Epizootic Malignant Catarrhal Fever in Three Bison Herds: Differences from Cattle and Association with Ovine Herpesvirus-2

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Abstract. Three bison herds in Colorado experienced high mortality from malignant catarrhal fever (MCF). In comparison with cattle, the bison had a more rapidly progressive disease, fewer clinical signs, and milder inflammatory histologic lesions. There was consistent association with ovine herpesvirus-2 (OHV-2). Contact with sheep was not consistent. Of 17 animals in herd A, 15 died of acute MCF; 1 was slaughtered while healthy; and 1 developed clinical signs of MCF, was treated with corticosteroids and antibiotics, and died of fungal abomasitis and rhinitis after 5 months. In herds B and C, approximately 300 of 900 and 18 of 20 died of MCF following brief clinical disease. The nearest sheep were 1 mile away from herd A, but direct contact with sheep could be documented in herds B and C. Complete gross and histologic examinations were conducted on 34 animals, including all animals in herd A, and MCF was diagnosed in 31. In addition, field necropsies were performed on all dead animals in herd B and most in herd C and MCF was diagnosed on the basis of the gross lesions in most animals. Clinical signs of each animal in herd A were recorded. Illness was brief, usually 8–48 hours. Clinical signs were subtle; separation from the herd was often observed. In all 3 herds, hemorrhagic cystitis and multifocal ulceration of the alimentary tract were consistently found at necropsy. Mild lymphocytic vasculitis was present in multiple organs. Ovine herpesvirus-2 was found by polymerase chain reaction (PCR) in 71 of 105 formalin-fixed tissue specimens from 29 of 31 animals with MCF. In herd A, blood samples from 13 animals were collected at 5 time points and tested by PCR for the presence of OHV-2 viral sequences in peripheral blood leukocytes. Nine bison with a positive PCR test and 4 with negative results prior to clinical illness died of MCF.

Malignant catarrhal fever (MCF) is an infectious disease characterized by widespread lymphocytic vasculitis and epithelial cell necrosis. Most cases are rapidly progressive. The disease affects a variety of ruminants[12] and has been reported in pigs.[10] Most of the cases of MCF in cattle appear to be sporadic, but herd outbreaks with high mortality have been reported in cattle.[2,12] A previous description of MCF in bison included an outbreak of 7 cases in a herd of 13 animals.[15] Corneal opacity, lacrimation, nasal discharge, anorexia, salivation, diarrhea, melena, depression, and hematuria have been reported in bison.[16]

Classically, two epidemiologic forms of the disease have been described, wildebeest associated and sheep associated. The cause of wildebeest-associated MCF is alcephaline herpesvirus 1 (AHV-1).[13,14] A similar virus, ovine-herpesvirus 2 (OHV-2), has been found in sheep-associated disease in cattle and bison using a polymerase chain reaction (PCR) test to detect OHV-2 genetic sequences.[14,16] Quantitation of OHV-2 viral sequences is possible.[5] In addition, a competitive enzyme-linked immunosorbent assay (c-ELISA) can detect presence of serum antibody to a common epitope shared by MCF-associated herpesviruses.[6,7]

The PCR and c-ELISA tests have shown that the blood of a high percentage of healthy adult sheep are positive for genetic sequences of OHV-2 and antibody to malignant catarrhal fever virus.[7] Although transmission of the virus among sheep has been shown,[8] any mode of transmission of the virus from sheep to cattle or bison or transmission among cattle and bison is not understood.

This paper describes three epizootic outbreaks of MCF in bison. The clinical signs, mortality rates, postmortem lesions, and results of PCR testing for OHV-2 are reported. These findings are compared with those in cattle.

Materials and methods

Bison examinations. Three bison herds (A, B, and C) from which animals were submitted to the Colorado State University Veterinary Diagnostic Laboratory were studied. Descriptions of clinical disease were provided by veterinary practitioners and animal owners. Histologic examination of multiple tissues from 34 bison was conducted at Colorado State University, and duplicate samples of some animals in herd A were examined at the University of Wyoming. Gross necropsies of these 34 animals were performed in the field by veterinary practitioners or in the laboratory by veterinary pathologists. These 34 animals included all 17 animals of herd A, 12 from herd B, and 5 from herd C. In addition,

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complete gross necropsies were performed in the field by veterinary practitioners on approximately 300 animals from herd B and 10 in herd C.

Cases of MCF in cattle. The clinical records, gross necropsy reports, histologic slides, and PCR results from 22 cases of MCF in dairy cows or feedlot cattle seen at Colorado State University were reviewed.

