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Partitioning of genetic effects on lifetime performance of mice *

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Summary. A total of 2,457 lifetime performance records of 29 genetic groups of mice was analyzed using multiple regression of records on the proportion of gene contribution from 6 lines (designated as Lines M_P, M_Q, W_P, W_Q, C_P and C_Q). Genetic effects were partitioned into line additive, line maternal, direct heterosis, maternal heterosis and paternal heterosis effects. The line additive and line maternal effects were expressed as deviations from Line C_Q. Seventeen of 25 line additive effects differed significantly ($P<0.05$) from Line C_Q whereas only 4 of 25 line maternal effects deviated significantly from Line C_Q. Deviations in line additive effects from C_Q were negative in all lines examined whereas deviations in line maternal effects from C_Q were all positive, indicating a negative relationship between line additive and line maternal effects. Direct heterosis effects were all positive and significant ($P<0.01$) except in the $M_P \times W_P$ cross which was produced by mating Lines M_P and W_P of the same base population (P). Maternal heterosis effects were significant in 10 of 20 cases whereas paternal heterosis effects were significant in 13 of 20 cases. Although direct heterosis is a major component of total heterosis effects (sum of direct, maternal and paternal heterosis), the results suggest that parental heterosis may need to be considered in producing multiple way crosses. The fitting of line additive, line maternal, direct heterosis, maternal heterosis and paternal heterosis effects in the multiple regression model effectively accounted for all genetic effects in lifetime performance.

Key words: Mice – Lifetime – Performance – Additive – Maternal – Heterosis

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Introduction

Crossbreeding has been used extensively to capitalize on heterosis as a means of improving animal production.

Literature reviews on crossbreeding are abundant (Dickerson 1973; Turton 1981; Sheridan 1981; Sellier 1982). Different methods of analyzing crossbreeding data (Henderson 1952; Griffing 1956; Gardner and Eberhart 1966; Robison et al. 1981) have been presented. Among them is the multiple regression of performance records on the proportion of genes contributed by different lines. This method has been used frequently in recent years. Touchberry (1970) and Koger et al. (1975) used the regression of breed group means on the proportion of gene contributions whereas other researchers (Robison et al. 1980, 1981; Neville et al. 1984; Lin et al. 1984; Jungst and Kuhlers 1984) used individual observations instead of breed group means as dependent variables. Multiple regression analysis of cross data (breeds, lines or strains) has two general advantages: firstly, a general genetic effect can be partitioned to provide detailed information on its genetic components; secondly, it provides a logical means of predicting the potential performance of various breed compositions without testing all possible crosses. Therefore, it allows maximum use of results from analysis of cross data.

Analyses of cross data by multiple regression as reported in the literature are generally concerned with breed additive, breed maternal and direct heterosis effects. In the majority of these studies maternal and paternal heterosis effects have not been examined either because the data did not allow for estimation of these effects or because these two effects were assumed to be negligible. For example, both paternal and maternal heterosis effects are not estimable in diallel cross data. Sires and dams must be crossbreds to allow for simultaneous estimation of paternal and maternal heterosis effects on the performance of their progeny. The purpose of this study was to estimate the line additive, line maternal, direct heterosis and maternal

and paternal heterosis effects on lifetime performance in mice.

Materials and methods

Genetic lines

Six straightbred lines of mice, M_P, M_Q, W_P, W_Q, C_P and C_Q, were developed from two populations (designated as P and Q) of different origin. These lines and their origin have been described by Nagai and McAllister (1982). The P population was synthesized from four inbred strains in 1966 while the Q population was synthesized from two randombred strains maintained by Dr. D. S. Falconer, Edinburgh. The M lines (M_P and M_Q) were selected for postnatal maternal performance as measured by 12-day body weights of a crossfostered first litter. The W lines (W_P and W_Q) were selected for individual body weight at 42 days in the first litter while the C lines (C_P and C_Q) were maintained unselected for 20 generations.

These 6 lines were used both as 6 straightbred lines (STR) and to set up 5 lines of crisscross (CC) and 5 lines of repeat hybrid male cross (RHMC). They were maintained under the defined mating systems (STR, CC and RHMC) for three time periods as shown in Table 1. Consequently, 29 genetic groups (6 types of straightbreds and 23 types of crossbreds) were available (Table 2) for estimation of various genetic effects.

