Estimation of maternal effects on birth and weaning weight of Hereford cattle
by Rodolfo Juan Carlos Cantet

A thesis submitted in partial fulfillment of the requirements for the degree of Master of Science in Animal Science
Montana State University

Abstract:
Evidence has been found that maternal effects are important sources of variation in mammals. In beef cattle, evidence has been found to support the hypothesis that maternal effects can reduce the expected response to selection for growth traits, especially preweaning growth. It is not clear whether maternal effects in preweaning growth of beef cattle are genetic or environmental. Therefore, the present research was conducted to study the nature and. magnitude of maternal effects in birth and weaning weight of Hereford cattle. The data used were the records of 4,423 noncreep-fed beef calves raised at the Northern Agricultural Research Center, Havre, Mt from 1938 to 1983. Least-squares, fitting constants method (Henderson III) and Restricted Maximum Likelihood procedures were used to estimate eighteen covariances between different types of relatives. The basic models included the fixed effects of line-year, age of dam, sex of calf, age of dam by sex interaction and the regressions of birth weight on birthdate of calf and weaning weight on weaning age of calf. The source of variation depicting the relative relationship was considered a random effect. Multiple regression procedures were used to obtain the nine parameters: additive genetic, dominance genetic and environmental direct and maternal variances and the covariance between the direct and maternal source in each case.

All solutions showed a negative additive genetic correlation between additive direct effects and additive maternal effects (rG). The results were not clear regarding to the magnitude of rG for birth weight but the probable value for weaning weight was around -.60 to -.75. There was also evidence for a negative environmental correlation for maternal phenotype of the dam and daughter in the case of weaning weight. This path coefficient (fm) was calculated to be .08 for birth weight and -.10 for weaning weight. After correcting the expectations of the covariances between relatives for fm, rG was still present and negative for both weights. The solutions showed that dominance was also involved in determining maternal effects in preweaning growth of beef cattle but its effects may be confounded with epistasis.

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A thesis submitted in partial fulfillment
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APPROVAL

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Rodolfo Juan Carlos Cantet

This thesis has been read by each member of the thesis committee and has been found to be satisfactory regarding content, English usage, format, citations, bibliographic style, and consistency, and is ready for submission to the College of Graduate Studies.

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Date May 25, 1984

To my wife, to my mother and

to the memory of my father

VITA

Rodolfo Juan Carlos Cantet was born to Mr. and Mrs. Rodolfo Manuel Cantet in Buenos Aires, Argentina, on March 17, 1954.

He attended Liceo Militar General San Martin Highschool and was admitted to the Facultad de Agronomia, Universidad de Buenos Aires in 1972. He graduated as an Ingeniero Agronomo in 1978.

He has worked as a researcher in Beef Cattle Breeding at the Departamento de Zootecnia, Universidad de Buenos Aires, from 1978 to present.

In September 1982, he was admitted to Montana State University for his Master's degree in Animal Science (Animal Breeding).

He married Patricia Prandini in 1982.

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ABSTRACT

Evidence has been found that maternal effects are important sources of variation in mammals. In beef cattle, evidence has been found to support the hypothesis that maternal effects can reduce the expected response to selection for growth traits, especially preweaning growth. It is not clear whether maternal effects in preweaning growth of beef cattle are genetic or environmental. Therefore, the present research was conducted to study the nature and magnitude of maternal effects in birth and weaning weight of Hereford cattle. The data used were the records of 4,423 noncreep-fed beef calves raised at the Northern Agricultural Research Center, Havre, Mt from 1938 to 1983. Least-squares, fitting constants method (Henderson III) and Restricted Maximum Likelihood procedures were used to estimate eighteen covariances between different types of relatives. The basic models included the fixed effects of line-year, age of dam, sex of calf, age of dam by sex interaction and the regressions of birth weight on birthdate of calf and weaning weight on weaning age of calf. The source of variation depicting the relative relationship was considered a random effect. Multiple regression procedures were used to obtain the nine parameters: additive genetic, dominance genetic and environmental direct and maternal variances and the covariance between the direct and maternal source in each case.

All solutions showed a negative additive genetic correlation between additive direct effects and additive maternal effects (r_G). The results were not clear regarding to the magnitude of r_G for birth weight but the probable value for weaning weight was around -.60 to -.75. There was also evidence for a negative environmental correlation for maternal phenotype of the dam and daughter in the case of weaning weight. This path coefficient (fm) was calculated to be .08 for birth weight and -.10 for weaning weight. After correcting the expectations of the covariances between relatives for fm, r_G was still present and negative for both weights. The solutions showed that dominance was also involved in determining maternal effects in preweaning growth of beef cattle but its effects may be confounded with epistasis.

INTRODUCTION

The success of a breeding scheme to improve performance characteristics of farm animals depends on how genetic and environmental sources of variation are taken into account. Like other domestic mammals, beef cattle are subject to maternal environment from the early moments of gestation through weaning time. A maternal effect is an effect contributed to the phenotypic value of an individual by its dam (Willham, 1980). Although maternal effects are environmental so far as their influence on offspring is concerned, they are determined by genetic and environmental factors (Koch, 1972).

Birth weight is the result of gestational growth rate and gestation length. Weaning weight is the consequence of birth weight and growth during the suckling period. Both traits are measured as the phenotypic value of the calf, but they are composed of at least two components, offspring genetics for growth and a maternal effect contributed by the dam. This latter influence is produced by nutrients provided by the uterus (in the case of birth weight) and the mammary gland (in the case of weaning weight). As Robison (1981) comments, it has recently become apparent that other factors may be involved in this action, perhaps through circulating hormones, cytoplasm, etc.

The other contribution of the dam to the phenotypic value of the offspring is a sample half of her genes to the calf. Therefore, while the sire contributes to the phenotypic value of the offspring by passing a sample half of his genes to the offspring, the dam

contributes in at least two ways. Willham (1980) points out that the confounding of the two contributions from the dam and the possibility of a negative genetic correlation between the direct and maternal effect constitute the bases for the paramount problems in estimating maternal effects. It is clear that heritabilities (h^2) can be biased because of the presence of maternal effects (Robison, 1981).

