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A study of genetic maternal effects in a designed experiment using Tribolium

Khorsand Bondari
Iowa State University

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A study of genetic maternal effects in a designed experiment using Tribolium

by

Khorsand Bondari

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Dean of Graduate College

Iowa State University
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1971
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INTRODUCTION

Reproduction is a complex organization of many physiological mechanisms. Certain of these mechanisms in the female, such as gestation and lactation (in mammals) have a strong influence on pre- and post-partum development of the young. The dependence of the offspring on the mother for growth and development makes the maternal influence part of the early environment of the offspring. Thus, a dam contributes to the growth of her offspring by the maternal environment she provides and also by the genes for growth she transmits. Although the maternal performance of the dam is usually environmental with regard to the offspring, it is partly conditioned by genes in the dam (Lush 1949). A sample of these genes will also be transmitted to the offspring. Willham (1963) defined such environmental effects on the offspring which are due to the genotypic differences among their dams as genetic maternal effects. The non-genetic portion of the maternal effects, which is due to the environmental differences among dams expressed in the phenotypic measurements of their offspring, is classified as the environmental maternal effect.

The interest of the breeders in genetic maternal effects is based on:
1. Improvement in maternal performance
2. Elimination of its influence on the trait so that selection can be for the direct genetic effect.

If a genetic correlation exists between the genotypic value for the direct effect and the genotypic value for the maternal effect, then selection response for a trait influenced by both a direct and maternal effect will depend on the correlation. Should this correlation be negative, selection based on the phenotypic values of the individuals (mass
selection) in the positive direction may have an adverse effect on the maternal ability of the dams. This is because the genotypic differences among those selected offspring becoming future dams will be expressed in the phenotype of their offspring. Information concerning direction and magnitude of such genetic correlations is of great importance in predicting a reliable response to selection. Providing such information is no simple matter due to the following problems:

1. The expression of maternal effects is limited to only one sex.
2. There is a generation delay for the expression of maternal performance since it cannot be directly measured on the individual himself.
3. The joint expression of the direct and maternal components of a character on the phenotypic value of a trait.

However, the correlations between relatives as applied to the problem of maternal effects by Dickerson (1947), Cockerham (1952), Kempthorne (1955), Koch and Clark (1955), Willham (1963), etc. provide a tool for exploring this area. The accuracy of the estimates of the genetic parameters derived by this method depends on:

1. Genetic relationships between relatives involved
2. Number of groups of relatives (e.g. sire groups)
3. Number of progeny per group (group size)
4. Design of the experiment and type of the relationships utilized
5. Assumptions made (no epistasis, no dominance, etc.).

The importance of maternal effects was brought to the attention of researchers when the inconsistency of the heritability estimates computed from different relationships was observed. This resulted because the relative magnitude of the variance components and the genotypic covariance between relatives computed for the traits influenced by maternal effects
vary greatly with the sign and magnitude of the genetic correlation which results from both direct and maternal causes. For instance, the dam component of variance ($c_{D/S}^2$) in a hierarchal classification is expected to be larger than the sire component, $c_S^2$, since $c_{D/S}^2 - c_S^2$ measures the total contributions of the maternal effects. But this is not always the case and a high negative direct-maternal covariance can alter the situation. The heritability estimate from the regression of offspring on dam may be overestimated if this covariance is positive and may be underestimated if it is negative. Falconer (1965) has also indicated in his litter size data that the inconsistency in heritability estimates can be accounted for after the maternal effect is considered.

This study, using a laboratory organism (Tribolium castaneum), was undertaken to develop, conduct, and analyze an experiment designed to estimate direct and maternal genetic variance and the direct-maternal genetic correlation for two traits influenced by maternal effects. Such a study provides a design and a pilot examination of such a design using biological material. The parameters estimated for Tribolium castaneum should indicate the possible magnitudes of the parameters to be found in economically important species. The designs used in this experiment are chosen to be feasible to farm animals.
REVIEW OF LITERATURE

To gain insight into this investigation, literature reports concerning maternal effects and their influences on the growth and development of the offspring were reviewed. The reports dealing with this problem were numerous since several traits of economic importance (e.g. birth weight, weaning weight, and litter size) are influenced by maternal effects. There is a genetic association between the development of such traits and the maternal contributions of a related individual. This fact creates difficulties in obtaining unbiased estimates of genetic parameters independent of the contribution of the maternal effects. Furthermore, the lack of consistency in estimates and the difficulties in interpretation of genetic parameters made many researchers become deeply interested in finding a means of evaluating genetic and environment maternal influences. This continued interest is clearly reflected in a series of publications by each of several authors, e.g. Dickerson, Falconer, Koch and Clark, Willham, and others.

These publications which have developed the basic concept and understanding of maternal effects and serve as a tool and guidance for other researchers are classified as theory. The rest of the reports which are directly or indirectly concerned with the results of these papers are classified as results. The results are subdivided into two classes, designed and non-designed experiments. Designed experiments include cross-fostering and other experiments which were specifically designed for the evaluation of genetic and environmental maternal effects. The non-designed subdivision does not necessarily imply that the experiments were not designed for anything, but that they were not originally planned and
carried out with a pertinent mating scheme designed for the study of maternal effects.

Theory

Hazel and Lamoreux (1947) undertook a study to investigate the probable influence of maternal effects and nicking upon variation in body weight at 22 weeks of age and in sexual maturity. Three sets of diallel mating, using White Leghorn, provided the data. The difference between dam and sire component of variance was utilized to estimate the importance of maternal effects. The estimated maternal effects were 5.1% for body weight and zero for sexual maturity. The existence of maternal effects for body weight was attributed to the differences in quantity of nutrition (egg size), quality of nutrition, disease organisms, and protective antibodies transmitted through the eggs to the offspring.

Dickerson (1947), in analyzing swine data, defined heritability of the maternally influenced traits as the regression of transmitting ability (genotypic value of an individual for a trait plus his genotypic value for maternal effects) on individual performance. The genetic components of this regression were obtained by a path coefficient diagram. Although the author did not separately measure variations due to the transmitted and direct maternal influence of the dam and their covariance, he examined the consequences of their existence. The results of this study, which in general agreed with the findings of Dickerson and Grimes (1947), indicated that a genetic antagonism may exist between good milking ability and rapid, economical fattening ability. This speculation resulted when the regression of offspring on sire for feed requirement exceeded the
corresponding value for the regression of offspring on dam. The author suggested that the maximum litter performance may be achieved through the crossing of sows of one line with good milking ability with the boars of another line with good rate and economy of post-weaning gains. This was suggested because the results indicated that the genes which cause pigs of a line to gain more economically may also cause the sows of that line to become poorer mothers.

Cockerham (1954) examined the type of variation that may influence the relationship between different relatives. A path coefficient diagram as shown in Figure 1 was used to show the dam-offspring relationship for a character influenced by a maternal effect. The phenotype of the offspring \((y)\) was considered to be influenced by his own additive genetic value \((G_{oy})\), environmental effects \((E_y)\), and additive genetic value of the dam's maternal ability \((G_{my})\),

\[
y = \mu_y + G_{oy} + G_{my} + E_y.
\]

By a similar description, the dam's phenotypic value \((x)\) is

\[
x = \mu_x + G_{ox} + G_{mx} + E_x.
\]

where \(G_{mx}\) is the additive genetic effect of the genes of the granddam in maternally influencing the growth of the dam. The offspring-dam covariance \((\text{Cov} \ xy)\) was computed as

\[
\text{Cov} \ xy = 1/2 \sigma_G^2 + 1/2 \sigma_G^2 + 5/4 \rho G_o G_m \sigma_G \sigma_G
\]

where \(\rho G_o G_m\) represents the correlation between the additive genetic effect of the dam's own genes for her growth \((G_{ox})\), and the additive genetic effect of the dam's own genes in maternally influencing the growth of
her offspring \( G_{my} \). This correlation results from the pleiotropic effects of the genes of the dam. The correlation between \( G_{oy} \) and \( G_{mx} \) is \( 1/4 \rho_{G_{o}G_{m}} \). The author suggested that this covariance accompanied by the sire-offspring covariance \( (1/2 \sigma_{G_{o}}^{2} + 1/4 \rho_{G_{o}G_{m}} \sigma_{G_{o}} \sigma_{G_{m}}) \) and the paternal half-sib covariance \( (1/4 \sigma_{G_{o}}^{2}) \) be utilized to estimate the two genetic standard deviations \( (\sigma_{G_{o}} \text{ and } \sigma_{G_{m}}) \) and the genetic correlation \( (\rho_{G_{o}G_{m}}) \).

In this procedure, dominance and epistatic effects were assumed to be zero.

![Diagram](image)

**Figure 1.** Path coefficient diagram showing the relationship between offspring and dam for a character that is influenced maternally by the genes of the dam and directly by the individual's own genes (Cockerham, 1954, p. 107)
Koch and Clark (1955) utilized the theoretical composition of the dam-offspring, sire-offspring, and paternal and maternal half-sib correlations to estimate the influence of maternal environment and the direct-maternal genetic correlation on the performance of the offspring. Although the number of unknown genetic parameters exceeded the number of equations which did not yield a particular solution, a range of values was determined. The equations were obtained by use of path coefficient diagrams. The results of this study indicated that a negative direct-maternal genetic correlation may exist for some traits of economic importance in beef cattle (e.g. weaning gain and score).

Kempthorne (1955) has considered genetically determined maternal effects under the control of a single locus with pleiotropic effects. He assumes that the genotypic value of an individual is determined additively by the joint effect of an individual's own genes and by the effect of the maternal genotype. Furthermore, he indicated that evaluation of the relationships involving maternal effects would require knowledge of seven parameters and cannot be understood from the total variance, sire-offspring, dam-offspring, and full-sib covariances.

Willham (1963) extensively examined the composition of the covariance between relatives when a maternal effect was involved. Although no data were available, the author hypothetically illustrated how each correlation between certain relatives was affected by a maternal influence. An investigation of several relationships outlined in this study indicated that various cousin relationships were well-suited for the study of genetic maternal performance.

Willham (1964) has indicated that the problem of obtaining estimates
of Cov (G_0, G_m) and V(G_m) can be solved by using grandchildren of a set of bulls. G_0 is the additive genetic value of an individual for the trait o and G_m is the additive genetic value of a related individual (dam) for the component trait m (maternal effect). Although the relationships are rather low, the estimates are shown to be free of environmental correlations.

The author has also pointed out that because of the high sampling errors of such estimates, one could only hope to detect the existence of any genetic antagonism in order to formulate a hypothesis which could be tested in selection studies.

Falconer (1965) using the data reported elsewhere (Falconer, 1955 and 1960a) showed that inconsistency in heritability estimates from the daughter-dam regression (zero), full-sib correlation (21%), and response to selection (24%) can be attributed to a maternal effect. Maternal effect (M) was defined as a linear function of the mother's phenotypic value (P') such that M=mP'. In this relationship, m is the partial regression coefficient relating phenotypic values of daughters to their mothers in the absence of genetic variation among the mothers. This coefficient was estimated to be -.133 indicating that so weak a maternal effect was enough to account for the wide discrepancy between the response to selection and the daughter-dam regression.

Eisen (1967) proposed three mating designs to yield 13, 10, and 12 different types of relatives, respectively. The expected genetic covariances between relatives (in the absence of epistasis) may be utilized to estimate eight genetic and environmental parameters. These parameters include direct additive and dominance variances, maternal additive and dominance variances, direct-maternal additive and dominance covariances,
and random and maternal environmental variances. The estimates of the eight parameters may be obtained by employing a least-square procedure to solve a set of simultaneous linear equations.

Koch (1969) developed a technique to evaluate the influence of the environment of a dam on the phenotype of her offspring. A path coefficient diagram is used to obtain the theoretical expectation of dam-offspring correlation. Restricting this correlation to an intra-granddam basis removes the direct effect of genotype for maternal ability. Since in this case all dams have the same granddam, the genetic variance among dams is reduced by 1/4, but the correlated effects of environmental influences remain unchanged. Thus, the difference between the two correlations does not include an environmental correlation. The results of analyzing weaning weight data (in cattle) in this way suggested a negative association between the environment affecting the growth of a dam and the maternal environment she provides her offspring.

Results

1. Designed experiments:

An asymmetry of response to selection for characters influenced by maternal effects was reported by Falconer (1955). The character selected for 30 generations of upward selection and 24 generations of downward selection was body weight up to six weeks of age. This response was divided into two components--weight at three weeks of age (weaning weight) which is mainly determined by the mother, and the post-weaning growth which is mainly determined by the individual itself. There was evidence
that the asymmetry affected only the maternal component of the weight and not the post-weaning growth. The weaning weight increased very little in the large line but decreased markedly in the small line. This asymmetrical response was attributed to the change of mothering ability under selection and not to the growth of the young themselves. Thus, a genetic correlation between body weight and maternal performance was suggested.

For an explanation of the asymmetrical response to selection, the author suggested a hypothesis based on Lerner's (1954) concept of genetic homeostasis. Based on this hypothesis, the maternal performance which was thought to be mainly a matter of milk yield, has two anatomical and physiological components. The anatomical component, represented by mammary gland size, should be directly related to body size. This component will increase continuously as body size increases in the large line and will decrease in the same way in the small line. In contrast, the physiological component should not be directly related to body size, but rather should be a component of natural fitness and shows overdominance as postulated by Lerner (1954). This component should then decline when body weight is changed by selection in either direction. Thus, the combined effect of the two components should be a very small change in the maternal effect when weight is increased but a marked reduction when weight is reduced.

Falconer (1958) has shown that the theory of genetic correlation may be applied to the problem of interaction between genotype and environment. This application makes it possible to estimate how much of the improvement gained by selection carried out in one environment will
be maintained if the improved strain is transferred to a different environment. The phenotypic measurement of any trait evaluated in two different environments may be regarded as measurements of two different "characters". The degree of genetic likeness between the two "characters", arising from pleiotropy, may be expressed as a genetic correlation.

An experiment with mice was constructed based upon this idea. The growth between three and six weeks of age was measured in two different environments made of high and low planes of nutrition. Two lines for each of upward and downward growth were selected, one reared on the high plane while the other on the low. The selections were made from the first generation litters, which were transferred to the other environment to rear the second generation litters. The genetic correlations estimated from a comparison of the direct response with the correlated response for the two characters agreed well when the calculations were based upon the divergence between the upward and downward selections. However, there was no agreement among the four estimates based upon the upward and downward responses separately. This discrepancy which was attributed to the asymmetry of the response in the two directions, is thought to be connected with maternal effects.

Falconer (1960a) studied some aspects of the genetics of litter size in mice under inbreeding and selection. Litter size is a character influenced by maternal effect. It is partly an attribute of the mother and partly an attribute of the members constituting the litter. Of the three surviving highly inbred lines, litter size was reduced whereas the body size was not. This suggested that the reduction of litter size brought
about an improved maternal environment which removed any decline of intrinsic growth that there may have been.

The daughter-dam correlation, which is influenced by maternal effects, was virtually zero. This suggested that the mothers having a large litter rear their daughters in a competitive environment which retards their growth, which in turn tends to reduce the size of their litters. This was an indication of maternal effect contributing negatively to the daughter-dam correlation which could counterbalance any positive genetic correlation that there may have been.

Selection was also practiced for increased and decreased litter size over 20 generations. Each generation consisted of ten full-sib families. Within each family, sisters were mated to the same male chosen at random, and the female with the best litter was selected. Such a within-family selection applied to females circumvented the negative maternal effect. This was because each group of females from which the selection was made was subjected to the same maternal environment.

DeFries and Touchberry (1961) studied the inheritance of body weight in Drosophila affinis. Body weight measurements were taken between the emergence time and 12 hours after it. A path coefficient diagram was used to investigate the relationships between weight of the male parent, weight of the female parent, and the number of offspring with the average weight of the offspring. The regression of the average weight of offspring on the weight of the male parent was found to be higher than that of the weight of the female parent. The paternal half-sib component of variance was also negative. These results indicated that a negative
maternal effect exists in the inheritance of body weight in Drosophila and that it operates through the number of offspring.

Dawson (1964a) examined the significance of maternal effects on the developmental rate in Tribolium. The results indicated that the proportion of variance attributable to maternal effects was approximately half as large as and equal to that due to heritability in Tribolium castaneum and Tribolium confusum, respectively. In a more extensive study, using five strains of beetles which were crossed in all possible combinations, maternal effects were most pronounced for early stages of development and diminished in importance with increasing age of offspring. Thus, it was suggested that there are differences in substances deposited in eggs among females. The advantageous utilization of these substances included in eggs by superior females occurred in the early developmental period. In the later stages, the progeny’s own genotype assumed a greater importance.

Many reports in the literature are concerned with the cross-fostering technique in litter bearing mammals to study the maternal influences of the dam on the body weight of the offspring. Cox et al. (1959) established groups of three unrelated litters, each consisting of at least six mice. The litter members of each group were divided among the three dams so that each dam kept two of her own and received two from each of the other two females in the group. An effort was made to determine the portion of the total variance of the different body weights due to the influences of prenatal and postnatal effects and their interaction.

The prenatal component includes variance due to the genetic differences between full-sib progenies (reflecting their own genotypes) and the
environmental variance (resulting from the differences in the uteri within which they developed). The postnatal component includes the variance due to the differences in the direct maternal effects of the dams (such as the genetic ability of a dam to produce milk) on the weight of the litters they nurse. Such influences are purely environmental as far as the young mice are concerned, but from the standpoint of the dam, they may be classified as both genetic and environmental. The prenatal by postnatal interaction was regarded as a genotype by environmental interaction.

The results of this study showed that the postnatal maternal influence was the most important factor in determining the weight through weaning. The postnatal effects were responsible for 71.5% of the total variance of the 12-day weight which suggested use of such weight as the measure of lactational performance of the dam. This result did not agree with the result indicated by Bateman (1954) who attributed only 32% of the variation to postnatal effects. Bateman's result had suggested that the 12-day weight should be regarded as an insensitive measure of maternal performance.

