Influence of Feeding Pooled Colostrum or Colostrum Replacement on IgG Levels and Evaluation of Animal Plasma as a Milk Replacer Protein Source

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(ABSTRACT)

Newborn Holstein (n = 48) and Jersey (n = 30) calves were studied to compare the absorption of immunoglobulin G (IgG) from maternal colostrum (n = 39) or a colostrum replacement product derived from bovine serum (n = 39). Calves were also fed milk replacer with (n = 38) or without (n = 40) animal plasma to 29 d of age to determine the effect of plasma protein on IgG status, health, and growth. Colostrum or colostrum replacement was fed at 1.05 and 13.5 h of age and provided a total of 250, 180, 249, or 186 g IgG for Holsteins and Jerseys fed replacement or colostrum, respectively. Milk replacer (12.5% DM) was fed at 31% of metabolic birth weight (2 feedings/d). Jugular blood was sampled at 0 h, 24 h, and weekly to determine plasma IgG. At blood collection calves were weighed and measured to determine growth. Health scores, fecal scores, and grain intake were measured daily. Mean plasma IgG at 24 h did not differ between calves fed colostrum (13.78 ± 0.39 g/L) and replacement (13.96 ± 0.38 g/L). Average daily gain, wither height, hip height, body length, heart girth, health, and incidence of diarrhea were not different between treatment groups. Plasma IgG and performance were not affected by addition of animal plasma to milk replacer. The colostrum substitute successfully replaced colostrum as the source of IgG for newborn calves. Animal plasma was an acceptable source of protein, but did not enhance growth or immunity.

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ABBREVIATIONS

IgG Immunoglobulin G

IgA Immunoglobulin A

IgM Immunoglobulin M

IgE Immunoglobulin E

FPT Failure of passive transfer

WPC Whey protein concentrate

NAHMS National Animal Health Monitoring Service

NDHEP National Dairy Heifer Evaluation Project

RP Treatment on which calves were fed colostrum replacement and milk replacer with animal plasma

RN Treatment on which calves were fed colostrum replacement and milk replacer without animal plasma

CP Treatment on which calves were fed colostrum and milk replacer with animal plasma

CN Treatment on which calves were fed colostrum and milk replacer without animal plasma (control)

IGF-I Insulin-like growth factor-I

GH Growth hormone

PUN Plasma urea nitrogen

AEA Apparent efficiency of absorption

DMI Dry matter intake

CHAPTER 1. INTRODUCTION

The importance of colostrum in determining calf health and survival is well established. Timely, adequate colostrum intake is the single most important management factor affecting morbidity and mortality in preweaned calves. The calf has essentially no immune protection at birth because maternal immunoglobulins cannot cross the placenta, and the calf's own immune system is functionally immature. Therefore, the calf's immune system must be fortified, via colostrum, to fight infections effectively. Colostrum provides antibodies, primarily immunoglobulin G (**IgG**), that protect the neonate until the calf's immune system becomes fully functional. The two most important factors affecting the amount of IgG absorbed from colostrum are the time of first feeding and the amount of IgG consumed.

A national survey of heifer management practices in 1991 by the National Animal Health Monitoring System found that 8.4% of dairy heifers born alive died prior to weaning. A follow-up survey in 1995 found the mortality rate had increased to 10.8%. Considering that a well-managed herd can achieve mortality of less than 5%, these numbers indicate that the dairy industry has difficulty managing newborn calves.

Management of the newborn calf can be complicated by disease risk and high milk production. Johne's disease, which is present in an estimated 20 to 40% of U.S. dairy herds, is transmitted to calves through feces, colostrum, and milk. Therefore, colostrum from animals with Johne's infections should not be fed to calves. Particularly in herds with widespread incidence of the disease, dumping all colostrum from infected cows can greatly reduce the amount of available colostrum. In addition to disease concerns, continued selection for higher milk production may reduce the quality of available colostrum. Research at Washington State has shown that first milking production of greater than 8.5 kg is associated with lower concentration of IgG in colostrum. The greater volume of colostrum dilutes the IgG concentration. Therefore, feeding colostrum from a cow with high first milking production to calves in the same volume as from a cow with average production will result in less total IgG.

In response to concerns about management of newborns, disease, and reduced colostral quality, several companies have developed products to supplement colostrum. These products vary in composition, and the range of ingredients includes dried colostrum, dried whey, purified colostral immunoglobulins, and purified blood proteins. Blood proteins cost less than milk proteins, but more than commonly used soy and wheat proteins, and interest in utilizing these blood proteins has increased in the past 5 yr due to their favorable amino acid profile. Unfortunately, supplements tend to provide less IgG than colostrum, and have not been widely accepted or used by producers. In general these products are not recommended for use as colostrum replacements, meaning they are usually added to colostrum, not fed in lieu of it. Since bacteria or viruses can be transmitted in colostrum, the practice of supplementing colostrum does not alleviate concerns about colostrum as a vehicle for spreading disease. On the other hand, true replacement of colostrum with a synthetic product may be able to break the cycle and prevent calves from acquiring diseases from their dams.

Interest in using readily available animal proteins in other ways has also increased. Milk replacer feeding and formulation are driven by economics. If animal proteins could effectively substitute for milk proteins, then the cost of milk replacer could be reduced without detrimentally affecting its nutritional qualities. Actually, it has been suggested that feeding animal proteins increases the nutritional value of milk replacer due to the provision of immunoglobulins. These immunoglobulins may exert a local protective effect in the intestine that would improve animal health and performance. In addition, because IgG absorbed from colostrum into blood is largely returned to the gastrointestinal tract, it is possible that IgG contained in animal protein could reduce the clearance of IgG from blood by equilibrating vascular and extravascular IgG pools. This balance could increase the calf's immune protection level against both blood-borne and enteric pathogens.

This investigation compared the absorption of IgG from colostrum and from a serum protein-based colostrum replacement made by American Protein Corporation, Inc. (LifelinePlus). The amount of colostrum fed was adjusted to provide a total dose of IgG equal to that provided by the replacement. Plasma concentration of IgG from calves fed milk

replacer with and without animal plasma was compared as well. Interaction of the IgG source and the milk replacer was investigated to provide additional information about the effectiveness of using animal plasma in milk replacer to enhance passive immunity.

It was hypothesized that calves would absorb more IgG from colostrum than from the replacement product, and that the use of animal plasma in milk replacer would decrease the rate of IgG clearance and exert a local protective effect in the digestive tract. Further hypothesis proposed that calves fed colostrum replacement would receive greater benefit from animal plasma in milk replacer than calves fed colostrum. This interaction was predicted based on the expectation that initial absorption of IgG from the replacement product would be lower than from colostrum.

Objectives of the study were as follows:

1. Compare the 24-h IgG concentrations in calves fed colostrum or a colostrum replacement product.

2. Compare the plasma IgG status and calculated IgG clearance rate of calves fed milk replacer with or without animal plasma.

3. Estimate the clearance rate of passively acquired IgG using changes in IgG concentrations over the first month of life.

4. Determine correlations between diet, supplement, growth and blood components that may be related to IgG:
    a. Plasma urea nitrogen, which indicates protein utilization and metabolism
    b. Insulin-like growth factor-I, which may be associated with IgG absorption and is associated with growth
    c. Growth hormone, which is associated with IGF-I and growth

5. Determine growth patterns of young calves including measurements of growth rate (weight, height at withers and hips, body length and heart girth), feed efficiency, and starter intake.

6. Determine the relationship between IgG status, growth, and disease in the first month of life.

CHAPTER 2. LITERATURE REVIEW

Neonatal Immune Status

The immune status of the newborn calf is widely accepted as hypogammaglobulinemic (Roy, 1990). The syndesmochorial structure of the bovine placenta prevents passage of immunoglobulins from maternal circulation into fetal circulation (Senger, 1997). Thus, a newborn calf has virtually no passively acquired immunoglobulins to fight infections. In addition the calf's own immune system is classified as functionally immature; the humoral immune system is incapable of mounting an effective response to invading pathogens (Roy, 1990). Hypogammaglobulinemia in the neonatal calf must be overcome quickly to protect the calf from early infection by bacteria present in the birth environment (Acres, 1985). Passive immune protection is transferred to the calf by ingestion and absorption of intact immunoglobulins, particularly immunoglobulin G (**IgG**), from colostrum. Once absorbed, immunoglobulins equilibrate to vascular and extravascular pools in a ratio of approximately 1:1 (Kruse, 1970; Payne et al., 1967). Reportedly, 68% of cleared immunoglobulin was returned to the lumen of the intestine each d over an 8 d period (Besser et al., 1988), where it retained antigen-binding activity and exerted a local protective effect (Besser, 1993; Besser et al., 1988). When determined by clearance of ^{125}I-labeled IgG_1, passively acquired IgG has an estimated half-life of 11.5 to 17.9 d (Besser, 1993; Besser et al., 1988; Sasaki et al., 1977). When half-life is determined by the decrease in plasma IgG_1 concentration, it appears to be 19.9 to 21.5 d (Logan et al., 1972; Sasaki et al., 1977). Endogenous production of IgG began at 4 wk of age in calves with high initial IgG concentrations (Logan et al., 1974). However, in colostrum-deprived and hypogammaglobulinemic calves, endogenous production began within a wk of birth (Logan et al., 1974). Husband et al. (1972) showed that endogenous production of IgG_1 and IgG_2 began 8 to 16 d after birth. Using ^{125}I-labeled IgG_1, Devery et al. (1979) found endogenous IgG_1 production of 1 g/d began around 36 h and continued up to 3 wk of age.

In summary, the newborn calf has virtually no immune protection at birth and receives passive immunity from colostral immunoglobulins. Absorbed immunoglobulin is cleared from the body, and the majority is returned to the intestinal lumen, where it protects

the mucosal surface. The half-life IgG_1 is 11.5 to 17.9 d, and endogenous production of IgG_1 begins as early as 3 d after birth.

Bovine Immunoglobulins

Bovine mammary secretions contain four classes of immunoglobulins. The same immunoglobulins are present in colostrum and in milk, but they are found in much higher concentration in colostrum. While milk has less than 1 g/L of immunoglobulins, colostrum typically contains 50 to 100 g/L (Larson et al., 1980; Roy, 1990). Immunoglobulin G comprises 85 to 90% of colostral immunoglobulin. Two subclasses of IgG are present in the bovine: IgG_1 and IgG_2. Immunoglobulin G_1 is found abundantly in colostrum (80 to 90% of all immunoglobulins), but the two subclasses are found in approximately equal amounts in the blood (Butler, 1983; Roitt et al., 1998; Roy, 1990). Immunoglobulin G_1 is the major antibody of secondary immune responses, fixes complement, acts as the principle opsonin for macrophages, and is the primary immunoglobulin involved in transferring passive immunity to the neonate (Butler, 1983; Butler, 1969; Roitt et al., 1998). The mammary gland selectively transports IgG (primarily IgG_1) in large amounts from the blood to colostrum via an intracellular transport mechanism (Larson et al., 1980). Immunoglobulin G_2 fixes complement, mediates the cytotoxicity of poly-morphonuclear neutrophils, and precipitates antigen (Butler, 1983). Immunoglobulins A (**IgA**) and M (**IgM**) are also found in colostrum, although in much smaller quantities. The secretory form of IgA, which is a dimer connected by a J-chain and attached to the secretory component, comprises about 5% of colostral immunoglobulins (Butler, 1969; Larson et al., 1980). Immunoglobulin A protects the surface of mucosal membranes, including the intestine, and prevents pathogens from attaching to the surface of cells (Butler, 1983; Roitt et al., 1998). Immunoglobulin M is a pentamer that makes up 7% of colostral immunoglobulins. Immunoglobulin M is the primary protective mechanism against septicemia, fixes complement, and is the major agglutinating antibody (Butler, 1969; Larson et al., 1980). Both IgA and IgM are synthesized locally by the mammary gland and concentrated in colostrum (Butler, 1969; Larson et al., 1980). Immunoglobulin E (**IgE**) is also present in bovine colostrum and can be transferred to the neonate. The role of IgE is less understood than the other immunoglobulins, but it does have skin-sensitizing activity (Butler, 1983).

Colostrum Composition and Quality

Colostrum is a mixture of lacteal secretions and components of blood serum, primarily immunoglobulins and other proteins, which accumulates in the mammary gland during the prepartum period and is harvested immediately after parturition (Foley and Otterby, 1978). Although milk produced during the first three d postpartum is unsalable, only the first milking produces true colostrum containing the highest amount of IgG (Foley and Otterby, 1978). From the perspective of the newborn dairy calf, colostrum quality is determined by the amount of immunoglobulin it provides. Colostrum contains 50 to 200 times more IgG, 60 to 100 times more IgM, and 25 to 85 times more IgA than milk (Foley and Otterby, 1978; Norcross, 1982; Roy, 1990). Colostrum has two times more total solids, two times more fat, and four times than whole milk (Foley and Otterby, 1978). Colostrum also contains a number of growth factors and non-specific antimicrobial factors in higher concentration than milk (Koldovský, 1989; Odle et al., 1996; Reiter, 1978). Insulin-like growth factors I and II, epidermal growth factor, insulin, and prolactin concentrations are elevated in colostrum compared to milk (Koldovský, 1989; Odle et al., 1996). Concentrations of insulin-like growth factor-I in colostrum range from 4 to 62 times those in milk, and epidermal growth factor is 2 to 4 times higher (Odle et al., 1996). In addition colostrum contains lysozyme, lactoferrin, and the components of the lactoperoxidase/thiocyanate/hydrogenperoxide system in quantities greater than those in milk (Reiter, 1978). These antimicrobial substances provide non-specific protection against infection and may aid the newborn during the gap between passive immunity and the development of the active immune system (Reiter, 1978).

Colostral composition and quality vary according to breed, parity, season, production at first milking, postpartum milking number, prepartum milking or leakage, and the health of the mammary gland (Devery-Pocius and Larson, 1983; Foley and Otterby, 1978; Kume and Tanabe, 1993; Maunsell et al., 1998; Muller and Ellinger, 1981; Pritchett et al., 1991; Quigley et al., 1994; Shearer et al., 1992). Jerseys have higher concentrations of IgG than Holsteins (Muller and Ellinger, 1981; Parrish et al., 1950; Quigley et al., 1994; Shearer et al., 1992), and in a small survey (Muller and Ellinger, 1981), Holsteins (n = 19) had the lowest

IgG concentration of the five breeds compared. Cows in third or greater lactation have higher IgG content and a broader range of antibodies to more pathogens, regardless of breed (Devery-Pocius and Larson, 1983; Oyeniyi and Hunter, 1978; Pritchett et al., 1991; Quigley et al., 1994; Shearer et al., 1992). First milking production greater than 8.5 kg is associated with lower IgG concentrations due to dilution (Pritchett et al., 1991). The increased concentrations of colostral components are greatest in the first milking postpartum and decline quickly to normal concentrations by the fourth postpartum milking (Parrish et al., 1950; Stott et al., 1981). Cows milked intensively, or that experience excessive colostrum leakage before calving, produce colostrum with reduced immunoglobulin content (Petrie, 1984). Most reports found no effect of season on colostral immunoglobulins (Roy, 1990). However, Shearer et al. (1992) observed that cows calving in August or September produced colostrum with higher IgG concentration. Caution must be taken when interpreting these (Shearer et al.) results since IgG concentrations were measured using a colostrometer. Measurements were made immediately after colostrum was removed from the cow. While the colostrometer is a useful tool to estimate colostral IgG content, its accuracy can be affected by temperature (Mechor et al., 1991). If the colostrum was not measured at a uniform temperature, results could be skewed. Nardone et al. (1997) observed that heifers exposed to heat stress in the last 90 d of gestation and in the first wk postpartum produced colostrum with lower IgG, IgA, total protein, and fat than non-heat-stressed heifers. While prepartum mastitis infection did not decrease IgG concentrations, it did increase somatic cell count and probably increases the number of bacteria present, which may make colostrum from persistently infected cows harmful to calf health (Maunsell et al., 1998; Roy, 1990). As previously mentioned, colostrum quality is determined by IgG content. Generally accepted quality standards are: less than 20 g/L, poor quality; 20 to 50 g/L, moderate quality; and greater than 50 g/L, excellent quality (Shearer et al., 1992; Stott and Fellah, 1983).

Mechanism of Absorption

At birth the cells lining the calf's small intestine are immature and are able to absorb large molecules, such as immunoglobulins, intact. This absorption process had been thought to occur only in the terminal portion of the small intestine (Comline et al., 1951a; Hardy, 1969; Staley et al., 1972). However, later work found that absorption occurred throughout the

small intestine (James et al., 1979; Jochims et al., 1994), although absorptive activity increased from duodenum to ileum and was greatest in the ileum. The absorption mechanism is a combination of micropinocytosis and receptor-mediated transport (Jochims et al., 1994). Micropinocytotic absorption occurs when molecules come into contact with the surface of intestinal villus cells. Pinocytosis of the molecules is followed by entrance into the apical tubular complex and encapsulation in vacuoles. The vacuoles then migrate to the basal membrane and release their contents into the extracellular space (exocytosis) (Comline et al., 1951a; Hardy, 1969; James, 1978; Staley et al., 1972). Once in the extracellular space, immunoglobulins are absorbed into the villus lacteals and travel to the thoracic duct, where they enter the bloodstream. Complete absorption from the intestinal lumen to the bloodstream takes 1 to 2 h (Balfour and Comline, 1962; Comline et al., 1951b).

Other evidence shows that some immunoglobulin is also absorbed via receptor-mediated transport. Using electron microscopy to describe intestinal ultrastructure, Staley et al. (1972) observed coated vesicles, which are associated with receptor-mediated transport, in the ileum. Staley et al. reported that ferritin was not absorbed into jejunal villus cells, but these cells took up a ferritin-IgG complex. Ileal cells absorbed both ferritin and ferritin-IgG, but neither was transported into the blood. At the same time, IgG that was not attached to ferritin passed into the intestinal cells and appeared in the bloodstream. They (Staley et al.) concluded that the intestinal cells exhibited a degree of selectivity based on protein structure of molecules. Jochims et al. (1994) used immunoelectron microscopy to confirm the findings of Staley et al. (1972). Jochims et al. observed clathrin, a protein associated with receptor-mediated endocytosis, in the coated vesicles of ileal cells. Using gold-labeled protein Jochims et al. saw clathrin bound to IgG in the duodenum and jejunum. Jochims et al. observed that feeding colostrum stimulated pinocytosis by the apical membrane. Pinocytosed IgG molecules were then moved by peristaltic contractions through the apical tubular complex to the basal membrane.

Closure and Period of Absorption

Intestinal villus cells lose the ability to absorb intact macromolecules soon after birth. The termination of intestinal permeability to immunoglobulins was termed closure by Leece

and Broughton (1973). The definition, “cessation of the uptake or internalization of macromolecules via pinocytosis into intestinal epithelium,” divides the loss of absorptive capability into the same two phases as the absorption mechanism; movement from the gut lumen to the villus cell, followed by movement from the cell to the circulatory system. The closure process apparently occurs in reverse, transport to the blood ends first, and eventually villus cells stop absorbing molecules from the intestinal lumen (Bush and Staley, 1980). Jochims et al. (1994) described the closure process as multifactorial. Mature cells replace fetal enterocytes; the cessation of transfer begins in retrograde; and lysosomic proteolysis within intestinal cells increases. First, the basal or lateral membrane fails to discharge protein. This soon leads to an overloading, and ultimately the destruction of, transport vesicles, leaving free IgG in the cytoplasm. Lysosomes, which first appeared 24 h after birth, then destroy the IgG, preventing its transfer into the blood (Jochims et al. 1994).

The factors that influence the length of the absorptive period and the time of closure in the calf remain obscure. In the piglet nutritional factors associated with colostrum intake have been shown to affect the length of the absorptive period. Colostrum intake precipitated closure, and delaying intake of colostrum delayed closure by several h (Leece et al., 1964; Payne and Marsh, 1962; Werhan et al., 1981; Zhang et al., 1998). However, in the calf, investigations of nutritional factors have found conflicting results. Early work by Deutsch and Smith (1957) attempted to extend the period of absorption by feeding lactose-dextrose mixtures or Aminosol instead of colostrum, but were unsuccessful. They concluded that a specific factor in milk was not responsible for initiating closure. The same workers also examined the effects of deoxyribonucleic acid hydrolysis and trypsin activity and found no difference in closure times. McCoy et al. (1970) reported no delay in closure time past 31 h due to starvation or maintenance on a 1% glucose diet. On the other hand, Stott et al. (1979a) estimated the time of closure for calves fed at birth compared to first feedings at 4, 8, 12, 16, 20, and 24 h. They observed that as the feeding of colostrum was delayed, the estimated time of closure also was delayed. However, the length of the period of absorption also decreased as first feeding time was delayed. Calves fed at birth experienced closure at 21 h and had a 21 h period of absorption. Calves fed at 24 h experienced closure at 33 h, but had an 8 h period of absorption. In addition, Stott et al. (1979b) found that the rate of absorption

following first feeding was high, regardless of age at first feeding. Stott et al. (1979a) concluded that although feeding colostrum did accelerate closure, spontaneous closure occurred in colostrum-deprived calves at about 24 h of age. Therefore, early feeding of colostrum shortens the period of absorption by 2 to 3 h, but late feeding shortens the absorptive period by 6 or more h. Calves fed after 12 h may experience spontaneous closure before any absorption occurs (Stott et al., 1979a). Michanek et al. (1989) observed that within the first 8 h of life, closure appears to begin after ingestion of large amounts of colostrum regardless of the time of first feeding. More recently, Tyler and Ramsey (1993) proposed that closure depends on the availability of an energy substrate. They treated calves with exogenous insulin to induce hypoglycemia, and reported a delay in the time of closure for insulin-treated calves (40 h versus 29 h for saline-treated calves). Furthermore, calves deprived of colostrum and treated with insulin had a greater delay in closure than calves either deprived of colostrum or treated with insulin. Tyler and Ramsey concluded that closure in calves is likely similar to closure in pigs and lambs and is controlled by nutritional factors.

The mechanism of closure and factors affecting it remain uncertain, but the time of closure is generally accepted to be 24 h. Stott et al. (1979a) used blood samples collected at 4-h intervals for 40 h to perform join point analysis and determine the time of closure. Closure was estimated at about 24 ± 4 h of age and did not differ for IgG, IgM or IgA. Penhale et al. (1973) found similar closure times by extrapolating linear data collected in the first 9 h of life. However, these workers reported that the time of closure differed for each class of immunoglobulin (IgG 27 h, IgM 16 h, IgA 22 h). Calves deprived of colostrum experienced spontaneous closure at about 24 h (Stott et al., 1979a). In addition, a majority of the work investigating closure has shown it cannot be delayed past 36 h. The more recent work by Tyler and Ramsey (1993) is interesting in light of previous work and the known impact of glucose on closure time in piglets. Further investigation into the factors affecting closure in the calf bears merit. The results of these studies (Penhale et al., 1973; Stott et al., 1979a) have led to recommendations that calves receive colostrum as soon as possible after birth to maximize IgG absorption.

Amount of IgG Absorbed

The amount of IgG that calves absorb from colostrum depends on several factors. The most important concerns are time of first feeding and mass of IgG consumed. Stott et al. (1979a) reported that 57% of calves fed colostrum after 24 h were unable to absorb colostral immunoglobulins. If feeding was delayed 12, 16 or 20 h, 3, 17, and 30% of calves showed no immunoglobulin absorption. On the other hand, all calves fed before 12 h absorbed all three classes of immunoglobulin. Kruse (1970) found absorption was reduced linearly to about one half by delaying feeding from 2 to 20 h. Smith and Erwin (1959) observed that calves 48 to 60 h old did not absorb colostral immunoglobulins. Age at initial feeding also affects the rate of absorption. After 12 h of age the absorption rate progressively declined with increasing age (Stott et al., 1979b).

The mass of IgG consumed is another important factor in determining how much IgG a calf absorbs. Kruse (1970) reported that mass of IgG fed accounted for 50% of the variation between calves in serum IgG concentration. The mass of IgG consumed is affected by volume of colostrum fed, IgG concentration of the colostrum, and efficiency of absorption of colostral IgG. Increasing the amount fed from 0.5 L to 2 L linearly increased serum IgG concentration in calves (Stott et al., 1979c). On the other hand, Morin et al. (1997) reported that doubling the volume of colostrum fed at birth did not increase serum IgG at 48 h (Morin et al., 1997). However, feeding an additional time (at 6 h) did increase IgG at 48 h. Hammon and Blum (1998) observed prolonged elevation of IgG concentrations from continued feeding of colostrum, but Petrie (1984) found no significant increase in IgG concentrations if calves were allowed to suckle at 0 h and again at 12 h. Morin et al. (1997) showed that higher colostrum concentrations of IgG increased serum IgG at 48 h. Therefore, they concluded that it was advantageous to feed a high volume of high quality colostrum at birth to improve the calf's immune status. Others (Besser et al., 1985; McEwan et al., 1970; Petrie, 1984; Stott and Fellah, 1983) also reported a positive relationship between serum IgG and colostrum IgG concentrations. While colostral IgG concentration does influence serum IgG concentration, its effect on efficiency of absorption is less clear. Both linear (McEwan et al., 1970; Stott and Fellah, 1983) and curvilinear (Besser et al., 1985; Besser et al., 1991) relationships have been reported for colostral and serum IgG concentrations. If the relationship is linear, then the

efficiency of absorption is constant, and the limit on IgG absorption is probably outside the range of typical IgG intake. If, however, the relationship is curvilinear, then IgG absorption may have an upper limit and absorption may become less efficient above a maximum amount of IgG fed (Quigley and Drewry, 1998).

Many other factors have been investigated to determine their influence on the amount of IgG that calves absorb. Considerable research effort has been directed towards corticosteroids (Deutsch and Smith, 1957; James, 1978; Johnston and Oxender, 1979; Patt, 1977; Rafai et al., 1981; Stott and Reinhard, 1978) due to findings in rodents and pigs that showed high cortisol concentrations decreased immunoglobulin absorption (Halliday, 1959). Some research showed that high cortisol concentrations decreased IgG absorption in calves (Rafai et al., 1981), but others found the opposite. James (1978) and Johnston and Oxender (1979) observed increased IgG absorption when calves had increased cortisol concentrations. However, the prevailing theory is that corticosteroid concentrations in the calf at birth have no effect on IgG absorption (Roy, 1990; Stott, 1980). Stott (1980) proposed that the reason corticosteroids affect the rodent, but not the calf, is the difference in the normal period of absorption between the two species. Rodents normally have absorption periods of up to 21 d (Morris and Morris, 1977), which would allow more time for cortisol to have an effect. Stott (1980) also suggested that although both induced and naturally occurring hyperadrenalemia have been shown to have no effect on intestinal absorption, there is evidence that stressful conditions imposed on the dam prepartum may affect the calf's absorption capabilities. This effect could be due to cortisol affecting intestinal cells at an earlier stage of maturation, more similar to the effect seen in rodents (Stott 1980). Factors such as nutritional status of the dam and induced calving fall into this category of prepartum influences.

Gillette and Filkins (1966) found evidence that in dogs the time of cortisol exposure was a factor influencing absorption. Colostrum-deprived puppies injected with cortisol at birth had antibody absorption similar to control puppies. On the other hand, when bitches were injected with ACTH or cortisol just prior to delivery, puppies from treated bitches had lower absorption than controls. Similar results were found in cattle treated with a slow-release corticosteroid used to induce parturition (Brandon et al., 1975; Husband et al., 1973).

Calves born to induced cows had inhibited absorption of colostral IgG. Furthermore, in trials using dexamethasone and betamethasone, more rapid acting glucocorticoids, Muller et al. (1975) and Langley and O'Farrell (1976) were unable to show a difference in IgG absorption in calves. The faster action of these compounds and the shorter period of cortisol action on the calf may have caused the difference in results (Stott, 1980). Poor nutrition during the prepartum period reduced IgG absorption in the calf as well (Appleman and Owen, 1975; Hough et al., 1990). However, other researchers have shown no difference in immunoglobulin absorption in calves from dams with restricted prepartum diets (Fishwick and Clifford, 1975; Halliday et al., 1978; Olson et al., 1981b).

