ABSTRACT

Title of Document: ROLE OF MATERNAL AND CYTOPLASMIC EFFECTS IN EARLY CALF GROWTH IN A CLOSED BREEDING NUCLEUS ANGUS HERD

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Maternal and cytoplasmic inheritance was investigated in a closed Angus herd. Observed traits were birth weight, weaning weight, adjusted body weight, average daily gain, hock length and scrotal circumference. Each animal in the herd was traced to one of 18 female founders. Data was analyzed with a model including contemporary group, gender and the random effects of animal, maternal, permanent environment, and cytoplasmic line. Ratios of cytoplasmic to phenotypic variances ranged from 0.000 ± 0.002 to 0.005 ± 0.006. Genetic maternal variances had ratios ranging from 0.044 ± 0.046 to 0.156 ± 0.029. Desired genetic gains indexes were computed for all traits. Inclusion of the cytoplasmic information in the index resulted in small reductions in genetic gains in direct and maternal values that can be compensated for a corresponding increase in cytoplasmic breeding value. Selection for cytoplasmic effects will lead to increased inbreeding unless new variation is created by mutations.

ROLE OF MATERNAL AND CYTOPLASMIC EFFECTS IN EARLY CALF GROWTH IN A CLOSED BREEDING NUCLEUS ANGUS HERD

By

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Advisory Committee:
Assistant Professor Frank Siewerdt, Chair
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Dedication

This thesis is dedicated especially to my wife Maripaz and my daughters Montserrat and Macarena. I used much of our family time to complete this project. They always have been supportive and never complained. They are my most valuable treasure and the reason for the accomplishment of this project.

To my parents Eladio and Leonor who inspired me during my youth to always go further and instilled in me that nothing important is obtained without sacrifice.

Acknowledgements

This research project would have not been possible without the support of many people. I would like to express a special gratitude to my advisor Frank Siewerdt who was always helpful and offered invaluable guidance and encouragement.

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The committee members Dr. William Ray Stricklin and Dr. Michael Scott Updike gave invaluable suggestions to complete this thesis. I would like to express my appreciation and thankfulness to them.

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Chapter 1: Introduction

In mammals, the inheritance mechanism for transmitted traits has been explained by Mendelian segregation, which is broadly accepted to describe this phenomenon. The result from mixing the parent's genetic materials during fertilization is a new cell that shares DNA from both, but strictly this happens only with the nuclear material and not the rest of the cell.

In almost all species the female gamete is larger than the male gamete and provides the cytoplasm for the new embryo. The male gamete makes almost no contribution for this cellular compartment. Sutovsky *et al*. (2000) reported that the sperm mitochondrion is destroyed shortly after fertilization and the mechanism responsible for this process is called ubiquitination and occurs inside the oocyte cytoplasm. The cytoplasm contains organelles and proteins that preexist at the time of fertilization, having been encoded by the female nucleus. These organelles have their own genetic information and its inheritance can be defined as non-Mendelian because they are only inherited from the mother. Traits that are controlled or influenced by the genetic material in these organelles express maternal inheritance.

Maternal effect is defined as the determination of progeny characters by the female parent or mediation by the genetics of the mother. Expanding on this definition, there are intrinsic genes of the mother that affect the progeny. For example, milk yield will affect the performance of the progeny but is determined only by the mother.

True maternal inheritance is due to the presence of DNA in the cytoplasmic organelles, typically mitochondria in animals and chloroplasts in plants. Cytoplasmic

and nuclear DNA are inherited independently. Replication of nonnuclear DNA occurs without recombination or regulatory influence of the nucleus. The replication mechanism of cytoplasmic DNA is different from nuclear DNA, resulting in different mutation rates (Lopez *et al.*, 1997). Mammals' mitochondrial DNA is believed to have a higher mutation rate than their nuclear DNA (Linnane *et al.*, 1989).

Most of the literature refers to cytoplasmic DNA as mitochondrial DNA (mtDNA) because mitochondria are the major carriers of DNA in the mammal cytoplasmic compartment. In plants chloroplasts are the main cytoplasmic containers of DNA. These terms are used interchangeably in this text for description and discussion purposes.

Figure 1 shows that mitochondrial DNA is passed directly from the mother to the progeny. In the absence of mutation, both should share identical mtDNA.

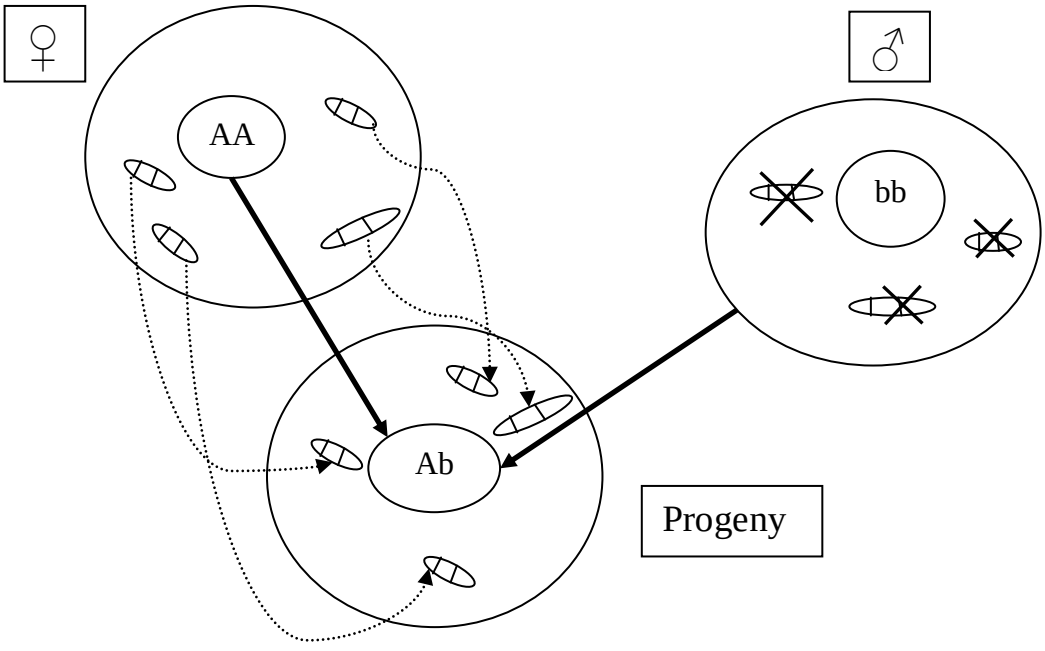

Figure 1. Inheritance of nuclear and cytoplasmic genes

Mitochondrial DNA does not undergo recombination of genes from both parents as the nuclear DNA does during meiosis. Both types of DNA are independently inherited.

According to the previous concepts, it can be said that all the progeny from a female will have the same cytoplasmic DNA. This is not quite right if there are mutations but they would be very similar. The similarity would be indirectly proportional to the mutation rate.

Some cytoplasmic inherited characteristics show an extensive phenotypic variation due to mitochondrial random segregation during cellular division. The resulting cells contain different proportions of cytoplasmic genes and most are inside the mitochondria. This process is illustrated in Figure 2.

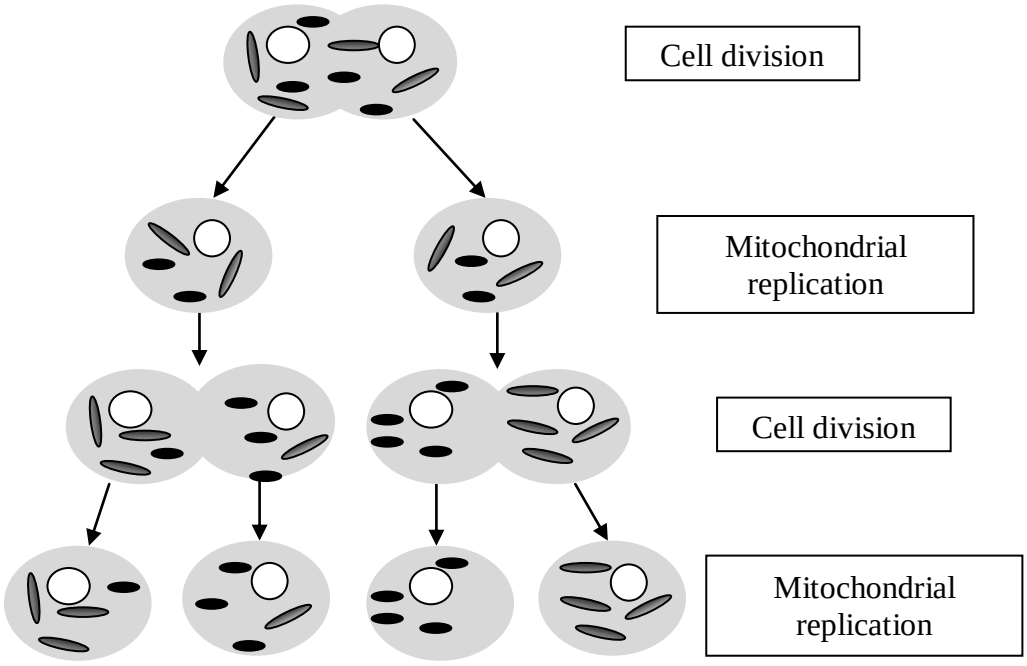

Figure 2. Mitochondrial replication and segregation during cell division

Cytoplasmic mutations can be transmitted to new cells during this process; this could explain the different number and type of mitochondria observed in new cells. Variation observed in the phenotype influenced by these genes could be maintained by these processes. Wagner (1972) reported an 8.5 fold increase in the rate of liver protein synthesis among four inbred lines of mice. Milk production is another trait that is influenced by the mitochondria; milk requires a lot of energy to be produced and this organelle plays a major role in energy production. The variability observed in these traits in closely related and highly inbred animals could be a consequence of the segregation suffered by cytoplasmic organelles during cell division.

The objectives of this study were to analyze and quantify maternal and cytoplasmic effects in order to provide essential information to understand the influence exerted by both effects in preweaning traits in beef cattle. Novel information may serve as a reference point for future studies in closed breeding nucleus herds. The inclusion or exclusion of these effects in different models will explain their importance in genotypic and phenotypic determination of preweaning traits, helping animal breeders to reach selection goals faster by accelerating genetic gain.

Chapter 2: Literature Review

Phenotypic components

Traditionally selection of animals for breeding was based on external phenotypic traits which could be either quantitative or qualitative attributes. As an initial strategy it amassed a lot of progress and many breeds were developed in domesticated species before the advent of improved genetic knowledge. Genetic progress in traits selected for occurred due to combined use of several pieces of information in the decision process, even without full realization that the phenotypic changes, partly due to the underlying genetics, could be accurately quantified.

A phenotype may be described as a combined effect of an intrinsic component in the animal (its genotype) and an external influence (the environment). This simple model can be elegantly written as:

$$P = G + E$$

This model describes a phenotype (P) being influenced by the genes of the animal (G) and the environment (E) to which it is exposed. The environmental effect encompasses a broad array of effects, including but not limited to diet, husbandry, housing conditions, and climatic factors.

Maternal effect

The previous model can be expanded by adding new components that further describe the genetic makeup of an animal and the influence of genotypes of other animals on the individual in question.

Adding the effect of the genes of an animal's mother (G_m) to the model will take into account the influence of other genes that influenced the animal's phenotype:

$$P = G + G_m + E$$

Explicitly, maternal genetic effects can be observed in the offspring due to expression of the mother's genes independently of the animal's own genotype. According to Falconer and MacKay (1996) there are two sorts of maternal effects. The first is described as the mother's phenotypic value influencing the offspring's phenotypic value of the same trait. An example of this is that larger cows produce more milk and also give birth to heavier calves. The second category of maternal effect causes the progeny's resemblance to be greater or lesser; but there is no correlation between the offspring and mother phenotypic observations. This is the case when the covariance that is observed is not the same character that produces the maternal effect. The previous authors offer an example where the tails of mice are influenced by the temperature in the nest. The nursing behavior of mothers varies, resulting in different nest temperatures. This creates an environmental covariance between the progeny of the same nest in respect to the tail length, but the mother's tail length is not related to the temperature of the nest. There is no environmental covariance between the mother and its progeny regarding tail length.

The existence of maternal effects has been proved and quantified in many species (Eisen, 1970; Rutledge *et al*., 1972; Van Vleck, 1976; Robinson, 1996; Bernardo, 1996; Bijma, 2006). The mechanism of action and how it influences the progeny varies, going from intrauterine medium regulation to milk production. The first example by Falconer and MacKay (1996) on the previous paragraph

demonstrates how the mother's genes can influence the offspring. It could also occur between fertilization, when the ovum received the paternal genetic material, and its birth. At this stage, the survival of the new fetus depends only on the mother's genes. Females that are immunologically incompatible will develop successive reactions that could induce abortion. For milk production, the form of influence is totally different. It happens after birth in an extra corporeal environment but is still regulated by maternal genes.

Cundiff (1972) summarized different point of views on maternal effects and how this concept can be applied to understanding different phenomena observed in animal breeding. To mention some of these Stormont (1972) discussed passive immunity in newborn animals resulting from the supply of gamma globulin from the mother and the importance for early survival, observing that its influence diminishes with the age of the newborn animal as they produce their own antibodies. These antibodies can be delivered pre- or post-partum; the importance of these mechanisms varies by species. Primates and guinea pigs receive antibodies transplacentally before birth; cattle, swine and horses, from the colostrum after birth. Rodents and carnivores have both mechanisms but the most relevant one is the colostral.

In economic traits the importance of these maternal effects varies depending on the species and the biology behind the observed characteristic. The importance of maternal effects has been investigated in many species and traits using different statistical models to compute their values (Rohrer *et al*., 1994; Robinson, 1996; Van Vleck, 2005). The maternal effect is an important factor in selection programs because taking it into consideration usually accelerates the rate of genetic progress.

Cytoplasmic effect

While the inheritance of nuclear and cytoplasmic genes has been elucidated, another uncertainty has emerged when trying to understand the influence of these genes located in the cytoplasmic compartment. Their importance is known and some are expressed during oxidative phosphorylation, a process that is critical for survival. Trying to apprehend the implications was not only a biological challenge: designing statistical models than could detect this effect efficiently became the next logical step.

