the numerator or denominator were of unlike sign. This condition occurred in the majority of cases.

In general, the results obtained using the arithmetic mean of the two covariances in the numerator were equally as poor as those previously obtained for the additive model, whether selection was by upper or by lower truncation. Since to present all the results for both types of selection would be of little value,

a sample of results has been presented only for selection by upper truncation. These are shown in Table 3 as deviations of the genetic correlations computed by parent-offspring covariances, from the product-moment correlations of genotypic values. The same group of treatment combinations and examples for the first replicate in selected generations as for the additive model are presented. The asterisk beside some estimates indicates that the two covariances within the numerator and within the denominator were of the same sign. More estimates had covariances of unlike sign than not. In those cases where the correlation could be computed by the geometric means in both numerator and denominator, seldom did the correlation by geometric mean agree with that by arithmetic mean.

Table 3 indicates that an interpretation of the results would be unwise, apparently random fluctuation prevents observing a predictable pattern. There is, however, the same tendency for the correlation to be underestimated as in the additive model.

Of the 300 deviates presented in Table 2, 194 were negative, and the overall average deviation was - 0.40. When the level of selection was low and the number of observations small, the average deviation was - 0.37 while at high intensity of selection the average deviation was - 0.43. These averages

TABLE 3. Deviation of the genetic correlation by parent-offspring covariances from the product-moment correlation of genotypic values (Complete Dominance - Upwards Selection)

				GENERATIONS									
r_G	b			2	3	4	5	6	10	15	20	25	30
.25	0.8	0.1	0.1	1.13*	0.52	-0.02	0.54	-0.11	-2.96	-1.72	-2.53	0.00	-0.34
"	"	0.1	0.7	-0.79	-0.39	-0.52*	0.69*	-0.40	-1.14	-0.53	0.48	-6.01*	-0.75
"	"	0.4	0.4	0.06	-0.82	-0.57	0.49*	0.10	1.13	1.22	0.00	0.01	-1.13*
"	"	0.7	0.1	3.67	0.11	1.55	-0.03	0.09	-0.25	-0.25	-0.44	-0.50	-1.03
"	"	0.7	0.7	1.08	-1.27*	-1.15	1.13	7.52	-0.65	-2.22	0.12	-0.98	0.07*
"	0.2	0.1	0.1	-3.08	-2.25*	-1.05	2.34*	-0.60	1.37	-0.02	-0.80*	-4.70	4.22*
"	"	0.1	0.7	-1.65	0.64	-1.35	-0.87*	-1.48	-0.98	-1.73	0.46	-1.08*	-0.48
"	"	0.4	0.4	-0.99*	-0.42	-0.24	-0.45	-1.11	-1.37	-5.08	-6.51*	1.08	1.11
"	"	0.7	0.1	0.21	3.85	-0.69	1.14	0.92	-4.02	-1.12	0.49	-3.49	0.90
"	"	0.7	0.7	0.79*	1.17	0.48	-0.63	-0.52	2.24	1.31*	0.78	1.33*	-0.50
.50	0.8	0.1	0.1	3.88	-0.65	-0.73	-0.50	1.36	0.38	-2.29*	0.54*	-4.44	-0.76
"	"	0.1	0.7	-0.81	0.91*	0.28	-0.71	-3.02	0.35	1.69	1.12*	0.08	-1.12*
"	"	0.4	0.4	-1.03	-1.76	-1.69	0.48	-1.26	-3.46	0.46	-0.37*	1.33*	-1.48
"	"	0.7	0.1	-0.02	-0.91	-0.42	5.59	-3.12	-7.86	0.84	0.67	0.24*	-0.61
"	"	0.7	0.7	-0.01	-1.07	1.44*	0.57	-2.90	0.08*	-1.28*	1.34*	-0.53	-1.16
"	0.2	0.1	0.1	-0.90	0.04	-0.66	-0.38	-1.75	-0.91*	1.67	-1.02*	-1.75*	-1.59
"	"	0.1	0.7	-1.00*	0.35*	-0.83	1.22*	0.33	-0.77	-2.17*	-0.68	-3.91	0.04
"	"	0.4	0.4	1.37*	3.29	-0.17	-1.19*	-1.62*	-1.00*	-3.16*	-0.02	-0.47	-1.10
"	"	0.7	0.1	-1.62	-1.07	4.09*	-0.45	-0.65	-1.09	-0.11	0.70	0.08	0.49
"	"	0.7	0.7	3.07	0.72	0.05	-0.39	0.24*	0.02	-4.05*	-1.05	0.26	-1.06

TABLE 3. (continued)

