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A. R. Swartz · T. R. Famula

The effects of selection for gain in mice on the direct-maternal genetic correlation

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Abstract Components of genetic variation for post-weaning growth traits were estimated for both control and growth stocks of mice. The effect of phenotypic selection for gain, which genetically combines selection for additive direct and maternal effects, on additive genetic variance components, heritability, and additive genetic correlations, is discussed. Quantitative genetic theory predicts that simultaneous selection for two metric traits in the same direction will cause the genetic correlation between the two traits to become more negative. The results presented in this paper conflict with this theory. The direct-maternal additive genetic correlation was more negative in the control line (with 356 mice) than in the growth-selected line (with 320 mice) for the three traits analyzed (0.310 vs 0.999 for 21-day weight, 0.316 vs 1.000 for 42-day weight, and 0.506 vs 1.000 for gain from 21–42 days). Estimates were obtained by restricted maximum likelihood (REML) computed under a derivative free algorithm (DFREML).

Key words Genetic correlation · Maternal effects Mice · Selection responses · Variance components

Introduction

Livestock selection decisions are often based on measurements of weight or growth rate. Postnatal maternal effects are of practical significance to livestock producers because the milk-producing ability of the dam plays a major role in determining offspring growth rate (e.g., Ahlschwede and Robison 1971; Hohenboken and Brinks 1971). Offspring selected on growth traits may also exhibit an undesirable maternal phenotype when mature (e.g., Van Vleck et al. 1977; Koch et al. 1982; Cantet et al. 1988).

Postnatal maternal influence is an embedded trait measurable only as a component of offspring phenotype. However, in mice, maternal effects are commonly studied by cross-fostering pups in order to separate prenatal and postnatal maternal effects. Mouse growth from 0–7 days of age is due primarily to postnatal maternal differences rather than direct genetic differences. About the time of weaning (21 days of age) the importance of postnatal maternal effects begins to decline while direct genetic effects become more important (Rutledge et al. 1972). The nature of the covariance between postnatal maternal and direct genetic effects is unclear due to conflicting experimental results (Rutledge et al. 1972).

Selection further complicates an understanding of the relationship between direct and maternal genetic effects and their role in influencing growth traits. Little is known about the effect of simultaneous selection for two quantitative traits, in this case direct and maternal effects. Simultaneous selection for two traits in the same direction is theorized to cause a negative change in the genetic correlation, and simultaneous selection in opposite directions to cause a positive change. Experimental results have both supported and contradicted this theory (Sheridan and Barker 1974; Falconer 1981). However, no experiments have studied the effect of simultaneous selection on an embedded trait.

The objectives of the present paper are: (1) to estimate additive components of genetic variation, heritabilities, and direct-maternal genetic correlations in cross-fostered control and growth-selected mouse populations, and (2) to compare direct-maternal genetic correlations in selected and control populations.

Materials and methods

Management of experimental animals

This research was conducted in accordance with the "Principles of laboratory animal care" (National Institutes of Health publication No. 85-23, revised 1985). Mice were housed in a room maintained at 21–25 °C with a 24 h light/dark cycle, 14 h light and 10 h dark (lighted from 06.00 to 20.00). Mice were fed, free choice, a pelleted diet (Simonsen Laboratories, Gilroy, Calif.) with a guaranteed analysis of at least 24% CP and 6% fat and a maximum of 3.5% CF. Cages were changed and fresh food and water supplied twice a week.

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A. R. Swartz (✉) · T. R. Famula
Department of Animal Science, University of California, Davis CA 95616, USA

At mating, mice were housed 2–3 per cage (either a male-female pair or two females and one male). The male was separated from the female(s) as soon as the female(s) appeared obviously pregnant. Females were checked daily after 3–4 days before the expected date of pupping. The actual date of birth for each litter and number of pups born per litter were both recorded. After pupping, one female and her litter were housed per cage. At weaning, mice were sexed and housed 4–6 of the same sex per cage.

