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Sexual Dimorphism and Direct and Maternal Genetic Effects on Body Weight in Mice[1]

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Summary. Genetic and phenotypic parameters for three-, six- and eight-week body weight and for weight gain between three and six weeks of age were estimated from data collected over 14 generations in a randombred control population. Genetic parameters were also estimated for sexual dimorphism in body weight and gain. Heritability estimates were substantial for body weight at all ages and for body weight gain. Additive maternal variances were also large. Estimates of the covariance between direct and maternal genetic effects were negative and substantial for three- and six-week weights and gain. Also the covariance between maternal effects on weaning weight and direct genetic effects on six- and eight-week weights were negative. These results indicate a consistent antagonism between maternal and direct genetic effects in this population.

The analysis of sexual dimorphism yielded estimates of 0.87 ± .09 and 0.71 ± .14 for the correlation between additive direct effects on males and females for six-week weight and body weight gain respectively. Corresponding heritability estimates were 0.07 ± .09 and 0.11 ± .09. Heritability estimates for sexual dimorphism in three- and eight-week weights were negative.

Introduction

Sexual dimorphism in animals is common. However, evidence suggesting a genetic basis for this dimorphism is limited. Further, the combined effects of a genotype x sex interaction and maternal effects on the estimates of genetic parameters have not been considered. The possible effects of such an interaction on selection response also need to be explored.

Robertson (1959) showed that the existence of a genotype x environment interaction required that the genetic correlation between the same trait measured in two environments be less than unity. Eisen and Legates (1966) extended this concept to the genotype x sex interaction and developed an expression for the heritability of sexual dimorphism as measured by the difference between male and female family means.

This paper examines the effects of a genetic correlation of less than unity on the covariances between relatives and the implications this has for the estimation of causal components of variance. An expression is derived for the heritability of sexual dimorphism where selection is based on individual phenotypes. Estimates of the genetic correlation between the sexes for body weight and gain in a randombred control stock of mice are given together with estimates of genetic and phenotypic parameters for these traits within sexes. These genetic parameter estimates include additive direct and maternal variances and additive direct-maternal covariances.

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Theory

In the following development it was assumed that genetic control of the trait under investigation was completely additive and autosomal. Random mating and the absence of linkage and inbreeding were assumed also. The maternal effects model given by Willham (1963) was generalized to include a genetic correlation of less than unity between the sexes and unequal sex variances. Thus, the model used to describe the phenotype of an individual of the i^{th} sex ($i = 1$ = male; $i = 2$ = female) was

$$P_i = \mu_i + A0_i + A^d m_i + E_i \,, \qquad (1)$$

where

μ_i = population mean for the i^{th} sex,
$A0_i$ = genotypic value for direct (transmitted) effects,
$A^d m_i$ = genotypic value of the dam of the individual for maternal effects,
E_i = environmental effect.

In a polytocous species, such as the mouse, the environmental term, E_i, can be partitioned into a random environmental component, e_i, peculiar to each individual and a portion, C_i, common to litter-mates of the i^{th} sex. The following variances (heritabilities) and covariances (correlations) are defined:

$\sigma^2_{A0_i}(h^2_{0i})$ = additive genetic variance (heritability) for direct effects in the i^{th} sex,
$\sigma^2_{A m_i}(h^2_{mi})$ = additive genetic variance (heritability) for maternal effects in the i^{th} sex,
$\sigma^2_{E_i} = \sigma^2_{C_i} + \sigma^2_{e_i}$ = environmental variance in the i^{th} sex,

$\sigma_{A\,0_i\,A\,m_j}(r_{0_i\,m_j})$ = covariance (correlation) between additive direct effects in the i^{th} sex and additive maternal effects in the j^{th} sex,

$\sigma_{A\,0_1\,A\,0_2}(r_{0_{12}})$ = covariance (correlation) between additive direct effects in male and female environments,

$\sigma_{A\,m_1\,A\,m_2}(r_{m_{12}})$ = covariance (correlation) between additive maternal effects in male and female environments,

$\sigma^2_{P_i} = \sigma^2_{A\,0_i} + \sigma^2_{A\,m_i} + \sigma^2_{A\,0_i\,A\,m_i} + \sigma^2_{C_i} + \sigma^2_{e_i}$ = phenotypic variance of the i^{th} sex.

