

RESPONSE TO SELECTION FOR PUPAL WEIGHT
WITH AND WITHOUT ASSORTATIVE MATING IN
TRIBOLIUM CASTANEUM

BY

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THESIS

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Joint effects of pre-selection mating system (random: R; assortative: A), selection mating system (R;A) and percent truncation selected (20%; 50%; 0% (control)) were studied in a $2 \times 2 \times 3$ factorial experiment with 5 replicates to examine direct response to selection for pupal weight (PW), and correlated responses for larval weight (LW) and pupation time (PT) in Tribolium castaneum. Prior to selection two groups, each with 50 males mated to 100 females were either R-mated or A-mated for 3 generations. At generation 3, 6 lines were formed in each group, one for each selection mating system-selection intensity combination, and selection began. Each line was maintained during 2 generations of selection by 10 pair-matings.

Half-sib (R-mated group) and minimum chi-square estimates of heritability (A-mated group) were, respectively, for PW: $.26 \pm .12$ and $.39 \pm .02$; LW: $.36 \pm .13$ and $.49 \pm .04$; and PT: $.29 \pm .08$ and $.25 \pm .03$.

Estimates of realized heritability of PW, calculated as the ratio of total response (deviated from the control lines) to cumulative selection differential, were:

Pre-selection x selection mating system

Percentage

<u>Selected</u>	<u>RR</u>	<u>RA</u>	<u>AR</u>	<u>AA</u>
20%	$.30 \pm .04$	$.31 \pm .11$	$.29 \pm .07$	$.39 \pm .04$
50%	$.12 \pm .04$	$.19 \pm .16$	$.23 \pm .08$	$.30 \pm .05$

Realized heritability of PW was increased by pre-selection assortative mating ($P < 0.10$), and by more intense selection ($P < 0.05$); the mating system during selection did not affect realized heritability. There were no clear correlated responses to selection for PW.

The experiment indicated the following: 1) assortative mating of parents for a few generations prior to selection enhanced direct short-term response to selection compared to random mating of parents; 2) assortative mating during selection did not significantly increase realized heritability, irrespective of selection intensity; 3) pre-selection assortative mating would not enhance short-term response per unit time (generation) of pre-selection and selection compared to selection from the onset; and 4) applying low selection intensity for the purpose of not constraining the effectiveness of assortative mating was not useful. The effects of assortative mating on long-term direct and correlated responses to selection need to be investigated.

DEDICATION
TO MY PARENTS AND MY WIFE

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CHAPTER ONE

INTRODUCTION

The effects of systems of mating on means, variances and covariances are of considerable importance in animal breeding theory and applications because these parameters play a major role in selection. Assortative mating (AM), defined here as the mating of individuals that are more phenotypically similar than if they were chosen at random from the population, is one of such mating systems.

Animal improvement programs usually aim to obtain maximum genetic change in a merit function (G) per generation following selection based on a predictor ($\hat{G}$). The expected genetic change per generation (ΔG) is given by

$$\Delta G = i\rho(G, \hat{G})\sigma_G \quad (1.1)$$

where i is the standardized selection differential; $\rho(G, \hat{G})$ is the correlation between the merit function and the predictor (also known as accuracy of selection); and σ_G is the standard deviation of the merit function. An increase in any of the above factors would be expected to also increase ΔG .

With random mating of selected parents, the only factors which can be manipulated by the breeder are the intensity and the accuracy of selection. Selection intensity can be increased by reducing the proportion of individuals saved for breeding, and the accuracy of selection can be enhanced

by collecting more information on the candidates for selection, e.g., progeny testing as a supplement to an individual record. On the other hand, AM offers an additional mean of enhancing ΔG because it can increase the genetic variance and the correlation between relatives. These two factors enter non-trivially in σ_G and in $\rho(G, \hat{G})$ respectively. Therefore, experimental information on the effects of AM on direct and correlated responses to selection is important for possible application in animal breeding programs.

Although AM in conjunction with selection appears to be a potentially useful tool to enhance the rate of improvement of animal populations, there has been little experimental work aimed to test its effectiveness. Further, the few experiments reported in the literature had design features that might have constrained the full realization of the benefits from AM of parents. These include lack of attempt to increase genetic variance in the base population by AM before selection, in order to elicit a larger response, and perhaps excessively high selection intensity. There has been no work aimed to study the effects of AM on correlated responses to selection.

The effects of AM for pupa weight in Tribolium castaneum were investigated in this study with the following objectives: 1) to estimate genetic and environmental parameters of pupation time, larval and pupal weights in unselected randomly mated or assortatively mated populations; and 2) to examine the joint effects of pre-selection mating system (random: R; assortative: A), selection mating system (R;A) and percent truncation selected (20%; 50%; 0% (control)) on direct and correlated responses to selection for pupal weight.

CHAPTER TWO

REVIEW OF LITERATURE

2.1. AN OVERVIEW OF MATING SYSTEMS

2.1.1 Introduction.

There are mainly two tools for genetic improvement of farm animals: selection and mating systems. Selection involves decisions on the extent to which individuals will contribute genes to the next generation. Mating systems involve determining which of these selected animals will mate with which or, equivalently, the control of the extent to which parents of the next generation will be genetically or phenotypically related to each other. The combination of a specific selection method with a mating system is often referred to as a breeding plan (Turner and Young, 1969). The breeding plan applied to a population greatly affects its genetic properties and the dynamics of genetic change.

2.1.2 Types of mating systems

Six different types of mating systems have been recognized in the literature. These are random mating, outbreeding, inbreeding, assortative mating, disassortative mating and selective or preferential mating. Under random mating, parents mate strictly at random, i.e., the probability of a mating occurring between two individuals of different genotypes is strictly a function of the genotypic frequencies in the population. Inbreeding and outbreeding are mating systems based on ancestral relationships. In inbreeding, mated animals are more genetically related to each other than a

pair sampled at random from the population; in outbreeding, ancestral relationships are deliberately avoided. Assortative and disassortative mating consist of mating individuals on the basis of their phenotypic similarity or dissimilarity, respectively. A selective or preferential mating system has been defined (Lutz, 1905) as one in which males or females belonging to a certain class of individuals find it more difficult to mate with individuals in other classes. Lewontin et al. (1968) have given precise definitions of the different systems of mating discussed above.

2.1.3 Consequences of mating systems: general.

Studies of the effects of mating systems on the genetic structure of populations began early in this century. Earlier work included contributions of Pearson (1904), Hardy (1908), Jennings (1912, 1916), Pearl (1913), Fisher (1918), Wright (1921), and Haldane (1924). These studies were primarily concerned with effects of mating systems on gene and genotypic frequencies, and on the correlations between relatives.

Systems of inbreeding were treated in great detail by Jennings (1917) Robbins (1917), Wright (1921) and Fisher (1949). Assortative mating was studied primarily by Fisher (1918) and Wright (1921), and dissasortative mating was considered by Hogben (1946), Li (1955) and Naylor (1962).

These authors showed that selective mating is character specific and that it involves change in gene frequencies. Assortative mating is character specific and it does not change gene frequencies. Inbreeding is not character specific, it does not affect gene frequencies and it does not change the mean of a population for a given trait except when genes are dominant. In this case, inbred individuals will have a lower mean than noninbred ones,

to an extent depending on the amount of inbreeding practiced. Inbreeding decreases the additive genetic variance within lines $V_w = (1 - F)V_0$, where V_0 is the random mating genetic variance and F is the inbreeding coefficient. However, it increases the additive genetic variance between lines, $V_B = 2FV_0$, and the total additive genetic variance, $V_T = (1 + F)V_0$ (Falconer, 1981).

The effects of inbreeding with non-additive gene action (epistasis and dominance) were studied by Robertson (1952). He found that an increase in the frequency of homozygotes resulting from inbreeding is accompanied by a dispersion of allelic frequencies and, generally, by a depression of mean values. However, King (1921), Hyde (1924), Sang (1964) and Dobzhansky (1970) have shown these effects to be secondary using laboratory experiments.

Outbreeding within a population results in effects opposite to those of inbreeding. The rate of loss of heterozygosity in an outbred finite population is approximately half of that under random mating (Turner and Young, 1969).

Rao and Narain (1967), among others, showed that assortative mating decreases the mean of a population only under dominance; the decrease in mean value is directly proportional to the decrease in heterozygosity. Assortative mating results in an increase in additive genetic variance (e.g., Fisher, 1918; Crow and Felsenstein, 1968); the opposite occurs under disassortative mating. Unlike inbreeding and outbreeding, which affect heterozygosity in all loci, assortative and disassortative mating affect heterozygosity in only a fraction of the total number of loci (Wright, 1921).

Evolutionary aspects of non-random mating systems in natural populations have been investigated by Wright (1964, 1965, 1969a), Parsons, (1965) and

Manning (1967). These authors concluded that mating systems have significant evolutionary consequences on the structure of populations.

2.2 THEORETICAL CONSEQUENCES OF ASSORTATIVE MATING WITHOUT SELECTION.

2.2.1 Introduction.

The method of study of the effects of assortative mating on a population depends on whether differences between individuals are of kind or of degree, i.e., qualitative or quantitative inheritance. Therefore, effects of assortative mating on qualitative and quantitative characters will be discussed separately in this section.

Qualitative differences are generally associated with the familiar Mendelian ratios and, in their study, gene and genotypic frequencies over generations and their limiting distributions are of primary interest. Quantitative differences depend on genes at many loci, the effects of which are not individually distinguishable. In this case Mendelian ratios are not observed and study centers on means, variances and covariances.

2.2.2 Qualitative inheritance.

Assortative mating for simple Mendelian traits does not occur frequently in animal populations. Assortative mating for pigment color or pattern, however, has been reported (Parsons, 1967). In spite of this, the theory of assortative mating for Mendelian traits is well developed.

The propensity to assortative mating can vary among phenotypic classes (Karlin, 1968). The following is an illustration using two alleles (A,a)

at an assorting locus. If A is dominant over a , there are two phenotypic classes, $A-$ and aa , with mating propensities α and β , respectively (Naylor, 1962). In the no dominance case, the mating propensities are assumed to be α , β and γ within AA , Aa and aa classes, respectively (Karlin and Farkash, 1978). In both cases, $0 \leq \alpha, \beta, \gamma \leq 1$. When $\alpha = \beta = \gamma = 0$, mating is at random, and when $\alpha = \beta = \gamma = 1$, every individual in the population is mating assortatively (complete assortative mating). If α , β and γ have values other than these two extremes, then some individuals are mating assortatively and the remainder are mating at random (incomplete or partial assortative mating). In these two models (complete and incomplete assortative mating) formulae have been derived describing effects of assortative mating on genic and genotypic frequencies, their limiting distributions at equilibrium and the number of generations required to reach equilibrium.

The analytical procedure used in these studies consists of stating mating types and frequencies in the base population and then deriving recursive relationships which link one generation with the next. Equilibrium frequencies are obtained from the recursive relationships, and local stability and polymorphic equilibria are assessed through evaluation of the eigenvalues of the gradient matrix at each equilibrium point (O'Donald, 1960). Basic assumptions usually employed include discrete generations, no selection and large population sizes so that random genetic drift is negligible and changes in genotypic frequency can be followed deterministically.

2.2.2.1 Assortative mating and the Hardy-Weinberg equilibrium.

The effect of assortative mating for a qualitative trait can be examined,

as a starting point, by investigating departures from the Hardy-Weinberg (HW) equilibrium found under random mating.

Wright (1922) recognized two sources of departure from HW frequencies (inbreeding and assortative mating) and used the following formula as a general expression of genotypic frequencies in the absence of mutation, migration and selection:

$$(p^2 + Fpq)AA + 2(pq - Fpq)Aa + (q^2 + Fpq)aa \quad (2.1)$$

where p and q are gene frequencies for A and a alleles, respectively, and F is the "fixation index" (Wright, 1969a, p. 174), a measure of departure from HW frequencies arising from inbreeding or assortative mating ($0 \leq F \leq 1$). When F is used as coefficient of inbreeding, Wright (1969, p. 174) described this formula as used in the "narrow sense", and when F is used as a measure of correlation between uniting gametes, arising either from a system of inbreeding or assortative mating, he referred to the formula as being used in the "broad sense". From the above expression, it is clear that when $F > 0$, genotypic frequencies depart from HW frequencies and the maximum departure occurs when $F = 1$.

In the absence of mutation, migration and selection, Price (1971) and Stark (1976) have shown that the deviation from HW equilibrium depends, in general, on the degree of assortative mating between parents and on differences in weighted mean gene frequencies between male and female parents. They also showed that this deviation is not influenced by the deviation in the parental generation.

2.2.2.2 Complete assortative mating.

A model of complete assortative mating assuming dominance at one locus with two alleles, was initially treated by Jennings (1916), extended by Wentworth and Remick (1916), and discussed later by other authors (e.g., Wright, 1921; Li, 1978). With this model, gene frequencies do not change as a result of assortative mating. However, there is usually a reduction in the proportion of heterozygotes (H) such that after an infinite number of generations of assortative mating H tends to zero.

A character controlled by two loci each with two alleles ($A,a;B,b$), without dominance, was discussed by Wright (1921). He found that complete assortative mating leads eventually to two extreme classes ($AABB$ and $aabb$). This is in contrast to inbreeding, where the frequencies of the four homozygote types ($AABB, AAbb, aaBB, aabb$) tend to become equal. This result holds, however, only if the frequency of each allele in the base population is 0.5. When the alleles in the two loci have different frequencies Ghai (1974) showed that in the limit, the population will consist of 3 classes of homozygotes. If the frequency of A is smaller than that of B , then the 3 homozygote classes in the limit will be $AABB, aaBB$ and $aabb$; if the opposite is true, the homozygote genotypes will be $AABB, AAbb$ and $aabb$. He also considered an n -locus model without dominance and found that when gene frequencies at all loci are different, complete assortative mating will result in $n + 1$ classes of homozygote genotypes present in the equilibrium population, one for each phenotypic group. However, gene frequencies remain unchanged.

2.2.2.3 Incomplete assortative mating.

In a one-locus situation where genotypes have distinct phenotypes, the

results of incomplete assortative mating are similar to those of partial selfing. This was first worked out by Wright (1921) who showed that incomplete assortative mating resulted in a reduction in the proportion of heterozygotes. After t generations, the inbreeding coefficient is given by

$$F_t = \frac{\alpha}{2 - \alpha} + (F_0 - \frac{\alpha}{2 - \alpha}) (\frac{\alpha}{2})^t \quad (2.2)$$

where α is the proportion of individuals in the population mating assortatively and F_0 is the initial proportion of heterozygotes. If, however, genotypes (AA, Aa, aa) differ in their propensity to mate assortatively, the above expression does not reduce to the one obtained with mixed selfing and random mating (O'Donald, 1960). In this case, no simple recursive expressions are available although formulae for equilibrium heterozygosity can be obtained for very small values of α , β and γ , the assorting propensities for AA, Aa and aa individuals, respectively.

O'Donald (1960), Karlin (1968) and Lange (1976), among others, considered a one locus-two alleles case with complete dominance. A proportion α of dominants and recessives mated assortatively within their classes, and a proportion $(1 - \alpha)$ mated randomly. It was shown that a unique stable equilibrium exists, provided that α is sufficiently small, which is approached at a geometric rate proportional to α . If fractions α and β of dominants and recessives, respectively, mate assortatively and the rest of the individuals mate at random, an unstable equilibrium results (Karlin, 1968). For $\alpha > \beta$, the population's final state is AA; for $\alpha < \beta$ the final state is aa. The approach to the "pure recessive" state is faster than that to a "dominant state".

Some workers (e.g., O'Donald and Davis, 1977) observed that in natural populations there is a significant preference for heterozygote mates among heterozygotes, and for homozygote mates among homozygotes. This was investigated by Naylor (1962), Otto (1977) and Karlin and Farkash (1978). When the preference rate for heterozygotes (β) exceeds that for homozygotes (α), the unique stable solution is the central Hardy-Weinberg equilibrium with $\text{Prob.}(AA) = \text{Prob.}(aa) = 1/4$ and $\text{Prob.}(Aa) = 1/2$. This equilibrium is approached at a geometric rate. If, however, $\beta < \alpha$, fixation occurs.

2.2.2.4 Assortative mating and incomplete penetrance.

The effect of assortative mating on a population depends on the exactness with which potential parents are assigned to particular mating groups. Factors influencing the expression of a genotype such as dominance, affect the consequences of assortative mating. It may also happen that genes do not produce their full effects in combination with certain other genes or under certain environmental conditions, i.e., when penetrance is incomplete. Several autosomal dominant genes have been found to exhibit this phenomenon (Allen, 1952).

Raddy and Narain (1972), and Otto (1981), investigated the consequences of assortative mating under incomplete penetrance for one locus, with and without dominance. In these cases, the population attains equilibrium with all three genotypes present unlike, for example, complete penetrance and no dominance where the population ultimately becomes homozygous. Equilibrium is attained, however, at a slower rate. The distribution of genotypes at equilibrium depends on the initial gene frequency and on the penetrance parameter. The inbreeding coefficient at equilibrium decreases as penetrance decreases irrespective of the initial gene frequency.

2.2.2.5 Assortative mating and mutation.

From an evolutionary viewpoint, it is of interest to evaluate the "chance of success" of a new allele resulting from mutation in a population undergoing assortative mating.

The work of Parsons (1962) and Moore (1979) indicates that when alleles mutate from one form to another, a stable nontrivial equilibrium results. However, one of the two alleles becomes rare under incomplete assortative mating. In the case of dominance and complete assortative mating, with mutants affecting fitness in some way, Parsons (1962) gave conditions that must be met for the new allele to increase in frequency. Let k , h and b be the relative viabilities of AA , Aa and aa individuals, respectively. When $\underline{A}$ is introduced in the population the condition is $h > 2b$ or $k > 2b$; when $\underline{a}$ is introduced, the condition is $h > k$ or $b > k$. In the case of no dominance and complete assortative mating, the conditions for the new genes to increase in frequency are those found under complete selfing, namely: $h > 2k$ and $b > k$ when the $\underline{a}$ gene is introduced and $h > 2b$ and $k > b$ when the $\underline{A}$ gene is introduced.

2.2.3 Quantitative inheritance.

Many traits of interest in animal breeding are inherited quantitatively. Parameters of interest in quantitative inheritance are means, variances and covariances. These depend on the distribution of parental mating types and on the frequency distribution of phenotypic values in the offspring. Three groups of models of assortative mating for quantitative traits have been described in the literature. The first group includes notably the models of Fisher (1918), Wright (1921) and Bulmer (1980). The second and third groups include the models of Karlin (1979a-d) and of Cavalli-Sforza and Feldman

(1976, 1978), respectively. Models in the first and third groups have been referred to by Karlin (1980) as of "linear assortative mating". Those in the second group were referred to by the same author as of "nonlinear assortative mating". Differences between these classes of models will be discussed later.

Most of these models follow Fisher's (1918) theory of assortative mating. Fisher (1918) assumed that the phenotypic values (X, Y) of members of a mating pair followed a bivariate normal distribution with correlation coefficient μ . If females and males with standardized values x and y have frequencies N and M , respectively, following Fisher the frequency of occurrence of $x - y$ pairs would then be

$$f_{xy} = NM(1 + \mu xy) \quad (2.3)$$

with $f_{xy} = NM$ under random mating. When μ is positive, f_{xy} is larger than under random mating; with negative μ , then f_{xy} is smaller. Fisher (1918) constructed distributions for progenies out of these matings and derived means, variances and covariances.

More recent formulations (e.g., Karlin, 1980) assume that the relationship between phenotypic values of parents and offspring can be written as

$$\underset{\sim}{X}_0 = T(\underset{\sim}{X}_m + \underset{\sim}{X}_f) + \underset{\sim}{\epsilon} \quad (2.4)$$

where $\underset{\sim}{X}_0$, $\underset{\sim}{X}_m$ and $\underset{\sim}{X}_f$ are vectors of phenotypic values in offspring, male and female parents, respectively and $\underset{\sim}{\epsilon}$ is an independent random residual component arising in connection with the segregation process. Under "equal

transmission of inheritance", $\underline{T} = (1/2)\underline{I}$, where $\underline{I}$ is an identity matrix; otherwise, $\underline{T}$ is a matrix which weights differentially the contributions of the two parents. Let $\text{Var}(\underline{X}_{\underline{t}}) = \underline{\Sigma}_{\underline{t}}$ (the variance is the same in the two sexes); $\text{Cov}(\underline{X}_{\underline{m}}, \underline{X}_{\underline{f}}) = \underline{M}$; $\underline{T} = (1/2)\underline{B}$ and $\text{Var}(\underline{\epsilon}) = \underline{E}$. From (2.4) the following recursive relationship can be established

$$\underline{\Sigma}_{\underline{t}+1} = 1/2(\underline{B}\underline{\Sigma}_{\underline{t}}\underline{B}' + \underline{B}\underline{M}\underline{B}') + \underline{E} \quad (2.5)$$

At equilibrium (Karlin, 1980)

$$\underline{\Sigma} = 1/2(\underline{B}\underline{\Sigma}\underline{B}' + \underline{B}\underline{M}\underline{B}') + \underline{E} \quad (2.6)$$

This recursion is linear on $\underline{\Sigma}$ and $\underline{M}$ and this is the reason why Karlin (1980) has termed models of this sort as of "linear assortative mating". In this model it is assumed that the marital covariance matrix ($\underline{M}$) is time invariant and that the number of mating encounters is infinite. Karlin (1979 a-d) relaxed this assumption using models in which the establishment of a particular type of mating depends on the frequency of encounters between males and females. These are mediated by a mate selection function that determines whether the encounter culminates in reproduction or not. This model causes the marital covariance matrix to change over time.

2.2.3.1 Consequences of assortative mating on means.

The effect of assortative mating on the mean of a quantitative character in a population has been studied using single locus models and then generalizing results by summing over all loci affecting the trait in question.

Rao and Narain (1967) considered a segregating locus. When dominance was complete the mean of the population decreased following one generation of assortative mating. This decrease was proportional to the decrease in heterozygosity. Without dominance, assortative mating had no effect on the mean value. With two linked loci and complete dominance, the mean of the population decreased more after assortative mating than when there was no linkage.

Crow and Felsenstein (1968) have shown that under assortative mating the inbreeding coefficient F (a measure of decrease in heterozygosity) is

$$F = \frac{r}{2 N_e(1 - r) + r} \quad (2.7)$$

where N_e is the effective number of loci, and r is the correlation between mates. Hence, with a large number of loci and with additive gene action, little change in the mean of the population should be expected following assortative mating. Likewise, only a slight change in the mean value of a polygenic trait should occur when dominance is present.

2.2.3.2 Consequences of assortative mating on the genetic variance.

In the absence of epistasis, the variance of a quantitative trait can be partitioned into additive, dominance and environmental components. As discussed in the following section, the variance among phenotypic values in progeny obtained from assortative mating of parents can be decomposed into variance among family means, variance within families and covariance between family means and parental values.

Breese (1956) discussed the decomposition of variability for a two locus model with two alleles each, with and without dominance. In the case of no dominance, he showed that assortative mating increased the variance among

family means but it did not affect the genetic variance contained within families. The increase in the variance between family means resulted from an increase of homozygote-homozygote (AABB and aabb) combinations; this became more important as the number of factors increased. With dominance, Breese (1956) found that the increase in the variance of family means depended on "re-enforcement" effects of heterozygote-heterozygote and homozygote-heterozygote combinations. With a large number of loci affecting the trait, the within-family genetic variance also remained unaffected by assortative mating under dominance.

