On the Negative Estimates of Direct and Maternal Genetic Correlation
- A Review**

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ABSTRACT : Estimates of genetic correlation between direct and maternal effects for weaning weight of beef cattle are often negative in field data. The biological existence of this genetic antagonism has been the point at issue. Some researchers perceived such negative estimate to be an artifact from poor modeling. Recent studies on sources affecting the genetic correlation estimates are reviewed in this article. They focus on heterogeneity of the correlation by sex, selection bias caused from selective reporting, selection bias caused from splitting data by sex, sire by year interaction variance, and sire misidentification and inbreeding depression as factors contributing sire by year interaction variance. A biological justification of the genetic antagonism is also discussed. It is proposed to include the direct-maternal genetic covariance in the analytical models. *(Asian-Aust. J. Anim. Sci. 2001. Vol 15, No. 8 : 1222-1226)*

Key Words : Maternal Effects, Parameter Estimation, Variance Component, Weaning Weight

INTRODUCTION

Genetic sources for mothering ability of cows as well as for growth potential of calves are integral components in genetic evaluation for weaning weight in beef cattle. Conventional analysis of the additive maternal genetic effects employed a maternal grandsire model while a separate analysis was conducted for reckoning additive direct genetic effects. Simultaneous estimation of additive direct and maternal genetic effects became possible with a Hendersonian model as represented in Quaas and Pollak (1980). Easy inference on the genetic covariance between additive direct and maternal effects is advantageous in the use of the mathematical model. The covariance has been of concern to producers and breeders because growth traits are often the basis for selection. Estimates of the genetic correlations obtained from analyses of field data were negative for many beef cattle breeds (Pollak et al., 1994). This genetic antagonism was incorporated into the national genetic evaluation for many beef cattle breeds. Genetic correlations used were -0.28 for Beefmaster, -0.29 for Brangus, -0.21 for Gelbvieh, -0.28 for Hereford, -0.27 for Polled Hereford, and -0.32 for Simmental (BIF, 1996). There has been much debate over the biological existence of this genetic correlation. Some researchers perceived such estimate to be an artifact from poor modeling. Recently further research was directed to scrutinize the potential causes of the negative correlation between additive direct and maternal genetic effects on weaning weight.

The objective of this review is to integrate the studies on the negative direct and maternal genetic covariance in

beef cattle. The need for including the direct and maternal genetic covariance in analytical models is discussed. The review begins with analytical modeling for assessment of the genetic correlation, and then a biological justification on the negative genetic correlation is presented.

NEGATIVE ESTIMATES OF DIRECT AND MATERNAL GENETIC CORRELATION

Importance of variance and covariance components

Nowadays, genetic merits of individual animals are estimated using mixed model methodology under the assumption of known (co)variances, and the estimated values are called BLUP. In reality, (co)variances are, however, unknown in field data, and they should be estimated with the data. Thus, accurate genetic merits of individuals are based on accurate (co)variance estimates, and many animal breeders have made research endeavors to develop a variety of statistical approaches and computing algorithms for variance component estimation (Lee, 2000). Furthermore, inclusion and exclusion of such parameters in an analytical model is an important issue for accurate genetic evaluation. Mallinckrodt et al. (1995) brought up a reliability problem of negative direct-maternal genetic covariance estimate in selectively reported data.

Selective reporting and its influence on direct-maternal genetic correlation

Garrick et al. (1989) pointed out unequal reporting of data by sex to the American Simmental Association (ASA) for weaning weight: twice more female calves than males. Selective reporting was suspected by assuming that birth rate and survival to weaning were approximately equal for males and females.

Mallinckrodt et al. (1995) reported the impact of data

falsification and selective reporting on the correlation between additive direct and maternal genetic effects for weaning weight. They simulated weaning weight data with three combinations of contemporary group and pedigree structures from field data sets. They found that data falsification reduced heritabilities but did not change the correlation in any consistent way. However, selective reporting caused changes in the estimates of the correlation. When negative or zero values for the correlation were used to simulate data, selective reporting produced larger negative estimates in 32 out of 44 samples. On the other hand, a positive bias was observed in 11 out of 17 samples when the true correlation was positive. In addition, using only the direct and maternal breeding value estimates for animals with records yielded larger negative correlation estimates than those for all animals in all 13 selectively reported populations. Mallinckrodt et al. (1995) also reported that the use of a large negative correlation (-0.28) led to a less accurate prediction of genetic merit than the use of zero in a population with a true correlation of -0.09. They recommended the use of a zero correlation in genetic evaluation if one suspected selective reporting in their data.

