EPIDEMIOLOGICAL INVESTIGATION OF HEALTH AND PERFORMANCE OF HOLSTEIN DAIRY HEIFERS

A Thesis

Submitted to the Graduate Faculty

for the Degree of

Master of Science

in the Department of Health Management

Faculty of Veterinary Medicine

University of Prince Edward Island

G. Arthur Donovan

Charlottetown, P.E.I.

August, 1993

ISBN 0-315-88454-1

SIGNATURE PAGES

ii-iii

REMOVED

ABSTRACT

A prospective cohort study was undertaken to determine calf-level factors that affected calf health status and performance (growth) from birth to 14 months of age. A convenience sample of approximately 3300 Holstein calves from two large Florida dairy farms were used for the study. Data collected on each calf at birth included farm of origin, weight, height at the pelvis, birth date, and serum total protein (a measure of colostral immunoglobulin absorption). Birth season was dichotomized into summer and winter using meteorological data collected by University of Florida Agricultural Research Stations. Data collected at approximately 6 and 14 months included age, weight, height at the pelvis, and height at the withers. Growth in weight and stature (height) was calculated for each growth period; growth period 1 (GP1) = birth to 6 months and growth period 2 (GP2) = 6 to 14 months. Health data including date of initial treatment and number of treatments were collected monthly for the diseases diarrhea, omphalitis or 'navel infection', septicemia, pneumonia and keratoconjunctivitis or 'pinkeye'.

Serum total protein (TP) was found to be a significant risk factor for mortality. This relationship of TP with mortality was quadratic and showed a dramatic decrease in mortality as TP increased from 4.0 to 5.0 g/dl, a small improvement from 5.0 to 6.0 g/dl and virtually no improvement in mortality rates as TP increased over 6.0 g/dl. The hazard mortality ratio was constant over the period from birth to six months. No interactions between TP, farm, season, or birth weight were found in these analyses.

Serum total protein concentration was a significant risk factor for the occurrence, age of onset and severity of septicemia and pneumonia. The association between TP and septicemia was linear and an interaction with birth season was found. The association between TP and pneumonia was quadratic, and contrary to the TP and septicemia relationship, the morbidity hazard ratio for pneumonia was not constant over the time measured; that is, colostral immunity protected the calf from developing pneumonia early in life, but this effect disappeared as the calf got older. Total protein was not a significant risk factor for diarrhea or omphalitis.

No significant associations were found among any of the diseases monitored.

Passive transfer of colostral immunoglobulins had no significant effect on rate of body weight or pelvic height growth. Season of birth and occurrence of diarrhea, septicemia and respiratory disease were significant variables affecting heifer growth (height and weight) in GP1. These variables, along with with farm, birth weight and age when 6 month data were collected, explained 20% and 31% of the variation in body weight and pelvic height gain, respectively, in GP1. The number of days treated for pneumonia before 6 months significantly decreased average daily weight gain in GP2 ($P<0.025$), but did not affect stature growth. Treatment for pneumonia after 6 months did not significantly affect weight or height gain. Neither omphalitis or pinkeye were helpful in explaining the variability in growth in either of the growth periods.

ACKNOWLEDGEMENTS

The realization of this research and thesis is the result of the mentoring and guidance of three outstanding veterinarians. Dr. Bob Curtis, whether he knows it or not, was the first person to really get me turned on to food animal practice and was instrumental in getting me my first veterinary job. He has been supportive and a friend ever since. Dr. Ken Braun, my mentor and boss at the University of Florida, coerced me into staying in academia 'just for a year or two'. Ken sets a fast pace that few can keep up with and it is his forward thinking and enthusiasm for herd health practice and teaching that I have tried to emulate. Lastly, I would like to acknowledge Dr. Ian Dohoo, my graduate supervisor, problem-solver and friend. Ian has been tremendously supportive of me and he always found a way, without breaking stride, to get around the obstacles thrown up in the path of my 'patchwork' graduate program. His financial support and confidence in me is greatly appreciated.

My supervisory committee needs special recognition. Ian Dohoo, Mary McNiven, Luis Bate and Dave Montgomery probably have never served on a committee as unique as this one. This long distance relationship seemed to work and I thank each and every one for their dedication and guidance.

I could not have completed this program had it not been for two groups of people that are very dear to me. I am grateful of the support and encouragement of the 'family', the

faculty, residents and staff of the Rural Animal Medicine Service in the College of Veterinary Medicine, University of Florida. It is to Peter Chenoweth, Owen Rae, Carlos Risco, Richard Barker, Juan Velez and Billy Smith that I owe a debt of gratitude that will be impossible to repay. To Fred Bennett, my technician, I owe nearly everything. Early morning trips to far away dairies, lifting and bleeding 3300 calves in all kinds of weather, what more can I say Fred, but "Thank you so very much for your help". The numerous friends that were developed while I was on the Island are the backbone of the network of people that encouraged me and supported me throughout the program. Thanks, eh.

The two dairy farms upon which this research was performed need special recognition. I cannot possibly acknowledge everyone who played a part in this effort at the farms, but several important players need to be recognized. At North Florida Holsteins, Bell, Florida, Mr. Don Bennink and Mr. David Sumrall and at McArthur Farms, Okeechobee, Florida, Kent Bowen, Jay Lemmerman and Chuck Zahn have been overwhelmingly supportive and have been eager and active participants from the first day.

Lastly, and most importantly, I want to thank my wife, Patti, and my two wonderful daughters, Jessie and Chris for putting up with me during this project. Patti was supportive throughout even when it meant being a single parent for 3 months. Living in PEI in 1990 was one of the highlights for our young family and the long winter of '93 will always be remembered and never be repeated.

TABLE OF CONTENTS

TABLE OF CONTENTS (continued)

TABLE OF CONTENTS (continued)

CHAPTER 1

GENERAL INTRODUCTION

1.1 Overview of Dairy Replacement Enterprise

Dairy replacement rearing is a specialized area of management within the dairy industry that accounts for 9 to 20% of the total expense to produce milk. Only feed cost for the lactating herd, and possibly labour expenses, are greater expenses for the dairy enterprise (39). The data presented in an enterprise budget for a Florida dairy farm (Appendix A) support this claim. In most cases, rearing of the replacement heifer occurs on the farm into which the heifer will enter as a lactating cow. The efficiency of this practice may be questioned in light of research results. Surveys of the cost to raise a heifer to calving range from $750 to $1300 (9, 47), yet Holstein heifers are readily available for $900 to $1100. The factors involved in this broad range of costs are not well defined. Feed and interest expenses account for 48 to 68% of the cost to raise the heifer (39, Appendix A). Other factors known to influence the economics of heifer rearing include morbidity and mortality patterns, age at first calving, weight at first calving, and housing and breeding expenses (12, 24, 25, 35, 39).

Purchasing the replacement heifer from other dairy farmers or commercial heifer growers is a management strategy that is employed by some producers. However, in this case, control and knowledge of genetics, health and growth of these heifers is lost. Less

frequently, farm management may elect to have their heifers reared under contract by someone dedicated solely to this task. The cost and quality of heifers reared under these circumstances vary greatly.

1.2 Morbidity and Mortality

Health problems can add directly to the cost of raising a heifer to calving by increasing treatment, labour and veterinary costs. The value of those calves dying must also be added to those that survive to calving (mortality cost). Indirect cost are also seen with excessive mortality because the opportunity to sell extra heifers is not realized and additional heifers may need to be purchased at a cost that is greater than that of heifers raised on the farm.

Of the management factors utilized to reduce health problems in calves and heifers, proper management of colostrum is one of the most important. The significance of colostrum to the newborn calf has been recognized since at least 1922 (40). Research on the negative effects of failure of passive transfer on calf health, especially diarrhea and respiratory disease, is well documented (4, 8, 11, 15, 20, 29, 43). These studies reported a 2- to 4-fold increase in mortality in calves with failure of passive transfer (FPT) compared to those that have received adequate amounts of colostral immunoglobulins. However, most studies have had short follow-up periods after birth, were based on relatively small sample sizes and primarily addressed mortality due to

enteric and respiratory disease. The effects of colostrum on severity and age of onset of disease has not been examined.

In several studies, the effects of colostrum intake on body weight gain was determined with conflicting results being reported. Researchers from the British Isles (3, 8, 14) failed to find an association between passive transfer status and calf growth or calf health, while most North American studies have observed a positive effect of level of passive transfer on growth in the pre-weaning period (7, 11, 30). Robison et al. (36) are the only researchers to find a significant positive effect of passively acquired immunity on heifer growth rate through 180 days of age. No attempts have been made to determine if any positive effect seen were due to some factor in the colostrum or if it was mediated through an improvement in calf health.

Associations amongst calfhood diseases have not been well defined. In most calf disease studies, the only well defined diseases have been diarrhea and respiratory disease, probably because these diseases are easy to define and there is a low probability of misclassification. Waltner-Toews, et al. (45) found a 3-fold increase in the odds of pneumonia in calves that had been treated for diarrhea. In this study, diarrhea did not necessarily precede pneumonia, but the median age of onset of diarrhea was less than that of pneumonia. In a New York study (10) in which a similar association between diarrhea and pneumonia was found, calves that had diarrhea were also 10 times more likely to develop 'dull calf syndrome'. This syndrome was vaguely defined with clinical signs that

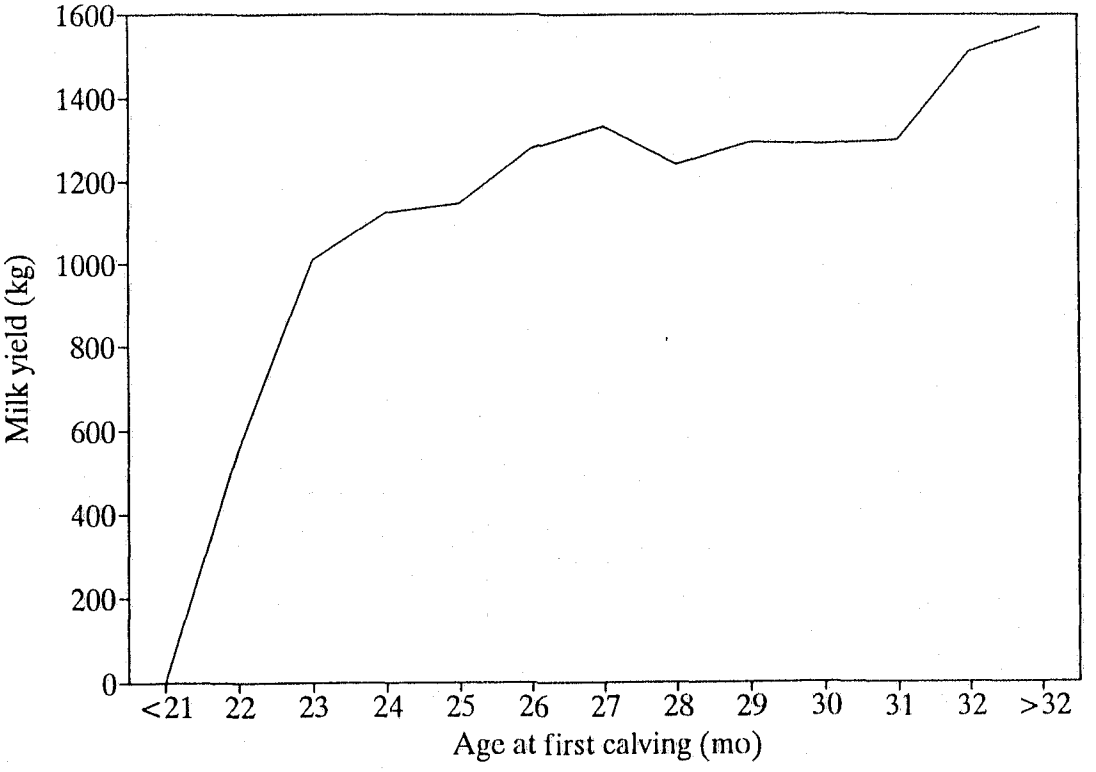
included listlessness, droopy ears, dullness or anorexia within 3 months of birth and may have been due to any number of disease conditions. Other common calf health problems such as navel infection, septicemia and pinkeye have not been investigated.

1.3 Age and Weight at Calving

The identification of a target age of calving for dairy heifers has been extensively studied (1, 24, 35, 42, 44). Keown, et al. (24) found that first lactation milk yield in Holstein heifers calving between 23 and 25 months was greater than that in heifers calving younger than 23 months and was the same as production in heifers calving at older ages (Figure 1). The same pattern of milk production was seen in second lactation cows. In an Israeli study (35), first lactation net income, total lifetime milk yield and lifetime gross income were maximized at calving ages between 22 and 24 months. Heifers calving at 23 to 24 months also had a greater probability of staying in the herd than those calving earlier or later (42). However, in spite of this knowledge, the average age at first calving in most herds ranges from 26 to 29 months (24, 25, 33, 37, 44).

The cost of delayed first parturition can be substantial. Values of $1US to $3US per day in excess of 24 months have been reported (9, 47, Appendix A). Feed costs make up the bulk of this expense, but other variable costs such as interest, as well as the fixed costs associated with a larger replacement herd must be considered. There is also a one time opportunity cost (gain) that can be realized if the age at first calving is reduced to

Figure 1 Relationship between first lactation milk yield and age at first calving; milk yield presented as increase over baseline age at first calving of ≤21 months.

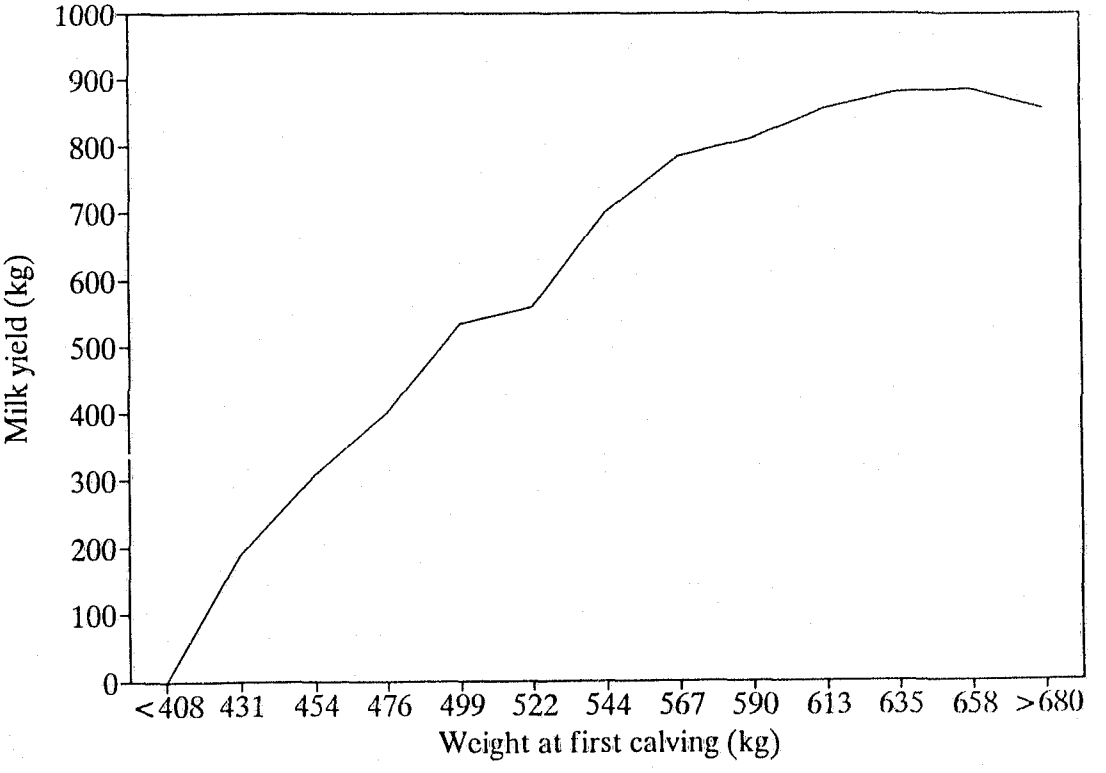


24 months. Extra heifers will be available to enter the herd which allows management the option to increase herd size, cull non-productive cows or sell the additional heifers. Weight at calving was not evaluated in the studies on age at first calving. However, Heinrichs and Hargrove (19) found a negative correlation ($r=-0.22$) between age at first calving and herd average milk yield, which in turn was positively correlated with weight ($r=0.34$) and height ($r=0.41$) at 24 months.

The average weight at which Holstein heifers calve varies from less than 410 kg to greater than 680 kg (25). Keown, et al. (25) found that first lactation milk yield increased in a curvilinear manner as weight at calving increased (Figure 2). They concluded that a calving weight of 550 to 600 kg was ideal to maximize first lactation milk production.

Attaining and maintaining proper growth in dairy heifers is of paramount importance. To achieve these target ages and weights at calving, producers must balance a multitude of factors, some of which are in conflict with each other (21). Calves and heifers must be fed in a cost effective manner to gain 0.7 to 0.8 kg per day throughout the growth period. Any deviation below this goal will result in lighter weight heifers, or older heifers at calving. Smaller heifers not only produce less milk, they are at increased risk of dystocia and other related periparturient health problems (13, 35, 38, 44). Older heifers add considerably to the cost of raising the replacement (9, 12, 47, Appendix A).

Figure 2 Relationship between first lactation milk yield and body weight at first calving; milk yield presented as increase over baseline weight at first calving of ≤408 kg.



The effects of diseases on growth of the dairy heifer have not been documented. Parasitism has been shown to cause a reduction in growth rate in all species of livestock (6, 22, 27, 32). The effects of pneumonia on growth have been documented in feedlot cattle (23, 26, 28) and swine (2, 5, 31, 48). Karren, et al. (23) demonstrated reduction in growth rate of 0.14 and 0.08 kg/d in studies of 68 and 95 days in length, respectively. In a study of veal calf health, both treatment for respiratory disease and area of lung consolidation at slaughter were directly related to a reduction in daily weight gain (34).

There is a strong body of evidence, although it is now somewhat dated, that growing heifers at a rate in excess of the recommended 0.8 kg/day during the period when heifers reach puberty may be detrimental to future milk production (18, 35, 41, 46). The allometric phase of mammary parenchymal growth occurs during the peripubertal period and appears to be dependent on somatotrophin and somatomedins. It is during this period that overfeeding causes a decrease in these hormones and mammary parenchymal growth is impaired (16, 17, 41).

1.4 Overall Objectives

Calf morbidity and mortality, heifer growth and age at first calving are important factors in the economics of raising dairy replacements, yet the interrelations amongst these variables and other calf-level variables have not been explored. Passive transfer of colostral immunoglobulins is beneficial in affording some protection against some of the

calfhood diseases, but more detailed relationships need to be defined, as do the relationship between passive transfer and growth. The overall objectives of this research were to undertake a prospective cohort study with well defined disease conditions and a large sample size to assess the above mentioned associations and relationships. The specific objectives of this study were as follows:

a) determine the effects of passive transfer of colostral immunoglobulins, birth weight and birth season on the incidence, severity and age of onset of the four calfhood diseases: diarrhea, septicemia, navel infection and pneumonia;
b) determine the associations amongst the four calfhood diseases listed above plus keratoconjunctivitis (pinkeye);
c) determine the effects of the five diseases on body growth as measured by weight gain and pelvic height gain during two growth periods: birth to 6 months, and 6 to 14 months.

1.5 References

1. AMIR S. The effect of age at first calving on subsequent milk yield of dairy cows. Agicultural Research Organization Special Publication No. 119, Division of Scientific Publications, the Volcani Center, Bet Dagen, Israel, 1978.

2. BACKSTROM L, HOEFLING DC, MORKOC AC, COWART RP. Effect of atrophic rhinitis on growth rate in Illinois swine herds. J Am Vet Med Assoc 1985; 187:712-715.

3. BARBER DML. Serum immune globulin status of purchased calves: An unreliable guide to viability and performance. Vet Rec 1978; 102:418-420.

4. BELKNAP EB, BAKER JC, PATTERSON JS, WALKER RD, HAINES DM, CLARK EG. The role of passive immunity in bovine respiratory syncytial virus infected calves. J Infect Dis 1991; 163:470-476.

5. BERNARDO TM, DOHOO IR, DONALD A. Effect of ascariasis and respiratory diseases on growth rates in swine. Can J Vet Res 1990; 54:278-284.

6. BORGSTEEDE FH, HENDRIKS J. Experimental infections with *Cooperia oncophora* (Railliet, 1918) in calves. Results of single infections with two graded dose levels of larvae. Parasitology 1979; 78:331-342.

7. BRADLEY JA, NIILO L. Immunoglobulin transfer and weight gains in suckled beef calves force-fed stored colostrum. Can J Comp Med 1985; 49:152-155.

8. CALDOW GL, WHITE DG, KELSEY M, PETERS AR, SOLLY HJ. Relationship of calf antibody status to disease and performance. Vet Rec 1988; 122:63-65.

9. CHASE L, OTTERBY D. Raise diary replacements economically. Dairy Herd Management 1986; 1:16-19.

10. CURTIS CR, ERB HN, WHITE ME. Risk factors for calfhood morbidity and mortality on New York dairy farms. Proc Cornell Nutrition Conf 1985; 90-99.

11. DAVIDSON JN, YANCEY SP, CAMPBELL SG, WARNER RG. Relationship between serum immunoglobulin values and incidence of respiratory disease in calves. J AM Vet Med Assoc 1981; 179:708-710.

12. DONOVAN GA, BRAUN, RK. Evaluation of dairy heifer replacement-rearing programs. Comp Contin Educ Pract Vet 1987; 9:F133-F139.

13. ERB HN, SMITH RD, OLTENACU PA, GUARD CL, HILLMAN RB, POWERS PA, SMITH MC, WHITE ME. Path model of reproductive disorders and performance, milk fever, mastitis, milk yield and culling in Holstein dairy cows. J Dairy Sci 1985; 68:3337.