Rao and Narain (1967), using a single locus model, showed that if assortative mating is practiced for an infinite number of generations, all the genetic variance tends to concentrate in the variance among family means. They also showed that under dominance, the total genetic variance after one generation of assortative mating would be larger than with random mating if $q > q_0$ (q : frequency of the recessive allele and $0.6 \leq q_0 \leq 0.7$), but smaller otherwise.

Binet (1963) concluded that as assortative mating proceeds, the total genetic variance might: a) decrease throughout, or b) increase over some generations and decrease over other generations, or c) increase throughout. He stated that the outcome depends on whether the more or the less frequent allele is dominant, and on the relationship between the degree of dominance and the difference in frequencies of the alleles. In the case of equal frequency of the two alleles (irrespective of the degree of dominance), or in the absence of dominance (irrespective of differences between allelic frequencies) the total genetic variance would increase throughout with complete assortative mating. This agrees with the results of Breese (1956) which were based on the assumption of equal gene frequencies.

The phenotypic variance under assortative mating (e.g., Crow and Felsenstein, 1968) can be written as

$$P_{t+1} = A_{t+1} + D + E \quad (2.8)$$

where A_{t+1} , D and E are additive, dominance and environmental components of variance after t generations of assortative mating. Crow and Felsenstein (1968), Vetta (1976) and Bulmer (1980) have argued that dominance and environmental components of variance do not change appreciably, if at all, with assortative mating. Thus, assortative mating affects primarily the additive genetic variance. Crow and Felsenstein (1968), among others, have shown that

$$A_{t+1} = 1/2[(1 + \rho h_t^2)A_t + A_0] \quad (2.9)$$

where ρ is the phenotypic correlation between mates, h_t^2 is the heritability of a trait at generation t and A_0 is the additive genetic variance under random mating. The increase in phenotypic variance resulting from assortative mating thus depends on an increase in additive genetic variance. This, in turn, depends on ρ and on h^2 . At equilibrium (e.g., Felsenstein, 1981)

$$P = \frac{A}{1 - r} + D + E \quad (2.10)$$

where r is the genetic correlation between mates. Feldman and Cavalli-Sforza (1979) have criticized this equilibrium formulation because as r approaches 1, the variance goes to infinity, a situation which has not been observed in any known biological system. The model of Karlin (1979a,b) does

not present this problem. However, noting that $r = \rho h^2$, there is only one situation in which r may be equal to 1, i.e., $\rho = h^2 = 1$. This situation is unlikely to occur in living organisms.

2.2.3.3 Consequences of assortative mating on correlations between relatives.

The assessment of the degree of resemblance between relatives is of basic importance in the study of quantitative traits. Resemblance can be measured in terms of covariances between relatives which, in turn, provide means of estimating additive genetic and other components of variance in a population.

Fisher (1918) derived correlations between various close relatives and expressed them in terms of the correlation between spouses (μ) and of broad and narrow sense heritabilities (c_1 and c_2 , respectively). For example:

$$E[\text{Corr}(\text{parent, child})] = 1/2 c_1 c_2 (1 + \mu), \quad (2.11a)$$

$$E[\text{Corr}(\text{full-sibs})] = 1/4 c_1 (1 + c_2 + 2c_2 A) \quad (2.11b)$$

where A is the genetic correlation between spouses ($A = c_1 c_2 \mu$). The parameters (e.g., c_1 , c_2 , μ) can be estimated by equating observed correlations to their expectations. The above expressions indicate that correlations between relatives are larger under assortative mating than under random mating ($\mu = 0$). The increase depends on μ and on the heritability of the trait in question. Nagylaki (1978) showed that with Fisher's (1918) assumptions, the formulae for the correlation between various close relatives hold irrespective of linkage relationships.

Vetta (1976) disagreed with Fisher's (1918) formulae for parent-offspring and sib correlations. He argued that in the presence of dominance, the sib correlation should be greater than the parent-offspring correlation. He introduced the following corrections to the parent-offspring (r_{PO}) and sib (r_{OO}) correlations:

$$r_{PO} = 1/2 c_1 c_2 [1 + A(1 - A)^2] + 1/2 c_1 (1 - c_2) A(1 - A) \quad (2.12a)$$

$$r_{OO} = 1/2 c_1 c_2 [1 + A(1 - A)^2] + 1/4 c_1 (1 - c_2). \quad (2.12b)$$

These correlations are also larger under assortative mating than under random mating. Bulmer (1977) has pointed out that with Vetta's (1976) corrections, the correlations are no longer an increasing function of μ .

Wright (1921) used path coefficients to derive parent-offspring and sib correlations under assortative mating without dominance, and obtained results similar to those of Fisher (1918). Crow and Felsenstein (1968) extended Wright's (1921) results, allowing for dominance and for correlations between allelic effects at different loci. Their results are shown in Table 1.

In humans, the choice of a mate may be based on phenotype or on socio-economic status. Two major types of assortative mating have been distinguished in humans (Cloninger et al., 1979): phenotypic homogamy and social homogamy. Models of phenotypic homogamy in the presence of "cultural" inheritance are used in the study of behavioral traits such as coefficient of intelligence (e.g., Wright, 1978). Models of social homogamy have been used in studying physiological traits such as lipoprotein concentration and blood pressure (e.g., Morton and Rao, 1979). The correlations between relatives derived from these models are larger than those obtained under random mating

Table 1. Equilibrium correlations between relatives in a random mating and in an assortatively mated population* (modified from Crow and Felsenstein, 1968)

Relationship	Correlation	
	Random	assortative
	mating	mating
Parent-offspring	$1/2 h^2$	$1/2 h^2(1 + \rho)$
Grandparent-grandoffspring	$1/4 h^2$	$1/4 h^2(1 + \rho)(1 + A)$
Great grandparent-great grand-offspring	$1/8 h^2$	$1/8 h^2(1 + \rho)(1 + A)^2$
Sibs	$1/2h^2 + 1/4D$	$1/2h^2(1 + A) + 1/4D$
Double first cousins	$1/4h^2 + 1/16D$	$1/4h^2(1 + 3A) + 1/16D$
Uncle-niece	$1/4h^2$	$1/4h^2(1 + A)^2 + 1/8DA$
First cousins	$1/8h^2$	$1/8h^2(1 + A)^3 + 1/16DA^2$

* h^2 : equilibrium heritability; A: equilibrium genetic correlation between mates; D: ratio of dominance variance to total variance at equilibrium; ρ : phenotypic correlation between mates.

particularly when "cultural" transmission is assumed. These correlations were derived using the method of path analysis. Results obtained by Eaves (1975) for correlations between relatives under "cultural" transmission and assortative mating are presented in Table 2; letting $A = 0$, the results apply to random mating with "cultural" transmission.

It is important to investigate the effects of assortative mating for trait X on the covariance between X and Y . Reeve (1953, 1961) studied in detail the estimation of heritability and of genetic correlations in a progeny test involving assortative mating of parents sampled from a random mating population. This author gave particular emphasis to the expectations of the regression of offspring on mid-parent and of the components of variance and covariance arising in the analysis of full-sib family groups. He observed that assortative mating for X did not affect the expected values of the regression coefficients of X and Y on the mid-parent values of X . However, the expected values of the regression coefficients of X and Y on the mid-parent values of Y were altered by assortative mating. This was a function of the increases in the mid-parent variance of X and Y caused by assortative mating and of the heritabilities of these traits.

Latter (1965) derived similar formulae appropriate for half-sib families in a population at equilibrium under assortative mating for trait X . He also considered the effects of genotype $\times$ environment interaction on estimates of genetic parameters under assortative mating with the assumption of additive genetic effects. He found that the parent-offspring covariance for trait Y increased by an amount which depended on the genetic correlation between traits X and Y , on the phenotypic correlation between mates and on the heritability of each trait. The covariance between traits X in the parents and Y in the offspring, and the covariance between trait

Table 2. Correlations between relatives under "cultural" transmission and assortative mating (modified from Eaves, 1975).

Correlation with	Expected correlation*
Parent	$\frac{1}{2} \left[1 + \frac{2A(1-A)}{H} \right] \left(\frac{1}{1-A} \right) H + E_C$
Grandparent	$\frac{1}{2} \left[1 + \frac{2A(1-A)}{H} \right] \left(\frac{1}{1-A} \right) \left(\frac{1+A}{2} \right)^2 H$
Monozygotic twin	$\frac{1}{2(1-A)} H + \frac{1}{4} D + E_C$
Full-sibs	$\frac{1}{2(1-A)} \left(\frac{1+A}{2} \right) H + \frac{1}{16} D + E_C$
Uncle or aunt	$\frac{1}{2(1-A)} \left(\frac{1+A}{2} \right)^2 H + \frac{A}{32} D$
First cousins	$\frac{1}{2(1-A)} \left(\frac{1+A}{2} \right)^3 H + \frac{A^2}{64} D$

* H and D are the additive and dominance components of variance as a % of the total; A: additive genetic correlation between mates; E_C : environmental correlation.

Y in the parents and trait X in the offspring also increased with assortative mating.

Gianola (1982), using the approach of Bulmer (1980), described the effects of assortative mating on the additive genetic correlation between traits X and Y, and on the equilibrium covariances for traits X and Y between sibs, offspring and parents, and grand-offspring and grandparents (Table 3). He also discussed the effect of "mixed" assortative mating, a system which induces a correlation between the phenotypic values of traits X in one sex and Y in the other. It was observed that "mixed" assortative mating increased the genetic correlation between traits X and Y. This suggests that this mating system could be used to enhance larger correlated responses to selection. Fernando and Gianola (1983), investigated "index" assortative mating, where the ordering of mating pairs is based on a linear combination of traits, and found that the sign of a genetic correlation could be changed using this mating system. Changes in the genetic correlation depended on the correlation between mates, on the heritabilities of the traits involved, and on the initial genetic correlation between traits.

Risch (1979) investigated equilibrium phenotypic correlations between various relatives due to autosomal and X-linked loci with proportional gene effects in the two sexes under assortative mating. Assuming an additive genetic model and normality, the author derived correlations between father and son (FS), mother and son (MS) father and daughter (FD) and mother and daughter (MD). These were:

Table 3. Expectation of offspring-parent and sib covariances under assortative mating for trait X (modified from Gianola, 1982).

COVARIANCE	EXPECTATION*
A. Offspring-parent	
1. (X, X)	$\sigma_{A_x}^2(1 + \rho)/2$
2. (X, Y)	$(\sigma_{A_{xy}} + h_x^2 \rho \sigma_{xy})/2$
3. (Y, X)	$\sigma_{A_{xy}}(1 + \rho)/2$
4. (Y, Y)	$\sigma_{A_y}^2(1 + \theta^2 \rho h_x^2)/2$
B. Full-sibs	
5. (X, X)	$\sigma_{A_x}^2(1 + \rho h_x^2)/2$
6. (X, Y)	$\sigma_{A_{xy}}(1 + \rho h_x^2)/2$
7. (Y, Y)	$\sigma_{A_y}^2(1 + \theta^2 h_x^2 \rho)/2$
C. Half-sibs	
8. (X, X)	$\sigma_{A_x}^2[1 + h_x^2 \rho(2 + \rho)]/4$
9. (X, Y)	$\sigma_{A_{xy}}[1 + h_x^2 \rho(2 + \rho)]/4$
10. (Y, Y)	$\sigma_{A_y}^2[1 + \theta^2 h_x^2 \rho(2 + \rho)]/4$

* ρ : phenotypic correlation between mates; $\sigma_{A_x}^2$, and $\sigma_{A_y}^2$: equilibrium additive genetic variances for X and Y; h_x^2 : equilibrium heritability for X; $\sigma_{A_{xy}}$: equilibrium additive genetic covariance; θ : equilibrium genetic correlation; σ_{xy} : equilibrium phenotypic covariance.

$$r_{FS} = \frac{1}{2\beta\lambda} \rho(s_f^2 + c_f) + \frac{1}{2\beta\lambda} \rho(a_f^2 + c_f) + \frac{1}{2} a_m^2 + \frac{1}{2} c_m \quad (2.13a)$$

$$r_{MS} = \frac{1}{2\beta\lambda} (s_f^2 + c_f) + \frac{1}{2\beta\lambda} (a_f^2 + c_f) + \frac{1}{2} \rho a_m^2 + \frac{1}{2} \rho c_m \quad (2.13b)$$

$$r_{FD} = \frac{1}{2} \rho h_f^2 + \beta\lambda(s_m^2 + c_m) + \frac{\beta\lambda^2}{2} (a_m^2 + c_m) \quad (2.13c)$$

$$r_{MD} = \frac{1}{2} h_f^2 + \rho\beta\lambda(s_m^2 + c_m) + \frac{\beta\lambda^2}{2} \rho(a_m^2 + c_m) \quad (2.13d)$$

where ρ is the phenotypic correlation between mates; f and m designate female and male respectively; λ is the ratio of male phenotypic variance to female phenotypic variance; β represents "dosage compensation" (e.g., when effects in male and females are identical, that is with no X-inactivation, then $\beta = 1$; when X-inactivation is complete, $\beta = \frac{1}{2}$); a_f^2 and s_f^2 are ratios of autosomal and X-linked genetic variance to phenotypic variance; c_f and c_m are ratios of covariances between autosomal and X-linked effects to phenotypic variance. Again, these correlations are large under assortative mating than under random mating; the latter are obtained by letting $\rho = 0$.

2.2.3.4 Assortative mating and linkage equilibrium.

The effect of introducing assortative mating in a population which is in linkage equilibrium is of evolutionary interest. Vetta (1975) showed that assortative mating would disturb linkage equilibrium which would be eventually reestablished after a number of generations irrespective of the recombination frequency.

Wilson (1978), using the approach of Crow and Kimura (1970), showed that linkage disequilibrium occurs when assortative mating is introduced into a population in linkage equilibrium. Contrary to Vetta's (1975) results,

Wilson (1978) found that this disequilibrium would continue as long as assortative mating is practiced in the population. Wilson (1978) pointed out that the difference between these two results arises from the fact that Vetta (1975) did not take into consideration the effect of crossing over which would make it possible to obtain coupling heterozygotes AB/ab from the union of Ab and aB gametes, and repulsion heterozygotes Ab/aB from the union of AB and ab gametes. Fisher (1918), however, showed that when the two loci are tightly linked, the linkage equilibrium would not be disturbed by assortative mating.

2.3 GENETIC CONSEQUENCES OF ASSORTATIVE MATING WITH SELECTION: THEORY AND EXPERIMENTAL RESULTS.

2.3.1 Introduction.

Selection occurs when there is differential reproductive success among genetically distinct individuals. This stems from differences in the ability of individuals to reproduce or to produce surviving offspring. Differential reproduction or survival can arise from natural or artificial causes and selection is then termed natural or artificial. Carmelli and Karlin (1975) stated that the basic difference between natural and artificial selection is that the latter is always frequency dependent, i.e., the types removed by selection depend on their frequency in the population. In natural selection, the types removed depend on their individual potential to survive and reproduce.

The basic effect of selection is the elimination from the population of alleles that lower the fitness of their homozygote or heterozygote carriers. Thus, selection can change the mean and the variance of a character. Bulmer (1980) pointed out that selection affects genetic variability by changing

gene frequencies and by inducing linkage disequilibrium, which causes correlations between loci. Bulmer (1976) partitioned the changes in genetic variance as a result of selection into those due to changes in gene frequencies, linkage disequilibrium and departures from Hardy-Weinberg. He then demonstrated that a population will first respond to a change in intensity of selection by a change in the linkage disequilibrium component. This response is gradually replaced by a change in the gene frequency component.

When there is assortative mating with selection, mating types and their frequencies and the viability of each genotype need to be specified in order to calculate the genotypic distribution in the following generation. Many models used in the study of assortative mating with selection are concerned with the conditions required for the establishment of stable equilibria. The joint effects of assortative mating and selection are discussed in the following sections.

2.3.2 Theory.

2.3.2.1 Assortative mating under both natural and artificial selection.

Carmelli (1977) and Carmelli and Karlin (1975) studied one-locus models combining artificial and natural selection under random and assortative mating. They considered a case with dominance, in which culling favored the dominant phenotypes but the recessives were the most fit, i.e., artificial selection opposing natural selection. They found that the population evolves to fixation and whether A or a is fixed depends on its initial composition. Assortative mating speeds up the rate of fixation to the desired type just slightly over that occurring under random mating. They

found that the approach to a pure recessive state takes place at a faster rate than that to the dominant state. When culling favors a dominant character and the heterozygotes are the most fit, the population does not evolve to fixation (Carmelli and Karlin, 1975). However, the equilibrium frequency of the dominant phenotype under assortative mating exceeds that under random mating, and the equilibrium is achieved with a lower degree of culling. In this case, the effect of natural selection is overriding that of artificial selection; even with severe culling, complete fixation of the desirable allele cannot occur.

2.3.2.2 Assortative mating under natural selection.

Karlin and Farkash (1978) investigated a model in which fractions α of homozygotes (AA,aa) and β of the heterozygotes (Aa) mate assortatively. The viability parameter for homozygotes was 1 and that for heterozygotes was $(1 + s)$; the selective advantage of the heterozygotes then depends on s being greater than zero. They observed that in the absence of natural selection ($s = 0$) and with $\beta > \alpha$, a unique stable realization is the central Hardy-Weinberg equilibrium. However, if $\alpha > \beta$ then fixation of one allele occurs. With selection, three stable states are possible: fixation of A or a, or symmetric equilibrium. The final state depends on α , β and s . For example, if $\alpha - \beta - 2s/(1 + s) > 0$, the population evolves towards fixation; if $(1 + s)(\alpha - \beta) - s(2 + s) < 0$ the population evolves to a symmetric equilibrium.

Campbell (1980) extended the model of Karlin and Farkash (1978). In addition to the case where homozygotes assort with homozygotes without discriminating whether the mate is AA or aa, he considered a situation where a proportion α of the homozygotes is mated only with individuals of

the same genotype. In the latter case, the likelihood of a symmetric equilibrium increases with β , the "assorting tendency" among heterozygote individuals. It was also observed in this case that the proportion of heterozygotes at equilibrium decreases as α increases. This is in contrast to the "no discrimination" case where the equilibrium frequency of heterozygotes is independent of the "assorting tendencies". The reason for this is that in the case of discrimination among homozygote types, matings involving homozygotes do not produce any heterozygotes.

Falk (1971) considered a one locus-two alleles model, with and without dominance. He showed that with complete assortative mating and no dominance, a stable polymorphism occurs if the fitness of the heterozygotes is more than twice that of the homozygotes. This is a stricter condition than under random mating where only higher fitness of the heterozygotes is required to attain a polymorphic equilibrium. With dominance and complete assortative mating a polymorphic equilibrium occurs if

$$\frac{h}{b} > \frac{h-a}{h-b} \quad (2.14)$$

where a , h and b are the relative viabilities of AA , Aa and aa , respectively. Falk (1971) pointed out that the above inequality is always satisfied when the fitness of AA individuals is greater than that of aa individuals.

2.3.2.3 Assortative mating under artificial selection.

A common form of artificial selection is truncation selection. This involves establishing a threshold value and keeping as parents of the next generation those individuals exceeding it.

Snow (1912) reported that the regression of offspring on parent increased as the intensity of selection and the phenotypic correlation between mates increased. Selection and assortative mating, however, decreased the correlation between parent and offspring.

Baker (1973) considered the effect of truncation selection followed by assortative mating of parents on genotypic frequencies and the genetic correlation between mates, and on the effectiveness of selection. He found that selection caused negative correlations between and within loci. These correlations were more strongly negative at lower gene frequencies or with fewer genes. Selection substantially reduced the large genetic correlation between mates that can be achieved with assortative mating of parents without selection. He argued that the effectiveness of assortative mating in increasing additive genetic variance and subsequent response to selection would be reduced by selection of parents. The net effects of assortative mating and selection on additive genetic variance ranged from an increase of 1.3% for a lowly heritable trait at high selection intensity, to one of 23.2% at high heritability and low selection intensity. Baker (1973) concluded that assortative mating might increase response to selection from 1 to 10% depending on heritability and selection intensity.

2.3.3 Experimental results.

There has been limited experimental work aimed at investigating the effects of assortative mating in plants and animals. Breese (1956) studied assortative mating in Nicotiana rustica and found that it increased significantly the variance among family means for flowering time, by as much as 43% over that under random mating. It was also observed that assortative mating for flowering time also resulted in a significant

correlation between mates for plant height, a character positively correlated with flowering time. This work drew attention to the possibility of increasing response to selection by using assortative mating of parents.

McBride and Robertson (1963) studied the effectiveness of assortative mating of selected individuals in increasing response to selection for chaeta score in Drosophila melanogaster. They reported a larger selection response with assortative mating than with random mating. Realized heritability was estimated at .52 for assortative mating and at .30 for random mating. They also attributed this increase in selection response to a larger selection differential resulting from assortative mating.

Wilson et al. (1965), conducted an experiment to evaluate the effects of mating systems and of selection on pupa weight of Tribolium castaneum. They reported that the average response in mass selected and assortatively mated lines was larger (realized heritability = 0.35) than in mass selected and randomly mated lines (realized heritability = 0.29). However, the difference in realized heritability was not statistically significant.

In the two experiments described above no attempt was made to increase genetic variance in the base population by assortative mating before selection. This might lead to a larger response immediately after selection begins. In the experiment of McBride and Robertson (1963), selection intensity was high (10% selected) so the theoretical effectiveness of assortative mating in increasing response to selection might have not been fully realized (Baker, 1973).

Rico et al. (1973), simulated effects of mating and of selection systems on response to selection using 3 linkage groups, each with 4 equally spaced loci. Two selection intensities ("high" and "low") were employed. They observed that the largest response to directional selection was obtained

with additive gene action, assortative mating and high selection intensity. In as far as selection intensity is concerned, this is in conflict with the theoretical results of Baker (1973). They also found that in the presence of dominance, assortative mating resulted in a negative response to intense selection. This result is in agreement with the theory that, with dominance, assortative mating of parents results in a reduction of progeny mean value proportional to the loss of heterozygosity (Rao and Narain, 1967).

De Lange (1974) simulated response to selection under random mating, assortative and disassortative mating. He reported that with equal additive gene effects and independent segregation, the conditions required for significant differences in response were: a large number of loci affecting the trait, high heritability and low selection intensity. These conditions are in agreement with Baker's (1973) results. When these conditions were simulated, he observed that assortative mating of selected parents gave the largest response followed by random mating.

2.4 ESTIMATION OF GENETIC PARAMETERS IN ASSORTATIVELY MATED POPULATIONS

2.4.1 Introduction.

The concepts of breeding value, dominance and epistatic deviation for a quantitative trait developed by Fisher (1918) and others depend upon unknown gene frequencies and genotypic values, the latter being unobservable. However, the observed variance can be partitioned into additive, dominance, epistatic and environmental components (e.g., Cockerham, 1954) which render the above concepts useful for practical purposes.

Genetic gain from selection depends upon the fraction of the phenotypic variance that is genetic. Hence, estimates of variance components provide

information which is useful for designing effective breeding plans. In addition, the estimation of variance components is intimately related to prediction of breeding value of candidates for selection (Falconer, 1981).

