sanitary research facility, processed (identified, weighed, and bled), and fed either the product to be evaluated dissolved in 2 l of water or a non-medicated kid milk replacer within 2-4 hours of age. Colostrum fed calves are allowed to suckle the cow and stay with the cow for 4 hours for the mothering benefit and to encourage maximum absorption of colostral antibody. They are then similarly transported and processed.

Calves are inoculated orally at 15-18 hours of age with an 11 hour culture of 10^9 invasive JL-9 *E. coli* (078:NM:F41). Cultures are grown in N-ZAB broth, a minimal media composed of casein derived amino acids, on a 37°C shaker water bath. Calves receive 20 ml of the culture *per os* with a 2-3 pint feeding of milk replacer. Rectal temperature, heart rate, respiratory rate, fecal consistency, joint and umbilical tenderness, and appetite are monitored twice daily using a modified form of the foal sepsis scoring system developed at the University of Florida. Blood samples collected at 0, 24, and 120 hours. Some groups of calves are not necropsied until 240 hours to allow time for secondary septic arthritis to develop; an additional sample is collected in these calves at 240 hours (pre-necropsy).

Serum samples are used to determine immunoglobulin levels using single radial immunodiffusion plates[1]. Blood samples are submitted to a clinical pathology laboratory for CBC, differential cell count, and fibrinogen. Animals developing signs of septicemia are humanely euthanatized and promptly necropsied.

Necropsies include gross examination of body cavities and serosal surfaces for evidence of sepsis; bacterial cultures from the heart, liver, spleen, and two joints; and histological examination of the adrenal glands. Bacterial isolates are identified using biochemical test strips.[2] Adrenal histological samples are scored by a pathologist on a scale of 0-5 based on tissue congestion, hemorrhage and necrosis.

Trials using this model yield consistent and significant differences in morbidity and mortality between colostrum fed and colostrum deprived calves. Several commercial products evaluated have also been shown to provide substantial protection to the neonate. This model may not simulate the cumulative environmental bacterial challenge the colostrum deprived calf faces daily, but the controlled environment in this model minimizes the extraneous variables present in field studies that make direct comparisons difficult.

[1]*SRID Kits, VMRD Inc., Pullman, Washington 99163.*
[2]*API 20E® System, Analytab Products, Sherwood Medical, Plainview, New York 11803.*

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Protective Effects of a Commercial Cheese Whey Derived Immunoglobulin Product[1] Against Septicemic *Escherichia coli* Challenge in Colostrum Deprived Calves

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A major cause of worldwide calf mortality is coliseptic-emia secondary to failure of passive transfer of colostral antibody. Several new products[1,2,3,4] have been marketed in recent years to combat this problem. These products often consist of immunoglobulins derived from cheese whey or colostral whey. Usefulness of some of these products has been challenged due to low total immunoglobulin (Ig) content. The purpose of this study was to use the JL-9 *Escherichia coli* model (described previously) to evaluate a commercial cheese whey-derived product that has been marketed for calves since 1987[1].

Colostrum fed (N = 7) and colostrum deprived calves (N = 10) were fed and processed as described in the previous abstract. Calves receiving the product (N = 6) to be evaluated (Colostrx™) were colostrum-deprived; between 2 and 4 hours of age they received a 454 gram bag of Colostrx™ (containing no less than 24 grams of total bovine IgG) mixed as per package directions. 10^9 virulent *E. coli* in 20 ml of culture medium were administered orally to all three groups of calves between 15 and 18 hours of age. Blood samples were taken prior to product or control feeding and compared to 24 hour and pre-necropsy samples.

Calves were scored twice daily using a modified neonatal sepsis scoring system.

All 10 calves in the colostrum deprived group were dead or moribund by 120 hours of age. None of the colostrum fed or Colostrx™ fed calves experienced disease or developed signs of localization of infection during this time. Pre-feeding blood samples were similar between the groups of calves. Twenty-four hour blood samples were also similar between the three groups of calves, except that percent and absolute numbers of neutrophils were significantly elevated in Colostrx™ fed calves, and colostrum fed calves had significantly higher total serum and plasma protein levels. Terminal blood samples from colostrum deprived calves demonstrated leukopenia, neutropenia, and band cell proliferation; colostrum and Colostrx™ fed calves demonstrated no such tendencies. Fibrinogen levels were highest in Colostrx™ fed calves (120 hour samples).

Pre-feeding mean serum IgG_1 levels for all sampled calves were less than 28 mg/dl. In colostrum deprived calves this value did not change significantly in 24 hours. Mean serum IgG_1 levels 24 hours following first feeding were 1976.2 ± 514.2 mg/dl (colostrum fed calves), and 222.7 ± 75.2 mg/dl (Colostrx™ fed calves).

Adrenal histology was similar between colostrum fed and Colostrx™ fed calves; adrenal hisology from colostrum deprived calves demonstrated varying degrees of hemorrhage and congestion. *E. coli* was isolated from heart blood, liver, joints, or spleen in 1 of 28 collections from colostrum fed calves; 38 of 40 collections in colostrum deprived calves; and 1 of 24 calves receiving Colostrx™. One Colostrx™ fed calf developed septic arthritis of the carpal joint; at necropsy *E. coli* was recovered from the joint.

Although total absorbed Ig mass appeared clinically inadequate to protect colostrum deprived calves, ingestion of Colostrx™ provided protection for hypogammaglobulinemic calves against a single septicemic coliform challenge. Some factor(s) other than absorbed Igs provided by the cheese whey derivative may have been responsible for the protection afforded against coliform challenge 12-15 hours later. It is unknown if this factor exerted a systemic effect or provided local specific enteric immunity against the coliform challenge.

[1] Colostrx™, Protein Technology, Inc. Minneapolis, Minnesota 55415.
[2] CL Replacer, Cuprem, Kenesaw, Nebraska 68956.
[3] Gold Label™, Cuprem, Kenesaw, Nebraska 68956.
[4] ID-1®, Immuno-Dynamics Inc., Perry, Iowa 50220.

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Successful Treatment of Bovine Retained Placenta by Umbilical Cord Injection of Collagenase

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Collagen in the uterus is rapidly degraded during early postpartum by enzyme collagenase. However, during placenta retention there is a persistence of type III collagen in the bovine placentome (Biol Reprod 43:229, 1990; Theriogenology 32:485, 1989). This could be related to placenta retention, since collagen is one of the most dynamic and abundant tissue binding proteins in the pregnant uterus. Isolated placentomes were perfused during two hours or more with blood containing different quantities of bacterial collagenase. This resulted in a significant ($P = 0.05$) loosening of fetal membranes as measured by a manometric technique developed in this laboratory. Collagenolysis caused by collagenase and other proteolytic enzymes was measured by hydroxyproline and nitrogen content. Injection of collagenase into the umbilical cord vessels of cows with placenta retention ($n = 20$) resulted in 80% release of the retained membranes within 24 hours, whereas none of the control cows released retained membranes. No clinical complications were found within a month of treatment. It was concluded that collagenase treatment is affordable, is highly effective, it can be given as soon as the diagnosis is done without losing its effectiveness. A study of the feasibility of this treatment for preventing placenta retention subsequent to a cesarean section is underway.