14. FALLON RJ. The effect of immunoglobulin levels on calf performance and methods of artificially feeding colostrum to the newborn calf. Ann Rech Vet 1978; 9:347-352.

15. FALLON RJ, HARTE FJ, HARRINGTON D. The effect of calf purchase weight, serum IG level and feeding systems on the incidence of diarrhoea, respiratory disease and mortality. Bovine Practitioner 1987; 22:104-106.

16. GARDNER RW, SCHUH JD, VARGUS LG. Accelerated growth and early breeding of Holstein heifers. J Dairy Sci 1977; 60:1941-1948.

17. HARRISON RD, REYNOLDS IP, LITTLE W. A quantitative analysis of mammary glands of dairy heifers reared at different rates of live weight gain. J Dairy Res 1983; 50:405-412.

18. HEAD HH. Heifer performance standards: Rearing systems, growth rates and lactation. *In*: Van Horn HH, Wilcox CJ, eds. Large Dairy Herd Management. Champaign, Ill: American Dairy Science Association, 1992:422-433.

19. HEINRICHS AJ, HARGROVE GL. Standards of weight and height for Holstein heifers. J Dairy Sci 1987; 70:653-660.

20. HOWARD JC, CLARKE MC, BROWNLIE J. Protection against respiratory infection by bovine virus diarrhea virus by passively acquired immunity. Vet Microbiol 1989; 19:195-203.

21. HOFFMAN PC, FUNK DA. Applied dynamics of dairy replacement growth and management. J Dairy Sci 1992; 75:2504-2516.

22. JOHNSON JC, STEWART TB, HALE OM. Differential responses of Duroc, Hampshire, and crossbred pigs to a superimposed experimental infection with the intestinal threadworm, *Strongyloides ransomi*. J Parisitology 1975; 61:517-524.

23. KARREN DB, BASARAB JA, CHURCH TL. The growth and economic performance of preconditioned calves and their dams on the farm and of calves in the feedlot. Can J Anim Sci 1987; 67:327-336.

24. KEOWN JF. What New York dairymen are telling us about the freshening age of their first-calf heifers. Northeast Improver 1984; 3:24-25.

25. KEOWN JF, EVERETT RW. Effect of days carried calf, days dry, and weight of first calf heifers on yield. J Dairy Sci 1986; 69:1891-1897.

26. LEBLANC PH, BAKER JC, GRAY PR, ROBINSON NE, DERKSEN FJ. Effects of bovine respiratory syncytial virus on airway function in neonatal calves. Am J Vet Res 1991; 52:1401-1406.

27. LELAND SE, DAVIS GV, CALEY HK, ARNETT DW, RIDLEY RK. Economic value and course of infection after treatment of cattle having a low level of nematode parasitism. Am J Vet Res 1980; 41:623-633.

28. MARTIN SW, BATEMAN KG, SHEWEN PE, ROSENDAL S, BOHAC JE. The frequency, distribution and effects of antibodies to seven putative respiratory pathogens on respiratory disease and weight gain in feedlot calves in Ontario. Can J Vet Res 1989; 53:355-362.

29. MECHOR GD, ROUSSEAUX CG, RADOSTITS OM, BABIUK LA, PETRIE L. Protection of newborn calves against fatal multisystemic infectious bovine rhinotracheitis by feeding colostrum from vaccinated cows. Can J Vet Res 1987; 51:452-459.

30. NOCEK JE, BRAUND DG, WARNER RG. Influence of neonatal colostrum administration, immunoglobulin, and continued feeding of colostrum on calf gain, health, and serum protein. J Dairy Sci 1984; 67:319-333.

31. NOYES EP, FEENEY DA, PIJOAN C. Comparison of the effect of pneumonia during lifetime with pneumonia detected at slaughter on growth in swine. J Am Vet Med Assoc 1990; 197:1025-1029.

32. PARKER CF. The sheep industry: trends, economics and production aspects. Veterinary Clinics North America: Food Animal Practice 1986; 2 (2):329-333.

33. POWELL RL. Trend of age at first calving. J Dairy Sci 1985; 68:768-772.

34. PRITCHARD DG, CARPENTER CA, MORZARIA SP, HARKNESS JW, RICHARDS MS, BREWER JI. Effect of air filtration on respiratory disease in intensively housed veal calves. Vet Rec 1981; 109:5-9.

35. REMER Y. Economic evaluation of early breeding of dairy cattle heifers under farm conditions in Isreal. Proc 27 Ann Meeting European Assoc Anim Prod 1976; 56:1-14.

36. ROBISON JD, STOTT GH, DeNISE SK. Effects of passive immunity on growth and survival in the dairy heifer. J Dairy Sci 1988; 71:1283-1287.

37. SILVA HM, WILCOX CJ, SPURLOCK AH, MARTIN FG, BECKER RB. Factors affecting age at first parturition, life span, and vital statistics of Florida dairy cows. J Dairy Sci 1986; 69:470-476.

38. SIMERL NA, WILCOX CJ, THATCHER WW, MARTIN FG. Prepartum and peripartum reproductive performance of dairy heifers freshening at young ages. J Dairy Sci 1991; 74:1724-1729.

39. SLATER K, THROUP G. Dairy Farm Business Management. Ipswich: Farming Press Ltd, 1983.

40. SMITH T, LITTLE RB. The significance of colostrum to the new-born calf. J Exper Med 1922; 36:181-189.

41. SWANSON EW. Optimal growth patterns for dairy cattle. J Dairy Sci 1967; 50:244-252.

42. SYRSTAD O. Survival rate of dairy cows as influenced by herd production level, age at first calving, and sire. Acta Agric Scand 1979; 29:42-44.

43. THOMAS LH, SWANN RG. Influence of colostrum on the incidence of calf pneumonia. Vet Rec 1973; 92:454-455.

44. THOMPSON JR, POLLAK EJ, PELISSIER CL. Interrelationships of parturition problems, production of subsequent lactation, reproduction and age at first calving. J Dairy Sci 1983; 66:1119-1127.

45. WALTNER-TOEWS D, MARTIN SW, MEEK AH. Dairy calf management, morbidity and mortality in Ontario Holstein herds. III. Association of management with morbidity. Prev Vet Med 1986; 4:137-158.

46. YOUNG CW, EIDMAN VR, RENEAU JK. Animal health and management and their impact on economic efficiency. J Dairy Sci 1985; 68:1593-1602.

47. WILLETT GS, THOMASON E, BERNARD J. What it costs to raise dairy heifers. Hoard's Dairyman 1984; 129:1257.

48. WILSON MR, TAKOV R, FRIENDSHIP RM, MARTIN SW, McMILLAN I, HACKER RR, SWAMINATHAN S. Prevalence of respiratory diseases and their association with growth rate and space in randomly selected swine herds. Can J Vet Res 1986; 50:209-216.

CHAPTER 2

ASSOCIATIONS BETWEEN PASSIVE IMMUNITY, AND MORBIDITY AND MORTALITY IN DAIRY CALVES

2.1 Introduction

Successful dairy replacement rearing is dependent upon a multitude of complex, interrelated factors of which colostrum management is but one. The neonatal calf, which is born with little or no humoral immunity, is totally dependent upon absorption of colostrally derived immunoglobulins for its early disease resistance (16,62). A 2- to 4-fold increase in mortality in calves with failure of passive transfer (FPT) compared to those that have received adequate amounts of colostral immunoglobulins has been reported (6, 9, 10, 18, 29, 36, 38, 39, 52). Only two researchers in Great Britian have failed to find this protective association (2, 10). The results in these last 2 studies may have been due to the very low mortality rate (10) or small population size (2).

The protective effects of colostrum in relation to the incidence and severity of neonatal septicemia and early calfhood pneumonia is well established (3, 10, 13, 18, 27, 40, 61). Davidson et al. (13) found that not only was respiratory disease risk reduced and fewer treatment days required in colostrum satisfied calves but onset of disease was delayed by 5 to 7 days - from 14 to 16 days in calves with FPT to 21 to 22 days in those receiving adequate amounts of colostral immunoglobulin. The authors imply that the older the

calf is when it first gets diseased, the better are its chances of survival.

With respect to neonatal calf diarrhea, the preventive effects of passive immunity are less clear. Results of epidemiological and experimental studies are divided between those showing no effect (10, 24, 38) and those demonstrating a reduction in incidence and severity of diarrhea and/or a decrease in mortality attributable to enteritis (7, 18, 46). These discrepencies can be explained mainly by differences in the primary causative organism involved. Diarrhea caused by *E coli* can be effectively controlled by feeding the neonate colostrum containing antibodies directed specifically at the enterotoxigenic form (7, 35). Colostral antibodies are not as efficacious against enteridities caused by *Salmonella* sp. and viruses and protozoa (1, 24, 54), probably due to the later age at which these disease problems occur (1, 10, 24, 54). Promising results have been seen when colostrum is fed throughout the period of risk (up to 21 days-of-age). Unfortunately, in most commercial calf rearing systems continued feeding of high quality colostrum for this length of time is not practical.

Investigations into the factors that are associated with the ability of the neonatal calf to absorb the immunoglobulins show that the mass of immunoglobulin fed and the time after birth at which it is fed are the two most important predictors of passive immune status in the calf (14, 37, 42, 46, 50, 60). Still, these factors only account for approximately 20% of the variation in calf serum immunoglobulin concentration (14). Other factors identified as important, but of lesser significance, include presence of the dam at

colostrum feeding (17, 58), method of colostrum feeding (17, 21, 44), acid-base status of the calf (5), environment or season of birth (15, 20, 38, 43, 56), stress (59), parity of the dam (14, 15), dystocia (14, 57) and genetics (15, 22, 48).

The most commonly used methods for assessing passive transfer status in the calf are radial immunodiffusion (RID), zinc sulfate turbidity (ZST) and serum total protein (TP) determined by refractometry. The RID is considered the gold standard against which all others are measured (11). Calves with serum IgG1 concentrations of less than 10 mg/ml are considered colostrum deprived. Serum total protein concentration is highly correlated with immunoglobulin concentrations measured using RID ($r=0.88$) (45). The sensitivity and specificity of TP are dependent upon the cutoff used to define failure of passive transfer and adequate passive transfer (APT). Using a cutoff for TP of <5.3 g/dl indicating FPT, Curtis et al. (11) found a sensitivity and specificity of 0.68 and 0.94, respectively. In the author's lab, sensitivity and specificity with FPT set at TP <5.4 was found to be 0.85 and 0.82.

Braun and Tennant (8) categorized calves into three groups based upon risk of early calfhood diseases; FPT was evident when TP was less than 5.0 g/dl and serum total protein concentrations greater than 5.4 g/dl indicated APT. The range from 5.0-5.4 g/dl was an intermediate range with moderately increased risk of neonatal disease.

Farm and/or calf level factors that are associated with mortality include farm size, calf

housing and season or birth. Studies in Michigan (49), Virginia (30) and Ontario (66) indicated a positive relationship between herd size and calf mortality, whereas Jenny, et al. (31) in South Carolina found the opposite to be true. The former finding is often explained as primarily being due to a dilution of management and inadequate calf rearing facilities as farms gradually grew from being a "small" farm to a "large" farm. In the South Carolina study, herd milk and fat production was found to be higher in the larger herds with the implication that herds with higher productivity are better managed and thus have lower calf mortality.

Seasonal trends in mortality have been reported from many different geographic locations and climates (8, 43, 56, 65, 66). Some of this variation can be explained by the seasonal variation in calf serum immunoglobulin levels and its subsequent effects on calf health. The remainder is due to direct environmental influences on the calf such as extremes on cold, heat or humidity plus unknown variables.

The method of housing calves, especially during the milk feeding period, is an important determinant in calf health. Not all studies are in agreement in this area but subjective assessment of most dairy veterinarians is that hutch housing is superior to individual penning of calves which in turn is better than group rearing. Only a large scale study in Ontario by Waltner-Toews, et al. (65, 66) substantiated this objective assessment.

The objective of this prospective cohort study was to define the associations between

passive immune status, as determined by serum total protein, and morbidity and mortality during the first six months of life. A secondary objective was to provide descriptive epidemiologic data of morbidity and mortality in a large population of dairy calves.

2.2 Materials and Methods

2.2.1 Study population

The animals used for this investigation were Holstein heifer calves from a convenience sample of two large dairy farms in Florida. One farm (Barn 1), in north central Florida, consisted of approximately 3000 adult animals and 2000 replacements. All cows were managed as one herd and milked through one large parlour. The second farm was located in south central Florida and had approximately 6000 adult cows and 5000 replacement animals. This latter farm was operated as 5 distinct and separate units, each under separate and independent management regimes. Four of these units were comprised either exclusively, or predominantly, of Holstein cows (study Barns 2, 3, 4 and 5) and one was exclusively Jersey cattle. Calves from this last unit were not included in the study.

All heifer calves born from January 16, 1991 through January 15, 1992 were enrolled in the study. This cohort was used for descriptive epidemiology but only those that lived longer than 48 h and had serum total protein concentration determined were used for

statistical analysis.

2.2.2 Dry cow and colostrum management

Dry cow and colostrum management was essentially the same in each of the 5 barns. At approximately 14 to 21 days prepartum, dry cows were moved to a maternity dry cow lot in which they were to calve. These lots were large pastures with artificial and/or natural shade provided. Cows were monitored at least every 2 hours during the day and every 3 to 4 hours at night and calving difficulty was dealt with promptly in a prescribed manner. Calves born between 6:00 and 18:00 were fed 2 to 3 L of colostrum via esophageal tube feeder within 3 to 4 h of birth. Calves born at night (18:00 to 6:00) were fed 2 L within 4 to 6 h of birth and another 1 to 2 L of colostrum 6 to 8 h later. Calves were picked up from the maternity lot twice daily (8:00 and 18:00) and taken to the calf rearing area of the farm.

Colostrum was collected in a similar manner in all barns. Cows were milked within 12 hours of parturition into individual cans and the quality determined shortly after milk harvest using a colostrometer and a variation of the methods of Fleenor and Stott (19). The temperature of the colostrum when tested for quality was 30-35°C, not 21°C as described by the above authors. Measuring at this temperature would tend to underestimate the true Ig content of the colostrum (41). Only colostrum with colostrometer Ig readings of greater than 50 mg/ml was used for feeding of calves during

the first 24 h. Good quality colostrum was refrigerated at 4°C for a maximum of 4 days. Excess colostrum was stored frozen (-20°C) at Barn 1 only.

2.2.3 Calf housing and management

All Barn 1 calves were housed in 2.5m x 1.5m calf hutches. The farm in south Florida had a central calf rearing facility so calves from Barns 2-5 were housed together in either hutches or an open-sided calf barn with elevated stalls.

Milk and grain feeding practices, vaccination programs and deworming protocols were similar between the two farms (5 barns).

2.2.4 Birth data collection

All birth data were collected within the first 7 days of life with the exception of serum total protein concentration (see 2.2.6 - Serum total protein). Birth weight was obtained using a platform scale (Terraillon, Versailles, France) accurate to ± 0.9 kg. Concerns over the accuracy of obtaining weights on some calves 5 to 7 days after birth versus getting weights within 4 days of birth were addressed by determining the difference between weights taken on a group of 58 calves at 0 to 4 days and again at 5 to 7 days. The mean change in calf weight from the first half to the second half of the first week was +0.75 kg. This value was used to adjust all birth weights to the 0 to 4 day time

period. Details of this analysis are included in Appendix B.

2.2.5 Health and culling data collection

Calf morbidity, mortality and culling information were collected and recorded on a daily basis using on-farm database management software (Barn 1 - VisiCow•, Haas Chemical Co, Mobile, AL, USA; Barns 2-5 - DBase III•, Aston-Tate, Torrance, CA, USA). Morbidity data recorded included date of disease event, diagnosis made by trained farm personnel (see 2.2.7 - Definitions of diseases) and treatment. Date and reason for death or disposal were recorded for deaths and culls.

2.2.6 Serum total protein

Blood samples were collected via jugular venipuncture into evacuated tubes (Vacutainer•, Becton Dickinson and Company, Rutherford, NJ, USA) between 2 and 8 days-of-age. Serum Ig does not peak until 24 to 36 h after colostrum ingestion (28, 34, 59) and stable levels are maintained for at least 8 days (15). Blood was placed on ice (4 to 8° C) immediately after collection and serum was separated within 24 h by centrifugation at 3000 g for 20 minutes. Serum total protein was determined using a refractometer (AO Scientific Instruments, Buffalo, NY, USA) as described by Reid and Martinez (51). The refractometer was calibrated monthly using sterile deionized water and known standards ranging from 3.0 to 9.0 mg/dl.

2.2.7 Disease definitions

Farm personnel were trained to recognize and treat the most common calfhood diseases. The four disease conditions of interest, diarrhea, septicemia, omphalitis and pneumonia, were chosen by the investigator because they represent the most commonly diagnosed diseases in dairy calves. An incident of disease was considered when the criteria for the disease, as described in Table I, were met. If a calf was retreated for diarrhea within 4 days of the end of the previous treatment, it was considered the same "case". Otherwise, it was considered a new case. In considering the three other diseases, retreatment within 14 days for the same condition was considered the same case of disease.

Diagnostic skills of farm personnel were monitored closely by the investigator and feedback was provided by assisting in diagnosis and treatment on a biweekly basis and via confirmation of diagnoses by performing necropsies on approximately 15% of calf deaths.

2.2.8 Data handling and storage

Data were categorized as birth data, that which was collected upon assignment of the calf to the study, or health/culling data which were retrieved from on-farm computer databases monthly. These data were maintained in a spreadsheet program (Quattro Pro®,

Table I Definition of calf diseases diagnosed by farm personnel

Disease	Diagnostic Definition	Analysis Definition
Diarrhea (Scours)	Diarrhea +/- dehydration requiring treatment with diarrhea specific products (kaolin-pectate, neomycin, etc.) and/or electrolytes	Treated for 2 or more consecutive days or for a total of 3 or more treatment days (unless died on second day)
Septicemia	Weak calf, off feed, depressed, +/- fever, +/- diarrhea; less than 30 days-of-age; treat with recommended anti-biotics +/- electrolytes	Calf less than 30 days-of-age at start of treatment and treated with recommended antibiotics at least 2 consecutive days (unless died on second day); calves treated for navel infection that died were recorded as septicemia
Navel Infection	Navel swollen or has abnormal discharge, no fever or other systemic signs; all navels are checked at 2-4 days of age; treat with recommended anti-biotics +/- local therapy	Treated for 2 or more consecutive days or for a total of 3 or more treatment days (if died, coded as septicemia)
Pneumonia (Respiratory)	Weak calf, +/- off feed, laboured breathing, +/- nasal discharge, fever; greater than 29 days-of-age; treat with recommended anti-biotics +/- electrolytes	Calf greater than 29 days-of-age at first treatment and treated with recommended antibiotics at least 2 consecutive days (unless died on second day)

Borland International Inc., Scotts Valley, CA, USA). Data were entered by one person and proofed/validated by one of two other people.

A subset of the on-farm health and culling data input records (original paper records from daily log) was compared to the computer database. Minimal input errors were found.

2.2.9 Definition of seasons

There are only two seasons of concern in the sub-tropical climate of Florida, summer and winter. Meteorological data for 1991 was collected at two University of Florida, Institute of Food and Agricultural Sciences Agricultural Research Stations, one each within 80 km of the two study sites. Summer was defined as beginning when the mean daily temperature for a 15 day period was above 25°C, which is the upper thermoneutral temperature, or comfort temperature, for dairy cattle and calves (53). May 7 to Sep 23, and April 23 to Oct 7 were the summer seasons for Barn 1 and Barns 2 to 5, respectively. Details of the season determination appear in Appendix C.

2.2.10 Statistical analysis

All analyses were performed using SAS® statistical software (SAS Institute Inc, Cary,

NC, USA). Descriptive statistics and residual diagnostics were performed using Proc Freq and Proc Univariate procedures. Categorical response variables (morbidity and mortality) were analysed for differences in occurrence of disease using multivariable logistic regression (Proc Logistic) and for differences in time of onset and proportional hazards using life table analysis (Proc Lifetest) and Cox proportional hazards survival analysis (Proc Phreg). The continuous variable, disease specific treatment days, was analysed using multiple linear regression and analysis of covariance (Proc Glm).

Variables considered for inclusion in the models are listed in Table II. Because of the small number of possible predictors per model, all possible combinations of variables were evaluated and remained in the model if Log likelihood χ^2 test or the partial F test or multiple partial F test was significant. The predetermined acceptable level of significance was set at $P < 0.05$ in all models. A second order polynomial of serum total protein was considered in each model after evaluation of the relationship between mortality and 0.5 g/dl groupings of TP from 4.0 to 8.0 g/dl demonstrated a decreasing curvilinear response. Multicollinearity between TP and TP^2 was reduced when necessary by use of the centering technique described by Glantz (23). Interaction was evaluated using cross product terms of variables that were found to be significant predictors in the full model. Confounding was considered a problem when inclusion of a variable in the model changed the parameter estimate by +/-50%.