Theory and methods of estimating components of variance with linear statistical models have been reviewed by many workers, e.g., Crump (1951), Searle (1968, 1971a), Anderson (1979) and Searle (1979), and details of different methods of estimation will not be discussed here.

The estimation of genetic parameters under random mating assumptions proceeds in the following manner: the expected values of estimates of variance (covariance) components are written in terms of covariances between relatives which, in turn, are linear combinations of components of variance and covariance due to additive, dominance, epistatic and environmental effects (Fisher, 1918; Wright, 1935; Cockerham, 1954; Willham, 1963). In most methods, mean squares or reductions in sums of squares are equated to their expectations in terms of a genetic model, the parameters of which are then solved for directly (e.g., environmental variance) or calculated indirectly (e.g., heritability).

Under assortative mating, correlations between relatives can be non-linear in the parameters and the usual assumptions made, such as lack of correlation between sire and dam effects, are not tenable. The usual methods are, therefore, no longer suitable. Methods that have been described in the literature for this case are discussed below.

2.4.2 Estimation of genetic parameters under assortative mating.

As pointed out before, correlations between relatives under assortative mating can be expressed as nonlinear functions of parameters of a given genetic model. Estimation procedures which have been used in the literature,

particularly in human populations where assortative mating is frequent, include ordinary least squares, weighted least squares, minimum chi-square and maximum likelihood. In this section we describe minimum chi-square and maximum likelihood procedures as employed in human genetics. For details, the reader is directed to Jinks and Fulker (1970), Burt (1971), Jinks and Eaves (1974), Eaves (1975), Goldberger (1978) and Rao and Morton (1978).

An $m \times 1$ vector $\tilde{x}$ of observed correlations between relatives can be described by the model

$$\tilde{x} = f(\tilde{\theta}) + \tilde{\epsilon}, \quad (2.15a)$$

where $f(\tilde{\theta})$ is a vector of functions of the $q \times 1$ parameter vector $\tilde{\theta}$, and $\tilde{\epsilon}$ is an $m \times 1$ vector of residuals. In minimum chi-square (Eaves, 1975), the estimator of $\tilde{\theta}$ is the vector minimizing the weighted sum of squared deviations

$$\begin{aligned} \chi^2 &= \tilde{\epsilon}' \tilde{W} \tilde{\epsilon} \\ &= [\tilde{x} - f(\tilde{\theta})]' \tilde{W} [\tilde{x} - f(\tilde{\theta})] \end{aligned} \quad (2.15b)$$

where $\tilde{W}$ is a non-singular matrix of weights. In general, $\tilde{W} = \tilde{V}'\tilde{V}$ with $\tilde{V}$ being a non-singular matrix; when $\tilde{W}$ is diagonal, $\tilde{V}$ is a diagonal matrix with typical element $v_{ii} = w_{ii}^{1/2}$. Let

$$\tilde{b} = \tilde{V}[\tilde{x} - f(\tilde{\theta})] \quad (2.16)$$

of order $m \times 1$, and

$$\underline{\underline{P}} = \frac{\partial \underline{\underline{b}}'}{\partial \underline{\underline{\theta}}} \quad (2.17)$$

be the $q \times m$ matrix of first partial derivatives of the m weighted deviations, $\underline{\underline{b}}$, with respect to the q parameters of $\underline{\underline{\theta}}$. Then

$$\frac{\partial \chi^2}{\partial \underline{\underline{\theta}}} = -2 \underline{\underline{P}} \underline{\underline{b}} \quad (2.18)$$

and $\underline{\underline{P}} \underline{\underline{b}} = \underline{\underline{0}}$ needs to be solved for $\underline{\underline{\theta}}$. However, this system is non linear and an iterative procedure is required to solve it, e.g., Newton-Raphson or Gauss-Newton.

Goldberger (1978) regards the elements of $\underline{\underline{x}}$ as approximately normally distributed and use "maximum likelihood" procedures to estimate $\underline{\underline{\theta}}$. The logarithm of the joint likelihood function, $L(\underline{\underline{\theta}})$, is then

$$\ln L(\underline{\underline{\theta}}) = c - \frac{1}{2} \underline{\underline{b}}' \underline{\underline{b}} \quad (2.19)$$

where c is a constant. Note that the log-likelihood is maximized when $\underline{\underline{b}}' \underline{\underline{b}}$ is minimized. Hence, in this case "maximum likelihood" and minimum-chi square lead to the same estimates of $\underline{\underline{\theta}}$. In general, the matrix $\underline{\underline{W}}$ needed to compute the "maximum likelihood" estimates is a function of $f(\underline{\underline{\theta}})$. One possible approach is to evaluate $\underline{\underline{W}}$ in terms of the solution for $\underline{\underline{\theta}}$ at each iterate. When the elements of $\underline{\underline{x}}$ are uncorrelated, $\underline{\underline{W}}$ is diagonal and Eaves (1975) suggested using the observed vector $\underline{\underline{x}}$ as a trial estimate of $f(\underline{\underline{\theta}})$, and then taking

$$\frac{(n_i - 2)}{[1 - f_i^2(\underline{\underline{\theta}})]^2} \quad (2.20)$$

as the i th diagonal element of $\tilde{W}$. In this case n_i is the number of pairs used in calculating the i th correlation.

Goldberger (1978) and Rao and Morton (1978) work with z-transforms of $\tilde{x}$ in order to obtain a better approximation to normality, and to reduce the dependence of $\tilde{W}$ on $\tilde{\theta}$. Fisher (1921) showed that if x_i is an estimated inter-class correlation based on n_i pairs of observations with $E(x_i) = \rho_i$, then the bias-corrected z-transform

$$z_i = \frac{1}{2} \ln \left(\frac{1 + x_i}{1 - x_i} \right) - \frac{\rho}{2(n_i - 1)} \quad (2.21)$$

is distributed normally with mean

$$E(z_i) = \frac{1}{2} \ln \left(\frac{1 + \rho_i}{1 - \rho_i} \right) \quad (2.22)$$

and variance

$$\text{Var}(z_i) = \frac{1}{n_i - 3}. \quad (2.23)$$

In the case of an intra-class correlation, x_i , the bias-corrected z-transform is

$$z_i = \frac{1}{2} \ln \left(\frac{1 + x_i}{1 - x_i} \right) + \frac{1}{2} \ln \left(\frac{n_i}{n_i - 1} \right) \quad (2.24)$$

with mean as before, and variance

$$\text{Var}(z_i) = \frac{1}{n_i - 1.5} \quad (2.25)$$

Then $\tilde{\theta}$ is estimated by minimizing the criterion

$$\chi^2 = \sum_{i=1} n_i [z_i - E(z_i)]^2 \quad (2.26)$$

with

$$E(z_i) = \frac{1}{2} \ln\left(\frac{1 + \rho_i}{1 - \rho_i}\right),$$

and $\rho_i = f_i(\tilde{\theta})$. Under the null hypothesis $H : E(\tilde{z}) = \tilde{0}$, with $\tilde{z}$ being a vector of z -transforms, then χ^2 has a central chi-square distribution with $m - q$ degrees of freedom. This provides a basis for testing the goodness of fit of the model.

The residual χ^2 can be used for testing hypotheses about genetic models (Rao and Morton, 1978). For example, if χ^2_{m-q-w} is the value of χ^2 when fitting $q + w$ parameters and χ^2_{m-q} is its value when only q parameters are fitted, then

$$\chi^2_w = \chi^2_{m-q} - \chi^2_{m-q-w} \quad (2.27)$$

can be referred to a central chi-square statistic with w degrees of freedom. This provides a likelihood ratio test for the hypothesis that the additional w parameters are null.

Smith (1980) reviewed methods of estimating correlations between relatives starting from the work of Galton (1877). He proposed a simple method for simultaneous estimation of the elements of x , i.e., correlations and cross-correlations (e.g., sister's height and brother's weight) between relatives. The correlations are obtained as

$$\rho = \frac{B}{A + B} \quad (2.28)$$

where B and A are "between" and "within" sibs variance components, respectively. The following is a summary of how A and B are obtained.

Let

$$\mu = \frac{\sum_f w_f \bar{x}_f}{\sum_f w_f} \quad (2.29)$$

be a weighted estimator of the population mean (μ) where $\bar{x}_f$ is the observed mean of the family f and

$$w_f = \left(\frac{A}{n_f} + B \right)^{-1} \quad (2.30)$$

where n_f is the number of individuals in the family. Let

$$\xi_f = \bar{x}_f - \mu \quad (2.31)$$

and calculate

$$S_A = \sum_f w_f^2 n_f^{-1} \xi_f^2 + \alpha^2 \Delta \quad (2.32a)$$

$$S_B = \sum_f w_f^2 \xi_f^2 \quad (2.32b)$$

respectively, where $\Delta = \sum_f \sum_i (x_{fi} - \bar{x}_f)^2$, and

$$\alpha = A^{-1} \quad (2.33)$$

Smith (1980) showed that nearly enough

$$E(S_A) = A(\sum_f w_f^2 n_f^{-2} + \alpha^2 v) + B \sum_f w_f^2 n_f^{-1} \quad (2.34a)$$

$$E(S_B) = A \sum_f w_f^2 n_f^{-1} + B \sum_f w_f^2 \quad (2.34b)$$

where $v = \sum (n_f - 1)$. Then

$$\begin{aligned}
 \underset{\sim}{S} &= \begin{bmatrix} S_A \\ S_B \end{bmatrix} = \underset{\sim}{T} \begin{bmatrix} A \\ B \end{bmatrix} \\
 &= \begin{bmatrix} \sum w_f^2 n_f^{-2} + \alpha^2 v & \sum w_f^2 n_f^{-1} \\ \sum w_f^2 n_f^{-1} & \sum w_f^2 \end{bmatrix} \begin{bmatrix} A \\ B \end{bmatrix} \quad (2.35)
 \end{aligned}$$

and

$$\begin{bmatrix} A \\ B \end{bmatrix} = \underset{\sim}{T}^{-1} \underset{\sim}{S} \quad (2.36)$$

Equations (2.29) to (2.36) can be used to find μ , A , B , w_f and ξ_f

iteratively as follows:

- (a) Let A = the mean square within-families, and $B = A$ provisionally;
- (b) Set $\alpha = A^{-1}$, and find w_f from (2.30), μ from (2.29), ξ_f from (2.31), S_A and S_B from (2.32a,b) and T from (2.35). Obtain new "improved" values of A and B from (2.36);

(c) With the "improved" values of A and B , repeat (b) until A and B stabilize. Then calculate ρ with (2.28). Smith (1980) also showed how this technique can be extended to correlations between different classes of relatives and to "cross-correlations". When distributions are normal and homoscedastic, the method gives maximum likelihood estimates and their error variances and covariances (Smith, 1980).

Most workers have used the method of maximum likelihood to estimate correlations between relatives (e.g., Rao and Morton, 1978). The following

illustrates the procedure. Suppose X_1 and X_2 are the phenotypic values of the sire and dam, respectively, and X_3 through X_{p+2} are phenotypic values of p progeny. Let $E(X_1) = \mu_1$; $E(X_2) = \mu_2$; $\text{Var}(X_1) = \sigma_1^2$; $\text{Var}(X_2) = \sigma_2^2$; $E(X_i) = \mu_3$ and $\text{Var}(X_i) = \sigma_3^2$, for $i = 3, \dots, p+2$. Thus

$$\text{Cov}(X_i, X_j) = \begin{cases} \sigma_i^2 & \text{for } i = j \leq 2 \\ \sigma_3^2 & \text{for } i = j > 2 \\ \rho_{ij} \sigma_i \sigma_j & \text{for } i < j \leq 2 \\ \rho_{i3} \sigma_i \sigma_3 & \text{for } i \leq 2; j > 2 \\ \rho_3 \sigma_3^2 & \text{for } j > i > 2 \end{cases} \quad (2.37)$$

Assuming a multivariate normal distribution for

$$Y' = [X_1 - \mu_1, X_2 - \mu_2, X_3 - \mu_3, \dots, X_{p+2} - \mu_3] \quad (2.38)$$

and $\text{Var}(Y) = \Sigma$, the log-likelihood for the i th family is given by

$$\ln L_i \propto -\frac{1}{2} (\ln |\Sigma| + Y' \Sigma^{-1} Y) \quad (2.39)$$

The log-likelihood is then a function of 4 correlations, 3 variances and 3 means, i.e., 10 unknowns. With n independent families, the overall log-likelihood is then

$$\ln L = \sum_i \ln L_i, \quad (2.40)$$

Estimates of the unknown parameters can be obtained by maximizing the overall likelihood.

From the preceding discussion, estimates of genetic parameters in an assortatively mated population depend on: 1) the method of estimating the elements of x , the vector of correlations between relatives, and 2) the method chosen to estimate θ , the genetic and environmental parameters of the assumed genetic model.

2.5 DESIGN OF SELECTION EXPERIMENTS AND ESTIMATION OF REALIZED GENETIC PARAMETERS

2.5.1 Introduction.

Selection experiments are usually designed so that maximum information can be obtained given constraints on the amount of experimental material, available facilities, time and cost. It is important to use designs which yield unbiased estimates of selection response, and of realized genetic parameters having satisfactory confidence limits. After the work of Robertson (1960), it became clear that artificial selection in a finite population results in the chance loss of desirable alleles. In turn, this leads to a decreased limit to selection.

Prout (1962), Hill (1971) and Bohren (1975) showed that in a finite population, genetic drift can be an important source of variation among selection lines, thus producing variation in response among lines treated identically. Bulmer (1976) and Avery and Hill (1977) showed that genetic drift can also be an important source of variation within lines. Inbreeding depression (Robertson, 1961) and environmental trend can cause bias in estimates of response to selection.

Precise interpretation of results and of estimates of parameters from selection experiments can be hampered by: a) lack of an experimental design

permitting separation of response to selection from the effects of genetic drift, inbreeding depression and environmental trend; and b) difficulties in measuring the amount of artificial selection actually applied for a given trait when natural selection is operating (Dickerson, 1969). The problem of design then becomes that of allocating experimental units to selection treatments such that the influence of the factors mentioned above can be controlled or accounted for.

2.5.2 Design of selection experiments.

2.5.2.1 Control of environmental trend:

Bell et al. (1955) and Dickerson (1969) stated that unbiased comparisons between the effectiveness of different methods of selection can be made even if the magnitude of environmental trend and of absolute response to selection are not measured. They also stated that for this to be possible, differences in environmental effects affecting experimental units allocated to alternative selection treatments need to be random during the period in which comparisons are made. On the other hand, estimation of actual response to selection requires accounting for the influence of environmental trend. Two methods are usually employed for this purpose: unselected control lines, and repeat mating designs; divergent selection can also be used to remove environmental trend (Dickerson, 1969). Nicholas (1980) showed that the difference in variance of response between a selection experiment with a control line and one without it is given by $\sigma_d^2 - 2\sigma_c^2$, where σ_d^2 is the drift variance and σ_c^2 is the environmental variance common to all individuals. Therefore, when $\sigma_c^2 > .5 \sigma_d^2$ the variance of response is smaller with a control. In this case a selection experiment would have less

chance of success if a control line is not included in the design.

Similarly, the population size required to attain a given probability of success would be larger without a control line.

Genetic change occurring in control populations as a result of random changes in gene frequencies, natural selection or epistatic effects on performance, can still bias estimates of response to selection (Dickerson, 1969). This suggests that the usefulness of control lines depends on the validity of the assumption that they have remained unchanged genetically during the course of selection. This assumption is expected to hold better in short-term than in long-term selection experiments.

An improvement over the relative accuracy of the measurement of response achieved using an unselected control line can be obtained with divergent selection. Each selected line then acts as a control for the other (Falconer, 1981) and the response is measured as the difference between the two divergent lines. The expected response to mass selection when an unselected control line is used is half of that under divergent selection (Hill, 1980). Thus, the coefficient of variation of response to divergent selection is half of that using an unselected control line (Hill, 1980; Nicholas, 1980). This suggests that a design incorporating divergent selection is more efficient than one using a control line. An unselected control line, however, is preferable to divergent selection if one is interested only in the response in one direction. Divergent selection does not yield estimates of selection response in each direction alone (Dickerson, 1969) and the response is not always equal in the two directions (Falconer, 1981).

In repeat-mating designs (Dickerson, 1969), sires used in a previous generation are re-mated together with those which are to be used in the

current generation. All sires are mated to "current" dams. Repeat mating thus results in overlapping generations. Genetic change is then measured by making comparisons between contemporary individuals belonging to sires of different generations (Falconer, 1981). Smith (1962) showed that with this design 4 times as many records are required to measure with the same accuracy a response of 0.1 standard deviations as one of 0.2 standard deviations; this indicates that larger facilities are required to establish a small response to selection than a large one. Thus, this design seems to be best suited to measure response to selection involving highly heritable traits. Smith (1962) calculated the total number of progeny required by this design and by one in which an unselected control was used, to obtain the same precision for an estimate of response over a number of years. He found that the numbers required by the unselected control line method were 25% of those needed by the repeat-mating scheme. He concluded that the unselected control line should be the method of choice to measure response to selection.

2.5.2.2 Control of inbreeding depression.

The effect of inbreeding in finite populations undergoing selection was discussed by Robertson (1961). He developed expressions describing the magnitude of the effects of inbreeding and showed that these were greater under intense selection or when the heritability of the selected character was high. Bohren (1975) has argued that inbreeding depression might be less severe in selected than in unselected lines of the same effective size because desirable dominant alleles would tend to be held at high frequencies by selection.

Bias from inbreeding depression can be reduced by maintaining a large effective population size (Robertson, 1961) and by avoiding, as much as

possible, matings between relatives. Also, inbreeding depression can be accounted for in a selection experiment by maintaining the same effective population size in both selected and unselected control lines. However, the expected rate of increase of inbreeding is higher in a selected than in a control line (Robertson, 1961).

Bohren (1975) suggested to maintain replicate selected and unselected control lines in order to cross them at the end of the experiment. This would permit estimation of the total response to selection free from inbreeding depression. Hill (1980) pointed out that under additive gene action, the coefficient of variation of the response in the cross between two sublines is the same as that of a single line of twice the size. Hence, the method suggested by Bohren (1975) does not result in a loss of efficiency.

2.5.2.3 Control of drift variance.

Hill (1980) showed that the drift variance of a mass selected line is a function of: a) size of the population undergoing selection; b) duration of the selection experiment; c) intensity of selection; and d) heritability and genetic standard deviation of the trait under consideration. In designing selection experiments for a given trait the first three factors can be manipulated.

Comstock (1974, 1977) discussed a method of calculating the minimum population size required to ensure a certain probability of fixation of a desirable allele. This approach requires knowledge of the initial frequency and of the selective value of the allele. These are usually unknown so the approach is not practical.

Hill (1980) gave expressions for finding the minimum effective population size needed to attain a certain coefficient of variation of the response

given the selection intensity, the duration of the experiment and the heritability of the trait. His formulae are appropriate for short term experiments as they require the assumption of constant variance.

Nicholas (1980) investigated two practical criteria which could be used to calculate the size of a selection line. These were the coefficient of variation of the response and the chance of success. A "successful" selection experiment was defined as one in which the observed response is greater than a certain proportion (β) times the expected response. For a mass selection program with intensity of selection i and heritability h^2 , the population size required for the coefficient of variation of the response to be θ after t generations is approximately $2/(\theta ih)^2 t$. Likewise, the size required for the chance of success to be α after t generations is approximately $2[z_\alpha/(\beta - 1)ih]^2/t$ where z_α is the standard normal deviate corresponding to a probability α , and β is as defined above. When selection is applied in both sexes, ih above is replaced by the average genetic superiority of the selected individuals in the two sexes. Nicholas (1980) also gave results applicable to overlapping generations.

For a fixed total number (N) of individuals to be scored, Hill (1971) showed that the variance of response in a line of size N is the same as the variance of mean response in n lines each of size N/n . Therefore, replication has no effect on precision in this case. However, the major advantage of replication is that the variance among lines and the variance of realized heritability estimates can be estimated directly without stringent assumptions and without the requirement of knowing the true parameter values. In addition, replication is essential for estimating the sampling variance of realized genetic correlations because the drift variance cannot be estimated from estimates of genetic variance (Hill, 1980). The theory developed by

Nicholas (1980) can be used to determine the number of replicates required to bring the coefficient of variation down or the chance of success up to a particular level.

Basically, the duration of an experiment depends primarily on its objectives. If these are to estimate genetic parameters or to test hypotheses about the base population from which the selection lines are drawn, the experiment is usually of short duration, e.g., five generations or less (Hill, 1980). If the objectives are to measure or utilize changes in rates of response, the duration is longer than five generations (Hill, 1980). It has been shown (e.g., Hill, 1971) that if a control population is used or if divergent selection is practiced, the greater part of the sampling variance in response due to drift depends primarily on the total number of individuals recorded in the experiment rather than on its duration.

Bohren (1975) suggested the use of single generation selection experiments to obtain unbiased estimates of genetic parameters in the base population. He argued that in this case drift would not contribute to the variance between replicates. The drift variance in a selection line is $t\sigma_A^2/N$, (Nicholas, 1980), where t is the number of generations of selection, σ_A^2 is the additive genetic variance and N is the number of individuals selected as parents each generation. From this expression it is clear that Bohren's (1975) argument would be true only in cases where N is very large.

For a given effective population size, duration of experiment and heritability of the trait, Soller and Genizi (1967) and Hill (1972) have shown that there is a selection intensity which minimizes the drift variance and, therefore, the coefficient of variation of response to selection. For a normally distributed trait and a long-term experiment this optimal selection

intensity is 27%. They pointed out that selection intensities between 15 and 25% will give good efficiency even when the optimal intensity is lower.

2.5.3 Estimation of realized heritability and of realized genetic correlations.

Falconer (1954, 1981) defined realized heritability as the ratio between response to selection obtained and selection actually applied. This statistic can be used to check predictions made prior to the experiment, or to provide estimates of heritability which can be more precise than those obtained from covariances between relatives (Hill, 1972).

Four estimators of realized heritability were studied by Hill (1972).

These are: 1) regression of cumulative response on cumulative selection differential (h_C^2); 2) regression of individual generation response on selection differential (h_I^2); 3) ratio of total response to total selection differential (h_T^2); and 4) a maximum likelihood estimator (h_M^2). A comparison of the sampling variances of these estimators was made by Hill (1972). He observed, assuming a constant selection intensity, that except when heritability is very close to zero, the linear estimators h_C^2 , h_T^2 and h_I^2 were almost as good, in the sense indicated above, as h_M^2 . He recommended the use of h_C^2 for most circumstances, and h_T^2 when heritability is high or when the experiment is of long duration. The estimate of sampling variance of h_C^2 obtained from the method of least-squares is biased downwards because, with drift, the variance increases with generation number and generation means are correlated. The ordinary least-squares assumptions do not hold in this case. Hill (1972) presented formulae that give "almost unbiased" estimates of the sampling variance.

If a selection experiment can be replicated, the sampling variance of realized heritability can be estimated from the variance between replicates, independently of the parameter estimates and without requiring stringent assumptions (Hill, 1980). However, when there are only a few replicates, the estimate of sampling variance would be unreliable and it is not clear whether the variance between replicates or Hill's (1972) formulae should be used.