				GENERATIONS									
rG	b			2	3	4	5	6	10	15	20	25	30
.75	0.8	0.1	0.1	0.17	-1.79*	1.43	-0.19*	0.84	-1.32	0.88	-2.70*	-0.09	-2.90
"	"	0.1	0.7	0.50*	-1.47	-2.78	0.36*	-0.37*	5.51	-1.77	-0.37	-1.67*	-0.89
"	"	0.4	0.4	0.74*	-6.21*	0.58	1.06	-2.48*	-3.19*	-0.13	-0.85	-2.45	0.26*
"	"	0.7	0.1	-5.33	-1.21*	-0.38	2.79	2.35	-0.04	-2.54*	-0.46	-0.18	2.10*
"	"	0.7	0.7	0.13*	-0.69	-1.49	1.57*	2.49*	0.29	-1.54	-0.71	2.20	-1.62
"	0.2	0.1	0.1	-1.55	0.05	-1.82*	-2.28	-0.83	0.82	1.30	-2.72	-2.27	4.28
"	"	0.1	0.7	-0.47	-0.78	-1.32*	-0.42	-0.02	-0.55	-1.38	-1.24	0.04*	-0.98
"	"	0.4	0.4	-2.05	2.58	-0.71	4.14	-3.11*	-8.64	-1.48*	-0.71	-1.53*	1.88
"	"	0.7	0.1	0.89	-3.06*	-0.42	-0.45	-2.60*	1.07	-0.57	-0.61	-0.67	8.56*
"	"	0.7	0.7	-1.44	0.31	-0.66	1.51	-1.21	-1.25	1.42	-0.28	2.28	1.02

agree fairly well with those computed in the additive model. For example, the overall average deviation in the additive model was - 0.38, compared to - 0.40 in the complete dominance model. There could be some mechanism operating to cause these underestimates, but the complexities of the situation would require a more precise examination than was conducted here.

Correlated Response to Selection.

For the complete dominance model the expected genotypic mean in the first generation of offspring was 72 for both traits X and Y whether selection was by upper or lower truncation. The change in the genotypic mean of trait X and the correlated change in the genotypic mean of trait Y at all treatment combinations have again been presented graphically over the 30 generations for both methods of selection in Figures 8.1, 8.2 and 8.3. The solid line represents the response in trait X, directly selected, while the broken lines indicate the correlated response of trait Y to selection for X at the three degrees of genetic correlation between X and Y. The upper four curves in each graph are the results of selection by upper truncation of phenotype of X while the lower four indicate the response to selection by lower truncation. As was done in the additive