In each of the 29 genetic groups, females at 42 days of age were pairmated with males and maintained continuously for 155 days, allowing successive production of litters. Litter size was not standardized and all young born alive were left with the mother until disposal at day 18. Total performance of the pair during 155 days of reproduction was defined as lifetime performance. Commercial pellet feed (Purina Mouse Chow) and tap water were supplied ad libitum. Temperature and humidity in mouse rooms ranged from 20° to 24 °C and 40% to 50%, respectively. The lighting regimen was 12 h light/12 h dark.

Data on 2,457 lifetime performance records of 29 female genetic groups were analyzed using multiple regression to partition the genetic effects of lifetime performance into various genetic components. The traits of lifetime performance examined for each female were total number of young born alive, total body weight (g) of young born alive, total number of young weaned at day 18, total body weight (g) of young at day 18, and number of parturitions.

Statistical analysis

The data were analyzed with a multiple regression model to partition genetic effects into line additive, line maternal, direct heterosis, specific maternal and paternal heterosis effects.

$$Y_{ijkl}=\mu+\Sigma b_i k_i+\Sigma b_j k_j+\Sigma h_{ij} k_{ij}+\Sigma m_j k^*_j+\Sigma f_{ij} c_{ij}+\Sigma p_{ij} c^*_{ij}+G_k+e_{ijkl}$$

where μ is a constant,

k_i and k_j are proportions of genes contributed by the i^{th} line through the sire and the j^{th} line through the dam, respectively,
b_i and b_j are the line additive effects of the i^{th} and j^{th} lines,
k_{ij} is the expected proportion of loci with one gene contributed by the i^{th} line and the other by the j^{th} line,
h_{ij} is the direct heterosis effect between the i^{th} and j^{th} lines,
k^*_j is the proportion of genes in the dam from the j^{th} line,
m_j is the line maternal effect due to the j^{th} line,
c_{ij} is the expected proportion of loci in dam with one gene from the i^{th} line and the other from the j^{th} line,
f_{ij} is the maternal heterosis effect corresponding to c_{ij},
c^*_{ij} is the expected proportion of loci in sire with one gene from the i^{th} line and the other from the j^{th} line,
p_{ij} is the paternal heterosis effects corresponding to c^*_{ij},
G_k is the time period effect, and
e_{ijkl} is a random residual effect [$NID(0,\sigma^2_e)$].

It should be noted that the above analyses were based on lifetime performance of the females (i.e. mothers) that produced pups. The proportions of gene contributions (k_i, k_{ij}, k^*_j, c_{ij} and c^*_{ij}) of the above model refers to the genetic makeups of the mothers rather than the pups. Therefore, sire and dam

Table 1. Matings for crisscross (CC), repeated hybrid male cross (RHMC) and straightbred (STR) lines

Mating system		Time period 1	Time period 2	Time period 3
		Male × Female	Male × Female	Male × Female
CC	1)	$M_Q \times M_P$	$M_P \times (M_Q M_P)$	$M_Q \times [M_P \times (M_Q M_P)]$
	2)	$M_P \times M_Q$	$M_Q \times (M_P M_Q)$	$M_P \times [M_Q \times (M_P M_Q)]$
	3)	$W_P \times W_Q$	$W_Q \times (W_P W_Q)$	$W_P \times [W_Q \times (W_P W_Q)]$
	4)	$C_P \times C_Q$	$C_Q \times (C_P C_Q)$	$C_P \times [C_Q \times (C_P C_Q)]$
	5)	$M_P \times W_P$	$W_P \times (M_P W_P)$	$M_P \times [W_P \times (M_P W_P)]$
RHMC	1)	$(M_Q M_P) \times (M_P M_Q)$	$(M_P M_Q) \times (M_Q M_P \times M_P M_Q)$	$(M_Q M_P) \times [(M_P M_Q) \times (M_Q M_P \times M_P M_Q)]$
	2)	$(M_P M_Q) \times (M_Q M_P)$	$(M_Q M_P) \times (M_P M_Q \times M_Q M_P)$	$(M_P M_Q) \times [(M_Q M_P) \times (M_P M_Q \times M_Q M_P)]$
	3)	$(W_P W_Q) \times (W_Q W_P)$	$(W_Q W_P) \times (W_P W_Q \times W_Q W_P)$	$(W_P W_Q) \times [(W_Q W_P) \times (W_P W_Q \times W_Q W_P)]$
	4)	$(C_P C_Q) \times (C_Q C_P)$	$(C_Q C_P) \times (C_P C_Q \times C_Q C_P)$	$(C_P C_Q) \times [(C_Q C_P) \times (C_P C_Q \times C_Q C_P)]$
	5)	$(M_P W_P) \times (W_P M_P)$	$(W_P M_P) \times (M_P W_P \times W_P M_P)$	$(M_P W_P) \times [(W_P M_P) \times (M_P W_P \times W_P M_P)]$
STR	1)	$M_P \times M_P$	$M_P \times M_P$	$M_P \times M_P$
	2)	$W_P \times W_P$	$W_P \times W_P$	$W_P \times W_P$
	3)	$M_Q \times M_Q$	$M_Q \times M_Q$	$M_Q \times M_Q$
	4)	$W_Q \times W_Q$	$W_Q \times W_Q$	$W_Q \times W_Q$
	5)	$C_P \times C_P$	$C_P \times C_P$	$C_P \times C_P$
	6)	$C_Q \times C_Q$	$C_Q \times C_Q$	$C_Q \times C_Q$