Stress on the newborn calf itself has been investigated for its effect on immunoglobulin absorption. Donovan et al. (1986) reported that dystocia decreased absorption of colostral immunoglobulins, but Stott and Reinhard (1978) found no difference in immunoglobulin absorption between dystocial and eutocial calves, even though cortisol concentrations in both groups were high enough to indicate hormonal stress. Environmental stressors imposed on the calf have produced conflicting results as well. Extreme temperatures, both hot (Donovan et al., 1986; Stott et al., 1972; Stott et al., 1976) and cold (Gay et al., 1983; Olson et al., 1981a; Rafai et al., 1981) reduced immunoglobulin absorption. However, moderately cold temperatures had no effect on absorption (Olson et al., 1981a; Olson et al., 1981b). Calves born into dirty or crowded environments with high pathogen loads are at greater risk for reduced absorption of IgG. If bacteria enter the small intestine before colostrum is fed, they can be absorbed readily (Corley et al., 1977; James et al., 1981; Logan et al., 1977) by the same mechanism as immunoglobulins. Not only do early infections allow bacteria to enter villus cells, the bacteria destroy the cells and reduce the absorptive capacity of the intestine (Corley et al., 1977; James et al., 1981).

Neonatal calves commonly experience some degree of metabolic or respiratory acidosis at birth (Besser et al., 1990; Szenci, 1985). Disturbances in acid-base balance may affect the absorption of colostral IgG. Some researchers have reported reduced absorption of IgG as a result of acidosis in neonatal calves (Besser et al., 1990; Boyd, 1989). However, others have found no effect of acid-base balance on absorption (Ayers and Besser, 1992;

Drewry et al., 1999; López et al., 1994). Tyler and Ramsey (1991) observed that hypoxia in newborn calves extended the period of absorption from 20 to 40.5 h when colostrum was fed at 0 and 12 h. Hypoxia had no effect in fasted calves though, so the researchers indicated that hypoxia was not the primary mechanism affecting the extended absorption period.

Calves allowed to nurse their dam generally have lower serum IgG concentrations and are more susceptible to disease than hand-fed calves (Barber, 1979; Besser et al., 1991; Brignole and Stott, 1980; Logan et al., 1981; Nocek et al., 1984; NAHMS, 1993; NAHMS, 1994). However, calves assisted in nursing or fed in the presence of their dam had serum IgG concentrations greater than hand-fed calves (Petrie, 1984; Quigley et al., 1995a). Other research showed no effect of nursing on IgG concentrations (Rajala and Castrén, 1995). Use of an esophageal feeder decreased IgG absorption in some reports (Hopkins and Quigley, 1997; Lateur-Rowet and Breukink, 1983), but had no effect in others (Adams et al., 1985; Molla, 1978).

Recently interest in the effects of growth factors, particularly insulin-like growth factor I (IGF-I), have received much attention in an attempt to elucidate their role in the absorption of IgG from colostrum. Estimates of bovine colostral IGF-I concentrations range from 4 to 62 times those in milk (Odle et al., 1996). Other species (including humans and rats) do not exhibit such high concentrations of IGF-I in colostrum (Odle et al., 1996). Interestingly, the accumulation of IGF-I and of IgG in colostrum occurs at about the same time and at the same rate (Baumrucker and Blum, 1993). Thus it seems reasonable that a relationship between IGF-I and IgG exists in the newborn calf. However, to date no research has shown conclusively that IGF-I affects IgG in any way. Feeding IGF-I caused an increase in intestinal growth in both calves and piglets in some studies (Baumrucker and Blum, 1993; Baumrucker et al., 1994b; Bühler et al., 1998; Xu et al., 1994; Xu et al., 1996). However, feeding IGF-I in milk replacer to neonates has transiently increased (Baumrucker and Blum, 1993; Baumrucker and Blum, 1994; Grütter and Blum, 1991) or had no effect (Vacher et al., 1995) on blood concentrations of IGF-I in other research conducted by the same group. In another study (Hammon et al., 2000), plasma IGF-I decreased markedly, but transiently, in calves fed colostrum at 12 or 24 h compared to calves fed at birth or 6 h. In addition, feeding

recombinant human IGF-I had no effect on total protein, albumin or globulin concentrations in calves (Baumrucker et al., 1994a). Current belief is that although IGF-I is barely absorbed by the intestine of the newborn calf, it may affect the growth and development of intestinal epithelium and may affect or interact with other growth factors and thereby influence the neonatal intestine (Odle et al., 1996; Vacher et al., 1995). The mechanism of colostral IGF-I action and the specific influence it may have on the neonate remain unclear.

Other factors suggested to increase absorption in the calf include addition of trypsin inhibitor to colostrum (Quigley et al., 1995b) and curd formation of colostrum in the abomasum (Cruywagen, 1990). Factors observed to decrease absorption include addition of ascorbate to colostrum (Cummins and Brunner, 1989), hyperthyroid conditions (Cabello and Levieux, 1978), and housing newborns in groups of prepartum and recently calved cows (Michanek and Ventorp, 1993). Factors investigated but shown to have no effect on absorption include replacement of colostral milk fat with coconut oil (Rajaraman et al., 1997) and injection of somatotrophin at birth (Smith et al., 1964).

Failure of Passive Transfer

Inadequate absorption of colostral immunoglobulins is known as failure of passive transfer (**FPT**). The definition of FPT is generally accepted as a serum IgG concentration of less than 10 g/L (Paré et al., 1993; Rea et al., 1996; NAHMS, 1993). The National Animal Health Monitoring Service (**NAHMS**) evaluated calf and heifer rearing practices in 1991 and 1992 using representative national surveying techniques (NAHMS 1993, NAHMS 1994). The project, the National Dairy Heifer Evaluation Project (**NDHEP**), found that 64% of calves were fed colostrum by hand feeding and 33.7% were allowed to nurse the dam (more than half of these were unassisted). In addition, 25.6% of calves received less than 1.89 L of colostrum in the first 24 h (NAHMS, 1993). Heinrichs et al. (1994) interpreted these results and suggested that the calves fed less than 1.89 L of colostrum probably suffered FPT based on the average IgG concentration of Holstein colostrum. Furthermore, the NDHEP (NAHMS, 1993) reported 31% of calves had serum IgG concentrations less than 10 g/L. Experimental trials involving fewer calves have observed FPT in 9.2 to 35% of calves (Hancock, 1985; Paré et al., 1993; Perino et al., 1993; Rea et al., 1996; Robison et al., 1988).

Results of the NDHEP (NAHMS, 1993) showed a preweaning mortality rate of 8.4%. The rate was increased to 10.8% in a 1996 follow-up survey by NAHMS (1996). The mortality rate for calves with serum IgG less than 10 g/L was more than twice that of calves with higher concentrations (NAHMS, 1993). Others have confirmed (Hancock, 1985; Perino et al., 1993; Robison et al., 1988; White and Andrews, 1986; Wittum and Perino, 1995) that calves that experienced FPT had greater risk of morbidity and mortality prior to weaning than those with adequate passive transfer. In discussing the NDHEP results, Wells et al. (1996) suggested that 31% of heifer mortality in the first 21 d could be prevented by changes in first colostrum feeding method, timing, and volume. Hancock (1985) reported that calves with the same serum IgG concentration had different mortality risk depending on the mortality rate of the herd. Calves with low IgG born in herds with a low mortality rate were more likely to survive than calves with the same IgG concentration born in a herd with high mortality. Furthermore, beef calves with the lowest serum IgG concentrations had lower daily gains and expected weaning weights than calves with high serum IgG concentrations (Wittum and Perino, 1995). In addition, serum IgG at 24 to 48 h was a significant source of variation in average daily gain for dairy calves in the first 180 d of life (Robison et al., 1988). Warnick et al. (1995) reported no significant effect of owner-diagnosed calfhood disease on first lactation milk production, but affected heifers had higher mortality before calving and were less likely to enter the milking herd.

Preventing Failure of Passive Transfer

Failure of passive transfer is associated with increased risk of morbidity and mortality in preweaned calves. Therefore, management practices must be implemented to minimize FPT and enhance calf health. Several approaches to the problem of FPT exist. First and foremost, timely feeding of high quality colostrum in adequate amounts must occur. One logical and fairly common way to reduce FPT in calves is to save excess colostrum and freeze it for calves born to dams with low quality colostrum. Colostrum can be frozen in plastic containers or bags in amounts suitable for single feedings. Frozen colostrum can be stored at -18ºC to -25ºC for at least six mo without changing its quality (Roy, 1990; White, 1993). Colostrum is thawed as needed, typically in warm water, although successful thawing can be accomplished in a microwave oven (Jones et al., 1987). Slow thawing at temperatures

below 50ºC does not affect colostrum quality, but temperatures above 50ºC cause colostral proteins, including immunoglobulins, to denature (Roy, 1990; White, 1993). Once thawed, colostrum should be used immediately, as repeated freeze-thaw cycles decrease the amount of viable immunoglobulin protein (Roy, 1990; White, 1993).

Over the past several years, interest in formulating supplemental products or colostrum substitutes to reduce the occurrence of FPT has grown. Supplement products are generally intended for addition to colostrum, to increase the amount of IgG provided to calves. To be labeled as a colostrum supplement, a product must be tested to demonstrate that it improves serum IgG concentrations compared to colostrum deprivation (Garry et al., 1996). To be effective in replacing colostrum these products must confer immunity, provide energy, and be a source of vitamins and minerals (Quigley et al., 1998b; White, 1993). However, in most cases (Abel Francisco and Quigley, 1993; Bouchard et al., 1992; Constant et al., 1994; Crowley et al., 1994; Garry et al., 1996; Mee et al., 1996; Mulvey, 1996; Poffenbarger et al., 1991; Quigley et al., 2000a; Quigley et al., 1998b; Todd et al., 1993; Zaremba et al., 1993) these products contained only 25 to 45 g/L of IgG and did not improve the absorption of IgG over maternal colostrum. Furthermore, because the products have not shown much effectiveness, their acceptance and use by dairy producers has been limited. In spite of findings that many calves experienced FPT, the NDHEP reported that only 0.3% of calves were given colostral supplements from 1991 to 1992 (NAHMS, 1994).

Colostrum supplements fall into four categories: dried colostrum products, whey protein-based products, serum protein-based products, and injectable products. Dried colostrum products were the first type of supplement investigated. Chelack et al. (1993) compared three methods of drying colostrum and concluded that spray drying was the most cost-effective method. Spray-dried colostrum was then reconstituted and fed to calves. Amounts of IgG fed were equal at 126 g (in two feedings) for spray-dried and frozen colostrum. Serum IgG concentration at 48 h was not different for calves fed the spray-dried colostrum compared to calves fed frozen colostrum (11.6 g/L and 10.57 g/L respectively). Todd et al. (1993) found that calves fed colostrum plus a fortified colostrum powder, providing 128, 78, or 52 g of IgG depending on amount of powder fed and solvent

(colostrum, milk, or water), achieved adequate passive immunity and remained healthy throughout the preweaning period. On the other hand, Zaremba et al. (1993) reported that calves fed 85 g of dried colostrum powder (9.6 g IgG) had lower serum IgG at 24 h than calves fed either 3 kg of pooled colostrum (288 g IgG) or 3 kg of pooled colostrum supplemented with 85 g of dried colostrum powder (297.6 g IgG). The addition of dried colostrum powder did not improve IgG concentrations compared to colostrum alone (Zaremba et al., 1993).

Supplement products based on whey protein concentrate have also been introduced. Abel Francisco and Quigley (1993) found a change in the timing of absorption when they fed a colostrum supplement containing lyophilized colostrum and dried whey. The IgG_1 concentration was highest in calves fed colostrum plus the supplement (198.7 g IgG_1) at 12 h, but at 24 h the colostrum calves (fed 198.8 g IgG_1) had the higher IgG_1 concentrations. Arthington et al. (2000a) also reported higher IgG concentrations in calves fed colostrum compared to calves fed either of two whey protein-based supplements. Intake of IgG for the three groups was 200, 50, and 60 g for colostrum, supplement 1, and supplement 2, respectively. Garry et al. (1996) fed colostrum and three different supplement products. Calves were fed 164.7, 156.8, 107.7, or 126.0 g IgG in colostrum and supplement groups 1 through 3, respectively. The colostrum group had the highest serum IgG concentrations at 24 h and was three times more efficient in absorbing IgG. In addition, the colostrum-fed calves experienced significantly fewer episodes of disease prior to weaning than calves fed supplements. Mee et al. (1996) found calves fed whey protein concentrate as a colostrum supplement (69.1 g IgG) or colostrum substitute (17.7 g IgG) had significantly lower serum IgG and total protein concentrations than colostrum-fed calves (123.6 or 117.2 g IgG). In addition, in one of two trials, calves fed only the supplement product had much greater mortality rate than colostrum-fed calves (27.6 and 3.4%, respectively). On the other hand, Seymour et al. (1995) reported similar health parameters and greater feed efficiency during the preweaning period for calves fed a whey protein concentrate substitute instead of colostrum, total IgG intakes were not reported.

Research suggests that products made from bovine serum may hold more promise for effective substitution of colostrum in newborn calves. Work with animal proteins in other species has shown potential for their use as colostrum substitutes. Plasma proteins are used extensively to replace milk proteins in the diets of young pigs (Hansen et al., 1993; Kats et al., 1994). Other work has been done with goat kids (Constant et al., 1994) and puppies (Bouchard et al., 1992; Poffenbarger et al., 1991). Crowley et al. (1994) reported that in calves, injection of immunoglobulins or supplementation of milk replacer with immunoglobulins purified from abattoir blood increased serum immunoglobulin concentrations over feeding milk replacer alone, but not to the same extent as feeding colostrum. In an investigation where three supplements were compared to colostrum, Arthington et al. (2000a) found that although the bovine serum-based product (90 g IgG) did not provide IgG absorption equal to colostrum (200 g IgG) in calves, the serum product was absorbed more efficiently than any of the other feeds, including colostrum. Furthermore, there were no differences in mortality between groups, but the serum-fed calves tended to need fewer treatments. Todd et al. (1993) determined that calves fed 3.2 L (77 g IgG), but not 2.4 L (58 g IgG) of bovine serum could achieve adequate passive immunity. Quigley et al. (1998b) fed colostrum or a colostrum replacer derived from bovine serum to calves in two blocks. Due to differences in colostrum quality between blocks, the amount of replacer fed was changed for block 2. This change provided an interesting result. Calves fed the serum product at a high dose (750 g, 150 g IgG) had reduced efficiency of absorption compared to calves fed the replacer at a low dose (266 g, 53.2 g IgG). At the high dose calves fed the replacer had lower 24 h plasma IgG than colostrum-fed calves (who were also fed 150 g IgG), but at the low dose the replacer calves absorbed more IgG than those fed colostrum (IgG intake was 53.2 g). Quigley et al. proposed that the large mass of non-IgG protein in replacement products might impair IgG absorption by competing for intestinal binding sites.

Further investigation by this group led to a series of experiments designed to test the theory of competition for binding sites. They found that the addition of casein or whey protein concentrates to colostrum supplements or maternal colostrum had no effect on plasma IgG concentration unless total protein in the product exceeded 500 g (Arthington et al., 2000b). Other experiments by this group found that calves fed a bovine serum-based product

achieved higher 24 h serum IgG than calves fed colostrum or porcine serum with the same IgG concentration (90 g IgG fed) (Arthington et al., 2000b). This group also reported that bovine serum added to colostrum of high (95.8 g IgG fed, 0% IgG from serum), medium (95.2 g IgG fed, 47% from serum) or low (98.8 g IgG fed, 70% from serum) quality had higher IgG absorption when added to medium or low quality colostrum. In addition, the apparent efficiency of absorption was greater for medium and low quality colostrum diets (Arthington et al., 2000b). Arthington et al. proposed that bovine serum contains a concentrated source of immunoglobulin that is efficiently absorbed by newborns. Furthermore, supplementing colostrum with bovine serum product or feeding bovine serum product alone can improve passive transfer in newborns. They also suggested that the ratio of IgG_1 to IgG_2 (42%: 46%) in serum-based products might increase absorption efficiency compared to colostrum (IgG_1 to IgG_2 is 95%: 5%; Butler, 1983). Arthington et al. (2000b) proposed that the more uniform isotype distribution in serum-based products, compared to colostrum, allowed greater absorption efficiency.

Other attempts to prevent FPT have used plasma transfusions and injections of purified immunoglobulins (Crawford et al., 1995; Pederson et al., 2000; Quigley and Welborn, 1996; Zaugg, 1994). Transfusion of plasma obtained from mature cows failed to provide IgG concentrations similar to calves fed colostrum (Zaugg, 1994). Infusion of a purified IgG solution into calves 3 to 8 d of age failed to increase serum IgG when administered subcutaneously, but did increase serum IgG when administered intravenously (Quigley and Welborn, 1996). Injection of purified IgG increased serum IgG compared to no colostrum, but did not increase it to the concentrations obtained by feeding colostrum (Crawford et al., 1995). More recently, Pederson et al. (2000) reported increased plasma IgG concentrations at 24 h when calves were injected at birth with bovine antiserum. In addition, administration of antiserum increased apparent efficiency of absorption by 42% over colostrum alone (Pederson et al., 2000).

Although producers have not used the currently available colostrum supplement products extensively, there is growing interest in using supplement or replacement products. Perhaps the most compelling reason for further investigation of these products is the risk of

disease transmission. Johne's disease, bovine viral diarrhea, and salmonella can be passed to calves by ingestion in colostrum, particularly if colostrum is contaminated with fecal matter (Roy, 1990; Stabel, 2001). Due to the economic impact of these diseases, it is prudent to avoid feeding infected colostrum to newborn calves that have essentially no immune capabilities. Any replacement product must contain enough readily absorbable IgG to effectively mimic colostrum in establishing passive immunity. Aside from disease control, such products must be cost-effective, readily available, easy to handle, and palatable if they are to be accepted and utilized by dairy producers. Although immunoglobulins contained in commercial products could not provide protection to farm-specific pathogens, substitute products have other potential advantages in consistent composition and quality. Effective replacement products would provide a viable alternative for producers facing low quality or contaminated colostrum supplies.

Feeding the Preweaned Calf

After initial feedings of colostrum, several liquid feed options exist for the preweaned calf, including whole milk, surplus colostrum or transition milk, unsalable/discard milk, and milk replacer. The NDHEP (NAHMS, 1993) revealed that 32.7% of farms surveyed fed whole milk from the bulk tank, 51.9% fed milk from recently calved cows (i.e. transition milk), 37.7% fed unsalable milk, and 59% fed milk replacer. It should be obvious that whole milk is a high-quality liquid feed that supports calf growth and health very well. Unfortunately, in most cases it is also the most expensive liquid feed when compared on the basis of total solids (Davis and Drackley, 1998). Surplus colostrum or transition milk and discard milk are the cheapest options for liquid feeds. Both types of milk are unsalable and have no market value. Therefore when they can be used to feed calves, these feeds gain value and provide cost savings relative to buying milk replacer or using salable milk (Davis and Drackley, 1998). On many farms excess colostrum and transition milk create an abundant supply of milk that can be used to feed calves from birth to weaning (Foley and Otterby, 1978). Transition milk composition approaches that of whole milk throughout the first six milkings postpartum (Foley and Otterby, 1978). Total protein, immunoglobulins, fat, total solids, solids non-fat, and ash decrease with time postpartum, while lactose increases (Parrish et al., 1950). Pooled colostrum and transition milk have a final composition of 14 to 18%

total solids, 5 to 7% protein (Daniels et al., 1977; Otterby et al., 1977), and 4 to 5% fat (Otterby et al., 1977). Storage of this resource was a problem prior to research that showed it could be frozen (Carlson and Muller, 1977) or fermented, either naturally or by chemical treatment (Carlson and Muller, 1977; Daniels et al., 1977; Muller et al., 1976; Otterby et al., 1977; Rindsig and Bodoh, 1977; Rindsig et al., 1977), with only small changes in composition. Frozen and fermented colostrum provided cheap sources of feed that were palatable and supported growth and health as well as whole milk (Foley and Otterby, 1978; Muller et al., 1974). The storage space and extra labor required to handle frozen milk limit its use (Foley and Otterby, 1978). Poor fermentation and putrefaction at warm ambient temperatures require that chemical preservatives be used to treat colostrum in warm climates. The extra labor and caustic chemicals involved in treating colostrum may limit the use of fermented colostrum, especially in warm climates (Muller et al., 1976). Increases in herd size, extra labor requirements for maintaining milk quality, large storage space needs, and disease transmission concerns may make the use of colostrum and transition milk less desirable (Davis and Drackley, 1998). Milk obtained from cows treated with antibiotics cannot be sold and, thus, is another potential source of liquid feed for calves. Chardavoyne et al. (1979) reported that calves fed fresh waste milk from antibiotic-treated cows had similar growth compared to calves fed whole milk or fermented colostrum. In addition, Keys et al. (1976 and 1979) showed that milk from cows treated with antibiotics for mastitis infections fermented more slowly than normal milk for the first two post-treatment milkings, but not for third or greater milkings. Inoculating treated milk with bacterial cultures or buttermilk did not decrease fermentation time (114 h if inoculated, 113 h if naturally fermented), but increasing the temperature did reduce fermentation time (108 h at 4.4ºC, 38 h at 32.2ºC). Furthermore, Keys et al. (1980) determined that the incidence of health disorders and mastitis in the first lactation of cows fed fermented mastitic milk as calves was not different from the occurrence of disease in cows fed fresh milk or fermented colostrum as calves. In a review of the issue, Kesler (1981) concluded that mastitic milk can provide acceptable growth and health in young calves; with no differences in weight gain or scouring compared to calves fed milk or fermented colostrum.

Milk replacers, fed to young calves instead of milk, were first developed in the 1950s (Davis and Drackley, 1998; Heinrichs, 1994). Compared to the other options for liquid feeds, milk replacer has several advantages, including: reduced labor and storage space requirements, increased consistency of physical and nutritional components, reduced risk of disease transmission, and lower cost than whole milk. Milk replacer quality and ability to support the growth and health of calves depends heavily on the ingredients used in formulating the product (Davis and Drackley, 1998). The most important criterion in selecting milk replacer ingredients is their digestibility in the developing gastrointestinal tract of the young calf. At birth the calf produces mostly lactase and has very little ability to digest proteins due to low secretion of pancreatic proteases. Production of pancreatic enzymes, both proteases and lipases, increases in the first week of life and continues to increase with age (Heinrichs, 1994; Otterby and Linn, 1981). By 3 wk proteolytic and lipolytic enzymes are fully functional, and the calf is more able to digest non-milk proteins and fats (Heinrichs, 1994). Other criteria, such as the cost of the ingredient, anti-nutritional factors, and solubility also influence the relative value of milk replacer ingredients (Davis and Drackley, 1998). A wide variety of milk replacer formulations are commercially available. These formulations differ in the amount of protein and fat and in the ingredients used to supply protein (Davis and Drackley, 1998). Differences in formulation drive price differences between products. Although milk fat is 95 to 97% digestible (Raven, 1970; Toullec et al., 1980), it is consistently more expensive than fats such as tallow and lard (Davis and Drackley, 1998). Therefore, most milk replacers contain an alternative fat. Tallow and lard are less digestible than milk fat (87 to 94% and 88 to 96%, respectively), but calves fed these fat sources perform very well (Toullec et al., 1980). Other fat sources, such as coconut oil and palm kernel oil, are used to a lesser extent (Davis and Drackley, 1998); vegetable oils are seldom used due to poor utilization and resultant diarrhea in young calves when they are fed (Jenkins et al., 1985; Jenkins et al., 1986). In contrast to fat sources, many different ingredients are used to provide protein, the most expensive ingredient, in milk replacer.

From the time that milk replacers were introduced there have been two primary objectives in formulating them: first, to find ingredients containing available, digestible protein, and second, to reduce the cost of feeding calves. Balancing these two objectives is

not easy. Historically as the price of ingredients has changed, milk replacer manufacturers have changed their formulations to reduce the cost of their products. Once a source of protein is identified, processing techniques are refined to maximize utilization of the protein. The increased processing typically opens up new uses for the protein and increases its value so that manufacturers begin to search for another protein source (Heinrichs, 1994). Many different protein sources have been investigated in the attempt to balance the cost of a milk replacer with its suitability for feeding the young calf. These sources can be divided into two general categories, milk and non-milk. Milk proteins are preferred because they are more digestible, but they are more expensive. Early milk replacers were made from dried skim milk, dried whey, dried buttermilk, and animal fat (Otterby and Linn, 1981). These products contained large amounts of overheated protein that was not well digested by calves and caused diarrhea and poor performance (Shilliam and Roy, 1963; Shilliam et al., 1962). Processing improvements were made to heat the milk at lower temperatures. When the price of dried skim milk increased drastically during the 1960s, the industry was forced to search for alternative ingredients (Heinrichs, 1994). At that time whey was a useless by-product of cheese making, but it soon became a valuable source of milk replacer protein. Today nearly all milk protein in milk replacers is derived from whey (Davis and Drackley, 1998; Lammers et al., 1998).

Early attempts to use whey proteins showed that in high amounts they caused decreased performance and increased diarrhea (Roy, 1980). However, other researchers reported satisfactory average daily gains and health when whey proteins were used (Bouchard et al., 1973; Cruywagen and Horn, 1985; Morrill et al., 1971; Strudsholm, 1988; Volcani and Ben-Asher, 1974). Advances in technology used to process whey led to the production of whey protein concentrates (**WPC**). Ultrafiltration and low-temperature evaporation techniques allowed uniform production of high quality proteins with little denaturation (Davis and Drackely, 1998). Whey protein concentrates have a gross composition equivalent to skim milk, contain vitamins and highly available amino acids equal to milk, and support acceptable gains and health in young calves (McDonough et al., 1976). Calves fed diets where WPC contributed 25% of crude protein (remainder from whole milk) had similar weight gain when compared to calves fed whole milk or frozen colostrum

(Muller et al., 1974). Babbela et al. (1988) fed milk replacer containing 25, 50, 75 or 100% of protein from WPC, with the remainder from dried skim milk. Digestibilities were similar for the 25, 50 and 75% formulations, but protein in the 100% WPC replacer was less digestible. Average daily gains were highest in calves fed 75% WPC, and no diarrhea was observed in any of the groups (Babbela et al., 1988). In a similar experiment, Terosky et al. (1997) fed diets with 0, 33, 67, or 100% of crude protein from WPC with the remainder from skim milk. No differences in number of days scouring were observed between groups. Feed efficiency and average daily gains also showed no differences between groups. No differences were detected in apparent digestibility of crude protein. Terosky et al. concluded that dried skim milk and whey protein concentrate were comparable protein sources. Two trials conducted by Lammers et al. (1998) used the same feeding rates as Terosky et al. (1997). In the first trial, calves were fed only milk replacer; in the second trial calves were allowed ad libitum starter intake. In trial 1 calves fed 67 or 100% WPC had significantly higher average daily gains and feed efficiency than calves fed 100% dried skim milk. However, in the second trial no differences in gains or efficiency were detected.

High quality proteins are the most expensive ingredients in milk replacer. Due to increasing demand for milk proteins in human food products, the price of these ingredients has steadily increased and is expected to continue to rise (Davis and Drackley, 1998). As a result, the milk replacer industry has attempted to use many non-milk proteins that are less expensive. Soy proteins have been targeted for use in milk replacers because they are widely available, relatively inexpensive and generally favorable in amino acid content (Davis and Drackley, 1998). However, soybean protein is less digestible than milk protein and contains many anti-nutritional factors that can be detrimental to calf health (Lallès, 1993). These anti-nutritional factors include protease inhibitors, antigenic proteins, indigestible carbohydrates, lectins, tannins and other phenolic compounds, phytate, and saponins (Lallès, 1993). These compounds have many effects on the calf, but in general they decrease protein digestibility and absorption and lead to increased diarrhea and decreased growth (Huisman, 1989; Lallès, 1993). Processing of soy protein reduces the anti-nutritional components, increases the availability of protein, and increases the cost of the ingredient. Common soy ingredients, in

order of least to most acceptability (and therefore cost), are soy flour, soy protein concentrates, and soy protein isolates (Davis and Drackley, 1998).

