Effect of disease on calf performance
by Tracie-Marie Margaret Bernardini

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

Abstract:
Health and performance records of beef cattle from inbred and outbred populations were evaluated to determine incidence and effects of calfhood disease on weaning (WWT) and final weights (FWT). Animals were maintained at the Northern Agricultural Research Center near Havre, Montana. Inbred animals were linebred Hereford cattle (Fx= 25). Outbred animals were Hereford, Angus x Hereford, Simmental x Hereford crosses and backcrosses, and Tarentaise x Hereford crosses and backcrosses. Incidences of scours (SCS) and respiratory illness (RI) were analyzed by CATMOD of SAS. The model for SCS included year, age of dam, and line (inbred or outbred). The model for RI was the same, except SCS was included. All main effects for the SCS and RI models were important (P<.01), except SCS (P< .14) in the RI model. WWT and FWT were analyzed by GLM of SAS. The model for WWT included year, age of dam, line, and date of birth (DOB) as a covariate. The model for FWT was the same, except sex of calf was. included. All main effects for the WWT and FWT models were important (P<.01). Interactions of year x age of dam (P<.01) and year x line (P<.01) were detected for the WWT and FWT models. In the FWT model additional interactions of year x RI, line x age of dam, and age of dam x sex of calf were detected (P<.01). Mean incidences of SCS and RI over all years were 35 and 22%, respectively. Incidences of SCS and RI were greatest in inbred calves bom to 2-yr-old dams at 47 and 37%, respectively. For calves contracting SCS and RI, there was a 9 or 12 kg difference in WWT and FWT, respectively. Non-significant line x SCS (P<.98) and line x RI (P<.84) interactions revealed effects of calfhood disease were not different for inbred versus outbred cattle. Economic loss per scouring animal over all years averaged $34.84, and $39.80 for animals contracting RI. These results indicate incidence of calfhood disease was highest in calves bom to young, inbred dams, and negatively impacted calf WWT and FWT. Key words: Beef Cattle, Scours, Respiratory Illness, Weaning Weight, and Final Weight.

EFFECT OF DISEASE ON CALF PERFORMANCE

by

Tracie-Marie Margaret Bernardini

A thesis submitted in partial fulfillment of the requirements for the degree

of

Master of Science

in

Animal Science

MONTANA STATE UNIVERSITY
Bozeman, Montana

December 1996

APPROVAL

of a thesis submitted by

Tracie-Marie Margaret Bernardini

This thesis has been read by each member of the graduate 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.

Dec 5, 1996
Date

W D Kress
Chairperson, Graduate Committee

Approved for the Major Department

Nov 26, 1996
Date

M. W. Lee
Head, Major Department

Approved for the College of Graduate Studies

12/20/96
Date

R L Brown
Graduate Dean

STATEMENT OF PERMISSION TO USE

In presenting this thesis in partial fulfillment of the requirements for a master's degree at Montana State University, I agree that the Library shall make it available to borrowers under rules of the Library.

If I have indicated my intention to copyright this thesis by including a copyright notice page, copying is allowable only for scholarly purposes, consistent with "fair use" as prescribed in the U.S. Copyright Law. Requests for permission for extended quotation from or reproduction of this thesis in whole or in parts may be granted only by the copyright holder.

Signature Janice-Marie Margaret Bernardini

Date November 26, 1996

ACKNOWLEDGEMENTS

I am thankful for the faculty and staff in the Department of Animal and Range Sciences who gave so that I may succeed. I would especially like to recognize:

Dr. Don Kress for teaching me patience and perseverance;

Dr. Jan Bowman for showing me how elegantly grace and hard work blend;

Dr. Mark Jutila for his insight and kindness;

K.C. Davis, Don Anderson, and Cheryl Horning for keeping my rowboat afloat;

Mary Huenergardt, Linda McDonald, Robin Rieger, and Peggy Kelly for laughter;

Lisa and Shane Surber, and Tim Milner for friendship rarely found in this life;

Rob McCray, second only to my brother;

Cheryl Ross for her thoughtfulness and love of life;

Dr. Verl Thomas for leaving the door open and a hand on my shoulder;

Dr. Rodney Kott for keeping the door open;

Chris Thomas for showing me what true strength and courage are;

Irene Decker for her deep, abiding faith;

John, Christa, and Zoe Kozlowski, my family, for unconditional love;

Gloria and Douglas Roark, my parents, for being the wind beneath my wings;

Brent Roeder, brave heart, for the beauty of shared flight.

TABLE OF CONTENTS

APPROVAL ii

STATEMENT OF PERMISSION TO USE iii

ACKNOWLEDGMENTS iv

LIST OF TABLES vii

LIST OF FIGURES viii

ABSTRACT xi

CHAPTER 1

INTRODUCTION 1

CHAPTER 2

LITERATURE REVIEW 3
Calfhood Disease 3
Treatment 3
Economic Cost 3
Evaluation of Immunity 4
Immune Responsiveness 5
Major Histocompatability Complex 7
Calf Immunity 9
Colostral Immunoglobulin 10
Failure of Passive Transfer 11
Inherited Disease Resistance 13
Heritability 13
Heterosis 14
Production Traits 14

CHAPTER 3

MATERIALS AND METHODS 18
Cattle 18
Scours 19
Respiratory Illness 20

TABLE OF CONTENTS (Cont.)

Statistical Analysis 20
Incidence of Calf Illness 20
Weaning Weight 21
Final Weight 21

CHAPTER 4

RESULTS AND DISCUSSION 23
Calf Morbidity 23
Incidence of Scours 23
Incidence of Respiratory Illness 24
Calf Performance Traits 26
Weaning Weight 26
Final Weight 28
Economic Loss 30
Weaning Weight 30
Final Weight 30
General Discussion 31

CHAPTER 5

SUMMARY 34

LITERATURE CITED 39

APPENDIX 47

LIST OF TABLES

Table | Page

1. Categorical analysis of variance for effect of year, age of dam, and line on incidence of scours 35

2. Categorical analysis of variance for effect of year, age of dam, line, sex of calf, and scours on incidence of respiratory illness 35

3. Analysis of variance for effect of year, age of dam, line, and scours on calf weaning weight including zero weaning weights for calves dying of scours 36

4. Analysis of variance for effect of year, age of dam, line, and scours on calf weaning weight without zero weaning weights for calves dying of scours 36

5. Analysis of variance for effect of year, age of dam, line, sex of calf, and respiratory illness on calf final weight including zero final weights for calves dying of respiratory illness 37

6. Analysis of variance for effect of year, age of dam, line, sex of calf, and respiratory illness on calf final weight without zero final weights for calves dying of respiratory illness 38

LIST OF FIGURES

Figure		Page
1.	Incidence of calf scours over 14 years at the Northern Agricultural Research Center	48
2.	Incidence of calf scours in inbred and outbred cattle over 14 years at the Northern Agricultural Research Center	49
3.	Effect of age group of dam on incidence of calf scours at the Northern Agricultural Research Center	50
4.	Effect of age of dam and line on incidence of calf scours at the Northern Agricultural Research Center	51
5.	Incidence of respiratory illness over 12 years at the Northern Agricultural Research Center	52
6.	Incidence of calf respiratory illness post-weaning in inbred and outbred cattle at the Northern Agricultural Research Center	53
7.	Incidence of respiratory illness in weaned calves by age group of dam at the Northern Agricultural Research Center	54
8.	Effect of age of dam and line on incidence of respiratory illness in weaned calves at the Northern Agricultural Research Center	55
9.	Effect of sex of calf and line on incidence of respiratory illness in weaned calves at the Northern Agricultural Research Center	56
10.	Least squares mean weaning weights of calves over 14 years at the Northern Agricultural Research Center	57
11.	Effect of age of dam on least squares mean weaning weights of calves at the Northern Agricultural Research Center	58
12.	Effect of age of dam on least squares mean weaning weights of calves over 14 years at the Northern Agricultural Research Center	59

LIST OF FIGURES (Cont.)