The genetic model with the maternal effect can be expanded so that the genetic portion (G) is partitioned into nuclear and cytoplasmic components.

$$P = (G_{n+}G_k) + (G_{n+}G_k)_m + E$$

This modification affects two pieces of the model: the genetic (G) and the maternal genetic (G_m). For the purposes of this study the nuclear genetic (G_n) and cytoplasmic genetic (G_k) portions are indicated as additive genetic and cytoplasmic effects, respectively. The effect of the maternal cytoplasmic effect $(G_k)_m$ is expected to be very small and is not accounted for in the model used.

Mitochondrial DNA

Mitochondria are cytoplasmic organelles that play a critical role in energy production; one of their most important tasks is to convert energy from food into a form that cells can use. In humans mtDNA represents only a small fraction of the total cellular DNA, about 0.0005%. There are 37 genes in the human mtDNA; all of them are essential for normal mitochondrial function. Thirteen of these genes regulate the production of enzymes involved in oxidative phosphorylation, a process that

produces adenosine triphosphate (ATP) from simple sugars and oxygen. The other 24 genes regulate the synthesis of transfer RNA (tRNA) and ribosomal RNA (rRNA), both of them participating in protein assembly.

The human mitochondrial genome is defined by a single type of circular double-stranded DNA whose complete nucleotide sequence has been determined (Anderson *et al*., 1982). Human mtDNA has 16,569 base pairs (bp) and 44% of them are constituted by guanine and cytosine. The two strands have significantly different base composition: The light strand (L) is rich in cytosine (C) and the heavy strand (H) in guanine (G). Most of the mtDNA is double-stranded but a small section is a DNA structure with a triple strand, known as the 7S DNA. Twenty-eight of the genes are encoded by the heavy strand, and the other nine by the light strand. Figure 3 illustrates the human mitochondrial DNA structure.

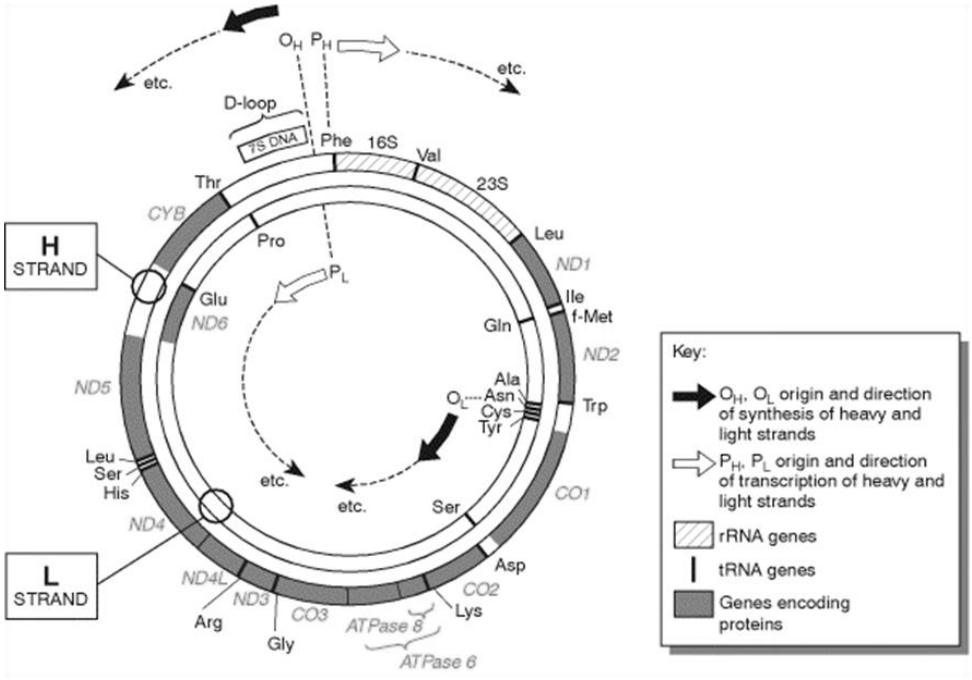

Figure 3. Human mitochondrial DNA structure
Source: (Strachan, 1999)

The human mitochondrial DNA is extremely compact, unlike the nuclear DNA. Ninety-three percent of the sequence represents coding sequence. The only section that lacks coding DNA is the displacement loop region. This section corresponds to the 7S DNA.

Replication for both strands is unidirectional and begins in predetermined and specific places. For the H strand, it starts in the D loop using the L strand as a template and the posterior displacement of the old H strand. When two-thirds of the H chain is replicated the L strand becomes exposed. In the next step the replication of the L strand occurs is the opposite direction using the H strand as a template. Figure 4 illustrates the bovine mtDNA. It consists of 16,338 nucleotides, having a sequence that is homologous to the human with genes organized virtually identically, but the D loop in the bovine mtDNA is only slightly homologous to its human counterpart.

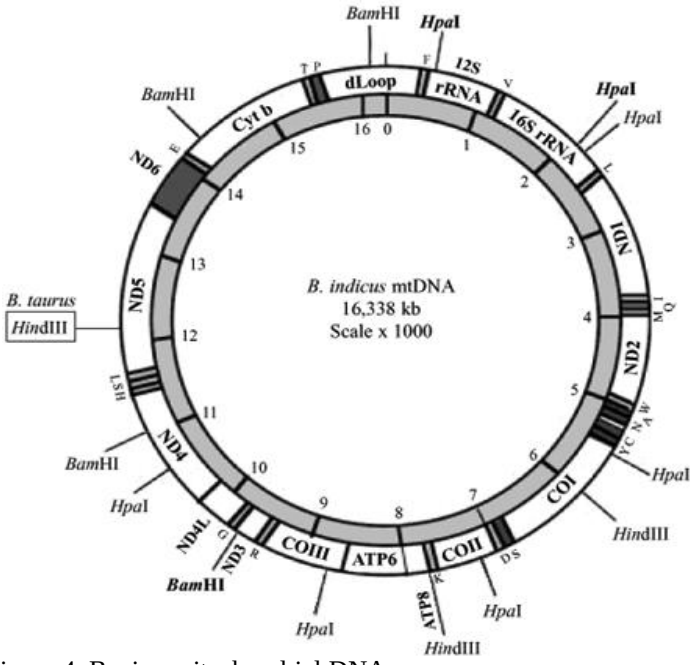

Figure 4. Bovine mitochondrial DNA
Source: (Meirelles *et al.*, 1999)

The length variability observed in this section is responsible for the bulk of the size difference between the bovine and human mtDNA (Anderson *et al*., 1982).

Wagner (1972) reported the mitochondrial importance as an inheritance mechanism due to presence of DNA and its mutation capabilities. In the same article it was mentioned that the ovum contains 100 to 1000 mitochondria and the sperm accommodate apparently just one, indicating a possible cytoplasmic inheritance process. Different rates of protein synthesis by liver mitochondria were observed between and within four inbred strains of mice, suggesting a potential mechanism of cytoplasmic inheritance.

Mothers have an important influence in many phenotypic traits. Some of them are economically important and the desire for improvement has stimulated studies to understand the mechanism of how it works (Bijma, 2006).

Maternal effects have been shown to play a role in beef cattle, especially in preweaning traits (Koch *et al*., 1972; Bell *et al*., 1985). In swine, Robison (1972) reported that direct and maternal effects have an important influence on early postnatal growth.

Practical calculation on maternal effect values always has been difficult and complex. Results have been contradictory and sometimes biased; at times the results were regarded with skepticism. Estimates of genetics correlations between direct and maternal effects are typically strongly unfavorable (Robinson, 1996; Meyer, 1997; Cheverud, 2003). Models used to analyze maternal effects do not account for the environmental covariances between a dam and its progeny (Quaas and Pollack, 1980). If a statistical model ignores the environmental covariance between dam and

offspring, the estimated correlation between direct and maternal effect may be biased (Koch, 1972).

Maternal effects are important because they affect the animal's performance and are based on an external genetic component that cannot be regulated by the individual itself. Legates (1972) added to the controversy surrounding the definition of maternal effect by defining it as any influence from a dam on its offspring, *"…excluding the effects of directly transmitted genes that affect performance of the offspring."* If cytoplasmic genes are assumed to be transmitted by the mother only, this definition is no longer acceptable and needs to be expanded to exclude the nuclear transmitted genes that are inherited from both parents.

Any factor that can affect the animal's phenotype is important to be observed and if possible to quantify how much influence it exerts. There is no agreement in the literature about the appropriate statistical power for detection of cytoplasmic effects (Brown *et al*., 1989; Tess, 1990; Van Vleck *et al*., 2003). Theories formulated trying to biologically explain this effect include cytoplasmic inheritance, intrauterine and postpartum nutrition provided by the dam, antibodies and pathogens transmitted from dam to offspring, and maternal behavior.

It has been suggested that maternal lineage effects, considered as cytoplasmic line effects, can affect yield and reproductive traits of dairy and beef cows (Albuquerque *et al*., 1998). Cytoplasmic origin was significant for total yield of fat plus protein and for milk returns (Huizinga *et al*., 1986). In Holstein cows, for milk and fat yield cytoplasmic effects accounted for about 2% and for fat percentage about 3.5%, of the total variation. Cytoplasmic effects accounted for 2%, 5% and 5% of

variance for weight at birth, average daily gain and weight adjusted at 205 days (Tess *et al*., 1987). Differences within species and even within breeds in the mtDNA have been reported in cattle (Bell *et al*., 1985). A possible biological explanation for this variability is that the mtDNA evolves faster than nuclear DNA. Brown *et al*. (1979) mentioned that species that show similar sequences in their single nuclear DNA copies demonstrate significant divergences in their mtDNA sequences. Their analyses reported a 5- to 10-fold increase in rates of evolution in primates and support the fact that high mitochondrial DNA mutation rate was responsible for this evolutionary fact.

Chapter 3: A pilot study on selection for cytoplasmic genetic effects

Introduction

Selection for cytoplasmic effects is not very well-documented in the literature. This is justified because cytoplasmic effects are small when compared to direct genetic and maternal genetic effects. In commercially important livestock species the emphasis in selection experiments has been placed on improvement of direct and maternal genetic effects due to their clear influence on economic traits. Experimentally there is a limitation in conducting selection trials that include cytoplasmic effects due to the large number of cytoplasmic lines (founder females) that needs to be established. The number of cytoplasmic lines can rapidly be reduced to one or two, rendering further improvement on cytoplasmic breeding values negligible (two founders) or even impossible (one founder). Nevertheless, a pilot study using a model organism was performed to prove that at least some gain can be achieved in cytoplasmic breeding values when selection for cytoplasmic effects is pursued in combination with direct and maternal effects.

The hypothesis for this pilot study was that genetic gain can be achieved for cytoplasmic genetic effects when the corresponding breeding values are included in a selection index along with direct and maternal breeding values.

Methodology

Five populations were formed with the red flour beetle (*Tribolium castaneum* Herbst). Two populations were assigned to each of the two selection strategies described below and one population was used as a randomly selected control group. There were two initial generations of preparatory matings, where 40 founder mating pairs were established with females from distinct genetic lines kept in laboratory conditions. These genetic lines had been reproductively isolated for 26 generations before being used in this trial. It was assumed that the females represented distinct cytoplasmic lines. There were seven generations in this trial.

At each generation, a population consisted of 50 single-pair matings, housed in individual glass jars with medium composed of enriched wheat flour (95%) and brewer's years (5%), by weight. The progeny were counted at day 28 after cohabitation. Total progeny was obtained by adding the number of larvae, pupae and young adults resulting from each mating.

The genetic model assumed the existence of an additive genetic effect (*a*), maternal genetic effect (*m*) and cytoplasmic effect (*k*). An animal model was adjusted to the data. The cytoplasmic effect was assumed to be an uncorrelated random effect in the model. Data analysis was done with the program MTDFREML (Boldman *et al.*, 1995). The covariance structure for the random effects was:

$$V\begin{bmatrix} a \\ m \\ k \end{bmatrix} = \begin{bmatrix} \boldsymbol{A}\sigma_a^2 & \boldsymbol{A}\sigma_{am} & 0 \\ \boldsymbol{A}\sigma_{am} & \boldsymbol{A}\sigma_m^2 & 0 \\ 0 & 0 & \boldsymbol{I}\sigma_k^2 \end{bmatrix}$$

The matrix shows the genetic variances on the diagonals and the covariances between the random effects on the off-diagonals. Values of zero indicate that there is no genetic covariance between the corresponding components. In the right-hand side of the equation above, **A** is the additive genetic relationship matrix (Henderson and Quaas, 1976) and **I** is an identity matrix. It can be appreciated that the cytoplasmic component is inherited independently from the other components.

All the progeny from each mating pair had the same predicted breeding value at the time of selection, since there is no data collected on the progeny yet, only on their parents. For each mating pair, three distinct breeding values were obtained, one for each random genetic effect. In the selection lines this information was combined in a selection index that included the additive plus maternal effects ($a+m$) or the additive plus maternal plus cytoplasmic effects ($a+m+k$). An index was calculated for each mating pair and the values were ranked. Each of these two lines was replicated twice.

For additive and maternal effects one genetic standard deviation (σ) of selection differential was used as relative desired genetic gains. For cytoplasmic effect 20 standard deviations were used. A few attempts were made to determine the relative weight of the cytoplasmic breeding value. This weight was decided upon because it was large enough to cause a change in the ranking between mating pairs but not too large to place an exaggerated emphasis on the cytoplasmic component with prejudice against the two other components.

The vector *b* shows the selection index used in the pilot study. The coefficients for each effect were obtained by applying the expression derived by Pesek and Baker (1969):

$$b = G^{-1}h$$

where

$$G = \begin{bmatrix} \sigma_a^2 & \sigma_{am} & 0 \\ \sigma_{am} & \sigma_m^2 & 0 \\ 0 & 0 & \sigma_k^2 \end{bmatrix}$$

and

$$h = \begin{bmatrix} \sigma_a \\ \sigma_m \\ 20\sigma_k \end{bmatrix}$$

are, respectively, the matrix of genetic covariances and the vector of desired relative genetic gains. In the selection line where no cytoplasmic effects were used, the last row and column of the matrix *G* and the last row of vector *h* were removed. The mating pairs with the eight highest index values were selected in each population as breeders. Male and female pupae were placed to produce the next generation, maintaining the population size at 50 mating pairs. All the new mating pairs were established avoiding of the placement of brother and sister (full siblings) in the same jar.

