Bison Diseases Field Guide

John Berezowski DVM
David Hunter DVM
David Love DVM
Patrick Toomey BSc
Murray Woodbury DVM

Published by

The National Bison Association
8690 Wolff Ct., Suite 200
Westminster, CO 80031
(office) 303.292.2833 (cell) 303.594.4420
https://bisoncentral.com/

Front and back cover photo courtesy of Patrick Toomey and the Intertribal Buffalo Council.

Table of Contents

Table of Contents (con't)

Foreword

For millennia, 30 to 60 million bison roamed the Great Plains and Western North America. European settlement brought domesticated livestock that replaced the large herds of bison, and in less than 150 years the entire bison population dropped below 1,000 animals. Along with other pressures, bison were exposed to pathogens brought by European and African livestock species.

In 2017, private bison ranchers, tribal leaders, First Nations communities, and conservationists launched an ambitious commitment to restore one million bison to North America over the next two decades. This campaign, known as Bison 1 Million, would more than double the population of bison grazing across the United States, Canada, and northern Mexico.

The first step in restoration is protecting the health of existing bison herds. Those that manage bison herds operate without access to many of the resources readily available to mainstream livestock commodity species. Also, the pathogens affecting bison health often differ in their expression from those experienced by cattle, thus limiting the ability of bison producers to adapt the interventions and treatments proven effective in beef herds. One goal of bison managers is to understand what disease issues are affecting bison and how to better manage away from various health problems.

The undomesticated nature of bison compounds that challenge. The prey/predator instinct is deeply ingrained in our animals. As a prey species, bison have evolved to hide any outward symptoms that would tip off predators that an animal is weak or suffering. That's why a common phrase in the bison business is, "A sick bison is usually a dead bison."

For more than two decades, a dedicated group of veterinarians and university researchers in the United States and Canada have been working to better understand the physiology of bison, and to develop new resources that will enable ranchers to protect the health of our herds. This understanding has been critical for the "herd health" programs needed to manage disease in bison. One critical and vital aspect is that ranchers have access to information the moment they come across an ill—or a dead—animal in our herds.

That's where this handbook comes in. This guide is an in-the-field resource that can be used to help diagnose potential health issues, and suggest possible remedies that will protect the overall health of our herds.

This book contains information compiled from many sources. The format is adapted from an earlier version based on a literature review written by veterinarian John Berezowski and published by Dr David Love and the InterTribal Buffalo Council. The National Bison Association Science and Research Committee, and in particular veterinarians Murray Woodbury and Dave Hunter, meticulously edited and updated the information, and added photos to increase the usability of this handbook. The National Bison Association and Canadian Bison Association thank them for their hard work.

All of us—whether private ranchers, tribal producers or public land managers—are stewards of these magnificent animals. This handbook is a tool to help us better fulfill that responsibility.

- Dave Carter, Executive Director, the National Bison Association
- Terry Kremeniuk, Executive Director, Canadian Bison Association

Note: The National Bison Association and National Buffalo Foundation have compiled an extensive library of resources on bison herd health and production. Summaries of all publications in this library can be found at: https://www.zotero.org/groups/2186447/bison_research/items. NBA members have full access to the contents of those publications database by logging into the Members' Section of www.bisoncentral.com and joining a similar Zotero group named "Bison publications."

Diseases of the Digestive System

Calf Scours

Causes:
- Viruses - mainly rotavirus, coronavirus.
- Bacteria - *E. coli*, Salmonella.
- Parasites - coccidia, giardia, cryptosporidium.
- Stress factors - overcrowding, accumulation of feces, extreme weather conditions, poor nutrition of the dam, inadequate consumption of colostrum, are thought to predispose calves to disease.
- Multiple causes, often depending on age of animal, management practices, and previous ranch history.

Transmission:
- Ingestion of some of the above organisms.

Clinical Signs:
- Watery diarrhea (scours) - often foul smelling, and can be white, yellow, grey, or blood-stained.
- Animals become dehydrated, weak, and depressed due to energy depletion, water loss, and electrolyte abnormalities.
- Intestinal lining can be damaged in some cases and lead to septicemia and staggering and recumbent prior to death in severe cases.
- Bison calves hide their clinical signs well until disease is severe. Observing the perineal area of bison calves (and adults) as well as bedding can help identify diarrhea cases.

Diagnosis:
- Fecal examination for parasites and parasite eggs, fecal culture, and fecal virus isolation are used to determine the exact cause of scours.
- Postmortem findings - dehydration, emaciation, and fluid feces in the intestinal tract.

Treatment:
- Oral or intravenous replacement of the fluid and electrolytes - correct electrolyte and acid-base imbalances - supportive care is essential.

Treatment con't:
- Use antibiotics, anti-inflammatory medication, and dewormers based on the exact cause. Do lab tests to find out what is the cause.
- Consider hand rearing the calf - bottle feed bison calves with lamb milk replacer, or by using nanny goats as surrogate mothers. (make sure goat surrogate is free of Johne's disease).

Prevention:
- Ensure calf receives colostrum within 8-12 hours of birth, if and when possible, to help establish a competent immune system.
- Keep stress levels low by avoid overcrowding, excessive handling, and early weaning.
- Avoid abrupt changes in feed - when finishing or changing diet, ease into new feed over three or four weeks to allow for the animals to adjust and avoid gastrointestinal disruption.
- Clean feeding equipment after each use.
- Consider environmental factors that may be significant contributors to the development of the disease - wet environmental conditions, overcrowding and poor nutrition of the dam.

Johne's Disease

Causes:

- Bacteria - *Mycobacterium avium subspecies paratuberculosis* (MAP).

Transmission:

- Ingestion of feed or water contaminated by the bacterium.
- MAP bacteria are shed in feces and milk.
- MAP bacteria can cross the placenta and infect the fetus.
- Clinically affected bison shed MAP organisms in feces and these bacteria can persist in feces on pasture for a year or more.
- Infection usually occurs early in life (generally <6 mos old). Average age of onset of clinical disease is about 8 yrs (range 4-12 yrs).
- Progression of clinical disease is rapid in bison (approx.6 mos).
- Bison older than 1 yr are generally resistant to MAP infections.

Clinical signs:

- The bacteria primarily affects the immune tissue in the intestines causing inflammation and thickening of the intestinal wall, preventing nutrient absorption.
- The most common clinical signs are chronic diarrhea and loss of body weight leading to emaciation.
- Bison may take up to a year to die.

Diagnosis:

- The clinical signs of chronic diarrhea and emaciation are not specific to Johne's disease.
- PCR and serology tests are available at https://johnes.org/testserv/forms/submission.PDF .
- Fecal cultures are the most reliable diagnostic tests - samples must be taken from a number of individuals in the herd and must be repeated several times.
- There is a high probability of identifying an infected herd but it is not possible to consistently diagnose the presence of the bacterium in individual animals.
- The only reliable method of establishing a diagnosis in bison is postmortem examination.

Treatment:

- Generally considered untreatable. Clinically sick bison will inevitably die.

Control:

- Difficult to control in a herd - there is no reliable test to determine if an individual animal is infected, making it difficult to identify and remove infected animals before they develop clinical signs with shedding.
- Fecal PCR testing will work in late stages of disease.
- Fecal cultures of all animals every 6 months and culling of infected animals and their offspring has reduced, but not completely eradicated the disease from cattle herds.
- As a last possible option, eradicate the disease by depopulating the herd and then repopulating from clean stock but pastures must also be decontaminated.
- Purchase animals from known Johne's-free herds, or place new animals in strict quarantine and perform fecal culture/PCR tests repeatedly to increase likelihood of identifying positive individuals.

