GENETIC, PHENOTYPIC, AND MATERNAL INTERRELATIONSHIPS OF GROWTH IN MICE¹

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CONSIDERABLE speculation has been presented regarding the genetic correlation between growth and postnatal maternal performance (lactation) in mammals, particularly since DICKERSON (1947) and DICKERSON and GRIMES (1947) found evidence which has been interpreted as indicating a negative genetic relationship between these two traits in swine. The study of this relationship in mammals is complicated due to problems involved in separating the transmitted and direct effects of the dam's genes upon the growth of her young. The dam transmits a sample half of her genes to her young, and her genotype is also expressed directly in the uterine and postnatal circumstances which influence their growth. The relationship is further confounded in most mammals by the necessity to measure lactational performance by the preweaning growth of the young. These points have been discussed in detail by KOCH and CLARK (1955) and WILLHAM (1963).

Knowledge of the relationship between growth and postnatal maternal performance is most important for an accurate interpretation of selection response and in devising breeding programs for traits affected by maternal influences. If the genetic correlation is negative, maternal performance may decline under continuous selection for growth to the extent that preweaning viability and growth may be retarded. Such retardation, if extreme, would not likely be offset by compensatory growth during a later period despite the presence of genes for rapid growth. The deterioration of maternal performance could be prevented by including some measure of it, properly weighted, in the selection criteria. Should the genetic correlation between growth and maternal performance be strongly negative, other breeding methods might be more desirable for overcoming the genetic antagonism between the two traits. Selection for maternal performance in one line and for growth in a second for eventual crossing has been suggested in such situations, although its applicability depends upon the nature of the action of the genes influencing the traits and the reproductive pattern of the species.

These investigations were undertaken to estimate the genetic correlation between measures of growth and postnatal effects, utilizing cross-fostering to separate the influence of prenatal and postnatal factors. Phenotypic, genetic, and ma-

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ternal correlations between weights at different ages and gains in weight were also determined to provide a more complete assessment of the interrelationships among these traits.

EXPERIMENTAL PROCEDURE

The experimental data were obtained from the cross-fostering experiments with mice reported by COX, LEGATES and COCKERHAM (1959) and YOUNG, LEGATES and FARTHING (1965). The population of mice studied and the methods employed have been described in these papers. The mice were taken from Generations 7, 8, 9, 13, 14 and 15 of a population arising from a cross of four inbred lines and maintained randomly thereafter. The cross-fostering experiments consisted of forming groups of three dams that had littered within a 12-hour period. Each dam was allowed to nurse two of her own young and two young from each of the other two litters. Individual weights were taken, and analyses of variance and covariance were made on these weights and other traits to estimate components of variance and covariance due to prenatal, postnatal, prenatal $\times$ postnatal and residual factors. In addition, covariances were obtained between certain prenatal and postnatal litter totals to obtain estimates of covariance components for postnatal maternal performance and the various weights and gains. These components of variance and covariance were utilized to estimate the variances and covariances needed to determine the genetic correlations between measures of growth and postnatal maternal performance, and the genetic, maternal and phenotypic correlations among the traits measured.

In the developments which follow, the letters g , d , and i denote the additive genetic effects, dominance deviations and epistatic deviations, respectively. The sum of these genetic effects has been denoted by the letter h . The effect of environmental differences of a permanent nature has been designated as pe . The genetic and permanent environmental effects indicated above are also identified for each trait by using the letters m , u and w to represent postnatal maternal performance, uterine environment, and weight or gain, respectively. Hence, g_m is defined as the additive genetic effect for postnatal maternal performance, and $\sigma^2_{g_m}$ is defined as the variance due to additive genetic differences for postnatal maternal performance. The other effects and variances for the three types of traits are defined in a similar manner.

The variances and covariances used in this investigation and the assumptions which were necessary to obtain the desired estimate are described below.

Prenatal variance. The prenatal component of variance (σ^2_a) includes variance due to hereditary and uterine factors which cause members of a litter to be more alike than mice from unrelated litters. The uterine factors result partly from the inherent qualities of the dam, but they act wholly as environmental stimuli to the growing young. If the hereditary influences of the young and the uterine effects of the dam are uncorrelated, the prenatal variance can be written as the sum of the variances for these two effects ($\sigma^2_a = \sigma^2_{h_w} + \sigma^2_u$). The hereditary variance for weight operating between full-sib progenies includes one half of the additive genetic variance ($\sigma^2_{g_w}$), one fourth of the dominance variance ($\sigma^2_{d_w}$), plus one fourth or less of the epistatic variance ($\sigma^2_{i_w}$). In the case of the variance due to uterine differences, the full impact of similar components ($\sigma^2_{g_u}$, $\sigma^2_{d_u}$, $\sigma^2_{i_u}$) would be expressed. An additional component due to permanent environmental effects in the dam ($\sigma^2_{pe_u}$) also may influence the intra-uterine growth and development of her young.