The Hosmer-Lemeshow goodness-of-fit χ^2 statistic was used to assess the fit of logistic

Table II Variables considered for inclusion in logistic and linear regression, life tables analysis and survival analysis

Variable Name	Variable Description
TP	Serum total protein (continuous variable, range 4.0 - 8.0 g/dl)
TP^2	Quadratic term for TP
Barn	Class variable (1 - 5) for barn
Season	Dichotomous variable for season of birth (1=summer, 0=winter)
W0	Birth weight (continuous variable, range 15 - 60 kg)
NUMDIA	Treatment days for diarrhea (continuous variable, range 0 - 22 days)
DIA	Dichotomous variable for diarrhea (0=not treated for diarrhea, 1=treated for diarrhea)
NUMSEP	Treatment days for septicemia (continuous variable, range 0 - 31 days)
SEP	Dichotomous variable for septicemia (0=not treated for septicemia, 1=treated for septicemia)
NUMPNU	Treatment days for pneumonia (continuous variable, range 0 - 32 days)
PNU	Dichotomous variable for pneumonia (0=not treated for pneumonia, 1=treated for pneumonia)
NUMNAV	Treatment days for navel infection (continuous variable, range 0 - 17 days)
NAV	Dichotomous variable for navel infection (0=not treated for navel infection, 1=treated for navel infection)

regression models. This was accomplished by outputting from SAS the observed and predicted responses and performing the chi-square calculations on deciles of predicted response as described by Hosmer and Lemeshow (26). Linear regression diagnostics employed included plots of studentized residuals against predicted values and normal plot of residuals.

Intra herd correlation coefficients were calculated for each response variable using the methods decribed by Snedecor and Cochran (55).

2.3 Results

2.3.1 Descriptive epidemiology

During the year-long assignment period, 3287 calves were born of which 3103 had complete morbidity, mortality, birth weight and serum total protein data. Four hundred and thirteen calves died during the first 6 months of life. Septicemia was the major disease specific cause of death (Table III). Over fifty percent of mortality to which a cause could not be attributed, ie. cause of death was listed as unknown, occurred during the first 4 to 5 d of life. Detailed descriptive epidemiology of disease specific mortality and age specific mortality are provided in Appendix D.

There were no significant differences in monthly mean serum total protein concentrations

Table III Descriptive epidemiology of mortality in a cohort of calves born on 2 large dairy farms in Florida in 1991.

	n=	%[a]
Total calves born alive	3287	
Calves living >48 hr	3253	
Calves with complete data	3103	95.4
Calf mortality before 180 days	379	11.7
Calves culled before 180 days	142	4.4
Disease Specific Cause of Death	**n=**	**% of deaths**
Diarrhea	38	10.0
Septicemia	210	55.4
Pneumonia	83	21.9
Other	7	1.8
Unknown	41	10.8
Calves born per barn	**Barn #**	**# calves**
	1	1134
	2	726
	3	734
	4	551
	5	142
Calves born per season	Summer	1314
	Winter	1973

[a] Calculated using calves living >48 h as denominator

or the proportion of calves born with failure of passive transfer (TP < 5.0 g/dl) in the two seasons.

Morbidity and mortality generally decreased as the study progressed with a much lower mortality being evident during the last 6 months than during the first (Figure 3).

The intraherd correlation coefficients for each of the outcome variables ranged from 0.04 to 0.16 (Appendix E).

2.3.2 Mortality

Failure of passive transfer is the true risk factor of interest but since TP ia s good surrogate measure of FTP, it will be described as the risk factor. The logistic regression model of factors associated with mortality is given in Table IV. Serum TP is a significant risk factor contributing to mortality. Since the risk of mortality was not linear with respect to TP the higher order quadratic function of TP was required. This non-linear protective effect of absorbed colostral immunoglobulins can be seen in Figure 4. The Hosmer-Lemeshow goodness-of-fit test for this model was 9.41 (with 8 degrees of freedom, $P > 0.25$) which indicates that there was no reason to suspect that this model did not adequately fit the data.

Proportional hazards survival analysis (Table V) also demonstrated that serum total

Figure 3 Mortality risk for Holstein dairy calves by birth-month cohort

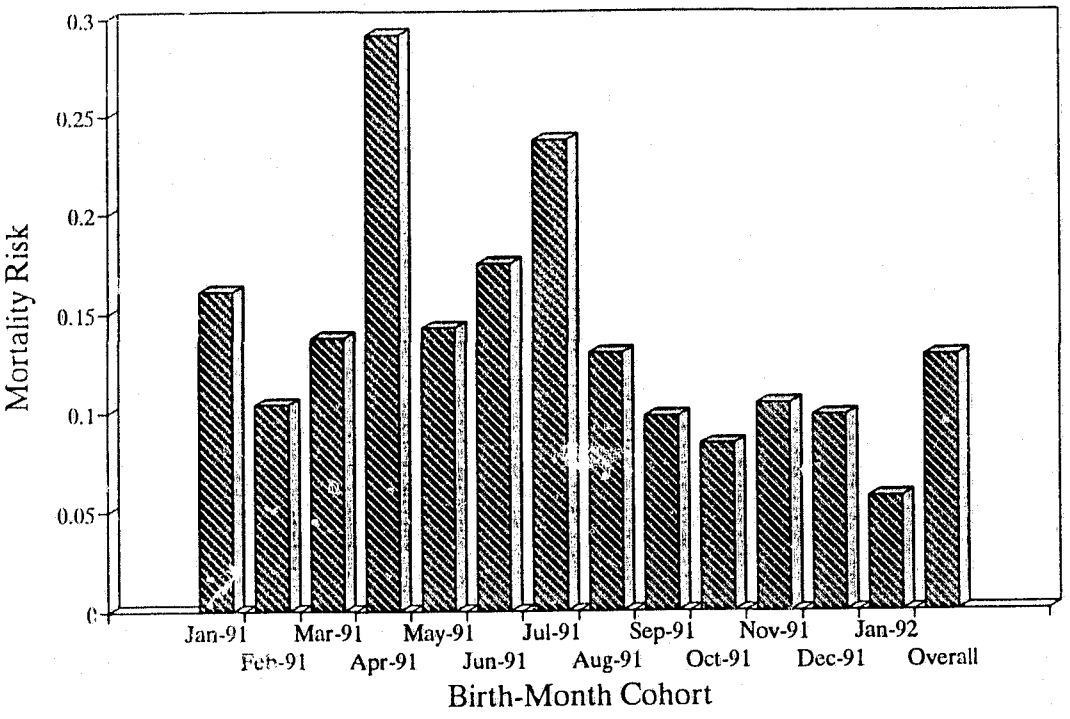


Table IV Logistic regression model of the effect of serum total protein on calf mortality through the first six months of age[a]

Variable	Parameter Estimate	Odds Ratio	*P-value*
Intercept	8.2743		0.0013
TP	-3.1333	[b]	0.0006
TP^2	0.2303		0.0041
Barn 1	-0.0000	1.00	.
Barn 2	-0.0583	0.94	0.7041
Barn 3	-0.5355	0.59	0.0015
Barn 4	-0.5705	0.57	0.0026
Barn 5	0.4323	1.54	0.0857

[a] Hosmer-Lemeshow goodness-of-fit statistic = 9.41 ($P>0.25$)

[b] Odds Ratio of population with TP(g/dl)=4.0, 4.5, 5.0, 5.5 and 6.0 versus referent population with TP=6.5 are 5.98, 3.32, 2.07, 1.45 and 1.14 respectively.

Figure 4 The effects of serum total protein on mortality risk; actual total protein cohort mortality versus logistic regression estimate from model presented in Table IV

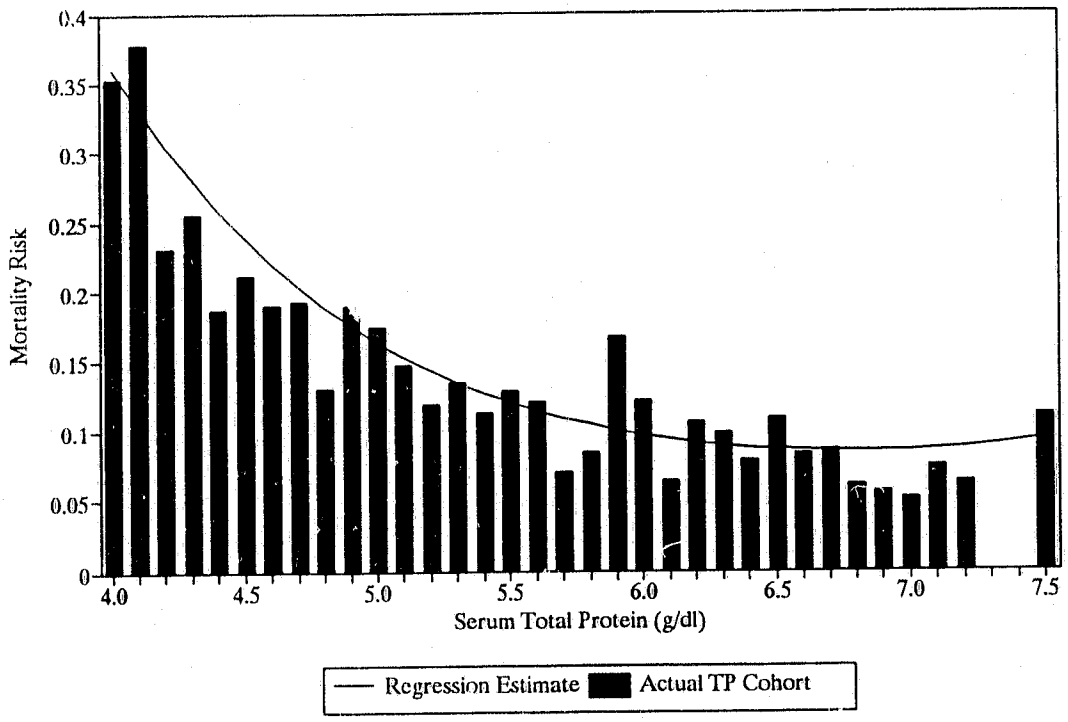


Table V Cox proportional hazards model of the association between serum total protein and calf mortality through the first six months of age[a]

Variable	Parameter Estimate	Risk Ratio	*P-value*
TP	-2.6982	[b]	0.0006
TP^2	0.1978		0.0041
Barn 1	0.0000	1.00	.
Barn 2	-0.1807	0.84	0.7041
Barn 3	-0.5819	0.60	0.0015
Barn 4	-0.6556	0.52	0.0026
Barn 5	0.2707	1.31	0.0857

[a] The Log likelihood χ^2 of inclusion of "time-dependent" covariates (i.e. interaction term between time and TP, and time and TP^2) was not significant (P=0.22), indicating that the assumption of constant proportional hazards over time was appropriate.

[b] The risk ratio of population with TP(g/dl)=4.0, 4.5, 5.0, 5.5 and 6.0 versus referent population with TP=6.5 are 4.73, 2.84, 1.89, 1.38 and 1.12 respectively.

protein was a significant risk factor for mortality. The increased risk of mortality associated with low TP was not only significant, the relative risk was also found to be constant over time through 160 days of age (see Appendix F for presentation of this relationship). Results from the lifetable analysis of the effect of TP (using the three serum total protein groupings described earlier) are presented in Figure 5.

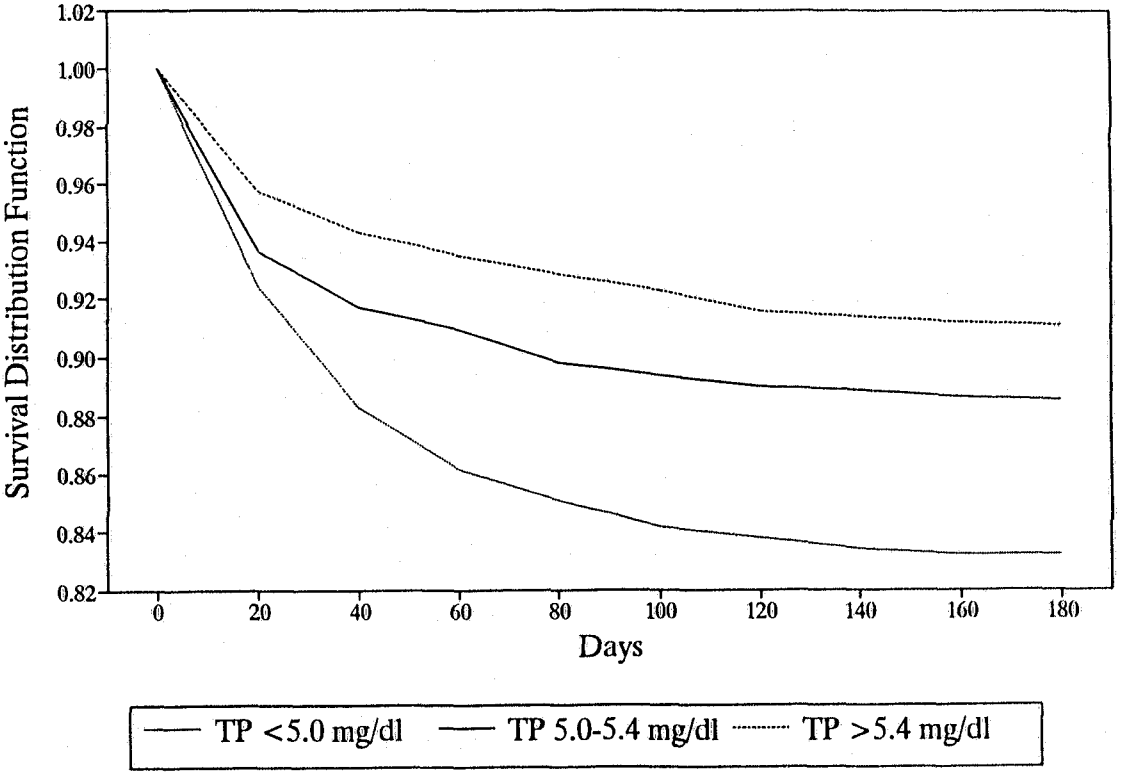
2.3.2 Morbidity

Nearly all calves experienced some degree of diarrhea during the first 15 to 20 days of life. Only those that were treated with supportive therapy, electrolytes $\pm$ antibiotics and anti-inflammatory agents, were included in the analysis.

Serum concentration of absorbed colostral antibodies was not associated with incidence, onset of occurrence or severity of diarrhea or omphalitis in these calves. However, the above mentioned disease measures for septicemia and pneumonia during the first 6 months were strongly associated with serum total protein (Table VI).

The logistic regression models of the association between TP and septicemia and TP and pneumonia are exhibited in Tables VII and VIII. The protective effect of TP on septicemia was a linear relationship but was seen mainly during the summer. Odds ratios during the summer months were 4.9 to 1.9 in animals with TP between 4.0 and 5.5 g/dl when compared to the reference population with serum total protein of 6.5 g/dl. The

Figure 5 The effects of serum total protein (using three discrete categories[a]) on survival distribution function using life tables analysis



[a] Braun and Tennant (8)

Table VI Statistical significance of the association of serum total protein with 4 calfhood diseases as measured by occurrence (yes/no), time of onset and duration of treatment

Parameter	Disease			
	Diarrhea	Septicemia	Navel Ill	Pneumonia
Logistic regression on occurrence of disease[a]	NS	0.0001	NS	0.0001
Survival Analysis[a]	NS	0.0001	NS	0.0001
Duration of treatment[b]	NS	0.0025	NS	0.0243

[a] *P-values* determined from likelihood ratio Chi-square test of the addition of TP or $TP+TP^2$ to the models.

[b] *P-values* determined from partial F test of the addition of TP or $TP+TP^2$ to the models.

Table VII Logistic regression model of the relationship between serum total protein and the occurrence of septicemia in Holstein dairy calves[a]

Variable	Parameter Estimate	*P-value*
Intercept	0.2235	0.7251
TP[b]	-0.1864	0.0173
Barn 1	0.0000	.
Barn 2	1.3798	0.0001
Barn 3	1.3325	0.0001
Barn 4	1.5752	0.0001
Barn 5	2.1144	0.0001
Season	0.6520	0.5487
Birth Weight	-0.0342	0.0029
Interaction terms		
TP*Season	-0.4484	0.0023
Barn 1*Season	-0.0000	.
Barn 2*Season	-0.3779	0.1480
Barn 3*Season	-0.7302	0.0079
Barn 4*Season	-1.1761	0.0001
Barn 5*Season	-0.4958	0.2585
Birth WT*Season	0.0508	0.0060

[a] Hosmer-Lemeshow goodness-of-fit statistic = 6.215 (P>0.50)

Odds Ratios of populations with varying serum total protein concentration in the two seasons (referent population TP=6.5 g/dl).

	Odds Ratio	
TP (g/dl)	Winter	Summer
4.0	1.59	4.89
4.5	1.45	3.56
5.0	1.32	2.59
5.5	1.20	1.89
6.0	1.10	1.37
6.5	1.00	1.00

Table VIII Logistic regression model of the relationship between serum total protein and the incidence risk of pneumonia to 6 months of age in Holstein dairy calves[a]

Variable	Parameter Estimate	*P-value*
Intercept	6.7003	0.0017
TP[b]	-2.3238	0.0018
TP^2	0.1744	0.0072
Barn 1	0.0000	.
Barn 2	-0.9105	0.0001
Barn 3	-0.7903	0.0001
Barn 4	-0.9100	0.0001
Barn 5	-0.0581	0.8165
Season	-0.6572	0.0001
Interaction terms		
Barn 1*Season	0.0000	.
Barn 2*Season	0.5851	0.0327
Barn 3*Season	0.5789	0.0282
Barn 4*Season	0.3145	0.2989
Barn 5*Season	0.9576	0.0312

[a] Hosmer-Lemeshow goodness-of-fit statistic = 3.839 ($P > 0.50$)

Odds Ratio of population with TP(g/dl)=4.0, 4.5, 5.0, 5.5 and 6.0 versus referent population with TP=6.5 are 3.42, 2.25, 1.61, 1.26 and 1.07 respectively.

same pattern of increased risk at lower serum total protein concentrations is seen in the pneumonia model, although the odds ratios are somewhat lower and the relationship was quadratic in nature.

The hazard ratios of developing septicemia are constant over the time interval measured, birth to 45 days of age (Table IX). As in the logistic model, survival analysis shows significant seasonal interaction. Similarly, the survival analysis model for the onset of pneumonia shows that FPT is a significant risk factor for earlier development of the disease when compared to calves receiving adequate colostral antibodies (Table X).

The number of treatment days for animals that had septicemia or pneumonia was significantly greater for those with lower TP, although the regression models accounted for a very small proportion of the variation in treatment days needed (Table XI). On average, reduction of 1 g/dl in TP only resulted in an extra 3/4 of a treatment day for each condition and the R^2 for the regression model was only 3.6 and 4.9% for septicemia and pneumonia respectively.

2.4 Discussion

2.4.1 Analyses

Three separate measures of disease were evaluated using the analyses described. Logistic

Table IX Cox proportional hazards model of the relationship between serum total protein and septicemia[a]

Variable	Parameter Estimate	*P-value*
TP[b]	-0.1611	0.0117
Barn 1	0.0000	.
Barn 2	1.2077	0.0001
Barn 3	1.1602	0.0001
Barn 4	1.3447	0.0001
Barn 5	1.7099	0.0001
Season	0.8588	0.3673
Birth Weight	-0.0289	0.0025
Interaction terms		
TP*Season	-0.4614	0.0004
Barn 1*Season	0.0000	.
Barn 2*Season	-0.1926	0.4163
Barn 3*Season	-0.5123	0.0411
Barn 4*Season	-0.9035	0.0006
Barn 5*Season	-0.2812	0.4168
Birth WT*Season	0.0442	0.0057

[a] Inclusion of "time-dependent" covariate (i.e. interaction term between time and TP) was not significant (P=0.33), indicating the assumption of constant proportional hazards over time was appropriate.

Risk Ratios of populations with varying serum total protein concentration in the two seasons (referent population TP=6.5 g/dl).

	Odds Ratio	
TP (g/dl)	Winter	Summer
4.0	1.50	4.74
4.5	1.38	3.47
5.0	1.27	2.54
5.5	1.17	1.86
6.0	1.08	1.37
6.5	1.00	1.00

Table X Cox proportional hazards model of the relationship between serum total protein and pneumonia during the first 6 months of age[a]

Variable	Parameter Estimate	*P-value*
TP[b]	-14.7073	0.0158
TP^2	1.0776	0.0434
Barn 1	0.0000	.
Barn 2	-0.6924	0.0001
Barn 3	-0.5739	0.0001
Barn 4	-0.6524	0.0001
Barn 5	0.2226	0.2745
Season	-0.5321	0.0001
Interaction terms		
Barn 1*Season	0.0000	.
Barn 2*Season	0.4786	0.0527
Barn 3*Season	0.4787	0.0412
Barn 4*Season	0.2143	0.4358
Barn 5*Season	0.8305	0.0187
TP*(log(CENSORPNU[b]))	3.2084	0.0338
TP^2*(log(CENSORPNU))	-0.2350	0.0749

[a] Log likelihood χ^2 of inclusion of "time-dependent" covariates (i.e. interaction term between time and TP, and time and TP^2) was significant ($P < 0.05$), indicating hazards ratios were not constant over time.

[b] CENSORPNU = Age of calf at initial diagnosis of pneumonia; if age > 180 or calf did not develop pneumonia then CENSORPNU = 180.

Risk Ratios of populations at varying ages in the two serum protein groups (referent population TP=6.5 g/dl).

	Risk Ratios	
Age (days)	TP=4.5 g/dl	TP=5.5 g/dl
60	1.83	1.20
80	1.28	1.08
100	0.97	0.99
120	0.77	0.92

Table XI Linear regression models of the relationship between serum total protein and treatment days required for septicemia and pneumonia

Outcome variable = Ln (# days treated for septicemia)

Variable	Parameter Estimate	*P-value*
Intercept	2.08070	0.0001
TP	-0.10335	0.0013
Barn 1	0.0000	.
Barn 2	0.1053	0.1121
Barn 3	0.1203	0.0736
Barn 4	0.1936	0.0062
Barn 5	0.3070	0.0006
R^2 = 0.0364		

Outcome variable = Ln (# days treated for pneumonia)

Variable	Parameter Estimate	*P-value*
Intercept	2.1754	0.0001
TP	-0.0996	0.0234
Barn 1	0.0000	.
Barn 2	-0.3461	0.0001
Barn 3	-0.2998	0.0002
Barn 4	-0.1932	0.0497
Barn 5	-0.0986	0.4157
Season	0.1340	0.0343
R^2 = 0.0491		

regression was used to determine the relationship between serum total protein and mortality and overall disease ocurrence. Time to onset of the various conditions and the assumption of constant risk over time were evaluated using lifetable and survival analyses. Linear regression was used to determine if the level of total protein influenced the severity of disease as measured by the duration of treatment. The last analysis was done using all calves treated for a specific disease condition and with only those that survived the disease. We had postulated that two separate models would evolve. Calves that die, do so early in the course of the disease and thus number of days treated would not be a good measure of severity in those calves. In fact, the parameter estimates in the two models were nearly identical.