Figure

Page

13. Effect of scours on least squares mean weaning weights of calves for age group of dam at the Northern Agricultural Research Center 60

14. Effect of line on least squares mean weaning weights of calves over 14 years at the Northern Agricultural Research Center 61

15. Effect of scours on least squares mean weaning weights of calves for inbred versus outbred cattle at the Northern Agricultural Research Center 62

16. Least squares mean difference in weaning weights of scouring versus non-scouring calves over 14 years at the Northern Agricultural Research Center . . . 63

17. Least squares mean final weights of calves over 12 years at the Northern Agricultural Research Center 64

18. Effect of age of dam on least squares mean final weights of calves at the Northern Agricultural Research Center 65

19. Effect of age of dam on least squares mean final weights of calves over 12 years at the Northern Agricultural Research Center 66

20. Effect of age of dam on least squares mean final weights of calves for inbred versus outbred cattle at the Northern Agricultural Research Center 67

21. Effect of line on least squares mean final weights of calves over 12 years at the Northern Agricultural Research Center 68

22. Effect of sex on least squares mean final weights of calves for each age group of dam at the Northern Agricultural Research Center 69

23. Effect of respiratory illness on least squares mean final weights of calves over 12 years at the Northern Agricultural Research Center 70

24. Least squares mean difference in final weight of calves contracting respiratory illness and calves free of respiratory illness over 12 years at the Northern Agricultural Research Center 71

LIST OF FIGURES (Cont.)

Figure Page

25. Estimated loss in income due to calf scours over 14 years at the Northern Agricultural Research Center 72

26. Estimated loss in income per scouring animal over 14 years at the Northern Agricultural Research Center 73

27. Estimated loss in income due to respiratory illness over 12 years at the Northern Agricultural Research Center 74

28. Estimated loss in income per calf contracting respiratory illness over 12 years at the Northern Agricultural Research Center 75

ABSTRACT

Health and performance records of beef cattle from inbred and outbred populations were evaluated to determine incidence and effects of calfhood disease on weaning (WWT) and final weights (FWT). Animals were maintained at the Northern Agricultural Research Center near Havre, Montana. Inbred animals were linebred Hereford cattle (F_x=.25). Outbred animals were Hereford, Angus x Hereford, Simmental x Hereford crosses and backcrosses, and Tarentaise x Hereford crosses and backcrosses. Incidences of scours (SCS) and respiratory illness (RI) were analyzed by CATMOD of SAS. The model for SCS included year, age of dam, and line (inbred or outbred). The model for RI was the same, except SCS was included. All main effects for the SCS and RI models were important (P<.01), except SCS (P<.14) in the RI model. WWT and FWT were analyzed by GLM of SAS. The model for WWT included year, age of dam, line, and date of birth (DOB) as a covariate. The model for FWT was the same, except sex of calf was included. All main effects for the WWT and FWT models were important (P<.01). Interactions of year x age of dam (P<.01) and year x line (P<.01) were detected for the WWT and FWT models. In the FWT model additional interactions of year x RI, line x age of dam, and age of dam x sex of calf were detected (P<.01). Mean incidences of SCS and RI over all years were 35 and 22%, respectively. Incidences of SCS and RI were greatest in inbred calves born to 2-yr-old dams at 47 and 37%, respectively. For calves contracting SCS and RI, there was a 9 or 12 kg difference in WWT and FWT, respectively. Non-significant line x SCS (P<.98) and line x RI (P<.84) interactions revealed effects of calfhood disease were not different for inbred versus outbred cattle. Economic loss per scouring animal over all years averaged $34.84, and $39.80 for animals contracting RI. These results indicate incidence of calfhood disease was highest in calves born to young, inbred dams, and negatively impacted calf WWT and FWT. Key words: Beef Cattle, Scours, Respiratory Illness, Weaning Weight, and Final Weight.

CHAPTER 1

INTRODUCTION

More than any other time in history consumers are concerned with the quality of their food and the environment in which it is produced. Cattlemen face tremendous social and economic pressures to produce a high quality product at low consumer cost.

To meet consumer demand scientists, veterinarians, and stockmen have increased the nutritional, reproductive, and genetic performance of cattle. These advances, however, have not come without economic cost. To offset the expense of an increasingly systematic and technical industry, stockmen depend on the survival of every calf from birth through the feedlot.

While we can estimate rate of gain, efficiency of feed conversion, and heritability of carcass traits, we have few tools to estimate which calves will be susceptible to and die of calfhood disease. Management techniques, environmental circumstances, and genetic predisposition all affect calf survivability (Odde, 1988). For these reasons survivability is perhaps the most complex trait in the livestock industry (Green, 1993). Additionally, research indicates selecting for increased carcass quality and milk production has antagonistic effects on disease susceptibility and immune responsiveness of animals (Gross and Siegel, 1988; Uribe et al., 1995).

National and regional morbidity and mortality data for calfhood disease is available (USDA:APHIS:VS, 1994). Information is also obtainable on the economic costs of calfhood disease (Wittum et al., 1993). However, little data is available evaluating the impact of calfhood disease on performance traits or the effects of inbreeding on susceptibility to disease.

The primary objective of this study was to determine the incidence and effects of calfhood disease on calf performance. A secondary objective was to determine if the incidence and effects of calfhood disease were different for inbred versus outbred cattle.

CHAPTER 2

LITERATURE REVIEW

Calfhood Disease

Treatment

Part V of the Beef Cow/Calf Health & Productivity Audit (USDA:APHIS:VS, 1994) reported 31% of beef cattle producers, either agreed or strongly agreed, calf scours had a significant economic impact on their cow/calf operation within the previous 12 months. In the same study, 23% of beef cattle producers, either agreed or strongly agreed, calf respiratory illness had an economic impact as well. Of all Sandhills, Nebraska ranches, 68% treat cattle for scours, and 51% treat cattle for respiratory illness each year using procedures other than vaccinations (Coady and Clark, 1993).

Economic Cost

Mean cost of death associated with scours estimated by Wittum et al. (1993) was $215 per case with greater than $10 attributed to treatment beyond the value of the calf. The study also estimated mean cost of death associated with respiratory illness at $263 per calf with greater than $12 attributed to treatment beyond the value of the calf. The higher cost per calf death in relation to respiratory illness resulted from calves being older at the

time of death. Wittum and Perino (1995) reported morbidity during the first 28 d of life was associated with a 16 kg decrease in weaning weight, while respiratory morbidity in the feedlot resulted in a 0.04 kg decrease in average daily gain.

Evaluation of Immunity

Attempts by producers and veterinarians to medicate newborn livestock for calfhood diseases are effective when the animal survives. However, economic loss in terms of labor, medication, and depressed calf growth may never be offset prior to weaning and sale (Wittum and Perino, 1995). While drug treatments are necessary for humane and proper veterinary care, they cover up natural differences in immunity, and therefore work against identification of disease resistance (Green et al., 1993).

Warner et al. (1987) defined disease as environmental insult meeting genetic predisposition. The evaluation of genetic predisposition, however, is the difficult factor. Researchers have divided their attention between two different evaluation methods. The first method is to evaluate an animal's immune system by its responsiveness to foreign antigens. The second method is to correlate the presence of alleles from the highly polymorphic major histocompatability complex (MHC) with disease resistance. The following sections will address each of these methods.

Immune Responsiveness

The belief behind evaluation of an animal's immune responsiveness to foreign antigens is that the type and amount of immunity produced is an indication of the animal's ability to fight disease. The type of immunity raised to counter foreign antigens is divided into two classes; humoral (B lymphocytes) and cell-mediated (T lymphocytes), both of which are acquired (Glick et al., 1956; Claman et al., 1966). While an animal has innate immunity (i.e., skin, low gastric pH, lysozyme, polymorphonuclear leukocytes, and natural killer cells), these responses are non-specific in defending against foreign antigens. Acquired immunity, in contrast, has specificity and memory when combating a particular foreign antigen, and self/non-self recognition to prevent an autoimmune response.

The traditional approach to the evaluation of immune responsiveness is through humoral immunity which characterizes antibodies as effectors (Kuby, 1992). Antibodies are synthesized by B lymphocytes, and known collectively as immunoglobulins (Ig). There are five classes of immunoglobulin; IgM, IgD, IgG, IgA, and IgE. In the bovine all classes of immunoglobulin are present. A unique feature of bovine IgG is that it has two subclasses (i.e., IgG_1 and IgG_2). This is in contrast to human IgG which has four subclasses (i.e., IgG_1, IgG_2, IgG_3, and IgG_4). Each class is distinguished by a pair of heavy and light polypeptide chains which form the antigen binding site. The structure of the heavy chain denotes the class of immunoglobulin (ie. IgG molecules have γ chains).