The prenatal component of variance is important as it provides the means for estimating the additive genetic variance for measures of weight and gain ($\sigma^2_{g_w}$). The use of σ^2_a for this purpose requires that certain assumptions be made regarding the uterine and nonadditive genetic effects. Only meager evidence is available upon which to base such assumptions, as the influence of uterine environment and nonadditive gene action upon weights and gains in mice have received little attention. BRUMBY (1960) studied the uterine effects on body weight in a large and a small strain of mice resulting from many generations of selection. He found an important uterine effect on birth weight, but no appreciable effect was evident on weekly gains to six weeks of age. BRUMBY's results concerning the importance of prenatal and postnatal influences on body weights were in agreement with those of BATEMAN (1954), and much different from those ob-

tained from the population under study here (Cox *et al.*, 1959; YOUNG *et al.*, 1965). Therefore, BRUMBY's findings appear to have limited value in clarifying the situation in the present population. In the case of non-additive genetic variance, both Cox *et al.* (1959) and MILLER *et al.* (1963) found evidence that non-additive genetic variance for body weight was unimportant in the population studied here. Hence, the variance due to both uterine and nonadditive genetic effects were assumed to be negligible for weights and gains at 12 days or later, and σ^2_{gw} was estimated by doubling σ^2_a .

Postnatal variance: The postnatal variance (σ^2_p) is simply the postnatal counterpart of variance in uterine environment, and it consists of additive genetic (σ^2_{gm}) dominance (σ^2_{dm}), epistatic (σ^2_{im}) and permanent environmental (σ^2_{pem}) variances for postnatal maternal performance. The first three components (σ^2_{gm} , σ^2_{dm} and σ^2_{im}) of the postnatal variance are due to genetic differences between dams in their influence on the growth of the young mice which they nurse. The remaining component (σ^2_{pem}) is due to any environmental effects which have a permanent influence upon the postnatal maternal performance of the dam. Although three of the four components comprising σ^2_p are due to genetic differences in the dams, all four components are environmental in respect to their effect upon the growth of the young.

Again, it was necessary to accept certain assumptions to obtain an estimate of σ^2_{gm} , the component of primary interest. The assumption made was that the additive genetic variance for postnatal maternal performance (σ^2_{gm}) was equal to the sum of σ^2_{dm} , σ^2_{im} and σ^2_{pem} , the other three components included in σ^2_p . Numerous heritability and repeatability estimates for lactation milk yield in dairy cattle indicate a relationship of this order in that species. Estimates of heritability and repeatability for lactational performance in mice are much less numerous and are based upon much smaller numbers. Because of the lack of reliable estimates from mouse populations, and because those estimates available for mice tend to correspond to values obtained from dairy cattle, σ^2_{gm} was estimated by dividing the postnatal variances (σ^2_p) by two.

Prenatal-postnatal covariances: Genetic covariances between body weight or growth measurements and postnatal maternal performance were estimated from the covariance between prenatal totals for the various measures of growth or weight and the postnatal total for traits most directly associated with differences in lactational performance. Inspection of Table 1 will be helpful in visualizing the covariances that were used.

In Table 1, the Y_{klm} represent observations on the m th individual belonging to the l th prenatal litter and the k th postnatal litter. The $Y_{k..}$, $Y_{l.}$, and $Y_{...}$ terms are row (prenatal), column (postnatal), and group totals, respectively. For these analyses, only cross-fostering groups with

TABLE 1

Cross-fostering group showing model symbolism for calculation of prenatal-postnatal covariances

Prenatal dam	Postnatal dam			Prenatal dam totals
	A	B	C	
A	Y_{111}	Y_{121}	Y_{131}	$Y_{1..}$
	Y_{112}	Y_{122}	Y_{132}	
B	Y_{211}	Y_{221}	Y_{231}	$Y_{2..}$
	Y_{212}	Y_{222}	Y_{232}	
C	Y_{311}	Y_{321}	Y_{331}	$Y_{3..}$
	Y_{312}	Y_{322}	Y_{332}	
Postnatal dam totals	$Y_{.1.}$	$Y_{.2.}$	$Y_{.3.}$	$Y_{...}$

a value for each Y_{klm} were used, so that each prenatal and postnatal total consisted of the sum of six observations, and the group total consisted of the sum of 18 observations.

The expectation of the mean cross product between the prenatal totals for the i th trait and the postnatal totals for the j th trait was derived and found to be $36 \sigma_{a_{ij}}$ (See APPENDIX). Division of the mean cross product by 36 provided an estimate of the covariance between prenatal effects in the i th trait and postnatal effects in the j th trait.

