Direct genetic and maternal variance and covariance component estimates from Angus and Hereford field data

Brad Richard Skaar

Iowa State University

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DIRECT GENETIC AND MATERNAL VARIANCE AND COVARIANCE COMPONENT ESTIMATES FROM ANGUS AND HEREFORD FIELD DATA

Iowa State University

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Direct genetic and maternal variance and
covariance component estimates from
Angus and Hereford field data

by

Brad Richard Skaar

A Dissertation Submitted to the
Graduate Faculty in Partial Fulfillment of the
Requirements for the Degree of
DOCTOR OF PHILOSOPHY

Department: Animal Science
Major: Animal Breeding

Approved:

Signature was redacted for privacy.

In Charge of Major Work

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For the Major Department

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For the Graduate College

Iowa State University
Ames, Iowa

1985
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INTRODUCTION

Major beef breed associations in the United States are currently conducting or sponsoring National Sire Evaluation Programs based on the mixed model methodology presented most notably by C. R. Henderson. Sires are evaluated for the economically important traits of birth, weaning, and yearling weight based primarily on progeny records. Best linear unbiased predictors of their transmitting abilities are reported to breeders for use in selection decisions. Further developments in these procedures by D. E. Wilson (1984) provide methodology for the unification of within herd evaluations with national sire evaluations by use of a model that accounts for both the sire and dam contributions to the phenotype of a calf. By partitioning the effects of the dam into maternal and genetic contributions, dams can be more objectively compared for their genetic ability to provide a maternal environment (uterine effects, milk production, maternal behavior, etc.). As a consequence, a sire's transmitting ability for maternal effect can also be determined by analyzing the records of progeny from female relatives, primarily his daughters. These estimated transmitting abilities for maternal effect can be reported to breeders and allow more objective selection of bulls that will sire replacement females for the herd. These will replace currently computed maternal breeding values.

The imminent implementation of this timely and badly needed evaluation of maternal capability provides the impetus for this study. The methodology used for these evaluations requires the use of known variances and covariances of all random effects in the model. Unfortunately, these variance components are not known and, in fact, need to be developed from
the population that is to be evaluated. Therefore, the objective of this study is to develop estimates of the variances components required in order to evaluate Angus and Hereford sires for both their direct genetic and maternal genetic transmitting abilities for birth and weaning weight. Genetic population parameters (variances and covariances, heritabilities, and correlations) were also computed for the additive direct and additive maternal effects of both traits.
REVIEW OF LITERATURE

The basis for concern about maternal effects manifests itself in the conclusions of Dickerson (1947) and Willham (1963). Reported from these works, the portion of the selection differential that is realized when selection is made on the expression of a maternally influenced trait can be quantified by the expression

\[(\sigma_a^2 + (1.5)\sigma_{A\cdot M} + (.5)\sigma_m^2)/\sigma_p^2,\]

where \(\sigma_p^2\) is the phenotypic variation of the trait, \(\sigma_a^2\) and \(\sigma_m^2\) are additive direct and additive maternal variance, and \(\sigma_{A\cdot M}\) is the genetic covariance between direct and maternal effects. Without maternal influences on \(p\), this proportion is simply heritability in the narrow sense. But clearly the magnitude of the maternal influence can enhance or impede selection progress for \(p\). In fact, antagonistic (negative) relationships between direct and maternal effects could result in a deterioration of maternal performance if continued selection on only the direct effect were practiced.

Typically, maternal effects in mammals are assumed to be at work during the time the offspring is in contact with his dam. Koch and Clark (1955) determined that maternal effects play an important role in the expression of growth of an offspring until they are weaned. However, Mavrogenis et al. (1978) and Rutledge et al. (1972) determined that these maternal effects even play an important role into the postweaning period of life. Extensive reviews present evidence of maternal effects in small
rodents (Legates, 1972), sheep (Bradford, 1972), swine (Robison, 1972), and beef cattle (Koch, 1972). Much of the current knowledge surrounding the influences that a dam has on her offspring's phenotype is contained in this series of articles. Cundiff (1972) summarized the series by concluding that maternal effects are likely more important than direct effects of the genes in the offspring for early postnatal growth while they are nursing their dams but that they have diminishing effects in later life.

Maternal effects have also been established in nonmammalian species (Bondari, 1971) but will not be discussed here. Discussion instead will be limited to livestock species and primarily beef cattle.

Legates (1972) defined two periods of maternal influence which define well the stages of the maternal influence in beef cattle. First are those effects that occur during the formation of the calf in utero, termed prenatal effects. These uterine influences are a result of the genotype of the dam, as well as environmental influences on the young that are mediated by the dam. Secondly, there are those influences termed postnatal effects. The dam's influence on her young after parturition is of concern here. Temperament and maternal instinct may play a role (Poindron and Neindre, 1978), but the nutritional environment of the calf through the dam's ability to lactate is the primary issue in beef cattle (Neville, 1962; Jeffery et al., 1971; Rutledge et al., 1971).

The role that maternal effects play in the selection of breeding stock needs to be determined before effective selection decisions can be made. This role can be quantified in part by the estimated heritability for maternal effects and by the genetic correlation between direct and
maternal effects. A review of some reported heritabilities and correlations for direct and maternal birth and weaning weight is presented, followed by a discussion of methodology surrounding estimation of these parameters.

Parameter Estimates

Birth weight

Birth weight is an economically important trait in beef cattle, primarily because of its positive association with calving difficulty (Bellows et al., 1971; Brinks et al., 1973) and its negative association with neonatal livability (Martinez et al., 1983). Prenatal maternal effects are believed to influence the expression of birth weight.

Koch (1972) examined evidence for birth weight maternal effects in beef cattle through an extensive literature review and analysis of Fort Robinson Experiment Station data on Hereford cattle. He summarized that 15 to 20 percent of birth weight variation in beef cattle is due to the genetic and permanent environmental components of the maternal influence. Of this, he suggests that 10 to 15 percent is additive genetic maternal variance.

Heritability estimates for the additive direct effects for birth weight are numerous in the literature; no attempt was made to review all of them. Woldehawariat et al. (1977) completely summarized estimates reported in the literature to that date and should adequately provide a basis for comparison. Some 136 paternal half sib heritability estimates alone were reviewed by Woldehawariat, producing a weighted average of .40. Bertrand (1983) reviewed more recent literature and stated an arithmetic average
birth weight heritability of about .20, nearly half of the more extensive review reported by Woldehawariat. Bertrand's review seemed to incorporate more estimates from breed association field data bases and from a larger proportion of research on European derived cattle (Simmental, Charolais, and Limousin).

More specific to the purpose of this dissertation, Table 1 is presented to review estimates from research and reviews that dealt with maternal effects on birth weight.

From the values in Table 1, the conclusion is drawn that relatively higher proportion of direct than maternal variance exists for birth weight, agreeing with general conclusions of Bourdon and Brinks (1982). They concluded that direct effects for birth weight account for three to four times more variation than do maternal effects. Koch's review of birth weight parameter estimates suggests that this proportion is somewhat lower (Koch, 1972). He infers that the heritability for additive direct effect ($h^2_A$) for birth weight is between .2 and .4, while that for additive maternal effect ($h^2_M$) ranges from .1 to .15. While these were observational averages, the literature reviewed by Koch actually reported a wider range in heritability estimates for maternal effect from 4 to 30 percent.

The estimates reported by Everett and Magee (1965), Burfening et al. (1981), and Bourdon and Brinks (1982) generally concur with Koch's averages but still show variety. Brown and Galvez (1969) disagreed slightly from Koch's conclusions, as they found that $h^2_M$ was slightly larger than $h^2_A$ in Angus but not so in Herefords.
Table 1. Reviewed estimates of additive direct (A) and maternal (M) heritabilities and genetic correlations for birth weight

<table>
<thead>
<tr>
<th>Author and date</th>
<th>$h_A^2$</th>
<th>$h_M^2$</th>
<th>$r_{A-M}$</th>
<th>Method$^a$</th>
<th>Breed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Everett &amp; Magee (1965)</td>
<td>.22</td>
<td>.04</td>
<td>-.93</td>
<td>PHS,MAS,MGS,COV(PGS,MGS)</td>
<td>Holstein</td>
</tr>
<tr>
<td></td>
<td>.65</td>
<td>.15</td>
<td>-.98</td>
<td>PHS,MHS,PGS,MGS,COV(PGS,MGS)</td>
<td>Holstein</td>
</tr>
<tr>
<td>Brown &amp; Galvez (1969)</td>
<td>.56</td>
<td>.30</td>
<td>-.57</td>
<td>PHS,MHS,MGS,OD</td>
<td>Hereford</td>
</tr>
<tr>
<td></td>
<td>.14</td>
<td>.25</td>
<td>-.39</td>
<td>PHS,MHS,MGS,OD</td>
<td>Angus</td>
</tr>
<tr>
<td>Vesely &amp; Robison (1971)</td>
<td>.67</td>
<td>.05</td>
<td>-.89</td>
<td>PHS,MHS,OD;($\sigma_M^2$,$\sigma_{Ep}^2$) = 1:4</td>
<td>Hereford</td>
</tr>
<tr>
<td></td>
<td>.67</td>
<td>.29</td>
<td>-.56</td>
<td>PHS,MHS,OD;($\sigma_M^2$,$\sigma_{Ep}^2$) = 4:1</td>
<td>Hereford</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>($\sigma_M^2$ = maternal variance; $\sigma_{Ep}^2$ = permanent maternal environment variance)</td>
<td></td>
</tr>
<tr>
<td>Koch (1972)</td>
<td>.20-.30</td>
<td>.10-.15</td>
<td>-.41</td>
<td>Literature review</td>
<td>Hereford</td>
</tr>
<tr>
<td></td>
<td>.45</td>
<td>.10</td>
<td>+.07</td>
<td>Average of six combinations using PHS,MHS,OS,OD,MGS,COV(PHS,MGS)</td>
<td></td>
</tr>
<tr>
<td>Philipsson (1976)</td>
<td>.17</td>
<td>.12</td>
<td>-.19</td>
<td>(Least squares; sire variance/(pooled) mgs variance computed separately. Indirect sire-mgs variance estimated.)</td>
<td>Holstein</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>.19</td>
<td></td>
<td>Holstein</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>.04</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

$^a$Indicates types of relatives used in estimation procedure or if another method was used: PHS=paternal half sib; MHS=maternal half sib; OS=offspring and sire; OD=offspring and dam; PGS=paternal grandsire sibs; MGS=maternal grandsire sibs; OM=offspring and midparent.
<table>
<thead>
<tr>
<th>Author and date</th>
<th>$\hat{h}_A^2$</th>
<th>$\hat{h}_M^2$</th>
<th>$\hat{r}_{A,M}$</th>
<th>Method(^a)</th>
<th>Breed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Burfenning et al. (1981)</td>
<td>.21</td>
<td>.11</td>
<td>-.24</td>
<td>Mixed model; sire variance/mgs variance computed separately. Indirect sire/mgs covariance estimated.</td>
<td>Simmental</td>
</tr>
<tr>
<td>Bourdon &amp; Brinks (1982)</td>
<td>.39</td>
<td>.12</td>
<td>--</td>
<td>PHS,MHS: assumed $r_{A,M}$ = 0</td>
<td>(Angus, Red (averaged)</td>
</tr>
<tr>
<td></td>
<td>.38</td>
<td>.10</td>
<td>--</td>
<td>PHS,MHS: assumed $r_{A,M}$ = 0</td>
<td>(Angus, Hereford)</td>
</tr>
<tr>
<td>Nelsen et al. (1984)</td>
<td>.36</td>
<td>.82</td>
<td>-.51</td>
<td>PHS,OS,OD,OM (various combinations of relationships and of data subsets were averaged)</td>
<td>Hereford</td>
</tr>
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Vesely and Robison (1971) report variances for direct effect that were higher than most others reviewed. The estimation procedures required a restriction that the maternal variance be a certain proportion of permanent environmental error variance, and a range of possible solutions was presented. No possible suggestions were given as to why these heritabilities were high, and it is not certain if these were a result of the restrictions. This was not suspected.

Nelsen (1984) studied data originating from two replicates of a randomly mated herd of Hereford cattle in Montana. Birth weights were taken on 1,012 calves, and the whole and subsets of the data were analyzed for the proportion of additive genetic direct and maternal variance and covariance present. Subsets were designed to depict combinations of matings that were above or below the mean birth weight for the herd. Estimates for the heritability of direct effects ($\hat{h}_A^2$), heritability of maternal effects ($\hat{h}_M^2$), and the genetic correlation between direct and maternal effects ($r_{A,M}$) were .36, .82, and -.51, respectively. However, estimated maternal heritabilities from various subsets of the data and from different genic expectations of relative covariances ranged anywhere from -.50 to +2.85. The author cited large experimental errors and the inability of the procedures to account for more sources of variation as factors inhibiting exact interpretation. Evidence of the presence of nonadditive genetic effects was considered as well when comparing different mating schemes. Assortative and disassortative mating schemes were shown to badly inflate $\hat{h}_M^2$. 
To draw general conclusions about the covariance between direct and maternal effects for birth weight, other than it being negative, would be difficult to do from the estimates reviewed. As Willham (1980) suggests, a negative covariance possibly exists, or at least can be argued. Still, literature estimates range from nearly 0 to -1.0, making it difficult to interpret the literature.

Koch and Clark (1955) took covariances between relatives and computed genetic direct-maternal correlations over a range of possible values for the component parts of direct genetic and total maternal variance. Their suggestions were that the genetic correlation is likely to be small and positive (.0 to .2) unless direct effect heritability is high and permanent environmental factors are very small. The calculations of direct heritabilities presented by Woldehawariat (1977) and those in Table 1 would suggest this is a likely possibility.

Koch's (1972) review found that literature estimates averaged -.44 for $r_{A\cdot M}$, obviously much larger than was anticipated possible by Koch and Clark (1955). Estimates reviewed ranged from -.33 to -.93. Koch's own research on Hereford cattle at Ft. Atkinson Experimental Station in Nebraska yielded estimates from -.17 to +.30 depending upon what type of relative structure was considered. His was one of few papers found to report a positive covariance.

Philipsson (1976) and Burfening et al. (1981) computed the genetic covariance by an indirect method proposed by Calo et al. (1973). Their estimates of -.19 and -.24, respectively, were similar and nearer zero than most estimates. Their methods had the advantage of using expectations that
avoided some unreasonable assumptions and dealt only with genes passed through male relatives. Both researchers nonetheless indicated that the correlations should be treated as rough estimates due to the approximate nature of the method. Bourdon and Brinks (1982) even made the assumption that the covariance of direct and maternal birth weight was zero because of the absence of convincing evidence to the contrary.

More recent work by Nelsen et al. (1984) listed a wide range in estimates (0 to -1.07) depending upon the type of data and relatives considered. He did observe that estimates more negative than -.6 occurred when only dams with heavy birth weights were used for analysis. They also suggested that nonadditive gene action such as epistasis was responsible for varying estimates. The dam-offspring covariance seemed to cause more variation in solutions than was possible to account for.

Weaning weight

As for birth weight, considerable research has produced parameter estimates for the direct effect of weaning weight. Woldehawariat et al. (1977) presents the most comprehensive review of weaning weight heritability estimates found. A heritability of .29 was derived from a weighted average of 144 paternal half sib estimates.

Unlike the expression of birth weight, a calf's weaning weight is known to be highly influenced by his dam's mothering ability. Koch (1972) examined evidence for weaning weight maternal effects in the literature and summarized that 35 to 45 percent of the variation in weaning weight is attributable to the total maternal influence, with additive genetic
maternal effects comprising 30-35 percent. Hohenboken and Brinks (1971a) concluded from their analysis of linebred and inbred range Hereford cattle that maternal effects contributed 7 to 17 percent more variability to weaning weight than did direct effects. Table 2 presents a review of some studies dealing with maternal effects and weaning weight.

A majority of the heritability estimates presented are in agreement with Koch (1972) with a direct effect heritability of about .25 and maternal effect heritability of .35. An exception was Vesely and Robison (1971) whose direct estimates were noticeably greater. Deese and Koger (1967) reported lower heritabilities on Brahman data than for Brahman x Shorthorn crossbreds but indicated that the level of possible inbreeding in the crossbred cows was not accounted for. Inbreeding should cause the parameters to be upwardly biased (Hohenboken and Brinks, 1971a).

Estimates larger than .35 for maternal effect heritability were generally extracted from analyses that considered the regression of offspring record on dam record. Hohenboken and Brinks (1971a) found that by replacing sire-offspring (OS) regressions with dam-offspring (OD) regressions increased estimated maternal variance by 63 percent. In analysis where OD regressions were removed, Koch (1972) computed maternal effect variances ranging from 27 to 29 percent of the total variance. But with OD regressions included, percent maternal variance estimates were erratic, ranging from 60 to -16 percent. The inability of procedures used to effectively account for permanent environmental variance and the possibility of a large environmental correlation are suggested as probable causes. Vesely and Robison (1971) are supportive of these conclusions. In all, the need
Table 2. Reviewed estimates of additive direct (A) and maternal (M) heritabilities and genetic correlations for weaning weight

<table>
<thead>
<tr>
<th>Author and data</th>
<th>$\hat{h}^2_A$</th>
<th>$\hat{h}^2_M$</th>
<th>$\hat{r}_{A-M}$</th>
<th>Method$^a$</th>
<th>Breed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hill (1965)</td>
<td>.34</td>
<td>.00</td>
<td>3.48</td>
<td>120 day wt.</td>
<td>Hereford</td>
</tr>
<tr>
<td></td>
<td>.32</td>
<td>.29</td>
<td>-.33</td>
<td>180 day wt.</td>
<td>Hereford</td>
</tr>
<tr>
<td></td>
<td>.32</td>
<td>.51</td>
<td>-.46</td>
<td>210 day wt.</td>
<td>Hereford</td>
</tr>
<tr>
<td>Deese &amp; Koger (1967)</td>
<td>.18</td>
<td>.15</td>
<td>0</td>
<td>PHS,MHS,MGS,OD</td>
<td>Brahman</td>
</tr>
<tr>
<td></td>
<td>.40</td>
<td>.46</td>
<td>-.73</td>
<td>PHS,MHS,MGS,OD</td>
<td>Shorthorn x-bred</td>
</tr>
<tr>
<td>Hohenboken &amp; Brinks (1971a)</td>
<td>.27</td>
<td>.40</td>
<td>-.28</td>
<td>PHS,MHS,MGS,OD,OS</td>
<td>Inbred Hereford</td>
</tr>
<tr>
<td></td>
<td>.23</td>
<td>.34</td>
<td>-.28</td>
<td>PHS,MHS,MGS,OS</td>
<td>Inbred Hereford</td>
</tr>
<tr>
<td></td>
<td>.23</td>
<td>.54</td>
<td>-.79</td>
<td>PHS,MHS,OD</td>
<td>Inbred Hereford</td>
</tr>
<tr>
<td>Vesely &amp; Robison (1971)</td>
<td>.51</td>
<td>.06</td>
<td>-.72</td>
<td>PHS,MHS,OD;($\sigma^2_M/\sigma^2_{Ep}=1:4$)</td>
<td>Hereford</td>
</tr>
<tr>
<td></td>
<td>.52</td>
<td>.33</td>
<td>-.57</td>
<td>PHS,MHS,OD;($\sigma^2_M/\sigma^2_{Ep}=4:1$)</td>
<td>Hereford</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>($\sigma^2_M$=maternal variance; $\sigma^2_{Ep}$=permanent maternal environmental variance)</td>
<td></td>
</tr>
<tr>
<td>Koch (1972)</td>
<td>.23</td>
<td>.19</td>
<td>-.05</td>
<td>Average of six combinations using PHS,MHS,OS, OD,MGS,COV(PHS,MGS)</td>
<td>Hereford</td>
</tr>
<tr>
<td></td>
<td>.25-.35</td>
<td>.35-.45</td>
<td>-.3 - -.78</td>
<td>Literature review</td>
<td></td>
</tr>
<tr>
<td>Crow &amp; Howell (1982)</td>
<td>--</td>
<td>.16-.23</td>
<td>--</td>
<td>Fitted maternal grandsire Angus, Charolais to mixed model</td>
<td>Hereford</td>
</tr>
</tbody>
</table>

$^a$Indicates types of relatives used in estimation procedure or if another method was used: PHS = paternal half sib; MHS=maternal half sib; OS=offspring and sire; OD=offspring and dam; PGS=paternal grandsire sibs; MGS=maternal grandsire sibs; OM=offspring and midparent.
to avoid using the dam-offspring relationship when estimating maternal variance is suggested by these results.