The modeling process to estimate maternal effects has been initiated by Dickerson (1947). Kempthorne (1955) was an important paper in solving the problem of the estimation of genetic and environmental variances based on covariances between relatives. The presentation included the basis of the actual theory for measuring maternal and direct variation. Koch and Clark (1955b) was the first paper to estimate the additive genetic covariance between direct and maternal effects (σAoAm) in beef cattle, by using path coefficients theory. Finally, Willham (1963) developed a linear model theory for estimating direct and maternal genetic covariances and variances by an extension of the procedures first developed by Kempthorne (1955).

The use of Willham's method applied to cattle records has been reported by Everett and Magee (1965), Hill (1965), Brown and Galvez (1969), Vesely and Robison (1971), Koch (1972), Philipsson (1976), Fisher and Williams (1978) and Burfening et al (1981) for birth weight. Hill et al (1965), Deese and Koger (1967), Hohenboken and Brinks (1967), Vesely and Robison (1971), Koch (1972), Beltran Bru (1978) and Kress et al (1979) dealt with weaning weight. The general conclusions from the reviewed literature were that heritability for additive genetic direct effects (h^2o) was larger for birth weight than

the contributions of additive genetic maternal effects (hm^2). The converse was true for weaning weight. A negative genetic antagonism between additive genetic direct and additive genetic maternal effects (i.e., negative value of $\sigma AoAm$) has been found for both traits. The magnitude of $\sigma AoAm$ is likely to be relatively greater for weaning weight than for birth weight. However, the exact value of $\sigma AoAm$ has been the matter of some discussion (Koch, 1972; Baker, 1980). Van Vleck et al (1977) have shown that the value of $\sigma AoAm$ determines the long term response to selection for weaning weight.

Totusek (1968), Mangus and Brinks (1971), Kress and Burfening (1972), Martin et al (1981) and Ochoa et al (1981) have found that the environment in which the heifer calf is raised affects her future maternal phenotype that she provides for her calves. This complicates the matter of the relative magnitude of environmental and genetic sources of variation on the expression of maternal effects, as Koch (1972) and Hohenboken (1973) discuss.

The objectives of the present study were:

1) to estimate the amount of variation due to additive genetic direct and additive genetic maternal effects in birth weight and weaning weight of Hereford beef calves,

2) to clarify the problem of the relative importance of genotype and environment for the expression of maternal effects on both traits, and

3) to estimate the additive genetic covariance between direct and maternal effects for both birth weight and weaning weight of Hereford beef calves.

REVIEW OF LITERATURE

Estimation of direct and maternal sources of variation

As in most other animal breeding problems, maternal effects theory is strongly related to quadratic estimation. The models used to estimate variance components due to maternal effects started from the variance of the classic model:

$$\sigma^2P = \sigma^2G + \sigma^2E \qquad (1)$$

where σ^2P is the total phenotypic variance, σ^2G is the variance due to genetic effects, and σ^2E is the environmental variance. During the entire modeling process, no attempt has been made to incorporate the genotype by environmental interaction source of variation (σ^2GE) into maternal effects quadratic estimation.

A further partition of σ^2G can be made as follows:

$$\sigma^2G = \sigma^2A + \sigma^2D + \sigma^2I \qquad (2)$$

where σ^2A is the variance due to additive genetic effects, σ^2D is the variance due to dominance effects (i.e., intralocus interaction) and σ^2I is the variance due to epistasis (i.e., interlocus interaction).

Dickerson (1947) made the first partition of the variances in model (1) to incorporate maternal effects. He did not consider dominance or epistasis in his model as shown below:

$$\sigma^2P = \sigma^2Ao + \sigma^2Am + \sigma Ao\ Am + \sigma^2Em + \sigma^2Eo \qquad (3)$$

The terms of model (3) are

σ^2Ao = variance due to additive direct effects,

σ^2Am = variance due to additive maternal effects,

$\sigma AoAm$ = covariance between additive maternal and additive direct effects,

σ^2Em = variance due to maternal environmental effects common to full-sibs and maternal half-sibs, and

σ^2Eo = variance due to random environment.

The model was used to estimate maternal and direct covariances in carcass data of swine. Dickerson (1947) developed the path coefficient diagram shown in Figure 1.

The phenotype of X (Px) is the result of its own genotype for direct effects (Gox) plus a commmon or maternal environment component among litter mates (Pmw), where w indicates the dam of X. Dickerson (1947) did not include the random environmental source of variation in the diagram but in the analysis. The transmissible genotype or additive genetic effects are split into two components: o (direct) and m (maternal). The Gmx component will be expressed only if individual X subsequently becomes a dam (Willham, 1963). The component Pmw is affected by the maternal genotype of W (i.e., Gmw). There was no intention to relate the maternal phenotype of w with the maternal phenotype of her dam, her maternal grand dam and so on.