The definitions of the prenatal and postnatal covariances were implied in the preceding definitions of the corresponding variances. The covariance between prenatal and postnatal effects can be written as follows:

$$\sigma_{ap} = \frac{\sigma_{g_w g_m}}{2} + \frac{\sigma_{d_w d_m}}{4} + \frac{\sigma_{i_w i_m}}{4} + \sigma_{g_u g_m} + \sigma_{d_u d_m} + \sigma_{i_u i_m} + \sigma_{p_{e_u} p_{e_m}}.$$

The first six terms on the right side of the above equation are genetic, and will exist only if the same genes or gene combinations affect both traits. Again retaining the previous assumption that dominance and epistatic effects are negligible, the expression reduces to:

$$\sigma_{ap} = \frac{\sigma_{g_w g_m}}{2} + \sigma_{g_u g_m} + \sigma_{p_{e_u} p_{e_m}}$$

DICKERSON and GRIMES (1947) suggest that the additive genetic effects for uterine environment (g_u) and suckling ability (g_m) are negatively correlated, although the supporting evidence is not conclusive. Knowledge of the covariance between permanent environmental effects for uterine environment (p_{e_u}) and postnatal maternal performance (p_{e_m}) is also lacking. Therefore, both $\sigma_{g_u g_m}$ and $\sigma_{p_{e_u} p_{e_m}}$ were assumed to be negligible, and $\sigma_{g_w g_m}$ was estimated by doubling σ_{ap} . The genetic correlations between postnatal maternal performance and the various measures of weight and gain were computed according to the following expression:

$$r_{g_w g_m} = 2\sigma_{ap} / (\sigma_{g_w} \cdot \sigma_{g_m})$$

Relationships between different measures of growth: The other relationships considered in this study were those between gains during different periods, and between weights taken at different ages. To obtain these relationships, covariances were estimated from analyses of covariance as shown in Table 2. This covariance analysis is similar to the analysis of variance applied to the traits individually, the results of which have been reported previously (Cox *et al.*, 1959; YOUNG *et al.*, 1965). From Table 2, it is seen that covariance estimates can be obtained for $\sigma_{(ap)_{i(sp)j}}$, $\sigma_{a_{ij}}$, $\sigma_{p_{ij}}$, and $\sigma_{e_{ij}}$, where the i and j subscripts refer to the two traits considered in the covariance analysis.

Genetic correlations between the various traits were obtained from the prenatal components of variance and covariance for a pair of traits. These prenatal components were taken to equal one half of the respective genetic components. Maternal correlations were obtained from the postnatal components of variance and covariance; hence, these computed correlations would involve only the postnatal maternal influences, uterine influences are not included. Phenotypic correlations were computed after summing the four components of variance for each of the traits and the four components of covariance between each pair of traits.

RESULTS AND DISCUSSION

Genetic relationship between weight and postnatal maternal performance: Estimates of the genetic covariances and genetic correlations between various measures of weight and gain and postnatal maternal performance are given in Table 3. These estimates were obtained from the cross-fostering groups for which data were complete at the indicated age. The number of such groups contributing to each correlation can be obtained by halving the degrees of freedom indicated in the footnotes to Table 3. The two measures of postnatal maternal performance used were 12-day weight and preweaning gain for litters of six mice. Previous analyses of the data (Cox *et al.*, 1959; YOUNG *et al.*, 1965) had shown them to be satisfactory measures of this trait.

TABLE 2

Analysis of covariance for traits i and j for a single cross-fostering group

Source	Degrees of freedom	Expectations of mean cross-products
Total	17	
Prenatal (rows)	2	$\sigma e_i e_j + 2 \sigma ap_i ap_j + 6 \sigma a_i a_j$
Postnatal (columns)	2	$\sigma e_i e_j + 2 \sigma ap_i ap_j + 6 \sigma p_i p_j$
Prenatal $\times$ postnatal	4	$\sigma e_i e_j + 2 \sigma ap_i ap_j$
Within full sibs	9	$\sigma e_i e_j$

The genetic correlations given in Table 3 range from $-.23$ to 0.57 . Negative estimates were obtained between postnatal maternal performance and weights taken at or prior to weaning, and between postnatal maternal performance and gain from 42 to 56 days. The negative estimates of the genetic correlations between postnatal maternal performance and the early weights probably should be viewed with caution, as uterine influences would most likely affect the weights taken at these ages. If the genetic covariance between additive genetic effects for uterine environment and additive genetic effects for postnatal maternal performance ($\sigma_{u^*g_m}$) is negative, as suggested by DICKERSON (1947), then $\sigma_{u^*g_m}$ rather than $\sigma_{u^*g_m}$ may be responsible for these negative estimates. The negative estimates between postnatal maternal performance and gain from 42 to 56 days are not likely to be explained in this manner.

Inspection of Table 3 reveals that the genetic correlations between postnatal maternal performance and weights tend to increase with age. This tendency, if real, may reflect a changing genetic relationship between weight and postnatal

TABLE 3

*Estimates of prenatal-postnatal covariances from prenatal and postnatal totals and of genetic correlations from these covariances and corresponding variances**

Growth trait	Prenatal effect	Postnatal maternal measurement	
		p_{12}	p_{21-0}
12-day weight	a_{12}	$-.0215^\dagger$ $-.23$	$-.0196^\ddagger$ $-.13$
21-day weight	a_{21}	$0.0130^\ddagger$ 0.09	$-.0050^\ddagger$ $-.02$
42-day weight	a_{42}	$0.0856^\S$ 0.29	$0.1057^\S$ 0.23
56-day weight	a_{56}	$0.1279^\parallel$ 0.42	$0.2661^\parallel$ 0.57
21—0 day gain	a_{21-0}	$0.0186^\ddagger$ 0.14	$0.0085^\ddagger$ 0.04
42—21 day gain	a_{42-21}	$0.0444^\S$ 0.23	$0.1072^\S$ 0.35
56—42 day gain	a_{56-42}	$-.0008^\parallel$ $-.01$	$-.0266^\parallel$ $-.17$

* Upper member of each pair of values is the covariance; lower member the correlation.