The fact that there was some evidence of "clustering" of the data (intra-herd correlation coefficients between 0.04 and 0.16), the standard errors of the coefficients in the logistic regression models may have been underestimated. However, for those conditions in which TP was associated with the dependent variable (ie. mortality, septicemia and pneumonia), the actual P value of TP/TP^2 was always less than 0.005. Consequently, it is unlikely that clustering resulted in any spuriously significant observations.

2.4.2 Mortality

Mortality rates of calves in this study were similar to those reported previously (25, 30, 31, 53, 61). The tendency for mortality to decrease as the study progressed could be

explained in a number of ways. The "healthy farmer effect" that is described by Curtis, et al. (12) is the most likely. In this scenario, the farmer becomes more aware of the subject area and becomes a better manager because of data collection. Also, an improvement in the calf's environment could be a contributing factor as there was a higher mortality rate in the summer compared to the winter season.

The data from this study provide strong evidence that serum TP concentration is a good surrogate measure for passive transfer status in the neonatal calf. Calves with low TP values (<5.0 g/dl) are 2 to 4 times more likely to die within the first six months of life than those with serum total protein concentrations of >6.0 g/dl. This is in agreement with mortality rate ratios estimated from most of the current literature (6, 9, 10, 18, 29, 36, 38, 39, 52). The sample size of this study allows for more detailed assessment of the shape of the association between serum total protein and mortality. The quadratic relationship of TP to mortality shows a dramatic decrease in mortality as TP values increase from 4.0 to 5.0 g/dl, a small improvement from 5.0 to 6.0 g/dl and virtually no difference in mortality rates in calves as serum TP increases over 6.0 g/dl (Figure 4).

Evaluation of possible interactions among predictor variables is also a benefit of large sample size. In the mortality models, no significant interactions were found. The effect of TP on mortality was constant across barns, seasons and birth weight which allows for valid extrapolation of this inference to other populations of calves.

For a variety of reasons, serum total protein was not determined in 121 calves. These calves were evenly distributed between barns. Thirty-seven (30.6%) of these calves died within 60 days of birth. A selection bias may have existed if these calves had a different distribution of TP values than calves included in the study. However, given the relatively small number of calves, any bias present was probably very small.

The fact that the mortality hazard ratio was constant throughout the period from birth through 6 months (i.e. the relative risk of mortality was constant (2 to 3) during each 20 day period) was suprising. It has always been felt that colostral protection was evident only early in life, and therefore most research efforts were concentrated in the preweaning to 3 month time frame. Close examination of data presented by Robison et al. (52) reveals that in a study with relatively low mortality, calves with serum Ig concentration of less than 18 mg/ml when compared to those with Ig concentrations greater than 18 mg/ml had crude relative risks of mortality in the age ranges of 0-35 d, 36-70 d, 71-105 d and 106-180 d of 1.28, 3.44, 1.52 and 3.29 respectively. However, other calf and herd level factors that may have confounded these results were not noted in the paper.

Even with high apparent levels of absorbed colostral immunoglobulin mortality risk was close to 10% (Figure 4). This emphasizes that other factors are involved with calf mortality besides level of humoral immunity. Colostrum does not completely overcome poor environmental or nutritional management. This may explain why some calves with

adequate passive transfer get sick and die, and conversely, why many well managed calves that have failure of passive transfer survive and thrive.

2.4.3 Morbidity

2.4.3.1 Diarrhea

The lack of a significant association between TP and diarrhea could be expected. These farms practiced a sound *E coli* vaccination program resulting in rare clinical or laboratory diagnosis of enteritis caused by *E coli*. The primary enteric pathogens on these farms are rotavirus, coronavirus, cryptosporidia and *Salmonella* sp. Systemically absorbed colostral Ig does not effectively prevent enteritis caused by these agents (1, 10, 24, 54).

2.4.3.2 Septicemia

Colostral protection of the calf from septicemia shown here agrees with other studies (10, 38, 39). The relationship between TP and the occurrence of septicemia is linear. There was a seasonal interaction, increased risk in the summer, that can best be explained by the environment into which the calf is born. Coincident with the summer season in Florida is a significant increase in precipitation (Appendix B) which presumably would lead to an increase in the pathogen load in the calving area and inside the calf housing area.

As with mortality there was a delay in onset of septicemia with higher TP levels and there was a constant hazard ratio from birth to 45 days. Also, calves with higher serum total protein concentrations that were treated for septicemia required fewer treatment days although the reduction was of little practical significance.

2.4.3.3 Omphalitis

Lack of demonstrable efficacy of passive Ig transfer against navel infections runs counter to conventional wisdom, but remarkably, very few scientific studies have been able to quantify any protection afforded by passive transfer or dipping of the navel in a disinfectant solution (46, 65). Two factors could play a role in these negative findings. First, the health management practices on these farms are such that all navels are evaluated at 2 to 4 days of age. If the navel was swollen or had an abnormal discharge at that time, the animal was started on a course of antibiotic therapy. The possibility for non-differential misclassification of navel infections may be quite high, thus reducing the chances of finding an association if there was one. The second possibility is that there is truly no association between colostrally absorbed immunoglobulins and the occurrence of navel infections.

2.4.3.4 Pneumonia

Results shown here generally agree with the literature (3, 13, 18, 27, 40, 61), however,

as in the mortality model, the association between TP and the occurrence of pneumonia was a quadratic relationship that was independent of season and farm. The lack of seasonal interaction would be expected because of the delay in the onset of pneumonia to after 30 days of age; some calves born in one season would be at risk of getting diseased in another. The fact that the hazard of developing pneumonia was not constant over time also makes biological sense. The number of sufficient causes for pneumonia are numerous and can occur at almost any time in the growing period. It would be hard to expect colostral immunity to protect the calf after three of four months when factors such as overcrowding, parasitism and nutritional stress are probably much larger contributors to the occurrence of disease.

2.5 Conclusions

Colostrally derived immunoglobulins help protect the calf from morbidity due to septicemia and pneumonia and from mortality. The preventive effect of colostrum with regards to mortality was quadratic in nature, implying that a much greater improvement in calf mortality can be seen when TP is increased from 4.0 to 5.0 g/dl than there is when TP increases from 5.5 to 6.5 g/dl. These effects on mortality last longer than previously thought as evident by a constant mortailty hazard ratio up to 6 months of age.

The positive relationship between TP and pneumonia also showed a decreasing curvilinear association (quadratic) whereas that for septicemia was linear. Once calves

became ill with one of these diseases, serum total protein concentration had a significant, but biologically minimal effect on the number of treatment days needed. Passive transfer of immunoglobulins did not show any relationship to neonatal diarrhea or omphalitis.

2.6 References

1. ARCHAMBAULT D, MORIN G, ELAZHARY Y, ROY RS, JONCAS JH. Immune response of pregnant heifers and cows to bovine rotavirus inoculation and passive protection to rotavirus infection in newborn calves fed colostral antibodies or colostral lymphocytes. Am J Vet Res 1989; 49:1084-1091.

2. BARBER DML. Serum immune globulin status of purchased calves: An unreliable guide to viability and performance. Vet Rec 1978; 102:418-420.

3. BELKNAP EB, BAKER JC, PATTERSON JS, WALKER RD, HAINES DM, CLARK EG. The role of passive immunity in bovine respiratory syncytial virus infected calves. J Infect Dis 1991; 163:470-476.

4. BERNADINA WE, van LEEUWEN MAW, HENDRIKX WML, RUITENBERG EJ. Serum opsonic activity and neutrophil phagocytic capacity of newborn lambs before and 24 to 36 hours after colostrum intake. Vet Immunol Immunopathol 1991; 29:127-138.

5. BESSER TE, SZENCI O, GAY CC. Decreased colostral immunoglobulin absorption in calves with postnatal respiratory acidosis. J Am Vet Med Assoc 1990; 196:1239-1243.

6. BOYD JW. The relationship between serum immune globulin deficiency and disease in calves: a farm survey. Vet Rec 1972; 90:645-649.

7. BOYD JW, BAKER JR, LEYLAND A. Neonatal diarrhea in calves. Vet Rec 1974; 95:310-313.

8. BRAUN RK, TENNANT BC. The relationship of serum gamma globulin levels of assembled neonatal calves to mortality caused by enteric diseases. Agri-Practice 1983; 4:14-24.

9. BRIGNOLE TJ, STOTT GH. Effect of suckling followed by bottle feeding colostrum on immunoglobulin absorption and calf survival. J Dairy Sci 1980; 63:451-456.

10. CALDOW GL, WHITE DG, KELSEY M, PETERS AR, SOLLY HJ. Relationship of calf antibody status to disease and performance. Vet Rec 1988; 122:63-65.

11. CURTIS CR, SHEARER JK, KELBERT DK. Evaluation of 3 field tests compared to a laboratory test to assess colostrum management in a large dairy herd. J Dairy Sci 1986; 69 (Suppl 1):244.

12. CURTIS CR, ERB HN, WHITE ME. Risk factors for calfhood morbidity and mortality on New York dairy farms. Proc Cornell Nutrition Conf 1985; 90-99.

13. DAVIDSON JN, YANCEY SP, CAMPBELL SG, WARNER RG. Relationship between serum immunoglobulin values and incidence of respiratory disease in calves. J Am Vet Med Assoc 1981; 179:708-710.

14. DOBBELAAR P, NOORDHUIZEN JPTM, van KEULEN KAS. An epidemiologic study of gammaglobulin levels in newborn calves. Prev Vet Med 1987; 5:51-62.

15. DONOVAN GA, BADINGA L, COLLIER RJ, WILCOX CJ, BRAUN RK. Factors influencing passive transfer in dairy calves. J Dairy Sci 1986; 69:754-759.

16. DUHAMEL GE, OSBURN BI. Neonatal immunity in cattle. Bovine Practitioner 1984; 19:71-77.

17. FALLON RJ. The effect of different methods of feeding colostrum on calf blood serum immunoglobulin levels. Anim Prod 1977; 24:142 (Abstr).

18. FALLON RJ, HARTE FJ, HARRINGTON D. The effect of calf purchase weight, serum IG level and feeding systems on the incidence of diarrhoea, respiratory disease and mortality. Bovine Practitioner 1987; 22:104-106.

19. FLEENOR WA, STOTT GH. Hydrometer test for estimation of immunoglobulin concentration in bovine colostrum. J Dairy Sci 1980; 63:973-977.

20. GAY CC, FISHER EW, McEWAN AD. Seasonal variations in gamma globulin levels in neonatal market calves. Vet Rec 1965; 77:994.

21. GAY C ?, McGUIRE TC, PARISH SM. Seasonal variation in passive transfer of imm' globulin G1 to newborn calves. J Am Vet Med Assoc 1983; 183:566-568.

22. GILBERT RP, GASKINS CT, HILLERS JK, BRINKS JS, DENHAM AH. Inbreeding and immunoglobulin g1 concentrations in cattle. J Anim Sci 1988; 66:2490-2497.

23. GLANTZ SA, SLINKER BK. Primer of applied regression and analysis of variance. Toronto: McGraw-Hill, Inc, 1990.

24. HARP JA, WOODMANSEE DB, MOON HW. Effects ot colostral antibody on susceptibility of calves to *Cryptosporidium parvum* infection. Am J Vet Res 1989; 50:2117-2119.

25. HARTMAN DA, EVERETT RW, SLACK ST, WARNER RG. Calf mortality. J Dairy Sci 1974; 57:576-578.

26. HOSMER DW, LEMESHOW S. Applied logistic regression. New York: John Wiley and Sons, 1989.

27. HOWARD JC, CLARKE MC, BROWNLIE J. Protection against respiratory infection by bovine virus diarrhea virus by passively acquired immunity. Vet Microbiol 1989; 19:195-203.

28. HUSBAND AJ, BRANDON MR, LASCELLES AK. Absorption and endogenous production of immunoglobulins in calves. Aust J Exp Biol Med Sci 1972; 50:491-498.

29. IRWIN VCR. Disease incidence in colostrum deprived calves under commercial conditions and the economic consequences. Vet Rec 1974; 94:406.

30. JAMES RE, McGUILLIARD ML, HARTMAN DA. Calf mortality in Virginia dairy herd improvement herds. J Dairy Sci 1984; 67:908-911.

31. JENNY BF, GRAMLING GE, GLAZE TM. Management factors associated with calf mortality in South Carolina dairy herds. J Dairy Sci 1981; 64:2284-2289.

32. JONES PW, COLLINS P, AITKEN MM. Passive protection of calves against experimental infection with *Salmonella typhimurium*. Vet Rec 1988; 123:536-541.

33. KIMMAN TG, ZIMMER GM, WESTENBRINK F, MARS J, van LEEUWEN E. Epidemiological study of bovine respiratory syncytial virus in calves: Influences of maternal antibodies on the outcome of disease. Vet Rec 1988; 123:104-109.

34. LOGAN EF, McMURRAY CH, O'NEILL DG, McPARLAND PJ, McRORY FJ. Absorption of colostral immunoglobulins by the neonatal calf. Br Vet J 1978; 134:258-262.

35. LOGAN EF, STENHOUSE A, ORMROD DJ. The role of colostral immunoglobulins in intestinal immunity to enteric colibacillosis in the calf. Res Vet Sci 1974; 17:290-301.

36. LOPEZ JW, ALLEN SD, MITCHELL J, QUINN M. Rotavirus and *Cryptosporidium* shedding in dairy calf feces and its relationship to colostrum immune transfer. J Dairy Sci 1988; 71:1288-1294.

37. MATTE JJ, GIRARD CL, SEOANE JR, BRISSON GJ. Absorption of colostral immunoglobulin G in the newborn dairy calf. J Dairy Sci 1982; 65:1765-1770.

38. McEWAN AD, FISHER EW, SELMAN IE. Observations on the immune globulin levels of neonatal calves and their relationship to disease. J Comp Path 1970; 80:250-265.

39. McGUIRE TC, PFEIFFER NE, WEIKEL JM, BARTSCH RC. Failure of colostral immunoglobulin transfer in calves dying of infectious disease. J Am Vet Med Assoc 1976; 169:713-718.

40. MECHOR GD, ROUSSEAUX CG, RADOSTITS OM, BABIUK LA, PETRIE L. Protection of newborn calves against fatal multisystemic infectious bovine rhinotracheitis by feeding colostrum from vaccinated cows. Can J Vet Res 1987; 51:452-459.

41. MECHOR GD, GRÖHN YT, VAN SAUN RJ. Effect of temperature on colostrometer readings for estimation of immunoglobulin concentration in bovine colostrum. J Dairy Sci 1991; 74:3940-3943.

42. MICHANEK P, VENTORP M, WESTRÖM B. Intestinal transmission of macromocules in newborn dairy calves of different ages at first feeding. Res Vet Sci 1989; 46:375-379.

43. MOHAMMED HO, SHEARER JK, BRENNEMAN JS. Transfer of immunoglobulins and survival of newborn calves. Cornell Vet 1991; 81:173-182.

44. MOLLA A. Immunoglobulin levels in calves fed colostrum by stomach tube. Vet Rec 1978; 103:377-380.

45. NAYLOR JM, KRONFELD DS. Refractometry as a measure of the immunoglobulin status of the newborn dairy calf: Comparison with the zinc sulfate turbidity test and single radial immunodiffusion. Am J Vet Res 1977; 38:1331-1334.

46. NAYLOR JM, KRONFELD DS, BECH-NIELSEN S, BARTHOLOMEW RC. Plasma total protein measurement for prediction of disease and mortality in calves. J Am Vet Med Assoc 1977; 171:635-638.

47. NOCEK JE, BRAUND DG, WARNER RG. Influence of neonatal colostrum administration, immunoglobulin, and continued feeding of colostrum on calf gain, health, and serum protein. J Dairy Sci 1984; 67:319-333.

48. NORMAN LM, HOHENBOKEN WD, KELLEY KW. Genetic differences in the concentration of immunoglobulin G1 and immunoglobulin M in serum and colostrum of cows and in serum of neonatal calves. J Anim Sci 1981; 53:1465-1471.

49. OXENDER WD, NEWMAN LE, MORROW DA. Factors influencing dairy calf mortality in Michigan. J Am Vet Med Assoc 1973; 162:458-460.

50. PATT JA. Factors affecting the duration of intestinal permeability to macromolecules in newborn animals. Biol Rev 1977; 52:411-429.

51. REID JFS, MARTINEZ AA. A modified refractometer method as a practical aid to the epidemiological investigation of disease in the neonatal ruminant. Vet Rec 1975; 96:177-179.

52. ROBISON JD, STOTT GH, DeNISE SK. Effects of passive immunity on growth and survival in the dairy heifer. J Dairy Sci 1988; 71:1283-1287.

53. SHEARER JK, BEEDE DK. Thermoregulation and physiological responses of dairy cattle in hot weather. Agri-Practice 1990; 11(4):5-17.

54. SMITH BP, HABASHA FB, REINA-GUERRA M, HARDY AJ. Immunization of calves against salmonellosis. Am J Vet Res 1980; 12:1947-1951.

55. SNEDECOR GW, COCHRAN WG. Statistical methods. 7th ed. Ames: The Iowa State University Press, 1980.

56. STOTT GH, WIERSMA F, MENEFEE BE, RADWANSKI FR. Influence of environment on passive immunity in calves. J Dairy Sci 1976; 59:1306-1311.

57. STOTT GH, REINHARD EJ. Adrenal function and passive immunity in the dystocial calf. J Dairy Sci 1978; 61:1457-1461.

58. STOTT GH, MARX DB, MENEFEE BE, NIGHTENGALE GT. Colostral immunoglobulin transfer in calves. IV. Effect of suckling. J Dairy Sci 1979; 62:1908-1913.

59. STOTT GH. Immunoglobulin absorption in calf neonates with special consideration of stress. J Dairy Sci 1980; 63:681-688.

60. THATCHER EF, GERSHWIN LJ. Colostral transfer of bovine immunoglobulin E and dynamics of serum IgE in calves. Vet Immunol Immunopathol 1989; 20:325-334.

61. THOMAS LH, SWANN RG. Influence of colostrum on the incidence of calf pneumonia. Vet Rec 1973; 92:454-455.

62. TIZZARD IR. An introduction to veterinary immunology . Toronto: WB Saunders, 1982.

63. TYLER JW, CULLOR JS, THURMOND MC, DOUGLAS VL, PARKER KM. Immunologic factors related to survival and performance in neonatal swine. Am J Vet Res 1990; 51:1400-1406.

64. VARLEY MA, WILKINSON RG, MAITLAND A. Artificial rearing of baby piglets: The effect of colostrum on survival and plasma concentration of immunoglobulin G. Br Vet J 1987; 143:369-378.

65. WALTNER-TOEWS D, MARTIN SW, MEEK AH. Dairy calf management, morbidity and mortality in Ontario Holstein herds. III. Association of management with morbidity. Prev Vet Med 1986; 4:137-158.

66. WALTNER-TOEWS D, MARTIN SW, MEEK AH. Dairy calf management, morbidity and mortality in Ontario Holstein herds. IV. Association of management with mortality. Prev Vet Med 1986; 4:159-171.

CHAPTER 3

ASSOCIATIONS AMONGST CALFHOOD DISEASES IN DAIRY CALVES

3.1 Introduction

The pathogenesis of diseases of the neonatal calf and growing heifer have been extensively reported (1, 2, 3, 4, 5, 7, 8, 9, 11) yet, knowledge about the relative importance of predisposing factors in the etiology of calf diseases is still inadequate. Diarrhea, septicemia, omphalitis and respiratory diseases are the most common medical conditions encountered (2, 3, 6, 7, 10). The risk factors involved with each of these disease syndromes are only partially understood. In Chapter 2 it was found that passive transfer of colostral immunoglobulins is an important and significant determinant for the occurrence, age of onset and severity of septicemia and pneumonia but had no protective effect on neonatal diarrhea or on the calf's susceptibility to navel infection.

Waltner-Toews, et al. (9) found that calves that were treated for diarrhea early in life were 2.5 times more likely to die after 90 days of age when compared to calves that did not have diarrhea. Reasons for mortality were not given. These researchers also found that diarrhea increased risk of pneumonia (11). In the individual calf, neonatal diarrhea increased odds of pneumonia by a factor of three. Not all cases of diarrhea occurred before pneumonia but median peak treatment for diarrhea occurred at a much earlier age

than median peak pneumonia treatment (10 days versus 6 weeks). At the farm level, a similar association was observed.

Using a study population of approximately 750 calves, researchers in New York found an even stronger "causal" association between scours and respiratory disease (OR=4), after accounting for herd differences (2). They also found that calves that had scours were 10 times more likely to develop dull calf syndrome. A diagnosis of dull calf syndrome was made when a calf showed signs of listlessness, droopy ears, dullness or anorexia within 3 months of birth.

A study conducted in Norway on feeder pigs also found a strong association between diarrhea and respiratory disease (12). These researchers describe a "double interaction" phenomena whereby piglets having diarrhea shortly after introduction into the feeder barn had a higher incidence of respiratory disease during the subsequent growth period and those pigs having respiratory disease during the early growth period in turn had a larger proportion developing diarrhea in the subsequent period.