Selective reporting vs complete reporting

For purebred Simmental data with selective reporting, the additive direct and maternal genetic correlations estimated by Garrick et al. (1989) were -0.27 for males and -0.34 for females. Lee et al. (1997) reported the variance and covariance component estimates using Simmental weaning weight data from herds where complete reporting occurred. The estimates of the additive direct and maternal genetic correlations were -0.46 and -0.38 respectively for males and females, both larger than those obtained by Garrick et al. (1989). This was not expected in the light of the findings by Mallinckrodt et al. (1995). Lee et al. (1997) maintained that selective reporting was not the cause of the negative correlation between additive direct and maternal genetic effects in Simmental data.

Zero correlation vs nonzero correlation

Use of incorrect variance and covariance matrix in a mixed model does increase the prediction error variances (PEVs) of the solutions to the mixed model equations (Henderson, 1975). Schaeffer (1984) also discussed the effects of incorrect correlations on multiple trait analyses. He concluded that PEV increased with the use of incorrect covariances, and this increase was directly related to the differences between true and estimated correlations. Therefore, one might conclude that if the true parameter was -0.3 and the estimated parameter was closer to this value than to zero, using zero should not be preferred.

Pollak et al. (1994) established influences from use of zero and nonzero correlations at different stages in an

animal's life cycle. Once an animal has a record, maternal expected progeny difference (EPD) for the high (low) growth animal can be decreased (increased) using negative correlation while the EPD for maternal ability is still the pedigee index using zero correlation.

Another major difference is displayed after the animal becomes a bull. The weight information obtained on the progeny will not influence the maternal EPDs of a bull if the correlations between direct and maternal effects are zero. The estimate is still the pedigree index for that trait. For nonzero correlations between direct and maternal effects, the maternal EPDs will once again change when the progeny performance information causes a change in the EPDs for the weight traits. It is not until the bull's daughters have calves with records that information on maternal ability becomes available directly. If a zero correlation is used, the EPD for maternal ability is the pedigree index until those data are obtained.

The impact of using a negative correlation on maternal EPDs was also investigated empirically for Simmental cattle by Pollak et al. (1994). The differences between maternal EPDs from zero and nonzero (-0.32) correlations in nonparent and in parent evaluations confirmed that maternal EPDs for larger growth bulls decreased when the negative correlation was used. It was emphasized that for the top 10% bulls, change in the parent evaluation from the nonparent evaluation was smaller when using the negative correlation than when using zero. Furthermore, using a zero correlation decreased the accuracy for additive maternal effects.

Heterogeneity of genetic correlation by sex

It was of concern that, from the results of Garrick et al. (1989), the smaller negative correlation was estimated for male data where selective reporting was likely to be more serious than for female data. This was the other way round from the findings of Mallinckrodt et al. (1995). The disagreement might have been possible for the following reasons. First, the results were not always identical in the different populations used by Mallinckrodt et al. (1995), i.e. it was not unusual to get a less negative estimate of direct-maternal genetic correlation in the Simmental population used by Garrick et al. (1989). The second potential reason was a true heterogeneity by sex. Finally, sources affecting the correlation estimate other than selective reporting may have produced confusion. An example was the problem created from partitioning data. The heterogeneity of variance and covariance components by sex is accounted for in the national genetic evaluation for Simmental cattle (Garrick et al., 1989). Garrick et al. (1989) partitioned data into male and female populations to estimate the parameters for each sex. However, selection bias might have been introduced by splitting the data because analyzing only

male data does not account for selection on females and vice versa. Partitioning of data can be considered as an extreme case of selective reporting. The selection in males and females is different in terms of both accuracy and intensity of selection. Selection influenced estimates of the variance and covariance components (Lee and Pollak, 1997b); hence not accounting for different levels of selection across sex contributed to heterogeneous estimates.

An attempt to remove selection bias caused by partitioning data was made by introducing genetic groups (Lee and Pollak, 1997b). However, the definitions of group effects are often arbitrary. Results showed that biases were not completely removed. This analysis with genetic group effects in an analytical model was also separately performed by sex, which suggested a simultaneous estimation of parameters by sex.