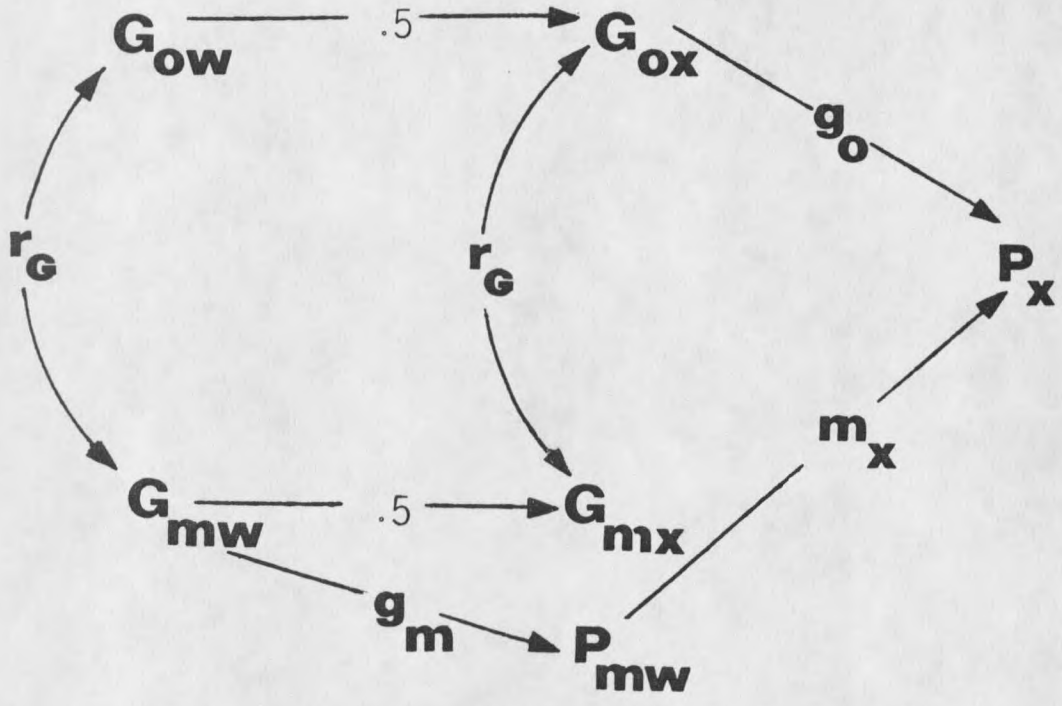


Figure 1. Path coefficient diagram of Dickerson's(1947) model.

In terms of the path coefficients of Figure 1, the heritability or regression of transmitted ability (Gox + Gmx) on individual performance (X) is

$$b(Go + Gmx)\ Px = go^2 + 3/2\ gogm\ r_{gogm} + 1/2\ gm^2 \qquad (4)$$

This expression is the numerator for what is called heritability for total effects where "total" stands for additive genetic direct plus additive genetic maternal effects.

The main contributions of Kempthorne (1955) to the theory of maternal effects was the inclusion of dominance in the model and the description of the basis for the coefficients of the direct and maternal covariances into the expectation of the covariance between relatives. His theory assumes that the genotypic value of an individual is additively determined by the joint effects of the genes possessed by the individual and its mother. The genetic variance of his model is

$$\sigma^2 G = \sigma^2 Ao + \sigma^2 Am + \sigma AoAm + \sigma^2 Do + \sigma^2 Dm + \sigma DoDm \qquad (5)$$

where $\sigma^2 Do$ and $\sigma^2 Dm$ are the variances due to dominance direct and dominance maternal deviations, respectively, and $\sigma DoDm$ is the covariance between the dominance effects.

Kempthorne (1955) did not consider the common or maternal source of environmental variation (i.e., $\sigma^2 Em$). The other expressions derived are the covariances between offspring and sire (cov(O,S)), offspring and dam (cov(O,D)) and among full-sibs (cov(FS)) as

$$cov\ (O,S) = pq\ \sigma^2 Ao + 1/2\ pq\ \sigma AoAm,$$

$$cov\ (O,D) = pq\ \sigma^2 Ao + pq\ \sigma^2 Am + 5/4\ \sigma^2 AoAm + \sigma DoDm, \quad \text{and}$$

$$\text{cov (FS)} = 1/2\ \sigma^2 Ao + \sigma^2 Am + 2\ pq\ \sigma AoAm + 1/4\ \sigma^2 Do + \sigma^2 Dm.$$

p and q are the gene frequencies for genes A and a, respectively.

The most commonly used model for estimating maternal effects of birth weight and weaning weight in cattle was derived by Willham (1963). Basically, he generalized the work of Kempthorne (1955) adding epistasis to the model. However, this interlocus gene expression has always been assumed to be zero or negligible for both birth weight and weaning weight. Perhaps the most useful result of Willham's paper was the derivation of the coefficients for the additive and dominance covariances in the expected values of various kinds of relatives.

The coefficients that Kempthorne (1955) had expressed as functions of Wright's coefficient of relationship with no inbreeding or Malecot's "coefficient de parente" received concrete numerical values with the work of Willham.

Willham (1963) showed that the genotypic covariance between the phenotypes of individuals X and Y, with respective mothers W and Z, is equal to

$$\text{cov}(Gx, Gy) = \text{cov}(Gox,Goy) + \text{cov}(Gox,Gmz) + \text{cov}(Gmw,Goy) + \text{cov}(Gmw,Gmz) \qquad (6)$$

where Gox and Goy are the genotypic values of X and Y for direct effects, and Gmw and Gmz are the genotypic values of the dams W and Z for maternal effects. Thus, the problem was reduced to calculating the four covariances in (6).

The first covariance between X and Y for component o was given by Kempthorne (1957) as

$$2p_{xy}\ \sigma^2 Ao + u_{xy}\ \sigma^2 Do + \sum_{r,s} (2p_{xy})^r\ (u_{xy})^s\ \sigma^2(A^rD^s)_o$$

for $2 \leq r+s \leq N$

where

$\sigma^2(A^rD^s)_o$ is the epistatic component of genotypic variance for the direct component o. For this term, r and s refer to the number of loci involved in the interaction and N refers to the number of loci segregating for component o. The term p_{xy} is Wright's coefficient of relationship with no inbreeding or twice Malecot's "coeffiecient de parente". The coefficient u_{xy} is the probability that the two genes at a locus in individual x are identical by descent with the two genes at that locus in individual y. The value of u_{xy} is zero unless the two individuals are related by two lines of descent such as full sibs or double first cousins.