Covariance commonly used to estimate causal components of variance for a quantitative trait are listed in Table 1, assuming either the absence or presence of genotype × sex interaction. Linear functions of these covariances provide estimates of the causal components in the absence of genotype × sex interaction.

Applying these functions in the presence of genotype × sex interaction yields unbiased estimates of $\sigma^2_{A\,0_i}$ and $\sigma^2_{e_i}$. Also, there is an unbiased estimate of $\sigma_{A\,0_1\,A\,m_1}$. In contrast, the additive maternal variance for males, $\sigma^2_{A\,m_1}$, has expectation

$$\sigma_{A\,m_1} r_{m_{12}} h_{m_2} \sigma_{p_1} - \sigma_{A\,0_1}(\sigma_{A\,0_1} - r_{0_{12}} h_{0_2} \sigma_{p_1}) + \\ + \sigma_{p_1}(2\, r_{0_2 m_1} h_{0_2} \sigma_{A\,m_1} + 1/2\, r_{0_1 m_2} h_{m_2} \sigma_{A\,0_1} - \\ - 5/2\, r_{0_1 m_1} h_{0_1} \sigma_{A\,m_1}) . \quad (2)$$

In a similar fashion the bias in estimates of $\sigma^2_{C_i}$ and $\sigma_{A\,0_2\,A\,m_2}$ may be determined. The expected value of $\sigma^2_{A\,m_1}$ indicates several sources of possible bias. Even when $r_{0_{12}} = 1$ the absence of bias involving $\sigma_{A\,0_1}$ requires that $h_{0_2} = h_{0_1}$. The sign of the coefficient of $\sigma_{A\,0_1}$ depends on the relative magnitude of $r_{0_{12}}$ and the ratio h_{0_1}/h_{0_2}. In order that the coefficient of $\sigma_{A\,m_1}$ be equal to $\sigma_{A\,m_1}$ it is necessary that $r_{m_{12}} = h_{m_1}/h_{m_2}$. These potential biases exemplify the need for caution when using covariances between relatives of opposite sex in arriving at estimates of causal components of variance.

The genotype × sex interaction component of variance was derived by Robertson (1959) as

$$\sigma^2_{gs} = 1/2\,(\sigma_{A\,0_1} - \sigma_{A\,0_2})^2 + \sigma_{A\,0_1}\sigma_{A\,0_2}(1 - r_{0_{12}}) . \quad (3)$$

When the model includes maternal effects, the interaction component becomes

$$\sigma^2_{gs} = 1/2\,(\sigma_{A\,0_1} - \sigma_{A\,0_2})^2 + \sigma_{A\,0_1}\sigma_{A\,0_2}(1 - r_{0_{12}}) + \\ + 1/2\,(\sigma_{A\,m_1} - \sigma_{A\,m_2})^2 + \sigma_{A\,m_1}\sigma_{A\,m_2}(1 - r_{m_{12}}) + \\ + 1/2\,(\sigma_{A\,0_1\,A\,m_1} + \sigma_{A\,0_2\,A\,m_2} - \sigma_{A\,0_1\,A\,m_2} - \sigma_{A\,0_2\,A\,m_1}) = \\ = \sigma^2_{g\,s_0} + \sigma^2_{g\,s_m} + 2\,\sigma_{g\,s_0\,g\,s_m} , \quad (4)$$

where $\sigma^2_{g\,s_0}$ = genotype × sex interaction component of variance due to direct effects, $\sigma^2_{g\,s_m}$ = genotype × sex interaction component of variance due to maternal effects and $\sigma_{g\,s_0\,g\,s_m}$ = covariance of direct and maternal genotype × sex interaction effects.

Eisen and Legates (1966) have pointed out that only the contribution $\sigma_{A\,0_1\,A\,0_2}(1 - r_{0_{12}})$ is important from the selection standpoint when maternal effects are not included. In the present case the genotype × sex interaction component of variance may be viewed in the same way if the breeding value is defined as $G_i = A\,o_i + A^d_{m_i}$. Then an expression analogous to (3) is

$$\sigma^2_{gs} = 1/2\,(\sigma_{G_1} - \sigma_{G_2})^2 + \sigma_{G_1}\sigma_{G_2}(1 - r_{G_1 G_2}) . \quad (5)$$

Thus, from a selection standpoint $(1 - r_{G_1 G_2})$ must be greater than zero before any net advance can be achieved in selection for sexual dimorphism.