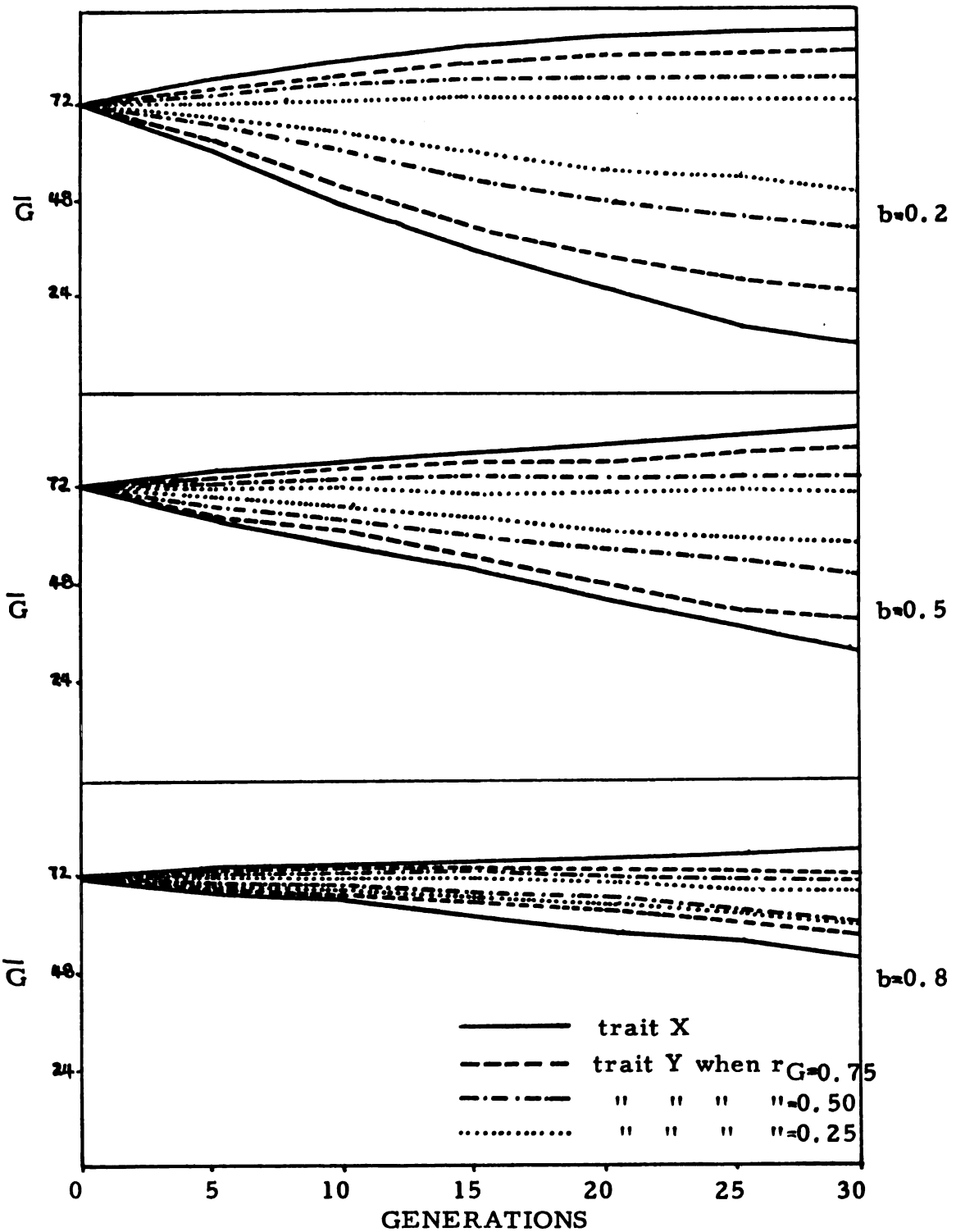

Figure 8.1 Mean genetic progress at three levels of selection when $h^2_x = 0.1$ (complete dominance). Upper four curves indicate selection by upper truncation, lower four selection by lower truncation.