The second and third terms in (6) are the genotypic covariances between the individuals and their mothers for components o expressed in x or y and component m expressed in y or x, respectively. Since Mode and Robinson (1959) showed that the genotypic covariance between characters, or in this case components of a character can be partitioned in an analoguos manner to the genotypic variance, those terms are

$$2\ p_{xz}\ \sigma\, AoAm + u_{xz}\ \sigma\, DoDm + \sum_{r,s}(2p_{xz})^r(u_{xz})^s\ \sigma(A^rD^s)_o\ (A^rD^s)_m$$

$2 \leq r+s \leq M$

and

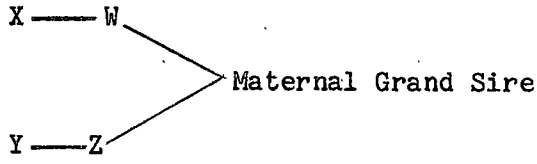
$$2\ p_{yw}\ \sigma AoAm + u_{yw}\ \sigma DoDm + \sum_{r,s}^{2\le r+s\le M} (2p_{yw})^r\ (u_{yw})^s\ \sigma(A^rD^s)_o\ (A^rD^s)_m$$

$\sigma(A^rD^s)_o\ (A^rD^s)_m$ is the epistatic covariance between o and m. M is the number of loci segregating that affect both o and m.

A general expression for the cov(Gx,Gy) assuming that epistasis is equal to zero is:

$$\text{cov}(Gx,Gy) = 2\ p_{xy}\ \sigma^2Ao + (2\ p_{xz} + 2\ p_{yw})\ \sigma AoAm + 2\ p_{wz}\ \sigma^2Am + u_{xy}\ \sigma^2Do + (u_{xz} + u_{yw})\ \sigma DoDm + u_{wz}\ \sigma^2Dm \qquad (7)$$

Table 1 shows the coefficients 2p and u for several relatives and is based on the relationships considered by Willham (1963) and Van Vleck and Hart (1966) for the genetic components of (7) with the more general expressions for the environmental components of variation as shown in Thompson (1976). Then, when x and y have the same maternal grand sire we have



$2\ p_{xy} = (1/2)^4 = 1/16;\ 2\ p_{xz} = 1/8;\ 2\ p_{yw} = 1/8;\ 2\ p_{zw} = (1/2)^2 = 1/4$

Since x and y are not related by two lines of descent all u's are zero. Therefore, the expected value of σ^2_{MGS} is

$$E(\sigma^2_{MGS}) = 2\ p_{xy}\ \sigma^2Ao + (2\ p_{xz} + 2\ p_{yw})\ \sigma AoAm + 2\ p_{wz}\ \sigma^2Am$$

$$E(\sigma^2_{MGS}) = 1/16\ \sigma^2Ao + 1/4\ \sigma AoAM + 1/4\ \sigma^2Am$$

Any kind of relative relationship can be evaluated in the same way.

Covariances in Table 1 were divided into three groups. In the first group sire-type relationships are considered; hence, the

TABLE 1. COEFFICIENTS FOR THE DIRECT AND MATERNAL VARIANCES AND COVARIANCES IN THE EXPECTED VALUES OF THE COVARIANCES BETWEEN RELATIVES

Relatives	$\sigma^2 A_o$	$\sigma A_o A_m$	$\sigma^2 A_m$	$\sigma^2 D_o$	$\sigma D_o D_m$	$\sigma^2 D_m$	$\sigma^2 E_o$	$\sigma E_o E_m$	$\sigma^2 E_m$
				Sire-type group					
Paternal Half-Sibs (PHS)	1/4	0	0	0	0	0	0	0	0
Paternal Grand Sire-Sibs (PGS)	1/16	0	0	0	0	0	0	0	0
Maternal Grand Sire-Sibs (MGS)	1/16	1/4	1/4	0	0	0	0	0	0
Maternal Great Grand Sire-Sibs (MGGS)	1/64	1/16	1/16	0	0	0	0	0	0
Maternal Grand Dam-Sibs (MGD)	1/16	1/4	1/4	0	0	0	0	0	0
				Covariance group					
Offspring and Sire (COV(O,S))	1/2	1/4	0	0	0	0	0	0	0
Offspring and Dam (COV(O,D))	1/2	5/4	1/2	0	1	0	0	1	0
Offspring and Maternal Grand Dam (COV(O,MGD))	1/4	5/8	1/4	0	0	0	0	0	0
Maternal Grand Sire Progeny and Grand Offspring COV(S,MGS)	1/8	1/4	0	0	0	0	0	0	0
Maternal Full-Sib Aunt and Offspring (COV(MA,N))	1/4	3/4	1/2	0	1/4	0	0	0	0
Paternal Full-Sib Aunt and Offspring (COV(PA,N))	1/4	3/4	1/2	0	1/4	0	0	0	0
				Relative relationships involving dominance					
Full-Sibs (FS)	1/2	1	1	1/4	0	1	0	0	1
Maternal Half-Sibs (MHS)	1/4	1	1	0	0	1	0	0	1
Single First Cousins (SFC)	1/8	1/2	1/2	0	0	1/4	0	0	0
PHS plus Dams MHS (PHS + MHSD)	5/16	1/4	1/4	1/16	0	0	0	0	0
PHS + SFC	3/8	1/2	1/2	1/8	0	1/4	0	0	0
FS plus PHS parents (FS + PHS)	5/8	5/4	1	25/64	0	1	0	0	1
PHS + PHS Dams (PHS + PHSD)	5/16	1/4	1/4	1/64	0	0	0	0	1

expectations only involves additive effects. The second group contains covariances between the offspring generation and a closely related individual of the parental generation. The last group is composed by the terms involving dominance. The error terms for models in which there is only one family component have not been extensively used. An unclear interpretation of the components involved in the expectation for those error terms is the possible reason.

So far it has been assumed there is no effect of the dam environment on the future maternal ability of the female offspring. This is likely to occur for weaning weight in beef cattle, as Koch (1972) pointed out. If this is true, maternal environment will be affected by grand maternal environment and the latter by maternal great grand dam environment and so on. In this sense, the maternal effect is viewed as partially affected by maternal environment from previous generations.

