Alimentary and Respiratory Tract Lesions in Eight Medically Fragile Holstein Cattle with Bovine Leukocyte Adhesion Deficiency (BLAD)

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Alimentary and Respiratory Tract Lesions in Eight Medically Fragile Holstein Cattle with Bovine Leukocyte Adhesion Deficiency (BLAD)


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Abstract. Lesions in the alimentary tract were studied in eight medically fragile Holstein cattle homozygous for the bovine leukocyte adhesion deficiency (BLAD) allele as determined by polymerase chain reaction and restriction endonuclease analysis. These cattle received institutional medical care but died or were euthanatized because of chronic debilitation associated with diarrhea (6/8) and pneumonia (4/8). The six cattle with diarrhea had acute (n = 3) or chronic (n = 3) intestinal ulcers, but the other two remained relatively healthy for 3 years and did not develop intestinal tract ulcers. Ulcerated areas were present in the small intestine in six animals, and two of these also had ulcers in the large intestine. Ulcers were covered by thick exudates that, in chronic lesions, partially occluded the intestinal lumen. Intramural and serosal fibrosis also contributed to lumen constriction. *Pseudomonas aeruginosa* was isolated from the intestine of four cattle. Bovine viral disease virus and *Salmonella* were not isolated from the five cattle that were tested. Respiratory tract lesions consisted of dense infiltrates of neutrophils in bronchi, bronchioles, and alveoli. This study suggests that intestinal lesions are integral to the demise of BLAD cattle that receive intensive medical care and that neutrophils do infiltrate the lung and enter airway lumina, despite the adhesion deficiency.

Key words: Cattle; CD18; integrins; neutrophils; ulcerative enteritis.
for the CD68 antigen of macrophages using the EBM11 antibody (Dako, Carpinteria, CA). Sections of ileum from animal No. 7 were tested for immunoreactivity to bovine papillomavirus (BPV) antigen with a commercially acquired rabbit anti-BPV-1 antibody (Lot 052A; Dako) according to the manufacturer’s instructions. Sections of papillomatous skin lesions were included as a positive control; primary antibody was excluded in sections of ileum and skin that served as negative controls. For electron microscopy studies, two or three selected areas of small intestine from No. 7 were cut into six 1-mm cubes, immersion fixed in 2.5% glutaraldehyde with 0.1 M cacodylate buffer, and embedded routinely in an acrylic resin. Lesions from the remaining areas of the digestive tract and other tissues were fixed in neutral-buffered formalin and processed for histology as above.

Ulcerated lesions of intestine from calf Nos. 2 and 4–6 and mucosa from animal Nos. 7 and 8 were submitted for microbiological culture (Clinical Veterinary Microbiology Laboratory, ISU, Ames, IA). Spleens from these animals were tested for bovine viral diarrhea virus (BVDV; Dr. S. Bolin, NADC, Ames, IA, and the ISU Veterinary Diagnostic Laboratory, Ames, IA). Calf No. 1 was tested for BVDV (Dr. S. Bolin) and bovine immune deficiency virus (BIV; Dr. M. Van Der Maaten, NADC).

Results

Clinical features

Calf No. 1 developed diarrhea and pneumonia 2 weeks after birth (Tables 1, 2). Treatment included penicillin/dihydrostreptomycin (Pfizer, New York, NY) (2 ml/100 lbs, intramuscularly [IM], once daily [SID]), and oxytetracycline (Beecham Laboratories, Bristol, TN) (3–5 mg/lb, IM, SID), but the animal’s condition deteriorated until it was euthanatized at 46 days of age. Calf No. 2 was produced from a controlled mating of a carrier cow (heterozygous for BLAD defect) with a carrier bull sire. This calf was derived by Caesarean delivery and remained in acceptable health until 2 years of age. Neither reached maturity but were stunted and showed signs of chronic respiratory tract disease. Both reached maturity but were stunted and showed signs of chronic respiratory tract disease.