A range of lower maternal heritability estimates presented by Crow and Howell (1982) is worth mention. The estimates were derived using a mixed model procedure for variance component estimation (Henderson, 1980) on field data from Angus, Charolais, and Hereford cattle. Their estimates represent the heritability of a maternal grandsire's total contribution to his daughter's performance as a mother and represents a composite measure of a sire's direct and maternal genetic contributions to his offspring. Their averaged estimates are admittedly lower than other literature values. This was explained by Crow as large sampling errors in the Charolais data causing some negative estimates to occur. Crow also reported estimates of $h^2_{mgs}$ by parity within breed. Estimates from the second and third parity were near zero for Angus and were small negative estimates for Charolais, but larger estimates were reported in the Hereford analysis. Crow cites erratic nutritional needs of second and third parity females as a cause for less explainable variation as compared to first calf and older cows.

As for the relationship between direct and maternal weaning weight, another look at Table 2 indicates that while generalities may be drawn in support of a nonzero covariance between direct and maternal effects, it appears that quantifying this value is not so simple. Earlier work by Koch and Clark (1955) had suggested that even over a large range of possible variances for direct and permanent environmental effects, the genetic correlation between direct and maternal weaning weight ($r_{A-M}$) is likely large and negative (-.65 to -.77). A later literature review by Koch
(1972) found that estimates of the genetic correlation were between -.3 to -.8 and averaged -.44. His own research on Hereford cattle implied that $r_{A \cdot M}$ was nearer to zero (-.05), but a range in estimates (from -.78 to 0.0) showed that they were dependent upon which relative covariances were considered. Most of Koch's estimates that were near zero did not consider the dam-offspring relationship, which is in agreement with the findings of Hohenboken and Brinks (1971a).

Deese and Koger (1967) discovered that Brahman cattle in Florida showed little maternal variance for weaning weight (15 percent of total) and reported a zero value for $r_{A \cdot M}$ for weaning weight. However, the same study showed a greater maternal component in a closed Brahman x Shorthorn crossbred cow herd and reported a value for $r_{A \cdot M}$ of -.73. Thus, in most situations reviewed, it appears that an increase in maternal variation brings with it a larger negative value for $r_{A \cdot M}$.

An exception is that reported by Vesely and Robison (1971). In solving for parameters, they imposed the restriction that maternal variance ($\sigma_M^2$) be estimated as a fixed proportion of permanent environmental variance ($\sigma_{PE}^2$). Estimates for $r_{A \cdot M}$ were from -.57 to -.72 when the ratio of $\sigma_M^2:\sigma_{PE}^2$ was 4:1 and 1:4, respectively.

The covariances reported by Hill (1965) also holds conclusions of interest. He found that the estimated genetic covariance between direct and maternal effects was larger negative at weight closer to 210 days. However, positive associations were found on 90 and 120 day weights ($\sigma_{A \cdot M}^2 = 195$ and 71 lbs$^2$, respectively). A small negative covariance was found at birth along with a small negative maternal variance. His results agreed
with earlier conclusions made by Dickerson and Grimes (1947) and Young and Legates (1965) that maternal influences are greatest during the period shortly after birth but decrease as the offspring matures. Further, it was stated that these results suggest a positive genetic correlation for milking ability and protein deposition but a negative association for milking ability and fat deposition. Hill recommended that selection for maternal ability improvement be made on 90-day progeny weights and that selection for direct growth be on progeny weights taken post-weaning.

In all but one instance reported in Tables 1 and 2, the environmental covariance between dam and offspring records was assumed to equal zero. However, this covariance is known to have a substantial influence on a calf's weaning weight (Christian et al., 1965; Martin et al., 1970; Mangus and Brinks, 1971). Koch (1972) eluded to the possibility of large environmental covariances between offspring and dam as a possible bias in computing the genetic relationships between direct and maternal effects for weaning and birth weight. Mangus and Brinks (1971) concluded that high levels of nutrition for a young heifer resulted in subsequent reduction of her productivity as a cow. To avoid this confounded effect, Hohenboken and Brinks (1971b) computed most probable producing abilities (MPPA) on paternal half sisters and correlated these to the weaning weight records of their paternal half brothers. In essence, this was a correlation between progeny proofs of a sire for both MPPA and weaning weight. A genetic correlation of +.49 was reported.

Earlier work by Langlet (1965) reported a correlation of .22 between milk production of 2,450 German Holstein daughters with 10 month gain in
236 sons of 25 sires. Bar-Anon (1965) reported a correlation of .06 between contemporary comparison milk production proofs of daughters and the yearling rate of gain of paternal half brothers of Israeli Holstein breeding. And Mason (1964) found a near zero genetic correlation between weight per day of age in steer progeny with the milk production records of their dams but reported a genetic correlation of +.19 between paternal half brother and sister groups from British Red Poll and Dairy Shorthorn field data.

Methodology

The task of estimating the direct-maternal covariance in cattle has been accomplished through the use of correlations between relatives. Dickerson (1947), Cockerham (1954), and Koch and Clark (1955) defined path diagrams which defined the genetic and environmental influences on the phenotype of an individual, as well as the relationships of these influences between individuals. Partial regression coefficients from path diagrams allowed Koch and Clark to partition observed correlations into their theoretical components, thus statistically separating maternal from direct effects.

Willham (1963) examined the composition of the covariance between relatives when a maternal effect was involved. A linear genotypic model was used to develop a general expression for the covariance between two individuals X and Y. From this expression, a set of linear equations can be solved, with each equation representing a different covariance. The method has since been used extensively to estimate the variances and covariances of maternal, dominance, and environmental effects (e.g.,
Bondari, 1971; Burfening et al., 1981; Hohenboken and Brinks, 1971a; Mavrogenis et al., 1978; Martinez et al., 1983).

In many cases such as research cited in Tables 1 and 2, the method of equating expectations of relative covariances involves the use of either maternal half sib or dam-offspring relationships. The problems of confounded effects (dam maternal and dam genetic) contributing to the phenotype of the calf and of a possible direct-maternal genetic covariance are discussed by Willham (1980) as two of the major problems inherent with maternal effect estimation. Eisen (1967) proposed that three mating systems producing 10 types of relatives would be required to obtain reliable estimates of the genetic and nongenetic parameters involved in a complete model. Unfortunately, its use is not practical for application to beef cattle research due to a slow reproductive rate.

Willham (1972) proposed that the covariance of second cousins could be estimated and provide solutions to genetic variance components that are theoretically free of dominance effects, environmental variances, and covariances, as well as a grand maternal effect. Bondari (1971) applied this plan to Tribolium castaneum to determine the maternal genetic components involved in family size and pupae weight. His plan is practical for application to beef field data, but he warns that the low level of relationships involved can decrease accuracy. Of course, when using low levels of relationship, accuracy can be increased by increasing the number of observations analyzed. Field data currently collected by beef breed associations can provide large numbers of records with relationships desired (see Wilson, 1984). Mixed model estimation procedures are applicable to such data.
Mixed model variance component estimation

Several methods of variance analysis procedures have been developed that are applicable to the mixed models defined in this dissertation. Among these include Henderson's Method 3 (1953), Maximum Likelihood (ML) (Hartley and Rao, 1967), Restricted Maximum Likelihood (REML) (Patterson and Thompson, 1971), and Minimum Norm Quadratic Unbiased Estimation (MINQUE) (Rao, 1971). In general, all are similar in that they are quadratic estimation procedures that are translation invariant (do not depend upon the scale of defined fixed effects). Some yield unbiased estimates of variance components (MINQUE, METHOD-3), but as a result, there is a nonzero probability that estimated variance components will not fall in the parameter space required for their use (Searle, 1971). An example would be a negative sire variance estimate.

However, unbiasedness is not necessarily a rigid requirement when estimates are to be used in a mixed model analysis. Use of any translation invariant estimate from a symmetric population will lead to unbiased predictors of breeding values when applied to the usual mixed model (Kennedy, 1981). The ML and REML estimation procedures are solved interatively and require normality of the data (REML, ML). They do not yield unbiased estimates. Although estimates from ML and REML are restricted to fall within a specified parameter space, convergence to a global maximum is not guaranteed by these procedures (Schaeffer, 1983). In fact, Schaeffer (1983) indicates that if negative estimates are obtained using an unbiased procedure, it is not likely that convergence would occur using one of the iteratively solved methods.
Numerous publications exist which detail the specific characteristics of each of these methods. Additional detailed reviews of Henderson's Methods and MINQUE are presented by Searle (1971) and Henderson (1984b), and a review for ML and REML may be found in Harville (1977).

Schaeffer (1983) clearly demonstrates through the use of examples that each of these methods is computationally very extensive. Depending upon the nature of the model (number of levels of random effects, single or multiple trait analysis, covariate analysis, etc.), direct inverses of a large coefficient matrix are required, as well as the traces of products of large nondiagonal matrices.

Harville (1977) suggested that when computations of quadratic forms for REML cannot be easily computed, an approximate quadratic form may exist which will approximate the true REML quadratic. Henderson (1980) presented computing algorithms for a new method of variance analysis frequently referred to as MINQUE-D which approximates the quadratic forms of a MINQUE estimator. This method entails inverting only the diagonal elements of the coefficient matrix of the mixed model equations after absorption of fixed effects. Then, quadratic forms are computed from the solutions to random effects obtained from the product of this inverse and the absorbed right hand sides. Quadratic values are equated to their expectations, and the variance components are solved for simultaneously. Henderson (1984a) extended this methodology to a multiple trait analysis and generalized his approach in Henderson (1984b).

With simulated data, Henderson (1980) compared the relative sampling variances of MINQUE-D estimates with those of Method 3 and MINQUE.
Comparisons were made over a large range of prior variance ratios used. MINQUE-D yielded smaller sampling variances than Method 3 in all classes of priors used and produced sampling variances that were only slightly larger than those found for MINQUE estimates.

Kennedy (1981) compared the characteristics of Henderson's new method (to be referred to as MINQUE-D) with ML, REML, and MINQUE procedures and the consequences of applying estimates from each to the mixed model prediction of breeding value. Sire rank for breeding value was unchanged by use of variances obtained from each method, although use of ML estimates resulted in downward biased estimates of heritabilities when applied to a smaller data set. Consequently, estimated sire breeding values were regressed more towards the mean when using ML estimated variances. Method 3, REML, MINQUE, and MINQUE-D yield nearly equal estimates of sire and error variance, although the advantage in computational simplicity was given to MINQUE-D. Kennedy admitted that the properties of MINQUE-D solutions are not defined.

Dempfle et al. (1983) used a Swiss Braunvieh data base of milk yield records to compare MINQUE-D estimates of sire variance with MINQUE estimates. They concluded that MINQUE-D always produced inferior estimates to MINQUE but maintained high efficiency when compared to the large sample variances of REML which were considered best. They also concluded that MINQUE-D is a useful alternative estimation procedure when computational difficulty forbids the use of MINQUE.

Few references were found of applications of MINQUE-D to specific data sets. Crow and Howell (1982) applied MINQUE-D techniques to estimate the
variance of maternal grandsire effect for weaning weight in Canadian beef field data. Wilson (1984) applied MINQUE-D towards the evaluation of Angus and Hereford birth and weaning weights and yearling gain records from field data. Silcox (1985) used MINQUE-D methods to estimate the sire, region, and sire x region interaction variance components for age at first calving in Angus field data. Although not required by the procedure, both Wilson and Silcox reported iterative estimates for variance components.

Hudson and Van Vleck (1982) computed MINQUE-D estimates for sire variance from northeast United States dairy records from five breeds. They reported both first round and iterative solutions for variance components but clearly stated that the properties of iterative MINQUE-D are not known. In fact, only first round solutions to MINQUE-D are unbiased and translation invariant by definition (Henderson, 1984a). However, if convergence occurs within the parameter space allowed as a restriction, then iterative MINQUE estimates are equivalent to REML estimates with the assumption of normality (Harville, 1977). Hudson and Van Vleck did find that convergence occurred quickly (usually less than five rounds) and that final estimates were only slightly different than initial ones. They also discovered that iterative solutions were identical despite choice of prior although first round estimates did vary somewhat, thus concluding that iterative MINQUE-D is insensitive to reasonable choice of priors.

Finally, the abstract presented by Cady and Burnside (1982) was the only report found that made use of MINQUE-D for the estimation of a covariance. Approximately 22,000 dystoria records of calves out of 1,073 sires and 1,933 maternal grandsires were analyzed. The model used included
terms for various fixed effects, sire of calf effect, sire of dam effect, and residual error. Variance components for sire effect and maternal grandsire effect were computed as well as their covariance using an iterative version of MINQUE-D. Solutions were obtained after 11 rounds of iteration.
MATERIALS AND METHODS

Two variations of a linear mixed model were applied to birth and weaning weight records from Angus and Hereford field data for the purpose of estimating variance components of random effects. These variance components can in turn be utilized in currently operational national sire evaluations of these two breeds. A description of the data bases used for these analyses is discussed first in this section followed by a detailed description of Analysis I. Analysis II is simply a variation of Analysis I and so the discussion is more abbreviated. A description of methods used to estimate genetic parameters concludes the section.

Data Description

Birth and weaning weight records were provided courtesy of the American Angus Association, St. Joseph, Missouri, and by the American Hereford Association, Kansas City, Missouri. Evaluations for sire direct variance, sire maternal variance, and the covariance between sire direct and sire maternal effects were computed within breed for each trait. In order to perform these evaluations, the following information was provided for each record.

1. herd identification
2. sex of calf
3. date weaned
4. weaning management code (creep or noncreep)
5. source of data (designed test or field data)
6. performance record (birth or weaning weight)
7. sire registration number
8. maternal grandsire registration number
Contemporary groups for both birth and weaning weight were defined in accordance with procedures used for sire evaluations currently conducted for both breed associations (Wilson, 1984). A group is defined by all calves of the same sex, raised in the same management conditions to weaning, and weaned at the same time. A distinction was made between records derived from designed evaluation tests and from field data as well. Also, weaning weights were initially adjusted to a constant 205 days of age and were additively corrected for age-of-dam by its respective association according to the correction factors determined by Anderson (1977) (for Angus data) and Leighton (1979) (for Hereford data).

From these performance data files, 20 Angus herds and 25 Hereford herds were selected to form the data base evaluated. The herds were selected by officials of their respective recording associations. They represent long histories of performance testing and provide common ties between contemporary groups through the use of artificial insemination. Angus records used were recorded between 1972 and 1984, and Hereford records used were recorded between 1970 and 1984. Further descriptive information concerning these data sets is given in Tables 3 and 4.

Variance Component Estimation - Analysis I

The purpose of the analysis is to quantify the relationship between a sire's transmitting ability for the direct effect of a trait and his ability to transmit genes that determine his daughter's maternal influence on that trait. Initially, this required that a model be developed which identified the sources of direct and maternal contributions to the
Table 3. Descriptive statistics for Angus data

<table>
<thead>
<tr>
<th>Trait</th>
<th>Number of records</th>
<th>Mean&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Std. dev.&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Number of sires&lt;sup&gt;b&lt;/sup&gt;</th>
<th>Number of maternal grandsires&lt;sup&gt;c&lt;/sup&gt;</th>
<th>Number of groups</th>
<th>Sire-daughter matings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Birth weight</td>
<td>25,586</td>
<td>75.92</td>
<td>11.08</td>
<td>718 (295)</td>
<td>1,319 (896)</td>
<td>884</td>
<td>110</td>
</tr>
<tr>
<td>Weaning weight</td>
<td>34,190</td>
<td>476.05</td>
<td>73.58</td>
<td>941 (368)</td>
<td>1,576 (1003)</td>
<td>1,197</td>
<td>141</td>
</tr>
</tbody>
</table>

<sup>a</sup>Expressed in units of pounds.

<sup>b</sup>( ) = number represented only as sires and not maternal grandsires.

<sup>c</sup>( ) = number represented only as maternal grandsires and not sires.

Table 4. Descriptive statistics for Hereford data

<table>
<thead>
<tr>
<th>Trait</th>
<th>Number of records</th>
<th>Mean&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Std. dev.&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Number of sires&lt;sup&gt;b&lt;/sup&gt;</th>
<th>Number of maternal grandsires&lt;sup&gt;c&lt;/sup&gt;</th>
<th>Number of groups</th>
<th>Sire-daughter matings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Birth weight</td>
<td>14,436</td>
<td>81.20</td>
<td>9.85</td>
<td>566 (275)</td>
<td>1,134 (843)</td>
<td>399</td>
<td>68</td>
</tr>
<tr>
<td>Weaning weight</td>
<td>46,616</td>
<td>503.08</td>
<td>84.09</td>
<td>1,366 (535)</td>
<td>2,169 (1338)</td>
<td>1,289</td>
<td>172</td>
</tr>
</tbody>
</table>

<sup>a</sup>Expressed in units of pounds.

<sup>b</sup>( ) = number represented only as sires and not maternal grandsires.

<sup>c</sup>( ) = number represented only as maternal grandsires and not sires.
phenotype of an individual. Then, a method of variance component estimation was employed that will facilitate the estimation of variances and covariances of these contributions.

**Model description**

In most basic terms, the phenotypic record $y$ of an individual is expressed as the sum of the influences made from his two parents plus environmental influences that alter the expression of his parental effects. Let the phenotypic record $y$ be defined as:

$$y = s + d + e,$$

where $s$ and $d$ represent the total contribution of the sire and dam, respectively, to $y$, and $e$ is random temporary error peculiar to the formation of $y$. The assumptions are made that sire and dam contribute a sample half of their genes to the direct effect of $y$ and that the dam contributes additionally to the expression of $y$ through her maternal ability. Thus, $y$ is now defined as:

$$y = \frac{1}{2}g_{A_s} + \left(\frac{1}{2}g_{A_d} + G_m\right) + G_{\phi A} + e$$

where

- $g_{A_s}$ = additive genetic value of the sire(s) or dam(d) for the direct effect.
- $G_m$ = the total maternal effect on the expression of $y$.
- $G_{\phi A}$ = a genetic effect due to Mendelian sampling.
The dam's contribution to $y$ can be further decomposed to represent the origins of her value for direct and maternal effects. Reflecting this, $y$ is now determined by:

$$y = \frac{1}{2}g_{A_s} + \frac{1}{2}(\frac{1}{2}g_{A_{mgs}} + \frac{1}{2}g_{A_{mgd}}) + \left(\frac{1}{2}g_{M_{mgs}} + \frac{1}{2}g_{M_{mgd}} + E_p\right) + G_p + e,$$

where

- $g_{M}$ = the additive genetic value of the maternal grandsire (mgs) or maternal granddam (mgd) for the maternal effect on $y$,
- $E_p$ = permanent environmental effect on the maternal ability of the dam plus a genetic effect due to Mendelian sampling,

and other terms remain defined as before.

Model development to this point has been discussed by Wilson (1984) and Quass and Pollak (1980) for the purpose of within-herd estimation of breeding values ($\hat{g}_A$ or $\hat{g}_M$) where traits are influenced by a direct and maternal effect.

To simplify, let $s_{A_s}$ represent $\frac{1}{2}g_{A_s}$ and $m_{A_{mgs}}$ represent $\frac{1}{2}g_{A_{mgs}}$; in other words, the estimated transmitting abilities for direct effect of the sire and maternal grandsire, respectively. Also, $m_{A_{mgd}}$ will represent the estimated transmitting ability of the maternal grandsire for maternal effect. A calf's phenotype can now be expressed in terms of the effects received from male descendants only, placing all other effects into the error term. It should be clear that both $g_{A_s}$ and $m_{A_{mgs}}$ represent the additive genetic value for the direct trait but estimated from progeny or maternal grandprogeny, respectively.
Therefore, $y$ is given as:

$$y = f + s_A + 1/2mg_A + mg_M + e'$$  \hspace{1cm} (4)$$

where $f$, a designation of fixed effects, including the population mean, a sex effect, management effects, etc., and $e' = (1/4g_{mgd} + 1/2g_{mgd} + G_{dA} + Ep_{d} + e - f)$. Other effects are defined as before.

There are several advantages to forming an expression for $y$ in this manner. The model is clearly adaptable to mixed model methodology of Henderson (1972). Thus, concurrent evaluations can be performed for both the direct effect of a sire ($s_A$ and $mg_A$), as well as an evaluation of a sire's potential to transmit maternal ability to his daughters. This potential is expressed in the phenotypes of his maternal grandoffspring.

Secondly, with enough progeny and/or maternal grandprogeny records, the plus and minus effects of permanent environmental effects of calf records should negate themselves in the estimation of maternal effects.

