EFFECT OF PASTEURIZATION OF COLOSTRUM ON ABSORPTION OF IMMUNOGLOBULINS BY DAIRY CALVES

By

RUBEN DARIO CONTRERAS N.

Medico Veterinario

Universidad Central de Venezuela

Maracay, Edo. Aragua, Venezuela

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BY DAIRY CALVES

Thesis Approved:

L. J. Bush
Thesis Adviser

F. E. Staley

Stanley E. Bellland

Norman N. Durham
Dean of the Graduate College

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CHAPTER I

INTRODUCTION

The high mortality of dairy calves during the first week of life and, more especially, during the first two days of life has intrigued investigators for decades. However, reports from Michigan, New York and California indicate little or no advancements in reducing calfhood mortality during the last 25 years (Otterby and Linn, 1981). There are two major health disorders affecting young calves. These are, the enteric disorders which in the form of neonatal diarrhea affect calves within the first few days of life, and respiratory disorders which are more common in older calves and closely related to environmental conditions.

The importance of colostrum consumption by calves soon after birth in relation to disease resistance is well established. The newborn calf, as well as the horse and pig, does not receive resistance through placental transfer like some other species (man, guinea pig and rabbit). Shortly after ingestion, colostral antibodies, or immunoglobulins appear unchanged in the serum of the newborn calf conferring the required passive immunity.

In newborn calves the absorption of colostral immunoglobulins occurs in the intestinal absorptive cells for a limited period after birth. The intake of these macromolecules into the cells appears to be nonselective. Ultrastructural studies suggest that the intestinal epi-

thelial cell may take in bacteria in a similar fashion (Staley et al., 1972), which may account to some extent for the high incidence of infections in newborn calves. Therefore, feeding colostrum heavily contaminated with bacteria to newborn calves at the critical period when defense mechanisms are hypoplastic may be risky.

The objective of this work was to determine the effect of pasteurization of colostrum on immunoglobulin absorption in newborn dairy calves.

CHAPTER II

REVIEW OF LITERATURE

Introduction

It is well recognized that the health of newborn calves is very dependent on adequate management, and the intake of colostrum shortly after birth plays a vital role in their survival. Certainly bovine colostrum provides nutrients and confers specific protection against many pathogenic bacteria and viruses by virtue of its content of immunoglobulins. It also contains a variety of nonspecific factors that strongly and selectively influence the growth of different microorganisms and may play an important part in protecting the newborn against infection. Therefore, in this chapter an attempt has been made to review the extensive literature about bovine colostrum. A special emphasis has been placed on its constituents that may confer protection against infections, as well as on the limited literature about pasteurization of colostrum and the possible effects of heating on the antibody molecule.

Since there has been an increase in interest among research scientists about the possible effect of heat on the different constituents in human colostrum and its significance on immune properties to the infant, the author has included information concerning some constituents in human colostrum. Although these constituents in bovine colostrum have not been studied extensively, they deserve special attention. Finally, the literature concerning the characterization of the bovine

immunoglobulins and absorption by the calf have been reviewed.

Bovine Colostrum

Definition

Bovine colostrum consists of the mixture of lacteal secretions and constituents of blood serum, notably immunoglobulins and other serum proteins, that accumulate in the mammary gland during the prepartum dry period and can be harvested immediately preceding or following parturition (Foley and Otterby, 1978). Milk is usually not considered normal until about the fifth day after calving. Therefore, colostrum differs markedly in composition from normal milk. Colostrum has a higher concentration of practically all milk components except lactose, potassium, panthothenic acid, and water (Webb et al., 1974).

Availability of Colostrum

The first six postpartum milkings, collected during the period of transition from colostrum to milk are unmarketable. Most healthy dairy cows produce colostrum in excess of the calf's requirement during this time (Foley and Otterby, 1978). Huber (1974) reported average colostrum yields of 32.7 kg for heifers and 41.7 kg for cows. Colostrum production by Holstein cows for the first six postpartum milking are presented in the following table, with data obtained from Muller et al. (1975) and Rindsig (1976), as summarized by Foley and Otterby (1978). With an average consumption of about 10% of birthweight or 3.5 kg of colostrum (Selman et al., 1971) there is a surplus of an unmarkable product. Even more, common management practices require removing calves from their dams shortly after birth and for feeding limited amounts of colostrum,

TABLE I

COLOSTRUM PRODUCTION BY HOLSTEIN COWS FOR THE FIRST SIX POSTPARTUM MILKINGS

Parity	No. of Cows	Average Yield (kg)[a]
1	36	33.2
2	19	45.4
3	17	58.4
4 or more	34	46.0
All parities	106	43.5

[a]Weighted means summarizing two sets of data.

usually 14 to 35% of the average colostrum production per cow as reported by Kaeser et al. (1948) and by Payne (1953). With an average production of colostrum of 43.5 kg per cow (Table I) and an average consumption of 11 kg per calf during the first 3 days postpartum, 32.5 kg of colostrum would be available per cow for feeding to calves over 3 days of age (Foley and Otterby, 1978). Other reports estimate that sufficient colostrum should be available to feed heifer calves to 5 weeks of age (Yu et al., 1976).

Composition of Colostrum

Colostrum markedly differs from milk secreted in established lactation. As early as 1875, Eugling (cited by Macy et al., 1953) had shown that colostrum was extremely rich in nitrogenous constituents. Famulener in 1912 conveyed the results of Ehrlich in 1892 and his own and postulated that the colostrum contained, in addition to normal milk proteins, immune serum proteins derived from the maternal blood. However, most of the early investigators partitioned the nitrogenous constituents of colostrum into two main groups--casein and albumin. The latter fraction was then partitioned into lacta-albumin and lacto-globulin fractions (Crowther and Raistrick, 1916) and the lacto-globulin was further partitioned by precipitation from sodium sulfate solutions (Howe, 1921). Smith (1946) showed electrophoretically that immune lactoglobulin is the predominant protein in bovine colostrum.

Other early studies (Howe 1921b and 1924; Orcutt and Howe, 1922; Smith and Little, 1922) clearly demonstrated that when colostrum was fed soon after birth these globulins appeared in the blood of the calf and when late-lactation milk was fed to calves the amount of globulin in

the blood of calves remained practically nil. Rook (1961) stated that the concentrations of proteins synthesized in the mammary gland, i.e., casein, β-lactoglobulin and α-lactalbumin, are about twice the mid-lactation values, but the major increase is in globulin and, to a lessor extent, in the proteases. There is a gradual change from colostrum to normal milk (Figure 1), as shown with data obtained from Engel and Schlag, cited by Webb et al. (1974). Numerous other workers have presented data which are in substantial agreement with those of Engel and Schlag. The composition of colostrum approaches that of whole milk starting 24 hours after calving (Figure 1). During this time, total protein, fat, total solids, non-fat solids, and ash decrease with time postpartum while lactose increases (Parrish et al., 1948, 1950). Most of the decline in total protein is traced to the reduction of the globulin content and Parrish et al. (1950) found ranges of values of first-milking colostrum from 4.0 to 24.6 percent. Bush et al. (1971) found a less precipitous decline in the total protein and immunoglobulin in colostrum at successive milkings after calving. Fat content of colostrum varies considerably among breeds and among cows of the same breed (Parrish et al., 1950). Johnson et al. (1961) reported fat content is high after parturition and Garret and Overman (1940) showed an upsurge of fat between 8 and 15% a few hours after calving, whereas other authors have reported a decrease of fat during the same period (Figure 1).

Colostrum has a higher concentration of Vitamin A, carotene and Vitamin D than normal milk. Cow's colostrum is up to ten times and two to three times richer in carotene, Vitamin A and Vitamin D, respectively, than milk but after the first few days the levels become steady and

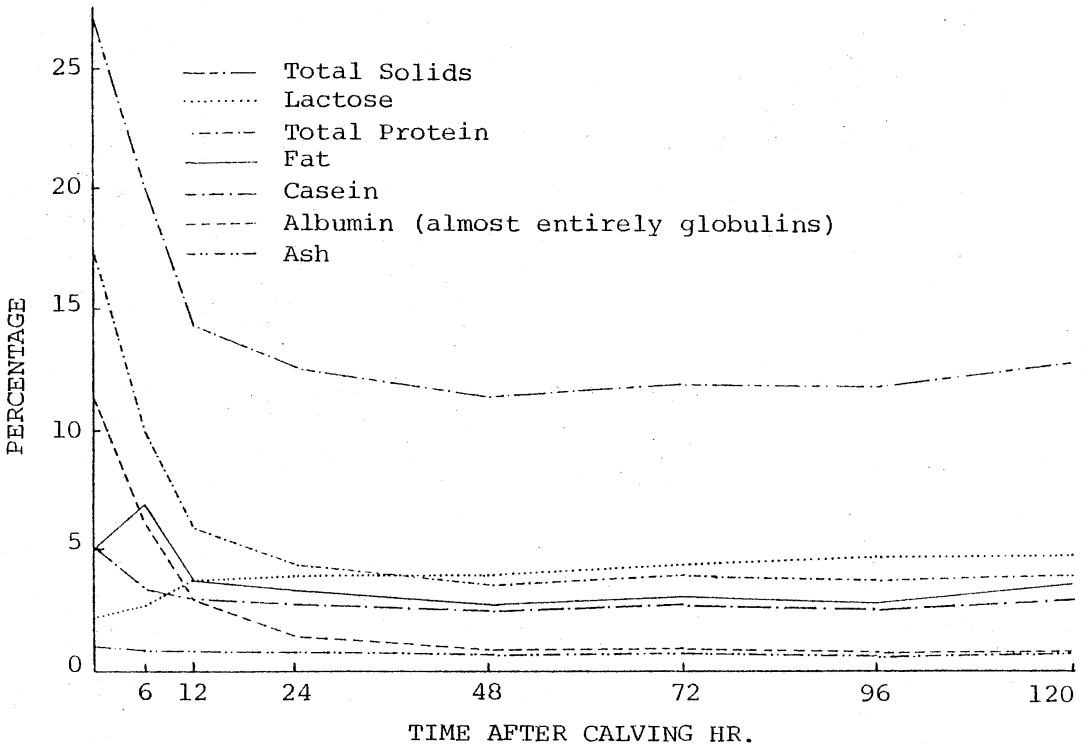

Figure 1. Transition From Colostrum to Normal Milk

there are no further lactational trends. Parrish et al. (1949) reported that Vitamin A and E contents decrease logarithmically during the first 4 days postpartum. Dann (1953) showed how little Vitamin A the newborn calf possesses and that the high vitamin content of colostrum is consequently of considerable value.

