

Additive, dominance, and epistatic loss effects on preweaning weight gain of crossbred beef cattle from different *Bos taurus* breeds¹

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ABSTRACT: (Co)variance components, direct and maternal breed additive, dominance, and epistatic loss effects on preweaning weight gain of beef cattle were estimated. Data were from 478,466 animals in Ontario, Canada, from 1986 to 1999, including records of both purebred and crossbred animals from Angus, Blonde d'Aquitaine, Charolais, Gelbvieh, Hereford, Limousin, Maine-Anjou, Salers, Shorthorn, and Simmental breeds. The genetic model included fixed direct and maternal breed additive, dominance, and epistatic loss effects, fixed environmental effects of age of the calf, contemporary group, and age of the dam \times sex of the calf, random additive direct and maternal genetic effects, and random maternal permanent environment effects. Estimates of direct and maternal additive genetic, maternal permanent environmental and residual variances, expressed as proportions of the phenotypic

variance, were 0.32, 0.20, 0.12, and 0.52, respectively. Correlation between direct and maternal additive genetic effects was -0.63 . Breed ranking was similar to previous studies, but estimates showed large SE. The favorable effects of direct and maternal dominance ($P < 0.05$) on preweaning gain were equivalent to 1.3 and 2.3% of the phenotypic mean of purebred calves, respectively. The same features for direct and maternal epistatic loss effects were -2.2% ($P < 0.05$) and -0.1% ($P > 0.05$). The large SE of breed effects were likely due to multicollinearity among predictor variables and deficiencies in the dataset to separate direct and maternal effects and may result in a less reliable ranking of the animals for across breed comparisons. Further research to identify the causes of the instability of estimates of breed additive, dominance, and epistatic loss genetic effects, and application of alternative statistical methods is recommended.

Key Words: Beef Cattle, Epistatic Loss, Genetic Parameters, Heterosis

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Introduction

In beef cattle improvement programs, dominance effects associated with breed heterozygosity are generally considered in the estimation of breeding values of crossbred animals through additive-dominance models, which account for additive and heterotic effects. These models have been used in large beef cattle populations in Canada (Sullivan et al., 1999), Brazil (Roso and Fries,

1998), Australia (Johnston et al., 1999), and the United States (Pollak and Quaas, 1998; Klei et al., 2002).

Justification for additive-dominance models is based on the assumption that heterosis is mainly due to dominance effects. According to Gregory et al. (1991), heterosis observed for growth traits in beef cattle is likely due to dominance effects and represents the recovery of accumulated inbreeding depression within populations that have been genetically isolated from each other for many generations. Research by Gregory et al. (1991) suggested that retention of heterosis is linearly proportional to heterozygosity. A similar relationship between heterosis and heterozygosity was observed by Arthur et al. (1999) and Fries et al. (2000). In these two studies, however, authors suggested that another component, epistatic loss, could be added to the additive-dominance model to provide a better explanation of the genetic differences between animals of different breed compositions.

Epistatic loss in crossbred animals represents the breakdown of favorable interactions between loci in purebred animals, which have been built by both natu-

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ral and artificial selection within breeds (Koch et al., 1985). Research studies to estimate both dominance and epistatic loss in beef cattle are not abundant, particularly with field data.

The objectives of this study were to estimate (co)variance components and direct and maternal breed additive, dominance, and epistatic loss effects on preweaning weight gain in a large multibreed beef cattle population.

Materials and Methods

Data

The data used in this study were preweaning weight gain of animals from beef herds enrolled by Beef Improvement Ontario, from 1986 to 1999. The dataset after preliminary edits consisted of 869,050 records, including records of both purebred and crossbred animals. Purebred and crossbred animals from the 10 breeds with the largest number of records, including Angus, Blonde d'Aquitaine, Charolais, Gelbvieh, Hereford, Limousin, Maine-Anjou, Salers, Shorthorn, and Simmental, were used in the analysis. Only records of animals with complete information for calculating direct and maternal dominance and epistatic loss were kept.

Connectedness Analysis

An analysis to check for connectedness among contemporary groups (herd-year-season-management group) across breeds was performed. The method used was the total number of direct genetic links between contemporary groups due to common sires and dams (Fries, 1998; Roso et al., 2004). Contemporary groups with more than 10 calves and with at least 10 direct genetic links and two classes of direct or maternal heterozygosities were considered connected and retained for the analysis. There were nine classes of direct and maternal heterozygosities with an interval of 0.125, ranging from 0 to 1. The resulting dataset included 23,059 contemporary groups, 478,466 calves, 19,908 sires, and 234,608 dams. A pedigree file of 714,220 animals was used in the analysis.