† Based on 96 d.f. within groups. ‡ Based on 82 d.f. within groups. § Based on 64 d.f. within groups. || Based on 40 d.f. within groups.

maternal performance, or it may mean that the assumption that uterine environment was not influential is more nearly fulfilled as maturity is approached.

When the postnatal gains to 42 days of age were considered, the estimates of the genetic correlations between these gains and postnatal maternal performance were all positive. The estimates are small (0.14 and 0.04) for gain from birth to 21 days, but they are similar in magnitude to the 0.30 for gain from 21 days (weaning) to 42 days of age. The estimated genetic correlations between gain from 42 to 56 days and postnatal maternal performance were $-.01$ and $-.17$.

There is an additional avenue through which the genetic correlations between growth and postnatal maternal performance can be estimated using the data available on cross-fostering. It has been pointed out that the prenatal component of variance for the 12-day weights of litters reared by the females which were cross-fostered (YOUNG *et al.* 1965, Table 5) provides an estimate of the variance of the transmitted effect for postnatal maternal performance. The prenatal variance components for the individual measurements taken earlier in the life cycle of these females provide estimates of the genetic variances for the weights or gains. In addition to the estimates of the genetic variances for maternal performance and for the weights or gains, the covariances between these effects are needed. These were obtained by computing the covariance between the 12-day weights of litters from cross-fostered females and the individual weights or gains of these females taken earlier in their life cycle. The same assumptions regarding nonadditive genetic effects and uterine effects were made as were held heretofore. Under these circumstances the genetic correlation between postnatal maternal performance and weight or gain was computed as follows:

$$r_{a_w a_m} = \frac{2\sigma_{a_i a_j}}{\sqrt{2(\sigma^2_{a_i}) \cdot 2(\sigma^2_{a_j})}} = \frac{\sigma_{a_i a_j}}{\sigma_{a_i} \cdot \sigma_{a_j}}$$

where the a_i are for the weight or gain measurements indicated in Table 4, and a_j is for the 12-day litter weight of litters reared by the cross-fostered females. Estimates of the variances and covariances obtained and the genetic correlations computed from them are given in Table 4.

In some cases the genetic correlations in Table 4 differ considerably from those presented in Table 3. The estimate of the genetic correlation between 12-day weight and postnatal maternal performance is $-.06$, which is somewhat closer

TABLE 4

Estimates of the genetic variances and covariances and the genetic correlations between postnatal maternal performance and weights or gains

Estimate	Measurement						
	12-day weight	21-day weight	42-day weight	56-day weight	21-42 day gain	42-56 day gain	56-84 day gain
$\sigma^2_{a_w}$	0.1514	0.3690	1.5180	1.577	0.2904	0.6652	0.1652
$\sigma^2_{a_m}$	7.6670	7.6670	7.6670	7.6670	7.6670	7.6670	7.6670
$\sigma_{a_w a_m}$	-.0656	0.7710	1.2482	0.8762	0.6728	0.4774	-.3744
$r_{a_w a_m}$	-.06	0.46	0.37	0.25	0.45	0.21	-.33

to zero than the negative estimates of $-.13$ and $-.23$ presented in Table 3. Estimates of the genetic correlations between postnatal maternal performance and later weights were all positive, but decreased as age increased. This is a reversal of the trend noted in the earlier computations where the correlations increased with age.

The genetic correlations between postnatal maternal performance and gains during the periods from birth to weaning, weaning to 42 days, and 42 to 56 days were 0.45 , 0.21 and $-.33$, respectively. The first of these estimates is much larger than the corresponding estimates of 0.04 and 0.14 from Table 3, however, the estimates for the latter two periods are in general agreement with those obtained from the previous approach.

None of the individual estimates given in Table 3 or Table 4 were found to be significantly different from zero when tested by the procedure of MODE and ROBINSON (1959). However, it does seem of some significance that all estimates of the genetic correlations between postnatal maternal performance and gains prior to 42 days of age were positive, whereas all estimates of genetic correlations between postnatal maternal performance and gains from 42 to 56 days of age were negative. Hence, the results of these investigations are at least suggestive of a positive genetic relationship between both preweaning and early postweaning gains and postnatal maternal performance, and of a negative genetic relationship between later gains and postnatal maternal performance.

Genetic, maternal and phenotypic correlations: The changing genetic relationships noted in the preceding section suggest that weight changes during the life cycle of the mouse may be controlled by different genes during different parts of the cycle. The four variance components for each trait, and the four components of covariance between each pair of traits were required to investigate further these genetic relationship, and to obtain the maternal and phenotypic relationships between the various weights and gains. The number of observations and the degrees of freedom available for estimating these components are given in Table 5. The complete variance-covariance matrix for all four sources of variation and covariation for each trait is given in Table 6.