Above: Johne's disease in a bison bull showing extreme weight loss,

Diseases of the Respiratory Tract

(Bovine) Respiratory Disease Complex (Shipping Fever)

Causes:

- This is a multifactorial disease syndrome caused by low-grade viral respiratory infection, which (combined with stress) lowers the immune system, allowing a severe bacterial infection to develop.
- Viruses - Bovine herpesvirus-1, bovine respiratory syncytial virus (BRSV), parainfluenza-3 (PI-3).
- Bacteria - *Pasturella multocida*, *Histophilus somni, Mannheimia haemolytica*, *Mycoplasma bovis.*
- Stress: Social, environmental, nutritional, transport, parasites.

***Transmission*:**

- Aerosol spread from coughing is the major mode of transmission.
- Oral or nasal contact with contaminated surfaces and objects.
- Under intensive management conditions disease occurrence involves a complex interaction of infectious agents, management associated stressors, and environment associated stressors.

Clinical Signs:

- Depression, inappetence, fever, and dullness are the most common signs.
- Rapid shallow breathing, coughing, and social isolation.
- Nasal and ocular discharge can be seen in more advanced cases.
- Death due to infection occurs more often in calves and compromised or weak adults.

Diagnosis:

- Generally diagnosed by symptoms in combination with a recent history of stressful event (shipping, handling, weaning, etc.).
- Bacterial culture and virus isolation from nasal or ocular discharge can identify organisms.

Treatment:

- Broad-spectrum, long-acting antibiotics that concentrate in the respiratory system can be used to treat bacterial infection. Supportive care including high quality nutrition and adequate clean water.
- Critical to reduce stress as much as possible.
- Careful surveillance of bison calves at risk with minimal intervention and handling is critical to reduce mortalities.

Prevention:

- Habituate bison herds to handling areas and chutes to decrease stress during processing.
- Avoid overcrowded pens, especially when weaning bison calves.
- Use appropriate deworming protocols to minimize immune system suppression from parasites.
- Quarantine new animals (especially younger animals) after shipping to see if symptoms appear.
- Use multivalent respiratory vaccines available for domestic cattle.
- Purchase backgrounded (pre-vaccinated, wormed) calves.

Infectious Bovine Rhinotracheitis (IBR)

Causes:

- Virus - Bovine herpesvirus-1.
- Stress - Social, environmental, nutritional, parasites.
- Co-infection with other respiratory pathogens may occur.

Transmission:

- Aerosol spread from coughing is the major mode of transmission.
- Nasal exudate, semen, and fetal fluids may contain the virus.
- A serologic survey of bison from Yellowstone National Park in Wyoming demonstrated antibodies in 31% of bison tested.

Clinical signs:

- The clinical signs of IBR in bison have not been described but it's something to consider.
- In cattle: fever, anorexia, reddening of the nasal mucosa, ocular discharge, nasal discharge, rapid breathing, and coughing. Some animals may develop reddened eyes that may be mistaken for pinkeye.
- In adult cattle, abortion is a common sequel of the disease, occurring up to 90 days after infection.

Postmortem findings:

- Postmortem findings include lesions in the nasal cavities, pharynx, larynx, trachea, and on the muzzle.

Diagnosis:

- Based on clinical signs.
- Virus can be isolated in swabs taken from nasal and ocular exudate.
- Serological testing may be done to detect rising titers to IBR virus - two blood samples must be taken 2 to 3 weeks apart.
- Most commonly the diagnosis is made on postmortem examination of aborted fetuses and submission of samples or the whole fetus to a pathology laboratory for histopathology and virus isolation.

Treatment:

- Protocols have not been reported for treating IBR in bison.
- Antibiotics do not work against viral infections.
- Infected bison should be separated from non-infected bison.
- Vaccinating in the face of an outbreak may reduce the spread of the disease in the herd.

Control:

- In Canada and the United States there are many commercially available IBR vaccines - none of them have been approved for use in bison.
- Most IBR vaccines provide good protection against the occurrence of IBR in cattle, but their efficacy in bison has not been established.
- Killed virus vaccines should be repeated in 3 weeks and then boosted annually.
- Modified live virus IBR vaccine given intramuscularly may provide adequate protection to bison but refrain from using a modified live IBR vaccines on recently weaned calves.
- Modified live virus IBR intranasal vaccine provides protection to cattle and presumably bison but they are difficult to administer properly to bison.

Parainfluenza 3 Virus (PI3)

Causes:
- Virus - Parainfluenza-3 virus.
- Co-infection with other respiratory pathogens may occur.

Transmission:
- Aerosol spread from coughing is the major mode of transmission.
- A serology survey in Yellowstone National Park showed 36% of all bison tested to have antibody titers to PI3 virus.

Clinical signs:
- There has been no known disease, or reduced productivity associated with PI3 infection in bison.

Diagnosis:
- Submit blood samples to a diagnostic pathology laboratory for serology tests.

Treatment:
- Since there has been no disease associated with PI3 infection in bison, there is no treatment required.

Control:
- Vaccination is unnecessary.

Bovine Respiratory Syncytial Virus (BRSV)

Causes:
- Virus - Bovine respiratory syncytial virus.
- Co-infection with other respiratory pathogens may occur.

Transmission:
- Aerosol spread from coughing is the major mode of transmission.
- Oral or nasal contact with contaminated surfaces and objects.

Clinical signs:
- There has been no known disease, or reduced productivity associated with BRSV infection in bison.

Diagnosis:
- Submit blood samples to a diagnostic pathology laboratory for serology tests.

Treatment:
- Since there has been no disease associated with BRSV infection in bison, there is no treatment required.

Control:
- Vaccination is unnecessary.

Mannheimia hemolytica (formerly *Pasteurella hemolytica*)

Causes:

- Bacteria - *Mannheimia hemolytica.*
- *M. hemolytica* has been found in the tonsils and upper respiratory tract of clinically normal bison.
- Stress - Transport, weaning, mixing, overcrowding, starvation, water deprivation, and handling.
- Co-infection with other respiratory pathogens may be important.

Transmission:

- Contact with animals infected with *M. hemolytica.*
- Oral or nasal contact with contaminated surfaces and objects.
- Aerosol spread from coughing is the major mode of transmission.

Clinical signs:

- Increased respiratory rate, cough, nasal discharge, depression, anorexia, and weight loss.
- Pneumonia can be a frequent cause of sudden death in bison.

Diagnosis:

- Diagnosis of pneumonia is usually based on clinical signs.
- Signs such as anorexia, depression and an increase in respiratory rate may not be easily observed in bison with pneumonia. Anorexic bison calves will often stand alongside of their pen mates with their heads in grain or hay feeders and mimic eating.
- Most often the diagnosis of Mannheimia pneumonia is made by postmortem examination and submission of samples to a diagnostic pathology laboratory for bacterial culture.
- The pathological changes associated with *M. hemolytica* pneumonia in bison can be described as fibrinopurulent bronchopneumonia, with fibrinous pleuritis and pericarditis.

