Summary

Field records from the American Simmental Association were used to estimate genetic parameters for direct and maternal effects of traits associated with dystocia. Estimates of the additive genetic variances of direct and maternal effects and of the additive genetic covariances of direct and maternal effects were calculated by equating the sire variance component, maternal grandsire variance component and covariance component between sire and maternal grandsire to their biological causal components. Independent variables in the mixed-model, least-squares analyses were herd, sire or maternal grandsire of the calf, sex of calf and percentage Simmental in the calf. The dependent variables were calving ease (score), assisted births, birth weight and gestation length. Sire and maternal grandsire were significant sources of variation for all traits studied. Maternal grandsire accounted for two to three times more variation in calving ease and assisted births than did sire, whereas sire accounted for a larger percentage of the total variation in birth weight and gestation length. Heritability estimates for the maternal effects were .20, .13, .10 and .09 for calving ease, assisted births, birth weight and gestation length, respectively. Genetic correlations between the direct and maternal effects were −.53, −.55, −.24 and −.38 for calving ease, assisted births, birth weight and gestation length, respectively. The effect of these correlations on selection is discussed.

(Key Words: Maternal Effects, Calving Ease, Birth Weight, Gestation Length, Beef Cattle.)

Introduction

Dystocia is an economically important trait to the cow-calf producer. Many breed associations and artificial insemination organizations are currently using progeny evaluations of bulls for dystocia for potential use by producers in selecting bulls that sire calves with less dystocia. However, little is known about the relationship between the direct effect of the bull on dystocia of his calves and the maternal component associated with dystocia in his daughters. Pollak and Freeman (1976) reported a rank correlation of .16 for the sire and maternal grandsire rankings for dystocia in Holsteins. Philipsson (1976) estimated the heritability of maternal effects associated with dystocia to range from .00 to .18 and the correlations between the direct and maternal effects to be −.19 and −.53 for birth weight and dystocia, respectively.

The objectives of this study were to estimate some of the genetic parameters for the direct and maternal effects associated with dystocia and to determine the correlation between the direct and maternal effects.

Materials and Methods

Data were field records provided by the American Simmental Association from its performance registration and sire evaluation programs. Records on approximately 450,000 progeny sired by more than 700 purebred Simmental bulls were edited so that each calf record included breeder identification number, sire, maternal grandsire, sex of calf, age of dam (BIF, 1976), percentage Simmental (75 or 88%), birth date, calving ease score (1 = no assistance, 2 = easy pull, 3 = hard pull usually with a mechanical puller, 4 = Caesarian section and 5 = abnormal presentation) and birth weight. All calving ease scores of 5 were deleted in the first edit.

Contemporary groups (herds) were estab-
lished on the basis of birth date of the calf within a given breeder identification number. Calves were assigned to a contemporary group based on the first calf born in a breeder's herd. All calves within a breeder's herd were sorted on the basis of birth date of the calf within a given breeder identification number. Calves were assigned to a contemporary group based on the first calf born in a breeder's herd. All calves within a breeder's herd were sorted on the basis of birth date, and calves born within 90 days after the birth of the first calf were assigned to the same contemporary group. Any time the birth dates of two consecutive calves were more than 45 days apart, another contemporary group was formed within that breeder's herd. Herds that had the same gestation lengths for all calves were removed from the data. Only bulls that were represented as sire and maternal grandsire were used. Only progeny from 2-year-old dams were used, for the following reasons: (1) when calving difficulty was measured as a trait of the calf, the variances among age of dam groups were heterogeneous (Burfening et al., 1979), and (2) selection of dams may have been practiced on the basis of their calving performance as 2-year-olds. These edits resulted in data sets that contained 11,552 and 11,026 records and 69 and 58 sires or maternal grandsires for model I and II analyses, respectively. The coefficients for the sire and maternal grandsire variance components in the expectation of their mean squares were 70.4 and 97.8, respectively.