Nonetheless, the problem of confounded effects on $y$ still exists, the inseparability of the genetic effect and the maternal influence on $y$. A maternal grandsire influences $y$ both by $1/4$ of his additive direct effect and by $1/2$ of his additive maternal effect, both of which are expressed through his daughter. By expressing $y$ as in Equation 4, advantage can be taken of the ability of Henderson's mixed model methods to partition the maternal grandsire's contribution to $y$ into direct and maternal effects through use of the variance-covariance matrix of random effects (Henderson, 1975).
In order to discuss the absorption of fixed effects, a more specific mathematical statement of \( y \) is required. For both weaning and birth weight, a record \( y \) on calf \( p \) is defined as:

\[
y_{ijklp} = \mu + c_{gij} + s_k + m_{d1} + m_{m1} + e_{ijklp},
\]

where

- \( \mu \) = overall mean of progeny records (fixed),
- \( c_{gij} \) = effect of the \( i \)th group in the \( j \)th herd (fixed),
- \( s_k \) = the direct additive genetic contribution of sire \( k \) (random),
- \( m_{d1} \) = \( 1/2 \) of the direct additive genetic contribution of mgs 1 (random) through the dam of \( p \),
- \( m_{m1} \) = genetic maternal effect of mgs 1 as expressed in the maternal ability of the dam of \( p \),

and

- \( e_{ijkl} \) = remaining random error after fitting other effects associated with the record of the \( p \)th calf.

**Mixed model equations**

Variance component estimation procedures used in this study are developed from mixed model methodology yielding best linear unbiased predictions (BLUP) of random effects other than error. Consequently, discussion of these procedures necessitate the use of linear sets of equations of progeny records denoted in matrix form. The model for Analysis I, defined in equation (5), is denoted in matrix notation as:

\[
y = Xb + Z_{1}u_{1} + Z_{2}u_{2} + e
\]
where

\[ y = \text{a known N x 1 vector of adjusted birth weight or weaning weight records.} \]

\[ b = \text{an unknown cg x 1 vector of fixed contemporary group effects, including the overall mean.} \]

\[ X = \text{a known N x ng incidence matrix which relates the elements of b to y.} \]

\[ u_1, u_2 = \text{unknown ns x 1 vectors of random effects for sire direct and sire maternal effect, respectively.} \]

\[ Z_1, Z_2 = \text{known N x ns incidence matrices that relate elements of } u_1 \text{ and } u_2 \text{ to } y \text{ (} Z_1 \text{ containing 1's, .5's, and 0's; } Z_2 \text{ containing 1's and 0's).} \]

\[ e = \text{an N x 1 vector of residual random errors, assumed } NID(0, \sigma_e^2). \]

Note that elements of \( u_1 \) represent direct effect evaluations for bulls represented as sires and maternal grandsires. Therefore, \( Z_1 \) was constructed to reflect all available progeny and maternal grandprogeny information as it relates to the direct effect of a bull. Three situations could occur in the data. First, a bull may be represented as a sire, thus a value of 1.0 is placed in the element of \( Z_1 \) that relates him to his progeny's record. Second, a bull may be represented as a maternal grandsire; hence, a value of .5 is placed in \( Z_1 \). Finally, it is possible that a bull is both the sire and maternal grandsire of a calf, and so a 1.5 is placed in \( Z_1 \) indicating that the direct effect of the calf's sire is represented in his phenotype, as is an additional 1/2 of the direct effect as his maternal grandsire. Because of the small number of sire-daughter matings represented in the data, the problems in estimation associated with records of inbred progeny were not considered in this study. If a greater percentage occurred, certainly the use of a relationship matrix which considered inbreeding would be mandated.
The $E(y) = (X\beta)$ and the variance of $y$ is defined as

$$V(y) = ZGZ' + R$$  \hspace{1cm} (7)

where

$$G = V(u) = \begin{bmatrix} \sigma_1^2 & \sigma_{12} \\ \sigma_{12} & \sigma_2^2 \end{bmatrix} \ast I, \ Z = (Z_1:Z_2), \ u = (u_1:u_2)'$$

and

$$R = V(e) = I\sigma_e^2.$$ 

Henderson (1984a,b) and Schaeffer (1983) found it useful to define the $V(y)$ in a linear form for the purpose of variance component estimation. For Analysis I then,

$$V(y) = \sum_{i=1}^{b} \sum_{j=1}^{b} Z_i' G_{ij} Z_j + \sum_{i=1}^{c} \sum_{j=1}^{c} R_{ij}$$  \hspace{1cm} (8)

where

- $b$ = the number of subvectors of $u$, (2 for this analysis)
- $c$ = the number of subvectors of $e$, (2 for this analysis)
- $Z = (Z_1:Z_2)$
- $Z_{ij}$ = the variance (or covariance) of $u_i$ with $u_j$
- $R_{ij}$ = the variance (or covariance) of $e_i$ with $e_j$
- $G_{11} = \begin{bmatrix} G_{11} & 0 \\ 0 & 0 \end{bmatrix}; \ G_{12} = \begin{bmatrix} 0 & G_{12} \\ G_{12} & 0 \end{bmatrix}$ etc.

$$R_{11} = \begin{bmatrix} R_{11} & 0 \\ 0 & 0 \end{bmatrix}; \ R_{12} = \begin{bmatrix} 0 & R_{12} \\ R_{12} & 0 \end{bmatrix}$$ etc.
and where each $b_{ij}$ and $R_{ij}$ corresponds to the $ij^{th}$ quadrant partition of $G$ or $R$, respectively. For example, $G_{11}$ corresponds to equations used to solve for $\hat{u}_1$.

Both analyses used in this study assume that $R_{ii} = I$. Because no environmental covariance was assumed to exist (a condition not true if $y$ results from a sire-daughter mating), the values for $R_{ij}$ and $r_{ij}$ are null, where $i \neq j$. Because of these assumptions, the $V(y)$ for these analyses is reduced to the form

$$V(y) = \sum_{i=1}^{2} \sum_{j=1}^{2} Z G_{ij} Z^\prime G_{ij} + I_N \sigma_0^2$$

or, in a more simple form

$$V(y) = \sum_{i=0}^{d} V_{i} \theta_i$$

where

$$\sigma_0^2 = \sigma_e$$

$$\theta_i = \sigma_i^2, \sigma_2^2, \text{ or } \sigma_{12} \text{ (for } i = 1-3, \text{ respectively)}$$

and

$$V_0 = I.$$

These analyses also assumed that sires were unrelated. Consequently, all $G_{ij}$ are equal to $I$. However, if sire relationships were considered, $G_{ij}$ would equal $A$, a numerator matrix of relationships among elements in $u_1$ and $u_2$. 
The application of Equation (9b) is perhaps more easily seen if the summations are carried through and the equation is written out. This gives us

\[ V(\gamma) = (Z_1:Z_2) \begin{bmatrix} G_{11} & 0 \\ 0 & 0 \end{bmatrix} \begin{bmatrix} Z_1^2 & (Z_1:Z_2) \\ Z_2^2 & \end{bmatrix} \begin{bmatrix} \sigma_1^2 + (Z_1:Z_2) \begin{bmatrix} Z_1^2 \\ Z_2^2 \end{bmatrix} \end{bmatrix} + (Z_1:Z_2) \begin{bmatrix} 0 & G_{12} \\ G_{12} & 0 \end{bmatrix} \begin{bmatrix} Z_1^2 \\ Z_2^2 \end{bmatrix} \sigma_{12} + I_N \sigma_0^2 \]  

and corresponds to Equation (9b) written out as:

\[ V(\gamma) = V_1 \theta_1 + V_2 \theta_2 + V_3 \theta_3 + V_0 \theta_0. \]  

The mixed model equations for the model defined by Equation (6) are:

\[
\begin{bmatrix}
X'X & X'Z_1 & X'Z_2 \\
Z_1'X & (Z_1'Z_1 & Z_1'Z_2) + G^{-1}\sigma_{12} \\
Z_2'X & (Z_2'Z_1 & Z_2'Z_2)
\end{bmatrix}
\begin{bmatrix}
\hat{b} \\
\hat{u}_1 \\
\hat{u}_2
\end{bmatrix}
= \begin{bmatrix}
X'\gamma \\
Z_1'\gamma \\
Z_2'\gamma
\end{bmatrix}
\]

where

\[ G = \begin{bmatrix}
\sigma_1^2 & \sigma_{12} \\
\sigma_{12} & \sigma_2^2
\end{bmatrix}
\]

- \( \sigma_1^2 \) = the variance of sire direct effects,
- \( \sigma_2^2 \) = the variance of sire maternal effects,
- \( \sigma_{12} \) = the covariance between sire direct and sire maternal effects.

The matrices \( Z_1'Z_1, Z_1'Z_2, Z_2'Z_1, \) and \( Z_2'Z_2 \) are each square, symmetrical, and of equal order \( n_s \) (the total number of sires). The \( X'X \) matrix is a square,
diagonal matrix with order equal to ng (the number of contemporary groups). Although not necessary, this study required that \( Z_1'Z_1 \) and \( Z_2'Z_2 \) be of equal order. Each sire received an estimate for both his direct and maternal effect. The requirement of ties across contemporary groups along with the use of the G matrix allowed fair comparisons of sires for both traits, even though some sires may not have maternal grandprogeny records available.

Absorption of fixed effects

The equations for fixed effects were absorbed into equations for random effects in order to make computations more manageable for finding the predictors in \( \hat{u} \). Absorption is a necessary feature of the variance component estimation procedures detailed later in this section and is accomplished by solving for the effects in \( \hat{u} \) in terms of the fixed effects in \( \hat{b} \).

The coefficient matrix of Equation (10) is partitioned into quadrants determined by fixed and random effects and is defined as

\[
\begin{bmatrix}
A & B \\
B' & D
\end{bmatrix}
\begin{bmatrix}
b' \\
\hat{u}
\end{bmatrix}
= 
\begin{bmatrix}
X'Y \\
Z'Y
\end{bmatrix}
\]

where

\[
A = X'X
\]

\[
B = X'Z
\]

and

\[
D = Z'Z + G^{-1} \sigma^2_0.
\]

Absorption of equations for \( \hat{b} \) into those for \( \hat{u} \) would result in the expression
By replacing the true expressions of A, B, C, and D, and after algebraic simplification, the full set of absorbed equations in matrix notation is defined as follows.

\[(D - B'(A^{-1})B)(\hat{\mathbf{u}}) = Z'Y - B'(A^{-1})X'Y.\]

or more specifically

\[
\begin{bmatrix}
Z'MZ_{11} & Z'MZ_{12} \\
Z'MZ_{21} & Z'MZ_{22}
\end{bmatrix}
+ G^{-1}
\begin{bmatrix}
1 & 0 \\
0 & 0
\end{bmatrix}
\begin{bmatrix}
\mathbf{u}_1 \\
\mathbf{u}_2
\end{bmatrix}
= \begin{bmatrix}
Z'MY_{11} \\
Z'MY_{22}
\end{bmatrix}
\tag{12b}
\]

where \(M\) is an idempotent matrix \((M'\mathbf{M} = \mathbf{I})\) and is defined as

\[
M = (I - X(X'X)^{-1}X').
\]

The mathematical algorithms used to form the partitioned segments of the coefficient matrix and right-hand sides of (12b) are given in Equations 13 through 17. Dot notation is used to describe the necessary computations performed for the absorption of fixed effects in Analysis I. Subscripts of variables follow the notation defined in Equation (5) but are redefined for clarity as they are used in the absorption formulae. Variables used in describing the absorption process are:

\[N_{ij} = \text{the total number of progeny in the } j^{\text{th}} \text{ group of the } i^{\text{th}} \text{ herd.}\]

\[N_{ij} = \text{the total number of progeny by sire } s \text{ in the } j^{\text{th}} \text{ group of the } i^{\text{th}} \text{ herd.}\]

\[N_{ij} = \text{the total number of maternal grandprogeny of } s \text{ in the } j^{\text{th}} \text{ group of the } i^{\text{th}} \text{ herd.}\]

\[N_{ij} = \text{the total number of progeny in group } j \text{ that are sired by } s \text{ and of maternal grandsire } s'.\]
\[ N_{ijss.} = \text{the total number of progeny in group } j \text{ whose sire is also their maternal grandsire.} \]

\[ Y_{ij...} = \text{the sum total of progeny records in the } j^\text{th} \text{ group of the } i^\text{th} \text{ herd.} \]

\[ Y_{ijs..} = \text{the sum total progeny records of sire } s \text{ in the } j^\text{th} \text{ group of the } i^\text{th} \text{ herd.} \]

\[ Y_{ij.s.} = \text{the sum total records of the maternal grandprogeny of } s \text{ in the } j^\text{th} \text{ group of the } i^\text{th} \text{ herd.} \]

\[ nh = \text{the total number of herds.} \]

\[ ng = \text{the total number of contemporary groups.} \]

With these definitions in mind, what follows are the algorithms used to perform the absorption process of this analysis.

\[ Z_{i}^{\prime}MZ_{i} = Z_{i}^{\prime}Z_{i} - Z_{i}^{\prime}X(X'X)^{-1}X'Z. \quad (13) \]

a. The \( s^\text{th} \) diagonal element of \( Z_{i}^{\prime}MZ_{i} \) is

\[
\sum_{i=1}^{nh} \left( N_{i.s..} + .25(N_{i..s.} + N_{i.ss.}) - \right.
\]

\[
\sum_{j=1}^{ng} \left[ \frac{N_{ijs..} + .5(N_{ij.s.})^2}{N_{ij...}} \right]
\]

b. The offdiagonal element of the \( s^\text{th} \) row and \( s^\text{th} \) columns of \( Z_{i}^{\prime}MZ_{i} \) is

\[
\sum_{i=1}^{nh} \sum_{j=1}^{ng} .5N_{ijss.} - \frac{[N_{ijs..} + .5N_{ij.s.}][N_{ijs..} + .5N_{ij.s.}]}{N_{ij...}}
\]

\( Z_{i}^{\prime}MZ_{i} \) is a symmetrical matrix of order \( ns \).
B. \[ Z_2^\prime Z_2 = Z_2^\prime Z_2 - Z_2^\prime X(X'X)^{-1}X'Z_2 \quad (14) \]

a. The \( s \)th diagonal element of \( Z_2^\prime Z_2 \) is

\[
\sum_{i=1}^{n_h} N_{i..s} - \sum_{j=1}^{n_g} \frac{(N_{ij.s})^2}{N_{ij..}} \]

b. The off-diagonal element of the \( s \)th row and \( s \)th column of \( Z_2^\prime Z_2 \) is

\[
\sum_{i=1}^{n_h} N_{i..s} (N_{ij.s}) (N_{ij.s'}) \quad \sum_{j=1}^{n_g} \frac{N_{ij..}}{N_{ij..}}
\]

\( Z_2^\prime Z_2 \) is a symmetrical matrix of order \( n_s \).

C. \[ Z_1^\prime Z_2 = Z_1^\prime Z_2 - Z_1^\prime X(X'X)^{-1}X'Z_2 \quad (15) \]

(note that \( Z_2^\prime Z_1 = (Z_1^\prime Z_2)^\prime \)).

a. The \( s \)th diagonal element of \( Z_1^\prime Z_2 \) is

\[
\sum_{i=1}^{n_h} .5N_{i.s..} + N_{i..s.s} - \sum_{j=1}^{n_g} \frac{(N_{ij..}) (N_{ij.s}) + .5(N_{ij.s})^2}{N_{ij..}} \]

b. The off-diagonal element of the \( s \)th row and the \( s \)th column of \( Z_1^\prime Z_2 \) is

\[
\sum_{i=1}^{n_h} \frac{[N_{ijs..} + .5N_{ijs..}] [N_{ij.s}s..]}{N_{ij..}} - \sum_{j=1}^{n_g} \frac{N_{ij..}}{N_{ij..}}
\]

\( Z_1^\prime Z_2 \) is a square but asymmetrical matrix of order \( n_s \).
The element of the vector $Z_1^{My}$ is defined as:

$$
Z_1^{My} = Z_1^{y} = Z_1^{X(X'X)^{-1}X'Y}
$$

The $s^{th}$ element of the vector $Z_1^{My}$ is defined as:

$$
\sum_{i=1}^{nh} y_{i.s.} + .5y_{i.s.} - \sum_{j=1}^{ng} \left[ N_{ijs..} + .5N_{ijs..} \right] / N_{ij...}
$$

The element of the vector $Z_2^{My}$ is defined as:

$$
Z_2^{My} = Z_2^{y} - Z_2^{X(X'X)^{-1}X'Y}
$$

The $s^{th}$ element of the vector $Z_2^{My}$ is defined as:

$$
\sum_{i=1}^{nh} (y_{ij.s.} - \sum_{j=1}^{ng} (N_{ij.s..}) / N_{ij...}
$$

**Quadratic forms and their expectations**

The method of variance component analysis selected is an adaptation of techniques discussed by Harville (1977) and Henderson (1980, 1984a). The method has been referred to as approximate-REML, MINQUE-D, approximate MINQUE, and Henderson's New Method in various publications. The method has the advantage over competing methods such as REML, MINQUE, and ML in that it is more computationally feasible for mixed models with large numbers of levels per random factor. The models used for this study have such characteristics. In contrast to ML, REML, and MINQUE procedures, the inversion of only diagonal and block diagonal coefficient matrices are required to yield unbiased estimates that are translation invariant. Further comparisons are made in the Literature Review section of this dissertation.
The quadratic forms used to estimate variance components for this study were computed by methods derived by LaMotte (1973). LaMotte proved that MINQUE of these components could be obtained by first computing

\[(y - Xb^*)' H^{-1} V_i H^{-1} (y - Xb^*), \quad i = 1 \text{ to } d.\]  

(18)

where

- \(y\) = the vector of individual records
- \(X\) = incidence matrix for fixed effects
- \(b^* = (X' H^{-1} X)^{-1} X' H^{-1} y\)
- \(H\) = prior value of \(V(y)\), the variance of \(y\)
  \[= \sum V_i \sigma^2_i \text{ (see Equations (9a)-(9c)).}\]
- \(d\) = the number of factors in \(H\).

These quadratics are then equated to their expectations and simultaneously solved for \(\sigma^2_i\).

Henderson (1984a) demonstrated that an equivalent form to Equation 18 exists which depicts the quadratic forms in terms of solutions to the mixed model equations. Henderson's forms are shown by replacing \(V_i\) with \(ZG^*Z'\) (taken from Equation (9b) defining the variance of \(y\), ignoring error variance) with the resulting quadratic forms defined by:

\[(y - Xb^*)' H^{-1} ZG^* G^{-1} G^* H^{-1} (y - Xb^*).\]  

(19)

where \(\hat{\gamma}\) = a priori values given to \(G\) (see Equation 18). It can be shown that \((y - Xb^*)' H^{-1} ZG = \hat{\gamma}^- (Henderson, 1984b). Hence, the general equation depicting the quadratic forms used in Analysis I are defined by
For example, when $G_{ij}^* = \begin{bmatrix} G_{11} & 0 \\ 0 & 0 \end{bmatrix} = \begin{bmatrix} I_{ns} & 0 \\ 0 & 0 \end{bmatrix}$, then the quadratic form to be computed is given as:

$$\frac{(u_1'u_1)/\sigma_1^2}{\sigma_1^2}$$

where $\sigma_1^2$ is the a priori value given to $\sigma_1^2$. Each $\sigma_1$ is considered a constant, and so when the quadratic form defined by $G_{ij}^*$ is equated to its expected value, the $\sigma_1^2$'s are cancelled. Using this methodology, the quadratic used to compute estimates for $\sigma_1^2$, $\sigma_2^2$, and $\sigma_{12}$ is $u_1'u_1$, $u_2'u_2$, and $u_1'u_2 + u_2'u_1$.