Dickerson (1969) stated that one of the purposes of estimating genetic correlations is to predict the genetic change in one character (Y) that will result from selection for another (X). Therefore, reliable estimates of genetic correlations can be obtained from well designed selection experiments. The direct response for trait X (R_X) and the correlated response for the other trait Y (C_Y) can be estimated in one line, and the direct response for trait Y (R_Y) and the correlated response for trait X (C_X) can be estimated in a second line. Following Dickerson (1969), an estimator of realized genetic correlation ($r_{g_{XY}}$) from one line is then

$$r_{g_{XY}} = \frac{C_Y}{R_X} \cdot \frac{\sigma_{A_X}}{\sigma_{A_Y}} \quad (2.41)$$

where σ_{A_X} and σ_{A_Y} are the genetic standard deviations for trait X and Y, respectively. These genetic standard deviations are estimated from the estimates of realized heritabilities (h_X^2 and h_Y^2), and from estimates of phenotypic variances ($\sigma_{P_X}^2$ and $\sigma_{P_Y}^2$) as

$$\sigma_{A_i} = (h_i^2 \sigma_{P_i}^2)^{1/2}, \quad (2.42)$$

where $i = X$ or Y . Thus, two estimates of genetic correlation are obtained, one from each line. The geometric mean of the two estimates is (Falconer, 1981)

$$r_{g_{XY}} = \left(\frac{C_Y}{R_X} \cdot \frac{C_X}{R_Y} \right)^{1/2} \quad (2.43)$$

The realized phenotypic correlation ($r_{p_{XY}}$) may be obtained (Dickerson, 1969) as

$$r_{p_{XY}} = \frac{S_Y}{S_X} \cdot \frac{\sigma_{p_X}}{\sigma_{p_Y}} \quad (2.44)$$

where S_Y is the selection differential of the correlated trait Y, and S_X is the selection differential for the selected trait X; σ_{p_X} and σ_{p_Y} are phenotypic standard deviations for traits X and Y, respectively. The realized environmental correlation ($r_{e_{XY}}$) may be estimated from the estimates of r_p , r_g , h_X^2 and h_Y^2 mentioned above as (Dickerson, 1969)

$$r_{e_{XY}} = (r_p - r_g h_X h_Y) / e_X e_Y \quad (2.45)$$

where $e_X = (1 - h_X^2)^{1/2}$ and $e_Y = (1 - h_Y^2)^{1/2}$.

Hill (1971) derived formulae for the approximate sampling variance of an estimated realized genetic correlation. His assumptions were that the correlation had a small coefficient of variation, that the experiment was of long-term, and that heritabilities were estimated from the same experiment. He stated that reliable estimates of the sampling variance can be obtained from the variance between replicates in well replicated experiments.

2.6 SELECTION EXPERIMENTS IN TRIBOLIUM CASTANEUM

2.6.1 Introduction.

Selection studies using laboratory animals, such as Tribolium castaneum, are used as checks on the validity of genetic models or theory. They are also used to provide information on the nature of phenomena such as

genetic asymmetry, plateaus in response to selection and segregation distortion, which in many instances are not predicted by theory (Bell, 1969). Hence, experiments often provided the impetus for further theoretical development.

Tribolium castaneum was first used as a laboratory animal in ecological studies. A review of work in this area was made by King and Dawson (1972). These studies suggested the potential usefulness of Tribolium as an animal model for quantitative genetic research. Early studies in genetics using Tribolium started in 1954 at Purdue University (Bell, 1982).

2.6.2 Biology and genetics of Tribolium castaneum.

The biology of this organism has been discussed in detail by Sokoloff (1972). Yamada (1974) summarized basic aspects of the biology of Tribolium and its advantages and disadvantages as an animal model in quantitative genetics. Only the most important points from these reviews will be presented here.

Four stages have been identified in the life cycle of Tribolium castaneum: egg, larva, pupa and adult stages. Each stage can be easily identified for study. Larvae hatch at an average 3.5 days after oviposition, and pupation takes place 17-18 days after hatching. Adults emerge at about 5 days after pupation. Females start egg production 1 week after emergence as an adult, and peak egg production occurs at about 10-15 days after emergence. The number of eggs a female lays per day averages about 15. Sexes are easily distinguished at both the pupa and adult stages by examination of the genital lobes. The sex ratio is 1:1.

Advantages of this organism as an animal model in quantitative genetic study include the following: 1) it has 10 pairs of chromosomes including the sex chromosomes, recombination takes place in both sexes and segregation

ratios are normal; 2) numerous mutants and genetic markers are available for use in studies of population genetics; 3) it is easy to rear on diets containing 95% wheat flour and 5% dry yeast; 4) it can be reared at a wide range of humidity and temperature. Temperature and relative humidity generally used are 32°C and 70%, respectively. This wide range of easily controlled environmental conditions facilitates studies of genotype X environment interactions; and 5) parents can be stored at very low cost over several generations for subsequent repeat mating.

Tribolium has two main disadvantages: 1) chromosomes are small, thus making cytological studies difficult, and 2) cannibalism by larvae and adults on eggs and pupae is marked under high population density.

Yamada (1974) pointed out that in Tribolium the composition of genetic variance for growth in terms of additive and non-additive contributions varies with age and culturing environment. In addition, maternal and environmental effects on growth vary with age, being larger at the juvenile than at the adult stage. Therefore, the picture on the genetics of growth characteristics in Tribolium (e.g., larval weight, pupal weight and pupation time) is similar to that encountered in *Drosophila*, mice and larger animals.

2.6.3 Selection experiments.

Many selection experiments using Tribolium castaneum have been carried out to test the validity of the theory of quantitative genetics in areas such as: 1) properties of control populations; 2) selection methods; 3) genotype X environment interaction; 4) selection indexes and 5) selection limits.

Bell (1969) summarized much of the work on some of the above areas done in his laboratory and elsewhere. Other reviews include the work of King and Dawson (1972), of Yamada (1974), covering growth, and an update made by Bell

(1982) with emphasis on experiments of Enfield (1977) and Bell (1981) on long term selection response. These reviews indicate, in general, that most of the studies seem to support theory. The present review will discuss some of the important results in the five areas of quantitative genetics listed above.

2.6.3.1 Control populations.

One early problem in quantitative genetics investigated using Tribolium castaneum was the design of an optimum control population. Ideally, genetic changes in a control population should be identical to those in a selected one, except for changes directly related to the selection treatment. In this case, the difference between selected and control populations permits estimation, without bias, of the effect of selection.

Bray et al. (1962) compared 15 methods of maintaining control populations over 8 generations of replicated selection for pupal weight. These methods included using inbreds, crosses between inbreds, random bred individuals with and without restrictions in matings, relaxed selection and repeated matings. They observed significant genotype X environment interactions and concluded that a control line should be closely related to the selected one both in origin and time, so that their reaction to environmental shifts would be similar in nature. If this were not the case, genotype by environment interaction might lead to biased comparisons. Bray (1966) analyzed the effects of restricting the choice of mates to reduce inbreeding, and the effects of unequal contribution of families on phenotypic variability of control population means. Restrictions and equal contribution of families were found to be effective in reducing the variance between

control lines, particularly in small populations of less than 50 breeders of each sex.

2.6.3.2 Genotype X environment interactions.

Assessment of genotype X environment interactions (GE) is needed to understand the nature of genetic changes and to formulate effective breeding plans (Bell, 1969). The finding by Bray et al. (1962) of GE during selection stimulated further studies in this area using Tribolium castaneum. These include the work of Bell and McNary (1963), Costantino et al. (1966, 1967, 1968), Hardin and Bell (1967) and Yamada and Bell (1969).

Bell and McNary (1963) selected for increased pupal weight in each of two environments ("WET" and "DRY"). Selection for pupal weight in the "DRY" environment gave a correlated response in the "WET" environment equal to the direct response to selection in "WET". However, the reciprocal was not true. They observed that the magnitude of the "effective" genetic correlation between growth in the "WET" and in the "DRY" environments was a function of the environment in which selection took place. This was not evident when the "effective" genetic correlation was calculated as

$$r = \left(\frac{C_W \cdot C_D}{R_W \cdot R_D} \right)^{1/2} \quad (2.46)$$

where R_W and R_D are direct responses to selection in the "WET" and "DRY" environments, respectively, and C_W and C_D are the correlated responses. They concluded that the "effective" genetic correlation between two traits in a population is a function of the trait selected and of the environment. This illustrates a shortcoming of current selection theory and pitfalls in its utilization for the prediction of correlated responses.

The work of Costantino et al. (1966) suggested that the effect of a gene on a metric trait can vary from small to large depending on the environment. In this study, 13-day larval weight of a base population raised in a "GOOD" diet was about twice that on a "POOR" diet (2303 ± 25 vs 1215 ± 37 μg). After 12 generations of selection for small 13-day larval weight in the "GOOD" diet, average larval weight was much greater in the "POOR" than in the "GOOD" diet (906 ± 36 vs 305 ± 15 μg). The reason for this was that corn oil in the "GOOD" diet retarded larval growth. The genetic basis of this effect was found to be an autosomal recessive gene, *cos*, corn-oil sensitive. Further analysis indicated that selection for small size in the presence of corn oil had fixed the mutant gene, *cos*, which blocks the oxidation of unsaturated fatty acids. Later work by Costantino et al. (1967, 1968) revealed that the effects of this gene on 13-day larval weight varied from small to large depending on the amount of unsaturated fatty acids in the diet. Thus, the same gene was "minor" in one environment and "major" in another.

Hardin and Bell (1967) and Yamada and Bell (1969) studied response to selection for 13-day larval weight in "GOOD" and "POOR" environments. They observed that direct selection responses in the two environments were generally larger than correlated ones. However, contrary to theoretical expectations, populations selected for average performance in both environments were not superior. They argued that selection for a compound trait such as 13-day larval weight might affect its components differently. In the case of 13-day larval weight, selection in the "GOOD" environment would place primary emphasis on ultimate body size; selection in the "POOR" environment would emphasize both developmental time and ultimate body size. As the component characters become differentiated by the direction and the

environment of selection, changes in a compound trait may reflect parametric changes and enhanced genotype X environment interaction. They concluded that such changes hamper the precision of current selection theory for predicting response to selection even in the short run.

2.6.3.3 Selection methods.

Alternatives to purebred selection, e.g., inbreeding and hybridization, recurrent selection to a tester line for specific combining ability, and reciprocal recurrent selection, are attractive because, in theory, these methods can exploit both additive and non additive genetic variation. Response to purebred selection, on the other hand, is dependent purely on the additive genetic variance. Many workers (e.g., Bell and Moore, 1958; Wong and Boylan, 1970; Bell, 1972; Orozco and Bell, 1974; and McNew and Bell, 1976) have investigated the relative efficiency of these methods using Tribolium castaneum. Their work was reviewed by Bell (1982) so that only the most important findings will be discussed here.

Bell and Moore (1972) compared reciprocal recurrent selection (RRS), and within-line selection (WLS) for pupal weight, a trait with an estimated heritability of 62%. As expected, because of the high heritability of the trait, WLS was superior to RRS. McNew and Bell (1974) investigated the same problem using larval weight, which was found to be moderately heritable ($h^2 = 0.21$); RRS was less effective than WLS. However, RRS crossbreds performed as well or better than WLS crossbreds. This suggested that RRS and WLS were exploiting different kinds of gene effects. Purebred and crossbred covariances indicated that RRS might have utilized some kind of non-additive genetic variance which was not used by purebred selection. This was in agreement with the theory of RRS developed by Comstock et al. (1949).

McNew and Bell (1976) compared selection for 13-day larval weight on the basis of crossbred performance (RRS) with within-line selection based on purebred performance (WLS). In this study the base population had been subjected to 30 generations of purebred selection. After 11 generations of selection, RRS purebreds showed a much smaller response than WLS purebreds; RRS crossbreds responded slightly less than crossbreds in WLS. To explain these results, the authors argued that in highly selected populations dominant and overdominant loci are more important determinants of response to selection than elsewhere. Thus, with greater fitness of heterozygotes, fixation of the dominant allele would be opposed by natural selection to the same extent in WLS and RRS. However, natural selection would have a large effect in retarding fixation at overdominant loci in RRS while there would be no similar effect in WLS. Therefore the use of RRS would not produce the required response in this case.

2.6.3.4 Index selection.

After Kempthorne and Nordskog (1959) published the first paper on restricted indexes, there has been a growing interest, both theoretical and practical, on their use. Scheinberg et al. (1967) examined its biological validity using 13-day larval and pupal weights and pupation time. Three indexes were calculated to obtain maximum gain in one of the traits while holding the other two at a constant level; the experiment was replicated 3 times. After 10 generations of selection, the indexes failed to attain their objectives; the attempted restriction for larval weight and pupal weight resulted in negative response in all cases. Possible causes of the results were non-additive components in the assumed model; biased estimates of parameters or possible changes in genetic and environmental parameters not

accounted for in the indexes. Okada and Hardin (1967) examined the same problem using 14-day larval weight and 30-day adult weight. They reported that after 7 generations of selection, the results were in general agreement with expectations, although there was considerable variation among replicates within selection methods. The response of 14-day larval weight after 13 generations of selection (Okada and Hardin, 1970) was as large in the index population as in the population directly selected for 14-day larval weight. However, 30-day adult weight of the index lines was considerably less than that in the lines under direct selection. Results of Bell and Burris (1973); Fairfull et al. (1977), Berger (1977) and Orozco et al. (1980) were reviewed by Bell (1982). He stated that in most cases, the selection indexes were not the most efficient, contrary to the expectation. The main reason for the discrepancy in almost all cases was primarily errors in parameter estimation, as demonstrated by Lin et al. (1979). This author selected for larval and pupal weights using indexes constructed with known parameter errors.

2.6.3.5 Selection limits.

Short term responses can be predicted from knowledge of heritabilities and selection differentials. However, prediction of long term response depends on usually unknown factors such as population size, number of segregating loci, gene interactions and gene frequencies. Empirical evidence on the nature of selection limits has helped to understand this phenomenon (Bell, 1982).

Bell (1982) reported that most workers have observed fairly linear responses over the first 20 generations or so of selection for body size of Tribolium. However, experiments spanning over 20 generations have reached plateaus in response. This seems to be related to the relationship between

body size and reproductive fitness which changed from positive to negative in the course of selection. Bell (1981) concluded that aspects of long term responses such as response plateaus, duration of plateaus and ultimate limits to response are not predictable from theory. He also pointed out that mutagenic substances are not useful for circumventing plateaus. Also, he indicated that polygenic mutation rates are probably higher than those reported from studies with genetic markers. This could account for a large part of the genetic variation present in populations which have reached a plateau. As pointed out, the genetic correlation between a metric trait and reproductive fitness can change from positive to negative within a few generations of selection, with linkage being perhaps more important than pleiotropy.

Enfield (1977) observed continued response to selection for 21-day pupal weight for about 75 generations. This was at variance with other experiments reported prior to this where the common pattern was to reach plateaus in 30 generations or less. He attributed this to the large effective population size employed in his study. There was no additional response in the next 30 generations even though genetic variability was present ($h^2 = 0.20$). Enfield (1977) argued that the plateaus were caused by a strong negative correlation between pupal weight and fitness, possibly due to both linkage and pleiotropy.

2.6.4 Genetic parameters of larval and pupal weights and of pupation time.

Bell (1969), King and Dawson (1972) and Yamada (1974) tabulated published work on selection for quantitative traits of Tribolium and the reader is referred to these papers for specific parameter estimates. The general picture, following a broad classification made by the authors with reference

to heritability, degree of heterosis and genetic correlations is presented here. Larval weight has medium heritability and exhibits a moderate degree of heterosis. Pupal weight has been found to be highly heritable and slightly heterotic. Pupation time has low to medium heritability and a moderate degree of heterosis. The genetic correlation between larval weight and pupal weight is positive and high, and that between larval weight and pupation time is negative. The genetic correlation between pupal weight and pupation time is sometimes negative and sometimes positive (Englert and Bell, 1969). There seems to be very little difference between sexes in parameter estimates (Scheinberg, 1969; Enfield, 1977) and only larval weight seems to be affected by maternal influences (Bondari et al., 1978). However, some workers have not found significant maternal influences on larval weight (e.g., Bartlett, et al. 1966).

CHAPTER THREE

MATERIALS AND METHODS

3.1 STOCK AND LABORATORY CONDITIONS.

An outbred population of Tribolium castaneum, Purdue "f" Foundation, was used in this study. The origin and history of this population is given by Bell and Moore (1972) and its biology has been discussed in a previous section of this thesis.

A sample of approximately 1600 individuals was obtained from the Population Genetics Institute, Purdue University, in 1982. The sample was introduced to the Tribolium Laboratory of the Department of Dairy Science, University of Illinois, and maintained in an incubator at $32.1 \pm 0.7^{\circ}\text{C}$ and $69.9 \pm 0.5\%$ of relative humidity. A medium containing 95% of all purpose wheat flour and 5% of dried brewers yeast was used to maintain the beetles.

3.2 VARIABLES RECORDED.

Larval and pupal weights, and pupation time were recorded. Larval weight (LW), in micrograms, was taken 13 days after egg collection. Pupal weight (PW), also in micrograms, was taken within 24 hours of pupation; pupation time (PT) was defined as the number of days between egg collection and pupation.

3.3 TREATMENT DESIGN.

A 2x2x3 factorial treatment design was used including the following factors and levels:

A) pre-selection mating system, with levels: 1) random mating (R), and 2) assortative mating for pupal weight (A);

B) selection mating system, with levels: 1) random mating, and 2) assortative mating for pupal weight; and

C) percent truncation selected for pupal weight, with levels: 1) 20%, 2) 50%, and 3) 0% (control). Each treatment was replicated 5 times and the replications were spaced at weekly intervals to ease the work load.

The pre-selection phase of the experiment lasted 3 generations, at which time the assortatively mated population was expected to be close to reaching equilibrium (Crow and Kimura, 1970; Bulmer, 1980). These generations will be referred to in this study as -3, -2 and -1. Fifty males were mated to 100 females (1 male paired with 2 females) per generation in each group (R vs. A) of the pre-selection phase. The expected rate of increase of inbreeding under random mating is then .2% per generation (Falconer, 1981). The expected rate of increase of inbreeding under assortative mating should have been close to that under random mating. This is because Crow and Felsenstein (1968) have shown that when a trait is affected by a large number of genes, which is probably the case for the traits considered in this study (Sokoloff, 1972; Bell, 1982), the decrease in heterozygosity, as measured by the inbreeding coefficient, is very slight.

The selection phase of the experiment lasted 2 generations. Ten mating pairs (1 male: 1 female) were used to propagate each of the 12 lines (there were 12 mating system-selection intensity combinations) in each replicate. The expected rate of increase of inbreeding in the randomly mated lines is 2.5% per generation (Falconer, 1981). With random mating of parents the expected variance of realized heritability estimates obtained in individual

selection lines, per replicate, at generation 2 is 0.01 and 0.04 for 20 and 50 percent selected, respectively (Hill, 1980).

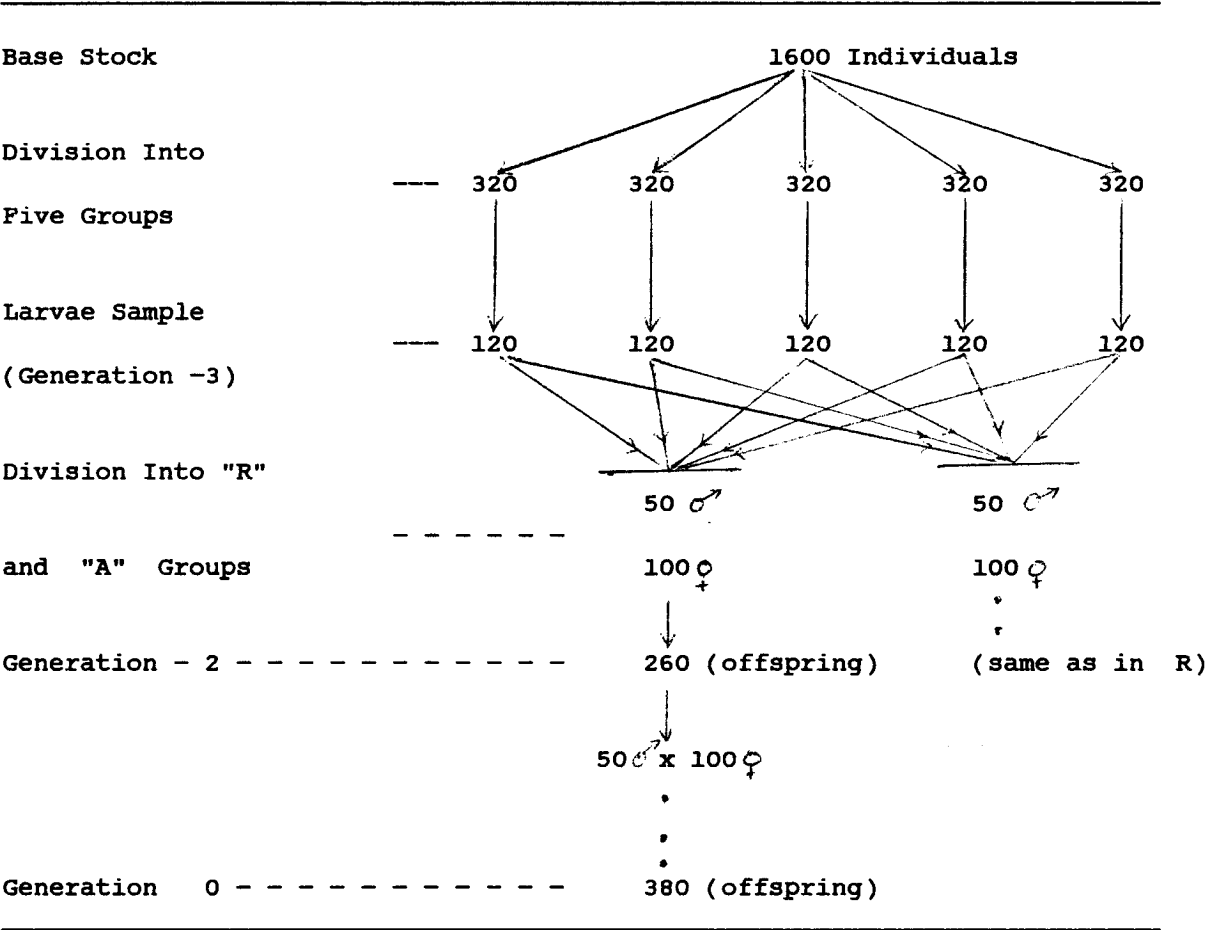
3.4 EXPERIMENTAL PROCEDURES

3.4.1 Pre-selection phase.

Procedures followed in this phase are illustrated in Figure 1. The stock obtained from Purdue was not identified by sex so it was not possible immediately to mate randomly individuals to produce generation -3. Therefore, upon introduction, the stock was divided randomly into 5 groups, each with approximately 320 individuals. The groups were maintained in half-pint bottles for 7 days. On the 8th day, individuals in each group were transferred to fresh medium for a 1-day egg collection. After collection, adults were kept for another 7-day mating period. A random sample of 120 larvae was taken from each group on the 13th day after egg collection. These larvae were then weighed and placed in individual vials. Pupal weight and pupation time were recorded as each individual pupated. Then 40 adult females and 20 adult males were chosen randomly from each group of 120 individuals.