In the randomly selected control line, male and female pupae were picked from eight randomly selected mating pairs and placed as described above. Variance components were re-estimated at each generation with cumulative data, pooling the

data of all lines and adding the differences between selection lines as a fixed effect in the statistical model.

Results and Discussion

The average values for predicted additive, maternal and cytoplasmic breeding values genetic values across generations, taken as deviations from the means of the randomly selected control line, are presented graphically below. The data in the next three figures are presented in standard deviation units to allow comparison of relative gains in each genetic component.

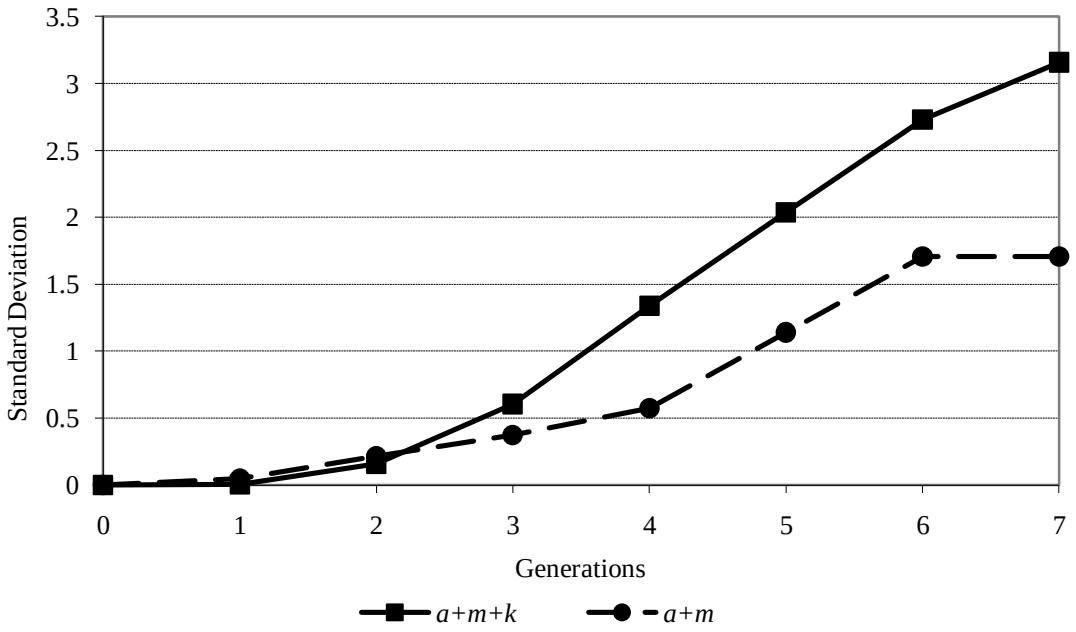

Figure 5. Additive genetic values expressed as standard deviation from the control group
(a+m+k) = Model with additive, maternal and cytoplasmic effect
(a+m) = Model with additive and maternal effects

In Figure 5 it can be seen that the complete model with additive, maternal and cytoplasmic effects *(a+m+k)* resulted in higher direct genetic gains than the model lacking cytoplasmic effect *(a+m)*. The increment was smaller from generation 1 to 3, then increased and stayed linear from generations 3 to 6. In the last generation the slope decreased a bit but there were still gains in additive genetic value.

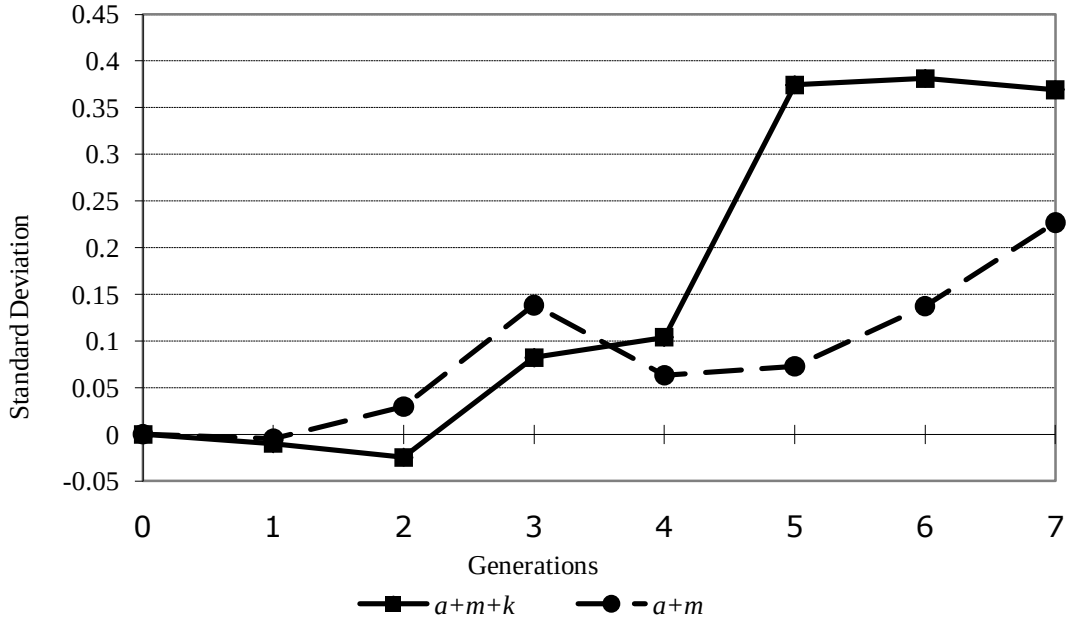

Figure 6. Maternal genetic values expressed as standard deviation from the control group
(a+m+k) = Model with additive, maternal and cytoplasmic effects
(a+m) = Model with additive and maternal effects

Analyzing the maternal genetic effect in Figure 6, it can be observed that the model with three effects resulted in greater genetic progress on maternal genetic value than the model only with additive and maternal values. A large gain was observed from generation 4 to 5. On generations 6 and 7 there was no gain in genetic maternal value in line (*a+m+k*). It is possible that maternal genetic variance has been

exhausted or had been severely reduced by generation 5, leaving no room for further genetic improvement.

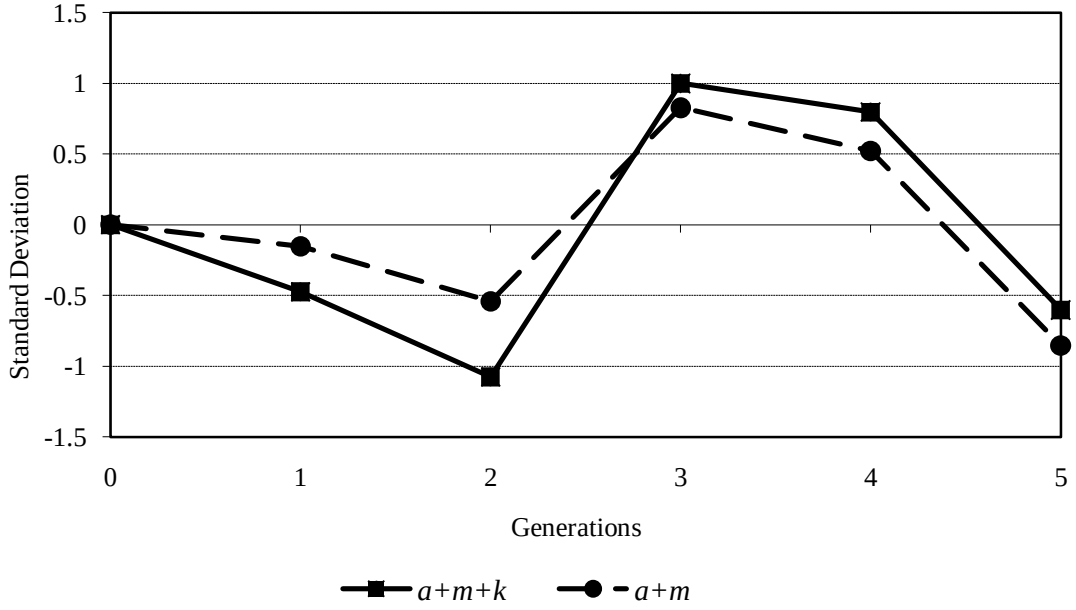

Figure 7. Cytoplasmic genetic values expressed as standard deviation from the control group
(a+m+k) = Model with additive, maternal and cytoplasmic effects
(a+m) = Model with additive and maternal effects

Figure 7 depicts only five generations because of the cytoplasmic line preparation and the cytoplasmic lines are always traced back two generations in order to be meaningful. The important result here is the genetic gain accrued between generations 2 and 3. This increment was just over two standard deviations going from –1.08 to +1.00. After generation 3 all individuals traced back to very few founders; hence there was little genetic variability available in cytoplasmic effects. Even though the cytoplasmic lines were redefined at each generation, the variance between females was very low and, despite being defined as having no covariance with the other two genetic components, it is possible that the covariance became negative, which could

explain the nominal drop in average values after generation 3. It must be remembered that only when data on generation 7 was collected could the cytoplasmic genetic values be obtained for generation 5, since they represent the newly defined founders from two generations back.

Conclusion

The data supports the hypothesis that it is possible to make genetic progress for cytoplasmic genetic effects but that the gains will be rapid and limited to a few generations. When cytoplasmic effect is combined with direct and maternal effects the response obtained is higher than using only the last two genetic components.

Chapter 4: Material and Methods

No animal care and use protocol was required by University of Maryland since this work was conducted with existing pedigree information and farm production records. Data were obtained from the records of the Wye Research and Education Center, University of Maryland, which is responsible for the management of the herd.

Wye Angus Herd

The Wye Angus herd is owned by the University of Maryland College Park Foundation and was established in 1937 with the acquisition of the first two bulls and 18 yearling heifers (Lingle *et al*., 2001). Two years later, eight additional heifers from a different source were introduced in the herd. Between 1942 and 1958, 19bulls from the British Isles were imported, all of them unrelated to the founder animals. Since 1958 the herd has been closed to outside breeding (Brinks and Katsigianis, 1982). The imported bulls contributed about 75% of the current germplasm in the herd. Full pedigrees of the newly added animals were recorded by the American Angus Association. The first calf crop was in 1939. The herd was reopened for a short period of time for a research project but the progeny of the imported animals were not kept for breeding. All these unique characteristics make this herd a valuable resource to perform population genetics studies.

The set of records includes information collected from 1939 to 2006 (67 years). There are 10,841 animals in the pedigree but not all of these have recorded phenotypic observations. Genetic relationships between the animals have been

established and reported in a previous study (Carrillo and Siewerdt, 2010). Records were screened and checked for inconsistent or erroneous information and only reliable ones were used. The number of records for different traits differs and will be treated according to the situation, taking into consideration the nature of the trait (e.g., scrotal circumference is measured only in male calves) and the statistical model that best fits each trait.

Statistical model

Four traits were measured directly on the animals. Body weights were recorded at birth (WB) within the first 24 h and at weaning (WW); hock length (HL) was also measured at birth while scrotal circumference (SC) was measured at weaning. Weaning weights were adjusted to a constant 205 days of age (W205) basis by employing an additive correction factor generated specifically for this herd. Average daily gain (ADG), from birth to 205 d, was obtained for every weaned calf.

Data was initially analyzed with an animal model that includes fixed effects and the direct genetic, maternal genetic and permanent environmental effects:

$$y = X\beta + Z_1 a + Z_2 m + Z_3 c + e$$

where y is a vector with the phenotypic observations on a trait, β is a vector of fixed effects (gender if appropriate, year of birth, age of cow, and age at weaning if appropriate), a is a vector with breeding values for direct genetic effects, m is a vector with breeding values for maternal genetic effects, c is a vector with permanent environmental maternal effects, e is a vector with the random errors, and X, Z_1, Z_2

and Z_3 are design and incidence matrices that relate each observation to the corresponding levels and values of its fixed and random effects. This model acknowledges the presence of maternal genetic effects, but is not adequate to determine the cytoplasmic effect. The model needs to be completed with the founder line information assuming that the cytoplasmic genes will be the same for every animal in the same founder line except for mutations that will accumulate along the number of generations between the founder female and the observed animal. Further details are provided below.

The expanded animal model with cytoplasmic effects is:

$$y = X\beta + Z_1 a + Z_2 m + Z_3 c + Z_4 k + e$$

Z_4 is an incidence matrix tracing each individual to its founder female and k is a vector with the cytoplasmic effects for each animal; all other matrices and vectors have been defined in the preceding model. In each row of Z_4 all the elements are zero except for one, which takes the value $(1 - \omega)^t$ where t is the number of generations between the founder female and the animal (t = 0, 1, 2, …) and ω is the mutation rate for cytoplasmic DNA. The inclusion of the cytoplasmic effect requires building this novel matrix (Z_4) that establishes the relationships of the cytoplasmic genes between all animals in the pedigree. A small example is used to illustrate the process in Figure 8. Assume that V and W are the founder females in this pedigree and that they are unrelated on the maternal side of their pedigrees.