A fourth quadratic was needed in order to estimate error variance or $\sigma_0^2$. Henderson (1980) suggests that any logical estimator for $\sigma_0^2$ will suffice (e.g., within smallest subclass mean square), foregoing the need for another quadratic; certainly the preceding described derivations from LaMotte's (1973) quadratic forms will work for error terms as well (Henderson, 1984b). However, for computational simplicity, the quadratic

$$\gamma'M\gamma = \gamma'(I - X(X'X)^{-1}X')\gamma$$

was computed and equated to its expectation. Schaeffer (1983) suggested that it should yield equally reliable estimates as other quadratics that could be used to obtain an estimate for $\sigma_e^2$. The next section defines the expectations of these quadratic forms derived from approximate solutions for $\hat{u}$ and the quantity $\gamma'M\gamma$. 
In order to compute quadratics, one must first compute the values for \( \hat{u}_1 \) and \( \hat{u}_2 \) from the absorbed equations shown in Equation (12b). At this point, the coefficient matrix \((Z'MZ)\) would be inverted and premultiplied to the right-hand sides of \((12b)\) in order to obtain solutions for \( \hat{u} \). However, a direct inverse of the coefficient matrix is not computationally feasible if the order of \( Z'MZ \) is very large. Harville (1977) suggests that an alternative coefficient matrix be substituted which would be simpler to invert, yet still allow \( \hat{u} \) to approach \( u \). Henderson (1980), with an identical concept, provided algorithms which yield unbiased estimates of \( \sigma^2_1 \) from approximate solutions to a mixed model.

Henderson extended these concepts to a multi-trait situation in Henderson (1980, 1984a). The key to deciding what approximate solution for \( \hat{u} \) to use is given by the pattern of the elements in \( Z'MZ \). In the case of the absorbed equations presented in Equation (12b), diagonal dominance is likely to be exhibited in each of \( Z_1'MZ_1, Z_2'MZ_2, \) etc., so long as each sire or maternal grandsire has a rather sizeable number of progeny and/or maternal grandprogeny. Also, recall that \( \hat{u}_1 \) and \( \hat{u}_2 \) are of equal order; in other words, every sire represented receives an estimate for both sire direct and sire maternal effect. As a result, the absorbed mixed model equations for \( \hat{u}_1 \) and \( \hat{u}_2 \) can be rearranged such that they are ordered by effect within sire. These equations exhibit a clear 2 x 2 block diagonal dominance. Thus, the off-diagonal elements of \( Z_1'MZ_1, Z_1'MZ_2, Z_2'MZ_1, \) and \( Z_2'MZ_2 \) were disregarded for solutions to \( \hat{u} \). At this point, the equations used to approximate \( \hat{u}_1 \) and \( \hat{u}_2 \) appear as:
\[ u = D^{-1} R \]  

where

\[ u = \begin{bmatrix} u_1^2 \\ u_1^2 \\ u_2^2 \\ \vdots \\ u_s^2 \end{bmatrix} \]

\[ D^{-1} = \begin{bmatrix} d_{11} & d_{12} \\ d_{21} & d_{22} \\ \vdots & \vdots \\ d_{s1} & d_{s2} \end{bmatrix} \]

and

\[ R = \begin{bmatrix} Z_i M \gamma \\ Z_i M \gamma \\ \vdots \\ \vdots \\ Z_i M \gamma \end{bmatrix} \]
It should be made clear that:

\[ \hat{u}_{ij} \] = approximate estimate of the \( i^{th} \) sire's transmitting ability for the \( j^{th} \) trait,

\[ d_{11} = \text{the diagonal element of } Z_{11} + G_{11} \]

\[ d_{12} = \text{the diagonal element of } Z_{12} + G_{12} \]

\[ d_{22} = \text{the diagonal element of } Z_{22} + G_{22} \]

(\text{and so forth})

and that

\[ Z_{1s} \]

\[ Z_{2s} \]

are the elements of the right-hand sides that correspond to the \( s^{th} \) sire.

After the elements of \( D \) are formed, a direct inverse is easily computed as a series of block 2 x 2 inversions. These are in turn premultiplied to their corresponding right-hand sides and solutions for \( \hat{u} \) are computed.

The next step in the estimation of variance components involves determining the expectations of the quadratics provided by Equations (20) and (21). The general rules for developing expectations of quadratic forms can be found in Schaeffer (1983) along with numerous examples. The four quadratic forms considered for this analysis include \( \hat{u}_1\hat{u}_1, \hat{u}_2\hat{u}_2, 2\hat{u}_1\hat{u}_2, \) and \( \hat{y}\hat{y} \). The expectations of each are derived in the following paragraphs.

For the purpose of the following derivations, we restate Equation (22); however, in this case, the equations are simply reordered so that they appear sequenced sires within effect. These equations are given again as
\[ \mathbf{u} = \mathbf{D}^{-1} \mathbf{r} \]

but in this case:

\[ \mathbf{u}^2 = \begin{bmatrix} \mathbf{u}_1^2 \\ \mathbf{u}_2^2 \end{bmatrix} = \begin{bmatrix} \text{sire direct effects} \\ \text{sire maternal effects} \end{bmatrix} , \]

\[ \mathbf{D}^{-1} = \begin{bmatrix} \mathbf{D}_{11} & \mathbf{D}_{12} \\ \text{sym.} & \mathbf{D}_{22} \end{bmatrix} \]

and

\[ \mathbf{r} = \begin{bmatrix} \mathbf{r}_1 \\ \mathbf{r}_2 \end{bmatrix} = \begin{bmatrix} Z_{1}^\prime \mathbf{y} \\ Z_{2}^\prime \mathbf{y} \end{bmatrix} . \]

Four items should be noted in order to facilitate derivations of expectations.

1. Each quadrant of \( \mathbf{D} \) is a square, diagonal matrix. Thus, \( \mathbf{D}_{21} = \mathbf{D}_{12} \).
   Also,
   \[ \mathbf{u}_1 = (\mathbf{D}_{11}:\mathbf{D}_{12}) \mathbf{r} , \]
   and
   \[ \mathbf{u}_2 = (\mathbf{D}_{12}:\mathbf{D}_{22}) \mathbf{r} . \]

2. The variance of \( \mathbf{y} \), \( \mathbf{V}(\mathbf{y}) \), defined in Equation (9b), was given the general form
   \[ \mathbf{V} = \sum_{i=0}^{4} \mathbf{V}_i \sigma_i^2 , \quad \mathbf{V}_0 = \mathbf{I}_N . \]

3. The expectation of any general quadratic form is
   \[ \mathbf{E}(\mathbf{r}' \mathbf{A} \mathbf{r}) = \mathbf{E}(\mathbf{r}' \mathbf{r}) \mathbf{A} \mathbf{E}(\mathbf{r}) + \text{tr}(\mathbf{A} \mathbf{V}(\mathbf{r})) . \]
   when \( \mathbf{r} = Z^\prime \mathbf{y} \), the \( \mathbf{E}(\mathbf{r}) = 0 \), thus the \( \mathbf{E}(\mathbf{r}' \mathbf{A} \mathbf{r}) = \text{tr}(\mathbf{A} \mathbf{V}(\mathbf{r})) . \)
4. $M = (I - X(X'X)^{-1}X')$ is symmetric and idempotent; thus

$$MM^r = M.$$

With these in mind, the values of $E(u_1^ru_1^r)$, $E(u_2^ru_2^r)$, $E(u_1^ru_2^r + u_2^ru_2^r)$, and $E(y^rMy)$ are defined in as follows.

1. $E(u_1^ru_1^r)$  

$$E(u_1^ru_1^r) = E \left( r' \begin{bmatrix} D_{11} & D_{12} \\ D_{12} & D_{22} \end{bmatrix} r \right)$$

$$= \text{tr} \left( \begin{bmatrix} D_{11}^2 & D_{11} \cdot D_{12} \\ D_{11} \cdot D_{12} & D_{12}^2 \end{bmatrix} V(r) \right)$$

$$= \text{tr} [Q_1 V(r)]$$

2. $E(u_2^ru_2^r)$  

$$E(u_2^ru_2^r) = E \left( r' \begin{bmatrix} D_{12} & D_{22} \end{bmatrix} r \right)$$

$$= \text{tr} \left( \begin{bmatrix} D_{12}^2 & D_{12} \cdot D_{22} \\ D_{12} \cdot D_{22} & D_{22}^2 \end{bmatrix} V(r) \right)$$

$$= \text{tr} [Q_2 V(r)]$$

3. $E(2u_1^ru_2^r)$  

$$E(2u_1^ru_2^r) = E \left( r' \begin{bmatrix} D_{11} \cdot D_{12} & D_{11} \cdot D_{22} \\ D_{12}^2 & D_{12} \cdot D_{22} \end{bmatrix} r \right) + E \left( r' \begin{bmatrix} D_{12} \cdot D_{11} & D_{12}^2 \\ D_{22} \cdot D_{11} & D_{22} \cdot D_{11} \end{bmatrix} r \right)$$

$$= \text{tr} \left( \begin{bmatrix} 2(D_{11} \cdot D_{12}) & ((D_{11} \cdot D_{22}) + D_{12}^2) \\ (D_{11} \cdot D_{22}) + D_{12}^2 & 2(D_{22} \cdot D_{12}) \end{bmatrix} V(r) \right)$$

$$= \text{tr} [Q_3 V(r)]$$
4. $E(y'y)$

\[ = E(y'y) - E(y'X(X'X)^{-1}X'y) \]
\[ = \text{tr}(Z_1'MZ_1)\sigma_1^2 + \text{tr}(Z_2'MZ_2)\sigma_2^2 \]
\[ + 2\text{tr}(Z_1'MZ_2)\sigma_3^2 + N - r(x)\sigma_0^2 \]

where

\[ r(X) = \text{the rank of } X \text{ which equals the number of fixed effects in } b \]

and

\[ N = \text{the number of elements in } y. \]

In the first three quadratics, the $V(r)$ appears and is defined as:

\[ V(r) = V(Z'y) = Z'MVMZ \]  \hspace{1cm} (27a)

where $V$ is the variance of $y$. By replacing $V$ with its equivalent, $V(r)$ may now be defined as

\[ V(r) = \sum_{i=0}^{3} K_i \sigma_i^2 \]  \hspace{1cm} (27b)

where

\[ K_1 = Z_1'MZ_1 Z_1'MZ \]
\[ = \begin{bmatrix} Z_1'MZ_1 \cdot Z_1'MZ_1 & Z_1'MZ_1 \cdot Z_1'MZ_2 \\ Z_2'MZ_1 \cdot Z_1'MZ_1 & Z_2'MZ_1 \cdot Z_1'MZ_2 \end{bmatrix}, \]
\[ K_2 = Z'MZ_2^2MZ \]
\[ = \begin{bmatrix} Z_1'^2M_2^2Z_2Z_2Z_2 + Z_1Z_2Z_2M_2^2Z_2Z_2Z_2 \\ Z_2^2M_2^2Z_2Z_2^2 + Z_1Z_2Z_2M_2^2Z_2Z_2Z_2 \end{bmatrix} \]

\[ K_3 = Z'M[Z_1Z_2^2 + Z_2Z_1^2]MZ \]
\[ = \begin{bmatrix} (Z_1'^2M_2^2Z_1Z_1 + Z_1Z_2Z_2Z_1)(Z_2^2M_2^2Z_2Z_2 + Z_1Z_2Z_2M_2^2Z_2Z_2) \\ (Z_2^2M_2^2Z_2Z_2Z_1 + Z_1Z_2Z_2Z_1)(Z_2^2M_2^2Z_2Z_2 + Z_1Z_2Z_2M_2^2Z_2Z_2) \end{bmatrix} \]

and

\[ K_0 = Z'MZ \]
\[ = \begin{bmatrix} Z_1'^2M_1Z_1 \\ Z_2Z_2M_2 \end{bmatrix} \]

Having defined \( V(r) \) in terms of the variance components \( \sigma_i^2 \), we can now form a set of equations that equate the expectations of the quadratic forms to their computed values and solve simultaneously for \( \sigma_i^2 \). For example, the \( E(\hat{u}_1\hat{u}_1) \), equated to its computed value \( \hat{u}_1\hat{u}_1 \), is:

\[ \hat{u}_1\hat{u}_1 = \text{tr}(Q_1K_1)\sigma_1^2 + \text{tr}(Q_2K_2)\sigma_2^2 + \text{tr}(Q_3K_3)\sigma_3^2 + \text{tr}(Q_4K_4)\sigma_0^2 \]

The final form of each quadratic equated to its expectation used for Analysis I is given as:
The computation necessary to quantify the values in (28) are anything but easy. The absorption is in itself a difficult task, and equation (28) shows that traces of products of numerous large matrices are required. However, computer work may be simplified by taking advantage of the fact that only the diagonal elements of each product forming the quadrants of each $K_i$ need be calculated. Also, each $K_i$ matrix is symmetrical; therefore, the lower left quadrant of each is considered by simply multiplying the upper right quadrant's diagonal elements by 2. For example, the term $\text{tr}(Q_i K_i)$ in (28) may be written out as:

$$\begin{bmatrix}
\text{tr}(Q_1 K_1) & \text{tr}(Q_1 K_2) & \text{tr}(Q_1 K_3) & \text{tr}(Q_1 K_0) \\
\text{tr}(Q_2 K_1) & \text{tr}(Q_2 K_2) & \text{tr}(Q_2 K_3) & \text{tr}(Q_2 K_0) \\
\text{tr}(Q_3 K_1) & \text{tr}(Q_3 K_2) & \text{tr}(Q_3 K_3) & \text{tr}(Q_3 K_0) \\
\text{tr}(Z_1^T M Z_1) & \text{tr}(Z_2^T M Z_2) & 2\text{tr}(Z_1^T M Z_2) & (N-r(x))
\end{bmatrix}
\begin{bmatrix}
\hat{\sigma}_1^2 \\
\hat{\sigma}_2^2 \\
\hat{\sigma}_{12}^2 \\
\hat{\sigma}_0^2
\end{bmatrix}
= 
\begin{bmatrix}
\hat{u}_1^2 \hat{u}_1 \\
\hat{u}_2^2 \hat{u}_2 \\
2\hat{u}_1 \hat{u}_2 \\
\gamma \gamma
\end{bmatrix}
\quad (28)$$

where only the diagonal elements of each product of the portions of $Z^T M Z$ are needed.

First round (unbiased) and iterative solutions were computed for all data sets and as reported in Results and Discussion.
Variance Component Estimation - Analysis II

The possibility exists that the covariance between sire and maternal grandsire effects are zero or that errors introduced to one effect cause bias in the estimation of other effects or their variances. In this case, separate evaluations for the variances of $u_1$ and $u_2$ are justified. Also, the variance components obtained from Analysis I are not appropriate for evaluations where sires and maternal grandsires are evaluated through separate models. Crow and Howell (1982) estimated the total heritability of a maternal grandsire’s contribution to his daughter’s offspring. This contribution includes both a sample one-fourth of his genes determining the direct effect and a sample one-half of his genes determining maternal effect expressed through the mothering ability of his daughter in the phenotype of his maternal grandoffspring.

Their mixed model analyses of calf weaning weights included fixed effects and a random effect of the maternal grandsire. Heritability of this effect, $h^2_{mgs}$, was computed as

$$h^2_{mgs} = \frac{4\hat{a}^2_{mgs}}{a^2_{mgs} + e^2}$$

Burfeneng et al. (1981) used a similar model to that of Crow and Howell and estimated both sire direct effect and maternal grandsire effect ($\hat{u}_s$ and $\hat{u}_{mgs}$) for birth weight. Two analyses were run with the same model and data base, once with the sire and the next with maternal grandsire in the model. Philipsson (1976) performed a similar analysis on Swedish Holstein birth weight records.
In a similar fashion, the purpose of Analysis II is to estimate the variance of sire effects ($\sigma_s^2$) and the variance of total maternal grandsire effects ($\sigma_{mgs}^2$) from separate analyses of the data. One model is defined which applies to both analyses. Also, methods used for variance component estimation in Analysis II are simply a modification of those used for Analysis I. Therefore, the details of method development have been left to those described for I and a more abbreviated discussion is presented for II.

**Model description and mixed model equations**

The general model defining a phenotypic record $y$ used in Analysis II is

$$y_{ijkp} = \mu + c_{gi} + s_k + e_{ijkp}, \quad (29)$$

where

- $\mu$ = the overall progeny record mean,
- $c_{gi}$ = the fixed effect of the $i$th group in the $j$th herd,
- $s_k$ = the random effect from the (1) $k$th sire or (2) $k$th maternal grandsire of $p$,

and

- $e_{ijkp}$ = remaining random error, NID(0, $\sigma_e^2$) of the phenotypic record of the $p$th individual in the $i$th group and $j$th herd and either (1) sired by the $k$th sire or (2) born to the daughter of the $k$th sire.

In matrix notation, Equation 29 is expressed as

$$y_i = X_i b_i + Z_i u_i + e_i, \quad i = s \text{ or } mgs \quad (30)$$
where

\[ y_i \] = a known \( N_i \times 1 \) vector of adjusted progeny records for birth or weaning weight.

\[ b_i \] = an unknown \( c_{g_i} \times 1 \) vector of fixed effects including the overall mean.

\[ X_i \] = a known \( N_i \times n_{g_i} \) incidence matrix which relates the elements of \( b_i \) to \( y_i \).

\[ u_i \] = an unknown \( n_s \times 1 \) vector of random effects measuring either
   \( i = s: \) sire's transmitting ability for the direct effect
   \( i = mgs: \) the sum of a sire's transmitting ability for maternal effects plus one-half of his transmitting ability for direct effect.

\[ Z_i \] = a known \( N_i \times n_{s_i} \) incidence matrix which relates the elements of \( u_i \) to \( y \).

\[ e_i \] = a \( N_i \times 1 \) vector of residual random effects assumed NID \((0, \sigma^2_{e_i})\) remaining after \( i = s: \) fixed and sire effects are removed or \( i = mgs: \) fixed and maternal grandsire effects are removed.

and \( i \) represents

\( s: \) a sire evaluation based on his progeny records in \( y_s \).

or

\( mgs: \) a maternal grandsire evaluation based on his maternal grandprogeny records in \( y_{mgs} \).

\( N_i \) = the number of progeny records considered in evaluation \( i \)

\( n_{g_i} \) = the number of fixed effects considered in evaluation \( i \)

\( n_{s_i} \) = the number of either (1) sires or (2) maternal grandsires estimated in evaluation \( i \).

Again, the \( E(y_i) = X_i b_i \) and the variance of \( y \) will be defined as:

\[ V(y_i) = Z_i G_i Z_i^T + R_i \quad (i = s \text{ or } mgs), \]
where
\[ G_i = V(u_i) = I_{N_i} \cdot \sigma^2_u, \]
and
\[ R_i = V(e_i) = I_{N_i} \cdot \sigma^2_e. \]

The mixed model equations formed from (30), after absorption equations for fixed effects, are shown as:

\[ [Z^M Z_i + G_i^{-1} \cdot \sigma^2_e][\hat{u}_i] = [Z^M y_i] \quad (31) \]

When sires are estimated, \( \hat{u}_i \) represents a measure of \( 1/2g_A \) or one-half the sire's additive genetic value for direct effect. However, a maternal grandsire evaluation yields a best linear unbiased prediction of \( 1/2g_M + 1/4g_A \), where \( g_M \) is the additive genetic value of the sire for maternal effect. The two terms are inseparable in this evaluation because the growth record of a calf is comprised of a confounded genotype and maternal influence. In Analysis I, the relationship between these confounded effects is assumed known to proportionality. Use of a covariance prior is made to appropriate a portion of maternal grandprogeny's record towards the direct effect evaluation of his maternal grandsire. The separate evaluation of sires and maternal grandsires such as in Analysis II does not allow this. However, a separate evaluation of maternal grandsires is warranted when bulls are selected that will sire daughters with improved total performance as mothers. The variances computed in Analysis II are useful when such evaluations are made.
Quadratic forms and their expectations

Using methods described for Analysis I, quadratic forms were computed from solutions to the mixed model equations given in Equation (31). For \( i = s \) (the evaluation of sire direct effects), the quadratic forms required for estimation for \( \sigma_{s}^2 \) and \( \sigma_{e}^2 \) were defined as

\[
\begin{align*}
(1) & \quad V_{s}V_{s}^{\prime} \quad , \\
(2) & \quad y_{s}^{\prime}M_{s}y_{s} .
\end{align*}
\]

Expectations to these quadratic forms were developed and were equated to their computed value. These equations were given as follows.

\[
tr(D_{s}^{2})(Z_{s}^{\prime}M_{s}Z_{s}) \quad tr(D_{s}^{2})(Z_{s}^{\prime}M_{s}Z_{s}) \\
tr(Z_{s}^{\prime}M_{s}Z_{s}) \quad N_{s} - r(X_{s})
\]

\[
\begin{bmatrix}
\sigma_{s}^{2} \\
\sigma_{e}^{2}
\end{bmatrix} = \begin{bmatrix}
\frac{\nu_{s}\gamma_{s}}{u_{s}u_{s}} \\
\frac{\nu_{s}\gamma_{s}}{u_{s}u_{s}}
\end{bmatrix}
\]

where

\[
D_{s} = \text{the inverse of a matrix formed from the diagonal elements of } Z_{s}^{\prime}M_{s}Z_{s} + G^{-1}s, \\
r(X_{s}) = \text{the number of fixed effects defined for the evaluation of sires (others defined previously). }
\]

Computations involved while forming Equation 33 are simplified because \( D_{s}^{2} \) is a diagonal matrix. Hence, only the diagonal elements of \( (Z_{s}^{\prime}M_{s}Z_{s})^{2} \) are required. Identical equations are defined for \( \sigma_{mgs}^{2} \) and \( \sigma_{emgs}^{2} \) by substituting mgs for s in the subscripts found in Equation (33).
These equations were solved simultaneously, and unbiased estimates of $\sigma_s^2$ and $\sigma_e^2$ were obtained from first round estimates. These and final iterative solutions to these equations are reported in Results and Discussion for all data sets defined.