Treatment:

- Many broad-spectrum antibiotics have been used to treat individuals and to mass medicate pens.
- If possible, long acting, antibiotic preparations should be used.
- Careful surveillance of bison calves at risk with little or no intervention is critical to minimizing the mortalities caused by pneumonia.
- Sick calves should be carefully removed from the group, treated, and then returned to the group.

Above: Typical “marbled” appearance on the cut surface of a lung affected with *Mannheimia hemolytica*, giving the lung the appearance of marble stone. The lower (ventral) portions of the lungs are firm, red, and inflamed.

Mycoplasma bovis Associated Disease

Mycoplasma bovis causes severe respiratory disease but is also associated with arthritis and reproductive disease in bison.

Causes:

- Bacteria - *Mycoplasma bovis* .
- It is primary pathogen in bison; it does not need other contributory organisms to cause disease.
- Stress - Environmental, social, nutritional as well as management stress (transport, weaning, overcrowding, handling).

Transmission:

- Contact with animals infected with *M. bovis.*
- Oral or nasal contact with contaminated surfaces and objects.
- Aerosol spread from coughing is the major mode of transmission.

Clinical signs:

- The first sign of illness is often weight loss and a reluctance to move or intolerance to exercise.
- It can take 7-14 days after infection before clinical signs are evident.
- A low grade fever, mild depression and runny eyes are often observed in but most of these signs are often too subtle to be of any use in bison.
- Mycoplasmas do not produce toxins like other pneumonia-causing pathogens so affected animals remain alert and usually eat with their cohorts.
- Coughing and an increased respiratory rate.
- Arthritis appears to be an inconsistent feature of mycoplasma disease in bison.
- *M. bovis* may cause abortion and infertility in bison.

Diagnosis:

- *M. bovis* organisms can be cultured from upper airways in live animals or from post mortem specimens.
- Immunohistochemistry (IHC) can be used to identify *M. bovis* organisms associated with lesions from post mortem specimens.
- ELISA can detect *M. bovis*-specific antibodies in serum.
- Polymerase chain reaction (PCR) testing is valuable in identifying *M. bovis* in tissues from clinical cases and can also identify various strains or types.

Diagnosis con't:

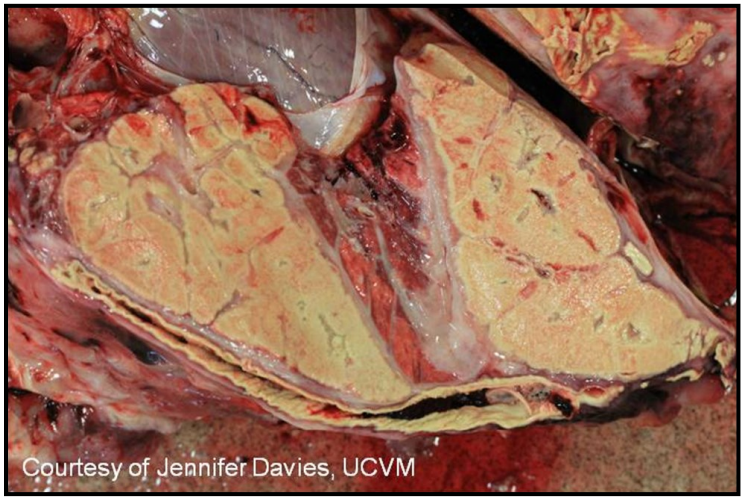
- On necropsy there are typical lesions associated with *M bovis* infection: severe, sometimes unilateral, fibrino-necrotizing pneumonia, sometimes pleuropneumonia with prominent pulmonary sequestra formation.
- Arthritic joints contain fibrinous or cheesy exudates and the surrounding tendons, synovial sheaths, connective tissue, and muscle contain foci of cheesy necrosis and extensive fibrosis.

Treatment:

- Mycoplasma organisms do not have a cell wall, so antimicrobial drugs whose action is directed at the bacterial cell wall are not effective (eg. penicillins or cephalosporins).
- Tulathromycin (Draxxin) is labelled for use in cattle *M bovis* infections and probably works in early infections but the nature of *M bovis* lesions will prevent antimicrobial drugs from reaching the bacteria in later stages.
- Treatment of *Mycoplasma bovis* pneumonia is not rewarding and is therefore not warranted.

Control:

- Commercial vaccines are unlikely to be effective.
- Autogenous bacterins have been used in bison but their effectiveness remains unproven



Above: Cut surface of lung shows lung tissue has been replaced with dead cells and pus. There is lots of fluid and fibrin covering the lungs and heart.

Bovine Tuberculosis (bTb)

Causes:

- Bacteria - *Mycobacterium bovis.*
- *Mycobacterium bovis* is NOT the same as *Mycoplasma bovis,* although both are sometimes abbreviated as *M. bovis.*

Transmission:

- Direct contact, inhalation of aerosolized bacteria, ingestion from contaminated soils/feed/water, and contact with urine/feces of infected animals.
- Wildlife reservoirs of the bacteria are important, and include white-tailed deer and elk.

Clinical signs:

- Chronic weight loss, chronic moist cough and enlarged lymph nodes.
- bTb often manifests as a slowly progressive, chronic disease (can take years to cause death).

Postmortem findings:

- Bison with tuberculosis may have tubercular abscesses or granulomas in head and throat lymph nodes, and thoracic and abdominal cavities.

Diagnosis:

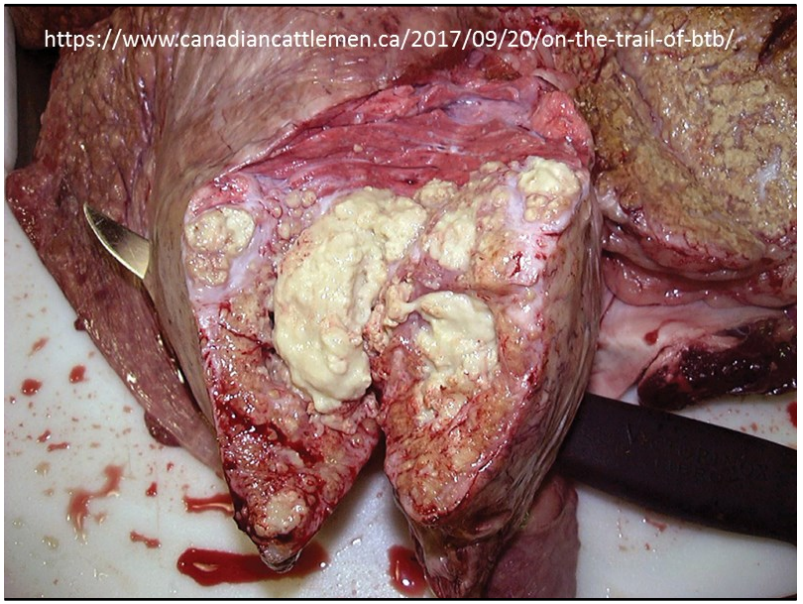
- The diagnostic test used in live bison is the intradermal caudal fold skin test done by a CFIA/USDA accredited veterinarian.
- When conducting a necropsy large granulomas (very firm, thick yellow-white-gray abscesses) within lungs, lymph nodes, and liver can be seen.
- If these granulomas are noted stop the necropsy and contact your state or CFIA veterinarian.
- Culture of the bacteria from lesions is the gold standard, most often done postmortem.

