GENETIC COVARIATION AMONG CHARACTERISTICS OF SWINE

by

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A Dissertation Submitted to the
Graduate Faculty in Partial Fulfillment of
The Requirements for the Degree of
DOCTOR OF PHILOSOPHY

Major Subjects: Animal Breeding
Genetics

Approved:

Signature was redacted for privacy.

Signature was redacted for privacy.

In Charge of Major Work

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1952

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INTRODUCTION

Only within the last few years have data from swine become available for the study of selection pressure applied, and of the response to this selection pressure. As the data accumulated it seemed to some of the workers in swine breeding research that certain economically important traits were acting stubbornly against their efforts to extend improvement. A cooperative study was initiated by workers of the Regional Swine Breeding Laboratory to study the amount of selection practiced, and its effect on improvement. The results were reported for the over-all study by Dickerson (1951), and for the Iowa station by Kottman (1952).

The selection study showed clearly that much selection had been practiced. Conclusions as to the effectiveness of the selection, however, were not so clear. Time trends and the possibility of improper corrections for extraneous influences did not warrant clear-cut conclusions, but there was little evidence that the selection was effective.

Selection is expected to increase the mean of the population each generation by an amount equal to the selection differential times the heritability (in the narrow sense). If the increase is nil in spite of sizeable selection differentials, the magnitude of heritability must be questioned. On the other hand, evidence on heritability seems fairly consistent from various experiments, and where various techniques of estimation were used.

Such a dilemma, if it really exists, requires an explanation. Lush (1949) discusses five possible explanations which are, in brief:

1. Overdominance
2. Epistasis
3. Negative genetic correlations
4. Positive selection for a character at one stage of the life cycle and negative selection at another stage of the life cycle
5. Selection for a character in one herd, or ecological niche, and against it in another.

One is not concerned with item five when considering the results from a single experiment station where any selection that is practiced for a character is generally always for or always against it. Item four was discredited as an explanation of the ineffectiveness of selection by the selection study (Dickerson, 1951). It is difficult, if not impossible with present techniques, to distinguish between overdominance and epistasis. The net effect of each, however, is to reduce the fraction of the total variance that can be additively genetic (heritability). A lot of epistatic variance could lead to an apparent dilemma, however, because epistasis would contribute something to most of the estimates of heritabilities that have been reported.

Even where the additive genetic variances are substantial for several characters, the net effect of simultaneous selection for all characters might be zero in the presence of negative genetic relationships. For example, the genes which have a positive effect on one character could have a negative effect on another character. Such genes would be alternately selected for and against and, if this selection were perfectly balanced, their frequency would not change.

It must not be overlooked, in spite of previous evidence, that heritabilities may not be far different from zero. Heritabilities could be negligible either because the hereditary variance is composed almost entirely of dominance and epistatic variance or because there is little or no hereditary variability. In the latter case gene frequencies are near 0 or 1 for genes which have any important effect on the character.

The amount of additive genetic variation for litter size and for growth in swine, and the genetic relationships between these characteristics, are investigated in this study. In addition, certain phenotypic relationships are investigated to aid in interpreting the results.

REVIEW OF LITERATURE

Production merit in swine is a composite of fecundity, mothering ability, livability, rate of gain, feed economy and carcass quality. Litter size and weight for age, which are considered in this study, have received considerable attention in the past.

The composition of the gain in weight changes gradually throughout the growing period. Growth prior to birth consists largely of structures and organs essential to life processes. After birth the body tissues exhibit marked differential growth behavior. Skeleton, muscle and fat develop in that order. McMeekan (1940) states that most of the pig's skeletal and muscular growth is made during the first 116-120 days, and that most of the later increase in weight is in fat deposition. Genetic influences on growth might then vary in importance with the stage of growth.

The conditions under which growth occurs during prenatal development, the suckling period, and after weaning also differ. The pig becomes more independent of maternal influences as development proceeds. In fact, weight or growth cannot be considered to be entirely a function of the pig since the dam also exerts considerable influence on the pig's growth from conception until weaning at least. An extreme example of maternal effect on size of offspring is the foal size in reciprocal crosses between the Shire horse and Shetland pony (Walton and Hammond, 1938). The size of foal at birth appeared to be almost completely determined by the mare. At birth the average weight of the three crossbreds from the Shetland mare was less than one-half as much as the average of the two crossbreds from the Shire mare. Although the relative difference decreased with age, the ratio was still roughly three-fourths at 38.5 months of age. Only two animals were involved in the latter comparison, however. In swine, Lush *et al.* (1934) found about 6 percent of the variation in birth weight to be hereditary, of which one-half was attributed to breed and sex. The environment common to litter mates, other than litter size, year, ration and gestation length, accounted for 29 percent. If birth weight is in part an expression of genetic qualities of the dam, a portion of the 29 percent is due to hereditary differences in maternal abilities of dams. Environment peculiar to the individual pig accounted for 47 percent of the variation.

Several studies have partitioned the variance of different measures of growth into three portions: the variance attributable to the heredity of the pig, environment common to litter mates, and environment not common to litter mates. The method of partitioning is given by Baker *et al.* (1943). The results are collected in Table 1.

Individual environment seemed to account for the largest portion of the variation in growth throughout the studies cited. Environment common to litter mates accounted for more of the variance early in life than later, but even at 168 days of age litter environment accounted for about 20 percent of the total variance in weight. It

Table 1

The Percent of the Variance in Different Measures of Growth Attributed to Heredity, to Litter Environment and to Environment Peculiar to the Individual

Measure	Heredity						Litter environment					Individual environment				
Weight for age at	(1)[a]	(2)	(3)	(5)	(6)	(7)	(1)	(2)	(3)	(5)	(7)	(1)	(2)	(3)	(5)	(7)
Birth	0	-	5				49	34	40			51	59	55		
56 days	15	-	14		7		48	32	33			37	46	54		
112 days	28	0	14[b]		(8)		34	30	24[b]			38	60	62[b]		
168 days	25	26	24[c]	20[c]	43[c]	34[c]	24	15	14[c]	24[c]	26[c]	51	54	62[c]	56[c]	40[c]
Gain between	(1)	(2)	(4)				(1)	(2)	(4)			(1)	(2)	(4)		
Birth and 21 days	7						54					39				
21 and 56 days	15	-					46	31				39	47			
56 and 84 days	20	18	18				30	24	8			50	57	74		
84 and 112 days	31	26					21	21				48	50			
112 and 140 days	4	28					40	14				56	54			
140 and 168 days	13	25					26	16				61	59			
Rate of gain from	(2)	(4)	(7)				(2)	(4)	(7)			(2)	(4)	(7)		
Birth to slaughter	22						16					57				
56 days to slaughter	40	18	31				8	9	16			50	73	53		
112 days to slaughter		14						14					72			

[a]The numbers in parenthesis indicate the source of the figures beneath them as follows: (1) Baker et al. 1943. Duroc. 55 sires and 252 litters.; (2) Nordskog et al. 1944. Seven inbred lines. 64 sires and 340 litters.; (3) Krider et al. 1946. Hampshire. 34 sires and 91 litters.; (4) Blunn and Baker, 1947. Duroc. 41 sires and 152 litters.; (5) Whatley, 1942. Poland China. 30 sires and 260 litters.; (6) Bywaters, 1937. Poland China. 20 sires and 271 litters.; (7) Dickerson, 1947. Poland China, Landrace and PL crossbreds. 62 sires and 174 litters.; (8) Whatley and Nelson, 1942. Duroc. 193 litters.

[b]150 days.

[c]180 days.

was pointed out before that a portion of litter environment would be genetic if part of the variation in weight is the result of genetic differences in maternal abilities. Dickerson (1942) partitioned the variance to illustrate this point. Heredity of the pig accounted for little variation at birth, but increased in importance with age to be about equal with litter environment at 168 days. The method of computing the hereditary fraction amounted to multiplying the paternal half-sib correlation in a random breeding population by four.

The hereditary variance has also been estimated by regression (b) of offspring (y) on dam (d), sire(s), and midparental average $\frac{s+d}{2}$. These estimates are collected in Table 2.

The Illinois selection experiment for rapid and slow growth rate (Krider _et al_. 1946) furnished another estimate of the heritable portion of variation in growth. The averages of four estimates of the heritable fraction of intra-line differences were 16 and 19 percent for 150 and 180-day weights, respectively. Each estimate was obtained by dividing the mean difference between unselected progeny of the high and low line by the total amount of selection practiced up to that generation. There were four generations.

To obtain evidence on the genetic and environmental relationships between different phases of growth, Hazel _et al_. (1943) utilized three periods: gain to 56 days (X_1), gain from 56 to 112 days (X_2), and gain from 112 to 168 days (X_3). The authors considered gain to be a function of a genetic source (G_i), a litter environment source (L_i), and a residual source (E_i). The correlations of the variables were found to be:

Table 2

The Percent of the Variance in Different Measures of Growth Attributed to Heredity

Measure	Method Obtained $2b_{ys}$		$2b_{yd}$						$b_{y\frac{s+d}{2}}$
Weight for age at	(8)[a]	(9)	(2)	(5)	(6)	(8)	(9)	(10)	(9)
Birth		-38	14				12		-23
72 days		4			10[c]		16	-19[b]	9
180 days	22			62		-32		14	
Gain between			(2)						
21 and 56 days			-2						
84 and 112 days			10						
140 and 168 days			10						
Rate of gain from	(9)		(2)	(9)	(10)				(9)
weaning to slaughter	44		22	58	31				43

[a]The numbers in parenthesis indicate the source of the figures beneath them as follows: (2) Nordskog et al., 1944. Seven inbred lines. 312 dams.; (5) Whatley, 1942. Poland China. 150 dams.; (6) Bywaters, 1937. Poland China. 85 dams.; (8) Whatley and Nelson, 1942. Duroc. 193 litters.; (9) Dickerson and Grimes, 1947. Duroc. 62 sires and dams.; (10) Comstock et al., 1942. Poland China and Minn. No. 1. 172 dams.

[b]56 days.

[c]Correlation between offspring and dam at 60 days.

	X_2	X_3		G_2	G_3		L_2	L_3		E_2	E_3
X_1	.44	.16	G_1	.71	.45	L_1	.54	.26	E_1	.31	.02
X_2		.55	G_2		.70	L_2		.19	E_2		.66

Although the genetic variance constituted only about one-fifth of the observed variance in each of the three periods (15, 28 and 17 percent, respectively), the genetic correlations were larger than the corresponding environmental correlations. This indicated that genes with persistent effects were responsible for much of the genetic variation. Although no figures were presented, Nordskog _et al_. (1943) found negative genetic correlations between genotypes for growth before and after weaning.

Dickerson and Grimes (1947) correlated three phases of growth of the progeny (birth weight (X), 72 day weight (Y), daily gain from 72 days to 225 pounds (Z)) and pounds of feed consumed per pound of gain (W) with the same measures of the parents. The correlations were:

		Dam				Sire				Midparent			
		X	Y	Z	W	X	Y	Z	W	X	Y	Z	W
	X	.06	.18	.10	-.15	-.19	-.17	-.14	.09	-.23	.02	-.08	-.00
Progeny	Y	-.15	.08	.12	.09	-.00	.02	.13	-.11	-.18	.09	.23	-.09
	Z	-.00	.16	.29	-.07	.10	.11	.22	-.22	.15	.25	.43	-.29
	W	-.07	-.14	-.19	.01	-.10	-.20	-.26	.23	-.23	-.31	-.40	.26

These correlations were actually deduced from regressions of progeny on parent to eliminate the bias in correlations introduced by selecting the parent and averaging the progeny. Dickerson and Grimes concluded that these correlations indicated that the genes which cause rapid post-weaning growth rate also tend to be responsible for good uterine nutrition but poor suckling ability. Also the more rapid post-natal growth of a female enables her to provide better uterine nourishment for her litter but is associated with slower inherited prenatal growth rate in her pigs. The authors also found indications that the same genetic factors tend to increase 72 day weight and the rate and efficiency of gain thereafter. There was more evidence for genetic antagonism between good suckling ability and inherently lower feed requirements than between good suckling ability and rapid growth rate.

Dickerson (1947) presented evidence which, in general, confirmed the findings of Dickerson and Grimes (1947). Heritable differences in rate of gain due to the pig's own genes were more largely in fat deposition than in bone and muscle growth. Rapid fat deposition and low feed requirements tended to be caused by the same genes. A tendency for poor suckling ability to be caused by the same genes responsible for rapid fat deposition and low feed requirements was strongly suggested.

Weight of gilts at mating time was found by Stewart (1945b) to be associated with a larger number of pigs born to them. Age and weight of the gilt at mating, however, accounted for only 4 percent of the variance in litter size at birth. Wentworth and Aubel (1916) compared 1,000 litters of "large type" Poland Chinas with 1,100 litters of the "small type" and found no difference in litter size. Their data probably included sow litters. Also, herd differences could have been present and influential. Warnick _et al_. (1951) found correlations of -.54 and -.58 between weight at 56 days and age at puberty and weight at 154 days and age at puberty, respectively, within lines. This means that there is a positive correlation between rate of growth and rate of maturity. Although the correlations were negative within lines, the correlation between 154 day weight and age at puberty was .45 between lines. There were only 5 inbred lines, however: three inbred Chester White lines, 1 inbred Yorkshire line, and one inbred line originating from a cross between Chester White and Yorkshire. Ovulation rate increased with order of heat period. When the breeding season is postponed until most of the gilts have expressed heat, one might expect the heaviest gilts within lines to farrow somewhat larger litters, since they would on the average be bred in later heat periods. A positive correlation between the average age at puberty and the average weight at 154 days of the lines (Warnick et al., 1951) indicates that a positive relationship between weight at mating and litter size need not exist among gilts of different lines of breeding. In fact, it suggests the opposite relationship.

Estimates of the heritable portion of variation in litter size are collected in Table 3. Most of the estimates have been obtained by dam-offspring regressions or correlations. In all cases litter size is considered to be a function of the dam of the litter.

The entries in Table 3 under the heading, "correlation of records by the same dam", are estimates of repeatability, rather than of heritability. According to Lush and Molln (1942) repeatability measures the fraction of the difference between single records of dams which

Table 3

Percent of the Variance in Litter Size Estimated to be Hereditary

Measure of litter size	Method of Estimation														
	Daughter-dam regression			Daughter-dam correlation					Paternal half-sib correlation		Correlation of records by the same dam				
	(1)[a]	(2)	(8)	(3)	(4)	(5)	(5)	(9)	(2)	(9)	(1)	(2)	(6)	(7)	(9)
Number of pigs born	25	14				34	44		16		24	13	17	20	
Number of pigs born alive	24	16	22	18	17			4	18	11	21	13			12
Number at weaning	19		32							12	25		17	21	7
Number at 168 days	42										22				

[a]The numbers in parenthesis indicate the following authorities: (1) Blunn and Baker, 1949. Duroc. 528 daughters.; (2) Stewart, 1945a. 12 Poland China lines and 2 crossbred lines. 42 sires and 245 litters. 250 dams and 475 daughters.; (3) Morris and Johnson, 1932. Poland China. 1,035 daughter-dam pairs from the herdbook.; (4) Rommel and Phillips, 1906. Poland China. 6,145 daughter-dam pairs from the herdbook.; (5) Henke, 1935. 89 Tamworth litters. 71 Berkshire litters.; (6) Lush and Molln, 1942. Various breeds. 7,415 litters born.; (7) Hetzer *et al.* 1940. Chester White. 362 litters.; (8) Cummings *et al.* 1947. Inbred lines. 279 dams. (9) Korkman, 1947. Large White and Landrace. 573 dams. 2,292 litters.

will most likely be found between other records of those dams. The other entries in Table 3 would not be expected to be as large as the estimates of repeatability unless (a) there were no permanent effects of environment and (b) there were no dominance or epistatic deviations. Under those conditions, differences between any two entries in Table 3 result from sampling variation.

Size of litter has been noted to have an adverse effect on average weight of the pigs in the litter at birth by Carmichael and Rice (1920) and Lush et al. (1934) and at weaning by Bywaters (1937). The regression is curvilinear, however. In each case, pigs in litters of 1 and 2 were not as heavy as those in litters of 3, and there was a general decline in average weight with size of litter greater than 3. Lush et al. (1934) found litter size to account for 7 percent of the variation in birth weight, while Bywaters (1937) found only 3 percent of the variation in weaning weight to be attributed to litter size. Smith and Donald (1939) found post-weaning growth in pigs to be independent of litter size.

CONCEPTS AND DEFINITIONS

The basic concepts and definitions relating to genetic parameters have been developed and clarified by Fisher, Wright and Lush during the past 35 years. Those pertinent to this study will be outlined.

Partitioning the Phenotypic Variance

The deviation of the phenotypic expression of a character from the population average can be considered to be the sum of a hereditary effect and an effect attributable to environment and interaction between the heredity and environment. This consideration is written symbolically as $X = \mu + H + E$, where X is the phenotype, μ is the population average, H is the hereditary effect and E is the deviation of H and μ from X. If the heredities are randomly distributed among the environments, the phenotypic variance is $\sigma^2_X = \sigma^2_H + \sigma^2_E$.

In his study on the correlation between relatives Fisher (1918) found that these correlations could be expressed simply in terms of certain components of σ^2_H. He named these components (1) additive genetic variance, (2) variance due to dominance deviations from the additive scheme and (3) variance due to epistatic deviations from the additive and dominance scheme. These components will be symbolized as σ^2_G, σ^2_D and σ^2_I, respectively.

The partitioning of the hereditary variance can be exemplified by two loci, each with two allelic genes (A, a and B, b). This excludes the case of multiple alleles. If the coupling and repulsion double heterozygotes are considered to be identical phenotypically, there are then 9 genetic types as in Table 4a.

Table 4a

Mean Yields and Frequencies of the Nine Genetic Types for Two Loci, Each with Two Genes

AABB	AABb	AAbb	
K_{11}	K_{12}	K_{13}	$K_{1.}$
f_{11}	f_{12}	f_{13}	$f_{1.}$
AaBB	AaBb	Aabb	
K_{21}	K_{22}	K_{23}	$K_{2.}$
f_{21}	f_{22}	f_{23}	$f_{2.}$
aaBB	aaBb	aabb	
K_{31}	K_{32}	K_{33}	$K_{3.}$
f_{31}	f_{32}	f_{33}	$f_{3.}$
$K_{.1}$	$K_{.2}$	$K_{.3}$	$K_{..}$
$f_{.1}$	$f_{.2}$	$f_{.3}$	$f_{..}$

The K's and f's in Table 4a are the yields and frequencies of the indicated types, respectively. A dot (.) indicates a marginal frequency or mean.

$$f_{i.} = \sum_j f_{ij}$$

$$K_{i.} = \sum_j f_{ij} K_{ij}/f_{i.}$$

If the frequencies at one locus are uncorrelated with frequencies at another locus (algebraically, all $f_{ij} = f_{i.}f_{.j}$), the partitioning of the variance can be accomplished with the eight orthogonal scales (W's) in Table 4b. Regardless of linkage, frequencies at one lucus will be uncorrelated with frequencies at another locus in a freely interbreeding population whose gene frequency is not changing. Frequencies of different loci will also remain uncorrelated in a population where mates are related only through descent, and where gene frequency does not change. If frequencies are correlated, which they would be under assortative mating, the following partitioning of the variance does not hold. Other causes of correlated frequencies are discussed by Lush (1948) under the subject of disequilibrium.