Heated soy flour can be used in milk replacers, but typically supports lower weight gain and growth than milk-based diets (Akinyele and Harshbarger, 1983; Dawson et al, 1988; Lallès et al., 1995; Miller et al., 1991; Silva et al., 1986). Protein from soy flour has lower digestibility than milk protein (soy flour 29 to 65%, milk 81 to 90%), and thus is more appropriate for calves greater than 3 wk old than for younger calves (Akinyele and Harshbarger, 1983; Dawson et al, 1988; Ramsey and Willard, 1975; Silva et al., 1986). Soy protein concentrate included at up to 50% of crude protein supported acceptable growth and health in calves (Akinyele and Harshbarger, 1983; Campos et al., 1982; Erickson et al., 1989; Dawson et al, 1988; Huber and Campos, 1982; Morrill et al., 1971; Tomkins et al., 1994), although gains were less than those obtained when all milk proteins were fed. Digestibility of protein in soy protein concentrate ranges from 57 to 65% (Akinyele and Harshbarger, 1983; Dawson et al, 1988). Calves fed soy protein isolates and modified soy proteins had fewer incidences of scouring and supported similar gains compared to calves fed all-milk milk replacers (Fowler et al., 1991; Lallès et al., 1995; Miller et al., 1991). Others reported reduced growth when soy protein isolates or modified soy proteins were fed (Khorasani et al., 1989a; Silva et al., 1986). Soy protein isolates have 60.5% digestible protein (Khorasani et al., 1989a), while modified soy proteins have about 83% digestible protein (Silva et al., 1986). Seegraber and Morrill (1986) observed atrophy and deterioration of intestinal villi when soy proteins were fed to provide one-third of protein in the diet. In the same study (Seegraber and Morrill, 1986), milk-fed calves exhibited greater daily weight gain and protein digestibility than calves fed soy or fish proteins.

Wheat protein is another alternative for providing protein in milk replacers, although it is very low in lysine (Davis and Drackley, 1998). Wheat gluten had lower apparent digestibility than a skim milk based replacer (87% versus 91%) in 8 wk old calves (Branco-Pardal et al., 1995). Soluble wheat proteins have been used successfully in veal calves in the Netherlands (Davis and Drackley, 1998). Substituting enzymatically hydrolyzed wheat protein for up to 50% of WPC in milk replacer produced similar growth performance to

100% WPC replacers in calves up to 6 wk of age (Terui et al., 1996). On the other hand, Tomkins et al. (1994) found average daily gains were lower in calves fed 10 to 20% enzyme modified wheat gluten (other 80 to 90% of protein from soy protein concentrate) than in calves fed an all-milk milk replacer.

Plasma protein purified from bovine and porcine blood is another alternative for supplying milk replacer protein. Spray-dried plasma protein is highly soluble and has an amino acid profile similar to that of skim milk, and was utilized at 95% of casein utilization in rats (Duarte et al., 1999). Diets for young pigs commonly include porcine plasma protein, and it has been shown to increase feed intake and growth (Davis and Drackley, 1998; Hansen et al., 1993; Kats et al., 1994). Very little scientific data is available concerning the use of plasma proteins in calf milk replacers. Morrill et al. (1995) fed 120 bull calves four diets containing all milk protein, 25% porcine plasma protein, 25% bovine plasma protein, or all milk protein plus a probiotic for 6 wk. Calves fed porcine or bovine plasma had heavier body weights and consumed more starter by the end of the 6-wk trial than calves fed all milk protein. No differences in fecal scores, height at withers, or heart girth were detected. Quigley and Bernard (1996) used bovine plasma protein to substitute for 0 or 25% of whey protein in milk replacers fed to 68 calves for 56 d. No differences were observed in weekly body weight, body weight gain, dry matter intake, efficiency of gain, or fecal scores. They concluded that plasma proteins included at 25% of crude protein could support growth equal to that supported by whey proteins. However, calves fed milk replacer with 50% soy protein concentrate, 10% enzyme modified wheat gluten, and 10% porcine plasma protein had lower average daily gains than calves fed an all-milk protein replacer (Tomkins et al., 1994). Calves fed porcine plasma to provide 10% of crude protein (remainder from soy protein concentrate) had lower starter intake than calves fed milk replacer containing 40% soy protein concentrate and 20% enzyme modified wheat gluten (Tomkins et al., 1994). A potential advantage of using plasma proteins in milk replacer is that they contain immunoglobulins. Drew (1994) suggested that adding immunoglobulins to milk replacer may reduce scour incidence and improve animal performance by providing local immune protection against enteric pathogens in the intestinal lumen. Further research is necessary to define the effectiveness of immunoglobulins in milk replacer in providing this type of

protection. Moreover, from a public health standpoint, if plasma proteins are used in milk replacer, research must identify and processing techniques must eliminate any potentially zoonotic pathogens.

Many other alternative sources of protein have been tested for use in milk replacers. However, these are less suitable replacements for milk protein than high quality soy products, modified wheat proteins, and animal plasma. While these protein sources are not acceptable for calves less than 21 d of age, they may be used for older calves in limited amounts (Davis and Drackley, 1998). These alternative proteins include pea protein (Lallès, 1993), lupin protein (Turkur et al., 1995), potato protein (Branco-Pardal et al., 1995), fish proteins (Campos et al., 1982; Diaz-Castañeda and Brisson, 1987a; Diaz-Castañeda and Brisson, 1987b; Huber, 1975; Huber and Campos, 1982; Jenkins et al., 1982), whole blood proteins (Raven, 1972), red blood cell proteins (Quigley et al., 2000b; Scott et al., 1999; Ziegler et al., 1996), bacterial proteins (Bouchard et al., 1973; Guilloteau and Toullec, 1980; Sedgman et al., 1985), yeast proteins (Sedgman et al., 1985), egg proteins (Scott et al., 1999), and meat solubles (Khorasani et al., 1989b; Polzin et al., 1976).

Summary

The importance of colostrum in determining calf health and survival is well established. Timely, adequate colostrum intake is the single most important management factor affecting morbidity and mortality in preweaned calves. Calves have essentially no immune protection at birth because maternal immunoglobulins cannot cross the placenta, and the neonatal immune system is functionally immature. Therefore, calves must consume colostrum, which is rich in IgG, to protect against infection. Immunoglobulins are absorbed, primarily by an indiscriminate pinocytotic process, in the small intestine for about 24 h after birth. Factors affecting the amount of IgG absorbed from colostrum include time of first feeding and amount of IgG consumed.

Surveys of calf management revealed that about 10% of calves died prior to weaning. Failure to achieve adequate passive immunity has been associated with increased morbidity and mortality, and poor colostrum feeding and management has been suggested as the

primary reason for high mortality in calves. Products have been introduced to supplement colostrum and attempt to reduce the incidence of calf mortality and disease transmission. These products vary in composition, and the range of ingredients includes dried colostrum, dried whey, purified colostral immunoglobulins, and purified blood proteins. Supplements tend to provide less IgG than colostrum, and have not been widely accepted or used by producers. In general these products are not recommended for use as colostrum replacements, meaning they are usually added to colostrum, not fed in lieu of it. Since bacteria or viruses can be transmitted in colostrum, the practice of supplementing colostrum does not alleviate concerns about colostrum as a vehicle for spreading disease. On the other hand, true replacement of colostrum with a synthetic product may be able to break the cycle and prevent calves from acquiring diseases from their dams.

Interest in using readily available animal proteins in other ways has also increased. If animal proteins could effectively substitute for milk proteins in milk replacer formulations, the nutritional value of milk replacer may be increased due to the provision of immunoglobulins, which may exert a local protective effect in the intestine and improve animal health and performance.

CHAPTER 3. MATERIALS AND METHODS

Experimental Treatments and Design

Calves were blocked according to gender and sex such that treatment blocks were defined as Holstein females, Holstein males, Jersey females, and Jersey males. Treatments were assigned to each of the four blocks independently prior to the start of the trial. Seventy-eight calves born between June 5 and December 3, 2000, were included in the study and assigned to one of four treatments: colostrum replacement + milk replacer with animal plasma (**RP**), colostrum replacement + milk replacer without animal plasma (**RN**), Colostrum + milk replacer with animal plasma (**CP**), Colostrum + milk replacer without animal plasma (control, **CN**).

Animals and Facilities Management

Calves were housed at the Virginia Tech Dairy Cattle Center in individual stalls in a ventilated calf barn. Thirty-six stalls were used throughout the experiment. Prior to the beginning of the trial, stalls were numbered and randomly ordered to reduce the potential effect of pen location (microenvironment). Calves had no physical contact with each other. Stalls measured 1.22 m by 1.83 m with 1.12-m wooden partitions and concrete floor. Straw and/or sawdust were added daily to maintain the condition of the stalls. Before the start of the experiment, the entire barn was cleaned thoroughly with a power-washer using a solution containing 8.4% sodium hypochlorite. During the trial, stalls were cleaned, swept, and dried between calves, but were not washed or disinfected.

Most cows calved in one of two pastures near the main dairy facility. Cows calving later in the trial (late November and December) were moved to individual box stalls bedded with straw just prior to parturition. All calvings were supervised to assure that calves had no opportunity to nurse the dam. Any calf whose birth was not observed was not used in the study. As soon as possible after birth (typically within 15 minutes), calves were moved to the calf facility, identified, weighed, and had their navels dipped with 7% iodine tincture. Vaccinations were administered for bovine rhinotracheitis and parainfluenza 3 (TSV-2, Pfizer Animal Health, Exton, PA) and rota- and coronavirus (Calf Guard, Pfizer Animal

Health, Exton, PA). Calves received injections of vitamins A and D (Holsteins 500,000 IU vitamin A, 75,000 IU vitamin D; Jerseys 250,000 IU vitamin A, 37,500 IU vitamin D; Phoenix Pharmaceutical, Inc., St. Joseph, MO) and selenium and vitamin E (BoSe, Holsteins 3 mg selenium, 204 IU vitamin E; Jerseys 1.5 mg selenium, 102 IU vitamin E; Schering-Plough Animal Health, Union, NJ). Calving difficulty score, time, and date of birth were recorded.

Calves were measured for growth to determine performance throughout the trial. At birth, 24 h after birth and weekly for 4 wk, the following measurements were taken: weight, wither height, hip height, body length, and heart girth. Body temperature was also determined at birth 12 and 24 h, and weekly for the first month of life. Weights were determined with a digital scale. Wither and hip heights were measured using a wooden height stick with horizontal crossbar and level, calibrated in .635-cm increments (actual calibrations were in English units; Nasco Metre, Nasco, Fort Atkinson, Wisconsin). Body length (measured from point of shoulder to caudal projection of pin bone) and heart girth were determined with a measuring tape calibrated in 1.27-cm increments (actual calibrations were in English units). Body temperatures were measured rectally with a digital thermometer calibrated in 0.1-degree increments (Quicktemp Thermometer, Agri-Pro Enterprises, Iowa Falls, Iowa). Error of measurement associated with the thermometer was ± 0.5°C.

Feeding and Feed Management

Calves were fed colostrum replacement or pooled colostrum containing approximately equal amounts of IgG within 1.5 h of birth and 12 h after the first feeding (see Table 1 for description of actual outcomes). Colostrum was collected from cows calving prior to and throughout the study. Colostrum from first and second milkings was tested with a colostrometer, and only excellent quality colostrum was used in the pool. All colostrum was stored at -20°C prior to pooling. Pooling was accomplished by thawing colostrum in warm water and mixing it in a clean, sanitized 189 L container. Samples were collected for IgG analysis, and the pool was divided into plastic jugs containing 2 L each. Aliquots were stored at -20°C until feeding. Approximately 68 L of colostrum from primiparous and multiparous Holstein and Jersey cows were pooled initially. When the first pool was almost

depleted, a second pool of about 56 L was made following the same procedures. However, 1.5 L aliquots were frozen in plastic bags to facilitate the thawing process. Holstein calves fed pool 1 and 2 colostrum received 1.42 L at each feeding. Jerseys fed pool 1 and 2 colostrum received 0.94 and 1.18 L per feeding, respectively. All colostrum was thawed in warm water (approximately 54°C) prior to feeding. Colostrum replacement powder was mixed with warm water (approximately 38°C) prior to feeding. Holsteins received 454 g powder dissolved in 1.89 L water. Jerseys received 340.5 g powder dissolved in 1.42 L water. Nutrient composition of feeds on a dry matter basis is reported in Table 2.

Table 1. Description of outcome of procedures described in experimental design. Treatments: colostrum at birth, no plasma in milk replacer (CN); colostrum at birth, plasma in milk replacer (CP); replacement at birth, no plasma in milk replacer (RN); replacement at birth, plasma in milk replacer (RP).

	Treatment				
Variable	CN Mean	CP Mean	RN Mean	RP Mean	SE
Calves, initial number	20	19	20	19	…
Calves, final number	19	16	20	19	…
Birth weight (kg)	34.0	35.5	35.9	36.4	2.0
Age at feeding (h)					
First feeding	1.2	1.1	1.0	0.9	0.2
Second feeding	13.2	13.1	13.0	12.9	0.2
IgG intake (g)					
Holsteins	250.3	250.3	250.3	250.3	…
Jerseys	180.7	180.7	180.7	180.7	…

All calves were fed colostrum or colostrum replacement by nipple bottle unless the bottle was refused; in which case, calves were fed using an esophageal feeder. Colostrum replacement was formulated to provide 125 g IgG/feeding. Based on this constraint, amounts of colostrum and replacement fed were adjusted to provide equal total g of IgG. Holsteins and Jerseys were offered different amounts of IgG due to the smaller size of Jerseys and the potential for concentration of IgG in the feed to influence IgG absorption. A standard dose of the product provided 9.83 g/kg of metabolic body weight (body weight$^{0.75}$). Using the average body weight of calves at the Virginia Tech Dairy Cattle Center (calculated separately for Holsteins and Jerseys), it was determined that Jerseys should be fed 75% of the standard dose. Holsteins fed the replacement received 250 g of IgG in total (2 feedings). Holsteins fed colostrum received 249 g. Jerseys fed colostrum replacement received 180 g of IgG in total (2 feedings). Jerseys fed colostrum received 186 g. Colostrum amounts were adjusted based on the IgG concentration of colostrum, as determined after pooling.

Calves were fed milk replacer with or without animal plasma from a nipple pail twice daily beginning 24 h after birth. Daily feedings were at 0800 h and 1630 h. Milk replacer was reconstituted to 12.5% dry matter and fed at a rate of 31% of metabolic birth weight for 29 d. After d 29 calves were released from the study. To determine the rate of feeding, the energy requirement of calves gaining 227 g/d and the energy content of an average milk replacer were calculated. It was determined that feeding 31% of metabolic body weight each d would meet the energy needs of calves. This feeding rate was compared to a more standard rate of 10% of body weight daily. The 31% rate more closely met the needs of both large and small calves and was therefore chosen as the feeding rate. The Virginia Tech Dairy farm crew fed calves in the mornings; the research team carried out evening feedings. Feed equipment was rinsed with water between calves and thoroughly cleaned daily.

Table 2. Nutrient composition of feeds on a dry matter basis.

		Colostrum	Milk Replacer		
	Colostrum[1]	Replacement	No Plasma	Plasma	Starter
Dry matter (%)	24.0	96.02	96.81	97.3	88.88
Crude protein (%)	62.5	44.35	22.02	22.81	20.44
Milk protein (% of CP)		…	100	80	…
Plasma protein (% of CP)		…	0	20	…
Fat (%)	27.9	3.44	21.27	21.08	4.67
Ash (%)		4.69	7.46	8.59	6.09
Acid detergent fiber (%)		…	…	…	4.74
Ca (mg/100 g)		373	1090	1510	1020
P (mg/100 g)		619	983	1240	709
K (mg/100 g)		142	1210	1170	999
Mg (mg/100 g)		80.6	130	178	213

[1]Composition of colostrum was not analyzed, average values (Foley and Otterby, 1978) are reported for comparison.

Beginning on d 1, calves were offered starter grain containing decocquinate at a rate of 250 g/d. Starter intake was measured daily by weighing refusals. The amount of starter fed was increased by 50 g if daily orts were less than 25 g. Calves were limited to no more than 2.5 kg/d, but no calves reached this limit. Fresh, clean water was available free-choice beginning 1 to 2 d after birth.

Description of number of calves in treatment, breed, and gender groups is presented in Table 3.

Sampling and Data Collection

Table 3. Number of calves in each treatment (colostrum or colostrum replacement at birth and milk replacer with or without animal plasma), breed, and gender group.

Treatment	No Plasma	Plasma	Total
Colostrum	20	19	39
Replacement	20	19	39
Total	40	38	78
Breed-Gender	Female	Male	Total
Holstein	23	25	48
Jersey	11	19	30
Total	34	44	78

Blood samples were drawn at 1 h (0 d), 24 h (1 d), 8 d, 15 d, 22 d and 29 d. All samples were collected pre-feeding. At 1 h and 24 h packed cell volume was measured via hematocrit. Hematocrit samples (sub-sample drawn from original blood sample) were centrifuged for 5 min at 13,700 × *g* (Autocrit Ultra3, Clay Adams). Blood samples were collected by jugular venipuncture into evacuated glass tubes containing potassium EDTA. Blood was immediately centrifuged (1745 × *g* for 15 min at 4°C) to separate plasma. Plasma was split into two samples and stored at –20°C. At the end of the trial, one set of plasma samples was sent to Ames, IA, where American Protein Corporation, Inc. conducted IgG and total protein analyses. Remaining plasma samples were analyzed at Virginia Tech for urea N, insulin-like growth factor-I (**IGF-I**), and growth hormone (**GH**).

Calves were monitored during each feeding and for the duration of the study for signs of health problems. All problems and treatments were recorded in a daily logbook. In addition, fecal scores were evaluated once daily using a three-point scale (0 = firm, 1 = loose, and 2 = watery). Clinical health was scored based on ability to stand and presence or absence of suckle reflex. The score had a maximum of 2 points, 1 for each category (does not stand = 0, stands = 1 plus suckle reflex absent = 0, suckle reflex present = 1). Body temperature also was measured at birth, 12 h, 24 h and weekly for the first mo of life.

Laboratory Methods

Immunoglobulin G and total protein. Technicians at American Protein, Ames, IA, quantified IgG in plasma samples by turbidimetric immunoassay according to the methods of Etzel et al. (1997). Plasma total protein was determined by biuret method (Smith et al., 1985).

Insulin-like growth factor-I. Plasma concentrations of IGF-I were determined by double antibody radioimmunoassay technique as described by Brier et al. (1991). Before beginning the IGF-I assay, plasma samples were incubated with acid-ethanol (100 μL sample: 900 μL extraction buffer) for 1 h at room temperature to separate IGF-I from its binding proteins. Acidified samples were centrifuged at 13,290 × *g* for 10 min at 4°C to remove binding proteins. The resulting supernatant liquid containing IGF-I was transferred to a glass tube and neutralized with 200 μL of 0.855 M tris base solution. Neutralized samples were incubated for 1 h at -20°C and then centrifuged at 3290 × *g* for 30 min at 4°C. Following centrifugation, supernatant was decanted into plastic storage tubes and held at -20°C until assaying.

Recombinant human IGF-I used for standards and iodination and mouse anti-human IGF-I antibody (first antibody) were gifts of Dr. Michael Akers (Virginia Tech). Goat anti-mouse antiserum (second antibody) was purchased from Sigma Chemical Company (St. Louis, MO). The IGF-I molecule has an identical 70-amino acid sequence in the bovine, human and pig (Weber, 1998) and exhibits strong cross-reactivity between these species. Therefore utilization of human IGF-I is appropriate when determining bovine IGF-I concentrations. For assay, duplicates of each sample or standard were diluted (30 μL sample plus 470 μL buffer). Assay buffer (pH 7.5) contained 4.41 g/L 30 mM sodium phosphate monobasic, 3.72 g/L 10 mM EDTA, 10 ml/L 0.02% sodium azide, 1 g/L 0.1% bovine serum albumin, 9 mg/L phenol red, 0.5 ml/L 0.05% Tween 20, and 200 mg/L protamine sulfate. First antibody (100 μL) and IGF-I tracer (^{125}I-labeled IGF-I, 100 μL) were added to each tube and incubated at 4°C for 24 h. Subsequently, 100 μL of second antibody was added to each tube and incubated for 72 h at 4°C. Tubes were decanted and a gamma counter measured binding of radioactivity. Inter and intra assay variation averaged less than 15%, which is acceptable variation (R. Michael Akers, personal communication).

Growth hormone. Plasma concentrations of GH were determined by double antibody radioimmunoassay technique as described by Barnes et al. (1985). Purified bovine GH used for standards and iodination, rabbit anti-ovine GH antibody (first antibody), and sheep anti-

rabbit IgG (second antibody) were gifts of Dr. Michael Akers (Virginia Tech). Duplicates of each sample or standard were diluted (200 μL sample plus 300 μL buffer) prior to beginning the assay. Assay buffer (pH 7.5) contained 0.5% bovine serum albumin in 0.05 M phosphate buffered saline. First antibody (100 μL) was added and to each tube and samples were incubated for 24 h at room temperature. Then GH tracer (^{125}I-labeled GH, 100 μL) was added to each tube and incubated at room temperature for 24 h. Subsequently, 100 μL of second antibody was added to each tube and incubated for 72 h at 4°C. Tubes were decanted and a gamma counter measured binding of radioactivity. Inter and intra assay variation averaged less than 20%, which is acceptable variation (R. Michael Akers, personal communication).

Plasma urea nitrogen. Plasma urea nitrogen (**PUN**) concentration was determined using urease and the indophenol reaction (Chaney and Marbach, 1962; Weatherburn, 1967). Urea standards were prepared by dissolving 0.2142 g urea in 100 ml deionized water to form a stock solution and then further diluting the stock with deionized water to prepare a set of standards. Plasma was assayed in 5 μL aliquots with three replications per sample. Each plasma sample was incubated with urease solution (pH 6.7, 0.104 U urease: 1 μL sample) for 20 min at room temperature. Subsequently, 500 μL of reagent 1 (50 g phenol, 250 g sodium nitroferricyanide dissolved in 1 L deionized water) was added and tubes were mixed thoroughly by vortex. Then 500 μL of reagent 2 (25 g sodium hydroxide and 42 ml bleach, brought to 1 L by adding deionized water) was added and tubes were again vortexed. Finally, 2.5 ml of deionized water was added to each tube, and tubes were vortexed and incubated overnight at room temperature. Samples were then transferred from glass tubes into 96-well plates. Each plate contained blanks and a standard curve. Absorbance of the samples was read on a Microplate Autoreader (Bio-Tek Instruments, Frederick, MD) at dual wavelengths of 405 nm and 625 nm. Inter and intra assay variation averaged less than 5%.

Manufacture of Colostrum Replacement

Bovine blood from abattoirs was collected into stainless steel containers and USDA approved for human consumption. Blood was centrifuged to separate plasma from red blood cells, and plasma was chilled (5°C) and transported to the processing facility. Lipid was

removed from plasma. Immunoglobulin G was then concentrated by removal of bovine serum albumin, centrifugation, ultrafiltration, and spray-drying. The resulting powder contained greater than 90% crude protein, of which more than 50% was IgG. The IgG concentrate was then mixed with lactose, dry fat, dextrose, glycine, salt, emulsifier, lecithin, vitamin/mineral premix, potassium chloride, magnesium sulfate, and flavoring to produce the final colostrum replacement product (Quigley et al., submitted 2001).

Statistical Analysis

Data were analyzed as a split-plot design with repeated measures on the sub-plot (time) using Proc MIXED of SAS (Littell et al., 1996). The model was:

$$Y_{ijklmno} = \mu + S_i + M_j + SM_{ij} + B_k + G_l + BG_{kl} + SB_{ik} + SG_{il} + MB_{jk} + MG_{jl} + SMB_{ijk} + SMG_{ijl} + SBG_{ikl} + MBG_{jkl} + SMBG_{ijkl} + R_m + SMBGR_{ijklm} + A(SMBGR)_{(ijklm)n} + T_o + ST_{io} + MT_{jo} + SMT_{ijo} + BT_{ko} + GT_{lo} + BGT_{klo} + SBT_{iko} + SGT_{ilo} + MBT_{jko} + MGT_{jlo} + \varepsilon_{ijklmno}$$

Where:

S = source of IgG, colostrum or replacement (i = 1, 2); fixed effect

M = milk replacer, no plasma or plasma (j = 1, 2); fixed effect

B = breed, Holstein or Jersey (k = 1, 2); fixed effect

G = gender, female or male (l = 1, 2); fixed effect

R = replication, colostrum pool (m = 1, 2); random effect

A = calf (n = 1…20); (total of 78 calves) random effect

T = time, days or weeks, depending on variable tested, if days (o = 1, 8, 15, 22, 29), if weeks (o = 1…4), fixed effect

ε = residual

Replication and calf were random effects; all other effects were fixed. Tests of the effect of IgG source, milk replacer, breed, gender, and replication were conducted using calf within the interaction of source by replacer, breed, gender, and replication as the error term. Significance was declared at $P < 0.05$. When interactions of main effects were significant, the slice option was used to clarify which effects were significant.

Tested dependent variables included:

a. Plasma IgG, urea N, total protein, IGF-I, GH (6 measurements)
b. Change in IgG, urea N, total protein, IGF-I, GH from baseline (calculated values)
c. Calculated efficiency of absorption of IgG at 24 h

   Calculated as: (plasma IgG at 24 h × BW at birth, kg × 0.091)/IgG intake (g)
d. Weight, wither height, hip height, body length, heart girth (5 measurements)
e. Weekly change in weight, wither height, hip height, body length, heart girth (4 calculated values)
f. Overall change in weight, wither height, hip height, body length, heart girth (calculated values)
g. Starter intake by week and overall (daily measurements summed in 4 weekly or 1 overall total(s))
h. Calculated gain to feed ratio by week and overall (calculated values)

When the variable tested was an overall change, the effect of time (d or wk) was removed from the model. Health and fecal scores were tested by performing chi square tests in Proc FREQ of SAS. In addition, correlations were calculated between selected variables to determine relationships between blood parameters and growth.

After the analysis described above was completed, the effect of birth weight was considered as a covariate in a second analysis to determine if initial weight affected treatment differences. Birth weight was significant, but did not change the significance of treatment effects. Furthermore, the previous inclusion of calf and breed in the model also accounted for

weight differences. Therefore, birth weight was not included as a covariate in the analyses that produced the results reported.

CHAPTER 4. RESULTS AND DISCUSSION

Birth Weight as a Covariate

Birth weight was added to the model as a covariate after initial analysis was conducted. However, it did not change the results of tests for treatment effects and did not produce changes of practical importance in the values obtained for dependent variables. As an example, when weight was added to the model used to test IgG concentrations, the *P* value for the covariate term (weight by breed interaction) was 0.0052, but the estimates of weight effect for each breed were small. For Holsteins, a 5-kg increase in birth weight translated into a 0.1 g/L decrease in plasma IgG. For Jerseys, a 5-kg increase in birth weight would result in a 0.04 g/L decrease in plasma IgG. Since least squares means of plasma IgG were in the range of 13 to 14 g/L, it is unlikely that such small changes are of practical significance.

Colostrum pools (replication) did not differ in IgG content and did not have a significant effect on any of the variables tested.

