GENOTYPE-SEX INTERACTION AND THE GENETIC CORRELATION BETWEEN THE SEXES FOR BODY WEIGHT IN *MUS MUSCULUS*[1,2]

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PHENOTYPIC sexual dimorphism is a commonly occurring phenomenon among mammalian species. In a large random mating population with a diploid mode of inheritance, male and female progeny receive, on the average, a similar sample of autosomal genes from the parental generation. Considering such a population, the expectations of autosomal gene frequencies in male and female progeny are identical. However, the presence of sexual dimorphism for a given quantitative character (e.g., body weight) in a species suggests that natural selection for a sex difference may have occurred at some period during the evolution of the species. In order for this type of natural selection to have been effective, the genetic variance of the difference between the same trait measured in males and females must have been present.

The internal physiological environments of males and females differ considerably (e.g., the type and quantity of hormone present). The external environments of males and females also may differ due to such factors as physical activity and social characteristics attributed to the male. Hence, the complement of autosomal genes controlling a quantitative trait may be expressed differently, depending on whether the gene effects are expressed in the male or female internal or external environments. This interaction between the male or female environments and the autosomal gene effects is a special case of a genotype-environment interaction. In order for this interaction to be present, the genetic correlation between the sexes for the same quantitative trait must be less than unity (ROBERTSON 1959). It is, of course, conceivable that sex-linkage could result in an observed genotype-sex interaction. However, the present development is concerned only with the effects of autosomal genes.

This study formulates the theoretical interrelationship among (1) the genetic variance of the difference between the same trait measured in males and females, (2) the genotype-sex interaction and (3) the genetic correlation between the sexes for the same trait. In order for either natural or artificial selection for a sexual dimorphism controlled by additive genes to be effective, these three factors must be present. Data from a laboratory population of *Mus musculus* provide

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evidence on the magnitude of these parameters and their biometrical interelationship.

THEORY

The following development assumes that the trait under investigation is controlled primarily by the additive effects of genes at autosomal loci. Thus, the effects of sex-linkage, dominance and epistasis are not included in the model.

The genetic variance of the difference between males and females for the same quantitative trait will be shown to be closely related to the genotype-sex interaction. Let subscripts "1" and "2" represent males and females respectively. Then the additive genetic variance of the difference between males and females for a trait such as body weight is given by the following expression:

$$\sigma^2_{G(1-2)} = \sigma^2_{G_1} + \sigma^2_{G_2} - 2\sigma_{G_1}\sigma_{G_2}r_{G'} , \qquad (1)$$

where $\sigma^2_{G_1}$ and $\sigma^2_{G_2}$ are the genetic variances for the same quantitative trait measured in males and females, respectively, and $r_{G'}$ is the genetic correlation between the sexes for the trait under consideration. The prime notation is used henceforth to denote a parameter which is free of scaling effects. The one-way analyses of variance and covariance given in Table 1 provide estimates of these parameters. The measurements made for each sex may be thought of as two separate traits or, alternatively, as the same trait measured in the internal and external environments of males and females.

The genotype-sex interaction component of variance may be obtained from the factorial analysis of variance presented in Table 2. The expected mean squares are given for the mixed model under the assumption that genetic groups are random and sexes are fixed effects (YAMADA 1962; EISEN *et al.* 1963).

The genotype (additive)—sex interaction variance component is defined as

$$\sigma^2_{GS} = \tfrac{1}{2}(\sigma_{G_1} - \sigma_{G_2})^2 + \sigma_{G_1}\sigma_{G_2}(1-r_{G'}) \qquad (2)$$

(ROBERTSON 1959). Note that expression (2) is greater than zero if $\sigma_{G_1} \neq \sigma_{G_2}$ and/or $r_{G'} < 1$. However, it is the latter condition, i.e. when the genetic correlation between the sexes for the trait is less than one, which is important from a selection standpoint. This is due to a differential ranking of genotypes, depending

TABLE 1

One-way analyses of variance and covariance with expected mean squares and expected mean product

Sources	Degrees of freedom	Expected mean squares			Expected mean product
		Males	Females	Males — Females	Males, Females
Genetic groups	$g-1$	$\sigma^2_1 + n\sigma^2_{G_1}$	$\sigma^2_2 + n\sigma^2_{G_2}$	$(\sigma^2_1 + \sigma^2_2) + n\sigma^2_{G(1-2)}$	$n\sigma_{G_1}\sigma_{G_2}r_{G'}$
Random error	$g(n-1)$	σ^2_1	σ^2_2	$(\sigma^2_1 + \sigma^2_2)$	