The orthogonal scales serve here the same purpose as orthogonal comparisons or polynomials (Snedecor, 1946) in computing a portion of the total variance for each degree of freedom. The requirements for orthogonal comparisons, as they are usually presented when frequencies are equal, are that the sum of the terms in each is zero and that the sum of the products of corresponding terms of any two comparisons is zero. The first requirement accounts for the name comparison and also insures that deviations about the mean are compared. The second requirement accounts for the comparisons being called orthogonal, which means simply that they are uncorrelated. The term scale is used here to differentiate the present case, where frequencies are unequal but proportional to the marginal frequencies, from the general presentation of orthogonal comparisons, where frequencies are equal. The requirements for the orthogonal scales are that the sum of the frequencies times the

Table 4b

Mean Yields, Frequencies, and Eight Orthogonal Scales Which are Utilized in Partitioning the Hereditary Variance

Scale	Genetic type								
	AABB	AABb	AAbb	AaBB	AaBb	Aabb	aaBB	aaBb	aabb
K	K_{11}	K_{12}	K_{13}	K_{21}	K_{22}	K_{23}	K_{31}	K_{32}	K_{33}
f	f_{11}	f_{12}	f_{13}	f_{21}	f_{22}	f_{23}	f_{31}	f_{32}	f_{33}
W_1	$2v$	$2v$	$2v$	$v-u$	$v-u$	$v-u$	$-2u$	$-2u$	$-2u$
W_2	$1/f_{1.}$	$1/f_{1.}$	$1/f_{1.}$	$-2/f_{2.}$	$-2/f_{2.}$	$-2/f_{2.}$	$1/f_{3.}$	$1/f_{3.}$	$1/f_{3.}$
W_3	$2y$	$y-x$	$-2x$	$2y$	$y-x$	$-2x$	$2y$	$y-x$	$-2x$
W_4	$1/f_{.1}$	$-2/f_{.2}$	$1/f_{.3}$	$1/f_{.1}$	$-2/f_{.2}$	$1/f_{.3}$	$1/f_{.1}$	$-2/f_{.2}$	$1/f_{.3}$
W_5	$4vy$	$2v(y-x)$	$-4vx$	$2y(v-u)$	$(v-u)(y-x)$	$-2x(v-u)$	$-4uy$	$-2u(y-x)$	$4ux$
W_6	$2v/f_{.1}$	$-4v/f_{.2}$	$2v/f_{.3}$	$v-u/f_{.1}$	$-2(v-u)/f_{.2}$	$(v-u)/f_{.3}$	$-2u/f_{.1}$	$4u/f_{.2}$	$-2u/f_{.3}$
W_7	$2y/f_{1.}$	$(y-x)/f_{1.}$	$-2x/f_{1.}$	$-4y/f_{2.}$	$-2(y-x)/f_{2.}$	$4x/f_{2.}$	$2y/f_{3.}$	$(y-x)/f_{3.}$	$-2x/f_{3.}$
W_8	$1/f_{11}$	$-2/f_{12}$	$1/f_{13}$	$-2/f_{21}$	$4/f_{22}$	$-2/f_{23}$	$1/f_{31}$	$-2/f_{32}$	$1/f_{33}$

terms in each scale is zero and that the sum of the frequencies times the products of corresponding terms of any two scales is zero. There are eight scales or partitions of the variance, one partition for each of the eight separate degrees of freedom in a 3 x 3 table.

The partition of the variance corresponding to any particular scale is found by first computing the products of K and the corresponding term of the particular scale. These products in turn are multiplied by their corresponding frequencies and then summed. The sum is squared and divided by the sum of the frequencies times the square of corresponding terms of the scale. This yields the desired partition of the variance. For example, the eighth partition of the variance, σ_8^2 is:

$$\sigma_8^2 = \left(K_{11}\frac{1}{f_{11}}f_{11} - K_{12}\frac{2}{f_{12}}f_{12} + K_{13}\frac{1}{f_{13}}f_{13} - K_{21}\frac{2}{f_{21}}f_{21} + K_{22}\frac{4}{f_{22}}f_{22} - K_{23}\frac{2}{f_{23}}f_{23} + K_{31}\frac{1}{f_{31}}f_{31} - K_{32}\frac{2}{f_{32}}f_{32} + K_{33}\frac{1}{f_{33}}f_{33}\right)^2 \Big/ \left(f_{11}\frac{1}{f^2_{11}} + f_{12}\frac{4}{f^2_{12}} + f_{13}\frac{1}{f^2_{13}} + f_{21}\frac{4}{f^2_{21}} + f_{22}\frac{16}{f^2_{22}} + f_{23}\frac{4}{f^2_{23}} + f_{31}\frac{1}{f^2_{31}} + f_{32}\frac{4}{f^2_{32}} + f_{33}\frac{1}{f^2_{33}}\right) = \frac{(\mathrm{cov}\ K\ W_8)^2}{\sigma^2_{W_8}} = b^2_{K\,W_8}\,\sigma^2_{W_8}\ .$$

where cov K W_8 is the covariance between K and the eighth orthogonal scale and b_{KW_8} is the regression of K on the eighth orthogonal scale. The other seven partitions are found in a similar manner.

The choice of this particular set of scales (there are an indefinitely large number of sets of eight scales which would be orthogonal and would partition the variance between nine items of data), and of course this particular partitioning of the variance, depended entirely on this set being most useful in separating the variance into parts ascribable to each locus separately and to joint effects (interactions) of the two different loci which cannot be partitioned logically among the individual loci. The particular scales chosen to separate the marginal variance for each locus into an additive and a dominance portion have been shown in the past to be most useful for expressing simply the correlation between parent and offspring. It turns out that the scales representing the interactions among the loci also allow one to express simply the correlations among the interactions of the parents and those of the offspring. This after all is the primary purpose of introducing the orthogonal scales.

The new symbols u, v, x and y in Table 4b are the frequencies of the genes A, a, B and b respectively. The first two scales are concerned only with the means for the rows in Table 4a and thus only with the marginal variance for the A locus. For example, the means and frequencies for the A locus are:

Mean	$K_{1.}$	$K_{2.}$	$K_{3.}$
Frequency	$f_{1.}$	$f_{2.}$	$f_{3.}$
W_1	$2v$	$v-u$	$-2u$
W_2	$1/f_{1.}$	$-2/f_{2.}$	$1/f_{3.}$.

The marginal variance of K can be broken into two parts, one part being the variance due to the regression of the marginal means on the linear scale, W_1, and the other part being the variance due to deviations from this regression. The variance due to the regression is the additive genetic variance for the A locus and the variance due to deviations from the regression is the variance due to dominance at the A locus. The additive genetic deviations for the A locus, g's, where $g = b_{KW_1}W_1$, are the same as the deviations of Wright's (1935) G values from their mean, $\bar{G}$. The dominance deviations, d's, where $d = b_{KW_2}W_2 = K_m - \bar{K} - g$, are the same as Wright's dominance deviations, where K_m represents marginal means for the A locus. The variance among the g's is of course the additive genetic variance and the variance among the d's is the dominance variance. Rather than introduce a new notation for each of the partitions of the variance, they will be numbered the same as the orthogonal scales. For example,

$$\sigma_1^2 = b_{KW_1}^2 \sigma_{W_1}^2 = \rho_{KW_1}^2 \sigma_K^2$$

is the additive genetic variance caused by A and

$$\sigma_2^2 = b_{KW_2}^2 \sigma_{W_2}^2 = \rho_{KW_2}^2 \sigma_K^2$$

is the dominance variance caused by A. These two sum to the marginal variance for the A locus. In a similar manner σ_3^2 and σ_4^2 are the additive and dominance variances, respectively, which sum to the marginal variance for locus B; i.e. the variance between the means for the columns in Table 4a. The partitioning of the variance to this point is identical with that of Fisher (1918) and Wright (1935).

The last four components (σ_5^2 through σ_8^2) account for the remaining or epistatic portion of the variance of K. The naming of the epistatic components is founded on the relationships among the orthogonal scales:

$$W_5 = W_1 \times W_3 \quad \text{(additive x additive)}$$

$$W_6 = W_1 \times W_4 \quad \text{(additive x dominance)}$$

$$W_7 = W_2 \times W_3 \quad \text{(dominance x additive)}$$

$$W_8 = W_2 \times W_4 \quad \text{(dominance x dominance).}$$

The epistatic variance, therefore, consists of four parts: σ_5^2 is the additive by additive, σ_6^2 is the additive in A by dominance in B, σ_7^2 is the dominance in A by additive in B and σ_8^2 is the dominance by dominance. Fisher (1918) and other subsequent workers in expressing the epistatic variance for two loci in a population mating at random, obtained one epistatic component which is actually the sum of the four components indicated above.

In summary then the t^{th} partition of the variance of K is

$$\sigma_t^2 = b_{KW_t}^2\, \sigma_{W_t}^2 \qquad (t = 1 \ldots 8)$$

and

$$\sigma_K^2 = \sum_t b_{KW_t}^2\, \sigma_{W_t}^2 = \sum_t \sigma_t^2 ,$$

where b_{KW_t} is the regression of K on the t^{th} orthogonal scale.

The orthogonal scales are useful in another way which may not have been noticed thus far. For example, the additive genetic deviations for the A locus are $b_{KW_1}W_1$. In finding the correlation of these deviations with any other variable, b_{KW_1} may be omitted except for sign (+ or -), since it is a constant multiplier for each deviation. Therefore, the correlation of K with another variable may be found by computing the correlation of each scale with the other variable.

Where e's indicate the following comparisons among the yield values:

$$e_{11} = K_{11} - K_{12} - K_{21} + K_{22} \qquad e_{12} = K_{12} - K_{13} - K_{22} + K_{23}$$

$$e_{21} = K_{21} - K_{22} - K_{31} + K_{32} \qquad e_{22} = K_{22} - K_{23} - K_{32} + K_{33} \quad ,$$

the eight regression coefficients and variances of the orthogonal scales under random mating are:

Orthogonal scale	b_{KW}	σ^2_W
W_1	$u(K_{1.} - K_{2.}) - v(K_{3.} - K_{2.})$	$2uv$
W_2	$u^2v^2(K_{1.} - 2K_{2.} + K_{3.})$	$1/u^2v^2$
W_3	$x(K_{.1} - K_{.2}) - y(K_{.3} - K_{.2})$	$2xy$
W_4	$x^2y^2(K_{.1} - 2K_{.2} + K_{.3})$	$1/x^2y^2$
W_5	$uxe_{11} + uye_{12} + vxe_{21} + vye_{22}$	$4uvxy$
W_6	$x^2y^2 \,[u(e_{11} - e_{12}) + v(e_{21} - e_{22})]$	$2uv/x^2y^2$
W_7	$u^2v^2 \,[x(e_{11} - e_{21}) + y(e_{12} - e_{22})]$	$2xy/u^2v^2$
W_8	$u^2v^2x^2y^2(e_{11} - e_{12} - e_{21} + e_{22})$	$1/u^2v^2x^2y^2$

The extension to the 3 loci case is apparent. There are 3 additive components, 3 dominance components and 20 epistatic components. The 20 epistatic components are 3 a by a, 6 a by d, 3 d by d, 1 a by a by a, 3 a by a by d, 3 a by d by d and 1 d by d by d (a = additive, d = dominance). For many purposes the epistatic components may be combined. They are designated separately because they present different properties in the correlations among relatives which will be discussed later.

The partitioning of the variance for any number of loci with two alleles each would follow a pattern similar to that outlined. Although the method presented here does not lend itself to multiple alleles, Fisher (1918) did partition the marginal variance for a locus with any number of alleles in a random mating population into an additive part and a dominance part. Correlations among the additive deviations and dominance deviations of relatives were the same as those for two alleles at a locus. It may be possible to partition the epistatic variance for multiple alleles into components which will bear definitions similar to those where only two alleles are considered. Should the correlations among these epistatic deviations of relatives be the same as those when only two alleles are considered, the development herein is general for any number of alleles. At present, this must remain as a conjecture.

The phenotype is now expressed as

$$X = \mu + G + D + I + E,$$

where μ is the mean, G is the sum of the additive genetic deviations from all loci, D is the sum of the dominance deviations from all loci, I is the sum of all epistatic deviations, and E is the deviation of H and μ from X. The variance of X is $\sigma_X^2 = \sigma_G^2 + \sigma_D^2 + \sigma_I^2 + \sigma_E^2$, since G, D and I are independent by definition and the genotypes occur among the environments at random in this example.

Another quantity, heritability, is often estimated rather than the additive genetic variance. Heritability in the narrow sense is defined by Lush (1948) to be the fraction of the phenotypic variance that is additively genetic:

$$h = \frac{\sigma_G^2}{\sigma_X^2}$$

Relationship Between Two Characters

One is generally interested in more than one important characteristic in each individual. It is desirable, therefore, to consider two phenotypes observed on the same individual.

$$X_1 = \mu_1 + H_1 + E_1$$
$$X_2 = \mu_2 + H_2 + E_2$$

The two phenotypes are X_1 (for example, weight) and X_2 (quality). The definitions and variances of each are the same as those previously outlined.

The relationship between X_1 and X_2 may be considered from the standpoint of correlation.

$$\rho_{X_1X_2} = \frac{1}{\sigma_{X_1}\sigma_{X_2}}\left[\text{cov } H_1H_2 + \text{cov } E_1E_2\right] ,$$

since the H's are uncorrelated with the E's in this example. The covariance between H_1 and H_2 (cov H_1H_2) is the sum of the covariances between the component parts of each $H(G_1, D_1, I_1$ and $G_2, D_2, I_2)$. The relationship among these parts may be exemplified again by two loci with two genes each and where the frequencies of loci are uncorrelated. Let K be the mean yield for one characteristic and K' be the mean yield for the other characteristic. The mean yields, frequencies and the first orthogonal scale are:

K	K_{11}	K_{12}	K_{13}	K_{21}	K_{22}	K_{23}	K_{31}	K_{32}	K_{33}
K'	K'_{11}	K'_{12}	K'_{13}	K'_{21}	K'_{22}	K'_{23}	K'_{31}	K'_{32}	K'_{33}
f	f_{11}	f_{12}	f_{13}	f_{21}	f_{22}	f_{23}	f_{31}	f_{32}	f_{33}
W_1	$2v$	$2v$	$2v$	$v-u$	$v-u$	$v-u$	$-2u$	$-2u$	$-2u$

The remaining W's are the same as those in Table 4b.

It can be shown that

$$\rho_{KK'} = \frac{\sum\limits_{t}\sum\limits_{t'} \rho_{(b_{KW_t}W_t)(b_{K'W_{t'}}W_{t'})}\ \sigma_t\ \sigma'_{t'}}{\sigma_K\ \sigma_{K'}} \quad .$$

$$(t, t' = 1 \ldots 8)$$

Since

$$\rho_{(b_{KW_t}W_t)(b_{K'W_{t'}}W_{t'})} = \pm\ \rho_{W_tW_{t'}} \quad ,$$

and since

$$\begin{aligned} \rho_{W_tW_{t'}} &= 1 \text{ if } t = t' \\ &= 0 \text{ if } t \neq t', \end{aligned}$$

it follows that

$$\rho_{KK'} = \frac{\sum\limits_{t} \pm\ \sigma_t \sigma'_t}{\sigma_K\ \sigma_{K'}} \quad ,$$

where σ_t and σ'_t are square roots of the t^{th} partition of the variances of K and K', respectively. Plus or minus is determined by b_{KW_t} and $b_{K'W_t}$; plus, if the two regressions are of the same sign and minus if they are of different signs.

Since there is no correlation between a component of one phenotype and the other components of the other phenotype, when the frequencies of loci are uncorrelated, the covariance between the hereditary components of the two phenotypes may be written as

$$\mathrm{cov}\, H_1H_2 = \rho_{G_1G_2}\sigma_{G_1}\sigma_{G_2} + \rho_{D_1D_2}\sigma_{D_1}\sigma_{D_2} + \rho_{I_1I_2}\sigma_{I_1}\sigma_{I_2},$$

and the correlation between the phenotypes is

$$\rho_{X_1X_2} = (\rho_{G_1G_2}\sigma_{G_1}\sigma_{G_2} + \rho_{D_1D_2}\sigma_{D_1}\sigma_{D_2} + \rho_{I_1I_2}\sigma_{I_1}\sigma_{I_2} + \rho_{E_1E_2}\sigma_{E_1}\sigma_{E_2})\frac{1}{\sigma_{X_1}\sigma_{X_2}}.$$

A genetic correlation was defined by Hazel (1943) to be $\mathrm{cov}\, G_1G_2/\sigma_{G_1}\sigma_{G_2}$, which of course is the same as $\rho_{G_1G_2}$. When the frequencies among loci are uncorrelated the genetic correlation can be written as

$$\rho_{G_1G_2} = \frac{\sum_i \pm \sigma_{g_{1i}}\sigma_{g_{2i}}}{\sqrt{\sum_i \sigma^2_{g_{1i}}}\ \sqrt{\Sigma\sigma^2_{g_{2i}}}}$$

where $\sigma^2_{g_{1i}}$ is the additive genetic contribution to $\sigma^2_{G_1}$ for the i^{th} locus, and $\sigma^2_{g_{2i}}$ is the additive genetic contribution to $\sigma^2_{G_2}$ for the i^{th} locus. Of course, $\sigma^2_{g_{1i}}$ or $\sigma^2_{g_{2i}}$ is zero for those loci which affect one character but not the other. When frequencies among loci are uncorrelated, which they would be in a random mating population or in a population where mates were related only through descent and gene frequencies were not changing, a genetic correlation can result only from pleiotropic effects of genes. Pleiotropic effects are considered here to be a multiplicity of effects coming from a common cause (genes at a locus), and whether pleiotropy is "genuine" or "spurious" (Gruneberg 1938) makes no difference.

If the frequencies of loci are correlated, two characters may be genetically correlated for this reason. Lush (1948) discusses this aspect of a genetic correlation, particularly with respect to linked genes. Lush also discusses causes of this correlation or disequilibrium (in his terminology) in the gametic array.

The correlation between dominance deviations is written in a manner similar to that of $\rho_{G_1G_2}$,

$$\rho_{D_1D_2} = \frac{\sum_i \pm \sigma_{d_{1i}}\sigma_{d_{2i}}}{\sqrt{\sum_i \sigma^2_{d_{1i}}}\sqrt{\sum_i \sigma^2_{d_{2i}}}} \quad .$$

The correlation between the epistatic deviations is more cumbersome to write. There are 3^n-2n-1 epistatic components, where n loci are involved. Rather than write the correlation in the same manner as $\rho_{G_1G_2}$ and $\rho_{D_1D_2}$, it will be put in the following form,

$$\rho_{I_1I_2} = \frac{\sum_k \rho_{I_{1k}I_{2k}}\sigma_{I_{1k}}\sigma_{I_{2k}} + \rho_{I_{1d}I_{2d}}\sigma_{I_{1d}}\sigma_{I_{2d}}}{\sigma_{I_1}\sigma_{I_2}} \; .$$

$$(k = 2,3 \ldots n)$$

where I_{1k} is the sum of all epistatic deviations for trait 1 which are entirely of the additive sort and involve k loci, and I_{1d} is the sum of all epistatic deviations for trait 1 which involve dominance for one or more factors. Similar definitions hold for I_{2k} and I_{2d}. The relationship between the partitions of I are:

$$I_1 = \sum_k I_{1k} + I_{1d}$$
$$I_2 = \sum_k I_{2k} + I_{2d} \; .$$

Although the partitioning of the epistatic variance may be beyond the limits of estimation techniques, the utility of this type of partitioning will be seen in expressing the correlations between parent and offspring.