Predictor Variables of Fixed Genetic Effects

Breed Additive Effects. Coefficients for direct and maternal breed additive effects were equal to the proportion of each breed in the breed composition of the calf and in the breed composition of the dam (Rodríguez-Almeida et al., 1997), respectively. The estimates of direct and maternal breed additive effects were expressed as differences relative to Angus.

Breed compositions of the animals are depicted in Figures 1 and 2. Figure 1 shows that less than 40% of the calves were purebred, indicating the degree of crossbreeding in the dataset. Most crossbred calves

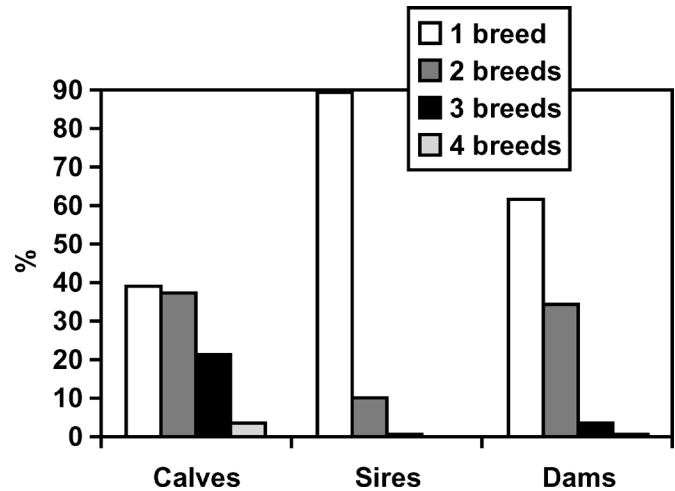


Figure 1. Percentage of calves, sires, and dams with breed composition formed by either one, two, three, or four breeds in the data containing 478,466 calves, 19,908 sires, and 234,608 dams.

originated from various two-breed crosses. In contrast, most sires (89.3%) and dams (61.3%) were purebred. Figure 2 shows that breeding practices in the beef herds studied resulted in an unbalanced number of animals among breeds. There were substantially more Angus, Charolais, Hereford, Limousin, and Simmental calves, sires, and dams than Blonde d'Aquitaine, Gelbvieh, Maine-Anjou, Salers, and Shorthorn.

Dominance Effects. Coefficients of direct (H_D) and maternal (H_M) dominance effects were equal to expected direct and maternal breed heterozygosities (Rodríguez-Almeida et al., 1997), respectively. Coefficients H_D and H_M were calculated using the following equations:

$$H_D = 1 - \sum_{i=1}^{nb} S_i \times D_i$$

and

$$H_M = 1 - \sum_{i=1}^{nb} MGS_i \times MGD_i;$$

where nb is the number of breeds, and S_i , D_i , MGS_i , and MGD_i are the fractions of the i th breed for the sire, dam, maternal grandsire, and maternal granddam breed composition, respectively.

Epistatic Loss Effects. For the estimation of epistatic loss effects, it was assumed that parents with large heterozygosities would produce more recombinant gametes. Thus, the coefficients for direct (E_D) and maternal (E_M) epistatic loss effects were calculated as the average breed heterozygosities in uniting gametes that generated the individual (Fries et al., 2000). Epistatic loss was assumed to be proportional to the average heterozygosity observed in parents, and it would have the largest value when both parents of an individual are F_1 . The E_D and E_M were calculated as follows:

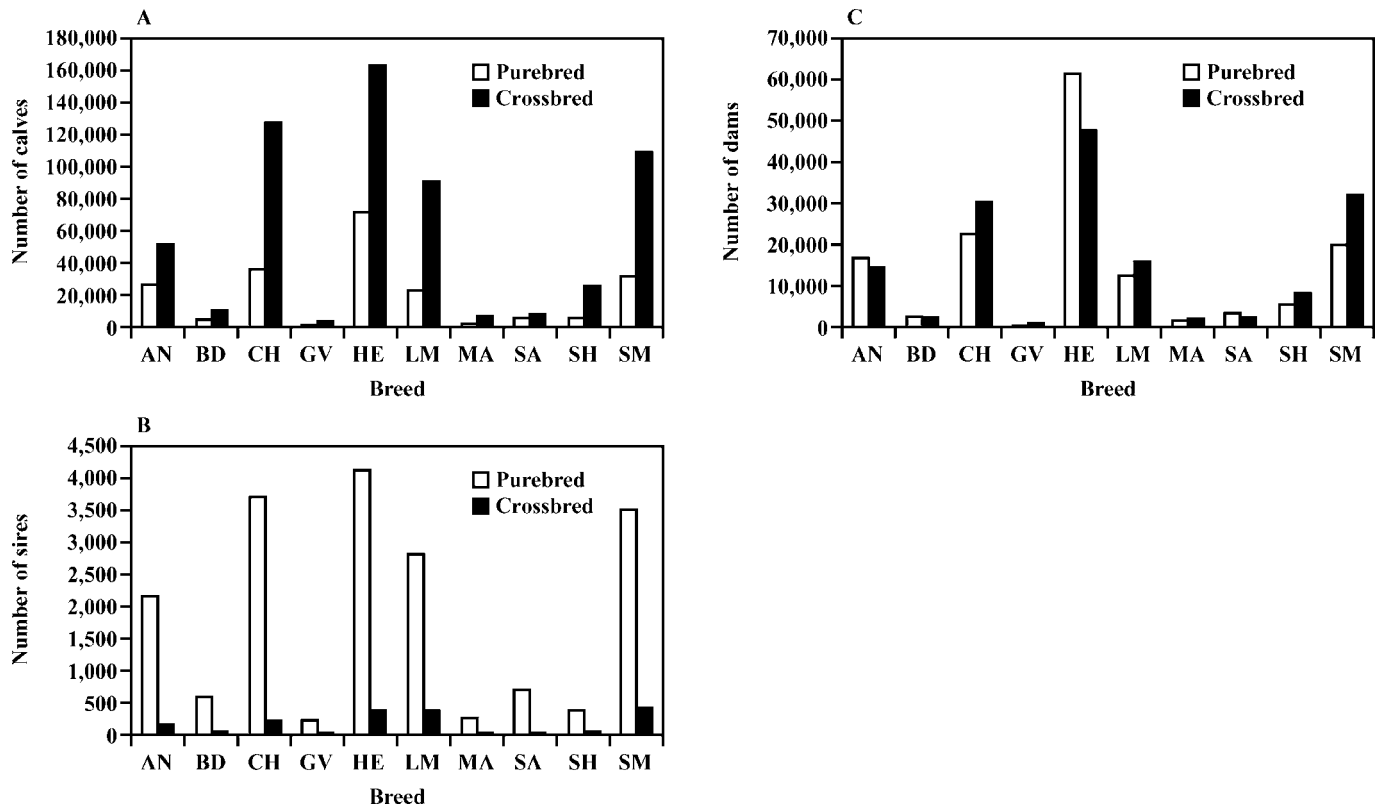


Figure 2. Number of purebred and crossbred calves, sires, and dams containing some proportion of the indicated breed in the breed composition in data including 478,466 calves, 19,908 sires, and 234,608 dams. The breeds are Angus (AN), Blonde d'Aquitaine (BD), Charolais (CH), Gelbvieh (GV), Hereford (HE), Limousin (LM), Maine-Anjou (MA), Salers (SA), Shorthorn (SH), and Simmental (SM).

$$E_D = 0.5 (H_{Sire} + H_{Dam})$$

$$E_M = 0.5 (H_{MGS} + H_{MGD})$$

where H_{Sire} , H_{Dam} , H_{MGS} , and H_{MGD} are the expected breed heterozygosities of the sire, dam, maternal grand-sire, and maternal granddam, respectively. The average epistatic loss due to the breakdown of all kinds of gene interactions involving two or more loci, as deviation from the average additive and dominance effects, will be estimated by E_D and E_M (Fries et al., 2002). For illustration, Table 1 shows coefficients of direct and maternal dominance and epistatic loss genetic effects for different mating systems involving only two breeds, A and B.