Despite treatment, the calf had recurring fevers, pneumonia, and diarrhea. It also had marked dermatitis and diarrhea at 10 days of age, although IBR virus was never isolated. This treatment was unsuccessful, and the calf was euthanatized at 36 days of age. Calf No. 4 developed recurrent episodes of diarrhea and pneumonia and was treated with fluacin meglumine (Schering Animal Health) and ceftriaxone sodium (UpJohn) as above. The animal was euthanatized for experimental purposes at 32 days of age. Calf No. 5 was immunized with an experimental vaccine containing killed Cryptosporidia parvum in phosphate-buffered saline at 2 days of age and orally infected with 1 × 10^5 C. parvum at 7 days of age. Oocysts were present in the feces for the next 10 days, but no oocysts were found after this time. The calf developed diarrhea at 10 days of age, and periodic episodes of diarrhea occurred until the calf was euthanatized at 29 days of age. Calf No. 6 was born at ISU’s I-O State Dairy Farm and treated there for stomatitis, diarrhea, and pneumonia with penicillin/dihydrostreptomycin (Pfizer) (6,000 μg/kg, IM, SID) at 65 days of age. After 7 weeks of the antibiotic therapy, the calf was presented to the ISU Veterinary Teaching Hospital with recurrent diarrhea, oral ulcers, pneumonia, and a general small body size. It was unresponsive to treatment with ceftriaxone sodium (UpJohn) (2 mg/kg, IM, SID) and died at 131 days of age. Calf Nos. 1–6 were all <1 year of age when they died (Nos. 1, 6) or were euthanatized (Nos. 2–5). All suffered from some degree of diarrhea, and several were treated for pneumonia (Table 1).

Animal Nos. 7 and 8 had longer lifespans than those above but had recurrent episodes of pneumonia, diarrhea, lameness, joint swelling, and subcutaneous abscesses. Both reached maturity but were stunted and weighed only 420 kg. Animal No. 7 had chronic recurring pneumonia and diarrhea. It also had marked cutaneous papillomatosis and dermatophytosis but remained in acceptable health until 2 years of age. Throughout this time, the animal was treated for periodic episodes of pneumonia with ceftriaxone sodium (UpJohn) (2 mg/kg, IM, BID), fluacin meglumine (Schering Animal Health) (1.1 mg/kg, IM, SID), procaine penicillin G (Beecham Laboratories, Bristol, TN) (6,000 μg/kg, IM, SID), lincomycin HCl (UpJohn) (10 mg/kg, IM, SID), and tilmicosin phosphate (Elanco Product Co., Indianapolis, IN) (10 mg/kg, SQ, SID). Dermatophytic lesions were treated with oral griseofulvin (Schering Animal Health) boluses (10–15 mg/
Table 1. Clinical problems requiring treatment, lesions, and microbiologic features of eight cattle with BLAD.

<table>
<thead>
<tr>
<th>Animal No.</th>
<th>Fate</th>
<th>Age</th>
<th>Major Clinical Problems</th>
<th>Distribution of Intestinal Lesions*</th>
<th>Associated Intestinal Microorganisms</th>
<th>Mucosal Lesion</th>
<th>Serosal Lesion</th>
<th>Vascular Leukocytosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Died</td>
<td>46 days</td>
<td>Diarrhea, stomatitis, pneumonia</td>
<td>SI</td>
<td>ND</td>
<td>Ulcers Yes (fibrinous, early fibrosis)</td>
<td>Yes</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Euth†</td>
<td>61 days</td>
<td>Diarrhea, stomatitis, dermatitis, pneumonia</td>
<td>SI, LI</td>
<td>Pseudomonas aeruginosa, Bacillus sp.‡</td>
<td>Ulcers Yes (fibrinous, early fibrosis)</td>
<td>Yes</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Euth</td>
<td>36 days</td>
<td>Diarrhea, pneumonia</td>
<td>SI</td>
<td>ND</td>
<td>Ulcers Yes (fibrosis)</td>
<td>Yes</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>Euth</td>
<td>32 days</td>
<td>Diarrhea, pneumonia</td>
<td>SI, LI, cecum</td>
<td>Pseudomonas aeruginosa, Enterobacter sp.‡</td>
<td>Ulcers Yes (fibrosis)</td>
<td>Yes</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>Euth</td>
<td>29 days</td>
<td>Diarrhea</td>
<td>SI</td>
<td>E. coli</td>
<td>Ulcers Yes (fibrosis)</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>6</td>
<td>Died</td>
<td>131 days</td>
<td>Pneumonia,§ diarrhea</td>
<td>SI</td>
<td>E. coli, C. perfringens, Pseudomonas aeruginosa‡</td>
<td>Ulcers Yes (fibrinous)</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>7</td>
<td>Euth</td>
<td>3 years</td>
<td>Diarrhea pneumonia,¶ dermatitis</td>
<td>SI (ileum)</td>
<td>Pseudomonas aeruginosa, Enterobacter sp.‡</td>
<td>None No; polypoid thickening, perianal fibrosis, and stricture</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>8</td>
<td>Euth</td>
<td>3 years</td>
<td>Pneumonia</td>
<td>None</td>
<td>None‡</td>
<td>None No</td>
<td>No</td>
<td>No</td>
</tr>
</tbody>
</table>