The first attempt to incorporate these effects into the model was made by Falconer (1965). The variance of his model is:

$$\sigma^2P = \sigma^2A + \sigma^2M + 2\ \sigma AM + \sigma^2D + \sigma^2Em + \sigma^2Eo \qquad (8)$$

where σ^2A is the variance of additive effects, σ^2M is the variance due to the maternal effects to which the individual is subject, σAM is the covariance between additive and maternal effects; σ^2D is the variance due to dominance deviations (Falconer included here also the epistatic deviations involving dominance), σ^2Em is the variance due to maternal or common environment, as before, and σ^2Eo is the variance due to random environment. The maternal effects (M) are defined as a linear function, fm, of the mother's phenotypic maternal

value, P', measured as a deviation from the population mean, so that

$$M = fm\ P' \qquad (9)$$

Falconer (1965) admitted that the way M is defined is rather restricted because it excludes all maternal influences that are not correlated with the mother's phenotypic maternal value. But he also points out that if these other influences are present they will be included with the rest of the common or maternal environment ($\sigma^2 Em$). The coefficient fm is defined to be "a partial regression coefficient relating daughters to mothers' phenotypic values in the absence of genetic variation among the mothers". Whether the relationship is really linear is, of course, open to question. Obviously, a linear relationship is easier to evaluate. It should also be noted that the D, Em and Eo terms in the model are uncorrelated with P'. Hence,all other covariances in (8) are zero but σAM. An interpretation of model (8) in terms of path coefficients is shown in Figure 2.

The prime in the graph indicates the previous generation. The arrow pointing at M' from the left indicates the carrying over of maternal effects from previous generations. Falconer (1965) derived the cov(O,D) in terms of fm as

$$cov(O,D) = \sigma^2 A\ (1/(2 - fm)) + fm\ \sigma^2 Pm'$$

It is worth noting the derivation of σAM.

By definition $M = fm\ Pm' = fm\ (A' + M' + D' + Em' + Eo')$. Since D', Em' and Eo' are not correlated with A, they can be omitted while deriving σAM. Therefore, the relevant part of M can be written as

$$M = fm\ (A' + fm\ P'')$$

$$= fm\ (A' + fm\ (A'' + fm\ P''''))$$

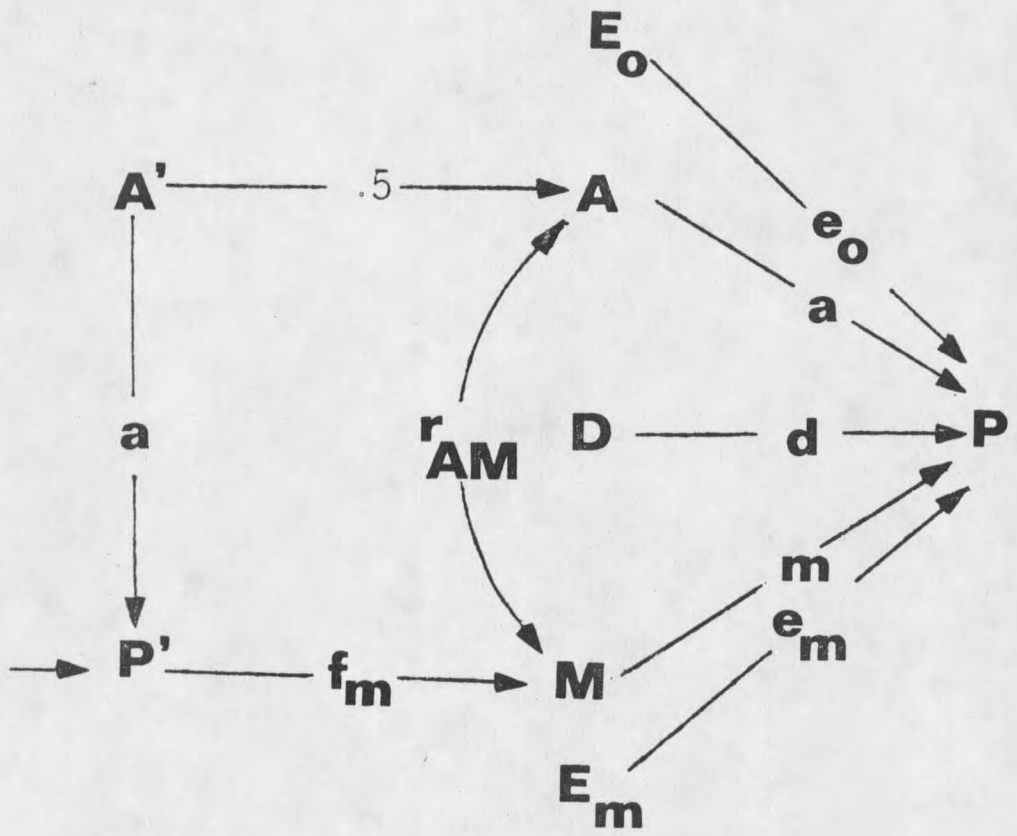


Figure 2. A path coefficient diagram describing Falconer's (1965) maternal effects model. P is the phenotype of the individual; A, his additive genotype; D, his dominance deviation; Em are the environmental factors common to full-sibs and maternal half-sibs that are not included in the maternal effect; M is the maternal effect; Eo are other environmental factors particular to the individual and r_{AM} is the correlation between additive genetic and maternal effects. Path coefficients (e_o, a, d, m, e_m, .5 and fm) are standard partial regression coefficients.

$$= fm\,(A' + fm\,(A'' + fm\,(A''' + \ldots.\text{etc})$$

Therefore

$$\sigma AM = fm\ \sigma AA' + fm^2\ \sigma AA'' + fm^3\ \sigma AA''' + \ldots.$$

$$\sigma AM = \sigma^2 A\ (1/2\ fm + 1/4\ fm^2 + 1/8\ fm^3 + \ldots.$$

The expression in brackets is the geometric series with common ratio 1/2 fm. If $|fm| < 1$ the series converges with sum

$$\frac{(1/2)\ fm}{1 - fm/2} = \frac{fm}{2 - fm}$$

Hence

$$\sigma AM = \sigma^2 A\ \frac{fm}{2 - fm}$$

Thompson (1976) has derived the coefficients for fm in the expected values of some relative relationships.