In ruminants all the vitamins of the B complex are synthesized in the rumen; thus, they are largely independent of an exogenous supply. For this reason the level of these vitamins in their milk varies less than in that of simple-stomached animals, for example in human milk (Ling et al., 1961). Most representatives of the vitamin B complex, on the other hand, are essential in the synthesis of the important metabolic enzymes and this fact also is of considerable significance. Pearson and Darnell (1940) reported that colostrum had higher thiamine, lower panthothenic acid and about the same niacin content as milk. They also reported that riboflavin in the first milking colostrum was about 3 to 3.5 times as much as normal milk. Sutton et al. (1947) found higher concentration of biotin in colostrum than in milk.

Bowland et al. (1949) reported the vitamin C content of colostrum was up to 10 times higher than in true milk. Milk is known for its low level of vitamin C.

The composition of milk ash is mainly K, Ca, Na, Mg, Fe, P, Cl and S. Several authors have reported that potassium is the only salt that is lower in colostrum than in normal milk and that P, Ca, Mg and Na salts are higher during the early hours of lactation but rapidly decline towards a fairly constant level as the milk becomes normal. The magnesium has a mild laxative effect and assists in the expelling the meconium (Heidrich and Renk, 1967).

The presence in colostrum of large amounts of enzymes deserve special mention. For the newborn the availability of these enzymes in large quantities is of the greatest significance in supporting the 'as yet incompletely developed' somatic and digestive metabolism (Heidrich and Renk, 1967). Jenness (1974) compiled a list of 44 enzymes detected in bovine, human and other milks. It is not the scope of this review to deal in detail with so many enzymes and the enzymatic activity detected in milk. However, it is not yet clear whether the enzymes in colostrum serve some purpose or whether they should be considered as extraneous material introduced into milk during the secretory process. Colostral milk and milk from cows in the last stage of lactation contain a large percentage of catalase. This enzyme is particularly high in colostrum and in milk from mastitic udders. Furthermore, it tends to parallel the leukocyte count. Catalase increases with the multiplication of bacteria in milk (Jenness and Patton 1951). The catalase content of milk has been proposed as a means of determining the quality of milk and of detecting milk from diseased udders (Webb et al., 1974). Cow milk also is rich in lactoperoxidase which, in the presence of thiocynate and peroxide comprise an antibacterial system which inhibits a variety of bacteria and viruses (Oram and Reiter, 1966). The lactoperoxidase system (LP system) becomes active below the natural pH of bovine milk (~6.6) which is important because the acidity of the stomach (pH < 2.0), which is the most effective barrier against enteric infection, is destroyed by the buffering effect of the milk, i.e., the pH increases up to pH 5.0 in the calf (Reiter, 1978).

Due to the antibacterial properties lysozyme deserves special attention. This enzyme, a muramidase, which cleaves peptidoglycans of

bacterial cell walls is some 300 times more abundant in human milk than in cow milk (Goldman and Smith, 1973). Lysozyme has been shown to be active against a number of Gram-positive and Gram-negative organisms and its role in the lytic action of the complement antibody system also is well known, i.e., IgA binds complement only in the presence of lysozyme making this class of immunoglobulins bactericidal (Adinolfi et al., 1966).

Milk contains an inhibitor against several bacteria such as staphylococci and some members of the E. Coli group. This inhibitor has been identified as the red iron-binding protein, lactoferrin, and its inhibitory activity is due to the ability of robbing the organisms of iron, impairing their growth. During the main period of lactation cow's milk contains only low concentrations of lactoferrin, but the concentration gradually increases during the drying-off period (Reiter and Oram 1967). Although bovine colostrum does contain some lactoferrin (up to 6 mg/ml), it is not inhibitory at its natural pH because of the high concentration of citrate (~3.6 mg/ml) which competes with lactoferrin for iron and makes it available to the bacteria for their growth. It appears that citrate is rapidly absorbed from the intestine of the valf and as bicarbonate is the main intestinal buffer secreted, the conditions in the intestine should be favorable for inhibition by lactoferrin (Reiter 1978). More recent studies suggest that lactoferrin plays a prominent role in protection of the mammary gland of the cow against infection (Gaunt 1980).

Cellular Content of Colostrum

The presence of cells or "cell bodies" in colostrum obtained from

normal animals was first described by Donne' in 1844, cited by Head and Beer (1978), who subsequently called them colostral corpuscles. The importance of cellular elements of milk was recognized with the advent of the commercial dairy industry, however, the significance of the cellular elements in immunity to the calf is largely unknown.

At the beginning and end of lactation the cell count is different from that of the mid-lactation period. The cell count falls during the first three weeks of lactation from about 1,000,000 to 70,000 cells/ml in healthy cows (Cullen 1966). The different cell types appearing at different stages of lactation are shown in the following table adapted by Cullen (1966).

TABLE II

CELL COUNTS IN NORMAL MILK (MILLIONS PER ML)

Stage of Lactation	Epithelial Cells	Polymorphs	Lymphocytes	No. of Samples
Colostrum 0-5 Days	1.19	2.28	0.72	44
MID	0.12	0.10	0.05	84
Last 4 Weeks Before Drying Off	0.85	0.28	0.59	36

The effects of these maternal cells upon the newborn calf have not been investigated, but their characteristics have been examined *in vitro*. Studies by Smith and Goldman in 1968 have shown that the colostral cor-

puscles described by Donne'--about 125 years ago--are, in fact, large macrophages containing many lipid vacuoles. Colostrum is a relatively rich source of T-lymphocytes, and recently Parmerly and Beer (1977) have examined these cells for evidence of cell-mediated immune activity. They have shown that approximately 11% of milk cells are small and large lymphocytes, with the majority of the remaining cells being monocytes or macrophages. In 1973, Goldman and Smith reported that macrophages comprised about 90% of the leukocytes in human colostrum. Diaz-Jouanen and Williams (1974) reported that approximately 50% of human colostrol lymphocytes were T cells and 34% were B lymphocytes. Macrophages in human colostrum have the same functional and morphological features as macrophages from other tissue sources. In specific tests of phagocytic functions it has been shown that a very high percentage of colostral macrophages engulf staphylococci. Macrophages in mammary secretions also have the capacity to produce complement components and lysozyme as well as lactoferrin (Goldman and Smith, 1973; Murillo and Goldman, 1970). Smith and Goldman (1970) showed that lymphocyte-macrophage interactions are an important facet of the immune response to many antigens which occur readily both in fresh colostrum and in colostral cell cultures.

There is also in vitro evidence that colostral cells synthesize immunoglobulins. In fact, Murillo and Goldman (1970) demonstrated by radioimmunoelectrophoresis the synthesis of IgA by the cells of human colostrum. They found no evidence for the formation of IgG or IgM by the colostral cells and this is in contrast to blood lymphocytes which produce those three immunoglobulins.

Recent awareness that viable leukocytes and macrophages are

ingredients of colostrum and milk of all species tested, has stimulated interest in the immunologic significance of these cells. In man, on the basis of in vitro analyses, milk lymphocytes exposed to a variety of stimuli respond in a manner indicating that both the T and B lymphocytes are primarily reactive against mucosal invading organisms (Head and Beer, 1978).

In summary, apart from its nutritional significance, colostrum affords newborn calves many immunologic benefits which traditionally have been thought to be mediated exclusively by soluble milk proteins known as immunoglobulins. The importance of those immunocompetent cells and their role in confering cellular immunity to the suckling deserves special attention.

Characterization of Bovine Immunoglobulins

The immunoglobulins of bovine colostrum and post-colostral calf serum have been the subject of numerous investigations which have been adequately reviewed by Buttler in 1969. Quantitative studies of the major bovine immunoglobulins have been described (Klaus et al., 1969; Penhale and Christie, 1969; Mungle, 1972 and Porter, 1972).

Immunoglobulins are a family of high molecular weight proteins that share common physicochemical characteristics and antigenic determinants (Butler, 1969). They form the humoral immune response and are manufactured by cells of the reticulo-endothelial system. Their prime function is to inactivate or destroy antigens which threaten the integrity of the host, and their chief characteristic is their ability to combine specifically with the antigen which stimulated their production.

In the bovine serum and lacteal secretions three molecular classes

of immunoglobulins have been identified, namely IgG, IgM and IgA. In each case, Ig refers to immunoglobulin and the third letter to some distinctive property of that immunoglobulin. Some of these classes can be further divided into sub-classes, e.g., in the case of IgG there are two, IgG_1 and IgG_2, whereas IgA can exist as serum IgA and secretory IgA.

The basic unit of an immunoglobulin molecule consists of two identical H (heavy or large) polypeptide chain with a molecular weight between 50,000 and 70,000 depending upon the Ig class in question, and two identical L (light or short) polypeptide chain with a MW. of 20,000, linked by three or more disulfide bonds and by noncovalent interactions such as hydrophobic bonds and electric for-es (Nisonaff, 1971). Quantitatively, IgG and IgM are the principal immunoglobulins of plasma whereas IgA is the major one in many external secretions. IgG accounts for 85-90 per cent of the immunoglobulin in serum and colostrum (Klaus, et al., 1969; Penhale and Christie, 1969). IgG_1 is the principal immunoglobulin in the serum and lacteal secretions, and the MW is approximately 163,000. Cow IgG_1 accumulates to very high levels in colostral and precolostral lacteal secretions by a mechanism which selectively transports it from the blood. IgG_2 usually makes up less than half of the total serum IgG immunoglobulins.