Returning to a model which excludes maternal effects, Eisen and Legates (1966) have defined the heritability of sexual dimorphism as

$$h^2_{0\,(1-2)} = \sigma^2_{A\,0(1-2)}/\sigma^2_{P\,(1-2)} , \quad (6)$$

where

$$\sigma_{A\,0(1-2)} = (\sigma_{A\,0_1} - \sigma_{A\,0_2})^2 + \sigma_{A\,0_1}\sigma_{A\,0_2}(1 - r_{0_{12}}) ,$$

$$\sigma^2_{P(1-2)} = (\sigma_{P_1} - \sigma_{P_2})^2 + \sigma_{P_1}\sigma_{P_2}(1 - r_{P_{12}}) ,$$

and $r_{p_{12}}$ = the phenotypic correlation between the same individual measured in male and female environments. The heritability adjusted for scale effects is

$$h^2_{0\,(1-2)'} = \frac{(1 - r_{0_{12}})}{1 - r_{P_{12}}} h_{0_1} h_{0_2} , \quad (7)$$

Table 1. *Expectations of a selected set of covariances between relatives in the presence and absence of genotype × sex interaction*

Covariance	Genotype-sex Interaction	
	Absent	Present[a]
Paternal half-sibs	$1/4\,\sigma^2_{A\,0}$	$1/4\,\sigma^2_{A\,0_i}$
Full sibs within sire	$1/4\,\sigma^2_{A\,0} + \sigma^2_{A\,m} + \sigma_{A\,0\,A\,m} + \sigma^2_C$	$1/4\,\sigma^2_{A\,0_i} + \sigma^2_{A\,m_i} + \sigma^2_{A\,0_i\,A\,m_i} + \sigma^2_{C_i}$
Individuals within full-sib family	$1/2\,\sigma^2_{A\,0} + \sigma^2_e$	$1/2\,\sigma^2_{A\,0_i} + \sigma^2_{e_i}$
Offspring-sire	$1/2\,\sigma^2_{A\,0} + 1/4\,\sigma_{A\,0\,A\,m}$	$(1/2\,\sigma_{A\,0_i\,A\,0_1} + 1/4\,\sigma_{A\,0_i\,A\,m_1})\sigma_{P\,i}/\sigma_{P1}$
Offspring-dam	$1/2\,\sigma^2_{A0} + 1/2\,\sigma^2_{A\,m} + 5/4\,\sigma_{A\,0\,A\,m}$	$(1/2\,\sigma_{A\,0_i\,A\,0_2} + 1/2\,\sigma_{A\,m_i\,A\,m_2} + \\ + \sigma_{A\,m_i\,A\,0_2} + 1/4\,\sigma_{A\,0_i\,A\,m_2})\,\sigma_{P\,i}/\sigma_{P2}$

[a] For individuals of the i^{th} sex, where $i = 1$ for males and $i = 2$ for females.

where the prime denotes a parameter free of scale effects. This heritability (7) is appropriate when each selection unit (i.e., individual or family) is measured in both male and female environments. However, for individual selection the sex dimorphism cannot be measured and selection must be based on individual phenotype in a particular sex. In this case the scale-free response may be derived as follows.

Consider selection in a population for increased male and decreased female scores. If male and female responses are expressed in terms of their respective phenotypic standard deviations, the expected response of males is

$$\Delta G_{0_1} = 1/2\ (\bar{i}_1\, h^2_{0_1} + \bar{i}_2\, r_{0_{12}}\, h_{0_1}\, h_{0_2})\ , \qquad (8)$$

while that for females is

$$\Delta G_{0_2} = 1/2\ (\bar{i}_2\, h^2_{0_2} + \bar{i}_1\, r_{0_{12}}\, h_{0_1}\, h_{0_2}) \qquad (9)$$

where $\bar{i}_i$ = standardized selection differential in the i^{th} sex. The sexual dimorphism response is

$$\Delta G_D = \Delta G_{0_1} - \Delta G_{0_2} = 1/2\ (\bar{i}_1\, h^2_{0_1} - \bar{i}_2\, h^2_{0_2}) + \\ + 1/2\, r_{0_{12}}\, h_{0_1}\, h_{0_2}\, (-\bar{i}_1 + \bar{i}_2)\ . \qquad (10)$$