Willham (1972) considered a model in which the effect of the maternal grand dam is present. The genotypic covariance between any two relatives is evaluated as in (6), but with nine covariances instead of four. An additive genetic term for the variation due to grand maternal effects (i.e. $\sigma^2 An$) is also defined. For genetic effects only the variance of this model is

$$\sigma^2 A = \sigma^2 Ao + \sigma AoAm + \sigma AoAn + \sigma^2 Am + \sigma AmAn + \sigma^2 An \qquad (9)$$

where $\sigma AoAn$ and $\sigma AmAn$ are the covariances between direct and grand maternal additive effects and maternal and grand maternal additive effects, respectively.

From a theoretical point of view, it is not clear whether to prefer the maternal-grand maternal additive model to the maternal

additive model. Using the same reasoning, it is possible to include great grand maternal effects into the model and so on.

Additive genetic effects are carried by the dam side through several generations. However, the same theory of the covariance between relatives (Kempthorne, 1955) shows a high level of reliability in the evaluation of the additive genotype or breeding value by considering only one generation previous to the one considered. As a result of these ideas, Koch (1972) defined the most biologically meaningful model to evaluate maternal effects in beef cattle birth weight and weaning weight. The model combined Willham's (1963) additive and dominant components with Falconer's (1965) concept of transmitted maternal phenotype effects. The covariance between random environmental and maternal or common environmental effects (σ EoEm) was also added. The variance of Koch's (1972) model can be expressed as

$$\sigma^2 p = \sigma^2 Ao + \sigma AoAm + \sigma^2 Am + \sigma^2 Do + \sigma DoDm + \sigma^2 Dm + \\ + \sigma^2 Eo + \sigma EoEm + \sigma^2 Em \qquad (10)$$

The original expression in the paper does not differentiate between σ^2Do and σ^2Eo. It also does not split σ^2Dm and σ^2Em. These four variances appear in two terms: σ^2Do + σ^2Eo and σ^2Dm + σ^2Em. The classical genetic theory assumes no covariance between dominance and environmental deviations of any kind (Falconer, 1981). Koch (1972) justified his procedure by saying that the relationships usually available in beef cattle data do not provide critical contrasts for separating dominance and environmental deviations.

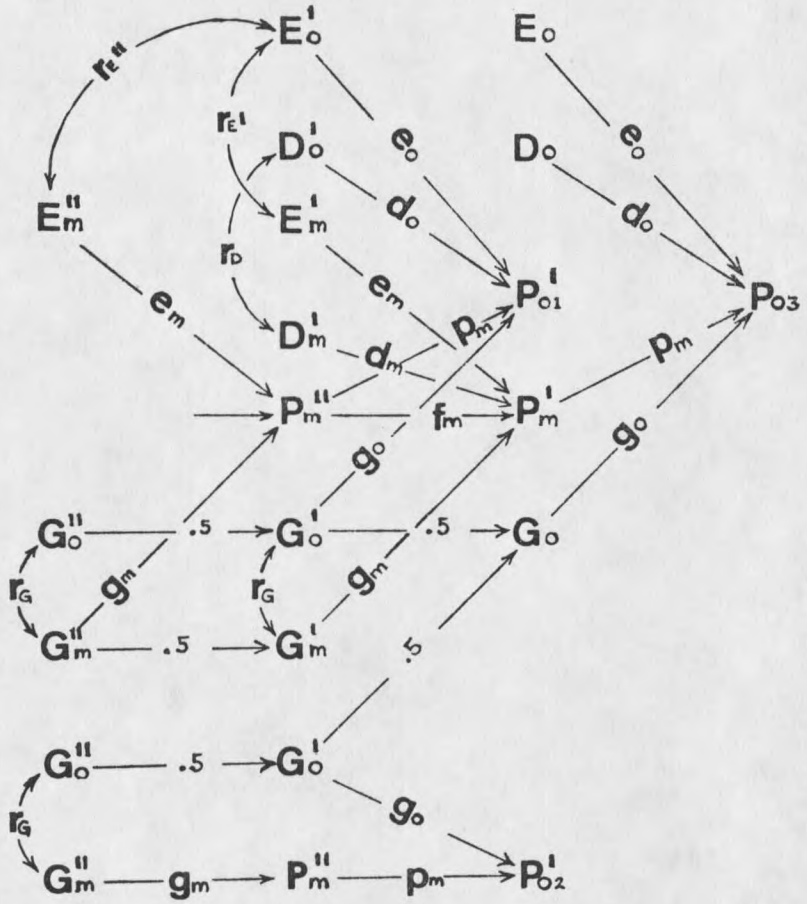


Figure 3. A path coefficient diagram describing Koch's (1972) model. Po is the phenotypic, Go is the additive genetic, Do is the dominance and Eo is the environmental value for birth weight or weaning weight expressed by individuals. Pm, Gm, Dm and Em are corresponding maternal effects. Primes on values represent parental values and double primes represent grandparent values. Path coefficients between symbols (.5, g, d, e, p, fm) are standard partial regression coefficients. Double arrows represent residual correlations between traits.

Figure 3 is the path diagram which summarizes the concepts of Koch (1972). In this diagram P_{o3}, P'_{o2} and P'_{o1} represent the phenotype for birth weight or weaning weight of the offspring, sire and dam, respectively. If all the possible paths exist, then

$$\text{cov}(O,D) = 1/2\ \sigma^2 Ao + 5/4\ \sigma AoAm + 1/2\ \sigma^2 Am + \sigma DoDm + f^2 m + \\ + (1+fm)\ \sigma EoEm + fm/(2-fm)\ (1/2\ \sigma^2 Am + 5/4\ \sigma AoAm) \quad (11)$$

Note that if fm = 0, this expression reduces to

$$\text{cov}(O,D) = 1/2\ \sigma^2 Ao + 5/4\ \sigma AoAm + 1/2\ \sigma^2 Am + \sigma DoDm + \sigma EoEm \quad (12)$$

as shown in Thompson (1976). The formulas (11) or (12) show that if genetic or environmental covariance terms are negative, the cov(O,D) can be lower than anticipated from direct genetic or maternal genetic effects (Koch,1972). This explains part of the disagreement between the estimates of h^2 by paternal half-sib analysis as compared to regression of offspring on dam ($h^2 b_{OD} = 2\ \text{cov}(O,D)/\sigma^2 P$, Falconer, 1981).