Plasma IgG Concentrations

Plasma IgG was undetectable in 0 h samples for all treatments and all breed-gender combinations, which verified that calves had no opportunity to nurse their dams prior to the first experimental feeding. After administration of colostrum or colostrum replacement at 1 and 13 h, plasma IgG concentrations rose to 13.76 ± 0.66 g/L in colostrum-fed calves and 13.84 ± 0.65 g/L in replacement-fed calves at 24 h. No difference was detected between treatments (P = 0.9301). Apparent efficiency of absorption (**AEA**) also did not differ between treatments. Mean efficiencies were 19.19 ± 0.79 and 20.31 ± 0.77% for colostrum- and replacement-fed calves respectively. Plasma IgG and AEA results for each treatment are presented in Table 4. Milk replacer effects were not significant in any 0 or 24 h measurements, which was expected because milk replacer treatments had not been applied at these times.

Observed concentrations of IgG at 24 h were within the range of 3 to 21 g/L reported by other researchers (Abel Francisco and Quigley, 1993; Arthington et al., 2000a; Arthington

et al., 2000b; Chelack et al., 1993; Crowley et al., 1994; Fiems et al., 1989; Garry et al., 1996; Mee et al., 1996; Mulvey et al., 1996; Quigley et al., 1998b). Concentrations of IgG obtained by calves fed colostrum replacement were greater than those previously reported (5.0 to 8.3 g/L) for bovine serum-based products (Arthington et al., 2000a; Arthington et al., 2000b; Quigley et al., 1998b; Quigley et al., 2000a). It is likely that feeding a greater amount of IgG to calves caused this difference. In previous research, this colostrum replacement product was fed to provide 90 total g of IgG (Arthington et al., 2000a; Arthington et al., 2000b; Quigley and Drewry, 1998; Quigley et al., 2000a). In the present experiment, calves received 250 (Holsteins) or 180 (Jerseys) total g of IgG in the first 24 h.

The greater mass of IgG fed could also account for differences in AEA between the present study and previous ones. In this experiment, AEA was in the low end of the range of previously reported values for maternal colostrum (30 to 35 %) and this replacement product (50 to 60%) (Arthington et al., 2000a; Arthington et al., 2000b; Besser and Osborn, 1993; Garry et al., 1996; Mee et al., 1996; Quigley and Hopkins, 1997; Quigley et al., 1998b; Quigley et al., 2000a). Increased mass of protein fed reduced absorption efficiency in a previous trial (Besser and Osborn, 1993). However, depression in AEA was not observed until protein mass exceeded 535 g in other reports (Davenport et al., 2000; Quigley et al., 1998b). Although AEA was lower than in other reports, it was similar to a recent experiment conducted at Virginia Tech using an earlier formulation of the same product (Quigley et al., 2000a). Therefore it is possible that herd management factors associated with the Virginia Tech dairy are responsible for decreased AEA.

While no treatment differences in these variables were found, there were differences between Holsteins and Jerseys (Table 5). Jersey calves had higher ($P < 0.0001$) 24-h IgG concentrations than Holstein calves (16.47 ± 0.71 and 11.12 ± 0.60 g/L, respectively). In addition, Jersey calves absorbed IgG with 21.90 ± 0.87 % efficiency compared to 17.00 ± 0.69% for Holsteins ($P = 0.0003$). No differences in IgG concentration or AEA were found between male and female Jerseys, but Holstein males absorbed IgG more efficiently and attained higher IgG concentrations at 24 h than females (Table 4).

Table 4. Least squares means of plasma protein, IgG, IGF-I, growth hormone (GH), and urea nitrogen (PUN) in calves fed colostrum (C) or colostrum replacement (R) and milk replacer with (P) or without (N) animal plasma at birth and at 24 h of age.

	Colostrum				Colostrum Replacement				Differences of Means[1]			
	N		P		N		P		N minus P		C minus R	
Variable	Mean	SE	Mean	SE	Mean	SE	Mean	SE	C	R	N	P
0-h IgG (g/L)	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
24-h IgG (g/L)	13.74	0.89	13.77	0.98	14.05	0.90	13.63	0.92	-0.03	0.42	-0.31	0.15
AEA of IgG[2] (%)	18.73	1.07	19.65	1.17	20.28	1.08	20.35	1.11	-0.92	-0.06	-1.55	-0.69
0-h Total protein (g/dl)	4.312	0.128	4.323	0.139	4.107	0.128	4.282	0.132	-0.012	-0.175	0.205	0.042
24-h Total protein (g/dl)	6.063	0.158	5.946	0.165	5.416	0.158	5.505	0.161	0.116	-0.088	0.647**	0.442**
0-h IGF-I (ng/ml)	141.50	14.75	142.02	16.10	141.61	14.75	127.32	15.25	-0.52	14.28	-0.11	14.69
24-h IGF-I (ng/ml)	109.55	16.21	116.66	17.70	152.11	16.21	115.94	16.76	-7.11	36.18	-42.56	0.72
0-h GH (ng/ml)	11.146	4.459	13.166	4.765	20.009	4.473	14.380	4.589	-2.021	5.630	-8.864	-1.213
24-h GH (ng/ml)	29.193	6.968	25.124	7.606	49.465	7.038	50.069	7.219	4.069	-0.603	-20.272*	-24.945*
0-h PUN (mg/dl)	13.858	1.038	14.569	1.050	15.329	1.050	15.776	1.074	-0.711	-0.446	-1.471	-1.206
24-h PUN (mg/dl)	12.291	0.922	12.900	0.982	10.214	0.925	9.840	0.948	-0.609	0.374	2.077*	3.060**

[1] Significance determined by slicing interaction of colostrum source and milk replacer.
[2] Apparent efficiency of IgG absorption, calculated as: (plasma IgG at 24 h × BW at birth, kg × 0.091)/IgG intake (g).
* $P < 0.05$
** $P < 0.01$

Breed differences in IgG absorption have been reported previously, and have been proposed to result from differences in body size and plasma volume (Quigley et al., 1998a). Jerseys have lower plasma volume as a percentage of body weight, and therefore could appear to have higher amounts of blood constituents than Holsteins as a result of concentration differences (Quigley et al., 1998a). Quigley et al. (2000b) reported that Jersey calves had higher concentrations of IgG at 24 h than Holstein calves (8.5 versus 6.1 g/L). Another study (Quigley et al., 1995a) found 24-h IgG concentrations in 48 Jersey calves ranging from 6.2 to 55.4 g/L (mean 32.4 g/L). Tennant et al. (1969) also reported higher IgG concentrations in the serum of Jersey calves 1 to 5 d old compared to Holstein calves of the same age. In the current study, Jerseys (n = 30) had 24-h IgG ranging from 10.8 to 21.09 g/L, while Holsteins (n = 48) ranged from 0.01 to 17.85 g/L. Other data supports the suggestion that Holsteins have lower average values for IgG at 24 h. Nocek et al. (1984) reported a range of 3.2 to 17.0 g/L in 129 Holstein calves. Tennant et al. proposed that the difference between breeds was caused by differences in absorption efficiency. This postulation was supported by the results of the present experiment. Results of Quigley et al. (2000a) showed AEA for Jerseys and Holsteins similar to the present experiment, however the difference was not significant. Greater number of observations in the current study may account for this discrepancy. The data set of Quigley et al. (2000a) included only 15 Jersey calves out of 60 total, which may have been an inadequate number of calves to detect breed differences.

Table 5. Least squares means of plasma protein, IgG, IGF-I, growth hormone (GH), and urea nitrogen (PUN) in Holstein (H) and Jersey (J), male (M) and female (F) calves at birth and 24 h of age.

	Holstein				Jersey				Differences of Means[1]			
	F		M		F		M		F minus M		H minus J	
Variable	Mean	SE	Mean	SE	Mean	SE	Mean	SE	H	J	F	M
0-h IgG (g/L)	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
24-h IgG (g/L)	9.90	0.86	12.35	0.83	16.75	1.09	16.19	0.90	-2.44*	0.56	-6.85**	-3.84**
AEA of IgG[2] (%)	15.48	1.00	19.74	0.95	21.54	1.37	22.25	1.07	-4.27**	-0.71	-6.07**	-2.51
0-h Total protein (g/dl)	4.284	0.115	4.440	0.110	4.196	0.168	4.104	0.126	-0.155	0.091	0.089	0.335
24-h Total protein (g/dl)	5.203	0.150	5.723	0.150	6.032	0.180	5.972	0.160	-0.520**	0.060	-0.829**	-0.249
0-h IGF-I (ng/ml)	154.88	13.25	152.42	12.70	121.28	19.40	123.86	14.61	2.46	-2.58	33.60	28.56*
24-h IGF-I (ng/ml)	123.93	14.57	111.26	13.96	105.13	21.32	153.94	16.05	12.67	-48.81†	18.80	-42.68*
0-h GH (ng/ml)	15.063	4.189	12.405	4.037	10.917	5.495	20.317	4.454	2.658	-9.400	4.146	-7.912
24-h GH (ng/ml)	48.744	6.643	46.771	6.345	35.467	8.633	22.869	7.024	1.973	12.597	13.277	23.901*
0-h PUN (mg/dl)	13.949	1.001	14.388	0.959	16.008	1.267	15.187	1.049	-0.439	0.821	-2.059	-0.799
24-h PUN (mg/dl)	9.902	0.871	10.408	0.841	12.526	1.125	12.408	0.922	-0.506	0.118	-2.624	-2.000

[1] Significance determined by slicing interaction of breed and gender.

[2] Apparent efficiency of IgG absorption, calculated as: (plasma IgG at 24 h × BW at birth, kg × 0.091)/IgG intake (g).

† $P < 0.10$

* $P < 0.05$

** $P < 0.01$

Plasma IgG concentrations less than 10 g/L (the cutoff point for failure of passive transfer) were observed in 15 calves (19.2%), and 24 h IgG concentrations ranged from 0.01 to 21.09 g/L. Failure of passive transfer (**FPT**) rate and IgG ranges for various subgroups within the study are reported in Table 6. More calves fed colostrum experienced FPT than calves fed replacement (20.5 versus 17.9%). No Jersey calves experienced FPT, and more females than males had 24-h IgG less than 10 g/L. Reports of other research trials have shown FPT in 9.2 to 35% of calves (Hancock, 1985; Paré et al., 1993; Perino et al., 1993; Rea et al., 1996; Robison et al., 1988). NAHMS (1993) reported FPT in 31% of calves surveyed in the National Dairy Heifer Evaluation Project. Even though the percentage of calves that had FPT in this study falls within the range of normally observed values, it is surprisingly high given the tightly controlled conditions of the experiment. Average age of calves at first feeding was 1.05 h, and only 5 calves were more than 2 h old at first feeding. All calves received high quality colostrum or replacement containing 91.3, 84, or 66 g/L of IgG for first pool colostrum, second pool colostrum, and colostrum replacement, respectively. Calves were fed by strictly following best management practices, yet still experienced a high rate of FPT. These results support recommendations that calves be fed high quality colostrum as soon as possible after birth. The results also provide support for the belief that low IgG concentrations are not a death sentence, since only four calves died over the course of the experiment. The mortality rate for calves with serum IgG less than 10 g/L was more than twice that of calves with higher concentrations (NAHMS, 1993). Others (Hancock, 1985; Perino et al., 1993; Robison et al., 1988; White and Andrews, 1986; Wittum and Perino, 1995) have confirmed that calves that experienced FPT had greater risk of morbidity and mortality prior to weaning than those with adequate passive transfer. However, Hancock (1985) reported that calves with the same serum IgG concentration had different

Table 6. Failure of passive transfer incidence.

		% Calves	Range of IgG (g/L)	
	n	< 10 g/L	min	max
Overall	78	19.2	0.01	21.09
Treatment				
Colostrum	39	20.5	6.80	19.95
Replacement	39	17.9	0.01	21.09
Breed				
Jersey	30	0.0	10.80	21.09
Holstein	48	31.3	0.01	17.85
Gender				
Male	44	11.4	7.95	20.39
Female	34	29.4	0.01	21.09

mortality risk depending on the mortality rate of the herd. Calves with low IgG born in herds with a low mortality rate were more likely to survive than calves with the same IgG concentration born in a herd with high mortality.

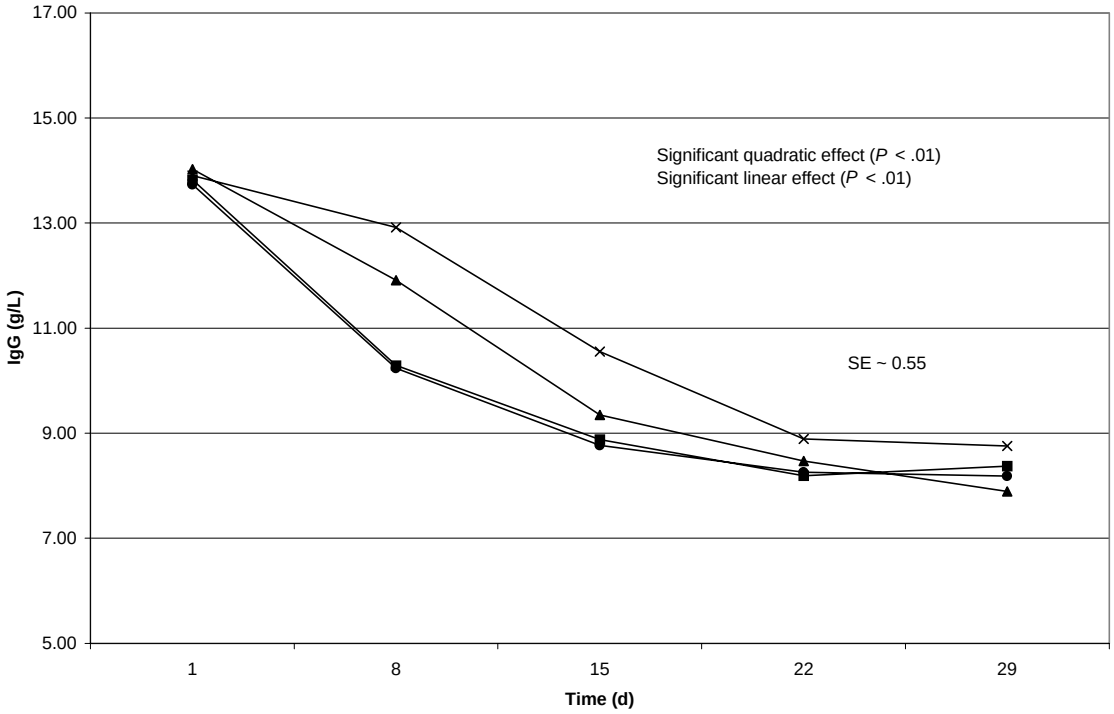

Figure 1. Plasma IgG concentrations (g/L) on d 1 to d 29 in calves fed colostrum at birth and milk replacer without animal plasma for 1 mo (•), colostrum at birth and milk replacer with animal plasma for 1 mo (■), colostrum replacement at birth and milk replacer without animal plasma for 1 mo (▲), or colostrum replacement at birth and milk replacer with animal plasma for 1 mo (×) Significant source by time interaction; differences detected on d 8 and 15.

Clearance of IgG

Concentrations of IgG declined in a quadratic manner ($P < 0.0001$) from d 1 to d 29. As shown in Figure 1, IgG concentrations in calves fed colostrum replacement were higher than in colostrum-fed calves on d 8 ($P = 0.0001$) and d 15 ($P = 0.0416$). There was no difference in IgG concentrations between calves fed plasma in milk replacer and calves fed control milk replacer. The half-life of passively acquired IgG appears to be 22 d, when estimated from the decrease in IgG over time. This estimate agrees with previous work that utilized the same method of calculation (Logan et al., 1972; Sasaki et al., 1997). However, compared to estimates of half-life calculated by clearance of ^{125}I-labeled IgG_1, the current

method overestimates half-life by 4 to 10 d (Besser, 1993; Besser et al., 1988; Sasaki et al., 1977).

To better understand plasma IgG concentrations it is necessary to consider endogenous production and clearance of IgG from plasma. Devery et al. (1979) determined that endogenous production begins by 3 d of life at a rate of 1 g/d. This calculation was based on calves observed in the first 8 d of life, so extrapolation beyond that age is merely speculation. Besser et al. (1988) measured daily clearance rates of 2.6 to 4% of IgG_1 in calves up to 10 d of age. Again, extrapolation of this data should be interpreted cautiously. However, similar clearance rates have been reported in mature cattle (Besser et al., 1988). To estimate clearance rate in the current experiment, change in IgG from d x to d y was divided by IgG on d x (e.g. [(d 1 – d 8)/d 1] × 100). Clearance rates were calculated for each 7-d period (Figure 2). Calves fed colostrum had a clearance rate of 3.5% per d in the first wk, which agrees well with the estimates of Besser et al. (1988). Rate of clearance in the second week (1.98%) was also similar to calculations of Besser et al. Based on these estimates, calves either cleared less IgG or produced more IgG as age increased. It is most likely that endogenous production increased with age, resulting in an apparent decrease in clearance rate in the third wk and a gain in IgG over the fourth wk.

While these estimates are probably appropriate for calves fed colostrum, they may not apply to calves fed colostrum replacement. The ratio of IgG_1 to IgG_2 in serum is approximately 1:1, but in colostrum it is skewed toward IgG_1 (95:5; Butler, 1983). In calves fed colostrum replacement, half of the passively acquired IgG was likely the IgG_2 isotype. The half-life of IgG_2 is unknown; therefore, clearance of IgG may not operate the same way in calves fed colostrum replacement. Calves fed colostrum replacement appeared to clear IgG less rapidly. Alternatively, increased endogenous IgG production may have occurred. Previous work has shown that hypogammaglobulinemic calves begin synthesizing IgG earlier than normogammaglobulinemic calves (Logan et al., 1974). If replacement-fed calves did not have “adequate” IgG_1 after initial absorption, then production of IgG_1 could have been enhanced. The increased IgG_1 production and additional IgG_2 (acquired from the colostrum replacement) may have resulted in a greater ability to fight infections during the

first 2 wk of life (Besser, 1993; Devery et al., 1979) due to larger quantities of antibody and the different actions of IgG_1 and IgG_2. However, differences in the health of calves on each treatment, as evidenced by incidence of diarrhea, were not detected in this trial. The difference in the ratio of IgG_1 to IgG_2 may have another influence; increased concentrations of IgG_2 may inhibit IgG_1 production and lead to lower concentrations of immunity as IgG is cleared from the blood.

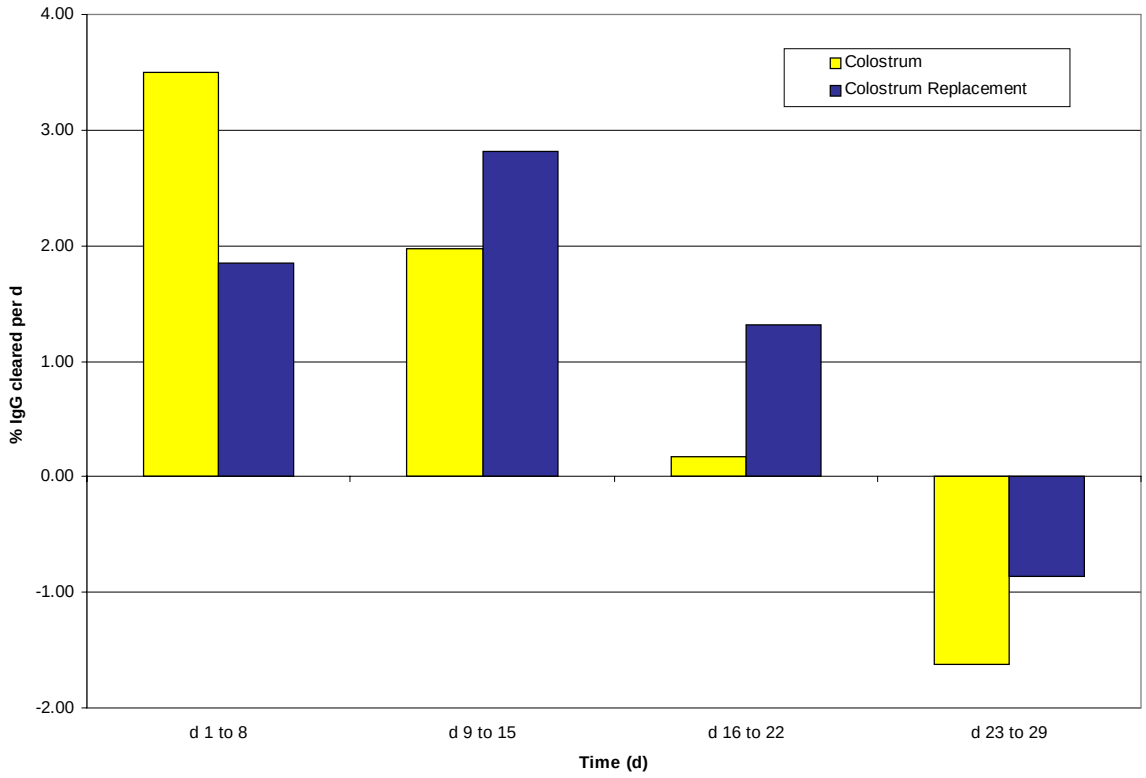

Figure 2. Calculated estimates of plasma IgG clearance rates (% of total plasma amount cleared per d) from d 1 to d 29 of age in calves fed colostrum or colostrum replacement.

Calves with higher initial concentrations of IgG cleared it at a faster rate than calves starting at lower concentrations (Tennant et al., 1969), which is in contrast to the current experiment where all treatment groups started at the same concentration of IgG, and differences in apparent clearance rate were observed. Besser et al. (1988) reported no differences in amount of ^{125}I-labeled IgG_1 in the intestine of calves fed 1 or 3.5 L of colostrum (serum IgG_1 at 5 d was 3.5 and 16.4 g/L, respectively). The authors concluded that

calves with high concentrations of IgG in blood would clear larger total amount of IgG into the intestine, which could increase local immune protection.

Jerseys attained higher IgG concentrations than Holsteins (Figure 3) on d 1 ($P < 0.0001$) and retained higher concentrations on d 8 ($P < 0.0001$) and d 15 ($P = 0.0024$), which may have increased their immunity to disease. This theory was not supported by health data, as Jersey males had greater incidence of diarrhea than all other groups (full discussion in the Health section). The increased IgG in Jersey calves could be due to the greater amount of IgG originally absorbed. If clearance rate was equivalent for Jerseys and Holsteins (as suggested by Besser et al., 1988), then more time would be required to excrete the larger quantity of IgG in the plasma of Jersey calves, and Jerseys would excrete more IgG into the intestine over time.

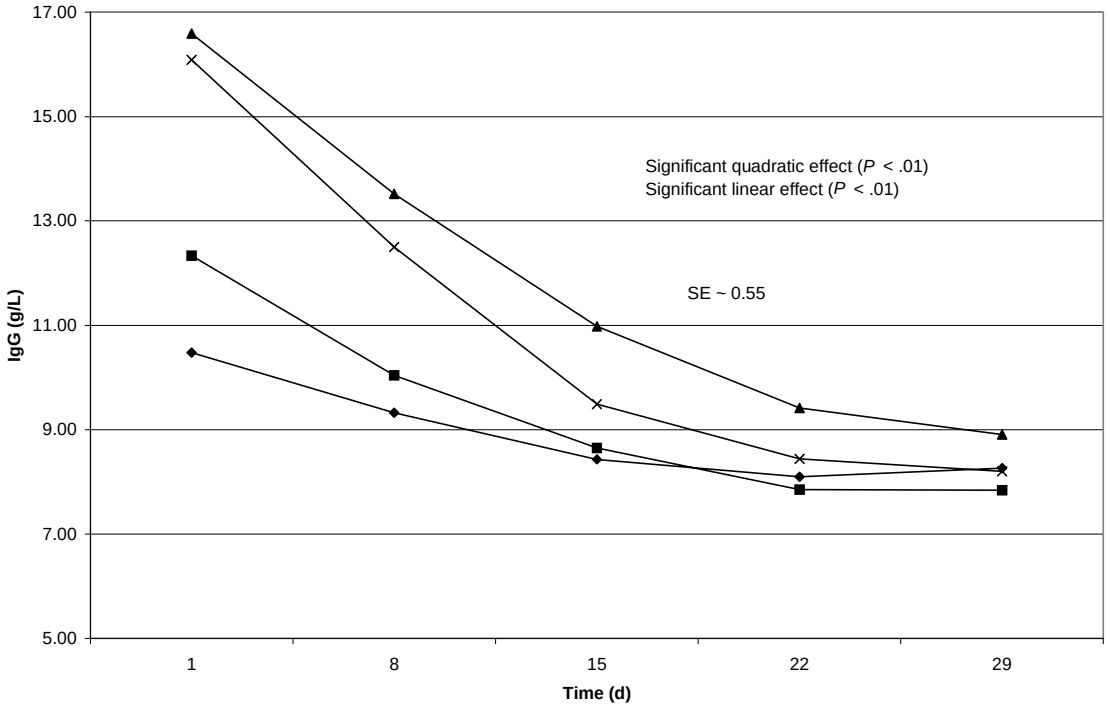

Figure 3. Plasma IgG concentrations (g/L) on d 1 to d 29 in Holstein females (♦), Holstein males (■), Jersey females (▲), and Jersey males (×). Significant breed by time interaction; differences detected on d 1, 8, and 15.

Plasma IGF-I and GH

No differences were detected between treatment groups (Figure 4). Among male calves, Jerseys had greater concentrations of IGF-I on d 1 than Holsteins (P 0.0469), but no differences in breed or gender were detected throughout the rest of the experiment (Appendix A), probably due to extreme variability in results. Plasma IGF-I decreased from d 0 to d 1 in all but the RN treatment group (Table 4). In all groups, plasma IGF-I decreased until d 22 and then increased from d 23 to d 29. Testing of contrasts showed significant quadratic ($P <$ 0.0001) and linear ($P = 0.0021$) effects on the shape of the curve. Concentrations of IGF-I are consistent with the range of published results in newborn calves in the first wk of life (Hadorn et al., 1997; Hammon et al., 2000; Kühne et al., 2000; Rauprich et al., 2000). Published results for wk 2 to 4 were not found. Changes in IGF-I and GH were opposite in the first 24 h, and concentrations of the two hormones were not correlated throughout the study. These findings agree with the reports of Kühne et al. (2000) and Grütter and Blum (1991), who suggested that in the very young calf, physiological concentrations of GH do not increase IGF-I. Considerable research effort has been directed at determining the relationship of IGF-I and IgG in the young calf in recent years. Colostrum contains very high concentrations of IGF-I compared to whole milk, and accumulation of IGF-I and of IgG in colostrum occur at about the same time and at the same rate (Baumrucker and Blum, 1993). Thus it seems reasonable to think that a relationship between IGF-I and IgG exists in the newborn calf. However, to date no research has shown conclusively that IGF-I affects IgG in any way. Feeding IGF-I caused an increase in intestinal growth in both calves and piglets in some studies (Baumrucker and Blum, 1993; Baumrucker et al., 1994b; Bühler et al., 1998; Xu et al., 1994; Xu et al., 1996). Current belief is that although IGF-I is barely absorbed by the intestine of the newborn calf, it may enhance development of the neonatal digestive system and may affect or interact with other growth factors to influence the neonatal intestine (Odle et al., 1996; Vacher et al., 1995). The mechanism of colostral IGF-I action and the specific influence it may have on the neonate remain unclear.