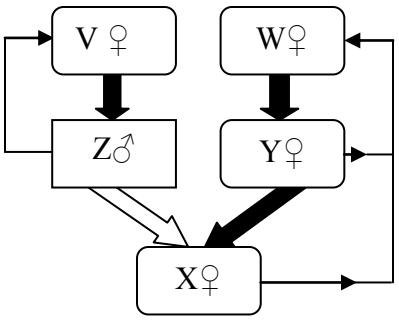

Figure 8. Cytoplasmic genetic material flow and founder line traceability

Male Z inherited the cytoplasmic DNA from its dam V; both Y and X inherited the cytoplasmic DNA from W. Barring mutation, V and Z have the same cytoplasmic genotype and the same applies to W, Y, and X. Mutations in cytoplasmic DNA will result in slight differences in the cytoplasmic DNA of a dam and its progeny. The differences will accumulate across generations. The 5 animals from Figure 8 are represented in this order in the next matrix: V, W, Z, Y and X. the new matrices in this model can be written as:

$$Z_4 = \begin{bmatrix} 1 & 0 \\ 0 & 1 \\ (1-\omega) & 0 \\ 0 & (1-\omega) \\ 0 & (1-\omega)^2 \end{bmatrix} \quad k = \begin{bmatrix} k_V \\ k_W \end{bmatrix}$$

The covariance matrix between cytoplasmic effects uses as a reference the cytoplasmic effects from the founder females:

$$V = \begin{bmatrix} \sigma_k^2 & 0 & (1-\omega)\sigma_k^2 & 0 & 0 \\ & \sigma_k^2 & 0 & (1-\omega)\sigma_k^2 & (1-\omega)^2\sigma_k^2 \\ & & (1-\omega)^2\sigma_k^2 & 0 & 0 \\ \text{Symm.} & & & (1-\omega)^2\sigma_k^2 & (1-\omega)^3\sigma_k^2 \\ & & & & (1-\omega)^4\sigma_k^2 \end{bmatrix}$$

Two assumptions are that the mutations are directional and accumulate exponentially. The quantity (1–ω) will be referred to as the discount. The power to which the discount is raised corresponds to the number of steps that can be traced between the two individuals, routing through the founder female.

This is a general approach valid for any mutation rate ω and assumes that the mutation rate is equal for each cytoplasmic genotype of the founder females. If there is no mutation rate then ω=0 and all the coefficients of matrix Z_4 will be 1 or 0. Using this matrix implies knowing the value of ω. As generations accrue, then the accumulation of mutations will weaken the original cytoplasmic effects from the founder females. The overall cytoplasmic effect could be greater or smaller than the one from the founder female, depending on the result of the mutation. However, it is not possible to predict the effect of cytoplasmic mutations with the animal model because these are not traceable to a founder female.

Values of ω will be very low so the function $(1 - \omega)^t$ can be approximated by a linear regression to estimate the cumulative mutation rate. Using this approximation allows circumventing a computationally extensive strategy of including another random effect in the model (Bell *et al*., 1985; Kennedy, 1986). The portion of the model Z_4k can be removed and a regression over the number of generations from founder female to the target animal is added to the fixed portion of the model. The maternal effect was analyzed as one component, consisting of the nuclear and cytoplasmic components, represented by the mother, and the cytoplasmic effect represented by the female founder in the model.

Data was analyzed using the software suite Multiple Trait Derivative-Free Restricted Maximum Likelihood (MTDFREML) (Boldman *et al*., 1995). Table 1 shows the structure of the fixed effects, covariables and uncorrelated random effects used for the analysis of each trait.

Table 1. Trait component structures used for the analysis

	Fixed Effects				Covariable	Uncorrelated Random effects	
Traits	Generations	Gender	Year of birth	Age of cow	Age at weaning	Permanent environment	Founder female
Birth weight	Yes	Yes	Yes	Yes		Yes	Yes
Weaning weight	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Adjusted weight, 205 d	Yes	Yes	Yes	Yes		Yes	Yes
Average daily gain	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Scrotal circumference	Yes		Yes	Yes	Yes	Yes	Yes
Hock length	Yes	Yes	Yes	Yes		Yes	Yes

Age at weaning was not used as a covariable in the model for WB, W205, and HL. Gender was not included as a fixed effect for scrotal circumference.

MTDFREML software requires the use of starting values for the analysis of each trait. The algorithm used by this program searches for the most likely values for the parameters given the data, from the numbers assigned as priors. Table 2 shows the starting values used for the analysis.

Table 2. Genetic parameters for preweaning traits on the Wye Angus herd

	Birth weight (kg)	Weaning weight (kg)	Adjusted weight, 205d (kg)	Average daily gain (kg)	Scrotal circumference (cm)	Hock Length (cm)
σ^2_p	17.605	658.74	601.64	0.01219	4.875	1.180
σ^2_a	6.952	149.12	169.06	0.00317	1.243	0.485
σ^2_m	2.023	65.91	64.87	0.00128	0.286	0.117
ρ_{am}	-0.084±0.109	-0.158±0.124	-0.104±0.122	-0.198±0.121	-0.200±0.285	-0.259±0.157

σ^2_p=Phenotypic variance; σ^2_a=Additive genetic variance; σ^2_m=Maternal genetic variance; ρ_{am}=Additive-maternal correlation (Carrillo et al., 2011)

Twice the natural logarithm of the likelihood given the data was used as a comparative method to determine the efficiency of the models. Smaller values mean that the model fits better the present data set.

After analyzing each trait with the complete model, alternative models were used for subsequent analyses. The objective of doing this was to see how the variance components changed and identify where the variance they explained goes when a factor is removed from the complete model. Adding factors to a model also has statistical and practical inconveniences that were taken into consideration during the analysis.

Once the genetic parameters and proportions were known, a selection index was created based on desired gains. For additive genetic and maternal effects one standard deviation was used; for cytoplasmic genetic effects 20standard deviations were defined. The numbers were adjusted to be the same as the ones used in the pilot study to facilitate interpretation of the results and comparison.

The large number used for cytoplasmic effect (20σ) is justified by the small proportion of it in the total variance; thus, to obtain a change in the animals' ranking this value must be used. To obtain the coefficients the following expression was used:

$$b = G^{-1}h$$

where *b* is a vector of the coefficients to be obtained, G^{-1} is the inverse of the genetic variance and covariance matrix and *h* is a vector of the desired gains. In this case the desired gains were defined as standard deviations. The previous formula was expanded to matrix notation for both cases.

The first one includes the cytoplasmic effect:

$$b = \begin{bmatrix} a \\ m \\ k \end{bmatrix} \quad G = \begin{bmatrix} \sigma_a^2 & \sigma_{am} & 0 \\ \sigma_{am} & \sigma_m^2 & 0 \\ 0 & 0 & \sigma_k^2 \end{bmatrix} h = \begin{bmatrix} \sigma_a \\ \sigma_m \\ 20\sigma_k \end{bmatrix}$$

The *b* vector has the coefficients for additive, maternal and cytoplasmic effects. It can be observed in the *G* matrix that the off diagonal values for cytoplasmic are zeros; this is due to the independent inheritance of the cytoplasmic genetic material. In vector *h*, additive and maternal effects have a coefficient of one while cytoplasmic is multiplied by twenty.

The second one shows the matrix notation without the cytoplasmic effect. The elements that stayed in the formula are the same as in the first one. But the coefficients obtained in vector *b* would be different.

$$b = \begin{bmatrix} a \\ m \end{bmatrix} \; G = \begin{bmatrix} \sigma_a^2 & \sigma_{am} \\ \sigma_{am} & \sigma_m^2 \end{bmatrix} h = \begin{bmatrix} \sigma_a \\ \sigma_m \end{bmatrix}$$

After solving for *b* and using their respective values, expected gains for each index were computed using the following formulas.

Starting from the basic principle

$$Pb = Gv$$

and knowing that expected genetic gain is calculated from

$$g = \frac{b'G}{b'Pb}$$

then replacing *Pb* with *Gv*, the formula used for computation was obtained as

$$g = \frac{b'G}{b'Gv}$$

where, depending on which index is employed

$$b' = [a\ m\ k] \quad \text{or} \quad b' = [a\ m]$$

$$v = \begin{bmatrix} 1 \\ 1 \\ 1 \end{bmatrix} \quad \text{or} \quad v = \begin{bmatrix} 1 \\ 1 \end{bmatrix}$$

Vector *v* represents the economic values for each effect. In this case all of them have the same importance and it does not matter which factor the gain is obtained from. For this reason a value of one was assigned for each vector element.

Chapter 5: Results and Discussion

Population Descriptors

Age of cows

The herd structure regarding the age of cows is an important observation that could explain the biology behind the numbers obtained in the analysis. Figure 9 describes the historical herd age composition. Comparison with management information could illuminate the policy used for different decisions, like age of animal replacement, precocity of the herd or even the proportion of animals used as donors.

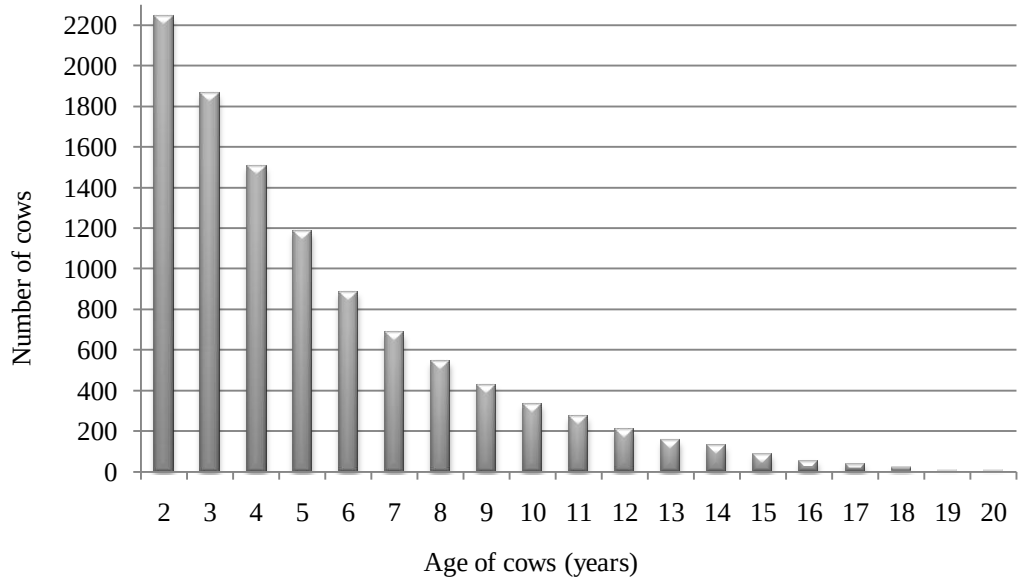

Figure 9. Historical distribution of cow age at the time of birth of their progeny

Two-year-old cows represent approximately 21% of the females, being the largest group. Around 63% of the females were five years old or younger. Cows of

nine years old or less constitute 86% of the total cows. These numbers tell that the replacement rate for females was high and most of the newborn females were kept for reproduction. Almost all females had a first opportunity to breed and after that they were subjected to the first culling. The cows were under a continuous selection process and the progressive decay in the breeder's number as they aged is a manifestation of the yearly culling. In a closed nucleus herd the breeders can only be replaced by the calves from new generations of the same herd. Records of some of the females result from their use as donors for embryo transfer. It does not mean that they were physically present at the moment of their calf's birth. This is an important observation when the data is analyzed because the interpretation of the information may otherwise be distorted.

New technologies allow for selection of animals to be bred in the future, taking advantage of genetic evaluation programs that require a long period of time to produce results. Progeny testing in bulls, for example, has been and is still used as one of the best options to evaluate males. Frozen semen technology was an important development to preserve gametes from animals that were dead at the time they were selected as breeders, but embryo transfer allows breeding animals from genetic parents that do not exist anymore. Multiple stage selection systems also are beneficiaries of new reproduction technologies; the animals can undergo many stages of selection until the breeder decides which one will be reproduced.

The use of previously mentioned technologies could have an effect on the progeny. How old the embryo was at the moment of the transfer or how well it was

preserved are unknown factors that could be partially accommodated for by including age of cow in the animal model.

Birth weight by age of cows

The average birth of weight across all cow ages was 34.34 ± 0.05 kg. Figure 10 summarizes the birth weight across all female ages and individually by group.

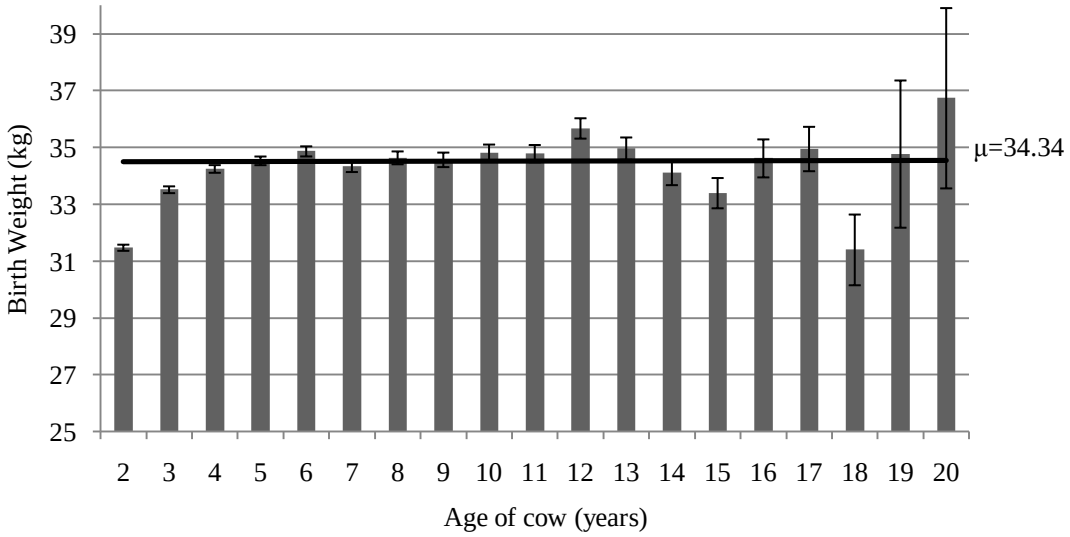

Figure 10. Calves' birth weight averaged by cow ages

Eighteen-year-old cows gave birth to the lighter calves weighing 31.40 ± 1.24 kg, but these were only 13 of 7984 animals with a recorded birth weight. The second lightest group by calf weight was the two-year-old cows with a mean of 31.47 ± 0.10 kg. Representatively this is the more important group of the two because it includes 1721 animals. The difference observed could be explained by the use of bulls that have lighter progeny with the first parturition cows. Smaller differences could be consequences of physiological events that occur at different stages of the animal's life

and could affect the trait in question. The heaviest calves belonged to the twenty-year-old group with 36.74 ± 3.17 kg, but it was represented only by two animals, resulting in a large standard error.