The models for estimating direct and maternal variation of birth weight and weaning weight in beef cattle have been considered in order of complexity. Some difficulties arise in the application of the more complex models to beef cattle. That is the subject of the next section.

Problems in estimating direct and maternal genetic covariances

There are some problems in estimating maternal variation in any animal species. Also, there are specific problems in estimating maternal and direct covariances in beef cattle. The order in which the problems are presented implies no order of importance.

1. Standard error of the estimates and non independence of the coefficients in the expected values

This is a general problem. The large standard error of the estimates of maternal covariances is due to the generally small number of relatives involved (specially in beef cattle) and the multipliers used (i.e., 1/2, 1/4, 1/8, 1/16) (Koch, 1972; Willham,1980). As Koch (1972) points out, errors of estimates in one component tend to cause other components to differ in the opposite direction since the sum of components is forced to equal the whole.

In general, it is expected that increasing the number of relative relationships involved would decrease the standard errors of estimation.

Designed experiments that yield specific useful covariances that are uncorrelated have been suggested by Willham (1963) and Eisen (1967). Bondari et al (1978) used two designs suggested by Willham (1963) in Tribolium castaneum to investigate genetic maternal influences on pupa weight and family size. The designs were based on creating a system of matings between families of full-sibs and paternal half-sibs. Matings between different full-sib families are also involved. At least three generations are required in all the designs. The generation interval in Tribolium is 30 days (Bondari et al, 1978); while in beef cattle the generation interval is 4.3 years (Koch et al, 1982). The problem is aggravated by the fact that the fecundity rate in cattle is low and repeated matings to produce full-sib families should take place in different years. The result

increases the generation interval. Therefore, the designs are better suited for laboratory animals than for beef cattle.

In case there are more covariances between relatives than direct and maternal covariances to evaluate, a method to solve simultaneously for the parameters should necessarily be used. Van Vleck and Hart (1966) have used least-squares, weighting the equations by the numbers of observations used in the estimate of the relative relationship. For the designs discussed in the previous paragraph, Eisen (1967) has suggested the use of the diagonal elements of $(X'X)^{-1}$ to weight the equations. He has also indicated that the procedure is not fully efficient if the variances of the estimates of the covariances between relatives are not homogeneous. This is likely to occur when different methods are used to estimate the variance components, and when the number of records used for the estimate are entirely different, as usually happens with beef cattle. To overcome this problem, Thompson (1976) has developed a modified maximun likelihood procedure. After assuming that the observations are normally distributed, the log of independent matrices of sum of squares is maximized. It is necessary to solve the equations iteratively. The case where parents are subject to culling was also considered by Thompson (1976). However, the fact that independent sum of squares is required precludes the use of the procedure when the data are not collected under a designed experiment. Another problem is that the method can not avoid the effects of the correlations between the coefficients even though the design is good (Thompson, 1976).

2. Small number of relatives involved in the estimation in beef cattle field data

This problem has partially been explained in the preceding section. Hohenboken (1973) said that it is difficult to locate enough types of families to equal the number of unknowns of interest, and it is difficult to account statistically for all environmental causes of likeness between relatives. Koch (1972) used seven relative relationships and the error of maternal half-sibs model. There is no other paper using more relative relationships than Koch(1972). The direct consequence is that some terms must be dropped from the model to solve for the rest. If the term dropped is not zero, the solution is biased and the other components are either underestimated or overestimated. The magnitude of the error depends on the correlation among the terms for that particular set of relatives and the size of the term dropped.

3. Maternal effects evaluation lenghtens the time to conduct the study

As Willham (1980) points out maternal effects are:

1) at least a generation behind the direct effects in their expression,

2) sex limited, and

3) occur late in the life of the female.

All these factors lengthen the evaluation time. If the records are taken over a long period of time, there is the possibility of introducing environmental correlations (Eisen, 1967); year by sire interaction is also possible to occur which in turn tends to inflate the sire variance component (Koch, 1972).

Estimates of direct and maternal genetic variances and covariances for birth weight and weaning weight in beef cattle

The estimates reviewed in this section are the results of applying the Willham (1963) and Koch (1972) procedures. The first approach to the problem was made by Koch and Clark (1955b).

1. Birth weight

Table 2 summarizes the estimates of published reports on birth weight. Three estimates of dairy cattle are also included.

In general, there is a trend for h^2o to be greater than h^2m. The means suggest that additive direct influences are two times the amount of additive maternal influences.

The correlation between additive direct and additive maternal effects (r_G) was negative in almost all the estimates. The few estimates and the big range of the values produce uncertainty about the real magnitude of r_G. A possible biological explanation given by Fisher and Williams (1978) is that the two opposing additive effects tend to prevent excesses in birth weight thereby protecting the survival of both calf and cow at parturition.

This hypothesis is supported by the fact that the estimates of r_G for dystocia, a trait strongly dependent on birth weight (Burfening et al, 1978), were -.19 for Philipsson (1976), -.54 for Burfening et al (1981) and -.38 (heifers) and -.25 (cows) for Thompson et al (1981).