Each B lymphocyte makes antibody molecules with a unique antigen binding site. Because of variable regions in the heavy and light chains of immunoglobulin it is theoretically possible to have 10^6 unique antigen binding sites. In virgin B lymphocytes

antibody is presented on the surface of a cell membrane (Vitetta et al., 1984). Antigen binding and helper T lymphocytes activate the B lymphocyte to multiply and mature into memory cells or antibody-secreting cells. Free antibody is secreted by mature antibody-secreting cells at the site of B lymphocyte activation, usually lyphoid organs (ie. lymph nodes, spleen, appendix, tonsils, adenoids, and Peyer's patches), and rapidly disperses throughout the body to combat foreign antigens. It is the level of antibody in the blood which has been the primary method of evaluating immune responsiveness in animals. Levels of certain leukocytes (i.e., lymphocytes, monocytes, neutrophils, basophils, and eosinophils) are often evaluated as well.

Using immune responsiveness as the sole measurement to evaluate an animal's ability to fight disease has limitations. Biozzi et al. (1979) showed, given a normal distribution of mice, animals at the apex of the curve would have the greatest level of general disease resistance because humoral and cell-mediated responses to disease occur with equal frequency. Therefore, breeding for extreme degrees of humoral or cell-mediated resistance creates animals highly susceptible to classes of infectious agents associated with the form of immunity not selected.

Kehrli et al. (1992) in a review article of β_2 Integrin Deficiency, an inherited disease in Holstein calves, noted neutrophil lysates lacked a β subunit of the β_2 integrins causing a failure of normal leukocyte-endothelial cell adherence and emigration. The concern β_2 Integrin Deficiency raises is that presence of neutrophils does not necessarily correlate positively with proper function. Therefore, using the presence or absence of antibodies, lymphocytes, monocytes, neutrophils, basophils, and (or) eosinophils as sole

indicators of immunity without determining functional competence of the cells may yield incorrect conclusions and mask genetic abnormalities relating to proper cellular function.

Major Histocompatability Complex

The second evaluation method correlates the presence of alleles from the highly polymorphic major histocompatability complex (MHC) with disease resistance. The MHC is a set of genes coding for the predominant surface proteins on the cells and tissues of each individual animal species (Snell et al., 1976; Goetze et al., 1977). In cattle the cell surface proteins are known as bovine leukocyte antigens (BOLA), and represent the means of self/non-self recognition (Amorena and Stone, 1978; Spooner et al., 1978)..

The MHC is intimately associated with acquired, cell-mediated immunity (CMI). In contrast to humoral immunity, cell-mediated immunity works locally instead of over long distances. The effector functions of CMI are helper T cell (Th) and cytotoxic T cell (Tc) activity. Helper T cell effector function is through a subclass of T cells expressing cell surface glycoproteins with the cluster designation marker (CD) of CD4. Cytotoxic T cells express cell surface glycoprotein CD8. Helper T cells activate B lymphocytes, while Tc cells are antigen-specific effector cells which bind to and kill their targets specifically and directly.

Unlike B lymphocytes which can recognize foreign antigen, T lymphocytes can only recognize foreign antigen in the context of the MHC (Kronenberg et al., 1986). There are three classes of MHC molecules; I and II, and III. Class I MHC molecules are expressed on the surface of all nucleated cells, Class II molecules are expressed on the

surface of cells involved with immune responsiveness, and Class III molecules are complement components. Each class of MHC molecules bind specifically to a T lymphocyte class. The MHC molecules present antigens on the surface of either antigen presenting cells or target cells. Th cells recognize foreign antigen on the surface of antigen presenting cells and B lymphocytes in association with MHC II molecules. Tc cells recognize foreign antigen on the surface of target cells in association with MHC I molecules. The binding of the T lymphocyte to foreign antigen in association with the appropriate MHC molecules initiates cell-mediated immunity.

The bovine immune system, however, has some unique features relating to cell-mediated immunity. The T lymphocytes of cattle are classified as γδ lymphocytes unlike their human counterparts which are αβ. The designation relates to the form of T-cell receptors expressed on the lymphocyte. Bovine γδ lymphocytes, which comprise 95% of the bovine T lymphocyte population, do not always require the MHC to recognize foreign antigen. The function of bovine γδ lymphocytes is not entirely understood, and is the focus of intense research.

The MHC is highly polymorphic, meaning the genotype of the MHC is extremely variable. Determination of the MHC genotype is called a haplotype. Correlating an animal's MHC haplotype with disease resistance may in time provide information which allows for identification of bovine haplotypes most resistance to disease. Stockmen may then be able to breed animals for optimal immune system performance much as they do carcass characteristics.

Calf Immunity

Syndesmochorial placentation in the ruminant animal prevents transplacental transfer of antibodies during gestation (Brambell, 1970). Because of this type of placentation, the humoral response of ruminants is immature at birth. Offspring of ruminants are born agammaglobulinemic (Campbell, 1977). Newborns are, therefore, dependent on the colostrum of dams to provide passive transfer of immunoglobulins (Brambell, 1970). The cell-mediated component of the ruminant neonatal immune system, however, is developed enough to provide a good immune response in comparison to other species. The myeloid lineage is intact, and T lymphocytes are functional. Also, innate immunity exhibited by the neonatal ruminant is quite heterogeneous.

Consumption of dam colostrum by newborn ruminants confers near adult levels of immunoglobulin immunity (Newby and Bourne, 1977). Colostrum protects young from septicemia, and respiratory and enteric infections. However, to be effective, colostral consumption must occur within the first 24 to 48 h of life prior to closure (Besser, 1994). Closure is a natural process which marks the end of non-specific macromolecular transport of immunoglobulin across the epithelium of the small intestine in the calf. To confer protective immunity, enough colostrum must be consumed by the calf to allow calf serum concentrations to reach $\geq$ 10 mg/ml.

Colostral Immunoglobulin

Approximately 80% of bovine colostral immunoglobulin is subclass IgG, specifically IgG_1, although all subclasses of immunoglobulin are present in colostrum. This is in contrast to other species where the principle colostral immunoglobulin is IgA. Initially, the immunoglobulin in mammary secretion was believed to be of two origins, either serum derived or produced locally by lymphoid cells found near the glandular epithelium. Applied research resolved that the majority of immunoglobulin in colostrum is serum derived. Smith et al. (1946) determined colostral immunoglobulin was similar to serum γ-globulin with respect to size, electrophoretic mobility, and precipitation characteristics. Larson and Kendall (1957) reported a decrease in serum γ-globulin levels as cows near calving. Dixon (1961) also reported a decrease in serum γ-globulin corresponding with a proportional accumulation in colostrum near calving.

The transfer of immunoglobulin, specifically IgG_1, from dam serum into the mammary gland is selective (Brandon and Lascelles, 1973). However, IgG_1, is the only immunoglobulin selectively transferred from serum to colostrum. The transfer of IgG_1 is active, selective, and receptor mediated. IgG_1 diffuses from the blood across the vascular epithelium and is bound by specific IgG_1-Fc receptors on the basal membrane of the mammary secretory epithelium. IgG_1 is then taken up by micropinocytotic vesicles transversing the epithelial cells which secrete the IgG_1 into colostrum. The number of IgG_1-Fc receptors increases dramatically during colostrum formation, accounting for the selective transfer of IgG_1 (Sasaki et al., 1977).

Colostral immunoglobulin concentrations are 5 to 10 times greater than serum concentrations. IgG concentrations in colostrum, however, are extremely variable. The average concentration is 100 mg/ml of colostrum (Halliday, 1978). Long gestations are correlated with increased immunoglobulin concentrations in the mammary gland prior to birth, with decreased colostral IgG concentrations associated with premature births. Production of colostrum has been reported to be proportional to number of offspring per litter. Halliday, (1978) indicated Finnish ewes have higher antibody titers depending on litter size prior to parturition, than other British breeds. Gilbert et al. (1988) determined litter size in six breeds of sheep (i.e., Polypay, Rambouillet, Targhee, Columbia, Finn and Finn cross) affected ($P<.05$) colostral concentrations of IgG_1. However, increased colostral IgG_1 concentrations in ewes having litters did not ensure increases in lamb serum concentrations. Triplet lambs in this study had significantly less serum IgG_1 concentrations at 36 hours old than single lambs. In contrast, Perino et al. (1995) indicated higher ($P<.01$) serum IgG concentrations in twin calves 24 hours old.