Correlation Between Parent and Offspring

The biometric relations between parent and offspring are given by Fisher (1918) and Wright (1921 and 1935). Under random mating the correlation between the additive deviations is one-half, and between the dominance deviations is zero.

The orthogonal scales may again be invoked to obtain these relationships for a random mating population where gene frequency is not changing. Let K_p and K_o be the yields of the parents and offspring respectively, and let W_t be the t^{th} orthogonal scale for the parents and $W'_{t'}$ be the t'^{th} orthogonal scale for the offspring. Of course, the offspring have the same yield values and orthogonal scales as the parents. The different notation simply designates whether the value or scale is used for the parent or offspring. Since

$$K_p = \overline{K} + \sum_t b_{K_p W_t} W_t, \quad (t = 1 \ldots 8)$$

and

$$K_o = \overline{K} + \sum_{t'} b_{K_o W'_{t'}} W'_{t'}, \quad (t' = 1 \ldots 8)$$

the correlation between K_p and K_o may be expressed as

$$\rho_{K_pK_o} = \frac{\sum_t \sum_{t'} \rho_{(b_{K_pW_t}W_t)(b_{K_oW'_{t'}}W'_{t'})} \sigma_t \sigma_{t'}}{\sigma_{K_p} \sigma_{K_o}} .$$

Remember that $\sigma_t^2 = b_{KW_t}^2 \sigma_{W_t}^2$.

Now,

$$\rho_{(b_{K_pW_t}W_t)(b_{K_oW'_{t'}}W'_{t'})} = \pm \rho_{W_tW'_{t'}} ,$$

because the regression coefficients are constants as far as the correlation is concerned. The sign of the correlation, $\rho_{W_tW'_{t'}}$ is the same as that of the product of the two regression coefficients. It is necessary then to determine the correlations between the eight orthogonal scales of the parent and the eight orthogonal scales of the offspring. This is done by constructing a nine by nine joint distribution or association (Fisher, 1918) table for parent and offspring. In a random mating population with no selection the correlations, which take values other than 0, among the scales of the parent and those of the offspring are:

$$\rho_{W_1W'_1} = 1/2$$
$$\rho_{W_3W'_3} = 1/2$$
$$\rho_{W_5W'_5} = 1/4.$$

Then

$$\rho_{K_pK_o} = \frac{\frac{1}{2}\sigma_1^2 + \frac{1}{2}\sigma_3^2 + \frac{1}{4}\sigma_5^2}{\sigma_K^2} .$$

It will be recalled that σ_1^2 and σ_3^2 are the additive variances for locus A and locus B, respectively, while σ_5^2 is the additive by additive epistatic variance. The only correlation, therefore, between the epistatic deviations involving two loci of parent and offspring is one-fourth for the additive by additive kind. A logical extension of these findings is that the correlation among epistatic deviations involves only the additive kind. For example, with 3 loci, there are 3 a, 3 a by a and 1 a by a by a partitions, and the remaining 19 partitions involve dominance for one or more factors. It has been found that the correlation between the one-factor or additive genetic deviations (a) of parent and offspring is one-half, that the correlation between the two-factor additive or a by a epistatic deviations of parent and offspring is one-fourth, and that the correlations between partitions involving dominance for two loci of parent and offspring are zero. Although not shown, the 3-loci case is believed to include only one other type of partition that is correlated between parent and offspring. This partition is the three factor additive or a by a by a epistatic deviations, and the correlation is one-eighth. A complete generalization for random mating and no selection, then, is that the correlations between the deviations of parent and offspring involve only the additive sort, and that the correlation is $(\frac{1}{2})^n$ where n is the number of factors or loci involved in the deviations under question.

The correlation between epistatic deviations then can be variable with a maximum of one-fourth. However, one would expect the correlation to be much less, since it involves only a portion of the possible epistatic deviations, and since the correlation, for the correlated portion involving more than two loci, is less than one-fourth.

The correlation between the phenotype of the parent and that of the offspring is

$$\rho_{X_pX_o} = \frac{\frac{1}{2}\sigma_G^2 + \sum_k (\frac{1}{2})^k \sigma_{I_k}^2}{\sigma_X^2} , \quad k = (2,3 \ldots n),$$

and

$$\frac{\sum_k (\frac{1}{2})^k \sigma_{I_k}^2}{\sigma_I^2} = \rho_{I_pI_o} \leq \frac{1}{4} ,$$

where $I = \sum_k I_k + I_d$ as in the previous section. The correlation between one trait in the parent and another in the offspring is

$$\rho_{X_{1p}X_{2o}} = \frac{\frac{\rho_{G_1G_2}}{2} \sigma_{G_1}\sigma_{G_2} + \sum_k (\frac{1}{2})^k \rho_{I_{1k}I_{2k}} \sigma_{I_{1k}}\sigma_{I_{2k}}}{\sigma_{X_1}\sigma_{X_2}} .$$

Epistasis may contribute then to the correlation between one trait in the parent and another in the offspring, as well as to the correlation between the same trait in the parent and offspring.

Although of no particular bearing on this study, the correlations among the yield values of half-sibs and full-sibs, in a random mating population where gene frequency is not changing, were computed for the two loci case. The method of computation was similar to that for parent and offspring. The half-sib correlation turned out to be

$$\rho_{\frac{K_s}{2}\frac{K_{s'}}{2}} = \frac{\frac{1}{4}\sigma_1^2 + \frac{1}{4}\sigma_3^2 + \frac{1}{16}\sigma_5^2}{\sigma_K^2} ,$$

and the full-sib correlation was

$$\rho_{K_sK_{s'}} = \frac{\frac{1}{2}\sigma_1^2 + \frac{1}{4}\sigma_2^2 + \frac{1}{2}\sigma_3^2 + \frac{1}{4}\sigma_5^2 + \frac{1}{8}\sigma_6^2 + \frac{1}{8}\sigma_7^2 + \frac{1}{16}\sigma_8^2}{\sigma_K^2} .$$

Only the additive kind of deviations are correlated between half-sibs as in the case of parent and offspring, while all deviations are correlated between full-sibs. These results lead to an even broader generalization than before. If one determines the correlation between the one factor additive deviations of two relatives to be b and the correlation between the one factor dominance deviations of these relatives to be c, then the correlation between a particular epistatic deviation of these two relatives is

$$(b)^{n_1} (c)^{n_2} ,$$

where n_1 is the number of loci involving additive and n_2 is the number of loci involving dominance in the particular epistatic deviation. For example, b = 1/2 and c = 1/4 for full-sibs. The correlation between a by d epistatic deviations of full-sibs is $(\frac{1}{2})(\frac{1}{4}) = 1/8$. The correlation for a by a is $(\frac{1}{2})^2(\frac{1}{4})^0 = \frac{1}{4}$, and the correlation for d by d is $(\frac{1}{2})^0(\frac{1}{4})^2 = 1/16$.

The influence of inbreeding on the relationship between parent and offspring is given by Wright (1921) for the additive genetic deviations. The correlation is then

$$\rho_{G_pG_o} = \frac{1 + 2F + F'}{2\sqrt{(1+F)(1+F')}} \quad .$$

where F is the inbreeding of the offspring and F' is the inbreeding of the parent. If there were no dominance and epistatic variance, the additive genetic variation increases in proportion to (1+F). Regardless of dominance and epistasis, the correlation of additive deviations between parent and offspring is 1.0 on complete inbreeding. The dominance variation disappears as F goes to one, but not linearly with F.

No simple generalization of the consequences of inbreeding with respect to epistatic deviations seems possible. When inbreeding is complete, the epistatic deviations become entirely the additive by additive kind and the correlation of these deviations between parent and offspring is one. Although not shown, the correlation between the additive kind of epistatic deviations of the parent and those of the offspring in an inbreeding population would logically follow the same pattern as those in a random breeding population, since the frequencies of loci are still uncorrelated in a population where mates are related only through descent. This correlation is then

$$\left[\frac{1 + 2F + F'}{2\sqrt{(1 + F)(1 + F')}}\right]^n \quad .$$

where n is the number of loci involved in the additive kind of epistatic deviations.

In contrast to what happens to the whole population of many more or less distinct inbred lines as inbreeding increases, it is desirable to consider the relationship between parent and offspring within inbred lines. In general, considering the whole population,

$$\rho_{G_pG_o} = \rho_{G_sG_s}\frac{\sqrt{1 + F}}{\sqrt{1 + F'}} \quad , \qquad \text{(Lush, 1948)}$$

where $\rho_{G_sG_s}$ is the correlation between additive genetic values of full-sibs. The sires and dams are considered to be equally inbred. When an analysis is done entirely within lines, the correlation among full-sibs is obtained from the following argument. If the parents within the line have the same inbreeding, and are no more related to their mates than to the rest of the parents in the line, the overall or population correlation among genetic values of non-sib members of the same line will be $2F / (1 + F)$, while the overall correlation between full-sibs is $(1 + 2F + F') / 2(1 + F)$, (Dickerson, 1942). The additive genetic variance in the population at a given time can be partitioned into 3 parts:

Between lines	$\rho\,\sigma^2_G$
Between litters in lines	$(\rho_{G_sG_s} - \rho)\,\sigma^2_G$
Between litter mates	$(1 - \rho_{G_sG_s})\,\sigma^2_G$
Total	σ^2_G

where ρ is the population correlation among non-sibs within the same line. The intraline correlation between full-sibs is

$$\rho'_{G_sG_s} = \frac{\rho_{G_sG_s} - \rho}{1 - \rho} = \frac{1 - 2F + F'}{2(1 - F)} = \frac{1}{2}\left(1 - \frac{F - F'}{1 - F}\right) .$$

This is approximately $\frac{1}{2}$ if F and F' are not very different. The inbreeding coefficient of parent and offspring cannot be very different in animals in a regular inbreeding system. On substituting, the intraline correlation between parent and offspring is

$$\rho'_{G_pG_o} = \frac{1 - 2F + F'}{2(1 - F)} \frac{\sqrt{1 + F}}{\sqrt{1 + F'}} ,$$

which is even closer to $\frac{1}{2}$ than the full-sib correlation, since F' will generally be a little smaller than F.

It appears then, for all practical purposes, that the genetic relationships among relatives within inbred lines are about the same as those under random mating. The same generalization is reached by considering the process of inbreeding to be a random fixation of certain genetic effects for each line. The correlations among the remaining additive genetic effects of relatives within a line would then be similar to those obtained when mating is random. Such a generalization would not be valid when considering large lines in which there are sublines or closely related groups or when mating was assortative within lines. Although the genetic relationships among relatives within inbred lines are similar to those for random mating, the phenotypic relationships are different. As was previously pointed out, the genetic variance within a line is reduced by inbreeding. For example, when there is no dominance or epistasis, the correlation between parent and offspring in a random breeding population is

$$\rho_{X_oX_p} = \frac{1}{2} \quad \frac{\sigma_G^2}{\sigma_G^2 + \sigma_E^2} \quad ,$$

while the intra-line parent-offspring correlation is

$$\rho'_{X_oX_p} = \frac{1}{2} \; \frac{(1 - F)\ \sigma_G^2}{(1-F)\ \sigma_G^2 + \sigma_e^2} \quad .$$

The intra-line phenotypic correlation, $\rho'_{X_oX_p}$, is therefore somewhat less than that in a random breeding population.

Selection for Several Characteristics

The progress one can expect from selection for several characters may best be illustrated by the principles involved in a selection index. The selection index was developed by Smith (1936) and Hazel (1943) as a criterion for effecting maximum genetic improvement. The index's more immediate utility, however, is in pointing out the problems confronting the animal breeder and the information that he needs.

A selection index is a linear function of the observed phenotypes:

$$Y = \sum_i b_i\ X_i \ ,$$

where Y is the index, X_i is the observation of the i^{th} character and b_i is the weight given X_i in the index. If one is selecting solely on such an index, the amount of genetic change in the i^{th} character, G_i, which may be expected to accompany a given amount of selection for Y, is obtained from the regression of G_i on Y,

$$B_{G_i Y} = \frac{\sum_j b_j \operatorname{cov} G_i G_j}{\sigma_Y^2}$$

When selection is wholly on Y, the intensity of such selection for Y, or the selection differential for Y may be written as $z\,\sigma_Y/p$, where z/p is the difference in standard deviations between the mean Y of the selected portion (p) of the parents and the average Y of the original unselected parents. Then the expected improvement in G_i from selecting entirely on Y is

$$\begin{aligned} \hat{G}_i &= \frac{z}{p}\,\sigma_Y\,B_{G_i Y} \\ &= \frac{z}{p}\,\frac{\sum_j b_j \operatorname{cov} G_i G_j}{\sigma_Y} \qquad \text{(Morley, 1950).} \qquad (1) \end{aligned}$$

The expected selection differential for an individual trait is found in a similar manner to be

$$\begin{aligned} \hat{X}_i &= \frac{z}{p}\,\sigma_Y\,B_{X_i Y} \\ &= \frac{z}{p}\,\frac{\sum_j b_j \operatorname{cov} X_i X_j.}{\sigma_Y} \qquad (2) \end{aligned}$$

Since the quantity $z\,\sigma_Y/p$ in Equation (1) is positive the genetic improvement in a trait will be proportional to

$$\sum_j b_j \text{ cov } G_i G_j = \sigma_{G_i} \left[b_i \sigma_{G_i} + \sum_j b_j \rho_{G_i G_j} \sigma_{G_j} \right].$$

It is evident that selection based on any sort of an index will be fruitless in effecting genetic improvement of a character either if (1) the genetic variance of that character is zero or (2) if the term in the brackets is zero. Selection could actually cause deterioration in the character since $\rho_{G_i G_j}$ may be negative and large enough to make the whole term within the brackets negative.

Knowing the progress that can be made through selection, then, depends on evaluating adequately the additive genetic variation for each characteristic and the genetic covariation among the characteristics.

Methods of Estimating Genetic Parameters

Lush (1940 and 1948) describes and discusses several methods of estimating heritability. The most popular methods for animals are: (1) correlation of offspring and parent, or regression of offspring on parent if parents are selected, (2) ratio of the difference between a high and low line to the amount of selection practiced in a selection experiment and (3) full-sib and half-sib correlations.

The estimation of genetic correlations has received less attention than heritability. Hazel *et al*. (1943) presented a method which utilizes paternal half-sib components of covariance and variance. Hazel (1943) indicated another method utilizing parent-offspring regressions.

The techniques which utilize the resemblance between parent and offspring are used in this study. It has been shown that the correlation between parent and offspring is

$$\rho_{X_oX_p} = \frac{1}{2}\ \frac{\sigma_G^2}{\sigma_X^2} + \leq \frac{1}{4}\ \frac{\sigma_I^2}{\sigma_X^2}\ .$$

This is equal to one-half of heritability,

$$h = \frac{\sigma_G^2}{\sigma_X^2}\ ,$$

only if there is no epistatic variance, or if the epistasis present is of the kind that is uncorrelated between parent and offspring. Therefore, twice the parent-offspring correlation may be a biased estimate of heritability because of epistasis and the bias is always in the direction of making the estimate too large.

Since parents are generally selected in a breeding program, offspring on parent regression is more often used as an estimator than the correlation. The regression is unbiased by selection of the parents, where parents are selected only on the characteristic for which the regression is computed, and where the relationship between phenotypes of parents and offspring is linear.

Hazel's (1943) method of estimating the genetic correlation is

$$r_{G_iG_j} = \sqrt{\frac{b_{i_2j_1} \cdot b_{j_2i_1}}{b_{i_2i_1} \cdot b_{j_2j_1}}} = \sqrt{\frac{\mathrm{cov}\ i_2j_1 \cdot \mathrm{cov}\ j_2i_1}{\mathrm{cov}\ i_2i_1 \cdot \mathrm{cov}\ j_2j_1}} \qquad (3)$$

where b is the regression coefficient, i and j are the i^{th} and j^{th} characters and 2 and 1 are the offspring and parent, respectively. Again, regressions are used instead of correlations because regressions are unbiased by selection of the parents. The regression coefficients or covariances in the numerator may be of opposite signs in some cases. Rationalizing that this discrepancy is the result of a fortuitous sample, the following form has been used:

$$r_{G_iG_j} = \frac{cov_{i_2j_1} + cov_{j_2i_1}}{2\sqrt{cov_{i_2i_1} \cdot cov_{j_2j_1}}} \quad \text{or} \quad \frac{cov_{i_2j_1} + cov_{j_2i_1}}{2\sqrt{\sigma^2_{G_i} \cdot \sigma^2_{G_j}}} \tag{4}$$

where $\sigma^2_{G_i}$ and $\sigma^2_{G_j}$ are estimated in some manner. This seems not to be a very advisable procedure, particularly when genetic variances are estimated in an unbiased manner and the covariances may be biased by selection. Also it is possible that the covariances are measuring slightly different things. This point will be dealt with later.

The genetic correlation in (3) may also be biased by epistasis. The correlation, written in terms of the expected values of the regressions, is

$$r_{G_iG_j} = \sqrt{\frac{\left(\frac{\rho_{G_iG_j}}{2} \sigma_{G_i}\sigma_{G_j} + \rho_{I_{i1}I_{j2}}\sigma_{I_i}\sigma_{I_j}\right)\left(\frac{\rho_{G_iG_j}}{2} \sigma_{G_i}\sigma_{G_j} + \rho_{I_{i1}I_{j2}}\sigma_{I_i}\sigma_{I_j}\right)}{\left(\frac{\sigma^2_{G_i}}{2} + \rho_{I_{i1}I_{i2}}\sigma^2_{I_i}\right)\left(\frac{\sigma^2_{G_j}}{2} + \rho_{I_{j1}I_{j2}}\sigma^2_{I_i}\right)}}$$

where $\rho_{I_{i1}I_{j2}}$ is the correlation between the epistatic deviations of the i^{th} trait in the parent with the j^{th} trait in the offspring, and $\rho_{I_{i1}I_{i2}}$ is the correlation between the epistatic deviations of i^{th} trait in the parent and the same trait in the offspring. The limits of $\rho_{I_{i1}I_{i2}}$ and $\rho_{I_{j1}I_{j2}}$ are 0 and $\frac{1}{4}$, while the limits of $\rho_{I_{i1}I_{j2}}$ are $-\frac{1}{4}$ and $\frac{1}{4}$. The type of bias in this procedure cannot be predicted. Of course, when there is no epistatic variance, the method estimates the desired correlation.

Sampling errors for methods (1) and (2), using large sample techniques, are given by Ercanbrack (1952) and Rae (1950), respectively.