Birth weight was the trait chosen to demonstrate how the age of the cow is a factor that should be taken in consideration for correctly building a statistical model for data analysis.

Cytoplasmic Line Generation Frequencies

Approximately 54% of the animals had founder females that were traced back between four and eight generations. The maximum number of generations was eighteen and only four animals traced back this far. About 33% of the individuals (3535) had founders with more than eight generations, constituting a good population to study this long-term effect. The presence of generation 0 in the chart is due to the 18 founder females.

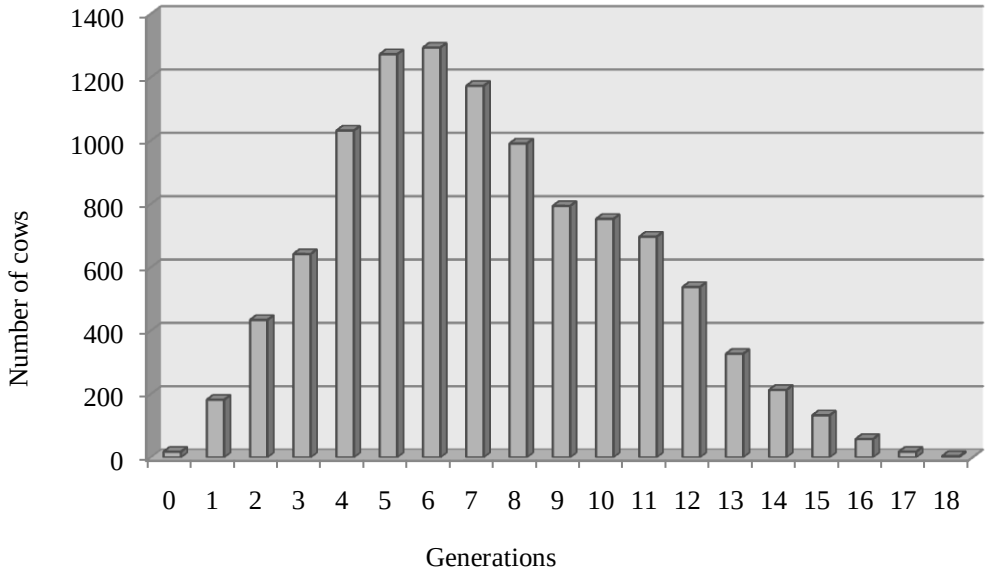

Figure 11. Number of generations from the founder female

Founder Contribution

Founder 19370077 was the most represented in the herd with a total of 1500 descendents. Cows 19370082 and 19370083 were ancestors of 1412 and 1371 animals, respectively. The next two female founders, 19390091 and 19390097, contributed 1197 and 944 descendants, respectively. These five animals were the founders of almost 60% of the total number of individuals in the herd. Founders 19370084, 19370086, 19390090, 19390092 and 19390094 have contributed less than 1% each to the herd. Female 19390094 was the least-important cow regarding number of progeny in the herd. There were only 268 calves without assigned founders in the whole herd. Figure 12 illustrates the contribution to the herd by each founder cow.

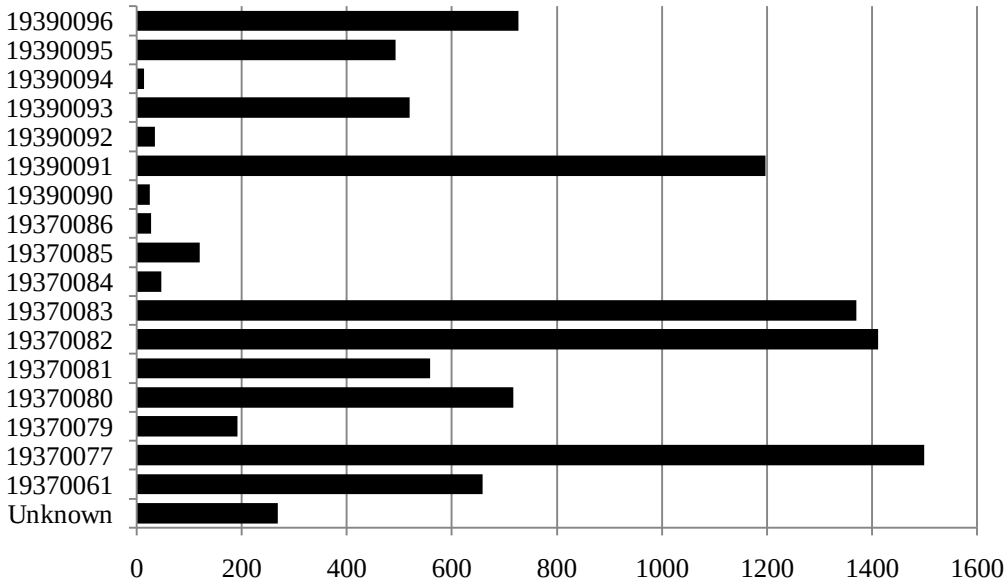

Figure 12. Total progeny number per founder in the Wye Angus herd

The number of progeny per founder female presented above was counted from 1939 until 2006. Figure 13 shows the founders' contributions in the last three calf crops, from 2004 to 2006. It could be helpful to analyze the current situation of the herd regarding the cytoplasmic lines as a methodological resource for making selection and management decisions.

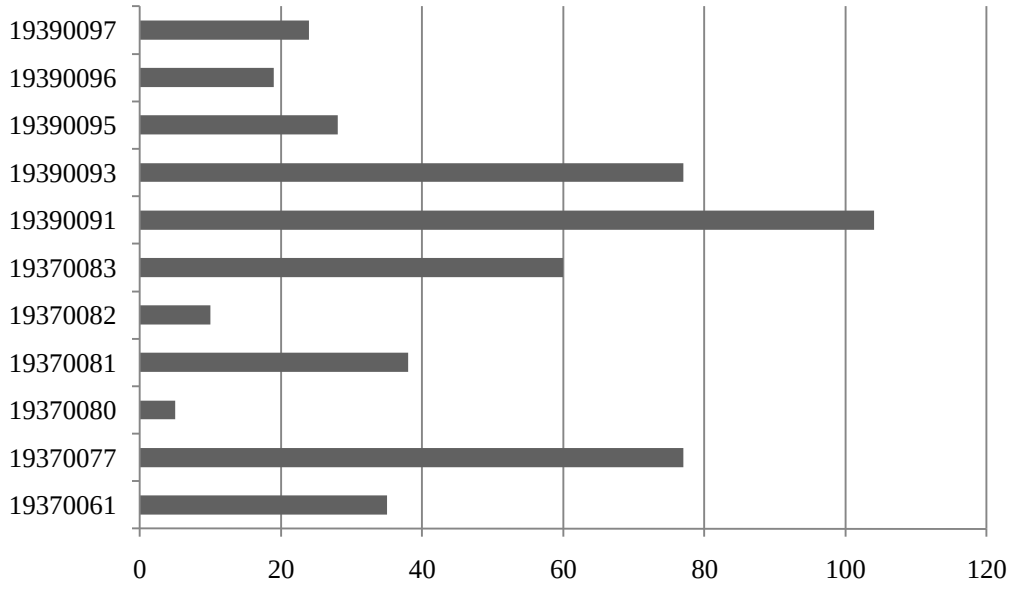

Figure 13. Number of progeny per founder from 2004 to 2006

It can be observed that only 11original founder lines are represented in the last three calf crops. The ranking changed completely when compared to the contribution of the founders for the entire herd across all years.

Cow number 19390091 was the animal with the highest representation in the last three years, contrary to the fourth place that she occupied in the general ranking. Animal 19370077 reduced its contribution to the herd, becoming third in the last three years but maintaining first place overall.

The selection criteria that have been taken place in the herd could contribute to the reordering of the founders and even would provide an explanation for the disappearance of influence of some founder cows like 19370079 and 19370085 during the last three years.

Population Trait Descriptors

Traits WB, WW, W205, and ADG had similar numbers of valid records. The data collection for these traits started at the same time. For SC and HL, collection started later (in 1983 and 1987, respectively) and the large difference in the numbers also reflects the fact that scrotal circumference is only measured in males. The population descriptors are summarized in Table 3. Values are biologically acceptable and could give a general idea about the quality of the herd. The maximum and minimum provide the range of values observed, and describe each trait's biological limits in the Wye Angus herd.

Table 3.Wye Angus herd population descriptors

Traits	Number of records	Mean	Standard Deviation	Min Value	Max Value
Birth weight (kg)	7984	33.71	4.49	13.61	56.70
Weaning weight (kg)	7986	232.42	36.98	74.39	396.90
Adjusted weight, 205 d (kg)	7985	232.00	28.99	80.29	323.87
Average daily gain (kg)	7983	0.921	0.138	0.245	1.470
Scrotal circumference (cm)	1846	21.63	3.05	14.00	41.00
Hock Length (cm)	3276	26.62	1.11	21.59	30.48

The values for birth weight and weaning weight were similar to those reported by Robinson *et al*. (1996). The mean scrotal circumference of 21.63 cm is smaller than the 34.70 cm reported by Garmyn *et al*. (2011). They reported a minimum of 20 cm and a maximum of 49 cm, giving a range of 29 cm. Their records corresponded to

bulls aging from 288 to 549 days. The smaller age range and the use of a single lineage in the present study justify the difference observed between these studies.

MTFREML Data Analysis Results

The complete model, accounting for additive genetic, maternal genetic, permanent environment and cytoplasmic genetic effects, was used for every trait.

Due to traits' natural diversity and the different scales used, presenting them in a table with absolute values for the variance components would be cumbersome. For this reason Table 4 shows the proportion of variance and covariance for different traits and models. This presentation makes it easier to determine the contribution that each of these effects made towards the total phenotypic variation.

Results from complete model will be used as a reference for comparison of the genetic values obtained in this study with others. For WB the genetic values obtained were similar to those reported by Robinson (1996) with values of six for additive and two for maternal variances. These were observed in an Australian Angus population. The size and variability of that herd compared to the Wye Angus one is larger by approximately 7,710 animals. The difference in the values could be explained by the larger size and also the closing of the Wye herd to new breeders resulting in more inbreeding than for the Australian herd. Rohrer *et al.* (1994) reported 8.850, 4.816 and -3.796 for the previously mentioned genetic values. The population in their study was constituted by Angus, Brahman and unknown breeds with 7,353 records. The larger diversity is reflected in the values but they still could be considered similar to those obtained in this study.

Cytoplasmic variance for WB was less than 0.0000 in the present study. It agreed with the values obtained by Rohrer *et al*. (1994) and Van Vleck *et al*. (2005). The latter authors' results were based on Polypay sheep, where he described a lack of cytoplasmic effect in four traits, WB being one of these.

WW values were 149.265, 67.919 and -14.968 for additive genetic variance, maternal variance and additive-maternal covariance. Permanent environmental variance with a value of 104.710 and cytoplasmic variance of 1.448 represented 0.16 and 0.0022 portions, respectively, of the total variance for WW. Robinson (1996) obtained 132 and 74 for additive and maternal variances in this trait, with proportions of 0.26 and 0.15, respectively, of the total variation. In this study the proportions were 0.23 and 0.10. respectively. The latter value is smaller than that obtained by Robinson (1996) but the model that he used lacked permanent environmental effect which could have been confounded within the maternal effect. Van Vleck *et al*. (2005) reported 0.24, 0.09, 0.04 and 0.00 for additive, maternal, permanent environment and cytoplasmic variance proportions, respectively, with sheep data. These values were similar to the ones found in the present study, except for the permanent environment, which was 0.12 lower in their study.

Table 4. Parameter estimates from fitting different models

	a^2	m^2	am	c^2	k^2	e^2	σ_p^2	-2Log
WB(kg^2)								
(a+m+c+k)	6.7479	3.2772	-0.1154	0.0669	0.0000	8.2655	18.2422	28927.65
(a+m+c)	6.7296	3.3923	-0.1691	0.0008	-	8.2743	18.2278	28929.77
(a+c+k)	7.3975	-	-	1.8258	0.0066	8.0186	17.2484	28967.98
(a+m+k)	6.6364	3.4329	-0.1297	-	0.0002	8.2981	18.2379	28929.85
*(a+m+c+k)**	6.6298	3.1991	-0.1117	0.0151	0.0000	8.3542	18.0865	28913.23
WW(kg^2)								
(a+m+c+k)	149.265	67.919	-14.968	104.710	1.448	354.765	663.138	57995.43
(a+m+c)	149.024	68.168	-14.435	104.769	-	355.011	662.538	57996.27
(a+c+k)	145.129	-	-	143.076	1.306	359.570	649.081	58031.67
(a+m+k)	143.795	240.610	-9.986	-	1.728	366.679	742.826	58072.88
*(a+m+c+k)**	143.731	58.883	-6.666	106.465	0.233	357.580	660.226	58047.34
W205(kg^2)								
(a+m+c+k)	167.274	61.261	-9.810	117.414	1.888	269.991	608.018	56735.04
(a+m+c)	167.248	61.077	-8.597	117.662	-	270.176	607.566	56736.50
(a+c+k)	163.769	-	-	153.270	1.423	274.234	592.697	56768.08
(a+m+k)	154.228	264.599	4.552	-	2.634	283.668	709.681	56831.22
*(a+m+c+k)**	169.230	60.765	-10.933	116.122	0.970	270.046	606.202	56790.16
ADG(kg^2)								
(a+m+c+k)	0.00318	0.00131	-0.00040	0.00222	0.00002	0.00593	0.01226	-28298.47
(a+m+c)	0.00339	0.00159	-0.00057	0.00214	-	0.00584	0.01239	-28296.88
(a+c+k)	0.00300	-	-	0.00293	0.00002	0.00607	0.01202	-28257.38
(a+m+k)	0.00297	0.00508	-0.00031	-	0.01399	0.00003	0.00622	-28200.59
*(a+m+c+k)**	0.00320	0.00128	-0.00042	0.00222	0.00001	0.00593	0.01221	-28432.80
HL(cm^2)								
(a+m+c+k)	0.47431	0.12173	-0.05670	0.03743	0.00167	0.59949	1.17793	3332.40
(a+m+c)	0.47553	0.12407	-0.05801	0.03712	-	0.59900	1.17770	3332.57
(a+c+k)	0.45013	-	-	0.08863	0.00243	0.61647	1.15766	3343.29
(a+m+k)	0.46778	0.17380	-0.06254	-	0.00117	0.61063	1.19082	3334.52
*(a+m+c+k)**	0.48276	0.11657	-0.05882	0.04180	0.00101	0.59774	1.18107	3303.32
SC(cm^2)								
(a+m+c+k)	1.45715	0.19481	-0.08809	0.52878	0.02148	2.28728	4.40141	4471.39
(a+m+c)	1.45501	0.22084	-0.09179	0.52608	-	2.28910	4.39924	4472.38
(a+c+k)	1.46633	-	-	0.60278	0.02381	2.29478	4.38770	4472.42
(a+m+k)	1.39903	0.57341	-0.06820	-	0.02026	2.54205	4.46654	4482.73
*(a+m+c+k)**	1.45525	0.15736	-0.04723	0.54130	0.02138	2.29720	4.42525	4461.51

WB=birth weight; WW=weaning weight; W205=weight adjusted 205 days; ADG=average daily gain; HL=hock length; SC=scrotal circumference

*(a+m+c+k)** = Complete model without number of generation from founder as a fixed effect

On W205 the values were 167.274, 61.261, -9.810, 117.414 and 1.888 for additive variance, maternal variance, additive by maternal covariance and cytoplasmic variance, respectively. The proportions and correlation were 0.28, 0.10, -0.10, 0.19 and 0.003, respectively. In this case the phenotypic observations were adjusted to 205 days but showed the same behavior as WW because they were obtained from the same observation. Some studies used only WW or W205. Where weaning weight adjusted to 205 days-of-age is available, it is a better choice of comparison.