IgG is the best known and most fully studied of the immunoglobulins. Papain in a mild reducing environment cleaves the IgG molecule into three portions, two of which are identical and called the Fab fragments while the other is called the fragment crystallizable or Fc fragment. Each Fab fragment consists of the amino terminal half of the H chain plus one of the entire disulfide-bounded light chains. The NH_2-terminal

half of each of these chains is considerably variable; the variability is associated with variability of specificity or its biologic activity--the antigen-binding ability. The Fc fragment contains the COOH-terminal of two H chains which are held together by one or more interchain disulfide bonds. This fragment also contains most of the carbohydrates, is highly constant in their amino acid sequence and is antigen-binding deficient. This region of the molecule is essential for its role in complement fixation.

One interesting characteristic of the Fab fragments is that each contains only one antigen-binding site; and that a single Fab fragment or univalent antibody can not serve as a coupling device to link antigen particles together. In fact, bivalence provides a good example of the relationship between structure and function since two antigen-binding sites permits the formation of a framework, or aggregation, with the antigen and antibody alternating in the structure (Nisonaff, 1971). This aggregation of an antigen enhances its uptake by the macrophages and the subsequent elimination of the antigen from the body. Aggregation also activates the complement system and in this way facilitates lysis of many species of bacteria. On the other hand, univalent antibodies or single Fab fragments can actually inhibit agglutination. If they are exposed to antigen prior to the exposure to the bivalent antibodies, they bind the regions of the antigen which would otherwise combine with the bivalent antibody fragments.

IgM molecules, in contrast, are large and complex with a MW of 900,000 and a sedimentation coefficient of 19S. A molecule of IgM consists of 10 H chains and 10 L chains held together by disulfide bonds which are easily split by reducing agents. Apparently, IgM is a more

effective antibody than IgG, particularly in agglutination, phage neutralization, complement fixation, and hemolysis (Butler, 1969). IgM tends to be present in higher proportions during the very early stages after challenge and may be designed for rapid protection (Nisonoff, 1971). The primary immune response of the cow when assayed for complement-fixing antibody to Brucella is found exclusively in the IgM class (Rose and Roepke, 1964).

IgA is the principal immunoglobulin in the lacteal, lacrimal, and salivary secretions of man while bovine colostrum contains very low amounts. This immunoglobulin has been partially characterized and has a molecular weight of 385,000 and a sedimentation coefficient of 11S. It was first isolated in the bovine by Mach et al. in 1969. The majority of IgA is attached to a secretory piece which is thought to prevent enzyme degradation of the molecule with subsequent loss of activity in the small intestine (Tomasi and Bienotock, 1968). IgA also contains a covalently linked polypeptide called J-chain (Halpern and Koshland, 1970). Williams (1972) postulated that IgA represents a local immunity in external secretions of many species and proposed a mechanism which can explain how secretory immunoglobulins function in the disposal of bacterial antigen.

Immunoglobulin Absorption

The fact that the bovine fetus does not receive maternal immunoglobulins by placental transfer is well established (Branbell 1958). The newborn calf acquires its maternal passive immunity solely via the colostrum. Therefore, immunoglobulins are derived from feedings of colostrum, being secreted by the mammary gland into the colostrum, and

are rapidly absorbed from the gut of the newborn calf. The absorption of colostral immunoglobulins in newborn calves has been extensively reviewed by Bush and Staley (1980).

Histological evidence that absorption of IgG occurs mainly in the jejunum and not in the duodenum or ileum of the calf was presented by El-Nagheh in 1967. Ultrastructural studies of the intestinal epithelial cells of the calf were conducted by Staley et al. (1972). The microvilli in the jejunum of the newborn calf are more developed than those in the ileal cell and a characteristic cellular organelle which absorbs undigested proteins from the digestive tract is present. This organelle is a tubullar system found in the apical end of the cell variously named, apical tubular system, apical canaliculus, or endocytic complex. This tubular system is responsible for engulfing immunoglobulins and colostral proteins from the digestive lumen and is a feature common to all epithelial cells which take up undigested proteins. The system has a limited life span and is present in the absorptive cell only during the early postnatal period (Staley 1971). However, the period of time after birth for the absorption of immunoglobulins is generally considered to be limited to 24-36 hours after birth (Baljour and Comline, 1962; Butler 1969; Bush et al., 1971). On data from a large commercial dairy herd, Stott et al. (1979) estimated time of cessation of intestinal permeability to colostral immunoglobulins to be near 24 hours following a normal distribution with a standard deviation of approximately 4 hours.

It appears that immunoglobulins are transported from the gut lumen into the circulation following three basic steps. The first step involves the engulfing of immunoglobulin by the intestinal epithelial cell. The surface membrane of the intestinal epithelial cell between

the microvilli is extremely active in invaginating and extending into the cytoplasm for some distance as tubules. Immunoglobulins enter these invaginations and are thus carried into the cytoplasm. The second step involves enlargement of the tubular end-piece to form a vacuole. Immunoglobulin, a colostral protein, fills and distends the vacuole, the tubular connections are lost, and the vacuole then is transported toward the basal cell membrane. Once in contact with basal cell membrane the vacuole opens and discharges its contents into the lamina propia where it passes the lymphatic endothelium into the circulation (Staley, 1971).

The intestinal tract of the newborn calf absorbs various types of protein indiscriminately (Pierce and Feinstein, 1965; Bangham et al., 1968). Staley et al. (1972) established that heterologous proteins such as human serum proteins pass the intestinal absorptive cells into the circulation of the newborn calf in an equal manner. They concluded that the absorption of intact proteins is a relative nonselective process in calves and suggested that the intestinal epithelial cells of certain neonates may take in intact proteins and bacteria in a similar fashion. Nevertheless, it would appear that some differential in absorption between individual classes of immunoglobulins exist. Panhale et al. (1973) reported that approximately 90 per cent of the IgG was absorbed whereas only 59 per cent of the IgM and 45 percent of the IgA were absorbed. They showed there was individual variation in the duration of absorption being 27, 22, and 16 hours post partum for IgG, IgA and IgM respectively. However, Stott et al. (1979a) found no significant differences in the duration of absorption. They reported no difference ($P > .05$) among the three classes in mean closure time post partum (IgG 26.4, IgM 25.0, and IgA 26.0 hr.). However, at some starting ages of

colostrum feeding the closure times were declared significantly different, although no particular trend was observed. For example, at starting time of 12h closure time for IgG was late (30h) and for IgA it was early (25h); at 16h starting time IgM was earliest of the three Ig's. Further analysis of the data, Stott et al. (1979b) showed that all immunoglobulin classes have common characteristics of absorption following a rapid transfer during the first 4h after feeding, and that age at first feeding had an inverse effect on rate of absorption. There was a positive linear trend in the amount fed up to 2 liters but the rates of absorption in succeeding time periods following the initial feeding had decreasing linear trends. Only immunoglobulin M had a significant quadratic response for amount fed.

The mechanism whereby absorption of macromolecules from gut to blood ceases has been referred to as, "closure" (Lecce and Morgan, 1962). The mechanisms of closure are not well elucidated, although, they appear to vary somewhat with several factors as pointed out by Bush and Staley in 1980; these are specie differences, intraluminal environment, length of postnatal life, and to some degree circulating hormones. Nevertheless, the process of closure apparently occurs in rectrograde fashion, that is, the basal cell membrane ceases to release the evacuolated product, transport ceases, and eventually uptake by the tubule system ceases (Staley 1971).

The Effects of Heating Colostrum

Although it is well known that at certain temperatures antibodies are destroyed or inactivated, detailed experiments covering this point in bovine colostrum have not been reported. Some experiments have been

designed to differentiate agglutinins by means of heat. Beyer and Reagh (1904) differentiated the flagellar and somatic agglutinins by heating serum at 70°C for 20 minutes. Jones (1927) studied the effect of various temperatures on certain antibodies contained in the blood serum of rabbits and cows. The activity of all the antibodies tested was reduced appreciably by heating at 60°C for 20 minutes. Differential tests to distinguish between nonspecific and specific agglutinins for Brucella based on heat inactivation of the nonspecific agglutinins are well known. Yet, most of these studies have been carried out with blood serum and very few using bovine colostrum. Thus, the literature concerning the effects of heating on antibodies present in bovine colostrum is very scarce.

Studies with human milk and colostrum suggest that the extent of heat denaturation of immunoglobulins is related to the protein content in the samples examined. Frank and Dóbias (1976) reported that heat denaturation of immunoglobulins was more severe in human milk with low protein content (900 mg/100 ml) than in milk with average protein content (1300 mg/100 ml). Since protein content varies within species and individuals during lactation, it may explain the controversial results reported in the literature regarding colostrum pasteurization. For instance, Ford et al. (1977) studied to what extent the immunoglobulins and other protective proteins in human milk are stable to heating for the purpose of destroying contaminant bacteria, and found that the holder method (62.5°C 30 minutes) reduced the content of IgA by 20% and completely destroyed IgM. On the other hand, Raptopoulou-Gigi et al. (1977) reported that pasteurization did not damage proteins, or had little effect on the levels of IgA, lactoferrin, or antibody to E. Coli.

Previous research (Szöllőzy et al. (1974) also had shown that pasteurization caused no reduction in antibody titer against E. coli. Liebhaber et al. (1977) found a significant decrease in immunoglobulin concentration and in specific antibody titer to Escherichia Coli after pasteurization of human milk. Therefore, one can conclude that the effect of heating colostrum has not been studied extensively enough to elucidate the factors responsible for the variation in results reported by different investigators.