Development of experimental stocks

Three lines of UCD mice: C, J (defined as JU in Bradford and Famula 1984) and G were crossed to create two new stocks of outbred mice so that variation due to genetic differences among mice could be measured. C is an unselected control line, J is a line with high litter size, and G is derived from a line selected for postweaning gain for more than 60 generations. The pedigrees of lines C and J, which were maintained as closed lines, were given by Bradford and Famula (1984, their Fig. 1); the present line G was produced by crossing lines G and H (depicted in that figure) and extracting a line that did not carry the high-growth (hg) gene.

Although inbreeding was not deliberately practiced in these three lines, relatively high inbreeding coefficients, of over 0.75, would have accumulated over the nearly 100 generations that this colony has been closed to outside breeding. Crosses between these three lines were made to eliminate this accumulated inbreeding. In this study, J is used as a common background for the new control and growth stocks being created and to ensure adequate reproductive performance in both stocks. Thus, the results here are a comparison of two F_2 and F_3 populations (lines G and C), in which both inbred parent lines have been crossed to the same line (J), which has not been selected for postweaning growth. A decomposition of the genotypic value of an individual in each line can be derived from the work of Bulmer (1985, p. 58) or Mather and Jinks (1982, p. 135). This design allows the comparison of unselected and growth-selected populations to a common point of reference.

Table 1 presents the crosses made to generate the stocks used in this experiment. Individuals mated in generation 0 were a random sample selected from the available line C, J and G mice. A total of 20 pairs of mice were mated: 5 J × C, 5 C × J, 5 J × G and 5 G × J. Litters were sexed at 2 days of age and standardized to ten pups (when possible five males and five females). Weights at 21 (weaning) and 42 days were recorded to the nearest 0.1 g for all pups.

At 8 weeks of age, F_1 pups were randomly mated within stock, with the stipulation that at least one pup of each sex from each generation 0 litter be included in the F_1 mating plan to increase the effective population size and the amount of genetic variation within stocks. Within litters and sexes, selection of pups for mating was random. Fourteen reciprocal control crosses, seven CJ × JC and seven JC × CJ, and 18 reciprocal growth-selected crosses; nine GJ × JG and nine JG × GJ, were made. Litters produced by F_1 dams were sexed at 2 days of age, but not standardized to ten pups because various reproductive problems occurred that reduced the overall number of litters produced.

The F_2 pups were weighed at 21 and 42 days of age. At 8 weeks of age they were randomly harem-mated (two females per male) within stock according to the following mating plan: 24 control (CO) females × 12 CO males and 24 growth (GO) females × 12 GO males. Pups from these matings were sexed at 2 days of age and assigned to cross-fostering units. The cross-fostering units consisted of two dams of the same stock that pupped within the same 12-h period. Litters were standardized to eight pups and, where possible, each dam was allowed to raise four pups of her own and four pups belonging to the other dam in the cross-fostering unit. Weights at 21 and 42 days of age were recorded to the nearest 0.1 g. The relationships of these F_3 pups (noted as CO′ and GO′ in Table 1) among themselves and with their parents, both genetic and nurse, provide the basis for the estimation of genetic variances, especially for separating the additive direct, maternal, and direct-maternal components.

Analysis of data

Data were collected on both fostered and non-fostered offspring (F_3) as well as on parents of offspring (F_2) (no F_1 data were included in the analyses). The data for analysis consisted of both raw and log-transformed phenotypic measurements on three growth traits: 21-day weight (WT21), 42-day weight (WT42), and weight gain from 21 to 42 days (GAIN). Control and growth data were analyzed separately using DFREML (Meyer 1988). This method is based on the general mixed linear model and assumes the data are a sample from a multivariate normal distribution. Variance components were estimated using the following specific form of the general mixed linear model

$$\mathbf{y} = \mathbf{Xb} + \mathbf{Z_d d} + \mathbf{Z_m m} + \mathbf{Z_c c} + \mathbf{e} \quad (1)$$

where $\mathbf{y}$ is a vector of N records (e.g., 21-day weight), $\mathbf{b}$ is a vector of p fixed effects (i.e., sex), $\mathbf{d}$ is an unknown random vector of q additive direct genetic effects, m is an unknown random vector of q additive maternal effects, $\mathbf{c}$ is an unknown random vector of s common environmental (i.e., litter) effects, and $\mathbf{e}$ is an unknown random vector of N residuals.