A review of the literature revealed little with regard to the epidemiology of omphalitis or its association to other calfhood diseases. However, septicemia has been reported as one of the common sequella and causes of mortality (4).

The objective of this study was to study the associations amongst the calfhood diseases

diarrhea, septicemia, omphalitis and pneumonia in a group of Holstein dairy calves.

3.2 Materials and Methods

3.2.1 General design

Specifics of the overall study design were reported in Chapter 2 (Section 2.2).

3.2.2 Time ordering of disease variables

A temporal pattern of disease occurrence had to be established in order to evaluate one disease as a risk factor for other diseases. An obvious prerequisite for disease A to be considered a risk factor for disease B is that disease A must occur before disease B. This time ordering of diseases was accomplished in two ways. Matching (details in Section 3.2.3) was used for the evaluation of septicemia as a risk factor for diarrhea or navel infection, diarrhea as a risk factor for septicemia, navel infection or pneumonia, navel infection as a risk factor for diarrhea, septicemia or pneumonia, and pneumonia as a risk factor for diarrhea or navel infection. In the analysis of septicemia as a risk factor for pneumonia, matching was not necessary because, by disease definition (see Section 2.2.7, page 22), septicemia occurred before pneumonia. Diagnoses of pneumonia made within 14 days of the last treatment for septicemia were deleted from analysis because it was impossible to determine if this disease incident was a new case

of respiratory disease or a relapse of a septicemic episode.

3.2.3 Matching

Matching was accomplished by identifying all calves that had disease B (cases). The age of onset of disease B was calculated and determined to be the time period at risk (TPAR) for disease B. Each case was matched with 2-4 control calves that were not treated for disease B. The controls for each case were assigned the TPAR for the case calf. Calf records were then analyzed to determine if disease A (the risk factor) did or did not occur during the TPAR of disease B. These were coded with a 0 if disease A had not been observed in the calf and coded with a 1 if disease A had occurred in the TPAR of disease B.

3.2.4 Statistical analysis

All analyses were performed using multivariable logistic regression (Proc Logistic) in SAS® statistical software (SAS Institute Inc, Cary, NC, USA). Variables considered for inclusion in the models of the disease of interest and model building techniques were the same as for those described in Chapter 2, Section 2.2.10. Serum total protein, barn of origin, birth weight and season of birth were variables that were considered possible confounders. Interaction between these variables and the presence or absence of the disease of interest in the TPAR were assessed as described in Section 2.2.10.

3.3 Results

The only significant association found amongst the calfhood diseases studied in this population of calves was that of omphalitis being a risk factor for septicemia (Table XII). Septicemia was not a risk factor for the occurrence of pneumonia, but exclusion of the effect of TP when assessing the risk of pneumonia after septicemia presented entirely different results with septicemia becoming a strong risk factor ($P < 0.05$). Likewise, season of birth was a strong confounder in the model assessing diarrhea as a risk factor for septicemia and pneumonia.

3.4 Discussion

The association between navel infection and septicemia reported here may in fact be a spurious one. An assumption was made that calves that die during, or shortly after, treatment for navel infection did so because of septicemia. In the author's experience, this is a valid assumption but there may still exist some degree of disease misclassification.

The lack of any other associations does not agree with previous studies. Waltner-Toews, et al. (11) and Curtis, et al. (2) found statistically and biologically significant increases in risk of pneumonia in calves treated for scours compared to those that did not have diarrhea. The latter study also found a strong association between scours and subsequent

Table XII Statistical significance of associations amongst the calfhood diseases diarrhea, septicemia, omphalitis and pneumonia in a dairy calf population. Serum total protein, barn, season of birth and birth weight were included in the models if the likelihood ratio chi-square statistic for the variable was significant at $P < 0.05$.

Risk factor	Disease			
	Diarrhea	Septicemia	Navel Ill	Pneumonia
Diarrhea	--	0.1939	0.5324	0.0818
Septicemia	0.7125	--	0.6799	0.0997
Omphalitis	0.4487	0.0001	--	0.2768
Pneumonia	0.3791	--[a]	--[a]	--

[a] Analysis could not be performed because there were no diagnoses of septicemia or omphalitis subsequent to an episode of pneumonia

"dull calf syndrome", a diagnostic entity that may be similar to the definition of septicemia in this study.

Both of the above studies controlled for season of birth, however neither study controlled for the effects of passive transfer of colostral immunoglobulins on disease incidence. As was shown in Chapter 2, these effects on septicemia and pneumonia are highly significant and must be considered. In fact, serum total protein was a strong confounder in the model assessing septicemia as a risk factor for pneumonia. Total protein was not a confounder for the diarrhea-pneumonia relationship.

Exclusion of calves with respiratory disease diagnosis made within 14 days of the end of septicemia treatment from the pneumonia model was justified. A proportion of these calves were relapses of the septicemia episode and were not new cases of pneumonia, thus there exists some unknown degree of disease misclassification in this cohort of calves. The author feels that those calves diagnosed with pneumonia 2 weeks, or more, after ceasation of treatment for septicemia or omphalitis were much more accurately classified.

3.5 Conclusions

The finding of no associations between neonatal diarrhea, septicemia and pneumonia was unique. Passive transfer of colostral immunoglobulins was found to be a strong

confounder in the logistic model assessing septicemia as a risk factor for pneumonia and removed any apparent associations among these diseases. Season of birth was a significant variable in most models and was a confounder in those evaluating the association amongst diarrhea, and septicemia and pneumonia. These results suggest that previous studies which reported significant associations but did not control for level of absorbed colostral immunoglobulins may have reported spurious associations. Although controlling all caqlfhood diseases is important, reducing the incidence of one disease will not correspondingly decrease the incidence of other diseases, with the exception of the association between omphalitis and septicemia.

3.6 References

1. BLOOD DC, HENDERSON JA, RADOSTITS OM. Veterinary Medicine, fifth edition, Bailliere Tindall, London, 1979.

2. CURTIS CR, ERB HN, WHITE ME. Risk factors for calfhood morbidity and mortality on New York dairy farms. Proceedings of the Cornell Nutrition Conference, 1985, pp. 90-99.

3. CURTIS CR, ERB HN, WHITE ME. Epidemiologic study of risk factors for calfhood scours and respiratory syndromes in commercial dairy herds. J Dairy Sci 1985; 68(Suppl 1):198.

4. KASARI TR. Omphalitis and its sequelae in ruminants. *In* Current Veterinary Therapy 3: Food Animal Practice, WB Saunders, Philadelphia, 1992, pp. 101-103.

5. RANDLE RF. Urinary disorders associated with the neonate. *In* Current Veterinary Therapy 3: Food Animal Practice, WB Saunders, Philadelphia, 1992, pp. 821-822.

6. ROY JHB. Factors affecting susceptibility of calves to disease. J Dairy Sci 1980; 63:650-664.

7. SIMENSEN E, NORDHEIM K. An epidemiological study of calf health and performance in Norwegian dairy herds. III. Morbidity and performance: literature review, characteristics. Acta Agric Scand 1983; 33: 57-64.

8. SIMENSEN E, NORDHEIM K. An epidemiological study of calf health and performance in Norwegian dairy herds. IV. Factors affecting morbidity and performance. Acta Agric Scand 1983; 33: 65-74.

9. WALTNER-TOEWS D, MARTIN SW, MEEK AH. The effect of early calfhood health status on survivorship and age at first calving. Can J Vet Res 1986; 50:314-317.

10. WALTNER-TOEWS D, MARTIN SW, MEEK AH. Dairy calf management, morbidity and mortality in Ontario Holstein herds. II. Age and seasonal patterns. Prev Vet Med 1986; 4:125-135.

11. WALTNER-TOEWS D, MARTIN SW, MEEK AH. Dairy calf management, morbidity and mortality in Ontario Holstein herds. III. Association of management with morbidity. Prev Vet Med 1986; 4:137-158.

12. WILLEBERG P, GERBOLA MA, MADSEN A, MANDRUP M, NIELSEN EK, RIEMANN HP, AALUND D. A retrospective study of respiratory disease in a cohort of bacon pigs. Nord Veterinaermed 1978; 30: 513-525.

CHAPTER 4

CALF AND DISEASE FACTORS AFFECTING GROWTH IN DAIRY CALVES

4.1 Introduction

One objective of dairy replacement rearing is to maintain economically efficient growth of the heifer so that she will calve with a minimum of difficulty at an age and weight at which lifetime milk production and profitability are maximized (14, 18, 19, 24, 25, 40. The ideal calving age has been reported to be 24 to 25 months and the target weight at calving should be 515 to 600 kg (29, 36, 43). Growth standards that can be utilized to attain these goals have been established for Holstein cattle (23). Goals for age and weight at calving are attainable under good management, but have been debated as being in conflict with each other. In a discussion of dynamics of replacement heifer growth, Hoffman and Funk (25) stressed the need for a balance between promoting rapid growth and avoiding some of the negative effects of this practice. Accelerated growth to achieve the proper weight at first calving may adversely affect first lactation or lifetime milk yield (43, 54), possibly due to impairment of mammary gland development during the peripubertal period of allometric mammary parenchymal growth (19, 21, 49). The exact physiological mechanism involved is not known, but is believed to be due to a decrease in circulating growth hormone and somatomedins in heifers on high energy diets (46) . A reduction in rate of heifer growth, aimed at avoiding this problem, may produce a heifer that will either calve at an older age, thus increasing the cost of the heifer, or will

result in a smaller body size thereby increasing the risk of dystocia and associated health disorders (16, 43, 50).

The most obvious factor affecting body growth in the calf/heifer is the nutritional status of the individual or herd. Nutrient intake that deviates from NRC recommendations will normally result in altered growth (30, 37). This deviation can arise as a result of improperly balanced rations, improper amounts fed, and poor feed bunk and/or pasture management.

Several North American studies have found associations between serum immunoglobulin concentration in the neonatal calf, health status of the calf (6, 7, 9) and preweaning growth rate (8, 13, 38). Similar effects of colostrum on pre-weaning growth of piglets have been found (51). Studies undertaken in Great Britian and Ireland have found no relationship between passive transfer and growth or health status in purchased dairy beef calves (3, 12, 17). However, in the study by Barker (3), growth rate, in the subset of calves in which 89% of deaths occurred, was lower than in the other groups of calves. This phenomenon was explained by the author to be due to the effects of non-fatal morbidity. Robison et al. (44) are the only researchers to find a significant positive effect of passively acquired immunity on heifer growth rate through 180 days of age. In studies in which a growth promoting effect of colostrum was seen, no efforts were made to determine if it was a direct effect of colostrally derived immunoglobulins or other colostral compounds, or if these effects were mediated through a reduction in

disease incidence or severity.

Research to identify and quantify the effects of growth factors in colostrum has revealed some interesting results. Insulin, insulin-like growth factor, and other somatomedins have been isolated from bovine and porcine colostrum (1, 10, 11, 51). These proteins have been shown to stimulate growth of cells in vitro (10, 11) or to enhance energy metabolism (33). The importance and influence these proteins have on animal performance are not well defined.

The effects of parasitism on youngstock growth has been demonstrated in nearly all species of livestock (5, 26, 32). Evidence of an effect of non-parasitic diseases on growth in the neonate and in the later growth period is sparse. Neonatal diarrhea in the pig has been associated with decreased growth during, and in the period immediately after, the episode of disease (35, 45, 47, 48, 51). Evidence of long term stunting of growth is conflicting (35, 51). No direct associations such as those seen in pigs have been noted in dairy calves. In a Canadian study (52), calves that were treated for diarrhea within the first 90 days of life were nearly 3 times more likely to calve after 900 days than their nontreated contemporaries, which would imply that growth was retarded.

Respiratory diseases in swine (atrophic rhinitis and pneumonia) have been found to cause a reduction in weight gain during the growing period (2, 4, 15, 20, 39, 53). The association between respiratory disease and reduction in growth in feedlot cattle has also

been established (27, 31, 34). Karren, et al. (27) demonstrated reduction in growth rate of 0.14 and 0.08 kg/d in studies of 68 and 95 days in length, respectively. In a study of the effects of veal barn ventilation on calf health, both treatment for respiratory disease and area of lung consolidation at slaughter were directly related to reductions in daily weight gain (41). Anecdotal associations abound with regards to diarrhea, pneumonia and other diseases causing the "runt calf" syndrome.

The objective of this study was to determine the effects of colostral immune status and disease conditions on heifer growth from birth to 6 months and from 6 months to 14 months.

4.2 Materials and Methods

4.2.1 General design

Specifics of the overall study design were reported in Chapter 2 (Section 2.2, page 18).

4.2.2 Body growth data

Body weight, height, and body condition score were collected on heifers at birth and two other time periods. Data were collected at the time of calfhood vaccination for *Brucella abortus* which varied with each calf from just under 5 months to just over 6 months.

Similarly, prebreeding data were recorded when heifers entered the breeding herd for the first time at 14 ($\pm$1) months of age.

Six month and prebreeding weights were obtained using a portable platform scale (AllFlex New Zealand Inc., Palmerston North, New Zealand) which was accurate to $\pm$0.9 kg in the range from 1 to 200 kg and $\pm$2.2 kg in the range from 200 to 1000 kg.

Height was measured using a vertical standard with level calibrated to 0.5 cm and equipped with a crossbar (Nasco height stick®, Nasco, Fort Atkinson, Wisconsin, USA). Pelvic heights were recorded in all three data collection periods (birth, 6 and 14 months) and were measured at the highest point of the pelvic sacral vertabrae with the animal in the normal standing position. Withers heights were measured from a point directly above the most caudal point of the scapula with the animal in a normal standing position. This measurement was not taken at birth because of the difficulty in getting the neonatal calf to stand in a normal "headup" position.

4.2.3 Statistical analysis

All analyses were performed using multivariable linear regression (Proc GLM) in SAS® statistical software (SAS Institute Inc, Cary, NC, USA). Model building techniques and assessment of model fit were the same as for those described in Chapter 2, Section 2.2.10. Dependent variables were average daily body weight gain (kg/d) and average

daily pelvic height gain (cm/d) during each of the time period, birth to 6 months and 6 months to 14 months. Variables considered for inclusion in the models are listed in Table XIII.

4.3 Results

4.3.1 Descriptive epidemiology

Complete 6 month and 14 month data were available for 1972 and 1701 heifers, respectively. Mean average daily gain ($\pm$ standard deviation) during the time period birth to 6 months and 6 to 14 months was 0.74 (0.12) and 0.72 (0.11) kg/day, respectively. Descriptive statistics for other variables are presented in Table XIV.

The Pearson correlation coefficients for the growth variables body weight and pelvic height are shown in Table XV. In each growth period, weight gain was relatively highly correlated ($r > 0.40$) with growth in stature as measured by height at the hip. Also, growth by either measure in one growth period was negatively correlated with measures in the other growth period.

Table XIII Variables considered for inclusion in linear regression of factors influencing growth in dairy heifers

Variable Name	Variable Description
TP	Serum total protein (g/dl)
TP^2	Quadratic term for TP
Barn	Class variable (1 - 5) for barn
Season	Season of birth (1=summer, 0=winter)
W0, W6, W14	Body weight at birth, 6 and 14 mo (kg)
HP0, HP6, HP14	Pelvic height at birth, 6 and 14 mo (cm)
HS6, HS14	Withers height at 6 and 14 mo (cm)
NUMDIA	Diarrhea, number treatment days
DIA	Diarrhea, dichotomous variable (0=not treated, 1=treated)
NUMSEP	Septicemia, number treatment days
SEP	Septicemia, dichotomous variable (0=not treated, 1=treated)
NUMPNU1	Pneumonia 0-6 months, number treatment days
NUMPNU2	Pneumonia 6-14 months, number treatment days
PNU1	Pneumonia 0-6 mo, dichotomous variable (0=not treated, 1=treated)
PNU2	Pneumonia 6-14 mo, dichotomous variable (0=not treated, 1=treated)
NUMNAV	Navel infection, number treatment days
NAV	Navel infection, dichotomous variable (0=not treated, 1=treated)
NUMPEY	Pinkeye, number treatment days
PEY	Pinkeye, dichotomous variable (0=not treated, 1=treated)
Age6, Age14	Age at 6 and 14 month data collection

Table XIV Descriptive epidemiology of heifer growth data

Variable	Mean ±S.D.	Min	Max
Age at 6 mo data collection (days)	165 (±16)	132	205
Age at 14 mo data collection (days)	428 (±51)	354	526
Weight at 6 mo (kg)	159 (±25)	92	215
Weight gain birth to 6 mo (kg/d)	0.74 (±0.12)	0.21	1.34
Weight at 14 mo (kg)	358 (±76)	264	438
Weight gain 6 to 14 mo (kg/d)	0.72 (±0.11)	0.26	1.19
Pelvic height at 6 mo (cm)	106 (±4.8)	93	116
Pelvic height gain birth to 6 mo (cm/d)	0.17 (±0.04)	0.05	0.24
Pelvic height at 14 mo (cm)	129 (±4.0)	121	139
Pelvic height gain 6 to 14 mo (cm/d)	0.09 (±0.02)	0.03	0.17
Withers height at 6 mo (cm)	100 (±4.3)	90	110
Withers height at 14 mo (cm)	121 (±4.6)	112	133
Withers height gain 6 to 14 mo (cm/d)	0.08 (±0.02)	0.02	0.15

Table XV Correlation coefficients of selected growth variables[a]

	WG14[b]	PG6	PG14
WG6	-0.184	0.434	-0.249
WG14		-0.133	0.488
PG6			-0.325

[a] All r values significant at $P < 0.0001$

[b] WG6= Weight gain birth to 6 mo; WG14= Weight gain 6 to 14 mo; PG6= Pelvic height gain birth to 6 mo; PG14= Pelvic height gain 6 to 14 mo

4.3.2 Weight gain

Two sets of linear regression models were assessed; one using the presence or absence of disease and the second using severity of disease as measured by number of treatment days. The R-square of the models for weight gain from birth to 6 months and from 6 to 14 months using the latter of the two disease measures was higher and therefore it was used as the measure of disease conditions in the main effects models. The results from the models incorporating the dichotomized disease variable are reported as the alternate models and are included in Appendix G for comparison. In these models, barn of origin was a confounding variable. Because of the variability in age at 6 and 14 month data collection, age of the heifer at these time periods was entered into each model as a covariate. The fit of both models was considered acceptable using the criteria described in Section 2.2.10.

Factors that significantly affected calf body weight gain from birth to 6 months are presented in Table XVI. Together, the factors accounted for 19.7% of the variation in growth during this time period. Using the mean number days treated for diarrhea (3.76 d), septicemia (5.72 d), and pneumonia (5.63 d), a depression in 180 day weight gain of approximately 9.1, 4.8 and 10.6 kg, respectively was seen. Passive transfer of colostral immunoglobulins, as measured by serum total protein, and the occurrence of navel infection did not significantly alter growth in this cohort of calves.

Table XVI Linear regression model of factors affecting body weight gain in dairy heifers from birth to 6 months using number of days treated for disease

Outcome variable = Average daily gain (kg/d) from birth to 6 months of age

Variable	Parameter Estimate	*P-value*
Intercept	0.2310	0.0336
Diarrhea (NUMDIA)	-0.0134	0.0001
Septicemia(NUMSEP)	-0.0046	0.0012
Pneumonia(NUMPNU1)	-0.0105	0.0001
Birth weight (W0)	-0.0028	0.0089
Birth height (HP0)	0.0045	0.0034
Age at 6 mo (Age6)	0.0015	0.0001
Season	-0.0460	0.0001
R^2 = 0.1973		

Variables that were associated with growth rate in the second growth period (6 to 14 months) were rate of gain in the first growth period, age at prebreeding data collection and the number of days treated for pneumonia before 6 months of age (Table XVII). The effect of pneumonia was small, accounting for a 3.1 kg reduction in growth during this time period.

4.3.3 Height gain

Pelvic height growth was analysed in the same manner as body weight growth using continuous and dichotomous measures of disease. The model for height growth in the first evaluation period (birth to 6 months) using number of treatment days as the disease measure was determined to be better than that using dichotomous data by virtue of its higher R^2 value (0.31 vs 0.29). The model using continuous disease data is shown in Table XVIII. Appendix H contains the alternate model using occurrence of disease variables. The fit of all models was considered to be adequate using criteria described in Section 2.2.10.

Diarrhea, septicemia and pneumonia each had a significant effect on pelvic growth through the first 6 months. Based upon mean number of treatment days for each disease condition, a reduction of 5.1, 3.2 and 4.4% in pelvic height growth rate was observed to be due to diarrhea, septicemia and pneumonia, respectively. Serum total protein concentration at 2 to 8 days of age did not have any significant effect on height growth

Table XVII Linear regression model of factors affecting body weight gain in dairy heifers from 6 to 14 months using number of days treated for disease

Outcome variable = Average daily gain (kg/d) from 6 to 14 months

Variable	Parameter Estimate	*P-value*
Intercept	1.2188	0.0000
Weight Gain 0-6 mo	-0.1756	0.0001
Age at 14 mo	-0.0008	0.0001
Pneumonia 0-6 mo	-0.0023	0.0053
R^2 = 0.2108		

Table XVIII Linear regression model of factors affecting growth in stature (pelvic height) of dairy heifers from birth to 6 months using number of days treated for disease

Outcome variable = Average daily pelvic height growth (cm/d) from birth to 6 months

Variable	Parameter Estimate	*P-value*
Intercept	0.4604	0.0001
Diarrhea (NUMDIA)	-0.0024	0.0001
Septicemia(NUMSEP)	-0.0009	0.0002
Pneumonia(NUMPNU1)	-0.0014	0.0001
Birth weight (W0)	0.0005	0.0194
Birth height (HP0)	-0.0040	0.0001
Season	-0.0041	0.0070
$R^2 = 0.3116$		

in these heifers.