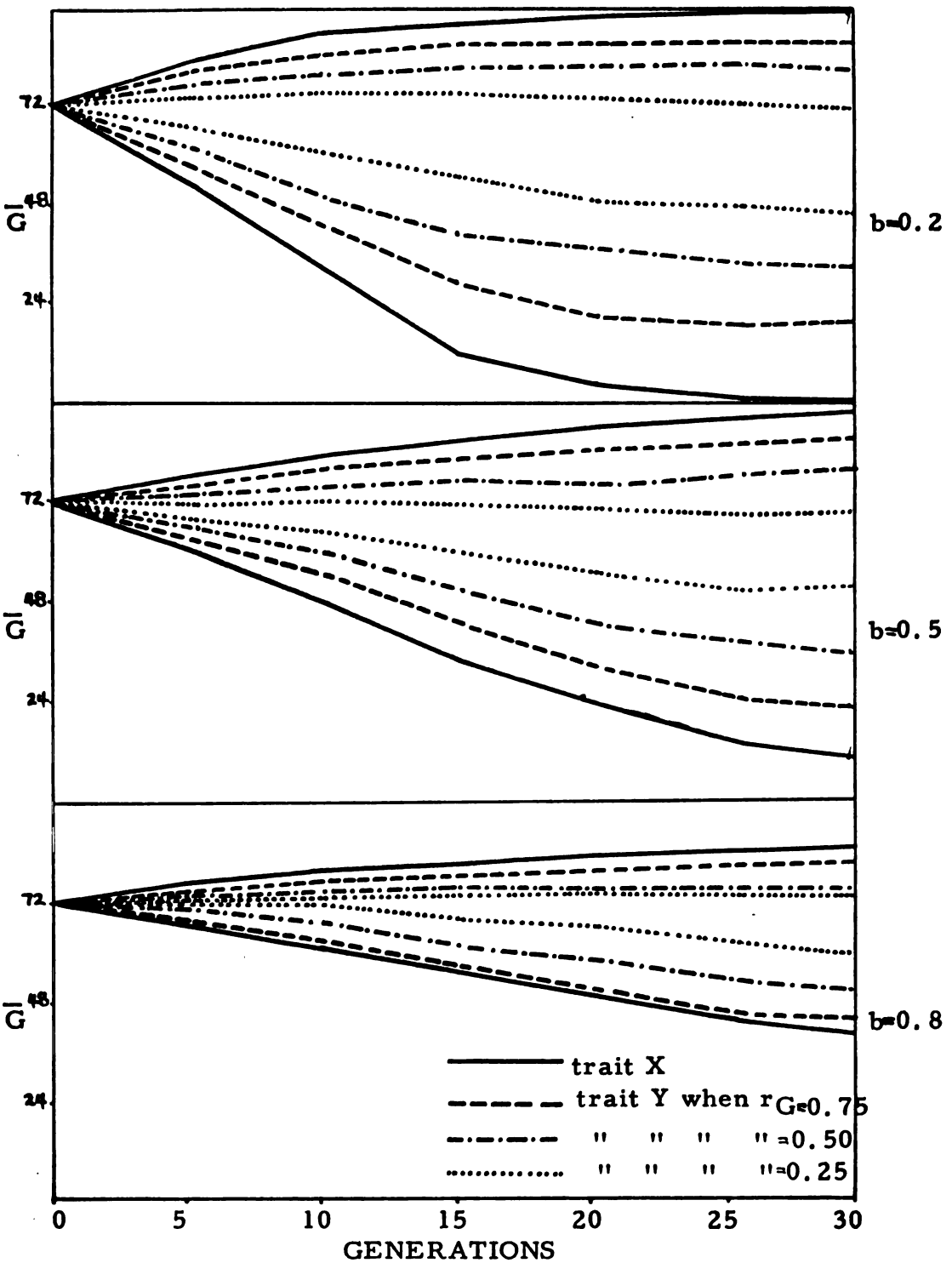

Figure 8.2 Mean genetic progress at three levels of selection when $h^2_x = 0.4$ (complete dominance). Upper four curves indicate selection by upper truncation, lower four selection by lower truncation.