$\mathbf{X}$, $\mathbf{Z_d}$, $\mathbf{Z_m}$ and $\mathbf{Z_c}$ are known incidence matrices that relate effects in $\mathbf{b}$, $\mathbf{d}$, $\mathbf{m}$ and $\mathbf{c}$ to phenotypes in $\mathbf{y}$, respectively. Note that the length of $\mathbf{y}$ is equal to the number of available phenotypes (N), while the lengths of $\mathbf{d}$ and $\mathbf{m}$ are the same and are equal to the total number of animals represented in the data set (q). Thus, $\mathbf{q}>\mathbf{N}$ since parents without measured phenotypes are included in the analysis to permit construction of the appropriate numerator relationship matrix. $\mathbf{c}$ is of the order of the total number of litters (s) in both F_2 (not cross-fostered) and F_3 (cross-fostered) generations. The appropriate columns of $\mathbf{Z_d}$ and $\mathbf{Z_m}$ are null for animals in $\mathbf{d}$ and $\mathbf{m}$ that either do not have phenotypes or are not mothers.

For the growth-selected line, N=320 records from s=36 litters. The number of animals represented in $\mathbf{d}$ and $\mathbf{m}$, including animals without phenotypes, was $\mathbf{q}$=391. In the control line, N=356 records from s=35 litters. The number of animals represented in $\mathbf{d}$ and $\mathbf{m}$, including those without phenotypes, was $\mathbf{q}$=420.

Further genetic information is included in this model through the covariance matrices of the elements in model (1). Specifically, $E[\mathbf{y}]=\mathbf{Xb}$, $E[\mathbf{d}]=E[\mathbf{m}]=\mathbf{0}$, $E[\mathbf{c}]=\mathbf{0}$ and $E[\mathbf{e}]=\mathbf{0}$, and

$$\mathrm{Var}\begin{bmatrix}\mathbf{d}\\ \mathbf{m}\\ \mathbf{c}\\ \mathbf{e}\\ \mathbf{y}\end{bmatrix} = \begin{bmatrix} \mathbf{A}\sigma_d^2 & \mathbf{A}\sigma_{dm} & 0 & 0 & \mathbf{AZ_d'}\sigma_d^2 \\ \mathbf{A}\sigma_{dm} & \mathbf{A}\sigma_m^2 & 0 & 0 & \mathbf{AZ_d'}\sigma_m^2 \\ 0 & 0 & \mathbf{R_c} & 0 & \mathbf{R_c Z_d'} \\ 0 & 0 & 0 & \mathbf{R_c} & \mathbf{R_c} \\ \mathbf{Z_d A}\sigma_d^2 & \mathbf{Z_m A}\sigma_m^2 & \mathbf{Z_c R_c} & \mathbf{R_c} & \mathbf{V} \end{bmatrix}$$

where $\mathbf{A}$ is the numerator relationship matrix among all animals with and without measured phenotypes, σ_d^2 is additive direct genetic var-

Table 1 Creation of control and growth stocks

Generation	Matings		
	Female	Male	Offspring
0	J	C	CJ[a]
	C	J	JC
	J	G	GJ
	G	J	JG
1	CJ	JC	JCJ[b]
	JC	CJ	CJC
	GJ	JG	JGJ
	JG	GJ	GJG
2	CO[c]	CO	CO′
	GO	GO	GO′