Table 2. Number of animals and contributions of female genetic groups to various genetic effects

Genetic group of females	No. of animals	Line additive						Line maternal						Direct heterosis				Maternal heterosis				Paternal heterosis			
		M_P	M_Q	W_P	W_Q	C_P	C_Q	M_P	M_Q	W_P	W_Q	C_P	C_Q	M_PM_Q	W_PW_Q	C_PC_Q	M_PW_P	M_PM_Q	W_PW_Q	C_PC_Q	M_PW_P	M_PM_Q	W_PW_Q	C_PC_Q	M_PW_P
M_P	199	1	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
M_Q	199	0	1	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
W_P	202	0	0	1	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
W_Q	198	0	0	0	1	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0
C_P	145	0	0	0	0	1	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0
C_Q	203	0	0	0	0	0	1	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0
M_PM_Q	107	½	½	0	0	0	0	0	1	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0
M_QM_P	109	½	½	0	0	0	0	1	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0
W_QW_P	54	0	0	½	½	0	0	0	0	1	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0
C_QC_P	53	0	0	0	0	½	½	0	0	0	0	1	0	0	0	1	0	0	0	0	0	0	0	0	0
W_PM_P	54	½	0	½	0	0	0	1	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0
W_PW_Q	46	0	0	½	½	0	0	0	0	0	1	0	0	0	1	0	0	0	0	0	0	0	0	0	0
C_PC_Q	53	0	0	0	0	½	½	0	0	0	0	0	1	0	0	1	0	0	0	0	0	0	0	0	0
M_PW_P	45	½	0	½	0	0	0	0	0	1	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0
$(M_QM_P)\times(M_PM_Q)$	54	½	½	0	0	0	0	½	½	0	0	0	0	½	0	0	0	1	0	0	0	1	0	0	0
$(M_PM_Q)\times(M_QM_P)$	53	½	½	0	0	0	0	½	½	0	0	0	0	½	0	0	0	1	0	0	0	1	0	0	0
$(W_PW_Q)\times(W_QW_P)$	55	0	0	½	½	0	0	0	0	½	½	0	0	0	½	0	0	0	1	0	0	0	1	0	0
$(C_PC_Q)\times(C_QC_P)$	52	0	0	0	0	½	½	0	0	0	0	½	½	0	0	½	0	0	0	1	0	0	0	1	0
$(M_PW_P)\times(W_PM_P)$	54	½	0	½	0	0	0	½	0	½	0	0	0	0	0	0	½	0	0	0	1	0	0	0	1
$M_P\times(M_QM_P)$	53	¾	¼	0	0	0	0	½	½	0	0	0	0	½	0	0	0	1	0	0	0	0	0	0	0
$M_Q\times(M_PM_Q)$	52	¼	¾	0	0	0	0	½	½	0	0	0	0	½	0	0	0	1	0	0	0	0	0	0	0
$W_Q\times(W_PW_Q)$	52	0	0	¼	¾	0	0	0	0	½	½	0	0	0	½	0	0	0	1	0	0	0	0	0	0
$C_Q\times(C_PC_Q)$	52	0	0	0	0	¼	¾	0	0	0	0	½	½	0	0	½	0	0	0	1	0	0	0	0	0
$W_P\times(M_PW_P)$	49	¼	0	¾	0	0	0	½	0	½	0	0	0	0	0	0	½	0	0	0	1	0	0	0	0
$M_PM_Q\times(M_QM_P\times M_PM_Q)$	54	½	½	0	0	0	0	½	½	0	0	0	0	½	0	0	0	½	0	0	0	1	0	0	0
$M_PM_Q\times(M_PM_Q\times M_QM_P)$	53	½	½	0	0	0	0	½	½	0	0	0	0	½	0	0	0	½	0	0	0	1	0	0	0
$W_QW_P\times(W_PW_Q\times W_QW_P)$	53	0	0	½	½	0	0	0	0	½	½	0	0	0	½	0	0	0	½	0	0	0	1	0	0
$C_QC_P\times(C_PC_Q\times C_QC_P)$	55	0	0	0	0	½	½	0	0	0	0	½	½	0	0	½	0	0	0	½	0	0	0	1	0
$W_PM_P\times(M_PW_P\times W_PM_P)$	49	½	0	½	0	0	0	½	0	½	0	0	0	0	0	0	½	0	0	0	½	0	0	0	1