Treatment:

- No treatment - slaughter only.

Control:

- In Canada and the US tuberculosis caused by *Mycobacterium bovis* is a reportable disease.
- The CFIA (Canada) and the USDA (US) maintains monitoring and surveillance of this disease.
- In Canada:
  - Bison herds must achieve and maintain a negative herd status from the CFIA.
  - All bison in the herd over 18 months of age must test negative to the caudal fold skin test.
  - The negative herd status will remain in effect for 5 years. Testing 10 % of the herd annually may extend the negative herd status.
- In the US:
  - Test, and slaughter those animals who test positive on screening and confirmatory tests.
  - Subsequent USDA testing and quarantine procedures will be initiated. Limit contact between reservoir hosts (deer) and bison and exclude wildlife from feeding areas.



Above: Cut surface of lung shows bovine TB lesions. They are usually abscesses with gritty lumps in the pus.

Diseases of the Reproductive System

Listeriosis

Causes:
- Bacteria - *Listeria monocytogenes*.
- Stress - Transport, weaning, mixing, overcrowding, starvation, water deprivation, and handling.

Transmission:
- Ingestion of vegetation or soil contaminated with feces .
- Spoiled silage can be a source of the bacteria.
- Once ingested, the bacteria often localizes in the intestine, brain, and/or placenta. If localized in the intestine this results in prolonged shedding of the bacteria.
- The bacterium is very widespread in nature. *L. monocytogenes* is found in about 45% of cattle manure samples and 80% of feed samples.

Clinical signs:
- Uterine infections at all stages of pregnancy, resulting in placentitis, fetal infection and stillbirth, abortion, deaths in newborn calves.
- Sporadic abortions occur in the last trimester of pregnancy.
- In cattle (presumably bison), *L. monocytogenes* causes brain infections with septicemia and nervous signs - weakness, loss of coordination, circling, head pressing.

Diagnosis:
- Submit an aborted fetus with placenta to a diagnostic pathology laboratory for histopathology as well as bacterial culture and identification.

Treatment:
- Antibiotics can be used to treat infected individuals.
- Once neurologic, it is difficult to treat with success.

Prevention:
- Avoid moldy/spoiled feed and improperly preserved ensiled forages.
- Isolate animals with clinical signs of the disease.

Brucellosis (Bang's Disease)

Causes:
- Bacteria - *Brucella abortus* (most often biovar 1).

Transmission:
- Spread by direct contact with aborted fetuses, placentas, fetal fluids, and vaginal discharge of infected individuals as well as contaminated fomites (shoes, boots, and work equipment).
- Also spread through milk and feces.
- Ingestion, mucous membrane contact, and breaks in the skin are avenues of infection.
- Infected individuals can become chronic carriers.
- Wildlife can be considered reservoir hosts of this bacteria (e.g. deer, elk, and bison).

Clinical signs:
- Females - abortion and retained placentas.
- Males - inflammation of testicles and seminal vesicles, resulting in decreased fertility.
- Other signs in both sexes include abscesses, chronic septic arthritis, lameness, and swelling over joints (hygroma).

Diagnosis:
- Bacteria can be cultured from fetal membranes, vaginal discharge, milk, semen, aborted fetuses, hygromas, and placentas.
- Blood samples can be used to detect Brucella-specific antigen as well as antibodies.

Treatment:
- In Canada and the US, brucellosis is not treated.
- Bison that are infected or test serologically positive for *B.abortus* are slaughtered.

Control:
- In Canada:
  - Farmed bison in Canada are brucellosis free but the disease is endemic in some northern wild herds.
  - The Canadian Food Inspection Agency (CFIA) administers a surveillance program that includes on-farm and slaughterhouse testing, and monitoring of the movement of bison.

- In the US:
  - Farmed bison in the US are brucellosis free but the disease is endemic in the wild Yellowstone Park herds. Montana, Wyoming and Idaho have Designated Surveillance Areas (DSA) around Yellowstone Park.
  - In DSAs, whole herd testing is done every 3 years. Positives result in a herd quarantine until reactors are removed and 3 consecutive negative herd tests are achieved.
  - Movement of bison off farms in the DSAs requires testing.
  - Vaccination is approved for domestic cattle, but efficacy/safety are not thoroughly tested in bison.

Above: Brucellosis positive cow with her calf.
Photo credit: Dave Hunter, Turner Enterprises

Bovine Virus Diarrhea (BVD)

BVD is a very complex disease. Little is known about the behavior of the virus in bison herds, other than that it is present and that it causes disease. In serosurveys, 60 to 80% of cattle tested have antibodies to the virus. A serosurvey in Yellowstone National Park demonstrated antibody titers in 31% of bison tested.

Causes:

- Virus - Bovine Virus Diarrhea Virus (BVDV).

Transmission:

- Thought to be by direct contact between animals (horizontal) and across the placenta to the fetus (vertical).
- BVD virus can be shed in nasal discharge, saliva, semen, feces, urine, tears, milk and uterine discharges following abortion.
- Some cattle can become persistently infected, shedding large quantities of the virus for the rest of their lives and are probably the main source of infection in cattle herds.
- Persistently infected bison have not been found, to date.

Clinical signs:

- There are several different manifestations of BVD (in cattle).
- *Acute (rapid onset) BVD:*
  - Occurs in animals that have not been previously exposed to the virus.
  - Can cause mortalities in bison at any age.
  - BVD infection in young calves may cause severe diarrhea (scours).
  - This form of BVD can be a symptomless infection or it can cause fever, diarrhea, fetal infections resulting in abortions/birth defects and immunosuppression leading to secondary infections (often respiratory).
- *Fetal infections:*
  - If a fetus is infected in the first 125-150 days of gestation it may become persistently infected (PI).
  - When these calves (cattle) are born they are infected and remain so for the rest of their lives, shedding virus and infecting naïve herds.
  - Fetal infections have not been reported in bison.
- *Persistently infected animals (PI's):*
  - Persistently infected animals (PI's) <u>have not been found</u> in bison.
- *Mucosal disease form:*
  - This disease only occurs in animals that are persistently infected.
- *Thrombocytopenic form:*
  - This form of BVD has not been reported in bison.

Diagnosis:

- Usually made on necropsy and submission of samples to a diagnostic lab.
- Blood samples for virus isolation or polymerase chain reaction (PCR) testing give reliable results.
- Skin samples from dead infected animals can be used to isolate the virus even if the carcass is decomposed or partly eaten by scavengers.

Treatment:

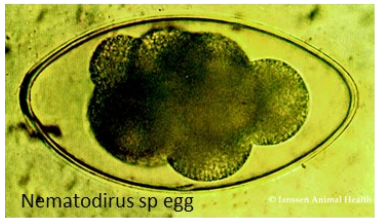
- No specific treatment protocols established for treating cattle or bison.
- Animals with BVD have not responded to any treatment.

Control:

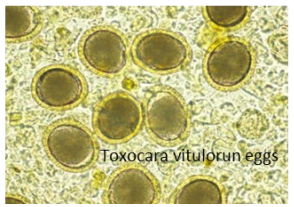
- In cattle, control of BVD has proven to be difficult for some forms of the disease.
- Vaccination may provide adequate protection against acute BVD infection.
- There are many commercially available BVD vaccines but none of them have been registered for use in bison.
- The adjuvants and immune stimulants found in killed BVD vaccines may have unfavorable effects on bison calves.
- Modified live BVD vaccines have been observed to cause diarrhea in recently weaned bison calves.
- DO NOT use live vaccine on pregnant bison - efficacy/safety not tested.