Cox and Willham (1962) reciprocally cross-fostered litters within two breeds of swine to explore the feasibility of a fostering scheme commonly practiced in smaller animals (mice). Six young pigs of each litter were identified and divided among the three sows in a set, so that each sow reared two of her own pigs and two from each of the two other females in the set. Each set was composed of three sows of the same breed, farrowing the same day, and with at least six live offspring. The weights at 21, 42, 98, and 154 days of age were taken on each individual pig.

The results indicated that prenatal effects arose from 6% of the total variance in weight at 21 days to 13% at 154 days. Postnatal influences accounted for over 20% of the variance in body weight at 21, 42, and 98 days.
and declined to 5% at 154 days. The design appeared to be also feasible for pigs.

Young et al. (1965) undertook a cross-fostering study similar to that of Cox et al. (1959) to assess the relative importance of prenatal and postnatal influences upon body weight and growth. Their main objective was to determine the usefulness of the 12-day weight of the suckling litters as an adequate measure of the lactational performance of the dam. Genetic and phenotypic relationships between different growth measurements and maternal characteristics were also examined.

The results of this study were in close agreement with those reported by Cox et al. (1959). The results of both investigations suggested that the postnatal maternal performance of the dam is by far the most important factor in determining the growth of the young mice in their suckling period. The genes of the young mice seem to have relatively small influence upon their preweaning growth.

Based on this study any one of the 12-day weight, 21-day weight (weaning), and gain from birth to weaning should be suitable for measuring the lactational performance of the dam. Prenatal influences had their largest effects on birth weight, accounting for 38% of the total variance in this trait, but were not important for any other trait. The interaction between prenatal and postnatal influences were unimportant for all traits studied. Postweaning weights and gains were expected to be considerably less influenced by the lactational performance of the dam. The results indicated that the postnatal effects were responsible for 22% and 16% of the total variance of 42 and 56-day weights, respectively, while 18% of the variance in both instances was due to prenatal effects. This showed that the postnatal influence of the dam has an important impact on the weights of the offspring until they near their
mature size. Postweaning gains were influenced more by prenatal than by the postnatal effects. There was indication that the lactational performance of the dam had little direct effect on the number of young born to her daughters and on the lactational performance of the young she nurses.

Young and Legates (1965), using the same data as the previous study, reported a positive genetic correlation between early gains and postnatal maternal performance. This relationship was negative when the later gains (from 42 to 56 days) were considered. With regard to these results, the authors suggested that the genetic association between protein anabolism and lactation is negative whereas between fattening and lactation it is positive. This suggestion was also based on Fowler's (1958) results which indicated that fat deposition in mice was primarily responsible for the gain made after 35 days of age and not prior to that time.

Maternal correlations (only the postnatal maternal effects were included) between preweaning and postweaning gains were negative. This negative relationship indicated that the young mice nursed by dams with good milking ability made reduced gains following weaning, whereas those nursed by poor milking dams tended to make an increased compensatory growth following weaning. The overall results of this study added information to the probable validity of the conclusion arrived at by Dickerson (1947).

White et al. (1968) conducted a reciprocal cross-fostering study on three lines of mice. Two of these lines had been subjected to long term within-family selection for high and low body weights measured at six weeks of age. Selection of this kind was practiced to avoid any direct selection for maternal environment. The third line was an unselected
control line. The cross-fostering technique used in this study followed one similar to that of Cox et al. (1959) and Young et al. (1965). This study was designed to investigate the magnitude and nature of line differences in prenatal and postnatal maternal influences upon growth and maternal ability.

The results of this study indicated that both prenatal and postnatal maternal effects were important in determining preweaning and postweaning growth of the three lines. An observed marked reduction in maternal performance of the low line confirmed the existence of an asymmetrical response to selection for six-week body weight as reported by Legates and Farthing (1962) and several other authors. The results also indicated that the postnatal maternal performance in the unselected line was superior to that in the line selected for high body weight. This superiority was attributed to several physiological and genetic mechanisms and their combinations. Three of the mechanisms discussed were genetic correlations between maternal effects and growth, inbreeding depression, and a hypothesis based upon Lerner's (1954) concept of genetic homeostasis.

Eisen et al. (1970) undertook a study to investigate selection response for increased 12-day litter weight in mice. The 12-day litter weight is a trait influenced by the maternal effect of the dam as well as by the genotype of the offspring itself. A mating scheme similar to one of the several suggested by Eisen (1967), for within-family selection, was designed to minimize the variation due to the genotypic effects of the suckling-young. This design included six lines each consisting of 15 full-sib families. Each family represented by four females and two
males. Four of the lines were subjected to a within-family selection whereas the remaining two were maintained as controls. Selection was based on choosing the litter (four females and two males) with the largest deviation in 12-day litter weight from the mean of each family. The four daughters from a selected litter were paired randomly with two full-sib males from another selected litter. Each male was mated to two of the daughters at random.

The genetic parameters were estimated from the results of the first ten generations of selection. These estimates expressed as the per cent of the total phenotypic variance were 22.2 for direct additive genetic variance \( \sigma^2_{A_C} \), 6.1 for maternal additive genetic variance \( \sigma^2_{A_m} \), 7.4 for direct-maternal additive genetic covariance \( \sigma_{A_mA_m} \), 50.1 for maternal environment variance \( \sigma^2_{o_m} \), and 14.2 for the random environmental variance \( \sigma^2_{e} \). Although the total postnatal maternal variance \( \sigma^2_{A_C} + \sigma^2_{o_m} \) accounted for 56.2% of the total phenotypic variance, only 10.8% of that was due to the genetic postnatal maternal influences. The genetic correlation between number of young born and the 12-day litter weight was .19.

2. Non-designed experiments:

There are numerous reports in this category but only a few were chosen. These chosen reports are not based on any priority in methods, techniques, etc. They were only chosen to indicate the importance of maternal effects on different traits of economic importance in farm animals.
King (1961) analyzed data collected over a two-year period from the pullets of 50 males, each mated to five females. By use of a sire shifting procedure, each male was mated to a total of ten females (five in each shift) and each female to two cockerels (one at a time in each shift). The data were analyzed using two different statistical models. The first model included sire, dam, and sire x dam interaction effects whereas the second model contained sires and dams within sires effects. The results of this study are summarized in Table 1. The maternal

Table 1. Summary of the results obtained by King (1961)

<table>
<thead>
<tr>
<th>Trait</th>
<th>( h^2_s )</th>
<th>( h^2_d:s )</th>
<th>Maternal effects</th>
<th>( h^2_{sd} )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age at first egg</td>
<td>.26</td>
<td>.57</td>
<td>7.7%</td>
<td>.04</td>
</tr>
<tr>
<td>32 week egg weight</td>
<td>.60</td>
<td>.73</td>
<td>3.1%</td>
<td>.24</td>
</tr>
<tr>
<td>32 week body weight</td>
<td>.62</td>
<td>.74</td>
<td>3.2%</td>
<td>.10</td>
</tr>
<tr>
<td>% egg production to Jan. 1</td>
<td>.06</td>
<td>.43</td>
<td>9.3%</td>
<td>.36</td>
</tr>
<tr>
<td>% egg production to 72 weeks</td>
<td>.16</td>
<td>.64</td>
<td>12.0%</td>
<td>.36</td>
</tr>
<tr>
<td>USDA albumen score</td>
<td>.10</td>
<td>.71</td>
<td>15.2%</td>
<td>.08</td>
</tr>
</tbody>
</table>

\( a \) and \( d \) denote sire and dam, respectively.

The heritability estimates utilizing the dam component of variance (\( h^2_{d:s} \)) were in every instance larger than the estimates from the sire com-
ponent \( h_s^2 \). The \( h_d^2 \) was thought to be inflated either by maternal effects, sire by dam interaction, or both. Even after the sire by dam interaction was separated as shown in Table 1, the results indicated the maternal effects were present for all traits studied.

The genetic correlations \( r_s, r_{s:d}, \) and \( r_{s:d} \) were not consistent between years, and in many instances they exceeded the range of -1 to +1 (e.g. -2.05 and 1.73). A negative genetic correlation was observed between egg weight and egg production and between egg production and albumen quality.

McCartney and Chamberlin (1961) analyzed data obtained from nine strains of turkeys, representing three varieties; Bronze, Large White, and Small White. Various systems of matings involving pure strains, variety crosses, backcrosses, and three-way crosses were utilized to determine the importance of general and specific combining ability and maternal and reciprocal effects on several economically important traits. The results of this study as the percentage of total variance is shown in Table 2. The results of this study clearly indicated that the maternal effect is by far more influential on fertility and hatchability than on either general and specific combining ability.

Dickinson et al. (1962) conducted two experiments involving the transfer of fertilized eggs from one breed of sheep to another. The first experiment included the reciprocal transfer of eggs between ewes of the large Lincoln breed and of the small Welsh Mountain breed. In the second experiment, eggs from two breeds of donor were transferred to one breed
of recipient (Scottish Blackface). This study aimed to investigate the influence of maternal and genetic factors on the size of lambs at birth and on their gestation length.

Table 2. Results of the study conducted by McCartney and Chamberlin

<table>
<thead>
<tr>
<th>Effects</th>
<th>Fertility</th>
<th>Hatchability</th>
<th>Poultry production</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Fert. eggs/All eggs</td>
<td></td>
<td></td>
</tr>
<tr>
<td>General</td>
<td>1.7</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Specific</td>
<td>0</td>
<td>1.2</td>
<td>4.1</td>
</tr>
<tr>
<td>Maternal</td>
<td>11.5</td>
<td>16.4</td>
<td>18.1</td>
</tr>
<tr>
<td>Reciprocal</td>
<td>11.4</td>
<td>0</td>
<td>13.3</td>
</tr>
<tr>
<td>Sampling error</td>
<td>75.4</td>
<td>82.4</td>
<td>77.5</td>
</tr>
</tbody>
</table>

The covariance between the size of the lambs at birth and the weight of the donor (mature) was regarded as genetic, whereas it was considered maternal with the recipient. The correlations of birth weight with the donor's weight and with the recipient's weight were .09 and .35, respectively. The corresponding correlations for cannon length were .23 and .31, respectively. The lamb's genotype and the maternal environment provided by the dam for the growth of the embryo accounted for 72% and 20% of the variation in birth weight, respectively. The corresponding values for cannon length were 97% and 1%, respectively.

Everett and Magee (1965) undertook a study to investigate maternal ability and genetic ability of birth weight and gestation length of
Holstein calves. Grand-offspring of paternal grand-sires, paternal half-sibs, grand-offspring of maternal grandsires, and maternal and paternal grand-offspring of a grandsire were utilized to estimate genetic parameters as computed by Willham (1964). The results obtained from this study indicated that the sire components of variance for both traits were larger than the corresponding dam components (zero). The genetic correlation between genetic ability and maternal ability of both traits was -.93.

Hill et al. (1966) undertook a study to determine the relative importance of the calf's genotype for weight (180-day) and the dam's genotype for maternal effects on calf weight. The covariances between paternal and maternal half-sibs, one-quarter sibs and offspring-dam were utilized to estimate genetic parameters. Dominance deviations, epistatic deviations, and non-maternal environmental correlations were assumed to be negligible. The additive genetic variance for weight and maternal effects and the genetic covariance between weight and maternal effects were estimated to be 100, 91, and -30, respectively. Thus, there was an almost equal contribution of the genotype of the calf for weight and the genotype of his dam for maternal effects on the 180-day weight. The genetic association between the two was negative.

The covariances among first-lactation milk records expressed as deviations from herdmate averages of Holstein cows were examined by Van Vleck and Hart (1966) to determine the importance of genetic maternal effects. Four mating patterns which yielded cousins of varying degree, daughter-dam, full-and maternal sibs, and aunt-nieces of varying degree
were utilized to estimate six genetic parameters. The results indicated that the additive genetic variance, accounting for 38% of the total variance, was the only important genetic parameter for the first lactation milk production. Earlier analyses had resulted in heritability estimates of .44 and .25 from daughter-dam regression and paternal half-sibs correlations. The difference between the two estimates was not accounted for by either genetic maternal effects or environmental covariances between records. It was suggested to be statistical in nature.

Deese and Koger (1967) analyzed weaning data from 725 purebred Brahman calves and 466 Brahman-Shorthorn crossbred calves. The estimated components, expressed as a per cent of total phenotypic variance, are shown in Table 3.

Table 3. Results of the study conducted by Deese and Koger (1967)

<table>
<thead>
<tr>
<th></th>
<th>$\sigma^2_A$</th>
<th>$\sigma^2_M$</th>
<th>$\sigma^2_D$</th>
<th>$\sigma^2_C$</th>
<th>$\sigma^2_E$</th>
<th>$\sigma^2_{M,M}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brahman herd</td>
<td>18</td>
<td>15</td>
<td>0</td>
<td>*</td>
<td>0</td>
<td>8</td>
</tr>
<tr>
<td>Crossbred herd</td>
<td>40</td>
<td>46</td>
<td>-30</td>
<td>*</td>
<td>7</td>
<td>38</td>
</tr>
</tbody>
</table>

$a^A$ and $D$ indicate additive and dominance deviation, respectively, for growth (N) and maternal (M). The starred component was originally assumed to be equal to zero in order to solve 6 equations with 8 unknowns.

$b^{\sigma^2_{E,E}} = \text{variance of permanent environmental influences on maternal effects, }^{\sigma^2_{E,E}} = \text{variance of non-permanent environmental effects.}$
The heritability estimate composed of both maternal and non-maternal effects and their covariance was .25 for the Brahman and .17 for the crossbred. $\sigma_A^2$ and $\sigma_M^2$ values are heritability estimates for growth and maternal effect, respectively.

Brown and Galvez (1969) undertook a study based on birth weight records of 789 Hereford and 932 Angus calves to evaluate maternal and non-maternal influences on birth weight. The estimates of the components obtained from this study expressed as a per cent of the total variance are shown in Table 4. A negative value for $\sigma_{ANM}^2$ indicates an antagonism.

Table 4. Results of the study conducted by Brown and Galvez (1969)

<table>
<thead>
<tr>
<th></th>
<th>$\sigma_A^2$</th>
<th>$\sigma_M^2$</th>
<th>$\sigma_{AN}^2$</th>
<th>$\sigma_{DM}^2$</th>
<th>$\sigma_{DN}^2$</th>
<th>$\sigma_{NM}^2$</th>
<th>$\sigma_{EC}^2$</th>
<th>$\sigma_E^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hereford</td>
<td>56</td>
<td>30</td>
<td>-24</td>
<td>-15</td>
<td>17</td>
<td>*</td>
<td>*</td>
<td>35</td>
</tr>
<tr>
<td>Angus</td>
<td>14</td>
<td>25</td>
<td>-7</td>
<td>-16</td>
<td>9</td>
<td>*</td>
<td>*</td>
<td>75</td>
</tr>
</tbody>
</table>

$^a$A and D indicate additive and dominance deviation for growth (N) and maternal (M) components of a character, respectively. The starred component was originally omitted in order to solve 6 equations containing 8 unknowns.

$^b$ $\sigma_{EC}^2$ = variance of permanent environmental influences on maternal effects. $\sigma_E^2$ = variance of non-permanent environmental effects.

between the genes for prenatal growth and the genes conditioning the intra-uterine environment for heavier weights at birth. The heritability estimate based on the total genetic contribution (maternal and non-maternal) was .36 in Hereford and .17 in Angus.
This section does not summarize all the literature reviewed. It attempts to relate these reports to show what problems are involved, what solutions are available, and what is the present status of the problem.

The development of a maternally influenced trait is under the control of at least two genetic components. These are the direct genetic effects of the individual and the maternal genetic effect of his dam on that trait. The influence of the dam on the phenotype of her offspring is solely environmental relative to the offspring, but it is composed of both genetic and environmental components with respect to the dam. The genetic maternal effect differs from its environmental portion in that genotypic differences among dams will also be expressed in the female progeny becoming future dams or in the daughters of males (Willham, 1963). Thus, the phenotypic value ($P_x$) of an individual for a trait influenced by a maternal effect of a related individual ($w$) may be shown as $P_x = G_{ox} + E_{ox} + G_{mw} + E_{mw}$ (Willham, 1963) where $G$ and $E$ indicate genotypic and environmental values, respectively. The subscript $(o)$ indicates a character expressed in offspring ($x$) under the influence of a component character ($m$) expressed in a related individual ($dam$).

The variance ($V$) of such a measurement is $V(P_x) = V(G_{ox}) + V(E_{ox}) + V(G_{mw}) + V(E_{mw}) + 2Cov(G_{ox},G_{mx}) + 2Cov(E_{ox},E_{mx})$ in the absence of genotype by environmental interactions and any correlation between genotypes and environmental deviations. Willham (1963) expressed the genotypic covariances between relatives in terms of these variances and covariances. To show the nature of the problem, suppose it is hypotheti-
cally assumed that the genotypic and environmental values of an individu-
ual are independent from the genotypic and environmental values of the
related individual and that the phenotypic expression ($P^m$) of such ma-
ternal influences is directly measureable. Now the modified equations
are as follows:

$$P_x = G_{ox} + E_{ox}$$

$$P_m = G_{mw} + E_{mw}$$

$$V(P_x) = V(G_{ox}) + V(E_{ox})$$

$$V(P_m) = V(G_{mw}) + V(E_{mw})$$

In this case the problem rests only on separating heredity variance from
the environmental variance. The solution to this problem has been avail-
able at least as early as 1918 by Fisher. Other authors, such as Wright
(1920), Lush (1940), Baker et al. (1943), Hazel et al. (1943), and Lush
(1949) developed solutions of this kind.

Obviously the problem resides on evaluating $\text{Cov}(G_{ox}, G_{mw})$ and
$\text{Cov}(E_{ox}, E_{mw})$ brought about by the dependence of the offspring on the dam.

The evaluation and separation of these two covariances from each other and
from other sources of variation are complicated since such an influence is
totally environmental on the offspring and is not directly measurable on
the dam. The genotypic covariance, $\text{Cov}(G_{ox}, G_{mw})$, is a function of $\text{Cov}(A_o,A_m)$
and $\text{Cov}(D_o,D_m)$ where A and D indicate additive and dominance genetic
effects, respectively (for a further breakdown of this covariance,
see Willham (1963)). The two covariance terms, $\text{Cov}(E_{ox}, E_{mw})$ and
Cov(D D ), are unlikely to be important but their presence will bias estimates of other parameters.