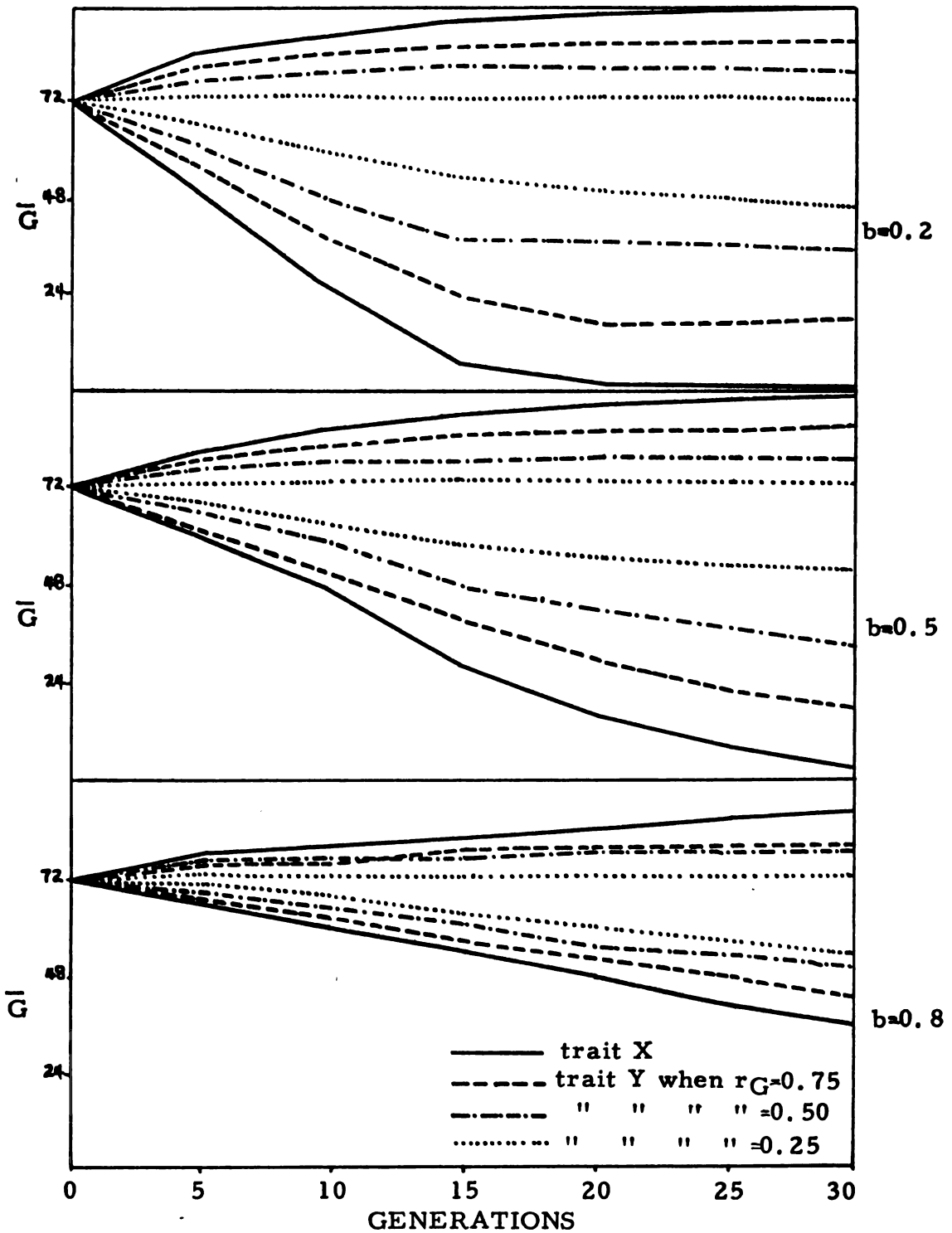

Figure 8.3 Mean genetic progress at three levels of selection when $h^2_x = 0.7$ (complete dominance). Upper four curves indicate selection by upper truncation, lower four selection by lower truncation.

model, results for the selected trait X were averaged over replicates, levels of environment of Y, and degrees of initial genetic correlation so that each point represents the average of 18 observations. Results for the correlated trait Y were averaged over replicates and levels of environment of Y, each point representing the average of six observations.

For complete dominance the expected maximum advance from selection was again different depending upon whether the trait was selected directly or whether the response came from indirect selection. In addition, the selection goal was different when selection was by lower truncation than when selection was by upper truncation. The differing selection goals for the two traits are shown in Table 4 for each direction of selection. The genotypic mean in the initial generation was 72 in every case.

Table 4. Advance possible in the genotypic mean of X and Y by different methods of selection and at different degrees of genetic correlation.

	Direction of Selection			
	Upwards		Downwards	
	Selection Goal	Max. Poss. Adv. (Units)	Selection Goal	Max. Poss Decl (Units)
Trait X	96	24	0	72
Trait Y(r_G=.25)	78	6	54	18
" " (r_G=.50)	84	12	36	36
" " (r_G=.75)	90	18	18	54

The maximum possible advance of the genotypic mean from selection by upper truncation was 24 units while the maximum possible decline from selection by lower truncation was 72 units. In every case the maximum response possible from downwards selection was three times that possible by upwards selection.

The results presented in Figures 8.1, 8.2, and 8.3 indicate that the response obtained from selection by lower truncation was considerably greater than that obtained from upper truncation. This asymmetry of response to selection made in opposite directions has been observed frequently in two-way selection experiments. In the population simulated here where there was complete dominance at each locus and all dominant alleles affected the trait in the same direction, genetic asymmetry of response to selection was expected. Falconer (1960) has referred to this condition as "directional dominance". If the initial gene frequency is 0.5, the response is expected to be greater in the direction which the alleles tend to be recessive. When the purpose of selection is to move the gene frequency of the desired gene from 0.5 to 1, then the rate of change of gene frequency is greater when selection is for the recessive allele. Lush (1945) has shown that selection for a dominant allele is most effective when the frequency of the allele is

about 0.3 while selection for a recessive allele is most effective when gene frequency is about 0.7.

The response to selection in opposite directions was quite symmetrical for the first few generations, which would be expected while the gene frequencies were still about 0.5 for the desired allele. However, as the discrepancy between the gene frequencies became greater, the response to selection for the recessive increased while selection for the dominant allele became much more difficult. When selection was upwards for the dominant allele, the gene frequency never rose higher than 0.95 even at high level of selection and low environmental variance. This simply illustrates that it is difficult to remove recessive genes when they are rare.

When level of selection was low ($b = 0.8$), the response was fairly linear at all levels of heritability for both directions of selection. As the intensity of selection increased, the response in both directions became distinctly curvilinear and the degree of asymmetry of response became greater. The response in both directions was considerably reduced in later generations when the frequency of the desired gene became low.

With regard to the correlated response to selection for the dominant allele, when the genetic correlation was only 0.25, the correlated response of trait Y was very small. In fact,

unless the level of selection was high (b = 0.2) and heritability was high, the response was generally negative over the 30 generations. Frequently some correlated response was observed in the early generations, only to be lost again by the 30th generation of selection. Clearly, when the genetic correlation was low, the correlated response to selection was less than was expected.

At intermediate genetic correlation (r_G = 0.5) the observed correlated response of the unselected trait again did not appear to be as great as would be expected from the direct response. The correlated response became curvilinear as levels of selection increased and as heritability increased, and most of the response had been achieved by the 15th generation of selection. Thereafter no further response was observed in Y and actually Y decreased in some cases (Figure 8.3, top graph).