Prevention:

- Good biosecurity practices and proper hygiene within bison facilities can help decrease spread.
- Maintain a closed herd, and test new animals if concerned.
- Use test-and-cull management to decrease transmission.



Above: Trichostrongyles, including Ostertagia, Nematodirus, Haemonchus all have “strongyle type” eggs and cannot reliably be distinguished from each other in the egg stage. DNA fingerprinting is the modern way to diagnose which species are involved in your parasite problems.



Above: Examples of other parasite eggs frequently found on fecal examination of bison.

Internal Parasitism (General)

Causes and pathogenesis:

- Parasites come in many forms including nematodes (round worms), protozoa (one-celled organisms), and flukes (flat worms).
- Bison can be hosts for the same parasites that cattle have.
- The following stomach and intestinal parasites have been identified in bison: Cooperia, Haemonchus, Monezia, Nematodirus, Oesophagostomum, Ostertagia, Setaria, Trichostrongylus, and Trichuris species.
- Pathogenesis of parasites varies by species and type of parasite.
    - Gastrointestinal parasites inhabit the abomasum and intestines, damaging the GI wall causing inflammation, and feeding on blood.
    - Some species invade the abomasal wall and enter a dormant state (hypobiosis).
    - When they re-emerge they damage the abomasal wall and prevent absorption of essential minerals and protein.
    - Liver fluke migration within the liver can trigger certain clostridial (bacterial) diseases.

***Transmission and life cycles*:**

- Transmission is through oral ingestion of parasite eggs or infective larvae, usually on pasture.
- Parasitism is often associated with overcrowded or wet conditions especially during the summer, when parasite survival on the ground is highest.
- The general life cycle of most internal parasites involves eggs laid by adult worms in the animal's GI tract or larvae which are then passed in feces onto pasture where they go through a few immature life stages in the soil or intermediate host species before being consumed again on forage.

Clinical Signs:

- Poor body condition, dull/shaggy hair coat, low pregnancy rates, and weak calves despite adequate feed and nutrition and access to bulls.
- Diarrhea and depression despite a good appetite. Generally these symptoms occur in gradients with dominant animals showing subclinical signs, with less dominant individuals showing more extreme symptoms.
- Parasites will weaken the immune system, making the animal more susceptible to infections.

Diagnosis:

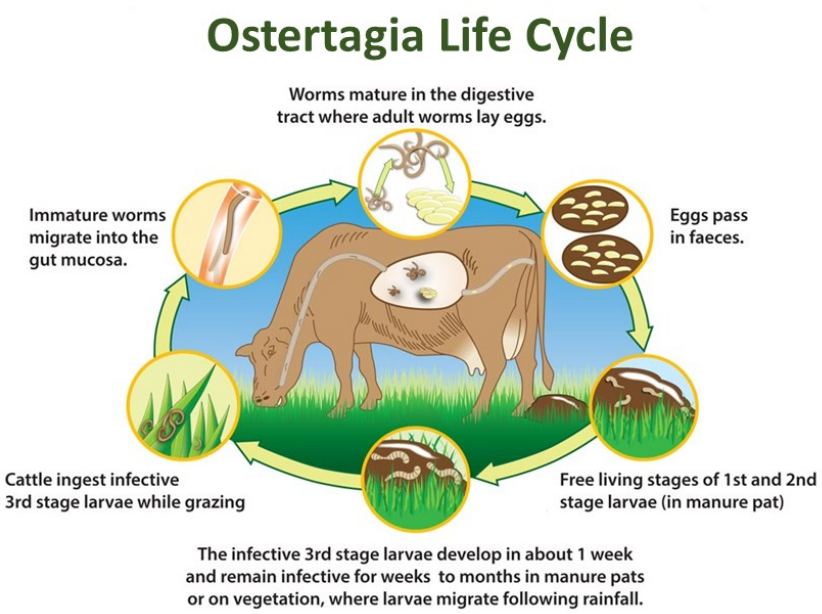
- Clinical signs in combination with a fecal egg count and visual identification can determine the exact parasite.

Treatment:

- Oral, pour-on and injectable dewormers are available for treatment, depending on the target parasite.

Prevention:

- Routine treatment at fall/spring handling is a good way to help manage some parasites.
- Because of the dense hair coat of bison, pour-on preparations often aren't absorbed well unless the drug is placed right on the skin along the back.
- Due to hierarchical nature of bison herds, appropriately dosing the herd with oral dewormer is difficult - dominant individuals will be overdosed while subordinate individuals will be underdosed.



https://www.bimectin.com/disease-information-cdn/cattle/stomach-worms

Ostertagiosis

Causes:

- Parasite: various *Ostertagia* species - usually *O. ostertagia.*

Transmission and life cycle:

- Ingestion of infective larvae on forage or on the ground.
- Ostertagia larva can penetrate the glands of the abomasum and there they can enter into a dormant (hypobiotic) state.
- They cause no damage to the abomasum until the larvae emerge from this state causing serious damage to the abomasum.
- This form of ostertagiosis is called Type II ostertagiosis .

Clinical signs:

- Loss of appetite, weight loss, weakness, dull hair coat, severe diarrhea, anemia.
- Mortalities are common.

Diagnosis:

- Examine feces (fecal flotation) to identify parasite eggs and larvae.
- Take blood samples and look for hypoproteinemia, decreased lymphocytes increased neutrophils (both are white cells).
- Increase in serum pepsinogen.
- Often leads to copper deficiency (poor absorption) and low liver copper levels.
- Post mortem exam shows emaciation, and irregular thickening of the abomasal mucosa, giving it a morocco-leather appearance.

Treatment:

- Ostertagia are susceptible to a wide variety of parasiticides, including benzimidazoles, avermectins and levamisole.
- The hypobiotic forms of Ostertagia are very resistant to treatment.

Control:

- Control programs will also vary with the geographic location of the bison.
- Programs should include routine fecal egg counts, pasture rotation and wise use of worming medications.

Anaplasmosis

Causes:

- Bacteria - *Anaplasma marginale.*
- *A. marginale* parasitizes the red blood cells of the host animal.

Transmission:

- It is transmitted between hosts by insect vectors, usually ticks. In the western USA the most common vector is the tick *Dermacentor andersoni*.
- 15.7% of bison tested at the National Bison Range in Montana, were seropositive to anaplasmosis on a compliment fixation test.

Clinical signs:

- Common clinical signs include anemia, jaundice, emaciation and debility.
- Bison calves experimentally infected with anaplasmosis demonstrated very mild clinical signs that included anemia, mild depression and listlessness.
- Bison may be more resistant to anaplasmosis than cattle.

Diagnosis:

- Examination of blood samples for the presence of *A. marginale* on red blood cells.
- Submit blood samples to a diagnostic pathology laboratory to detect antibodies to anaplasmosis.

Treatment:

- Animals with anaplasmosis respond well to treatments with long-acting tetracyclines.

Control:

- There have been no control programs reported for anaplasmosis in bison but there are vaccination protocols established for cattle.
- They do not provide complete protection against anaplasmosis for extended periods of time.
- Some of the vaccines are associated with serious side effects, so it may not be advisable to use these vaccines in bison.