Table 3. F-tests and residual mean squares for lifetime performance traits

Effect	d.f.	Total no. of young born alive	Total wt. of young at birth	Total no. of young at day 18	Total wt. of young at day 18	No. of parturitions
Generation	2	1.5	0.9	3.3*	1.9	3.1*
Line additive:						
M_P	1	3.5	5.2*	3.0	5.3*	0.9
M_Q	1	11.6**	10.5**	8.4**	6.9**	6.3*
W_P	1	7.7**	6.7**	5.9*	5.2*	2.9
W_Q	1	4.8*	3.4	3.5	1.6	4.0*
C_P	1	10.5**	12.2**	11.6**	19.9**	1.5
Line maternal:						
M_P	1	2.2	2.8	1.7	3.9*	2.1
M_Q	1	4.1*	4.8*	3.2	5.1*	2.0
W_P	1	1.4	1.4	0.8	1.0	0.9
W_Q	1	1.9	1.5	1.2	0.5	1.0
C_P	1	1.2	0.8	0.6	2.4	1.3
Direct heterosis:						
M_P and M_Q	1	59.4**	61.7**	69.5**	89.6**	21.8**
W_P and W_Q	1	33.7**	37.0**	34.3**	42.6**	12.6**
C_P and C_Q	1	30.7**	31.1**	43.6**	57.5**	12.0**
M_P and W_P	1	1.0	0.8	1.2	2.1	0.0
Maternal heterosis:						
M_P and M_Q	1	0.0	0.0	0.1	0.1	0.1
W_P and W_Q	1	5.9*	6.3*	4.8*	5.6*	3.3
C_P and C_Q	1	6.4*	5.4*	5.2*	5.2*	0.9
M_P and W_P	1	2.4	2.0	1.7	4.0*	4.8*
Paternal heterosis:						
M_P and M_Q	1	1.2	2.2	1.5	4.8*	1.3
W_P and W_Q	1	8.9**	11.0**	9.6**	14.2**	11.1**
C_P and C_Q	1	5.1*	6.7**	6.6**	10.7**	4.3*
M_P and W_P	1	2.3	1.9	1.6	5.1*	9.2**
Residual	2,432	354.1	1,003.8	345.0	39,785.8	2.6

* Significant at $P<0.05$; ** Significant at $P < 0.01$

as defined in the model refer to the sire and dam of the mother. Since males (as mates) of the same line were mated with females of different genetic groups, it was assumed that lifetime performance traits examined were basically female-oriented traits.

Since the proportions of gene contributions for either line additive or line maternal effects sum to one, there are dependencies among equations corresponding to line additive effects and among equations corresponding to line maternal effects. For this reason, line additive and line maternal effects for Line C_Q were set to zeros in obtaining the least squares solutions. This means that Line C_Q was used as a basis for comparison. As a consequence, all line additive and line maternal effects were expressed as deviations from the corresponding effects of Line C_Q.

In addition to the multiple regression analysis, the data were analyzed by a genetic group model which consisted of time period and genetic group effects. The difference in reduction in sums of squares between fitting genetic group model and multiple regression model provides a test for goodness of fit of the multiple regression model.