Tess *et al*. (1987) reported contrary results of 4.57% and 1.61% in two Hereford herds for W205, being much higher that the values founded in this study. The model used by these authors was different compared to this one; theirs included fixed effect of the overall mean, year-selection line combination, sex of calf and age of dam. In the case of age of dam they grouped by 2, 3, 4, 6 to 10 and more than 10 years old. Random effects were sire, cytoplasmic line and residual. It can be seen that this is a simpler model than the one used in this study. The model did not include generations from founder in the fixed effects portion and in the random portion maternal effect was not taken into account. The last item is critical because the cytoplasmic effect could be confounded within the maternal effect for some traits, as has been observed in the present study where maternal effect was removed from the model. Also, computational resources were more limited at that time so the cytoplasmic effect could be confounded with other factors that the model did not considered, giving a larger value in their case.

For ADG, cytoplasmic variance was 0.00002 with a diminutive proportion of the total variance of 0.0019, agreeing again with Rohrer *et al*. (1994), who reported

zero, but contrary to Tess *et al*. (1987) who reported 4.89 % and 1.54 % in two Hereford herds. The possible explanation for the difference is the same explained in the previous paragraph for the W205 trait.

For HL additive-by-maternal correlation was negative with a value of -0.24 and a covariance of -0.0567. Comparison of the HL values obtained with the literature was impossible because of the lack of similar studies. Therefore, this study is a valuable resource for upcoming research projects determining genetic parameters for this trait. Hock length is a measurement used as a descriptor of carcass size. It could be interesting to analyze it in studies with other frame-size indicators and try to establish its importance and correlation between them. It could be used as a predictor for other traits and also used as a selection criterion for specific goals.

In SC the proportion observed for cytoplasmic effect was the biggest one, reaching almost 0.5 % of the total variation. Values reported by Garmyn *et al*. (2011) were 0.46 and 3.3718 for heritability and additive variance. The cytoplasmic variance proportion reported was less than 0.001. The additive variance was larger than that reported in the present study. The use of different breeds and different age at measurement resulted in more variability and was reflected in the phenotypic variance of 7.33 reported by them, contrary to the 4.40 found in this study. The present study was conducted in a single-breed closed herd with less variability than the population observed by Garmyn *et al*. (2011).

Heritability is a function directly related to the additive genetic variance and previous values touched on it. The cytoplasmic genetic effect was significantly smaller in comparison to the number of 0.0049 founded in this study.

Table 5. Genetics parameters, correlations and variance proportions in preweaning traits

	Heritability		Genetic Correlation	Proportion of Total Variance		
Traits/Models	*a*	*m*	*am*	*c*	*k*	*e*
WB(kg)						
(a+m+c+k)	0.37 (0.040)	0.18 (0.028)	-0.02 (0.096)	0.0037 (0.012)	0.00000 (0.002)	0.45 (0.029)
(a+m+c)	0.37 (0.040)	0.19 (0.029)	-0.04 (0.095)	0.00004 2 (0.011)	-	0.45 (0.029)
(a+c+k)	0.43 (0.028)	-	-	0.11 (0.011)	0.00038 (0.002)	0.46 (0.026)
(a+m+k)	0.36 (0.039)	0.19 (0.022)	-0.03 (0.095)	-	0.000011 (0.002)	0.45 (0.029)
(a+m+c+k) *	0.37 (0.040)	0.18 (0.028)	-0.02 (0.096)	0.00084 (0.011)	0.0000016 (0.002)	0.46 (0.029)
WW(kg)						
(a+m+c+k)	0.23 (0.029)	0.10 (0.025)	-0.15 (0.125)	0.16 (0.019)	0.0022 (0.003)	0.53 (0.023)
(a+m+c)	0.22 (0.029)	0.10 (0.025)	-0.14 (0.125)	0.16 (0.019)	-	0.54 (0.023)
(a+c+k)	0.43 (0.028)	-	-	0.11 (0.011)	0.00038 (0.002)	0.46 (0.026)
(a+m+k)	0.19 (0.026)	0.32 (0.025)	-0.05 (0.090)	-	0.0023 (0.004)	0.49 (0.022)
(a+m+c+k) *	0.22 (0.027)	0.09 (0.023)	-0.07 (0.133)	0.16 (0.018)	0.00035 (0.002)	0.54 (0.022)
W205(kg)						
(a+m+c+k)	0.28 (0.033)	0.10 (0.026)	-0.10 (0.128)	0.19 (0.020)	0.0031 (0.004)	0.44 (0.024)
(a+m+c)	0.28 (0.033)	0.10 (0.026)	-0.09 (0.128)	0.19 (0.020)	-	0.44 (0.024)
(a+c+k)	0.28 (0.027)	-	-	0.26 (0.015)	0.0024 (0.003)	0.46 (0.023)
(a+m+k)	0.22 (0.028)	0.37 (0.024)	0.02 (0.085)	-	0.0037 (0.005)	0.40 (0.022)
*(a+m+c+k)**	0.28 (0.034)	0.10 (0.026)	-0.11 (0.126)	0.19 (0.020)	0.0016 (0.003)	0.45 (0.024)
ADG(kg)						
(a+m+c+k)	0.26 (0.034)	0.11 (0.026)	-0.20 (0.122)	0.18 (0.019)	0.0019 (0.003)	0.48 (0.025)
(a+m+c)	0.27 (0.035)	0.13 (0.029)	-0.25 (0.112)	0.17 (0.020)	-	0.47 (0.026)
(a+c+k)	0.25 (0.027)	-	-	0.24 (0.014)	0.0017 (0.002)	0.50 (0.023)
(a+m+k)	0.21 (0.030)	0.36 (0.025)	-0.08 (0.085)	-	0.0022 (0.004)	0.44 (0.023)
*(a+m+c+k)**	0.26 (0.034)	0.10 (0.025)	-0.21 (0.121)	0.18 (0.019)	0.00042 (0.002)	0.49 (0.025)
HL(cm)						
(a+m+c+k)	0.40 (0.063)	0.10 (0.040)	-0.24 (0.159)	0.032 (0.021)	0.0014 (0.004)	0.51 (0.046)
(a+m+c)	0.40 (0.063)	0.11 (0.040)	-0.24 (0.157)	0.032 (0.021)	-	0.51 (0.046)
(a+c+k)	0.39 (0.042)	-	-	0.077 (0.017)	0.0021 (0.004)	0.53 (0.039)
(a+m+k)	039 (0.062)	0.15 (0.035)	-0.22 (0.142)	-	0.0009 (0.004)	0.51 (0.046)
(a+m+c+k) *	0.41±.06 (0.063)	0.10±.04 (0.039)	-0.25±.16 (0.159)	0.035 (0.021)	0.00085 (0.004)	0.51 (0.046)
SC(cm)						
(a+m+c+k)	0.33 (0.068)	0.04 (0.046)	-0.17 (0.329)	0.12 (0.038)	0.0049 (0.006)	0.52 (0.055)
(a+m+c)	0.33 (0.068)	0.05 (0.047)	-0.16 (0.315)	0.12 (0.038)	-	0.52 (0.055)
(a+c+k)	0.33 (0.057)	-	-	0.14 (0.032)	0.0054 (0.006)	0.52 (0.053)
(a+m+k)	0.31 (0.067)	0.13 (0.053)	-0.08 (0.248)	-	0.0045 (0.007)	0.57 (0.055)
*(a+m+c+k)**	0.33 (0.067)	0.04 (0.043)	-0.10 (0.377)	0.12 (0.037)	0.0048 (0.006)	0.52 (0.055)

WB=birth weight; WW=weaning weight; W205=weight adjusted 205 days; ADG=average daily gain; HL=hock length; SC=scrotal circumference.

*(a+m+c+k)** = Complete model without number of generation from founder as a fixed effect.

Because variance proportions are easier to understand than absolute values, numbers from Table 5 will be used for the next analysis. Interpretation of the remaining components when a term was removed from the model was not trivial. The results were completely different for each trait. The easiest way to understand and identify patterns is to visualize the results graphically.

Regarding the WB trait, model (*a*+*c*+*k*) showed the largest additive effect. Figure 13 shows birth weight variance proportion for different models. The (*a*+*c*+*k*) model lacks the maternal effect and also the covariance for these two. Most of these two values were accounted for by the additive effect and the permanent environment which for this model also has the largest value. When all the models included maternal effect, it was detected in similar proportions but the largest were (*a*+*m*+*c*) and (*a*+*m*+*k*).

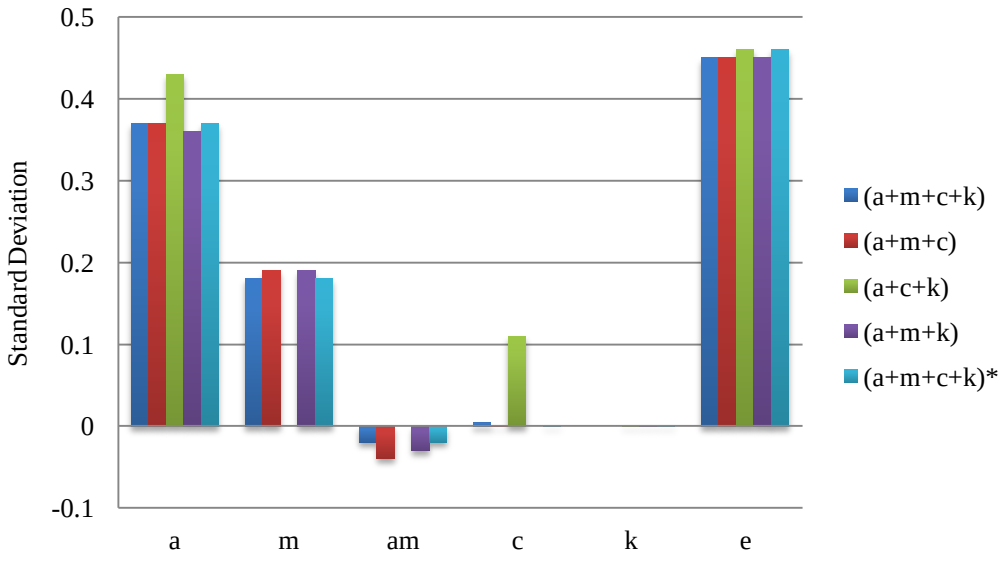

Figure 13. Birth weight variance proportions and correlation fitting different models
a=additive genetic; m=maternal genetic; am=additive-maternal correlation; c=permanent environment; k=cytoplasmic effect; e=error
*(a+m+c+k)**=Complete model without number of generation from founder as a fixed effect.

The additive-maternal correlation for (*a*+*m*+*c*) was larger in magnitude and also more negative than for (*a*+*m*+*k*). The lowest errors were for the complete model and for the model where cytoplasmic effect was removed. For WB it can be concluded that maternal effect accounted for an important proportion of the total variance and when it was removed that variance went to the additive and permanent environmental components.

The analysis for WW showed that when maternal effect was removed from the model the additive increased but the permanent environment decreased in comparison to the other models. This is different from what was observed in WB. When permanent environment was removed in (*a*+*m*+*k*); the maternal effect showed a large peak and the additive decreased a little compared to the rest of the models. The complete model (*a*+*m*+*c*+*k*), had the most negative additive-maternal correlation. Figure 14 summarizes WW variance proportions in different models.