None of the disease conditions evaluated had any significant effect on pelvic height growth during the growth period from 6 to 14 months of age (Table XIV).

4.4 Discussion

4.4.1 Model selection

The models developed using number of treatment days as the measure of disease were used because they explained a higher percentage of the variation in the dependent variable, and barn was not a confounding variable in the models. The external validity of models without a confounding farm effect may be better and results generated more readily applied to other populations of similar animals.

Pelvic height measurements and growth in pelvic height were used instead of withers height data because they generated higher R^2 values in respective models of growth. The author felt that pelvic height measurments were more accurate and precise than those taken at the shoulder because heifers stood squarely during pelvic height determination and tended to crouch when height measurements were taken at the withers.

Table XIV Linear regression model of factors affecting growth in stature (pelvic height) of dairy heifers from 6 to 14 months using number of days treated for disease

Outcome variable = Average daily pelvic height growth (cm/d) from 6 to 14 months

Variable	Parameter Estimate	*P-value*
Intercept	0.2287	0.0000
Weight Gain 0-6 mo	-0.0394	0.0001
Age at 14 mo	-0.0002	0.0001
Season	-0.0034	0.0001
R^2 = 0.4939		

4.4.2 Weight gain from birth to 6 months

The average rate of body weight gain in this cohort of heifers was within published recommendations (23, 37). Passive transfer of colostral immunoglobulins had no effect on rate of weight gain, a finding that is in disagreement with most published studies (8, 13, 38, 44). Two reasons could account for this discrepency. In other studies, disease conditions were not included in the analysis. Analysis of the data from our study, without controlling for disease status, produced a spurious association between serum total protein and rate of body weight gain through 6 months. Secondly, in most other studies, the growth period covered was relatively short, 60 to 90 days (8, 13, 38). Any preweaning growth advantage attributable to passive transfer may be diluted out over a longer follow-up period.

The negative effects of diarrhea and septicemia on rate of weight gain through 6 months were a unique finding. Indirect (52) or unsubstantiated (35) associations between diarrhea and reduction in growth of calves have been made. Waltner-Toews (52) found that calves with diarrhea in the first 90 days were 3 times more likely to calve after 900 days than their healthy herdmates. If one assumed that no confounding existed between age and weight at breeding and occurrence of diarrhea, then the delay in calving could be attributed to reduced growth in those heifers. For each day that a calf is treated for diarrhea, the reduction in weight gain would add approximately 3.3 days to the growth period of affected calves to get them to the same weight as unaffected calves. Mean days

treated for diarrhea in this cohort of calves was 3.76 which would add approximately 13 days to the growth period. Using the alternate model, which used dichotomous disease classification, occurrence of diarrhea resulted in an estimated 12.7 kg reduction in growth through 6 months.

The association between occurrence of pneumonia and growth rate to 6 months was similar to that seen in beef cattle. Other studies have used occurrence of respiratory disease as the measure of disease. Our results were of lower magnitude (0.07 kg/d) than others. This difference may be a function of the length of follow-up. In two similarly designed studies, Martin, et al. (34) found that the impact of respiratory disease on rate of gain in a feedlot was less with a longer follow-up period. This may be partially explained by compensatory growth after recovery from illness (22).

The magnitude of the effect of septicemia on growth was less than that of the previously mentioned diseases, but still amounted to a one day reduction in growth for every day treated. In this study, the septicemia case fatality rate was high at 27.6% (Appendix C, Figure 3). Herds with lower septicemia case fatality rates may experience somewhat different growth rates in survivors. The absence of any relationship between navel infection and growth rate was expected. Any such relationship cited in the literature is either weakly substantiated, or not substantiated at all (28, 42).

A complicating factor that could impact the outcome of this study, or any epidemiological

investigation, is loss of follow-up of some study subjects. Few of the calves entering the study were culled before 6 months of age; 51 of 129 heifers that left the herd did so because of the effects of chronic respiratory disease on growth. Had these "poor doers" been included in the analysis, a greater impact of pneumonia would likely have been seen.

Because of the design of this study, we cannot be absolutely certain that the diseases of interest actually caused a reduction in growth. An alternative explanation might be that slow growing calves are at higher risk of developing disease. However, reports of studies that had a shorter follow-up period support the former hypothesis (27, 34). In addition there is no known biological reason why slower growing calves should be at a higher risk of disease than those growing at a normal rate. Consequently, it was concluded that diarrhea, pneumonia and septicemia did adversely affect the growth of calves from birth to 6 months of age.

4.4.3 Weight gain from 6 to 14 months

The impact of pneumonia during the second growth period was significant ($P < 0.05$) but of marginal clinical importance. A heifer treated an average number of days (5.76) would be expected to have a reduction in growth to 14 months of 3.2 kg. This translates to an additional 4.4 days to reach the same weight as healthy herdmates. All of the observed effect was found to be due to disease occurring before 6 months of age.

Since many poor-doing heifers are culled during this time period, the actual effect of respiratory disease is most likely somewhat greater. None of the other diseases, including keratoconjunctivitis, had any significant influence on heifer body weight gain.

This model could also be interpreted as an example of "compensatory gain", which is a phenomenon in which animals can grow quite rapidly after a period of relatively slow growth (22). Animals that grew slowly during the period from birth to 6 months grew faster than the mean growth after 6 months. Alternately, calves that grew fast when younger, grew more slowly later on.

4.4.4 Growth in pelvic height

The clinical significance of the effect of the monitored diseases on pelvic height growth is unknown. Using the average number of treatments per calf for diarrhea, septicemia and pneumonia, a reduction in height growth of 1.6, 1.1 and 1.4 cm, respectively, was observed. Breeding data for heifers from Barn 1 showed that pelvic height is a significant predictor for first service conception after controlling for breeding season and viral vaccination status (Appendix I). No other data are available that objectively look into the effect of stature (height) on performance and production variables.

4.5 Conclusions

Health status of dairy calves and growing heifers had a significant effect on growth rate, especially during the first 6 months of life. Septicemia and pneumonia slowed growth by 13 to 15 days during the first 6 months. Diarrhea had a much smaller impact on growth. Passive transfer of colostral antibodies had no direct effect on growth but does influence weight and height growth through its effects on health, especially septicemia and respiratory disease (Chapter 2). The longer term effects of disease on growth and production are unknown and need to be addressed.

4.6 References

1. ARANDA P, SANCHEZ L, PEREZ MD, ENA JM, CALVO M. Insulin in bovine colostrum and milk: Evolution through lactation and binding to caseins. J Dairy Sci 1991; 74:4320-4325.

2. BACKSTROM L, HOEFLING DC, MORKOC AC, COWART RP. Effect of atrophic rhinitis on growth rate in Illinois swine herds. J Am Vet Med Assoc 1985; 187:712-715.

3. BARBER DML. Serum immune globulin status of purchased calves: An unreliable guide to viability and performance. Vet Rec 1978; 102:418-420.

4. BERNARDO TM, DOHOO IR, DONALD A. Effect of ascariasis and respiratory diseases on growth rates in swine. Can J Vet Res 1990; 54:278-284.

5. BORGSTEEDE FH, HENDRIKS J. Experimental infections with *Cooperia oncophora* (Railliet, 1918) in calves. Results of single infections with two graded dose levels of larvae. Parasitology 1979; 78:331-342.

6. BOYD JW. The relationship between serum immune globulin deficiency and disease in calves: a farm survey. Vet Rec 1972; 90:645-649.

7. BOYD JW, BAKER JR, LEYLAND A. Neonatal diarrhea in calves. Vet Rec 1974; 95:310-313.

8. BRADLEY JA, NIILO L. Immunoglobulin transfer and weight gains in suckled beef calves force-fed stored colostrum. Can J Comp Med 1985; 49:152-155.

9. BRAUN RK, TENNANT BC. The relationship of serum gamma globulin levels of assembled neonatal calves to mortality caused by enteric diseases. Agri-Practice 1983; 4:14-24.

10. BROWN KD, BLAKELEY DM. Cell growth-promoting activity in mammary gland secretions of the goat, cow and sheep. Br Vet J 1983; 139:68-78.

11. BURRIN DG, SHULMAN RJ, REEDS PJ, DAVIS TA, GRAVITT KR. Porcine milk and colostrum stimulate visceral organ and skeletal muscle protein synthesis in neonatal pigs. J Nutrition 1992; 122:1205-1212.

12. CALDOW GL, WHITE DG, KELSEY M, PETERS AR, SOLLY HJ. Relationship of calf antibody status to disease and performance. Vet Rec 1988; 122:63-65.

13. DAVIDSON JN, YANCEY SP, CAMPBELL SG, WARNER RG. Relationship between serum immunoglobulin values and incidence of respiratory disease in calves. J AM Vet Med Assoc 1981; 179:708-710.

14. DONOVAN GA, BRAUN, RK. Evaluation of dairy heifer replacement-rearing programs. Comp Contin Educ Pract Vet 1987; 9:F133-F139.

15. DUBERSTEIN LE, HESSLER JR. Porcine atrophic rhinitis: a model for studying nasal physiology and pathophysiology. Rhinology 1978; 16:31-39.

16. ERB HN, SMITH RD, OLTENACU PA, GUARD CL, HILLMAN RB, POWERS PA, SMITH MC, WHITE ME. Path model of reproductive disorders and performance, milk fever, mastitis, milk yield and culling in Holstein dairy cows. J Dairy Sci 1985; 68:3337.

17. FALLON RJ. The effect of immunoglobulin levels on calf performance and methods of artificially feeding colostrum to the newborn calf. Ann Rech Vet 1978; 9:347-352.

18. FISHER LJ, HALL JW, JONES SE. Weight and age at calving and weight change related to first lactation milk yield. J Dairy Sci 1983; 66:2167-2172.

19. GARDNER RW, SCHUH JD, VARGUS LG. Accelerated growth and early breeding of Holstein heifers. J Dairy Sci 1977; 60:1941-1948.

20. HALL WF, BANE DP, KILROY CR, ESSEX-SORLIE DL. A model for the induction of *Pasteurella multocida* type-A pneumonia in pigs. Can J Vet Res 1990; 54:238-243.

21. HARRISON RD, REYNOLDS IP, LITTLE W. A quantitative analysis of mammary glands of dairy heifers reared at different rates of live weight gain. J Dairy Res 1983; 50:405-412.

22. HEAD HH. Heifer performance standards: Rearing systems, growth rates and lactation. *In*: Van Horn HH, Wilcox CJ, eds. Large Dairy Herd Management. Champaign, Ill: American Dairy Science Association, 1992:422-433.

23. HEINRICHS AJ, HARGROVE GL. Standards of weight and height for Holstein heifers. J Dairy Sci 1987; 70:653-660.

24. HOCKING PM, McALLISTER AJ, WOLYNETZ MS, BATRA TR, LEE AJ, LIN CY, ROY GL, VESELY JA, WAUTHY JM, WINTER KW. Factors affecting culling and survival during rearing and first lactation in purebred and crossbred dairy cattle. Anim Prod 1988; 46:1-12.

25. HOFFMAN PC, FUNK DA. Applied dynamics of dairy replacement growth and management. J Dairy Sci 1992; 75:2504-2516.

26. JOHNSON JC, STEWART TB, HALE OM. Differential responses of Duroc, Hampshire, and crossbred pigs to a superimposed experimental infection with the intestinal threadworm, *Strongyloides ransomi*. J Parisitology 1975; 61:517-524.

27. KARREN DB, BASARAB JA, CHURCH TL. The growth and economic performance of preconditioned calves and their dams on the farm and of calves in the feedlot. Can J Anim Sci 1987; 67:327-336.

28. KASARI TR. Omphalitis and its sequelae in ruminants. *In* Current Veterinary Therapy 3: Food Animal Practice, WB Saunders, Philadelphia, 1992, pp. 101-103.

29. KEOWN JF, EVERETT RW. Effect of days carried calf, days dry, and weight of first calf heifers on yield. J Dairy Sci 69:1891-1897.

30. KERTZ AF, PREWITT LR, BALLAM JM. Increased weight gain and effects on growth parameters of Holstein heifer calves from 3 to 12 months of age. J Dairy Sci 1987; 70:1612-1622.

31. LEBLANC PH, BAKER JC, GRAY PR, ROBINSON NE, DERKSEN FJ. Effects of bovine respiratory syncytial virus on airway function in neonatal calves. Am J Vet Res 1991; 52:1401-1406.

32. LELAND SE, DAVIS GV, CALEY HK, ARNETT DW, RIDLEY RK. Economic value and course of infection after treatment of cattle having a low level of nematode parasitism. Am J Vet Res 1980; 41:623-633.

33. LEPINE AJ, BOYD RD, WHITEHEAD DM. Effect of colostrum intake on hepatic gluconeogenesis and fatty acid oxidation in the neonatal pig. J Anim Sci 1991; 69:1966-1974.

34. MARTIN SW, BATEMAN KG, SHEWEN PE, ROSENDAL S, BOHAC JE. The frequency, distribution and effects of antibodies to seven putative respiratory pathogens on respiratory disease and weight gain in feedlot calves in Ontario. Can J Vet Res 1989; 53:355-362.

35. MEBUS CA. Diarrhea in neonatal animals. J Clin Nutrition 1977; 30:1851-1855.

36. MOORE RK, KENNEDY BW, SCHAEFFER LR, MOXLEY JE. Relationship between age and body weight at calving and production in first lactation Ayrshires and Holsteins. J Dairy Sci 1991; 74:269.

37. NATIONAL RESEARCH COUNCIL. Nutrient requirements of dairy cattle. 6th rev. ed. Washington: National Academy Press, 1989.

38. NOCEK JE, BRAUND DG, WARNER RG. Influence of neonatal colostrum administration, immunoglobulin, and continued feeding of colostrum on calf gain, health, and serum protein. J Dairy Sci 1984; 67:319-333.

39. NOYES EP, FEENEY DA, PIJOAN C. Comparison of the effect of pneumonia during lifetime with pneumonia detected at slaughter on growth in swine. J Am Vet Med Assoc 1990; 197:1025-1029.

40. OTTERBY DE, LINN JG. Advances in nutrition and management of calves and heifers. J Dairy Sci 1981; 64:1365-1377.

41. PRITCHARD DG, CARPENTER CA, MORZARIA SP, HARKNESS JW, RICHARDS MS, BREWER JI. Effect of air filtration on respiratory disease in intensively housed veal calves. Vet Rec 1981; 109:5-9.

42. RANDLE RF. Urinary disorders associated with the neonate. *In* Current Veterinary Therapy 3: Food Animal Practice, WB Saunders, Philadelphia, 1992, pp. 821-822.

43. REMER Y. Economic evaluation of early breeding of dairy cattle heifers under farm conditions in Isreal. Proc 27 Ann Meeting European Assoc Anim Prod 1976; 56:1-14.

44. ROBISON JD, STOTT GH, DeNISE SK. Effects of passive immunity on growth and survival in the dairy heifer. J Dairy Sci 1988; 71:1283-1287.

45. SARMIENTO JI, DEAN EA MOON HW. Effects of weaning on diarrhea caused by enterotoxigenic *Escherichia coli* in three-week-old pigs. Am J Vet Res 1988; 49:2030-2033.

46. SJERSEN K, HUBER JT, TUCKER HA. Influence of amount fed on hormone concentrations and their relationship to mammary growth in heifers. J Dairy Sci 1983; 66:845-855.

47. SVENSMARK B, JORSAL SE, NIELSEN K, WILLEBERG P. Epidemiological studies of piglet diarrhoea in intensively managed Danish sow herds. I. Pre-weaning diarrhea. Acta Vet Scand 1989; 30:43-53.

48. SVENSMARK B, NIELSEN K, WILLEBERG P, JORSAL SE. Epidemiological studies of piglet diarrhoea in intensively managed Danish sow herds. II. Post-weaning diarrhea. Acta Vet Scand 1989; 30:55-62.

49. SWANSON EW. Optimal growth patterns for dairy cattle. J Dairy Sci 1967; 50:244-252.

50. THOMPSON JR, POLLAK EJ, PELISSIER CL. Interrelationships of parturition problems, production of subsequent lactation, reproduction and age at first calving. J Dairy Sci 1983; 66:1119-1127.

51. TYLER JW, CULLOR JS, THURMOND MC, DOUGLAS VL, PARKER KM. Immunologic factors related to survival and performance in neonatal swine. Am J Vet Res 1990; 51:1400-1406.

52. WALTNER-TOEWS D, MARTIN SW, MEEK AH. The effect of early calfhood health status on survivorship and age first calving. Can J Vet Res 1986; 50:314-317.

53. WILSON MR, TAKOV R, FRIENDSHIP RM, MARTIN SW, McMILLAN I, HACKER RR, SWAMINATHAN S. Prevalence of respiratory diseases and their association with growth rate and space in randomly selected swine herds. Can J Vet Res 1986; 50:209-216.

54. YOUNG CW, EIDMAN VR, RENEAU JK. Animal health and management and their impact on economic efficiency. J Dairy Sci 1985; 68:1593-1602.

CHAPTER 5

SUMMARY AND CONCLUSIONS

5.1 Justification for Study

The success of a dairy heifer replacement rearing program largely depends on effective disease control, proper growth with a cost effective feeding program and having an age at first calving of approximately 24 months (12, 18, 23, 36). Replacement rearing may be one of the least efficient aspects of the dairy farm enterprise. The total dollar loss to the dairy industry in the United States has not been well documented but estimates are as high as $200,000,000 per year (17). Calf mortality surveys from several areas have shown a range of mortality rates of 15 to 30% (17, 21, 26, 34). Martin, et al. (26), using a computer model, showed that a 20% mortality rate resulted in a 38% decrease in net profit for the calf raising unit. The economic losses on the average dairy farm due to delayed first calving may be even greater than that from calf mortality (12).

Identifying and quantifying the factors affecting morbidity, mortality, slow growth and delayed first calving could help the dairyman in making management and culling decisions earlier so that further losses on affected animals could be avoided. The calf that does not die after a bout of disease often is overlooked as a potential economic liability. Veterinary clinicians or dairymen often claim, without supporting data, that these animals are chronic slow growers and are non-productive. The interrelations

amongst passive transfer of colostral immunoglobulins, morbidity, mortality, and growth rate of heifers have not been explored perhaps due to the large number of study subjects required to make these associations.

With this in mind, a prospective cohort study with well defined disease conditions and a large sample size was designed with an overall objective of determining the associations amongst passive transfer status, disease occurrence and mortality, and growth in dairy replacement heifers.

5.2 Summary of Results

5.2.1 The Role of Passive Immunity in Calf Health and Growth

Since Smith and Little (39) published their classic dissertation on the significance of colostrum to the newborn calf in 1922, many researchers have focussed on this area of dairy calf management. Calves with failure of passive transfer (FPT) compared to those that have received adequate amounts of colostral immunoglobulins have a 2- to 4-fold increase in mortality (4, 6, 7, 14, 20, 25, 29, 30, 37). The protective effects of colostrum against morbidity due to calfhood diseases has also been demonstrated (3, 11, 19, 31, 40). These studies focused primarily on the association amongst passive transfer status, mortality and the occurrence of selected diseases, most notably enteric and respiratory diseases.

Results of our study substantiate many of those previous observations. In this study, definitions of disease conditions were more precise thus allowing for more indepth analysis of the relationship between passive transfer status and neonatal diseases. Data presented here show that adequate passive transfer delays onset of septicemia and pneumonia. This is of clinical importance because the neonatal calf's immune system matures in a linear fashion to full, adult function at 3-5 weeks of age (13, 41); thus older calves can more readily respond to disease. The severity of septicemia or pneumonia was slightly reduced in calves that had high levels of circulating colostral antibodies, but these findings were of minimal clinical significance.

The data also show that passive transfer was not associated with the occurrence, severity or age of onset of diarrhea or omphalitis. The enteric disease agent(s) known to be endemic in these herds, rotavirus, cryptosporidia and salmonella, are those for which colostrum will not afford protection (1, 16, 25, 38). The lack of any protective effect of colostrum against navel infection is intuitively surprising. However, a review of the literature shows no substantive reports to the contrary (15, 32, 43).

Because of the large study population, the shape of the relationship between passive transfer, as measured by serum total protein, and mortality could be determined. The quadratic relationship found (Table IV, page 31 and Figure 4, page 32) indicates that a great reduction in mortality can be achieved by increasing calf TP levels from 4.0 to 5.0 g/dl, but little benefit is derived by increasing TP above 6.0 g/dl. The large study size

also allowed us to evaluate possible interactions among predictor variables. In the mortality models, no significant interactions were found; the effect of TP on mortality was constant across barns, seasons and birth weight which allows for valid extrapolation of this inference to other populations of calves.

A unique and suprising finding was the constant mortality hazard ratio throughout the period from birth to 6 months (Table V, page 33, Figure 5, page 35 and Appendix F). The relative risk of mortality was 2 to 3 times higher in calves with FPT when compared to those receiving adequate amounts of colostrum. Previous studies could not evaluate this phenomenon because they did not have a long enough follow-up period, had a small sample size or did not include other calf and herd level factors that could be possible confounders.

Passive transfer of colostral immunoglobulins had no effect on rate of weight gain, a finding that is in disagreement with most published studies (5, 11, 33, 37). However, in other studies, disease conditions were not included in the analyses. Analysis of the data from our study, without controlling for disease status, produced a spurious association between serum total protein and rate of body weight gain through 6 months. Also, the growth period covered in previous studies was relatively short (60 to 90 days) (5, 11, 33). Any preweaning growth advantage attributable to passive transfer may be diluted out over the longer follow-up period used in this study.