The prenatal components of variance and covariance presented in Table 6 were used to estimate the genetic correlations, whereas, the postnatal components

TABLE 5

Number of observations for each trait and degrees of freedom available within groups to estimate the variance and covariance components

Line of analysis	12-day wt. ($\bar{X}_3$)	21-day wt. ($\bar{X}_4$)	42-day wt. ($\bar{X}_5$)	56-day wt. ($\bar{X}_6$)
Observations	1326	1306	1238	763
Prenatal	165	165	165	98
Postnatal	152	150	146	90
Prenatal $\times$ postnatal	284	279	268	171
Within	642	629	576	355

TABLE 6

*Variance-covariance matrix for the four sources of variation and covariation
for the indicated weights and gains*

Trait	Variance or covariance component	Trait						
		12-day wt. ($\bar{X}_3$)	21-day wt. ($\bar{X}_4$)	42-day wt. ($\bar{X}_5$)	58-day wt. ($\bar{X}_6$)	(21-0) day gain ($\bar{X}_7$)	(42-21) day gain ($\bar{X}_8$)	(56-42) day gain ($\bar{X}_9$)
X_3	Prenatal	0.0757	0.1067	0.1540	0.1454	0.0895	0.0430	-.0012
	Postnatal	0.4662	0.6243	0.5897	0.4429	0.6120	0.0058	-.0743
	Pre $\times$ Post	0.0215	0.0248	-.0045	0.0024	0.0250	-.0284	0.0042
	Within	0.1814	0.2157	0.3035	0.3034	0.1864	0.1192	-.0005
X_4			0.1845	0.3042	0.2951	0.1606	0.1210	-.0290
			1.1306	0.9559	0.7710	1.1264	-.1015	-.1600
			0.0316	-.0114	-.0086	0.0349	-.0525	0.0391
			0.4813	0.5787	0.5365	0.4473	0.1648	-.0314
X_5				0.7590	0.7018	0.2653	0.4537	0.0033
				0.9811	0.7810	0.9489	0.0244	-.1244
				-.1295	-.1650	0.0028	-.1166	-.0629
				2.3121	2.2028	0.5276	1.7335	-.1438
X_6					0.7885	0.2599	0.4060	0.0862
					0.7024	0.7619	0.0092	-.0792
					-.1155	0.0047	-.1565	0.0508
					2.8977	0.4818	1.6677	0.6949
X_7						0.1452	0.1081	-.0254
						1.1228	-.1005	-.1606
						0.0328	-.0401	0.0370
						0.4261	0.1433	-.0307
X_8							0.3326	0.0318
							0.1257	0.0349
							-.0660	-.1024
							1.5711	-.1155
X_9								0.0826
								0.0449
								0.1120
								0.8454

were employed to estimate the maternal correlations. The sum of the four variance components for each trait, and the sum of the covariance components for pairs of traits were utilized to obtain estimates of the phenotypic correlations between the various weights and gains. These estimates are presented in Table 7.

Table 7 shows that the phenotypic, genetic and maternal correlations between the various weights were large and positive. These results are not unexpected, since each represents a correlation between a part and a whole. It is likewise expected that the correlations will decrease as the interval between the weights increases, and the correlations in Table 7 verify that this is the case.

The more important genetic correlation estimates in Table 7 are those between

TABLE 7

*Genetic, maternal, and phenotypic correlations between the indicated weights and gains**

Trait	Correlation	Trait					
		X_4	X_5	X_6	X_7	X_8	X_9
X_3	Genetic	.90	.64	.60	.86	.27	— .02
	Maternal	.86	.87	.77	.85	.02	— .51
	Phenotypic	.83	.61	.50	.80	.12	— .08
X_4	Genetic	..	.81	.77	.98	.49	— .23
	Maternal	..	.91	.87	1.00	— .27	— .71
	Phenotypic	..	.68	.57	1.00	.07	— .13
X_5	Genetic	..	..	.91	.80	.90	.01
	Maternal	..	..	.94	.90	.69	— .59
	Phenotypic	..	..	.86	.67	.75	— .16
X_6	Genetic	..	..	..	.77	.79	.34
	Maternal	..	..	..	.86	.03	— .45
	Phenotypic	..	..	..	.56	.67	.35
X_7	Genetic	..	..	..	..	.49	— .23
	Maternal	..	..	..	..	— .27	— .72
	Phenotypic	..	..	..	..	.06	— .13
X_8	Genetic	..	..	..	..	..	.19
	Maternal	..	..	..	..	..	.46
	Phenotypic	..	..	..	..	..	— .10

* X_3 , 12-day wt.; X_4 , 21-day wt.; X_5 , 42-day wt.; X_6 , 56-day wt.; X_7 , (21-0) day gain; X_8 , (42-21) day gain; X_9 , (56-42) day gain.

gains during the three periods. These were found to be quite variable, ranging from 0.49 for preweaning gain with gain from 21 to 42 days to — .23 for preweaning gain with gain from 42 to 56 days. The genetic correlation between gains during the two postweaning periods was 0.19.