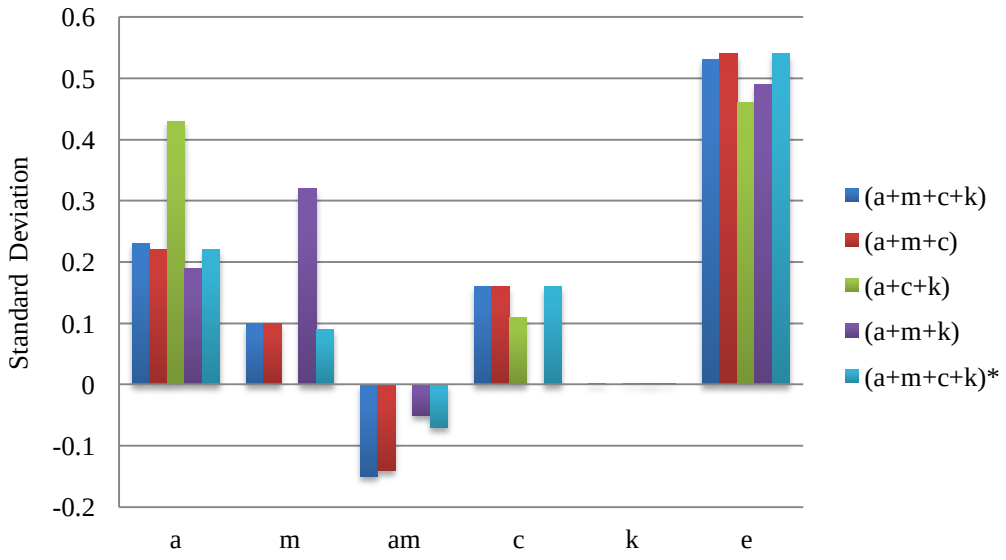

Figure 14. Weaning weight variance proportions and correlation fitting different models
a=additive genetic; m=maternal genetic; am=additive-maternal correlation; c=permanent environment; k=cytoplasmic effect; e=error
*(a+m+c+k)**=Complete model without number of generation from founder as a fixed effect.

From these observations it can be concluded that for WW maternal effect and permanent environment had important roles and should be considered for modeling; otherwise both of them may be absorbed by the additive effect, giving a biased result.

In the trait W205 the most notorious event was the peak observed in the maternal effect for the model (*a*+*m*+*k*), meaning that if permanent environment was removed maternal effect absorbed its portion of variation. Figure 15 describes variance proportion and correlation for W205.

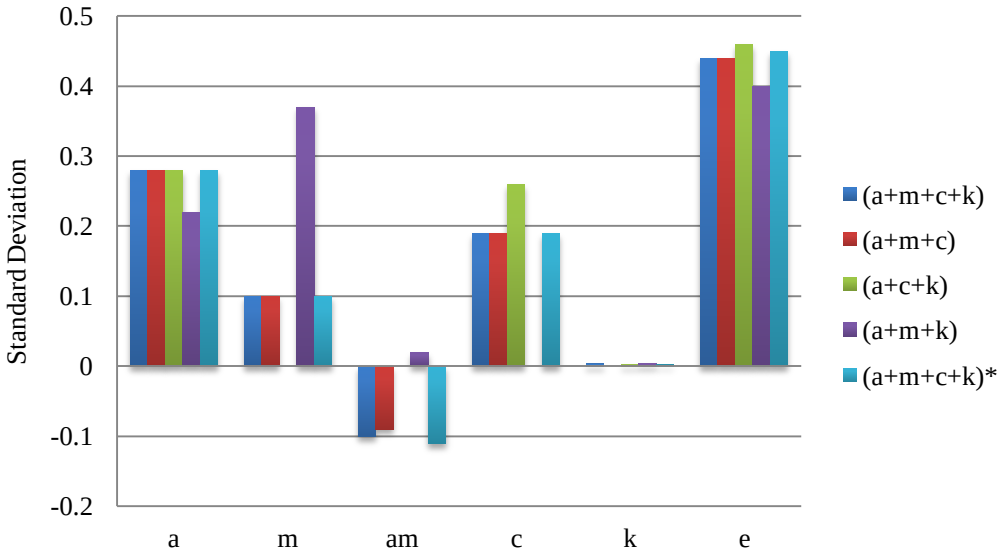

Figure 15. Weaning weigh adjusted to 205 days variance proportions and correlation fitting different models
a=additive genetic; m=maternal genetic; am=additive-maternal correlation; c=permanent environment; k=cytoplasmic effect; e=error
*(a+m+c+k)**=Complete model without number of generation from founder as a fixed effect.

In the (*a*+*c*+*k*) model an increase in the permanent environment could be noticed and also a bit of increase in the error. For W205 both complete models (*a*+*m*+*c*+*k*), (*a*+*m*+*c*+*k*)* and also (*a*+*m*+*c*) acted similarly.

In ADG the models acted like in the W205 trait with a notorious peak in maternal effect for the model lacking permanent environment effect, which can be

observed in Figure 16. The lack of cytoplasmic effect in the model (*a*+*m*+*c*) produced a more negative additive-maternal correlation. Comparing just the complete models, they resembled one another although with a bit of increase in the error for the model with no generations.

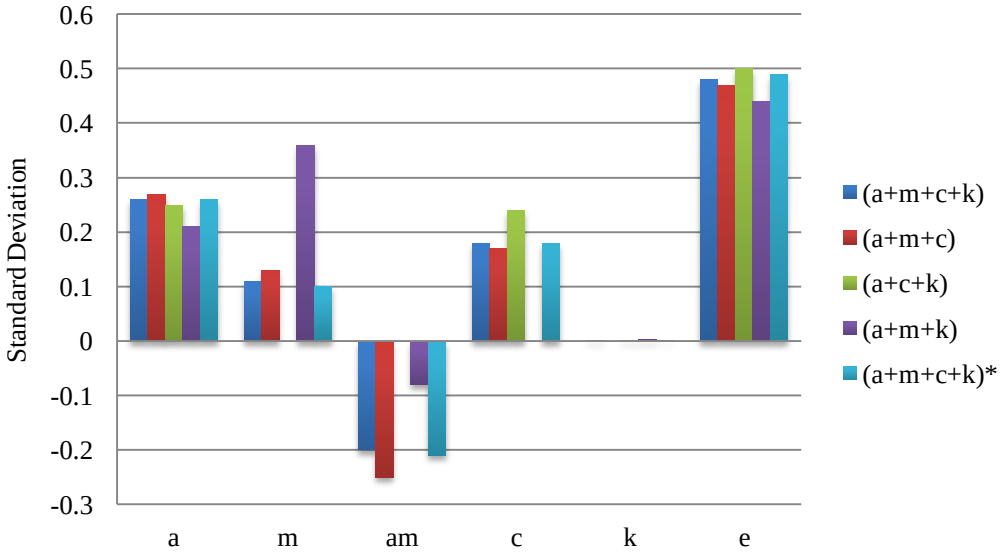

Figure 16. Average daily gain variance proportions and correlation fitting different models
a=additive genetic; m=maternal genetic; am=additive-maternal correlation; c=permanent environment; k=cytoplasmic effect; e=error
*(a+m+c+k)**=Complete model without number of generation from founder as a fixed effect.

For HL all the models produced similar results; (*a*+*m*+*k*) showed an increase in the maternal effect and (*a*+*c*+*k*) had a larger permanent environmental effect and error. The latter situation suggested that an effect removed from the model can end up being absorbed by any other of the remaining effects. Figure 17 demonstrates the previous situation and supports the inclusion of both effects when modeling for hock length. Also, it could be observed that (*a*+*m*+*c*+*k*), (*a*+*m*+*c*) and (*a*+*m*+*c*+*k*)* gave very similar proportion of variances.

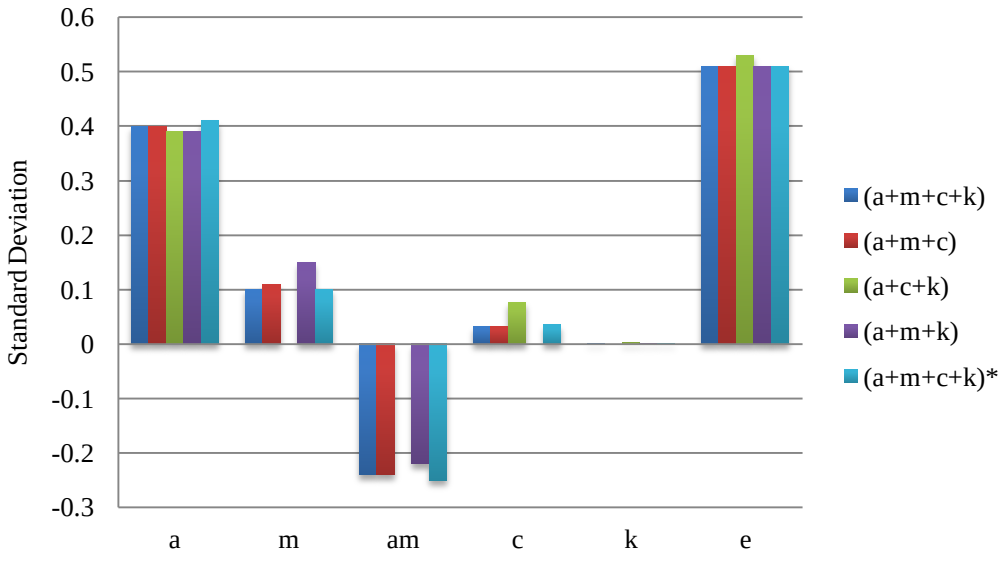

Figure 17. Hock length variance proportions and correlation fitting different models
a=additive genetic; m=maternal genetic; am=additive-maternal correlation; c=permanent environment; k=cytoplasmic effect; e=error
*(a+m+c+k)**=Complete model without number of generation from founder as a fixed effect.

Observing Figure 18 it can be seen that for SC all models showed a parallelism; only (*a+m+k*) manifested increases in maternal effect and error. This model proved the importance of the permanent environmental effect for SC. The complete model expressed that the larger additive-maternal negative correlation translated to a bit of decrease in the maternal effect.

If the consequence of taking out a term from the model was the same in all traits, a pattern should be observed. This never happened and it could be stated that how they act depends on the trait and should be analyzed independently. The biology behind each of these traits could explain some of the differences observed, but the fit of these models cannot be generalized for all analyzed traits and should be considered for each case individually.

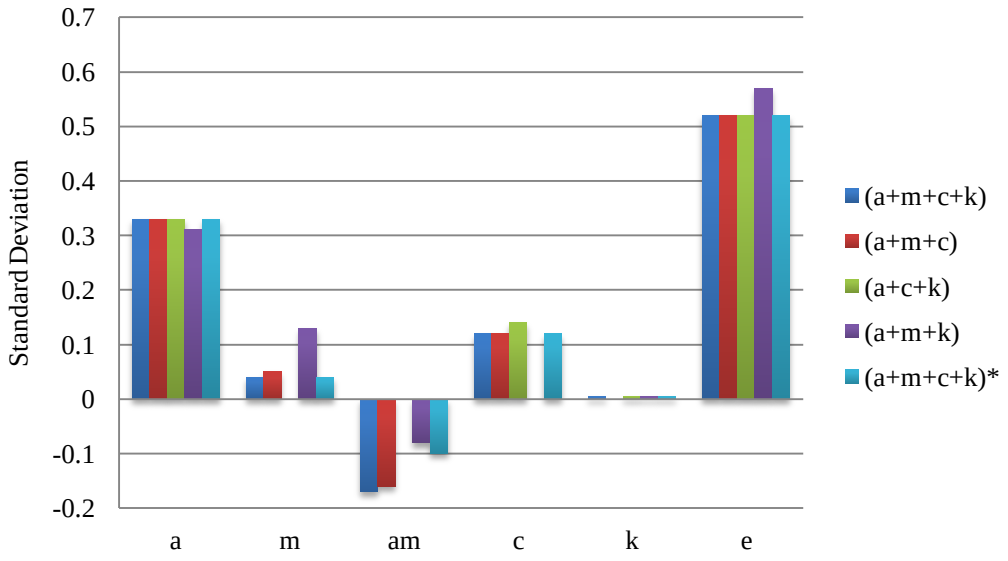

Figure 18. Scrotal circumference variance proportions fitting different models
a=additive genetic; m=maternal genetic; am=additive-maternal correlation; c=permanent environment; k=cytoplasmic effect; e=error
*(a+m+c+k)**=Complete model without number of generation from founder as a fixed effect.

Cytoplasmic breeding values

Seventeen females from the 18 original founders were considered for the calculation of cytoplasmic breeding values. Table 6 summarizes cytoplasmic breeding values for each founder and trait. Cow 19390094 is not shown in the founders breeding values table because of the lack of valid records. She had progeny represented in the herd but all of them with missing phenotypic information on the historical records. Cows 19370084, 19370086 and 19390090 did not have WB breeding values as a consequence of having no records for this trait.

Seventeen cows had cytoplasmic breeding values for WW, W205 and ADG. Founders 19370079, 19370084, 19370085, 19370086, 19390090 and 19390092 did not have progeny with HL and SC records, lacking breeding values for these traits.

Cells showing zeros are real values calculated as breeding values for the respective animal contrary to the dash that means a lack of valid records. In brief, seventeen founders had breeding values for WW, W205 and ADG; fourteen for WB and eleven for HL and SC.

Table 6. Cytoplasmic breeding values for preweaning traits in the founder females

	WB	WW	W205	ADG	HL	SC
19370061	0.00001	0.14649	0.08348	0.00049	0.02433	0.02084
19370077	-0.00002	-0.34449	-0.03831	-0.00163	-0.02997	-0.05752
19370079	-0.00001	-0.19798	-0.43775	-0.00046	-	-
19370080	-0.00002	-0.11547	-0.23383	0.00001	0.00000	-0.00989
19370081	-0.00001	0.02516	-0.03472	0.00042	-0.01215	-0.10760
19370082	0.00000	-1.09596	-1.45930	-0.00459	0.00583	-0.11572
19370083	0.00000	0.66324	0.84818	0.00290	0.00525	0.13672
19370084	-	-0.23834	-0.08880	-0.00092	-	-
19370085	0.00000	0.01783	0.04660	-0.00021	-	-
19370086	-	-0.07272	-0.08546	-0.00028	-	-
19390090	-	-0.01129	-0.06950	-0.00001	-	-
19390091	0.00003	0.89911	1.16493	0.00311	0.00472	0.06447
19390092	0.00000	0.17212	0.09321	0.00060	-	-
19390093	0.00000	0.80861	0.82822	0.00276	-0.00224	0.12782
19390095	0.00000	-0.22010	-0.31625	-0.00080	-0.00172	0.01011
19390096	-0.00002	-0.98623	-1.17916	-0.00361	-0.01704	-0.02769
19390097	0.00002	0.55003	0.87847	0.00223	0.02299	-0.04156

WB=birth weight; WW=weaning weight; W205=weight adjusted 205 days; ADG=average daily gain; HL=hock length; SC=scrotal circumference

Desired gain selection indexes

The difference between calculated selection indexes is presented in Table 7. The values are the coefficients that should be used in each case, including or excluding the cytoplasmic effect. These values should be interpreted carefully as proportions in the desired gain index. This means that if the first index is used the proportion is 1:1 for gains in additive and maternal genetic effects. Otherwise if the

second index is used the gain proportion is 1:1:20 for additive, maternal and cytoplasmic genetic effects.