Failure of Passive Transfer

As early as 1922, Smith and Little realized the significance of colostrum to newborn calves. Boyd (1972) in a farm survey noted the association of serum immunoglobulin deficiency and disease in calves. Increased susceptibility to disease and death loss has been associated with low lamb and calf serum immunoglobulin concentrations or failure of passive transfer of maternal immunity at birth from dams to lambs and calves (McGuire et al., 1976, 1983; Muggli et al., 1984; Gilbert et al., 1988a).

McDonough et al. (1994) reported decreased serum IgG concentrations (P<.001) in intensively managed veal calves contracting and(or) dying of diarrhea. Wittum et al. (1995) reported decreased IgG and plasma protein concentrations in calves 24 h old resulted in increased risk of preweaning mortality, neonatal morbidity, and preweaning morbidity. Calves having decreased plasma protein concentrations at 24 h old had an increased risk of feedlot morbidity and feedlot respiratory tract morbidity.

Historically, failure of passive transfer has been defined as calf serum concentrations <800 mg of IgG/dl by Perino et al. (1995) and < 900 mg of IgG/dl by McGuire et al. (1976). Failure of passive transfer of maternal immunity is often due to management factors, dystocia, and age of dam at parturition (Odde, 1988; Wittum et al., 1994). Age of dam is a limiting factor in that mammary and immune system development is not as great in younger dams (2-yr-olds) as in mature dams (Frerking and Aeikens, 1978). Norman et al. (1981) found age of dam affected prepartum cow serum and colostral concentrations of IgG_1 with younger dams having the lowest concentrations. Bradley et al. (1979) reported decreased serum concentractions of IgG_1 (18.6 mg/ml) in calves born to primiparous heifers versus calves (25.7 mg/ml) born to multiparous cows. This is supported by the work of Perino et al. (1995) indicating age of dam was associated with decreased plasma protein and IgG concentrations in calves 10 h after birth. However, calf serum IgG_1 concentrations increase with increasing age of dam (Mueller and Elleinger, 1981; Muggli et al., 1984).

Inherited Disease Resistance

Heritability

Within breed heritability of survival from birth to weaning is low. As a trait of the dam heritability is .08, and .04 as a trait of the calf (Cundiff, 1984). Researchers have attempted to determine heritabilities associated with passive immune transfer of maternal immunity to offspring. Halliday (1981) reported low heritabilities for maternal passive transfer of immunoglobulins. These findings were supported by the work of Muggli et al. (1984) who related moderate heritability estimates (.27±.17) for serum IgG_1 concentration in Hereford calves 36 h old selected for weaning weight, yearling weight, and muscling score. Gilbert et al. (1988a) reported low heritabilities for lamb serum IgG_1 concentrations at 36 h old when treated as both a trait of the lamb (.18) and ewe (.16).

These studies are in contrast to Norman et al. (1981) and Gilbert et al. (1988b) who reported relatively high heritabilities of .69±.30 and .56±.25, respectively, for calf serum IgG_1 concentrations. Cattle in the Norman study were Hereford and Angus x Hereford crosses, while cattle in the Gilbert study were linebred Hereford cattle (F_x = 0.0 to .68), Angus, Red Angus, and Simmental. Gilbert and coworkers accounted for differences in the heritability estimates because of a higher degree of variation between sires in their study versus sires in the Muggli et al. (1984) study.

Heterosis

Effects of individual heterosis for survival are large with a 3.4% increase in survival of weaned F_1 calves and an additional 1.3% increase in survival of calves raised by F_1 dams due to maternal heterosis (Long, 1980). Significant breed differences in pre-partum cow serum, colostral, and calf serum immunoglobulins have been reported (Halliday et al., 1978; Bradley et al., 1979; Norman et al., 1981). Muggli et al. (1984) noted breed differences ($P<.01$) in calf serum immunoglobluin concentrations with Angus calves highest (39.2 mg/ml), Red Poll calves intermediate (35.0), and Hereford calves least (30.0 mg/ml). Gilbert et al. (1988a) reported similar results in lamb serum concentrations of IgG_1 between different breeds of sheep with sire within breed being an important source of variation ($P<.01$). Norman et al. (1981) reported higher IgG_1 serum concentrations in Angus x Hereford calves, than straightbred Hereford calves.

Makerechian et al. (1980) reported a decreased incidence of scours in crossbred calves versus Hereford, beef synthetic, and dairy synthetic calves. Muggli et al. (1992) noted a significant ($P<.05$) difference between straightbred Hereford and crossbred cattle in incidence of respiratory illness in post-weaned calves, with crossbred calves having decreased levels of respiratory illness.

Production Traits

Within the last 100 yr, several co-operative producer organizations (ie. the Standardized Performance Assurance Program for beef cattle, Dairy Herd

Improvement Association, and National Sheep Improvement Program) have been formed for the express purpose of increasing the efficiency of production. The goal of these groups is to maximize kilograms of animal weaned or kilograms of milk produced given a particular set of environmental constraints. During the Great Depression and World War II eras, pressure was put on farmers to harvest enough food to support the people of this country. This pressure still exists, but has been magnified to a global level, and is coupled with economic viability.

Since 1980 production per beef cow has increased from 205 to 261 kg (Cattle and Beef Industry Statistics, 1996). Average annual milk production in dairy cattle has increased from 2,101 to 7,065 kg since 1940 (NDHIA, 1994). A tremendous percentage of the increase in production may be attributed to breeding for increased performance. While beef cattle are increasingly being matched to range environments (Kress et al., 1986), selection for weaning weight, yearling weight, and carcass traits continues. In dairy cattle the pressure of selection for milk production has become so extreme, the national dairy herd is 90% Holstein (Lawlor, personal communication) due to favorable breed characteristics. Concern has been raised about the consequences of selecting for such high levels of production from ethical considerations of general animal welfare (Solbu and Lie, 1984; Shook et al., 1989) to consequences on health and immunity.

Biozzi et al. (1982) reported larger spleens containing increased levels of B lymphocytes, and larger, less active macrophages in mice selected for high antibody response to antigens. Poultry research by Gross and Siegel (1988) support these findings. Poultry were inoculated with sheep erythrocytes, and birds were selected for high (HA)

and low antibody (LA) response. After five generations of selection, LA poultry were heavier, had superior feed efficiency, matured earlier, layed more eggs, maintained fertility longer, had larger thymuses and smaller spleens. However, LA poultry had higher mortality rates. These differences were apparent through the first 250 d of life. Shanks et al. (1978) reported an increase in health disorders associated with increased milk production in dairy cattle. Simianer et al. (1991) reported incidence of disease increased as a result of genetic impovement of milk production.

Halliday et al. (1978) found a positive correlation between IgG_1, IgG_2, and IgM concentrations and daily weight gain in calves, but only through the first 42 d of life. Bradley et al. (1979) reported Shorthorn calves selected for yearling weight had lower ($P<.05$) serum immunoglobulin levels of colostral origin than a randomly selected line of Shorthorn calves. Muggli et al. (1984) determined in four lines of Hereford calves selected for weaning weight, yearling weight, muscling score, and a control line; control calves had higher ($P<.05$) concentrations of serum IgG_1 compared to calves selected for yearling weight. Muggli-Cockett et al. (1992) reported positive genetic correlations between preweaning and post-weaning bovine respiratory disease (BRD) and birth weight. However, this may be a function of decreased calf vigor due to stress at calving.

While the previous studies suggest that selecting for increased production decreases immune responsiveness, the following studies indicate selection for production traits coupled with selection for MHC haplotype may provide improved immune system function without sacrificing production. Simpson et al. (1982) reported an association between the mouse major histocompatability complex (H-2) and relative body size. Gill

and Kunz (1979) and Kunz et al. (1980) determined a gene influencing body size was closely linked to the MHC of rats. Associations between the MHC of swine (SLA) and growth rate have also been reported (Rothschild et al., 1986). Stear et al. (1989) related gene substitutions of the cattle MHC (BoLA) within breeds were significant for increasing birth weight, preweaning, and post-weaning weight gain. Muggli-Cockett et al. (1990) found no association of growth rate traits (ie. birth weight, adjusted 205 d weight, preweaning and post-weaning average daily gain through 1 yr of age) and 10 unique MHC haplotypes in 145 Angus calves. However, MHC haplotype was significantly associated with bovine viral diarrhea virus (BVDV) and bovine respiratory syncytial virus (BRSV) antibody titers at 30 d of age.