The separation of Cov(A A ) from other sources of variation and the evaluation of its magnitude and direction have become goals of many research efforts. Dickerson (1947) and Dickerson and Grimes (1947), in analyzing swine data, speculated that this covariance may be negative. Due to the importance of this covariance, Dickerson (1947) redefined heritability as the regression of transmitting ability on individual performance. Cockerham (1954) suggested that the dam-offspring, sire-offspring, and paternal half-sib relationships may be utilized to evaluate this covariance. Koch and Clark (1955) took the initiative to evaluate it in beef cattle and even succeeded in determining a range of possible values for it. Kempthorne (1955) theoretically examined the consequences on the correlation between relatives when a maternal effect was involved and pointed out that the situation cannot be understood from the sire-offspring, dam-offspring, and full-sib relationships. Willham (1963) developed a general formula for the genotypic covariance between relatives and theoretically examined the application of evaluating and separating this covariance from other sources of variation in the absence of epistatic effects. Willham (1964) furthermore examined the practical aspect of the genotypic covariance between relatives and suggested that the grandchildren of a set of bulls by way of his son and by way of his daughter may be utilized to evaluate this covariance. This author also computed the theoretical expectations for the necessary genotypic covariances. Falconer (1965) showed that the discrepancy between heritability esti-
mates from a daughter-dam regression, full-sib correlation, and response to selection may be accounted for if the maternal effects are considered. Koch (1969) compared the offspring-dam correlation with the same correlation done on an intra-granddam basis. This technique provided a means of evaluating the influence of the dam's environment on the phenotype of the offspring. The results also confirmed that this covariance may be negative.

Although examining earlier literature reports in order to recognize a particular investigator or a group of authors for priority in this subject is not within the scope of this study, it is fair enough to conclude that many publications have contributed to this area of study. Certainly there are many other authors who have directly or indirectly contributed, but time and space prohibit citing them. Meanwhile it should be pointed out that the works of the authors in the results section have also greatly contributed to a clarification of the situation. These authors have approached the problem by different methods and techniques (e.g. cross-fostering, ova transplantation, reciprocal differences, etc.); by using different organisms (e.g. mice, Drosophila, Tribolium, beef cattle, etc.); by designing experiments (e.g. Eisen (1967)); by utilizing different relationships (full-sibs, half-sibs, etc.); by making different assumptions (e.g. no dominance effect, no epistatic effect, no certain interaction effect, etc.); and other differences. But as yet there is no certainty and agreement in the magnitude and direction of $\text{Cov}(A^o, A^m)$ for any particular trait of economic importance.

The uncertainty of the estimates of the $\text{Cov}(A^o, A^m)$ and other genetic
parameters of interest (e.g. $\sigma^2_{A_m}$ and $\sigma^2_{D_m}$) is a result of the following problems:

1. Estimates have relatively high sampling error.
2. Estimates may not be free of environmental correlations.
3. Estimates are based on correlations where the genetic relationship between individuals is small.
4. In most cases estimates are not unbiased in the sense that they are not separated from other genetic and environmental parameters (e.g. epistasis).
5. Other problems that may exist with respect to the type of designs, measurement errors, etc.

To combat these interfering factors, a pertinent design with an adequate number of observations and suitable to the estimation of a certain or a group of those parameters of interest should be utilized. Although this is the most reliable approach to this complicated problem, one can not be sure that all the interfering factors have been eliminated. Perhaps the maternal ability of the dam is also correlated with the maternal ability of the granddam or other relationships are involved which can not be easily separated.
Obtaining reliable estimates of the genetic parameters largely depends on the sample size and its composition (e.g., genetic structure and size of the family). An increase in the number of observations sometimes cannot be easily done with the population of interest. This is especially true in working with cattle and other large animals because of the long life cycle, small numbers of offspring, high handling costs, management problems, and lack of confinement area. In such cases, the investigators have the opportunity to choose other experimentally suited organisms which may serve the purpose without losing the implications of the results. These alternatives could be a computer simulation or a pilot organism (e.g., Drosophila, Tribolium, mice, etc.). Kojima and Kelleher (1963) and Robertson (1967) have extensively discussed the use of laboratory animals in selection studies.

For the purpose of this study, the flour beetle *Tribolium castaneum* was chosen. This pilot organism has been used extensively for laboratory studies in ecology, physiology, genetics, and to some extent animal breeding. The usefulness of this genetic material for research projects has been reported by many authors (e.g., Bell 1968; Lerner and Ho 1961; and Dawson 1968). Several expedient characteristics of this organism are short generation cycle, high reproductive rate, polygamous mating habits, small body size, ease of maintenance and handling, and known previous selection history. These considerations will become highly important if an investigation is to be carried out over several generations.
Maternal effects in Tribolium are due to the dam's transmitted materials and nutriments passed through the eggs to provide a developmental environment for her progeny. These materials and nutriments might vary in both quality and quantity. In mammals, maternal influences are both prenatal and postnatal since the development of the embryo takes place inside the body of the mother. But in Tribolium such influences are based strictly on whatever is included in the eggs which develop into larvae outside the body of the mother. Although the chosen laboratory organism and farm animals follow different reproductive patterns, the principle of the mother's influence on early environment of the offspring which in turn may affect other stages of life remains the same. Thus, the application of the proposed designs in the evaluation of the direction and magnitude of the direct-maternal genetic correlation, the main interest of this study, should be similar in both cases.

Three designs were planned and carried out simultaneously. Design 1 included 331 random sires each mated to two random dams from which one male and one female of each family were measured. The first generation offspring (for convenience called $F_1$) from design 1 were allowed to mate and yield second generation progeny (for convenience called $F_2$) following two different patterns which constituted designs 2 and 3. Thus, the sires and dams used in design 1 became grandsires (GS) and granddams (GD) for designs 2 and 3. The $F_1$ offspring from 208 of these grandsires were two paternal half-sibs of different sexes each mated to a random mate. One male and one female of each $F_2$ family were measured. Information obtained from these 208 grandsires' progeny formed design 2. Design 3
which included the progeny of the remaining 123 grandsires differed from design 2 only in that the F1 individuals were two paternal half-sibs of the same sex (females). The schematic structures of the three designs are illustrated in Figures 2, 3, and 4.

\[ \text{Figure 2. Schematic structure of design 1 for each sire (331 sires)} \]
Each 0 represents one offspring.

$^a$F (female) and M (male) are the two random mates chosen for $S_1$ and $D_2$, respectively.

$^b$Each 0 represents one offspring.

Figure 3. Schematic structure of design 2 for each GS (208GS)
a$M_1$ and $M_2$ are two random mates (males) chosen for $D_1$ and $D_2$, respectively.

Figure 4. Schematic structure of design 3 for each GS (123 remaining grandsires not used in design 2)
A general formula for the genotypic covariance between relatives x and y, each being maternally influenced respectively by w and z, is given by Willham (1963) as follows:

\[
\text{Cov}(P_x, P_y) = 2p_{xy} \sigma_{A_o}^2 + U_{xy} \sigma_{D_o}^2 + (2p_{xz} + 2p_{wy}) \sigma_{A_m} + \\
(U_{xz} + U_{wy}) \sigma_{D_o} D_m + 2p_{xz} \sigma_{A_m}^2 + U_{wz} \sigma_{D_m}^2 + \sum_{r,s} (2p_{xy})^r (U_{xy})^s \sigma_{A_D}^2 (r,s) + \\
\sum_{r,s} (2p_{xz})^r (U_{xz})^s \sigma_{A_D}^2 (r,s) + \sum_{r,s} (2p_{wy})^r (U_{wy})^s \sigma_{A_D}^2 (r,s) + \\
\sum_{r,s} (2p_{wz})^r (U_{wz})^s \sigma_{A_D}^2 (r,s). \quad 2 \leq r + s \leq N
\]

\(P_x\) and \(P_y\) represent the phenotypic values of x and y, respectively.

The coefficients \(2p_{xy}\), \(2p_{xz}\), \(2p_{wy}\), and \(2p_{wz}\) are Wright's coefficients of relationship with no inbreeding, or twice Malecot's coefficients of parentage (i.e. \(p_{xy}\) is the probability that a random gene from individual x is identical by descent with a random gene at the same locus in individual y). The coefficients \(U_{xy}\), \(U_{xz}\), \(U_{wy}\), and \(U_{wz}\) are expressed in probability forms (i.e. \(U_{xy}\) is defined as the probability that the two genes at a locus in individual x are identical by descent with two genes at that locus in individual y). \(A\), \(D\), and \(A_D\) represent additive, dominance, and epistatic effects for direct (o) and maternal (m) components of a character, respectively. Of the total number of loci (N) which are segregating, \(r\) loci with additive effects interact with \(s\) loci having dominance effects.

In the absence of epistasis this covariance is simplified as follows:
\[
\text{Cov}(P^x, P^y) = 2p_{xy} \sigma^2_{A_c} + U_{xy} \sigma^2_{D_o} + (2p_{xz} + 2p_{wy}) \sigma^2_{A_o A_m} + (U_{xz} + U_{wy}) \sigma^2_{D_o D_m} + 2p_{wz} \sigma^2_{A_m} + U_{wz} \sigma^2_{D_m}.
\]

A primary task is to determine coefficients of different variance and covariance components included in Cov \((P^x, P^y)\). To do this, members of design 2 and design 3 are listed in chronological order of oldest to youngest, left to right. This forms a square, symmetrical array (Table 5 and Table 6). Parents of each pedigree member (if known) are listed above the individual itself. In the absence of inbreeding, diagonal elements are equal to one. The off-diagonal elements are Wright's coefficient of relationship between individuals represented by a row and a column. The off-diagonal values are zero when two individuals have non-listed parents (individuals with non-listed parents should be totally unrelated and products of random mating). If the parents of a member are listed in a column, then the sum of the parents in the same row will be halved. For instance, the relationship between GS and \(S_1\) (Table 5) is obtained by halving the sum of the relationships of GS with GS and GS with GD or \(1/2(1 + 0) = 1/2\).

The "U" coefficients in the absence of inbreeding can be obtained as follows:

\[
U_{xx} = U_{yy} = 1
\]

\[
U_{xy} = 1/4 \left[ R_{xy} R_{xy} + R_{xy} R_{xy} \right], \text{ where the R's represent the relationships between two related individuals as subscripted. S and D indicate sire and dam of the subscripted individuals.}
\]
Table 5. Relationship between pedigree members of design 2 ($2p_{ij}$)

<table>
<thead>
<tr>
<th></th>
<th>GS-GD₁</th>
<th>GS-GD₁</th>
<th>GS-GD₂</th>
<th>GS-GD₂</th>
<th>S₁-F</th>
<th>S₁-F</th>
<th>M-D₂</th>
<th>M-D₂</th>
</tr>
</thead>
<tbody>
<tr>
<td>GS</td>
<td>1</td>
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<td>0</td>
<td>1/2</td>
<td>1/2</td>
<td>1/2</td>
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<tr>
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<td>1/2</td>
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<tr>
<td>GD₂</td>
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<td>0</td>
<td>0</td>
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<td>1/2</td>
<td>0</td>
</tr>
<tr>
<td>S₁</td>
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<td>1/2</td>
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<td>1/2</td>
<td>1/4</td>
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<td>1/2</td>
<td>1</td>
<td>1/4</td>
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<td>0</td>
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<tr>
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<td>0</td>
<td>1/2</td>
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<td>1/4</td>
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<td>0</td>
</tr>
<tr>
<td>D₂</td>
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<td>1/2</td>
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</tr>
<tr>
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<td>0</td>
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<td>1/2</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

Note: The table values represent genetic contributions and relationships based on the design of the experiment.
Table 6. Relationship between pedigree members of design 3 ($2p_{ij}$)

<table>
<thead>
<tr>
<th></th>
<th>GS-GD₁</th>
<th>GS-GD₁</th>
<th>GS-GD₂</th>
<th>GS-GD₂</th>
<th>M₁-D₁</th>
<th>M₁-D₁</th>
<th>M₂-D₂</th>
<th>M₂-D₂</th>
</tr>
</thead>
<tbody>
<tr>
<td>GS</td>
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<td>1/2</td>
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<tr>
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<td>1</td>
<td>0</td>
<td>1/2</td>
<td>1/2</td>
<td>0</td>
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</tr>
<tr>
<td>GD₂</td>
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<td>0</td>
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<td>1/2</td>
<td>1/2</td>
<td>0</td>
</tr>
<tr>
<td>S₁</td>
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<td>1/4</td>
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</tr>
<tr>
<td>S₂</td>
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<td>1/2</td>
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<td>1/4</td>
<td>1</td>
<td>1/2</td>
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<tr>
<td>D₂</td>
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<td>0</td>
<td>1/2</td>
<td>1/4</td>
<td>1/4</td>
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<td>0</td>
</tr>
<tr>
<td>M₁</td>
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<td>0</td>
<td>0</td>
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<td>0</td>
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<td>1/2</td>
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<td>0</td>
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<td>1/8</td>
<td>1/8</td>
<td>1/2</td>
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<tr>
<td>0₇</td>
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<tr>
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<td>1/8</td>
<td>1/4</td>
<td>1/2</td>
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</tr>
</tbody>
</table>
With these simplifications, the coefficients of the components of the covariances between relatives can easily be determined.

Example: Cov(O_1, O_3) of design 2 using Table 5 is as follows:

\[ 0_1 = x, 0_3 = y, F = w, D_2 = z \]

\[ 2p_{xy} = 2p_{0_1,0_3} = 1/16, \ U_{xy} = \ U_{0_1,0_3} = 1/4 \left[ R_{S_1} S_3 D_0 \right] + \]

\[ R_{S_1} D_0^2 \]

\[ (1/4) [(0)(0)] = 0, \ 2p_{xy} = 2p_{0_1,0_3} = 1/8, \ 2p_{wy} = 2p_{0_3,w} = 0 \]

\[ U_{xy} = U_{0_1,D_2} = 1/4 \left[ R_{S_1} S_0 D_0^2 \right] + \]

\[ R_{S_1} D_0^2 \]

\[ 1/4 \left[ R_{S_1} S_0 D_0^2 \right] = 1/4 \left[ (1/2)(0) + (0)(0) \right] = 0 \]

\[ U_{wy} = U_{0_3,F} = 0 \] since \( O_3 \) and \( F \) are unrelated.

\[ U_{zw} = U_{F,D_2} = 0, \ 2p_{zw} = 2p_{F,D_2} = 0. \] Thus using the formula (1) the

\[ \text{Cov}(O_1,0_3) = 1/16 \sigma^2_{A_{o}} + 1/8 \sigma^2_{A_{o,m}}. \]

By following the same procedure as in the above example, other genotypic covariances between different relatives are computed as follows:

Design 2:

1. \( \text{Cov}(O_1,S_1) = 1/2 \sigma^2_{A_{o}} + 1/4 \sigma^2_{A_{o,m}} \)

2. \( \text{Cov}(O_1,D_1) = 1/4 \sigma^2_{A_{o}} + 1/4 \sigma^2_{A_{o,m}} \)

3. \( \text{Cov}(O_1,D_2) = 1/8 \sigma^2_{A_{o}} \)
4. Cov(0, S) = $1/8 \sigma_{A_o}^2$

5. Cov(0, D) = $1/2 \sigma_{A_o}^2 + 5/4 \sigma_{A_{om}}^2 + 1/2 \sigma_{D_{om}}^2$

6. Cov(0, S) = $1/4 \sigma_{A_o}^2 + 3/4 \sigma_{A_{om}}^2 + 1/2 \sigma_{D_{om}}^2$

7. Cov(0, D) = $1/8 \sigma_{A_o}^2 + 1/4 \sigma_{A_{om}}^2$

8. Cov(0, D) = $1/8 \sigma_{A_o}^2 + 1/4 \sigma_{A_{om}}^2$

Design 3:

1. Cov(0, D) = $1/2 \sigma_{A_o}^2 + 5/4 \sigma_{A_{om}}^2 + 1/2 \sigma_{D_{om}}^2$

2. Cov(0, S) = $1/4 \sigma_{A_o}^2 + 3/4 \sigma_{A_{om}}^2 + 1/2 \sigma_{D_{om}}^2$

3. Cov(0, D) = $1/8 \sigma_{A_o}^2 + 1/4 \sigma_{A_{om}}^2$

4. Cov(0, S) = $1/8 \sigma_{A_o}^2 + 1/4 \sigma_{A_{om}}^2$

5. Cov(0, D) = $1/2 \sigma_{A_o}^2 + 5/4 \sigma_{A_{om}}^2 + 1/2 \sigma_{D_{om}}^2$

6. Cov(0, S) = $1/4 \sigma_{A_o}^2 + 3/4 \sigma_{A_{om}}^2 + 1/2 \sigma_{D_{om}}^2$

7. Cov(0, D) = $1/3 \sigma_{A_o}^2 + 1/4 \sigma_{A_{om}}^2$

8. Cov(0, S) = $1/8 \sigma_{A_o}^2 + 1/4 \sigma_{A_{om}}^2$

9. Cov(0, S) = $1/16 \sigma_{A_o}^2 + 1/4 \sigma_{A_{om}}^2 + 1/4 \sigma_{D_{om}}^2$
Although only one member of each set of $F_2$ full-sibs in designs 2 and 3 was utilized in the covariances computed above, the relationships will remain identical for the other members. Design 1, which is partitioned into designs 2 and 3, was planned to result in a reliable estimate of $\sigma^2_{A_o}$. The sire component of variance ($\sigma^2_{S}$) in this design has the expectation of $1/4 \sigma^2_{A_o}$, since it estimates the covariance between paternal half-sibs. The expectation of the grandsire component of variance ($\sigma^2_{GS}$) in design 2 is $1/16 \sigma^2_{A_o} + 1/8 \sigma^2_{A_o A_m}$. This component measures the covariance between the offspring of a grandsire by way of his son ($S_1$) and by way of his daughter ($D_1$). The covariance of this kind (e.g. $\text{Cov}(0_1, 0_3)$) was shown to be $1/16 \sigma^2_{A_o} + 1/8 \sigma^2_{A_o A_m}$. The grandsire component of variance in design 3 ($\sigma^2_{GS}$) estimates the covariance between the offspring of a grandsire by way of his two daughters ($D_1$ and $D_2$). This covariance is also shown to have the expectation of $1/16 \sigma^2_{A_o} + 1/4 \sigma^2_{A_o A_m} + 1/4 \sigma^2_{A_m}$ (e.g. $\text{Cov}(0_5, 0_7)$). Thus, the solution to these three equations with three unknowns should provide means of estimating the direct-maternal genetic correlation. Willham (1964) has also computed these expectations and discussed the approach to this problem.