If $\bar{i}_1 = -\bar{i}_2 = \bar{i}$, then

$$\Delta G_D = 1/2\, \bar{i}\, (h_{0_1} - h_{0_2})^2 + \bar{i}\, h_{0_1}\, h_{0_2}\, (1 - r_{0_{12}})\ . \qquad (11)$$

Equation (11) shows that expressing response in units of the phenotypic standard deviation for each sex does not eliminate all scale effects for the response unless $h^2_{0_1} = h^2_{0_2}$. The scale-free response in standard deviation units is given by

$$\Delta G_{D'} = \Delta G_D - 1/2\, \bar{i}\, (h_{0_1} - h_{0_2})^2\ . \qquad (12)$$

The most appropriate scale in which to express this response is that given by the scale-free phenotypic standard deviation of the difference between the sexes; i.e.,

$$\sigma_{p(1-2)'} = [(1 - r_{p_{12}})\, \sigma_{p_1}\, \sigma_{p_2}]^{1/2}\ .$$

Therefore the response becomes, from (12),

$$\bar{i}\, h_{0_1}\, h_{0_2}\, (1 - r_{0_{12}})\, \sigma_{p(1-2)'} = \bar{i}\, h^2_{0(1-2)'}\, (1 - r_{p_{12}})\, \sigma_{p(1-2)'} \qquad (13)$$

and the heritability is

$$h^2_{0(1-2)'}\, (1 - r_{p_{12}})\ . \qquad (14)$$

This expression for the heritability differs from that of Eisen and Legates (1966) by the factor $1 - r_{p_{12}}$ because they treated the case where the units of selection had measures in both environments and hence the sex dimorphism of each selection unit was observed.

The expected response to selection for sexual dimorphism for traits subject to maternal effects will be considered elsewhere (Eisen and Hanrahan, 1972).

Materials and Methods

Source of data and laboratory procedures: Data from generations 5 through 18 of a randombred ICR stock of mice were used in this study. Each generation at least 24 males were each mated to two females. Parents were chosen so that each dam in the previous generation was represented by one daughter and each sire was represented by one son. This procedure minimized the sampling variance of gene frequency and made the effective size as large as was operationally possible. Under this sampling procedure the level of inbreeding of generation 18 mice is expected to be less than 10 percent.

Females were between eight and ten weeks of age when joined with males. Mating was allowed to continue for 16 days at which time females were placed in separate cages. Litters were standardized to eight young at day five postpartum, with equal numbers of males and females, where possible. Progeny were weaned at 21 days postpartum. During lactation females were fed a high-energy diet (Emory Morse Company) *ad libitum*, while weaned mice were fed Purina Laboratory Chows *ad libitum*.

Individual body weight was recorded to the nearest 0.1 g at three, six and eight weeks of age. In addition, postweaning gain was defined as six-week minus three-week body weight. A total of 2558 male and 2562 female progeny with complete records was available. This represented the offspring of 355 sires and 661 dams.

Sterility, measured as the percentage of all females placed with males for 16 days who failed to produce a litter, was 9 percent. The average litter size at birth was 12.39 $\pm$ 0.08 when only fertile females were included.

Statistical procedures: Observational components of variance were estimated from the hierarchical analysis of variance based on the model

$$Y_{ijkl} = \mu + t_i + s_{j(i)} + d_{k(ij)} + w_{ijkl}\ ,$$

where Y_{ijkl} is an observation on the l^{th} mouse in the litter of the k^{th} dam mated to the j^{th} sire in the i^{th} generation; $w_{ijkl}, d_{k(ij)}$ and $s_{j(i)}$ are assumed normally and independently distributed with zero means and variances σ^2_w, σ^2_d and σ^2_s, respectively. Covariances between different traits were determined by nested covariance analysis of the same form as the analysis of variance. Heritability was estimated as 4 σ^2_s/σ^2_p where $\sigma^2_p = \sigma^2_s + \sigma^2_d + \sigma^2_w$. The genetic correlation between two traits was estimated as $\sigma_{sxy}/\sigma_{sx}\, \sigma_{sy}$ where σ_{sxy} is the sire component of covariance for traits x and y measured on each individual and σ^2_{sx} and σ^2_{sy} are the corresponding variance components. All analyses except those on sex differences were carried out separately for males and females.