Genetic Parameter Estimation

The preceding section describes methodology used to obtain variance components which can be equated to their expected genetic causal components. What follows is a description of procedures used to compute these genetic variance components, as well as heritabilities and genetic correlations for additive direct and additive maternal effects, as well as the heritability of the maternal grandsire's contribution to the maternal ability of his daughter's performance as mothers. The discussion is in order of analysis.

Analysis I

Recall that for Analysis I a mixed model was presented that partitioned the phenotype of a calf into sire direct effect, one-half maternal grandsire direct effect, maternal grandsire maternal effect, and error. Other effects such as dominance, epistasis, and permanent environmental influences were assumed as part of the error term. Linear mixed model techniques described by Henderson (1953, 1972) were used. Best linear unbiased predictions for sire direct and sire maternal effects were computed by approximating the inverse of the coefficient matrix after the equations for fixed effects were absorbed.
Variances and covariances were then obtained by employing an approximate MINQUE variance component estimation procedure. Quadratics of the approximate BLUP solutions for sire direct ($\hat{u}_s^*$) and sire maternal ($\hat{u}_m^*$) effects were equated to their expectations to solve for sire direct variance ($\hat{\sigma}_s^2$) and sire maternal variance ($\hat{\sigma}_m^2$). The values $\hat{u}_s^*$ and $\hat{u}_m^*$ represent estimates of one-half the additive genetic value of a sire for his direct or maternal genetic contribution to his offspring. If we define:

$$g_A = \text{the additive genetic value of a sire for direct effect},$$

and

$$g_M = \text{the additive genetic value for his maternal effect},$$

then

$$E(\hat{u}_s^*) = 1/2g_A^* ,$$

and

$$E(\hat{u}_m^*) = 1/2g_M^* .$$

The expected variances are then given as

$$E(\hat{u}_s^2) = 1/4E(g_A^2) = 1/4\sigma_A^2$$

and

$$E(\hat{u}_m^2) = 1/4E(g_m^2) = 1/4\sigma_M^2 ,$$

and the expected genetic covariance is given as

$$E(\hat{u}_s^*) (\hat{u}_m^*) = (1/2)(1/2)E(g_A^*, g_M^*) = 1/4\sigma_{A,M}^2 .$$
The variance of $y$, the phenotypic value of a calf, was defined by (9b); thus, for a single record, $V(y)$ is

$$V(y) = \sigma_s^2 + \sigma_{mg}^2 + \sigma_{s.mg}^2 + \sigma_e^2,$$

(39)

The expected values of these variances are defined as

$$E(\hat{\sigma}_s^2) = (1/4)\sigma_A^2$$

(40)

$$E(\hat{\sigma}_{mg}^2) = (1/4)\sigma_M^2$$

(41)

$$E(\hat{\sigma}_{s.mg}^2) = (1/4)\sigma_{A.M}^2$$

(42)

and

$$E(\hat{\sigma}_e^2) = (3/4)\sigma_A^2 + (3/4)\sigma_M^2 + (3/4)\sigma_{A.M}^2 + \sigma_E^2,$$

(43)

where

$\sigma_A^2$ = the additive genetic variance of direct effect on $y$.

$\sigma_M^2$ = the additive genetic variance of maternal effect on $y$.

$\sigma_{A.M}^2$ = the additive genetic covariance between direct and maternal effects influencing $y$.

$\sigma_E^2$ = the environmental influences on $y$, as well as any effects due to dominance or permanent environmental influences on the dam of $y$.

It follows that $\sigma_A^2$, $\sigma_M^2$, and $\sigma_{A.M}^2$ from Analysis I are estimated as $4\hat{\sigma}_s^2$, $4\hat{\sigma}_{mg}^2$, and $4\hat{\sigma}_{s.mg}^2$, respectively. The theoretical formulation for the heritability of direct effect is finally defined as:
\[ h_A^2 = \frac{\sigma_A^2}{\sigma_A^2 + \sigma_M^2 + \sigma_{A\cdot M}^2 + \sigma_E^2}, \quad (44) \]

and is estimated by:

\[ h_A^2 = \frac{4\sigma_s^2}{(4\sigma_s^2 + \sigma_{mg}^2 + \sigma_{s\cdot mg}^2 + \sigma_e^2)}. \quad (45) \]

The heritability for sire maternal effect is identically formulated as for sire direct effect as in (44) and (45) by simply replacing \( \sigma_A^2 \) with \( \sigma_M^2 \) and \( \sigma_s^2 \) with \( \sigma_{mg}^2 \) in the numerators of the two expressions.

Finally, the additive genetic correlation between direct and maternal

was defined by the formula:

\[ r_{A\cdot M} = \frac{\sigma_{A\cdot M}}{\sigma_A \cdot \sigma_M} \quad (46) \]

and was computed using the formula

\[ r_{A\cdot M} = \frac{\sigma_{s\cdot mg}^2}{(\sigma_s^2 \cdot \sigma_{mg}^2)^{1/2}} \quad (47) \]

Analysis II

A mixed model for Analysis II was described where the phenotypic

record of a calf was equated to certain fixed effects, random remaining

error, and either a sire or a maternal grandsire effect. In other words,

two analyses were run for the same data, one where the model included the
direct effect of the sire and the other where the total effect of the

grandsire (both direct and maternally through his daughters) was included.
Estimates of genetic parameters were computed from Analysis II following the logic presented for Analysis I.

First, we defined \( \hat{\nu}_s \) to estimate \( 1/2g_A \), or one-half of the additive direct effect for a sire, and \( \hat{\nu}_{mgs} \) to estimate \( 1/4g_A + 1/2g_M \), which is the total effect of a sire on his maternal grandoffspring. Therefore,

\[
E(\hat{\nu}_s^2) = E(\hat{\sigma}_s^2) = E(1/2g_A)^2 = 1/4\sigma_A^2
\]

and

\[
E(\hat{\nu}_{mgs}^2) = E(\hat{\sigma}_{mgs}^2) = E(1/4g_A + 1/2g_M)^2 = 1/16\sigma_A^2 + 1/4\sigma_M^2 + 1/4\sigma_{A,M}^2.
\]

As for the error terms \( e_s \) and \( e_{mgs} \) (from models which include a sire or maternal grandsire effect, respectively)

\[
E(e_s^2) = E(\hat{\sigma}_s^2) = (3/4)\sigma_A^2 + \sigma_E^2
\]

and
Separate heritabilities were computed for both additive direct effect \( h^2_A \) as well as the \( h^2_{mgs} \), which is termed the heritability of maternal grandsire effect. The maternal grandsire effect measures a sire's contribution to his daughter's performance as a mother (Crow and Howell, 1982). The estimated parameters for both were calculated from Analysis II as

\[
\hat{h}^2_A = \frac{4\sigma^2_s}{\hat{\sigma}^2_s + \hat{\sigma}^2_e_s} \tag{52}
\]

and

\[
\hat{h}^2_{mgs} = \frac{4\sigma^2_{mgs}}{\hat{\sigma}^2_{mgs} + \hat{\sigma}^2_e_{mgs}} \tag{53}
\]

and have theoretical compositions of

\[
\frac{\sigma^2_A}{\sigma^2_A + \sigma^2_E} \tag{54}
\]

and

\[
\frac{1/4\sigma^2_A + \sigma^2_M + \sigma^2_{A\cdot M}}{\sigma^2_A + \sigma^2_M + \sigma^2_{A\cdot M} + \sigma^2_E} \tag{55}
\]

for \( h^2_A \) and \( h^2_{mgs} \), respectively.
RESULTS AND DISCUSSION

The previous sections present the methodology used to estimate sire-direct and sire maternal variance components for birth and weaning weight from Angus and Hereford field data. Two mixed models were applied to each trait. Analysis I partitioned the direct and maternal genetic contributions of a sire to his daughter's progeny records and allows for the estimation of the covariance between sire direct and sire maternal effects \( \hat{\sigma}_{s-mg}^2 \). Analysis II estimated direct sire effect variance \( \hat{\sigma}_s^2 \) and the maternal grandsire effect variance \( \hat{\sigma}_{mgs}^2 \) from a model which considered one of these two random effects exclusive of the other.

The major purpose of this study was to compute variance components applicable to mixed model sire evaluations currently being developed for the American Angus and American Hereford Associations. Computational difficulty has prevented previous estimations of these variance components from sufficiently large enough numbers of sires to keep sampling errors of the estimates small. The procedures developed for this purpose are quite feasible on large data sets, although very little is known of its properties and behavior.

Discussion of this study is presented in two parts. First, follows a discussion of the methods employed for variance component estimation, including sensitivity to priors, as well as the effects of iteration. Secondly, variance component estimates from these procedures are presented as are the estimated genetic population parameters.
Estimation Procedures

A variation of MINQUE-D, otherwise called Henderson's new method (Henderson, 1980, 1984a), was used to estimate variance components from solutions to linear mixed model equations. This method was selected for this study because:

1. The computational simplicity of MINQUE-D was required because of the large number of sires and maternal grandsires in the population.

2. MINQUE-D holds a high degree of efficiency for estimation when compared to competing methods (Henderson, 1980; Kennedy, 1981; Dempfle et al., 1983).

Estimates derived from this study may be used for the evaluation of sires and maternal grandsires with national sire evaluation programs. Considerable reduction in the number of sires and maternal grandsires estimated would certainly allow the use of other, more precise methods such as MINQUE or REML. However, limited editing was performed so that estimates derived would be a true reflection of the population to which they will be applied. Simulation work is needed to determine a method of sampling that would reduce numbers of sires, yet still maintain population properties.

One of the major drawbacks of using MINQUE-D is a lack of knowledge concerning its properties. Henderson (1980) tells us that unbiased, translation invariant estimates are derived from initial solutions to the MINQUE-D equations. But the values of these first-round solutions are dependent upon the choice of priors, priors that are assumed known but usually are not. If the variance analysis procedure is robust to priors, then choice of priors is not critical.
In order to determine how sensitive the analyses were to priors, a range of reasonable priors was chosen, and first round solutions from Analysis I were examined for differences. Both birth and weaning weight records from the Angus data set were evaluated. Prior values used for $\sigma_s^2$, $\sigma_{mg}^2$, and $\sigma_{s.mg}^2$ only need to be known to proportionality with each other and with $\sigma_e^2$ when applied to these mixed model equations. Therefore, priors were computed from two sets of heritabilities for direct and maternal effect and from a range of estimated genetic correlations. Tables 5 and 6 list the first round solutions and percents of total variance for each change in priors.

Regardless of priors chosen, estimated sire direct variance ($\sigma_s^2$) was generally larger than estimated sire maternal variance ($\sigma_{mg}^2$). But unlike the results of Hudson and Van Vleck (1982), considerable differences in first round estimates were observed with a change in priors. Estimated sire direct variance generally comprised a greater percent of total estimated variability than did maternal effect variance. This is logical in those cases where $h_A^2 > h_M^2$ because solutions to the mixed model equations are regressed toward zero for incomplete heritability ($h^2 < 1.0$). However, even when $h_A^2 < h_M^2$, estimates for $\sigma_s^2$ were greater than for $\sigma_{mg}^2$, except for weaning weight when a large negative prior correlation was chosen. In these cases, a larger sire maternal effect variance than direct variance was computed. Large generalized conclusions cannot be drawn about the effect of prior heritabilities chosen because only two of many possible combinations were tested. Nonetheless, differences are observed in first round estimates, differences caused by choice of priors.
Table 5. Effect of priors on initial estimates of variance components for Analysis I on Angus birth weights\textsuperscript{a,b}

<table>
<thead>
<tr>
<th>(r_{A-M}^2)</th>
<th>(\hat{\sigma}_s^2)</th>
<th>(\hat{\sigma}_{mg}^2)</th>
<th>(\hat{\sigma}_{s\cdot mg}^2)</th>
<th>(\hat{\sigma}_e^2)</th>
<th>(\hat{\sigma}_s^2)</th>
<th>(\hat{\sigma}_{mg}^2)</th>
<th>(\hat{\sigma}_{s\cdot mg}^2)</th>
<th>(\hat{\sigma}_s^2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>(h_A^2 = .25)</td>
<td>(h_M^2 = .10)</td>
<td>(h_A^2 = .10)</td>
<td>(h_M^2 = .25)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>-.50 (%)</td>
<td>5.58 (8.0)</td>
<td>1.68 (2.4)</td>
<td>0.59 (.85)</td>
<td>61.97 (88.8)</td>
<td>5.81 (8.3)</td>
<td>3.31 (4.7)</td>
<td>-1.01 (-1.4)</td>
<td>61.63 (88.4)</td>
</tr>
<tr>
<td>-.25 (%)</td>
<td>6.01 (8.6)</td>
<td>1.4 (2.0)</td>
<td>1.09 (1.6)</td>
<td>61.32 (87.8)</td>
<td>5.94 (8.5)</td>
<td>2.57 (3.7)</td>
<td>-0.17 (-.2)</td>
<td>61.44 (88.0)</td>
</tr>
<tr>
<td>0.00 (%)</td>
<td>6.41 (9.2)</td>
<td>1.37 (2.0)</td>
<td>1.40 (2.0)</td>
<td>60.71 (86.9)</td>
<td>6.16 (8.8)</td>
<td>2.08 (3.0)</td>
<td>0.49 (.7)</td>
<td>61.10 (87.6)</td>
</tr>
<tr>
<td>+.25 (%)</td>
<td>6.78 (9.7)</td>
<td>1.45 (2.1)</td>
<td>1.59 (2.3)</td>
<td>60.08 (86.0)</td>
<td>6.40 (9.7)</td>
<td>1.88 (2.7)</td>
<td>0.98 (1.4)</td>
<td>60.62 (86.7)</td>
</tr>
<tr>
<td>+.50 (%)</td>
<td>7.14 (10.2)</td>
<td>1.72 (2.5)</td>
<td>1.63 (2.3)</td>
<td>59.45 (85.0)</td>
<td>6.64 (9.5)</td>
<td>2.00 (2.9)</td>
<td>1.26 (1.8)</td>
<td>60.01 (85.8)</td>
</tr>
</tbody>
</table>

\(s = \) direct sire effect; \(mg = \) maternal sire effect; \(e = \) error; \(A = \) direct additive genetic value; \(M = \) maternal additive genetic value; \(a\) indicates prior value; \(\wedge\) indicates estimate.

Because the effects of prior heritabilities are not constant across prior correlations suggests that an interaction exists between the influence that prior variances and covariances have on first round solutions. When \(h_A^2 > h_M^2\), little change is seen in \(\hat{\sigma}_{mg}^2\) when the prior correlation is changed (this is true for both traits). However, greater changes are created in \(\hat{\sigma}_{mg}^2\) when \(h_A^2 < h_M^2\). There seems to exist a particular combination of priors that causes the resulting \(\hat{\sigma}_{mg}^2\) to be at a minimum. For example, if \(h_A^2\) and \(h_M^2\) were held constant at .25 and .10, respectively, for birth
Table 6. Effect of priors on initial estimates of variance components for Analysis I on Angus weaning weights\(^a, b\)

<table>
<thead>
<tr>
<th>(r_{A\cdot M})</th>
<th>(\hat{\sigma}_s^2)</th>
<th>(\hat{\sigma}_{mg}^2)</th>
<th>(\hat{\sigma}_{s\cdot mg}^2)</th>
<th>(\hat{\sigma}_e^2)</th>
<th>(\hat{\sigma}_s^2)</th>
<th>(\hat{\sigma}_{mg}^2)</th>
<th>(\hat{\sigma}_{s\cdot mg}^2)</th>
<th>(\hat{\sigma}_e^2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>(-.50) (%)</td>
<td>121.1</td>
<td>100.3</td>
<td>-.10</td>
<td>2062.3</td>
<td>131.7</td>
<td>150.9</td>
<td>-48.8</td>
<td>2037.7</td>
</tr>
<tr>
<td></td>
<td>(5.3)</td>
<td>(4.4)</td>
<td>(.5)</td>
<td>(90.8)</td>
<td>(5.8)</td>
<td>(6.6)</td>
<td>(-2.1)</td>
<td>(89.7)</td>
</tr>
<tr>
<td>(-.25)</td>
<td>129.0</td>
<td>97.1</td>
<td>0.4</td>
<td>2046.8</td>
<td>132.4</td>
<td>136.6</td>
<td>-30.9</td>
<td>2034.5</td>
</tr>
<tr>
<td></td>
<td>(5.7)</td>
<td>(4.3)</td>
<td>(.02)</td>
<td>(90.0)</td>
<td>(5.9)</td>
<td>(6.0)</td>
<td>(-1.4)</td>
<td>(89.5)</td>
</tr>
<tr>
<td>0.00</td>
<td>138.5</td>
<td>95.2</td>
<td>7.7</td>
<td>2031.8</td>
<td>135.6</td>
<td>126.1</td>
<td>-15.8</td>
<td>2027.7</td>
</tr>
<tr>
<td></td>
<td>(6.1)</td>
<td>(4.2)</td>
<td>(.34)</td>
<td>(89.4)</td>
<td>(6.0)</td>
<td>(5.5)</td>
<td>(-.7)</td>
<td>(89.2)</td>
</tr>
<tr>
<td>(+.25)</td>
<td>146.9</td>
<td>96.5</td>
<td>12.9</td>
<td>2017.1</td>
<td>139.6</td>
<td>120.3</td>
<td>-3.3</td>
<td>2018.1</td>
</tr>
<tr>
<td></td>
<td>(6.5)</td>
<td>(4.2)</td>
<td>(.57)</td>
<td>(88.7)</td>
<td>(6.1)</td>
<td>(5.3)</td>
<td>(-.15)</td>
<td>(88.7)</td>
</tr>
<tr>
<td>(+.50)</td>
<td>156.5</td>
<td>99.4</td>
<td>16.4</td>
<td>2001.7</td>
<td>143.2</td>
<td>119.5</td>
<td>6.7</td>
<td>2006.5</td>
</tr>
<tr>
<td></td>
<td>(6.9)</td>
<td>(4.4)</td>
<td>(.72)</td>
<td>(88.0)</td>
<td>(6.3)</td>
<td>(5.3)</td>
<td>(.3)</td>
<td>(88.2)</td>
</tr>
</tbody>
</table>

\(^a\) Variance components reported as pounds

\(^b\) s = direct sire effect; mg = maternal sire effect; e = error; A = direct additive genetic value; M = maternal additive genetic value; \(^\circ\) = indicates prior value; \(^\wedge\) = indicates estimate.

weight, a minimum value for \(\hat{\sigma}_{mg}^2\) is found when \(r_{A\cdot M}\) is chosen to be near zero. When \(h_A^2 = .10\) and \(h_M^2 = .25\), this minimum occurs at about \(r_{A\cdot M} = .25\). In general for both traits, this low point value for \(\hat{\sigma}_{mg}^2\) shifts toward greater positive values of \(r_{A\cdot M}\) as \(h_A^2\) is made greater than \(h_M^2\).

Also, in the case of weaning weight, where maternal effects have a greater influence, this low point for \(\hat{\sigma}_{mg}^2\) is found to be at greater positive values for \(r_{A\cdot M}\) than for birth weight.
This behavior might be explained by reasoning that a fixed amount of phenotypic variance is partitioned out to a number of random effects. If sire variance is estimated to be quite large, then a component will necessarily need to be small in order for the sum of the components to equal the whole. Now at higher positive values of $r_{A-M}$, the percentage of $\hat{\sigma}_s^2$, $\hat{\sigma}_{mg}^2$, and $\hat{\sigma}_{s-mg}^2$ grows larger but at the expense of a decreasing $\hat{\sigma}_e^2$. This also seems reasonable as an increase in knowledge of the correlation between sire and maternal effect should allow us to explain a greater proportion of the phenotypic variance.