CHAPTER III

EFFECT OF PASTEURIZATION ON ABSORPTION AND ACTIVITY OF IMMUNOGLOBULINS

Abstract

Seven pooled batches of colostrum from cows at the first two milkings postpartum were collected. One-half of each batch was pasteurized at 62.5°C for 30 minutes, after which all was packaged in milk cartons and frozen. Forty-eight dairy calves obtained before nursing were assigned at random to pasteurized and unpasteurized groups within blocks. The calves were fed the appropriate type of colostrum at 10% of metabolic size soon after birth and at 12 and 24 hr afterwards. A sample of blood was taken from each calf before feeding and at 12, 16, 20, 24, 28, 32 and 36 hr for determination of immunoglobulin concentration by single radial immunodiffusion. Compared to the control group, the blood serum IgG of calves fed pasteurized colostrum was higher ($P < .005$) at 12 hr after first feeding. Thereafter, the differences between groups tended to be smaller, less consistent and then negligible at 36 hours. Concentration of IgM in serum of the calves was similar at each sampling period.

In a second experiment, batches of colostrum were pooled from cows immunized with rabbit erythrocytes. One-half was pasteurized and all was frozen until needed. Twenty-two dairy calves handled and grouped as in the previous experiment were fed the appropriate colostrum at 10%

of metabolic size. Blood samples from each calf were collected at the appropriate intervals and the serum IgG and the titer against rabbit erythrocytes were determined. The serum IgG concentration of the calves were similar at each sampling period, and agglutinating titers were comparatively higher at 12 through 32 hr in calves fed unpasteurized colostrum. From 24 to 32 hr after feeding colostrum the titers were significantly higher ($P < .025$ to .05) for calves fed the control colostrum. It was concluded that antibody activity may be reduced in serum of calves fed pasteurized colostrum.

In a third experiment colostrum from the third milking of a cow after calving was obtained and a portion was combined with serum from a calf immunized with Brucella abortus. Another portion of colostrum was combined with the gamma globulin fraction from the immune serum of this calf. Duplicated aliquots of these and the immune serum containing agglutinins for Brucella were heated at 62.5°C to simulate pasteurization. Neither the respective test colostrums nor the immune serum showed visible agglutination for the antigen used in this experiment. Evidence is presented to suggest that antibodies in colostrum can be destroyed or inactivated during pasteurization.

Introduction

Bacterial infection of newborn calves may be related to the occurrence of pathogenic organisms in colostrum due to udder infection in the cow. In a controlled study, Volovenko (1972) reported a mortality rate of 71.4% in calves fed colostrum from cows with staphylococcal mastitis, and 50% morbidity and 20% mortality in calves fed colostrum from cows with Streptococcus agalactiae mastitis. Kharinova and Abzalova

(1976), cited by Keys et al. (1980), have shown that calves developed dyspepsia when fed immediately after birth fresh colostrum from cows infected with mastitis. It appears that the gut of newborn calves may be more susceptible to organisms at birth. However, experiments by Corley et al. (1977) clearly demonstrated the beneficial effects of colostrum in limiting transepithelial migration of one serotype (055) of infectious Escherichia coli in newborn calves. Other researchers, Kesler (1981) and Barto et al. (1982) found no evidence of localized infection or health disorders in calves fed milk to which cells of Staphylococcus aureus were added. These results were interpreted as evidence that feeding milk containing mastitis-causing staphylococci was not detrimental to young calves. Nevertheless, Kesler (1981) suggested that feeding of mastitic milk be delayed until after the first day of life of the calf because of possible permeability of the gut to microorganisms.

Pasteurization of colostrum prior to feeding it to calves has been proposed to prevent the newborn calf from ingesting pathogenic microflora that could lead to illness or death. Although pasteurization is commonly practiced in the storage of human milk (Honour and Dolby, 1979), information is not available regarding the effects of this practice on the well being of calves.

The main protective factor in colostrum for the newborn is associated with the absorption of intact immunoglobulins during the first few hours of life. The possible effect of pasteurization of colostrum on immunoglobulin concentration appears to vary with the class of immunoglobulin in question. For instance, Szöllósy et al. (1974) found that treatment of human milk at 65^{o}C for 30 minutes caused no reduction in

E. coli type 055 antibody titer. Raptopoulon-Gigi et al. (1977) showed that pasteurization has little effect on the levels of IgA, lactoferrin and antibody to E. coli. Rogers and Synge (1978) showed that the bacteriostatic titer against a specific E. coli strain was stable to pasteurization. On the other hand, Ford et al. (1977) reported a decrease in IgA titer by 20% and destruction of the IgM. Liebhader et al. (1977) reported that pasteurization of human milk resulted in 33% loss of IgA ($P < 0.01$), a significant ($P < 0.01$) decrease in IgG and a large percentage decrease in IgM, but no statistical significance could be demonstrated for the change in IgM because of the small sample number.

The present study was undertaken to determine the possible effects of pasteurizing bovine colostrum on the absorption of immunoglobulins by newborn dairy calves and on the extent to which the immunoglobulins are inactivated by this process.

Experimental Procedure

Trial 1

Colostrum from several dairy cows at the first two milkings postpartum was pooled to make seven batches. One-half of each batch was pasteurized by heating at 62.5^{o}C for 30 minutes (holding method). Then, both the pasteurized and unpasteurized portions were packaged in half pint plastic coated paper cartons and frozen until needed.

A sample of each batch of unpasteurized colostrum was analyzed for total protein, fat and IgG concentration (Table III). Total protein was determined by Kjeldahl procedure and fat was determined by Babcock method. There was a wide variation in the colostral IgG concentration (29 to 62

TABLE III

COMPOSITION OF COLOSTRUM

Batch	Date Collected	Fat	Total Protein	IgG Concentration
		(%)	(%)	(%)
TRIAL I				
1	07-22-80	2.0	8.1	29.14
2	07-23-80	2.5	7.4	33.55
3	07-25-80	3.3	9.4	43.50
4	07-28-80	3.0	10.4	50.49
5	07-29-80	2.5	10.3	61.66
6	07-31-80	2.6	10.9	38.79
7	08-01-80	3.2	10.5	61.22
TRIAL II				
1	07-27-81	2.3	11.2	60.30
2-3	07-28-81	3.3	10.4	55.48
4	07-30-81	2.6	10.3	61.60
5	08-06-81	3.5	11.2	54.04

mg/ml) and total protein content (7.4 to 10.9%) among the seven batches of pooled colostrum. This is in agreement with other reports in that there is wide variation in the colostrum immunoglobulin concentration from cow to cow and in pooled colostrum. Stott et al. (1979) reported ranges of IgG concentration 28.4 to 46 mg/ml in five pooled colostrums. However, the fat content did not vary considerably among the batches fed in the present experiment. Other authors (Parrish et al. 1950) reported fat contents of first-milking colostrum ranging from .3 to 18.0% in individual cows.

Coliforms in the colostrum were enumerated using violet red bile agar as per procedures in Standard Methods for the Examination of Dairy Products (1978). Viable coliform counts for the raw and pasteurized colostrums showed the effectiveness of the pasteurization (Table IV).

Forty-eight dairy calves (32 Holsteins and 16 Ayrshires) from the University herd were separated immediately after birth, before nursing their dams. A commercial reovirus and corona virus vaccine was administered orally to all calves at birth. The navel of each calf was disinfected with a 7% iodine solution, then each calf was weighted, and transported to an individual calf pen for the course of the experiment. The calves were assigned at random to treatments within groups which were of a given breed and designated to receive a given batch of colostrum. One-half of the calves within each group were fed pasteurized colostrum, whereas the other half received unpasteurized (control) colostrum. All calves were fed colostrum by nipple bottle at 10% of metabolic size within 2 hours (average = 40 min.) after birth and at 12 and 24 hours afterwards.

Blood samples were taken from each calf by jugular venipuncture

TABLE IV

BACTERIAL COUNTS ON BATCHES OF COLOSTRUM

Batch	Coliform Count/ml		Total Count/ml	
	Unpasteurized	Pasteurized	Unpasteurized	Pasteurized
TRIAL I				
1	100,000	< 1		
2	1,800	< 1		
3	300	20		
4	450,000	< 1		
5	170	< 1		
6	2,800,000	< 1		
7	2,100	< 1		
TRIAL II				
1	4,900,000	< 1	3,000,000	160
2-3	2,000	< 1	310,000	160
4	220,000	< 1	5,000,000	76
5	250,000	< 1	4,100,000	95

immediately after birth (before feeding) and subsequently at 12, 16, 20, 24, 28, 32 and 36 hours. These were allowed to clot at room temperature, then centrifuged 10 min at 1650 x g and 5°C to separate the serum which was frozen until assayed for immunoglobulin concentration.

Immunoglobulins (IgG and IgM) were assayed by the single radial immunodiffusion technique as described by Fahey and McKelvey (1965). Purified bovine immunoglobulin G and M and antisera for these prepared in rabbits were obtained from Miles Laboratories.[1] Standard curves (IgG and IgM) were prepared by plotting the immunoglobulin concentration (mg/ml) of the standard reference samples against the diameter of the precipiting rings. In preparation for assay of colostrum for IgG, colostrum was centrifuged 10 min at 1650 x g and 5°C and the fat then removed, according to the procedure of Fleenor and Stott (1981). Skim milk was used as a diluent in preparing the IgG standards to resemble the composition of the fat-free colostrum.

Trial 2

Twenty-two calves (18 Holsteins and 4 Ayrshires) used in this experiment were handled and assigned to treatments within groups essentially as described in the first trial. No virus vaccine was administered at birth. Five batches of colostrum from 32 cows at the first two milkings post partum were collected and half of each batch pasteurized. Colostrum was analyzed for total protein, fat, and IgG concentration (Table III). Both total bacterial counts and viable coliform counts were determined on each batch of colostrum (Table IV). As in Trial I, a

[1]Research Products Division, Miles Biochemicals, Elkhart, IN.

drastic reduction in the number of bacteria by the pasteurization processes was demonstrated.

Of particular interest was the higher IgG concentration found in these batches (54 to 62 mg/ml) in comparison to those used in Trial 1. The cows were immunized prior to calving by subcutaneous injection of 1 ml of packed rabbit erythrocytes reconstituted to its original volume with pH 7.3, 0.15 M phosphate-buffer and saline (PBS). The rabbit erythrocytes, obtained fresh from two rabbits, were injected subcutaneously into four different areas on the neck of each cow.