The genetic correlation between gains during the preweaning and first postweaning period is positive and large enough to suggest that many of the same genes have like effects on gain during both periods. However, the genetic correlations between gains during these two periods and the second postweaning period suggest that gain during the latter period is controlled by different genes. If the same genes are involved certain of these have opposite effects during the two earlier periods and the later periods. It is well known that the kind of tissue comprising weight gain does change as maturity is approached, and FOWLER (1958) has shown that such a change occurs at about 35 days in some mice. Prior to this age, gains could be attributed largely to protein anabolism, whereas, gains subsequent to 35 days could be largely due to fat deposition.

The maternal correlations, as computed, include only the postnatal maternal effects. Maternal correlations between gains during the three periods were also quite variable, those between preweaning gain and postweaning gains being negative; whereas, the maternal correlation between gains during the postwean-

ing periods was 0.46. The negative maternal correlations between preweaning and postweaning gains indicate that the young mice that were nursed by below average dams, tended to compensate for this by increased growth following weaning. Compensatory growth is known to occur in mammals when favorable conditions follow periods of undernutrition. The negative maternal correlations between preweaning and postweaning gains would appear to be merely expressions of this phenomenon which has been reviewed by WILSON and OSBOURN (1960).

Gains during the three periods were apparently independent, or nearly so, on a phenotypic basis. According to the results given in Table 7, the phenotypic correlation between preweaning gain and gain from 21 to 42 days was 0.06, whereas that between preweaning gain and gain from 42 to 56 days was $-.13$. The gains during the two postweaning periods were negatively correlated phenotypically; the correlation being $-.10$. None of the phenotypic correlations between gains during different periods approached significance. Even if the estimates should be real, their magnitude are such that knowledge of gains during one period would not provide a reliable basis for predicting gains during another period.

GENERAL DISCUSSION

None of the genetic correlations between growth and postnatal maternal performance obtained in this study were found to be significantly different from zero when tested by a method given by MODE and ROBINSON (1959). However, in most instances more than one method of estimating the correlations was available, and for the most part the different avenues of approach led to essentially the same conclusions. While it is true that the different methods are themselves not wholly uncorrelated, the similarity of the results obtained was not likely due entirely to this factor. Hence, it is felt that the overall findings of this investigation add materially to the information available on this subject, and that in general they add to the probable validity of the conclusions arrived at by DICKERSON (1947).

With regard to DICKERSON's work, it is felt by the authors that his findings are often misinterpreted. DICKERSON's conclusion in this area was not that growth and lactation were negatively correlated genetically, but rather that the ability to fatten and the ability to lactate were negatively correlated genetically. DICKERSON's work was done during a period when lard-type swine were still popular; consequently, at that time his findings were appropriately interpreted as demonstrating a genetic antagonism between suckling ability and the production of desirable and heavy carcasses.

The positive genetic correlations between early growth and postnatal maternal performance, and the opposite genetic relationship between later gain and postnatal maternal performance found in this study lead to the same conclusions as were suggested by DICKERSON's results; provided that the chemical composition of gains was similar for the population employed here and that studied by FOWLER (1958). FOWLER's work indicated that fat deposition contributed very little

to gains made prior to about 35 days of age, but that it was primarily responsible for gains made thereafter. This change in the composition of gains could explain why the genetic relationship between gains and lactation might change as it did here, and likewise why the phenotypic relationships between gains during different periods might be so low. The accumulation of data on the chemical composition of gains for the population used in this study is now in progress and will assist in clarifying this issue.

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SUMMARY

A cross-fostering study was conducted with mice to estimate the genetic, phenotypic and maternal interrelationships of growth and postnatal maternal performance. Estimates of the genetic correlation between growth and postnatal maternal performance were obtained by two approaches. While the results of neither approach were conclusive, they both suggested a positive relationship between early gains and postnatal performance and a negative relationship between later gains and postnatal maternal performance. These results are suggestive of a positive genetic relationship between protein anabolism and lactation, and a negative genetic relationship between fattening and lactation.

Genetic and phenotypic correlations between weights taken in this study ranged from 0.50 to 0.94. Genetic correlations between gains made during different periods varied being 0.49 between preweaning gain and gain from weaning to 42 days, -0.23 between preweaning gain and gain from 42 to 56 days, and 0.19 between gain from weaning to 42 days and gain from 42 to 56 days. Phenotypic correlations between these gains were consistently near zero, and they were negative genetic relationship between protein anabolism and lactation, and a negative genetic relationship between fattening and lactation.

Maternal correlations between the various weights were large and positive. However, maternal correlations between preweaning gain and postweaning gains were negative. Thus, young mice that nursed dams that apparently milked well made reduced gains following weaning.