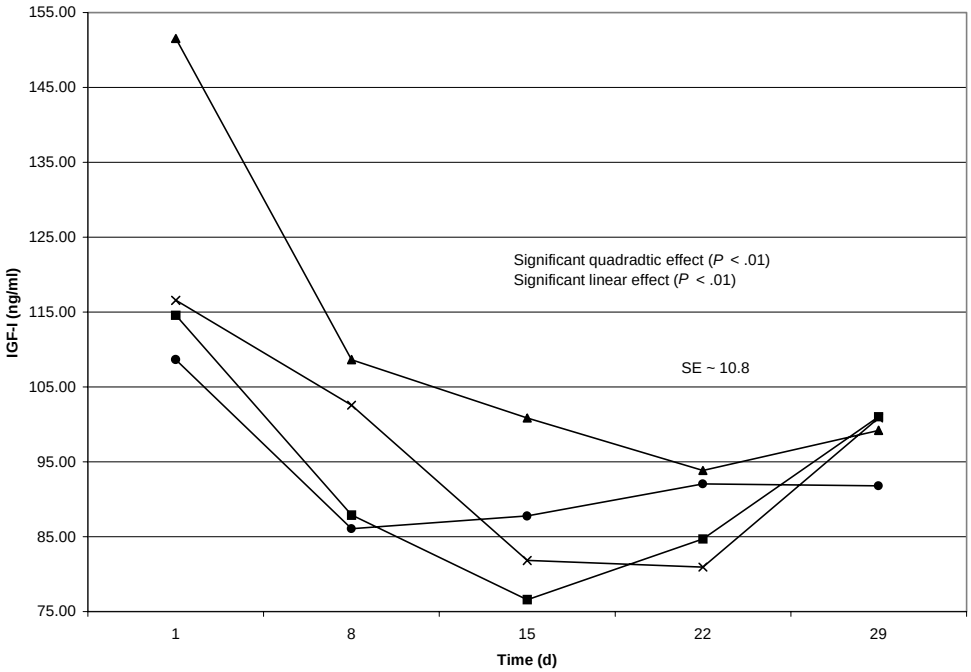

Figure 4. Plasma IGF-I concentrations (ng/ml) on d 1 to d 29 in calves fed colostrum at birth and milk replacer without animal plasma for 1 mo (●), colostrum at birth and milk replacer with animal plasma for 1 mo (■), colostrum replacement at birth and milk replacer without animal plasma for 1 mo (▲), or colostrum replacement at birth and milk replacer with animal plasma for 1 mo (×).

No differences were detected between groups in the concentration of GH on d 0 (P = 0.1885), and GH concentrations in plasma increased in all treatment groups from d 0 to d 1 (Table 4). However, concentrations of GH were higher (P < 0.0001) in replacement-fed calves than in colostrum-fed calves on d 1. Plasma GH concentrations decreased from d 1 to d 29 (Figure 5), with significant linear (P < 0.0001) and quartic (P = 0.0060) contrasts detected. Kühne et al. (2000) reported increased GH concentrations during the first wk of life in calves fed milk replacer compared to calves fed colostrum at birth. Calves fed milk replacer had a greater increase in GH after the initial feeding, and plasma GH remained higher through the first wk of life. Concentrations of GH reported in calves at birth and on the first d of life range from 10 to 25 ng/ml for samples taken from 2 to 4 h after birth and 10 to 30 ng/ml at approximately 24 h (Grütter and Blum, 1991; Hadorn et al., 1997; Hammon and Blum, 1997; Hammon et al., 2000; Kühne et al., 2000; Rauprich et al., 2000). Colostrum-fed calves in the current study had GH concentrations within these ranges, but replacement-fed calves had considerably higher GH at 24 h.

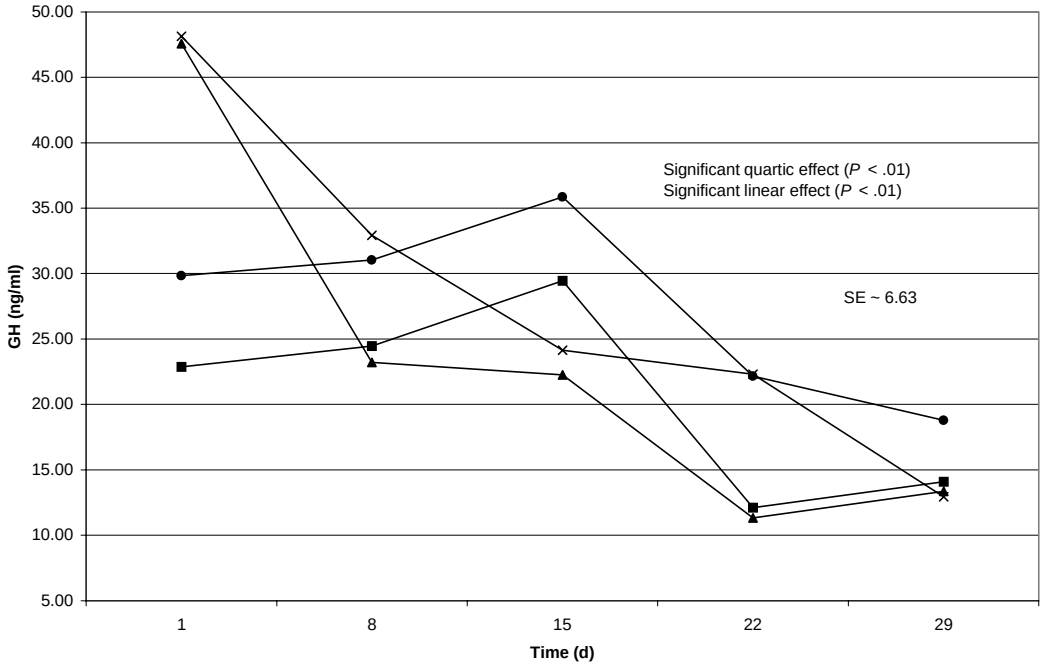

Figure 5. Plasma GH concentrations (ng/ml) on d 1 to d 29 in calves fed colostrum at birth and milk replacer without animal plasma for 1 mo (●), colostrum at birth and milk replacer with animal plasma for 1 mo (■), colostrum replacement at birth and milk replacer without animal plasma for 1 mo (▲), or colostrum replacement at birth and milk replacer with animal plasma for 1 mo (×). Significant source by time interaction; differences detected on d 1 and 15.

Transfer of GH across the placenta is minimal (Oxender et al., 1972), and colostrum contains very low concentrations of GH (Blum and Hammon, 1999). In spite of these facts, fetal and neonatal concentrations of GH are high. Oxender et al. (1972) reported that fetal serum GH increased from 90 to 260 d of gestation and was 10 to 20 times greater than maternal concentrations. Neonates had 36 ± 9 ng/ml of GH on the d of birth (sampled within 2 h of birth) and averaged 27 ng/ml throughout the first wk of life (Oxender et al., 1972). Furthermore, while colostrum normally contains low concentrations of GH, the GH concentration of the replacement product was unknown. If GH was present in high amounts, intestinal absorption of GH could account for differences between treatments. Since digestive enzyme activity is low in the neonate at birth and intestinal absorption is indiscriminate in the first 24 h, intact GH should be readily absorbed if present in the intestine. Differences in GH between treatment groups in the current study also may be related to nutritional factors. In 10-d old male calves, Coxam et al. (1989b) reported that increased triglyceride concentrations depressed GH secretion. Differences in the fat content and composition

between colostrum and the replacement product may have elicited differential responses in GH secretion in the calves of the current experiment during the first 24 h of life. Colostrum was not analyzed for fat content, but on average contains 6.7% fat (Foley and Otterby, 1978). The replacement product contained only 0.1% fat when reconstituted to 24% dry matter. Considerably higher fat concentrations in colostrum compared to the replacement probably influenced the energy balance of calves, and possibly affected GH secretion.

Colostrum-fed calves in this study had higher concentrations of GH on d 15 (P = 0.0454, Figure 5). These differences may also be related to nutrition. In contrast to younger calves, calves 30 d of age responded to high concentrations of triglycerides with increased GH secretion (Coxam et al., 1989a). If colostrum-fed calves in the present investigation initially received greater amounts of fat, their body stores of fat may have remained higher than those of replacement-fed calves. Thus, during a period of nutritional stress on the calf between 2 and 3 wk of age, body fat mobilization could have increased triglyceride concentrations in colostrum-fed calves and enhanced GH secretion. It is also important to note that GH secretion in the young animal is pulsatile. Using serial blood collections every 15 min for 6 h per d, Brückmann et al. (2000) observed that, at 3 d of age, calves had 3.3 ± 0.6 to 4.6 ± 0.4 episodes of GH secretion per h. Amplitude of the pulses ranged from 34.0 ± 7.2 to 40.5 ± 5.4 ng/ml in these calves. Frequency and amplitude of GH pulses were not reduced significantly by 21 d of age. Therefore, due to infrequent sampling in the current experiment, GH results could be influenced by natural variation due to pulsatile secretion, and these results must be interpreted with great caution.

Holstein calves had higher concentrations of plasma GH than Jersey calves on d 1 (P < 0.0001) and d 8 (P = 0.0111, Figure 6). Plasma GH concentrations in young Jersey calves were not found in the literature, therefore direct comparison of breed effect cannot be made. Concentrations of growth hormone in mature Jerseys and Holsteins were not found in the literature either, although differences in adult cattle would help to explain the differences seen in this study.

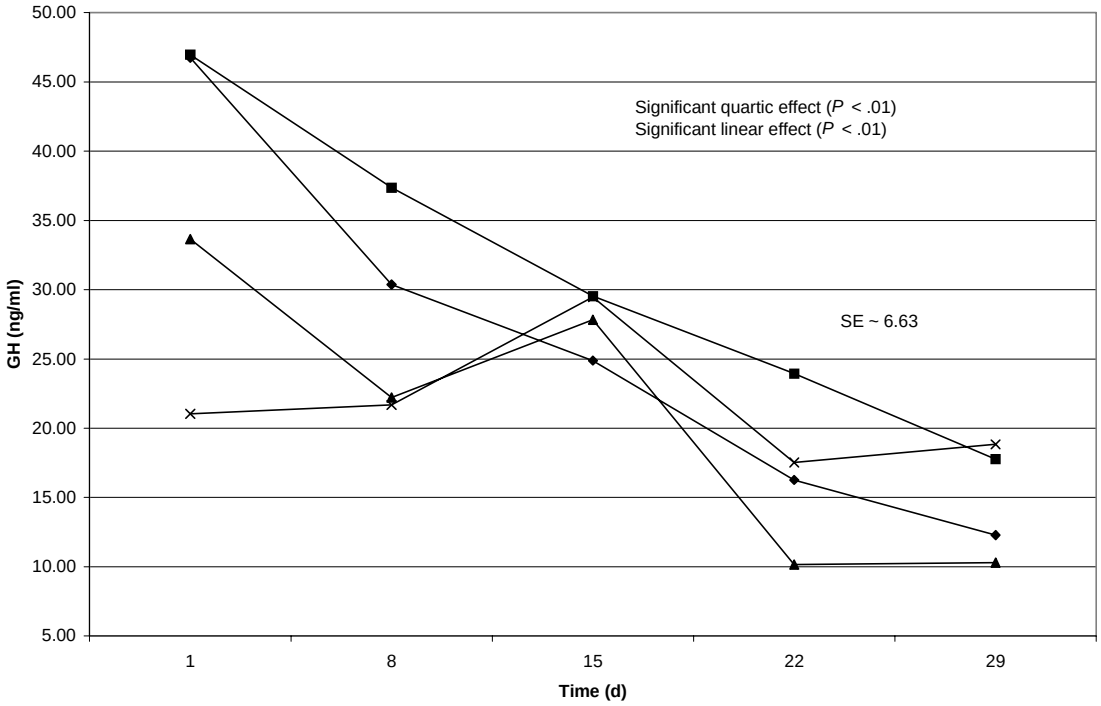

Figure 6. Plasma GH concentrations (ng/ml) on d 1 to d 29 in Holstein females (♦), Holstein males (■), Jersey females (▲), and Jersey males (×). Significant breed by time interaction; differences detected on d 1 and 8.

Growth

Body weights were not different between treatment groups at birth (Table 1). Furthermore, no differences between treatment groups were observed in body weight from d 8 throughout the rest of the study (Figure 7). Body weight on d 1 was greater in calves fed the replacement. The difference in weight was 3 kg, which is only slightly more than the difference in the weight of the liquid fed to calves on different treatments (2.2 kg). Replacement-fed calves received more liquid in the first 24 h than colostrum-fed calves due to the high quality of colostrum. (Lower volumes of colostrum with high IgG concentration were required since amount fed was based on IgG provided.) Due to differences in body weight between d 0 and d 1, and the assumption that added weight was merely fluid, not true body mass increase; d 1 weights were replaced with d 0 weights. Contrast tests and calculations of average daily gains were adjusted accordingly. Body weights increased with age in a quadratic fashion ($P < 0.0001$). The osmolarity of the replacement product was not

tested, but if it differs from colostrum, differences in d 1 weight also may have been affected by differential fluid retention.

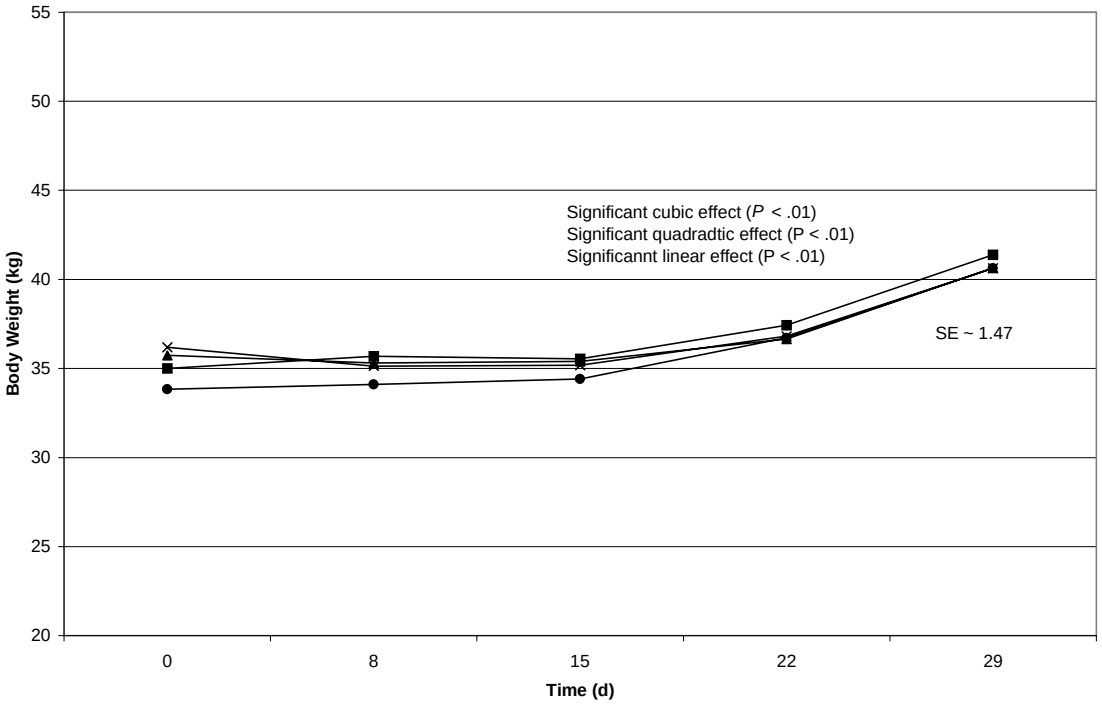

Figure 7. Body weight (kg) on d 0 to d 29 in calves fed colostrum at birth and milk replacer without animal plasma for 1 mo (•), colostrum at birth and milk replacer with animal plasma for 1 mo (■), colostrum replacement at birth and milk replacer without animal plasma for 1 mo (▲), or colostrum replacement at birth and milk replacer with animal plasma for 1 mo (×).

As expected, Holstein calves weighed more than Jersey calves (average weight over all d of 45.75 ± 1.7 and 27.50 ± 1.2 kg, respectively) on all d ($P < 0.0001$, Figure 8). Holstein calves (43.68 ± 1.78 kg) were slightly heavier at birth than target weights (40 kg) published by Kertz et al. (1998). Holsteins (51.21 ± 1.12 kg) were lighter at d 29 than published standards (62.1 ± 0.76 kg; Heinrichs and Hargrove, 1987). Jerseys (30.42 ± 1.31 kg) also were lighter than published standards (42.1 ± 0.79 kg; Heinrichs and Hargrove, 1991).

Rate of weight gain differed over time, being lowest from d 9 to d 15 and highest from d 23 to 29 (Figure 9). Kertz et al. (1979) also reported this decrease in gains during the second wk, but only in calves grouped as poor performers based on their overall weight gain from wk 1 to 4.

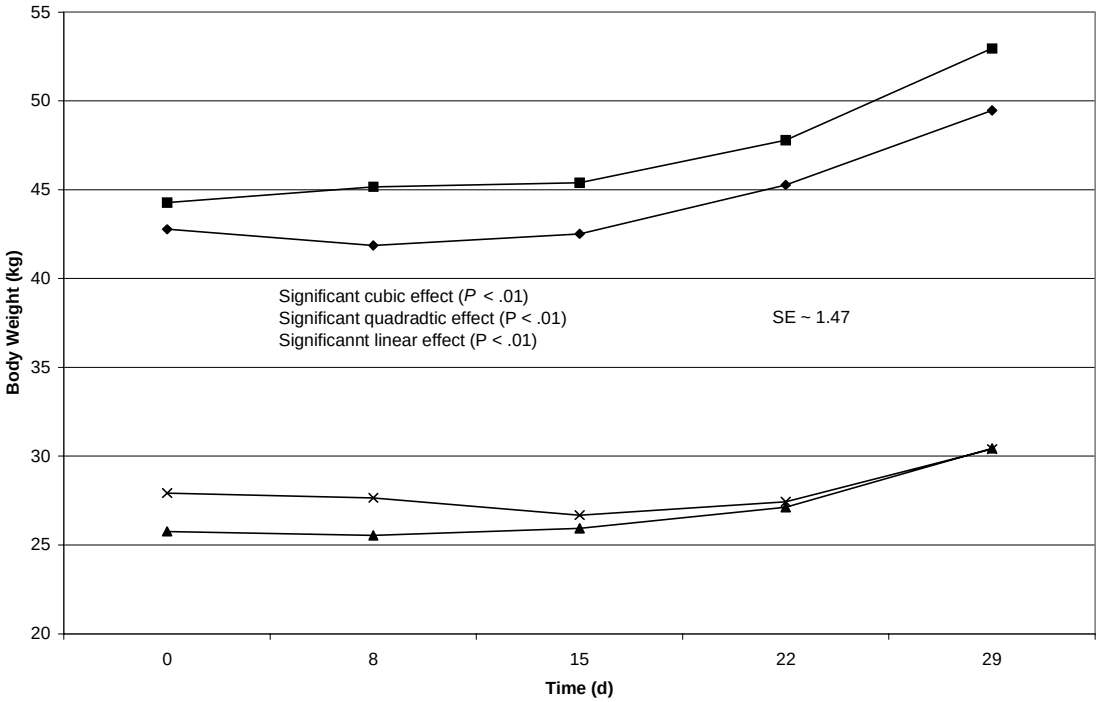

Figure 8. Body weight (kg) on d 1 to d 29 of Holstein females (♦), Holstein males (■), Jersey females (▲), and Jersey males (×). Significant breed by time interaction; differences detected on all d.

It is possible that this time period is critical in determining the growth of calves. Passive immunity is waning at this time and the calf's own immune system must take over responsibility for protecting the body (Besser, 1993). In addition, the calf must receive adequate energy and protein to support maintenance and growth requirements. These factors may become more important if the calf is exposed to a large load of infectious agents and at the same time is malnourished. From d 0 to d 8, calves fed replacement at birth tended to gain less weight than calves fed colostrum (P = 0.0560; -105.6 and 45.1 g/d, respectively). By d 15, there were no differences between treatments. Overall weight gain and average daily gains (ADG) were not different for the four treatment groups (Table 7). However, Holstein males had greater gains (8.591 kg overall and 309 g/d) than any other combination of breed and gender (Table 8). Appendix B presents changes in weight gain for breed-gender groups. Published values for ADG and growth rate vary widely due to differences in experimental conditions, and range from 199 to 790 g/d, with most values in the range of 400 to 500 g/d.

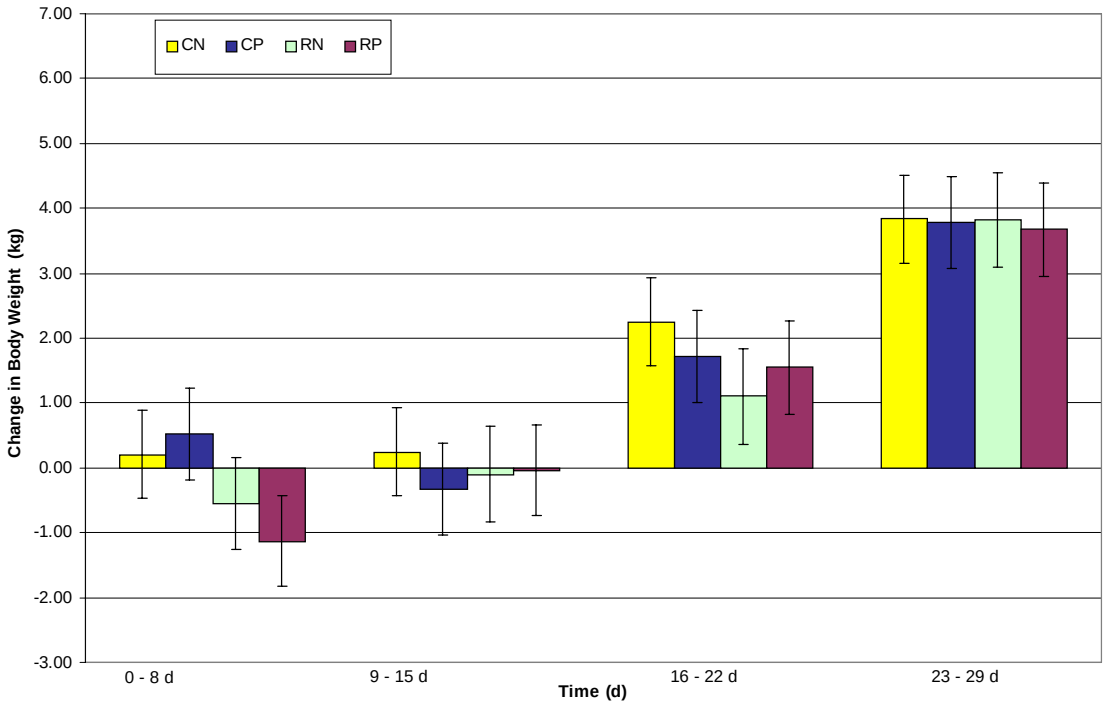

Figure 9. Body weight change (kg/wk) in calves fed colostrum at birth and milk replacer without animal plasma for 1 mo (CN), colostrum at birth and milk replacer with animal plasma for 1 mo (CP), colostrum replacement at birth and milk replacer without animal plasma for 1 mo (RN), or colostrum replacement at birth and milk replacer with animal plasma for 1 mo (RP).

Wither height did not differ by treatment and increased linearly from d 1 to d 29 (Appendix C). Holsteins were taller at the withers than Jerseys initially (76.7 cm, compared to 68.0 cm on d 1) and throughout the study (Figure 10). Increase in wither height from d 1 to d 29 was similar among males and females of the same breed, but Holstein females gained more than Jersey females (Table 8). Holsteins (80.77 ± 0.99 cm) were on target at d 29 compared to some published values (80.1 ± 0.30 cm; Heinrichs and Hargrove, 1987), and taller than others (74 to 78 cm; Kertz et al., 1998; Lammers et al., 1998). However, Jerseys (70.95 ± 1.16 cm) were shorter than published standards (73.3 ± 0.60 cm; Heinrichs and Hargrove, 1991). Rate of gain in wither height was highly variable, but calves fed milk replacer containing no animal plasma had greater gains from d 1 to d 8 (P = 0.0091) and d 16 to d 21 (P = 0.0341) than calves fed animal plasma protein in milk replacer (Appendix D).

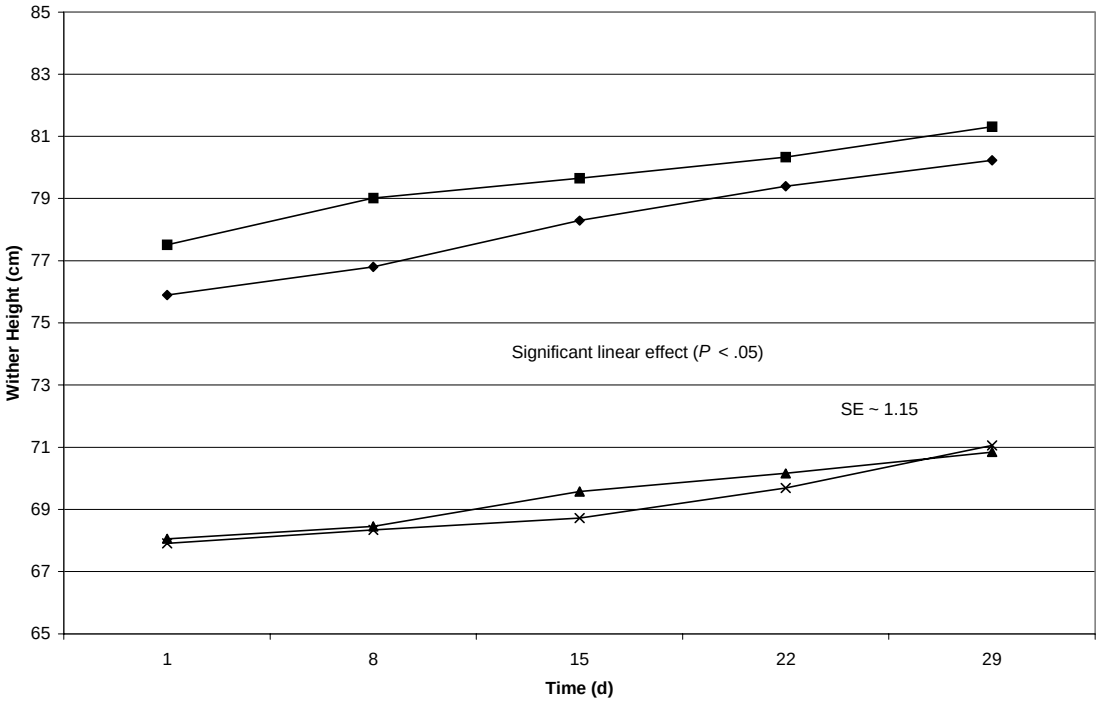

Figure 10. Wither height (cm) on d 1 to d 29 of Holstein females (♦), Holstein males (■), Jersey females (▲), and Jersey males (×).

Table 7. Least squares means of growth parameters in calves fed colostrum (C) or colostrum replacement (R) and milk replacer with (P) or without (N) animal plasma.

	Colostrum				Colostrum Replacement				Differences of Means[1]			
	N		P		N		P		N minus P		C minus R	
Variable	Mean	SE	Mean	SE	Mean	SE	Mean	SE	C	R	N	P
Weight gain d 0 – 29 (kg)	6.524	1.383	5.704	1.436	4.230	1.457	4.296	1.442	0.820	0.024	2.204	1.408
ADG d 0 – 29 (kg/d)	0.233	0.054	0.203	0.056	0.153	0.057	0.145	0.056	0.030	0.008	0.080	0.058
Wither height gain d 1 – 29 (cm)	4.06	0.60	2.67	0.64	3.62	0.66	3.02	0.64	1.39	0.59	0.44	-0.35
Hip height gain d 1 – 29 (cm)	3.30	0.77	3.28	0.84	2.91	0.87	5.16	0.83	0.02	-2.24	0.38	-1.88
Body length gain d 1 – 29 (cm)	3.82	1.03	3.22	1.13	3.85	1.17	3.11	1.12	0.60	0.74	0.03	0.11
Heart girth gain d 1 – 29 (cm)	5.82	0.53	5.18	0.58	3.68	0.61	3.76	0.58	0.65	-0.08	2.14*	1.42

[1] Significance determined by slicing interaction of colostrum source and milk replacer.
* $P < 0.05$

Table 8. Least squares means of growth parameters in Holstein (H) and Jersey (J), male (M) and female (F) calves.