The distribution of observations among coefficients of dominance and epistatic loss effects is presented in Table 2. For ease of presentation, coefficients of dominance and epistatic loss effects were grouped in classes of 0.125, ranging from 0 to 1. Numbers in Table 2 suggest that there were more observations for classes of coefficients greater than 0.625 for direct and maternal dominance compared with epistatic loss effects. Because approximately 10% of the sires were crossbred (Figure 1), there were relatively few observations in the classes of coefficients of epistatic loss effects larger than 0.625. The observed overall mean and SD of pre-

weaning gain and weaning age were 203.5 ± 49.4 kg and 204.0 ± 30.9 d, respectively.

Genetic Analysis

The genetic model for preweaning gain, defined in matrix notation, was as follows:

$$\mathbf{y} = \mathbf{Xb} + \mathbf{Fv} + \mathbf{Za} + \mathbf{Wm} + \mathbf{Sp} + \mathbf{e}$$

where \mathbf{y} = vector of observations; \mathbf{b} = vector of fixed regressions on coefficients for direct and maternal breed additive, dominance, and epistatic loss genetic effects; \mathbf{v} = vector of fixed environmental effects. This vector included age of the calf as a covariate (linear and quadratic effects), and age of the dam by sex of the calf and contemporary group (herd-year-season-management group) as classification variables; \mathbf{a} = vector of random direct additive genetic effects; \mathbf{m} = vector of random maternal additive genetic effects; \mathbf{p} = vector of random maternal permanent environmental effects; and \mathbf{e} = vector of random residual effects.

Incidence matrices \mathbf{X} , \mathbf{F} , \mathbf{Z} , \mathbf{W} , and \mathbf{S} relate records to fixed genetic, fixed environmental, direct genetic, maternal genetic, and permanent environmental effects, respectively.

Table 1. Coefficients of direct (H_D) and maternal (H_M) dominance and direct (E_D) and maternal (E_M) epistatic loss genetic effects for different mating systems involving two breeds, A and B

Sire	Dam	f_A^a	H_D	H_M	E_D	E_M
Parental						
A	A	1	0	0	0	0
B	B	0	0	0	0	0
F ₁						
A	B	½	1	0	0	0
B	A	½	1	0	0	0
Backcrosses						
A	AB	¾	½	1	½	0
B	AB	¼	½	1	½	0
AB	B	¼	½	0	½	0
AB	A	¾	½	0	½	0
Advanced generations						
F ₁	F ₁	½	½	1	1	0
F ₂	F ₂	½	½	½	½	1
F ₃	F ₃	½	½	½	½	½

^aFraction of breed A in the breed composition of the animal.

The vectors of random effects \mathbf{a} , \mathbf{m} , \mathbf{p} , and \mathbf{e} were assumed to have (co)variance matrices equal to $\mathbf{A}\sigma_a^2$, $\mathbf{A}\sigma_m^2$, $\mathbf{I}\sigma_p^2$, and $\mathbf{I}\sigma_e^2$, respectively, where \mathbf{A} is the additive numerator relationship matrix among animals and \mathbf{I} is an identity matrix. Covariance between \mathbf{a} and \mathbf{m} was assumed equal to $\mathbf{A}\sigma_{am}$. Homogeneity of variances and the same dominance and epistatic loss effects for crosses of different pairs of breeds, and no interactions between genetic and environmental effects were assumed.

Estimates of (co)variance components (σ_a^2 , σ_m^2 , σ_p^2 , σ_e^2 , and σ_{am}) and estimates of the effects included in the model were obtained using the DMU program (Madsen and Jensen, 2000). First, (co)variance components were estimated by the REML method, using a data subset containing 300,002 records from randomly sampled herds, which overcame computational limitations. Given the estimated (co)variance components, the estimates of the effects in the model were obtained using the complete dataset.

Results and Discussion

(Co)variance Components

Estimates of direct additive genetic variance, maternal additive genetic variance, maternal permanent environmental variance, residual variance, and direct by maternal additive genetic covariance of preweaning gain are presented in Table 3. For ease of interpretation, variances were expressed as proportions of phenotypic variance. Estimates of h_a^2 , h_m^2 , p^2 , and e^2 were compared with estimates from previous studies of Miller (1996) in Ontario and with average results reported by Koots et al. (1994a) in a review of a large number of published estimates of genetic parameters. Estimates of h_a^2 , h_m^2 , p^2 , and e^2 were 0.32, 0.20, 0.12, and 0.52, respectively. Estimates of h_a^2 and h_m^2 were in line with pooled estimates of 0.27 and 0.23, respectively, reported by Koots et al. (1994a). The estimate of h_a^2 did

