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.