At high genetic correlation (r_G = 0.75) the correlated response to selection followed the direct response of the selected trait more consistently although the same tendency to decrease after the 15th generation was again observed especially when level of selection was high.

When selection was by lower truncation for the recessive allele, the correlated response of the unselected trait was distinct and proportional to the direct response and, in most

cases, to the level of genetic correlation. However, in Figure 8.1, lower graph, the correlated response of Y to selection of X was actually greater when the genetic correlation was 0.25 than when the genetic correlation was 0.50. Random sampling in the correlated trait could account for this discrepancy of observed response from expectation.

The correlated response to selection was calculated as the per cent progress toward the selection goal for both directions of selection to illustrate more clearly the relative response at all levels of genetic correlation. The statistics for the 15th and 30 generations of selection were computed in exactly the same way as described previously for the additive model and are shown in Tables 5 and 6.

In Table 5 selection was by upper truncation for the dominant allele. The negative signs in the Table indicate that no progress toward the selection goal had been made for that treatment combination in that generation. The table illustrates more clearly those points already observed from the graphs. When the genetic correlation was low, no correlated response of Y to direct selection for X was achieved unless selection intensity was high. Where correlated response was achieved, more progress had been made toward the selection limit at the 15th generation than at the 30th generation. Unless the genetic

TABLE 5. The response in trait X and correlated response in Y at three levels of genetic correlation, measured as per cent of selection goal achieved at the 15th and 30th generation. (Complete dominance, selection by upper truncation.)

Level of Selection b	Level of Environment h_k^2	Generation	Response to Selection as Per cent of Selection Goal			
			Trait X	Trait Y r_G=0.25	Trait Y r_G=0.50	Trait Y r_G=0.75
0.8	0.1	15	10.4	-	6.3	4.3
		30	20.4	-	-	8.7
	0.4	15	29.6	-	8.9	21.8
		30	53.3	-	11.3	43.1
	0.7	15	41.3	-	38.9	31.2
		30	64.6	-	54.6	42.3
0.5	0.1	15	37.1	-	23.9	36.7
		30	58.3	-	19.8	54.6
	0.4	15	61.7	-	49.2	53.8
		30	86.7	-	63.5	74.8
	0.7	15	75.4	32.2	61.9	74.8
		30	94.2	19.7	61.4	87.9
0.2	0.1	15	60.4	26.3	50.6	54.6
		30	80.4	17.2	49.0	72.5
	0.4	15	83.3	41.7	76.4	75.1
		30	95.0	-	70.2	86.8
	0.7	15	91.7	38.8	77.8	81.3
		30	97.9	5.0	58.6	85.9

correlation is quite high and the gene frequency of the desired allele near the middle of the range, the correlated response to selection for a dominant allele will be very small.

When the genetic correlation was 0.5, correlated response to selection was achieved but never reached that which was expected. The table shows that the progress toward the selection goal was always less in the correlated trait than in the selected trait, and most of this progress had been achieved in the early generations of selection. In fact, when the level of selection was high, there was the same tendency for some of the progress achieved in the early generations to be lost in the later generations of selection.

When the genetic correlation was high ($r_G = 0.75$), there was much closer agreement between the correlated trait and the selected trait in per cent of selection goal achieved. Most of this progress was made in the first 15 generations, and the correlated trait never quite achieved as much relative progress as the selected trait.

In Table 6, where the results of selection by lower truncation are listed, a different outcome can be seen. Most progress toward the selection goal was made in the correlated trait when the genetic correlation was 0.25, and this greater relative progress was observed for all treatment combinations.

TABLE 6. The response in trait X and correlated response in Y at three levels of genetic correlation, measured as per cent of selection goal achieved at the 15th and 30th generation. (Complete dominance, selection by lower truncation.)

Level of Selection b	Level of Environment h_x^2	Generation	Response to Selection as per cent of Selection Goal			
			Trait X	Trait Y r_G=0.25	Trait Y r_G=0.50	Trait Y r_G=0.75
0.8	0.1	15	13.6	32.4	13.7	12.7
		30	27.6	74.4	32.9	26.0
	0.4	15	23.3	32.6	29.4	28.8
		30	46.2	63.5	60.5	52.4
	0.7	15	24.9	53.7	33.5	27.8
		30	52.8	100.6	63.6	56.1
0.5	0.1	15	28.6	39.0	30.2	29.3
		30	56.8	75.0	59.0	62.0
	0.4	15	50.8	66.9	57.6	51.6
		30	87.5	111.9	101.7	91.3
	0.7	15	59.2	79.8	67.2	59.4
		30	95.6	114.0	108.0	99.1
0.2	0.1	15	51.4	64.8	47.9	56.9
		30	84.2	115.7	89.3	89.6
	0.4	15	80.0	95.4	87.0	80.1
		30	99.3	143.9	109.6	99.2
	0.7	15	91.1	110.2	97.0	92.2
		30	100.0	141.8	108.5	104.4