	Holstein				Jersey				Differences of Means[1]			
	F		M		F		M		F minus M		H minus J	
Variable	Mean	SE	Mean	SE	Mean	SE	Mean	SE	H	J	F	M
Weight gain d 0 – 29 (kg)	6.420	1.363	8.591	1.346	3.789	1.572	2.045	1.434	-2.172*	1.744	2.631	6.546**
ADG d 0 – 29 (kg/d)	0.222	0.053	0.309	0.052	0.136	0.063	0.066	0.055	-0.087*	0.070	0.087	0.243**
Wither height gain d 1 – 29 (cm)	4.24	0.57	3.74	0.56	2.54	0.77	2.86	0.63	0.50	-0.32	1.70*	0.88
Hip height gain d 1 – 29 (cm)	4.74	0.69	4.23	0.68	2.25	1.06	3.42	0.82	0.51	-1.18	2.49*	0.81
Body length gain d 1 – 29 (cm)	4.58	0.93	4.28	0.91	2.49	1.43	2.65	1.10	0.30	-0.16	2.10	1.64
Heart girth gain d 1 – 29 (cm)	5.52	0.48	5.69	0.47	4.45	0.74	2.78	0.57	-0.17	1.68	1.08	2.92**

[1] Significance determined by slicing interaction of breed and gender.
* $P < 0.05$
** $P < 0.01$

Hip height was highly variable and did not differ between treatments. Hip height increased from d 1 to d 29 (Appendix E), and contrast tests to determine the shape of the curve showed significant quadratic ($P = 0.0281$) and linear ($P < 0.0001$) effects. Holsteins were taller at the hips (82.32 ± 0.73 cm) than Jerseys (70.99 ± 0.81 cm, $P < 0.0001$), which was expected (Appendix F). Increase in hip height from d 1 to d 29 was not different between males and females of the same breed, but Holstein females gained more than Jersey females (Table 8). There were no differences between treatments or between breed-gender combinations in the rate of increase in hip height. Values for hip height in calves were not found in the literature.

Body length also was highly variable and did not differ between treatment groups. Body length increased linearly ($P < 0.0001$) from d 1 to d 29 (Appendix G). Holsteins were longer than Jerseys ($P < 0.0001$, 74.00 ± 0.50 and 65.21 ± 0.62 cm, respectively; Appendix H), but overall increase in body length (d 1 to d 29) was not different between breeds (Table 8). Increases in body length also did not differ by gender. Values for body length of dairy calves were not found in the literature, although Wilson et al. (1997) reported an average of 72.8 ± 0.11 cm in 2-wk-old, Holstein male veal calves.

Heart girth increased linearly from d 1 to d 29 ($P < 0.0001$, Appendix I). No differences in heart girth were observed between treatments, but Holsteins had greater girth than Jerseys, as expected ($P < 0.0001$, 80.51 ± 0.83 and 68.97 ± 0.96 cm, respectively; Appendix J). Holstein calves had smaller girth than values (82.30 ± 1.02 cm at birth) published by Lammers et al. (1998), and were smaller than values (86.0 ± 0.11 cm) published for veal calves at 2 wk of age (Wilson et al., 1997). Overall increase in heart girth did not differ between breeds or genders (Table 8).

Calves fed milk replacer containing animal plasma grew at the same rate as calves fed control milk replacer, which indicates that animal plasma can successfully replace up to 20% of crude protein in milk replacers for young calves. Other research concerning the use of animal plasma found similar results, however, gains in the current experiment were lower than those previously reported (Morrill et al., 1995; Quigley and Bernard, 1996). Calves fed

milk replacer containing 25% of crude protein from bovine plasma protein gained 12.7 kg over a 6-wk period (302 g/d), while control calves gained 10.8 kg (257 g/d; Morrill et al., 1995). This difference was not significant. Increases in wither height and heart girth were 10.0 ± 0.8 and 2.3 ± 0.5 cm, respectively, and were not different between treatments (Morrill et al., 1995). Quigley and Bernard (1996) reported average daily gains over 28 d of 473 ± 17 g/d for 68 calves fed milk replacer with or without animal plasma. Treatment groups did not differ in body weight or body weight gain.

Intake and Feed Efficiency

Dry matter intake (**DMI**) was monitored for milk replacer and starter, but because milk replacer DMI remained constant throughout the study, starter intake drove changes in total DMI. Therefore, only total DMI results are discussed (starter and milk replacer intakes are reported in Tables 9 and 10). Dry matter intake (Figure 11) increased in a quadratic manner ($P < 0.0001$) from d 1 to d 29 (linear contrast was also significant, $P < 0.0001$). No differences were detected between treatments, but Holsteins consumed more dry matter than Jerseys ($P < 0.0001$, Tables 9 and 10). From d 1 to d 8 Holsteins consumed 48.1 ± 0.02 g/d of dry matter; Jerseys ate 32.4 ± 0.03 g/d (Appendix K). Part of this difference is accounted for by the difference in body weight between breeds. Milk replacer was fed at 31% of metabolic body weight, so Holsteins inherently had greater milk replacer intake. Between d 23 and d 29, Holsteins consumed 93.6 ± 0.02 g/d of dry matter, and Jerseys ate 58.7 ± 0.03 g/d. Holsteins more efficiently converted feed into body weight gain (Table 10). Over the 4-wk experiment, Holstein calves averaged 0.391 kg gain/kg DMI compared to 0.244 kg gain/kg DMI for Jerseys (Appendix L). Holstein males were much more efficient than Jersey males (0.445 versus 0.179 kg gain/kg DMI), which is not surprising due to increased incidence of diarrhea in Jersey males compared to all other groups (full discussion in Health section).

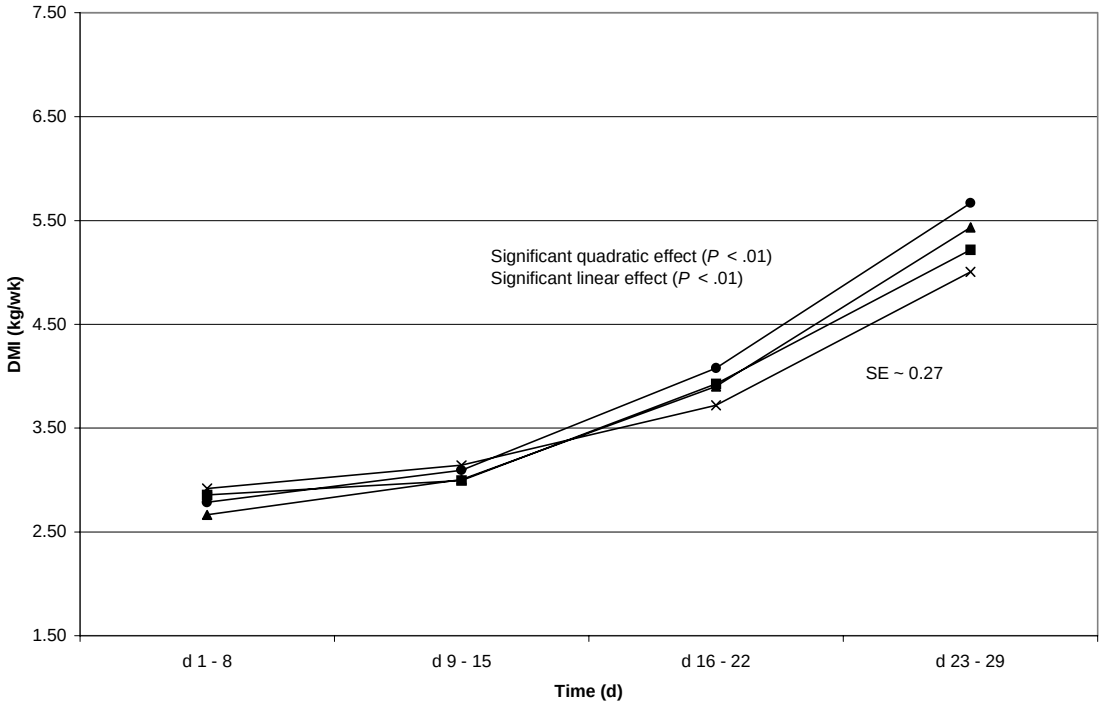

Figure 11. Dry matter intake (kg/wk) of calves fed colostrum at birth and milk replacer without animal plasma for 1 mo (●), colostrum at birth and milk replacer with animal plasma for 1 mo (■), colostrum replacement at birth and milk replacer without animal plasma for 1 mo (▲), or colostrum replacement at birth and milk replacer with animal plasma for 1 mo (×).

Table 9. Least squares means of dry matter intake (DMI) and feed efficiency in calves fed colostrum (C) or colostrum replacement (R) and milk replacer with (P) or without (N) animal plasma.

	Colostrum				Colostrum Replacement				Differences of Means[1]			
	N		P		N		P		N minus P		C minus R	
Variable	Mean	SE	Mean	SE	Mean	SE	Mean	SE	C	R	N	P
Total DMI (kg)	15.668	1.196	14.996	1.317	13.667	1.210	14.586	1.234	0.671	-0.918	2.000	0.411
Milk Replacer DMI (kg)	10.681	0.564	11.036	0.617	9.782	0.568	10.925	0.585	-0.355	-1.143	0.899	0.112
Starter DMI (kg)	4.951	0.744	3.940	0.819	3.851	0.753	3.705	0.770	1.011	0.145	1.100	0.234
Feed Efficiency[2]	0.432	0.086	0.357	0.087	0.224	0.088	0.258	0.087	0.075	-0.034	0.208**	0.099

[1] Significance determined by slicing interaction of colostrum source and milk replacer.
[2] Feed efficiency calculated as: (kg BW gain)/(kg DMI).
** $P < 0.01$

Table 10. Least squares means of dry matter intake (DMI) and feed efficiency in Holstein (H) and Jersey (J), male (M) and female (F) calves.

	Holstein				Jersey				Differences of Means[1]			
	F		M		F		M		F minus M		H minus J	
Variable	Mean	SE	Mean	SE	Mean	SE	Mean	SE	H	J	F	M
Total DMI (kg)	18.466	1.165	18.463	1.121	11.415	1.440	10.574	1.210	0.004	0.841	7.052**	7.889**
Milk Replacer DMI (kg)	12.812	0.524	12.653	0.499	8.336	0.722	8.623	0.565	0.159	-0.286	4.476**	4.031**
Starter DMI (kg)	5.560	0.721	5.798	0.692	3.154	0.905	1.935	0.753	-0.237	1.219	2.407*	3.863**
Feed Efficiency[2]	0.338	0.084	0.445	0.084	0.309	0.093	0.179	0.087	-0.107*	0.130*	0.029	0.266**

[1] Significance determined by slicing interaction of breed and gender.
[2] Feed efficiency calculated as: (kg BW gain)/(kg DMI).
* $P < 0.05$
** $P < 0.01$

Calves were least efficient at the beginning of the experiment; efficiency increased linearly from d 1 to d 29 ($P < 0.0001$). In the first 8 d of the trial, colostrum-fed calves did not lose weight, and therefore had greater feed efficiency than replacement-fed calves (0.145 ± 0.15 and –0.372 ± 0.15 kg gain/kg DMI, respectively, $P = 0.0009$, Figure 12). However, no differences existed between treatments after d 8. Gain to feed ratios reported by Quigley and Bernard (1996) for calves fed milk replacer with or without animal plasma were 0.442 and 0.469 kg gain/kg DMI, respectively. Calves in the current study gained less weight and were less efficient in converting dry matter into body weight. However, observed feed efficiencies fall within the range of values (0.330 to .680 kg gain/kg DMI) reported by other researchers (Crowley et al., 1994; Kertz et al., 1979; Lammers et al., 1998; Quigley et al., 2000b; Richard et al., 1988; Seymour et al., 1995; Terosky et al., 1997). Jersey calves (n = 48) had feed efficiencies of 0.164 to 0.305 kg gain/kg DMI (Quigley et al., 1995a) and 0.504 to 0.666 kg gain/kg DMI (n = 96; Quigley et al., 1995b). Holstein calves (n = 277) at the Purina Research Farm over a 3-yr period averaged 0.375 kg gain/kg DMI (Kertz et al., 1979).

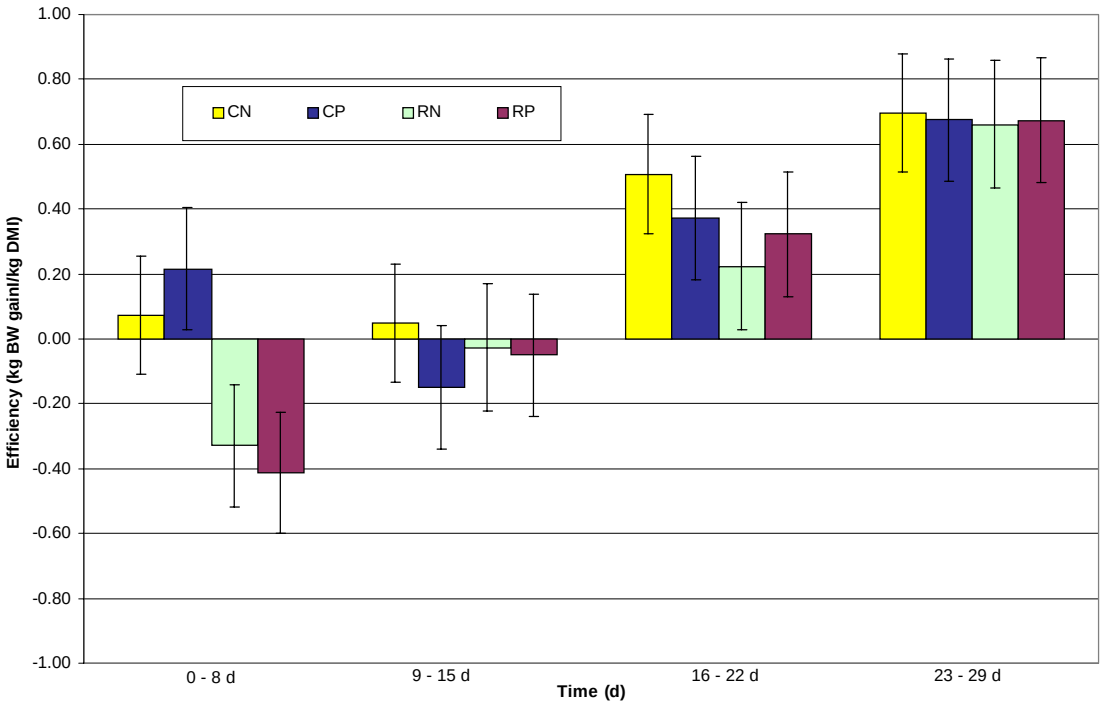

Figure 12. Feed efficiency (kg body weight gain/kg dry matter intake) in calves fed colostrum at birth and milk replacer without animal plasma for 1 mo (CN), colostrum at birth and milk replacer with animal plasma for 1 mo (CP), colostrum replacement at birth and milk replacer without animal plasma for 1 mo (RN), or colostrum replacement at birth and milk replacer with animal plasma for 1 mo (RP).

Plasma Urea Nitrogen

No differences in plasma urea nitrogen (PUN) were found between treatments or breed-gender groups at birth (Tables 3 and 4), however, calves fed colostrum had higher concentrations of PUN on d 1 ($P = 0.0012$) than calves fed colostrum replacement (12.60 ± 0.78 and 10.03 ± 0.77 mg/dl, respectively, Figure 13). Replacement-fed calves received a greater volume of liquid in the first 24 h of life, and experienced a greater increase in plasma volume from d 0 to d 1 than colostrum-fed calves (4.39 ± 0.75 compared to 2.63 ± 0.76%). Therefore, PUN concentrations may be diluted in replacement-fed calves on d 1.

Differences in crude protein content of feeds may also contribute to differences between calves fed replacement and calves fed colostrum. The colostrum was not analyzed for protein content, but on average colostrum contains about 15% protein (Foley and Otterby, 1978). The replacement product contained 10% protein (Table 2). Greater total protein content in the colostrum compared to the replacement product could cause an excess of dietary protein in the colostrum-fed calves. Calves fed colostrum had higher total plasma protein on d 1 than calves fed replacement ($P < 0.0001$), so it is likely that the colostrum provided more total protein than the replacement (Appendix M). In older animals, this excess protein would likely be metabolized into urea for excretion from the body, but the digestive system of the calf is not fully functional in the first 24 h. Activity of pancreatic enzymes and development of the ruminant digestive system increase over time. By 3 wk of age calves are more able to digest non-milk proteins (Davis and Drackley, 1998; Heinrichs, 1994). Therefore, differences in PUN concentrations are probably due to differences in plasma volume.

From d 1 to d 22, Jerseys had higher PUN concentrations than Holsteins ($P < 0.01$, Figure 14). This effect also could be attributed to differences in body size and plasma volume. Jersey calves fed the replacement experienced greater plasma volume expansion than Holstein calves fed replacement (5.72 ± 0.97 and 3.07 ± 0.82%, respectively), but Jerseys have lower plasma volume than Holsteins (Quigley et al., 1998a). Furthermore, Jersey calves had greater total plasma protein (Appendix N) than Holstein calves on d 1 ($P <$

0.0001), d 8 ($P < 0.0001$), and d 22 ($P < 0.0421$), which could also occur due to lower plasma volume. Alternatively, greater total plasma protein, as a result of greater absorption of protein from initial feedings of colostrum or replacement, could lead to increased PUN concentrations as the excess protein is metabolized and cleared from the body.

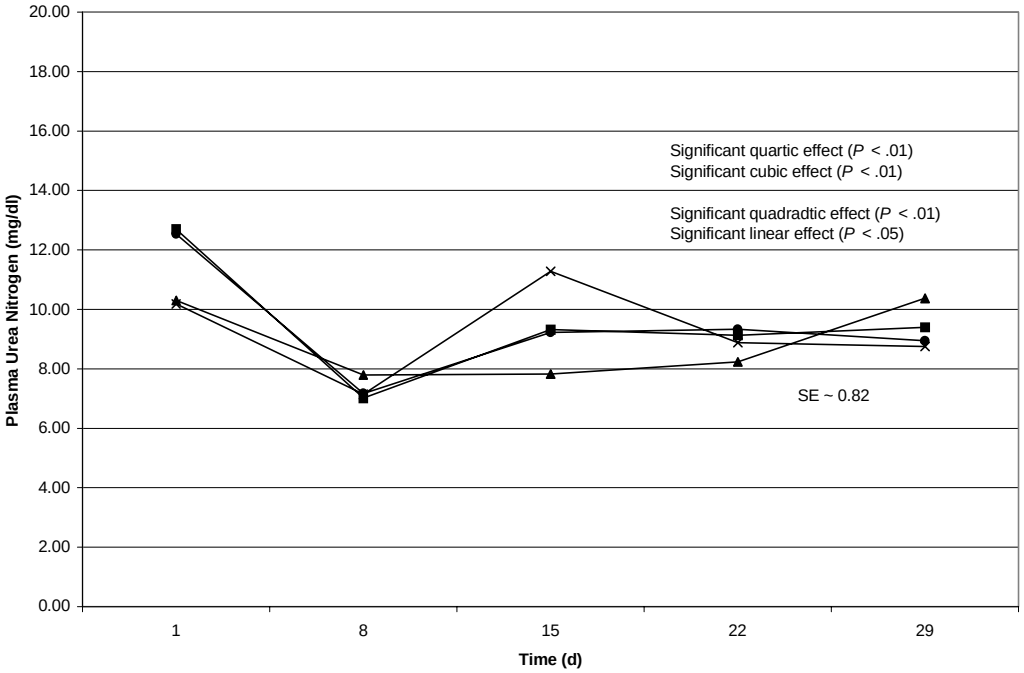

Figure 13. Plasma urea nitrogen concentrations (mg/ml) on d 1 to d 29 in calves fed colostrum at birth and milk replacer without animal plasma for 1 mo (•), colostrum at birth and milk replacer with animal plasma for 1 mo (■), colostrum replacement at birth and milk replacer without animal plasma for 1 mo (▲), or colostrum replacement at birth and milk replacer with animal plasma for 1 mo (×). Significant source by time interaction; differences detected on d 1.

Plasma urea nitrogen decreased from d 0 to d 1 in all groups, and further decreased from d 1 to d 8. Concentrations of PUN increased on d 15 and remained relatively stable for the rest of the experiment. In testing the shape of the PUN curve over time, significant contrasts were: quartic ($P < 0.0001$), cubic ($P < 0.0001$), quadratic ($P < 0.0001$), and linear ($P < 0.0497$). The biological reasons for changes in PUN indicate that the curve is most likely to be cubic. High concentrations of PUN at birth could result from maternal influences, as PUN in the young calf is much lower than that of the mature cow (Kitchenham et al., 1975). Continued clearance of maternal urea nitrogen as well as clearance of the glut of protein provided in the initial feedings account for further declines in PUN. Concentrations of PUN

then increase as calves begin to consume greater amount of grain and development of ruminal fermentation occurs (Quigley and Bernard, 1992). As the digestive system matures, the low PUN concentrations increase to adult concentrations (9.5 to 19.5 mg/dl; Kitchenham et al., 1975).

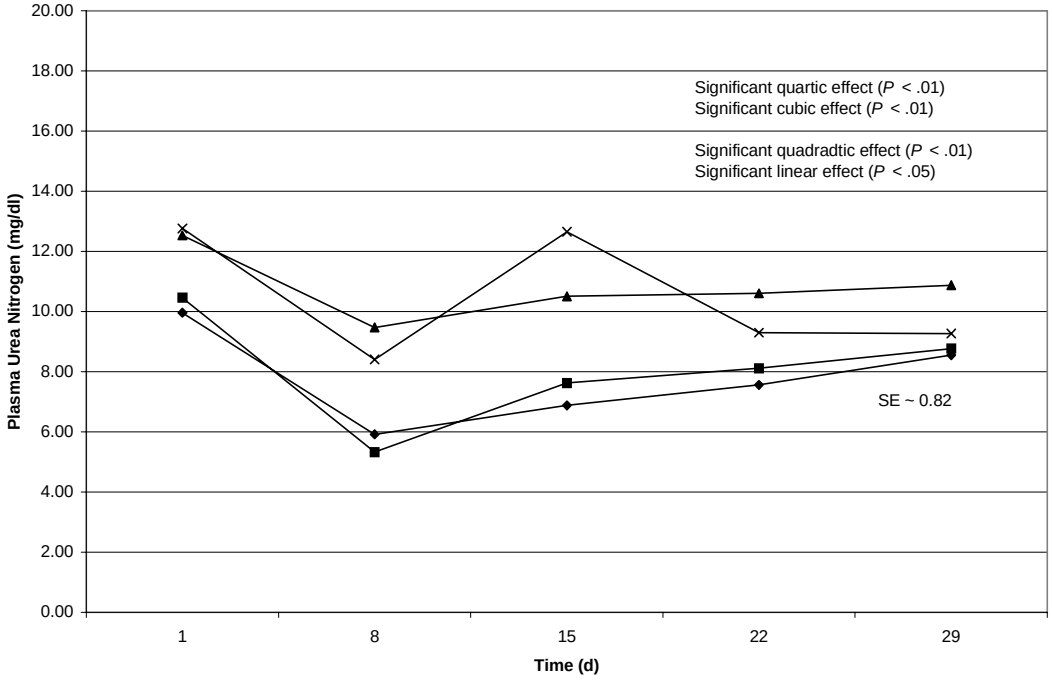

Figure 14. Plasma urea nitrogen concentrations (mg/dl) of Holstein females (♦), Holstein males (■), Jersey females (▲), and Jersey males (×) from d 1 to d 29. Breed differences detected on d 1, 8, 15, and 22.

Health

Fecal score was independent of treatment; the distribution of scores was similar for all four treatments. Fecal score was not independent of breed-gender combination however (Chi square test, $P = 0.0002$). The distribution of scores was different for breed-gender groups. Jersey males experienced more diarrhea (fecal score of 2) than other groups. Jersey males had fewer d scored 0 (firm feces) and more d scored 2 than the other groups. Holstein females had fewer d scored 0 than Holstein males and Jersey females, but more d scored 1 than any other group and the lowest incidence of d scored 2. Four calves died during the course of the experiment. Following necropsy, three of the deaths were attributed to diarrhea caused by *cryptosporidium* and *E. coli* infection. A fourth calf was euthanized due to an obstruction of the large intestine. Necropsy confirmed the defect was present at birth. Four

other calves experienced swelling and hardening of the salivary glands. Differences in calf deaths and incidence of salivary gland infection were not significantly different between treatments or breed-gender groups. Due to finding no differences between treatments in 24 h IgG concentrations, correlations between IgG, morbidity, and mortality were not calculated.

CHAPTER 5. CONCLUSIONS

Calves fed an equal amount of IgG from colostrum or a colostrum replacement attained equivalent plasma IgG concentrations at 24 h of age. Apparent clearance rate of passively acquired IgG was faster in calves fed colostrum than in calves fed replacement. No differences in growth performance or health status of calves were attributable to source of IgG in the first 24 h. It is therefore concluded that colostrum can be successfully replaced with products formulated to provide IgG and other essential properties of colostrum. However, while replacement products may provide adequate amounts of IgG, the viability and specificity of antibodies, as well as the ratio of IgG_1 to IgG_2, are potential concerns for the efficacy of these products. If IgG molecules are not viable and specific for pathogens in the local environment of calves, then the amount provided does not matter. The effects of the intense amount of processing required to isolate and concentrate IgG from animal proteins on the activity of IgG also needs to be determined. In addition, the risks associated with feeding animal proteins must be assessed. This replacement product is derived from human-edible grade plasma, but the risk of disease transmission can never be zero if animal proteins are fed to other animals. Furthermore, the ratio of IgG_1 to IgG_2 in colostrum is likely skewed in favor of IgG_1 for a physiological reason. The impact of changing this ratio needs to be evaluated more closely. If calves receiving colostrum replacement are stimulated to produce IgG_1 then the product could be very valuable in providing enough initial IgG_1 to effectively protect the calf until endogenous production begins. At the same time, IgG_2 would be present in excess of normal concentrations and may offer additional benefits.

No differences from controls were observed in the growth or health of calves fed milk replacer containing animal plasma, which indicates that it is an acceptable replacement for up to 20% of milk protein in milk replacers. Further investigation is required to determine if animal plasma provides health benefits to calves in addition to meeting nutritional requirements.

CHAPTER 6. IMPLICATIONS

Successful substitution of colostrum with colostrum replacement products provides a method of passive transfer of immunity that confers adequate immune protection on calves and can be used to break the cycle of diseases transmitted from cow to calf through colostrum. Furthermore, use of animal plasma in milk replacers can reduce the cost of feeding young calves while still providing functionally adequate protein for utilization in support of maintenance and growth. Further investigation into the viability, safety, specificity, and composition of IgG of replacement products and animal proteins is necessary to fully understand the potential benefits and/or risks of their use.