Offspring-parent covariances were estimated from analysis of covariance on individual progeny records. Consequently, each sire or dam record was repeated with each one of his or her progeny. The resulting covariance estimates are biased (Kempthorne and Tandon, 1953) except when all parents have an equal number of progeny. Since litters were standardized to equal numbers of each sex, there is little variation in the number of progeny records, and this source of bias was ignored. The data were further classified as to maternal and paternal grandparents which allowed the estimation of additional covariances between relatives.

Genetic and phenotypic covariances between males and females were estimated using paired male and female records as the observational unit. Pairing of male and female records within full-sib families was made at random until either male or female records were exhausted. Surplus male or female records, within a litter, were omitted. Thus, 2260 male-female pairs were available for analysis. The resulting constructed data records were treated in the same way as individual records.

Results and Discussion

Time trends: Since the data were to be pooled over time, it was necessary to establish if any time trends were evident in the traits measured. Generation means of body weight traits for each sex were analyzed by weighted regression on generation number. No significant change over time was detected for any trait with the exception of female six-week body weight, which had a regression of $0.077 \pm .036$ ($P < .05$). The logarithms of phenotypic variance and within full sib family variance estimates for each sex showed no linear time trends. The results indicated that the ICR population was in a relatively stable state between generations five and 18. Therefore, all data were pooled within generations to estimate observational variance components.

Individual body weight: Means, phenotypic variances and coefficients of variation for three-, six- and eight-week body weights and postweaning gain are given in Table 2. Most of the difference in six- and eight-week weight was due to the more rapid postweaning gain of males. Phenotypic variances for males were significantly greater than those for females for all traits. Examination of the coefficients of variation showed that these differences were due to scaling effects except in the case of postweaning gain where females exhibited a substantially greater variability.

Heritabilities and genetic and phenotypic correlations are given in Table 3. Although not significant, females had larger heritability estimates than males for all traits. Heritability estimates for three-week body weight were substantial in contrast to the estimates of zero for this parameter reported by Eisen and Legates (1966) based on three earlier generations of the same population.

The genetic correlation between three-week weight and postweaning gain was approximately zero. Thus, in this population, genes determining growth up to weaning were operationally independent of genes for postweaning growth. This result contrasts with the value of 0.27 reported by Young and Legates (1965). Eisen, Legates and Robison (1970) reported a value of 0.41 for the genetic correlation between body weight at 12 days of age and gain between 12 and 42 days. Their estimate was undoubtedly inflated by

Table 2. *Means, phenotypic variances and coefficients of variation of male and female body weights traits in ICR stock mice*[a]

	Trait	Males	Females	
				t-test
Means (g)	3-wk wt	13.92 ± .04	13.51 ± .04	7.32**
	6-wk wt	31.81 ± .96	26.53 ± .05	57.07**
	8-wk wt	35.00 ± .06	28.32 ± .05	82.47**
	(6−3)-wk wt	17.89 ± .05	13.02 ± .05	67.64**
				F-test
Phenotypic variances (g^2)	3-wk wt	3.41 ± .15	2.83 ± .12	1.20**
	6-wk wt	7.61 ± .26	5.43 ± .18	1.40**
	8-wk wt	9.56 ± .33	6.66 ± .22	1.43**
	(6−3)-wk wt	6.22 ± .21	4.96 ± .17	1.25**
Coefficients of variation (%)	3-wk wt	13.26 ± .19	12.44 ± .17	1.14*
	6-wk wt	8.67 ± .12	8.78 ± .12	0.97
	8-wk wt	8.83 ± .12	9.11 ± .13	0.94
	(6−3)-wk wt	13.94 ± .19	17.11 ± .24	0.66**

[a] Based on 2568 males and 2562 females
* $P < .05$, ** $P < .01$

Table 3. *Genetic and phenotypic correlation coefficients and heritability estimates*[a]