Pre-selection random mating (R) and assortative mating (A) groups were formed by randomly dividing these 20 males and 40 females in each of the five groups into R and A groups. Then, R and A groups each had 50 males and 100 females which were designated as in generation -3. Individuals in R were mated at random (2 females per male) while those in A were mated assortatively (2 females per male) on the basis of pupal weight. Females were identified by clipping the left or the right antenna. Random or assortative matings were also made within R and A groups,

Figure 1: Experimental Plan For The Pre-Selection Phase*



respectively, in generations -2 and -1. Hence, generation 0 was the result of 3 generations of either random or assortative mating of parents. No selection of parents was practiced during generations -3 through -1. Full-sib and half-sib matings were avoided.

The above procedures were followed in each of the 5 replicates of the experiment. Hence, each of the R and A group consisted of 250 males and 500 females mated per generation.

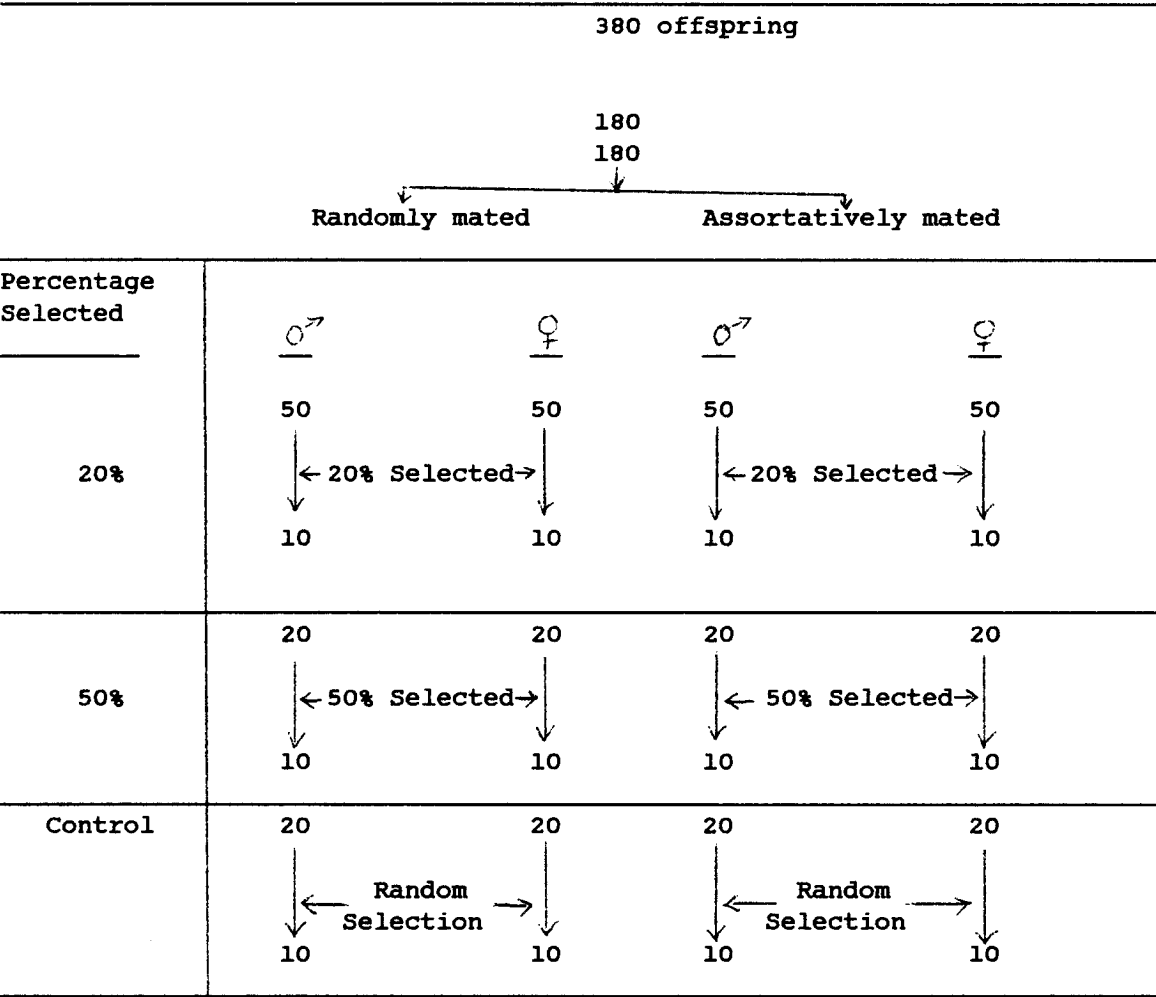
3.4.2 Selection phase.

In each of the R and A groups within each replicate, 6 lines were formed to represent all possible combinations of the 2 levels of selection mating system (R,A) with the 3 levels of percent truncation selected (20%; 50%; and 0% (control: CO)). Hence, there was a total of 12 lines in each replicate.

Lines derived from the group which was randomly mated during the pre-selection phase were designated as: RR20, RR50, RRCO, RA20, RA50 and RACO. Those derived from the group which was assortatively mated in the pre-selection phase were: AR20, AR50, ARCO, AA20, AA50 and AACO. In a given line, the first and second characters indicate the pre-selection and selection mating systems, respectively, and the last two characters indicate the percent truncation selected.

At generation 0, 180 males and 180 females within each of the R and A groups were randomly assigned, within families, to the 6 lines as illustrated in figure 2. In the control line, 10 males and 10 females were randomly selected to be parents of generation 1. In the case of 20% and 50% truncation selected lines, males and females were ranked separately (on the basis of individual pupal weight) and the top 10 individuals in

Figure 2: Experimental plan at generation 0 for the R group. (The same was done for the A group).



each sex were selected as parents, and mated following the appropriate selection mating system. Three spare matings were kept in each individual line. The experimental plan for individual lines during the selection phase is illustrated in figure 3.

3.4.3 Laboratory procedures.

Procedures were adapted from those of Scheinberg (1969) and followed throughout the experiment; details are presented in table 4.

3.5 ESTIMATION OF GENETIC PARAMETERS IN THE RANDOMLY MATED AND ASSORTATIVELY MATED BASE POPULATIONS

3.5.1 Heritability and genetic correlations under random mating.

Parent-offspring and sib covariances were calculated using analysis of variance (covariance) and regression techniques to estimate heritabilities of larval and pupal weights, and of pupation time, and genetic correlations between these traits. Data from generations -3, -2, -1 and 0 were used for this purpose.

3.5.1.1 Analysis of variance approach

A first analysis was carried out separately for each sex and for each replicate. The model used was (Model 1)

$$y_{ijkl} = \mu + t_i + s_{ij} + d_{ijk} + w_{ijkl} \quad (3.1)$$

where y_{ijkl} : an observation on the l th progeny from the k th dam mated to the j th sire in the i th generation;

Figure 3: Experimental plan for individual lines during the selection phase (illustrated with the RR20, RR50 and RRC0 lines).

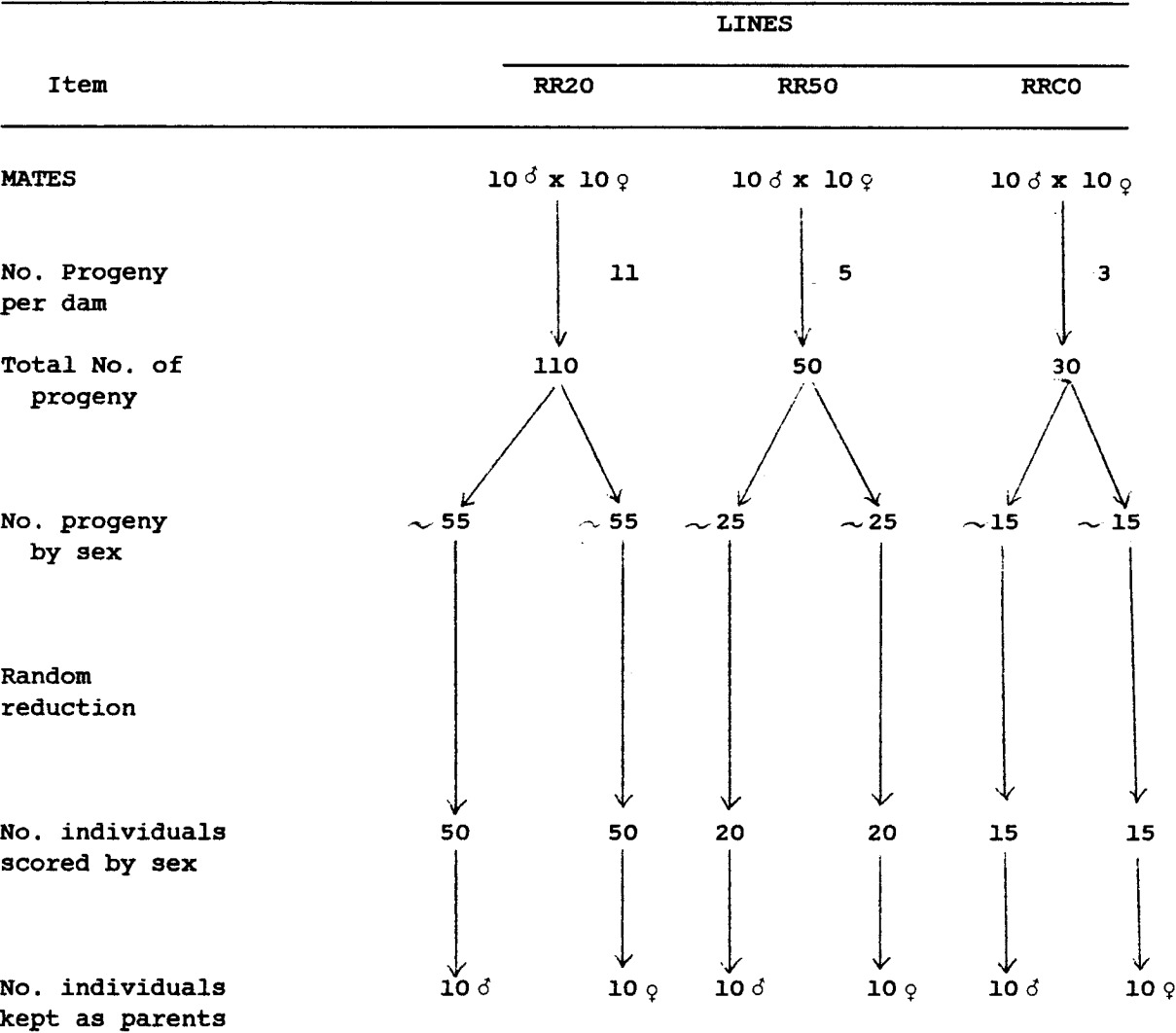


Table 4: Outline of laboratory procedures.

<u>Day of cycle</u>	<u>Procedure</u>
0	Three-day matings finish. Each female placed in 2g of fresh standard medium for 1-day egg collection.
1	Females removed from medium and discarded. Eggs returned to incubator.
13	Thirteen-day larvae sampled at random from each dam and weighed individually. Larvae placed in individual vials with 0.1g of standard medium.
14	Recording pupal weight and pupation time starts. Sex determined for each pupa, and 0.4g of standard medium added to the vial.
25	Final day of recording. All larvae not pupating at this time discarded.
32	Surviving adults checked for viability.
33	Parents of the next generation selected and mated.
35	Cycle finishes.

μ : a fixed effect common to all individuals in
the same replicate and sex;

t_i : the fixed effect of the i th generation
($i = 1, 2, 3$);

s_{ij} : the random effect of the j th sire in the i th
generation ($j = 1, \dots, s_i$);

d_{ijk} : the random effect of the k th dam mated to the
 j th sire in the i th generation ($k = 1, \dots, d_{ij}$);
and

w_{ijkl} : a random residual peculiar to the $ijkl$ th
individual ($l = 1, \dots, n_{ijk}$).

The following assumptions were made for this model: $E(y_{ijkl}) = \mu + t_i$,
 $E(s_{ij}) = E(d_{ijk}) = E(w_{ijkl}) = 0$, $\text{Var}(s_{ij}) = \sigma_s^2$, $\text{Var}(d_{ijk}) = \sigma_d^2$ and
 $\text{Var}(w_{ijkl}) = \sigma_w^2$. Further, sire, dam and residual effects were assumed to
be mutually uncorrelated so that

$$\text{Var}(y_{ijkl}) = \sigma_s^2 + \sigma_d^2 + \sigma_w^2 = \sigma_p^2 \quad (3.2)$$

Variance and covariance components were estimated by equating mean
squares and products to their expectations. Computations were carried out
using the NESTED procedure of SAS (SAS Institute, 1982). Table 5 contains a
summary of the nested analysis of variance (covariance) carried out
separately for each of the 10 sex-replicate combinations.

Table 5: Form of the nested analysis of variance^a.

Source	degrees of freedom	EMS ^b
Generation	$t - 1$	NOT NEEDED
Sires/generations	$\sum_i (s_i - 1)$	$\sigma_w^2 + k_2 \sigma_d^2 + k_3 \sigma_s^2$
Dams/sires/generations	$\sum_i \sum_j (d_{ij} - 1)$	$\sigma_w^2 + k_1 \sigma_d^2$
Residual	$\sum_i \sum_j \sum_k (n_{ijk} - 1)$	σ_w^2

a In the analysis of covariance, variance components are replaced by the appropriate covariance components.

b EMS: expected mean square. In the analysis of covariance, the EMS is replaced by the expected mean product. k_1 , k_2 and k_3 are coefficients of variance (covariance) components.

Variance and covariance components were estimated by equating mean squares and products to their expectations. Computations were carried out using the NESTED procedure of SAS (SAS Institute, 1982). Table 5 contains a summary of the nested analysis of variance (covariance) carried out separately for each of the 10 sex-replicate combinations.

Heritability estimates based on sire ($\hat{h}_s^2$), dam ($\hat{h}_d^2$) and sire plus dam components ($\hat{h}_{s+d}^2$) were calculated as

$$\hat{h}_s^2 = \frac{4\hat{\sigma}_s^2}{\hat{\sigma}_p^2}, \quad (3.3a)$$

$$\hat{h}_d^2 = \frac{4\hat{\sigma}_d^2}{\hat{\sigma}_p^2}, \quad (3.3b)$$

and

$$\hat{h}_{s+d}^2 = \frac{2(\hat{\sigma}_s^2 + \hat{\sigma}_d^2)}{\hat{\sigma}_p^2}, \quad (3.3c)$$

respectively, where $\hat{\sigma}_p^2$ is the estimated total phenotypic variance ($\hat{\sigma}_p^2 = \hat{\sigma}_s^2 + \hat{\sigma}_d^2 + \hat{\sigma}_w^2$). Standard errors of heritability estimates were obtained by using the approximations outlined by Funkhouser and Grossman (1982), and also from the variance between estimates in different replicates.

Genetic correlations from sire ($\hat{r}_{g_s}$), dam ($\hat{r}_{g_d}$) and sire plus dam ($\hat{r}_{g_{s+d}}$) components of covariance were calculated as

$$\hat{r}_{g_s} = \frac{\hat{\sigma}_{s_{ab}}}{\sqrt{\hat{\sigma}_{s_a}^2 \cdot \hat{\sigma}_{s_b}^2}}, \quad (3.4a)$$

$$\hat{r}_{g_d} = \frac{\hat{\sigma}_{d_{ab}}}{\sqrt{\hat{\sigma}_{d_a}^2 \cdot \hat{\sigma}_{d_b}^2}}, \quad (3.4b)$$

and

$$\hat{r}_{g_{s+d}} = \frac{\hat{\sigma}_{s_{ab}} + \hat{\sigma}_{d_{ab}}}{\sqrt{(\hat{\sigma}_{s_a}^2 + \hat{\sigma}_{d_a}^2)(\hat{\sigma}_{s_b}^2 + \hat{\sigma}_{d_b}^2)}}, \quad (3.4c)$$

respectively, where terms in (3.4a)-(3.4c) are appropriate components of variance and covariance for traits a and b. Standard errors of the estimates of genetic correlations were approximated using the formulae of Grossman (1970), and were also calculated from the variance between replicates. Estimates of phenotypic ($\hat{r}_{p_{ab}}$) correlations were

$$\hat{r}_{p_{ab}} = \frac{\hat{\sigma}_{s_{ab}} + \hat{\sigma}_{d_{ab}} + \hat{\sigma}_{w_{ab}}}{\sqrt{\hat{\sigma}_{p_a}^2 \cdot \hat{\sigma}_{p_b}^2}}, \quad (3.5)$$

Standard errors of phenotypic correlations were calculated from the variance between replicates.

Estimates of heritabilities, and of genetic, and phenotypic correlations were averaged, separately for each sex and unweighted across replicates, and the standard errors of these averages were calculated from the variance between estimates in individual replicates. Also, single estimates of the above parameters were obtained in a second analysis using a model similar to (3.1) (Model 2) but including generation-replicate subclasses, sires nested within such subclasses, dams nested within sires and progeny nested within dams. Standard errors of these estimates were calculated using formulae of Funkhouser and Grossman (1982) for heritabilities and of Grossman (1970) for the genetic correlations.

3.5.1.2 Regression approach.

Regressions of sons on sires, daughters on sires, sons or daughters on dams (intra-sire); individual offspring on sires or on dams (intra-sire); sons or daughters on mid-parents, and individual offspring on mid-parent were calculated for each pair of generations (-3 and -2, -2 and -1, -1 and 0). Sums of squares and of products were then pooled across pairs of generations; this was done separately for each replicate.

Heritabilities were calculated by doubling individual offspring-individual parent regressions, and directly from the offspring mid-parent regressions. Mean unweighted heritability estimates were calculated, and the standard errors of these means were obtained from the variance between estimates in the 5 replicates.

3.5.2 Heritabilities and genetic correlations under assortative mating.

With assortative mating, the assumptions of the nested model described above do not hold because sire and dam effects are correlated. Therefore, methods described in section 2.4.2 and variations thereof were applied to the data from generations -1 and 0, where the population mated assortatively in the pre-selection phase should have been close to equilibrium.

3.5.2.1 Non-linear approach: raw correlations.

Offspring-parent correlations were calculated as described in the previous section with data from generations -1 and 0. Correlations between full-sibs and half-sibs in generation 0 were calculated, using product-moment techniques. Within each replicate, unweighted sums of squares and of products were computed by pairing individuals within a family, such that specific pairs appeared twice, once with one member in the

left column and again with this member in the right column. To illustrate, consider 2 half-sib families in a given replicate of generation 0:

Sire 1		Sire 2	
<hr/>		<hr/>	
<u>Dam 1</u>	<u>Dam 2</u>	<u>Dam 3</u>	<u>Dam 4</u>
$X_{11} \ Y_{11}$	$X_{21} \ Y_{21}$	$X_{31} \ Y_{31}$	$X_{41} \ Y_{41}$
$X_{12} \ Y_{12}$	$X_{22} \ Y_{22}$	$X_{32} \ Y_{32}$	$X_{42} \ Y_{42}$

The variables X_{ij} and Y_{ij} denote measurements in the j th member of the i th full-sib family. In order to calculate the covariances between full-sibs for traits X or Y, the following pairs would be formed:

Trait X		Trait Y	
<hr/>		<hr/>	
<u>column 1</u>	<u>column 2</u>	<u>column 1</u>	<u>column 2</u>
X_{11}	X_{12}	Y_{11}	Y_{12}
X_{12}	X_{11}	Y_{12}	Y_{11}
X_{21}	X_{22}	Y_{21}	Y_{22}
X_{22}	X_{21}	Y_{22}	Y_{21}
X_{31}	X_{32}	Y_{31}	Y_{32}
X_{32}	X_{31}	Y_{32}	Y_{31}
X_{41}	X_{42}	Y_{41}	Y_{42}
X_{42}	X_{41}	Y_{42}	Y_{41}

The covariance between X, Y values of full-sibs would be calculated from the following pairs:

<u>column 1</u>	<u>column 2</u>
X_{11}	Y_{12}
X_{12}	Y_{11}
X_{21}	Y_{22}
X_{22}	Y_{21}
X_{31}	Y_{32}
X_{32}	Y_{31}
X_{41}	Y_{42}
X_{42}	Y_{41}

The covariance between X values of paternal half-sibs would be calculated from the pairs:

Trait X	
<u>column 1</u>	<u>column 2</u>
X_{11}	X_{21}
X_{21}	X_{11}
X_{11}	X_{22}
X_{22}	X_{11}
X_{12}	X_{21}
X_{21}	X_{12}
X_{12}	X_{22}
X_{22}	X_{12}

column 1 (cont.) column 2 (cont.)

X_{31}	X_{41}
X_{41}	X_{31}
X_{31}	X_{42}
X_{42}	X_{31}
X_{32}	X_{41}
X_{41}	X_{32}
X_{32}	X_{42}
X_{42}	X_{32}

A similar pairing would be done with Y values and with (X,Y) values involving paternal half-sibs

Consider now the 10 covariances between relatives presented in Table 3. Expressing these as correlations involving three traits (larval weight, pupal weight and pupation time), and including dominance ($d^2/4$) and maternal contributions (m^2) to the resemblance between full-sibs, the results of Table 6 are obtained. With known ρ , which can be calculated in each replicate as the correlation between pupal weights of the mates, the 20×1 parameter vector is

$$\begin{aligned} \tilde{\Psi}' = [& h_X, h_Y, h_Z, \theta_{XY}, \theta_{XZ}, \theta_{YZ}, d_X, d_Y, d_Z, r_{d_{XY}}, r_{d_{XZ}}, r_{d_{YZ}}, \\ & m_X, m_Y, m_Z, r_{m_{XY}}, r_{m_{XZ}}, r_{m_{YZ}}, r_{XY}, r_{XZ}] \end{aligned} \quad (3.6)$$

where X, Y and Z stand for pupal weight, larval weight and pupation time respectively. Let $\tilde{y}_i$ be the 20×1 vector of calculated correlations between relatives in the i th replicate ($i = 1, \dots, 5$). Then

Table 6: Expected value of correlations between different types of relatives for traits X, Y and Z under assortative mating for X (after Gianola, 1982).

Correlation		Expected values*
A. Offspring-parent		
1. (X,X)	$h_X^2(1 + \rho)/2$	
2. (X,Y)	$(\theta_{XY}h_Xh_Y + h_X^2\rho r_{XY})/2$	
3. (Y,X)	$\theta_{XY}h_Xh_Y(1 + \rho)/2$	
4. (Y,Y)	$h_Y^2(1 + \theta_{XY}^2\rho h_X^2)/2$	
5. (X,Z)	$(\theta_{XZ}h_Xh_Z + h_X^2\rho r_{XZ})/2$	
6. (Z,X)	$\theta_{XZ}h_Xh_Z(1 + \rho)/2$	
7. (Z,Z)	$h_Z^2(1 + \theta_{XZ}^2\rho h_X^2)/2$	
8. (Z,Y)	$\theta_{ZY}h_Zh_Y(1 + \theta_{XY}\theta_{XZ}\rho h_X^2)/2$	
B. Full-sibs		
9. (X,X)	$h_X^2(1 + \rho h_X^2)/2 + d_X^2/4 + m_X^2$	
10. (X,Y)	$\theta_{XY}h_Xh_Y(1 + \rho h_X^2)/2 + r_{d_{XY}}d_Xd_Y/4 + r_{m_{XY}}m_Xm_Y$	
11. (Y,Y)	$h_Y^2(1 + \theta_{XY}^2\rho h_X^2)/2 + d_Y^2/4 + m_Y^2$	
12. (X,Z)	$\theta_{XZ}h_Xh_Z(1 + \rho h_X^2)/2 + r_{d_{XZ}}d_Xd_Z/4 + r_{m_{XZ}}m_Xm_Z$	
13. (Z,Z)	$h_Z^2(1 + \theta_{XZ}^2\rho h_X^2)/2 + d_Z^2/4 + m_Z^2$	
14. (Z,Y)	$\theta_{ZY}h_Zh_Y(1 + \theta_{XY}\theta_{XZ}\rho h_X^2)/2 + r_{d_{ZY}}d_Zd_Y/4 + r_{m_{ZY}}m_Zm_Y$	

Table 6 Cont.