TABLE 2

Factorial analysis of variance and expected mean squares for estimating the genotype-sex component of variance

Sources	Degrees of freedom	Expected mean squares
Sexes (S)	1	Not relevant
Genetic groups (G)	g—1	$\sigma^2 + 2n\sigma^2_G$
S × G	g—1	$\sigma^2 + n\sigma^2_{GS}$
Random error	$2g(n-1)$	σ^2

upon whether the genotypes are measured in the male or female environments. On the contrary, the condition where $\sigma_{G_1} \neq \sigma_{G_2}$ is a scaling effect which can be removed either by a suitable transformation of the data or by adjusting formula (2) using the development suggested by DICKERSON (1962) and EISEN *et al.* (1963) as follows:

$$\sigma^2_{GS'} = \sigma^2_{GS} - \tfrac{1}{2}\,(\sigma_{G_1} - \sigma_{G_2})^2 \ . \tag{3}$$

Combining expressions (1) and (2) yields

$$\sigma^2_{GS} = \tfrac{1}{2}\,(\sigma^2_{G_1} + \sigma^2_{G_2}) - \sigma_{G_1}\sigma_{G_2} r_{G'} = \tfrac{1}{2}\,\sigma^2_{G(1-2)} \ , \tag{4}$$

which shows that the genetic variance of the difference between males and females is exactly twice the genotype-sex interaction. Expressing (1) in terms of the proportion of total variance gives a measure of the heritability (in the narrow sense) of the difference between the sexes for the trait, which may be written as follows:

$$h^2_{(1-2)} = 2\sigma^2_{GS}/\sigma^2_{P(1-2)} \ , \tag{5}$$

where $\sigma^2_{P(1-2)}$ is the phenotypic variance of the difference between the sexes for the trait. If there is heterogeneity of the genetic and/or phenotypic variances between the sexes, then formula (5) may be adjusted to give

$$h^2_{(1-2)'} = \frac{2\sigma^2_{GS} - (\sigma_{G_1} - \sigma_{G_2})^2}{\sigma^2_{P(1-2)} - (\sigma_{P_1} - \sigma_{P_2})^2} = \frac{2\sigma^2_{GS'}}{\sigma^2_{P(1-2)'}} \ . \tag{6}$$

From this development, it follows that the expected genetic advance is

$$\Delta G_{(1-2)} = i\, h_{(1-2)'}\, \sigma_{G(1-2)'} = \sqrt{2}\, i\, h_{(1-2)'}\, \sigma_{GS'} \ , \tag{7}$$

where i is the selection differential in standardized units. From equation (7) it can be seen that selection for sexual dimorphism in either direction is in fact selection for a directional genotype-sex interaction.

It can be shown that the genetic correlation between the trait as measured in males and in females, which is estimated by the variance-covariance technique given in Table 1, is unbiased by scaling effects. The formula for this genetic correlation is given by

$$r_{G'} = \sigma_{G_{12}}/\sigma_{G_1}\,\sigma_{G_2} \ , \tag{8}$$

where $\sigma_{G_{12}}$ is the genetic covariance between the same trait measured in males and females. However, the genetic correlation estimated from the intra-class

correlation in the factorial analysis of variance given in Table 2 is biased downward by scaling effects. The formula for this genetic correlation is given by

$$r_G = (\sigma^2_G - \tfrac{1}{2}\,\sigma^2_{GS})/(\sigma^2_G + \tfrac{1}{2}\,\sigma^2_{GS}) \qquad (9)$$

(YAMADA 1962; EISEN *et al.* 1963), where the numerators of (8) and (9) are equal. The intra-class genetic correlation given in expression (9) may be adjusted for scaling effects as follows:

$$r_{G'} = (\sigma^2_G - \tfrac{1}{2}\,\sigma^2_{GS})/[\sigma^2_G + \tfrac{1}{2}\,\sigma^2_{GS} - \tfrac{1}{2}\,(\sigma_{G_1} - \sigma_{G_2})^2] \qquad (10)$$

For further details see ROBERTSON (1959), DICKERSON (1962), YAMADA (1962), and EISEN *et al.* (1963).