* SI = small intestine; LI = large intestine.
† Euthanitized.
‡ Tested for BVDV and Salmonella and found negative.
§ Severe acute fibrinopurulent pneumonia involving >50% of the lung. Pseudomonas aeruginosa was isolated.
¶ Severe chronic bronchopneumonia involving roughly 50% of the entire lung. Pseudomonas aeruginosa was isolated.

Table 2. Clinical features* of eight cattle hospitalized with bovine leukocyte adhesion deficiency.

<table>
<thead>
<tr>
<th>Ailment</th>
<th>Animal No.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Oral ulcers</td>
<td>+</td>
</tr>
<tr>
<td>Diarrhea</td>
<td>++</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>+</td>
</tr>
<tr>
<td>Fever of unknown origin</td>
<td>+</td>
</tr>
<tr>
<td>Abscesses</td>
<td>-</td>
</tr>
<tr>
<td>Dermatophytosis</td>
<td>-</td>
</tr>
<tr>
<td>Papillomatosis</td>
<td>-</td>
</tr>
<tr>
<td>Lameness</td>
<td>-</td>
</tr>
<tr>
<td>Prolonged healing</td>
<td>+</td>
</tr>
<tr>
<td>Bloating</td>
<td>-</td>
</tr>
</tbody>
</table>

* Subjective scoring of clinical severity: - = no abnormalities; + = mild; ++ = moderate; +++ = severe.
gas. At the time of euthanasia (3 years of age), the cow was small in stature, had widespread dermatologic lesions, and was very thin.

Animal No. 8 was an intact male, born in Michigan, shipped to the NADC at 4 months of age, and raised for 3 years. During this time, the bull had several minor episodes of pneumonia but reached sexual maturity and served as a source for semen collection. The bull was in comparatively good condition when euthanized at 3 years of age, although he was markedly stunted. This animal also had numerous episodes of fevers and pneumonia, which were treated with ceftiofur sodium (UpJohn) (2 mg/kg, IM, BID), fluxinin meglumine (Schering Animal Health) (1.1 mg/kg, IM, BID), and procaine penicillin G (Beecham Laboratories) (6,000 µg/kg, IM, SID). The bull periodically developed swollen joints, subcutaneous abscesses, an enlarged mandible, dermatophytosis, and papillomatosis. Treatment included fluxinin meglumine (Schering Animal Health) (for joint swelling), surgical drainage of abscesses, and oral and topical antifungal therapies.

Leukograms and temperatures

As typical with BLAD, all cattle developed a persistent and progressive leukocytosis composed predominantly of neutrophils. Fluctuations in the number of circulating neutrophils were common (Fig. 1). Many of the rapid and large increases in neutrophil numbers were associated with elevated temperatures (Fig. 1) and clinical episodes of diarrhea or pneumonia (not shown) that required antibiotic and antipyretic therapy. Therapy was often associated with a sharp decline in both neutrophil numbers and body temperature, although neutrophil numbers never returned to within normal ranges. In the terminal stages of illness, the leukocytosis progressively increased despite treatment.