CHAPTER 3

MATERIALS AND METHODS

Cattle

All animals were maintained at the Northern Agricultural Research Center, near Havre, Montana. The cattle herd was comprised of inbred and outbred populations. Inbred animals were linebred Hereford cattle with an average inbreeding coefficient of .25. Outbred animals were Hereford, Angus x Hereford, Simmental x Hereford crosses and backcrosses, and Tarentaise x Hereford crosses and backcrosses.

Due to ongoing animal breeding research, not all breed groups were represented through the entire time period examined in this study with the exception of the linebred Hereford cattle. The purity and consistency of bloodlines maintained within the linebred Hereford cattle provide the foundation for this study. The cattle were considered inbred and outbred populations, with linebred Hereford cattle as the inbred population and all other breeds, including crossbreds and backcrosses, representing the outbred population.

Scours

Health and performance records of 3,637 calves from inbred and outbred populations were evaluated for a 14-yr period (1979, 1981 to 1989, 1991 to 1994) to determine the incidence and effect of calf scours on weaning weight. Years 1980 and 1990 were excluded from the study due to insufficient health records.

Calves were born during March and April. First calf heifers (2-yr-olds), representing 20 to 25% of breeding females, were bred to calve two weeks ahead of the main cow herd from 1979 to 1984, and 1989 to 1994. From 1985 to 1988 all dam age groups were bred to calve at the same time. During daylight hours first calf heifers were in a 16 hectare pasture. At night they were put in a smaller lot (15 x 46 m). When possible, first calf heifers were calved out in a shed. Older dams ($\geq$ 3-yr-olds), representing 75 to 80% of the breeding females, gave birth to calves in a 40 hectare calving pasture. Regardless of age of dam, within 24 to 48 h after calving, inbred and outbred animals were separated into one of two similar 40 hectare pastures. Both inbred and outbred cattle were managed similarly within these pastures. Calves dead at birth were removed from the data set.

An incidence of calf scours was defined as calves having been treated at least once for diarrhea within the first 4 mo of life (Makarechian et al., 1980). Calves observed scouring were treated with antibiotics and(or) electrolyte solutions.

Respiratory Illness

Health and performance records of 2,424 calves from inbred and outbred populations were evaluated for a 12-yr period (1981 to 1988, 1991 to 1994) to determine the incidence and effect of respiratory illness on calf final weight. Years 1979, 1980, 1989, and 1990 were excluded from the study due to insufficient health records.

After summer turn-out of inbred and outbred populations, calves were weaned the first week of October. At weaning calves were divided into groups by sex (ie. heifers, bulls, and steers) and managed differently. Heifers were placed in a large (90 x 305 m), atypical feedlot with a creek as the water source and fed to gain .6 to .7 kg per head per day. Bulls and steers were placed in typical feedlots (30 x 46 m) with a common waterer, and fed to gain 1.1 kg and 1.1 to 1.4 kg per head per day, respectively.

An incidence of respiratory illness was defined as calves having been treated at least once after weaning through the end of the calendar year for nasal discharge, fever, and(or) coughing (Muggli-Cockett et al., 1992). Calves observed with respiratory illness were treated with antibiotics.

Statistical Analyses

Incidence of Calf Illness

Incidence of calf scours was analyzed by a categorical analysis of variance procedure (SAS, 1995). The model included year, age of dam, and line (inbred or outbred). Incidence of calf respiratory illness was analyzed by a categorical analysis of

variance procedure (SAS, 1995). The model included year, age of dam, line (inbred or outbred), sex of calf (heifer, bull, or steer), and scours (calves contracting scours versus calves free of scours). Health and performance records for calves sold at weaning were not included in the analysis of respiratory illness.

Weaning Weight

Statistical analyses were run with two different data sets. The first data set included zero weaning weights for calves that died from scours. The second data set removed those calves. The purpose for analyzing the two data sets was to compare the impact of calf scours morbidity to calf scours mortality.

Weaning weight was analyzed by least squares analysis of variance (SAS, 1995). The model included year, age of dam, line (inbred or outbred), scours, and date of birth as a covariate. All two-way interactions were tested, and non-significant interactions were deleted from the final model.

Final Weight

Statistical analyses were run with two different data sets. The first data set included zero final weights for calves that died of respiratory illness. The second data set removed those calves. The purpose for analyzing the two data sets was to compare the impact of morbidity due to calf respiratory illness with mortality due to calf respiratory illness.

Final weight was analyzed by least squares analysis variance (SAS, 1995). The model included year, age of dam, line (inbred or outbred), sex of calf (heifer, bull, or steer), respiratory illness, and date of birth as a covariate. All two-way interactions were tested, and non-significant interactions were deleted from the final model.

CHAPTER 4

RESULTS AND DISCUSSION

Calf Morbidity

Incidence of Scours

Year affected ($P<.01$) incidence of scours (Table 1). Incidence of scours by year is summarized in Figure 1. Incidence of scours was lowest in 1982 at 13% and highest in 1991 at 64%. Mean rate of incidence was 35%, while median rate of incidence was 34%. Dargatz (personal communication) indicated large ($\geq$ 300 animals) Wyoming and Colorado cattle herds may have a scouring rate up to 72%. Clement et al. (1993) described scouring rates of 0.0 to 96.6% in North Dakota beef herds.

Over all years, 1.0% of calves died of scours. This mortality rate compares favorably with a national scours mortality rate of 5.1% (USDA:APHIS:VS, 1994). The difference in incidence of scours between inbred and outbred cattle was consistent over time (Figure 2), despite the introduction of Tarentaise cattle in 1989 to the Northern Agricultural Research Center.

Incidence of scours in calves was influenced ($P<.01$) by age of dam, and was greatest for calves born to 2-yr-old dams and least for calves born to older dams (Figure 3). These findings support the work of Makarechian et al. (1980) which

suggested incidence of scours is highest in calves born to 2-yr-old dams, and decreases with increasing age of dam.

Line affected (P<.01) incidence of scours with inbred calves contracting scours more frequently (41%) than outbred calves (28%). Incidence of scours was greatest in inbred calves born to 2-yr-old dams at 47% and lowest in outbred calves born to 5-yr-old and older dams at 25% (Figure 4). While little information is available on susceptibility of inbred versus outbred calves to scours, Makarechian et al. (1980) reported decreased incidence of calf scours in crossbred calves versus other breed groups monitored (ie. Hereford, beef synthetic, and dairy synthetic).

Incidence of Respiratory Illness

Year affected (P<.01) incidence of respiratory illness (Table 2). Incidence of respiratory illness is summarized in Figure 5 . Incidence of respiratory illness was lowest in 1991 at 5% and highest in 1986 at 43%. Mean rate of incidence was 22%, while median rate of incidence was 17%. Wittum and Perino (1995) reported a respiratory tract morbidity rate of 47% in post-weaned calves.

Over all years .003% of calves died of respiratory illness. This mortality rate compares favorably with a national respiratory disease mortality rate of 0.8% (USDA:APHIS:VS, 1994). The difference in incidence of respiratory illness between inbred and outbred cattle was consistent over time (Figure 6), despite the introduction of Tarentaise cattle in 1989 to the Northern Agricultural Research Center.

Incidence of respiratory illness in calves was influenced (P<.01) by age of dam, and was greatest for calves born to younger dams and least for calves born to older dams (Figure 7). These results were in contrast to Muggli-Cockett et al. (1992) who found calves born to 2-yr-old dams had increased preweaning, but lower post-weaning frequencies of bovine respiratory disease than calves born to older dams (P<.05). They concluded this may be due to interference of maternal antibodies in calves born to older dams with calf vaccinations. Enough maternal antibody may have been present so that the calf did not experience the immunological challenge necessary to mount its own independent response.

Line affected (P<.01) incidence of respiratory illness with inbred calves contracting respiratory illness more frequently (33%) than outbred calves (11%). Incidence of respiratory illness was greatest in inbred calves born to 2-yr-old and 3-yr-old dams at 37%, and lowest in outbred calves born to 4-yr-old dams at 7% (Figure 8).

