

# A TWO-YEAR STUDY OF VACCINATION IN THE PREVENTION OF BOVINE *E. COLI* DIARRHEA

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Investigations have demonstrated many causes of diarrhea in calves, including management and infectious agents (34, 73). The infectious agents presently recognized as the causative agents of diarrhea include many viruses such as rotavirus, IBR and BVD. In addition, bacteria including the *Salmonella spp.*, *Clostridium spp.*, and *Escherichia coli* (*E. coli*) have frequently been associated with calf diarrhea (84).

*E. coli* bacteria may cause diarrhea (colibacillosis) or septicemia enterotoxemia (enterotoxigenic *E. coli*) (ETEC) (22, 23, 87). Usually these forms of *E. coli* infection occur because of colostrum deprivation (11, 39, 46,62). *E. coli* organisms originate from the environment and colonize in the intestine after birth. The longer the calves are exposed to a confined environment the greater the chance for contamination and the greater the pathogenicity of the organisms that invade the digestive tract of the calf (68, 69, 71, 85).

Recent investigations indicate that colibacillosis caused by the ETEC organism is due to extensive proliferation of *E. coli* in the small intestine and its ability to adhere to the mucosal surface of the intestinal lining (colonization) (56). Proliferation and colonization of the ETEC on the intestinal mucosa is facilitated genetically through a pilus antigen on the bacterial surface (12, 13, 29, 33, 73). A pilus is an appendage of the bacterial capsule or envelope and is serologically designated as K88, K99, or 987 antigens (32, 34). The K99 pilus antigen is primarily associated with colibacillosis of calves, so it has been postulated that *E. coli* containing the K99 pilus antigen could be utilized as bacterins for vaccination of the dam as a means of preventing colibacillosis of calves during the neonatal period (three weeks following birth) (12, 19, 63, 64). Some strains of ETEC will produce more than one type of pilus, and pili distinct from K99, K88, or 987 are

often present on the non-enteropathogenic *E. coli* (20, 27, 57). Many infectious agents are associated with enteritis and they may act singly or in concert with the ETEC *E. coli* (3, 34). It has also been demonstrated that more than one strain of *E. coli* organisms can be associated with colibacillosis within a single herd and that vaccination with a single strain of *E. coli* will not provide demonstrable protection for the other pathogenic strains (35, 50).

To accomplish maximum prevention of colibacillosis, certain management procedures and immunological aspects must be considered.

First, the majority of antibodies (immunoglobulins — Igs) present in colostrum come from blood through selective absorption by the mammary gland and thus must be at their highest concentration in the blood when colostrum formation begins (14, 15, 51, 67). Colostrum formation begins five to four weeks before calving and reaches its maximum colostrum concentration about five days before calving with maintenance of maximal levels until parturition (5, 7, 8, 10, 40, 41). Immediately following parturition, there is a rapid decline of the Igs in the milk (7). Igs are quantitatively selectively absorbed from the blood into colostrum (6, 82). Colostrum Ig types are the same as those found in the blood (24, 42). IgG1 and IgG2 are the primary immunoglobulins absorbed from the blood into the mammary gland (16, 54, 59). Small quantities of other types of immunoglobulins are secreted in the mammary gland (36, 39, 52).

It is a basic immunological fact that to obtain maximum antibody formation a three-week period must elapse between vaccination and the establishment of **maximum** antibody levels (28, 62). It has also been demonstrated repeatedly that upon initial vaccination a minimum of two vaccinations (initial vaccination and a booster administration of vaccine) must be given if maximum immunity is to be obtained, regardless if a living (attenuated) or killed (inactivated) vaccine is employed (53). To obtain a maximum antibody concentration in colostrum through vaccination, the initial vaccination should be made not later than 15-12 weeks before calving with the second booster vaccination given not less than 12-9 weeks before calving.

Although antibodies are present in the colostrum, they will exist in the calf only if the calf nurses (43, 44,

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76, 86, 89). Newborn calves may be deprived of colostrum protection because they are too weak to nurse, because of poor mothering ability of the dam (particularly in first calf heifers), or because of dams' udders that are too large and pendulous or have large funnel-shaped teats that prevent the calf from nursing (46, 77, 79, 80.) Separating the cow from the calf will also contribute to calves being more susceptible to enteritis (75, 78).