Polymerase chain reaction tests for OHV-2. The PCR for OHV-2 sequences was conducted as previously described on 105 formalin-fixed tissues from 31 of the 34 bison that were examined histologically.[4,16] In herd A, blood samples from 13 animals were tested by PCR for the presence of OHV-2 sequences in peripheral blood leukocytes (PBLs). The blood tests of animals in herd A were conducted at Colorado State University or Washington State University; several were run in duplicate. Blood samples from all surviving animals were collected 41, 107, 357, 434, and 468 days after the date of the first deaths in the herd. The PCR was performed on DNA purified from blood using a commercial kit.[a] The PCR was performed with 5 μl from specimens containing 0.1–0.5 optical absorbance units at 260 nm. All animals were clinically normal when the blood samples were obtained.

Results

Herd histories, clinical and pathologic findings of bison with MCF

Herd A. Sixteen of 17 bison in a herd on the Colorado plains died of MCF in disease outbreaks that occurred in the winters of 1997–1998 and 1998–1999. At the time the outbreak began, there were 15 bison of various ages in the herd, and 2 calves were born the following spring. The herd had no health problems prior to the MCF outbreaks. When the outbreak began, the herd was kept in a 45-acre pasture and were observed carefully at least twice per day. The nearest sheep were 1 mile away.

Assembly of the herd had begun 7 years earlier with animals purchased at different times from several sources. During the 2 years prior to the outbreak, various animals were moved to and from different locations in Colorado and Wyoming. In the spring before the first year's MCF outbreak, a cow was purchased from a herd that had a single case of MCF 1 year previously. Two months before the MCF outbreak, 5 animals from more than 1 source were added to the herd. From July until September before the outbreak, the bison were kept in a pasture across a road from sheep. Then they were moved 20 miles away to a 45-acre pasture that had never housed sheep. After 7 animals died of MCF, the owners of the bison were concerned about possible contamination of their property so they moved the bison to a pasture 12 miles away. At the new location, 2 calves and a young bull died of MCF, and the survivors were moved back to the 45-acre pasture.

The 10 animals that died the first winter included two 6-month-old calves, a 1½-year-old heifer, 4 cows aged 3, 4, 8, and 9 years, 2 bulls 1½ years old, and a 5-year-old bull. The outbreak began in late November and continued to March. Two bison died day 1 of the outbreak, 2 more on day 33, and 1 each on days 59, 61, 64, 70, 85, and 112. The 11th bison was a male calf that died on day 273 in August, but its illness had begun in March. Three cows and 1 bull did not develop MCF the first year, remained healthy, and 2 of the cows had calves the following spring. Five of these 6 animals died of MCF in the second winter, 4 from December to February, and the fifth in May. Two deaths occurred on day 375 and 1 on days 404 and 446 after the first case. The cow that had been purchased before the MCF outbreak was the last animal to die. In April, this cow aborted a near-term fetus that had no lesions, then became anorectic and lost condition for approximately 20 days and developed acute MCF in May and died on day 522. In January of the second outbreak, a healthy bull was slaughtered for meat and multiple organs were collected at the time of butchering, and no gross or histologic lesions of MCF were found. In April, after the first MCF outbreak, a Holstein calf was introduced into the bison herd, and in December before the second outbreak, 2 beef cattle were added. These 3 animals have remained healthy.

Subtle clinical signs were observed from 8 to 48 hours in 14 MCF cases before the animals became recumbent. When animals became recumbent, death followed within a few hours. Separation from the herd and feed refusal were usually the first abnormalities noted in these bison. Other clinical signs included mild serous ocular or nasal discharge and low head carriage. Corneal opacity developed in 2 animals, coughing and diarrhea were each observed in 1 animal. Hematuria was not noted but stranguria was seen in one, although urination was rarely observed carefully. One cow aborted hours before death, another cow 21 days before death. One animal appeared normal in the morning and was found recumbent less than 8 hours later.

Treatment was attempted on 2 bison calves, and these lived 159 and 12 days after the onset of illness. In March of the first outbreak, a 10-month-old male calf developed a unilateral serous ocular discharge followed by bilateral discharge 2 days later, and then discharge appeared to resolve. A clinical diagnosis of MCF was made when corneal opacity in the right eye and a fever of 104.1 F developed 6 days after initial clinical abnormalities. The calf was treated with corticosterioids, a nonsteroidal anti-inflammatory drug, and antibiotics every other day for 18 days, during which time his health was improving. By the time drug treatments were halted, the calf appeared normal except for some cloudiness in the right cornea. Four months after initial illness, the calf developed inter-

mittent diarrhea and weight loss. For 1 month before death, the calf was thin, weak, had a poor appetite, and had corneal opacity in the right eye. Treatment with corticosteroids and antibiotics was resumed but the calf did not respond. Profuse watery diarrhea, intermittent serous nasal discharge, and profound weakness developed, so he was euthanized 159 days after illness began. During the second outbreak a 7-month-old female calf was observed to be depressed, anorexic, and pyrexic, so treatment with antibiotics, corticosteroids, and a nonsteroidal anti-inflammatory drug was initiated that day. This treatment continued until the fever resolved, and corticosteroids were administered at decreasing doses for 12 days, at which time the calf died of MCF.