Differences were detected (P<.01) in rate of respiratory illness due to sex of calf. Heifer calves had the lowest incidence of respiratory illness (8%), steers were intermediate (27%) and bulls were highest (31%). Incidence of respiratory illness was greatest at 48% in inbred bulls and least at 4% in outbred heifers (Figure 9). Differences between the sexes were attributed to management. Heifer calves were in a large (90 x 305 m), atypical feedlot with a creek for a water source, and rations were formulated for growth appropriate to reach puberty by 13 to 14 mo of age. Steer and bull calves were in a typical feedlot (30 x 46 m) with a common water source, and fed rations for higher rates of gain.

Contracting scours in the first 4 mo of life did not influence (P<.14) incidence of respiratory illness after weaning. However, when sex of calf was removed from the model, contracting scours in the first 4 mo of life influenced (P<.05) incidence of respiratory illness. The main effect of scours being non-significant in the model including sex of calf might be explained by the influence of sex, with steers and bulls experiencing greater stress (associated with increased average daily gain, smaller lot size, and common waterers) over-riding any residual effects scours in the first 4 mo of life may have had. Rate of respiratory illness in weaned calves that scoured in the first 4 mo of life was 21% and 18% for non-scouring calves.

Calf Performance Traits

Weaning Weight

There was no difference in significance of effects obtained from analysis of variance between the data set including calves that died from scours (Table 3), and the data set without those calves (Table 4). Therefore, data reported in this section reflect zero weaning weights included for calves that died of scours.

Year affected (P<.01) calf weaning weight (Table 3), and there was an increase in calf weaning weight over time (Figure 10). The increase was attributed to independent animal breeding projects that led to heightened calf performance (Kress et al., 1989a, 1989b, 1995).

Calf weaning weight was influenced (P<.01) by age of dam, and rose with increasing age of dam (Figure 11). A significant (P<.01) year x age of dam interaction indicated the effect of age of dam was different over time (Figure 12). Two-yr-old dams consistently weaned the lightest calves, while 3-yr-old dams consistently weaned the second lightest calves. A non-significant (P<.39) age of dam x scours interaction indicated the effect of scours on calf weaning weight was not different for each age group of dam (Figure 13).

Line affected (P<.01) calf weaning weight with inbred calves weighing 205 kg, and outbred calves weighing 219 kg. The 14 kg difference in weaning weight was attributed to an inbreeding depression and breed differences (Kress et al., 1990a, 1990b, 1995). A significant (P<.01) year x line interaction indicated the effect of line on calf weaning weight was different over time (Figure 14). However, inbred calves consistently weighed less than outbred calves. The non-significant line x scours interaction (P<.98) indicated the effect of scours on calf weaning weight was not different for inbred versus outbred cattle (Figure 15).

Contracting scours negatively impacted (P<.01) calf weaning weight. Scouring calves weighed 208 kg at weaning, while non-scouring calves weighed 217 kg. Wittum and Perino (1995) reported similar observations. Morbidity, including calves scouring during the first 28 d of life, was associated with a 16 kg decrease in expected weaning weight.

The least squares mean difference in weaning weight for scouring and non-scouring calves ranged from 2 kg in 1994 to 21 kg in 1981 (Figure 16), even though there

was no year x scours interaction (P<.34). The mean difference in weaning weight of scouring and non-scouring calves over all years was 9 kg, while the median difference was 8 kg.

Final Weight

There was no difference in significance of effects obtained from analysis of variance between the data set including calves that died of respiratory illness (Table 5), and the data set without those calves (Table 6). Therefore, the data reported in this section reflect zero final weights included for calves that died of respiratory illness.

Year affected (P<.01) calf final weight (Table 5). Figure 17 shows the least square mean final weights of calves over time. Calf final weight was influenced (P<.01) by age of dam, and rose with increasing age of dam (Figure 18). A significant (P<.01) year x age of dam interaction indicated the effect of age of dam was different over time (Figure 19). The significant line x age of dam interaction (P<.01) indicated the effect of age of dam on calf final weight was different for inbred versus outbred cattle (Figure 20). However, inbred calves consistently had lower final weights regardless of age of dam. A non-significant (P<.30) age of dam x respiratory illness interaction indicated the effect of respiratory illness on calf final weight was not different for each age group of dam.

Line affected (P<.01) calf final weight with inbred calves weighing 374 kg, and outbred calves weighing 412 kg. The 38 kg difference was attributed to an inbreeding depression and breed differences (Kress et al., 1990a, 1990b, 1995). A significant year x line interaction indicated the effect of line on calf final weight was different over time

(Figure 21). However, inbred calves consistently weighed less than outbred calves, with a minor exception in 1992. The non-significant line x respiratory illness interaction (P<.84) indicated the effect of respiratory illness on calf final weight was not different for inbred versus outbred cattle.

The sex of a calf influenced (P<.01) its final weight. The least squares mean final weight was 337, 403, and 439 kg for heifer, bull, and steer calves, respectively. A significant (P<.01) age of dam x sex of calf interaction indicated the effect of sex of calf on final weight was different for each age group of dam (Figure 22). However, the ranking of sex of calves for least squares mean final weights was constant for each age group of dam with heifers lightest, bulls intermediate, and steers heaviest.

Contracting a respiratory illness negatively impacted (P<.01) calf final weight. The final weight of calves contracting respiratory illness was 387 kg, while calves free of respiratory illness weighed 399 kg. These results are supported by the work of Thurmond and Pare´ (1993) which suggested as number of episodes of calf respiratory illness increase, the time required for dairy calves to reach 91 kg increased. A significant year x respiratory illness interaction indicated the effect of respiratory illness on calf final weight was different over time (Figure 23). However, calves contracting respiratory illness generally had lower final weights than calves free of respiratory illness. The least squares mean difference in final weight of calves contracting respiratory illness and calves free of respiratory illness ranged from -9 kg in 1983 to 30 kg in 1985 (Figure 24). The mean difference in final weight of calves contracting respiratory illness and calves

free of respiratory illness over all years was 10 kg, while the median difference was 13 kg.

Economic Loss

Weaning Weight

Using average calf prices for October (USDA, 1996), least squares mean differences in weaning weights for scouring versus non-scouring calves, scours treatment costs of $10.00 for labor and medicine (Wittum et al., 1993), and the Consumer Price Index (Bureau of Labor Statistics, 1996) to adjust to September, 1996 U.S. currency, loss in income due to scours over all years at the Northern Agricultural Research Center was estimated at $40,292.28. Estimated loss in income per year for all calves contracting scours ranged from $789.06 in 1982 to $6,302.63 in 1989 (Figure 25). Mean estimated loss in income per year was $2,878.02, while median estimated loss was $2,554.15. Estimated loss in income on a per animal basis for calves contracting scours was lowest in 1994 at $17.28 and highest in 1981 at $59.96 (Figure 26). Mean estimated loss in income on a per animal basis was $34.84, while median estimated loss was $32.36.

Final Weight

Using average calf prices for March (USDA, 1996), least squares mean differences in final weights of calves contracting respiratory illness versus calves free of respiratory illness, respiratory illness treatment costs of $12.00 for labor and medicine (Wittum et al., 1993), and the Consumer Price Index (Bureau of Labor and Statistics, 1996) to adjust to

September 1996 U.S. currency, total loss in income due to respiratory illness over all years at the Northern Agricultural Research Center was estimated at $15,400.26. Estimated loss in income per year for all calves contracting respiratory illness ranged from -$287.81 in 1983 (a negative number means a net gain for calves contracting respiratory illness) to $2,591.69 in 1985 (Figure 27). Mean estimated loss in income per year was $1,283.36, while median estimated loss was $1,116.61. Estimated loss in income on a per animal basis for calves contracting respiratory illness was lowest in 1983 at -$3.55 and highest in 1993 at $74.88 (Figure 28). Mean estimated loss in income on a per animal basis was $39.80, while median loss was $42.53.

The negative values for 1983 may be interpreted to mean calves experiencing respiratory illness actually had greater final weights than healthy calves. That year a relatively high number of calves (n=81) had respiratory illness which may have allowed for preferential treatment. While a net gain in final weight was ultimately posted by calves contracting respiratory illness, any additional feed and labor costs were not accounted for using the formula mentioned above.