Selection experiments also present an approach to the genetic relationship among different characters. It will be recalled from the previous section that the expected gain for the i^{th} trait from selecting on an index is

$$\hat{G}_i = \frac{z}{p} \; \frac{\sum_j b_j \text{ cov } G_i G_j}{\sigma Y} \quad ,$$

and the expected selection differential is

$$\hat{X}_i = \frac{z}{p} \; \frac{\sum_j b_j \text{ cov } X_i X_j}{\sigma Y} \quad .$$

If selection is solely on one characteristic, X_1, the expected gains and selection differentials are

$$\hat{G}_1 = \frac{z}{p} \; \frac{\sigma^2_{G_1}}{\sigma_{X_1}} \quad ,$$

$$\hat{X}_1 = \frac{z}{p} \, \sigma_{X_1} \quad ,$$

$$\hat{G}_{i\neq 1} = \frac{z}{p} \; \frac{\text{cov } G_i G_1}{\sigma_{X_1}}$$

$$\hat{X}_{i\neq 1} = \frac{z}{p} \; \frac{\text{cov } X_i X_1}{\sigma_{X_1}}$$

The observed gain divided by the selection practiced for the trait under selection,

$$\frac{\text{gain}}{\text{selection}} = \frac{\hat{G}_1}{\hat{X}_1} = \frac{\sigma^2_{G_1}}{\sigma^2_{X_1}} ,$$

estimates heritability for that trait. The observed gain of another trait divided by the observed gain of the trait under selection,

$$\frac{\hat{G}_{i/1}}{\hat{G}_1} = \frac{\text{cov } G_i G_1}{\sigma^2_{G_1}} = \beta_{G_i G_1} ,$$

estimates the regression of the additive genetic values of the observed character on the additive genetic values of the one selected for. Two selection experiments afford an estimate of the genetic correlation in that

$$\rho_{G_i G_j} = \sqrt{\beta_{G_i G_j} \beta_{G_j G_i}}$$

It should be noted that, even with large sampling techniques, an unbiased estimate of heritability and the genetic regression is obtained only when selection has been practiced on a single characteristic in one experiment.

THE DATA

The data used in this study came from records of twelve inbred lines of Poland China swine and one Danish Landrace inbred line developed and maintained by the Iowa Agricultural Experiment Station in cooperation with the U.S.D.A. Regional Swine Breeding Laboratory.

History of Animals

The foundation Poland China herd, later designated as the S line, was brought together at the Iowa station in 1930. It consisted of eight sows which were then in pig to four different boars, 19 gilts, two boar pigs and one boar, purchased from various breeders of purebred Poland China swine.

Twelve of the foundation animals were slightly inbred, but the inbreeding was less than 4 percent for any one of these. Pedigrees were traced to 1925 as a base date for the inbreeding coefficient. The highest relationship between any pigs in the foundation herd was 32 percent for non-litter mates and 51 percent for three pigs which were litter mates. The herd has been closed to outside animals ever since the Fall of 1930.

In the Fall of 1937, a two-sire line, H, and three one-sire lines, E, I and J, were separated from the original four-sire herd which was continued as the S line.

Additional purebred stock, consisting of 35 sows and four boars, was purchased in 1938 from various breeders. Out of this purchased stock were developed the one-sire C, D, F and G lines. Also in 1938, another one-sire line, K, was separated from the S line.

Two two-sire Poland China lines were established in 1938. They were the A line, made by combining K and G, and the B line, established by combining C and F.

A Danish Landrace line was started in 1934. The foundation animals consisted of four sows and two boars, but all the descendants of one sow were eventually discarded. These animals were imported from Denmark in cooperation with the United States Department of Agriculture. This line, L, has been continued as a closed two-sire line.

The dates of origin and the sources of the foundation stock for the inbred lines are summarized in Table 5. Although the original plan was to use the number of sires indicated in Table 5 for each line, breeding difficulties and other unforeseeable circumstances often prohibited strict adherence to the plan.

The H and K lines were discarded in the summer of 1947 because of poor performance in their own phenotypes. The I and J lines were merged in 1948. The new line which resulted from this cross was not included in this study. The F line was discarded in 1949.

Tentative selections of breeding stock at weaning were based on the individuality of the pig and the production record of its dam. Until the fall of 1943, pigs upon reaching a weight of 225 pounds

Table 5

Dates of Origin and Sources of the Inbred Lines of Swine Developed at the Iowa Experiment Station

Lines	Foundation Stock	Date of Origin	Date First Inbred Litters Farrowed	No. Sires	No. Dams
Poland					
S	Purchased	1930	1931	4	40
A	K x G	1938	1940	2	20
B	G x F	1938	1940	2	20
C	Purchased	1938	1939	1	10
D	Purchased	1938	1939	1	10
E	S	1937	1938	1	10
F	Purchased	1938	1939	1	10
G	Purchased	1938	1939	1	10
H	S	1937	1938	2	20
I	S	1937	1938	1	10
J	S	1937	1938	1	10
K	Purchased	1938	1939	1	10
Landrace					
L	Imported from Denmark	1934	1934	2	20

were described and scored by several members of the animal husbandry staff independently, including usually the project leader himself, and the scores were averaged for each animal and considered in the selections.

Beginning with litters farrowed in the fall of 1938 a selection index was introduced. This index gave two-thirds credit to weight at 180 days and one-third credit to the pig's score at 225 pounds. The dam's productivity was included by using factors based on the number of pigs farrowed and the number and weight of offspring at three weeks and at weaning (Lush and Molln, 1942). The index was modified in 1940, so that forty percent of the credit was based on weight at 180 days; 20 percent on market score at 225 pounds; and 40 percent on the productivity of the dam. Provision was made for full-sib credits or penalities. After a gilt weaned her first litter, her productivity was added to the index, which served as a basis for any subsequent decisions about whether she should be kept to produce a second litter.

Another modification in the index was made in 1941 in accordance with the findings of Hazel (1943). The new index was:

$$\text{Index} = .5W - .06S + 1.0\,P + B$$

W = weight at 180 days

S = score at 225 pounds

P = dams productivity (Lush and Molln, 1942)

B = sib-credit for weight and score computed on a sliding scale according to the number of sibs (Hazel, 1943).

In 1943 W was changed to weight at 154 days and market score was dropped from the index, which then took the form: Index = $(.65\ W + P + B)(1.0 - 0.1\ N)$, where N is the number of defects in the litter, and the factor .1 N is an arbitrary deduction. Such defects include crytorchidism, umbilical hernia, scrotal hernia, blindness and atresia ani.

A comprehensive account of the amount of selection practiced is given by Kottman (1952).

Complete breeding and farrowing records were kept on each line. By the 1938 spring season, in which were taken the first data used in this study, pig weights were taken at birth, and at approximately 3 weeks, 8 weeks and 6 months of age. In 1943 the 6-month weight was changed to 5 months. Litter size at the various ages was computed from the number of weights recorded. Birth weights of all pigs dead or alive were recorded and the sex of each pig was noted at that time. The inbreeding of each litter was entered on the litter sheets.

Characters Studied

The characters chosen for study in this investigation were:

(1) size of litter at birth, ...n_b ,

(2) size of litter at 56 days, ... n_{56} ,

(3) size of litter at 154 days, ... n_{154} .

(4) weight of pig at 56 days, ... w_{56} ,

(5) weight of pig at 154 days, ... w_{154} .

Size of litter at birth included all pigs born.

Adjustment of Data

Since the eight-week and five-month weights were sometimes taken a few days earlier or later than these exact ages, the actual weights were adjusted for age. Eight-week weights were adjusted to a standard age of 56 days by the formula developed by Whatley and Quaife (1937):

$$W = Z\left(\frac{41}{X - 15}\right),$$

where W = adjusted 56-day weight,

Z = actual weight,

X = actual age in days when weighed.

Five-month weights were corrected to 154 days by a formula derived by Lush and Kincaid (1943):

$$W = Z\left(\frac{142.5}{0.0032143\,X^2 + 0.58X - 23}\right),$$

where W = adjusted 154-day weight,

Z = actual weight,

X = actual age in days when weighed.

Prior to the fall of 1942 weights were recorded at approximately 180 days instead of the 154 days used later. These weights were first adjusted to 180 days according to Whatley's (1942) findings:

$$W = Z\left(\frac{180-60}{X-60}\right),$$

where W = 180-day adjusted weight,

Z = actual weight,

X = actual age in days when weighed.

Since this study included weights from both periods, the 180-day weights were in turn reduced to 154-day weights by the 154-day adjustment formula. Each 154-day or 180-day weight was actually based on two different weights taken about two weeks apart. Each of these weights was adjusted and the two adjusted weights were then averaged.

Other extraneous sources of variation, which allow partial statistical control, are the general differences between lines, years, seasons, ages of dams, inbreeding of dam and litter and sex of the animal. The data were not corrected for these items. Instead, the analyses were done within lines, years, seasons and ages of dam. This should remove all additive and non-additive contributions from these sources. However, the estimates obtained may not be entirely free from the influences of these sources. This point will be dealt with later.

Sex of the animal was ignored entirely. Bywaters (1937) found sex to account for only 0.2percent of the variance in weaning weight. While sex is more important in influencing later weights, it was found by Whatley (1942) to contribute only 2 percent to the total variance of 180-day weights. Sex has even less influence on number in the litter. Vernon (1948) found mortality from birth to 21 days to be .41 for boars and .38 for gilts. Mortality in later periods was larger for boars than for gilts but the difference was extremely small. That the contribution of sex to the variation in litter size is small may be seen by considering, as an extreme case, the situation where litter size at birth is constant and where one-half the litters are of one sex and the other half are of the other sex. If litter size at birth were 8 and if the difference in mortality between the sexes were .03, the sex contribution to the total variance of a later litter size would be .029. Varying litter size at birth should not alter the results appreciably. The fact that the actual sex ratio varied from litter to litter would reduce the sex contribution still further. Such small sources of variation, although real, were not deemed worth adjustment. This is particularly true for the influence of sex on weight since the method of genetic analysis averages out most of the contributions of sex.

The effects of inbreeding of litter and of dam have been reported by Blunn and Baker (1949), Stewart (1945), Dickerson _et al._ (1946) and Dickerson _et al._ (1947). The findings of the first three of these are generally in agreement with those of Dickerson _et al._ (1947), in which, for each 10 percent rise in inbreeding of litter, there was a decline in litter size of 0.2 pigs at birth and 0.5 pigs at 56 and 154 days, no decline in weight to 56 days and a decline of 3.6 pounds at 154 days. Such corrections would, at the most, remove about 1 percent of the total variance in each characteristic in this study. The above authors report that the declines accompanying the inbreeding of the dam were generally less than those from the inbreeding of the litter. Although the phenotypic effects of inbreeding were ignored, the genetic consequences of inbreeding were utilized when interpreting the genetic analyses.

ESTIMATES OF PHENOTYPIC PARAMETERS

For this part of the study data from 9,147 pigs weaned in 1,980 litters were used. The data were entered on punched cards with classifications for line, individual, sire, dam, inbreeding of individual, age of the dam, and the size of the litter at birth. The identifications of individuals, sires and dams included their year and season of birth.

The means of the various characters for lines, ages of dam, years and seasons are given in Table 6. Comparisons among these means may not yield an unbiased estimate of the real difference between the groups for which these means are listed. One line, for example, may have more fall litters than another and the difference between the means of these two lines may, therefore, be due in part to a difference between fall and spring litters. To obtain unbiased comparisons among these means would require adjusting them by a least squares analysis to correct or allow for differences among other classifications. However, comparisons among the means are not of great importance in this study.

Further analyses were conducted within groups. In each group all animals were born in the same line, year and season, and had dams of the same age to the nearest one-half year. Such a grouping is termed LYSA. There were 531 LYSA groups.

Table 6

Phenotypic Means for Lines, Ages of Dam, Years and Seasons

Grouping	No. of litters	Means						
		n_b	n_{56}	n_{154}	w_{56}	w_{154}	Age of dam (years)	Inbreeding of litter (%)
Line								
S	439	7.43	5.06	4.76	30.5	137	1.37	29.9
A	210	7.77	5.12	4.85	28.4	140	1.21	29.8
B	191	6.17	4.23	4.02	30.0	137	1.30	25.9
C	136	7.97	4.68	4.35	24.2	120	1.42	37.0
D	104	7.12	4.07	3.79	27.8	130	1.51	35.1
E	123	6.32	3.87	3.43	28.5	129	1.39	50.2
F	94	6.12	4.31	3.95	29.3	127	1.59	33.2
G	105	7.90	4.90	4.61	26.7	140	1.39	37.4
H	142	6.70	3.96	3.57	29.4	134	1.38	35.7
I	99	7.62	4.45	4.03	29.3	129	1.42	39.2
J	94	6.66	4.35	4.11	30.6	135	1.36	48.7
K	66	7.83	4.05	3.47	27.8	130	1.41	38.1
L	177	8.19	5.12	4.45	32.3	140	1.49	27.4
Age of dam in years								
1.0	1179	6.64	4.30	3.93	27.0	131	1.00	34.6
1.5	340	7.49	5.02	4.61	31.0	134	1.50	32.3
2.0	326	8.50	5.17	4.91	32.8	143	2.00	35.3
2.5	70	8.43	5.10	4.91	32.4	139	2.50	29.4
3.0	48	9.63	5.00	4.71	32.5	139	3.00	30.1
3.5	10	9.60	4.50	3.90	32.7	139	3.50	24.1
4.0	3	9.33	4.67	4.67	30.5	121	4.00	15.3
4.5	1	11.00	7.00	7.00	39.1	140	4.50	24.0
5.0	3	12.33	5.33	5.33	38.8	161	5.00	11.0
Year								
1938	108	8.19	5.44	5.18	33.4	131	1.57	19.5
1939	144	8.28	5.49	5.15	31.1	136	1.36	19.7
1940	154	7.73	5.00	4.76	32.3	148	1.39	23.5
1941	245	7.11	4.78	4.50	30.8	140	1.40	26.8
1942	195	7.11	4.98	4.72	28.8	136	1.24	30.4
1943	198	7.10	4.60	4.17	28.9	139	1.35	34.7

Table 6 (continued)

Grouping	No. of litters	Means						
		n_b	n_{56}	n_{154}	w_{56}	w_{154}	Age of dam (years)	Inbreeding of litter (%)
<u>Year</u>								
1944	181	6.80	4.25	3.73	26.0	119	1.44	37.2
1945	199	7.11	4.47	3.97	27.7	129	1.40	39.3
1946	173	7.22	4.42	4.01	28.3	132	1.32	41.3
1947	123	6.73	3.31	3.00	24.3	117	1.20	44.1
1948	94	7.32	4.74	4.38	28.0	131	1.48	44.8
1949	80	6.70	3.58	3.21	26.7	136	1.13	45.8
1950	86	7.29	4.45	4.30	31.1	146	1.60	48.4
<u>Season</u>								
Spring	1596	7.28	4.58	4.24	28.9	136	1.33	34.7
Fall	384	7.19	4.78	4.38	30.7	130	1.55	30.4
Total or Average	1980	7.26	4.62	4.27	29.3	135	1.38	33.9

The variances of and covariances among the litter sizes were obtained from the sums of squares and products which were first found within LYSA groups and then pooled. These variances and covariances are given in Table 7. The simple correlations, computed in the usual manner, are also presented in Table 7.

Table 7

Variances of and Covariances and Simple Correlations Among Litter Sizes

Character	d.f.	Variance - Covariance			Correlation	
		n_b	n_{56}	n_{154}	n_{56}	n_{154}
n_b	1449	4.69	2.10	1.90	.52	.48
n_{56}	1449		3.49	3.20		.94
n_{154}	1449			3.34		

With the use of Bartlett's test, as described by Snedecor (1946), variances for all litter sizes were found to be heterogeneous. The probability of such differences being chance ones is five percent or less. The test was not applied to all groups simultaneously, but to various classifications. For example, sums of squares were pooled for each line, and the variances among lines were tested for heterogeneity. This same procedure was carried out for years, for seasons and for ages of dam. The tests showed significant heterogeneity for all of the above classifications, except seasons.

Sources of variation which act multiplicatively can cause heterogeneity of variance, provided the means vary from group to group. However, sources of variation within groups may differ markedly from group to group. For example, the temperature may remain relatively stable throughout a particular farrowing season, while for another season it may fluctuate widely. Mortality would increase for litters born in extremely cold spells with inadequate heating facilities. Diseases may be prevalent for only part of the season. Pigs in other seasons may be relatively free of diseases. Such sources of variation and others that could be mentioned, many of which do not lend themselves to experimental control or statistical analysis, can cause heterogeneity of variance.

For a given litter size at birth, the litter size at weaning may be approximately binomially distributed, provided mortality does not differ genuinely from litter to litter. However, Kincaid (1946) found evidence that mortality to weaning was not independent of the litter. Mortality would be expected to vary more from litter to litter than the binomial distribution indicates because litter mates prior to weaning are much more contemporary than individuals chosen at random from the same LYSA group. Also, litter mates are more alike genetically and would respond more alike to diseases and adverse conditions than randomly chosen individuals. In addition to the litter differences in mortality among those litters which are of the same size at birth, many workers have found mortality to differ with the size of litter at birth. In the present analysis variation among sizes of litter at weaning included all litters within a LYSA group, many of which differed in size at birth. Just what sort of distribution size of litter at weaning follows under these conditions is not clear. The suggestion of a binomial distribution, however, leads one to suspect that the mean and variance are correlated. The correlation between the mean and variance is not large when mortalities are near the middle of the range (.5). Here mortality was about .36 from birth to 56 days but only .08 from 56 days to 154 days. That a slight correlation does exist is indicated by the relationship between the average litter size and the variance of litter size of different aged dams as follows:

Age of Dam	d.f.	n_b	n_{56}	n_{154}	$s^2_{n_b}$	$s^2_{n_{56}}$	$s^2_{n_{154}}$
1.0	998	6.64	4.30	3.93	4.26	3.35	3.12
1.5	199	7.49	5.02	4.61	5.35	4.15	4.27
2.0	213	8.50	5.17	4.91	5.79	3.40	3.32

Although these means are not the most reliable comparisons, as indicated previously, they show about the same order of differences as those of Lush and Molln (1942). The relationship is most apparent for litter size at birth. Why the variance of litter size at 56 and 154 days is less for two-year-old dams than one-and-a-half-year-old dams is not known. Since older dams are more selected than younger ones, a very slight reduction in variance would be expected because of selection, but not nearly as much as was found. Although quantitatively not large in this case, selection increases the mean but reduces slightly the variance among selected individuals. The question of transformations to homogenize the variances is discussed in detail by Bartlett (1947). There does not appear to be a clear case for using any particular transformation for these data.

Pooling the sums of squares may not be the best method of obtaining an over-all variance. If one could assume that one was sampling from a population with common variance, an unbiased estimate of the population variance is obtained by pooling the sums of squares and dividing by the total degrees of freedom. But in this case, the variance is not really the same in all groups. An average variance, no matter how it is weighted, need not be an unbiased estimate of the variance in any particular one of these groups but is an average of genuinely unequal variances.

The correlations between size of litter at birth and size of litter at 56 and 154 days were also different among the classifications tested except for seasons. The correlation between size of litter at 56 days and size of litter at 154 days varied little from group to group. The consistency of the later correlations is not surprising because there is little mortality from 56 to 154 days. Therefore, size of litter at 154 days is the major part of size of litter at 56 days. The over-all correlation coefficients were computed from sums of products and squares which were found within LYSA groups and then pooled together.