SOURCE OF DATA AND EXPERIMENTAL PROCEDURES

Data from three generations of the randombred ICR Albino strain of laboratory mice originally obtained from the Institute of Cancer Research in Philadelphia were utilized in this study. A hierarchal mating plan was used in each generation. Matings were at random except for avoiding full- and half-sib matings. Only virgin females between eight and ten weeks old at time of mating were included.

Females were placed in separate cages 16 days after mating. Litters were standardized to eight mice at five days of age, at which time the sex ratio of each litter was equalized as nearly as possible. All mice were weaned at three weeks of age. Four mice of like sex from litters weaned the same day were randomly allotted to a cage. The parents and progeny were fed a standard mouse laboratory chow throughout the experiment. The laboratory was maintained at 72 ± 2°F and 50 ± 5% relative humidity.

Individual body weights to the nearest 1/10 gram were obtained at 3, 6 and 8 weeks of age. In addition, postweaning growth rate defined as 6 minus 3-week weight was recorded as a fourth trait. A recorded score for each of the four traits observed and survival at selection time were required for each individual entering into the statistical analysis. This was in addition to the requirement that each full-sib family had at least one live individual of each sex. These two factors accounted for the deletion of 3.8% of all mice weaned. This will result in very little or no bias in the reported results. A total of 781 male progeny and 767 female progeny from 95 sires and 214 dams were included in the analysis.

Analyses of variance within generations as outlined in Table 3 were carried out for each trait on individual male and female progeny separately in order to estimate the components of phenotypic variance (FALCONER 1960). Male and female full-sib family means were then obtained, and analyses of variance and covariance were used to partition the variability among sire family means and among dam family means within sire families (Table 4). This partitioning of variances and covariances from experimental data permits the estimation of the genetic correlation between the body weight trait measured in males and females in a manner similar to the analyses given in Table 1. The genetic variance and heritability of the difference between the same trait measured in males and females were obtained from an analysis of variance of differences between male and female full-sib family means (Table 4).

RESULTS AND DISCUSSION

Mean values of the four traits for males and females are given in Table 5. Males weighed significantly ($P < .01$) more than females at six and eight weeks of age. This difference is accounted for by the more rapid postweaning growth rate of males (4.32 g) since the difference in weight between males and females at weaning, although statistically significant, is small in actual magnitude (.33 g).

The phenotypic variances of males are significantly larger ($P < .01$) than

TABLE 3

Hierarchal analysis of variance on an individual basis with expected mean squares and degrees of freedom for males and females

Sources	Degrees of freedom		Expected mean squares*
	Males	Females	
Generations (T)	2	2	Not relevant
Sires (S)/T	92	92	$\sigma^2_w + k_1\sigma^2_d + k_2\sigma^2_s$
Dams (D)/S/T	109	109	$\sigma^2_w + k_3\sigma^2_d$
Progeny (W)/D/S/T	577	563	σ^2_w

* See KEMPTHORNE (1957) for calculation of coefficients in the expected mean squares.

TABLE 4

*Analyses of variance and covariance among and within sire family means**

Sources	df	Expected mean squares	
		Males	Females
Generations (T)	2	Not relevant	Not relevant
Sires (S)/T	92	$(k_4\sigma^2_{w_1} + \sigma^2_{d_1}) + k_5\sigma^2_{s_1}$	$(k'_4\sigma^2_{w_2} + \sigma^2_{d_2}) + k_5\sigma^2_{s_2}$
Dam Family Means $(\bar{D})$/S/T	109	$(k_6\sigma^2_{w_1} + \sigma^2_{d_1}) = \sigma^2_{\bar{d}_1}$	$(k'_6\sigma^2_{w_2} + \sigma^2_{d_2}) = \sigma^2_{\bar{d}_2}$

Sources	df	Expected mean squares	Expected mean product
		Males — Females	Males, Females
Generations (T)	2	Not relevant	Not relevant
Sires (S)/T	92	$[k_4\sigma^2_{w_1} + k'_4\sigma^2_{w_2} + \sigma^2_{d(1-2)}] + k_5\sigma^2_{s(1-2)}$	$\sigma_{d_{12}} + k_5\sigma_{s_{12}}$
Dam Family Means $(\bar{D})$/S/T	109	$[k_6\sigma^2_{w_1} + k'_6\sigma^2_{w_2} + \sigma^2_{\bar{d}(1-2)}] = \sigma^2_{d(1-2)}$	$\sigma_{d_{12}} = \sigma_{\bar{d}_{12}}$

* See KINNEY and SHOFFNER (1965) for calculating the coefficient for $\sigma^2_{w_1}$ and $\sigma^2_{w_2}$, and KEMPTHORNE (1957) for calculating the coefficient for $\sigma^2_{s_1}$, $\sigma^2_{s_2}$, $\sigma^2_{s(1-2)}$ and $\sigma_{s_{12}}$.