After editing for gestation lengths, the data sets contained 4,196 and 3,715 records and 63 and 56 sires or maternal grandsires for model I and II analyses, respectively. The coefficients for the sire and maternal grandsire variance components in the expectation of the mean squares in this analysis were 29.4 and 38.5, respectively. The larger data sets were used for the dependent variables calving ease, assisted births and birth weight, while the smaller data set was used for the dependent variable gestation length. When the phenotypic correlation between a sire's offspring group average and maternal grandsire offspring group average was calculated, only sires common to both data sets were used.

Data were analyzed by mixed-model least-squares procedures (Harvey, 1977) according to the following linear model:

\[
Y_{ijkl} = \mu + H_i + S_j + X_k + P_l + e_{ijkl},
\]

where

\[
Y_{ijkl} = \text{each record of the dependent variable,}
\]

\[
\mu = \text{population mean,}
\]

\[
H_i = \text{random effect of the } i^{th} \text{ herd (contemporary group),}
\]

\[
S_j = \text{fixed effect of the } j^{th} \text{ sire or maternal grandsire,}
\]

\[
X_k = \text{fixed effect of the } k^{th} \text{ sex,}
\]

\[
P_l = \text{fixed effect of the } l^{th} \text{ percentage of Simmental and}
\]

\[
e_{ijkl} = \text{random error.}
\]

In model I, sire of the calf was included, and, in model II, the maternal grandsire of the calf was used instead of sire of the calf. Sire or maternal grandsire were analyzed as fixed effects so that sire or maternal grandsire constants could be obtained from the analyses in order to calculate the phenotypic correlation

\[
(r_{FSFMG})
\]

between sire group averages and maternal grandoffspring group averages. However, when the sire (\(\sigma_S^2\)) or maternal grandsire (\(\sigma_{MG}^2\)) variance components were estimated, sires or maternal grandsires were considered to be random effects and the variance components were calculated accordingly.

The dependent variables in the analyses were calving ease (score), assisted births, birth weight and gestation length. Assisted births were calculated by recording all scores of 1 as 0 and all scores of 2, 3 and 4 as 1.

Estimates of the additive genetic variance of direct effects (\(\sigma_A^2\)), additive genetic variance of maternal effects (\(\sigma_M^2\)) and additive genetic covariance of direct and maternal effects (\(\sigma_{A\cdotM}\)) were calculated by equating the sire variance component (\(\sigma_S^2\)), maternal grandsire variance component (\(\sigma_{MG}^2\)) and covariance component between sire and maternal grandsire (\(\sigma_{S\cdotMG}\)) to their biological causal components (Koch, 1972), as shown below:

\[
\sigma_S^2 = 1/4 \sigma_A^2,
\]

\[
\sigma_{MG}^2 = 1/16 \sigma_A^2 + 1/4 \sigma_{A\cdotM} + 1/4 \sigma_M^2
\]

and

\[
\sigma_{S\cdotMG} = 1/8 \sigma_A^2 + 1/4 \sigma_{A\cdotM}.
\]
The additive genetic variances and covariances were then calculated as follows:

\[ \sigma_A^2 = 4 \sigma_S^2, \]
\[ \sigma_{A \cdot M} = 4 \sigma_{S \cdot MG} - 2 \sigma_S^2 \text{ and} \]
\[ \sigma_M^2 = 4 \sigma_{MG}^2 - 4 \sigma_{S \cdot MG} + \sigma_S^2. \]

The estimates of sire and maternal grandsire variance components were obtained from models I and II, respectively.