Why this should be is not entirely clear unless the smaller goal possible in the correlated trait could cause a small deviation from the expected response to be magnified when expressed as a percentage. The trend, however, was quite distinct and consistent and occurred to a lesser degree in the relative response of the correlated trait when the genetic correlation was 0.5 and 0.75. When the genetic correlation was 0.75, the progress in the correlated trait was quite close to that achieved in the selected trait but had a tendency to be slightly greater in most cases. Random sampling in the unselected part of the correlated trait should not consistently result in changes in the direction of selection. Direct selection for a recessive allele when the frequency of the allele is greater than 0.5 will result in distinct response in a correlated trait not selected for.

The measurement of correlated response to selection indicates that the mode of gene action has a considerable effect on the amount of response to selection which can be achieved. The same population mechanisms which control the amount and rate of response to direct selection also apply to the correlated response of an unselected trait, at least in the simple models simulated in this study. For example, in the same way that response becomes more difficult to achieve in selecting for

a dominant allele as the recessive allele becomes rare, a correlated response in an unselected trait is also difficult to achieve. This difficulty feasibly could be greater in the more complex genetic systems operating in economic species. .

APPLICATION OF RESULTS AND SUGGESTIONS FOR FURTHER RESEARCH

In a discussion of the implications of the results presented, the limitations of the simple genetic models simulated should be borne in mind. Direct application of the results to the genetic improvement of economic species may not be appropriate. Yet, two points should be mentioned here. Firstly, estimates of genetic correlation computed from phenotypic covariances between parent and offspring should be regarded with considerable caution, especially if the number of observations is small. Secondly, if most of the genetic variance is additive, then correlated response to selection should conform rather well to theoretical expectation predicted from genetic covariance.

With regard to the direction and choice of parameters in further research the following suggestions may be useful:

1. A more detailed examination of the factors influencing the present method of estimating genetic correlations is required. A greater range of combinations of number of observations and selection intensity should be simulated. In the present study, either the number of observations was small or the selection intensity was high and both of these factors could reasonably

lead to unreliable estimates. Combinations where the number of observations was varied over the same level of selection and level of selection varied over the same number of observations would be interesting.

2. More complex models of gene action could be simulated including overdominance and epistatic models.

3. Different methods of selection would be of interest. For example, selection could be made simultaneously for both traits, or each trait could be selected in alternate generations.

4. The inclusion of linkage in some future model could be informative. The linkage relations simulated could either be within the pleiotropic loci or between pleiotropic and independent loci.

5. Genetic correlations other than the simple positive correlations simulated in the present study should be tried. More complex systems could include loci affecting each trait in the same direction or in opposite directions or both, leading to a net correlation which could range from -1 to +1. An initial genetic correlation of zero would be of interest, as would pleiotropic loci having unequal effects on the correlated traits.

Finally, although more complex systems should be simulated, the need for clear interpretation of the results obtained should

be stressed. Care must be taken that the complexity of the population simulated does not prevent interpretation.

SUMMARY AND CONCLUSIONS

This study was undertaken to examine the effects of truncation selection of a primary trait upon the genetic correlation and the correlated response of a secondary trait. Genetic populations and the process of selection were simulated through the use of random numbers generated by a high-speed computer.

Upper or lower truncation selection based on phenotype was made for only one of two quantitative traits, and the correlated response in the other trait was measured in each of 30 non-overlapping generations. The population was bisexual diploid, and the traits were expressed in both sexes. The size of the population of parents was 24 males and 24 females in each generation, and mating was random by sampling with replacement, the number of offspring produced being determined by the level of selection desired. Each trait was controlled by 48 loci segregating independently, and the genic effects were equal at every locus. Gene frequency was arbitrarily set at 0.5 at each locus in the initial generation.

Three degrees of genetic correlation, 0.25, 0.50, and 0.75, between the traits in the initial generation of offspring were simulated. Three levels of selection were simulated saving

the upper 20, 50 and 80 per cent of the offspring each generation. Three levels of environmental variation designed to produce heritabilities of 0.1, 0.4 and 0.7 under the additive scheme were considered for both traits. These factors and levels were then combined in a 3^4 factorial experiment and replicated to produce 162 parameter sets.