When a change is made toward negative values of $r_{A-M}$, there is not a corresponding decrease in $\hat{\sigma}_e^2$ as one might first expect. Perhaps this occurs because a move towards a negative $r_{A-M}$ is antagonistic with the nature of this particular data and model. Results of this dissertation show that the relationship between direct and maternal sire effects is indeed positive and so would support this idea.

Perhaps this may suggest an approach to obtaining priors in an analysis with a covariance. Provided that a reasonable choice of heritabilities can be made, a reasonable range of covariances can be tested to find one that yields a minimum value for the variance of one of the effects. Certainly, the general effect of priors on first round solutions is untouched in the literature and would constitute a research effort by itself.

Table 7 is provided as a means of determining the effect that a change in priors has on first round estimates of the same parameter. Reported in this form, it appears that choice of prior heritabilities has just a small
Table 7. Effect of priors on initial estimates of heritabilities ($h^2$) and genetic correlations ($r$) from Analysis I of Angus birth and weaning weights

<table>
<thead>
<tr>
<th>Trait</th>
<th>$r_{A\cdot M}$</th>
<th>$\hat{h}_A^c$</th>
<th>$\hat{h}_M^c$</th>
<th>$\hat{r}_{A\cdot M}$</th>
<th>$\hat{h}_A^b$</th>
<th>$\hat{h}_M^b$</th>
<th>$\hat{r}_{A\cdot M}$</th>
<th>$\hat{h}_A^d$</th>
<th>$\hat{h}_M^d$</th>
<th>$\hat{r}_{A\cdot M}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Birth</td>
<td>-.5</td>
<td>.32</td>
<td>.09</td>
<td>+.19</td>
<td>.33</td>
<td>.18</td>
<td>-.23</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>weight</td>
<td>+.5</td>
<td>.41</td>
<td>.09</td>
<td>+.47</td>
<td>.37</td>
<td>.11</td>
<td>+.34</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Weaning</td>
<td>-.5</td>
<td>.21</td>
<td>.17</td>
<td>-.09</td>
<td>.23</td>
<td>.26</td>
<td>-.35</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>weight</td>
<td>+.5</td>
<td>.27</td>
<td>.17</td>
<td>+.13</td>
<td>.25</td>
<td>.21</td>
<td>+.05</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Notation used: $^0 = \text{parameter from which priors were determined}$; $A = \text{direct additive genetic value}$; $M = \text{maternal additive genetic value}$.

$^b h_A^2 = .25$ and $h_M^2 = .10$.

$^c h_A^2 = .10$ and $h_M^2 = .25$.

influence on first round estimates. The same relationships and interactions discussed for Tables 5 and 6 apply here. It is clear that the estimates for $r_{A\cdot M}$ are greatly influenced by choice of priors. When greater emphasis is given to maternal sire effects by way of $h_M^2$, the estimated correlation moves in a negative direction. The change is greater for negative values of $r_{A\cdot M}$.

Although peculiar to these data and models, it is apparent that first round estimates of variance components from MINQUE-D cannot be fully relied upon when priors are not known accurately. If priors are known close to their actual values, MINQUE estimates are shown to have minimum sampling variance (Rao, 1971; Schaeffer, 1983). Unfortunately, the same is not
known of MINQUE-D estimates. Also badly needed is research that indicates the effect that errors in estimated variance components have on solutions to a set mixed model equation and on subsequent selection progress made from use of these solutions.

Hudson and Van Vleck (1982) state that MINQUE-D estimation procedures lend themselves to iteration. Iteration involves replacing priors with estimated variances and then solving for new estimates. This process is continued until a specified lack of change in final solutions is achieved. The properties of iterative solutions to MINQUE or MINQUE-D have not been examined and, consequently, are not known. Hudson and Van Vleck solved iteratively for MINQUE-D estimate and found convergence to occur quickly even if chosen priors were far from the final estimates. This study found solutions to converge quickly as well.

Iteration destroys the properties of unbiasedness that were initially desired. Even though first round estimates are derived to be unbiased, they are unbiased estimates of the quadratics formed from approximations and not from the best solutions to the population that are theoretically possible. Because inferences of these approximate estimates are to be made back to the population from which they are estimated, the question arises whether the properties of unbiasedness relative to the population were lost in the first place. Also, Kennedy (1981) reminds us that even though individual effect variances are unbiased estimates, the ratio of these estimates may not be unbiased estimates of the true ratio. However, unbiased estimates of priors are not necessary to obtain best linear unbiased predictors of random effects (Kennedy, 1981; Kackar and Harville, 1981).
Iterative as well as first round solutions were reported for variance components computed in this dissertation. In support of this decision, two sets of first round solutions each, taken from the values given by Tables 5 and 6, were iterated until convergence was achieved or until negative estimates were obtained. The results of these iterations are given in Tables 8 and 9 for birth weight and weaning weight, respectively.

Table 8. Effect of prior genetic correlation ($r_{A\cdot M}$) on iterative solutions to Analysis I of birth weight

<table>
<thead>
<tr>
<th>$r_{A\cdot M}$</th>
<th>Round of iteration</th>
<th>Component estimates $^{b}$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$\sigma_{s}^{2}$</td>
<td>$\sigma_{mg}^{2}$</td>
</tr>
<tr>
<td>-.5</td>
<td>1</td>
<td>5.58</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>6.75</td>
</tr>
<tr>
<td></td>
<td>6$^{c}$</td>
<td>6.48</td>
</tr>
<tr>
<td>+.5</td>
<td>1</td>
<td>7.14</td>
</tr>
<tr>
<td></td>
<td>2$^{c}$</td>
<td>7.32</td>
</tr>
<tr>
<td></td>
<td>14$^{c}$</td>
<td>6.73</td>
</tr>
</tbody>
</table>

$^{a}$Prior heritabilities for additive direct genetic value ($h_{A}^{2}$) = .25 and for additive maternal genetic value ($h_{M}^{2}$) = .10.

$^{b}$Variance components reported as pounds$^{2}$.

$^{c}$Convergence not pursued due to negative estimates.

From these results, iterative solutions to MINQUE-D equations appear useful when prior values are not accurately known. Despite a range in the assumed value of $r_{A\cdot M}$, solutions to all four variance components were basically equivalent when solved for iteratively. As for weaning weight,
Table 9. Effect of prior genetic correlation \( r_{A-M} \) on iterative solutions to Analysis I of weaning weight

<table>
<thead>
<tr>
<th>( r_{A-M} )</th>
<th>Round of iteration</th>
<th>( \sigma^2_s )</th>
<th>( \sigma^2_{mg} )</th>
<th>( \sigma^2_{s-mg} )</th>
<th>( \sigma^2_e )</th>
</tr>
</thead>
<tbody>
<tr>
<td>-.5</td>
<td>1</td>
<td>121.1</td>
<td>100.3</td>
<td>-10.7</td>
<td>2062.3</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>127.5</td>
<td>110.4</td>
<td>1.3</td>
<td>2034.9</td>
</tr>
<tr>
<td></td>
<td>12</td>
<td>136.4</td>
<td>103.1</td>
<td>17.9</td>
<td>2018.9</td>
</tr>
<tr>
<td>+.5</td>
<td>1</td>
<td>156.5</td>
<td>99.4</td>
<td>16.4</td>
<td>2001.7</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>134.2</td>
<td>99.6</td>
<td>23.6</td>
<td>2018.8</td>
</tr>
<tr>
<td></td>
<td>9</td>
<td>136.3</td>
<td>103.1</td>
<td>17.9</td>
<td>2019.1</td>
</tr>
</tbody>
</table>

\(^a\)Prior heritabilities for additive genetic direct value \( h^2_A \) = .25 and for additive genetic maternal value \( h^2_M \) = .10.

\(^b\)Variance components reported as pounds.²

\(^c\)Convergence criterion met: ± .1 pounds.²

Convergence criterion were met after 12 and 9 rounds of iteration for prior values of \( r_{A-M} \) equal to -.5 and +.5, respectively. Conclusions cannot be so clearly drawn from the birth weight analysis because of the negative estimates for \( \sigma^2_{mg} \). Note, however, that iterative solutions fell out of the parameter space at approximately the same magnitude, and so this is not suggestive as contrary to the results for iterative weaning weight analysis. Therefore, based on these results and the discussion of Hudson and Van Vleck (1982), iterative solutions to MINQUE-D equations are recommended in most applied circumstances.
Applications of MINQUE-D procedures are infrequently found in the literature, and in only one case found was the methodology applied towards computing a covariance of two random effects in one model. Consequently, discussion of the method's strengths and weaknesses is rare. Several questions of MINQUE-D arise from this study which will require answers if the methodology is to be used extensively. These are:

1. What are the consequences of ignoring sire relationships in the procedures? It is suspected that sires with a large number of relative ties but few progeny records would receive unnecessary weighting on the diagonal elements without the benefit of off-diagonal ties (since they are ignored in the approximation).

2. Are there more useful or accurate ways of approximating the predictors of sire effects? Guidelines need to be established relative to data and model types, as well as computer capabilities.

3. What are more exact procedures of prior selection? And what are the consequences of incorrect priors on selection results?

4. In the case of Analysis I, what are the consequences of using progeny records of sires that are not represented as maternal grandsires (or vice versa)? A high proportion of one or the other may bias estimated variances, especially when off-diagonal elements providing ties are ignored. If more selected data are necessary to provide estimates that are unbiased by the procedure, how should the data be selected and how valid are the references from these estimates to the whole population?
(5) In the case of covariance analysis, is a negative estimate in one component because the effect has little or no variance or is it just overwhelmed by the larger variance of another effect in the model?

Concerns can also be raised about the ways in which data are edited previous to an analysis. In the situation of Analysis I, it is conceivable that a high proportion of sires represented only as maternal grandsires would bias the covariance estimate in a positive direction. In each trait and breed analyzed, there were a great number of sires that had both progeny and maternal grandprogeny records. This tie is essential. However, there were a great number of maternal grandsires without progeny records. And even though all sires received estimates for both direct and maternal effect, this may still result in variance components that reflect the number of sires in this category rather than a comparison of their ability to perform as both sire and maternal grandsire.

These questions are all not necessarily answered by this study but really are raised as a result. Other discussions of these concerns were not found in the literature and certainly would warrant further studies. In this way, properties of estimates obtained using MINQUE-D would be better understood. However, the method has been compared favorably to other methods for single random effect analyses such as REML, MINQUE, and Henderson's Method III.
Variance Component Estimates

To begin, both analysis procedures (Analyses I and II) required the use of variances and covariances assumed known prior to the analyses. A drawback of most estimation procedures is this necessary assumption. However, if the true population parameters were indeed known, there would be no need to estimate them. Good estimates of priors need to come from previous research, perhaps from pooling the results of many experiments to increase accuracy. Unfortunately, prior estimates for sire direct and sire maternal effect variances and covariances do not exist in the literature for the models proposed in this dissertation, but estimates from similar work provide some clue as to the range of realistic values that could be used. Priors for models used were inferred from the literature reviewed in this dissertation, primarily from the suggestions of Koch (1972). These "best guess" estimates were computed from the parameters chosen for weaning weight of:

\[ h_A^2 = .25 \text{ (additive direct effect heritability)} \]
\[ h_M^2 = .35 \text{ (additive maternal effect heritability)} \]
\[ r_{A\cdotM} = -.25 \text{ (genetic correlation between direct and maternal effects)} \]

and for birth weight of:

\[ h_A^2 = .25 \]
\[ h_M^2 = .10 \]
\[ r_{A\cdotM} = .00 \]

Because Analysis II utilizes different models than Analysis I, prior values appropriate for one method are not expected to be entirely
appropriate for the other. However, no previous estimates were found that utilized these models on similar populations. Thus, prior estimates for \( \sigma^2_{mg} \) and \( \sigma^2_{mgs} \) were developed from the same value of heritability.

**Birth weight**

Initial and iterative solutions for Analysis I for birth weight are presented in Table 10. For both breeds, each round of iteration increased estimates of the direct effect variance (\( \hat{\sigma}^2_s \)) and the direct-maternal effect covariance (\( \hat{\sigma}^2_{s.mg} \)). Maternal effect variance (\( \hat{\sigma}^2_{mg} \)) decreased steadily toward zero with each round of iteration. Iteration was concluded after eight and three rounds in the Angus and Hereford analyses, respectively, because unreasonably large estimates were obtained for \( \hat{\sigma}^2_{mg} \), \( \hat{\sigma}^2_{s.mg} \), and \( \hat{\sigma}^2_e \). A negative estimate of \( \hat{\sigma}^2_s \) was obtained as well, so it was clear that convergence would not occur in the parameter space allowed.

The problem of negative estimates remains unsolved. Nelsen et al. (1984) reported several negative variance components estimates for birth weight. Inability of methodology to account for sources of variance was blamed as was large sampling error due to a small number of observations. Crow and Howell (1982) reported negative estimates for second and third parity analyses of weaning weights of three breeds; sampling error was considered the cause.

Schaeffer (1983) suggested five possible actions that can be taken should negative variances be computed. His suggestions included:

1. to set the negative estimate to zero.

2. to report the estimate as is (averages of many estimates will be more accurate when these are included).
Table 10. Initial and final variance component estimates from Analysis I of birth weight

<table>
<thead>
<tr>
<th>Breed</th>
<th>Round of iteration</th>
<th>Variance components&lt;sup&gt;a&lt;/sup&gt;</th>
<th>(\hat{\sigma}^2_s)</th>
<th>(\hat{\sigma}^2_{mg})</th>
<th>(\hat{\sigma}^2_{s.mg})</th>
<th>(\hat{\sigma}^2_e)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Angus</td>
<td>0</td>
<td></td>
<td>4.05 (6.3)</td>
<td>1.60 (2.5)</td>
<td>0.00 (0.0)</td>
<td>59.00 (91.2)</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td></td>
<td>6.41 (9.2)</td>
<td>1.37 (2.0)</td>
<td>1.40 (2.0)</td>
<td>60.71 (86.9)</td>
</tr>
<tr>
<td></td>
<td>7</td>
<td></td>
<td>7.15 (10.2)</td>
<td>0.68 (.9)</td>
<td>3.21 (4.6)</td>
<td>59.02 (84.2)</td>
</tr>
<tr>
<td></td>
<td>8&lt;sup&gt;b&lt;/sup&gt;</td>
<td></td>
<td>-48.07 (*)</td>
<td>32.90 (*)</td>
<td>18.96 (*)</td>
<td>71.39 (*)</td>
</tr>
<tr>
<td></td>
<td>Hereford</td>
<td></td>
<td>0.05 (6.3)</td>
<td>1.60 (2.5)</td>
<td>0.00 (0.0)</td>
<td>59.00 (91.2)</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td></td>
<td>5.83 (9.1)</td>
<td>0.56 (.87)</td>
<td>1.61 (2.5)</td>
<td>56.04 (87.5)</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td></td>
<td>7.24 (11.3)</td>
<td>0.18 (.28)</td>
<td>2.37 (3.7)</td>
<td>54.37 (84.7)</td>
</tr>
<tr>
<td></td>
<td>3&lt;sup&gt;b&lt;/sup&gt;</td>
<td></td>
<td>-42.37 (*)</td>
<td>30.18 (*)</td>
<td>26.25 (*)</td>
<td>55.72 (*)</td>
</tr>
</tbody>
</table>

<sup>a</sup>Components expressed as pounds<sup>2</sup>.

<sup>b</sup>Convergence not pursued due to negative estimates.

3. to conclude high sampling variances are at fault and collect more data.

4. to use another method of estimation.

5. to remove the factor from the model and re-estimate other components.
Based on the results of other researchers, it is not likely that sire variance is near zero. Burfening et al. (1981) reported that the sire component was twice the size of the maternal grandsire component. Philipsson (1976) reports similar findings and also concludes that the maternal grandsire component is likely near zero for birth weight. Actually, no reports were discovered that supported a near zero sire variance component. Based on these findings and that large numbers of sires were estimated from a considerable quantity of birth weight records, suggestions 1, 2, and 3 were considered inappropriate for this analysis. Use of another method of estimation also was not attempted, although merit in this approach exists. If sire variance can be assumed positive (and indeed it can), then biased procedures restricting nonnegativity (e.g., REML) may be helpful.

Suggestion 5 offered useful advice to the problem. Koch (1972) and Hohenboken and Brinks (1971a) determined that the genetic covariance between direct and maternal effects is likely zero. If this were true, then $\sigma_{s\cdot mg}$ would be near zero in Analysis I. Therefore, the restriction was made that $\sigma_{s\cdot mg}$ was zero (both prior and final estimates were restricted to zero), and the remaining components were re-estimated using the procedures of Analysis I. Table 11 provides the results of this analysis.

These results indicate that for both breeds, $\sigma^2_{mg}$ is near zero. Ten rounds of iteration brought convergence to solutions for Angus data when $\hat{\sigma}_{s\cdot mg}$ was forced to equal zero. Also, sire direct variance was 4 percent greater than the seventh round solutions when no restriction was imposed.
Table 11. Initial and final variance component estimates obtained from Analysis I of birth weight: covariances restricted to equal zero

<table>
<thead>
<tr>
<th>Breed</th>
<th>Round of iteration</th>
<th>[\sigma_s^2] (and % of total)(^a)</th>
<th>[\sigma_{mg}^2] (and % of total)</th>
<th>[\sigma_e^2] (and % of total)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Angus</td>
<td>0</td>
<td>4.05 (6.2)</td>
<td>1.60 (2.5)</td>
<td>59.00 (91.3)</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>7.74 (12.0)</td>
<td>1.56 (2.5)</td>
<td>60.42 (93.5)</td>
</tr>
<tr>
<td></td>
<td>10(^b)</td>
<td>9.88 (14.2)</td>
<td>0.75 (1.1)</td>
<td>59.00 (84.7)</td>
</tr>
<tr>
<td>Hereford</td>
<td>0</td>
<td>4.05 (6.2)</td>
<td>1.60 (2.5)</td>
<td>59.00 (91.3)</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>6.19 (9.5)</td>
<td>0.02 (.03)</td>
<td>58.73 (90.4)</td>
</tr>
<tr>
<td></td>
<td>3(^c)</td>
<td>24.06 (28.1)</td>
<td>-1.03 (-1.2)</td>
<td>62.56 (73.1)</td>
</tr>
</tbody>
</table>

\(^a\) Variances expressed in pounds\(^2\).

\(^b\) Convergence criterion met: \(\pm .01\) pounds\(^2\).

\(^c\) Convergence was not pursued due to negative estimate.

However, in the Hereford analysis, restricting \(\hat{\sigma}_{s, mg}^2\) to zero still resulted in a negative variance estimate but this time for \(\sigma_{mg}^2\). Consequently, unreasonable solutions for \(\hat{\sigma}_s^2\) and \(\hat{\sigma}_e^2\) were the result. Only two rounds of iteration were performed before negative estimates for \(\sigma_{mg}^2\) were obtained. Rounds 1 and 2 showed again that \(\hat{\sigma}_{mg}^2\) was approaching a near zero value before a negative solution was computed. Convergence, if it were to occur, would be expected to come more quickly on the Hereford data than for Angus because of the greater number of records.
Analysis II estimated sire effect variance \( \hat{\sigma}_s^2 \) and maternal grandsire effect variance \( \hat{\sigma}_{mgs}^2 \) independently. In theory, \( \sigma_s^2 \) represents the same value in both analysis procedures; however, \( \hat{\sigma}_{mg}^2 \) (Analysis I) and \( \hat{\sigma}_{mgs}^2 \) (Analysis II) differ. We would expect \( \hat{\sigma}_{mgs}^2 > \hat{\sigma}_{mg}^2 \) because no attempt was made to partition the direct and maternal genetic contribution made by a maternal grandsire when calculating \( \hat{\sigma}_{mgs}^2 \). Hence, \( \hat{\sigma}_{mgs}^2 \) is a measure of the total variance contributed from maternal grandsires. The results of these separate analyses are presented in Table 12.

Estimates for sire variance remained only slightly greater than they were for Analysis I when the restriction was imposed. Estimated error variances for the sire model were essentially unchanged between rounds of iteration although the percent error variance decreased as the percent sire variance increased. An increase in estimated sire variance from Analysis I to II might suggest that interactions between sire direct and sire maternal were accounted for in Analysis I but remained associated with the direct sire contributions analyzed in II.