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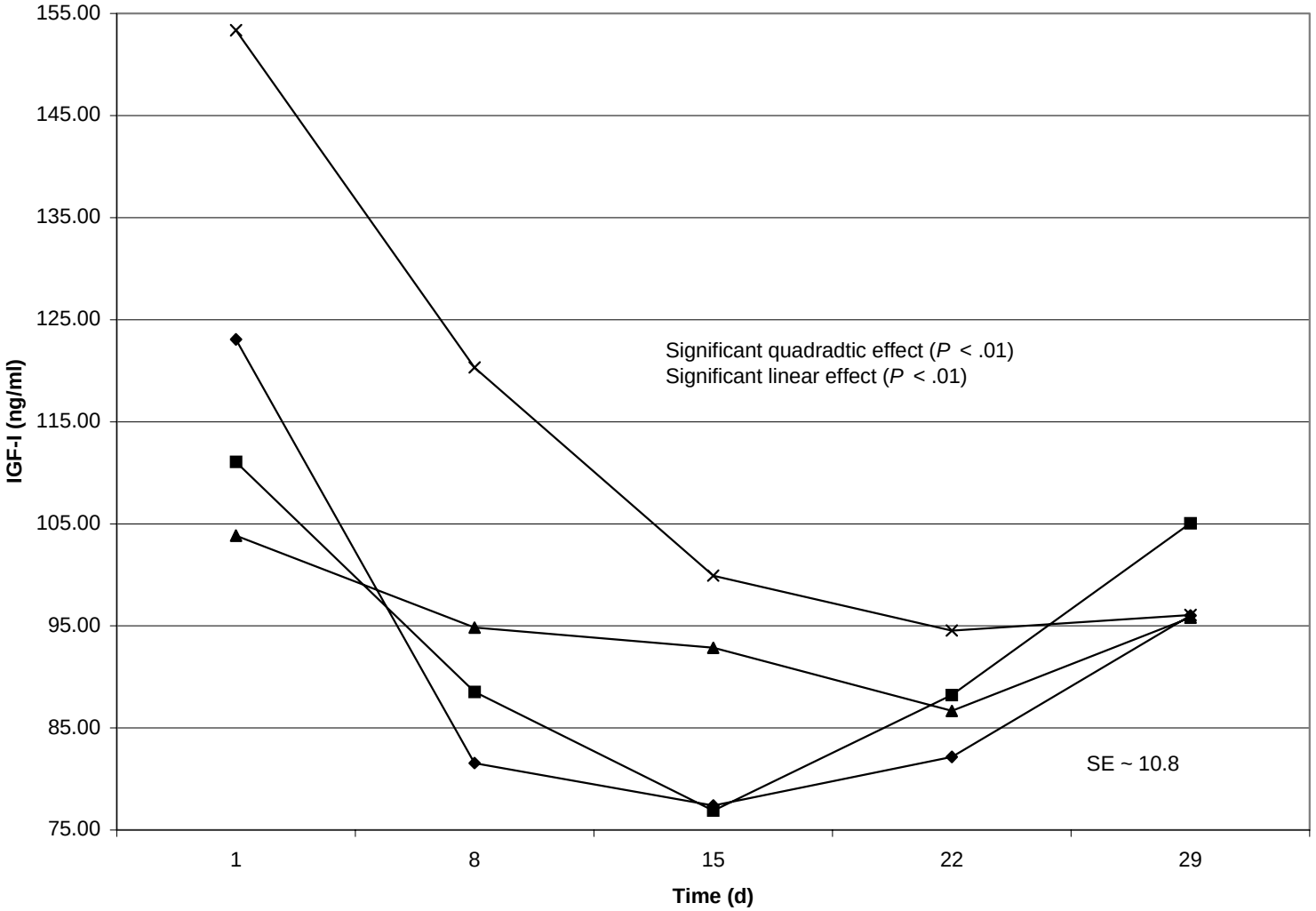
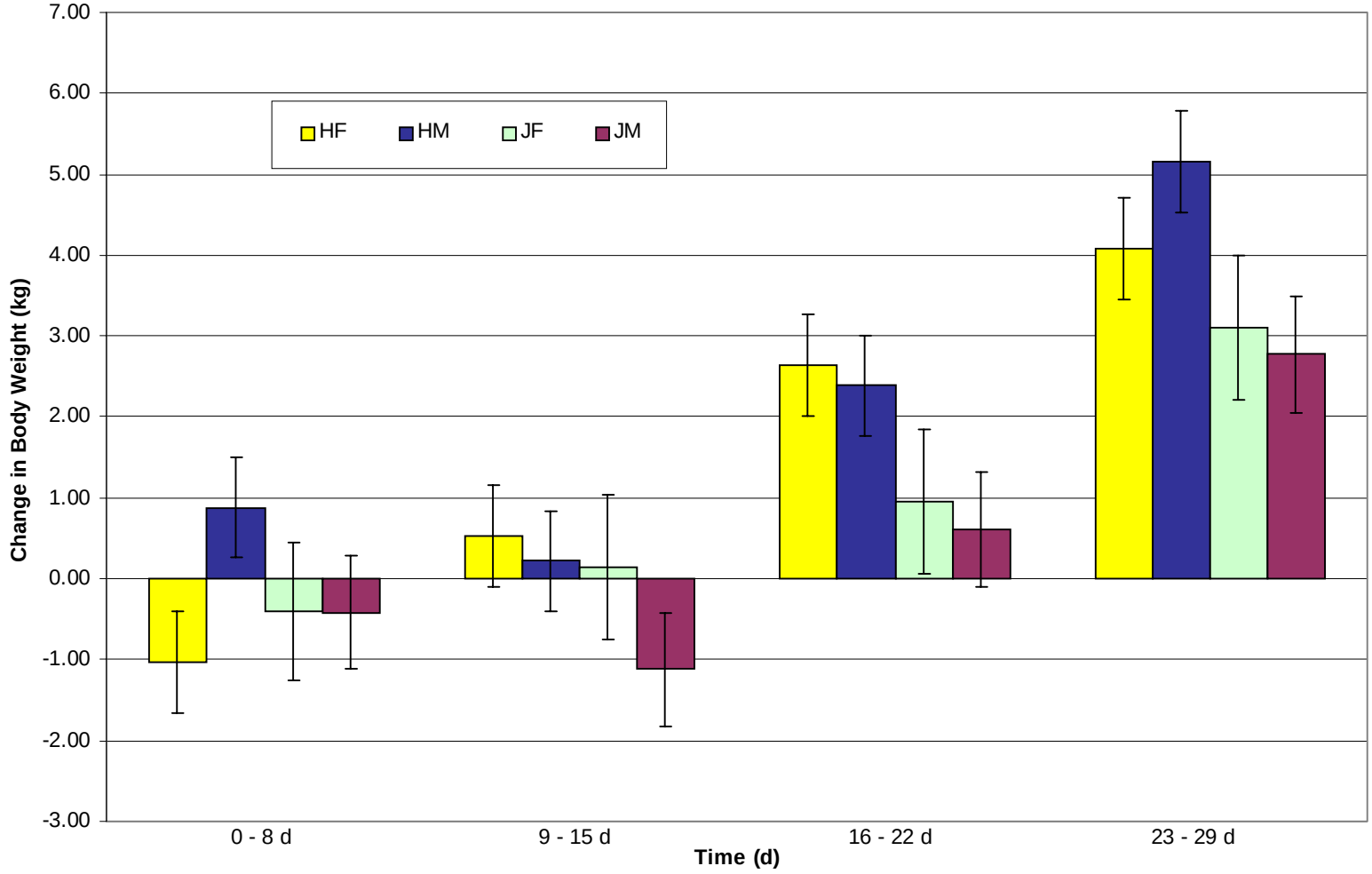
APPENDIX

APPENDIX A



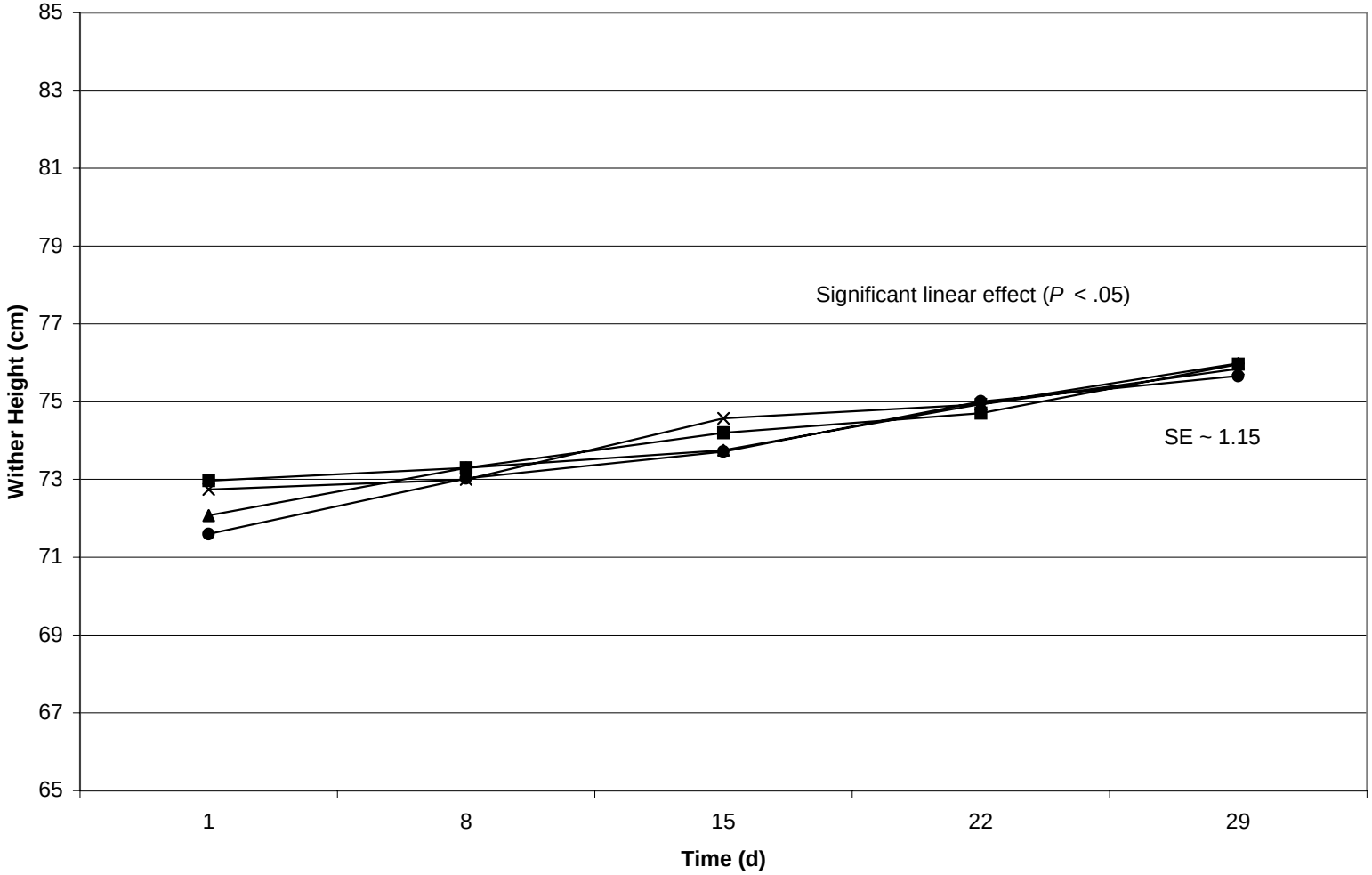
Appendix A. Plasma IGF-I concentrations (ng/ml) of Holstein females (♦), Holstein males (■), Jersey females (▲), and Jersey males (×) from d 1 to d 29.

APPENDIX B



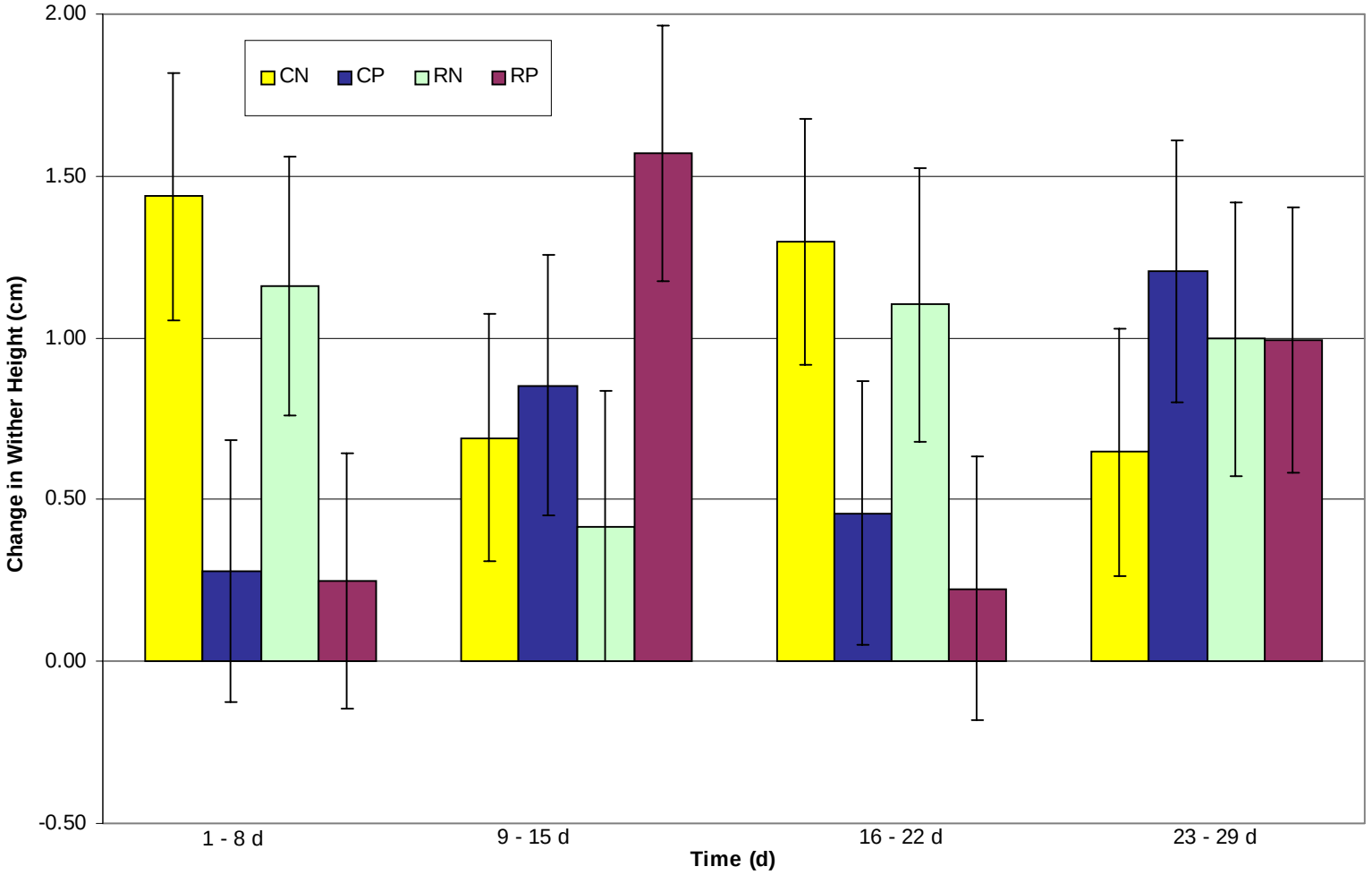
Appendix B. Changes in body weight (kg) by wk for Holstein females (HF), Holstein males (HM), Jersey females (JF), and Jersey males(JM).

APPENDIX C



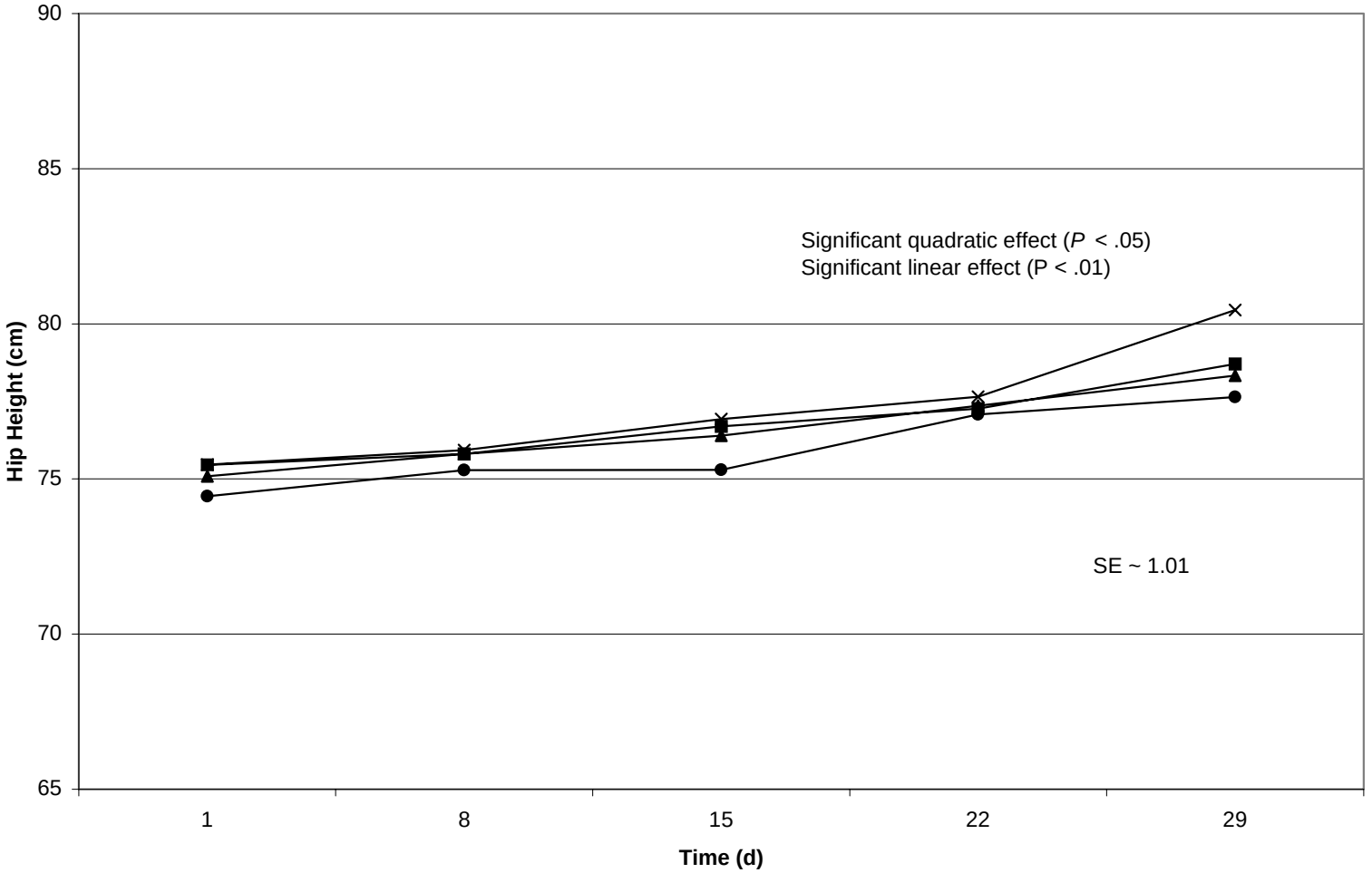
Appendix C. Wither heights (cm) on d 1 to d 29 in calves fed colostrum at birth and milk replacer without animal plasma for 1 mo (•), colostrum at birth and milk replacer with animal plasma for 1 mo (■), colostrum replacement at birth and milk replacer without animal plasma for 1 mo (▲), or colostrum replacement at birth and milk replacer with animal plasma for 1 mo (×). Significant replacer by time effect; no differences detected on individual d.

APPENDIX D



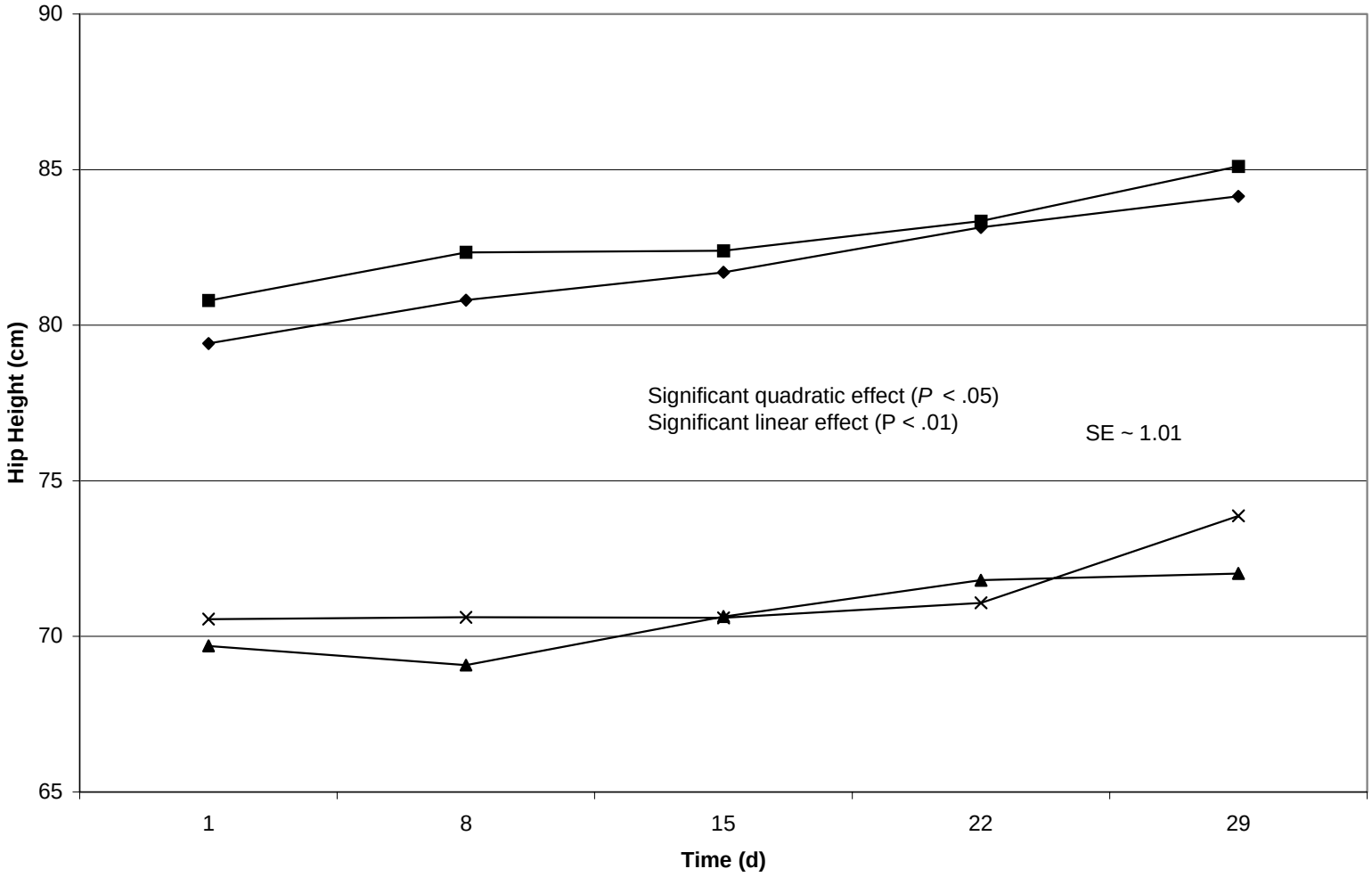
Appendix D. Change in wither height (cm) each wk in calves fed colostrum at birth and milk replacer without animal plasma for 1 mo (CN), colostrum at birth and milk replacer with animal plasma for 1 mo (CP), colostrum replacement at birth and milk replacer without animal plasma for 1 mo (RN), or colostrum replacement at birth and milk replacer with animal plasma for 1 mo (RP).

APPENDIX E



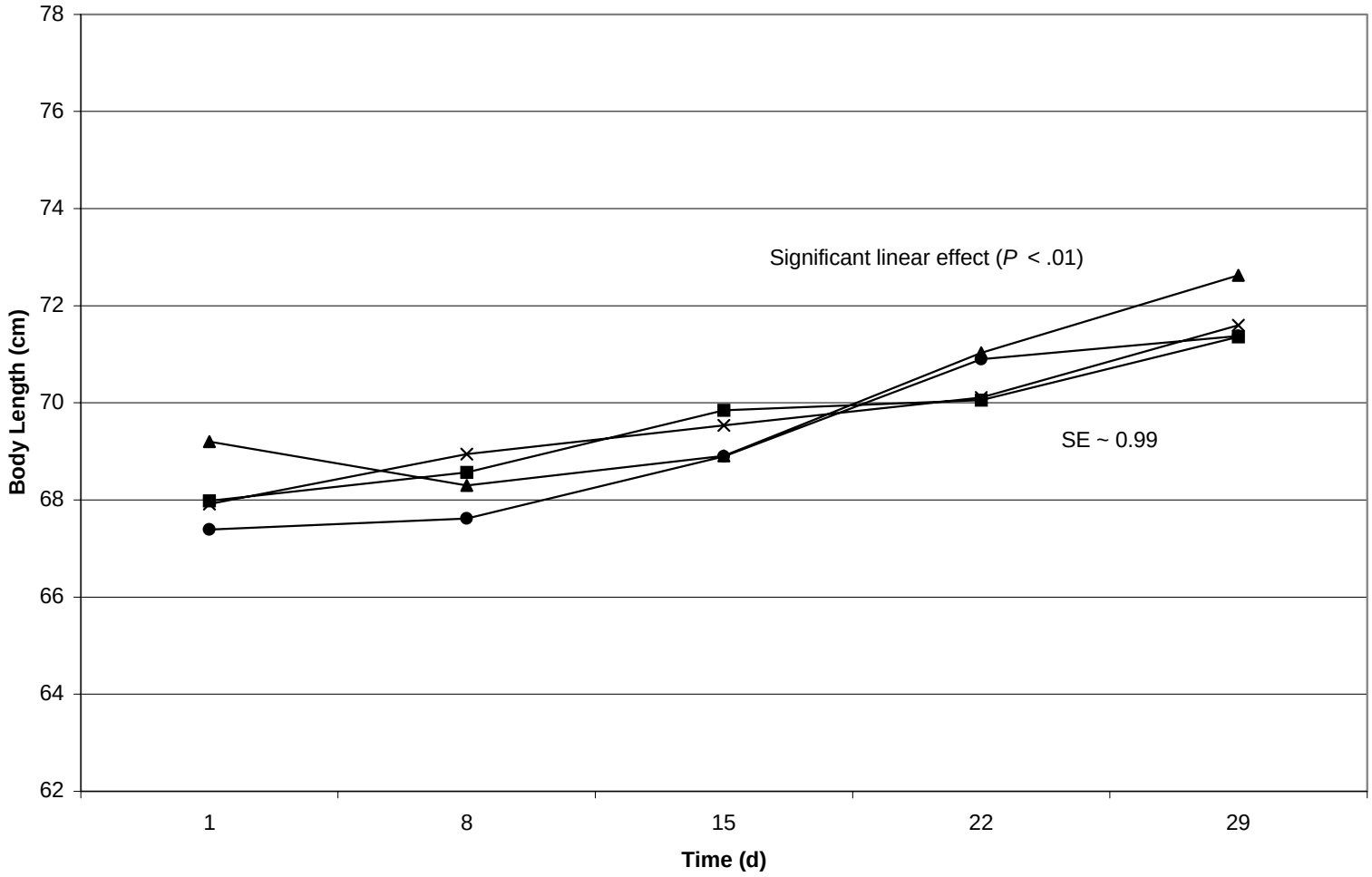
Appendix E. Hip height (cm) on d 1 to d 29 in calves fed colostrum at birth and milk replacer without animal plasma for 1 mo (•), colostrum at birth and milk replacer with animal plasma for 1 mo (■), colostrum replacement at birth and milk replacer without animal plasma for 1 mo (▲), or colostrum replacement at birth and milk replacer with animal plasma for 1 mo (×).