The covariance component between sire and maternal grandsire was estimated by an indirect procedure similar to that used by Calo et al. (1973) and Philipsson (1976). The phenotypic correlation between a sire's offspring group average and maternal grandoffspring group average as presented in equation (1) assumes that the environmental contribution is zero. However, no edits were performed on the data in order to ensure that a calf from a sire was not also a dam for the same sire in the maternal grandsire role.

\[ \hat{r}_{GS \cdot MG} = \sqrt{\frac{n_S \hat{h}_S^2}{4 + (n_S - 1) \hat{h}_S^2}} \cdot \sqrt{\frac{n_{MG} \hat{h}_{MG}^2}{4 + (n_{MG} - 1) \hat{h}_{MG}^2}} \cdot \hat{r}_{GS \cdot GMG,} (1) \]

where

\[ n_S = k \text{ value for sires,} \]
\[ n_{MG} = k \text{ value for maternal grandsire,} \]
\[ \hat{h}_S^2 = \frac{\sigma_A^2}{\sigma_S^2 + \sigma_{RS}} \]
\[ \hat{h}_{MG}^2 = \frac{4 \sigma_{MG}^2}{\sigma_{MG}^2 + \sigma_{RMG}^2} \]
\[ r_{GS \cdot GMG} = \text{correlation between sire (direct) and maternal grandsire (direct and maternal) additive genetic values and} \]
\[ \sigma_R^2 = \text{residual variance component from sire(s) or maternal grandsire (MG) analysis.} \]

What remains to be estimated is (1)

\[ \hat{r}_{GS \cdot MG}, \]

and it can be calculated. Now the covariance component between sire and maternal grandsire can be estimated from

\[ \hat{\sigma}_{S \cdot MG} = \hat{r}_{GS \cdot GMG} \cdot \sqrt{\hat{\sigma}_S^2 \cdot \hat{\sigma}_{MG}^2}. \]

Heritabilities of direct and maternal effects were calculated as:

\[ \hat{h}_A^2 = \frac{\sigma_A^2}{\sigma_P^2}, \]
\[ \hat{h}_M^2 = \frac{\sigma_A^2 \cdot \sigma_{A \cdot M} + \sigma_M^2 + \sigma_E^2}{\sigma_P^2}, \]

where

\[ \sigma_P^2 = \sigma_A^2 + \sigma_{A \cdot M} + \sigma_M^2 + \sigma_E^2. \]

The correlation between additive genetic direct effects and additive genetic maternal effects was estimated as follows:

\[ \hat{r}_{A \cdot M} = \frac{\sigma_{A \cdot M}}{\sqrt{\sigma_A^2 \cdot \sigma_M^2}}. \]

Dickerson (1947) showed that the portion of the selection differential that is realized is

\[ \left( \sigma_A^2 + 3/2 \sigma_{A \cdot M} + 1/2 \sigma_M^2 \right) / \sigma_P^2. \]

Hence, if \( \sigma_{A \cdot M} \) is negative, the realized selection differential will be decreased. This value was calculated and compared to the case of \( \sigma_{A \cdot M} = 0 \) for an assessment of the effect of \( \sigma_{A \cdot M} \) on selection.

**Results**

The least-squares analyses of variance indicated that sire and maternal grandsire were significant sources of variation for all traits studied (table 1). The maternal grandsire variance component for calving ease score and assisted births was approximately three times larger than the sire variance component. However, the sire variance component was approximately two times larger for birth weight and four times larger for gestation length than the maternal grandsire variance component (table 1). These results agree with those of Philipsson.
DIRECT AND MATERNAL EFFECTS

However, Brinks et al. (1973) did not find the same relationship between the sire and maternal grandsire variance components, and sire was not a significant source of variation in dystocia among 2-year-old dams.

Heritability estimates (table 2) of the direct effects ($h^2_d$) were similar to those reported by Brinks et al. (1973), Cundiff et al. (1975), Philipsson (1976) and Tong et al. (1976). The heritability estimates for the dam effects ($h^2_M$) were higher than those for the direct effects ($h^2_d$) for calving ease (score) and assisted births, and lower for birth weight. These results agree with those of Philipsson (1976), who observed that $h^2_M$ was higher for calving ease and lower for birth weight than was $h^2_d$. However, Brinks et al. (1973) reported a heritability estimate of 0 for calving difficulty in 2-year-olds when the trait was classified as a trait of the dam.