[a] Offspring are listed with the line of the male parent first
G, growth-selected line, J, high-litter-size line and C, control line
[b] Offspring are listed with the male parent symbolized by the first and second letters and the female parent symbolized by the second and third letters
[c] JCJ=CJC=CO=control stock of mice; JGJ=GJG=GO=growth stock of mice

iance, σ^2_m is additive maternal genetic variance, and σ_{dm} is the additive direct-maternal genetic covariance. Moreover, $\mathbf{R_c}=\mathbf{I_s}\,\sigma^2_c$, $\mathbf{R_e}=\mathbf{I_N}\,\sigma^2_e$ and $\mathbf{V}=\mathbf{Z_dAZ'_d}\;\sigma^2_d+\mathbf{Z_mAZ'_m}\;\sigma^2_m+(\mathbf{Z_dAZ'_m}+\mathbf{Z_mAZ'_d})\sigma_{dm}+\mathbf{Z_cR_cZ'_c}+\mathbf{R_e}$. In addition, σ^2_c is variance due to common, nongenetic litter effects and σ^2_e is variance of residual effects.

Heritability for additive direct effects was defined as $h^2_d=\sigma^2_d/\sigma^2_P$, where $\sigma^2_P=\sigma^2_d+\sigma^2_m+\sigma_{dm}+\sigma^2_c+\sigma^2_e$. Heritability for additive maternal effects was defined as $h^2_d=\sigma^2_m/\sigma^2_P$. Heritability for total direct and maternal additive effects was defined by Willham (1963) as $h^2_t=(\sigma^2_d+1.5\;\sigma_{dm}+.5\;\sigma^2_m)/\sigma^2_P$, and is the proportion of the phenotypic variance resulting from the combined influence of all additive genetic effects. The genetic correlation between additive direct and maternal effects was defined as $r_g=\sigma_{dm}/(\sigma^2_c\,\sigma^2_m)^{1/2}$. A common environmental component was defined as $c^2=\sigma^2_c/\sigma^2_P$, the ratio of common environmental variance to the phenotypic variance. Heritabilities and genetic correlations were estimated by replacing defined parameters with their DFREML estimates.

Table 2 Phenotypic means (g) for growth traits adjusted for sex, litter size and parity

Trait	Control		Growth Selected	
	Mean (n)	SD	Mean (n)	SD
WT21	10.6 (356)	1.58	13.3 (320)	1.92
WT42	24.5 (356)	2.44	32.7 (320)	2.77
GAIN	13.9 (356)	2.09	19.4 (320)	2.33

Table 3 Causal components of phenotypic variation for raw data estimated by DFREML

Trait	Stock	Estimates of Causal Components[a]				
		σ^2_d	σ_{dm}	σ^2_m	σ^2_c	σ^2_e
WT21	Control	2.046	–0.154	0.121	0.445	<0.001
	Growth	3.315	0.109	0.004	1.027	0.171
WT42	Control	5.544	0.233	0.098	0.269	0.003
	Growth	7.890	0.052	0.001	1.214	0.359
GAIN	Control	4.221	–0.170	0.027	0.046	0.292
	Growth	3.273	0.114	0.004	0.942	1.959

[a] Causal components are additive direct variance (σ^2_d), additive direct-maternal covariance (σ_{dm}), additive maternal variance (σ^2_m), common environment variance (σ^2_c), and residual environmental variance (σ^2_e)

Results and discussion

Phenotypic means of growth traits adjusted for sex, litter size, and parity, appear in Table 2. Variance component solutions from raw and log-transformed data appear in Tables 3 and 5. Tables 4 and 6 summarize estimates of heritability, additive genetic correlation and phenotypic variation for growth traits from raw and log-transformed data.

Direct and maternal effects

DFREML estimates indicate that additive genetic variation at both 21 days and 42 days is primarily of direct origin (Table 3). Similarly, weight gain from 21 to 42 days is heavily influenced by direct genes. Typically, studies with mice have reported larger maternal-effect influences at 21 days than were found in this study (e.g., El Oksh et al. 1967; Rutledge et al. 1972).