All 16 animals with clinical signs of MCF were necropsied less than 24 hours after death. Hemorrhage in the urinary bladder was found in 15 bison but only mild erythema and edema in the 16th. Ulceration in various parts of the alimentary tract was found in all animals, with the most common pattern being involvement of the oral cavity and abomasum, with variable involvement of the esophagus. Intestines had congestion, hemorrhages, fibrinous exudate on the mucosal surface, or ulceration. Corneal opacity was seen in 2 animals. Slightly enlarged lymph nodes were observed grossly in 8 animals. The principle histologic lesion was mild vasculitis with lymphocytes in the adventia and media of vessels in multiple organs, particularly the carotid rete and brain, liver, and kidney. Alimentary tract tissues had mucosal ulcers and mixed mononuclear inflammatory cells and neutrophils in adjacent mucosa and submucosa. Not all ulcerated tissues had distinct vasculitis. Most lungs were congested and edematous, but only 3 had vasculitis. Fibrinoid vascular necrosis was seen in only 2 animals. Lymph nodes of 8 animals had mild hyperplasia, 2 had lymphoid depletion, and the other 6 were unremarkable. The calf that was treated and survived 159 days had serous atrophy, healed ulcers in the oral cavity, severe ulcerative abomasitis, intrahepatic venous thrombosis, and mycotic-bacterial ulcerative rhinitis. Vasculitis and obliterative arteriopathy expected in chronic MCF[11] were not found.

Herd B. Approximately 900 bison were kept on winter pasture in the Colorado Plateau. Mouflon sheep were present on the same land. The herd lost approximately 300 animals from late November 1990 to April 1991. The sheep were removed from the bison herd as soon as MCF was diagnosed, but bison deaths continued. Most animals were found dead. Necropsies were performed on all animals that died; 12 were performed at Colorado State Veterinary Diagnostic Laboratory and the others by the local practitioner. Animals examined in the laboratory were adults of 1 to 4 years of age, both sexes. Hemorrhagic cystitis and alimentary track ulcers, primarily in oral cavity and intestines, were present in most animals. Of the 12 animals examined in the laboratory, 11 had gross and histologic lesions of MCF. Histologic lesions included mild lymphocytic vasculitis in a variety of organs, including meninges, carotid rete, liver, and kidney. Lymphoid tissues had hyperplasia, depletion, or no lesions.

Herd C. This herd of 20 bison was pastured on the Colorado plains. The herd had direct contact with sheep. Eighteen animals died from November 1990 to March 1991; the 15 that were necropsied were diagnosed as MCF cases. The owner assumed the other 3 deaths were due to MCF but did not have the animals necropsied. The owner reported that 1 survivor died in the fall of 1991 with a clinical diagnosis of MCF and the last animal died in winter of 1997, with MCF diagnosed by gross necropsy, but records of these 2 animals were not available. Hemorrhagic cystitis and alimentary track ulcers were found in the 15 animals that had gross necropsies. Lymphocytic vasculitis was found in multiple organs of the 5 that had histologic examinations.

Historical findings in dairy cattle with MCF

The records of the 22 dairy or feedlot cattle indicate that their average illness was 2–4 days duration. Corneal opacity and lymph node enlargement were seen consistently. Alimentary tract ulceration was seen consistently, but hemorrhage colitis was not a feature. The urinary bladder usually had erythema or focal erosions or ulcers but not extensive hemorrhage. The histologic lesions in cattle included severe lymphocytic vasculitis in many organs in all cases and fibrinoid vascular necrosis in half the cases. Herd morbidity was low and sheep contact was variable.