TABLE 5

Means, phenotypic variances and coefficients of variation of male and female body weight measurements

Statistic	Trait	Males	Females	t-test
Means (g)	3-week wt.	11.42 ± .04	11.09 ± .03	6.60**
	6-week wt.	30.03 ± .07	25.38 ± .06	52.60**
	6 minus 3-week wt.	18.61 ± .07	14.29 ± .06	48.87**
	8-week wt.	33.97 ± .09	27.79 ± .08	51.50**
				F-test
Phenotypic variances(g^2)	3-week wt.	3.252 ± .317	2.888 ± .300	1.13
	6-week wt.	7.140 ± .542	3.792 ± .319	1.88**
	6 minus 3-week wt.	5.678 ± .433	3.299 ± .224	1.72**
	8-week wt.	8.533 ± .621	5.157 ± .324	1.65**
	3-week wt.	15.80	15.32	
Coefficients of variation (%)	6-week wt.	8.89	7.68	
	6 minus 3-week wt.	12.79	14.14	
	8-week wt.	8.60	8.17	

** $P < .01$.

those of females for the three postweaning body weight measurements (Table 5). Heterogeneity of phenotypic variances probably is due to a scaling effect brought about by the larger mean weight of males. This is supported by the similarity of the coefficients of variation of males and females for each of the postweaning body weight traits.

Estimates of the sire, dam and progeny components of variance calculated from the analysis of variance of the form given in Table 3 are presented in Table 6 for the four traits measured on male and female progeny. The approximate standard errors of the estimates are based on the method given by KEMPTHORNE (1957). The components of variance of male progeny are larger than those for female progeny in all cases. These observational components of variance were used to estimate the genetic and environmental components of variance (FALCONER 1960). Additive genetic variance is estimated by $\hat{\sigma}^2_G = 4\hat{\sigma}^2_s$. The genetic and environmental maternal variances plus ¼ of the dominance variance is

TABLE 6

Observational components of variance of males and females for each body weight trait calculated from the analysis of variance of Table 3

Trait	Sex	$\hat{\sigma}^2_s$	$\hat{\sigma}^2_d$	$\hat{\sigma}^2_w$
3-week wt	males	.007 ± .245	2.310 ± .343	.935 ± .055
	females	.000 ± .222	2.209 ± .321	.679 ± .040
6-week wt	males	1.135 ± .474	2.813 ± .493	3.192 ± .188
	females	.354 ± .218	1.347 ± .375	2.091 ± .125
6 minus 3-week wt	males	.742 ± .358	2.069 ± .382	2.867 ± .169
	females	.341 ± .188	1.306 ± .237	1.652 ± .098
8-week wt	males	1.291 ± .520	2.756 ± .535	4.486 ± .264
	females	.400 ± .259	1.402 ± .317	3.355 ± .200

given by $\hat{\sigma}^2_q = \hat{\sigma}^2_d - \hat{\sigma}^2_s$. An estimate of the random environmental variance plus ¾ of the dominance variance is $\hat{\sigma}^2_V = \hat{\sigma}^2_w - 2\hat{\sigma}^2_s$. The phenotypic variance is defined as $\hat{\sigma}^2_p = \hat{\sigma}^2_s + \hat{\sigma}^2_d + \hat{\sigma}^2_w$, and the estimates of the respective proportions of total variance are $\hat{h}^2$, $\hat{q}^2$ and $\hat{v}^2$. This partition of the causal components of variance is used because previous studies with mice have shown that the maternal components of variance are large for postweaning body weight measurements (YOUNG *et al.* 1965), whereas the dominance and epistatic variances are negligible (MILLER *et al.* 1963).

Estimates of the causal components of variance and their proportion of the phenotypic variance are given in Table 7. Additive genetic variance is not significantly different from zero for three-week body weight. However, considerable additive genetic variance is present for postweaning growth rate and six- and eight-week body weights. The larger phenotypic variance for postweaning body weight and growth rate observed in males is due in part to the presence of more additive genetic variance for male progeny. The heritability for males is also consistently larger than that for females. Also contributing to the differential phenotypic variance is a larger maternal (plus ¼ dominance) component for males. However, when the maternal variance is expressed as a fraction of the total variance ($\hat{q}^2$), then there is little difference between male and female estimates. The random environmental (plus ¾ dominance) variance is smaller for male progeny for six- and eight-week weights but larger for postweaning growth rate and three-week weight.