Other covariance terms computed from designs 2 and 3 should provide additional information on dominance, epistasis, and environmental correlations. They also may be utilized to estimate the three above mentioned unknowns within designs 2 and 3.
DESCRIPTION OF DATA

The description of the foundation pearl stock, laboratory conditions, and the nutritional handling used in this investigation have been explained in detail by Moore (1969). The stock was established in 1960 at Purdue University from the systematic crossing of four unrelated laboratory stocks, one of which was homozygous for the recessive autosomal eye color mutation pearl, p. Beyond the four-way cross, this stock was reproduced as Purdue +, except the pearl mutation was selected to provide a genetic marker. Also, this marker can be easily used to detect any contamination by eggs in the media since the pearl eye-color will not be present. A sample of this stock was obtained by Iowa State University in 1966.

The present data were collected from three designs which were carried out simultaneously during six periods. Six periods were necessary as time and available assistance limited the number of pupae that could be sexed and weighed in one period on the 19th day.

The numbers of \( F_1 \) and \( F_2 \) larvae and pupae obtained from the three designs are classified in Table 7. The number of grandsires which contributed to the three designs in each period and those which left sufficient progeny of at least two offspring of different sex from each mating in \( F_1 \) and \( F_2 \) are shown in Table 8. The percentages of grandsires not leaving sufficient progeny in \( F_1 \) and \( F_2 \) were 24.8%, 25.5%,
Table 7. Numbers of larvae and pupae obtained from the three designs in $F_1$ and $F_2$

<table>
<thead>
<tr>
<th>Design number</th>
<th>$F_1$ Total</th>
<th>Pupae number</th>
<th>Larvae number</th>
<th>$F_2$ Total</th>
<th>Pupae number</th>
<th>Larvae number</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>11745</td>
<td>11615</td>
<td>130</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2</td>
<td>7366</td>
<td>7285</td>
<td>81</td>
<td>8271</td>
<td>8255</td>
<td>46</td>
</tr>
<tr>
<td>3</td>
<td>4379</td>
<td>4330</td>
<td>49</td>
<td>5366</td>
<td>5320</td>
<td>46</td>
</tr>
</tbody>
</table>

Table 8. Distribution of CS at the start and completion of the test over six periods

<table>
<thead>
<tr>
<th>Period</th>
<th>Design 1 Start</th>
<th>Design 1 End</th>
<th>Design 2 Start</th>
<th>Design 2 End</th>
<th>Design 3 Start</th>
<th>Design 3 End</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>60</td>
<td>38</td>
<td>40</td>
<td>25</td>
<td>20</td>
<td>13</td>
</tr>
<tr>
<td>2</td>
<td>60</td>
<td>33</td>
<td>40</td>
<td>21</td>
<td>20</td>
<td>12</td>
</tr>
<tr>
<td>3</td>
<td>100</td>
<td>78</td>
<td>60</td>
<td>45</td>
<td>40</td>
<td>33</td>
</tr>
<tr>
<td>4</td>
<td>100</td>
<td>78</td>
<td>60</td>
<td>46</td>
<td>40</td>
<td>32</td>
</tr>
<tr>
<td>5</td>
<td>60</td>
<td>53</td>
<td>40</td>
<td>36</td>
<td>20</td>
<td>17</td>
</tr>
<tr>
<td>6</td>
<td>60</td>
<td>51</td>
<td>40</td>
<td>35</td>
<td>20</td>
<td>16</td>
</tr>
<tr>
<td>Total</td>
<td>440</td>
<td>331</td>
<td>280</td>
<td>208</td>
<td>160</td>
<td>123</td>
</tr>
</tbody>
</table>
and 23.1% in designs 1, 2, and 3 respectively. From 109 prospective grandsires excluded from design 1 only 21 had no progeny. The respective numbers for designs 2 and 3 were 14 unsuccessful matings from 72 and 7 from 37. Although the remaining grandsires had some offspring in either generation or both, they did not reach the limit required and were dropped from the test.

The number of bottles containing larvae and pupae and the average number of larvae and pupae in those bottles are shown in Table 9. Only one bottle contained one adult (male) throughout the entire test. This may have resulted from accidental transfer of an egg adhering to the body of the female or from an exceptionally rapid development of the observed individual.

The generation interval was chosen to be 30 days. Pupa weighing in both generations was done on day 19 counting from the day of removing the females from the bottles after a 24-hour period of egg-laying. Extra caution was taken in removing the adults to not carry eggs from one bottle to another. For this purpose each spoon was sterilized with alcohol and dried over a vacuum before reusing. An effort was also made to keep the temperature and relative humidity as constant as possible. This obviously was important since the developmental rate was dependent on the environmental conditions. Matings were always made in the pupal stage since the collection of virgin females is most convenient in this stage (Dawson, 1964b). Details of the procedure followed in the laboratory throughout the course of this study are outlined in Table 10.
Table 9. Distribution of bottles containing larvae and pupae from the three designs

<table>
<thead>
<tr>
<th>Design Number</th>
<th>No. of bottles containing larvae</th>
<th>Average No. of larvae per bottle</th>
<th>F1 Larvae</th>
<th>F2 Larvae</th>
<th>F1 Pupae</th>
<th>F2 Pupae</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>98</td>
<td>1.3</td>
<td>-</td>
<td>-</td>
<td>662</td>
<td>17.5</td>
</tr>
<tr>
<td>2</td>
<td>58</td>
<td>1.4</td>
<td>38</td>
<td>1.2</td>
<td>416</td>
<td>17.5</td>
</tr>
<tr>
<td>3</td>
<td>40</td>
<td>1.2</td>
<td>28</td>
<td>1.6</td>
<td>246</td>
<td>17.6</td>
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</tbody>
</table>
Table 10. Outline of the laboratory work schedule

<table>
<thead>
<tr>
<th>Date</th>
<th>Weekday Period</th>
<th>Item</th>
</tr>
</thead>
<tbody>
<tr>
<td>Feb. 13</td>
<td>Fri. A.M. 1</td>
<td>Randomly select 60 male and 120 female pupae from the 39th generation of the random mating base population. Place one male and two females at random in a 3/4 oz. creamer bottle. Keep the bottles in a modified Jamesway incubator (approximately 32.8 C. and 65% relative humidity).</td>
</tr>
<tr>
<td>Feb. 21</td>
<td>Sat. A.M. 3</td>
<td>Repeat the same procedure as Feb. 12 except select 100 male and 200 female pupae.</td>
</tr>
<tr>
<td>Feb. 22</td>
<td>Sun. A.M. 1</td>
<td>Sex the adults and transfer each female to an individual bottle for 24 hours. This period of stay should remove eggs attached to the female's body (if any). Destroy the male with alcohol.</td>
</tr>
<tr>
<td>Feb. 23</td>
<td>Mon. A.M. 1</td>
<td>Remove the females to another bottle with fresh media for a 24-hour egg-lay.</td>
</tr>
<tr>
<td>Feb. 24</td>
<td>Tues. A.M. 1</td>
<td>Take the female out and destroy with alcohol. Incubate the collected eggs (day 1 for pupa age).</td>
</tr>
<tr>
<td>Feb. 27</td>
<td>Fri. A.M. 2</td>
<td>Repeat the procedure of Feb. 22.</td>
</tr>
<tr>
<td>Feb. 28</td>
<td>Sat. A.M. 2</td>
<td>Repeat the procedure of Feb. 23.</td>
</tr>
<tr>
<td>Mar.  2</td>
<td>Mon. A.M. 3</td>
<td>Repeat the procedure of Feb. 22.</td>
</tr>
<tr>
<td>Mar.  3</td>
<td>Tues. A.M. 3</td>
<td>Repeat the procedure of Feb. 23.</td>
</tr>
</tbody>
</table>
Table 10. (continued)

<table>
<thead>
<tr>
<th>Date</th>
<th>Weekday Period</th>
<th>Item</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mar. 6</td>
<td>Fri. A.M. 4</td>
<td>Repeat the procedure of Feb. 22.</td>
</tr>
<tr>
<td>Mar. 7</td>
<td>Sat. A.M. 4</td>
<td>Repeat the procedure of Feb. 23.</td>
</tr>
<tr>
<td></td>
<td>Sat. P.M. 5</td>
<td>Repeat the procedure of Feb. 22.</td>
</tr>
<tr>
<td></td>
<td>Sun. P.M. 5</td>
<td>Repeat the procedure of Feb. 23.</td>
</tr>
<tr>
<td>Mar. 9</td>
<td>Mon. P.M. 5</td>
<td>Repeat the procedure of Feb. 24.</td>
</tr>
<tr>
<td>Mar. 14</td>
<td>Sat. A.M. 1</td>
<td>Record the family size and randomly select two pupae of different sex from each family. Weigh the two selected $F_1$ pupae from each bottle to the nearest $\mu_g$. Make the matings for the $F_2$ progeny according to designs 2 and 3. Randomly choose the mates for $F_1$ progeny from the base population or its subdivisions.</td>
</tr>
<tr>
<td>Mar. 20</td>
<td>Fri. P.M. 6</td>
<td>Repeat the procedure of Feb. 13.</td>
</tr>
<tr>
<td>Mar. 23</td>
<td>Mon. A.M. 1</td>
<td>Sex the adults, take the male out and transfer the female to another bottle. Destroy the male with alcohol.</td>
</tr>
<tr>
<td>Mar. 24</td>
<td>Tues. A.M. 1</td>
<td>Repeat the procedure of Feb. 23.</td>
</tr>
<tr>
<td>Mar. 27</td>
<td>Fri. P.M. 5</td>
<td>Repeat the procedure of Mar. 14.</td>
</tr>
<tr>
<td>Mar. 28</td>
<td>Sat. A.M. 2</td>
<td>Repeat the procedure of Mar. 23.</td>
</tr>
</tbody>
</table>
Table 10. (continued)

<table>
<thead>
<tr>
<th>Date</th>
<th>Weekday</th>
<th>Period</th>
<th>Item</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mar. 29</td>
<td>Sun. A.M.</td>
<td>2</td>
<td>Repeat the procedure of Feb. 23.</td>
</tr>
<tr>
<td></td>
<td>Sun. P.M.</td>
<td>6</td>
<td>Repeat the procedure of Feb. 22.</td>
</tr>
<tr>
<td></td>
<td>Mon. P.M.</td>
<td>6</td>
<td>Repeat the procedure of Feb. 23.</td>
</tr>
<tr>
<td>Mar. 31</td>
<td>Tues. A.M.</td>
<td>3</td>
<td>Repeat the procedure of Mar. 23.</td>
</tr>
<tr>
<td></td>
<td>Tues. P.M.</td>
<td>6</td>
<td>Repeat the procedure of Feb. 24.</td>
</tr>
<tr>
<td>Apr.  1</td>
<td>Wed. A.M.</td>
<td>3</td>
<td>Repeat the procedure of Feb. 23.</td>
</tr>
<tr>
<td>Apr.  2</td>
<td>Thurs. A.M.</td>
<td>3</td>
<td>Repeat the procedure of Feb. 24.</td>
</tr>
<tr>
<td>Apr.  4</td>
<td>Sat. A.M.</td>
<td>4</td>
<td>Repeat the procedure of Mar. 23.</td>
</tr>
<tr>
<td>Apr.  5</td>
<td>Sun. A.M.</td>
<td>4</td>
<td>Repeat the procedure of Feb. 23.</td>
</tr>
<tr>
<td></td>
<td>Sun. P.M.</td>
<td>5</td>
<td>Repeat the procedure of Mar. 23.</td>
</tr>
<tr>
<td>Apr.  6</td>
<td>Mon. A.M.</td>
<td>4</td>
<td>Repeat the procedure of Feb. 24.</td>
</tr>
<tr>
<td></td>
<td>Mon. P.M.</td>
<td>5</td>
<td>Repeat the procedure of Feb. 23.</td>
</tr>
<tr>
<td>Apr.  7</td>
<td>Tues. P.M.</td>
<td>5</td>
<td>Repeat the procedure of Feb. 24.</td>
</tr>
<tr>
<td>Apr. 12</td>
<td>Sun. A.M.</td>
<td>1</td>
<td>Record the family size and randomly select two pupae of different sex from each family. Weigh the two selected pupae from each bottle to the nearest g. This is the end of the procedure for this period.</td>
</tr>
<tr>
<td>Apr. 17</td>
<td>Fri. A.M.</td>
<td>2</td>
<td>Repeat the procedure of Apr. 12.</td>
</tr>
<tr>
<td>Apr. 18</td>
<td>Sat. P.M.</td>
<td>6</td>
<td>Repeat the procedure of Mar. 14.</td>
</tr>
<tr>
<td>Apr. 20</td>
<td>Mon. A.M.</td>
<td>3</td>
<td>Repeat the procedure of Apr. 12.</td>
</tr>
</tbody>
</table>
Table 10. (continued)

<table>
<thead>
<tr>
<th>Date</th>
<th>Weekday</th>
<th>Period</th>
<th>Item</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apr. 24</td>
<td>Fri. A.M.</td>
<td>4</td>
<td>Repeat the procedure of Apr. 12.</td>
</tr>
<tr>
<td>Apr. 25</td>
<td>Sat. P.M.</td>
<td>5</td>
<td>Repeat the procedure of Apr. 12.</td>
</tr>
<tr>
<td>Apr. 27</td>
<td>Mon. P.M.</td>
<td>6</td>
<td>Repeat the procedure of Mar. 23.</td>
</tr>
<tr>
<td>Apr. 28</td>
<td>Tues. P.M.</td>
<td>6</td>
<td>Repeat the procedure of Feb. 23.</td>
</tr>
<tr>
<td>Apr. 29</td>
<td>Wed. P.M.</td>
<td>6</td>
<td>Repeat the procedure of Feb. 24.</td>
</tr>
<tr>
<td>May 17</td>
<td>Sun. P.M.</td>
<td>6</td>
<td>Repeat the procedure of Apr. 12.</td>
</tr>
</tbody>
</table>

The measured traits were pupal weight and family size at the 19th day of age. Family size was based on a 24-hour egg-lay period. Pupal stage is the easiest stage for sexing and handling and has been extensively used in biological research (Enfield et al. 1966 and Wilson et al. 1965).
METHODS OF ANALYSIS

The implementation of the three proposed designs resulted in a number of groups of related individuals. The relationship among these relatives gives rise to resemblance among them. The basis of selection is the resemblance between related individuals. Resemblance results from having genes in common or genes that are identical by descent. The degree of resemblance for a trait measures a fraction of the additive genetic variance since for the same relationship the degree will change by trait. This provides a means of estimating heritability since all methods of estimating heritability rest on measuring how closely relatives resemble each other as compared with unrelated individuals (Lush, 1940). All other relationships are compounded of chains or sums of chains of parent-offspring relationships (Lush, 1948).

Correlation or covariance among relatives measures the resemblance among them. As indicated by Falconer (1960b), both genetic and environmental sources of variance contribute to such correlation or covariance. Cockerham (1963) reasoned that the components of variance should first be translated into covariances between relatives which are then interpretable into components of genetic variance. Mode and Robinson (1959) showed genetic covariance may be partitioned in the same way and with the same number of terms as the genetic variance. The objectives of analyzing the present data are to express the observed covariances between relatives into genetic and environmental casual components. This provides a means of estimating various genetic parameters.
Design 1: This design resulted in one set of full-sib individuals from each mating. Since a sire is mated to two different dams, the two sets of full-sibs produced from each sire are paternal half-sibs. Each sire effect is estimated as a deviation of the mean of all his progeny from the overall mean. This effect not being the same for all sire groups, causes the paternal half-sib groups to be different. The component of variance due to the sire group differences ($c^2_S$) is the estimate of covariance of half-sibs which in turn estimates $1/4c^2_A$. Since this component of variance ($c^2_S$) may be computed by at least two different methods, the clarification of the choice of a model is necessary.

Assume that $s$ randomly chosen sires are mated to $d$ sets of dams. Furthermore, each mating is assumed to retain a constant number of progeny ($r$). The progeny are measured to provide data. A suitable model to be fitted to data is as follows:

$$Y_{ijk} = \mu + S_i + D_{ij} + e_{ijk}$$  \hspace{1cm} (1)

$i = 1, 2, \ldots, s$ sires \hspace{1cm} $j = 1, 2, \ldots, d$ dams \hspace{1cm} $k = 1, 2, \ldots, r$ offspring

where $Y_{ijk}$ is the record of the $k$th progeny of the $j$th dam mated to the $i$th sire; $\mu$ is the overall mean; $S_i$ is the effect of the $i$th sire; $D_{ij}$ is the effect of the $j$th dam mated to the $i$th sire and $e_{ijk}$ is the random error associated with the $k$th progeny of the $j$th dam mated to the $i$th sire.
The following assumptions are made:

\[ E(S_1) = E(D_{ij}) = E(e_{ijk}) = 0, \quad E(S_1^2) = \sigma_S^2, \quad E(D_{ij}^2) = \sigma_D^2, \]

\[ E(e_{ijk}^2) = \sigma_e^2, \quad \text{and} \quad E(e_{ijk}, e_{ijk'}) = 0 \text{ for } k \neq k'. \]

The analysis of variance is presented in Table 11. The sire component of variance can be calculated as follows:

\[ \sigma_S^2 = \frac{[SS_S/s-1]-[SS_D/S/s(d-1)]}{rd} \]

If the model fitted to the data is chosen to be:

\[ Y_{ij} = \mu + S_i + e_{ij}, \quad i = 1, 2, \ldots, s \text{ sires} \]

\[ j = 1, 2, \ldots, n \text{ progeny per sire} \]

which is actually reparameterized model (1), by pooling \( D_{ij} \) and \( e_{ijk} \) as \( e_{ij} \). In this model, \( Y_{ij} \) is the observation on the \( j^{th} \) progeny from the \( i^{th} \) sire; \( S_i \) is the same as before and \( e_{ij} \) is the random error associated with the \( j^{th} \) progeny of the \( i^{th} \) sire. The analysis of variance is in the form of Table 12. Although \( SS_S \) and \( SS_T \) are the same and are associated with the same DF in both tables (\( n = rd \)), and \( SS_W \) in Table 12 is equal to \( SS_D/S + SS_e \) in Table 11, yet the \( \sigma_S^2 \) calculated from Table 12 as

\[ \frac{[SS_S/s-1]-[SS_W/s(n-1)]}{n = rd} \]

differs from the \( \sigma_S^2 \) computed before.