Coccidiosis

Causes:

- Protoza (one-celled organisms): various species of Eimeria.
- Although Eimeria are routinely seen in the feces of bison, clinical disease is uncommon.

Transmission:

- Coccidian oocysts (eggs) will survive well in a wet environment.
- Clinical disease is associated with buildup of organisms from overcrowding, fecal build up in pens, fecal contamination of water sources, weaning, transport, and mixing of calves, and cold weather conditions.
- Dry, sunny conditions will dramatically reduce the survival of coccidian oocysts in the environment.

Clinical signs:

- In cattle, coccidia species cause bloody diarrhea in calves.
- There have been no clinical signs of disease reported in bison literature.
- Although clinical coccidiosis is unusual in bison, it is possible that poor management conditions may precipitate the occurrence of disease.

Diagnosis:

- Coccidia oocysts may be easily identified by examining fecal samples from bison (fecal flotation).
- The presence of coccidian oocysts in the feces of bison may not be an indication that coccidia is the cause of disease.
- Bison calves must have diarrhea that contains at least 5000 coccidian oocysts per gram of feces.

Treatment:

- Medications for treating coccidiosis in cattle include: sulfamethazine, nitrofurazone and amprolium.
- None of these drugs have been licensed for use in bison.
-

Control:

- Preventative programs for bison should be centered on the management of weaned bison calves:
    - Weaned bison calves should have lots of space, to prevent fecal build up in the pens.
    - Feed bunks and water sources should be well separated to prevent fecal build up around them.

Control con't:

- Remove feces when it builds up around sources of feed and water.
- Ensure that there is adequate feed bunk space for all the calves. Monitor the feeding behavior of calves closely. Weaned bison calves will quickly develop dominance relationships.
- Don't transport or mix calves until one to two months after weaning.
- Monensin and Lasalocid have been fed to beef calves:
- In feed yards to prevent the development of clinical coccidiosis, and
- to cows during the winter to reduce the shedding of coccidian oocysts onto winter feeding or bedding grounds.

Liver Fluke

Causes:

- Flatworm - *Fasciola hepatica.*

Transmission and life cycle:

- The life cycle of *F. hepatica* requires the passage of immature stages of the fluke through snails.
- Immature stages of the fluke leave the snail and encyst on vegetation.
- Bison are infected when they consume infected snails or cysts.
- Once eaten, the immature flukes leave the intestinal tract of the new host and migrate through the abdominal cavity to enter the liver, where they travel around causing damage to the liver.
- After 4 to 5 weeks the flukes enter the bile ducts of the liver and begin to lay eggs.
- These eggs are passed into the intestinal tract, where they hatch into immature forms of the fluke.
- The immature flukes pass out of the host in the feces and once on the ground the immature flukes can infect snails.

Clinical signs:

- Flukes cause acute hepatitis while they are migrating through the liver signs include sudden death, anemia, low blood protein, edema, and fluid in the abdomen.
- Chronic hepatitis may be associated with damage to the biliary system, caused by flukes residing in the bile ducts.
- Liver damage places animals at risk from bacillary hemoglobinuria or "Redwater" (*Clostridium haemolytica*).

Diagnosis:

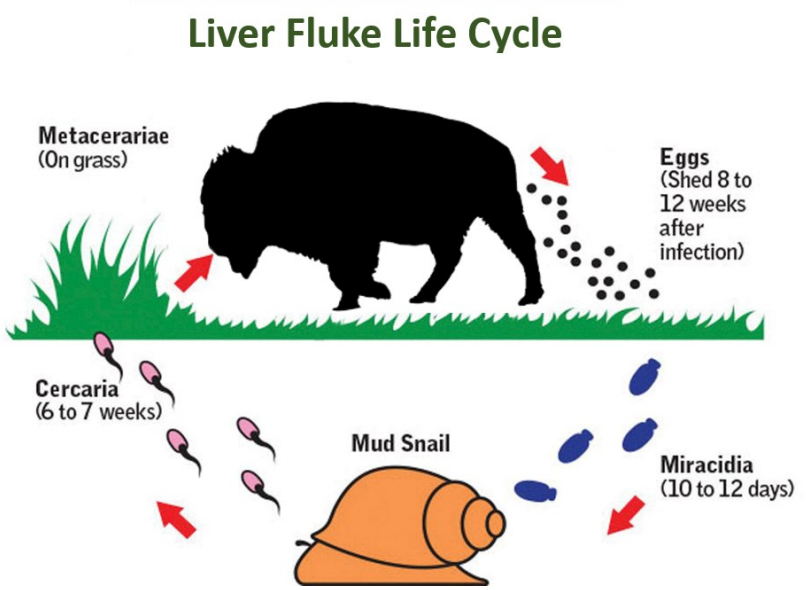
- In chronic cases fluke eggs can be observed in fecal samples.
- Acute (rapid onset) cases are most commonly diagnosed on postmortem examination - may have a large, damaged, swollen liver with perforations of the capsule and subcapsular hemorrhages.
- Chronic disease causes enlarged bile ducts with adult liver flukes in them.

Treatment:

- Triclabendazole (Fasinex) is a commonly used flukicide because it kills all stages of fluke development.
- Treatment should be administered early in the spring to reduce the number of flukes that infected bison pass onto pasture during the summer.

Control:

- Reducing the levels of immature flukes found in snails and in vegetation and prevent bison from consuming encysted flukes and infected snails.
- Generally means fencing off water sources or marshy areas contaminated with flukes and snails.



Adapted from www.canadiancattlemen.ca/2017/07/25/controlling-liver-flukes-in-beef-cattle/

Lungworm

Causes:

- Parasite: Various *Dictyocaulus species*.

Transmission and life cycle:

- Mature lungworms live in the bronchi of infected animals.
- They produce eggs that hatch into larva, which are swallowed and passed out in the feces.
- The larva can survive overwinter on pasture but dry, warm and sunny weather will destroy larva.
- Larvae consumed on pasture pass through the wall of the intestinal tract and enter the venous drainage of the intestine.
- Once in the circulatory system, the larvae pass through the heart and lodge in the lung.

Clinical signs:

- Most commonly seen in late summer.
- Increased respiratory rate, cough, slight nasal discharge, increased heart rate, mild fever.
- Clinical signs are difficult to recognize in bison on pasture.

Diagnosis:

- Fecal samples can be examined for the presence of lungworm larvae.
- Routine flotation techniques will not detect lungworm larva. A Baermann sedimentation procedure must be performed.
- Test in late July and August to see if bison are becoming infected while on pasture.
- Postmortem findings: pulmonary edema, emphysema and large quantities of bloody froth in the trachea, and bronchi containing adult lungworm.