	3-wk wt	6-wk wt	8-wk wt	(6-3) wk wt
3-wk wt	0.344 ± .177 0.449 ± .171	0.620 ± .091 0.569 ± .082	0.402 ± .108 0.478 ± .091	−0.013 ± .148 −0.129 ± .119
6-wk wt	0.471 0.420	0.388 ± .116 0.511 ± .113	0.966 ± .025 1.003 ± .021	0.776 ± .058 0.742 ± .054
8-wk wt	0.380 0.335	0.796 0.763	0.440 ± .118 0.551 ± .109	0.909 ± .059 0.819 ± .059
(6−3)-wk wt	−0.219 −0.316	0.757 0.729	0.599 0.545	0.293 ± .119 0.384 ± .123

[a] Upper entry of each pair is for males while lower entry is for females. Diagonal entries are heritabilities with standard errors (Osborne and Patterson, 1952). Upper triangular matrix contains genetic correlations with standard error (Tallis, 1959). Lower triangular matrix contains phenotypic correlation coefficients.

Table 4. *Total maternal impact, measured as the ratio* $(\hat{\sigma}_d^2 - \hat{\sigma}_s^2)/\hat{\sigma}_p^2$, *on body weights and gain*

Trait	Male	Female
3-wk wt	0.506 ± 0.028	0.428 ± 0.028
6-wk wt	0.159 ± 0.029	0.092 ± 0.028
8-wk wt	0.150 ± 0.029	0.067 ± 0.027
(6-3) wk wt	0.210 ± 0.031	0.193 ± 0.031

a high genetic correlation between 12-day weight and gain from 12 days to weaning. A further contrast between studies was the negative phenotypic correlation between weaning weight and postweaning gain in the present results and the positive value of 0.12 reported by Young and Legates (1965). The stock used in the present study was different from that used by Young and Legates (1965) and Eisen *et al.* (1970).

Considering the genetic parameter estimates, a substantial response to selection would be expected for any of the traits measured, together with positive correlated responses in the other traits. An exception is the case of weaning weight as a correlated response to selection for postweaning gain and vice versa. However, this interpretation neglects the role of maternal effects in the determination of these traits.

Maternal effects on body weight: Maternal influences are an environmental effect with respect to the individual offspring, but when considered as a trait of the dam they can be partitioned into genetic and environmental components. The total contribution of maternal effects to the phenotypic variance is defined as $(\sigma_{Am}^2 + \sigma_{A0Am} + \sigma_C^2)/\sigma_p^2$. This proportion was estimated by $(\hat{\sigma}_d^2 - \hat{\sigma}_s^2)/\hat{\sigma}_p^2$ (Table 4). As expected, the total impact of maternal performance was greatest at weaning and declined up to eight weeks. El Oksh, Sutherland and Williams (1967) reported a considerably higher value (40%) for the maternal impact on six-week weight and a value (41%) for weaning weight which was somewhat lower than the present values. Young, Legates and Farthing (1965) reported higher values for all weights but a considerably lower value (4%) for postweaning gain.

Interrelationships between direct and maternal genetic effects: The regression of male offspring on sire (Table 5) may be compared with the correlation between paternal half-sibs to assess the genetic covariance between direct and maternal effects on body weight in males. Regressions involving opposite sexes have been converted to the sex of offspring scale. Comparing the heritability estimate for male postweaning gain (0.293 ± .119) with twice the regression of male offspring on sire, 0.246 ± .060, yields an estimate of −0.047 for σ_{A0Am}/σ_p^2 or $\sigma_{A0Am} = -0.047 \times 6.22 = -0.292$. This shows that σ_{A0Am} made a small negative contribution to the variance of postweaning gain. In contrast the regression of offspring on sire for three-week weight was essentially zero which, when compared with the paternal half-sib estimate of heritability (0.344 ± .177), indicated a substantial negative correlation between direct and maternal genetic effects on weaning weight. Smaller regression estimates of heritability for six- and eight-week weight relative to the paternal half-sib estimates indicate that the direct-maternal genetic covariance was negative for these traits also, but of less relative magnitude.

Comparisons between regressions of offspring on sire and dam are difficult to interpret in view of the possible complexity of the components involved in the difference between these regressions. For example, in the simplified situation where there is no genotype × sex interaction and heritabilities are equal, the difference between the regression on dam and on sire has expectation equal to $^1/_4 h_m^2 + r_{0m} h_0 h_m$ (Table 1).