Table 2. Distribution of observations among coefficients of direct (H_D) and maternal (H_M) dominance and direct (E_D) and maternal (E_M) epistatic loss genetic effects

Class ^a	H_D	H_M	E_D	E_M
0.000	184,115 (38.5) ^b	285,368 (59.6)	261,239 (54.6)	413,347 (86.4)
0.125	18,825 (3.9)	9,159 (1.9)	32,202 (6.7)	10,324 (2.2)
0.250	15,414 (3.2)	9,476 (2.0)	29,755 (6.2)	11,710 (2.5)
0.375	3,322 (0.7)	759 (0.2)	10,764 (2.3)	1,563 (0.3)
0.500	62,701 (13.1)	27,320 (5.7)	119,984 (25.1)	33,332 (7.0)
0.625	8,622 (1.8)	5,568 (1.2)	15,871 (3.3)	6,137 (1.3)
0.750	6,722 (1.4)	2,193 (0.5)	4,184 (0.9)	1,647 (0.3)
0.875	2,630 (0.6)	352 (0.1)	434 (0.1)	10 (0.0)
1.000	176,115 (36.8)	138,271 (28.9)	4,033 (0.8)	396 (0.1)

^aCoefficients of dominance and epistatic loss effects were grouped in classes of 0.125, ranging from zero to one. Every class included fractions equal or smaller than the mentioned class.

^bPercents in parentheses were expressed relative to the total number of calves.

Table 3. Estimates of variance components and genetic parameters of preweaning weight gain

(Co)variance component ^a	Estimate, kg ²	Parameter ^b	Estimate ^c
σ_a^2	254.5 ± 8.6	h_a^2	0.32 ± 0.00
σ_m^2	161.2 ± 9.3	h_m^2	0.20 ± 0.00
σ_p^2	94.1 ± 5.4	p^2	0.12 ± 0.00
σ_e^2	408.2 ± 4.8	e^2	0.52 ± 0.00
σ_{am}	-128.6 ± 7.8	r_{am}	-0.63 ± 0.02

^a σ_a^2 = direct additive genetic variance; σ_m^2 = maternal additive genetic variance; σ_p^2 = maternal permanent environmental variance; σ_e^2 = residual variance; and σ_{am} = direct by maternal additive genetic covariance.

^bVariance component as a proportion of phenotypic variance. For σ_{am} , genetic correlation (r_{am}) is shown.

^cStandard errors for variance ratios were approximated by first-order Taylor series expansion (Mood et al., 1974).

not differ greatly from that of Sullivan et al. (1999), where a h_a^2 of 0.30 was used in the estimation of genetic trends and mean genetic differences among breeds in Ontario. Estimates of h_a^2 , h_m^2 , and p^2 were lower than the estimates of 0.44, 0.25, and 0.15, respectively, obtained by Miller (1996); however, this author analyzed a smaller data set from the same population of cattle used in the current study.

The correlation between direct and maternal genetic effects on preweaning gain ($r_{am} = -0.63$), although lower in absolute value than the estimate of -0.77 obtained by Miller (1996), was still strongly negative. This result is in marked contrast with an average estimate of -0.25 reported by Koots et al. (1994b), and larger than estimates of Meyer (1992) and Robinson (1996), where average values of -0.59 and -0.47 were reported. One possible factor contributing to the strong negative genetic correlation is the small proportion of female calves with records that later had their own progeny. There were only 23,508 cases where a female calf later became a cow, corresponding to approximately 10% of all female calves in the dataset.

Dominance and Epistatic Loss Effects

Estimates of direct and maternal dominance and epistatic loss effects on preweaning gain associated with breed heterozygosity are presented in Table 4. For both

Table 4. Estimates and SE of direct and maternal dominance (H) and epistatic loss (E) genetic effects on preweaning weight gain

Genetic effect	Direct	Maternal
H, kg	2.7 ± 0.2 (1.3%) ^a	4.6 ± 0.8 (2.3%)
E, kg	-4.5 ± 1.6 (-2.2%)	-0.2 ± 0.4 (-0.1%)

^aPercents in parentheses were expressed relative to phenotypic mean of purebred calves.

dominance and epistatic loss effects, the magnitude of the estimates was small. Expressed relative to the phenotypic mean of purebred calves, direct and maternal dominance effects were 1.3 and 2.3% ($P < 0.05$), respectively. Direct and maternal epistatic loss effects were -2.2 ($P < 0.05$) and -0.1% ($P = 0.41$), respectively. Estimates of maternal dominance and direct epistatic loss effects were of comparable magnitude, although opposite in sign. Standard errors of maternal dominance and direct epistatic loss effects were large compared with SE of direct dominance and maternal epistatic loss effects.