Treatment:

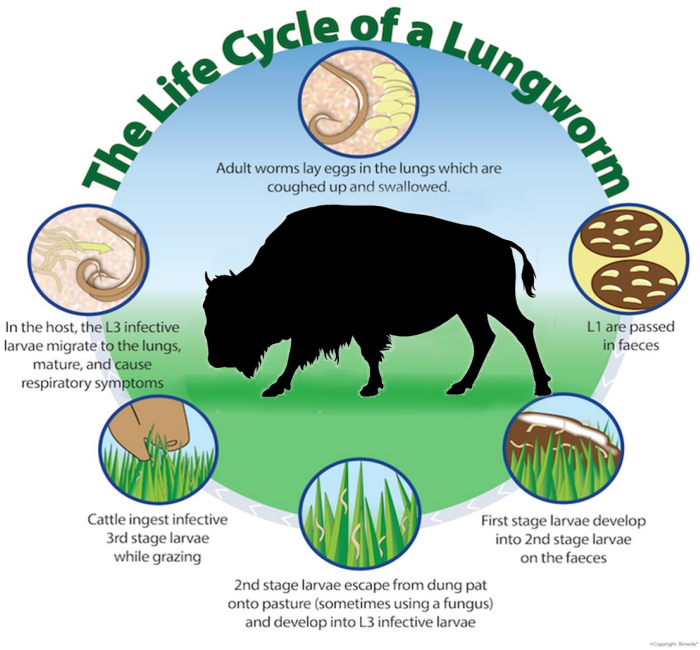
- Ivermectin, oxfendazole, fenbendazole, albendazole, febantel, and levamisole have all been used to treat lungworm infection in cattle.
- The problem is not what to treat with, but how and when to get the treatment into them.
- Lungworm treatments should be done in late spring, before the herd goes out to pasture.

Treatment con't:

- Oral fenbendazole (Safegard™) can be provided in the feed, or in salt.
- Reducing the worm load in bison at this time will reduce the number of eggs deposited on the pasture during summer.

Control:

- Control programs should include well-timed anthelmintic treatments as well as pasture management programs aimed at reducing the number of larvae consumed by bison while on pasture.
- Contaminated pastures should not be grazed early in the spring. A simple two-pasture rotation system can be setup where one pasture is used for spring to early summer grazing and another is used for summer and fall grazing.
- Fecal sampling surveys of the herd looking for larvae should be done in early spring (March, April, May) .



Adapted from www.bimectin.com/disease-information-cdn/

Nutritional Diseases

Copper Deficiency

Cause:

- Primary copper deficiency—insufficient copper in the diet.
- Secondary copper deficiency— dietary constituents prevent copper from being absorbed from the digestive tract.
- Secondary deficiency can be associated with high sulfur (sulfates) or molybdenum levels in the diet or drinking water.

Clinical signs:

- "Poor doing" animals, infertility.
- Stiff gait, lameness leading to recumbency (arthritis)
- Emaciation, diarrhea, and loss of coat color in adult animals.

Diagnosis:

- Herd history involving the above clinical signs would be suggestive of copper deficiency.
- Normal serum and liver copper levels have not been established for bison but cattle reference values are useful.
- Liver Cu values are more reliable and diagnostic than serum levels.
- In reported cases in bison, serum copper levels were 5.8 mol/l and liver copper levels were 0.02 mol/g.

Postmortem findings:

- Infertility resulting from Cu deficiency shows no specific post mortem lesions.
- Positive findings are mainly associated with degenerative lesions of the joints, which included thinning of articular cartilage, defects in the articular cartilage and rupture of joint ligaments and capsules.

Treatment:

- In advanced cases with degenerative joint lesions, treatment would probably be unsuccessful.
- Copper injections have been used when necessary.
- The diet of affected bison should be supplemented with copper.
- 25 mg of Cu per kg of dry matter of the total dietary intake is sufficient.
- Copper sulfate can be added to the salt-mineral mix to a level of 3 to 5% of the total mineral mixture.

Control:

- Feed, water, and pasture forage should be sampled to determine if there is adequate copper available in the diet.
- These should also be tested to see if there are any elements present, such as molybdenum, or sulfates, that may inhibit the absorption of copper from the diet.
- Over supplementation with copper can produce toxicities. Care should be taken when copper supplements are prescribed. A qualified nutritionist should be consulted to examine both feed and water analysis before supplementation of copper, or other trace minerals is recommended.

Diseases Causing Sudden Death

Clostridial Disease

Causes:

- Bacteria in the genus *Clostridium.*
- Clostridium bacteria release volatile and damaging toxins often resulting in rapid death of the host animal.
- *Blackleg* (*Cl. chauvei*) occurs after trauma to musculature resulting in bruising, providing an anaerobic environment for bacterial growth and reproduction. (See further description this section).
- *Tetanus* (*Cl. tetani*) occurs after puncture wounds or exposure of wound site to infective organisms providing an anaerobic environment for bacterial growth and reproduction. Can also occur from uterine infections.
- *Malignant edema* (various Clostridium species) occurs after puncture wounds or exposure of wound site to infective organisms, resulting in a fatal toxemia.
- *Botulism* (*Cl. botulinum*) occurs after ingestion of decomposing animal or plant material (i.e. dead mouse in hay) contaminated with the bacteria's toxins.
- *Enterotoxemia* (*Cl. perfringens* or *Cl. difficile*) caused by disruption in digestion, often when introduced to new high carbohydrate diets too rapidly – allowing normally present Clostridium organisms in the gastrointestinal tract to rapidly overgrow and proliferate.
- *Redwater disease* (bacillary hemoglobinuria by *Cl. hemolyticum*) caused by damage to liver (where spores can be present and inactive), often precipitated by liver flukes – results in intravascular rupture of red blood cells and anemia.

Transmission:

- Clostridium bacteria live in the environment in spore form, or are natural inhabitants of clinically normal animals.
- These bacteria thrive in damaged tissue under conditions of low oxygen (anerobic).
- Spores of some Clostridium are ingested from contaminated environments.
- Otherwise, Clostridium bacteria are often already present but rapidly increase in numbers and produce toxins under favourable conditions.

Clinical Signs:

- Symptoms vary dependent on specific infection.
- *Blackleg* - most common clostridial disease found in bison (see description this section).
- *Tetanus* - saw-horse stance/lockjaw, rigid paralysis, resulting in respiratory failure and death.
- *Malignant edema* - swelling, muscle tissue death, gangrene, fever, weakness.
- *Botulism* - floppy paralysis starting in hind quarters working its way forward. Drooling is often seen as well as difficulty chewing or swallowing.
- *Redwater disease* - weakness, red colored urine; often results in sudden death.
- *Enterotoxemia* - affects gastrointestinal tract causing severe diarrhea and systemic toxemia; may result in death before symptoms appear.

Diagnosis:

- Depends on strain of Clostridium, but can be cultured from feces or wounds of affected animals.
- Laboratory tests are used to identify toxins from specific clostridia.

Treatment:

- Most often there is no opportunity to treat – sudden death.
- High doses of penicillin can be attempted, but disease is usually fatal once noticed.

Prevention:

- Use multivalent vaccines targeting multiple clostridial species (often called 5-,7-, 8-, 9-way vaccine).
- No efficacy studies performed in bison, but vaccines commonly used amongst producers and vaccinated animals don't seem to get clostridial diseases.

Blackleg

Causes:

- Bacteria - *Clostridium chauvei.*
- *C. chauvei* is a soil-borne bacterium and can survive in the soil in spore form for many years.

Transmission and pathogenesis:

- The bacterium enters the body through consumption of contaminated grass or hay.
- Usually seen in the late summer when pasture is low, or bacteria can be picked up in hay.
- *C. chauvei* organisms are found in the spleen, liver and intestinal tract of normal animals.
- Disease occurs when bacterial spores lodged in normal tissue (usually muscle) proliferate secondary to bruising or injury of the tissue.
- As the bacteria proliferate they produce toxins that kill tissue and bring about toxemia.