The weights of pigs at 56 and 154 days were also analyzed as between and within litters within LYSA groups. Although the procedure is fairly common (Snedecor, 1946) the general outline will be presented. Two traits X and Y will be considered. X_{jk} is the observation of X on the k^{th} animal in the j^{th} litter of a particular LYSA group. A similar definition applies to Y_{jk}. The analysis of covariance for a LYSA group is:

Source	d.f.	Mean Product	E(MP)
Between litters in LYSA group	t-1	$\frac{1}{t-1}\left[\sum_j \frac{X_{j.}Y_{j.}}{n_j} - \frac{X_{..}Y_{..}}{n.}\right]$	$(\rho_{xy}-\rho_{xy'})\sigma_x\sigma_y + p\,\rho_{xy'}\sigma_x\sigma_y$
Within litters	$\Sigma n_j - t$	$\frac{1}{\Sigma n_j - t}\left[\sum_{jk}\sum X_{jk}Y_{jk} - \sum_j \frac{X_{j.}Y_{j.}}{n_j}\right]$	$(\rho_{xy} - \rho_{xy'})\sigma_x\sigma_y$

In this example k goes from 1 to n_j, the number of animals in the j^{th} litter, and is variable; and j goes from 1 to t, the number of litters. A dot (.) indicates summation over that subscript. The expectations of the mean products are found from the following considerations:

$$
\begin{aligned}
E(X_{jk}-\mu_X)(Y_{j'k'}-\mu_Y) &= \text{Cov } xy = \rho_{xy}\sigma_x\sigma_y \text{ if } j = j' \text{ and } k = k' \\
&= \text{Cov } xy' = \rho_{xy'}\sigma_x\sigma_y \text{ if } j = j' \text{ and } k \neq k' \\
&= 0 \text{ if } j \neq j' \text{ and } k \neq k',
\end{aligned}
$$

where μ is the mean, ρ_{xy} is the correlation between X and Y on the same individual, and $\rho_{xy'}$ is the correlation between X on one individual and Y on a full-sib. The quantity p is

$$p = \bar{n} - \frac{\sigma_n^2}{t\bar{n}},$$

where $\bar{n}$ is the average litter size. If the number of litters, t, is at all large, $\sigma_n^2 / t\bar{n}$ is small, and $\bar{n}$ may be used for p with little error. The number of litters was often quite small in a LYSA group and p was computed.

The expectations of the mean products are written in general form. They may be converted to the expected mean squares by letting Y = X. The expectations then become:

Source	E (mean square)
Between litters	$\sigma_X^2(\rho_{XX}-\rho_{XX'}) + p\ \rho_{XX'}\sigma_X^2 = (1-\rho_{XX'})\sigma_X^2 + p\ \rho_{XX'}\sigma_X^2$
Within litters	$\sigma_X^2(\rho_{XX}-\rho_{XX'}) = (1-\rho_{XX'})\sigma_X^2$

The mean squares and products for the weights, which were found by pooling the sums of squares and products of the LYSA groups, are given in Table 8.

Table 8

Mean Squares and Products for Weights at 56 and 154 Days

Source	d.f. for W_{56}	d.f. for W_{154}	Mean square W_{56}	Mean product W_{56}, W_{154}	Mean square W_{154}
Between litters within LYSA groups	1449	1449	126.1	255.5	1329.1
Within litters	7140	6467	33.3	77.2	505.6
p			4.50	4.15	4.15

From the mean squares and products in Table 8 the variances and correlations in Table 9 were found. The prime, as before, means that the trait which is primed is observed on a litter mate of the animal with the unprimed trait. The variances and correlations are very similar to those of Baker et al. (1943) and Nordskog et al. (1944).

The variances of weights within litters, $\sigma_x^2(1 - \rho_{xx'})$, were heterogeneous from group to group within the major classifications.

Table 9

Variances of and Correlations Between Weights at 56 and 154 Days

Trait	Variance	Correlation w'_{56}	w'_{154}	w_{154}
w_{56}	54	.38	.22	.62
w_{154}	704	.22	.28	

Again, to make this test, the sums of squares for weights within litters were pooled for each major subgroup within years, lines, seasons and ages of dam. The tests for homogeneity were applied to the variances computed from the pooled sums of squares. The variances were particularly variable for 154-day weights. Variable sex ratios could have caused little of the variation among variances of 154-day weights or 56-day weights because sex differences would be about averaged out between groups in which the variances within litters were pooled.

A possible cause of such heterogeneity is the relationship between the mean and variance or standard deviation in growth data. The coefficient of variation remains fairly constant over the period of growth to 154 days, with a gradual decline with age. Baker *et al.* (1943) found the coefficient of variation to decline from 19.7 at birth to 14.4 at 168 days. Nordskog *et al.* (1944) found the same sort of relationship between intra-litter coefficients of variation. The fact that the coefficient of variation of weights is fairly stable suggests a logarithmic transformation to stabilize the variance (Bartlett, 1947). However, since the coefficient of variation declines with age, a logarithmic transformation would probably overdo the matter. It does not necessarily follow, either, that the relationship between the mean and variance of groups of individuals which are substantially of the same age is the same relationship as exists between the mean and variance of groups of individuals which differ in age.

To obtain a rough idea of the relationship between the mean and variance, the linear correlation between the average weight of the litter at 56 days and the variance of weights within the litter at 56 days was computed. The sums of products and squares were found within LYSA groups and pooled together. Of course, litters with only one pig at 56 days had to be deleted since they gave no estimate of the variance. The correlation was found to be -.11 (1340 degrees of freedom), which is quite different from the correlation between the mean and variance of groups of pigs which differ markedly in age. Runt or very small pigs are believed to be the chief contributing cause to this negative relationship. Quite a few runt pigs, some weighing as little as 5-7 pounds, were recorded at

56 days. Runt pigs would tend to make the average weight of the litter low but cause the variance to be large, particularly if the number of pigs in the litter was small.

There is some question as to whether a runt or extremely small pig is a normal individual and whether it should be included in a study such as this. No satisfactory scheme for determining an abnormal pig could be decided on at the time, and all pigs were included in the study regardless of their weight. An arbitrary minimum, below which all pigs are deleted, is highly unsatisfactory because the distribution of weights is continuous through such a minimum. A runt is conspicuous when he is an exception in the litter and his littermates have done very well. It occurred to the author later in the analysis, that the comparison of a suspect with the average of his litter mates might afford a reasonable approach for the rejection of runts, somewhat according to the recommendations of Thompson (1934) concerning the rejection of aberrant items in general. However, this was not done.

Another possible cause of heterogeneity is intra-litter competition for food. Pigs in larger litters would on the average have a smaller milk supply per pig than pigs in smaller litters. Husky pigs in a large litter conceivably could maintain and increase their advantage at the expense of their less fortunate litter mates. If this were true, the variance within litters would be expected to increase with the size of the litter. The correlation between litter size at 56 days and the variance of weights at 56 days within the litters actually turned out to be negative, -.02, but practically zero. This correlation was also computed from sums of squares and products which were first found within LYSA groups and were then pooled together. It appears then that competition, as far as that varies with size of litter, has little effect on the variation of weights within the litter.

The relationship between size of litter and average litter weight is not a linear one, but the linear correlations were computed anyhow to get at least a rough indication of their relationship. Bywaters (1937), and Korkman (1947) found that the heaviest pig weights at 8 weeks and 3 weeks, respectively, occurred in litters of size 3. Weights were smaller in both larger and smaller litters. The major downward trend came with increasing size of the litter, however. The simple correlations between the litter sizes and the average litter weights are in Table 10, and were based on 1448 degrees of freedom.

There are two trends in Table 10. First, at any age the correlation of size of litter with 154-day weight is less than its correlation with 56-day weight. Second, the correlation of size of litter with a particular weight decreases as the size of litter was counted at older ages. The interpretation, which seemed reasonable at first, was that the milk supply per pig was less in larger litters than in smaller litters until weaning. After weaning, when they were on their own, the pigs tended to compensate for the initial handicap of litter size to the extent that weight at 154 days was uncorrelated with size of litter. That there is some compensatory growth is indicated by $r_{n_b\bar{w}_{154}}$ being less than $r_{n_b\bar{w}_{56}}$. That the pigs do not completely overcome the initial effects of litter size is indicated by $r_{n_b\bar{w}_{154}}$ being negative. The same results would be obtained in a situation where the genes for larger litter size were positively correlated with genes for growth. The initial

Table 10

Correlations Between Size of Litter and Average Weight of Pigs in the Litter

Trait	n_b	n_{56}	n_{154}
$\bar{w}_{56}$	-.278	-.148	-.072
$\bar{w}_{154}$	-.171	-.054	.005

negative direct effect of large litter size on weight of the pig would be gradually overcome by the genes for growth as they had a chance to express themselves. Quite aside from the initial effect of litter size on weight at birth, it is not easy to see how the correlation between the size of litter and weight decreases with an increase in age at which the size of litter and weight is measured, at least to the extent that $r_{n_{154}\bar{w}_{154}}$ is zero or positive. Two forces seem to be at work. Larger litters at birth are initially handicapped in weight per pig. However, for a given size of litter at birth, more pigs will survive to 56 days in those litters having the higher average birth weights. Also, for a given average birth weight, the larger size of litter at birth will have a larger average weight at 56 days. This latter relationship says that pigs under a greater handicap of large size of litter at birth, which have done as well as pigs under a lesser handicap of small size of litter at birth, are inherently better doers, and will continue to do better as they become more independent of the direct effects of size of litter. Thus, there is a concurrent see-saw relationship between size of litter and weight of the pigs. These forces are in opposition to the initial relationship of weight and size of litter to such an extent that by 154 days the size of litter is not related to average weight of the pigs in the litter. Since birth weights were not included in this study an example can be given only for 56 days and 154 days.

The double headed arrows in diagrams a, b and c in Figure 1 represent the simple correlations between the two variables to which the arrows are pointed. The single headed arrows are path coefficients (Wright, 1934) or standard partial regression coefficients (Snedecor, 1946) of the variables to which the arrows point on the variables from which the arrows point. Diagram c is a composite of diagrams a and b. Although $r_{n_{154}\bar{w}_{56}}$ is negative (-.07), $\beta_{n_{154}\bar{w}_{56} \cdot n_{56}}$ is positive (.07). Likewise, $r_{n_{56}\bar{w}_{154}}$ is negative (-.05) but $\beta_{\bar{w}_{154}n_{56} \cdot \bar{w}_{56}}$ is positive (.05). If one wished to predict the correlation between n_{154} and $\bar{w}_{154}$, knowing only the relationships in diagram c, the predicted correlation is

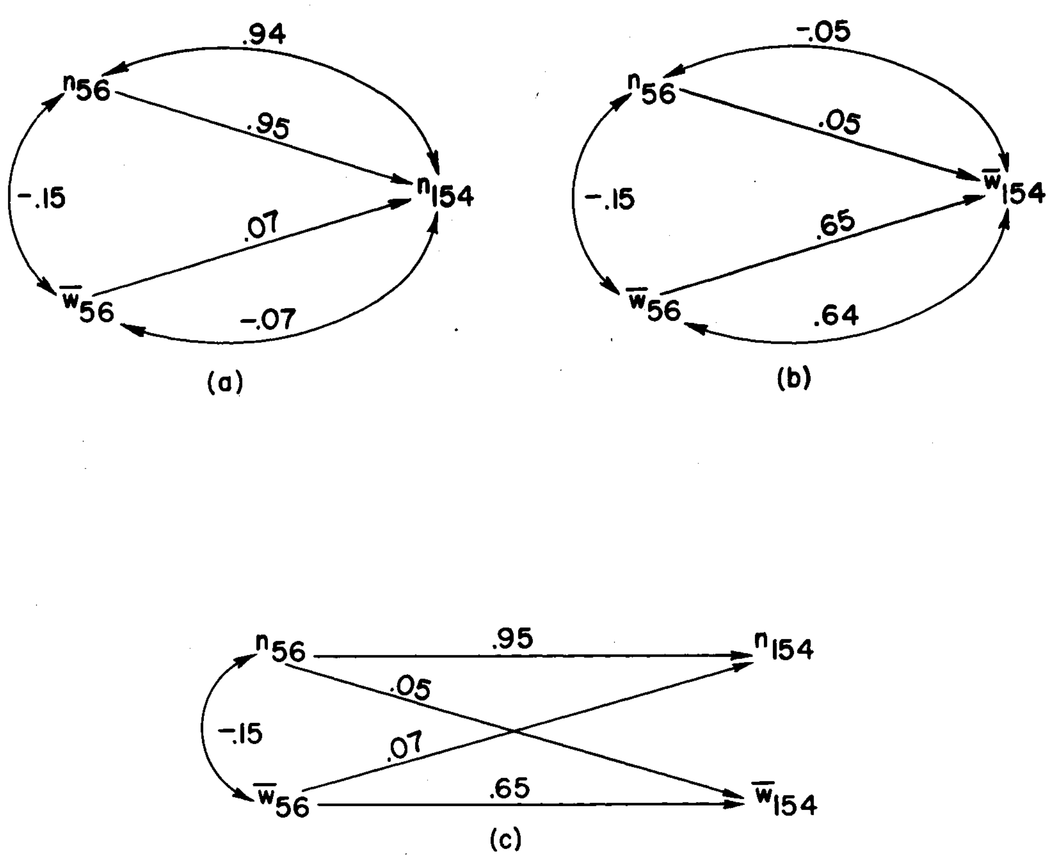

Fig. 1. Path Coefficient Diagram Indicating Relations Between Number and Average Weight in the Same Litter at a Later Age As "Dependent On" Number and Average Weight at an Earlier Age.

$$r_{n_{154}\bar{w}_{154}} = (.95)(.05)+(.07)(.65)+(.95)(-.15)(.65)+(.05)(-.15)(.07) = .00,$$

which agrees with the correlation obtained.

It does not necessarily follow that the relationships exemplified in the post-weaning period are the same ones present in the preweaning period. Size of litter in the preweaning period is thought to affect the food supply per pig, at least as far as the amount of milk per pig obtained from the dam is concerned (even here, mothering ability and milking capacity of the dam may be related to size of litter or growth of the pigs). In the post-weaning period the pigs are freed of any concurrent effects of size of litter, since the pigs are grouped together after weaning. However, the difference between $r_{n_b w_{56}}$ and $r_{n_{56} w_{56}}$ can be explained by the same arguments that were given in the post-weaning period.

ESTIMATES OF GENETIC PARAMETERS

Conceptually, at least, the variances and relationships of characteristics can be broken down into definable component parts. Problems arise in the proper evaluation of these parts. This study is concerned with the additive genetic variances of and the genetic correlations between litter size and weight for age.

Regression of Offspring on Parent with a Variable Number of Offspring for Each Parent

The technique used throughout this study for estimating genetic parameters was the phenotypic regression of offspring on dam. Since the number of progeny from each dam was variable, it seemed desirable to investigate what procedure would be most suitable for estimating the regression. Although this is an old problem in animal breeding research, the best procedures for obtaining estimates which have minimum variance have received little attention. In general, two methods have been used for this situation. One method has been to use the average of the offspring for a paired observation with the dam. Such a solution may be written as

$$b_{y_a x} = \frac{\sum_i (X_i - \bar{X}_a)(y_{i.} - \bar{y}_{i.})}{\sum_i (X_i - \bar{X}_a)^2}$$

where X_i is the observation on the i^{th} dam, $y_{i.}$ is the average of the n_i offspring of the i^{th} dam, $\bar{X}_a$ is the average of the k dams, and $\bar{y}_{i.}$ is the average of the averages of offspring $(\Sigma y_{i.}/k)$. The other procedure has been to dub in the observation of the dam for each offspring, and then compute the regression in the usual manner. This procedure is the same as using the average of the offspring for each dam, and weighting each dam by the number of offspring, which is written as

$$b_{y_d x} = \frac{\sum_i n_i (X_i - \bar{X}_d)(y_{i.} - y_{..})}{\sum_i n_i (X_i - \bar{X}_d)^2} ,$$

where $\bar{X}_d$ is the weighted average of the dams $(\Sigma n_i X_i / \Sigma n_i)$, and $y_{..}$ is the average of all offspring.

Complications arise not only because the number of offspring is variable but also because the offspring are correlated with each other. If the offspring are correlated with the dam, ρ_{xy}, the expected intra-class correlation between offspring with the same dam for this reason alone is ρ^2_{xy}. The correlation is usually larger than this because offspring by the same dam are likely to be correlated for other reasons, particularly in those species which have multiparous birth. Individuals within a litter not only usually have the same sire but also they are contemporary for many circumstances which may affect them phenotypically. In these data most of the offspring have one or more full-sibs.

The formulation of such a condition with allowance for linear regression of offspring on dam, is

$$Y_{ij} = \alpha + \beta X_i + e_{ij} ,$$

where α is a constant, β is the regression of offspring on dam and the e_{ij}'s are the deviations. Since the e_{ij}'s are correlated as between sibs, they will be considered to have the following expectations:

$$\begin{aligned} E\, e_{ij} &= 0 \\ E\, e_{ij}e_{i'j'} &= \sigma^2 \text{ if } i = i',\ j = j' \\ &= \rho\sigma^2 \text{ if } i = i',\ j \neq j' \\ &= 0 \text{ if } i \neq i'. \end{aligned}$$

The quantities ρ and σ^2 may be best understood in terms of the phenotypic variances and correlations, relative to the whole population of many litters. Since

$$\sigma^2 = E\, e_{ij}^2 = E\,(Y_{ij} - \alpha - \beta X_i)^2 = \sigma_y^2\,(1 - \rho_{xy}^2),$$

and since

$$\rho\sigma^2 = Ee_{ij}e_{ij'} = E(Y_{ij}-\alpha-\beta X_i)(Y_{ij'}-\alpha-\beta X_i) = \sigma_y^2(\rho_{yy'}-\rho_{xy}^2) ,$$

it follows that

$$\rho = \frac{\rho_{yy'} - \rho_{xy}^2}{1 - \rho_{xy}^2} ,$$

where ρ_{xy} is the correlation between dam and offspring, $\rho_{yy'}$ is the intraclass correlation between offspring with the same dam (full-sibs in these data) and σ_y^2 is the variance among offspring with different dams. The quantity ρ is then the intraclass correlation between offspring with the same dam, when those offspring's phenotypes had first been adjusted for their regression on the phenotype of the dam.

The method of maximum likelihood is used to find the estimators of α and β. If the errors are normally distributed, the likelihood function is

$$\frac{\sqrt{|P^{-1}|}}{(2\pi)^{\frac{\Sigma n_i}{2}}} \exp\left[-\frac{1}{2}\sum_{ij}\sum_{i'j'} p^{iji'j'}(Y_{ij}-\alpha-\beta X_i)(Y_{i'j'}-\alpha-\beta X_{i'})\right] ,$$

where $p^{iji'j'}$ is an element of the inverse of the variance-covariance matrix, P^{-1} (Mood, 1950). Sections of the variance-covariance matrix, P, are independent if $i \neq i'$. A section where $i = i'$ is

$$\sigma^2 \begin{bmatrix} 1 & \rho & \cdots & \rho \\ \rho & 1 & \cdots & \rho \\ \cdot & \cdot & & \cdot \\ \cdot & \cdot & & \cdot \\ \cdot & \cdot & & \cdot \\ \rho & \rho & \cdots & 1 \end{bmatrix}$$

and the size of the section is $n_i x n_i$, the number of rows and columns being equal to the number of offspring for the i^{th} dam.