In order to examine heterogeneous variance and covariance components for sex, Lee and Pollak (1997b) estimated the parameters simultaneously by introducing a multiple trait model for treating male data as one trait and female data as another trait. This approach was applied to weaning weight data of Simmental cattle (Lee et al., 1997).

Sire by year interaction

A question arose as to whether or not the large negative genetic correlation of -0.46 for males and -0.38 for females estimated by Lee et al. (1997) resulted only from a true genetic antagonism. Parameter estimates are generally affected by ignoring components such as covariances, interactions, and other effects in an analytical model. Ignoring a true interaction changes the expected values of the variance components as shown by Lee and Pollak (1997d).

Robinson (1996) empirically showed that direct and maternal genetic correlation estimates were influenced by ignoring other effects. She investigated, through simulation, the consequences on genetic parameter estimates when negative dam-offspring covariance or sire by year interaction variance were ignored. A total of 1,500 records were simulated from mating 15 sires each to 20 dams for 5 mating seasons.

Data simulated with a negative dam-offspring regression coefficient of -0.02 were analyzed using a model with additive direct-maternal genetic correlation and permanent environmental effects instead of the dam-offspring regression. The estimate of the direct-maternal genetic correlation was -0.48, and that of permanent environmental variance was 8% of the phenotypic variance estimate.

Robinson (1996) simulated seven herds with additional sire by year interaction, and the simulated data were analyzed using a model with direct and maternal genetic correlation instead of the sire by year interaction. Ignoring

sire by year interaction created a spurious negative correlation. Undesirably, parameter estimates obtained using the correct model differed (p<0.05) from the input values. The number of replicates in Robinson (1996) was not large enough to explain the effect from ignoring sire by year interaction.

Lee and Pollak (1997d) simulated data with sire by year interaction but without genetic correlation, and a spurious negative genetic correlation was also observed using a model with direct-maternal genetic correlation instead of sire by year interaction. Furthermore, they also found that inflation of negative direct and maternal genetic correlation estimate was caused by ignoring sire by year interaction effects in both simulated and Simmental field data. Lee (1998) analytically investigated the cause of an inflated negative genetic correlation estimate. The inflation could be due to a functional relationship of design matrices for additive direct and maternal genetic effects to that for sire effects within which sire by year interaction effects were nested. It was proven that the maternal genetic variance was inflated by the amount of reduction for sire variance, the direct genetic variance was inflated by four times the change for maternal genetic variance, and the direct and maternal genetic covariance was deflated by twice the change for maternal genetic variance.

Sire by year interaction effect was significant, but its origin unknown. Plausibility lacked in biological explanation of the sire by year interaction effect. It might be a true interaction, perhaps caused by different environmental factors associated with different years, or the effect is confounded with other unidentified sources of covariance between progeny records in the same year. Sire by year interaction effects would be caused by ignoring inbreeding depression for both direct and maternal genetic effects because inbreeding coefficients for progeny of each sire and for their dams increase every year in closed populations. The inbreeding depression affected weaning weight of the Simmental population (Ward, 1993) although inbred animals were not a large portion of the population. In the simulation study of Lee and Pollak (1997a), all genetic variance and covariance components increased by ignoring inbreeding depression, a 0.8 kg reduction of performance per 1% inbreeding. Yet, negligible sire-by-year interaction variance was observed.

Although plausible biological explanation for the paternal effects is still not presently available, it is possible that an artifact for paternal effects (and so sire by year interaction effects) might be created from misidentified male parents. An analysis of stochastically simulated data by Lee and Pollak (1997c) showed that 20% sire misidentification resulted in a spurious sire by year interaction variance, 1 to 3% of the phenotypic variance. Other potential factors contributing sire by year interaction

variance may be importation of genetically superior materials and nonrandom mating groups (Robinson, 1996).

A BIOLOGICAL POINT OF VIEW

Partitioning of nutrients

Bauman and Currie (1980) discussed partitioning of nutrients to various body tissues involved in maintenance and growth and to establishing body reserves such as lipids, glycogen, and labile protein. Substantial portions of maternal nutrients are utilized to develop the fetus and to support lactation. The partitioning involves two kinds of regulation. One is homeostasis, which involves maintenance of physiological equilibrium, and the other is homeorhesis, which involves orchestrated changes for the priorities of a physiological state. During lactogenesis, the coordinated adaptations in rates of lipogenesis and lipolysis in adipose tissue and of gluconeogenesis and glycogenolysis in liver is under homeorhetic control of partitioning nutrient. Genetic variation in partitioning and utilizing nutrients could exist in circulating hormones (extracellular signals) and in cellular recognition and expression of these signals (Gorski, 1979; Kiddy, 1978; Bauman and Currie, 1980).