APPENDIX F



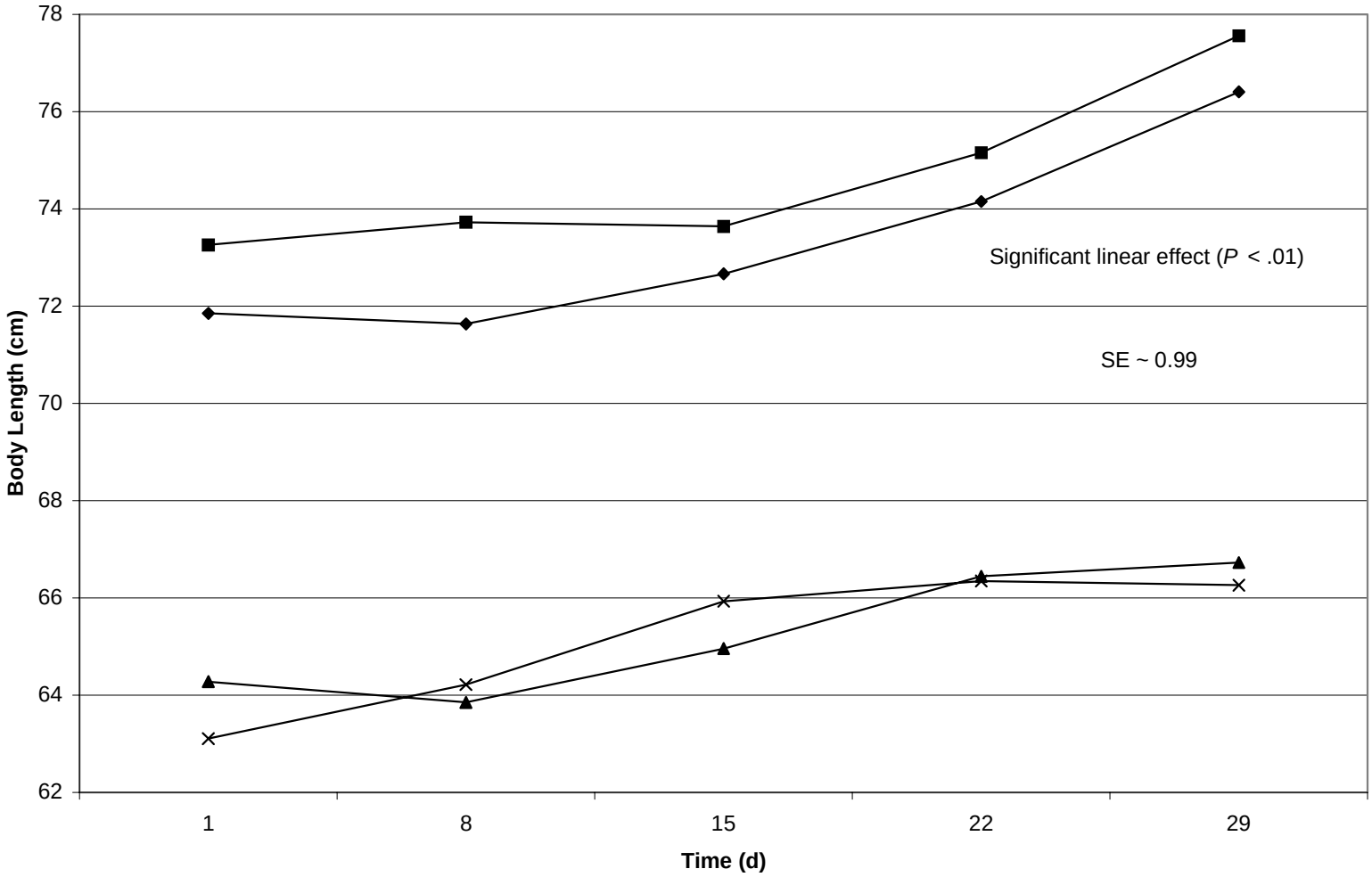
Appendix F. Hip height (cm) of Holstein females (♦), Holstein males (■), Jersey females (▲), and Jersey males (×) from d 1 to d 29. Significant breed by time and gender by time interactions; breed differences detected on all d.

APPENDIX G



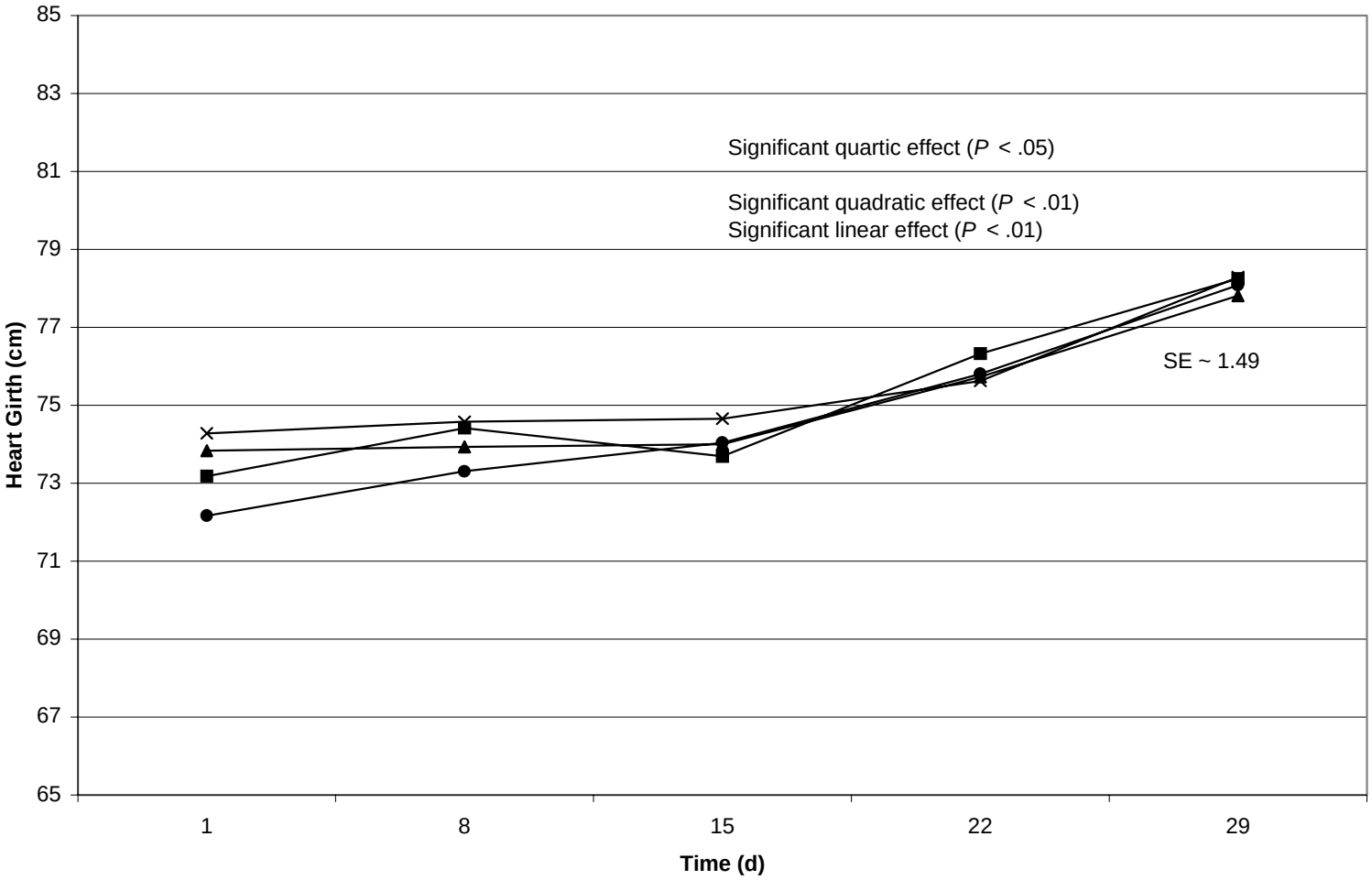
Appendix G. Body length (cm) on d 1 to d 29 in calves fed colostrum at birth and milk replacer without animal plasma for 1 mo (•), colostrum at birth and milk replacer with animal plasma for 1 mo (■), colostrum replacement at birth and milk replacer without animal plasma for 1 mo (▲), or colostrum replacement at birth and milk replacer with animal plasma for 1 mo (×).

APPENDIX H



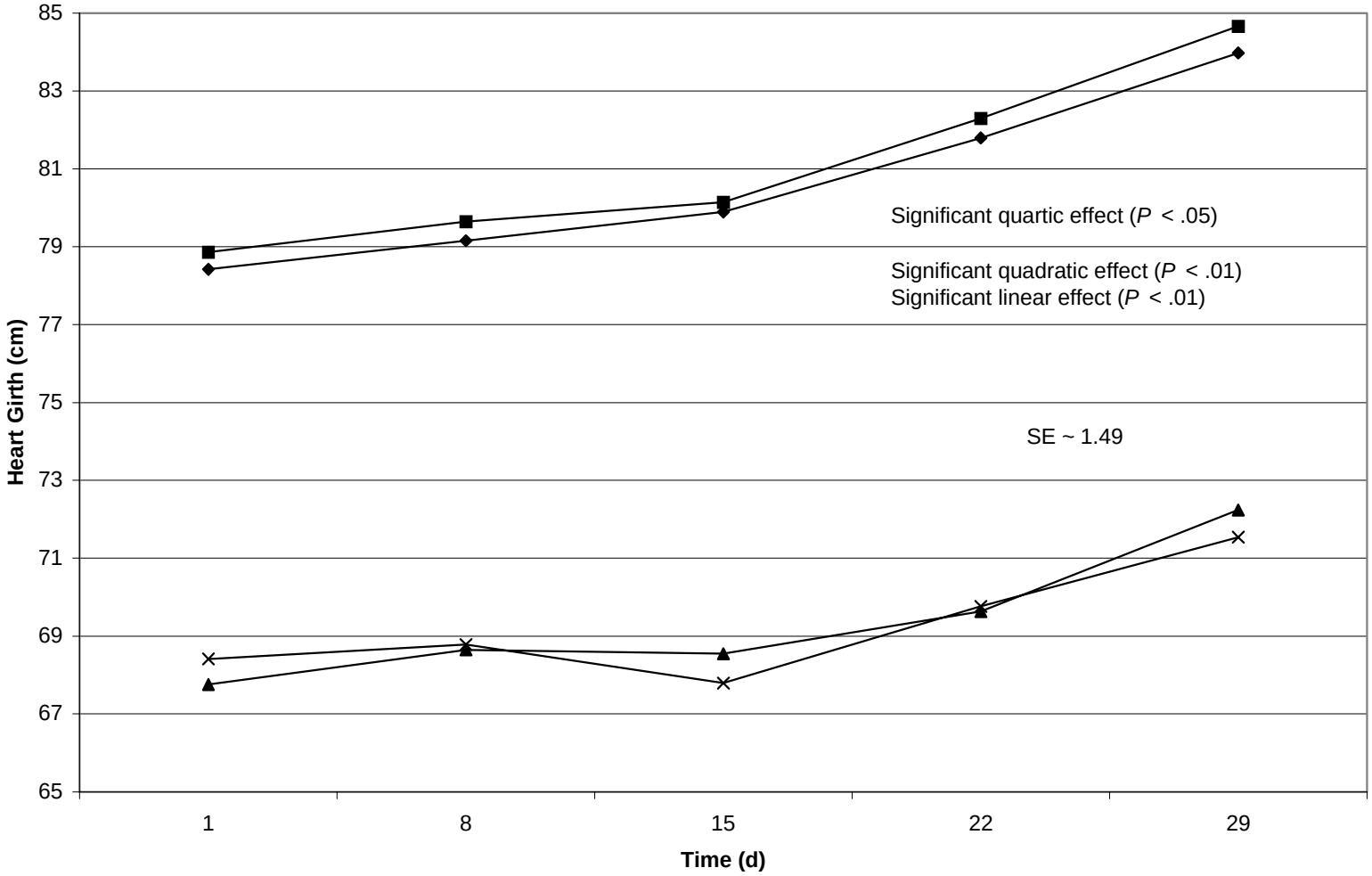
Appendix H. Body length (cm) of Holstein females (♦), Holstein males (■), Jersey females (▲), and Jersey males (×) from d 1 to d 29. Significant breed by time interaction; differences detected on all d.

APPENDIX I



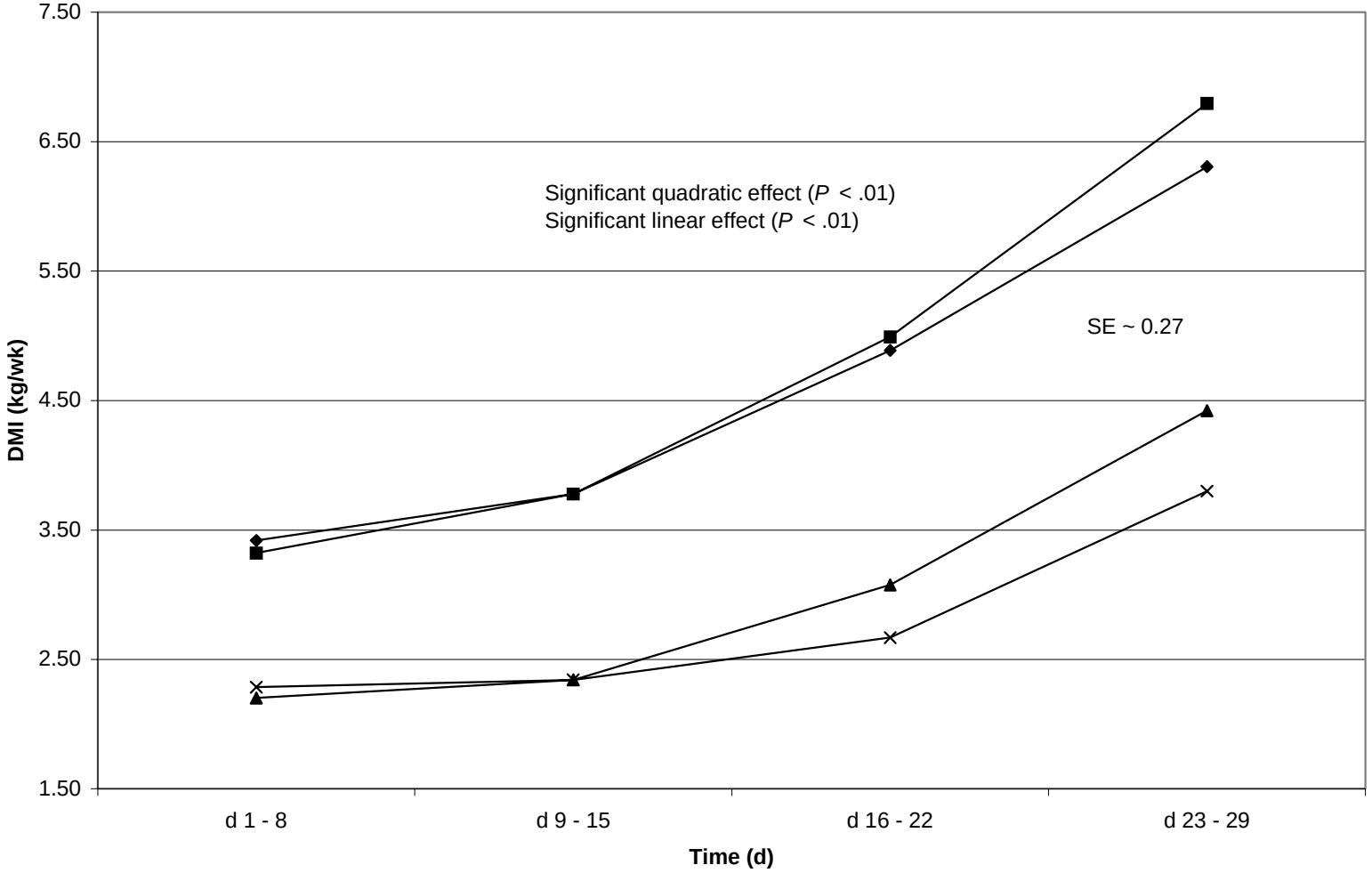
Appendix I. Heart girth (cm) on d 1 to d 29 in calves fed colostrum at birth and milk replacer without animal plasma for 1 mo (•), colostrum at birth and milk replacer with animal plasma for 1 mo (■), colostrum replacement at birth and milk replacer without animal plasma for 1 mo (▲), or colostrum replacement at birth and milk replacer with animal plasma for 1 mo (×).

APPENDIX J



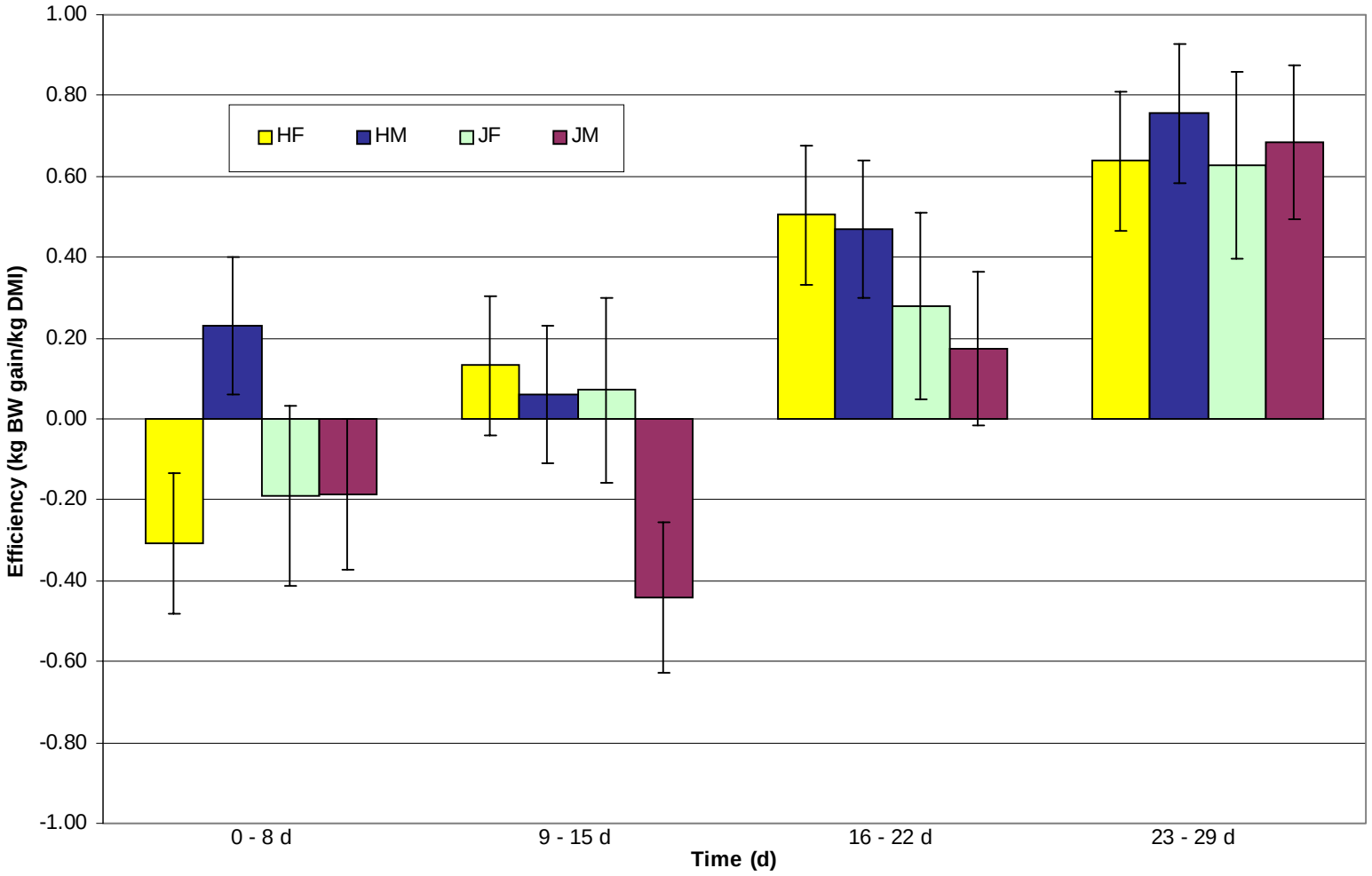
Appendix J. Heart girth (cm) of Holstein females (♦), Holstein males (■), Jersey females (▲), and Jersey males (×) from d 1 to d 29.

APPENDIX K



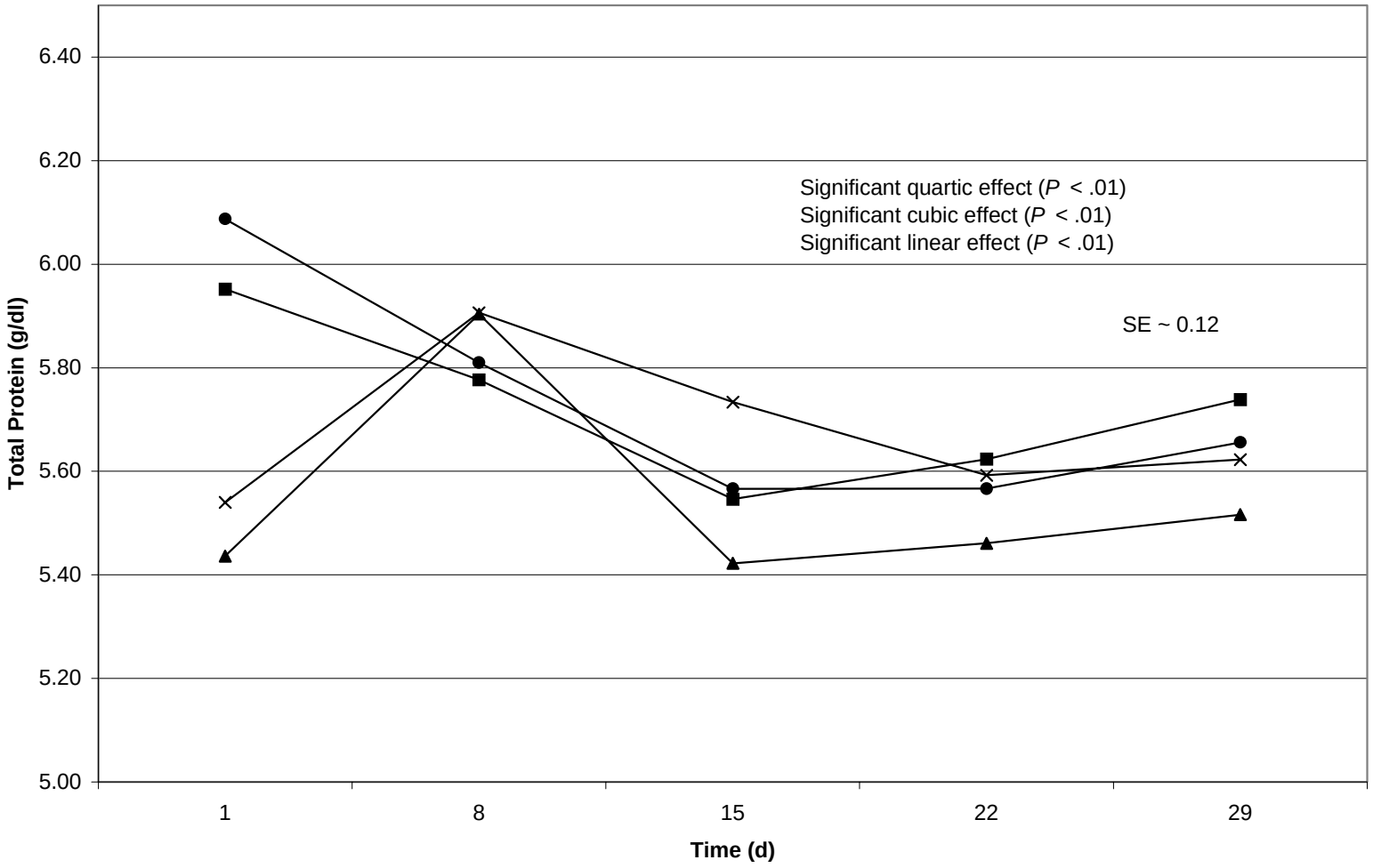
Appendix K. Dry matter intake (kg/wk) of Holstein females (♦), Holstein males (■), Jersey females (▲), and Jersey males (×) from d 1 to d 29. Significant breed by time interaction; differences detected in all 7-d time periods.

APPENDIX L



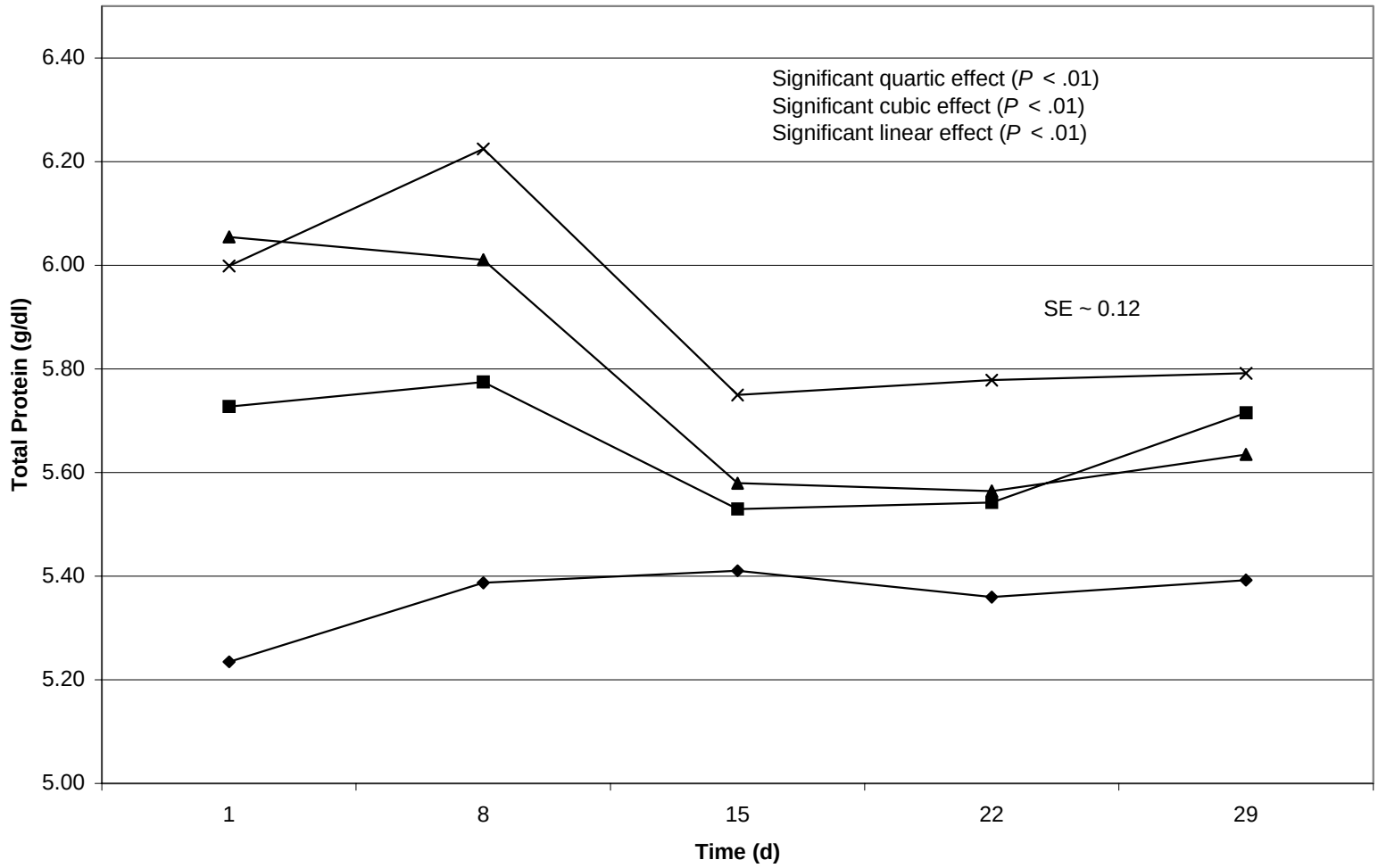
Appendix L. Feed efficiency (kg weight gain/kg dry matter intake) by wk for Holstein females (HF), Holstein males (HM), Jersey females (JF), and Jersey males (JM).

APPENDIX M



Appendix M. Total plasma protein (g/dl) on d 1 to d 29 in calves fed colostrum at birth and milk replacer without animal plasma for 1 mo (•), colostrum at birth and milk replacer with animal plasma for 1 mo (■), colostrum replacement at birth and milk replacer without animal plasma for 1 mo (▲), or colostrum replacement at birth and milk replacer with animal plasma for 1 mo (×). Significant source by time interaction; differences detected on d 1.

APPENDIX N



Appendix N. Total plasma protein (g/dl) of Holstein females (♦), Holstein males (■), Jersey females (▲), and Jersey males (×) from d 1 to d 29. Significant breed by time interaction; differences detected on d 1, 8, and 22.

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