There is considerable practical interest in the genetic correlation between postnatal maternal performance and postweaning growth (Young and Legates, 1965). In the present data the covariance between maternal genetic effects on weaning weights and direct genetic effects on postweaning weights and gain may be estimated for males from male offspring-sire covariances. The covariance between sire weaning weight and six-week weight of his male progeny, for example, has expectation equal to $^1/_2 \sigma_{A0_3 A0_6} + {}^1/_4 \sigma_{Am_3 A0_6}$ where the additional subscripts "3" and "6" refer to three- and six-week weights, respectively. The paternal half-sib covariance between these traits has an expectation of $^1/_4 \sigma_{A0_3 A0_6}$ so that

Table 5. *Regression of offspring on sire and offspring on dam for individual body weights and postweaning gain*[a]

Trait	Regression on			
	Sire		Dam	
	Male	Female	Male	Female
3-wk wt	−0.004 ± .034	0.019 ± .033	0.117 ± .037	0.103 ± .034
6-wk wt	0.114 ± .030	0.127 ± .028	0.201 ± .031	0.186 ± .029
8-wk wt	0.169 ± .030	0.152 ± .027	0.290 ± .033	0.236 ± .025
(6−3)-wk wt	0.123 ± .030	0.079 ± .029	0.110 ± .031	0.120 ± .030

[a] Regressions involving opposite sexes have been converted to the sex of offspring scale.

Table 6. *Covariances and correlations between maternal genetic effects on weaning weight and direct genetic effects on six- and eight-week weights and postweaning gain*

Trait	Sex	Covariance	Correlation
6-wk wt	*M*	−2.472	−1.551
	F	−0.692	−0.407
8-wk wt	*M*	−0.808	−0.455
	F	−1.720	−0.950
(6-3)-wk wt	*M*	−0.068	−0.056
	F	0.032	0.023

$\sigma_{A\,m_{12}A\,0_{18}}$ may be estimated without bias. An estimate of $\sigma_{A\,m_{12}A\,0_{28}}$ may be obtained by combining information from the covariance of three- and six-weak body weight obtained from female offspring-sire regression with the sire component of covariance between males and females for these traits. Table 6 shows the estimated covariances and corresponding correlations. The results show that there was a negative association for six- and eight-week weights with post-weaning gain showing a correlation of essentially zero. Young and Legates (1965) reported positive covariances for all three traits using estimates from a crossfostering study.

Simultaneous estimation of causal components: Several covariances between relatives are necessary for the simultaneous estimation of the causal components of variance. Additional covariances to those given in Table 1 were obtained from the maternal grandsire component of variance for each sex and the covariances of male offspring on maternal and paternal grandsires and female offspring on maternal and paternal granddams. In view of the possible influence of genotype × sex interactions, only covariances between relatives of the same sex were employed. Thus, a total of seven equations were used to obtain a least squares solution for the five causal variance components in each sex (Table 7).

The large negative estimates of σ_C^2 for male six-week weight coupled with the large positive estimates of σ_{Am}^2 may be a consequence of the high correlation (−0.9) between these estimates. This indicates the inadequacy of the design for the separation of the causal components involved in maternal effects (Eisen, 1967). The model was reduced by assuming $\sigma_C^2 = 0$ and the remaining components were reestimated (Table 8).

Additive maternal effects contributed a considerable portion of the variability in body weights and gain. The results again indicated a negative covariance between direct and maternal additive genetic effects. Although this negative covariance would tend to reduce selection response somewhat, genetic gain would still be expected.

Genotype × sex interaction: The heritability of the sex difference and the genetic correlations between the sexes for body weight and postweaning gain are presented in Table 9. The sex dimorphism for postweaning gain and six-week body weight had positive heritabilities while those for three- and eight-week weight were negative. However, none of the heritability estimates were significantly different from zero. The heritabilities, adjusted for scale effects, were not altered appreciably. The present results suggest that response to selection for dimorphism in body weight or gain would be relatively low.

The parameter estimates in Table 9 differ somewhat from those of Eisen and Legates (1966) who used data from the first three generations of the same mouse stock, and obtained positive estimates of heritability of sex dimorphism for all four traits. The two sets of data are consistent in that postweaning gain exhibited the largest heritability in both. In terms of the genetic correlation between the sexes for postweaning gain the present value of $0.713 \pm .143$ is comparable with the value of $0.680 \pm .195$ reported by Eisen and Legates (1966).