Microbiologic findings

Pseudomonas sp. was isolated from intestine of four of the cattle (Nos 2, 4, 6, 7) (Table 1). Lesser numbers of other bacteria isolated included Enterobacter sp. and Bacillus sp. Escherichia coli was isolated from calf Nos. 5 and 6. Salmonella sp. and BVDV were not isolated from animal Nos. 2, 5, 6, 7, or 8. Animal No. 1 was negative for BIV and BVDV but was not tested for Salmonella (Table 1). PCR analysis and cultures of intestine did not reveal Mycobacterium paratuberculosis in animal No. 7.

Postmortem findings

Oral lesions. All cattle suffered some degree of oral lesions, predominantly gingival, buccal, and lingual ulcers. Animal No. 7 had chronic ulcers on its chin (Fig. 2). Oral radiographs of animal Nos. 6–8 revealed loss of one or more molars. Remaining molars often protruded at irregular angles and were elongated with pointed ends. The pointed teeth were adjacent to ulcers on the dorsolateral surface of the tongue in animal Nos. 7 and 8 (Fig. 3). Adjacent to many of the elongated molars were deep recessions probably caused by tooth loss or fracture. These invaginations contained large amounts of compacted ingesta. Gingiva of the incisors was recessed in all cattle, and up to 0.4 cm of the roots of the incisors were exposed in animal No. 8.

Microscopically, oral and lingual ulcers had marked subjacent fibrosis with neocapillaries. Few neutrophils were present within these areas, but aggregates of macrophages immunoreactive to EBMI11 (anti-CD68) were present in some cattle.

Intestinal lesions. Intestinal ulcers in the small intestine and/or large intestine were present in animal Nos. 1–6. Several abomasal trichobezoars were present in animal Nos. 2, 4, and 5, but none of the cattle had abomasal ulcers or ulcers within the forestomachs or esophagus. In animal Nos. 1, 3, 4, and 5, there was adherence of serosal surfaces of adjacent areas of small intestine because of marked fibrosis (Fig. 4). Ulcers in
Ulcers were present in small intestine but not in large intestine in animal Nos. 1 and 5. Small and large intestinal ulcers were present in animal Nos. 2 and 6. Small intestine, cecum, and large intestine ulcers were present in animal No. 4 (Fig. 6). The ulcers...
in animal Nos. 1–5 were covered by thick, laminated, and dry fibrinonecrotic exudates. This material partially occluded the intestinal lumen and was often associated with chronic ulcers that had a firm fibrous enteric wall and fibrous serosal surface. These areas were often firm and poorly distendable. As a result, these sites had very small (2 cm) lumenal openings. Microscopically, intestinal ulcers in animal Nos. 1–6 had features similar to those previously described.8,11,13,14,21 Ulcerated surfaces were covered by thick exudates of fibrin, necrotic cell debris, and populations of cocci and filamentous bacteria. Subadjacent to the ulcerated areas were numerous fibroblasts, neocapillaries, lymphocytes, plasma cells, and macrophages.

Blood vessels within fibrous connective tissue subjacent to ulcers and in the adjacent mucosa were moderately dilated and contained moderate to large numbers of neutrophils in animal Nos. 1–4. This vascular leukocytosis was present in the serosal tissue of animal No. 5. The neutrophils often had bandlike nuclei or were only slightly segmented. Anti-CD68 immunohistochemically labeled only a small portion (<5%) of the intravascular cells. In the remaining cattle, there was no obvious increase in intravascular neutrophils. The serosa subjacent to the ulcers was covered by polymerized fibrin containing elongated mesothelial cells and small numbers of macrophages and neutrophils in animal Nos. 1 and 2. The serosa subjacent to ulcers was markedly thickened by dense fibrous connective tissue and neocapillaries in animal Nos. 3–5. No protozoal cysts were seen in sections of small intestine from animal No. 5, which was experimentally infected with Cryptosporidium parvum.