To show the source of this difference, the simplest way is to express the variance components of Table 12 in terms of the sums of squares in Table 11.
Table 11. Analysis of variance table for a hierarchical model

<table>
<thead>
<tr>
<th>Source</th>
<th>DF</th>
<th>SS</th>
<th>Mean square (MS)</th>
<th>Expectation of MS (EMS)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between sires (S)</td>
<td>s-1</td>
<td>SS$_S$</td>
<td>SS$_S$/s-1</td>
<td>$\sigma_e^2 + rC^2_{D/S} + rDc^2_S$</td>
</tr>
<tr>
<td>Between dams/sires (D/S)</td>
<td>s(d-1)</td>
<td>SS$_{D/S}$</td>
<td>SS$_{D/S}$/s(d-1)</td>
<td>$\sigma_e^2 + rC^2_{D/S}$</td>
</tr>
<tr>
<td>Within progeny</td>
<td>sd(r-1)</td>
<td>SS$_e$</td>
<td>SS$_e$/sd(r-1)</td>
<td>$\sigma_e^2$</td>
</tr>
<tr>
<td>Total (T)</td>
<td>sdr-1</td>
<td>SS$_T$</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*DF and SS represent degrees of freedom and sum of squares, respectively.*
Table 12. Analysis of variance table for model (2)

<table>
<thead>
<tr>
<th>Source</th>
<th>DF</th>
<th>SS</th>
<th>MS</th>
<th>EMS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between sires (S)</td>
<td>s-1</td>
<td>SS_S</td>
<td>SS_S/s-1</td>
<td>$\sigma^2_w + n\sigma^2_S$</td>
</tr>
<tr>
<td>Within sires (W)</td>
<td>s(n-1)</td>
<td>SS_W</td>
<td>SS_W/s(n-1)</td>
<td>$\sigma^2_W$</td>
</tr>
<tr>
<td>Total</td>
<td>sn-1</td>
<td>SS_T</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

$$M_S^W_{\text{of Table 12}} = \frac{SS_W}{s(n-1)} = \frac{SS_{D/S} + SS_e}{s(n-1)} = \frac{SS_{D/S}}{s(rd-1)} + \frac{SS_e}{s(rd-1)}$$

which differs from $$\frac{SS_{D/S}}{s(d-1)} + \frac{SS_e}{sd(r-1)}$$ of Table 11. Thus, $\sigma^2_S$ computed from Table 12 in terms of the values of Table 11 would be as follows:

$$\sigma^2_S = \frac{1}{rd} \left[ \frac{SS_S}{s-1} - \frac{SS_{D/S}}{s(d-1) + sd(r-1)} - \frac{SS_e}{s(d-1) + sd(r-1)} \right]$$

and differs from the $\sigma^2_S$ computed from Table 11.

The expectation (E) of the mean square within sires is equal to:

$$E[\frac{SS_{D/S} + SS_e}{s(rd-1)}] = \frac{E(SS_{D/S}) + E(SS_e)}{s(rd-1)}$$

where

$$E(SS_{D/S}) = s(d-1)(\sigma^2_e + r\sigma^2_{D/S})$$

and

$$E(SS_e) = sd(r-1)\sigma^2_e$$

as shown in Table 11.
Thus,
\[ E(\text{MS}_w) = \frac{s(d-l) (\sigma_e^2 + r\sigma_{D/S}^2) + s(d-r-l)\sigma_e^2}{s(rd-l)} = \]
\[ = \frac{(d-l)\sigma_e^2 + r(d-l)\sigma_{D/S}^2 + d(r-l)\sigma_e^2}{rd-l} + \frac{d-l + dr-d}{rd-l} \frac{r(d-l)}{rd-l} \sigma_{D/S}^2 \]

The values of Table 12 in terms of the values of Table 11 are shown in Table 13.

**Table 13. Modified table of analysis of variance for model (2)**

<table>
<thead>
<tr>
<th>Source</th>
<th>DF</th>
<th>SS</th>
<th>MS</th>
<th>EMS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between sires</td>
<td>s-1</td>
<td>SS_S</td>
<td>SS_S/s-1</td>
<td>(\sigma_e^2 + r\sigma_{D/S}^2 + rd\sigma_S^2)</td>
</tr>
<tr>
<td>Within sires</td>
<td>s(dr-l)</td>
<td>SS_D/S + SS_e (SS_D/S + SS_e)/s(dr-l)</td>
<td>(\sigma_e^2 + \frac{r(d-l)}{rd-l} \sigma_{D/S}^2)</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>sdr-l</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

By comparing Table 11 and Table 13, it can be shown that if \(r = 1\) (one observation per dam), then \(\sigma_S^2\) computed by either method will yield the same result. This indicates that the analysis of the data composed of only half-sib individuals from each sire group should be analyzed by model (2), while the existence of full-sibs in the data dictate the use of model (1). Thus, \(\sigma_S^2\) computed from Table 12 as modified in Table 13 is equal to:
which overestimates the heritability if \( r > 1 \). From the above discussion model (1) was chosen to analyze the measured pupal weight whereas model (2) was applied to the family size.

Design 2: The models chosen for the design 2 data are as follows:

1. Pupal weight

\[
Y_{ijk} = \mu + G_{i} + G_{ij} + \epsilon_{ijk}
\]

\( i = 1, 2, \ldots, 208 \) grandsires \( j = 1, 2 \) granddams \( k = 1, 2 \) \( F_2 \) progeny

where \( Y_{ijk} \) is the weight of the \( k \)th progeny of the \( j \)th granddam mated to the \( i \)th grandsire; \( \mu \) is the overall mean; \( G_{i} \) is the effect of the \( i \)th grandsire; \( G_{ij} \) is the effect of the \( j \)th granddam mated to the \( i \)th grandsire and \( \epsilon_{ijk} \) is the random error associated with the \( k \)th progeny of the \( j \)th granddam mated to the \( i \)th grandsire. \( G_{ij} \) may also be represented as the effect of the \( j \)th \( F_1 \) offspring of the \( i \)th grandsire. This is because the effect of each granddam is transmitted to the \( F_2 \) progeny by only one of her offspring. The following assumptions are made:
E(GS,) = E(GD_l^j) = E(e_i jk) = 0, E(GS,)^2 = \sigma_{GS}^2, E(GD_l^j)^2 = \sigma_{GD/GS}^2, \\
E(e_i jk)^2 = \sigma_e^2, \text{ and } E(e_i jk, e_i jk') = 0.

2. Family size

Since each grandsire resulted in only two family size measurements in each generation, the following model was chosen to analyze the data:

\[ Y_{ij} = \mu + GS_i + e_{ij} \]

\[ i = 1, 2, \ldots, 208 \text{ grandsires} \quad j = 1, 2 \text{ family size} \]

where \( Y_{ij} \) is the \( j^{th} \) family size of the \( i^{th} \) grandsire; \( \mu \) is the overall mean; \( GS_i \) is the effect of the \( i^{th} \) grandsire; \( e_{ij} \) is the random error associated with the \( j^{th} \) family size of the \( i^{th} \) sire. The following assumptions are made:

\[ E(GS_i) = E(e_{ij}) = 0, E(GS_i)^2 = \sigma_{GS}^2, E(e_{ij})^2 = \sigma_e^2, \text{ and} \]

\[ E(e_{ijk}, e_{ijk'}) = 0 \text{ for } k \neq k'. \]

Design 3: Since the structure of the data in design 3 was quite similar to that of design 2 (except for the differences due to the \( F_1 \) offspring), the design 3 data were also analyzed in the same way as design 2. Additional analyses in this design and design 2 were covariances between the records of two groups of relatives. For instance, the covariance between weights of parent (x) and offspring (y) was computed by using a combination of their weights (e.g. weight of x + weight of y).
Then from the identity:

\[ V(x + y) = V(x) + V(y) + 2\text{Cov}(x,y) \]

\[ \text{Cov}(x,y) = \frac{1}{2} [V(x + y) - V(x) - V(y)] \]

where \( V \) and \( \text{Cov} \) denote the variance and covariance, respectively.
RESULTS AND DISCUSSION

The arithmetic means of the 19 day pupa weight and family size for each of the three designs are given in Table 14. Family size is defined as the total number of live full-sib individuals (pupae and larvae) obtained from a 24-hour egg collection. They were reared in the same bottle. Family size was determined at the time of pupa-weighing. Thus, family size, as defined above, is dependent on the number of eggs laid by the female in a 24-hour period and the survival ability of the resulting larvae and pupae up to 19 days of age. This attribute was measured only on those mating groups (sire or grandsire groups) in which each mating succeeded in producing at least two offspring (one of each sex) in each generation. Failure to reach this limit caused all members of that mating group to be excluded from the design. The way of defining family size makes it possible to consider this trait as being influenced by the survival ability of the members of the family themselves (direct genetic effect), as well as by the nutritional substances provided by the dam (included in the eggs) necessary for the development of eggs into viable larvae (maternal effect).

Table 14. Arithmetic means of pupa weight and family size for each design

<table>
<thead>
<tr>
<th></th>
<th>Design 1</th>
<th>Design 2</th>
<th>Design 3</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>F_1</td>
<td>F_2</td>
<td>F_1</td>
</tr>
<tr>
<td>Pupa weight</td>
<td>2575^a</td>
<td>2572</td>
<td>2569</td>
</tr>
<tr>
<td>Family size</td>
<td>17.7</td>
<td>17.7</td>
<td>19.9</td>
</tr>
</tbody>
</table>

^aMicrograms.
The results show that family size increased during the investigation. Such an increase was also observed during a previous experiment in the same laboratory (Moore, 1969). At the start of the experiment the pupae were obtained from the base population which was not kept in the incubator. This may partly be responsible for the smaller $F_1$ family size. Should this prove to be the case, a similar experiment should be started by using a base population kept and reproduced in an incubator for several generations. The number of females mated to each male was two in the $F_1$ and one in the $F_2$. This may also be the cause of variation in family size. The accidental malfunction of the incubator in different stages of development, slight changes in temperature and humidity, an improvement over time in skill and technique in handling (e.g. length of the time of vacuuming, taking the adult parents out of the bottle, sexing the pupae and adults, etc.), freshness of the food, and other unknown factors may have also been responsible for the change in family size.

Only a limited number of literature reports are concerned with family size. These reports vary in the length of time for egg-collection (e.g. 24 hours or more), lab procedures and facilities (e.g. removing adults, amount of food placed in each bottle, etc.), the time of determining family size (e.g. number of larvae, pupae, adults, and any combinations of the three at various ages), etc. For these reasons, the cause of the increase in family size may not be determined until a standard procedure is followed.
Design 1:

The results of the analysis of the pupa weight obtained from the design 1 data are presented in Table 15. The sire component of variance

Table 15. Analysis of variance of pupa weight for design 1

<table>
<thead>
<tr>
<th>Source</th>
<th>DF</th>
<th>MS</th>
<th>EMS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between sires (S)</td>
<td>330</td>
<td>54417.53</td>
<td>$\sigma_e^2 + 2\sigma_{D/S}^2 + 4\sigma_S^2$</td>
</tr>
<tr>
<td>Bet. dams/sires (D/S)</td>
<td>331</td>
<td>40880.53</td>
<td>$\sigma_e^2 + 2\sigma_{D/S}^2$</td>
</tr>
<tr>
<td>Progeny/dams (e)</td>
<td>662</td>
<td>25852.42</td>
<td>$\sigma_e^2$</td>
</tr>
<tr>
<td>Total (T)</td>
<td>1323</td>
<td>36737.36</td>
<td>$\sigma_T^2$</td>
</tr>
</tbody>
</table>

($\sigma_S^2$) is due to the sire groups being different. Since these groups are composed of half-sib individuals which have the sire effect in common, this component is also an estimate of the covariance between half-sibs. This is shown, by using the model previously described, as follows:

$$ Y_{ijk} = \mu + S_i + D_{ij} + e_{ijk}. $$

$$ \text{Cov}(Y_{ijk}, Y_{ijk'}) = \text{covariance between half-sibs (HS)}. $$

In general,

$$ \text{Cov}(X, Y) = E[(X-E(X))(Y-E(Y))]. $$

Thus,

$$ \text{Cov}(Y_{ijk}, Y_{ijk'}) = E[(Y_{ijk}-E(Y_{ijk}))(Y_{ijk'}-E(Y_{ijk'}))]. $$

$$ E(Y_{ijk}) = E(Y_{ijk'}) = \mu $$
since all the terms in the model (except $u$) represent random effects which have zero expectations. Thus,

$$\text{Cov}(Y_{ij}, Y_{i'j'}) = E[Y_{ij} - \mu][Y_{i'j'} - \mu] =$$

$$E[S_i + D_{ij} + e_{ij}][S_i + D_{ij} + e_{ij}] =$$

$$E[S_i^2 + D_{ij}^2 + e_{ij}^2 + \text{cross products}] = \sigma_S^2$$

since the sires and the dams mated to each sire are unrelated making the covariances between effects not having identical subscripts zero.

The dam component of variance ($\sigma_{D/S}^2$) is due to the dam groups being different. The progeny of different dams within these groups are half-sibs and the progeny of a particular dam are full-sibs. Thus, $\sigma_{D/S}^2$ is the estimate of the covariance between full-sibs (FS) after the covariance between half-sibs is removed, $[\text{Cov}(FS) - \text{Cov}(HS)]$. This is shown as follows:

$$\text{Cov}(FS) = \text{Cov}(Y_{ij}, Y_{i'j'}) = E[Y_{ij} - E(Y_{ij})][Y_{i'j'} - E(Y_{i'j'})] =$$

$$E[Y_{ij} - \mu][Y_{i'j'} - \mu] = E[S_i + D_{ij} + e_{ij}][S_i + D_{ij} + e_{ij}] =$$

$$E[S_i^2 + D_{ij}^2 + e_{ij}^2 + \text{cross products}] = \sigma_S^2 + \sigma_{FS}^2.$$ 

Thus,

$$\text{Cov}(FS) - \text{Cov}(HS) = \sigma_S^2 + \frac{\sigma_{FS}^2}{\sigma_D} = \sigma_{D/S}^2.$$ 

Since the two components of variance together ($\sigma_S^2 + \sigma_{D/S}^2$) estimate the covariance between full-sibs, the component $\sigma_{FS}^2$ estimates the remainder of the total variance not accounted for by the covariance between full-sibs ($\sigma_T^2 - \text{Cov}(FS)$). Thus, the components $\sigma_S^2$, $\sigma_{D/S}^2$, and $\sigma_{FS}^2$ may be expressed
in terms of the covariances among relatives. The results are summarized in Table 16.

Table 16. Estimates of the different variance components for pupa weight of design 1

<table>
<thead>
<tr>
<th>Components</th>
<th>Covariances</th>
<th>Estimates of the components</th>
<th>% of the total variance</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \sigma^2_S )</td>
<td>Cov(HS)</td>
<td>3384.25</td>
<td>9.2</td>
</tr>
<tr>
<td>( \sigma^2_{D/S} )</td>
<td>Cov(FS)-Cov(HS)</td>
<td>7514.05</td>
<td>20.4</td>
</tr>
<tr>
<td>( \sigma^2_e )</td>
<td>( \sigma^2_T - \text{Cov(FS)} )</td>
<td>25852.42</td>
<td>70.4</td>
</tr>
<tr>
<td>( \sigma^2_T )</td>
<td></td>
<td>36750.72</td>
<td>100.0</td>
</tr>
</tbody>
</table>

The main objective of this design was to utilize the paternal half-sib covariance to estimate the additive genetic variance for each trait. This covariance estimates \( 1/4 \sigma^2_A \) in the absence of epistatic effects since the sire transmits a sample half of his genes to each offspring. From Table 16, \( \sigma^2_A \) is calculated to be 13537.00 for the pupa weight which accounts for 36.8\% of the total variance. The intra-class correlation

\[
\tau = \frac{\sigma^2_S}{\sigma^2_S + \sigma^2_{D/S} + \sigma^2_e} = .09.
\]

This correlation measures the proportion of the total variance that is common to members of the same sire group. This is the measurement of the likeness between paternal half-sibs as shown in the following. The correlation between half-sibs is
This way of obtaining $t$ from the ratio of the covariance of two variables to the geometric mean of their variances also indicates that the intraclass correlation is, in fact, intrinsically a correlation coefficient (Kempthorne, 1957). The computation of a simple correlation coefficient (inter-class correlation) requires information from only two individuals whereas the intraclass correlation utilizes the variance components which are based on all the data (Pirchner, 1969). Since $\sigma_S^2$ estimates $1/4 \sigma_{A_0}^2$, $t$ becomes

$$t = \frac{1/4 \sigma_{A_0}^2}{\sigma_S^2 + \sigma_{D/S}^2 + \sigma_e^2}$$

and $4t$ estimates

$$\frac{\sigma_{A_0}^2}{\sigma_S^2 + \sigma_{D/S}^2 + \sigma_e^2} = \frac{\sigma_{A_0}^2}{\sigma_T^2} = .37.$$

This estimates the extent to which phenotypic likeness parallels genetic likeness (in terms of the additive genetic effects) and is the estimate of heritability, $h^2$, (Lush, 1949).
Since $h^2$ is estimated from $4t$, the sampling error of $h^2$ will be 16 times that of the sampling error of $t$. Thus, the accuracy of the estimate of heritability ($h^2$) resides on the accuracy of the estimate of $t$, ($\hat{t}$). The accuracy of the estimate of $t$ in turn depends on the accuracy of determining the components of variance. This accuracy in turn rests on the number of degrees of freedom for each component. This is shown as follows: Assume that only 100 individuals are to be measured to provide the data to estimate the heritability of a particular trait. Furthermore, assume that the paternal half-sib relationship is to be utilized for this purpose. Obviously, there are several ways of utilizing the 99 degrees of freedom (DF) but only two cases are shown below.