Calves develop some "natural" resistance to colonization of *E. coli* by 36-96 hours of age and demonstrable self-produced antibodies to resident *E. coli* bacteria by four to six weeks of age (81, 83). The latter would indicate active immunity. Passive (colostral) immunity will interfere with the formation of active immunity, so calves should not be vaccinated until they are at least three months of age if an optimum immunological response is to be obtained (30, 78, 81).

The small intestine mucosa will allow unselected immunoglobulin passage into the circulation for approximately the first 24-36 hours following birth (8, 9, 76). No absorption occurs in the rumen, abomasum, or large intestine (17). Most rapid and complete absorption takes place during the first four hours following birth and absorbing ability decreases rapidly thereafter until it ceases at 24-36 hours (38). Blood serum levels of Igs will protect the calf from colisepticemia but is of less benefit in providing protection to colibacillosis than local immunity (47, 49, 62, 65). Intestinal protection can only be provided by local (gut) immunity due to the presence of colostrum containing specific Ig or antibodies for the disease producing organism within the small intestine (38, 45, 47). Local protection was demonstrated as early as 1956 but this information was not published because it contradicted the prevailing concept that protection depended only on antibodies in the circulation (70).

Colibacillosis usually occurs within five days of birth, but outbreaks have been reported in the postcolostral period. During the first three days, the Igs are detected in the greatest quantity in the colostrum, so local immunity in the calf is decreased extensively after the third to the fourth day after birth (55, 72, 75).

There have been numerous demonstrations under controlled laboratory experimental conditions of extensive protection from colibacillosis in calves from vaccinated dams. However, the data has been obtained by challenge with a specific *E. coli* organisms serologically identical to the vaccine organisms. In addition, all calves received colostrum containing antibodies homologous to the antigens of the challenge organism (37, 63). The challenge organism was administered immediately or within several hours following consumption of the specific antibody containing colostrum (1, 2).

Investigations under field conditions with natural exposure utilizing a single serotype of *E. coli* vaccine have indicated minimal benefits from vaccination as a means

of controlling clinical colibacillosis and/or death (25, 48, 62).

Other approaches to vaccination include inactivated or attenuated *E. coli* organisms given orally to dams or calves (7). No protection was demonstrated when given to dams, but some resistance to *E. coli* was reported after oral vaccination of calves (55, 89). A less practical approach has been the intrauterine vaccination of the fetus (18, 88).

## EXPERIMENTAL PROCEDURES

### Experimental Animals

Cattle utilized in this investigation were beef type breeds from herds whose owners volunteered their animals for "on-ranch evaluation" of three *E. coli* bacterins. The investigation was for two calving seasons. First-calf heifers were grouped separately for evaluation. At least two *E. coli* bacterins were used on each ranch. Approximately one-half of the cows and first calf heifers received bacterins as recommended by the manufacturer and one-half of the cows and first calf heifers served as controls (no vaccination). All animals were initially vaccinated twice with the bacterins with a minimum of three weeks between vaccinations and with the last vaccination being administered not less than three weeks before calving. Previously vaccinated animals received a (booster) vaccination at least three weeks previous to parturition.

During this investigation, 2,596 cows and 648 first-calf heifers from nine ranches were utilized during a two-year calving period.

### Determination of Incidence of Diarrhea

Incidence of diarrhea was based on the observation of clinical diarrhea as designated by the owner and/or herdsman.

## RESULTS

### Individual Herd Histories

In an attempt to better assess the efficacy of vaccination with *E. coli* bacterins in relation to varying management procedures, individual herd histories and management practices are presented.

#### Herd B

One hundred fifty-five cows were vaccinated and 138 served as controls with a 2.5 percent incidence of diarrhea in the vaccinates and a 3.5 percent incidence in the controls during the 1980-81 calving season. Weather conditions during the calving season were wet and cold. During the 1981-82 calving season, no clinical diarrhea was observed in either vaccinated or control calves. The weather was dry and sunshine was extensive during this calving season. Most calving was done on a range-type area, but a calving barn was available for inclement

weather. All calving was supervised by the owners only. Clinical calf diarrhea has never been a serious problem on this ranch and it is apparent that vaccination contributed nothing under this type of management.

Evaluation of climatic conditions in relation to the incidence of calf diarrhea indicated that there was a very close relationship between increased incidence of diarrhea and decreased percentage of solar radiation. There was minimal demonstrable relationship to temperature and humidity.