General Discussion

Incidence of disease was highest for calves born to young dams, however the effects of disease on weaning and final weight were the same regardless of age of dam. A biological explanation for the higher incidence, yet similar effects of disease in these calves, is the failure of passive transfer of maternal IgG_1 within the first 24 to 48 h of life.

This is in agreement with several studies where age of dam negatively influenced IgG_1 concentrations in colostrum and serum of newborn calves (Bradley et al., 1979; Norman et al., 1981; Muggli et al., 1984; Perino et al., 1995).

While colostrum and calf serum data were not available for animals in this study, the results of our analyses agree with the body of literature available for the failure of passive transfer of maternal IgG_1. Calves in our study exhibited increased disease morbidity and decreased weaning and final weights consistent with increases in morbidity for calfhood disease and decreases in weaning weight and average daily gain in calves experiencing failure of passive transfer of IgG_1 (Perino and Wittum, 1995).

Incidence of disease was highest in inbred calves, particularly calves born to young inbred dams. However, the effect of disease decreasing weaning and final weights was the same regardless of line. Very little literature is available comparing the incidence and effect of disease in inbred versus outbred cattle. Muggli et al. (1984) reported calves from a closed line of Hereford cattle (F_x unavailable) had higher mortality rates than other breeds represented in the project.

In a study (Gilbert et al., 1988b) investigating the effect of inbreeding on IgG_1 concentrations in newborns; line of sire, inbreeding of calf, and age of dam did not affect serum IgG_1 concentrations in calves 36 h old. These findings are in contrast to other research (Halliday et al., 1978; Bradley et al., 1979, Norman et al., 1980; Muggli et al., 1984; Gilbert et al., 1988a; Perino et al., 1995) which suggest line of sire and age of dam affect serum IgG_1 concentrations in newborn calves. Gilbert acknowledged these discrepancies and attributed a lack of age of dam difference to the possibility of

increased assistance being given to non-suckling calves. While not mentioned, this would also negatively bias any line differences.

Increased incidence of calf scours over time in the present study corresponded with increases in calf weaning weights (Figures 1 and 10). A possible explanation may be found in the work of Muggli et al. (1984) which indicated selecting for weaning weight, yearling weight, and muscling score may negatively impact immune responsiveness. The observation for scours and weaning weights did not hold when evaluating incidence of respiratory illness and final weights (Figures 5 and 17). When final weights were highest, no clear trend of increasing incidence of respiratory illness was apparent; suggesting the need for further research in this area.

CHAPTER 5

SUMMARY

Tremendous potential exists for immunogenetics research with the inbred, Hereford cattle at the Northern Agricultural Experiment Center. At the writing of this thesis, the author is unaware of an applied animal breeding research project evaluating failure of passive transfer of maternal immunity to calves. Historically, the failure of passive transfer has been defined on the basis of pre-parturition cow serum, colostral, and neonatal calf serum concentrations of IgG_1. While, this has provided a foundation for immunological evaluation, it represents a minute portion of the bovine immune system. A project which would combine haplotyping of cattle for immune system and growth traits; a thorough evaluation of bovine neonatal-maternal immunity which evaluates concentration and competence of T lymphocytes, immunoglobulins, and complement in pre-partum cow serum, colostrum, and newborn calf serum; level of inbreeding; and records for growth and performance traits would be a landmark study.

Adequately defining and scientifically determining if a genetic mechanism exists for failure of passive transfer is of utmost importance. Research of this kind is needed to correctly identify any antagonistic effects inbreeding and(or) selection may have on the development of proper immunity in the calf if efficiency of production remains the primary goal of livestock industry.

Table 1. Categorical analysis of variance for effect of year, age of dam, and line on incidence of calf scours.

Source	DF	Chi-Square	Probability
Year	13	276.49	.0001
Age of Dam	3	16.11	.0011
Line	1	51.09	.0001

Table 2. Categorical analysis of variance for effect of year, age of dam, line, sex of calf, and scours on incidence of respiratory illness.

Source	DF	Chi-Square	Probability
Year	11	116.81	.0001
Age of Dam	3	10.78	.0130
Line	1	107.13	.0001
Sex of Calf	2	160.43	.0001
Scours	1	2.18	.1402

Table 3. Analysis of variance for effect of year, age of dam, line, and scours on calf weaning weight including zero weaning weights for calves dying of scours.

Source	DF	MS	F ratio	Pr>F
Year	13	129,550.07	27.71	.0001
Age of Dam	3	856,313.04	183.14	.0001
Line	1	525,220.24	112.33	.0001
Scours	1	296,879.76	63.49	.0001
Year x AOD	39	23,857.64	5.10	.0001
Year x Line	13	13,572.08	2.90	.0003
DOB	1	2,441,319.16	522.12	.0001
Residual	3,565	4,675.82		

Table 4. Analysis of variance for effect of year, age of dam, line, and scours on calf weaning weight without zero weaning weights for calves dying of scours.

Source	DF	MS	F ratio	Pr>F
Year	13	115,416.75	36.58	.0001
Age of Dam	3	820,823.08	260.15	.0001
Line	1	407,621.35	129.19	.0001
Scours	1	53,426.15	16.93	.0001
Year x AOD	39	23,180.26	7.35	.0001
Year x Line	13	11,910.81	3.78	.0001
DOB	1	2,285,434.27	724.35	.0001
Residual	3,537	3,155.13		

Table 5. Analysis of variance for effect of year, age of dam, line, sex of calf, and respiratory illness on calf final weight including zero final weights for calves dying of respiratory illness.

Source	DF	MS	F ratio	Pr>F
Year	11	150,160.94	11.12	.0001
Age of Dam	3	77,137.45	5.71	.0007
Line	1	1,813,820.05	134.37	.0001
Sex of Calf	2	4,880,506.92	361.54	.0001
Respiratory	1	180,471.03	13.37	.0003
Year x AOD	33	110,107.92	8.16	.0001
Year x Line	11	112,730.79	8.35	.0001
Year x Resp	11	27,194.12	2.01	.0236
Line x AOD	3	98,330.73	7.28	.0001
AOD x Sex	6	126,957.41	9.40	.0001
DOB	1	986,793.19	73.10	.0001
Residual	2,340	13,499.11		

Table 6. Analysis of variance for effect of year, age of dam, line, sex of calf, and respiratory illness on calf final weight without zero final weights for calves dying of respiratory illness.

Source	DF	MS	F ratio	Pr>F
Year	11	146,408.27	12.87	.0001
Age of Dam	3	81,900.52	7.20	.0001
Line	1	1,719,845.47	151.13	.0001
Sex of Calf	2	4,576,948.40	402.20	.0001
Respiratory	1	42,198.86	3.71	.0543
Year x AOD	36	111,447.81	9.79	.0001
Year x Line	12	115,980.44	10.19	.0001
Year x Resp	11	24,488.03	2.15	.0145
Line x AOD	3	105,008.21	9.23	.0001
AOD x Sex	6	108,283.70	9.52	.0001
DOB	1	905,333.89	79.56	.0001
Residual	2,499	11141.20		

LITERATURE CITED

Amorena, B., and W.H. Stone. 1978. Serologically defined (SD) locus in cattle. Science. 201:159.

Biozzi, G., D. Mouton, A. Heumann, Y. Bouthillier, C. Stiffel, and J.C. Mevel. 1979. Genetic analysis of antibody responsiveness to sheep erythrocytes in crosses between lines of mice selected for high or low antibody synthesis. J. Immunol. 36:427.

Boyd, J.W. 1972. The relationship between serum immunoglobulin deficiency and disease in calves: A farm survey. Vet. Rec. 90:645.

Bradley, J.A., L. Nilo, and W.J. Dorward. 1979. Some observations on serum gammaglobulin concentrations in suckled beef calves. Can. Vet. J. 20:227.

Brambell, F.W.R. 1969. The Transmission of Passive Immunity from Mother to Young. In: Vol.18. Am. Elsevier Publishing Co., Inc. New York.

Brandon, M.R. and A.K. Lascelles. 1974. The effect of prepartum milking on the transfer of immunoglobulin into the mammary secretion of cows. Lactation.