The solutions for α and β, found by taking the log of the likelihood function, differentiating with respect to α and β, and solving simultaneously, turn out to be:

$$b_{y_m x} = \hat{\beta} = \frac{\sum_i c_i(X_i-\overline{X}_m)(y_{i.}-\overline{Y}_m)}{\sum_i c_i(X_i-\overline{X}_m)^2}$$

$$\hat{\alpha} = \overline{Y}_m - \beta\overline{X}_m ,$$

where $c_i = n_i / \left[1 + (n_i - 1)\rho\right]$, $\overline{X}_m = \Sigma c_i X_i / \Sigma c_i$ and $\overline{Y}_m = \Sigma c_i y_{i.} / \Sigma c_i$. The solution uses the average of the daughters for a paired observation with the dam, and weights the average of the daughters in the regression according to the reciprocal of their variance. For example,

$$V_{e_{i.}} = V\left(\sum_j e_{ij}/n_i\right) = \sigma^2 \left[1 + (n_i - 1)\rho\right] / n_i .$$

Since σ^2 is constant, it cancels out in the weighting.

To make use of maximum likelihood it is necessary to assume some sort of distribution. The normal distribution was used in this case. Little can be said about the application of this solution to data where the e_{ij}'s follow some distribution other than normal.

Unfortunately, no way was found by which ρ could be estimated simultaneously with α and β. However, ρ depends on $\rho_{yy'}$, which can be estimated separately, and on ρ_{xy} which is the same as the regression coefficient, β, that is being estimated. In special cases ρ is a function of ρ_{xy} only. If the offspring by the same dam are half-sibs, and if there is no environmental correlations between offspring by the same dam or between dam and offspring, $\rho_{yy'} = \rho_{xy}/2$ and $\rho = \rho_{xy}(\frac{1}{2} - \rho_{xy})/(1 - \rho_{xy}^2)$. If the offspring are full-sibs, and if there are no environmental correlations and no effects of dominance, $\rho_{yy'} = \rho_{xy}$ and $\rho = \rho_{xy}/(1 + \rho_{xy})$. In the present data, where full-sibs are born in litters, traits are likely to be correlated for environmental reasons. If $\rho_{yy'}$ is found to be as large as .3 or larger and if ρ_{xy} does not exceed .2, ρ may for all practical purposes be considered to be the same as $\rho_{yy'}$. The specific limits chosen are somewhat arbitrary, but they are believed to be representative for growth in swine. For example, the correlation between offspring by the same dam (full-sibs in these data) was found to be .38 and .28 for 56- and 154-day weights, respectively (Table 9). The parent-offspring regression or correlation has seldom been found to be larger than .2.

All three methods are unbiased, provided the weights (the c_i) are uncorrelated with the characteristic of the dam. The variance of the weighted regression is in general

$$\sigma^2_{b_{y_w x}} = \sigma^2 \frac{\sum_i w_i^2 (X_i - \bar{X}_w)^2 / c_i}{\left[\sum_i w_i (X_i - \bar{X}_w)^2\right]^2} ,$$

where w_i is the weight, c_i is the maximum likelihood weight and $\bar{X}_w$ is the weighted mean of dams. If $w_i = c_i$ the variance reduces to

$$\sigma^2_{b_{y_m x}} = \frac{\sigma^2}{\sum_i w_i (X_i - \bar{X}_w)^2} ,$$

and is a minimum in large samples. The reduction in variance of the regression coefficient, with increasing number of offspring may be considered here by letting the number of offspring for each parent be constant. Then,

$$\sigma^2_{b_1} = \frac{\sigma^2}{\sum\limits_i (X_i-\bar{X})^2}$$

and

$$\sigma^2_{b_n} = \frac{\sigma^2}{c\sum\limits_i (X_i-\bar{X})^2} = \frac{1 + (n-1)\rho}{n} \; \sigma^2_{b_1} \; .$$

which tends towards $\rho\sigma^2_{b_1}$ as a limit when n becomes very large. The subscript for b indicates the number of offspring per parent. When the number of offspring varies, the variance would be somewhat greater than the above formula would yield if $\bar{n}$ were used in place of n.

Note that the two procedures generally used are two special cases of the maximum likelihood solution.

$$b_{y_a x} = b_{y_m x} \quad \text{if } \rho = 1$$
$$b_{y_d x} = b_{y_m x} \quad \text{if } \rho = 0.$$

Actually, ρ can never be 0 or 1 when the offspring are full-sibs, unless the regression being estimated, β, is also 0. It would be desirable, with a knowledge of ρ, to be able to determine in advance whether $b_{y_a x}$ or $b_{y_d x}$ has the smaller variance. Although no procedure was found, the extreme cases (ρ near 1 or 0) are apparent from the relationship of the two regression coefficients and that of the maximum likelihood one.

The procedure chosen in this study was to average all offspring for each parent within a LYSA group (i.e. to compute $b_{y_a x}$). Also, the dam was dubbed in for some of the regressions in order that the two methods of estimation could be compared ($b_{y_d x}$ to $b_{y_a x}$).

Offspring on Dam Regression

Certain definitions are necessary to make clear which offspring are regressed on which dams. Three terms, dam, daughter and litter will be used. The daughter is an offspring of the dam, but the mother of the litter. Litter size of the dam is the size of litter in which the daughter was born. Litter size of the daughter is the size of the litter which the daughter produced. Weight of the daughter is the daughter's own individual weight. Average litter weight is the average weight per pig of the litter that the daughter produced.

To compute the regressions the data were sorted into LYSA groups (the same line, year, season and age of dam) for the daughters and for the litters. This means that the dams within a LYSA group were all born in the same line, year, and season but their mothers could have differed in age. For the regression of weight on weight the average weight of the litter was regressed on the individual weight of the daughter. The regression of litter size on weight utilized the same pairing as weight on weight, except litter size of the daughter was substituted for average weight of the litter. For the regression of litter size on litter size, the litter size of the daughter was regressed on the size of the litter in which the daughter was born. In this case, if daughters were full-sibs, the size of their litters were averaged for a single paired observation with the dam. To obtain the regression of weight on litter size the average weight of the litter was regressed on the litter size of the dam. Again, the average weights of litters from full-sib daughters were averaged for a single paired observation with the litter size of the dam. To summarize: the average weight of the pigs in the litter the daughter produced was paired with the individual weight of the daughter, the size of the litter the daughter produced was paired with the individual weight of the daughter, the size of litter the daughter produced (she and her full-sibs were averaged) was paired with the size of litter in which the daughter was born, the average weight of the pigs in the litter which the daughter produced (again average weights of pigs from full-sister daughters were averaged) was paired with the size of litter in which the daughter was born, and the regressions were computed within groups such that the effects of line, year, season, and age of dam were removed for both of the paired observations.

There were 1702 litter-daughter pairs for the weights. The 1702 pairs contained only 1245 different daughters, and there were 821 full-sib groups among these daughters. In other words, when daughters which were full-sibs were grouped together, there were 821 such groups. The restrictions imposed to remove line, year, season and age of dam effects, divided the data into 372 LYSA groups in each of which there were 2 or more litter-daughter pairs. The number in a group varied from 2 to 21 with an average of 3.7, after 309 litter-daughter pairs were eliminated because they appeared singly in a LYSA group. For the regression of weight and litter size on weight of the daughter there were then 1020 degrees of freedom (1702-309-372-1). The averaging of daughters which were full-sibs in a LYSA group, reduced the degrees of freedom to 557 for the regression of the traits on litter size of the dam.

The regression coefficients are given in Table 11. The regressions of average weight of the litter and litter size of the daughter on litter size of the dam were also computed by dubbing in the dam for each daughter or litter; i.e. both $b_{y_a x}$ and $b_{y_d x}$ were computed. These regression coefficients are given in Table 11 for comparison with the ones obtained when full-sib daughters or litters from full-sib daughters were averaged. In general, $b_{y_a x}$ and $b_{y_d x}$ are nearly alike. The standard errors in Table 11 were computed in the usual manner with the number of degrees of freedom based on the number of dams when litter size was the independent variable and on the number of daughters when weight was the independent variable. They are approximate only in that dams or daughters often appeared in more than one group, and the errors of groups would then be correlated. The variances of the offspring (litters or daughters) also cannot be considered to be homogeneous since varying numbers of offspring were averaged.

If these data were from a random bred population, heritability of a character would be estimated by doubling the corresponding diagonal element in Table 11. Heritability in an inbred population, such as this, may also be estimated in this manner. Generally, however, heritability as found in the sample is extrapolated to

Table 11

Regression Coefficients of Characteristics of the Offspring on Those of the Parent

Independent variable	Character-istic	Daughter's litter n_b	Daughter's litter n_{56}	Daughter's litter n_{154}	Daughter's pigs w_{56}	Daughter's pigs w_{154}
Dam's litter	n_b	-.053 ±.042	.004 ±.035	-.010 ±.034	.085 ±.106	.020 ±.361
		-.039[a]	-.008[a]	-.017[a]	.087[a]	-.023[a]
	n_{56}	-.092 ±.048	-.046 ±.041	-.056 ±.040	.106 ±.121	.373 ±.413
		-.059[a]	-.032[a]	-.041[a]	.093[a]	.335[a]
	n_{154}	-.122 ±.049	-.071 ±.041	-.074 ±.040	.110 ±.123	.332 ±.422
		-.093[a]	-.053[a]	-.052[a]	.097[a]	.312[a]
Daughter	w_{56}	.041 ±.011	.017 ±.010	.021 ±.009	.012 ±.029	-.063 ±.099
	w_{154}	.009 ±.001	.001 ±.001	.0023 ±.001	.003 ±.032	.025 ±.035

[a] These regression coefficients were computed by dubbing in the dam again for each daughter or litter, and are presented here for comparison with those directly above them, where full-sib daughters were averaged.

make it comparable with that in a random bred population. This is intended to remove differences which the special level of inbreeding in the sample studied would probably have caused in the observed regressions. If there is no dominance or epistasis, inbreeding reduces the additive genetic variation within a line in proportion to 1-F, where F is the inbreeding coefficient. In the event that dominance and epistasis do not alter the reduction with F very much, the relationship between the two additive genetic variances will still be approximately

$$\sigma^2_{G_L} = (1-F)\sigma^2_{G_r} \quad ,$$

where $\sigma^2_{G_L}$ is the additive genetic variance within a line and $\sigma^2_{G_r}$ is the additive genetic variance in the random bred population from which the line was derived. The relationship between heritability within an inbred line, h_L, and heritability in a random bred population, h_r, is then

$$h_r = \frac{h_L}{1-F(1-h_L)} \quad .$$

One other correction may be worth considering. In these data many of the daughters were full-sibs. Estimates of heritability are not generally corrected for the relationship of the dams or daughters in this case; however, it is rare that so many full-sibs appear as do in these data. When full-sib daughters were averaged for the regression of the various traits on litter size of the dam, there were only 558 degrees of freedom in contrast to 1021 degrees of freedom for the regressions of the traits on the weight of the daughter--a difference of 463 degrees of freedom. Full-sibs contributed about one-half of the 1021 degrees of freedom, since for each group averaged a single observation for the group appeared in the 558 degrees of freedom.

If all the dams in a group were full-sibs, the regression would be

$$b \cong \frac{\sigma_G^2 / 4}{(1-\rho)\sigma_x^2} ,$$

where ρ is the correlation among full-sibs, and epistasis is ignored. The heritability of differences among nonrelated dams ($h = \sigma_G^2/\sigma_x^2$) is

$$h \cong 4b(1 - \rho) \cong 2h'(1 - \rho) ,$$

where h' is the heritability of differences among full-sib dams ($h' \cong 2b$). When the only reason for correlation among phenotypes of full-sibs is additively genetic, ρ is $h/2$. In this case, the correction to make heritability of differences between full-sib dams applicable to heritability of differences between nonrelated dams may be of considerable size,

$$h = \frac{4b}{1 + 2b} ,$$

particularly if b is small. The correction is small, however, if ρ is near .5. Also, the correction becomes less as the proportion of dams which are full-sibs decreases. In these data the correlation among full-sibs for 56- and 154-day weights were .38 and .28, respectively. The size of these correlations, coupled with the fact that only about half of the dams were full-sibs, would make negligible the correction for relationship between the dams.

Heritability of weight was corrected for inbreeding. The inbreeding figure used was .32, which is the average of the daughters' and litters' average inbreeding of .29 and .35, respectively. The heritabilities thus corrected turned out to be:

w_{56} ... .03

w_{154} ... .07

These estimates are considerably lower than those found by other workers (Tables 1 and 2). The fact that these are much lower than have generally been found, and that the estimates for litter size are negative in comparison to an average of about .15 in Table 3, needs an explanation. Differences among lines and age of dams, or time trends could be important. The regressions are broken down according to line of breeding, year of birth of the daughter, and age of the daughter in Table 12.

Although the regression coefficients in Table 12 fluctuate considerably from line to line and from year to year, they were not significantly different from the average regression coefficient. The variability is about what one would expect when sampling from a population where the true value was zero. Since inbreeding increased with time, and the additive genetic variation within a line decreases with an increase in inbreeding, heritability would be expected to decrease with an increase in years. No time trend

Table 12

Regression of the Character of the Offspring on the Same Character of the Parent by Line, by Year, and by Age of Daughter

Grouping	d.f. for litter size	n_b	n_{56}	n_{154}	w_{56}	w_{154}	d.f. for weight
Line							
S	160	-.097	-.098	-.093	-.040	.027	289
A	88	.072	.001	-.009	.007	-.047	149
B	66	-.178	.035	.142	.066	.164	108
C	38	.102	.038	.007	.033	.178	69
D	21	-.075	-.270	-.284	.111	.272	41
E	28	-.054	.068	-.029	.058	.051	58
F	12	.155	-.514	-.652	.115	.021	31
G	13	-.005	.050	-.140	.306	.147	37
H	42	-.073	-.085	-.131	-.029	-.081	67
I	24	.015	.207	.188	.025	-.071	43
J	13	.172	-.272	-.364	.140	.107	30
K	10	-.092	-.151	-.365	-.101	-.087	18
L	43	-.172	-.004	-.090	-.050	-.125	81
Year							
1938	23	-.280	.112	.104	-.170	.035	43
1939	68	-.210	-.088	-.203	-.019	-.085	113
1940	63	.073	-.107	-.151	.066	-.004	129
1941	54	-.137	.074	-.001	.002	.148	123
1942	72	.188	.073	.018	.087	.065	116
1943	55	.108	-.241	-.179	.105	.145	95
1944	52	-.192	-.100	-.069	.074	.100	105
1945	43	-.053	-.019	-.001	.128	.299	77
1946	51	-.285	-.181	-.142	-.023	.146	88
1947	19	.292	.630	.330	-.097	-.283	38
1948	43	.160	-.096	-.099	-.214	-.213	67
1949	15	.037	.218	.237	-.184	-.290	27
Age of daughter							
1 year	401	-.067	-.048	-.070	.028	.062	756
>1 year	157	-.004	-.040	-.083	-.031	-.076	265

is apparent. However, the increase in average F over the period studied was only from about .20 to .48. Hence a trend would not be obvious unless it were extreme.

Sampling errors can be invoked to explain the negative estimates of heritability for litter size. Sampling errors are small enough, however, to make it highly unlikely that the parameters estimated are anywhere near the size of the estimates found in the past (Tables 1, 2 and 3).

The phenotypic consequences of inbreeding were ignored. If parents were mated so that the more inbred dams had less inbred offspring than the less inbred dams, there would be a negative relationship between the inbreeding of the dam and that of the offspring. This negative relationship would tend to make the correlation between the litter size of the daughter and that of the dam negative provided the phenotypic values decrease with an increase in inbreeding. The correlation actually found between the inbreeding of the dam and that of the offspring was slightly positive, .033, but essentially zero.

The direct effect of litter size on weight of individuals in the same litter, and of weight of the sow on the size of the litter she produces could be confusing sources of variation in the estimation of heritability. These effects will be investigated while considering the regression of one variable on another.

The genetic correlation between two characters is found by taking the ratio of the geometric mean of their two symmetrical elements (regression coefficients) in Table 11 to the geometric mean of their corresponding diagonal elements. No genetic correlations are estimated in these data since they involve taking the square root of a negative number or utilizing a negative heritability. Also, with such numerically small values for heritability, the sampling errors of estimates of genetic correlations would be extraordinarily high.

In order that symmetrical elements in Table 11 may be compared (b_{12} to b_{21}), the regression coefficients in Table 11 were converted to correlation coefficients in Table 13. This conversion was done by multiplying the regression coefficients by the ratio of the standard deviation of the independent variable to the standard deviation of the dependent variable. The standard deviations are the ones found in the phenotypic analysis.

Each correlation in Table 13 comes from the regression in the corresponding location in Table 11. In discussing the correlations the subscript order will be the same as that of the regression coefficients from which they were found. The first subscript denotes the characteristic of the daughter for litter size and of the litter for weight. The second subscript denotes the characteristic of the dam for litter size and of the daughter for weight.

Differences between reciprocal correlations (i.e. $r_{w_{56}w_{154}}$ and $r_{w_{154}w_{56}}$) are most likely the result of sampling, but this need not be so, particularly for the correlations involving size of litter and weight. The correlation between the size of litter the daughter produces and her own individual weight, r_{nw}, is a correlation between two characters of the same individual (to the extent that the size of the litter the sow produces really is a character of the sow and the sow's own weight really is a character of the sow), while the reciprocal correlation, r_{wn}, is between one character of a sow and another character of her grandchildren.

Table 13

Correlation Coefficients Between Characters of the Offspring and Those of the Parent

Character		Daughter's litter			Daughter's pigs	
		n_b	n_{56}	n_{154}	w_{56}	w_{154}
Dam's litter	n_b	-.053	.004	-.012	.026	.002
	n_{56}	-.079	-.046	-.057	.028	.027
	n_{154}	-.103	-.070	-.074	.029	.024
Daughter	w_{56}	.131	.065	.080	.012	-.017
	w_{154}	.111	.008	.032	.010	.025

It is not surprising then that the former correlation is generally larger than the latter correlation. About two-thirds of the litters were from gilts. Stewart (1945b) found weight of the gilt at mating to be correlated with subsequent litter size, and Warnick et al. (1951) found weight at 56 and 154 days to be negatively correlated with age at puberty. Growthier gilts within a line would then tend to be bred at a later heat period than slower growing gilts. This would be particularly true if the breeding season began late enough that even the latest of the gilts had started to come into heat. Since ovulation rate increases with order of heat period, heavier gilts would conceivably have larger litters. This sort of argument would not hold for older daughters. To check this point the correlations were computed by age of daughter in Table 14.