Rahnefeld *et al.* (1963) suggested that a genetic correlation of less than unity between the sexes was the cause of a discrepancy between predicted and

Table 7. *Estimates of causal components of variance from the least squares solution*

Trait	Sex	σ_{A0}^2	σ_{Am}^2	$\sigma_{A0\,Am}$	σ_C^2	σ_e^2
3-wk wt	*M*	0.503 ± .438	0.933 ± 1.263	−0.346 ± .786	1.307 ± .719	0.844 ± .304
	F	1.250 ± .148	0.182 ± .305	−0.428 ± .159	1.460 ± .181	0.358 ± .087
6-wk wt	M	2.721 ± .490	5.726 ± 1.282	−2.333 ± .798	−2.124 ± .730	3.563 ± .308
	F	3.094 ± .462	1.704 ± .950	−1.148 ± .498	−0.136 ± .564	1.991 ± .271
8-wk wt	*M*	3.373 ± .538	1.200 ± 1.406	0.082 ± .874	0.365 ± .800	4.329 ± .338
	F	3.512 ± .121	0.069 ± .249	−0.085 ± .130	0.437 ± .148	2.756 ± .071
(6−3)-wk wt	*M*	1.569 ± .206	2.337 ± .540	−1.133 ± .336	0.168 ± .307	3.216 ± .129
	F	2.084 ± .760	0.376 ± 1.563	−0.471 ± .819	1.009 ± .928	2.010 ± .445

Table 8. *Estimates of causal components of variance for six- and eight-week weights from the least squares solution assuming* $\sigma_c^2 = 0$

Trait	Sex	σ_{A0}^2	σ_{Am}^2	$\sigma_{A0\,Am}$	σ_e^2
6-wk wt	*M*	2.153	2.445	−0.895	3.847
	F	3.074	1.488	−1.054	2.001
8-wk wt	*M*	3.470	1.764	−0.165	4.280
	F	3.576	0.764	−0.387	2.724

Table 9. *Genetic correlations between the sexes and the heritability of sex differences for individual body weight traits and postweaning gain*[a]

	3-wk wt	6-wk wt	8-wk wt	(6−3)-wk wt
3-wk wt	1.084 ± .048 (−0.093) ± .084	0.757 ± .109	0.678 ± .115	0.009 ± .157
6-wk wt	0.670 ± .102	0.872 ± .094 0.074 ± .089	0.881 ± .097	0.522 ± .142
8-wk wt	0.523 ± .111	1.105 ± .093	1.117 ± .088 (−0.071) ± .088	0.948 ± .137
(6−3)-wk wt	−0.037 ± .156	0.534 ± .143	0.617 ± .141	0.713 ± .143 0.110 ± .091

[a] The row headings refer to traits measured in males, the column headings refer to the corresponding traits measured in females. Each diagonal position has two entries, the upper being the genetic correlation and the lower is the heritability of the sex difference. The upper and lower triangular matrices contain genetic correlations between the sexes for the traits indicated by row and column headings.

observed responses in a selection experiment for postweaning gain in mice. Enfield (1960) obtained an estimate of 0.83 ± .05 for the genetic correlation between male and female chickens for eight-week body weight. Enfield *et al.* (1966) reported a value of 0.97 ± .13 for the genetic correlation between male and female pupa weights in *Tribolium*. Vesely and Robison (1970) concluded that genotype × sex interactions were not important for body weight and fleece traits in sheep.

Basis of genotype × sex interaction: The obvious genetical explanation for a genotype × sex interaction would perhaps be sex-limited autosomal genes (Passmore, 1969). However, since a gene × environment interaction would not be compatible with the observed pattern of change in the genetic correlation between the sexes seen in the current data (Table 9). The correlation tended to decrease from unity at three weeks of age to 0.872 at six weeks and increased again to unity at eight weeks. The gain between three and six weeks of age exhibited a correlation of 0.713. If this pattern reflects the true situation, then an explanation for the genotype × sex interaction would perhaps be on the basis of a differential chronological time pattern of gene action between the sexes. Then if males and females were measured at the same developmental age, no genotype × sex interaction would be evident. Hormonal factors influencing rate of sexual maturation may play a role here. Timon and Eisen (1970) showed a significantly faster rate of development in females, as measured by age at the point of inflection on the growth curve, for lines of mice derived from the population used in the present study.

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