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APPENDIX

The expectation of the mean cross product between the prenatal totals for the i th trait and the postnatal totals for the j th trait is derived in terms of the row and column totals for a single cross-fostering group (Table 1). Thus the expectation of the following is desired:

$$\frac{1}{n-1} E \left[\frac{\sum_{k=1} (Y_{k..})_i (Y_{.l.})_j - (Y_{...})_i (Y_{...})_j / n \right]$$

$$= \frac{1}{n-1} E \left[\frac{\sum_{k=1} (Y_{k..})_i (Y_{.l.})_j \right] - \frac{1}{n(n-1)} E \left[(Y_{...})_i (Y_{...})_j \right]$$

where n equals the number of litters per cross-fostering group.

Model: $Y_{klm} = \mu + a_k + p_l + (ap)_{kl} + e_{klm}$

Where: Y_{klm} is an observation on the m th mouse of the l th postnatal and the k th prenatal litter

μ is the general mean for a cross-fostering group;

a_k is the prenatal effect of the k th litter; $k = 1, 2, 3$;

p_l is the effect of the l th postnatal dam, $l = 1, 2, 3$;

$(ap)_{kl}$ is the result of interaction between the prenatal and postnatal effects;

e_{klm} is a residual effect due to unidentified influences, $m = 1, 2$.

The different traits or measurements are denoted by subscripts i and j .

Expectations:

$$E(a_k) = E(p_l) = E(ap)_{kl} = E(e_{klm}) = 0$$

$$E[(\mu)_i (\mu)_j] = \mu^2 \text{ for } i = j; = (\mu_i \mu_j) \text{ for } i \neq j.$$

$$E[(a_k)_i (a_{k'})_j] = \sigma_a^2 \text{ for } i = j, k = k'; = \sigma_{a_i a_j} \text{ for } i \neq j, k = k'; = \text{zero otherwise.}$$

$$E[(p_l)_i (p_{l'})_j] = \sigma_p^2 \text{ for } i = j, l = l'; = \sigma_{p_i p_j} \text{ for } i \neq j, l = l'; = \text{zero otherwise.}$$

$$E[(ap)_{kl}]_i [(ap)_{k'l'}]_j = \sigma_{ap}^2 \text{ for } i = j, k = k', l = l'$$

$$= \sigma_{(ap)_i (ap)_j} \text{ for } i = j, k = k', l = l'$$

$$= \text{zero otherwise.}$$

$$E[(e_{klm})_i (e_{k'l'm'})_j] = \sigma_e^2 \text{ for } i=j, k=k', l=l', m=m' \\ = \sigma_{e_i e_j} \text{ for } i \neq j, k=k', l=l', m=m' \\ = \text{zero otherwise}$$

$$E[(a_k)_i (p_l)_j] = \sigma_{ap} \text{ for } i=j, k=l; = \text{zero otherwise.}$$

Consider the first portion of the expectation:

$$E[\sum_{k=1} (Y_{k..})_i (Y_{.1.})_j] = E[(Y_{1..})_i (Y_{.1.})_j] + E[(Y_{2..})_i (Y_{.2.})_j] \\ + E[(Y_{3..})_i (Y_{.3.})_j] \\ Y_{1..} = Y_{111} + Y_{112} + Y_{121} + Y_{122} + Y_{131} + Y_{132} \\ Y_{.1.} = Y_{...} + Y_{112} + Y_{211} + Y_{212} + Y_{311} + Y_{312} \\ E[(Y_{1..})_i (Y_{.1.})_j] = E[(Y_{111})_i (Y_{111})_j] + E[(Y_{111})_i (Y_{112})_j] + \dots + \\ E[(Y_{132})_i (Y_{312})_j]$$

Thus $E[(Y_{1..})_i (Y_{.1.})_j]$ involves 36 terms which can be evaluated for $i=j$ as follows:

$$E(Y_{111})^2 = E[\mu + a_1 + p_1 + (ap)_{11} + e_{111}]^2 \\ = \mu^2 + \sigma_a^2 + \sigma_p^2 + \sigma_{ap}^2 + \sigma_e^2 + 2\sigma_{ap} \\ E(Y_{111})(Y_{112}) = E[(\mu + a_1 + p_1 + (ap)_{11} + e_{111})(\mu + a_1 + p_1 + (ap)_{11} + e_{112})] \\ = \mu^2 + \sigma_a^2 + \sigma_p^2 + \sigma_{ap}^2 + 2\sigma_{ap} \\ E(Y_{111})(Y_{211}) = E[(\mu + a_1 + p_1 + (ap)_{11} + e_{111})(\mu + a_2 + p_1 + (ap)_{21} + e_{211})] \\ = \mu^2 + \sigma_p^2 + \sigma_{ap}$$

Similarly,

$$E(Y_{111})(Y_{212}) = \mu^2 + \sigma_p^2 + \sigma_{ap} \\ E(Y_{111})(Y_{311}) = \mu^2 + \sigma_p^2 + \sigma_{ap} \\ E(Y_{111})(Y_{312}) = \mu^2 + \sigma_p^2 + \sigma_{ap}$$

and

$$E(Y_{111})(Y_{.1.}) = 6\mu + 2\sigma_a^2 + 6\sigma_p^2 + 2\sigma_{ap}^2 + 2\sigma_e^2 + 8\sigma_{ap}.$$