OHV-2 assays

The OHV-2 sequences were identified by PCR in at least 1 formalin-fixed tissue from 27 of 29 animals with MCF. One MCF animal from herd B and 1 from herd C were negative. A positive PCR reaction occurred in lung of 10 of 11 bison, liver of 18 of 22 bison, kidney of 13 of 22, spleen of 8 of 9, lymph node of 9 of 14, abomasal ulcers of 3 of 3, intestinal ulcers of 2 of 3, oral ulcers of 2 of 2, carotid rete of 2 of 3, and urinary bladder of 2 of 6 bison. The OHV-2 was not found in the healthy bull from herd A that was slaughtered during the disease outbreak or the animal from herd B that did not have MCF lesions (Table 1).

In herd A, 10 of 26 PCR tests on PBLs were positive for OHV-2 sequences. The 7 animals tested at more than 1 time point had variable results with 4 converting from negative to positive during the disease

Table 1. Results of PCR testing for OHV-2 on formalin-fixed tissues collected at necropsy from 31 bison from 3 herds, 29 animals with MCF and 2 without. OHV-2 was found in at least 1 tissue of 27 of 29 bison with MCF.

Animal	Liver	Kidney	Node	Lung	Spleen	Other*
A1	+	+			−	
A2		+	+	−	+	
A3	+	+				
A4		−	−		+	
A5		−	+	+		colon −
A6	+	+		+	+	abo +/ub −
A7				+	+	ub +/rete −/oral +
A8						ub −/rete +
A9	+			+		abo +/rete +/ub −
A10	+	+	+	+	+	ub −
A11	−		+	+		colon +
A12				+	+	abo +
A13†	−	−	−			
A14	+	+				
A15	+	+		+		colon +
A16	+	+	+	+	+	oral +, ub +, eso +
A17	+	+		+	+	
B1	+	−				
B2†	−	−	−			
B3	+	+	+			
B4	+	+				rete −
B5	+					
B6	−	+				
B7‡	−	−	−	−	−	rete −
B8			+			
B9	+	−	−			
B10	+	−	−			
B11	+	+	+			
C1	+	−				abo +
C2‡	−	−	−	−		
C36	+	+	+			
Total + in MCF cases	18/22	14/22	9/14	10/11	8/9	10/17

* abo = abomasum; ub = urinary bladder; rete = carotid rete; oral = oral mucosa; eso = esophagus.
† Bison did not have MCF.
‡ Bison had MCF lesions but negative PCR results.

outbreak, 1 converting from positive to negative, and 2 staying positive. Time elapsed between an animal's first positive PCR test and MCF death ranged from 12 to 363 days. The 4 bison with negative PCR results were sampled while healthy at 5, 23, 29, or 44 days before MCF death. The slaughtered bull that did not have MCF was negative at 3 time points (Table 2). The blood of the healthy Holstein calf remained negative for OHV-2 for 1 year after his introduction to the bison herd, and the 2 beef cattle had negative blood PCR results 2 months after introduction to the bison herd.

Discussion

These outbreaks confirm that MCF can occur in epizootic form in bison herds. The disease was diagnosed on the basis of gross and histologic lesions plus PCR identification of OHV-2 in tissues. Clinical course of disease was more rapid than in cattle. Although many clinical signs have been described in bison,[15,16] very few clinical signs were observed in the carefully monitored animals of herd A. Ocular or nasal discharge was observed in only 4 of the 16 cases, and corneal opacity was noted in only 2 of these. In contrast, corneal opacity is seen in most cases of MCF in cattle. Although hemorrhagic cystitis was found in most bison at necropsy, hematuria had not been observed clinically but would have been difficult to detect. The depression, withdrawal from the herd, and anorexia that commonly occurred are not specific for MCF, so it does not seem possible to rely on clinical signs to make a diagnosis of MCF in bison.

The postmortem lesions in the bison MCF cases were consistent with those described previously.[16] However, lesions in these bison differed slightly from those in cattle examined at Colorado State University. Lymphadenomegaly is found in the majority of affected cattle, but it was noted grossly in less than half the bison. Histologic features in bison lymph nodes included lymphoid hyperplasia, depletion, or no le-

Table 2. Results of PCR testing for OHV-2 on blood samples collected from 13 bison in herd A. Samples were collected at 5 time points after the first death during the MCF outbreak. Four bison in this herd died before this testing was concluded.