Table 7. Index selection coefficients including and excluding cytoplasmic effect

Desired Gain	WB (kg)	WW(kg)	W205(kg)	ADG(kg)	SC(cm)	HL(cm)
Cytoplasmic effect included						
σ_a	0.3946	0.0961	0.0856	22.0556	0.9925	1.9004
σ_m	0.5662	0.1425	0.1414	34.3634	2.7144	3.7513
$20\sigma_k$	8716.8273	16.6220	14.5551	4102.2642	136.4744	489.7314
Cytoplasmic effect excluded						
σ_a	0.3996	0.0956	0.0845	22.7640	0.9892	1.9051
σ_m	0.5628	0.1413	0.1398	33.2391	2.5391	3.7297

WB=birth weight; WW=weaning weight; W205=weight adjusted 205 days; ADG=average daily gain; HL=hock length; SC=scrotal circumference
σ_a=additive standard deviation; σ_m=maternal standard deviation; σ_k=cytoplasmic standard deviation

Expected Genetic Gains

Values in Table 8 show the expected genetic gains for both indices. It can be noticed that for each trait the sum of the elements is 1. For practicality each cell number can be read as the effect contribution to the total genetic gain expressed as a proportion.

Table 8. Expected gains for preweaning traits using desired gain indexes

INDEX WITH CYTOPLASMIC EFFECT						
	WB	WW	W205	ADG	SC	HL
Additive	0.5832	0.2743	0.2681	0.2964	0.2635	0.3713
Maternal	0.4064	0.1850	0.1622	0.1902	0.0963	0.1881
Cytoplasmic	0.0103	0.5405	0.5696	0.5132	0.6400	0.4404
INDEX WITHOUT CYTOPLASMIC EFFECT						
	WB	WW	W205	ADG	SC	HL
Additive	0.5940	0.5980	0.6225	0.6318	0.7492	0.6667
Maternal	0.4059	0.4019	0.3774	0.3681	0.2507	0.3332

WB=birth weight; WW=weaning weight; W205=weight adjusted 205 days; ADG=average daily gain; HL=hock length; SC=scrotal circumference

Expected genetic gains for WB for both indices were similar. The tiny cytoplasmic effect on this trait could not provide nearly any gain. Considering the first index, the largest expected gains came from the cytoplasmic effect for WW, W205, ADG, SC and HL. The ranking of the expected gains were cytoplasmic, maternal and additive. The change observed in these traits in comparison to WB is due to a larger cytoplasmic effect accounted for in the MTDFREML analysis.

The largest cytoplasmic gain obtained was for SC. This could be explained by the 0.0049 of the total variation attributed to the cytoplasmic effect.

In the index without cytoplasmic effect WB, WW and W205 have gain ratios about 60:40 for additive and maternal. HL shows an intermediate gain ratio of 66:33. The corresponding ratio for SC was 75:25, having the larger additive expected value obtained with both indices.

Chapter 6: Conclusion and Implications

Maternal genetic effect accounted for 10% or more of the phenotypic variability in all traits in the complete model, except for scrotal circumference where it was responsible for only 4% of the total variation. In models with fewer elements, larger values were computed, reaching a peak of 37% in W205 for the model that omitted permanent environmental effect. These numbers demonstrate the importance of maternal effect in pre-weaning traits.

An efficient selection program should consider the maternal effect in the animal model for a better estimation of the causes of the observed phenotypic variation. It is crucial for a good selection program and for the prediction of expected genetic gains to be unbiased.

It was impossible to predict where the maternal effect is reallocated when it is excluded from the model. A pattern should be observed if the inclusion or not of terms in the model performs similarly for different traits, but such a pattern could not be distinguished. The biology of the trait and the correlation between additive and maternal effects could explain a portion of this behavior. Each trait should be analyzed individually because some are more influenced by the maternal effect than others. Traits that experience maternal effect through the modification of the environment by the mother's genes and are influenced by this effect for a long period of time normally express a larger negative additive-maternal correlation than should be considered for obtaining accurate breeding values.

Cytoplasmic effects accounted for less than 1% of the phenotypic variability in all traits. The largest cytoplasmic effect found was for SC with 0.49% and 0.54%

of the total variation for the complete model and for the one that excluded maternal effect, respectively.

For some traits it could be useful to consider cytoplasmic effects, especially if this information was available already without adding extra costs. The impact of the cytoplasmic effect inclusion could be observed in the computed expected gains.

Most of the traits experienced considerable changes when including cytoplasmic effect in the index with the exception of weigh at birth, where it had minuscule impact, resulting in almost no difference. The important change observed in the gains justifies the use of the cytoplasmic effect for at least one or two generations to get the maximum gain from it. Knowing that the selection intensity in the cytoplasmic effect was extremely high, after one or two generations it would be unlikely to obtain any more gain from the cytoplasmic effect due to the absence of variation. The only way to accrue cytoplasmic variation in this scenario is through mutation, and this occurs at very low rates comparing to the necessary mutation rate for maintaining variation in order to continue making genetic progress. In addition, individuals from the best lines will be selected as breeders, increasing the relatedness between the mated individuals, which will result in an increase of the inbreeding within the population.

Inbreeding depression could be dangerous and to prevent it, cytoplasmic effects could be employed in the index for a couple of generations; then its utilization should be stopped and breeders should continue with the additive-maternal index.

It was not possible to arrive to a generalized conclusion about the cytoplasmic genetic effect. Its importance depends on the trait in question and should be gauged independently.

A good knowledge of the trait biology, the results obtained in the present and other studies, and an acute judgment based on the production intensity could support the use of cytoplasmic effect in targeted situations in animal breeding programs.

Future studies combining molecular markers for cytoplasmic genes and phenotypic records should reveal more about the cytoplasmic inheritance mechanism and its potential use in animal breeding plans including genomic selection. Understanding better this process and the interrelation that it has with other inheritance mechanisms in determining a specific trait also would permit its application in other biological fields.

References

Albuquerque, L. G., J. F. Keown, and L. D. Van Vleck. 1998. Variances of direct genetic effects, maternal genetic effects, and cytoplasmic inheritance effects for milk yield, fat yield, and fat percentage. Journal of Dairy Science 81: 544-549.

Anderson, S., de Bruijn, M.H.L., Coulson, A.R., Eperon, I.C., Sanger, F., Young, I.G. 1982. Complete sequence of bovine mitochondrial DNA conserved features of the mammalian mitochondrial genome. Journal of Molecular Biology 156: 683-717.

Bell, B. R., B. T. McDaniel, and O. W. Robison. 1985. Effects of cytoplasmic inheritance on production traits of dairy cattle. Journal of Dairy Science 68: 2038-2051.

Bernardo, J. 1996. Maternal effects in Animal Ecology. American Zoologist 36: 83-105.

Bijma, P. 2006. Estimating maternal genetic effects in livestock. Journal of Animal Science 84: 800-806.

Boldman, K. G., L. A. Kriese, L. D. Van Vleck, C. P. Van Tassell, and S. D. Kachman. 1995. A manual for use of MTDFREML: A set of programs to obtain estimates of variances and covariances. ARS, USDA, Washington, DC.

Brinks, J. S., and T. S. Katsigianis. 1982. The genetic history and present structure of the Wye Angus University of Maryland herd. University of Maryland, College Park.

Brown, D. R., S. K. DeNise, and R. G. McDaniel. 1989a. Cytoplasmic genetic effects and growth of hybrid mice. Journal of Animal Science 67: 887-894.

Brown, D.R., Koehler, C.M., Lindberg, G.L., Freeman, A.E., Mayfield, J.E., Myers, A.M., Schutz, M.M., Beitz, D.C. 1989b. Molecular analysis of cytoplasmic genetic variation in Holstein cows. Journal of Animal Science 67: 1926-1932.

Brown, W. M., M. George, and A. C. Wilson. 1979. Rapid evolution of animal mitochondrial DNA. Proceedings of the National Academy of Sciences 76: 1967-1971.

Carrillo, J. A., and F. Siewerdt. 2010. Consequences of long-term inbreeding accumulation on preweaning traits in a closed nucleus Angus herd. Journal of Animal Science 88: 87-95.

Carrillo, J. A., J. Braccini Neto, E. C. Draper, and F. Siewerdt. 2011. Genetic parameters of preweaning traits in a closed Angus herd undergoing selection for maternal ability. Genomics and Quantitative Genetics (submitted).

Cheverud, J. M. 2003. Evolution in a genetically heritable social environment. Proceedings of the National Academy of Sciences of the United States of America 100: 4357-4359.

Cundiff, L. V. 1972. The role of maternal effects in animal breeding: VIII. Comparative aspects of maternal effects. Journal of Animal Science 35: 1335-1337.

Eisen, E. J. 1970. Maternal effects on litter size in mice. Canadian Journal of Genetics and Cytology.12: 209-216

Falconer, D. S., and T. F. C. Mackay. 1996. Introduction to quantitative genetics. 4th ed. Longman, Essex, England.

Garmyn, A. J., D. W. Moser, R. A. Christmas, and J. Minick Bormann. 2011. Estimation of genetic parameters and effects of cytoplasmic line on scrotal circumference and semen quality traits in Angus bulls. Journal of Animal Science 89: 693-698.

Henderson, C. R., and R. L. Quaas. 1976. Multiple trait evaluation using relatives' records. Journal of Animal Science 43: 1188-1197.

Huizinga, H. A., S. Korver, B. T. McDaniel, and R. D. Politiek. 1986. Maternal effects due to cytoplasmic inheritance in dairy cattle. Influence on milk production and reproduction traits. Livestock Production Science 15: 11-26.

Kennedy, B. W. 1986. A further look at evidence for cytoplasmic inheritance of production traits in dairy cattle. Journal of Dairy Science 69: 3100-3105.

Koch, R. M. 1972. The role of maternal effects in animal breeding: VI. Maternal effects in beef cattle. Journal of Animal Science 35: 1316-1323.

Legates, J. E. 1972. The role of maternal effects in animal breeding: IV. Maternal effects in laboratory species. Journal of Animal Science 35: 1294-1302.

Lingle, J. B., C. R. Koch, and S. M. Barao. 2001. The breed of noble bloods. University of Maryland Foundation, Adelphi.

Linnane, A., T. Ozawa, S. Marzuki, and M. Tanaka. 1989. Mitochondrial DNA mutations as an important contributor to ageing and degenerative diseases. The Lancet 333: 642-645.

Lopez, J. V., M. Culver, J. C. Stephens, W. E. Johnson, and S. J. O'Brien. 1997. Rates of nuclear and cytoplasmic mitochondrial DNA sequence divergence in mammals. Molecular Biology and Evolution 14: 277-286.

Meirelles, F.V., A.J.M. Rosa, R.B. Lôbo, J.M. Garcia, L.C. Smith and F.A.M. Duarte. 1999. Is the American Zebu really Bos Indicus? Genetics and Molecular Biology 22: 543-546.

Meyer, K. 1997. Estimates of genetic parameters for weaning weight of beef cattle accounting for direct-maternal environmental covariances. Livestock Production Science 52: 187-199.

Pesek, J., and R. J. Baker. 1969. Desired improvement in relation to selection indices No. 49. p 803-804. Canadian Journal of Plant Sciences.

Quaas, R. L., and E. J. Pollak. 1980. Mixed model methodology for farm and ranch beef cattle testing programs. Journal of Animal Science 51: 1277-1287.

Robinson, D. L. 1996. Estimation and interpretation of direct and maternal genetic parameters for weights of Australian Angus cattle. Livestock Production Science 45: 1-11.

Robison, O. W. 1972. The role of maternal effects in animal breeding: V. Maternal effects in swine. Journal of Animal Science. 35: 1303-1315.

Rohrer, G. A., J. F. Taylor, J. O. Sanders, and R. M. Thallman. 1994. Evaluation of line and breed of cytoplasm effects on performance of purebred Brangus cattle. Journal of Animal Science 72: 2798-2803.

Rutledge, J. J., O. W. Robison, E. J. Eisen, and J. E. Legates. 1972. Dynamics of genetic and maternal effects in mice. Journal of Animal Science 35: 911-918.

Stormont, C. 1972. The role of maternal effects in animal breeding: I. Passive immunity in newborn animals. Journal of Animal Science 35: 1275-1279.

Strachan, T. 1999. Human Molecular Genetics. 2nd ed. Bios Scientific, New York.

Sutovsky, P. et al. 2000. Ubiquitinated sperm mitochondria, selective proteolysis, and the regulation of mitochondrial inheritance in mammalian embryos. Biology of Reproduction 63: 582-590.

Tess, M. W., C. Reodecha, and O. W. Robison. 1987. Cytoplasmic genetic effects on preweaning growth and milk yield in Hereford cattle. Journal of Animal Science 65: 675-684.

Tess, M. W., and O. W. Robison. 1990. Evaluation of cytoplasmic genetic effects in beef cattle using an animal model. Journal of Animal Science 68: 1899-1909.

Van Vleck, L. D., K. J. Hanford, and G. D. Snowder. 2005. Lack of evidence for cytoplasmic effects for four traits of Polypay sheep. Journal of Animal Science 83: 552-556.

Van Vleck, L. D., G. D. Snowder, and K. J. Hanford. 2003. Models with cytoplasmic effects for birth, weaning, and fleece weights, and litter size at birth for a population of Targhee sheep. Journal of Animal Science 81: 61-67.

Vleck, L. D. V. 1976. Selection for direct, maternal and grandmaternal genetic components of economic traits. Biometrics 32: 173-181.

Wagner, R. P. 1972. The role of maternal effects in animal breeding: II. Mitochrondria and animal inheritance. Journal of Animal Science 35: 1280-1287.