<table>
<thead>
<tr>
<th></th>
<th>Case 1</th>
<th>Case 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between sires</td>
<td>1</td>
<td>24</td>
</tr>
<tr>
<td>Between dams/sires</td>
<td>2</td>
<td>25</td>
</tr>
<tr>
<td>Within progeny</td>
<td>96</td>
<td>50</td>
</tr>
<tr>
<td>Total</td>
<td>99</td>
<td>99</td>
</tr>
</tbody>
</table>

Note that the mating structure is chosen to be the same (one sire mated to two dams) in both cases to make the comparison easy. The differences between the progeny of different sires, $\sigma_5^2$, which measures the likeness between paternal half-sibs is estimated on progeny of only two sires in case 1. This is not nearly as accurate as in case 2 where the progeny of 25 sires are utilized.

The sampling error of $\sigma_5^2$ is computed by the method of Anderson and Bancroft (1952) as follows:
where $\sigma_i^2$ is a variance component estimated from the $i$th mean square ($V_i$) with $f_i$ degrees of freedom. The coefficient of the variance component being estimated is assumed to be $c$. Since $\sigma_S^2$ is estimated from the sire mean squares (MS$_S$) and the dam within sire mean squares (MS$_D/S$), then

$$\sigma_S^2 = \frac{MS_S - MS_D/S}{rd} \quad \text{(See Table 11)}$$

The importance of an increase in the number of sires ($s$) in reducing the sampling variance of $\sigma_S^2$ may readily be seen if the mean squares are expressed as the sum of squares (SS) divided by the corresponding degrees of freedom as follows:

$$V(\sigma_S^2) = \frac{2}{(rd)^2} \left[ \frac{SS_S^2}{s+1} + \frac{SS_D/S^2}{(s(d-1) + 2)} \right] .$$

The two sampling variances for the two cases are computed to be:

Case 1: $$V(\sigma_S^2) = \frac{2}{2500} \left[ \frac{SS_S^2}{3} + \frac{SS_D/S^2}{16} \right] = \frac{SS_S^2}{3750} + \frac{SS_D/S^2}{20,000}$$

Case 2: $$V(\sigma_S^2) = \frac{2}{16} \left[ \frac{SS_S^2}{14976} + \frac{SS_D/S^2}{16875} \right] = \frac{SS_S^2}{119,808} + \frac{SS_D/S^2}{135,000} .$$

The $SS_S^2$ and $SS_D/S^2$ in case 2 are reduced about 32 times and 7 times more than that of case 1, respectively. Thus, increase in number of sires should increase the accuracy of the estimate of $\sigma_S^2$. Furthermore, an increase in the number of offspring per dam ($r$) should also contribute in making the estimate of $\sigma_S^2$ more dependable. Lower standard errors could have been achieved in this study by weighing more individuals per genetic
group. But it was thought more important to have as many sets of grand-
sire groups as physically possible.

The components $\sigma_S^2$ and $\sigma_{D/S}^2$, expressed as Cov(HS) and Cov(FS)-Cov(HS), respectively, may further be expanded as shown by Willham (1963).

$$\sigma_S^2 = \frac{1}{4} \sigma_{A_0}^2$$

$$\sigma_{D/S}^2 = \frac{1}{4} \sigma_{A_0}^2 + \frac{1}{4} \sigma_{A_0A_m}^2 + \sigma_{A_m}^2 + \sigma_{D_m}^2 + \sigma_{E_m}^2$$

where $\sigma_{E_m}^2$ is the variance due to the maternal environment common to the full sibs. The difference, $\sigma_{D/S}^2 - \sigma_S^2$, is an estimate of $\frac{1}{4} \sigma_{D_0}^2 + \sigma_{A_0A_m}^2 + \sigma_{A_m}^2 + \sigma_{D_m}^2 + \sigma_{E_m}^2$ in the absence of epistatic effects. This difference is mainly attributable to the maternal effects (genetic and en-
vvironmental) since only $\frac{1}{4}$ of the direct dominance effect is involved. The results summarized in Table 16 indicate that $\sigma_{D/S}^2$ is about 2.2 times larger than $\sigma_S^2$. Thus, pupa weight is a trait at least in part influenced by maternal effects.

The component $\sigma_S^2$, may be larger than $\sigma_{D/S}^2$ if $\sigma_{A_0A_m}$ is negative with an absolute value larger than $\frac{1}{4} \sigma_{D_0}^2 + \sigma_{A_m}^2 + \sigma_{D_m}^2 + \sigma_{E_m}^2$. This requires the Cov(HS) to be greater than $[1/2] \text{Cov(FS)}$. In the absence of maternal effects and dominance, the two components ($\sigma_S^2$, $\sigma_{D/S}^2$) should approximately be of the same magnitude which requires Cov(HS) to be equal to $[1/2] \text{Cov(FS)}$. In this case, heritability estimates from

$$\frac{4\sigma_S^2}{\sigma_S^2 + \sigma_{D/S}^2 + \sigma_e^2}$$

$$\frac{4\sigma_{D/S}^2}{\sigma_S^2 + \sigma_{D/S}^2 + \sigma_e^2}$$

and

$$\frac{2(\sigma_S^2 + \sigma_{D/S}^2)}{\sigma_S^2 + \sigma_{D/S}^2 + \sigma_e^2}.$$
should be approximately equal. The respective values of the three heritabilities computed from Table 16 are .37, .82, and .59. The discrepancies between the estimates strongly suggest that maternal effects are present and are likely to be important. The heritability estimate computed from the sire component of variance (.37) is taken to be the estimate of heritability in the narrow sense for pupa weight since it is free of maternal effects. This estimate is consistent with other reports in the literature (e.g. Enfield et al. (1966) and Wilson et al. (1965)).

The standard error of the heritability estimate is calculated by the method of Osborn and Patterson (1952) and Kempthorne (1957) as presented by Dickerson (1969). The procedure is summarized as follows:

\[
h^2 = \frac{4\sigma_S^2}{\sigma_S^2 + \sigma_D/S^2 + \sigma_e^2} = \frac{4X}{Y}
\]

where \(X = \sigma_S^2\) and \(Y = \sigma_S^2 + \sigma_D/S^2 + \sigma_e^2\).

\[
\sigma_{h^2} = \frac{\sigma(4X/Y)}{\sqrt{Y^2V(X) + X^2V(Y) - 2XY\text{Cov}(X,Y)}}.
\]

\[
V(X) = V(\sigma_S^2) = \sqrt{\frac{\text{MS}_S - \text{MS}_{D/S}}{4}} = \frac{1}{2/16}\left[\frac{\text{MS}_S^2}{333} + \frac{\text{MS}_{D/S}^2}{331}\right]
\]

\[
(1/8) \left[\frac{(54417.53)^2}{332} + \frac{(40880.53)^2}{333}\right] = 1,742,269.06 \quad \text{(See Table 15 for the values used here.)}
\]

\[
V(Y) = V(\sigma_S^2 + \sigma_D/S^2 + \sigma_e^2) = V(\sigma_S^2) + V(\sigma_D/S^2) + V(\sigma_e^2) + 2\text{Cov}(\sigma_S^2, \sigma_D/S^2) + 2\text{Cov}(\sigma_S^2, \sigma_e^2) + 2\text{Cov}(\sigma_D/S^2, \sigma_e^2).
\]

\[
V(\sigma_D/S^2) = V\left[\frac{\text{MS}_{D/S} - \text{MSe}}{2}\right] = \frac{2}{4} \left[\frac{\text{MS}_{D/S}^2}{331+2} + \frac{\text{MSe}^2}{662+2}\right]
\]
\[ \left( \frac{40880.53}{333} \right)^2 + \left( \frac{25852.42}{664} \right)^2 = 3012609.75. \]

\[ V(\sigma_e^2) = \frac{2MS_e^2}{664+2} = \frac{2(25852.42)^2}{664} = 2013095.24. \]

\[ \text{Cov}(\sigma_S^2, \sigma_{D/S}^2) = \frac{-2}{(4)(2)} \left( \frac{MS_{D/S}^2}{333+2} \right) = (-1/4) \left( \frac{40880.53}{333} \right)^2 = -1254667.97. \]

\[ \text{Cov}(\sigma_S^2, \sigma_e^2) = 0 \]

\[ \text{Cov}(\sigma_{D/S}^2, \sigma_e^2) = - \frac{2}{2} \left[ \frac{MS_e^2}{664+2} \right] = - \frac{2(25852.42)^2}{664} = -1006547.62. \]

Thus,

\[ V(Y) = 1742269.06 + 3012609.75 + 2013095.24 + 2(-1254667.97) + 0 + 2(-1006547.62) = 2245542.87. \]

\[ \text{Cov}(X,Y) = \text{Cov}(\sigma_S^2, \sigma_S^2 + \sigma_{D/S}^2 + \sigma_e^2) = \text{Cov}(\sigma_S^2, \sigma_S^2) + \text{Cov}(\sigma_{D/S}^2, \sigma_{D/S}^2) + \text{Cov}(\sigma_{D/S}^2, \sigma_e^2) = 1742269.06 - 1254667.97 = 487601.09. \]

Thus,

\[ \sigma_h^2 = \left( \frac{4}{(36750.72)^2} \right) [((36750.72)^2(1742269.06) + (3384.25)^2(2245542.87) - 2(3384.25)(36750.72)(487601.09)]^{1/2} = .14. \]

The results of the analysis of the family size obtained from the design 1 data are given in Table 17. Since each sire group is made up

<table>
<thead>
<tr>
<th>Source of variation</th>
<th>DF</th>
<th>MS</th>
<th>EMS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between sires (S)</td>
<td>330</td>
<td>54.99</td>
<td>( \sigma_S^2 + 2\sigma_{D/S}^2 )</td>
</tr>
<tr>
<td>Within sires (W)</td>
<td>331</td>
<td>45.10</td>
<td>( \sigma_W^2 )</td>
</tr>
<tr>
<td>Total</td>
<td>661</td>
<td>50.03</td>
<td>( \sigma_T^2 )</td>
</tr>
</tbody>
</table>
of half-sib individuals, \( \sigma_S^2 \) is an estimate of the covariance among half-sibs. The variance component \( \sigma_W^2 \) represents the remainder of the total variance not included in the covariance among half-sibs \( (\sigma_T^2 - \text{Cov}(HS)) \). The estimates of the components derived from Table 17 are given in Table 18.

Table 18. Estimates of the different variance components for family size

<table>
<thead>
<tr>
<th>Components</th>
<th>Covariances</th>
<th>Estimates of the components</th>
<th>% of the total variance</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \sigma_S^2 )</td>
<td>Cov(HS)</td>
<td>4.94</td>
<td>9.9</td>
</tr>
<tr>
<td>( \sigma_W^2 )</td>
<td>( \sigma_T^2 - \text{Cov}(HS) )</td>
<td>45.10</td>
<td>90.1</td>
</tr>
<tr>
<td>( \sigma_T^2 )</td>
<td></td>
<td>50.04</td>
<td>100.0</td>
</tr>
</tbody>
</table>

The intra-class correlation, \( t \), is measured as \( \frac{\sigma_S^2}{\sigma_S^2 + \sigma_W^2} = .10 \).

The estimate of the additive genetic variance \( (\sigma_A^2) \) for this trait is computed from Table 18 to be 19.7 or \( 4\sigma_S^2 \). The heritability estimate for family size is computed as \( 4t = .40 \). The standard error of this heritability estimate by the method previously described is calculated to be \( .22 \). This standard error may also be calculated as follows:

\[
t = \frac{\sigma_S^2}{\sigma_S^2 + \sigma_W^2} = .10
\]

\[
V(t) = \frac{2[1 + (n-1)t]^2 (1 - t)^2}{n(n - 1)(N - 1)}
\]

where \( N \) is the number of half-sib families and \( n \) indicates the number of observations per family. Thus,
\[ V(t) = \frac{2(1 + (2 - 1)(1.1))^2(1 - 1)^2}{2(2 - 1)(331 - 1)} = 0.003 \]

\[ V(h^2) = 16V(t) = (16)(0.003) = 0.048, \text{ and} \]

\[ \sigma_{h^2}^2 = \sqrt{0.048} = 0.22. \]

**Design 2:**

The results of the analysis of the pupa weight are given in Table 19. The variance component \( \sigma_{GS}^2 \) is due to the grandsire groups being different. These groups are composed of \( F_2 \) progeny of each grandsire by way of a son and a daughter which makes them half first cousins (HFC). Thus, \( \sigma_{GS}^2 \) estimates the covariance between half first cousins (Cov(HFC)). The component \( \sigma_{GD/GS}^2 \) is due to the granddam groups within grand sires being different. Since the progeny of different \( F_1 \) offspring within each

<table>
<thead>
<tr>
<th>Source of variation</th>
<th>DF</th>
<th>MS</th>
<th>EMS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between grandsires (GS)</td>
<td>207</td>
<td>48498.76</td>
<td>( \sigma_{e}^2 + 2\sigma_{GD/GS}^2 + 4\sigma_{GS}^2 )</td>
</tr>
<tr>
<td>Between ( F_1 )/GS</td>
<td>206</td>
<td>44672.04</td>
<td>( \sigma_{e}^2 + 2\sigma_{GD/GS}^2 )</td>
</tr>
<tr>
<td>( F_2 ) progeny/( F_1 )/GS</td>
<td>416</td>
<td>23866.80</td>
<td>( \sigma_{e}^2 )</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>831</td>
<td>35710.73</td>
<td></td>
</tr>
</tbody>
</table>
grand sire are half first cousins and since the progeny of a particular $F_1$ individual are full-sibs, the $\sigma^2_{GD/GS}$ component may be expressed as the $\text{Cov}(FS) - \text{Cov}(HFC)$. The covariance expression and the estimate of each variance component of Table 19 are presented in Table 20.

Table 20. Estimates of variance components for pupa weight of design 2

<table>
<thead>
<tr>
<th>Components</th>
<th>Covariances</th>
<th>Estimates of the components</th>
<th>% of the total variance</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\sigma^2_{GS}$</td>
<td>$\text{Cov}(HFC)$</td>
<td>456.68</td>
<td>1.28</td>
</tr>
<tr>
<td>$\sigma^2_{GD/GS}$</td>
<td>$\text{Cov}(FS) - \text{Cov}(HFC)$</td>
<td>23866.80</td>
<td>66.80</td>
</tr>
<tr>
<td>$\sigma^2_e$</td>
<td>$\sigma^2_T - \text{Cov}(FS)$</td>
<td>35726.10</td>
<td>100.00</td>
</tr>
<tr>
<td>$\sigma^2_T$</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The $\text{Cov}(HFC)$ is $1/16 \sigma^2_{A_0} + 1/8 \sigma_{A_0A_m}$ (see the example in the Design of Experiment section). The main objective of this design is to estimate $\sigma_{A_0A_m}$, the direct-maternal additive genetic covariance. Since this covariance has the coefficient $1/8$, a large number of grandsires is required to reduce the sampling error. For this reason, about 63% of the total grandsires used in this experiment were assigned to this design. The estimate of $\sigma^2_{A_0}$ from design 1 (using $F_1$ progeny of all grandsires) is used in this design to estimate $\sigma_{A_0A_m}$ as follows:

$$456.68 = 1/16 \sigma^2_{A_0} + 1/8 \sigma_{A_0A_m} \quad \text{or}$$

$$\sigma_{A_0A_m} = 8(456.68 - (1/16)(13537.00)) = -3115.04.$$
This negative covariance accounts for 8.7% of the total variance in this design. The sampling error of this estimate may be calculated as follows:

\[
\sigma_{GS}^2 = \frac{1}{16} \sigma_{AO}^2 + \frac{1}{8} \sigma_{AOAm}^2 \quad \text{or}
\]

\[
\sigma_{AOAm}^2 = 8 \sigma_{GS}^2 - \frac{1}{2} \sigma_{AO}^2
\]

and since

\[
\sigma_{AO}^2 = 4 \sigma_{S}^2,
\]

\[
\sigma_{AOAm}^2 = 8 \sigma_{GS}^2 - 2 \sigma_{S}^2
\]

\[
V(\sigma_{AOAm}^2) = 64V(\sigma_{GS}^2) + 4V(\sigma_{S}^2) - 32Cov(\sigma_{GS}^2, \sigma_{S}^2).
\]

Considering that the two components of variance, \(\sigma_{GS}^2\) and \(\sigma_{S}^2\), are independent, then:

\[
V(\sigma_{AOAm}^2) = 64V(\sigma_{GS}^2) + 4V(\sigma_{S}^2).
\]

\[
V(\sigma_{GS}^2) = (2/16) \left[ \left( \frac{48498.76}{207} \right)^2 + \left( \frac{46672.04}{208} \right)^2 \right] = 2703370.98
\]

(see Table 19)

The variance of \(\sigma_{S}^2\) was previously calculated to be 1742269.06. Thus,

\[
V(\sigma_{AOAm}^2) = (64)(2703370.98) + (4)(1742269.06) = 179,984,818.96
\]

\[
\sigma(\sigma_{AOAm}^2) = \sqrt{V(\sigma_{AOAm}^2)} = \sqrt{179,984,818.96} = 13415.84.
\]

To illustrate how this covariance term is brought about, a path coefficient diagram is shown in Figure 5. In this diagram P, G, and E represent the phenotypic, genotypic, and environmental values of each individual for pupa weight, respectively. The subscripts o and m represent the direct and maternal components of pupa weight, respectively.
Figure 5. Path coefficient diagram showing the biometric relations between members of each grandsire group in design 2.
Primes ('') and double primes (""") are also used to distinguish between parent and grandparent of the F₂ progeny (P₀ and P₀₀), respectively. The subscripts S and D refer to male and female paternal half-sibs, respectively, and are also used to distinguish between their progeny. The genetic and environmental correlations between direct and maternal effects are represented by \( r_{G₀Gₘ} \) and \( r_{E₀Eₘ} \), respectively. These correlations in the offspring are assumed to be the same as in the parents and grandparents. The square of each path coefficient (\( m \), \( h₀ \), \( hₘ \), \( e₀ \), and \( eₘ \)) measures the fraction of the variation in the dependent values which is determined by the causal value. For instance,

\[
\frac{\sigma_{G₀}^2}{\sigma_{Pₐ}^2} = h₀^2
\]

since a fraction of the variation in \( Pₐ \) values is caused by changes in \( G₀ \) values. This is the measurement of heritability of pupa weight if the genotypic values of the trait are only the additive genetic values for that trait.