Both direct and maternal epistatic loss effects had the anticipated negative effect on preweaning gain. However, epistatic effects for a specific trait may be either favorable or unfavorable, depending on the selection history of the population and genetic correlations among traits. Favorable epistatic effects may result from direct selection for a particular trait, whereas unfavorable effects may result from correlated response of traits with antagonistic genetic correlation (Cassady et al., 2002). Crossing different breeds may break favorable epistatic relationships established by direct selection for a trait within breeds, resulting in epistatic loss.

The maternal epistatic loss effect was not statistically different from zero, probably reflecting the deficiency in the structure of data to estimate this genetic effect, as shown in Table 2. To detect a significant effect, a larger proportion of crossbred sires is likely required. The small proportion of crossbred sires (and grandsires) in the dataset had two consequences. First, it decreased the expression of epistatic loss because at least one allele at each locus would be from a parental breed in a large proportion of the crossbred progeny, which decreased the breakdown of favorable interactions established in the pure breeds (Kinghorn, 1983). Second, it increased the dependency between dominance and epistatic effects, causing collinearity between these two genetic effects.

According to results obtained by Gregory et al. (1991) in a large beef cattle crossbreeding experiment, the heterosis observed for growth traits in beef cattle is likely due to dominance effects. This observation allows fitting heterosis as being proportional to the probability that alleles at a locus come from different breeds, which is equal to breed heterozygosity. Additional analyses fitting only dominance effects in the model (excluding epistatic loss effects) resulted in estimates of direct and maternal dominance of 1.3 and 1.8%, respectively. Therefore, estimates of dominance effects from both models did not differ greatly. These results also were in close agreement with those of Miller (1996), who reported estimates of direct and maternal heterosis of 1.3 and 2.3%, respectively, assuming a dominance model.

Koch et al. (1985) evaluated dominance and epistatic loss effects on weaning gain of Angus × Hereford crosses. In their study, direct dominance and epistatic loss effects were not significant, despite the relatively

Table 5. Estimates and SE of direct and maternal breed additive genetic effects for preweaning weight gain, expressed as deviations from Angus

Breed	Direct, kg	Maternal, kg
Angus	0.0 ± 3.7	0.0 ± 1.9
Blonde d'Aquitaine	5.3 ± 4.1	-5.7 ± 2.3
Charolais	13.2 ± 3.6	-2.9 ± 1.8
Gelbvieh	10.4 ± 4.7	7.9 ± 3.1
Hereford	-6.3 ± 3.6	-3.2 ± 1.9
Limousin	-3.1 ± 3.7	0.6 ± 1.9
Maine-Anjou	12.3 ± 4.6	0.3 ± 2.5
Salers	0.6 ± 4.3	7.6 ± 2.5
Shorthorn	-9.3 ± 4.3	4.6 ± 2.2
Simmental	14.2 ± 3.6	5.3 ± 1.9

large negative values. They stated that more data and a more complete array of mating types would be needed to attain statistically significant results. In a review of a large number of experimental results including beef cattle, dairy cattle, pigs, poultry, and sheep, Sheridan (1981) found that, in many cases, the level of heterosis in crossbred populations other than F_1 was substantially below expectations based on heterozygosities. The conclusion of the review of Sheridan (1981) was that, based on the performance of purebred and F_1 populations, it was not possible to predict the level of heterosis in other various genotypes, suggesting the presence of epistatic effects.

According to Cunningham (1987), although in some cases epistatic loss effects can be safely neglected, their proper evaluation is one of the unsolved problems of animal breeding research. Recent studies have reported epistatic loss on preweaning gain in crosses between *Bos taurus* and *Bos indicus* (Fries et al., 2000; Piccoli et al., 2002; Demeke et al., 2003; Cardoso, 2004). Because *Bos taurus* and *Bos indicus* have a greater genetic distance (larger potential differences in gene frequencies), *Bos taurus* × *Bos indicus* crosses generally express a higher level of heterosis compared with crosses between *Bos taurus* breeds (Koch et al., 1989). Consequently, greater epistatic loss is expected in their crosses.