Correlation between body weight and milk production

A genetic relationship between the weight of a beef or dairy cow and milk production has been inconsistent (Badinga et al., 1985; Lin et al., 1985), while a positive phenotypic relationship has been repeatedly demonstrated (Sieber et al., 1988; Lewis et al., 1990; Freking and Marshall, 1992). Moore et al. (1991) estimated genetic parameters of weight at calving and production in first lactation using a multiple trait model. Test-day records of 11,457 Ayrshire and 112,371 Holstein cows from a subpopulation of Quebec Dairy Herd Analysis Service were collected from 1979 to 1986, and they were used to calculate cumulative lactation milk yield at 70, 90, and 305 days in lactation. The estimates of genetic correlations between weight at calving and milk production ranged from -0.29 to -0.33 and from -0.22 to -0.24 in Ayrshires and Holsteins, respectively.

In beef cattle, Meyer et al. (1994) obtained smaller genetic correlation estimates (-0.10 for Hereford and 0.00 for Wokalup) between weaning weight and milk yield. Recently, Lee and Pollak (2002) estimated genetic correlation between body weight and milk production in beef cattle. Data used for body weight included birth weight, weaning weight, and yearling weight of Korean cattle, and milk yields were actually measured at sequential intervals from one month to four months after calving. Posterior means of genetic parameters were estimated with multiple trait sire and maternal grandsire mixed models using Gibbs sampling. Genetic correlation estimates between body weight and milk yield were all negative. The estimates ranged from -0.08 to -0.16 for birth weight, from -0.04 to -0.21 for weaning weight, and from -0.12 to -0.19 for yearling weight, and most of them were more negative than the estimates of Meyer et al. (1994).

A hypothesis to negative direct and maternal genetic covariance

Biological explanation of genetic antagonism between direct and maternal genetic effects is currently unavailable. However, given the Bauman and Currie's (1980) idea of partitioning nutrients in mammals, the negative genetic correlation between weight and milk yield in the studies of Moore et al. (1991), Meyer et al. (1994), and Lee and Pollak (2002) implied genetic antagonism between direct and maternal effects.

CLOSING REMARKS

It is recommended from the studies discussed in this article that the influence from several sources on the direct-maternal genetic correlation estimates be considered in order to evaluate accurate additive direct and maternal genetic effects. The sources are potential heterogeneity of the correlation by sex (Lee et al., 1997), selection bias caused from selective reporting (Mallinckrodt et al., 1995), selection bias caused from splitting data by sex (Lee and Pollak, 1997b), sire by year interaction variance or paternal permanent environmental variance (Robinson, 1996; Lee and Pollak, 1997d) and their potential heterogeneity by sex, factors contributing sire by year interaction variance, i.e. sire misidentification (Lee and Pollak, 1997c), inbreeding depression (Lee and Pollak, 1997a) and interactions among these sources.

Direct-maternal genetic covariance is required to be in analytical models for accurate weaning weight genetic evaluation. Computational burden from addition of the covariance is not critical. This is because the number of nonzero elements in Henderson's mixed model equation increases, but number of equations does not increase. The computation, however, requires more iteration for estimation of genetic parameters by likelihood- or posterior-based methods. In a huge data set analysis, reduction of the equation number would be beneficial for (co)variance components estimation by using an equivalent model referred to as the reduced animal model (RAM) developed by Quaas and Pollak (1980) or, furthermore, the really reduced animal model (RRAM) by Quaas (1992, unpublished note). Incorporation of genetic group for both direct and maternal genetic effects in the analytical model may lead to more accurate genetic evaluation (Van Vleck, 1990).

This review attempted to explain a physiology of genetic antagonism between direct and maternal effects. The genetic antagonism of growth potential of calves with mothering ability of cows might be explained by the genetic antagonism of body weight with milk yield under the theory of partitioning nutrients. To arrive at an obvious biological explanation of the negative direct and maternal genetic correlation requires more research into identifying the nature of genes affecting such traits.

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