Phenotypic variance

Total phenotypic variance for all traits is greater in the growth-selected stock than in the control stock (Table 4). There are three potential explanations for this difference in phenotypic variance between the stocks. First, there is greater genetic difference between line G and J mice than between line C and J mice, so that more genetic variation in the former cross would be expected which, naturally, would be reflected in larger phenotypic variation. Second, the assumption that decreased genetic variation due to selection will lead to decreased phenotypic variation is seldom supported experimentally. In fact, phenotypic variation may even increase with selection (Falconer 1981). Third, based on a comparison of the coefficients of variation between stocks, there is some evidence that the difference in phenotypic variation is simply a scale effect. This possibility was investigated using a log transformation of the data, and the resulting genetic parameter estimates are presented in Table 6. A comparison of Tables 4 and 6 shows that the log transformation had little to no effect on interpretation of this data. The only area of inconsistent results across Tables 4 and 6 is the relative magnitudes of the phenotypic variance across growth and con-

Table 4 Heritability estimates, additive genetic correlations and phenotypic variances from raw data with DFREML

Trait	Stock	h^{2a}_d	h^{2b}_m	h^{2c}_t	c^{2d}	r^e_g	σ^2_P
WT21	Control	0.832	0.049	0.763	0.181	–0.310	2.459
	Growth	0.717	0.000	0.752	0.222	0.999	4.625
WT42	Control	0.902	0.016	0.967	0.044	0.316	6.147
	Growth	0.829	0.000	0.837	0.128	1.000	9.514
GAIN	Control	0.956	0.006	0.901	0.010	–0.506	4.416
	Growth	0.520	0.001	0.548	0.150	1.000	6.923

[a] Direct heritability
[b] Maternal heritability
[c] Total heritability
[d] Common environmental component
[e] Additive direct-maternal genetic correlation

trol lines. For the raw (untransformed) data (Table 4), the phenotypic variance of the growth line exceeds the variance of the control line. However, under the log transformation (Table 6) the phenotypic variance for postweaning gain is larger in the control line than the variance in the growth line.

The effect of selection on litter components

Tables 3 and 4 show not only an increase in phenotypic variance of growth-selected mice over control lines, but also a significant change in litter components. The additive maternal variance in growth-selected mice is significantly reduced from that of control mice. Coincidentally, the common environmental component increased with the practice of selection for growth.

The power of cross-fostering designs is evidenced here by the ability to separate the two litter components. Selection for growth has not constrained the variance in maternal effects. On the contrary, as the sum of these two components indicate, selection for growth appears to increase the variability of maternal performance. However, the nature of that change can not be specifically described. Selection for growth has apparently exhausted additive genetic variation in the maternal effects on postweaning growth. Yet, the variability of common environmental effects is dramatically increased. Taken together, mothers in the selected line are more variable in the contribution they make to the phenotype of their litter than are mothers in the control line. However, the source of this increased variability is not additive genes.

Mothers in the selected line are either more sensitive to environmental change or else nonadditive genetic components are responsible for this increase in variance of selected over control animals. The common environmental component, σ^2_c, is a combination of both nonadditive genetic effects and environmental contributions. Thus, the two causes of increased variability can not be separated with these data. Perhaps some interaction between genes in the original growth-selected stock and those of line J is being expressed. However, the evidence is that additive maternal gene effects are not responsible for this change in variation.

Table 5 Causal components (g^2) of phenotypic variation for log data estimated by DFREML

Trait	Stock	Estimates of Causal Components [a] ($\times 10^3$)				
		σ^2_d	σ_{dm}	σ^2_m	σ^2_c	σ^2_e
WT21	Control	21.48	−0.88	0.80	4.00	<0.01
	Growth	20.05	6.00	2.67	21.42	4.06
WT42	Control	8.17	0.10	0.08	0.33	<0.01
	Growth	7.17	0.13	<0.01	1.28	0.37
GAIN	Control	23.31	−0.14	0.12	0.02	2.64
	Growth	9.15	0.29	0.01	1.96	4.88

[a] Causal components are additive direct (σ^2_d), additive direct-maternal (σ_{dm}), additive maternal (σ^2_m), common environment (σ^2_c), and residual genetic and environmental variance (σ^2_e)

The effect of selection on additive genetic correlations

Growth-selected mice were selected for gain based on recorded phenotypic measurements of gain. This phenotypic measurement of gain contains a direct additive genetic component and an embedded maternal additive genetic component as well as various environmental components. Thus selection for increased gain is selection for an increase in all the components which influence the phenotype for gain. Genetically, selection for gain is simultaneous selection for two traits, additive direct effects and additive maternal effects.