The fifth column in Table 2 was included since Koch (1972) and Baker (1980) postulated that, when cov(O,D) is excluded from the analysis, the values of $\sigma A_o A_m$ and r_G are close to zero. The simple

TABLE 2. ESTIMATES OF DIRECT AND MATERNAL ADDITIVE GENETIC VARIANCES AND COVARIANCES ON BIRTH WEIGHT OF CATTLE

Heritabilities			Genetic correlation between additive direct and additive maternal effects (r_G)	Inclusion of COV (O,D) in the solution	Number of records used	Breed	Authors
Direct effects (h^2_o)	Maternal effects (h^2_m)	Total effects (h^2_t)					
.35	-	.42	>0	No	4,553	Hereford	Koch and Clark (1955c)
.22	.04	.00	-.93	No[a]	1,064	Holstein	Everett and Magee (1965)
.65	.15	.27	-.98	No[b]			
.56	.30	.36	-.58	Yes	789	Hereford	Brown and Galvez (1969)
.14	.25	.17	-.39	Yes	932	Angus	
.72	.48	-	-.55 to -.89	Yes	1,962	Hereford	Vesely and Robinson (1971)
.44 to .40	.05 to .12	-	-.17 to .27	Yes	4,060	Hereford	Koch (1972)
.44	.04 to .19	-	-.07 to .30	No			
.19	.08	-	-.53	No	6,724	Swedish-Friesian	Philipsson (1976)
.51	.26	.44	-.36	Yes	1,534	Holstein	Fisher and Williams (1978)
.21	.11	-	-.24	No	11,552	Simmental	Burfening et al. (1981)
.40	.17	.27	-.36				Averages

[a]Relatives included: σ^2PHS, σ^2MGS, COV(S,MGS)
[b]Relatives included: σ^2PGS, σ^2MGS, COV(S,MGS)

mean of the estimates without cov(O,D) is equal to -.35. This value does not seem to support that hypothesis.

The only two reports in which dominance and environmental maternal effects were included were Brown and Galvez (1969) and Koch (1972). However, both papers assumed $\sigma DoDm$ and $\sigma EoEm$ to be zero. The dominance direct effects accounted for 9 and 17 % of ^2P in Angus and Hereford, respectively (Brown and Galvez, 1969). The estimate of σ^2Dm was negative which is possibly suggesting that a negative source of variation was left out from the model. Koch (1972) presented a term for $\sigma^2Dm + \sigma^2Em$ that accounted for 10 % of σ^2P. This author concluded that the environmental maternal ability of dams did not have a significant direct effect on maternal ability in the next generation.

2. Weaning weight

Estimates of direct and maternal additive genetic sources of variation on preweaning growth are shown in Table 3.

Contrary to birth weight, average daily gain or weaning weight have more variation for additive maternal effects than for additive direct effects. The mean of the estimates of r_G is highly negative. Koch (1972) emphasized the fact that the estimates of r_G when the cov (O,D) was excluded from the solution were smaller and suggested an overestimation of $\sigma AoAm$. However, the last two estimates in Table 3 indicate that this is not necessarily the case. It should be noted that it is very difficult to compare estimates coming from different genetic models. The only study in which r_G was evaluated by a solution which only contains additive effects is the one of Kress et

TABLE 3. ESTIMATES OF DIRECT AND MATERNAL ADDITIVE GENETIC VARIANCES AND COVARIANCE OF CALF GROWTH THROUGH WEANING

Heritabilities			Genetic correlation between additive direct and additive maternal effects (r_G)	Inclusion of COV (O,D) in the solution	Number of records used	Breed	Authors
Direct effects (h^2_o)	Maternal effects (h^2_m)	Total effects (h^2_t)					
Average daily gain from birth to weaning							
.21	-	.12	-.65	No	4,553	Hereford	Koch and Clark (1955c)
.18	.15	.25	.0	Yes	725	Brahman	Deese and Koger (1967)
.40	.46	.17	-.73	Yes	466	Brahman x Shorthorn	
.20	.28	.32	-.05	No	4,060	Hereford	Koch (1972)
.26	.11	.12	-.40	Yes			
.26	.24	.17	-.45				Averages
Weaning Weight							
.32	.29	.34	-.31	Yes	717[a][b]	Hereford	Hill (1965)
.31	.50	.34	-.46	Yes			
.37	-	.17	-.73 to -1.07	Yes	1,692	Hereford	Vesely and Robison (1971)
.23	.54	.08	-.79	Yes	2,618	Hereford	Hohenboken and Brinks (1971a)
.23	.34	.28	-.28	No			
.14	.64	-	-1.14	Yes			
.14	.34	.32	-.07	No	228	Charolais	Baker (1980)
.20	.53	.09	-.90	Yes	3,765	Brahman	Beltran Bru (1978)
.20	.47	.02	-.75	No			
.12	.05	-	-.68	No	13,682	Simmental Crosses	Kress et al. (1979)
.22	.41	.20	-.65				Averages

[a]Relatives included: ^{2}PHS, ^{2}MGS, COV(S,MGS)
[b]Relatives included: ^{2}PGS, ^{2}MGS, COV(S,MGS)

al (1979). In the remaining ones, the solutions were obtained assuming that some of the direct and maternal covariances were zero. As discussed before, errors in the estimate of one covariance cause the other components to differ in the opposite direction, since the sum is forced to equal the total. When cov(O,D) was included in the solution, $\sigma DoDm$ and $\sigma EoEm$ were always assumed to be zero. If these terms are not null, then the inclusion of the cov(O,D) in the solution tends to overestimate the value of $\sigma AoAm$ and as a consequence, also of r_G.

The importance of determining the exact magnitude of $\sigma AoAm$ comes from the fact that response to selection for preweaning growth relies mostly on its value as shown by Van Vleck et al (1977). It is reasonable to suppose that $\sigma AoAm$ is negative since all its estimates have this sign (Table 3). This means that most of the progress made in one generation due to selection for growth rate is overcome by maternal effects in the next generation. After reviewing the literature of selection experiments in beef cattle, Koch et al (1982) concluded that the realized h^2 (the portion of the total or phenotypic change due to genetic progress) was .45 for birth weight, .24 for weaning weight, .35 for postweaning gain; .41 for final weight at the performance test and .33 for gain efficiency. Thus, selection would be less successful for weaning weight than for other growth and efficiency traits.

Important evidence for the presence of maternal effects on weaning weight are given by the comparison of maternal and paternal genetic parameters. Koch et al (1982) indicated that maternal half-