Results and discussion

Analyses of variance are shown in Table 3. Partial regression coefficients and standard errors are in Table 4. It should be noted that line additive effect in this paper refers to the additive effect of the individual's genotype (i.e., additive direct genetic effect) whereas line maternal effect is the additive maternal genetic effect. In this study, the decimal fractions of gene contributions rather than the percentage of gene contribution were used as independent variables in regression. Line additive and line maternal partial regression coefficients (Table 5) indicate the corresponding changes due to complete replacement of C_Q genes by genes of the other lines. The heterosis partial regression coefficients represent the amount of heterotic effects due to complete heterozygosity for each two-line combination.

Table 4. Partial regression coefficients and standard errors for genetic effects

Genetic effects	Total no. of young born alive	Total wt. of young at birth	Total no. of young at day 18	Total wt. of young at day 18	No. of parturitions
Line additive:					
M_P	−14.3±7.6	−29.3±12.8	−13.2±7.6	−185.5±80.6	−0.6±0.6
M_Q	−22.3±6.6	−35.8±11.0	−18.9±6.5	−182.7±69.5	−1.4±0.6
W_P	−23.0±8.3	−36.0±13.9	−20.0±8.2	−200.2±87.5	−1.2±0.7
W_Q	−20.0±9.0	−27.7±15.2	−16.6±8.9	−119.4±95.4	−1.5±0.8
C_P	−14.2±4.4	−25.9± 7.4	−14.9±4.4	−208.0±46.6	−0.5±0.4
Line maternal:					
M_P	11.1±7.4	20.9±12.5	9.6±7.4	154.5±78.7	0.9±0.6
M_Q	12.7±6.3	23.1±10.5	11.2±6.2	149.7±66.4	0.7±0.5
W_P	9.4±8.1	16.0±13.6	6.9±8.0	84.6±85.3	0.7±0.7
W_Q	12.3±8.8	17.9±14.8	9.6±8.8	63.5±93.4	0.8±0.8
C_P	4.1±3.8	5.6± 6.5	2.9±3.8	63.0±40.7	0.4±0.3
Direct heterosis:					
M_P and M_Q	14.1±1.8	24.1± 3.1	15.1±1.8	182.8±19.3	0.7±0.2
W_P and W_Q	12.3±2.1	21.7± 3.6	12.3±2.1	146.3±22.4	0.6±0.2
C_P and C_Q	11.6±2.1	19.7± 3.5	13.8±2.1	168.7±22.2	0.6±0.2
M_P and W_P	2.2±2.1	3.1± 3.6	2.3±2.1	32.8±22.5	0.0±0.2
Maternal heterosis:					
M_P and M_Q	0.1±2.0	0.5± 3.4	0.6±2.0	−2.4±21.1	−0.1±0.2
W_P and W_Q	−6.9±2.8	−12.0± 4.8	−6.2±2.8	−71.5±30.1	−0.4±0.2
C_P and C_Q	−7.2±2.8	−11.1± 4.8	−6.5±2.8	−68.4±30.2	−0.2±0.2
M_P and W_P	−4.5±2.9	−6.9± 4.9	−3.7±2.9	−61.4±30.8	−0.5±0.2
Paternal heterosis:					
M_P and M_Q	2.1±1.9	4.6± 3.2	2.3±1.9	43.4±19.9	0.2±0.2
W_P and W_Q	8.1±2.7	15.1± 4.6	8.3±2.7	108.5±28.8	0.8±0.2
C_P and C_Q	6.1±2.7	11.8± 4.5	6.9±2.7	93.2±28.6	0.5±0.2
M_P and W_P	4.2±2.8	6.6± 4.7	3.5±2.8	66.7±29.6	0.7±0.2

Table 5. Comparison of reductions in sums squares due to fitting multiple regression and genetic group models

	Reductions in sums of squares due to fitting		Error mean square for genetic group model	F-test[a]
	Multiple regression model	Genetic group model		
Degrees of freedom	24	30	2,426	
Total no. of young born alive	99,254	101,357	354	1.0
Total wt. of young at birth	271,016	278,854	1,003	1.3
Total no. of young at day 18	114,039	117,091	350	1.4
Total wt. of young at day 18	16,590,809	16,961,958	39,731	1.6
No. of parturitions	515	532	3	0.9

[a] Numerator of F-test = (Difference in reduction SS between genetic group and multiple regression models) divided by 6
Denominator of F-test = Error mean square for genetic group model