Calves were fed pasteurized or unpasteurized colostrum by nipple bottle at 10% of metabolic size at the same intervals as in Trial 1. Blood samples were collected before feeding and at the same intervals as in Trial 1. Duplicate aliquots of the serum collected from each calf at each sampling period were frozen for storage. These were assayed for IgG and tested for hemagglutination with rabbit erythrocytes in microtiter plates. The latter test was performed in duplicate as follows: 0.25 ml of phosphate-buffered saline containing 2% bovine serum albumin (BSA) was placed into each well of the microplate; to the first well in a row 0.25 ml of serum to be tested was added and dilutions were carried out using a microdilutor. Then, 0.5 ml of 2% rabbit erythrocyte suspension in PBS (containing 2% BSA) was transfered to each well. The microplates were allowed to stand overnight at room temperature, then each well was read for the presence of a shield (hemoagglutination) or the presence of a button (resembling the control). The highest dilution of serum which exhibited complete hemoagglutination was recorded as the hemagglutionation titer of that serum.

Trial 3

The purpose of this trial was to study the effect of heating colostrum on the functionality of specific antibodies. To provide the immune serum for this experiment a 350 lb male Jersey calf was immunized with Brucella antigen prepared from Brucella abortus strain 1119-3 diluted 1:100 with 0.85% NaCl solution containing 0.5% phenol. The antigen is commonly used in the diagnosis of brucellosis in animals by the serum-tube agglutination test method. The calf received six intravenous injections spaced every 2 days starting with 0.25 ml and then .50, .75 and 1 ml of the suspension of this antigen for the last three injections. The suspension contained approximately 4.5×10^{10} cells of B. abortus 1119-3 per milliliter. Blood samples were taken weekly and the serum was separated by centrifugation. The serum was then assayed by the standard tube test (STT) for brucellosis to monitor the antibody status of the animal. The serum with the highest titer (serum dilution = 3,200) was found about 50 days after the first inoculation.

Colostrum from a cow at the third milking after calving was obtained and mixed thoroughly in a sterilized container. Then 25 ml were placed in a dialysis bag which was suspended in a walk-in cooler at $4^{\circ}C$ for 48 hours; afterwards about 20 ml were recovered from the bag. A portion of this concentrated colostrum was adjusted to a suitable titer by the addition of 3.5 ml immune serum to 9 ml of colostrum. To another 9 ml portion of concentrated colostrum, 3.5 ml of serum globulin from the immunized calf were added. This globulin fraction was obtained by precipitation of globulin from the serum with saturated ammonium sulfate, centrifugation 30 min at 2000 x g and room temperature, and reconstitu-

tion of the isolated material to one-half the original volume with physiological saline. Duplicate aliquots of the colostrum plus immune serum, colostrum plus serum globulin, and serum containing specific agglutinins for Brucella were heated to simulate pasteurization, whereas two unheated aliquots of each served as a control. The colostrums and serum which were heated were handled identically as follows: the lower half of several 15 x 150 mm test tubes were immersed in a water bath adjusted to 62.5°C. The temperature was monitored by placing an aliquot of original colostrum in a tube and reading the temperature with a thermometer, graduated in 0.1°C, placed with the bulb in close proximity to the bottom of the inside of this tube. Three ml of serum or the respective test colostrums were placed in each of two immersed tubes for each material tested and then the tubes were tightly capped. After 4 minutes were allowed for temperature equilibrium (zero time), the tubes were heated for 30 minutes. Afterwards the test colostrums and the immune serum were cooled by holding the tubes under running cool water and the agglutination titer was then determined by the standard tube test for brucellosis. Colostrum samples were first defeated by centrifugation (Fleenor and Stott, 1981) and the whey separated by the addition of .3 ml of commential rennin solution.

Results and Discussion

Trial 1

Based on the serum concentrations of IgG and IgM at birth (Tables VII and IX) it is clear that calves are not totally agammaglobulinaemic at birth which is in agreement with reports by others (Pierce and Fein-

stein, 1965; Klaus et al. 1969; Bush et al., 1971 and Mungle 1972). In fact, in three of 48 calves concentration of IgG at birth, before nursing, resembled the concentration generally found several hours after colostrum feeding (Table VII). Also three of 35 of the calves for which concentration of IgM were determined had relatively high values at birth (Table IX). A similar high concentration of IgG in one calf known to be colostrum-free was observed by Mungle (1972). The reason for this occurrence is not known. Also concentration of IgG and IgM in these calves were high throughout the trial. As expected, in most calves the immunoglobulin concentration increased markedly after ingestion of colostrum. However, the blood serum IgG of calves fed pasteurized colostrum was higher ($P < 0.005$) at 12 hours after first feeding than in calves fed unpasteurized colostrum. Thereafter, the differences between groups tended to be smaller, less consistent and, at the end of the experimental period (36 hours), negligible (Figure 2).

On the other hand, concentration of IgM in serum of the calves was similar at each sampling period (Figure 3). The peak average concentration of both IgM and IgG occurred at 32 hours after first feeding, indicating that closure of the intestinal epithelium to immunoglobulins transfer probably was between 24 and 36 hours. Mungle (1972) reported peak immunoglobulin concentration in sera of calves fed pooled colostrum with lower IgG concentration than in the present study at 28 hours after birth. Penhale et al. (1973) reported closure at 27 and 16 hours for IgG and IgM respectively by an extrapolation of data from feeding calves colostrum at 1, 3 and 9 hours postpartum. Stott et al. (1979) by a joint point analysis on data from 210 calves estimated time of closure of intestinal permeability to colostral immunoglobulins being near 24 hours of age with a normal distribution having a standard deviation of