Animal	PCR					Days between first positive test and death*
	Day 41	Day 107	Day 357	Day 434	Day 468	
5	+					18
6	+					20
7	−					NA
8	−					NA
9	−					NA
10	−	−				NA
11	−	+				166
12	−	−	+			18
13†	−	−	−			NA
14	+	−	−			363
15‡			+			19
16‡			−	+		12
17	−	−	+	+	+	165

* NA = not applicable, no positive tests.
† Healthy bull slaughtered for meat did not have MCF.
‡ Calves born in the spring after the first winter outbreak of MCF.

sions, while hyperplasia occurs regularly in cattle. Hemorrhagic cystitis is supportive of a postmortem diagnosis of MCF since it occurs frequently in MCF and rarely in other conditions. The hemorrhage was usually more extensive in bison than in cattle. Also, extensive hemorrhage in colon was common in bison but not in cattle. The histologic lesions in these bison generally were more subtle than those found in cattle. In particular, the number of lymphocytes present in vascular and epithelial lesions is generally lower than in cattle. Fibrinoid vascular necrosis was rarely present in bison but was present in half of the cattle cases. Subtle inflammatory lesions that might be considered nondiagnostic in cattle appear to be important in bison.

The value of the therapies administered to 2 bison in herd A is uncertain. The calf with the 159-day illness died of ulcerative abomasitis and fungal rhinitis with systemic dispersion of fungi. There was no vasculitis, and MCF could not be diagnosed based on necropsy findings, but tissues were positive for OHV-2. The corticosteroids administered long term in high doses may have caused immunosuppresion and contributed to death from infectious agents; the drugs may also have suppressed host response to the causative agent, leading to lack of MCF-associated lesions at the time of death. Thus, it is possible that this calf recovered from MCF but died because of secondary fungal infections from drug-induced immunosuppression. The calf that died after a 12-day illness was treated less intensively and lived longer than untreated animals but still died of MCF, so the value of treatment is questionable.

Since these 3 outbreaks occurred in winter, it is possible that stress from the cold or another climatic factor contributed to the development of disease. There may have been more congregation of bison at feed or water thoughs or changes in exposure to or survival of the causative agent in the environment. These winter months do not coincide with lambing season, so there does not appear to be similarity to the occurrence of wildebeest-associated MCF in cattle at the time of wildebeest calving. The lack of direct sheep contact in herd A and the continuation of bison cases after cessation of sheep contact in herd B raise questions about how the disease could spread within the bison herds without the sheep contact that has been thought to be necessary. It is possible that the adult bison in herd A acquired OHV-2 from sheep at some time months to years before the disease outbreak, but the calves in the herd never had any direct contact with sheep. Bison-to-bison spread of disease may have occurred. The pattern of MCF cases in herd A is consistent with a propagated epidemic with an incubation period of approximately 33 days. The incubation period is similar to the 25-day incubation period in previous experimental transmission of MCF to a bison.[9] Further studies will be needed to answer questions about reservoirs of OHV-2 and transmission of MCF.

Sequences of OHV-2 can be readily demonstrated in tissues from bison with gross and histologic lesions of MCF, and in this study, were found in 27 of 29 animals examined. This agrees with results reported previously for cattle[4] and bison.[16] Except for the treated calf in herd A, all the animals that had OHV-2 sequences in tissues collected at postmortem had histologic lesions consistent with MCF. Also, of the 13 animals that were tested by PCR for OHV-2 sequences in blood, all 10 with a positive test died of MCF. While not proving that this is the agent of the disease, these findings strengthen the association of OHV-2 with MCF.

The gross lesions in the alimentary track of animals with MCF are similar to those of mucosal disease in cattle. In the past, the laboratory routinely attempted to identify bovine viral diarrhea (BVD) virus in cattle and bison with alimentary ulcers but did not find the virus in cases that are MCF disease.[3] BVD has not been found in any adult bison submitted to the laboratory for any reason. Some bison in these outbreaks were tested for BVD and found to be negative. As the laboratory gained more knowledge about the MCF and bison, routine testing for BVD was stopped.

The presence or absence of OHV-2 sequences in blood taken from clinically normal bison did not predict when or whether clinical MCF would develop in

an individual animal. Animals with positive results remained healthy for up to 363 days, and 4 animals with negative results died of MCF 5–44 days after testing. The number of days between testing blood and MCF deaths was variable, so it is not possible to conclude whether or not blood tests would have been more predictive if performed closer to the time of clinical illness. The change from a negative test to positive could be explained by acquisition of a new infection, reactivation of a latent infection, or increase in the amount of virus from a previously undetectable level. Change from positive to negative could indicate an infection has become latent or viral load too low to be detected. Although results of a blood test of a single animal are difficult to interpret, positive blood tests do indicate that the virus is present in the herd. Positive blood PCR tests have not been found in herds that have no history of disease (J. Collins, in press JVDI).[3] It is not known if positive animals are carriers of OHV-2 that could spread infections and subsequent clinical disease to other members of the herd. Prospective longitudinal studies are needed to define the meaning of blood PCR tests in clinically healthy bison.

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Sources and manufacturers

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