Line additive effects (additive direct genetic effects)

Of 25 line additive effects, seventeen differed significantly from C_Q (Table 3). The partial regression coefficients for line additive effects were all negative (Table 4). This indicates that replacement of Line C_Q genes by the genes of any other lines (M_P, M_Q, W_P, W_Q or C_P) would decrease the line additive effects for the five lifetime traits studied. As shown in Table 4, the decrease in line additive effects ranged from 14.3 to 23.0 for total number of young born alive, from 25.9 to 36.0 g for total weight of young at birth, from 13 to 20 for total number of young at day 18, from 119.4 to 208.0 g for total weight of young at day 18, and from 0.5 to 1.5 for number of parturitions. Among the six lines studied, W_P had the smallest line additive effects

for total number of young born alive, total weight of young at birth and total number of young at day 18 whereas C_P showed the lowest line additive effect for total weight of young at day 18.

Line maternal effects (additive maternal genetic effects)

Of 25 line maternal effects, only four showed significant deviations from the C_Q line (Table 3). The partial regression coefficients for line maternal effects were all positive (Table 4), suggesting that replacement of C_Q genes by the genes of other lines studied would improve line maternal effects. That is, Line C_Q had the poorest maternal effect. The absolute values of partial regression coefficients for the line additive effects are generally larger than those for the maternal effects (Table 4). This suggests that line maternal effects should not be expected to contribute more variation to the cross data than the line additive effects. Similar results have been reported in dairy heifers (Lin et al. 1984).

Line maternal effects for all traits in Lines M_P and M_Q were generally of greater magnitude than in Lines W_P, W_Q, C_P and C_Q (Table 4). This appears to be due to selection for increased maternal performance in M lines. Notably, maternal effects for total weight of young at day 18 in Lines M_P and M_Q were about twice as large as in Lines W_P, W_Q, C_P or C_Q (Table 4). Maternal effects in Lines W_P and W_Q in turn were greater than those in Lines C_P and C_Q. Selection for increased adult body weight in Lines W_P and W_Q appears to have increased maternal effects of the five lifetime traits as a correlated response.

All lines examined deviated negatively from C_Q in line additive effects for the lifetime traits but deviated positively from C_Q in line maternal effects (Table 4). These results indicate that there was a negative relationship between line additive and maternal effects for the lifetime traits studied. Lines with positive line additive effects consistently showed negative maternal effects.

The presence of a negative relationship between additive and maternal genetic effects has been reported in beef cattle (Kress et al. 1979; Bailey 1981) and in swine (Jungst and Kuhlers 1984). This antagonistic relationship constitutes a genetic barrier to animal improvement since selection gain in additive effects adversely affects maternal effects. However, line additive effects are usually large enough to overcome deterioration of line maternal effects. Restricted index selection might be used to improve line additive effects while holding line maternal effects constant. Maximum genetic response could be achieved through index selection for direct and maternal genetic effects weighted by their net economic values (Van Vleck 1970).

Direct heterosis effects

Direct heterosis effects of $M_P \times M_Q$, $W_P \times W_Q$ and $C_P \times C_Q$ crosses were significant ($P < 0.01$) for the lifetime traits studied (Table 3). However, direct heterosis effects for $M_P \times W_P$ cross were nonsignificant for all traits. This may be because Lines M_P and W_P originated from the same population, P. Heterosis effects are expected to arise from crossing of two lines with diverse genetic backgrounds. Partial regression coefficients for direct heterosis effects were all positive (Table 4) which is desirable for improvement of lifetime performance.

Direct heterosis effects are not necessarily positive as reported for dairy heifer reproduction traits (Lin et al. 1984). Negative heterosis has been reported for weaning rate of beef cattle (Peacock et al. 1977; Bailey 1981) and for some litter traits of swine (Jungst and Kuhlers 1984).

Of the four combinations of direct heterosis effects ($M_P \times M_Q$, $W_P \times W_Q$, $C_P \times C_Q$ and $M_P \times W_P$), $M_P \times M_Q$ showed the greatest heterotic effects for all traits (Table 4). The $M_P \times M_Q$ heterotic effect would increase total number of young born alive by 14.1, total weight of young at birth by 24.1 g, total number of young at day 18 by 15.1, total weight of young at day 18 by 182.8 g, and number of parturitions by 0.7. Line additive and direct heterosis effects (except for $M_P \times W_P$ heterosis) were generally greater (in absolute values) than line maternal effects (Table 3), suggesting that line additive and direct heterosis effects are a more important source of genetic variation in lifetime performance traits than line maternal effects as was also found for dairy heifer reproduction traits (Lin et al. 1984). The results imply that in animal production, more attention should be given to improvement of additive effects and exploitation of direct heterosis effect than to use of maternal effects.