Correlations		Expected Values*
C. Half-sibs		
15. (X,X)	$h_X^2 [1 + h_X^2 \rho(2 + \rho)]/4$	
16. (X,Y)	$\theta_{XY} h_X h_Y [1 + h_X^2 \rho(2 + \rho)]/4$	
17. (Y,Y)	$h_Y^2 [1 + \theta_{XY}^2 h_X^2 \rho(2 + \rho)]/4$	
18. (X,Z)	$\theta_{XZ} h_X h_Z [1 + h_X^2 \rho(2 + \rho)]/4$	
19. (Z,Z)	$h_Z^2 [1 + \theta_{XZ}^2 h_X^2 \rho(2 + \rho)]/4$	
20. (Z,Y)	$\theta_{ZY} h_Z h_Y [1 + \theta_{XY} \theta_{XZ} h_X^2 \rho(2 + \rho)]/4$	

* h_X^2, h_Y^2, h_Z^2 are equilibrium heritabilities; $\theta_{XY}, \theta_{XZ}, \theta_{ZY}$ are equilibrium genetic correlations; r_{XY} and r_{XZ} are equilibrium phenotypic correlations; d_X, d_Y, d_Z are dominance contributions; $r_{d_{XY}}, r_{d_{XZ}}, r_{d_{ZY}}$ are correlations between dominance deviations; m_X, m_Y, m_Z are maternal contributions; $r_{m_{XY}}, r_{m_{XZ}}, r_{m_{ZY}}$ are correlations between maternal deviations; ρ is phenotypic correlation between mates.

$$\begin{bmatrix} y_1 \\ \sim \\ y_2 \\ \sim \\ y_3 \\ \sim \\ y_4 \\ \sim \\ y_5 \\ \sim \end{bmatrix} = \begin{bmatrix} f_1(\psi) \\ \sim \\ f_2(\psi) \\ \sim \\ f_3(\psi) \\ \sim \\ f_4(\psi) \\ \sim \\ f_5(\psi) \\ \sim \end{bmatrix} + \begin{bmatrix} \epsilon_1 \\ \sim \\ \epsilon_2 \\ \sim \\ \epsilon_3 \\ \sim \\ \epsilon_4 \\ \sim \\ \epsilon_5 \\ \sim \end{bmatrix} \quad (3.7)$$

where $f_i(\psi)$ is a vector of non-linear functions of ψ , and ϵ_i is a vector of residuals $\sim(0, \Sigma_i)$. Further, $\text{Cov}(\epsilon_i, \epsilon_j) = 0$. In general, Σ_i is not diagonal; however, we approximated it by V_i , a diagonal matrix which contains estimates of the diagonal elements of Σ_i . This was done because of difficulties in deriving the off-diagonal estimates of Σ_i , i.e., covariances between different bilinear forms calculated from the same data.

The parameter vector ψ was estimated by minimizing

$$\chi^2 = [y - f(\psi)]' V^{-1} [y - f(\psi)] \quad (3.8)$$

with respect to ψ , where $y' = [y'_1, \dots, y'_5]$, $f'(\psi) = [f'_1(\psi), \dots, f'_5(\psi)]$ and

$$V^{-1} = \begin{bmatrix} V_1^{-1} & 0 & \dots & 0 \\ \sim & \sim & & \sim \\ 0 & V_2^{-1} & \dots & 0 \\ \sim & \sim & & \sim \\ \cdot & \cdot & & \cdot \\ \cdot & \cdot & & \cdot \\ 0 & 0 & \dots & V_5^{-1} \\ \sim & \sim & & \sim \end{bmatrix} \quad (3.9)$$

Now, $\underset{\sim}{f}(\underset{\sim}{\psi})$ is 100×1 , $\underset{\sim}{\psi}$ is 20×1 , and

$$\begin{aligned} \frac{\partial \chi^2}{\partial \underset{\sim}{\psi}} &= \frac{\partial \underset{\sim}{f}(\underset{\sim}{\psi})}{\partial \underset{\sim}{\psi}} \cdot \frac{\partial \chi^2}{\partial \underset{\sim}{f}(\underset{\sim}{\psi})} \\ &= -\underset{\sim}{K}' \underset{\sim}{V}^{-1} [\underset{\sim}{y} - \underset{\sim}{f}(\underset{\sim}{\psi})] \end{aligned} \quad (3.10)$$

where $\underset{\sim}{K}'$ is a 20×100 matrix containing the first derivatives of $\underset{\sim}{f}(\underset{\sim}{\psi})$ with respect to $\underset{\sim}{\psi}$. For example, the first row of $\underset{\sim}{K}'$ is

$$\left[\frac{\partial f_{1,1}(\underset{\sim}{\psi})}{\partial \psi_1} \cdot \cdot \cdot \cdot \frac{\partial f_{5,20}(\underset{\sim}{\psi})}{\partial \psi_1} \right]$$

where $f_{ij}(\underset{\sim}{\psi})$ is the expected value of the j th correlation in the i th replicate. Now, expand $\underset{\sim}{f}(\underset{\sim}{\psi})$ with a first order Taylor series about $\underset{\sim}{\psi}_0$.

$$\underset{\sim}{f}(\underset{\sim}{\psi}) = \underset{\sim}{f}(\underset{\sim}{\psi}_0) + \underset{\sim}{K}'_0 (\underset{\sim}{\psi} - \underset{\sim}{\psi}_0^0) \quad (3.11)$$

where $\underset{\sim}{K}_0$ is $\underset{\sim}{K}$ evaluated at $\underset{\sim}{\psi}_0^0$. Using (3.11) in (3.10), setting to 0 and rearranging yields a sequence of iterations

$$\underset{\sim}{\psi}^l = \underset{\sim}{\psi}^{l-1} + [\underset{\sim}{K}'_{l-1} \underset{\sim}{V}_{l-1}^{-1} \underset{\sim}{K}'_{l-1}]^{-1} \underset{\sim}{K}'_{l-1} \underset{\sim}{V}_{l-1}^{-1} [\underset{\sim}{y} - \underset{\sim}{f}(\underset{\sim}{\psi}^{l-1})]$$

where $\underset{\sim}{\psi}^l$ is a solution at the l th iterate, and $\underset{\sim}{K}'_{l-1}$ and $\underset{\sim}{V}_{l-1}^{-1}$ are the $\underset{\sim}{K}'$ and $\underset{\sim}{V}^{-1}$ matrices evaluated at the l th iterate. The computations were carried out with the NLIN procedure of SAS (SAS Institute, 1982) using the technique of segmented models.

The matrix $\underset{\sim}{V}_i^{-1}$ was calculated and recalculated at each iterate ($l = 0, 1, \dots$), following ideas of Eaves (1975), as

$$\underset{\sim}{V}_i^{-1} = \text{Diag} \left\{ \frac{n_{ij} - 2}{(1 - f_{ij}^2(\underset{\sim}{\psi}^l))^2} \right\}, \quad (3.12)$$

where $f_{ij}(\psi^l)$ is an estimate of $f_{ij}(\psi)$, the expected value of the j th correlation in the i th replicate ($i = 1, \dots, 5; j = 1, \dots, 20$), and n_{ij} is the number of pairs used in calculating such correlation. In order to ascertain whether convergence to a global minimum had occurred, two sets of trial values were used for iteration

$$a) \psi^0 = 0,$$

and

$$b) \psi^0 = \text{estimates obtained in the randomly mated population.}$$

During iteration, elements of ψ^l were kept within the parameter space

$$0 \leq (h_X, h_Y, h_Z, d_X, d_Y, d_Z, m_X, m_Y, m_Z) \leq 1$$

$$\text{and } -1 \leq (\theta_{XY}, \theta_{XZ}, \theta_{YZ}, r_{d_{XY}}, r_{d_{XZ}}, r_{d_{YZ}}, r_{m_{XY}}, r_{m_{XZ}}, r_{m_{YZ}}, r_{XY}, r_{XZ}) \leq 1.$$

Iteration ended when

$$||\psi^l - \psi^{l-1}||/20 < 10^{-8}.$$

The variance-covariance matrix of the estimates was computed as

$$\text{Var}(\hat{\psi}) = [K' V^{-1} K]^{-1} \quad (3.13)$$

where $\hat{\psi}$ is the minimum chi-square estimate of ψ .

3.5.2.2 Non-linear approach: z-transforms.

The above described procedure was repeated but now using z-transforms of the elements of y in order to obtain a better approximation to normality,

and to render $\tilde{y}$ "independent" of $\tilde{\psi}$. Let y_{ij} be the j th correlation in the i th replicate and take

$$z_{ij} = \frac{1}{2} \ln \left(\frac{1 + y_{ij}}{1 - y_{ij}} \right). \quad (3.14)$$

Then, following Fisher (1921)

$$E(z_{ij}) = \frac{1}{2} \ln \left(\frac{1 + f_{ij}(\tilde{\psi})}{1 - f_{ij}(\tilde{\psi})} \right) = \mu_{ij} \quad (3.15)$$

and

$$\text{Var}(z_{ij}) = 1/n_{ij}.$$

The parameter vector $\tilde{\psi}$ was estimated by minimizing

$$\chi^2 = (z - \mu)' N (z - \mu) \quad (3.16)$$

with respect to $\tilde{\psi}$, where

$$\tilde{z}' = [z_{11}, \dots, z_{5,20}], \quad \tilde{\mu}' = [\mu_{1,1}, \dots, \mu_{5,20}]$$

and $\tilde{N} = \text{diag}\{n_{ij}\}$. Calculations were carried out iterating with

$$\tilde{\psi}^l = \tilde{\psi}^{l-1} + [\tilde{Q}'_{l-1} \tilde{N} \tilde{Q}_{l-1}]^{-1} \tilde{Q}'_{l-1} \tilde{N} [z - \mu^{l-1}] \quad (3.17)$$

where

$$\tilde{Q}'_{20 \times 100} = \frac{\partial \mu_{ij}}{\partial \psi_p}, \quad i = 1, \dots, 5; j = 1, \dots, 20; p = 1, \dots, 20.$$

The computations were also carried out with the NLIN procedure of SAS (SAS Institute, 1982). The variance-covariance matrix of the estimates was calculated as

$$\text{Var}(\hat{\psi}) = [Q'NQ]^{-1}$$

using the value of $\hat{\psi}$ at convergence.

3.6 EVALUATION OF RESPONSE TO SELECTION

Line means for each trait were analyzed separately for each generation using the fixed effects model

$$\begin{aligned} \bar{y}_{ijkl} = & \mu + P_i + S_j + T_k + PS_{ij} \\ & + PT_{ik} + ST_{jk} + PST_{ijk} + e_{ijkl} \end{aligned} \quad (3.18)$$

where $\bar{y}_{ijkl}$ is the mean of the l th replicate ($l = 1, \dots, 5$) of the ijk th treatment combination, μ is an effect common to all lines in the same generation; P_i is the effect of the i th pre-selection mating system ($i = 1, 2$); S_j is the effect of j th selection mating system ($j = 1, 2$); T_k is the effect of the k th selection intensity ($k = 1, \dots, 3$); PS_{ij} , PT_{ik} and ST_{jk} are first-order interactions; PST_{ijk} is a second-order interaction, and e_{ijkl} is a residual assumed to be $N(0, \sigma^2/n_{ijkl})$ where n_{ijkl} is the number of observations in the ijk th mean, and σ^2 is a scalar. Let $\bar{y}$ be a 60×1 vector of means (12 lines in each of 5 replicates) and α be the 36×1 vector of parameters

$$\alpha' = [\mu, P_1, P_2, \dots, PST_{111}, \dots, PST_{223}] \quad (3.19)$$

The model in (3.18) can be written as

$$\tilde{y} = K\alpha + e \quad (3.20)$$

where K is a 60×36 known matrix and e is a 60×1 vector of residuals. The normal equations were solved as (Searle, 1971)

$$\alpha^\circ = (K'NK)^{-1} K'N\tilde{y} \quad (3.21)$$

where α° is a particular solution, N is a 60×60 diagonal matrix with typical element $\{n_{ijkl}\}$, and $(K'NK)^{-1}$ is a generalized inverse of $K'NK$. Testable hypotheses of the form $L'\alpha^\circ = 0$, where L' is an $s \times 36$ matrix of full-row rank, were tested by referring the statistic

$$F = \frac{\{\alpha^\circ' L [L' (K'NK)^{-1} L]^{-1} L' \alpha^\circ\} / s}{(\tilde{y}' N \tilde{y} - \alpha^\circ' K' N \tilde{y}) / 48} \quad (3.22)$$

to a central $F_{(s, 48)}$ distribution (Searle, 1971b).

Direct and correlated responses to selection were also evaluated by computing the mean of a given trait in the selection lines as a deviation from the mean of that trait on the respective controls each generation (0,1,2), and then regressing these deviations linearly on generation number. Since there were 12 lines including 4 controls, 8 such regressions were calculated. Fifteen data points (5 replicates per line in each generation) were used for calculating each regression. The model for individual regressions was $Z_{ij} = \beta_{0i} + \beta_{1i} X_{ij} + w_{ij}$; $i = 1, \dots, 8$, where Z_{ij} is a

deviation from the control in the j th generation, β_{0i} and β_{1i} are regression coefficients, X_{ij} is generation number and w_{ij} is a residual. A symmetric 95% confidence interval was calculated for each β_{1i} as (Searle, 1971b)

$$\beta_{1i} \pm \hat{\sigma}_i^2 t_{(13, .025)} \sqrt{a^{1i}} \quad (3.23)$$

where $\hat{\sigma}_i^2$ is an estimate of the residual error variance associated with the i th regression, and a^{1i} is the diagonal element associated with β_{1i} in $(X'X)^{-1}$.

Realized heritabilities were estimated in each replicate of each selection treatment by regressing cumulative response to selection on cumulative selection differential, and as the ratio of total response to total selection differential (Hill, 1972). Standard errors of the estimates of realized heritability were calculated from the variance between estimates obtained in replicate lines of the same selection treatment (4 degrees of freedom). An analysis of variance of 40 realized heritability estimates was performed. The form of the analysis of variance is given in Table 7. For the analysis, it was assumed that the realized heritability estimates were independent and normally distributed.

In an experiment in which single-trait selection for X and for Y is practiced, the realized genetic correlation between the two traits can be estimated (Falconer, 1981) as

$$r_g = \left(\frac{C_X \cdot C_Y}{R_X \cdot R_Y} \right)^{1/2} \quad (3.24)$$

Table 7: Analysis Of Variance Of Estimates Of Realized Heritability.

Source ^a	degrees of freedom	Expected mean square ^b
P	1	$\sigma_e^2 + Q_1$
S	1	$\sigma_e^2 + Q_2$
P × S	1	$\sigma_e^2 + Q_3$
I	1	$\sigma_e^2 + Q_4$
P × I	1	$\sigma_e^2 + Q_5$
S × I	1	$\sigma_e^2 + Q_6$
P × S × I	1	$\sigma_e^2 + Q_7$
Replicates	4	$\sigma_e^2 + 8\sigma_R^2$
Residual	28	σ_e^2

a P: pre-selection mating system; S: selection mating system;
I: selection intensity.

b Q_i , $i = 1 \dots 7$ denote quadratic forms; σ_e^2 is the residual variance,
 σ_R^2 is the variance between replicates.

where R_X and R_Y are direct responses to selection for traits X and Y respectively, C_X is the correlated response of trait X when selection is for Y, and C_Y is the correlated response of Y when selection is for X. In this study, direct response (R_X) can be estimated for only one trait, pupal weight, and correlated responses (C_Y) can be assessed for larval weight and pupation time. Therefore, genetic correlations were estimated indirectly in each replicate as (Dickerson, 1969)

$$r_g = \frac{C_Y}{\sigma_{g_Y}} \cdot \frac{\sigma_{g_X}}{R_X} \quad (3.25)$$

where σ_{g_Y} and σ_{g_X} are genetic standard deviations for traits Y and X, respectively. Estimates of σ_{g_Y} and σ_{g_X} were obtained from the analyses in the pre-selection phase, and used in the above formula. Standard errors of realized genetic correlations were calculated from the variance between estimates obtained in replicate lines of the same treatment.

CHAPTER FOUR

4. RESULTS AND DISCUSSION

4.1. ESTIMATES OF GENETIC PARAMETERS IN THE BASE POPULATIONS.

Estimates of heritability and genetic and phenotypic correlations obtained in the randomly mated base population (R) from the two analysis of variance models are presented in Tables 8 and 9. Because within models there were no consistent differences between sexes in estimates of genetic parameters, the analyses were repeated across sexes, i.e., ignoring the sex classification. Results of these analyses in the R-mated base population are shown in Table 10 for heritabilities and in Table 11 for genetic and phenotypic correlations.

The estimates of heritability (Table 10) were within the range of those reported in studies involving the same foundation strain (e.g., Yamada and Bell, 1969; Scheinberg et al., 1967 and Bell and Moore, 1972). Heritability estimates obtained with the two models were similar, but the standard errors of the estimates obtained with model 1, calculated from the variance among replicates, were somewhat smaller than those obtained with model 2, calculated from approximate expressions. Estimates of heritability based on the sire component of variance were smaller than those obtained from the dam component, for all traits. This difference was more pronounced for larval weight than for the other traits. Possible causes of these differences could be dominance and maternal effects. These effects have been reported to affect larval weight (e.g., King and Dawson, 1972) and pupal weight (e.g., Bondari et al., 1978). Yamada and Bell (1969) argued that larger estimates from the dam component of variance might be due to dominance deviations rather than to maternal effects.

Table 8. Estimates of heritability and genetic and phenotypic correlations from half-sib covariances and their standard errors obtained with Model 1 in the randomly mated population, by sex^a.

Trait	Sex ^c	Trait ^b		
		PW	LW	PT
PW	M	.19 ± .13	-.01 ± .42	.43 ± .36
	F	.35 ± .13	-.08 ± .37	.32 ± .33
LW	M	.01 ± .12	.37 ± .13	-.67 ± .22
	F	-.03 ± .05	.25 ± .11	-.60 ± .13
PT	M	.59 ± .03	-.69 ± .01	.31 ± .09
	F	.48 ± .05	-.71 ± .03	.38 ± .12

^a Estimates of heritability in the diagonal; estimates of genetic and phenotypic correlations above and below the diagonal, respectively.

^b PW: pupal weight; LW: larval weight; PT: pupation time.

^c M: males; F: females.

Table 9. Estimates of heritability and genetic and phenotypic correlations from half-sib covariances and their standard errors obtained with Model 2 in the randomly mated population, by sex^a.

Trait	Sex ^c	Trait ^b		
		PW	LW	PT
PW	M	.24 ± .14	-.44 ± .65	.53 ± .46
	F	.37 ± .13	.03 ± .72	.37 ± .40
LW	M	-.19 ± .03	.37 ± .12	-.77 ± .22
	F	-.16 ± .01	.25 ± .12	-.77 ± .45
PT	M	.50 ± .14	-.69 ± .18	.33 ± .10
	F	.49 ± .16	-.69 ± .41	.38 ± .14

^a Estimates of heritability in the diagonal; estimates of genetic and phenotypic correlations above and below the diagonal, respectively.

^b PW: pupal weight; LW: larval weight; PT: pupation time.

^c M: males; F: females.

Table 10. Heritability estimates in the randomly mated base population obtained with Models 1 and 2.^a

Estimate ^c	Model	Trait ^b		
		PW	LW	PT
$\hat{h}_s^2$	1	.26 ± .12	.36 ± .13	.29 ± .08
	2	.30 ± .12	.26 ± .14	.39 ± .10
$\hat{h}_d^2$	1	.54 ± .16	.81 ± .12	.48 ± .13
	2	.48 ± .17	.93 ± .18	.44 ± .15
$\hat{h}_{s+d}^2$	1	.42 ± .05	.58 ± .04	.40 ± .05
	2	.39 ± .15	.59 ± .16	.39 ± .09

^a Models 1 and 2 are described in section 3.5.1.1.

^b PW: pupal weight; LW: larval weight; PT: pupation time.

^c See equations (3.3a), (3.3b) and (3.3c).

Estimates of genetic and of phenotypic correlations (Table 11) were, in general, in agreement with previous studies conducted in the same foundation stock (Bell, 1969; Bell and Moore, 1972; Englert and Bell, 1969, 1970). In the present study the genetic correlation between PW and LW in the R-mated population was negative when estimated from the sire component of covariance but positive when the dam component was used. While this might reflect the large sampling variance of the estimates, it is possible that additive; dominance and maternal effects might contribute to the covariance between these traits in opposite directions. The genetic correlation between PW and PT was estimated as positive in the present research. However, studies in the same foundation population (Bell, 1969; Bell and Burris, 1973) yielded negative estimates of the genetic correlation between these traits. Nevertheless, Englert and Bell (1970) reported positive estimates of the genetic correlation between PW and PT in some related populations. Further, Howe (1961) demonstrated the dependence of the sign and of the magnitude of the genetic correlation between PW and PT on the frequencies of "quick" and "slow" developing individuals in the population. When one type was more frequent than the other, the correlation was high and negative; the opposite was true when their frequencies were about equal (Howe, 1961). In this study, the frequencies of the above two types could have been about the same.

There was little or no difference between the regressions of sons on sires and those of daughters on sires. The same occurred with regressions of sons on dams (intra-sires) and those of daughters on dams (intra-sires). Hence, only regressions of individual offspring on parents will be reported here. Heritability estimates based on these regressions are presented in Table 12 for the randomly mated base population. There were

Table 11. Estimates of genetic ($\hat{r}_g$) and phenotypic ($\hat{r}_p$) correlations obtained in the randomly mated base population with Models 1 and 2.^a

Estimate ^c	Model	Trait ^b		
		PW and LW	PW and PT	LW and PT
$\hat{r}_{g_s}$	1	-.05 ± .20	.37 ± .23	-.64 ± .12
	2	-.21 ± .23	.45 ± .10	-.77 ± .10
$\hat{r}_{g_d}$	1	.21 ± .16	.24 ± .19	-.60 ± .16
	2	.10 ± .19	.43 ± .41	-.72 ± .40
$\hat{r}_{g_{s+d}}$	1	.03 ± .03	.26 ± .10	-.36 ± .01
	2	-.15 ± .09	.44 ± .16	-.71 ± .35
$\hat{r}_p$	1	-.01 ± .06	.54 ± .03	-.70 ± .01
	2	-.17 ± .21	.49 ± .22	-.69 ± .25

^a Models 1 and 2 are described in section 3.5.1.1.

^b PW: pupal weight; LW: larval weight; PT: pupation time.

^c See equations (3.4a)-(3.4c) and (3.5a).