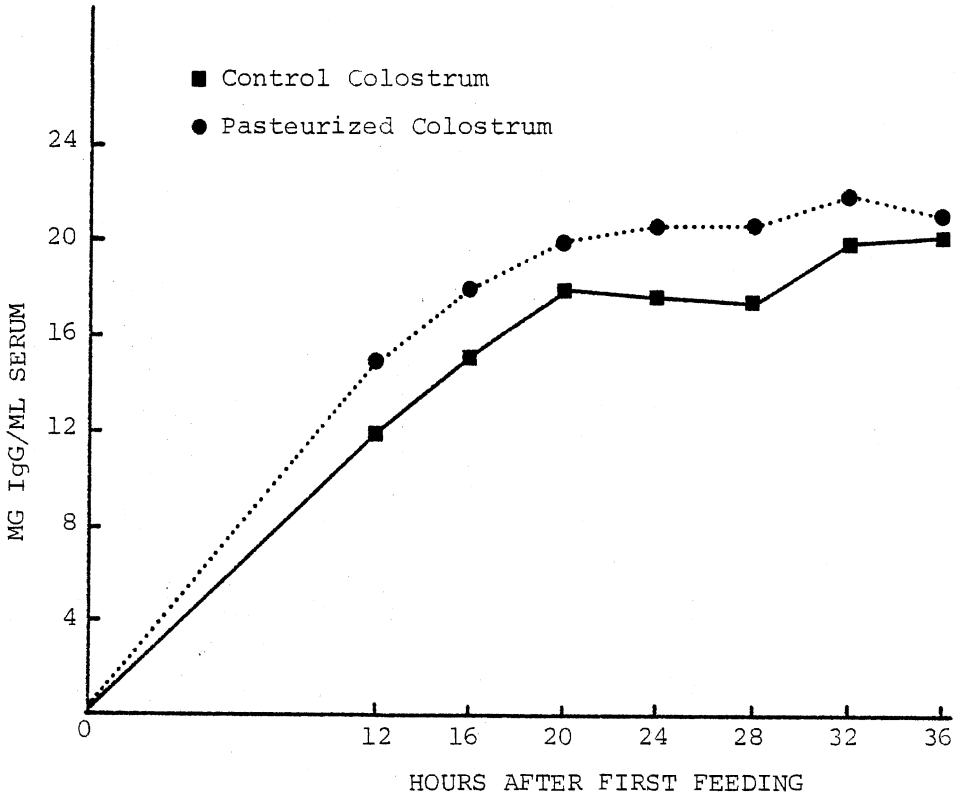

Figure 2. Concentration of IgG in Blood Serum of Calves - Trial I

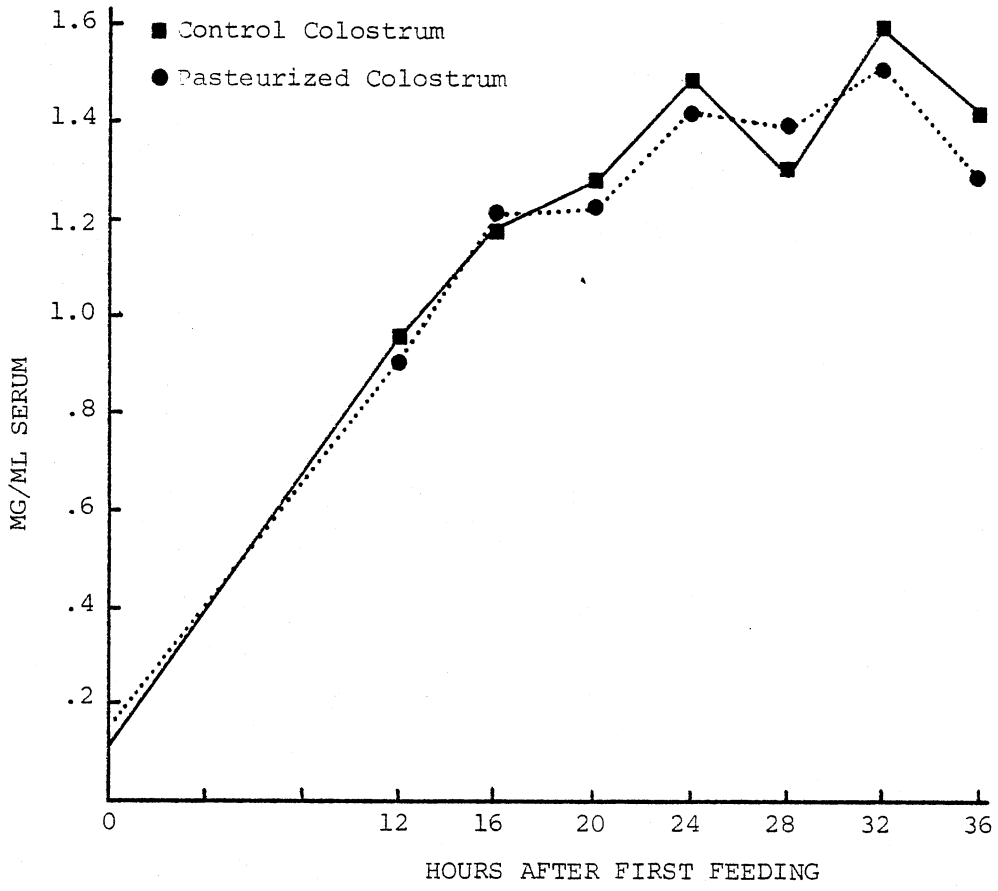

Figure 3. Concentration of IgM in Blood Serum of Calves - Trial I

approximately 4 hours. The colostral IgG concentration in that experiment ranged between 28.4 to 46.0 mg/ml which resembles the colostral IgG values in the present experiment. There was a wide variability among calves in concentration of IgG (9.85 to 36.41 mg/ml) in serum at 32 hours (peak immunoglobulin concentration) indicating that all calves absorbed immunoglobulins in this experiment and only one calf had serum IgG concentration (7.23 mg/ml) at the end of the trial comparable to levels reported by Penhale et al. (1973) in surviving calves where spontaneous disease occurred with high incidence. The levels of IgM were 4 to 30 times higher than levels reported by those workers.

It has been shown in numerous studies that calves which sucumbed to infectious diarrhea had lower immunoglobulin concentration in their sera than those that survived (Gay 1965; Dam 1968; Hurvell and Fey 1970; McEwan et al., 1970; Penhale et al., 1970; Penhale et al., 1973; McGuirre et al., 1976). Thus, more rapid uptake of IgG under the present experimental conditions would have significance in terms of resistance of calves to certain infectious agents, providing that the absorbed immunoglobulin retained its functionality. A possible explanation for the lower IgG concentration at 12 hours in serum of calves that received unpasteurized colostrum is that bacteria might bind the antibody molecule making it unavailable for intestinal absorption. A similar mechanism has been proposed to explain the malabsorption of Vitamin B_{12} where the increased numbers of bacteria such as Escherichia coli are believed to result in greater binding of Vitamin B_{12} making it unavailable for absorption (Tennant et al., 1971). Moreover, subnormal concentration of gammaglobulin has been reported in inoculated calves with a mixed population native to the duodenum of a milk-fed calf (James et al., 1976).

The same authors concluded that the rate and nature of the bacterial colonization of the intestine were related to the closure phenomenon. This in turn might explain the less consistent differences observed after 12 hours in this trial when it might be assumed that bacteria colonization was to a great extent similar in both groups of calves. However, James and Polan (1978) have presented evidence that inoculation of calves with bacteria of enteric origin did not interfer with absorption of immune globulins, providing that colostrum was fed prior to or simultaneous with the bacterial inoculation.

One of the principal points which has to be considered in relation to the results of this experiment is the validity of the method used to measure the immunoglobulin concentration in serum of calves. Single radial immune diffusion is an antibody-precipitant technique for quantitating specific proteins in a complex mixture without separating the individual protein to be measured. The method might not be indicative of the actual quantity of intact IgG in the serum of calves fed pasteurized colostrum. The activation of some interchain sulfhydryl groups during heating may result in a change in the spatial configuration and possible cleavage of some of the IgG molecules. Therefore, the characteristic properties of the protein are altered, i.e., loss of the antigen-binding ability. The disrupted IgG would retain its primary amino acid sequences, which in turn would still be precipitated by the antiserum used in this experiment. Moreover, if cleavage of some of the molecules occurred, the resulting values of IgG would tend to be higher since the final size of the precipitate ring in the gel-diffusion plate is a function of the MW of the protein measured.

Trial 2

This feeding trial was to determine whether the immunoglobulin concentrations measured in serum of calves fed pasteurized colostrum represent functional molecules capable of binding specifically with their antigens. As in the previous experiment calves were fed pasteurized or unpasteurized (control) pooled colostrum. In this trial colostrum was obtained from cows with a known agglutinating titer against rabbit red blood cells. The serum immunoglobulin concentration (IgG) of the calves for each treatment group averaged at each sampling period were similar (Figure 4). Although the IgG blood serum concentration was lower for the control group than for the treated group at a majority of the sampling periods, none of the differences were statistically significant ($P > 0.10$). The smaller number of calves in this experiment compared to the previous one (22 instead of 45) may explain the lack of statistically significant differences between treatment groups at 12 hours after feeding. No agglutinating antibodies against rabbit erythrocytes were detected in the serum of calves immediately after birth. The agglutinating titers were comparatively higher at 12 through 32 hours in calves fed unpasteurized colostrum indicating that antibody activity may be reduced in serum of calves fed pasteurized colostrum. From 24 to 32 hours after feeding the titers were significantly higher ($P < .025$ to .05) for calves fed the control colostrum. Nevertheless, since the agglutinating titers in sera of control calves were low, these results were not considered conclusive in terms of the relationship of heat to inactivation or loss of specificity of the antibody molecule. The low agglutinating titers did not reflect efficacy of the inoculation, i.e., in nearly 80% of the calves, at 12 hours after feeding, titers were

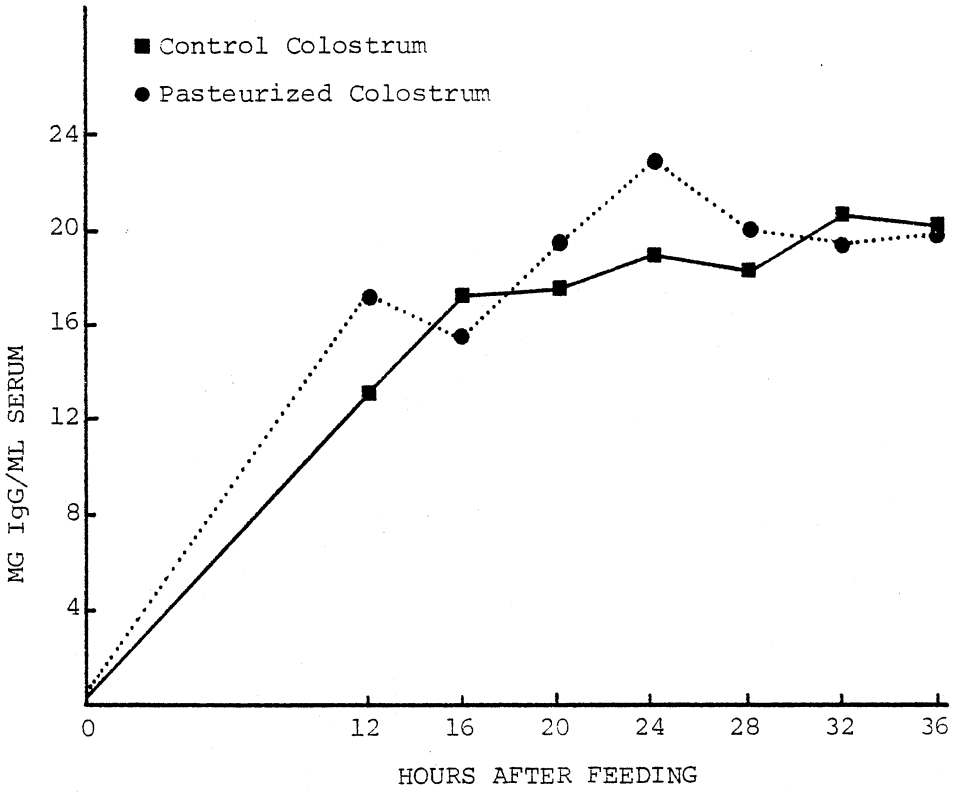

Figure 4. Concentration of IgG in Blood Serum of Calves - Trial II

below 1:2 and at 24 hours when peak average IgG concentration occurred, 63 and 29% of the calves had titers below 1:2 and 1:4, respectively. Under the present conditions, only one subcutaneous injection of 1 ml of the rabbit erythrocyte suspension to preparturient cows was not enough to elicit a satisfactory antibody response to be passively transmitted to their calves via colostrum, and that an additional exposure to the antigen would induce an anamnestic response with the subsequent increase in antibody titers.

Trial 3

After heating to simulate pasteurization neither the colostrum to which either the immune serum or the globulin fraction thereof had been added nor the immune serum showed agglutination for Brucella antigen used in this experiment (Table V). No agglutinated particles were found in the immune serum or test colostrums which had been heated at 62.5°C for 30 minutes. On the other hand, agglutinating particles were found tightly adhering to the bottoms of the test tubes containing either unheated immune serum or test colostrums.

Based on the results of this experiment, it appears that after heating colostrum at 62.5°C for 30 minutes antibodies against Brucella abortus strain 1119-3 are destroyed or inactivated. Possibly, the antigen used to stimulate agglutinin production in this study could excite the production of agglutinins that were relatively heat-labile. In fact, Amerault et al., 1961 devised a heat-inactivation test to inactivate certain agglutinins encountered in the standard tube agglutination test for brucellosis in bovine sera from herds apparently free of brucellosis. This test was based on the observation that certain types

TABLE V

AGGLUTINATION TITERS OF HEATED VS CONTROL COLOSTRUM AGAINST BRUCELLA ANTIGEN

	Heated	Control
Colostrum plus immune serum	< 50	800[a]
Colostrum plus serum globulin	< 50	400[b]
Immune serum	< 50	3600[a]

[a]Incomplete agglutination.