#### Herd F

Previous to becoming involved in this investigation, the incidence of clinical diarrhea averaged over 35 percent each year with a 50 percent mortality on this ranch. Calving was done in a remodeled cow and horse barn and the surrounding yard in which drainage was not optimum. When the preliminary discussions were underway on the cooperative project, it was brought to the attention of the owner that a vacant pasture area, adjacent to the ranch yard, had not been used for cattle. This area was well drained and no cattle had been in the pasture at any time for several years. This pasture was used for calving during the 1980-82 calving seasons. There were no cases of clinical diarrhea in either the vaccinates (337 cows) or control (250 cows) either calving season. Apparently the change in calving area was sufficient to control the enteric problem previously experienced.

Other investigators have provided information that appears to be applicable to herd F. This is that the *E. coli* bacteria within the digestive tract are of two recognized groups, i.e. resident and transient. The resident group consists of one or two strains predominant in numbers and are present for several weeks to months. There are three to four transient strains which are few in number and present for only a few days (26).

Epidemiological studies indicate that *E. coli* diarrhea develops spontaneously and that morbidity and mortality increase as the period of occupation of the calving area is prolonged. It is suggested that this is due to the natural selection of virulent strains of *E. coli* conditioned by the calves' deficiency of antibody to these strains and resulting in the build up of infection (31).

#### Herd H

Based on observations this herd was apparently a well-managed operation that has been constantly confronted with extensive losses from clinical diarrhea. History indicated that when bred cattle were sold from this operation little or no problem was usually encountered with clinical diarrhea by the new owner. A herd of cattle with very similar blood lines and located within five miles of this ranch has had no problem with clinical diarrhea for the past five years. Over the 1980-82 calving seasons the cooperators experienced 15.2 percent incidence of diarrhea in calves of control cows. Controls had a statistically significant decrease in clinical diarrhea from those of the calves of the vac-

inated dams. Observations of general management and of the feeding program provided no indication for changes that would possibly contribute to a decrease in the incidence of the clinical calf diarrhea, other than large amounts of corn silage was fed before and after calving.

Controlled investigations with two groups of 20 cattle per group that originated from this ranch were initiated. It was demonstrated that a lower incidence of diarrhea occurred in calves of cows fed silage for a roughage source than in the group of cows fed hay for a roughage source.

#### Herd D

Calving was done in an excellently drained extensive barnyard area. No clinical diarrhea was detected until the calves were 30 days or more of age. Wheat farming was the owner's primary activity and the seeding of wheat coincided with the 30 days postpartum period in which diarrhea was observed. Antimicrobial medication was useless and calves of vaccinated cows had an incidence of 15 percent diarrhea as compared to 2.3 percent in the controls, indicating a statistically significant lower incidence of diarrhea in the control groups.

#### Summary of All Ranches

Comparison of the incidence of clinical diarrhea in the 1980-81 calving season (Table 1) indicated a slightly better protection for the calves of cows vaccinated with

**Table 1. *E. coli* Bacterin Vaccination for Calf Diarrhea Summary — 1980-82**

Ranches and Experimental Animals	Bacterins Used			Controls
	A	B	C	
TOTAL FOR COWS — 1980-81				
Total Number Cows	471	412	34	366
Number of Calves Having Diarrhea	29	33	2	17
Percent Diarrhea	6.2	8.0	5.9	4.6
TOTAL FOR COWS — 1981-82				
Total Number Cows	427	212	92	582
Number of Calves Having Diarrhea	29	27	6	22
Percent Diarrhea	6.8	12.7	6.5	3.8
COMBINED TOTAL — COWS (2 Calving Seasons)				
Total Number	898	624	126	948
Number of Calves Having Diarrhea	58	59	8	39
Percent Diarrhea	6.5	9.5	6.3	4.1
COMBINED TOTAL — HEIFERS (2 Calving Seasons)				
Total Number	239	130	239	
Number of Calves Having Diarrhea	35	16	15	
Percent Diarrhea	14.6	12.3	6.3	

All bacterins were provided through the courtesy of the manufacturers.

Bacterin A — Vicoten™, Connaught Animal Health, Inc., Lenexa, Kansas 66215

Bacterin B — Coligen™, Fort Dodge Laboratories, Fort Dodge, Iowa 50501

Bacterin C — K99™, Dr. D.H. Hastings, Bismarck, North Dakota 58501

*E. coli* bacterin C as compared to A and D bacterins. The control group, however, had the lowest incidence of clinical diarrhea. Similar results were observed for the 1981-82 calving season.