Table 12. Heritability estimates from the regression of offspring on sire ($\hat{h}_{Os}^2$), on dam (intra-sire) ($\hat{h}_{Od}^2$) and of offspring on mid-parent ($\hat{h}_{Om}^2$) obtained in the randomly mated base population.

Estimate	Trait ^a		
	PW	LW	PT
$\hat{h}_{Os}^2$	.31 ± .08	.09 ± .54	.17 ± .03
$\hat{h}_{Od}^2$	.36 ± .08	.14 ± .08	.09 ± .11
$\hat{h}_{Om}^2$	.34 ± .02	.15 ± .02	.16 ± .02

^a PW: pupal weight; LW: larval weight; PT: pupation time.

small differences between estimates of heritability obtained from the three different types of regressions. The regression estimate of heritability of PW was larger than the one obtained from the covariance between half-sibs ($.26 \pm .12$, Table 10). However, the regression estimates for LW and PT were smaller than their half-sib counterparts in Table 10. This would suggest a negative covariance between additive direct and genetic maternal effects (Willham, 1963).

Note from Table 6 that in an assortatively mated population the offspring-parent regressions (with the exception of offspring-mid parent) would lead to upwardly biased estimates of heritability. In fact, these regressions (not presented here) were generally larger than those in the R-mated population.

In the assortatively mated (A) population, the iterative procedure of (3.12) failed to converge when the parameter vector $\tilde{\psi}$ was as in (3.6). Dominance and maternal contributions and associated correlations were then deleted from the model but the procedure still did not converge. At this point, the following further modifications of the model were made: a) correlations with expected values being a function of more than 5 parameters (e.g., offspring-parent correlation between Z - Y values; see Table 6) were deleted from the analysis. The parameter vector became $\tilde{\psi}' = [h_X, h_Y, h_Z, \theta_{XY}, \theta_{XZ}, r_{XY}, r_{XZ}]$; b) the restriction that the estimates of the parameters had to be within the parameter space during iteration was lifted; c) estimates of genetic parameters from the R-mated population were used to start iteration, except that genetic correlations were set to zero. With these modifications, Marquardt's (1963) algorithm converged in 7 iterations for raw correlations, and in 4 iterations for z-transformed correlations. The iteration procedure converged to the same values when estimates from

either model 1 or model 2 were used as starting values. Starting values from model 1 estimates and results of the iterations are presented in Tables 13 and 14 for the analysis with raw and z-transformed correlations, respectively. Estimates of the parameters were essentially the same in both cases. Note that only the estimate of the phenotypic correlation between PW and PT was outside the parameter space. The estimate of genetic correlation between LW and PT could not be obtained in the A-mated population because this parameter had to be dropped from the model.

Estimates of parameters in the R-mated and in the A-mated populations, with their approximate standard errors, are summarized in Table 15 for comparative purposes. Heritability estimates of PW and LW were larger in the A-mated population, but this was not the case for PT. The increase in heritability was highest for PW (46%), the trait on which assortative mating was practiced. This result was in agreement with theory because assortative mating for a trait with h^2 ranging from .26 to .30 ($p = .98$) would be expected to yield an equilibrium heritability between .35 and .42 (Bulmer, 1980).

The estimates of genetic correlation between PW and LW in the R-mated population were negative while those in the A-mated population were positive. This is at variance with the theory developed by Gianola (1982) which states that positive assortative mating for trait X should decrease a negative genetic correlation between X and Y in random mating. The genetic correlation between PW and PT was decreased by assortative mating, which is also in conflict with the theoretical results of Gianola (1982). However, as pointed out before, a positive genetic correlation between PW and PT in random mating is also in conflict with other studies (Bell, 1969; Bell and Burris, 1973).

Table 13. Assortatively mated population: values of the parameters during iteration (analysis with raw correlations).

Parameter ^(a)	Iterations			
	0	3	5	7
$\hat{h}_X$	.5100	0.6264	.6264	.6264
$\hat{h}_Y$	.6000	.6889	.6888	.6888
$\hat{h}_Z$	.5100	.4951	.4951	.4951
$\hat{\theta}_{XY}$	0	.2718	.2721	.2721
$\hat{\theta}_{XZ}$	0	.1457	.1457	.1457
$\hat{r}_{XY}$	-.0100	-.2353	-.2355	-.2355
$\hat{r}_{XZ}$	.5400	1.0138	1.0138	1.0138

(a) X: pupal weight; Y: larval weight; Z: pupation time. See footnote to Table 5 for definition of the parameters.

Table 14. Assortatively mated population: values of the parameters during iteration (analysis with z-transformed correlations).

Parameter ^(a)	Iterations			
	0	2	3	4
$\hat{h}_X$	.5100	.6394	.6381	.6381
$\hat{h}_Y$	.6000	.7029	.6956	.6956
$\hat{h}_Z$	.5100	.4961	.4957	.4957
$\hat{\theta}_{XY}$	0	.2696	.2620	.2620
$\hat{\theta}_{XZ}$	0	.1444	.1432	.1432
$\hat{r}_{XY}$	-.0100	-.2359	-.2215	-.2215
$\hat{r}_{XZ}$	.5400	1.0686	1.0508	1.0508

(a) X: pupal weight; Y: larval weight; Z: pupation time. See footnote to Table 5 for definition of the parameters.

Table 15. Summary of estimates of parameters in randomly mated (R) and assortatively mated (A) base populations.

Parameter ^a	Base populations	
	R ^b	A ^c
$\hat{h}_X^2$	.26 ± .12 (.30 ± .12)	.39 ± .02 (.41 ± .02)
$\hat{h}_Y^2$	.36 ± .13 (.26 ± .13)	.49 ± .04 (.48 ± .05)
$\hat{h}_Z^2$	.29 ± .08 (.39 ± .10)	.25 ± .03 (.25 ± .03)
$\hat{\theta}_{XY}$	-.05 ± .20 (-.21 ± .23)	.27 ± .10 (.26 ± .09)
$\hat{\theta}_{XZ}$	.37 ± .23 (.45 ± .10)	.15 ± .14 (.14 ± .12)
$\hat{\theta}_{YZ}$	-.64 ± .12 (-.77 ± .14)	not estimable
$\hat{r}_{XY}$	-.01 ± .06 (-.17 ± .21)	-.24 ± .38 (-.22 ± .33)
$\hat{r}_{XZ}$	.54 ± .03 (.49 ± .22)	1.01 ± .32 (1.05 ± .34)
$\hat{r}_{YZ}$	-.70 ± .01 -.69 ± .25	not estimable

^a See Table 5 for the definitions of parameters. X: pupal weight; Y: larval weight; Z: pupation time.

^b Entries are analysis of variance (half-sib) estimates from Models 1 and 2 (in parentheses)

^c Entries are minimum chi-square estimates from raw and z-transformed data (in parentheses).

4.2. DIRECT RESPONSE TO SELECTION.

Mean pupal weights for each of the 12 lines, averaged over replicates by generation of selection are presented in Table 16. These means were plotted against generations as shown in Figures 4 and 5 for the lines that had undergone pre-selection random or assortative mating, respectively. Environmental trends for pupal weight were negligible as suggested by the performance of the AACO and ARCO lines (Figure 5); nevertheless, the means of the RRCO and RACO lines varied somewhat from generation to generation.

Line means were deviated from their respective controls (RRCO, RACO, ARCO and AACO) and plotted against generation of selection as shown in Figure 6. As a result of a drop in mean pupal weight in the RACO line in generation 2 (Figure 4), the RA20 and RA50 lines showed a somewhat greater response to selection in this generation than expected from a linear trend (Figure 6). However, it is clear from Figure 6 that all lines, particularly those selected at higher intensity, responded to selection for PW.

The analysis of variance of line means (Table 17) indicated that pre-selection mating system and percent selected were the only significant sources of variation among line means, within generations. In the last generation, lines which had been mated assortatively in the pre-selection phase had, on average, higher mean pupal weight than those randomly mated. Similarly, lines selected at 20% had, on average, higher mean pupal weight than the lines selected at 50%.

Trends within individual selection lines were examined by regressing response (deviation from control) on generation number (Table 18). All regressions involving PW were positive and significant ($P < .05$) with

Table 16. Line means for pupal weight (μg) averaged over replicates, by generation of selection.

Line ^(a)	Generation		
	0	1	2
RR20	2485.7 $\pm$ 16.9	2586.2 $\pm$ 19.1	2719.5 $\pm$ 20.5
RR50	2477.3 $\pm$ 26.7	2515.7 $\pm$ 30.2	2591.0 $\pm$ 31.4
RRCO	2489.5 $\pm$ 30.8	2513.3 $\pm$ 34.9	2548.6 $\pm$ 37.4
RA20	2467.1 $\pm$ 16.9	2586.9 $\pm$ 19.1	2704.9 $\pm$ 20.5
RA50	2472.9 $\pm$ 26.7	2528.6 $\pm$ 30.2	2595.9 $\pm$ 32.4
RACO	2497.8 $\pm$ 30.8	2463.5 $\pm$ 34.9	2533.3 $\pm$ 37.4
AR20	2453.6 $\pm$ 16.9	2592.1 $\pm$ 19.1	2723.4 $\pm$ 20.5
AR50	2482.4 $\pm$ 26.7	2561.5 $\pm$ 30.2	2636.5 $\pm$ 32.4
ARCO	2546.7 $\pm$ 30.8	2542.5 $\pm$ 34.9	2548.6 $\pm$ 37.4
AA20	2477.5 $\pm$ 16.9	2650.5 $\pm$ 19.1	2785.9 $\pm$ 20.5
AA50	2510.3 $\pm$ 26.7	2540.2 $\pm$ 30.2	2654.5 $\pm$ 32.4
AACO	2543.1 $\pm$ 30.8	2539.7 $\pm$ 34.9	2558.3 $\pm$ 37.4

(a) The first and second letters indicate the mating system during the pre-selection and selection phases, respectively (R: random; A: assortative); 20, 50 and CO indicate the selection intensity treatment applied to a given line.

Figure 4: Mean pupal weight (μg) in the lines that had undergone pre-selection random mating, by generation of selection.

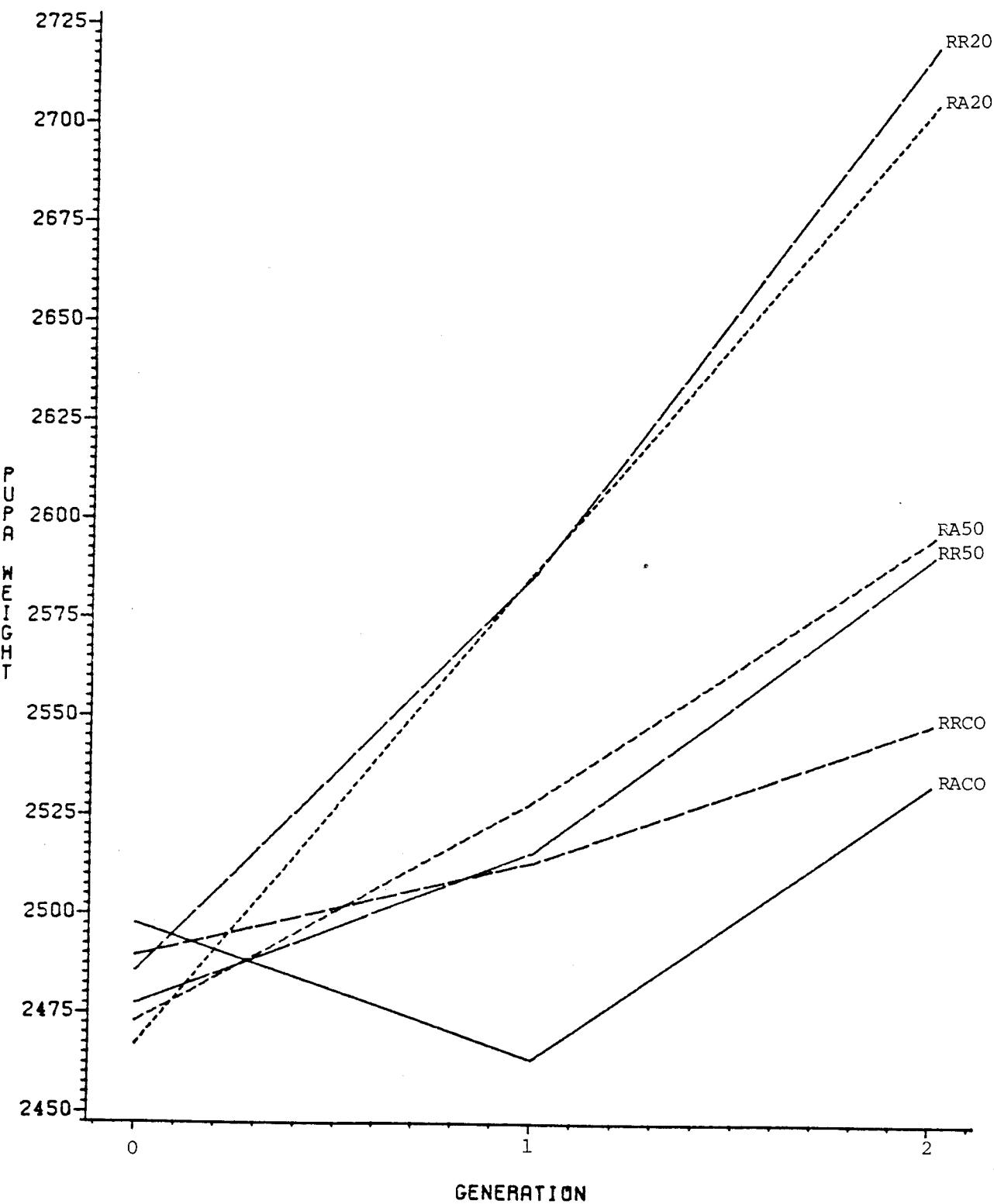


Figure 5: Mean pupal weight (μg) in the lines that had undergone pre-selection assortative mating, by generation of selection.

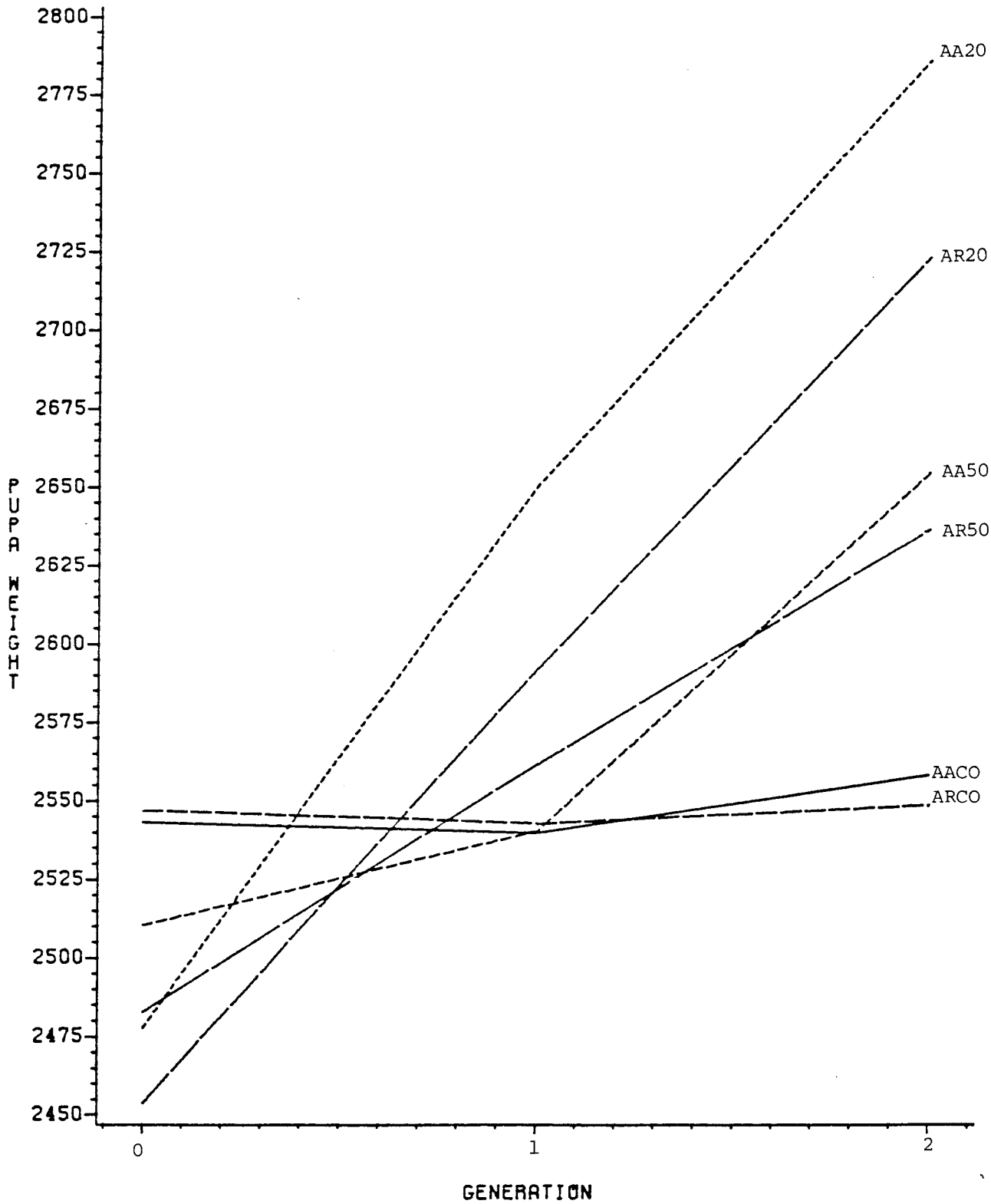


Figure 6: Mean pupal weight (μg) in the selection lines expressed as deviations from the controls, by generation of selection.

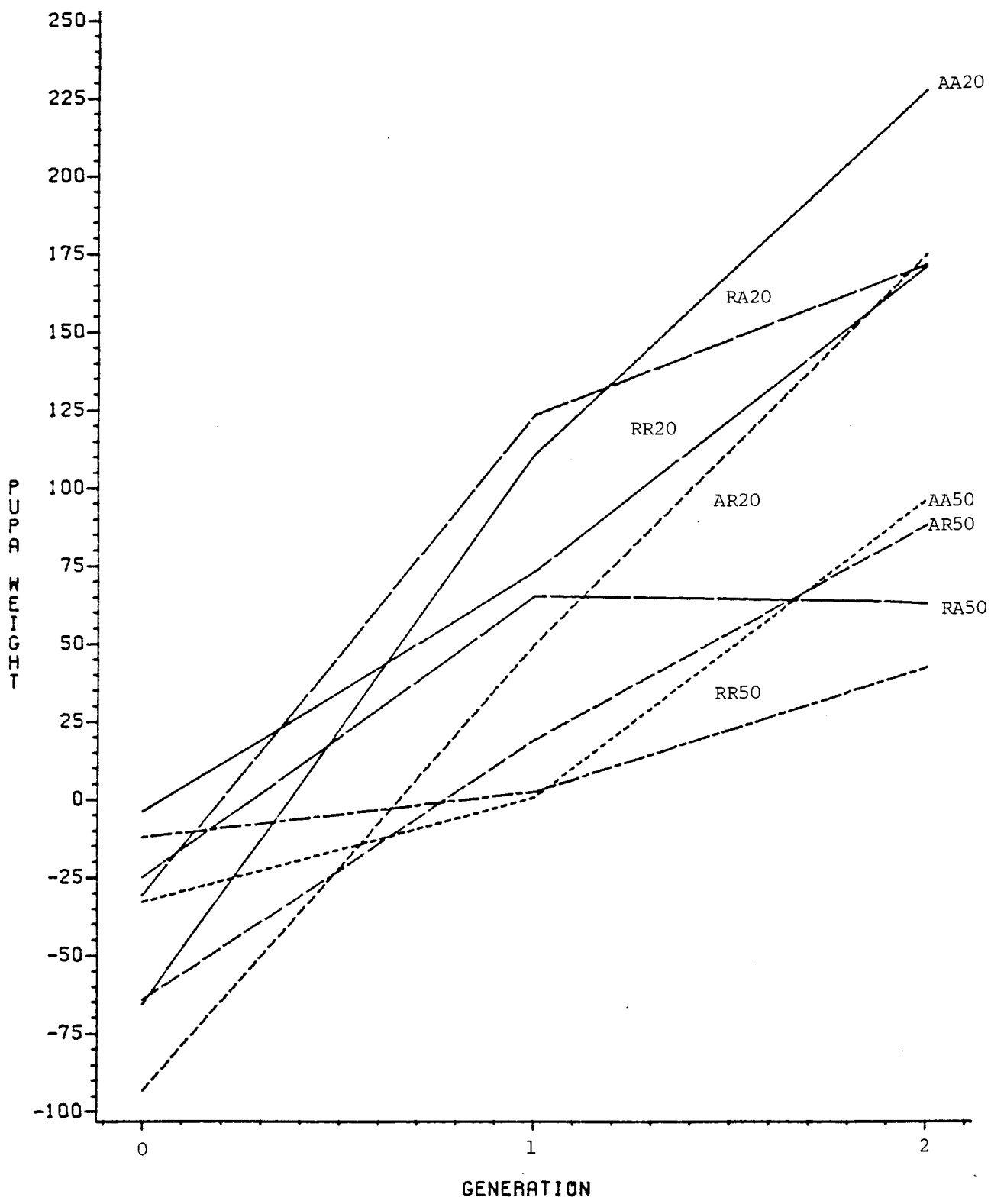


Table 17. Weighted analysis of variance of line means for pupal weight, by generation.

Source ^a	Degrees of freedom	Mean squares in generation		
		0	1	2
P	1	139,301.3	493,638.9 ^b	418,587.9 ^c
S	1	10,267.5	29.7	38,961.5
P × S	1	36,203.9	45,631.3	121,463.6
I	2	271,014.5	1,253,863.3 ^b	4,773,504.7 ^b
P × I	2	123,134.6	13,312.2	36,359.8
S × I	2	3,187.3	106,582.0	22,320.7
P × S × I	2	21,466.4	77,122.3	45,683.9
Residual	48	71,041.4	91,334.8	104,757.9

^a P: pre-selection mating system; S: selection mating system;
I: intensity of selection.

^b P < .05.

^c P < .10.

the exception of the one for the RA50 line. On average, lines which were A-mated during the pre-selection phase had a greater rate of response than those that had been R-mated (105.3 vs 64.9 $\mu\text{g/generation}$, respectively). Similarly, 20% selected lines had a larger average rate of response than the 50% selected lines (117.28 vs 52.92 $\mu\text{g/generation}$, respectively). The average rate of response in the lines that were assortatively mated during selection was similar to that in the randomly mated ones (89.03 vs 81.18 $\mu\text{g/generation}$, respectively).

Realized heritability estimates of PW, averaged over replicates, are presented in Table 19. Estimates from the regression of cumulative response on cumulative selection differential were generally smaller (with the exception of RA lines) than those calculated as the ratio of total response to total selection differential. However, the latter had smaller sampling variances. Note that the standard errors of realized heritability estimates in the RA20 and RA50 lines were much larger than those in other lines. This was due to the fact that some estimates in these lines were outside of the parameter range. For example, estimates in different replicates of the RA50 line ranged between -.02 and 1.81.