Maternal heterosis effects

Maternal heterosis effects for $W_P \times W_Q$ and $C_P \times C_Q$ crosses were significant ($P < 0.05$) for all traits except number of parturitions (Table 3). Maternal heterosis for $M_P \times M_Q$ cross was nonsignificant for all traits whereas maternal heterosis for $M_P \times W_P$ cross was significant ($P < 0.05$) for total weight of young at day 18 and number of parturitions. As shown in Table 4, 17 of 20 maternal heterosis effects were negative, suggesting that crossline dams would contribute negative heterotic maternal effects to the performance of their progeny in comparison with pureline dams.

Negative maternal heterosis was reported for ribeye area in beef cattle (Alenda et al. 1980). Neville et al. (1984) examined the average maternal heterosis for reproductive, preweaning, postweaning and carcass traits in beef cattle and found that average maternal heterosis effects could be negative or positive depending upon the traits. Jungst and Kuhlers (1984) found the average maternal heterosis effect to be significantly positive for some litter traits of swine. Whenever the average maternal heterosis effect is found to be significant, the average maternal heterosis should be further partitioned into specific maternal heterosis due to different combinations of lines (breeds or strains) as was done in this study so that the

most favorable line combination for maternal heterosis could be identified.

Paternal heterosis effects

Paternal heterosis effects were found to be significant in 13 out of 20 cases (Table 3). In contrast to maternal heterosis effect, paternal heterosis effects were all positive and favorable for lifetime performance (Table 4). The $W_P \times W_Q$ or $C_P \times C_Q$ combinations in the sires showed greater paternal heterosis than $M_P \times M_Q$ or $M_P \times W_P$ whereas the reverse was true for maternal heterosis. No research on paternal heterosis seems to have been reported in the literature. Of 2,457 lifetime performance records, 532 with crossbred sires contained information for estimation of paternal heterosis (Table 2). These 532 females were all mated with crossbred males. Therefore, paternal heterosis effects and the effects of using crossbred males as mates were confounded. Nagai et al. (1984) reported that females showed superior lifetime performance when pair-mated with F_1 cross males than when pair-mated with purebred males. This is probably the reason why paternal heterosis effects (Table 3) were all positive relative to maternal heterosis effects. Nevertheless, both paternal and maternal heterosis effects were generally much smaller than direct heterosis effects with the exception of direct heterosis for $M_P \times W_P$ cross (Table 4). This indicates that heterozygosity of a female is more important for the exhibition of heterosis than that of her sire or dam because the genetic makeup of a female has greater impact on her own performance than that of her ancestors. The more remote the ancestors are, the less impact their heterozygosities are likely to have on the offspring's lifetime performance.

Goodness of fitting multiple regression model

A model that fitted constants for time period and genetic group effects was applied to the data (genetic group analysis). Reduction in sums of squares due to fitting the genetic group model should account for all (additive and non-additive) genetic effects whereas multiple regression model used in this study should account for additive, maternal, direct heterosis and parental heterosis effects. Reduction in sums of squares due to fitting these two models are compared in Table 5. Differences in reductions are non-significant, suggesting that fitting the multiple regression model for additive, maternal, direct heterosis, maternal heterosis and paternal heterosis effects effectively accounted for all genetic components.

Conclusions

Information on lifetime performance is important for improving animal production but data are usually lacking. The present study on lifetime performances of various genetic groups in mice suggests that line additive and direct heterosis effects are a more important source of genetic variation in lifetime performance than line maternal or parental heterosis effects. In animal production, more attention should be given to improvement of line additive effects and exploitation of direct heterosis effects than to use of line maternal or parental heterosis effects. Lines with positive line additive effects consistently showed negative line maternal effects, presenting a genetic barrier to animal improvement. Fitting the multiple regression model for line additive, line maternal, direct heterosis and parental heterosis effects effectively accounted for all genetic effects in lifetime performance.

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