Estimates of the observational variance components calculated from the analyses of variance and covariance among and within sire family means are presented in Table 8. For a completely balanced design, the estimates of $\hat{\sigma}^2_{s_1}$ and $\hat{\sigma}^2_{s_2}$ would be identical to those given in Table 6. The discrepancies between these estimates are therefore due to the unequal numbers of progeny per dam. However, this type of analysis is necessary in order to obtain estimates of the genetic variances and heritabilities of the difference between males and females, the genetic correlations between the sexes for the trait, the genotype-sex interaction variance components and the expected response to selection for a sexual dimorphism. Estimates of these genetic parameters which are given in Table 9 were found by applying formulas (1) to (10) to the data.

The method of estimating $\hat{h}^2_{(1-2)}$ requires some explanation since there is no direct estimate for $\hat{\sigma}^2_{d(1-2)}$ and $\hat{\sigma}^2_{w(1-2)}$. Eight-week weight is used to illustrate the procedure. Since the coefficients for $\hat{\sigma}^2_{w_1}$ and $\hat{\sigma}^2_{w_2}$ in the expected mean squares given in Table 4 varied over a narrow range (.297 to .310), they are assumed equal to .3. Equating observed with expected within sire family mean squares for males minus females gives

$$\hat{\sigma}^2_{\bar{d}(1-2)} = .3\hat{\sigma}^2_{w(1-2)} + \hat{\sigma}^2_{d(1-2)}$$
$$= .3(\hat{\sigma}^2_{w_1} + \hat{\sigma}^2_{w_2} - 2\hat{\sigma}_{w_{12}}) + (\hat{\sigma}^2_{d_1} + \hat{\sigma}^2_{d_2} - 2\hat{\sigma}_{d_{12}}) = 2.852 ,$$

where the expectation of $\hat{\sigma}_{w_{12}}$ is assumed to be zero. Now substituting the values of

TABLE 7

Genetic and environmental components of variance and their proportion of the total phenotypic variance

Trait	Sex	Causal components			Proportion of phenotypic variance		
		$\hat{\sigma}^2_A$	$\hat{\sigma}^2_Q$	$\hat{\sigma}^2_V$	$\hat{h}^2$	$\hat{q}^2$	$\hat{v}^2$
3-week wt	males	.028 ± .301	2.303 ± .546	.921 ± .493	.008 ± .301	.708 ± .167	.284 ± .151
	females	.001 ± .307	2.209 ± .505	.679 ± .444	.000 ± .307	.765 ± .174	.235 ± .153
6-week wt	males	4.540 ± 1.896	1.678 ± .843	.922 ± 1.040	.636 ± .266	.235 ± .118	.129 ± .143
	females	1.416 ± .872	.993 ± .572	1.383 ± .560	.373 ± .230	.262 ± .150	.365 ± .147
6 minus 3-week wt	males	2.968 ± 1.432	1.327 ± .647	1.383 ± .826	.523 ± .252	.234 ± .113	.243 ± .145
	females	1.364 ± .752	.963 ± .384	.970 ± .488	.413 ± .228	.293 ± .116	.294 ± .147
8-week wt	males	5.164 ± 2.080	1.465 ± .918	1.904 ± 1.160	.605 ± .244	.172 ± .107	.223 ± .135
	females	1.600 ± 1.036	1.002 ± .517	2.555 ± .684	.310 ± .201	.194 ± .100	.496 ± .132

TABLE 8

Estimates of observational components of variance and covariance among and within sire family means