Following the approach illustrated above the other expectations can be shown to be:

$$E(Y_{112})(Y_{.1.}) = 6\mu^2 + 2\sigma_a^2 + 6\sigma_p^2 + 2\sigma_{ap}^2 + 2\sigma_e^2 + 8\sigma_{ap} \\ E(Y_{121})(Y_{.1.}) = 6\mu^2 + 2\sigma_a^2 + 8\sigma_{ap} \\ E(Y_{122})(Y_{.1.}) = 6\mu^2 + 2\sigma_a^2 + 8\sigma_{ap} \\ E(Y_{131})(Y_{.1.}) = 6\mu^2 + 2\sigma_a^2 + 8\sigma_{ap} \\ E(Y_{132})(Y_{.1.}) = 6\mu^2 + 2\sigma_a^2 + 8\sigma_{ap}$$

Summing these six expectations gives:

$$E[(Y_{1..})_i (Y_{.1.})_j] = 36\mu^2 + 12\sigma_a^2 + 12\sigma_p^2 + 4\sigma_{ap}^2 + 2\sigma_e^2 + 48\sigma_{ap}, \text{ where } i=j. \\ = 36\mu_i \mu_j + 12\sigma_{a_i a_j} + 12\sigma_{p_i p_j} + 4\sigma_{(ap)_i (ap)_j} + 2\sigma_{e_i e_j} \\ + 48\sigma_{a_i p_j} \text{ (where } i \neq j \text{).}$$

Likewise, it can be shown that

$$E[(Y_{1..})_i (Y_{.1.})_j] = E[(Y_{2..})_i (Y_{.2.})_j] = E[(Y_{3..})_i (Y_{.3.})_j]; \\ \text{hence, } 1/n-1 E[\sum_{k=1} (Y_{k..})_i (Y_{.k.})_j]$$

$$\begin{aligned}
&= \frac{1}{2} [3(36\mu^2 + 12\sigma_a^2 + 12\sigma_p^2 + 4\sigma_{(ap)}^2 + 2\sigma_e^2 + 48\sigma_{ap})] \\
&= 54\mu^2 + 18\sigma_a^2 + 18\sigma_p^2 + 6\sigma_{(ap)}^2 + 2\sigma_e^2 + 72\sigma_{ap}, \text{ where } i = j \\
&= 54\mu_i\mu_j + 18\sigma_{a_i a_j} + 18\sigma_{p_i p_j} + 6\sigma_{(ap)_i (ap)_j} + 3\sigma_{e_i e_j} + 72\sigma_{a_i p_j}, \\
&\quad \text{where } i \neq j.
\end{aligned}$$

Now the second expectation of the above term must be considered.

$$\begin{aligned}
E[(Y_{...})_i (Y_{...})_j] &= E[(Y_{111} + Y_{112} + \dots + Y_{332})_i \\
&\quad (Y_{111} + Y_{112} + \dots + Y_{332})_j] \\
&= E[(Y_{111})_i (Y_{111})_j] + E[(Y_{111})_i (Y_{112})_j] + \dots \\
&\quad + E[(Y_{332})_i (Y_{332})_j].
\end{aligned}$$

Taken individually 324 expectations are involved in the above breakdown. While they will not be written out here, it can be verified that the sum of these expectations is as follows:

$$\begin{aligned}
E[(Y_{...})_i (Y_{...})_j] &= 324\mu^2 + 108\sigma_a^2 + 108\sigma_p^2 + 36\sigma_{(pa)}^2 + 18\sigma_e^2 + 216\sigma_{ap}, \\
&\quad \text{where } i = j. \\
&= 324\mu_i\mu_j + 108\sigma_{a_i a_j} + 108\sigma_{p_i p_j} + 36\sigma_{(ap)_i (ap)_j} + 18\sigma_{e_i e_j} \\
&\quad + 216\sigma_{a_i p_j}, \text{ where } i \neq j.
\end{aligned}$$

Then

$$\begin{aligned}
\frac{1}{n(n-1)} E[(Y_{...})_i (Y_{...})_j] &= 54\mu^2 + 18\sigma_a^2 + 18\sigma_p^2 + 6\sigma_{(ap)}^2 + 3\sigma_e^2 + 36\sigma_{ap}, \\
&\quad \text{where } i = j. \\
&= 54\mu_i\mu_j + 18\sigma_{a_i a_j} + 18\sigma_{p_i p_j} + 6\sigma_{(ap)_i (ap)_j} + 3\sigma_{e_i e_j} \\
&\quad + 36\sigma_{a_i p_j}, \text{ where } i \neq j.
\end{aligned}$$

Combining the two expectations

$$\begin{aligned}
&\frac{1}{n-1} E[\sum_{k=1} (Y_{k..})_i (Y_{..l})_j] - \frac{1}{n(n-1)} E[(Y_{...})_i (Y_{...})_j] \\
&= 36\sigma_{ap} \text{ where } i = j. \\
&= 36\sigma_{a_i p_j} \text{ where } i \neq j.
\end{aligned}$$