The phenotypic correlations (table 2) between the traits studied were the same for models I and II. Calving ease (score) and assisted births were moderately correlated with birth weight but not with gestation length, and there was a low but positive correlation between birth weight and gestation length.

Genetic correlations (table 2) of calving ease (score) and assisted births with birth weight were high and agree closely with correlations reported by Philipsson (1976) and Burfening et al. (1979). Genetic correlations of calving ease (score) and assisted births with gestation length were moderate and were slightly higher than those reported by Philipsson (1976). In general, genetic correlations between traits from model II were lower than those from model I and agreed fairly closely with those reported by Philipsson (1976).

Correlations between the sire offspring averages and maternal grandsire grandoffspring averages

\[
\rho_{F_SF_{MG}}
\]

from models I and II are shown in table 3. Correlations between the progeny averages for calving ease (score) and assisted births were negative but not significantly different from 0. Pollak and Freeman (1976) found a rank correlation for dystocia score of .16 among 39 Holstein sires appearing as both the sire and maternal grandsire. Using data from the 1978 National Simmental Sire Summary, Burfening (unpublished data) found correlations for cal-
TABLE 2. HERITABILITIES, GENETIC AND PHENOTYPIC CORRELATIONS FOR MODELS I AND II FOR 2-YEAR-OLD DAMS

<table>
<thead>
<tr>
<th>Trait</th>
<th>Calving ease</th>
<th>Assisted births</th>
<th>Birth weight</th>
<th>Gestation length</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Calving ease (score)</td>
<td>.05 ± .01</td>
<td>1.00 ± .04</td>
<td>.94 ± .08</td>
<td>.58 ± .21</td>
</tr>
<tr>
<td></td>
<td>.20 ± .03</td>
<td>.98 ± .01</td>
<td>.76 ± .08</td>
<td>−.03 ± .22</td>
</tr>
<tr>
<td>Assisted births</td>
<td>.81**</td>
<td>.04 ± .01</td>
<td>.85 ± .11</td>
<td>.47 ± .24</td>
</tr>
<tr>
<td></td>
<td></td>
<td>.13 ± .03</td>
<td>.73 ± .10</td>
<td>.02 ± .23</td>
</tr>
<tr>
<td>Birth weight (kg)</td>
<td>.43**</td>
<td>.35**</td>
<td>.21 ± .03</td>
<td>.32 ± .17</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>.10 ± .02</td>
<td>.20 ± .25</td>
</tr>
<tr>
<td>Gestation length (days)</td>
<td>.09**</td>
<td>.09**</td>
<td>.18**</td>
<td>.25 ± .04</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>.09 ± .03</td>
</tr>
</tbody>
</table>

*aHeritabilities on main diagonal; genetic correlations above main diagonal; phenotypic correlations below main diagonal.

**Phenotypic correlation (P<.01).

Heritability estimates for the maternal effects (h²_M) (table 4) were larger than those for the direct effects (table 2) for calving ease (score) and assisted births but smaller than those for birth weight and gestation length. These results agree closely with those of Philipsson (1976), who observed heritabilities for the maternal effects to range from .00 to .18 for similar traits. Although there are larger opportunities for error in estimating the variance and covariance components for maternal effects, these results indicate that there was considerable genetic variation in the maternal components of calving ease. Koch (1972) reported that the additive genetic component of maternal ability accounted for 10 to 15% of the variance in birth weight.

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The estimated covariance components (Ω_A,M) (table 3) between the direct and maternal effects (h²_M) (table 4) were larger than those for the direct effects (table 2) for calving ease (score) and assisted births but smaller than those for birth weight and gestation length. These results agree closely with those of Philipsson (1976), who observed heritabilities for the maternal effects to range from .00 to .18 for similar traits. Although there are larger opportunities for error in estimating the variance and covariance components for maternal effects, these results indicate that there was considerable genetic variation in the maternal components of calving ease. Koch (1972) reported that the additive genetic component of maternal ability accounted for 10 to 15% of the variance in birth weight.