5.2.2 Associations Amongst Calfhood Diseases

Calves that develop navel infections are 1.8 times more likely to develop subsequent septicemia than their healthy herdmates. No other direct associations amongst the monitored diseases were seen. These findings do not agree with previous studies showing diarrhea and septicemia as strong risk factors for pneumonia (10, 43). In the evaluation of septicemia as a risk factor for pneumonia, passive transfer of colostral antibodies was a strong confounding variable that when uncontrolled in the analysis led to a spurious association between these two disease entities. Season of birth was similarly shown to be a confounder in the analysis of the association between diarrhea and pneumonia. These factors were not controlled in previous studies and this probably accounted for their finding of associations between diarrhea, septicemia and pneumonia.

The strengths of this study for evaluating associations among diseases lay in the sample size and definitions of the diseases. Health data were collected on over 2500 calves/heifers during the 14 month study period. Disease misclassification is inevitable in this type of study, however, disease conditions were defined as precisely as possible and every effort was made to minimize the problem of misclassification through continual personnel training and monitoring.

5.2.3 Disease Factors Affecting Growth

The health status of dairy calves and growing heifers had a significant effect on growth rate, especially during the first 6 months of life. Both body weight gain and pelvic height growth were affected by some disease conditions. Septicemia and pneumonia each slowed growth by 13-15 days during the first 6 months whereas diarrhea had a significant, albeit minimal, impact on growth. The residual effects of pneumonia in the first six months of life carried over into the second growth period monitored (6 to 14 mo), but this depression in growth was clinically insignificant. Navel infection status and pinkeye had no effect on growth rate.

To our knowledge these findings are the first to be reported from a longterm study of dairy replacement heifers. Data from 2 short-term studies of veal calves showed some reduction in growth due to pneumonia (8, 35) and one report obliquely implied that diarrhea caused slowing of growth and delayed first parturition (42). The magnitude of the effect of respiratory disease on heifer growth was less than that seen in feedlot animals (22, 24, 27). Two possible factors may minimize the observed impact of respiratory disease on weight gain. Calves may experience compensatory weight gain after recovery from illness or alternately, slow growing calves may have been culled before reaching the age at which they would be weighed. The latter scenario may have happen as it was a management practice on both farms to cull animals that were growing slowly and were deemed likely to be non-profitable. This became more apparent as the

study went on and has been documented in a previous study (10).

Using the data shown here, and an average cost of $2.00 per day to keep a replacement heifer in the herd (9, 44), diarrhea, septicemia and pneumonia cost $24.60, $13.00 and $28.60, respectively, in delayed growth costs alone. This calculation assumed that the study heifers will calve at a standardized weight and or height and no further negative effects of disease or compensatory gain will be seen. Had this been an experimental study and all poordoers kept in the herd, these costs would be much greater.

5.3 Conclusions

Using the data generated by this study, an estimate of the value of an improvement in colostrum management can be made. Several assumptions must be made in order to arrive at this estimate; these are listed in Table XX. Table XXI demonstrates potential savings per calf when the serum total protein concentration of calves with TP less than 5.5 g/dl is increased by 0.5 g/dl. The proportion of calves in this cohort that had TP less than 5.0 g/dl and less than 5.5 g/dl would be reduced from 18.0% and 46.7%, respectively, to 3.3% and 18%. These latter proportions are more in line with attainable goals set by many veterinary clinicians. This improvement in colostrum management would directly result in a decrease in mortality, decrease in morbidity due to septicemia and pneumonia, and fewer treatments for calves that became ill due to these two calfhood diseases. Indirectly, an improvement in growth would be seen via a decrease in the

Table XX Assumptions made in developing economic analysis of improving colostrum management

Value of a calf at death[a]	$200.00
Cost per treatment - septicemia	$ 2.00
Mean number treatments - septicemia	5.72
Cost per treatment - pneumonia	$ 3.00
Mean number treatments - pneumonia	5.82
Cost per day to raise a heifer	$ 2.00

[a] Calves with failure of passive transfer die at the same age as calves that have absorbed adequate amounts of colostral immunoglobulins.

Table XXI Potential economic benefits of improving colostrum management in a cohort of Holstein dairy calves. Serum total protein (TP) increased 0.5 g/dl in calves with TP < 5.5 g/dl.

Frequency distribution of serum total protein concentration (g/dl)								
TP Categories	4.0-4.4	4.5-4.9	5.0-5.4	5.5-5.9	6.0-6.4	6.5-6.9	7.0-7.4	7.5-8.0
Actual Distribution	93	429	829	714	495	219	85	28
Improved Distribution	0	93	429	1543	495	219	85	28

	Improvement	$ Saved	$ Saved / hd
Mortality	66 calves	$13,200	$4.56
Number of septicemia cases	17 cases	$198	$0.07
Number of pneumonia cases	59 cases	$1002	$0.35
Severity of septicemia cases	167 treatment-days	$333	$0.12
Severity of pneumonia cases	392 treatment-days	$1175	$0.41
Total Morbidity/Mortality Savings			**$5.51**
Growth loss avoided - septicemia	112 growth-days[a]	$224	$0.08
	849 growth-days	$1699	$0.59
Total Slow Growth Savings			**$0.67**
TOTAL SAVINGS FROM IMPROVED COLOSTRUM MANAGEMENT			**$6.18**

[a] One growth-day equals the number of extra days (0.74 kg/day) that diseased heifers take to reach the same weight at 6 months as non-diseased heifers.

number of calves that developed septicemia and pneumonia. The overall savings from this improvement in health would be at least $6.18 per calf born alive. There could also be an indirect effect of improved colostrum management on herd health. Since fewer animals in the herd become diseased, the probability of adequate contact between those that are diseased and those that are non-diseased would be reduced. According to the Reed-Frost model of herd immunity (28), the number of cases of disease should be reduced. Other factors such as reduction of stress, environmental (housing) and nutritional management, parasite control and a sound herd vaccination program are required to further improve herd health and profitability.

Timely administration of an adequate volume of high quality colostrum is of paramount importance for future health and performance of the neonatal calf. Few chores on the farm will give the monetary returns and job satisfaction as the time and effort expended ensuring that colostrum is ingested by the calf. Veterinarians can assess colostrum management quickly, easily and accurately via serum total protein concentrations in neonatal calves and make corrective action when needed.

The longer term effects of diseases on growth, reproduction and milk production in first and later lactations are still unknown. Data will be collected on this cohort of heifers through the end of their first lactation so that these associations can be elucidated. We will also evaluate the relationships between various body size measurements taken at breeding and conception rate and risk of dystocia at first calving.

5.4 References

1. ARCHAMBAULT D, MORIN G, ELAZHARY Y, ROY RS, JONCAS JH. Immune response of pregnant heifers and cows to bovine rotavirus inoculat' n and passive protection to rotavirus infection in newborn calves fed colostra antibodies or colostral lymphocytes. Am J Vet Res 1989; 49:1084-1091.

2. BARBER DML. Serum immune globulin status of purchased calves: An unreliable guide to viability and performance. Vet Rec 1978; 102:418-420.

3. BELKNAP EB, BAKER JC, PATTERSON JS, WALKER RD, HAINES DM, CLARK EG. The role of passive immunity in bovine respiratory syncytial virus infected calves. J Infect Dis 1991; 163:470-476.

4. BOYD JW. The relationship between serum immune globulin deficiency and disease in calves: a farm survey. Vet Rec 1972; 90:645-649.

5. BRADLEY JA, NIILO L. Immunoglobulin transfer and weight gains in suckled beef calves force-fed stored colostrum. Can J Comp Med 1985; 49:152-155.

6. BRAUN RK, TENNANT BC. The relationship of serum gamma globulin levels of assembled neonatal calves to mortality caused by enteric diseases. Agri-Practice 1983; 4:14-24.

7. BRIGNOLE TJ, STOTT GH. Effect of suckling followed by bottle feeding colostrum on immunoglobulin absorption and calf survival. J Dairy Sci 1980; 63:451-456.

8. CALDOW GL, WHITE DG, KELSEY M, PETERS AR, SOLLY HJ. Relationship of calf antibody status to disease and performance. Vet Rec 1988; 122:63-65.

9. CHASE L, OTTERBY D. Raise diary replacements economically. Dairy Herd Management 1986; 1:16-19.

10. CURTIS CR, ERB HN, WHITE ME. Risk factors for calfhood morbidity and mortality on New York dairy farms. Proc Cornell Nutrition Conf 1985; 90-99.

11. DAVIDSON JN, YANCEY SP, CAMPBELL SG, WARNER RG. Relationship between serum immunoglobulin values and incidence of respiratory disease in calves. J AM Vet Med Assoc 1981; 179:708-710.

12. DONOVAN GA, BRAUN, RK. Evaluation of dairy heifer repiacement-rearing programs. Comp Contin Educ Pract Vet 1987; 9:F133-F139.

13. DUHAMEL GE, OSBURN BI. Neonatal immunity in cattle. Bovine Practitioner 1984; 19:71-77.

14. FALLON RJ, HARTE FJ, HARRINGTON D. The effect of calf purchase weight, serum IG level and feeding systems on the incidence of diarrhoea, respiratory disease and mortality. Bovine Practitioner 1987; 22:104-106.

15. FRERKING H, AEIKENS T. About the importance of colostrum for the newborn calf. Ann Rech Vet 1978; 9:361-365.

16. HARP JA, WOODMANSEE DB, MOON HW. Effects of colostral antibody on susceptibility of calves to *Cryptosporidium parvum* infection. Am J Vet Res 1989; 50:2117-2119.

17. HARTMAN DA, EVERETT RW, SLACK ST, WARNER RG. Calf mortality. J Dairy Sci 1974; 57:576-578.

18. HEINRICHS AJ, HARGROVE GL. Standards of weight and height for Holstein heifers. J Dairy Sci 1987; 70:653-660.

19. HOWARD JC, CLARKE MC, BROWNLIE J. Protection against respiratory infection by bovine virus diarrhea virus by passively acquired immunity. Vet Microbiol 1989; 19:195-203.

20. IRWIN VCR. Disease incidence in colostrum deprived calves under commercial conditions and the economic consequences. Vet Rec 1974; 94:406.

21. JENNY BF, GRAMLING GE, GLAZE TM. Management factors associated with calf mortality in South Carolina dairy herds. J Dairy Sci 1981; 64:2284-2289.

22. KARREN DB, BASARAB JA, CHURCH TL. The growth and economic performance of preconditioned calves and their dams on the farm and of calves in the feedlot. Can J Anim Sci 1987; 67:327-336.

23. KEOWN JF. What New York dairymen are telling us about the freshening age of their first-calf heifers. Northeast Improver 1984; 3:24-25.

24. LEBLANC PH, BAKER JC, GRAY PR, ROBINSON NE, DERKSEN FJ. Effects of bovine respiratory syncytial virus on airway function in neonatal calves. Am J Vet Res 1991; 52:1401-1406.

25. LOPEZ JW, ALLEN SD, MITCHELL J, QUINN M. Rotavirus and *Cryptosporidium* shedding in dairy calf feces and its relationship to colostrum immune transfer. J Dairy Sci 1988; 71:1288-1294.

26. MARTIN SW, SCHWABE CW, FRANTI CE. Dairy calf mortality rate: Influence of management and housing factors on calf mortality rate in Tulare County, California. Amer J Vet Res 1975; 36:1111-1114.

27. MARTIN SW, BATEMAN KG, SHEWEN PE, ROSENDAL S, BOHAC JE. The frequency, distribution and effects of antibodies to seven putative respiratory pathogens on respiratory disease and weight gain in feedlot calves in Ontario. Can J Vet Res 1989; 53:355-362.

28. MARTIN SW, MEEK AH, WILLEBERG P. Veterinary epidemiology: Principles and methods. Ames: Iowa State University Press, 1987.

29. McEWAN AD, FISHER EW, SELMAN IE. Observations on the immune globulin levels of neonatal calves and their relationship to disease. J Comp Path 1970; 80:250-265.

30. McGUIRE TC, PFEIFFER NE, WEIKEL JM, BARTSCH RC. Failure of colostral immunoglobulin transfer in calves dying of infectious disease. J Am Vet Med Assoc 1976; 169:713-718.

31. MECHOR GD, ROUSSEAUX CG, RADOSTITS OM, BABIUK LA, PETRIE L. Protection of newborn calves against fatal multisystemic infectious bovine rhinotracheitis by feeding colostrum from vaccinated cows. Can J Vet Res 1987; 51:452-459.

32. NAYLOR JM, KRONFELD DS, BECH-NIELSEN S, BARTHOLOMEW RC. Plasma total protein measurement for prediction of disease and mortality in calves. J Am Vet Med Assoc 1977; 171:635-638.

33. NOCEK JE, BRAUND DG, WARNER RG. Influence of neonatal colostrum administration, immunoglobulin, and continued feeding of colostrum on calf gain, health, and serum protein. J Dairy Sci 1984; 67:319-333.

34. OXENDER WD, NEWMAN LE, MORROW DA. Factors influencing dairy calf mortality in Michigan. J Am Vet Med Assoc 1973; 162:458-460.

35. PRITCHARD DG, CARPENTER CA, MORZARIA SP, HARKNESS JW, RICHARDS MS, BREWER JI. Effect of air filtration on respiratory disease in intensively housed veal calves. Vet Rec 1981; 109:5-9.

36. RADOSTITS OM, BLOOD DC. Herd health: A textbook of health and production management of agricultural animals. Philadelphia: WB Saunders, 1985.

37. ROBISON JD, STOTT GH, DeNISE SK. Effects of passive immunity on growth and survival in the dairy heifer. J Dairy Sci 1988; 71:1283-1287.

38. SMITH BP, HABASHA FB, REINA-GUERRA M, HARDY AJ. Immunization of calves against salmonellosis. Am J Vet Res 1980; 12:1947-1951.

39. SMITH T, LITTLE RB. The significance of colostrum to the new-born calf. J Exper Med 1922; 36:181-189.

40. THOMAS LH, SWANN RG. Influence of colostrum on the incidence of calf pneumonia. Vet Rec 1973; 92:454-455.

41. TIZZARD IR. An introduction to veterinary immunology. Toronto: WB Saunders, 1982.

42. WALTNER-TOEWS D, MARTIN SW, MEEK AH. The effect of early calfhood health status on survivorship and age at first calving. Can J Vet Res 1986; 50:314-317.

43. WALTNER-TOEWS D, MARTIN SW, MEEK AH. Dairy calf management, morbidity and mortality in Ontario Holstein herds. III. Association of management with morbidity. Prev Vet Med 1986; 4:137-158.

44. WILLETT GS, THOMASON E, BERNARD J. What it costs to raise dairy heifers. Hoard's Dairyman 1984; 129:1257.

APPENDIX A

Enterprise budget for a Florida dairy farm

Table 1 - The Total Herd

Weekly Report for Period... Aug 29, 1992 thru Sep 5, 1992
Date: Sep 7, 1992
Head counted: 1846

	Week Total	%	Per Head
INCOME:			
MILK	$74,352.40	100.00%	$5.75
OTHER	$ 0.00	0.00%	$0.00
GROSS	$74,352.40	100.00%	$5.75
EXPENSES:			
FEED COST	$26,344.32	35.43%	$2.04
REPLACEMENTS	$12,570.77	16.91%	$0.97
LABOUR	$10,877.00	14.63%	$0.84
INTEREST	$ 4,468.00	6.01%	$0.35
HAULING	$ 2,008.20	2.70%	$0.16
PROPERTY REPAIR	$ 1,823.00	2.45%	$0.14
PAYROLL	$ 1,650.04	2.22%	$0.13
ELECTRIC	$ 1,367.00	1.84%	$0.11
OVERHEAD	$ 1,365.00	1.84%	$0.11
DRUGS/CLEANERS	$ 1,155.00	1.55%	$0.09
BREEDING	$ 760.00	1.02%	$0.06
ADVERTISING	$ 734.71	0.99%	$0.06
COOP DUES	$ 685.73	0.92%	$0.05
DHIA	$ 624.00	0.84%	$0.05
VET	$ 512.00	0.69%	$0.04
TAXES/INSURANCE	$ 239.00	0.32%	$0.02
PARLOR REPAIR	$ 118.00	0.16%	$0.01
TOTAL EXPENSES	$67,301.77	90.52%	$5.21
NET INCOME	$ 7,050.63	9.48%	$0.55

APPENDIX A

Enterprise budget for a Florida dairy farm

Table 2 - The Replacement Herd

Weekly Report for Period... Aug 29, 1992 thru Sep 5, 1992
Date: Sep 7, 1992
Head counted: 1044

EXPENSE ITEM	$/wk	% Expenses	$/hd/d
FEED COST	$ 4,299.69	34.20%	$0.59
INTEREST	$ 2,149.84	17.10%	$0.29
LABOUR	$ 2,111.00	16.79%	$0.29
TAXES/INSURANCE	$ 900.00	7.16%	$0.12
OVERHEAD	$ 421.00	3.35%	$0.06
PROPERTY REPAIR	$ 419.00	3.33%	$0.06
FUEL/OIL/GREASE	$ 358.00	2.85%	$0.05
PAYROLL	$ 320.24	2.55%	$0.04
BREEDING	$ 314.00	2.50%	$0.04
VET	$ 278.00	2.21%	$0.04
EQUIPMENT REPAIR	$ 253.00	2.01%	$0.03
ELECTRIC	$ 83.00	0.66%	$0.01
DRUGS/CLEANERS	$ 25.00	0.20%	$0.00
FERTILIZER	$ 0.00	0.00%	$0.00
TOTAL EXPENSES:	$12,570.77	100.00%	$1.63

HEIFER COST:	Per Hd Cost	Income Lost	
22 MONTH CALVING	$ 933.40		
23 MONTH CALVING	$ 969.40		Our average
24 MONTH CALVING	$1,006.60		calving age
25 MONTH CALVING	$1,042.60	$ 36.00	is 25.18 mo;
26 MONTH CALVING	$1,079.80	$ 73.20	Our heifer
27 MONTH CALVING	$1,115.80	$109.20	rearing cost
28 MONTH CALVING	$1,153.00	$146.40	is **$1,049.08.**
29 MONTH CALVING	$1,189.00	$182.40	
30 MONTH CALVING	$1,226.20	$219.60	

APPENDIX B

Evaluation of birth weights taken at 1-4 days of age versus birth weights taken at 4-8 days of age in a group of Holstein dairy calves

Materials and Methods:

Fifty-eight calves were weighed twice using a platform scale accurate to +/-0.9 kg. The first weight (WT1) was taken between 1 and 4 days of age (day 1 being the day of birth). The second weight (WT2) was taken 3 or 4 days later (4 to 8 days of age).

Data were analysed using a paired T-test in the computerized statistical analysis program Statistix® (Analytical Software, St. Paul, MN, USA).

Results:

a) Descriptive Statistics

	WT1	WT2
N	58	58
MEAN	35.82	36.60
SD	3.82	3.44
LO 95% CI	34.82	35.69
UP 95% CI	36.82	37.50
MINIMUM	27.22	27.68
1ST QUARTILE	34.03	34.03
MEDIAN	35.39	36.30
3RD QUARTILE	38.23	39.02
MAXIMUM	47.64	45.37

b) Paired T test for WT2 - WT1

MEAN	0.7475
STD ERROR	0.1754
T STATISTIC	4.42
DF	57
P	0.0001

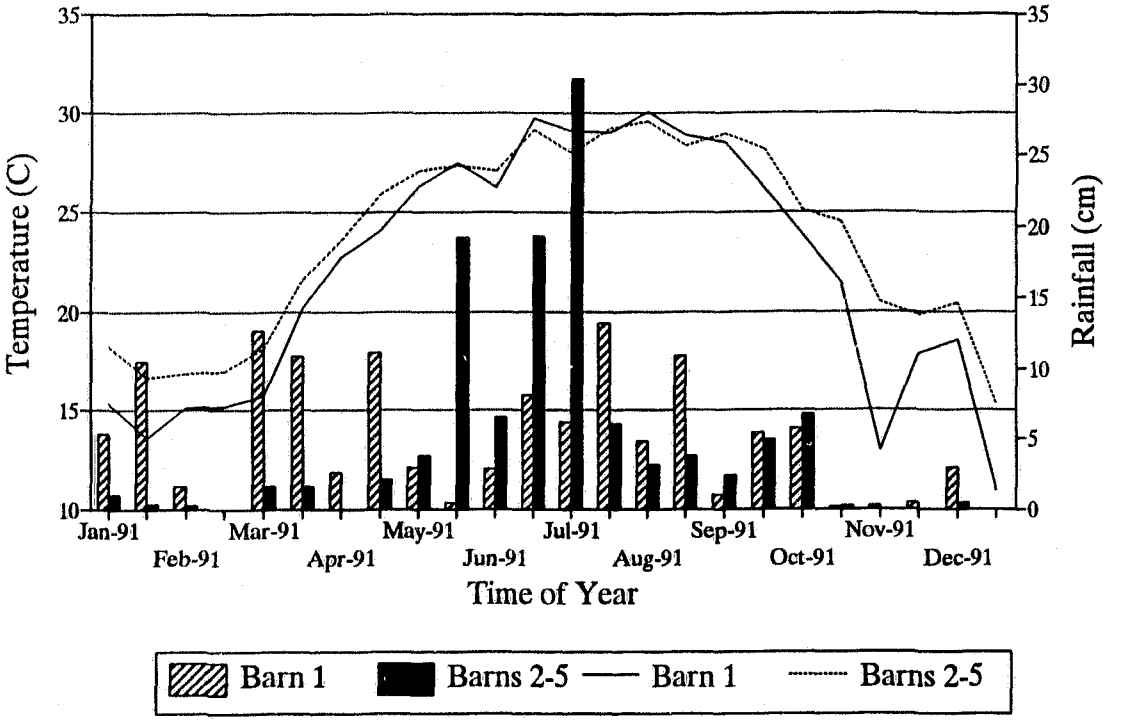
Conclusions:

Using these results, all birth weight data from calves that were 4 to 8 days of age on the day of collection was adjusted by subtracting 0.75 kg from the actual weight.