Table 14

Correlations of the Traits with Individual Weights of the Daughter, Grouped According to the Age of Daughter

Trait	Age of daughter	Daughter's litter n_b	Daughter's litter n_{56}	Daughter's litter n_{154}	Daughter's pigs w_{56}	Daughter's pigs w_{154}	d.f.
w_{56}	1 year	.142	.077	.086	.028	-.016	756
	>1 year	.101	.030	.064	-.030	-.021	265
w_{154}	1 year	.111	.012	.046	.049	.062	756
	>1 year	.112	-.002	-.006	-.097	-.026	265

There is little indication that the correlation between the litter size the daughter has and the daughter's weight at 56 or 154 days, differs as between first litters and later litters, particularly for litter size at birth. If one considers weight to be chiefly a function of the individual, and litter size a function of the sow which produces the litter, the correlation between the litter size the sow has and her own weight is a phenotypic correlation. This correlation may have both genetic and environmental contributions. Although the correlation, r_{nw}, is generally less for daughters which produced their litters when they were more than a year old than for daughters which produced their litters when they were a year old, the difference is not large. Genetic causes may then be the chief contributor to the correlation between litter size and weight. If this were true, r_{nw} would be expected to be about four times the size of r_{wn} (Table 13), since the latter correlation involves the relationship between the average weight of the dam's grandchildren and the size of litter the dam produces. Although the agreement between r_{nw} and $4r_{wn}$ in Table 13 is not close in any particular case, the average r_{nw} is .071 and four times the average r_{wn} is .091.

Why litter size at 56 days and later is less related to weight of the daughter that produced the litter than litter size at birth is not apparent (Table 14), unless rapid growth indicates a heavier, clumsier parent which overlays more pigs, or that rapid growth enables a sow to farrow more pigs to a greater extent than it enables her to care for them. It could also indicate a genetic antagonism between growth and mothering ability.

One other disturbing item in Table 13 is the negative correlation between average weight of the pigs in the litter at 154 days and the 56-day individual weight of the daughter which produced the litter. Weight at 56 days is influenced to a considerable extent by the milking and mothering ability of the dam, while post-weaning growth is chiefly a function of the pig. This negative relationship again suggests a genetic antagonism between milking ability and rapid growth. On the other hand, the average weight of the pigs in the litter at 56 days was positively correlated with the weight of the daughter at 154 days. The difference between these two correlations may not be real. It is possible, too, that the latter correlation may measure environmental influences of weight of the daughter on weight of the pigs that she produces. Larger gilts may be able on the average to feed their pigs better, simply because they have a larger body size and milking capacity. When sows over one year of age are considered, the correlation turns out to be negative (Table 14).

The negative correlations between size of litter which the daughter produces and size of litter which her dam produces might be explained in part by the relationship of litter size to weight of the pigs in it and by the relationship of weight of the gilt to the size of litter that she produces. Not only does litter size exert a direct influence on weight of the pig (Table 10), the relationship is made even more negative by simultaneous selection for the two traits. The correlations between weight of the daughters and the size of litter in which they were born, for selected daughters, are in Table 15.

The correlations in Table 15 which are concerned with n_{56} and n_{154} are more negative than the corresponding ones in Table 10.

Actually, the correlations in Table 10 may differ from those in Table 15 for two reasons. The former correlations are between size of litter and average weight of pigs in the litter, and thus are expected to be more negative than the correlations between the size of litter and the weight of a randomly chosen pig from the litter.

Table 15

Correlations Between Weight of the Selected Daughters and the Size of the Litter in Which They Were Born

Trait	n_b	n_{56}	n_{154}
w_{56}	-.25	-.20	-.17
w_{154}	-.12	-.12	-.09

Although the latter correlations (Table 15) are between size of litter and the weight of a single pig in the litter, simultaneous selection for size of litter and weight made the correlations involving n_{56} and n_{154} more negative than the corresponding ones in Table 10. Kottman (1952) found the selection pressure to be greater for n_{56} than for n_b (n_{154} was not included in the selection study), which would explain why the correlations involving n_b do not follow the same pattern as those involving n_{56} and n_{154}. Now, these negative relationships might in part explain the negative estimates of heritability of litter size. For example, if the size of the litter at birth in which the daughter was born is correlated with the 154-day weight of the daughter (-.12, Table 15), and if the 154-day weight of the daughter affects the size of litters to which she gives birth, (.111, Table 13) and if there are no other causal relationships, the correlation between litter size at birth of the daughter and that of the dam might be expected to be $-.013 = -.12 \times .111$. This could cause a negative estimate of heritability of litter size at birth but not nearly as large a negative as was found. Also, this sort of argument would explain little of the negativeness of the estimates of heritability for n_{56} and n_{154}. Although it is easy to visualize that the 154-day weight of a gilt might environmentally influence the subsequent litter size that she produces, the environmental influence of the 56-day weight of the daughter on her subsequent litter size should be reflected only through her 154-day weight. However, the correlation of 56-day weight of the daughter with the size of litter that she produced was higher than that between her 154-day weight and the size of litter that she produced (Table 14).

DISCUSSION

Negative parent-offspring regressions have generally been considered to be the result of a fortuitous sample. However, Lush (1949) pointed out that the parent-offspring regression would be negative, if the parents were survivors of really intense selection and if overdominance were an important source of variation. This point is investigated further here.

Parent-Offspring Regression with Overdominance and Selection

With a single pair of genes and random mating, the following situation may be visualized for the parents:

Genotype	Frequency of unselected parents f	Selective value S	Frequency of selected parents f_s	Mean yield K	Coded mean yield K_c	Linear scale W
AA	q^2	1	q^2/B	K_1	0	1
Aa	$2q(1-q)$	$1-hs$	$2q(1-q)(1-hs)/B$	K_2	$-k$	0
aa	$(1-q)^2$	$1-s$	$(1-q)^2(1-s)/B$	K_3	-1	-1

$$B = q^2 + 2q(1-q)(1-hs) + (1-q)^2(1-s) = 1-s(1-q)\left[1-q(1-2h)\right]$$

The measure or degree of dominance for this example is k for the yield values and h for the selective values, $k = (K_1-K_2)/(K_1-K_3)$.

When k is $\frac{1}{2}$, no dominance exists among the yield values; when k is 0, complete dominance exists among the yield values; and when k is less than 0, the yield value for the intermediate genotype exceeds that of the better homozygote and overdominance is present. The numerical value of k measures the degree of dominance within or between these broader classifications. The value of h describes the dominance situation for the selective values in the same manner that k does for the yield values. The selective values determine what happens to q, while the k values not only describe the location of K_2 relative to K_1 and K_3, but also may modify h, especially in artificial selection where man may vary his emphasis on K. The range of both h and k is from one to minus infinity, but dominance for the selective values may be quite different from that for the yield values. The range for s is from 0 to 1.

The additive genetic deviations for yields among the selected parents are $g = b_{K_c W}(W-\overline{W})$ and the dominance deviations are $d = K_c - \overline{K}_c - g$. Wright (1935) found additive genetic values, G's (in his notation), by minimizing $\Sigma f_s(K_c-G)^2$, subject to the restriction that $G_1-G_2 = G_2-G_3$. The g's here are simply $G - \overline{G}$ in Wright's notation; i.e. the g's are the additively genetic deviations from the mean rather than the additive genetic values themselves. The dominance deviations are the same as Wright's because $\overline{K}_c = \overline{G}$. The additive genetic deviations, g' 's, and the dominance deviations, d' 's, for the offspring, which result from random mating the selected parents are found in a similar manner. The regression of offspring on selected parents is

$$b_{K_c'K_c} = \frac{\rho_{g'g}\ \sigma_{g'}\ \sigma_{g}}{\sigma^2_{K_c}} ,$$

since the dominance deviations of parent and of offspring are not correlated with each other or with the additive genetic values. The yield values, the K's, are the same for both parent and offspring. A prime is used to distinguish the values for the offspring. The W scale is linear for both parent and offspring, but $b_{K_c'W}$ is likely to be different from b_{K_cW}. Thus g' will not be the same as g since they depend on frequencies of the K's which will differ as between the parents and the offspring when selection is being practiced. The sign of the regression, $b_{K_c'K_c}$, is determined by the correlation between the additive genetic values of the parents and that of the offspring,

$$\rho_{gg'} = \pm \frac{1}{2} \sqrt{1 - \frac{s\ q\ (1-q)(1-2h + sh^2)}{[q + (1-q)(1-hs)]\ [q(1-hs)+(1-q)(1-s)]}} .$$

Without selection this correlation is $+\frac{1}{2}$. The term under the square-root sign is the factor by which the correlation is reduced because of selection. The + or - consideration depends on whether the additive genetic effects in the parents, b_{K_cW}, and offspring, $b_{K_c'W}$, are of the same or different sign, respectively. The sign of the additive genetic effects are determined by the quantities:

$$q^2k + q(1-q)(1-hs) + (1-q)^2(1-s)(1-k)$$

for the offspring, and

$$q^2k(1-hs) + q(1-q)(1-s) + (1-q)^2(1-s)(1-hs)(1-k)$$

for the parents, which are actually the numerators of the regressions $b_{K'_cW}$ and b_{K_cW}, respectively.

The additive genetic effects for parents and offspring are of the same sign, and always positive, when k is greater than or equal to 0, or in the absence of over-dominance on the yield scale. In more general terms, if the yield values of the heterozygote coincides with the yield values of either homozygote or lies in between the two homozygotes, the parents cannot be selected in a manner such that the parent-offspring regression will be negative, provided the parents are mated at random. Further remarks will then be confined to the condition of overdominance, or k less than 0.

The first case to be considered is when the selective values are proportional to the yield values, $h = k$ and $s = (K_1-K_3)/K_1$. If gene frequency of all parents, prior to selection, is equal to $(1-h)/(1-2h)$, selection has no effect on the offspring-parent regression, $b_{K'_cK_c}$. The regression is zero, because the additive genetic effect for the offspring is zero. The condition, $q = (1-h)/(1-2h)$, is one of equilibrium at which the mean of the population is at a maximum under random mating (Lush, 1948). When q in the unselected parents is greater than the equilibrium value, the additive genetic effects for both parents and offspring will be negative and the offspring-parent regression will be positive. When the gene frequency of parents prior to selection is below the equilibrium value, selection can cause the additive genetic effect for the selected parents to be negative, but the additive genetic effect for the offspring will always be positive. The offspring-parent regression is negative under such a condition. To illustrate this point further, the shaded areas in Figure 2 indicate the values that s and q have to take for three values of $h(-.1,-1,-10)$ such that the parent-offspring regression will be negative. The straight lines which form the right boundaries of the shaded areas are the equilibrium values, when the additive genetic effects for the offspring are zero. The smooth curves which form the left boundaries of the shaded areas connect points for which the additive genetic effects of the parents are zero. The right and left boundaries of the shaded areas are then values for which the offspring-parent regression is zero.

The effectiveness of overdominance and selection in causing the offspring-parent regression to be negative, as far as the case where $h = k$ is concerned, does not appear to be very great. First, rather large selection coefficients are necessary to make the regression negative when q is as much as 0.1 below its equilibrium value. Second, if the gene frequency of the parents is below its equilibrium value but large enough that the parent-offspring regression is negative, selection will in a few generations carry

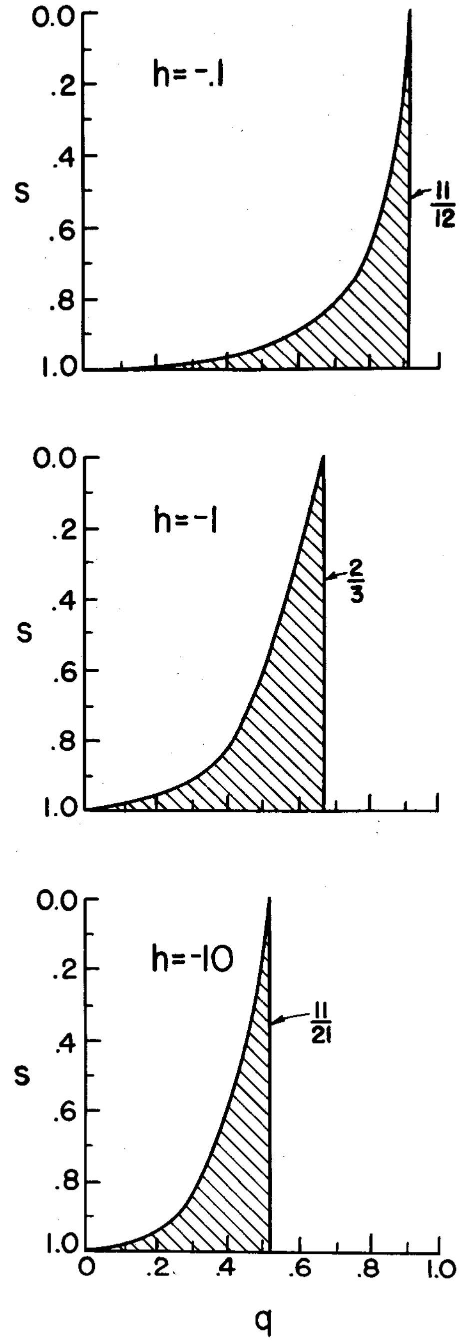

Fig. 2. The Shaded Areas Specify Conditions Necessary for a Negative Parent-Offspring Regression.

the frequency close to the equilibrium value. The attainment of equilibrium, however, is an asymptotic property.

The foregoing situation, where the selective value is proportional to the yield value, is not likely to be very realistic, especially when the intensity of selection is suddenly changed. This could be particularly true for genes which had more than one effect, and where selection was for several characters. No complete generalization seems possible, but some understanding of the problem may be gained by considering special cases where the selection and yield scales differ. In all cases the overdominance condition exists for the yield scale, k less than 0. Otherwise, the parent-offspring regression is always positive.

For the first case, there is overdominance on the selective scale, h less than 0, and gene frequency has reached equilibrium with respect to selection, $q = (1-h)/(1-2h)$. The sign of the parent-offspring regression then depends on the sign of

$$\frac{(1-2h + sh^2)}{(1-2h)^2} \; (k-h)$$

for the offspring, and

$$\frac{(1-2h + sh^2)}{(1-2h)^2} \left[k-h + sh(1-k)\right]$$

for the selected parents. If overdominance is greater on the yield scale than on the selection scale, k a larger negative than h, the regression will always be positive. In this case, which seems likely when artificial selection is not very intense, the selection holds gene frequency at an equilibrium value which is higher than is optimum on the yield scale (optimum is used for the yield scale rather than equilibrium because when $q = (1-k)/(1-2k)$ the mean of a random mating population is at a maximum). The counterpart to this condition, which is rather likely when artificial selection becomes very intense, is when overdominance is greater on the selection scale, h a larger negative than k. Here, gene frequency is held at an equilibrium value which is below the optimum on the yield scale. The genetic effect for the offspring is always positive, $(k-h) > 0$. The sign of the genetic effect for the parents depends on the sign of $\left[k-h + sh\ (1-k)\right]$. Although no k's are indicated in Figure 2, this figure can be used to illustrate the values of s, h and k that will make the parent-offspring regression negative. Let the h's in Figure 2 now be k's. The right boundaries of the shaded areas are then optimum values, q'', of q for the yield scale. If one picks for the selection scale some other equilibrium value, q', of q between .5 and q'', remembering that $h = (1-q')/(1-2q')$, the parent-offspring regression will be negative for any value of s that falls within the shaded area for that particular q'. For example, consider the middle graph in Figure 2. The optimum value of q for $k = -1$ is $q'' = 2/3$. If .6 is chosen as the equilibrium value, q', of q, $h = -2$, the parent-offspring regression will be negative for values of s from slightly less than .3 to 1. One important point to note here is that the parent-offspring regression is negative for a larger range of values of s as the difference between q' and q'' decreases. This means that a very small difference between h and k, h a greater negative than k, will cause the parent-offspring regression to be negative in the presence of small values of s. However, a more logical way of interpreting this is that small values of s restrict within narrow limits the values h can take for a given k and for the parent-offspring regression to be negative. It is not likely that s would ever be as large as .1 for an individual gene (unless sub-lethal or lethal). Although attaching values to s amounts to sheer speculation, it is believed that s is more likely to be of the order of .01 or less. Such numerically small values of s limit the conditions under which the parent-offspring can be negative. For example, when $s = .01$ and $k = -1$, the parent-offspring regression is negative if

$$k-h + s\,h\,(1-k) < 0 \quad \text{and} \quad k-h > 0,$$

$$-1-h + .02h < 0 \quad \text{and} \quad -1-h > 0,$$

and thus h has the following limitations,

$$-1.02 < h < -1.00,$$

or h must be a greater negative than -1.00 but a lesser negative than -1.02. For $k = -.1$ and $k = -10$ h must fall within the limits of

$$-.101 < h < -.100,$$

and

$$-11.25 < h < -10.00,$$

respectively. If h is outside the ranges indicated for that set of conditions, the parent-offspring regression is positive.

The other special case to be considered is where gene frequency is optimum with respect to the yield scale, $q = (1-k)/(1-2k)$, and selection is tightened up or relaxed so that gene frequency changes. If selection is changed such that q becomes larger than the optimum value (overdominance is greater on the yield scale), as seems likely when the selection intensity is relaxed, the parent-offspring regression is positive. On the other hand, if selection decreases gene frequency (overdominance is greater on the selection scale), as seems more likely when the intensity of selection is increased greatly, the parent-offspring regression is negative. When equilibrium frequency is reached with respect to the increased selection, $q = (1-h)/(1-2h)$, the conditions necessary for the parent-offspring regression to be negative are given in the case just previous to this one.

In terms of the present data, a parent-offspring regression could be negative, but would require overdominance on the yield scale. In addition, a persistent type of selection is required where overdominance is greater on the selection scale than the yield scale, and where an equilibrium value of q with respect to selection is slightly less than optimum with respect to yield. If selection is relaxed or h changes so that it is outside of very narrow limits, the parent-offspring regression is positive. Selection procedures changed some with time in the present data. Selection also varied considerably from year to year for the inbred lines depending on defects and difficulties which arose with this sort of breeding procedure. The exacting requirements that overdominance be only slightly greater on the selection scale than on the yield scale, makes it seem unlikely that the parent-offspring regression would be negative for a trait such as weight, where the selection pressure may be quite variable. However, for a trait such as size of litter or number of pigs reared, where h may be the same as k without any conscious selection (man's emphasis), the additional conscious selection could well cause overdominance to be greater on the selection scale than on the yield scale. This would cause the parent-offspring regression for litter size to be negative, provided that overdominance on the selection scale was only slightly greater than on the yield scale.

Difference Between Selective Scale and Yield Scale

The selection scale can differ from the yield scale either because of the intensity of selection or because of simultaneous selection for several characters. In the latter category, natural selection or survival rate must be included. Although the problem was not investigated, it is very likely that h varies with the intensity of selection or the proportion of individuals saved.

When selection is based on several characters, genes with manifold effects will be selected according to some average effect, which may be quite different from the effect for a particular character. For example, suppose one gives two traits equal weight in a selection index. Now, suppose the genetic effects of a locus are entirely additive, equally important for each trait, but negative in one trait and positive in the other trait. The yield values for each trait are:

Genotype	Trait 1	Trait 2	Average
AA	0	-1	- 1/2
Aa	- 1/2	- 1/2	- 1/2
aa	-1	0	- 1/2

If selection is proportional to average merit it would not effect any change in gene frequency. This result is identical with that of a negative genetic correlation. For this locus the genetic correlation between the two traits is -1. The two approaches are the same. The selective value for a gene depends on its average effect. If two traits are negatively correlated for genetic reasons, some genes which affect both traits in opposite directions must be present. Selection would change the frequency of these genes only in accordance with their average merit. The average merit scale could well be one of overdominance, even if dominance were only partial for any one trait. This point is easily seen by letting k take some value between 0 and 1/2, rather than 1/2, in the previous example. If the more favorable effect of a gene is generally dominant, negative genetic correlations then indicate the presence of genes which express overdominance on their average merit scale.