[b]Complete agglutination.

of agglutinins react differently to varying temperatures. However, White and Koepke (1962) reported that agglutinins in sera of Brucella-infected cows were only partially inactivated after 30 minutes at 63°C.

CHAPTER IV

SUMMARY AND CONCLUSIONS

Pasteurization of colostrum may accelerate absorption and consequently the IgG concentration in blood serum of calves soon after birth. These higher IgG levels in their serum could have significance in protecting calves against colisepticemia. However, it appeared that modifications in the structure of the gammaglobulin may occur during pasteurization with the consequent loss of antibody activity. Other antimicrobial factors known to participate in the immunologic system of calves may also be affected. It might be hazardous to feed colostrum lacking these factors to calves. It might well be that the heat treatment that is adequate to ensure the absence of pathogenic organisms in colostrum might actually destroy or inactivate the antibodies thereby increasing the risk of enteric infection in the newborn calf. The idea should be to collect clean colostrum, with minimal contamination and to store it immediately in a freezer until it is gently thawed for feeding to the calf.

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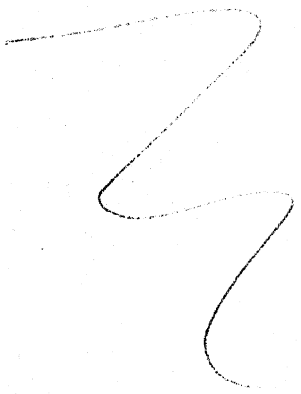

TABLE VI

GENERAL INFORMATION ABOUT CALVES USED IN TRIAL I

Block	Animal No.	Date of Birth	Body Weight	Breed	Sex	Treatment Group	Amount of Colostrum Per Feeding	Age at 1st Feeding
			(Kg)				(Kg)	(Min)
1	3304	7-22-80	33.11	Hol	F	1	1.380	60
1	218	7-23-80	44.91	Hol	M	1	1.734	45
1	3307	7-24-80	41.73	Hol	F	2	1.642	45
1	3306	7-23-80	35.83	Hol	F	2	1.465	75
2	226	7-27-80	41.73	Ayr	M	1	1.642	20
2	225	7-27-80	32.21	Ayr	M	1	1.352	60
2	3308	7-24-80	36.74	Ayr	F	2	1.492	30
3	3309	7-25-80	51.71	Hol	F	1	1.928	90
3	222	7-26-80	46.26	Hol	M	1	1.774	20
3	223	7-26-80	46.27	Hol	M	1	1.774	15
3	3314	7-28-80	38.10	Hol	F	1	1.536	45
3	3310	7-26-80	41.28	Hol	F	2	1.628	100
3	3312	7-27-80	39.92	Hol	F	2	1.588	25
3	3311	7-27-80	40.82	Hol	F	2	1.615	45
4	3317	7-30-80	42.18	Hol	F	1	1.655	30
4	235	7-30-80	47.63	Hol	M	1	1.813	60
4	240	7-31-80	43.54	Hol	M	1	1.695	30
4	3324	7-31-80	39.00	Hol	F	2	1.560	30
4	237	7-30-80	42.64	Hol	M	2	1.669	60
4	239	7-31-80	42.18	Hol	M	2	1.655	30
5	3322	7-31-80	39.46	Ayr	F	1	1.574	15
5	3323	7-31-80	30.84	Ayr	F	1	1.308	10
5	243	8-02-80	27.67	Ayr	M	1	1.206	25
5	250	8-06-80	36.74	Ayr	M	2	1.492	45

TABLE VI (Continued)

Block	Animal No.	Date of Birth	Body Weight	Breed	Sex	Treatment Group	Amount of Colostrum Per Feeding	Age at 1st Feeding
			(Kg)				(Kg)	(Min)
5	3318	7-30-80	39.00	Ayr	F	2	1.560	45
5	236	7-30-80	41.28	Ayr	M	2	1.628	30
6	242	8-01-80	43.09	Hol	M	1	1.682	60
6	3327	8-02-80	43.54	Hol	F	1	1.695	30
6	244	8-03-80	53.98	Hol	M	1	1.991	30
6	247	8-04-80	48.53	Hol	M	1	1.839	60
6	3336	8-08-80	42.18	Hol	F	1	1.655	15
6	255	8-09-80	51.71	Hol	M	1	1.928	60
6	256	8-09-80	37.65	Hol	M	1	1.519	30
6	3326	8-02-80	39.46	Hol	F	2	1.574	30
6	246	8-04-80	44.45	Hol	M	2	1.722	30
6	3331	8-04-80	40.37	Hol	F	2	1.602	30
6	248	8-05-80	45.36	Hol	M	2	1.748	30
6	3337	8-08-80	47.17	Hol	F	2	1.800	45
6	3338	8-09-80	49.90	Hol	F	2	1.877	30
7	251	8-07-80	33.57	Ayr	M	1	1.395	30
7	252	8-08-80	32.21	Ayr	M	1	1.352	15
7	3348	8-16-80	29.03	Ayr	F	1	1.251	45
7	3342	8-10-80	33.11	Ayr	F	2	1.380	45
7	3349	8-17-80	34.47	Ayr	F	2	1.423	45
7	257	8-09-80	34.47	Ayr	M	2	1.427	30

TABLE VII

CONCENTRATION OF IgG IN BLOOD SERUM OF CALVES AT DIFFERENT HOURS (gm/100 ml) - TRIAL I

Calves No.	Hours After First Feeding							
	0	12	16	20	24	28	32	36
3304	0.007	0.844	0.808	1.123	1.339	0.963	1.495	1.123
218	0.007	0.988	0.885	0.637	0.726	0.710	1.258	1.178
3307	0.014	1.032	1.127	1.500	1.231	1.674	1.344	1.231
3306	0.007	0.826	1.782	1.227	1.528	1.528	1.562	1.029
226	0.017	0.663	0.863	1.254	1.099	1.148	1.282	1.528
225	0.010	0.808	0.902	1.099	1.099	1.052	0.985	0.724
3308	0.009	0.678	0.844	0.863	0.844	0.607	1.052	0.844
3313	0.594	1.254	1.495	1.822	1.148	1.632	1.744	1.946
3309	0.007	1.052	1.597	1.562	1.744	1.947	2.079	2.079
222	0.008	1.093	1.734	1.852	1.696	1.362	1.660	1.697
223	0.008	1.275	1.696	1.936	1.936	2.576	2.520	2.751
3314	0.009	1.001	1.275	1.852	1.392	1.392	1.893	1.893
3310	0.030	1.632	1.528	2.221	1.904	2.891	2.891	2.706
227	0.553	1.247	2.160	1.362	1.696	2.633	2.067	2.113
3312	0.007	1.454	1.734	2.465	2.575	2.751	2.465	2.633
3311	0.008	1.392	1.660	1.487	1.812	1.734	1.936	1.979
3317	0.008	1.142	1.454	1.487	1.454	1.812	1.734	1.812
235	0.095	1.332	1.660	1.812	3.004	2.208	2.465	2.633
240	0.070	1.802	2.102	1.968	2.196	2.398	2.149	2.677
3324	0.008	1.111	1.925	1.925	2.506	2.736	2.451	2.562
237	0.008	1.487	1.046	1.773	2.067	1.979	1.979	2.208
239	0.067	1.773	1.696	1.893	2.812	2.160	3.504	2.520
3322	0.007	0.799	2.011	1.384	2.196	2.056	1.925	2.102
3323	0.021	1.579	1.725	3.262	2.988	2.923	2.562	2.295
243	0.009	1.725	2.562	2.196	2.677	3.335	3.191	3.485
250	0.006	2.797	4.247	3.122	2.677	2.797	3.641	2.245
3318	0.011	1.763	2.346	2.295	3.562	2.988	3.409	3.976

TABLE VII (Continued)

Calves No.	Hours After First Feeding							
	0	12	16	20	24	28	32	36
236	0.126	2.245	2.346	2.506	2.736	2.736	2.859	3.054
242	0.009	1.431	1.632	2.479	2.647	2.221	2.590	3.088
3327	0.018	1.123	1.052	1.029	1.174	1.099	1.744	1.822
244	0.047	1.105	1.347	1.957	1.641	1.471	1.792	1.606
247	0.098	1.261	2.233	1.537	2.137	1.678	2.438	2.282
3336	0.008	1.233	1.792	2.721	0.868	1.012	2.548	1.641
255	0.008	1.081	0.652	2.233	1.233	0.479	1.058	0.947
256	0.009	0.786	1.332	1.588	1.734	1.734	2.359	2.633
245	0.477	1.528	2.270	1.782	2.647	2.372	2.828	2.221
3326	0.029	1.863	2.079	1.946	2.034	2.125	2.079	2.590
246	0.003	1.261	0.868	1.957	1.914	1.606	1.914	1.289
3331	0.010	1.233	2.090	2.090	2.045	1.914	1.914	2.233
248	0.011	1.347	1.207	2.233	2.090	2.385	1.571	1.503
3337	0.081	2.233	2.233	2.548	1.873	2.492	1.678	3.465
3338	0.013	1.362	1.893	2.113	2.465	2.257	3.004	1.893
251	0.010	2.257	2.465	3.071	2.113	2.751	2.691	2.576
252	0.014	1.093	1.423	1.332	1.588	1.554	1.623	1.812
3348	0.011	1.275	1.812	2.465	2.208	2.257	2.160	2.160
3342	0.010	1.623	1.893	1.734	1.303	1.554	1.734	1.520
3349	0.010	1.193	1.773	1.696	1.660	1.554	1.852	1.454
257	0.089	1.734	1.454	2.208	2.308	1.623	1.812	2.023