In the multibreed genetic evaluation currently run in Ontario, records are preadjusted for heterosis on the basis of heterozygosity. For preweaning growth traits, a direct and maternal heterosis of 5% is assumed for an individual with heterozygosity of 100%, regardless of the breeds involved (Sullivan et al., 1999). Results from this study and from Miller (1996), however, give evidence that the level of direct and maternal heterosis on preweaning gain in Ontario's cattle is lower than 5%.

Breed Additive Effects

Estimates of direct and maternal breed additive effects on preweaning gain, expressed as deviations from Angus, are presented in Table 5. Estimates of direct breed additive effects of Hereford, Limousin, and Short-

horn were lower than estimates of Angus. Salers slightly exceeded Angus, whereas Charolais, Gelbvieh, Maine-Anjou, and Simmental exceeded Angus by more than 10 kg for direct effects.

Estimates of maternal breed additive effects of Blonde d'Aquitaine, Charolais, and Hereford were lower than Angus. Limousin and Maine-Anjou exceeded Angus by less than 1 kg. Gelbvieh, Salers, Shorthorn, and Simmental exceeded Angus by more than 4.5 kg.

Estimates of breed additive effects were in general agreement with what was expected based on a previous study with this population of cattle by Miller (1996). The SE of the estimates of both direct and maternal breed additive effects were large for all the breeds and greater for those breeds represented by a small number of calves (Blonde d'Aquitaine, Gelbvieh, Maine-Anjou, Salers, and Shorthorn). Further analysis assuming a zero genetic covariance between direct and maternal genetic effects resulted in small changes in the estimates of breed effects and no changes in the rank of the breeds (data not shown).

Sampling Correlations

The data used in this investigation included records from beef herds enrolled in a commercial genetic improvement program and therefore were not designed to estimate breed additive, dominance, and epistatic loss effects. Cunningham and Connolly (1989) showed that high correlation between estimates might jeopardize the precision of estimation of genetic effects. Even estimable functions may be highly confounded.

To obtain information with regard to degree of confounding between estimates, sampling correlations among additive, dominance, and epistatic loss effects were calculated (Table 6). The sample correlation between maternal dominance and direct epistatic loss effects was very high (-0.98), likely due to a structural deficiency of the data to separate maternal dominance and direct epistatic loss effects and/or due to linear dependencies (multicollinearity) involving predictor variables of maternal dominance (H_M) and direct epistatic loss (E_D) effects.

Sample correlations between breeds were generally high (from 0.58 to 0.96 and from 0.41 to 0.94 for direct and maternal breed additive effects, respectively). Sample correlations of direct breed additive effects with maternal breed additive effects within the same breed were higher than sampling correlations between different breeds. Thus, it was generally more difficult to separate direct and maternal additive genetic effects within breeds than between breeds. High SE of the estimates of breed effects and high sampling correlations between estimates, particularly between direct and maternal breed effects, could be a symptom of not having enough information to estimate both direct and maternal breed additive effects and/or multicollinearity among corresponding predictor variables. With a high degree of multicollinearity, estimates of regression coefficients

Table 6. Sampling correlations among estimates of direct (D) and maternal (M) fixed genetic effects