Although sires and grandsires lack the phenotypic expression for maternal effects, they do possess the genotype for it. Thus, there is no path m from the male parents to their offspring since they do not contribute to the growth of their offspring through a maternal effect as do the female parents. This is also true for the offspring which have not yet become a parent.

Some authors (e.g., Willham, 1964) have not included a phenotypic expression for a maternal effect in the female parents. This is reasonable
since the existence of such an expression and its independent measurement is questionable. In this case, two paths are drawn to connect $G'D_m$ and $E'D_m$ directly to $P_{D_0}$. If the two paths are called $m'$ and $e'$, respectively, the relationships between the two systems are as follows:

$$m' = \frac{\sigma G'D_m}{\sigma P_{D_0}} = \frac{\sigma G'D_m}{\sigma P_{D_0}} \cdot \frac{\sigma P'D_m}{\sigma P_{D_0}} = (h_m)(m)$$

$$e' = \frac{\sigma E'D_m}{\sigma P_{D_0}} = \frac{\sigma E'D_m}{\sigma P_{D_0}} \cdot \frac{\sigma P'D_m}{\sigma P_{D_0}} = (e_m)(m).$$

Thus, the two systems are identical if $m_{h_m}$ and $m_{e_m}$ are redefined as $m'$ and $e'$, respectively.

From Figure 5, the correlation between half first cousins ($r_{PS_0P_{D_0}}$) is

$$r_{PS_0P_{D_0}} = \frac{1}{16} h_0^2 + \frac{1}{8} m_{h_m^2} r_{G_0G_m}.$$

Obviously the path $m_{h_m}$, which is brought about through the influence of the dam on the performance of her offspring, is the cause of the covariance $\sigma_{A_0A_m}^2$. The computed values for $r_{PS_0P_{D_0}}$ and $h_0^2$ are approximately

$.01$ (from Table 20) and $.37$ (from design 1), respectively. Thus, $m_{h_m} h_0 r_{G_0G_m} = 8(.01 - .37/16) = -.08$ and $m_{h_m} r_{G_0G_m} = -.14$. Further knowledge of the values for $m_{h_m}$ and $r_{G_0G_m}$ is the main objective of design 3.

The results of the analysis of family size obtained from this design are shown in Table 21. The variance component $\sigma_{G}^2$ is the same as previously explained. The component $\sigma_{W}^2$ is the amount of the total variance.
not accounted for by the Cov (HFC). The estimate of each variance component computed from Table 21 is summarized in Table 22.

Table 21. Analysis of variance of family size for design 2

<table>
<thead>
<tr>
<th>Source of variation</th>
<th>DF</th>
<th>MS</th>
<th>EMS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between grandsires (GS)</td>
<td>207</td>
<td>38.94</td>
<td>$\sigma_{WS}^2 + \sigma_{GS}^2$</td>
</tr>
<tr>
<td>Within grandsires (W)</td>
<td>208</td>
<td>48.76</td>
<td>$\sigma_{W}^2$</td>
</tr>
<tr>
<td>Total (T)</td>
<td>415</td>
<td>43.86</td>
<td>$\sigma_{T}^2$</td>
</tr>
</tbody>
</table>

Table 22. Estimates of different variance components for family size of design 2

<table>
<thead>
<tr>
<th>Components</th>
<th>Covariances</th>
<th>Estimates of the components</th>
<th>% of the total variance</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\sigma_{GS}^2$</td>
<td>Cov(HFC)</td>
<td>0.00</td>
<td>0.00</td>
</tr>
<tr>
<td>$\sigma_{W}^2$</td>
<td>$\sigma_{T}^2 - Cov(HFC)$</td>
<td>48.76</td>
<td>100.00</td>
</tr>
<tr>
<td>$\sigma_{T}^2$</td>
<td></td>
<td>48.76</td>
<td>100.00</td>
</tr>
</tbody>
</table>

The computed value for $\sigma_{GS}^2$ was negative (-4.91) but it was assumed to be zero. The Cov(HFC) has the expectation of $1/16 \sigma_{AO}^2 + 1/8 \sigma_{AOA_m}$.

Using the estimate of $\sigma_{AO}^2$ obtained from design 1:

$$0 = (1/16)(19.76) + 1/8 \sigma_{AOA_m}$$

or

$$\sigma_{AOA_m} = 8(-1.23) = -9.84.$$
This negative covariance accounts for 20.3% of the total variance in this design. The standard error of this estimate is 25. Actually negative values of $\sigma_{GS}^2$ are possible since $1/8 \sigma_{AoAm}^2$ can be greater than $1/16 \sigma_A^2$. As will be seen later, using -4.91 results in a genetic correlation over one. Thus, for the present, zero was used for the covariance.

Design 3:

The results of the analysis of pupa weight for this design are given in Table 23. The components of variance of Table 23 are as previously defined in Table 19. Although the analyses of the data obtained from designs 2 and 3 follow the same pattern, the expectations of the variance components are not the same. This difference is brought about by the change in the type of relationships and not by the change in the degree of relationships.

Table 23. Analysis of variance of pupa weight for design 3

<table>
<thead>
<tr>
<th>Source of variation</th>
<th>DF</th>
<th>MS</th>
<th>EMS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between grandsires (GS)</td>
<td>122</td>
<td>48,335.97</td>
<td>$\sigma_e^2 + 2\sigma_{GD/GS}^2 + 4\sigma_{GS}^2$</td>
</tr>
<tr>
<td>Between $F_1$/GS</td>
<td>123</td>
<td>38,494.65</td>
<td>$\sigma_e^2 + 2\sigma_{GD/GS}^2$</td>
</tr>
<tr>
<td>$F_2$ progeny/$F_1$/GS</td>
<td>246</td>
<td>23,310.27</td>
<td>$\sigma_e^2$</td>
</tr>
<tr>
<td>Total</td>
<td>491</td>
<td>33,332.30</td>
<td></td>
</tr>
</tbody>
</table>

The covariance expression and the estimate of each variance component of Table 23 are summarized in Table 24. Although the component $\sigma_{GS}^2$ in this design is also referred to as the Cov(HFC) as in design 2,
Table 24. Estimates of variance components for pupa weight of design 3

<table>
<thead>
<tr>
<th>Components</th>
<th>Covariances</th>
<th>Estimates of the components</th>
<th>% of the total variance</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\sigma^2_{GS}$</td>
<td>Cov(HFC)</td>
<td>2,460.33</td>
<td>7.37</td>
</tr>
<tr>
<td>$\sigma^2_{GD/GS}$</td>
<td>Cov(FS) - Cov(HFC)</td>
<td>7,592.19</td>
<td>22.76</td>
</tr>
<tr>
<td>$\sigma^2_e$</td>
<td>$\sigma^2_T$ - Cov(FS)</td>
<td>23,310.27</td>
<td>69.87</td>
</tr>
<tr>
<td>$\sigma^2_T$</td>
<td></td>
<td>33,362.79</td>
<td>100.00</td>
</tr>
</tbody>
</table>

it has the expectation of $1/16 \sigma^2_{AO} + 1/4 \sigma_{AOA_m} + 1/4 \sigma^2_{Am}$ (see the Cov(05, Oγ) in the Design of Experiment section). This is different from the expectation of $\sigma^2_{GS}$ in design 2. The main objective of this design is to utilize this component in order to estimate $\sigma^2_{Am}$ (the additive genetic contribution for the maternal effect). For this purpose, estimates of the $\sigma^2_{AO}$ (from design 1) and $\sigma_{AOA_m}$ (from design 2) should also be utilized. The procedure is outlined as follows:

$$2460.33 = 1/16 \sigma^2_{AO} + 1/4 \sigma_{AOA_m} + 1/4 \sigma^2_{Am}$$

or

$$\sigma^2_{Am} = 4[2460.33 - (1/16)(13,537.00) - (1/4)(-3115.04)] = 9572.11$$

which accounts for about 29% of the total variance in this design. The standard error of this estimate is 15,814.20.

The success in estimating $\sigma^2_{Am}$ from this experiment resides in reducing the two sources of sampling errors as follows:
1. The direct sampling error associated with $\sigma_{Am}^2$ itself which in part results from the coefficient $1/4$.

2. The indirect sampling error or the sampling errors associated with the other two computed statistics ($\sigma_{A0}^2$ and $\sigma_{AOAm}$). The three statistics are not independent since $\sigma_{A0}^2$ is used to estimate $\sigma_{AOAm}$ and both are used to estimate $\sigma_{Am}^2$. But the estimates of the three covariances from which $\sigma_{A0}^2$, $\sigma_{AOAm}$, and $\sigma_{Am}^2$ are calculated are independent. This was accomplished by carrying out three designs that produced progeny by different sets of grandsires from which each covariance was independently estimated.

The problem often encountered in beef cattle data is using records of the same animals more than once in estimating the three covariances. In this case, the three equations

\[
\begin{align*}
1/4 \sigma_{A0}^2 \\
1/16 \sigma_{A0}^2 + 1/8 \sigma_{AOAm} \\
1/16 \sigma_{A0}^2 + 1/4 \sigma_{AOAm} + 1/4 \sigma_{Am}^2
\end{align*}
\]

are no longer independent of each other. There are not enough degrees of freedom for grandsires, such that they can be represented in more than one equation. Such dependency between the equations causes the number of unknowns to exceed the number of independent equations and makes it difficult to estimate the true values of the involved genetic parameters. This problem was taken into consideration in planning the design of this experiment.
The two sources of sampling errors were reduced in this experiment as much as possible by taking the following into consideration:

1. Estimating \( \sigma_{A_0}^2 \) from the combined information available on paternal half-sibs of designs 2 and 3.

2. Assigning 63\% of the total grandsires to design 2 in order to estimate \( \sigma_{A_0A_m} \) well.

3. Allotting a fairly large number of grandsires to design 3 for estimating \( \sigma_{A_m}^2 \).

4. Conducting the three designs simultaneously in order to minimize environmental variations among designs.

5. Eliminating environmental correlations by developing appropriate designs to yield relationships thought to be free of such correlations. This can readily be seen from Figure 5 (since \( r_{PSO_{PD}} \) does not include \( r_{EO_{Em}} \)). To show that \( r_{PSO_{PD}} \) does not include \( r_{EO_{Em}} \) for design 3, Figure 6 is drawn to illustrate the biometric relations between members of this design. In this figure

\[
r_{PD_{10}PD_{20}} = 1/16 h_o^2 + 1/4 m^2 h_m^2 + 1/4 mh_o h_m r_{G_oG_m}
\]

and is also free of environmental correlation, since the maternal likeness being measured has been transmitted from sire to his daughters. The phenotypic, genotypic, and the environmental values of the two dams (\( F_1 \) paternal half-sibs) and their progeny are distinguished between by
Figure 6. Path coefficient diagram showing the biometric relations between members of each grandsire group in design 3.
the use of subscripts $D_1$ and $D_2$. The rest of the symbols and notations are as previously defined for Figure 5.

The correlation, $r_{D_1D_2}$, is calculated to be .07 (see Table 24). The two other terms included in this correlation ($h^2$ and $mh_mh_o r_{G_oG_m}$) were calculated as .37 (from design 1) and -.08 (from design 2), respectively. Thus

\[
.07 = \frac{1}{16}(.37) + \frac{1}{4} m^2 h_m^2 + \frac{1}{4}(-.08)
\]
or

\[
m^2 h_m^2 = 4(.07 - \frac{1}{16}(.37) - \frac{1}{4}(-.08)) = .28.
\]

Since information on $h^2$, $mh_mh_o r_{G_oG_m}$, and $m^2 h_m^2$ is available, the genetic correlation between direct effects and maternal effects for pupa weight ($r_{G_oG_m}$ or $r_{A_oA_m}$ in terms of the additive genetic effects) can be calculated as follows:

\[
r_{G_oG_m} = \frac{mh_mh_o r_{G_oG_m}}{(h_o)(\sqrt{m^2 h_m^2})} = \frac{-.08}{(.60)(.52)} = -.26.
\]

This correlation may also be directly computed as:

\[
r_{G_oG_m} = \frac{\text{Cov}(A_oA_m)}{\sqrt{\sigma^2 A_o \sigma^2 A_m}} = \frac{-3115.04}{\sqrt{(13,537.00)(9572.11)}} = -27.
\]

The discrepancy between the two different methods is only due to the rounding errors.

The separate contribution of the two paths $m$ and $h_m$ is not evaluated since these two paths do not contribute to any relationship independent of each other. This dependency causes the number of unknowns to exceed
the number of equations.

The results of the analysis of family size for this design are presented in Table 25. The components of variance in this table are as follows:

Table 25. Analysis of variance of family size for design 3

<table>
<thead>
<tr>
<th>Source of variation</th>
<th>DF</th>
<th>MS</th>
<th>EMS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between grandsires (GS)</td>
<td>122</td>
<td>48.04</td>
<td>( \sigma_W^2 + 2\sigma_{GS}^2 )</td>
</tr>
<tr>
<td>Within grandsires (W)</td>
<td>123</td>
<td>37.85</td>
<td>( \sigma_W^2 )</td>
</tr>
<tr>
<td>Total (T)</td>
<td>245</td>
<td>42.93</td>
<td>( \sigma_T^2 )</td>
</tr>
</tbody>
</table>

defined for Table 21. The covariance expression and the estimated value of each component are listed in Table 26. Although the component \( \sigma_{GS}^2 \) in this design is also expressed as the Cov(HFC), its expectation is different from that of design 2. The expectation of this covariance is \( 1/16 \sigma_{A_o}^2 + 1/4 \sigma_{A_oA_m}^2 + 1/4 \sigma_{A_m}^2 \). Using the estimates of \( \sigma_{A_o}^2 \) (from design 1) and \( \sigma_{A_oA_m}^2 \) (from design 2), the estimate of \( \sigma_{A_m}^2 \) is obtained as...
follows:

\[ 5.10 = 1/16 \sigma^2_{A_0} + 1/4 \sigma^2_{A_0A_m} + 1/4 \sigma^2_{A_m} \quad \text{or} \quad \sigma^2_{A_m} = 4[5.10 - (1/16)(19.76) - 1/4(-9.84)] = 25.30. \]

This variance accounts for about 59% of the total variance in this design. The standard error of this estimate is 30.06.

Had the family size been defined as egg production which is expressed only in females, then the heritability estimate for such trait would have been computed as \( \frac{\mu \sigma^2_{GS}}{\sigma^2_T} \approx 0.47 \). The additive variance for egg number can only be measured from the progeny of the daughters of a group of sires. This estimate of heritability is in close agreement with the estimate computed from design 1. In either case, the estimate of heritability for family size seems to be fairly high. This may partly be due to excluding those mating groups which did not yield one offspring of each sex in each generation. Evidently survival ability plays an important role in the definition of family size as does egg production since the heritability for the direct effect of survival is 0.40 and the heritability for egg production is \( \frac{\sigma^2_{A_m}}{\sigma^2_T} = 0.59 \).

The genetic correlation between the direct effect and maternal effect is calculated as follows:

\[ r_{G_0G_m} = \frac{\sigma_{A_0A_m}}{\sqrt{\sigma^2_{A_0} \sigma^2_{A_m}}} = \frac{-9.84}{\sqrt{(19.76)(25.30)}} \approx -0.44. \]

This correlation will become -1.37 if the original computed value of \( \sigma^2_{GS}, -4.91, \) is taken into consideration (this component of variance can
be negative since it is a covariance term). The procedure is

\[-4.91 = \frac{1}{16}(19.76) + \frac{1}{8} \sigma_{AoAm}\]

or

\[\sigma_{AoAm} = 8(-6.14) = -49.12\]

and

\[5.10 = \frac{1}{16} \sigma_A^2 + \frac{1}{4} \sigma_{AoAm} + \frac{1}{4} \sigma_A^2\]

or

\[\sigma_A^2 = 4[5.10 - (\frac{1}{16})(19.76) - \frac{1}{4}(-49.12)] = 64.6\]

and

\[r_{GOGm} = \frac{\sigma_{AoAm}}{\sqrt{\sigma_{Ao}^2 \sigma_A^2}} = \frac{-49.12}{\sqrt{(19.76)(64.6)}} = -1.37\]

Since this correlation exceeds the range of -1 to +1, the previously calculated value of -0.44 is taken to be the estimate of direct-maternal genetic correlation.

To obtain an estimate of the total phenotypic variance \(\sigma_p^2\) for each trait, the average of the total variance resulting from the three designs was considered. This estimate is 35,279.87 and 47.25 for pupa weight and family size, respectively. The total variance may be expressed as

\[\sigma_p^2 = \sigma_A^2 + \sigma_m^2 + \sigma_{AoAm}^2 + \sigma_{Dm}^2 + \sigma_{Dm}^2 + \sigma_{Em}^2 + \sigma_{Bm}^2\]

To get a better picture of the relative magnitude of the three estimates, \(\sigma_A^2\), \(\sigma_{AoAm}^2\), and \(\sigma_m^2\), the corresponding value of each estimate may be expressed as the percentage of \(\sigma_p^2\). These values for \(\sigma_A^2\), \(\sigma_{AoAm}^2\), and \(\sigma_m^2\) are 38.4, -9.8, and 27.1 for pupa weight and 41.8, -20.1, and 53.5 for family size, respectively. The values 27.1% and 53.5% may be regarded as the estimate of heritability of the maternal effects for
pupa weight and family size, respectively. These estimates are also subject to a large sampling error.