Bureau of Labor Statistics, U.S. Federal Government. 1996. Consumer Price Index as of November 2, 1996. Series ID: CUUR0000SA0. Available: http://stats.bls.gov/cgi-bin/surveymost?cu.

Campbell, S.G., M. J. Siegel, and B.J. Knowlton. 1977. Sheep immunoglobulins and their transmission to the neonatal lamb. N.Z. Vet. J. 25:361.

Cattle and Beef Industry Statistics. 1996. National Cattlemen's Beef Association, Cattlemen's Beef Promotion and Research board. Statistics as of November, 1996. Available:http://www.ncanet.org/beef_stat/cbis_1196.html#2.1.

Claman, H. N., E.A. Chaperon and R.F. Triplett. 1966. Thymus-marrow cell combinations. Synergism in antibody production. Proc. Soc. Exp. Biol. Med. 122:1167.

Clement, J.C., M.E. King, T.E. Wittum, R.D. Biwer, M.J. Fleck, M.D. Salman, and K.G. Odde. 1993. Factors associated with the incidence of calf scours in North Dakota beef herds. Agri-Practice. 9:13.

Coady, S.A. and R.T. Clark. 1993. Ranch management practices in the Sandhills of Nebraska: Managing production. RB 318, Agric. Res. Div., Institute of Agric. and Nat. Res., Univ. of Nebraska, Lincoln. p.61.

Cundiff, L.V. 1984. Quantitative genetic approaches to breeding for genetic resistance to disease in cattle. In: Proceedings of Characterization of the Bovine Immune System and the Genes Regulating Expression of Immunity with Particular Reference to Their Role in Disease Resistance. W.C. Davis, J.N. Shelton, and C.W. Weems (Ed).

Dargatz, D. 1996. Personal communication. David Dargatz. USDA:APHIS:VS 533 South Howes, Suite 200, Fort Collins, CO 80521.

Dixon, F.J., W.O. Weigle, and J.J. Vazquez. 1961. Metabolism and mammary secretion of serum proteins in the cow. Lab. Invest. 10:226.

Frerking, H. and T. Aeikins. 1978. About the importance of colostrum for the newborn calf. Ann. Rech. Vet. 9:361.

Gilbert, R.P., C.T. Gaskins, J.K. Hillers, C.F. Parker, and T.C. McGuire. 1988a. Genetic and environmental factors affecting immunoglobulin G_1 concentrations in ewe colostrum and lamb serum. J. Anim. Sci. 66:855.

Gilbert, R.P., C.T. Gaskins, J.K. Hillers, J. S. Brinks, and A.H. Denham. 1988b. Inbreeding and immunoglobulin G_1 concentrations in cattle. J. Anim. Sci. 66:2490.

Gill, T.J., and H.W. Kunz. 1979. Gene complex controling growth and feritility linked to the major histocompatability complex in the rat. Am. J. Pathol. 96:185.

Glick, B., T.S. Chang and R.G. Jaap. 1956. The bursa of Fabricius and antibody production. Poul. Sci. 35:224.

Goetz, D. 1977. The Major Histocompatability System in Man and Animals. Springer-Verlag. Berlin.

Green, R.D. 1993. Genetic and environmental factors affecting survivability and disease resistance in beef cattle. Proc. Matching Beef Cattle to Western Environements. Gilbert Publishing, Inc., Saskatoon, Alberta, Canada. p.56.

Gross, W.B., and P.B. Siegel. 1988. Environment-genetic influences on immunocompetence. J. Anim. Sci. 66:2091.

Haggard, D.L., R.J. Farnsworth, and J.A. Springer. 1983. Subclinical mastitis of beef cows. J. Am. Vet. Med. Assoc. 182:604.

Halliday, R. 1978. Immunity and health in young lambs. Vet. Rec. 103:489.

Kehrli, M., M.R. Ackermann, D.E. Shuster, M.J. van der Maaten, F.C. Schmalstieg, D.C. Anderson, and B.J. Hughes. 1992. Animal model of human disease: Bovine Leukocyte Adhesion Deficiency, β_2 Integrin Deficiency in young, Holstein cattle. Am. J. Path. 140:1489.

Kress, D.D., D.E. Doornbos, D.C. Anderson, and K. Hanford. 1986. Performance of different biological types of cows: Cow traits. MT AgResearch. MT. Agric. Exp. Sta. Vol 3. Issue 1:13.

Kress, D.D., D.E. Doornbos, and D.C. Anderson. 1990a. Performance of crosses among Hereford, Angus, and Simmental cattle with different levels of Simmental breeding: IV. Maternal heterosis and calf production by two-year-old dams. J. Anim. Sci. 68:54.

Kress, D.D., D.E. Doornbos, and D.C. Anderson. 1990b. Performance of crosses among Hereford, Angus, and Simmental cattle with different levels of Simmental breeding: V. Calf production, milk production, and reproduction of three- to eight-year-old dams. J. Anim. Sci. 68:1910.

Kress, D.D., D.E. Doornbos, D.C. Anderson, and K.C. Davis. 1995. Tarentaise and Hereford breed effects on cow and calf traits and estimates of individual hetersosis. J. Anim. Sci. 73:2574.

Kronenberg, M., G. Siu, L.E. Hood, and H. Shastri. 1986. The molecular genetics of the T-cell antigen receptor and T-cell antigen recognition. Annu. Rev. Immunol. 4:529.

Kuby, J. 1992. Immunology. Freeman, New York.

Kunz, H.W., T.J.Gill, B.D. Dixon, F.H. Taylor, and D.L. Greiner. 1980. Growth and reproduction complex in the rat. Genes linked to the major histocompatability complex that affect development. J. Exp. Med. 152:1506.

Larson, B.L., and K.A. Kendall. 1957. Changes in specific blood serum protein levels associated with parturition in the bovine. J. Dairy. Sci. 40:659.

Lawlor, T. 1996. Personal communication. Thomas Lawlor. Holstein Association, 1 Holstein Place, Brattleboro, VT 05302.

Long, C.R. 1980. Crossbreeding for beef production: Experimental results. J.Anim. Sci. 51:1197.

Makarechian, M. and R.T. Berg. 1980. The effect of breed on the incidence of calf scours neonatal diarrhea in beef cattle under range conditions. Agric.-For. Bull. Edmonton, University of Alberta (special issue) p.17.

Malak, V.S., and E.P. Killehoj. 1994. Antibody Techniques. Academic Press. San Diego.

McDonough, S.P., C.L. Stull, and B.I. Osburn. Enteric pathogens in intensively reared veal calves. Am. J. Vet. Res. 5:1516.

McGuire, T.C., N.E. Pfeiffer, J.M. Weikel, and R.C. Bartsch. 1976. Failure of colostrum immunoglobulin transfer in claves dying from infectious disease. J. Am. Vet. Med. Assoc. 169:713.

McGuire, T.C., J. Regnier, T. Kellom, and N.L. Gates. 1983. Failure in passive transfer of immunoglobulin G_1 to lambs: Measurement of immunoglobulin G_1 in ewes colostrum. Am. J. Vet. Res. 43:1064.

Mueller, L.D. and D.K. Elleinger. 1981. Colostral immunoglobulin concentrations among breeds of dairy cattle. J. Dairy Sci. 64:1727.

Muggli, N.E., W.D. Hohenboken, L.V. Cundiff, and K.W. Kelley. 1984. Inheritance of maternal immunoglobulin G_1 concentration by the bovine neonate. J. Anim. Sci. 59:39.

Muggli, N.E., W.D. Hohenboken, L.V. Cundiff, and D.E. Mattson. 1987. Inheritance and interaction of immune traits in beef calves. J. Anim. Sci. 64:385.

Muggli-Cockett, N.E., C.L. Kelling, and R.T. Stone. 1991. Associations of the bovine major histocompatability complex with measurements of growth and serum antibody titers specific for two viruses in beef cattle. J. Anim. Sci. 69:200 (Suppl.1).

Muggli-Cockett, N.E., L.V. Cundiff, and K.E. Gregory. 1992. Genetic analysis of bovine respiratory disease in beef calves during the first year of life. J. Anim. Sci. 70:2013.

National Dairy Herd Improvement Association. 1994. National Handbook, Status of U.S. Dairy Cattle.

Newby, T.J., and J. Bourne. 1976. The nature of the local immune system of the bovine mammary gland. J. Immunol. 118:461.