	H _D ^a	E _D	AN _D	BD _D	CH _D	GV _D	HE _D	LM _D	MA _D	SA _D	SH _D	SM _D
H _D	1.00											
E _D	-0.01	1.00										
AN _D	0.00	0.32	1.00									
BD _D	-0.01	0.28	0.84	1.00								
CH _D	0.01	0.32	0.94	0.86	1.00							
GV _D	-0.01	0.24	0.73	0.67	0.74	1.00						
HE _D	0.04	0.31	0.93	0.84	0.95	0.72	1.00					
LM _D	-0.00	0.31	0.93	0.85	0.96	0.73	0.94	1.00				
MA _D	0.00	0.23	0.74	0.67	0.75	0.58	0.74	0.75	1.00			
SA _D	-0.01	0.26	0.79	0.71	0.80	0.61	0.79	0.79	0.64	1.00		
SH _D	0.04	0.25	0.77	0.70	0.79	0.60	0.78	0.78	0.64	0.66	1.00	
SM _D	0.02	0.32	0.94	0.85	0.96	0.73	0.94	0.95	0.75	0.79	0.78	1.00
H _M	-0.01	-0.98	-0.31	-0.28	-0.32	-0.24	-0.31	-0.31	-0.23	-0.25	-0.24	-0.31
E _M	-0.01	-0.00	-0.01	-0.01	-0.01	-0.00	-0.01	-0.01	-0.00	-0.00	-0.00	-0.01
AN _M	0.01	-0.30	-0.96	-0.81	-0.91	-0.70	-0.89	-0.90	-0.71	-0.76	-0.74	-0.90
BD _M	0.06	-0.24	-0.74	-0.89	-0.76	-0.59	-0.74	-0.75	-0.59	-0.63	-0.61	-0.75
CH _M	0.03	-0.31	-0.92	-0.83	-0.97	-0.72	-0.92	-0.93	-0.73	-0.78	-0.76	-0.93
GV _M	0.05	-0.18	-0.55	-0.50	-0.55	-0.77	-0.54	-0.55	-0.44	-0.46	-0.45	-0.55
HE _M	-0.03	-0.30	-0.91	-0.82	-0.93	-0.70	-0.97	-0.92	-0.72	-0.77	-0.77	-0.92
LM _M	0.07	-0.30	-0.90	-0.82	-0.92	-0.71	-0.90	-0.96	-0.72	-0.76	-0.75	-0.91
MA _M	0.00	-0.21	-0.67	-0.61	-0.69	-0.53	-0.67	-0.68	-0.91	-0.58	-0.58	-0.68
SA _M	0.05	-0.22	-0.67	-0.61	-0.68	-0.53	-0.67	-0.68	-0.54	-0.86	-0.56	-0.68
SH _M	-0.04	-0.23	-0.74	-0.67	-0.76	-0.58	-0.75	-0.75	-0.61	-0.63	-0.96	-0.75
SM _M	0.01	-0.31	-0.91	-0.83	-0.93	-0.71	-0.91	-0.92	-0.73	-0.77	-0.76	-0.97
	H _M	E _M	AN _M	BD _M	CH _M	GV _M	HE _M	LM _M	MA _M	SA _M	SH _M	SM _M
H _M	1.00											
E _M	0.00	1.00										
AN _M	0.30	0.01	1.00									
BD _M	0.25	0.00	0.74	1.00								
CH _M	0.32	0.01	0.92	0.77	1.00							
GV _M	0.19	0.00	0.55	0.47	0.56	1.00						
HE _M	0.31	0.02	0.91	0.75	0.94	0.55	1.00					
LM _M	0.30	0.00	0.90	0.76	0.94	0.56	0.92	1.00				
MA _M	0.21	0.01	0.68	0.56	0.69	0.41	0.68	0.69	1.00			
SA _M	0.22	0.00	0.67	0.56	0.69	0.41	0.68	0.68	0.52	1.00		
SH _M	0.24	0.01	0.74	0.61	0.77	0.45	0.77	0.75	0.58	0.56	1.00	
SM _M	0.31	0.01	0.91	0.76	0.94	0.56	0.93	0.92	0.69	0.69	0.76	1.00

^aH = dominance; E = epistatic loss; AN = Angus; BD = Blonde d'Aquitaine; CH = Charolais; GV = Gelbvieh; HE = Hereford; LM = Limousin; MA = Maine-Anjou, SA = Salers; SH = Shorthorn; and SM = Simmental.

obtained by ordinary least squares methods typically have large SE, might be highly confounded, and are easily affected by changes in the dataset.

Implications

Estimates of (co)variance components of preweaning gain consistent with previous studies indicated that current parameters for genetic evaluation are appropriate. A large estimated negative genetic correlation between direct and maternal effects seemed to be a consequence of data structure rather than a true high negative relationship. The estimated level of direct and maternal heterosis on preweaning gain was less than the level of 5% assumed in the genetic evaluation of this population. Lack of information to adequately separate direct and maternal breed additive effects and multicollinearity among predictor variables of breed effects may affect the ranking of animals across breeds. Further

investigation of the causes of instability of the estimates and application of alternative statistical methods are warranted to improve the multibreed genetic evaluation of this population of beef cattle.

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