The combined data for 1980-82 indicated a similar incidence of clinical diarrhea in calves of cows vaccinated with bacterins A and C and a greater incidence in calves of all vaccinated cows as compared to the control groups. The first-calf heifer group received vaccines A and B, and a greater incidence of diarrhea was observed for calves of heifers receiving vaccine A then those receiving vaccine B. The control groups had a lower incidence of clinical diarrhea than those of heifers that were vaccinated with either bacterin A or B.

If those herds in which no clinical diarrhea appeared in either vaccinates or controls are not included on the assumption that they were not exposed to virulent enteric organisms, the problems of clinical calf diarrhea were 8.9 percent, 13.8 percent, 12 percent, and 7.2 percent respectively for bacterin A, B, C, and controls.

Chi-square tests of equal percentages of calves scouring in the treatment and control groups were applied to the data of herds which had incidence of diarrhea. Heifers were kept separate from cows. Two of the eight 1980-81 herd groups showed a significant ( $P < .25$  to  $.025$ ) difference in percentage of calves with diarrhea between treated and control animals. The controls had the lowest percentage of diarrheic calves in both cases. When the data of the eight herd groups were combined, the chi-square test for equal percentage of scours was not significant ( $P < .25$  to  $.5$ ). The 1980-81 overall percentages of scours for bacterin A, B, and C and control were 9.3, 12.6, 5.9, and 8.6, respectively. Three of the five 1981-82 groups showed significance ( $P < .25$  to  $.01$ ) in favor of the controls. When data of the five 1981-82 herds in which diarrhea occurred were combined, the test for equal scours incidence in all four groups was significant ( $P > .01$ ). The percentages of scours were 23.0, 24.1, 20.7 and 10.9 for A, B, C, and control, respectively.

### Discussion and Summary

From these investigations it is apparent that vaccination provided a small part in the control of colibacillosis under ranch conditions. A possible explanation for the greater incidence of clinical diarrhea in the calves of vaccinated cows as compared to control calves could be sensitization of calves to *E. coli* via colostrum of vaccinated cows (55). When or if calves were exposed naturally to *E. coli* of a like serotype, the sensitization could result in clinical diarrhea (60, 66, 90, 91).

Several management aspects appear to make a contribution to colibacillosis control including calving on clean, well-drained ground and avoiding concentration (over population) in the calving area (21, 26, 31). This was demonstrated in herds B and F.

These investigations indicated no demonstrable relationship between consumption of silage by the dam dur-

ing the precalving and postpartum periods and the incidence of colibacillosis.

Though the incidence of colibacillosis has been suggested to be genetically related in some animals, this could not be demonstrated in herd H.

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average seasonal weight gain of the tagged calves showed a weight advantage that ranged from 10-17 pounds per calf on the different farms. The average seasonal cash value advantage per calf for tagged versus untagged herds ranged from \$6.20 to \$10.54 on the different farms.

**Table 15. Burleigh County 1983 Calf Weight Gain Trials. Comparison of trials on 3 separate farms. Cows with 1 insecticide ear tag; calves untagged. The untreated control herd was pastured separately.**

Farm	Season Gains <sup>1</sup> (lbs.)		Advantage (lbs.)	Season Cash (\$) Value Advantage <sup>2</sup>
	Tagged	Untagged		
JH	271 ( 5)	261 ( 5)	10	6.20
MR	248 (33)	231 (37)	17	10.54
SW	302 (10)	288 ( 9)	14	8.68

<sup>1</sup>Average calf weight gain with the number of animals in parentheses.  
<sup>2</sup>Estimated calf market value of \$62.00 cwt.

When separate analyses of variance were run for each farm (Table 15), the differences in weight gains between calves of tagged and untagged animals never quite reached the significance level ( $P=0.05$ ). However, when the analysis was done over all herds and only on steer calves with starting weights as a covariate, significant differences ( $P=0.05$ ) were indicated between the tagged and untagged animals. Based on the adjusted mean weight gains per day, the calves of insecticide ear tagged cows gained an average of 14.7 pounds more than untagged animals over a 150-day trial period. Treatment resulted in a \$9.11 value increase per calf over the untreated animals at a calf market price of \$62.00 cwt.

After subtracting the cost of the one insecticide ear tag applied per cow (ca. \$1.25) and the cost of application, there is still sufficient cash value gain to make insecticide ear tags profitable.

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