Natural selection or survival rate must also be included in the picture, although little use may be made of it. If genes lower survival in some manner, but have desirable effects on certain characteristics, conscious selection for the genes would oppose natural selection against the genes. For example, Lerner (1951) studied number of eggs laid, number of chicks hatched and the percentage of hatchability with respect to egg weight. It was found that maximum values for all three measurements fell into the intermediate egg weight classes when birds were grouped according to the characteristic weight of eggs they produced. Lerner concludes that artificial selection is in the opposite direction from natural selection for egg weight and that artificial selection is maintaining egg weight somewhat above the optimum at the expense of performance in the other three characters. The result is similar to the previous example where genes had opposite effects on two traits, and were consciously selected for and against.

There is little evidence in this study for genes expressing overdominance in average merit because of negative genetic correlations (Table 13).

Heritabilities

Heritability as estimated in this study is not a fixed parameter, but a description of the relative importance of heredity and environment in determining differences among individuals in a particular environment, at a particular time and belonging to a particular breed. In comparing these estimates with those of other workers, the relative importance of sources of variation may differ, depending upon the experimental control of environmental variation, the method of analysis, and the gene frequencies of the population studied. Estimates of heritability may also contain contributions from dominance deviations and epistatic deviations and perhaps from environmental correlations which might have affected the observed resemblance between relatives.

Doubling the regression of daughter on dam includes part of the epistatic variance, if any exists, as well as the additive genetic variance. None of the dominance deviations contribute to the parent-offspring resemblance in a random mating population. Correlations between the environments of dam and offspring, which would contribute to the regression, are thought to be nil, since no effort was made to treat parent and offspring alike. It is possible, however, that the environment a gilt receives could carry over and influence the litter of pigs that she has. To test this point, one could compare heritabilities estimated from different-aged dams. Even then, the issue might be confused by genes which affect the gilt's own performance, but cause her maternal abilities to differ with age. For example, genes which increase growth may give a gilt not yet grown, an advantage in mothering her first litter, but make her so large and clumsy as a mature animal that she is a poor mother. In any case, these data were not extensive enough to test differences between heritabilities estimated from dams different in age.

The offspring-dam regression may have a genetic contribution for maternal ability of the dams for traits like 56-day weight. For example, differences among offspring's phenotypes, Y's, which are influenced maternally by the dams, may be considered to be a function of differences between the pig's own additive genetic values, G_{oy}'s, of differences between additive genetic values of the dams' maternal abilities, G_{my}'s, and of differences in the environments, E_y's,

$$Y = \mu_y + G_{oy} + G_{my} + E_y.$$

A similar description of the dam's phenotype is

$$X = \mu_x + G_{ox} + G_{mx} + E_x.$$

These considerations are also given in a path coefficient diagram in Figure 3, which in addition indicates the relationship between the components of X and Y. The correlation between the additive genetic effect of the dam's own genes in influencing her own growth, G_{ox}, and the additive genetic effect of the dam's own genes in maternally influencing the growth of her offspring, G_{my}, which results from the pleiotropic effects of the dam's genes, is written as $\rho_{G_oG_m}$. The correlation between G_{oy} and G_{mx} is $\rho_{G_oG_m}/4$, since it is the correlation between the additive genetic effect of the offspring's own genes in influencing its own growth and the additive genetic effect of the granddam's own genes in maternally influencing the growth of the dam. The offspring-dam regression may then be written as

$$b_{yx} = \frac{\frac{1}{2}\sigma^2_{G_o} + \frac{1}{2}\sigma^2_{G_m} + \frac{5}{4}\rho_{G_oG_m}\sigma_{G_o}\sigma_{G_m}}{\sigma^2_x} .$$

which is one-half the heritability of the pig's own influence on his growth, plus one-half the heritability of the dam's maternal influence on the pig's growth and plus an additional term which indicates the additive genetic relationship between the two influences.

A crude separation of the two genetic variances and the genetic correlation may be accomplished with the regression of offspring on sire and the correlation between paternal half-sibs, in addition to the regression of offspring on dam. Since the sire influences the offspring only through the gamete that he passes to them, the regression of offspring on sire is:

$$b_{yx_s} = \frac{\frac{1}{2}\sigma^2_{G_o} + \frac{1}{4}\rho_{G_oG_m}\sigma_{G_o}\sigma_{G_m}}{\sigma^2_x} .$$

Even here, the offspring's phenotype may be correlated with that of the sire because of pleiotropic genes which maternally affected the sire's phenotype and were passed on to the offspring and influenced the phenotype of the offspring directly. The paternal half-sib correlation, however, includes only the effects of the pig's own genes,

$$\rho_{y_sy_s} = \frac{\frac{1}{4}\sigma^2_{G_o}}{\sigma^2_x} .$$

There are then three unknowns and three equations with which to solve for the desired variances and correlation. Sampling errors of estimates obtained by this procedure would no doubt be very large. The present data did not lend themselves to estimating all three

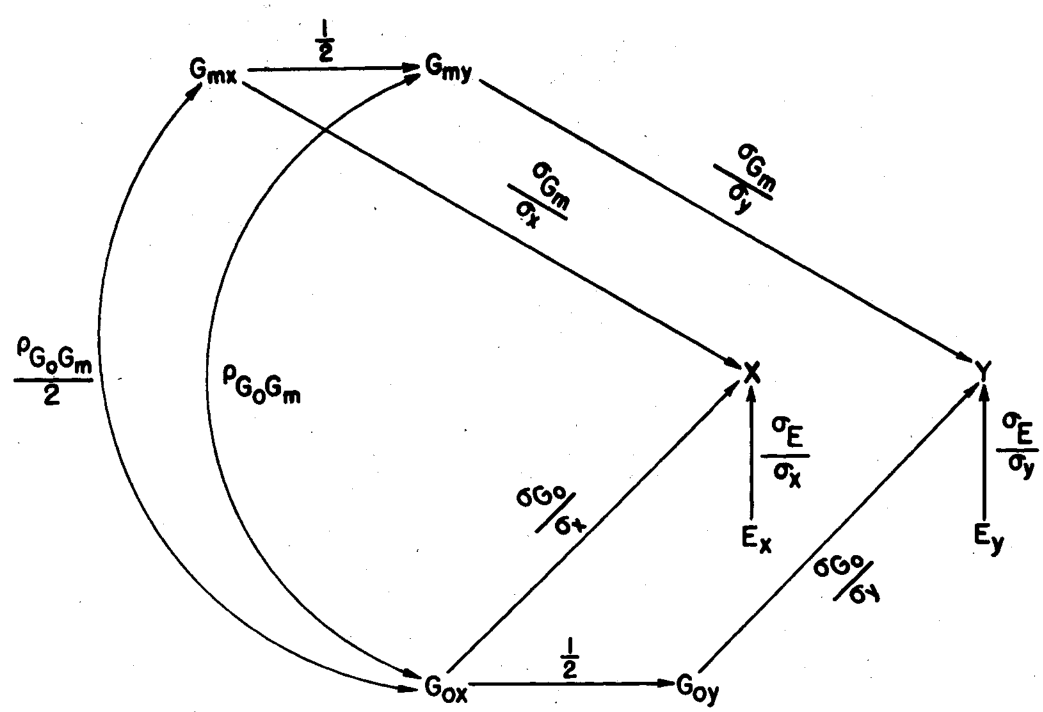

Fig. 3. Path Coefficient Diagram Showing the Relationship Between Offspring and Dam for a Character that is Influenced Maternally by the Genes of the Dam and Directly by the Individual's Own Genes.

quantities and this was not done. The method is mentioned merely to help clarify the type of variation that may influence the relationships between different relatives. To be absolutely complete, epistasis and dominance should have been included in the above considerations. The regressions measure the indicated quantities perfectly only in the absence of epistasis and dominance, and of environmental correlations other than those incident to G_m.

Four times the paternal half-sib correlation, the principal estimator of heritabilities in swine, other than resemblance between offspring and parent, approximates the fraction of the total variance that is additively genetic plus less than half the portion of the epistatic variance present in estimates derived from the regression of offspring on parent. If the progeny of one sire is not treated differently from that of another sire, there should be no correlation between the environments of half-sibs. Any differences in the treatment of progenies by different sires will contribute directly to the half-sib correlation. Such practices as mating boars to sows which are contemporary to them in age, for example, or mating one boar to the first sows that manifest heat and another boar to a later group of sows may bias the half-sib correlation considerably.

Much epistatic variance would tend to make the estimate of heritability from the parent-offspring regression larger than the one from the paternal half-sib correlation. If the environment a dam receives influences her offspring maternally, the offspring-dam regression may be increased or decreased relative to the paternal half-sib correlation. An increase would mean that a favorable environment of the dam affected the offspring favorably. Additive genetic maternal abilities of the dams may tend to make estimates of heritability from offspring-dam regressions larger or smaller than from paternal half-sib correlations, for traits like 56-day weight. They would be smaller if

$$\sigma_{G_m} + \frac{5}{2} \rho_{G_oG_m} \sigma_{G_o} < 0 \quad \text{or} \quad \rho_{G_oG_m} < -\frac{2}{5} \frac{\sigma_{G_o}}{\sigma_{G_m}} \quad ,$$

and thus depends on the genetic correlation between the direct and maternal influence and on the ratio of their additively genetic standard deviations.

The manner by which environmental sources of variation are removed in computing the estimators may influence the estimates obtained. The data are sometimes corrected for extraneous sources of variation such as years, seasons, ages of dam and lines. The estimates are then obtained from the corrected data. The method used in this study was to obtain the estimates from individuals which were contemporary with respect to the above sources of variation, thus insuring their removal. The two methods are not identical. Although no particular biases or correlations can be foreseen to be introduced by first correcting the data, a better procedure is to insure that none are introduced, particularly when estimating correlations of 0.1 or less. The question really asked of these data is whether differences among dams, which are contemporary with respect to line, year, season, and age of dam, are of any utility in predicting differences among their offspring. The significance of the answer is apparent, even without the genetic interpretation.

It does not seem desirable to compare the estimates obtained from these data with each of those reported by other authors, or to make a detailed inquiry into the possible causes of discrepancies in each case. The experiments include a variety of breeds, inbred lines and crosses among them. The number of animals vary. The methods of analysis differ, particularly with respect to the removal of environmental sources of variation. Sometimes, too, it is not wholly clear just what was done. For a general comparison, the heritabilities in Tables 1, 2 and 3 are ranked in Table 16, along with the ones from these data. Only those measuring the same traits, which were taken at approximately the same ages as the traits in this study, are included.

The heritabilities of weight, found in the present data, fall within the range of those found elsewhere, but they are somewhat lower than the average of the others, particularly for 154-day weight. The heritabilities of litter size from these data are far below the other estimates reported. It is hard to reconcile oneself to accepting these differences for litter size as sampling differences. Overdominance and selection could cause the parent-offspring regression to be negative. Whatever the answer is, these data make it seem very unlikely that heritability of litter size is much larger than zero.

Table 16

Estimates of Heritability (in percent)

Estimator	From Other Experiments	In Table	Trait	From these Data
4(Paternal half-sib correlation)	0 7 14 15	1	w_{56}	
2(Offspring on dam regression)	-19 10 16	2	w_{56}	3
2(Offspring on sire regression)	4	2	w_{56}	
(Offspring on mid-parent regression)	9	2	w_{56}	
4(Paternal half-sib correlation)	20 24 25 26 34 43	1	w_{154}	
2(Offspring on dam regression)	-32 14 62	2	w_{154}	7
2(Offspring on sire regression)	22	2	w_{154}	
4(Paternal half-sib correlation)	16	3	n_b	
2(Daughter on dam regression)	14 25	3	n_b	-11
2(Daughter-dam correlation)	34 44	3	n_b	
4(Paternal half-sib correlation)	12	3	n_{56}	
2(Daughter on dam regression)	19 32	3	n_{56}	-9
2(Daughter on dam regression)	42	3	n_{154}	-15

Low or negligible heritabilities are in accord with the results of the selection study, Dickerson (1951) and Kottman (1952).

If heritabilities are zero or low, is there any hereditary variance? If so, is it primarily dominance, overdominance or epistasis? These questions cannot be answered definitely here, but different sorts of information may be used as indicators. The different measures of heritability include some epistasis. Negligible estimates of heritability, then, indicate that the additive kind of epistatic interactions are of little importance. Since heritabilities from paternal half-sib correlations contain something less than half as much epistatic variation as those from offspring-parent regressions, a comparison of these two estimators may indicate the importance of the additive kind of epistasis. Although the comparisons in Table 16 are not reliable enough for conclusive evidence, there is little indication that the two estimators give different results.

The depression of the mean as inbreeding proceeds, which has been noted in many other organisms as well as in swine, indicates that there is hereditary variation. No argument would seem to explain the inbreeding depression, without admitting the existence of hereditary variation at the onset of inbreeding. The effect of inbreeding is to produce homozygosity or to eliminate heterozygosity. If the heterozygote is not on the average better than the average of the two homozygotes, it is difficult to see how inbreeding could cause the mean to decrease. This implies that there is at least partial dominance of favorable genes.

The Illinois selection experiment (Krider, et al. 1946) furnishes evidence on the question of dominance. The mean of the superior line actually decreases, but not as much as the mean of the inferior line. Although the data are too few to be conclusive, and year differences were confounded in time trends of the means, such results might be expected when the major variation was of the overdominance type and gene frequencies were in equilibrium. The intensifying of selection in a selection experiment could make overdominance greater on the selection scale than on the yield scale for the high line. The mean of the high line would then go down slightly, depending on the intensity of selection. On the other hand, the existence of overdominance of favorable genes would permit selection to decrease the mean in the low line. If such conditions were true, heritability estimated from the selection experiment is an erroneous indicator of the improvement that can be made through selection. The heritability is valid in that it is indicative of possible conditions (i.e., low frequency of favorable overdominant genes in a low line) where considerable improvement may be accomplished by selection.

Genetic Relationships

Two characters might be uncorrelated genetically, even when many genes exist which affect the two traits in different directions. For this to be true, however, would require that there be equally effective genes which affect the two traits in the same direction. One would think that the latter kind of genes, those which have the same sort of effect on two or more traits, would soon become nearly fixed and would contribute little to the variation. The remaining variation then might be expected to stem from genes which had both good and bad effects. When that stage was reached the two characters would be negatively correlated genetically.

These data indicate the genetic relationship between litter size and growth to be positive. Even if one suspected an environmental contribution in the correlation between the growth rate of a dam and the size of her subsequent litter, the relationship between the size of litter the dam produced and the growth rate of her grandchildren was also positive (Table 13).

The slightly negative relationship between the average 154-day weight of the pig and the 56-day weight of the dam may be interpreted as an indication of an antagonism between the dam's transmitted and direct influence on growth. It could also result from genes which cause the pig's own ability to grow to differ in the two periods of growth. In either case, the result is due to genes which actually affect the two phases of growth in different directions. Nordskog _et al_. (1944) indicated that they found an estimate of the genetic correlation between genotypes before and after weaning to be negative but the method of obtaining it was not given. Hazel _et al_. (1943) found the genetic correlation between the two periods of growth to be positive. Their correlation included line differences, however, and is not necessarily comparable to the one of Nordskog _et al_. (1944).

The correlation between the average weight of the pig at 56 days and the dam's weight at 154 days was positive (.010, Table 13). If there were no environmental contributions to the correlation and if the maternal influence of the dam were independent of her transmitted influence, this correlation is expected to be the same as that between the 56-day weight of the dam and 154-day weight of her offspring (-.017, Table 13). An environmental contribution could cause the difference. An antagonism between the dam's maternal and transmitted influence would tend to make the former correlation even more negative than the latter one. Both correlations are small and the difference between them is small and statistically non-significant.

SUMMARY

At the onset of this study two contradictory pieces of evidence were at hand. Heritabilities of litter size and growth, although not large, had been estimated to be of such a size that one could expect some improvement from selection. On the other hand results of the selection study (Dickerson et al. 1951) indicated that improvement in these traits did not result, in spite of sizeable selection differentials. The contradiction could be a real one and not merely due to sampling errors if negative genetic correlations between desired characters were prevalent, if an important part of the variation consisted of the additive kind of epistasis, or if natural selection holds gene frequencies at some value other than optimum for the characters in spite of conscious selection for them.

The primary purpose of this study was to examine further the heritability of litter size and of growth in swine, and to investigate the genetic relationship between them. Litter size was measured at three different ages: birth, 56 days, and 154 days. Growth was measured by weight at 56 and 154 days.

The data came from twelve inbred lines of Poland China swine and one inbred Danish Landrace line, and encompassed a total of 1980 litters. The analyses were of the variation within groups in which all individuals were contemporary with respect to line, year, season and age of dam.

Heritability was estimated as twice the regression of daughter or litter on dam. The numerical values thus obtained were as follows:

Number born	$-.11 \pm .07$
Number at 56 days	$-.09 \pm .08$
Number at 154 days	$-.15 \pm .08$
Weight at 56 days	$.03 \pm .08$
Weight at 154 days	$.07 \pm .10$

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Correlations between the traits of the offspring and those of the dam are given in Table 13. No genetic correlations were computed since they involved either a negative heritability or correlations which differed in sign. The relationship between additive genetic effects for litter size and weight were indicated to be positive, while additive effects for growth before weaning appeared to be independent of those for growth after weaning.

Sampling errors or overdominance and selection seem to be the only plausible explanations for negative estimates of heritability for litter size. Consequently, heritability of litter size was concluded to be zero. Even for weight, the estimates are quite small and are not significantly different from zero.

This study then conforms to the results of the selection study but contradicts previous estimates of heritability.

The fact that the mean is depressed with inbreeding and that inbred lines generally become differentiated from each other distinctly is rather conclusive evidence that there is hereditary variation of some sort. Whether more of this variation stems from overdominance than from epistasis is not clear. The inbreeding depression coupled with the results of this study suggest that overdominance is an important factor.

A breeding plan which will capitalize on both overdominance and epistasis is to form many distinct families or inbred lines as rapidly as possible. The lines which perform the best in crosses are then used in the production of market hogs. It may be necessary to use these lines in a rotational crossbreeding system to avoid the lowered performance levels of inbred dams. However, foster rearing of pigs might overcome part of this handicap. New inbred lines would be made as fast as the test-crossing procedure permits.

Foreign breeds of swine warrant consideration in the above crosses because they are a possible source of desirable genes which may not be present in the domestic breeds. Although isolation has already created distinct breeds, it may be desirable to make inbred lines from the foreign breeds for use in the crosses.

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ACKNOWLEDGMENTS

The author expresses his appreciation to Dr. L. N. Hazel for his counsel and criticism during this investigation, to Dr. J. L. Lush for his constructive criticisms during the preparation of the manuscript and to Dr. W. A. Craft for his encouragement throughout the study.