Animal No. 7 lacked intestinal ulcers; however, 0.80 m of ileum had a markedly thickened mucosa containing multiple polypliod projections (Fig. 7). This condition was due to marked proliferation of enterocytes resulting in the formation of numerous irregularly branching crypts and polypliod projections supported by a thin fibrous stalk. At the base of the mucosa–submucosa junction were numerous oval to round and dilated cysts of various sizes that formed small cystlike structures. These cysts often contained only small amounts of cell debris and were largely empty. Throughout the remaining mucosa, there were occasional dilated crypts that contained large numbers of neutrophils and lesser numbers of sloughed cells and cell debris. Special stains did not reveal any bacilli. The lamina propria of the thickened mucosa contained moderate multifocal infiltrates of lymphocytes, plasma cells, and macrophages. Few cells immunoreactive for EBM11 (CD68), the anti-macrophage antibody, were present in the lamina propria. This finding was in marked contrast to the condition in cattle with Johne’s disease, in which infiltrates of macrophages are present in the lamina propria. Moreover, the lesion did not resemble those of Johne’s disease either grossly or histologically. Sections of ileum lacked specific immunoreactivity to BPV-1 antigen. There were no serosal adhesions or proliferative serosal areas.

Because of the unusual histologic features in the mucosa in animal No. 7, samples were examined by electron microscopy. Ileal mucosa from this animal was lined by mature enterocytes and many intervening goblet cells. The lamina propria contained dilated lymphatic vessels (lacteals) and was expanded by a pale, lightly osmiophilic flocculent ground substance. Ultrastructural analysis did not reveal bacteria, inclusion bodies, or virions.

Respiratory lesions. Although all of the cattle at some time had some degree of clinical pneumonia, four cattle (Nos. 1, 2, 6, 7) had pulmonary lesions at the time of death. Animal Nos. 1 and 2 had moderate multifocal cranioventral bronchopneumonia characterized microscopically by moderate multifocal infiltrates of neutrophils in bronchi, bronchioles, and al-
Numerous neutrophils were present in the airway lumina (Figs. 8, 9). These infiltrates also contained modest amounts of fibrin and necrotic cell debris. Animal No. 6 had severe diffuse fibrinopurulent bronchopneumonia involving nearly 80% of the organ. Numerous neutrophils were present in the airway lumina and interlobular septa. Animal No. 7 had severe chronic bronchopneumonia with marked bronchiectasis and peribronchial and peribronchial fibrosis. Neutrophils were present within the airway lumina and the fibrous stroma, along with numerous macrophages and a lesser number of lymphocytes and plasma cells. Pseudomonas aeruginosa was isolated from the lungs of animal Nos. 6 and 7.

Other findings at necropsy included dermatitis (papillomatosis and dermatophytosis) (animal Nos. 1, 2, 4, 7, and 8) and ulcerative dermatitis and vasculitis (animal No. 1).

Discussion

Although cattle with BLAD typically die of respiratory or enteric infections, this study suggests that debilitation and death from enteric disease occurs in cattle receiving intensive antibiotic therapy. The importance of enteric lesions in this group of BLAD cattle is underscored not only by the severity of the lesions but also by the early age of deterioration in health of those six cattle with enteric ulcers in contrast to the relatively long lifespan of the two animals lacking ulcerative lesions. Antibiotic therapy may prevent fulminating pneumonia in young hospitalized cattle with BLAD but does not prevent or effectively treat enteric disease.

The intestinal and oral ulcerations in the six younger cattle may have contributed to debilitation and generalized weakening by at least three possible mechanisms. First, the combination of intramural and extramural fibrosis in association with dry fibrinonecrotic material covering the ulcers led to a very narrow lumen that may have restricted passage of ingesta. The series of chronic ulcerations along the intestinal tract likely exacerbated the reduced flow of ingesta and may also have interfered with intestinal contractions and absorption. In addition, protein lost through these ulcerated foci may lead to decreased serum protein levels (not measured) and protein-losing enteropathy.

Second, chronic exposure of the intestinal submucosa to bacterial pathogens (and lipopolysaccharide) and concomitant release of inflammatory cytokines (interleukin-1, interleukin-6 and tumor necrosis factor) may affect food intake and metabolic function. Although levels of these cytokines were not measured, increases would be expected in these cattle because of the numerous lesions and the absorption of bacterial products, namely lipopolysaccharide.

Third, pain associated with tooth loss, open wounds associated with tooth loss, oral ulcers, gingivitis, and intestinal ulcers may also affect feed intake and mastication. The oral lesions are constantly exposed to the sharp edges of teeth and dry stems of plants. Oral ulcers occur relatively frequently in BLAD cattle. Clinically, loss of molar teeth can go unnoticed and is identified only through careful oral examination at necropsy.