Trait	$\hat{\sigma}^2_{s_1}$	$\hat{\sigma}^2_{s_2}$	$\hat{\sigma}^2_{s(1-2)}$	$\hat{\sigma}_{s_{12}}$
3-week wt	.010 ± .282	.006 ± .228	.005 ± .239	.005 ± .231
6-week wt	1.131 ± .498	.350 ± .220	.345 ± .253	.568 ± .231
6 minus 3-week wt	.702 ± .353	.314 ± .196	.375 ± .213	.319 ± .167
8-week wt	1.440 ± .569	.347 ± .261	.662 ± .343	.563 ± .241
Trait	$\hat{\sigma}^2_{\bar{d}_1}$	$\hat{\sigma}^2_{\bar{d}_2}$	$\hat{\sigma}^2_{\bar{d}(1-2)}$	$\hat{\sigma}_{\bar{d}_{12}}$
3-week wt	2.853 ± .383	2.394 ± .321	1.926 ± .259	2.457 ± .330
6-week wt	3.927 ± .527	1.956 ± .263	2.327 ± .312	1.778 ± .239
6 minus 3-week wt	2.945 ± .395	1.744 ± .234	1.834 ± .246	1.407 ± .189
8-week wt	4.259 ± .572	2.416 ± .324	2.852 ± .383	1.912 ± .256

$\hat{\sigma}^2_{w_1} + \hat{\sigma}^2_{w_2}$ estimated from the individual analysis of variance (Table 6) yields $\hat{\sigma}^2_{d(1-2)} = .500$ and $\hat{\sigma}^2_{w(1-2)} = 7.841$. Thus

$$\hat{h}^2_{(1-2)} = \frac{4\hat{\sigma}^2_{s(1-2)}}{\hat{\sigma}^2_{s(1-2)} + \hat{\sigma}^2_{d(1-2)} + \hat{\sigma}^2_{w(1-2)}} = \frac{2.648}{9.003} = .294 .$$

The genetic variance estimates for males ($\hat{\sigma}^2_{G_1}$) and females ($\sigma^2_{G_2}$) for all four traits closely approximate those given in Table 7 as expected. The genetic variances of the difference between males and females are large enough to be of biological significance for postweaning growth rate and eight-week weight, since the estimates are only slightly less than twice their standard errors. However, the estimate of $\hat{\sigma}^2_{G(1-2)}$ is not significant for six-week body weight. As would be predicted from the absence of measurable additive genetic variance within male and female groups, $\hat{\sigma}^2_{G(1-2)}$ is negligible for three-week weight.

It follows from equation (3) that the genotype-sex interaction component ($\hat{\sigma}^2_{GS}$) is of biological importance for postweaning growth rate and eight-week body weight. However, owing to the presence of heterogeneous genetic variances for body weight between the males and females in this population, the interaction components must be corrected for the effects of scaling. The corrected estimates of the interaction components of variance for postweaning growth rate and eight-week body weight, although reduced in magnitude, still are relatively large. Scaling will also affect estimates of the heritability of the difference between males and females [formula (5)] and the intra-class genetic correlation between the sexes for the trait [formula (9)].

The presence of genotype-sex interaction for postweaning growth rate and eight-week weight also is reflected in the estimates of the genetic correlations (corrected and uncorrected) being less than unity. Note that the correction for scaling has had a marked effect on the estimate of the genetic correlation for eight-week weight, but not for postweaning growth rate. While these genetic

TABLE 9

Estimates of genetic components of variance

Trait	$\hat{\sigma}^2_{G_1}$	$\hat{\sigma}^2_{G_2}$	$\hat{\sigma}^2_{G(1-2)}$	$\hat{\sigma}_{G_{12}}$	$\hat{\sigma}^2_{GS}$	$\hat{\sigma}^2_{GS'}$
3-week wt	.040 ± 1.128	.024 ± .912	.020 ± .956	.020 ± .924	.010 ± .478	.008
6-week wt	4.524 ± 1.992	1.400 ± .880	1.380 ± 1.012	2.272 ± 1.012	.690 ± .506	.146
6 minus 3-week wt	2.808 ± 1.412	1.256 ± .784	1.500 ± .852	1.276 ± .668	.750 ± .426	.597
8-week wt	5.760 ± 2.276	1.388 ± 1.044	2.648 ± 1.372	2.252 ± .964	1.324 ± .682	.578

Trait	$\hat{r}_G$	$\hat{r}_{G'}$	$\hat{h}^2_{(1-2)}$	$\hat{h}^2_{(1-2)'}$	$\Delta G_{(1-2)}$*
3-week wt	.625 ± 4.227‡	.640 ± 4.095	.013 ± .621	.010	.000
6-week wt	.767 ± .147	.903 ± .066	.217 ± .159	.084	.142
6 minus 3-week wt	.630 ± .219	.680 ± .195	.282 ± .160	.240	.585
8-week wt	.629 ± .229	.796 ± .132	.294 ± .152	.142	.436

* For a selection differential equal to one.
‡ Approximate standard errors of genetic correlations calculated by the method of ROBERTSON (1959).

correlations are not significantly less than unity owing to the large sampling errors generally associated with such estimates, they follow a pattern similar to the genotype-sex interaction components.