Negative estimates for \( \hat{\sigma}_{mgs}^2 \) resulted for birth weight evaluations of both breeds. Analysis II of Angus birth weights was carried out for five rounds. Maternal grandsire variance quickly approached zero for each round, and with it an expected increase in the proportion of error variance estimated was observed. First round solutions from Hereford birth weights yielded estimates of \( \hat{\sigma}_{mgs}^2 \) that were out of the parameter space as well.

In the summary of Tables 10, 11, and 12, the direct effect variance for birth weight ranges from 10 to 15 percent of the total phenotypic variance. As would be expected, the percentage of direct effect variance
Table 12. Initial and final variance component estimates obtained from Analysis II of birth weight

<table>
<thead>
<tr>
<th>Breed</th>
<th>Round of iteration</th>
<th>Components from sire model</th>
<th>Components from MGS model</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>$\sigma^2_s$</td>
<td>$\sigma^2_e$</td>
</tr>
<tr>
<td>Angus</td>
<td>0</td>
<td>3.90</td>
<td>59.00</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(6.2)</td>
<td>(93.8)</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>9.77</td>
<td>59.80</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(14.0)</td>
<td>(86.0)</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>10.55</td>
<td>59.02</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(15.2)</td>
<td>(84.8)</td>
</tr>
<tr>
<td></td>
<td>$^b,c$</td>
<td>10.56</td>
<td>59.02</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(15.2)</td>
<td>(84.8)</td>
</tr>
<tr>
<td>Hereford</td>
<td>0</td>
<td>3.90</td>
<td>59.00</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(6.2)</td>
<td>(93.8)</td>
</tr>
<tr>
<td></td>
<td>$^c$</td>
<td>7.91</td>
<td>55.89</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(12.4)</td>
<td>(87.6)</td>
</tr>
<tr>
<td></td>
<td>$^d$</td>
<td>9.27</td>
<td>54.55</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(14.5)</td>
<td>(85.5)</td>
</tr>
</tbody>
</table>

$^a$Variances are reported as pounds$^2$.

$^b$Convergence for sire model met at $\pm .01$.

$^c$Convergence of MOS solutions not pursued due to negative estimate.

is slightly greater, when fewer variance components are estimated from the model. A smaller percentage was reported by Burfening et al. (1981) (4 percent); however, their model considered herd effect as random and included its estimated variance into the total.

The negative estimates for sire direct variance seen in Analysis I were apparently due to lack of capability of the methodology to account for
maternal grandsire variance or the covariance of sire and maternal grandsire contributions. Perhaps a dependency occurs in the MINQUE-D equations as the estimate for $\hat{\sigma}_{mg}^2$ approaches zero. The problem can occur because the sum of the components estimated must equal the whole. A large proportion of one component to another (say $\hat{\sigma}_s^2$ to $\hat{\sigma}_{mg}^2$) could cause negative estimates to occur.

Variance of sire maternal effects as it contributes to the phenotypic variance of birth weight appears very small. Although the existence of a direct-maternal covariance is still possible, it appears to be small, even though Analysis I would first lead us to believe otherwise. An asymptotic approach to zero for $\hat{\sigma}_{mg}^2$ may bring with it an asymptotic approach towards an infinitely large value for $\hat{\sigma}_{s\cdot mg}^2$ which, of course, is unreasonable.

Next, genetic variance components, heritabilities, and correlations were computed from the values in Tables 10 to 12 as described in the Methods section. These results are presented in Tables 13, 14, and 15.

Negative parameter estimates are a result of negative estimates obtained for effect variances; a discussion of these negative values has been presented. Of course, exceedingly large or negative estimates, although possible, are out of the accepted parameter space and are of no use in predicting response to selection. However, they are included for completeness.

Table 13 provides genetic parameter estimates from Analysis I. By following the results of iteration, the heritability of direct effects ($h_A^2$) approaches a value near .4 to .45, and the heritability of maternal effect ($h_M^2$) is suspected to be near zero for both breeds. The estimated
Table 13. Additive genetic variance components, heritabilities, and correlations for direct (A) and maternal (M) birth weight from Analysis I

<table>
<thead>
<tr>
<th>Breed</th>
<th>Round of iteration&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Parameter estimates&lt;sup&gt;a&lt;/sup&gt;</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>$\hat{\sigma}_A^2$</td>
<td>$\hat{\sigma}_M^2$</td>
<td>$\hat{\sigma}_{A\cdot M}$</td>
<td>$\hat{h}_A^2$</td>
</tr>
<tr>
<td>Angus</td>
<td>1</td>
<td>25.6 (.37)</td>
<td>5.4 (.08)</td>
<td>5.6 (.47)</td>
<td>-192.3 (*)</td>
</tr>
<tr>
<td></td>
<td>7</td>
<td>28.6 (.41)</td>
<td>2.7 (.04)</td>
<td>12.8 (1.46)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>8</td>
<td>-192.3 (*)</td>
<td>131.6 (*)</td>
<td>75.8 (*)</td>
<td></td>
</tr>
<tr>
<td>Hereford</td>
<td>1</td>
<td>23.3 (.36)</td>
<td>2.2 (.03)</td>
<td>6.4 (.89)</td>
<td>-169.5 (*)</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>28.9 (.45)</td>
<td>0.7 (.01)</td>
<td>9.5 (2.08)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>-169.5 (*)</td>
<td>120.7 (*)</td>
<td>105.0 (*)</td>
<td></td>
</tr>
</tbody>
</table>

<sup>a</sup>Variances are reported as pounds<sup>2</sup>.  
<sup>b</sup>Convergence not pursued due to negative estimates.

The genetic correlation ($\hat{r}_{A\cdot M}$) is positive and becomes quite large after initial solutions.

By restricting the covariance estimated by Analysis I to be 0, the results in Table 14 are obtained. Convergence was accomplished for the Angus birth weight analysis but not for Hereford weights. The Angus results indicate that $\hat{h}_A^2$ is .57 but that $\hat{h}_M^2$ is .04 or for all practical purposes 0. Iterative solutions for each round of the Hereford analysis
Table 14. Genetic variance components and heritabilities for additive direct (A) and maternal grandsire (mgs) effects from Analysis I of birth weight: covariances restricted to equal zero

<table>
<thead>
<tr>
<th>Breed</th>
<th>Round of iteration</th>
<th>Parameter estimates</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>$\sigma_A^2$</td>
</tr>
<tr>
<td>Angus</td>
<td>1</td>
<td>31.0 (.44)</td>
</tr>
<tr>
<td></td>
<td>10 $^b$</td>
<td>39.5 (.57)</td>
</tr>
<tr>
<td>Hereford</td>
<td>1</td>
<td>24.76 (.38)</td>
</tr>
<tr>
<td></td>
<td>3 $^c$</td>
<td>96.24 (*)</td>
</tr>
</tbody>
</table>

$^a$Variances reported in pounds$^2$.

$^b$Convergence criterion met: $\pm$ .01 pounds$^2$.

$^c$Convergence not pursued due to negative estimate.

exhibited the same trend as did Angus evaluation. However, negative solutions occurred quickly, and the iteration process was stopped at that point.

The results in Table 15 would suggest that the heritability for direct birth weight is slightly less than that for Angus (.58 and .61, respectively). Negative solutions for $h_{mgs}^2$ were obtained for both breeds, but iterative trends would again suggest that $h_{mgs}^2$ has a value near .00 for both breeds.
Table 15. Genetic variances and heritabilities for additive direct (A) and maternal grandsire (mgs) effects from Analysis II of birth weight

<table>
<thead>
<tr>
<th>Breed</th>
<th>Round of iteration</th>
<th>Parameter estimates</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>$\hat{\sigma}_A^2$</td>
</tr>
<tr>
<td>Angus</td>
<td>1</td>
<td>39.1 (.56)</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>42.2 (.61)</td>
</tr>
<tr>
<td></td>
<td>$5^{b,c}$</td>
<td>42.2 (.61)</td>
</tr>
<tr>
<td>Hereford</td>
<td>1$^c$</td>
<td>31.64 (.49)</td>
</tr>
<tr>
<td></td>
<td>6$^b$</td>
<td>37.08 (.58)</td>
</tr>
</tbody>
</table>

$^a$Variances reported as pounds$^2$.

$^b$Convergence for sire model achieved at $\pm .1$ pounds$^2$.

$^c$Convergence of MGS solutions not pursued due to negative estimate.

In summary of Tables 13, 14, and 15, it seems that the heritability of direct birth weight is between .4 and .6. Final iterative solutions would suggest that $h_A^2$ falls in the upper half of this range, while first round estimates are nearer to .4. This may be due in part to the use of only .25 as a prior value in the estimation process. First round estimates would be expected to be regressed toward zero by a prior that is closer to zero than the final iterative estimates. This range agrees with the conclusions of
Woldehawariat (1977), Koch (1972), and Vesely and Robison (1971) who report values for $h_A^2$ of about .4, .45, and .67, respectively. Brown and Galvez (1969) computed a value for $h_A^2$ of .56 for Herefords but only .14 for Angus. The results of Burfening et al. (1981) and Philipsson (1976) are much lower, where far fewer numbers of sires were estimated than in the present study. Also, in none of the mentioned cases were models, methods, or data sets employed that were very similar to those used for Analysis I.

Furthermore, the results of this study indicate that the heritability of maternal birth weight and the genetic covariance between direct and maternal birth weight are both zero. To the contrary, Koch (1972) suggested that $h_M^2$ is nearer to 10-15 percent. Estimates for $h_M^2$ were slightly above zero for Everett and Magee (1965) and Philipsson (1976), which are in agreement with the present findings. Vesely and Robison (1971) found that if permanent environmental effects are high relative to maternal variance, then $h_M^2$ is near to zero; otherwise, their estimates are from .1 to .3. Perhaps this can explain discrepancies between the present study and those studies from which larger values for $h_M^2$ were obtained. By using the covariances of sire proofs, the dominance and environmental variances and covariances associated with maternal half sib and offspring dam analyses are avoided.

Koch's review reported genetic correlations near to zero for several relative covariances, and he settled on an average of .07. Bourdon and Brinks (1982) decided to assume $r_{A-M} = 0$ because of lack of literature evidence to the contrary. These results would concur with conclusions from this study. As discussed previously, a wide range of estimates have been reported for $r_{A-M}$ depending upon method employed.
All indications point to the conclusion that birth weight is a highly heritable trait and that selection pressure applied should bring rapid genetic changes for the trait in both breeds. Sufficient genetic variation exists to identify sires that would bring this change about. On the other hand, to select for sires that will genetically alter the prenatal environment that affects the birth weight of their daughters' calves would be fruitless. The direct effect of the maternal grandsire is still present, but other influences that determine the birth weight of a calf cannot be attributed to genetic differences among females.

Therefore, it is not recommended that maternal influences on birth weight be a major consideration in the national sire evaluation programs of these two breeds. Because the heritability for maternal birth weight has been estimated near zero and that the genetic covariance between direct and maternal effects is near zero, the portion of the selection differential realized by selection on birth weight is approximately equal to heritability in the narrow sense. Selection progress is not expected to be hampered by any influence of genetic maternal effects on birth weight.

Weaning weight

Prior values, first round, and final iterative solutions were computed for Analysis I of weaning weight records. Positive estimates were obtained for all variance components including the covariance between sire direct and sire maternal effects. These components are presented in Table 16.

Although equivalent prior values were assumed for both breeds, the Hereford evaluation yielded larger estimates for all components than the
Table 16. Initial and final variance component estimates from Analysis I of weaning weight

<table>
<thead>
<tr>
<th>Breed</th>
<th>Round of iteration</th>
<th>$\hat{\sigma}^2_s$</th>
<th>$\hat{\sigma}^2_{mg}$</th>
<th>$\hat{\sigma}^2_{s\cdot mg}$</th>
<th>$\hat{\sigma}^2_e$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Angus</td>
<td>0</td>
<td>158.0 (6.2)</td>
<td>221.0 (8.7)</td>
<td>-46.7 (-1.8)</td>
<td>2212.1 (86.9)</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>109.4 (4.8)</td>
<td>127.8 (5.6)</td>
<td>-04.9 (-0.2)</td>
<td>2042.7 (89.8)</td>
</tr>
<tr>
<td></td>
<td>11</td>
<td>136.4 (6.0)</td>
<td>103.2 (4.5)</td>
<td>18.3 (0.8)</td>
<td>2017.8 (88.7)</td>
</tr>
<tr>
<td>Hereford</td>
<td>0</td>
<td>158.0 (6.2)</td>
<td>221.0 (8.7)</td>
<td>-46.7 (-1.8)</td>
<td>2212.1 (86.9)</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>100.0 (3.4)</td>
<td>192.7 (6.5)</td>
<td>17.4 (0.6)</td>
<td>2648.3 (89.5)</td>
</tr>
<tr>
<td></td>
<td>9</td>
<td>138.3 (4.6)</td>
<td>169.4 (5.7)</td>
<td>38.6 (1.3)</td>
<td>2648.8 (88.4)</td>
</tr>
</tbody>
</table>

Component reported as pounds$^2$.

$s = \text{direct sire effect}; \ mg = \text{maternal sire effect}; \ e = \text{error}.$

Convergence criterion met: $\pm .1 \text{ pounds}^2$.

Angus evaluation. A greater phenotypic variance may be due to a larger number of records used and sires estimated. As a proportion of total variance, estimated error variances were nearly equivalent for both breeds for each round of iteration. Angus records expressed a greater percentage of sire direct variance than Herefords, although the Hereford analysis yields a greater percentage of total variance attributed to maternal effects. This suggests that a greater amount of maternal genetic variability is
present among sires in the Herefords than in the Angus evaluated. This
greater amount of variance due to maternal effects brings with it a larger
covariance component for Herefords than Angus. Both covariances (representing one-fourth of the genetic covariance between direct and maternal
effects) are positive and contribute .8 and 1.3 percent of the total
variance observed in Angus and Hereford weaning weights.

As presented for birth weights, Analysis II was performed on the
weaning weight records of both breeds. All components estimated by
Analysis I of weaning weights were defined and in the accepted parameter
space. Consequently, these values represent recommended values to use as
priors in current Angus and Hereford National Sire Evaluations. The
results of Analysis II would be applicable to mixed model sire evaluations
where sire effects to be estimated independent of maternal grandsire
effects. The resulting variance components of Analysis II are presented in
Table 17.

All variance components estimated by Analysis II are larger than their
counterpart from I. In particular, \( \sigma^2_{mgs} \), which is the variance of one-half
of a sire's genetic maternal value plus one-fourth of his direct effect
value, is larger than \( \sigma^2_{mg} \). This would be expected because \( \sigma^2_{mgs} \) contains
the additional values of \( 1/16\sigma^2_A + 1/4\sigma^2_{A,M} \) in its expectations (\( A = \) additive
direct, \( M = \) additive maternal value). Also expected was the increase in
\( \sigma^2_e \) over \( \sigma^2_{mg} \) because the sire direct effects were not accounted for in the
MGS model.

Crow and Howell (1982) employed MINQUE-D techniques and estimated the
variance due to maternal grandsires in a manner most similar to that used
Table 17. Initial and final variance component estimates from Analysis II of weaning weight\textsuperscript{a}

<table>
<thead>
<tr>
<th>Breed</th>
<th>Round of iteration</th>
<th>Components for sire model\textsuperscript{b}</th>
<th>Components for MGS model\textsuperscript{c}</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>$\sigma^2_s$</td>
<td>$\sigma^2_{e_1}$</td>
</tr>
<tr>
<td>Angus</td>
<td>0</td>
<td>147.5</td>
<td>2212.1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(6.3)</td>
<td>(93.7)</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>214.7</td>
<td>1750.2</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(10.9)</td>
<td>(89.1)</td>
</tr>
<tr>
<td></td>
<td>6\textsuperscript{d}</td>
<td>229.2</td>
<td>2035.8</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(10.1)</td>
<td>(89.9)</td>
</tr>
<tr>
<td>Hereford</td>
<td>0</td>
<td>147.5</td>
<td>2212.1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(6.3)</td>
<td>(93.7)</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>239.8</td>
<td>2734.2</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(8.1)</td>
<td>(91.9)</td>
</tr>
<tr>
<td></td>
<td>6\textsuperscript{d}</td>
<td>270.9</td>
<td>2703.3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(9.1)</td>
<td>(90.9)</td>
</tr>
</tbody>
</table>

\textsuperscript{a}Components reported as pounds\textsuperscript{2}.

\textsuperscript{b}s = sire effect; $e_1$ = error after fitting.

\textsuperscript{c}mgs = maternal grandsire effect; $e_2$ = error after fitting mgs.

\textsuperscript{d}Convergence criterion met: $\pm .1$ pounds\textsuperscript{2}.

in Analysis II. They reported that from 9 to 10 percent of total variation is accounted for by maternal grandsires. This is nearly identical to results reported for this study and gives confidence to the estimate. No other studies were found using similar techniques. Crow and Howell also reported that variance remaining after accounting for maternal grandsire effects comprised an average of 90 to 95 percent of the total estimated
variances, which again is in complete agreement with the results shown in Table 17.

Not expected was a large percentage increase of estimated sire direct variance estimated by Analysis II over Analysis I. The variance of sire direct effects determined by II did not have the additional variance as contributed by one-fourth of the direct effect estimated from maternal grandprogeny records in Analysis I. Possibly, this result stems from the part of whole theory discussed for birth weight. In Analysis I, the sum of each component is forced to equal the whole, and so an increase in the magnitude of one component estimated (such as the covariance) causes another component to be estimated at a lower value. Nonetheless, in a similar study on Angus and Hereford field data, Kennedy and Henderson (1975) included sire effect in the model. A Henderson Method II analysis determined that 10 and 6 percent of total variation, after adjustment for fixed effects, was explained by sire direct effects, which is in nearly identical agreement with the results of their study.

Also noted from both Analyses I and II was that in all cases iterative solutions are considerably different from first round (unbiased estimates). This subject has been discussed. Iterative solutions are expected to more accurately represent the nature of the data.

The results presented in the following tables are the additive genetic parameters derived from the results in Table 16 and 17 for weaning weight. General differences between breed estimates and between analysis method have been discussed.
In general, no reports were found that utilized models and methodology that were employed for this study. Hence, differences from literature estimates are expected because, in these cases, they represent different estimates or different gene pools or both.

The direct effect heritabilities of .24 and .18 are slightly lower but are in general agreement with most estimates discussed in the Review of Literature. Woldehawariat's (1977) review average of .29 from paternal half sib estimates is somewhat larger than this range. However, more sources of variance were accounted for, and estimates were solved for simultaneously from the same data set in the present study. This would likely reduce the variation attributable to sire direct effects, and so estimates from the study could be expected to be somewhat lower than other literature reports. Other papers reviewed presented direct heritability estimates from about .2 to .5.

Two values are reported for maternal heritability in Tables 18 and 19. The value from Analysis I, $h^2_{mg}$, represents the heritability of additive direct maternal ability and is useful for predicting selection progress for genetic maternal ability. The estimates of .18 and .23 reported here would be comparable to most reports in the literature. Koch's (1972) suggestion that 30-35 percent of the variation in weaning weights is due to additive maternal effects is only slightly larger than the Hereford analysis (.25) and more so than the Angus analysis (.16). Hohenboken and Brinks (1971a) and Koch (1972) both made the observation that estimated maternal variance is much larger when the covariance of dam and offspring is considered. Certainly the nature of field data such as used in the present study is
Table 18. Additive genetic direct (A) and maternal (M) weaning weight variance components, heritabilities, and correlations from Analysis I of weaning weight

<table>
<thead>
<tr>
<th>Breed</th>
<th>Round of iteration</th>
<th>Parameter estimates $^a$</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>$\hat{\sigma}^2_A$</td>
<td>$\hat{\sigma}^2_M$</td>
<td>$\hat{\sigma}^2_{A\cdot M}$</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>($h_A^2$)</td>
<td>($h_M^2$)</td>
<td>($r_{A\cdot M}$)</td>
<td></td>
</tr>
<tr>
<td>Angus</td>
<td>1</td>
<td>437.6</td>
<td>511.3</td>
<td>-19.9</td>
<td>(.19)</td>
</tr>
<tr>
<td></td>
<td>11</td>
<td>545.9</td>
<td>412.9</td>
<td>73.6</td>
<td>(.24)</td>
</tr>
<tr>
<td>Hereford</td>
<td>1</td>
<td>400.3</td>
<td>771.1</td>
<td>69.8</td>
<td>(.14)</td>
</tr>
<tr>
<td></td>
<td>9</td>
<td>553.3</td>
<td>677.8</td>
<td>154.5</td>
<td>(.18)</td>
</tr>
</tbody>
</table>

$^a$Variances and covariances reported as pounds$^2$.