The analysis of variance of realized heritability estimates of PW (Table 20) indicated that realized heritability of PW varied with pre-selection mating system ($P < 0.10$) and with intensity of selection ($P < 0.05$). Lines that were assortatively mated in the pre-selection phase had higher estimates of realized heritability than lines randomly mated. Also, estimates in the 20% selected lines were higher than those selected at 50%. This last trend is depicted in Figure 7 where a plot of the fitted regressions of cumulative response to selection for PW on cumulative selection differential is presented. Larger realized heritability estimates

Table 18. Regressions of line means deviated from their controls on generation number for pupal (PW) and larval (LW) weights and pupation time (PT), and their standard errors.

Line ^a	Trait		
	PW	LW	PT
RR20	87.35 ± 18.00 ^a	-32.74 ± 22.90	.14 ± .10
RR50	27.29 ± 12.63 ^b	-13.83 ± 24.99	-.03 ± .11
RA20	101.18 ± 36.18 ^c	21.82 ± 24.99	.13 ± .13
RA50	43.77 ± 25.54	25.76 ± 29.11	.19 ± .17
AR20	133.95 ± 15.17 ^c	45.82 ± 39.34 ^b	.01 ± .10
AR50	76.10 ± 19.37 ^c	82.97 ± 35.13 ^b	.08 ± .13
AA20	146.63 ± 18.37 ^c	74.66 ± 28.07 ^b	.14 ± .08
AA50	64.54 ± 13.86 ^c	64.25 ± 24.03 ^b	.12 ± .09

^a The first and second letters indicate the mating system during the pre-selection and selection phases, respectively (R: random, A: assortative); 20, 50 and CO indicate the selection intensity treatments applied to a given line.

^b P < .05.

^c P < .01

Table 19. Realized heritability estimates for pupal weight by treatment combination, averaged over replicates.^a

Percentage selected	Pre-selection X selection mating system ^b			
	RR	RA	AR	AA
20%	.30 ± .04 (.23 ± .06)	.31 ± .11 (.42 ± .18)	.29 ± .07 (.16 ± .06)	.39 ± .04 (.37 ± .15)
50%	.12 ± .04 (.05 ± .08)	.19 ± .16 (.41 ± .35)	.23 ± .08 (.09 ± .08)	.30 ± .05 (.03 ± .11)

^a Values without parentheses are the ratio of total response to total selection differential. Values in parentheses are the regressions of cumulative response on cumulative selection differential.

^b R: random mating; A: assortative mating.

Table 20: Analysis of variance of realized heritability estimates for pupal weight.^a

Source ^b	Degrees of freedom	Mean squares
P	1	.0625 ^c
S	1	.0314
P × S	1	.0027
I	1	.1408 ^d
P × I	1	.0088
S × I	1	.0016
P × S × I	1	.0024
Replicates	4	.0204
Residual	28	.0196

^a Ratio of total response to total selection differential.

^b P: pre-selection mating system; S: selection mating system;
I: intensity of selection.

^c $P < 0.10$.

^d $P < 0.05$.

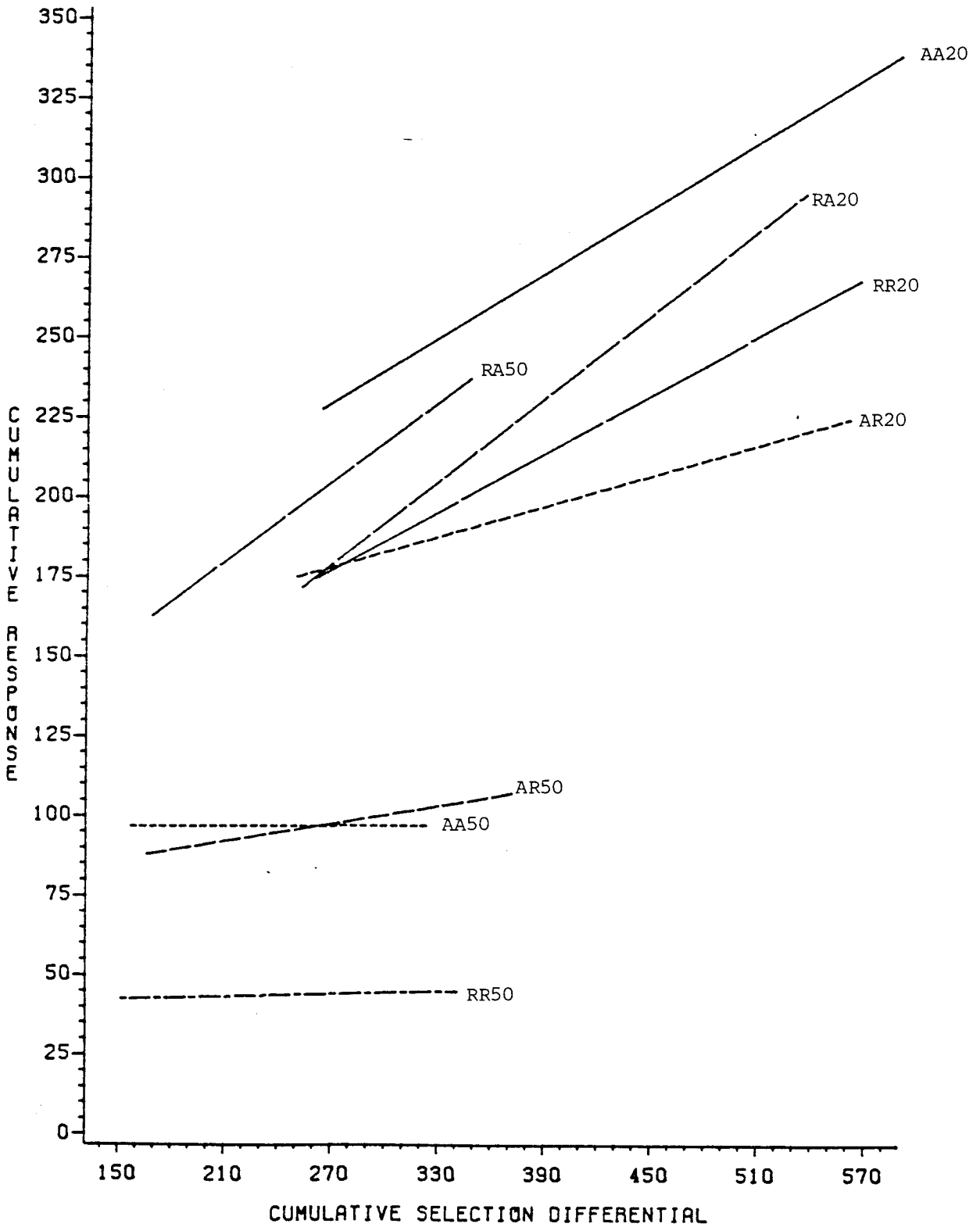
at higher selection intensities were also obtained by Hanrahan et al. (1973). They reported larger estimates of realized heritability for body weight in mice when 25% of the parents were selected than when 50% were selected. The population size ($N_e = 8$) used by Hanrahan et al. (1973) compares to $N_e = 20$ (10 mating pairs) in the present study. They argued that when population size is small, high selection intensity would reduce genetic drift and observed that estimates of heritability in the base population were in closer agreement with realized heritability at high selection intensity than with estimates of this parameter obtained at low selection intensity. This was not the case in larger population sizes used by Hanrahan et al. (1973).

Estimates of realized heritability for PW were in agreement with those found by other workers in the same foundation population. For example, Wilson et al. (1965) reported realized heritability estimates between .12 - .39; Bell and Burris (1973) found an estimate of $.30 \pm .06$. However, estimates obtained in the 50% selected lines were lower than those obtained in the base population from correlations between relatives. King and Dawson (1972) argued that estimates in the base population are often biased because of the method of estimation and because of the presence of non-additive gene effects. However, this explanation is not suitable here as the realized heritability estimates obtained in 20% selected lines were in general agreement with those obtained in the base population, as it occurred in the study of Hanrahan et al. (1973)

4.3. CORRELATED RESPONSES TO SELECTION.

Line means (deviated from controls) for LW and PT plotted against generations of selection for PW are presented in Figures 8 and 9, respectively. Analyses of variance of line means by generations (Table 21)

Figure 7: Fitted regression of response to selection for pupal weight cumulative selection differential (μg), by line.



indicated that larval weight was affected by pre-selection mating system ($P < .05$) in all generations, and by the selection mating system in generation 1. Larval weight was heavier in the lines that had been A-mated during pre-selection, and the difference remained throughout selection (Table 22). In generation 1, lines that were assortatively mated during selection phase were heavier ($P < .05$) than their randomly mated counterparts. There is no obvious explanation for these differences. Pupation time was not affected by any of the sources of variation considered (Table 21); line means for pupation time are presented in Table 23.

Regressions of LW and PT (deviated from controls) on generation of selection for PW are in Table 18. Most correlated responses were not different from zero ($P < .05$). Exceptions were positive regressions for LW in the AR50, AA20 and AA50 lines. Considering that selection intensity did not affect LW in any generation (Table 21), it seems safe to conclude that correlated responses in this study were negligible or nonexistent. Realized genetic correlations in the lines that were R-mated in the pre-selection phase were:

	<u>PW and LW</u>	<u>PW and PT</u>
RR20	$-.17 \pm .23$	$.05 \pm .19$
RR50	$.00 \pm .11$	$-.41 \pm .14$
RA20	$.21 \pm .26$	$.14 \pm .13$
RA50	$.10 \pm .21$	$.06 \pm .22.$

The realized genetic correlations in the lines that were A-mated in the pre-selection phase were not estimated because the iterative procedure did not yield estimates of additive genetic variance.

Figure 8: Line mean for 13 day larval weight (μg), deviated from their controls, plotted against generations of selection for pupal weight.

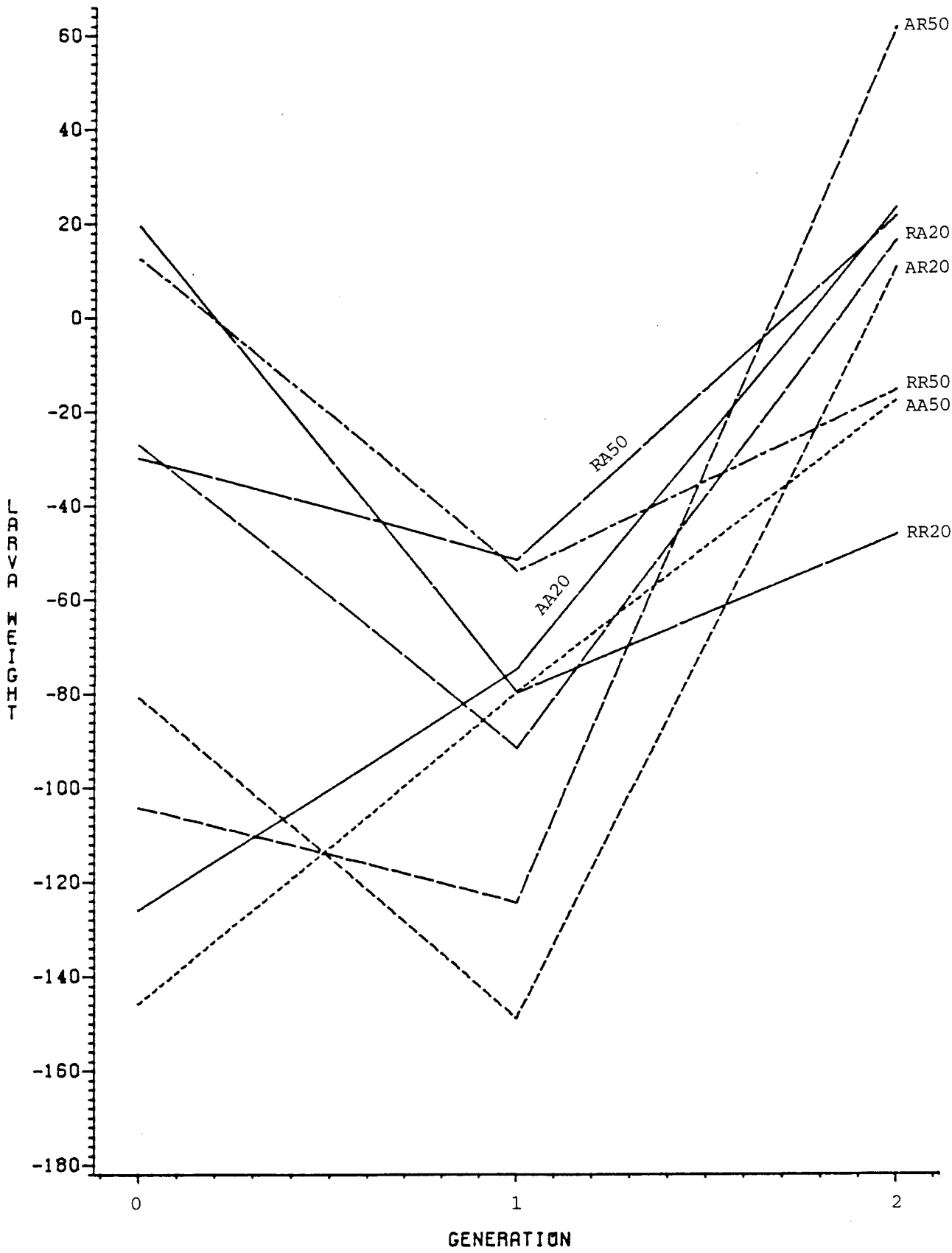


Figure 9: Line mean for pupation time (days), deviated from their cont.
plotted against generation of selection for pupal weight

Table 21. Weighted analysis of variance of line means for larval weight (LW) and for pupation time (PT), by generation.

Source ^a	d.f. ^b	Mean squares (LW) by generation			Mean squares (PT) by generation		
		0	1	2	0	1	2
P	1	4484281.5 ^(c)	7174769.5 ^(c)	1456 822.1 ^(c)	4.715	3.96	9.912
S	1	73161.4	1824913.8 ^(c)	90904.5	.667	.618	.086
P × S	1	39515.6	1041448.9	128656.5	2.647	1.723	1.876
I	2	431445.9	1127089.7	21369.7	.653	6.903	3.353
P × I	2	346197.1	52187.6	30503.6	3.999	.625	2.446
S × I	2	62301.8	28181.5	36531.2	2.615	.957	2.413
P × S × I	2	20.6	58968.1	75580.7	.302	.133	1.791
Residual	48	161211.7	414606.4	258485.5	4.126	2.711	5.452

^a P: pre-selection mating system; S: selection mating system;
I: intensity of selection.

^b d.f.: degrees of freedom

^c P < 0.05.

Table 22. Line means for larval weight (μg) by generation.

Line ^(a)	Generation		
	0	1	2
RR20	987.6 $\pm$ 25.4	1017.9 $\pm$ 40.7	968.8 $\pm$ 32.2
RR50	980.5 $\pm$ 40.2	1043.7 $\pm$ 64.4	999.7 $\pm$ 50.8
RRCO	968.1 $\pm$ 46.4	1097.7 $\pm$ 74.4	1014.7 $\pm$ 58.7
RA20	995.0 $\pm$ 25.4	1027.4 $\pm$ 40.7	1034.7 $\pm$ 32.2
RA50	992.2 $\pm$ 40.2	1067.4 $\pm$ 64.4	1039.8 $\pm$ 50.8
RACO	1022.1 $\pm$ 46.4	1119.1 $\pm$ 74.4	1018.1 $\pm$ 58.7
AR20	1085.8 $\pm$ 25.4	1086.6 $\pm$ 40.7	1067.4 $\pm$ 32.2
AR50	1062.4 $\pm$ 40.2	1111.2 $\pm$ 64.4	1118.3 $\pm$ 50.8
ARCO	1166.5 $\pm$ 46.4	1235.7 $\pm$ 74.4	1056.5 $\pm$ 58.7
AA20	1075.1 $\pm$ 25.4	1251.9 $\pm$ 40.7	1099.1 $\pm$ 32.2
AA50	1055.0 $\pm$ 40.2	1247.0 $\pm$ 64.4	1058.2 $\pm$ 50.8
AACO	1200.9 $\pm$ 46.4	1326.6 $\pm$ 74.4	1075.6 $\pm$ 58.7

(a) See Table 16 for line identification.

Table 23. Line means for pupation time (days), by generation.

Line ^(a)	Generation		
	0	1	2
RR20	19.8 ± .1	19.7 ± .1	20.4 ± .2
RR50	19.8 ± .2	19.6 ± .2	20.1 ± .2
RRCO	20.0 ± .2	19.6 ± .2	20.0 ± .3
RA20	19.7 ± .1	19.8 ± .1	20.3 ± .1
RA50	19.5 ± .2	19.7 ± .2	20.2 ± .2
RACO	19.9 ± .2	19.5 ± .2	20.2 ± .3
AR20	19.8 ± .2	19.9 ± .1	20.4 ± .1
AR50	20.0 ± .2	19.8 ± .2	20.4 ± .2
ARCO	19.7 ± .2	19.7 ± .2	20.4 ± .3
AA20	20.0 ± .1	19.8 ± .1	20.7 ± .1
AA50	19.9 ± .2	19.8 ± .2	20.6 ± .2
AACO	19.8 ± .2	19.5 ± .2	20.2 ± .3

(a) See Table 16 for line identification.

4.4. GENERAL DISCUSSION.

The work of Blair et al. (1961), McBride and Robertson (1963) and Wilson et al. (1965) had suggested that for assortative mating to enhance response to selection greatly, the heritability of the selected trait (h^2) and the phenotypic correlation between mates (ρ) should be high enough so that the genetic correlation between mates ($m = \rho h^2$) is at least 0.5. Under perfect assortative mating ($\rho = 1$) this implies that h^2 should be at least 0.5. Prior to the beginning of this experiment, previous work with this stock (Bell, 1969) suggested that the heritability of PW would have been at least 0.5 in a randomly mating population and, in theory, even larger in an assortatively mated population. Also ρ was expected to be greater than 0.9. It turned out that heritability of PW was estimated at $.26 \pm .12$ and $.38 \pm .02$ in the R and A unselected populations, respectively, and the estimate of ρ was $.98 \pm .01$. Thus, while there was a trend for assortative mating during selection to enhance the effectiveness of selection (Table 19), its effect was small and not statistically significant. In this respect, this study was in agreement with previous ones. However, an increase in heritability in an unselected population following assortative mating was indicated by this study (Table 15). This is in agreement with theory (e.g., Bulmer, 1980) as pointed out previously.

The analytical work of Baker (1973) and the simulation study of De Lange (1974), indicated that assortative mating of selected individuals would be expected to increase response to selection more at high heritability and low selection intensity than at other combinations of these factors. This is because truncation selection reduces the genetic correlation between mates by reducing heritability. The higher the intensity of selection, the more the reduction in heritability in long term. This reduction also

depends on the number of generations of selection. In the present study, and contrary to Baker's (1973) results, realized heritability was larger when 20% of the parents were selected than when 50% were selected. This was true both in assortatively and randomly mated lines. The low heritability of PW observed in this study and the short duration of the experiment might have constrained the realization of Baker's (1973) expectations.

The study indicated that assortative mating of parents prior to selection enhanced the effectiveness of short-term response to selection. However, this cannot be suggested in practical animal breeding programs because short-term response to selection per generation of pre-selection and selection would not be enhanced by assortative mating. Given a fixed number of generations, the total response would be larger if selection were started from the onset than if a few generations were used to boost the genetic variance via assortative mating prior to selection. However, assortative mating prior to selection could be helpful in the development of synthetic breeds or strains. In breed development programs, assortative mating could replace random mating during the period when selection is not being practiced. This would increase genetic variance within the synthetic breed, hence, the subsequent response to selection.

Estimates of genetic parameters in the randomly mated base population indicated that selection for PW should have elicited a negligible correlated response in LW, and a positive correlated response in PT. This was not found in the selection phase of the experiment. It is possible, however, that if the experiment had continued for more generations, correlated responses might have been observed. Work on the effects of assortative mating on long-term direct and correlated responses to selection is, therefore, indicated.

CHAPTER FIVE

SUMMARY AND CONCLUSIONS

Joint effects of pre-selection mating system (random: R; assortative: A), selection mating system (R;A) and percent truncation selected (20%; 50%; 0% (control: CO)) were studied in a $2 \times 2 \times 3$ factorial experiment with 5 replicates to examine direct response to selection for pupal weight (PW), and correlated responses of larval weight (LW) and pupation time (PT) in Tribolium castaneum. Prior to selection two groups each with 50 males mated to 100 females, were either R-mated or A-mated for 3 generations. At generation 3, 6 lines were formed in each group, one for each treatment combination, and selection began. Each line was maintained during 2 generations of selection by mating 10 pairs of individuals.

Variance components from a hierarchical linear model and regressions of progeny on parents were used to estimate genetic parameters in the R-mated group prior to selection. "Equilibrium" genetic parameters in the A-mated group prior to selection were estimated by a non-linear minimum chi-square procedure. Estimates of heritability in the R-mated (from the sire components of variance) and in the A-mated groups were, respectively, for PW: $.26 \pm .12$ and $.38 \pm .02$; LW: $.36 \pm .13$ and $.49 \pm .04$; and PT: $.29 \pm .08$ and $.25 \pm .03$. Estimates of heritability agreed well with others found in previous studies in the same foundation stock. In agreement with theory, the estimate of heritability of PW in the A-mated population was larger than in the R-mated group. Estimates of genetic correlation between PW and LW were negative in the R-mated group and positive in the A-mated

one. This is in conflict with theory. The genetic correlation between PW and PT in the R-mated group was decreased by assortative mating, which is also at variance with the theory.

Response to selection was assessed using weighted analysis of variance of line means, by generation, and unweighted analysis of variance of realized heritability estimates calculated as the ratio of total response to cumulative selection differential. Rates of responses to selection were evaluated by regressing line means (deviated from the appropriate control) on generation number. Analysis of line means for pupal weight within generations of the selection phase of the experiment indicated that pre-selection mating system ($P < 0.1$) and percent selected ($P < 0.05$) were the only significant sources of variation among such means. Mean pupal weight in lines that were A-mated prior to selection was larger than in corresponding R-mated lines. Similarly, pupal weight was higher in lines where 20% of parents were selected than in those where 50% were selected. Estimates of realized heritability, calculated as the ratio of total response (deviated from the control lines) to cumulative selection differential were by treatment: RR20: $.30 \pm .04$; RR50: $.12 \pm .04$; RA20: $.31 \pm .11$; RA50: $.19 \pm .16$; AR20: $.29 \pm .07$; AR50: $.23 \pm .08$; AA20 $.39 \pm .04$; and AA50: $.30 \pm .05$. Realized heritability of PW was increased by pre-selection assortative mating ($P < 0.10$) and by more intense selection ($p < 0.05$). Mating system during selection had no effect on realized heritability. Estimates of realized heritability calculated as the regression of cumulative response on cumulative selection differential were generally smaller than those calculated as the ratio of total response to total selection differential. Realized heritability estimates in the lines selected at 50% intensity were generally lower than those found in the base population.

However, the latter were in good agreement with the realized estimates in the lines where 20% of the parents were kept. No significant correlated responses to selection for PW was observed.

The study suggested the following: 1) assortative mating of parents for a few generations prior to selection seemed to enhance heritability in the base population, and short-term response to selection; 2) assortative mating did not seem to enhance short-term response per generation of pre-selection and selection; and 3) high selection intensity was more effective than low when parents were randomly or assortatively mated during selection. The effects of assortative mating on long-term direct and correlated responses to selection require study.

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