Growth of Pseudomonas aeruginosa in the intestine of several of the animals may have been enhanced by the broad spectrum antimicrobial therapy. Antibiotics used to treat the BLAD cattle included a cephalosporin (ceftiofur), penicillin, and an aminoglycoside (gentamicin). Except for gentamicin, these antibiotics are not effective against Pseudomonas sp. Moreover, resistance of Pseudomonas sp. to aminoglycosides is well documented. It is unlikely that Pseudomonas sp. caused initial development of the ulcers; it probably grew, along with other resistant organisms, under a selective advantage after the ulcers developed. The bacterial overgrowth may have prevented any attempt of mucosal repair. The antibiotic therapy may also have affected the entire enteric microflora by enhancing growth of other types of nonsusceptible or resistant bacteria. The inability of Cryptosporidium parvum to establish prolonged colonization and induce diarrhea in calf No. 5 suggests that this animal formed an appropriate immune response to this pathogen. Vaccination of the animal may have enhanced the protective response.

The thickened ileal mucosa seen in animal No. 7 is unusual and cannot be explained. It lacked features of proliferative enteritis seen in pigs, ferrets, hamsters, and rabbits. In addition, spirochetal pathogens were not seen with special stains or by electron microscopy. It also lacked gross and histologic features of Johne’s disease, and M. paratuberculosis was not identified by acid-fast stains, culture, or PCR analysis. Proliferative intestinal lesions have been reported in cases of cattle with enteric papillomatosis, although ileal sections lacked immunoreactivity to anti-BPV-1 antibody and virions were not seen ultrastructurally. The mature epithelium (enterocytes with abundant cytoplasm and goblet cells) lining the crypts and the paucity of mitotically active cells suggest that the thickening occurred gradually. Also, cytologic features of the enterocytes lacked characteristics of neoplasia (nuclear changes, high mitotic indices, etc.). Enteric ulcers were not present in this animal, which may have been beneficial to its survival into adulthood. Severe, chronic pneumonia involving nearly 50% of the pulmonary parenchyma contributed to the chronic debilitation of this animal more than did the thickened ileal mucosa.

The neutrophilic infiltrates in the lungs of four cattle...
in this study are similar to those reported previously in cattle with BLAD and in humans with severe forms of LAD, type 1. However, the pulmonary infiltrates contrast sharply to the relatively few neutrophils that enter other tissues (e.g., intestine and oral cavity). Pulmonary infiltrates of neutrophils into the lungs of BLAD cattle and LAD children suggest that CD18-independent mechanisms (L-selectin, sialglycoproteins) of adherence mediate neutrophil infiltration in the lung. In animals with normal CD18 expression, L-selectin and its receptor mediate initial adherence of neutrophils to endothelial cells. Upon neutrophil and endothelial activation, stable adherence is mediated between CD18 and receptors such as ICAM 1, 2. But in lung, recent evidence suggests that CD18 mediates neutrophil adherence during acute pulmonary lesions, and CD18-independent molecules mediate adherence during subacute injury. CD18-dependent and-independent adherence in lung are also contingent upon the type of inflammatory stimulus.

Although neutrophils enter the lung parenchyma of cattle with BLAD, antibiotic therapy is probably essential for resolution of respiratory infections. Retrospective clinicopathologic studies of outpatient cattle with BLAD from Japan, the United States, Germany, The Netherlands, and Denmark all report fatalities that are often secondary to pneumonia. Recurring infections in the alimentary and respiratory tracts, mucosal surfaces, and skin in cattle with BLAD demonstrate the importance of neutrophils in immunity. Loss of CD18 expression by leukocytes leads to inadequate passage of these cells into the perivascular interstitial tissue and the overlying epithelium. It appears that without neutrophil entrance into the epithelium, bacterial colonization occurs. Although antibiotic therapy can eliminate certain types of bacterial strains, resistant and nonsusceptible bacteria remain. Those agents can then enter mucosal surfaces and grow within small epithelial defects caused by foreign bodies or trauma. Once this nidus of infection is established, it likely persists because neutrophils from cattle with BLAD have severely impaired adherence, chemotaxis, and iC3b-mediated phagocytosis.

Acknowledgements

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References


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