Two models of gene action were simulated, an additive model in which the contributions to the genotypic value were 2, 1, and 0 for the + +, + -, and -- phases at each locus, and a model of complete dominance in which the contributions were 2, 2, and 0 at each locus. In the model of complete dominance, the experiment was carried out separately for two directions of selection, upwards for the dominant allele and downwards for the recessive allele.

The genetic correlation caused solely by pleiotropy was determined by the number of loci which affected both traits in the same direction. The remaining loci of the 48 affecting each trait affected each trait independently. The genetic correlation was measured each generation as the product-moment correlation of genotypic values of each individual, and also by two variations of the method proposed by Hazel (1943) utilizing phenotypic covariances between parent and offspring.

Statistics of interest were calculated for each of the 30 generations of selection in a given parameter set, both in the unselected offspring and in those offspring selected to become parents of the next generation.

In the additive model the genetic correlation measured as the product-moment correlation of genotypic values in the offspring generation remained consistently near its initial level at all levels of environment and at all levels of genetic correlation when the fraction of offspring saved as parents was as high as one-half. When the fraction of offspring saved became as low as one fifth, some decrease in the genetic correlation was observed but only became extreme in later generations and only when the environmental variance was low. The levels of selection practised for traits in animal species would not have much effect on the magnitude of the genetic correlation unless heritability of the selected trait was high.

A closer examination of the genetic correlation indicated that at low selection intensity the genetic covariance between the traits was maintained over the 30 generations of selection. With greater selection intensity there was a decrease in the genetic covariance. But the genetic variances of the traits declined proportionately over the same period causing the genetic correlation to be maintained.

The effect of linear truncation of one variable on the genetic correlation was examined by comparing the correlation in the offspring generation with that from those offspring selected to become the parents of the next generation. Truncation selection caused a decrease in the genetic correlation in the truncated part of the distribution. The decrease became greater as heritability increased rather than as level of selection increased, the amount of the reduction depended on the heritability of the selected trait rather than on the degree of truncation selection.

Estimates of genetic correlation obtained from phenotypic covariances between parent and offspring were found to fluctuate markedly from the true correlation and no pattern was apparent although the true correlation tended to be underestimated. The results emphasize that it is unwise to be confident of genetic correlations from parent-offspring covariances in a population of the size simulated in this study and when selection is intense.

The correlated response of the unselected trait to selection of the primary trait agreed closely with response expected from theoretical considerations. The amount of response in the unselected trait was directly proportional to that in the selected trait in most cases and depended on the genetic covariance between the traits. Only when the genetic covariance was low

or when level of selection was low, did chance have any noticeable effect on the correlated response.

In the model of complete dominance, the change in the genetic correlation followed essentially the same pattern as in the additive model when selection was by upper truncation. The genetic correlation in each generation of offspring remained consistently high when level of selection was low or environmental variance high. At high level of selection and low environmental variance, the genetic correlation again tended to decline over the 30 generations although the amount of the decline was not as great as in the additive model. A detailed study of the genotypic variances and covariance showed that similar conditions were acting to maintain the genetic correlation as in the additive model.

When selection was by lower truncation, the behaviour of the genetic correlation under selection conformed closely to that for the additive model although the decrease in the correlation at high intensity of selection was more rapid than previously.

As in the additive model, truncation selection caused a decrease in the genetic correlation in the offspring selected to be parents. Similar results were obtained for both upper and lower truncation selection, the amount of reduction increased as heritability increased. Level of environmental variance was

again more important than level of selection in causing a decrease in the correlation in the selected group.

Estimates of genetic correlation computed from phenotypic covariances between parent and offspring were equally as poor in the model of complete dominance as those obtained previously for the additive model whether selection was by upper or lower truncation. In the majority of cases the two covariances in the numerator or denominator were of unlike sign and no estimate of the correlation was possible. The same tendency for the true genetic correlation to be underestimated was noted.

The response of the genotypic mean of the unselected trait to selection of the primary trait in opposite directions was quite symmetrical for the first few generations of selection but became distinctly asymmetrical in later generations. At low levels of selection the response was fairly linear at all levels of heritability for both directions of selection. But, as the intensity of selection increased, the response in both directions became distinctly curvilinear, and the degree of asymmetry of response became greater. The asymmetry was probably due to "directional dominance", the response being greater when selection was downwards for the recessive allele than when upwards for the dominant allele. The mode of gene action affects the amount

of response to selection which can be achieved, although the same mechanisms which control the amount of direct selection also apply to the correlated response of an unselected trait.

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