In this case, quantitative genetic theory predicts that the genetic correlation between the two traits being selected for will become more negative. Possible reasons (Sheridan and Barker 1974) for this are: (1) alleles with a positive effect on both traits are rapidly fixed in the population; (2) alleles with a positive effect on one trait and a neutral effect on the other trait also become fixed, but at a slower rate; (3) alleles with a positive effect on one trait and a negative effect on the other trait remain segregating in the population; and (4) alleles with a negative effect on both traits, or a negative effect on one trait and a neutral effect on the other trait, are eliminated from the population. Elimination of alleles contributing only negative effects theoretically causes the correlation between the two traits to become more negative. This prediction assumes: (1) the original

Table 6 Heritability estimates, additive genetic correlations and phenotypic variances from log data with DFREML

Trait	Stock	h^{2a}_d	h^{2b}_m	h^{2c}_t	c^{2d}	r^e_g	σ^2_P
WT21	Control	0.846	0.032	0.809	0.158	−0.035	0.025
	Growth	0.521	0.000	0.533	0.458	1.000	0.038
WT42	Control	0.941	0.009	0.963	0.038	0.123	0.009
	Growth	0.800	0.000	0.823	0.142	1.000	0.009
GAIN	Control	0.898	0.050	0.892	0.001	−0.082	0.026
	Growth	0.562	0.001	0.589	0.120	1.000	0.016

[a] Direct heritability
[b] Maternal heritability
[c] Total heritability
[d] Common environmental component
[e] Additive direct-maternal genetic correlation

genetic correlation between the two traits is primarily due to pleiotropy; and (2) selection is practiced long enough for alleles to become fixed in the population.

Estimates from this experiment are that the direct-maternal additive genetic correlations are more negative in the control stock than in the growth-selected stock for all three growth traits (Table 4). Phenotypic selection for gain, which in genetic terms is simultaneous selection for additive direct and maternal effects, resulted in more positive additive direct-maternal genetic correlations for all three growth traits. These results are unexpected and conflict with accepted quantitative genetic theory (Sheridan and Barker 1974; Falconer 1981). However, other experimental results have also contradicted this genetic theory so that the effect of simultaneous selection for two traits on the genetic correlation between the traits remains unclear (Friars et al. 1962; Sheridan and Barker 1974).

Adding to this confusion is the apparently illogical change seen in the direct-maternal correlation in the control line for 21-day weight, 42-day weight and the gain between these two periods. The direct-maternal correlation for 21-day weight is 0.310 (Table 4), with a change to 0.316 for 42-day weight. Yet, the direct-maternal correlation for the gain in weight is 0.506; outside the bounds of 0.310 and 0.316 for each weight. The simplest explanation for this inconsistency, and perhaps most likely, is sampling error. Estimates of genetic correlation from a data set of this size are quite susceptible to variation caused by sampling.

Other explanations, based on the modelling of maternal traits, can also be attempted. A likely source is in the direct-maternal covariances among the two weight-traits; specifically, the covariances between direct effects for 21 (42)-day weight and maternal effects for 42 (21)-day weight. Both of these covariances contribute to the direct-maternal covariance of postweaning gain. Unfortunately, the two parameters cannot be directly estimated. Speculation on the magnitude and direction of these parameters, with the data at hand, would be unwarranted.

In summary, the effect of selection on the genetic covariance between direct and maternal traits remains unclear. A priori the design in the experiment would be expected to yield useful answers. Cross-fostering is a powerful statistical design to separate genetic and environmental maternal effects. In addition, the methods of variance estimation used are the most powerful available. Though many useful results can be found in this analysis, a definitive answer to the original question remains to be provided.

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