Heritability estimates for the traits influenced by maternal effects may also be calculated by the method of Dickerson (1947). In this method heritability is defined as the regression of transmitting ability (direct genotypic value plus the maternal genotypic value for a trait) on individual performance. Thus,

\[ h_o^2 = \frac{(\sigma^2_{A_0} + 1.5 \sigma^2_{A_0A_m} + .5 \sigma^2_{A_m})/\sigma^2_F}{\sigma^2_F}. \]

For pupa weight,

\[ h_o^2 = \frac{13,537.00 + 1.5(-3115.04) + .5(9572.11)}{35,279.87} = .39, \]

and for family size,

\[ h_o^2 = \frac{19.76 + 1.5(-9.84) + .5(25.30)}{47.25} = .37. \]

These estimates are in close agreement with those estimates previously obtained from the paternal half-sib relationships. This is because 1.5 \( \sigma^2_{A_0A_m} \) and .5 \( \sigma^2_{A_m} \) almost cancel each other out for both traits.

The estimate of heritability was also calculated from the parent-offspring relationship. This estimate was obtained from the pooled F_1 parents used in designs 2 and 3. The covariance between average of the F_2 progeny and the measurement of their F_1 parent (either sire or dam since only one parent was measured) for each trait was doubled and divided by the average of the respective total variance. The computed values for the heritability of pupa weight and family size were .38 and .31, respectively. Although the sire-offspring and the dam-offspring relationship...
ships were pooled together and the maternal effect was not taken into account, the estimate of heritability for pupa weight is in close agreement with all previous estimates. They should be most like the heritability as defined by Dickerson (1947). The estimate of the heritability calculated for family size is somewhat lower than the estimates obtained by other methods. This may have been due to the fact that the dam-offspring relationships were more numerous in the calculation than the sire-offspring relationship and that family size is more influenced by maternal effects than pupa weight.

The standard error of the estimate of the genetic correlation may be computed according to the method outlined by Dickerson (1969) as follows:

\[
\sigma_{r_{GOGM}} = r_{GOG} \frac{V(\sigma_{AOAm}) + V(\sigma_{AO}^2) + V(\sigma_{Am}^2)}{\left(\sigma_{AOAm}^2 \right)^2 + 4(\sigma_{AO}^2)^2 + 4(\sigma_{Am}^2)^2} - \frac{\text{Cov}(\sigma_{AOAm}, \sigma_{AO}^2)}{\left(\sigma_{AOAm} \sigma_{AO}^2 \sigma_{Am}^2 \right)^{1/2}}
\]

where

\[
\text{Cov}(\sigma_{AOAm}, \sigma_{AO}^2) = \text{Cov}(8\sigma_{GS}^2 - 2\sigma_{S}^2, 4\sigma_{S}^2) = -8V(\sigma_{S}^2), \text{ and}
\]

\[
\text{Cov}(\sigma_{AOAm}, \sigma_{Am}^2) = \text{Cov}(8\sigma_{GS}^2 - 2\sigma_{S}^2, 4\sigma_{G}^2 - 3\sigma_{S}^2 - 8\sigma_{GS}^2) = -64V(\sigma_{GS}^2) + 6V(\sigma_{S}^2).
\]

Prime is used for the \(\sigma_{GS}^2\) of design 3 and no prime for the \(\sigma_{GS}^2\) of design 2.
\[
\text{Cov}(\sigma_{A_o}^2, \sigma_{A_m}^2) = \text{Cov}(4\sigma_{S}^2, 4\sigma_{G,S}^2 - 3\sigma_{S}^2 - 8\sigma_{GS}^2) = -12\sigma_{S}^2.
\]

Now all the necessary information for the computation of this standard error for both traits has been previously calculated, see Table 27.

Table 27. Summary of the results obtained from analyses of pupa weight and family size

<table>
<thead>
<tr>
<th>Estimated items</th>
<th>Estimated values for pupa weight</th>
<th>Estimated values for family size</th>
</tr>
</thead>
<tbody>
<tr>
<td>(\sigma_{A_o}^2)</td>
<td>13,537.00</td>
<td>19.76</td>
</tr>
<tr>
<td>(\sigma_{A_oA_m})</td>
<td>-3115.04</td>
<td>-9.84</td>
</tr>
<tr>
<td>(\sigma_{A_m}^2)</td>
<td>9572.11</td>
<td>25.30</td>
</tr>
<tr>
<td>(V(\sigma_{A_o}^2))</td>
<td>179,984,818.96</td>
<td>625</td>
</tr>
<tr>
<td>(V(\sigma_{A_oA_m}^2))</td>
<td>250,088,896.02</td>
<td>903.69</td>
</tr>
<tr>
<td>(V(\sigma_{A_m}^2))</td>
<td>1,742,269.06</td>
<td>7.61</td>
</tr>
<tr>
<td>(V(\sigma_{GS}^2))</td>
<td>2,703,370.98</td>
<td>9.29</td>
</tr>
<tr>
<td>(r_{G_oG_m})</td>
<td>-.27</td>
<td>-.44</td>
</tr>
</tbody>
</table>

The estimated standard errors for the genetic correlations were .96 and 1.55 for pupa weight and family size, respectively. The high values of these standard errors prohibit one from drawing many conclusions. Even the sign of the correlation is questionable.

The estimates of covariances and correlations between different relatives of designs 2 and 3 are summarized in Tables 28 and 29, respectively. The coefficients of the components calculated by the method
explained in the Design of Experiment are also given. Other components with zero coefficient are not listed.

Table 28. Estimates of different covariances and correlations between different relatives of design 2 for pupa weight

<table>
<thead>
<tr>
<th>Relatives involved</th>
<th>Estimated covariance</th>
<th>Estimated correlation</th>
<th>Components of the covariance</th>
</tr>
</thead>
<tbody>
<tr>
<td>((0_1+0_2)/2, S_1)</td>
<td>2978.50</td>
<td>.1040</td>
<td>(\sigma^2_{A_0}) (\sigma_{A_0A_m}) (\sigma^2_{A_m}) (\sigma_{D_0D_m})</td>
</tr>
<tr>
<td>((0_1+0_2)/2, D_1)</td>
<td>2606.50</td>
<td>.0840</td>
<td>1/2 1/4</td>
</tr>
<tr>
<td>((0_1+0_2)/2, D_2)</td>
<td>1023.36</td>
<td>.0356</td>
<td>1/8</td>
</tr>
<tr>
<td>((0_1+0_2)/2, S_2)</td>
<td>4296.26</td>
<td>.1756</td>
<td>1/8</td>
</tr>
<tr>
<td>((0_1+0_2)/2,(S_2+D_2)^2)</td>
<td>2659.81</td>
<td>.1223</td>
<td>1/8</td>
</tr>
<tr>
<td>((0_3+0_4)/2, D_2)</td>
<td>9746.38</td>
<td>.3466</td>
<td>1/2 5/4 1/2 1</td>
</tr>
<tr>
<td>((0_3+0_4)/2, S_2)</td>
<td>5746.47</td>
<td>.2399</td>
<td>1/4 3/4 1/2 1/4</td>
</tr>
<tr>
<td>((0_3+0_4)/2, S_1)</td>
<td>2302.68</td>
<td>.0821</td>
<td>1/8 1/4</td>
</tr>
<tr>
<td>((0_3+0_4)/2, D_1)</td>
<td>713.34</td>
<td>.0235</td>
<td>1/8 1/4</td>
</tr>
<tr>
<td>((0_3+0_4)/2,(S_1+D_1)/2)</td>
<td>1508.01</td>
<td>.0613</td>
<td>1/8 1/4</td>
</tr>
</tbody>
</table>

These results may be utilized to estimate \(\sigma_{D_0D_m}\) and the environmental correlation, \(r_{D_0D_m}\), for pupa weight. As can be seen from Tables 28 and 29, the estimated values for covariances and for correlations are not consistent in all cases. This indicates that the type of the relationships utilized and the sampling errors involved play an important role in estimating components of genetic variance and covariances. Thus, estimating genetic parameters with low sampling errors requires
Table 29. Estimates of different covariances and correlations between different relatives of design 3 for pupa weight

<table>
<thead>
<tr>
<th>Relatives involved</th>
<th>Estimated covariance</th>
<th>Estimated correlation</th>
<th>Components of the covariance</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>σ^2_{A_0}</td>
<td>σ_{A_0A_m}</td>
</tr>
<tr>
<td>(0^j+0^q)/2, D_1</td>
<td>11,817.98</td>
<td>0.4631</td>
<td>1/2</td>
</tr>
<tr>
<td>(0^j+0^q)/2, S_1</td>
<td>4,802.92</td>
<td>0.1776</td>
<td>1/4</td>
</tr>
<tr>
<td>(0^j+0^q)/2, D_2</td>
<td>932.62</td>
<td>0.0323</td>
<td>1/8</td>
</tr>
<tr>
<td>(0^j+0^q)/2, S_2</td>
<td>8,439.62</td>
<td>0.2958</td>
<td>1/8</td>
</tr>
<tr>
<td>(0^j+0^q)/2, (S_2+D_2)/2</td>
<td>4,686.12</td>
<td>0.1970</td>
<td>1/8</td>
</tr>
<tr>
<td>(0^j+0^q)/2, D_2</td>
<td>12,024.26</td>
<td>0.3919</td>
<td>1/2</td>
</tr>
<tr>
<td>(0^j+0^q)/2, S_2</td>
<td>5,386.58</td>
<td>0.1777</td>
<td>1/4</td>
</tr>
<tr>
<td>(0^j+0^q)/2, D_1</td>
<td>7,266.45</td>
<td>0.2680</td>
<td>1/8</td>
</tr>
<tr>
<td>(0^j+0^q)/2, S_1</td>
<td>4,569.48</td>
<td>0.1590</td>
<td>1/8</td>
</tr>
<tr>
<td>(0^j+0^q)/2, (S_1+D_1)/2</td>
<td>5,917.97</td>
<td>0.2584</td>
<td>1/8</td>
</tr>
</tbody>
</table>

well-suited, planned designs to yield certain relatives of sufficient numbers.

As the results indicate, the genetic correlations between the additive genetic value for direct effect and for maternal effect are negative in both traits studied. However, due to the large sampling error and elimination of some effects which may have been important (e.g. epistatic effects) in calculations, the magnitude of these correlations in reality is uncertain. Should these estimates be near the real value, the results suggest that many of the same genes have opposite effects on...
direct and maternal components of pupa weight and family size. Antagonisms of this sort have also been indicated by many other authors such as Dickerson (1947) and Dickerson and Grimes (1947) for suckling ability and economical gaining ability in swine; Koch and Clark (1955) between milking ability and both growth response from birth to weaning and weaning score in beef cattle; Young and Legates (1965) for postnatal maternal performance and fat deposition in mice; Falconer (1965) between size of litter produced by the dam and the size of the litter produced by her offspring in mice; Everett and Magee (1965) between genetic ability and maternal ability for both birth weight and gestation length in Holstein cows; Hill et al. (1966) for the calf's genetic ability and the dam's maternal ability for 180-day weight in beef cattle; Brown and Galvez (1969) between genes for prenatal growth and genes conditioning the intra-uterine environment for heavier birth weight in beef calves; etc. Several reports (e.g. Dickerson (1947), Dickerson and Grimes (1947), Koch and Clark (1955), Willham (1963), Willham (1964), Falconer (1965), etc.) have extensively discussed the consequences of such a negative correlation in a selection program.

The existence of a genetic antagonism between the two components of a character causes any successful attempt in improving one to bring about an unfavorable change in the other. Thus, direct selection for heavy pupa weight or large family size may lower the maternal ability of the dams that provide for the offspring of the succeeding generation. Since improvement in the performance of such a character in the offspring
is controlled by favorable effects of both components of that character, this antagonism may in turn offset the performance of the offspring. Thus, it may be concluded that the dams which transmit genes for heavier pupa weight and larger family size may also provide poorer nutritional substances required for the development and viability of their young which to some extent may nullify the possible improvement in these characters. This one might expect if a relatively constant supply of nutrients were available for egg production. In this case any attempt to increase family size would cause less nutrients to be available per egg. Also the nutrients might be of lower quality. The net result then would be a reduction in survival ability of the larvae that emerged from these eggs. They would be poorly supplied with the nutritive materials necessary for the early stage of their life. On the other hand, a decrease in the number of eggs would cause production of eggs richly supplied with sufficient substances for early needs resulting in well-formed embryos which develop into larvae with a high probability of survival. Although this mechanism may represent a simple and logical explanation of the results of this study, it is nothing but an interpretation. Possibly such negative correlations are only a matter of chance or the method of analysis. Possibly such results are brought about under laboratory or experimental farm conditions which bear little relationship to what goes on in nature. Further investigations need to be carried out to clarify the situation especially when most traits so far studied have indicated a negative correlation between direct and maternal effects.
One suggestion for further study might be that two selection schemes, between family and within family selection, be practiced for a trait. Provided the families are those individuals having a common maternal effect, within family selection will select primarily for the direct effect since within family the maternal effect is common. Between family selection involves both the direct and maternal effect. The two selection schemes should be carried out simultaneously and the traits such as number of eggs produced by each female, possibly the weight of the eggs, and the number of larvae and pupae surviving up to a certain age be compared among schemes. Although the problem may not be as simple as stated here, such investigation should help in the solution.

Based on the results of this study, the high negative genetic correlation between the direct and maternal effect of family size means that individuals from large families will have fewer offspring in forthcoming generations. This should make the natural populations self-regulatory as far as family size is concerned. From the evolutionary viewpoint, since natural selection operates to increase family size to protect the species, family size may be kept in check by the maternal effect of dams from large families having fewer offspring. Although it is not the intention to investigate this matter completely within the scope of this study, two comments on how natural selection may have operated follows:

Natural selection has operated in favor of large family size but the environmental factors (climatic conditions, natural enemies, competition of other organisms of the same or different species for food and
space, etc.) have opposed the increase in family size.

2. Family size is negatively correlated genetically with other traits (e.g. body weight). In this case natural selection has been directed toward the heavier weight as well as the larger family size. This also makes a self-regulatory mechanism for both traits (or several traits) to remain in some optimum state.

If as the results suggest, genotypic values for rapid pupa growth are associated with the genotypic values for poor maternal effects, an emerged larva in a poor environment (provided by the egg) may compensate for the growth that has been denied him by increased growth following emergence. Such compensatory growth may be similar to those seen in farm animals when a period of undernutrition is followed by favorable conditions. A growth of this kind is also reported to take place in mice by Young and Legates (1965). Since most of the reports in literature concern an antagonism between maternal effects and some measurements of growth in various species, whether compensatory growth is always the cause of it needs further investigation.

Developing two selected groups simultaneously, one based on between family selection and the other based on within family selection, and comparing average daily gain up to a certain age may be a way to solve this problem. Comparison of the average daily gain from emergence to pupation between the two lines of Tribolium, one selected for heavy weight and the other for light pupa weight may be helpful in solving this problem. Such tests may not result in a definite solution to the problem, since
pupa weight does not seem to be independent of family size. In this case there may exist a genetic correlation between the genotypic values for direct effects (additive, dominance, and epistatic) of pupa weight and the respective values of family size. These correlations may be either positive or negative. Thus, the change in pupa weight may also be brought about by the change in family size. In this case a technique should be developed to calculate genetic correlations between various components of two traits influenced by maternal effects. This should be very helpful in calculating an index for such pairs of traits when both direct and maternal effects can be separated (see Van Vleck, 1970).

If as the results of this study suggest, an antagonism exists between the direct effect and maternal effect of traits selection procedures other than mass selection should be planned. Several selection schemes have been proposed by several authors to prevent the deterioration of maternal performance. Dickerson (1947) suggested that sows of a line selected for good maternal performance be crossed to boars of a second line selected for rate and economy of post-weaning gains to secure the maximum performance. Legates (1968) and other authors (e.g., Falconer (1960a)) have discussed within-family selection in such situations to avoid direct selection for maternal performance. Further investigation of these methods, their applicability, and other alternatives would be of practical value.
SUMMARY

Three designs were planned and carried out simultaneously on *Tribolium castaneum* to investigate genetic maternal influences on pupa weight and family size. Design 1 included 331 sires each mated to two random dams from which one male and one female of each family were measured. The paternal half-sib correlations were 0.09 and 0.10 for pupa weight and family size, respectively. Designs 2 and 3 yielded first generation and second generation offspring from 208 and 123 grandsires, respectively. These grandsires were the sires used in design 1. Each grandsire was randomly mated to two granddams. The first generation offspring were two paternal half-sibs of different sexes in design 2 and of the same sex (females) in design 3. Of the progeny from mating each first generation individual to a random mate, one male and one female were measured. Components of variance for sires in design 1 and for grandsires in designs 2 and 3 had expectations of $1/4 \sigma_{A_o}^2$, $1/16 \sigma_{A_o}^2 + 1/8 \sigma_{A_oA_m}^2$, and $1/16 \sigma_{A_o}^2 + 1/4 \sigma_{A_oA_m}^2 + 1/4 \sigma_{A_m}^2$, respectively. $A_o$ and $A_m$ represent additive genetic effects for direct and maternal components of a character, respectively. Estimates of the components $\sigma_{A_o}^2$, $\sigma_{A_oA_m}^2$, and $\sigma_{A_m}^2$ expressed as the percentage of their corresponding total variance were 36.8, -8.7, and 28.7 for pupa weight and 39.5, -20.3, and 59.0 for family size, respectively.

These estimates expressed as the percentage of the average of the total variance resulting from the three designs were 38.4, -8.8, and 27.1 for pupa weight and 41.8, -20.1, and 53.5 for family size, respectively.
Heritability estimates from the regression of transmitting ability on individual performance were .39 and .37 for pupa weight and family size, respectively. These estimates are in close agreement with those estimates previously obtained from the paternal half-sib relationships.

Genetic correlations between direct and maternal effects were estimated to be -0.27 and -0.44 for pupa weight and family size, respectively.

These results suggest that many of the same genes have opposite effects on direct and maternal components of pupa weight and family size. The existence of a genetic antagonism between the two components of a character causes any successful attempt in improving one to bring about an unfavorable change in the other. Thus, direct selection for heavy pupa weight or large family size may lower the maternal ability of the dams which provides for the offspring of the next generation.

The estimates of the various correlations and covariances between relatives based on the information obtained from only one design were not consistent. This suggests that correctly estimating genetic parameters would require well-suited, planned designs to yield certain relatives with sufficient numbers.
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