TABLE VIII

CONCENTRATION OF SERUM IgG IN CALVES BY BLOCKS (gm/100 ml) - TRIAL I

Block	Hours After First Feeding							
	0	12	16	20	24	28	32	36
1	0.009	0.923	1.151	1.122	1.206	1.107	1.415	1.140
2	0.011	0.707	1.169	1.020	0.972	0.854	1.092	0.985
3	0.012	1.299	1.608	1.929	1.895	2.139	2.234	2.272
4	0.043	1.441	1.647	1.810	2.340	2.216	2.380	2.402
5	0.030	1.818	2.539	2.461	2.806	2.806	2.931	2.859
6	0.026	1.348	1.581	2.041	1.852	1.757	2.051	2.083
7	0.024	1.529	1.803	2.084	1.863	1.882	1.979	1.924

TABLE IX

IgM CONCENTRATION IN BLOOD SERUM OF CALVES AT DIFFERENT HOURS (gm/100 ml) - TRIAL I

Calves No.	Hours After First Feeding							
	0	12	16	20	24	28	32	36
252	0.012	0.083	0.136	0.069	0.138	0.099	0.144	0.163
3342	0.006	0.070	0.009	0.006	0.026	0.120	0.157	0.138
3348	0.008	0.021	0.008	0.012	0.015	0.005	0.005	0.056
3349	0.009	0.097	0.007	0.104	0.011	0.067	0.062	0.058
257	0.011	0.090	0.148	0.125	0.189	0.123	0.128	0.098
251	0.007	0.219	0.197	0.248	0.367	0.359	0.461	0.243
3338	0.012	0.048	0.088	0.182	0.154	0.185	0.269	0.214
256	0.009	0.066	0.104	0.079	0.167	0.098	0.125	0.123
255	0.015	0.047	0.063	0.048	0.065	0.034	0.072	0.055
3337	0.027	0.219	0.215	0.148	0.123	0.197	0.186	0.161
3336	0.016	0.108	0.136	0.189	0.113	0.142	0.142	0.125
248	0.008	0.056	0.125	0.072	0.104	0.041	0.081	0.039
247	0.102	0.167	0.189	0.178	0.210	0.185	0.205	0.242
3331	0.021	0.092	0.151	0.139	0.214	0.219	0.151	0.160
244	0.008	0.063	0.128	0.122	0.136	0.090	0.110	0.113
246	0.011	0.026	0.047	0.035	0.067	0.035	0.061	0.031
3327	0.119	0.163	0.350	0.180	0.284	0.342	0.241	0.322
3326	0.049	0.101	0.117	0.163	0.169	0.129	0.122	0.135
242	0.013	0.089	0.141	0.141	0.180	0.153	0.135	0.129
245	0.021	0.037	0.099	0.091	0.169	0.095	0.127	0.055
243	0.008	0.077	0.127	0.135	0.200	0.152	0.204	0.217
250	0.016	0.132	0.132	0.140	0.176	0.221	0.180	0.180
3323	0.027	0.146	0.169	0.195	0.166	0.184	0.191	0.188
3318	0.011	0.064	0.114	0.180	0.132	0.208	0.169	0.217
3322	0.011	0.133	0.174	0.174	0.144	0.138	0.201	0.181
236	0.012	0.174	0.209	0.189	0.174	0.227	0.232	0.201
240	0.010	0.104	0.101	0.125	0.090	0.130	0.108	0.110
3324	0.009	0.029	0.047	0.038	0.082	0.052	0.078	0.057

TABLE IX (Continued)

Calves No.	Hours After First Feeding							
	0	12	16	20	24	28	32	36
235	0.008	0.111	0.096	0.154	0.154	0.076	0.206	0.167
237	0.013	0.081	0.139	0.128	0.154	0.094	0.113	0.125
3317	0.009	0.072	0.075	0.096	0.154	0.145	0.118	0.123
239	0.009	0.088	0.238	0.161	0.269	0.136	0.253	0.171
3316	0.009	0.130	0.159	0.177	0.204	0.209	0.196	0.141
3314	0.013	0.213	0.196	0.267	0.316	0.365	0.350	0.303
227	0.213	0.309	0.458	0.350	0.477	0.279	0.529	0.380

TABLE X

IgM CONCENTRATION IN SERUM OF CALVES BY BLOCKS (gm/100 ml) - TRIAL I

Block	Hours After First Feeding							
	0	12	16	20	24	28	32	36
4	0.010	0.088	0.122	0.125	0.158	0.120	0.153	0.128
5	0.014	0.121	0.154	0.169	0.165	0.189	0.196	0.197
6	0.017	0.079	0.118	0.117	0.138	0.118	0.132	0.112
7	0.009	0.097	0.084	0.094	0.124	0.129	0.159	0.126

TABLE XI

DATA ON INDIVIDUAL CALVES USED IN TRIAL II

Block	Animal No.	Date of Birgh	Body Weight	Breed	Sex	Treatment Group	Amount of Colostrum Per Feeding	Age at 1st Feeding
			(Kg)				(Kg)	(Min)
1	336	07-27-81	37.65	Hol	M	2	1.520	45
1	337	07-27-81	36.29	Hol	M	1	1.478	10
1	3415	07-27-81	27.22	Hol	F	2	1.188	60
1	3416	07-27-81	44.00	Hol	F	1	1.708	20
1	3420	08-01-81	37.65	Hol	F	1	1.520	30
1	338	07-27-81	47.99	Hol	M	2	1.823	30
2	3418	07-29-81	35.83	Ayr	F	1	1.465	60
2	3423	08-03-81	33.11	Ayr	F	2	1.380	60
2	342	08-03-81	40.82	Ayr	M	1	1.615	60
2	344	08-03-81	44.45	Ayr	M	1	1.722	60
3	3419	07-30-81	36.29	Hol	F	2	1.478	60
3	3422	08-02-81	42.19	Hol	F	1	1.655	45
3	3421	08-02-81	34.02	Hol	F	2	1.409	30
3	3424	08-04-81	44.45	Hol	F	1	1.722	30
4	345	08-05-81	48.99	Hol	M	1	1.852	60
4	3427	08-07-81	38.56	Hol	F	2	1.547	60
4	346	08-05-81	46.72	Hol	M	1	1.787	30
4	3428	08-09-81	46.72	Hol	F	2	1.787	30
5	365	08-23-81	48.53	Hol	M	1	1.839	120
5	361	08-16-81	51.71	Hol	M	1	1.928	45
5	377	08-28-81	36.74	Hol	M	2	1.492	45
5	3437	08-25-81	43.55	Hol	F	1	1.695	45

TABLE XII

CONCENTRATION OF IgG IN BLOOD SERUM OF CALVES AT DIFFERENT HOURS (gm/100 ml) - TRIAL II

Calves No.	Hours After First Feeding							
	0	12	16	20	24	28	32	36
336	0.011	2.265	1.756	2.037	2.363	2.217	2.081	1.871
337	0.009	1.391	1.101	1.101	1.683	1.174	1.579	1.305
3415	0.008	0.970	2.081	1.078	2.171	1.149	1.149	2.518
3416	0.009	1.012	1.391	0.752	1.278	1.451	1.683	0.524
3420	0.014	1.629	1.263	1.889	1.971	0.552	1.930	1.629
338	0.204	2.192	2.597	2.709	3.494	2.949	2.101	2.652
3418	0.014	1.971	2.145	2.013	1.811	1.664	1.889	2.101
3423	0.017	0.053	0.108	0.217	1.022	0.264	1.022	0.589
342	0.025	1.571	2.505	2.906	2.114	2.159	3.163	4.166
344	0.018	2.613	4.347	3.516	3.096	2.785	3.370	2.906
3419	0.010	2.336	2.101	2.239	2.767	2.652	3.143	2.437
3422	0.010	0.187	0.655	0.614	0.979	1.185	1.528	1.889
3421	0.014	1.375	0.497	1.112	1.185	1.089	1.434	0.919
3424	0.011	0.826	1.664	2.101	2.489	2.597	2.709	2.887
345	0.015	1.465	1.561	1.971	2.437	1.664	2.386	1.889
3427	0.011	1.465	0.919	2.652	1.629	2.287	1.773	1.699
346	0.011	0.601	1.022	2.287	1.736	1.930	1.375	2.287
365	0.022	1.595	2.386	1.263	2.336	2.192	1.629	2.336
361	0.011	0.904	0.849	0.617	1.243	1.537	1.982	1.217
377	0.013	2.024	1.569	2.398	3.227	2.299	1.708	1.637
3437	0.012	1.442	1.782	1.745	1.860	1.673	1.504	1.191
3428	0.010	3.006	2.537	3.006	2.821	2.703	3.070	2.761

TABLE XIII

CONCENTRATION OF IgG IN BLOOD SERUM OF CALVES BY BLOCKS (gm/100 ml) - TRIAL II

Block	Hours After First Feeding							
	0	12	16	20	24	28	32	36
1	0.043	1.576	1.698	1.595	2.160	1.582	1.754	1.750
2	0.018	1.552	2.276	2.163	2.011	1.859	2.361	2.440
3	0.011	1.181	1.229	1.516	1.855	1.881	2.204	2.033
4	0.012	1.634	1.510	2.479	2.155	2.146	2.151	2.159
5	0.014	1.491	1.646	1.506	2.166	1.925	1.706	1.595

VITA /

Ruben Dario Contreras N.

Candidate for the Degree of

Master of Science

Thesis: EFFECT OF PASTEURIZATION OF COLOSTRUM ON ABSORPTION OF IMMUNOGLOBULINS BY DAIRY CALVES

Major Field: Dairy Science

Biographical:

Personal Data: Born in Caracas, Venezuela, October 12, 1952, the son of Dr. and Mrs. F. Contreras.

Education: Graduated from Colegio Emil Friedman, Caracas, Venezuela in 1971; received Veterinary Medicine degree from the College of Veterinary Science in Maracay, Universidad Central de Venezuela, in 1979; completed the requirements for the Master of Science degree in Dairy Science at Oklahoma State University, May, 1983.

Professional Experience: Worked as student at the Pilot Milk Plant at the Universidad Central de Venezuela, Maracay; Veterinary Assistant in commercial dairy farms in Venezuela 1979; member of the Coleqio de Veterinarios de Venezuela; member of the American Dairy Science Association.