TABLE 3. STATISTICS FOR DIRECT AND MATERNAL EFFECTS

<table>
<thead>
<tr>
<th>Trait</th>
<th>Ω_P_S</th>
<th>Ω_P_MG</th>
<th>Ω_Δ</th>
<th>Ω_M</th>
<th>Ω_S_MG</th>
<th>Ω_A_M</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calving ease (score)</td>
<td>−.2131</td>
<td>.0336</td>
<td>.1538</td>
<td>−.0053</td>
<td>−.0382</td>
<td></td>
</tr>
<tr>
<td>Assisted births</td>
<td>−.1936</td>
<td>.0088</td>
<td>.0334</td>
<td>−.0012</td>
<td>−.0094</td>
<td></td>
</tr>
<tr>
<td>Birth weight (kg)</td>
<td>.05928</td>
<td>3.4868</td>
<td>2.2987</td>
<td>.0465</td>
<td>−.6856</td>
<td></td>
</tr>
<tr>
<td>Gestation length (days)</td>
<td>.1255</td>
<td>4.6752</td>
<td>2.2360</td>
<td>.1585</td>
<td>−1.2243</td>
<td></td>
</tr>
</tbody>
</table>

*aCalculated from 58 sire and maternal grandoffspring group averages.
TABLE 4. HERITABILITY ESTIMATES FOR MATERNAL EFFECTS ($h^2_M$) AND GENETIC CORRELATION BETWEEN DIRECT AND MATERNAL EFFECTS ($r_{A\cdot M}$)

<table>
<thead>
<tr>
<th>Item</th>
<th>Calving ease (score)</th>
<th>Assisted births</th>
<th>Birth weight (kg)</th>
<th>Gestation length (days)</th>
</tr>
</thead>
<tbody>
<tr>
<td>$h^2_M$</td>
<td>.20</td>
<td>.15</td>
<td>.11</td>
<td>.10</td>
</tr>
<tr>
<td>$r_{A\cdot M}$</td>
<td>-.53</td>
<td>-.55</td>
<td>-.24</td>
<td>-.38</td>
</tr>
</tbody>
</table>

Effects were negative for all traits (table 3).

Correlations between the additive genetic direct and maternal effects ($r_{A\cdot M}$) (table 4) were all negative and moderately high. Philipsson (1976) observed negative correlations between direct and maternal effects for calving ease, birth weight and gestation length, but the correlations were much smaller for calving ease score and much larger for birth weight than were those from this study. Koch (1972) observed a genetic correlation between direct and maternal effects for birth weight of .07. He reported an average from the literature of -.44.

Discussion

The results of this study, along with those reported by Philipsson (1976), suggest an antagonism between direct and maternal effects for traits related to dystocia. Although the heritability estimates for calving ease (score) were low, the frequency of assisted births ranged from 24 to 69% for bulls as sire of the calf and from 35 to 70% for bulls as maternal grandsire when they were 100 or more progeny for either trait. Although the phenotypic correlations between sire rankings of progeny groups classified as offspring and grandoffspring did not differ from 0, the results indicated that, because of the negative genetic correlation between the direct and maternal effects, progress from selection for reduced dystocia would be reduced. The estimated proportion of direct selection differential that would be realized as a result of the negative covariance between direct and maternal effects would be .50, .47, .80 and .72 for calving ease (score), assisted births, birth weight and gestation length, respectively.

These results, along with those of Philipsson (1976), give negative estimates of the correlation between direct and maternal effects for calving ease and birth weight. However, the estimates for $\sigma^2_M$ and $\sigma_{A\cdot M}$ are subject to large sampling errors since the variance and covariance components were estimated from different types of relatives and the multipliers used (2 and 4) may have inflated the errors of estimates. Therefore, the results indicate the need for further studies with other breeds on large data sets to estimate these parameters. If subsequent estimates substantiate the negative relationship between the direct and maternal effects on dystocia, selection methods will require modification for effective reduction of dystocia.

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