APPENDIX C

Fifteen day averages of mean daily temperature ([Min+Max]/2) and total 15-day rainfall as measured by University of Florida Institute of Food and Agricultural Sciences Agricultural Research Stations located within 80 km of farm sites. Summer is defined as the period of time when mean daily temperature is above 25° C, the upper range of thermoneutral zone of cattle.

(Bars=rainfall; Lines=temperature)

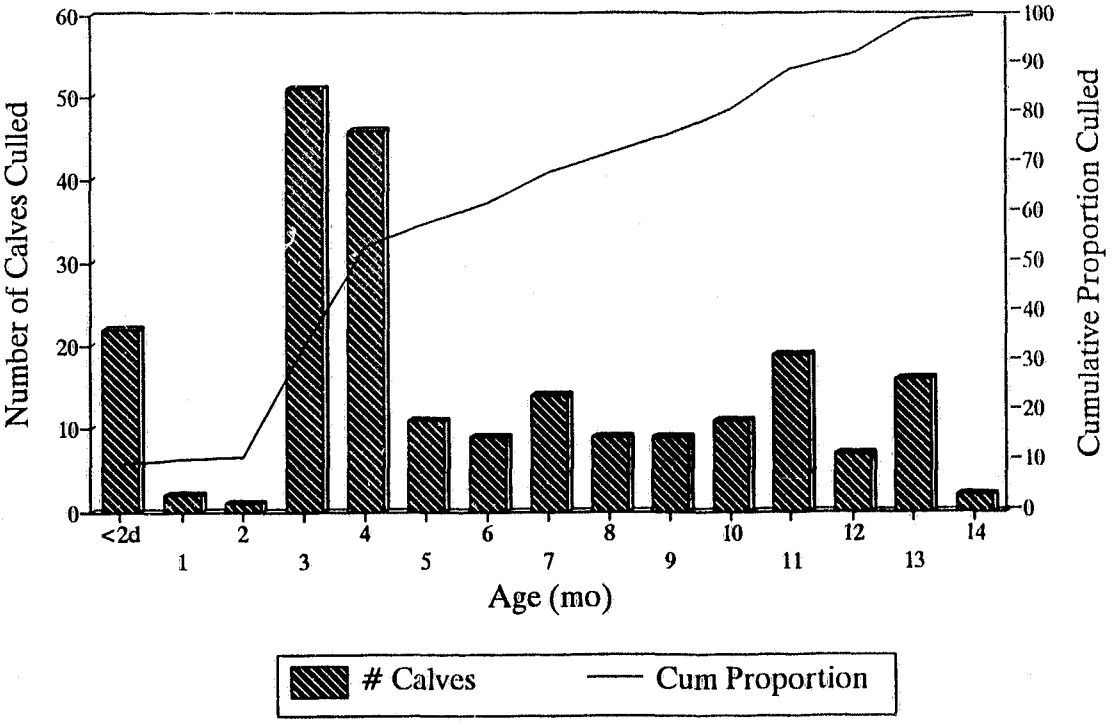


APPENDIX D

Descriptive epidemiology of mortality in a group of Holstein dairy calves

Figure 1 Culling of dairy heifers from birth to breeding age. Two periods of peak culling are noted; at birth, primarily due to size of calf and around the time calves are commingled for the first time at 3 - 4 months of age.

# calves at risk of being culled =	3253
# calves culled by 14 months =	229
Culling risk birth to 14 months =	7.0%

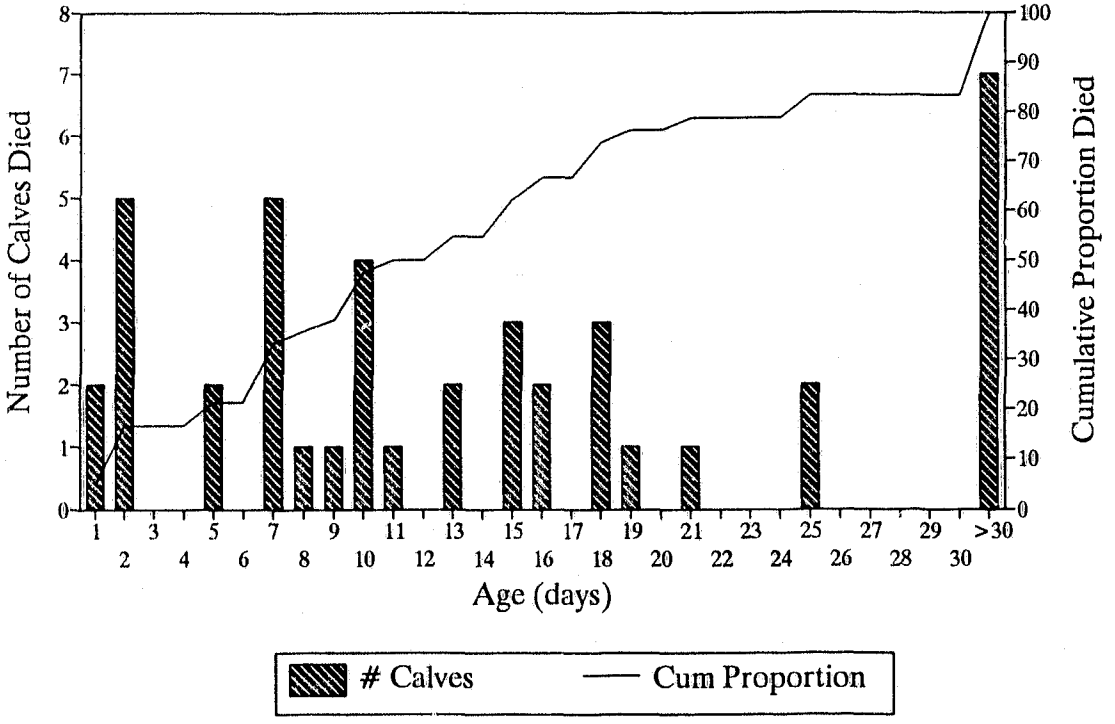


APPENDIX D

Descriptive epidemiology of mortality in a group of Holstein dairy calves

Figure 2 Age at death for dairy calves dying from diarrhea. The 7 calves that died after 30 days of age died beween 42 and 108 days. Calves dying less than 48 hrs of age are not included in statistics below.

# calves at risk of mortality due to diarrhea =	3253
# calves treated for diarrhea =	493
# calves whose death was attributed to diarrhea =	38
Mortality risk due to diarrhea (38/3253) =	1.2%
Case fatality risk =	7.7%
Age (days) 80% of cumulative proportion dead =	25

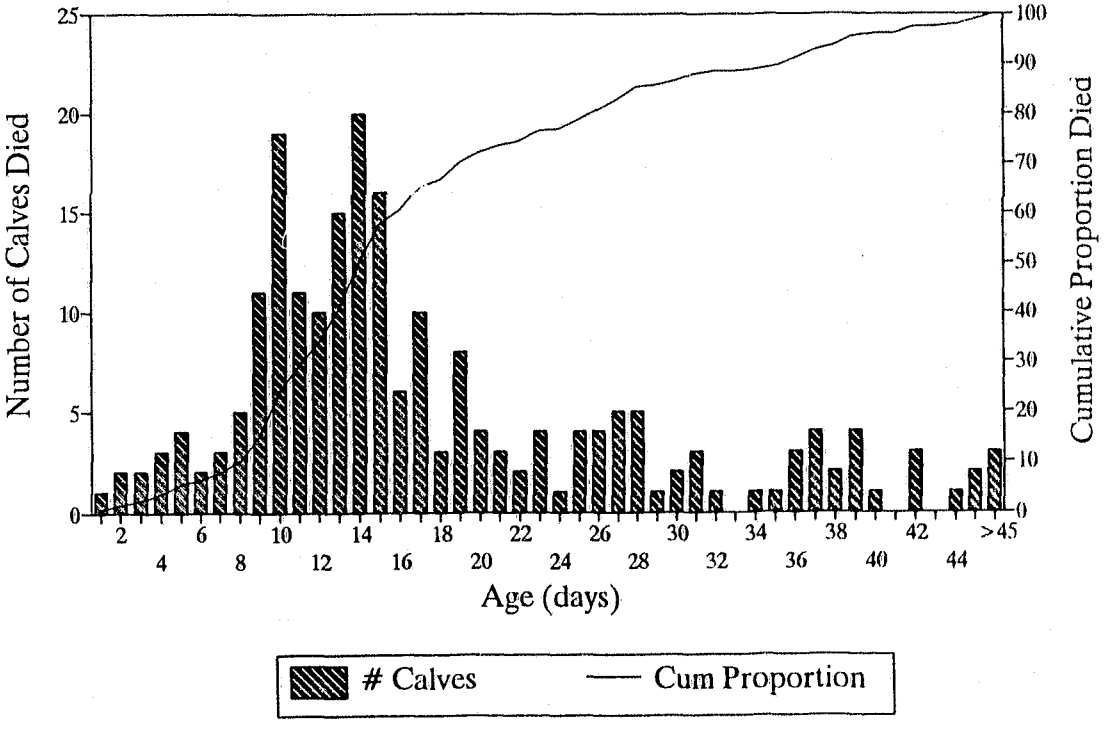


APPENDIX D

Descriptive epidemiology of mortality in a group of Holstein dairy calves

Figure 3 Age at death for dairy calves dying from septicemia. Calves that were treated for navel infection that died are included. Calves dying less than 48 hrs of age are not included in statistics below

# calves at risk of mortality due to septicemia =	3253
# calves treated for septicemia =	751
# calves whose death was attributed to septicemia =	207
Mortality risk due to septicemia (207/3253) =	6.4%
Case fatality risk =	27.6%
Age (days) 80% of cumulative proportion dead =	26

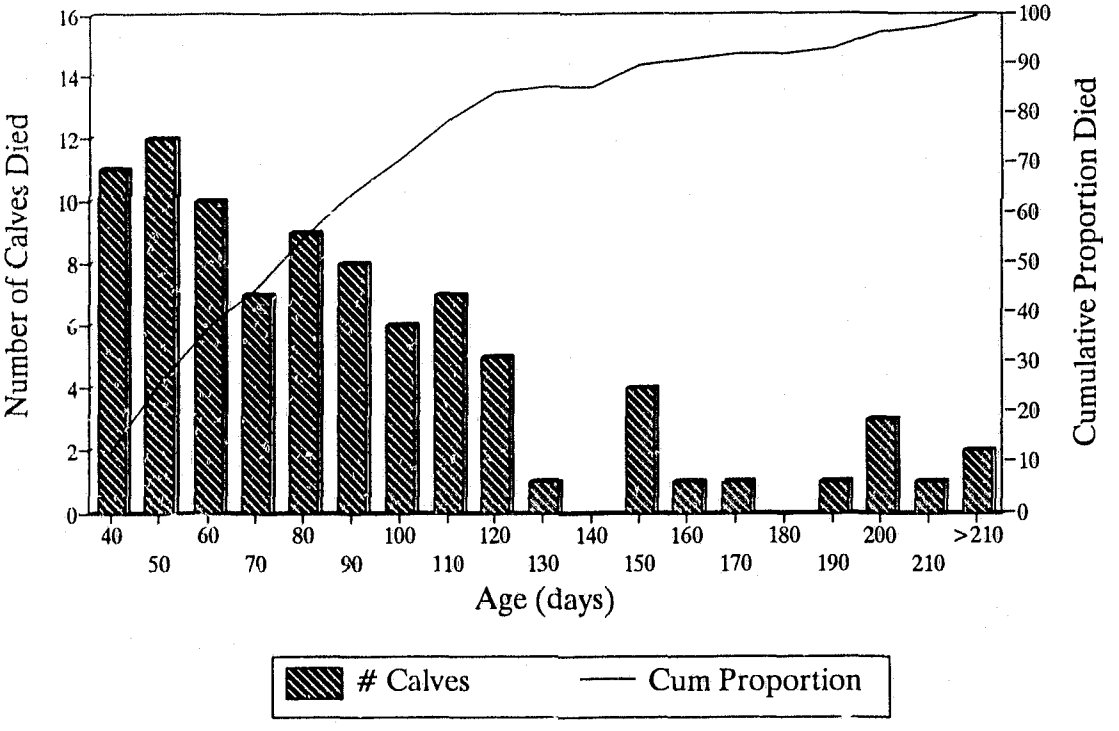


APPENDIX D

Descriptive epidemiology of mortality in a group of Holstein dairy calves

Figure 4 Age at death for dairy calves/heifers dying from pneumonia. By definition, calves whose first treatment with antibiotics was before 30 days of age were diagnosed as having septicemia

# calves at risk of mortality due to pneumonia =	2989
# calves treated for pneumonia =	600
# calves whose death was attributed to pneumonia =	83
Mortality risk due to pneumonia (83/2989) =	2.8%
Case fatality rate =	13.8%
Age (days) 80% of cumulative proportion dead =	120

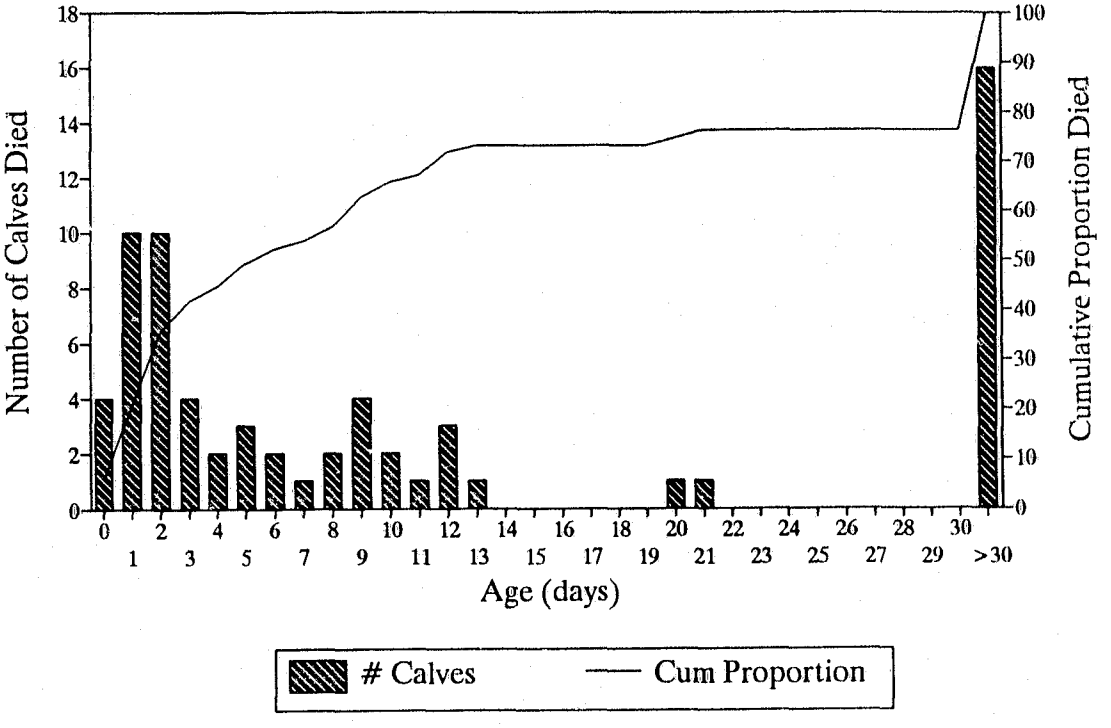


APPENDIX D

Descriptive epidemiology of mortality in a group of Holstein dairy calves

Figure 5 Age at death for dairy calves/heifers dying for unknown reasons.

Age (days) 80% of cumulative proportion dead = 42



APPENDIX E

Calculation of intraherd correlation coefficients for the dependent variables a) mortality and b) occurrence of diarrhea using the methods of Snedecor and Cochrane (55, Section 2.6, page 54).

Formula:-

$$\rho = \frac{MST - MSE}{MST + (m-1)MSE}$$

a) Intraherd correlation coefficient for mortality

Analysis of Variance Procedure

Source	DF	Sum of Squares	F Value	Pr > F
Model	4	1.6977	3.78	0.0045
Error	3282	368.3509		
Corrected Total	3286	370.0487		

$$\rho = \frac{MST - MSE}{MST + (m-1)MSE} = \frac{1.70/4 - 368.35/3282}{1.70/4 + (626.5-1)(368.35/3282)}$$

$$= \frac{0.4244 - 0.1122}{0.4244 + 625.5*0.1122}$$

$$= \frac{0.3122}{70.6266}$$

$$= \mathbf{0.0044}$$

b) Intraherd correlation coefficient for diarrhea

Analysis of Variance Procedure

Source	DF	Sum of Squares	F Value	Pr > F
Model	4	79.5382	122.21	0.0001
Error	2003	325.9044		
Corrected Total	2007	405.4426		

$$\rho = \frac{MST - MSE}{MST + (m-1)MSE} = \mathbf{0.1621}$$

APPENDIX E

Calculation of intraherd correlation coefficients for the dependent variables for occurrence of c) septicemia, d) navel infection and e) pneumonia using the methods of Snedecor and Cochrane (55, Section 2.6, page 54).

c) Intraherd correlation coefficient for septicemia

Analysis of Variance Procedure

Source	DF	Sum of Squares	F Value	Pr > F
Model	4	73.8389	93.56	0.0001
Error	3128	617.1972		
Corrected Total	3132	691.0361		

$$\rho = \frac{MST - MSE}{MST + (m-1)MSE} = \mathbf{0.1287}$$

d) Intraherd correlation coefficient for navel infection

Analysis of Variance Procedure

Source	DF	Sum of Squares	F Value	Pr > F
Model	4	16.9995	44.91	0.0001
Error	3282	310.5764		
Corrected Total	3286	327.5759		

$$\rho = \frac{MST - MSE}{MST + (m-1)MSE} = \mathbf{0.0655}$$

e) Intraherd correlation coefficient for pneumonia

Analysis of Variance Procedure

Source	DF	Sum of Squares	F Value	Pr > F
Model	4	7.0327	11.72	0.0001
Error	3282	492.2743		
Corrected Total	3286	499.3070		

$$\rho = \frac{MST - MSE}{MST + (m-1)MSE} = \mathbf{0.0168}$$

APPENDIX F

Relative risk of mortality for calves in three discrete serum total protein categories (15) in 20 day time increments. Data were analysed using Proc Lifetest in SAS. These data demonstrate that the relative risk of death is approximately equal over the first six months of life.

Age Interval (days)	Serum Total Protein (g/dl)	
	<5.0 vs >5.4	5.0-5.4 vs >5.4
0 - 20	1.80	1.20
21 - 40	3.02	2.21
41 - 60	2.97	2.85
61 - 80	1.78	1.01
81 - 100	1.85	2.12
101 - 120	0.56	1.15
121 - 140	2.26	3.45
141 - 160	1.95	0.86
161 - 180	.[a]	.[a]

[a] Mortality in calves with serum total protein greater than 5.4 g/dl was 0, so mortality risk ratios could not be calculated.

APPENDIX G

Linear regression model of factors affecting body weight gain in dairy heifers using dichotomous variables for disease conditions .

Table 1 Growth from birth to 6 months

Outcome variable = Average daily gain (kg/d)

Variable	Parameter Estimate	*P-value*
Intercept	0.5058	0.0001
Diarrhea (DIA)	-0.0703	0.0001
Septicemia (SEP)	-0.0448	0.0003
Pneumonia (PNU)	-0.0885	0.0001
Birth weight (W0)	-0.0030	0.0072
Birth height (HP0)	0.0041	0.0095
Age at 6 mo (Age6)	0.0008	0.0038
Barn 1	-0.1129	0.0001
Barn 2	-0.0487	0.1796
Barn 3	-0.0594	0.0785
Barn 4	-0.0319	0.3841
Barn 5	0.0000	.
Season	-0.0422	0.0001
$R^2 = 0.1663$		

Table 2 Growth from 6 to 14 months of age

Outcome variable = Average daily gain (kg/d)

Variable	Parameter Estimate	*P-value*
Intercept	1.2173	0.0000
Weight Gain 0-6 mo	-0.1706	0.0001
Age at 14 mo (Age14)	-0.0009	0.0001
Pneumonia 0-6 mo (PNU1)	-0.0142	0.0237
$R^2 = 0.2096$		

APPENDIX H

Linear regression model of factors affecting growth in stature (pelvic height) in dairy heifers using dichotomous variables for disease conditions.

Pelvic height grow from birth to 6 months

Outcome variable = Average daily pelvic height gain (cm/d)

Variable	Parameter Estimate	*P-value*
Intercept	0.4640	0.0001
Diarrhea (TT1)	-0.0123	0.0001
Septicemia(TT2)	-0.0061	0.0064
Pneumonia(TT3)	-0.0113	0.0001
W0	0.0005	0.0074
HP0	-0.0041	0.0001
Season	-0.0044	0.0046
R^2 = 0.2891		

APPENDIX I

Logistic regression model of the association between height at the pelvis and first service conception rate in Holstein dairy heifers. Data were collected from heifers from Barn 1 as described in this study (Section 2.2.1, page 18)

Variable	Parameter Estimate	Odds Ratio	*P-value*
Intercept	-3.1339		0.0001
Breeding season	1.2150	3.37	0.0001
IBR vaccination status	1.8845	6.58	0.0002
Pelvic height (cm)			
<122.0	0.0000		
122.0-124.5	-0.0325	0.97	0.8997
124.5-127.0	0.1959	1.22	0.3247
127.0-129.5	0.4868	1.63	0.0249
129.5-132.0	0.4069	1.50	0.0824
132.0-134.5	0.7700	2.16	0.0324
134.5-137.0	1.1330	3.10	0.1102

[a] Hosmer-Lemeshow goodness-of-fit statistic = 6.54 (P>0.10)