Scaling has had an appreciable effect on the magnitude of the genetic correlation for six-week weight ($\hat{r}_G = .767$ versus $\hat{r}_{G'} = .903$). This result emphasizes the necessity for removing scaling effects in these types of studies. The corrected genetic correlation of .9 suggests that little genotype-sex interaction is present for six-week weight relative to the total genetic variance.

The uncorrected heritabilities of the difference in weight between males and females are .013, .217, .282 and .295 for three-, six-, six minus three- and eight-week weights, respectively. Corresponding corrected heritability estimates are .010, .084, .240 and .142, respectively. No measurable response to selection for a sexual dimorphism is expected for weaning weight. Selection responses of .142, .585 and .436 g are predicted for six-, six minus three- and eight-week weights, respectively. In application, the response to selection for sexual dimorphism might be larger if selections are based on between family differences.

The population of mice studied indicates that for postweaning growth rate and eight-week weight the genetic correlation deviates sufficiently from one and the genotype-sex interaction is large enough to suggest that selection for a sexual dimorphism would be possible. RAHNEFELD *et al.* (1963) have suggested that the genetic correlation between the sexes for growth was less than perfect in the population of mice they studied. This hypothesis is further supported in the present data by the heritabilities of the difference in weight between males and females of .240 and .142 for postweaning growth rate and eight-week weight, respectively. The heritability of the difference between males and females for six-week weight suggests a negligible response to selection for a sexual dimorphism for this trait, whereas the expected response for three-week weight in zero.

KORKMAN (1957) has reported the only artificial selection experiment for a sexual dimorphism in mammals. He selected successfully for a smaller and larger sex difference with regard to body weight in two lines of mice. A control population was not utilized and realized heritabilities were not given, but it would appear from a plot of the data that the rate of genetic advance was not greater than would be expected on the basis of the genetic parameter estimates made in the ICR population.

The concept of selection for a sex difference (or a genotype-sex interaction) is closely associated with canalizing selection (WADDINGTON 1960) since the prediction equations for response to selection by the two methods are identical [formula (7)]. Canalizing selection is accomplished by submitting a portion of each family or genetic group of the population sampled to an environmental stress, and then selecting for an increase (poor canalization) or a decrease (good canalization) in sensitivity to the environmental treatment of the trait being studied. In selecting for a sexual dimorphism the environmental treatments in the form of the internal and external sexual environments are already present. WADDINGTON (1960) was successful in selecting for a reduced sensitivity of *Drosophila melanogaster* eyes to two temperature regimes. KINDRED (1965)

altered the canalization of scutellar bristles in *D. melanogaster* by selecting for temperature sensitivity and insensitivity during development. These experiments involved selection for an experimentally induced genotype-environment interaction.

In artificial selection experiments, the presence of a genetic correlation which is less than one may be important when the reproductive pattern dictates that selection be more intense in one sex. An example is the use of artificial insemination, which may result in a much greater selection intensity in males than in females. In such a situation the sexual dimorphism would tend to increase (BECKER *et al.*, unpublished). This dimorphism may be increased further if the heritability of the trait is greater in males, as was the case in the present study.

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SUMMARY

The biometrical interrelationship among the genetic variance of the difference between males and females, the genotype-sex interaction and the genetic correlation between the sexes for a quantitative trait expressed in both sexes has been described in the theoretical portion of this paper. The theoretical results clearly demonstrate that selection for an increase or decrease in a sexual dimorphism is selection for a directional genotype-sex interaction. The presence of a genotype-sex interaction will result in a genetic correlation of less than one between the sexes. The genotype-sex interaction is biased upward by the effects of scaling, while the genetic correlation is biased downward. An appropriate correction for scaling is presented and interpretations of the data are made on corrected estimates.—Data from a laboratory population of the house mouse provided evidence for a possible genotype-sex interaction for postweaning growth rate and eight-week body weight, but not for three- and six-week weight. This resulted in a genetic correlation of less than one between the sexes for the former two traits. The heritabilities of the difference between males and females for postweaning growth rate and eight-week body weight indicate that a favorable response to selection for a sexual dimorphism would be possible in this population.

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