$^b$Convergence criterion met: ± .1 pounds$^2$.

subject to many more errors of measurement and variability of environment that the data taken on more controlled research herds. These differences in reports are also likely due to the inability of procedures used to account for environmental covariances and dominance variances and covariances which are confounded with the maternal ability of the dam expressed through her calf. Almost all methods reviewed utilized either this covariance or maternal half sib covariances to obtain estimates for maternal effects and their covariance with direct effect. It is not surprising to see the maternal variances reported for this study to be lower than most literature estimates. In Analysis I, the variances were
Table 19. Genetic variances and heritabilities for additive direct (A) and maternal grandsire (mgs) effects from Analysis II of weaning weight

<table>
<thead>
<tr>
<th>Breed</th>
<th>Round of iteration b</th>
<th>Parameter estimates^a</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>( \sigma^2_A )</td>
<td>( \sigma^2_{mgs} )</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>( (h^2)^A )</td>
<td>( (h^2)_{mgs} )</td>
<td></td>
</tr>
<tr>
<td>Angus</td>
<td>1</td>
<td>858.8 (.44)</td>
<td>785.6 (.39)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>6</td>
<td>916.8 (.40)</td>
<td>525.2 (.23)</td>
<td></td>
</tr>
<tr>
<td>Hereford</td>
<td>1</td>
<td>959.2 (.32)</td>
<td>1082.0 (.36)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>6</td>
<td>1083.6 (.36)</td>
<td>907.6 (.30)</td>
<td></td>
</tr>
</tbody>
</table>

^a Variances and covariances reported as pounds^2.

^b Solutions converged at \( \pm .1 \) pounds^2.

estimated from the covariances of estimated transmitting abilities for sire direct and maternal effect; thus utilizing genes passed through male relatives over a large sample of females.

The heritability of total maternal effect is reported in Table 19. This value \( (h^2)_{mgs} \) is the heritability of the maternal grandsire's contribution to their daughter's performance as mothers. Crow and Howell (1982) report values for \( h^2_{mgs} \) from -.12 to .26, with values from .1 to .25 considered most accurate. Negative estimates were obtained by Crow and Howell on second and third parity analyses and from the Charolais data set where numbers were fewer, hence higher standard errors. However,
parameters that were in an acceptable parameter space compare favorably to the results of Analysis II. Their average $h^2_{mgs}$ reported of .23 for first and fourth parities is somewhat lower than those in Table 19 but were the result of some negative estimates in the average. Also, smaller numbers of progeny were used for the results of Crow and Howell than for the present study. And because estimates are regressed toward the mean for smaller numbers would suggest that their estimates may have somewhat smaller values.

Finally, genetic covariances between direct and maternal effect, reported in Table 18, were found to be small in Angus, larger in Herefords, and positive in both cases. Studies performed on beef cattle data would suggest that this value is negative and larger in magnitude. Koch's (1972) results are indicative of other literature reviewed. He found wide discrepancies among reported values for $r_{A\cdot M}$ but found an average value of $r_{A\cdot M} = -.44$. His own study on Hereford cattle yields estimates nearer to zero, especially when covariances between offspring and dam were not considered.

Work by Hohenboken and Brinks (1971b) provides support for the values of $r_{A\cdot M}$ reported here. Values of -.28 were computed for $r_{A\cdot M}$ by conventional means. Next, researchers estimated maternal abilities as MPPA (most probable producing abilities) on daughters of sires and correlated these values to the weaning weights of their paternal half sib brothers. A genetic correlation of .49 was reported. In concept, similar work by Langlet (1965), Mason (1964), and Bar-Anon (1965) was performed and reported correlations of .22, and .19 and .06 between sire milk proofs and
growth rate in various breeds. These studies may hold large similarities to the one reported here. By passing genes through male descendents, the problem of dealing with a possibly large negative environmental covariance is reduced. The likely existence of such a covariance will drive the covariance to a negative direction when not accounted for in the estimation procedure.
SUMMARY

Birth weight and weaning weight records collected by the American Angus and American Hereford Associations were used to determine sire direct and sire maternal effect variance components for use in mixed model sire evaluations. Data from 20 Angus and 25 Hereford herds were selected for study because of their long histories of credible and consistent use of performance evaluation programs offered by the associations. Each performance record included identification of the herd, sex of calf, weaning date and management code, source of data (designed test or field data), and calf sire and maternal grandsire. A total of 25,586 Angus birth weight records resulted from 718 sires and 1,318 maternal grandsires. For Angus weaning weight analyses, 34,190 records were used from 941 sires and 1,576 maternal grandsires. Hereford data contained 14,436 birth weight records from 566 sires and 1,134 maternal grandsires. For weaning weight analyses, 46,616 Hereford records resulted from 1,366 sires and 2,169 maternal grandsires.

Two mixed models were developed that accounted for fixed contemporary group effect, as well as random sire and/or maternal grandsire contributions to the calf's phenotype. Approximate solutions to Henderson's mixed model equations were computed by inverting a 2 x 2 matrix which included the diagonal elements of a bull's direct and maternal equations and the corresponding off-diagonal elements from the coefficient matrix after absorption of fixed effects. Quadratic forms were developed from these solutions and were equated to their expectations. Iterative
solutions produced estimates of either direct and maternal effect variances and covariances (from Analysis I) or sire and maternal grandsire variances (Analysis II).

First round estimates, although unbiased, were found to be sensitive to initial prior variance components used in their estimation. Nonetheless, final iterative solutions were found to converge quickly and to the same approximate point despite a range in priors tested. Although the properties of iterative solutions to the MIVQUE-D (or Henderson's New Method) procedures used in Analysis I are not known, they are computationally very feasible and produce consistent results. Iterative solutions are recommended when priors are not known with confidence.

Analysis I of weaning weight records yielded direct heritability estimates \( h^2_A \) of .24 for Angus and .18 for Herefords. Maternal weaning weight heritabilities \( h^2_M \) were .18 and .23 for each breed, and the direct-maternal covariance \( r_{A-M} \) was .16 and .25, respectively. Analysis II produced estimates for \( h^2_A \) of .40 and .36, and the heritability of total maternal grandsire effect was estimated as .23 and .30. These heritability estimates are in good agreement with other reports, suggesting that substantial genetic variance exists for the improvement of both direct and maternal weaning weight. Herefords seem to exhibit slightly more genetic variation for maternal ability than Angus when measured via progeny weaning weight records. The positive correlation estimated by this study is in agreement with reports of genetic correlations between sire proofs for milk production and growth rate. However, the correlation is in contrast to negative estimates reported from research that utilized covariances of dam
and offspring or of maternal half sibs to determine its value. Because this study only considered effects from male relatives, the need to ignore possible environmental and dominance influences was circumvented and so avoided a probable negative bias in the estimates. The positive covariance would suggest that genetic progress for maternal ability could be enhanced when replacement females are sired by bulls that were selected entirely for direct weaning weight.

For birth weight, negative variances were estimated from Analysis I for both breeds. If $\hat{r}_{A-M}$ were restricted to equal zero, the Hereford analysis still resulted in a negative estimate for maternal birth weight variance, but iterative estimates of $\hat{h}_{A}^2 = .57$ and $\hat{h}_{M}^2$ were obtained for Angus data. Analysis II produced estimates of $\hat{h}_{A}^2 = .61$ and .58 for sire direct effect, but positive estimates were not obtained for $\hat{h}_{M}^2$.

Direct effect heritabilities were somewhat higher than many literature estimates but still indicate that progress could be made with selection for direct birth weight. Results indicate that almost no maternal effect variance exists in either breed for birth weight and that a genetic relationship between direct and maternal birth weight is likely negligible as well. Sire evaluations for maternal birth weight is not recommended from these results.
LITERATURE CITED


Much gratitude is extended to Dr. R. L. Willham for his role as advisor during my graduate career and for his patient support of my interests in tanbark animal breeding.

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Uppermost, thanks be to the Lord for a patient, supportive bride and an encouraging family.
APPENDIX.
NUMERICAL EXAMPLE OF MIVQUE-D APPLICATION TO ESTIMATE SIRE DIRECT AND SIRE MATERNAL VARIANCE-COVARIANCE COMPONENTS

Development of Approximate Solutions

The method of variance component estimation described in the section entitled Methods is illustrated here by use of a small, hypothetical example. Analysis I deals with a mixed model which equates number of fixed effects (collectively termed group), sire direct effect (random), sire maternal effect (random), and random residual error. The goal is to estimate the variances of sire direct and sire maternal effects and their covariance (denoted as $\hat{\sigma}_e^2$, $\hat{\sigma}_{mg}^2$, and $\hat{\sigma}_{mgs}^2$) using a procedure termed MINQUE-D. For symmetric matrices, the left of diagonal elements are not displayed. Table A-1 details a set of hypothetical data that will illustrate the method.

The mixed model used in Analysis I was given as in matrix notation as:

$$ y = Xb + Z_1u_1 + Z_2u_2 + e $$

where $u_1$ represents sire direct effects and $u_2$ represents sire maternal effects. Based on the sample data, $X$, $Z_1$, and $Z_2$ are shown as follows.
Table A-1. Hypothetical data structure used for illustration of MIVQUE-D application

<table>
<thead>
<tr>
<th>Calf</th>
<th>Group</th>
<th>Sire</th>
<th>Maternal grandsire</th>
<th>Record (nos.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>445</td>
</tr>
<tr>
<td>2</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td>460</td>
</tr>
<tr>
<td>3</td>
<td>1</td>
<td>2</td>
<td>4</td>
<td>470</td>
</tr>
<tr>
<td>4</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>380</td>
</tr>
<tr>
<td>5</td>
<td>2</td>
<td>2</td>
<td>4</td>
<td>350</td>
</tr>
<tr>
<td>6</td>
<td>2</td>
<td>2</td>
<td>3</td>
<td>420</td>
</tr>
<tr>
<td>7</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>400</td>
</tr>
<tr>
<td>8</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>340</td>
</tr>
<tr>
<td>9</td>
<td>2</td>
<td>2</td>
<td>3</td>
<td>395</td>
</tr>
<tr>
<td>10</td>
<td>2</td>
<td>5</td>
<td>4</td>
<td>410</td>
</tr>
</tbody>
</table>

Assumed prior parameters used were:

\[
\begin{align*}
\sigma_s^2 &= 136.5 \\
\sigma_{mg}^2 &= 103.2 \\
\sigma_{s\cdot mg}^2 &= 18.4 \\
\sigma_e^2 &= 2018 \\
\end{align*}
\]

\[
(h_A^2 = .24) \\
(h_M^2 = .18) \\
(r_{A\cdot M} = .16)
\]

\[
X = \begin{bmatrix}
1 & 0 \\
1 & 0 \\
1 & 0 \\
1 & 0 \\
0 & 1 \\
0 & 1 \\
0 & 1 \\
0 & 1 \\
0 & 1
\end{bmatrix}
\]
Note that $Z_1$ and $Z_2$ are of equal order because each sire and maternal grandsire represented in the data is also represented in $Z_1$ and $Z_2$. A sire that has no maternal grandprogeny receives 1s in $Z_1$ and 0s in $Z_2$. Maternal grandsires only receive 1s in $Z_2$ and .5s in $Z_1$ which represent their direct
genetic contribution to the phenotype of their grand progeny. Notice also that sire 1 receives a 1.5 his row and column of $Z_1$ because he is both a sire and maternal grandsire to calf no. 1.

Following the rules for absorption of fixed effects described in the Methods section, the following absorbed least squares equations are built.

$$Z_{1\text{MZ}_1} = \begin{bmatrix} 1.6875 & -1.063 & -.3125 & -.3125 & 0 \\ 2.1875 & -.3125 & -.3125 & -.5 \\ 1.1875 & -.0625 & -.5 \\ .52083 & .16667 & .83333 \end{bmatrix}$$

$$Z_{1\text{MZ}_2} = \begin{bmatrix} 0.875 & 0.375 & -.625 & -.625 & 0 \\ -0.625 & -0.125 & 1.375 & -.625 & 0 \\ -0.125 & -0.125 & 0.375 & -.125 & 0 \\ -0.125 & -0.125 & -.7917 & 1.0417 & 0 \\ 0 & 0 & -.3333 & .33333 & 0 \end{bmatrix}$$

$$Z_{2\text{MZ}_2} = \begin{bmatrix} 0.75 & -0.25 & -0.25 & 0 \\ 0.75 & -0.25 & -0.25 & 0 \\ 2.0833 & -1.583 & 0 \\ 2.0833 & 0 \end{bmatrix}$$

$$Z_{1\text{My}} = \begin{bmatrix} 30.625 \\ -9.375 \\ -39.37 \\ -6.042 \\ 24.167 \end{bmatrix}$$
Recall that the absorbed mixed model equations were denoted by

\[
Z^*MZ = \begin{bmatrix}
6.25 \\
21.25 \\
-15.42 \\
-12.08 \\
0
\end{bmatrix}
\]

In order to solve these equations, a direct inverse of \(Z^*MZ + G^{-1}\sigma_0^2\) is needed. Solutions for the \(\hat{u}\)'s are needed to compute quadratics for...
variance component estimation, but an inverse is often unthinkable. Therefore, an approximation to $Z'WZ + G^{-1}o^2$ is needed. To obtain this approximate form, we can reorder the preceding equations so that they are ordered effect within sire. The $2 \times 2$ matrices along the diagonal of these reordered equations are easily inverted and allow approximate solutions for both effect on a sire to be computed. We call this reordered set of equations $D$ and illustrate its inverse as

$$D^{-1} = \begin{pmatrix}
.0599 & .0052 \\
.0485 & \\
.0590 & .0080 \\
.0492 & \\
.0621 & .0065 \\
.0459 & \\
.0643 & .0048 \\
.0455 & \\
.0640 & .0086 \\
.0510 & 
\end{pmatrix}$$

The approximate solutions $\hat{u}_1$ and $\hat{u}_2$ are then computed.

$$\hat{u}^*_1 = \begin{pmatrix}
1.869 \\
-.382 \\
-2.548 \\
-0.447 \\
1.547 
\end{pmatrix}$$
MINQUE-D Equations and Solutions

The quadratic forms used to compute $\hat{\sigma}_s^2$, $\hat{\sigma}_{mg}^2$, and $\hat{\sigma}_{s\cdot mg}$ are

\[
\hat{u}_2\hat{u}_2' = 12.729
\]

\[
\hat{u}_1\hat{u}_1' = 2.468
\]

and

\[
2\hat{u}_1\hat{u}_2' = 7.075
\]

Although not necessary, the quadratic $y'My$ was chosen to provide a fourth equation and allow calculation of $\hat{\sigma}_0^2$ (the estimated error variance or $\hat{\sigma}_e^2$). $y'My$ in this example equals 10340.

Notation used for the definition of the MINQUE-D equations are defined in the Methods section. The matrices from this example that form these equations are given as follows.

\[
Q_1 = \begin{bmatrix}
D_{11}^2 & D_{11}\cdot D_{12} \\
D_{11}\cdot D_{12} & D_{12}^2
\end{bmatrix}
\]
\[ Q_2 = \begin{bmatrix}
D_{12}^2 & D_{12} \cdot D_{22} \\
D_{12} \cdot D_{22} & D_{22}^2
\end{bmatrix} \]

\[
\begin{bmatrix}
0.0036 & 0 & 0 & 0 & 0 & 316E-6 & 0 & 0 & 0 & 0 \\
0.00348 & 0 & 0 & 0 & 0 & 473E-6 & 0 & 0 & 0 & 0 \\
0.00386 & 0 & 0 & 0 & 0 & 406E-6 & 0 & 0 & 0 & 0 \\
0.00414 & 0 & 0 & 0 & 0 & 310E-6 & 0 & 0 & 0 & 0 \\
0.0041 & 0 & 0 & 0 & 0 & 552E-6 & 0 & 0 & 0 & 0 \\
277E-7 & 0 & 0 & 0 & 0 & 643E-7 & 0 & 0 & 0 & 0 \\
643E-7 & 0 & 0 & 0 & 0 & 394E-6 & 0 & 0 & 0 & 0 \\
427E-7 & 0 & 0 & 0 & 0 & 300E-6 & 0 & 0 & 0 & 0 \\
232E-7 & 0 & 0 & 0 & 0 & 220E-6 & 0 & 0 & 0 & 0 \\
744E-7 & 0 & 0 & 0 & 0 & 441E-6 & 0 & 0 & 0 & 0 \\
0.00236 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0.00242 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0.00211 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0.00208 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0.00261 & & & & & & & & & 
\end{bmatrix}
\]
and

\[
Q_3 = \begin{bmatrix}
2(D_{11} \cdot D_{12}) & ((D_{11} \cdot D_{12}) + D_{12}^2) \\
((D_{11} \cdot D_{12}) + D_{12}^2) & (2D_{22} \cdot D_{12})
\end{bmatrix}
\]

\[
= \begin{bmatrix}
631E-6 & 0 & 0 & 0 & 0 & .00294 & 0 & 0 & 0 & 0 \\
946E-6 & 0 & 0 & 0 & 0 & .00297 & 0 & 0 & 0 & 0 \\
812E-6 & 0 & 0 & 0 & 0 & .0029 & 0 & 0 & 0 & 0 \\
620E-6 & 0 & 0 & 0 & 0 & .00296 & 0 & 0 & 0 & 0 \\
0.0011 & 0 & 0 & 0 & 0 & 0.00335 & 0 & 0 & 0 & 0 \\
511E-6 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
789E-6 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
600E-6 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
439E-6 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
881E-6 & \end{bmatrix}
\]

Note that for the \( K \) matrices, only the diagonal elements of each quadrant because the trace of \( C \cdot K \) is the desired value and the \( C \)'s are quadrant diagonal matrices themselves. The \( K \) matrices are computed as follows.

\[
K_1 = Z \cdot M_2 Z^T M_2
\]
\[ K_2 = Z^\top M Z \, Z^\top M Z \]

\[
\begin{bmatrix}
4.1719 & 2.2188 \\
6.3594 & -.5938 \\
1.8594 & 0.4270 \\
.4983 & 0.9965 \\
1.2222 & 0 \\
1.1875 & 0.1875 \\
0.1875 & 3.1597 \\
& 1.9931 \\
\end{bmatrix}
\]

\[
\begin{bmatrix}
1.6875 & 0.875 \\
2.6875 & -0.125 \\
0.1875 & 1.0417 \\
1.7431 & 3.4861 \\
.22222 & 0 \\
\end{bmatrix}
\]

\[
\text{(sym)}
\]

\[
\begin{bmatrix}
0.75 \\
0.75 \\
6.9722 \\
6.9722 \\
0 \\
\end{bmatrix}
\]
and

\[ K_3 = Z^* M \left[ Z_1 Z_2^* + Z_2 Z_1^* \right] M Z \]

= 
\[
\begin{bmatrix}
1.4688 & 0.6875 \\
0.15625 & -0.3125 \\
0.53125 & 0.14583 \\
(N.A.) & 0.67014 \\
1.6875 & 0.875 \\
2.0625 & -0.125 \\
2.7292 & 1.8472 \\
1.3403 & 2.6806 \\
0 & 0
\end{bmatrix}
\]

Note that the needed elements of KO are found from the diagonal elements of \( Z_1 M Z_1, Z_2 M Z_2, \) and \( Z_2 M Z_2 \).

After computing all needed expectations, the MINQUE-D equations to be solved are

\[
\begin{bmatrix}
0.05343 & 0.02825 & 0.0306 & 0.02544 \\
0.01612 & 0.03555 & 0.02815 & 0.01365 \\
0.3324 & 0.04435 & 0.06645 & 0.02122 \\
6.4167 & 5.6667 & 4.3333 & 8
\end{bmatrix}
\begin{bmatrix}
\hat{\sigma}_s^2 \\
\hat{\sigma}_{mg}^2 \\
\hat{\sigma}_{s,mg}^2 \\
\hat{\sigma}_e^2
\end{bmatrix}
= 
\begin{bmatrix}
12.729 \\
2.4683 \\
71.0753 \\
10340
\end{bmatrix}
\]
The final solutions for the variance components are

\[
\begin{bmatrix}
\hat{\sigma}_s^2 \\
\hat{\sigma}_{mg}^2 \\
\hat{\sigma}_{s-mg}^2 \\
\hat{\sigma}_e^2
\end{bmatrix}
= 
\begin{bmatrix}
-472.0 \\
-535.7 \\
54.9 \\
2020.8
\end{bmatrix}
\]

The negative estimates are possible and not surprising because of the arbitrary nature of the data and small number of records.