Clinical signs:

- Clinical signs are not often seen because the rapid onset and quick progression to death.
- Some clinical signs that might be observed:
  - the most common finding is sudden death.
  - animals found alive will be depressed, anorexic, have a high heart rate and high body temperature.
  - Swellings with the feel of bubble wrap may be seen under the skin in localized areas.

Diagnosis:

- The diagnosis of blackleg is most commonly made on postmortem examination.
- Affected tissue may be submitted to a diagnostic pathology laboratory for bacterial culture and identification.
- The following are some post mortem changes:
  - the carcass bloats and putrefies quickly after death.
  - body cavities often contain excess fluid, often reddish in color.
  - infected muscle masses are swollen, discolored and have a foul odor.

Treatment:

- Response to treatment is often poor and usually the animal is found dead or too sick to treat.
- *C. chauvei* is susceptible to most antibiotics commonly used in cattle.
- Vaccinate all animals in the face of an outbreak and treat all at risk animals with long acting antibiotic preparations to provide protection until immunity develops.

Control:

- All bison should be vaccinated against blackleg using a multivalent 8-way vaccine.
- Many bison ranchers vaccinate all of their bison with a clostridial vaccine annually.

Above: Anthrax carcass—typical “sawhorse position” with severe, rapid bloating as well as bloody discharge from body openings.

Anthrax

Causes:

- Bacteria - *Bacillus anthracis.*

Transmission:

- Anthrax is distributed along river valleys and flood plains.
- Outbreaks most often occur during summer months after prolonged wet periods.
- Infection is thought to occur by ingestion of spores present in recently disturbed soil or hay.
- If the carcass is opened to the air, the bacteria will produce resistant spores that may survive in the environment for up to 30 years.
- Spores can be spread by streams, insects, birds, dogs and other carnivores.
- Humans can contract anthrax if they eat infected meat or through contamination of skin wounds acquired from processing or performing a postmortem on an infected carcass.

Clinical Signs:

- Usually a very rapidly fatal disease.
- Sick bison are often unresponsive and stand with their head down.
- They may be reluctant to move and may be lame, stagger or have a stiff legged gait.
- Affected animals will often have edematous swellings of the preputial or umbilical region.

Diagnosis:

- Swabs may be taken of the exudate from a leaky orifice and sent to a diagnostic pathology laboratory for bacterial culture and identification.
- SNAP test kits are available but can be unreliable in decomposed animals.
- When examined microscopically *B. anthracis* has a characteristic rectangular "box-car" shape.

Do not perform a postmortem dissection without first examining exudate from body openings for the presence of anthrax organisms.

- The diagnosis is commonly made on postmortem exam of the carcass:
  - the most common accompanying history is sudden death.
  - the carcass bloats quickly after death, and has a "sawhorse" appearance.
  - there may be exudation of dark tarry blood from body orifices.
  - spleen and lymph nodes are typically enlarged and the blood may not clot after death.

Treatment:

- None - clinically affected animals usually die rapidly.

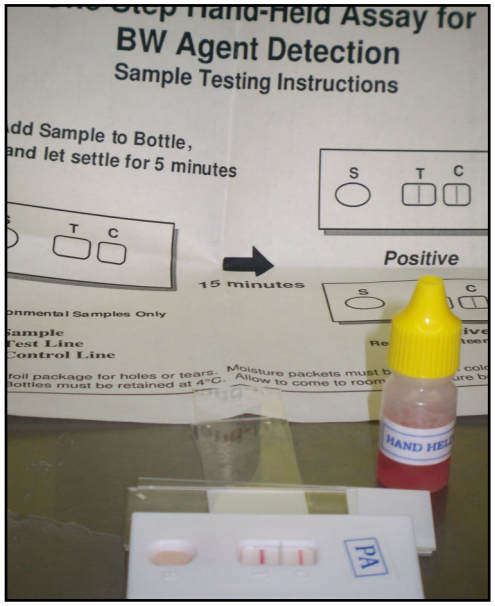
Control:

- Commercial cattle anthrax vaccine available - use 2x cattle dose.
- Probably only effective for 6 months in bison.
- Vaccinate bison 2-4 weeks prior to exposure to known infected pasture.
- Avoid recent flood areas with history of anthrax.

Special Notes:

- **Any bison that suddenly died, quickly bloated, and has blood coming from body orifices should be suspected of anthrax.**
- If the carcass of a dead animal is not opened after death, putrefaction will destroy the bacteria.
- This is a federally reportable disease – notify state/provincial veterinarian immediately if cases are suspected.
- Dispose of carcasses by burning or deep burial with local disinfection using 5% formaldehyde or Decon™ foam.
- Follow recommendations from your state/provincial veterinarian.

Above: Using the EasyDecon DF 200 foam to decontaminate an anthrax carcass



Left: SNAP tests are available for anthrax detection in the field.

Histophilosis

Causes:

- Bacteria: *Histophilus* (formery Hemophilus) *somnus.*
- Stress: Environmental, social, nutritional as well as management stress (transport, weaning, overcrowding, handling).

Transmission:

- Common organism - in Canada, 25% of cattle tested serologically positive to *H. somnus* and the organism has been isolated from the respiratory, urinary, and reproductive tracts of clinically normal animals.
- Outbreaks in bison occur most commonly in the fall and winter and have been associated with very cold weather and stressful situations such as handling.

Clinical signs:

- In cattle *H. somnus* causes disease in a wide range of body organs.
- In bison, only the meningitis form has been reported - called infectious thromboembolic meningoencephalitis or ITEME.
- Sudden death is common.
- Animals may be found recumbent, depressed, and have a high or low body temperature. They may have blindness in one or both eyes.
- Sick bison calves usually die within 24 to 48 hours.
- Convulsions often occur prior to death. If clinically affected calves are found standing, they may be weak with tremors, staggering, or ataxic.

Diagnosis:

- Diagnosis of this disease is difficult in live bison - clinical signs are suggestive but not definitive.
- The diagnosis is most often made from postmortem examination.
- There are hemorrhagic infarcts in the brain and spinal cord.
- Tissues from the carcass, especially the brain, should be sent to a pathology laboratory for microscopic exam as well as bacterial culture and isolation.

Treatment:

- Observation of at-risk calves is important for the detection of even advanced cases of disease.
- Oxytetracycline or florfenicol are commonly used antibiotics.
- Surveillance and early detection and treatment of bison calves is critical to reducing mortalities.

Control:

- There are many commercially available *H. somnus* vaccines.
- The efficacy of these vaccines for the protection of bison against *H. somnus* is unknown.

Malignant Catarrhal Fever (MCF)

Causes:

- Virus-Ovine Herpesvirus-2 (OHV-2).
- Stress-most cases occur in winter.

Transmission:

- Bison are very susceptible to OvHV-2.
- Domestic sheep are the main reservoir of this virus and shed the virus over their lifetime, mostly when stressed.
- Lambs become infected at birth and shed through respiratory secretions, mostly at weaning.
- Infection comes from contact and proximity to sheep (especially lambs) by direct contact, aerosolized fluids, and shared pasture/water sources.
- Bison closer than 1 km to sheep are at risk from air borne virus.
- The higher the concentration of sheep near bison herds, the higher the risk for infection (bison pastured near sheep feedlots have a higher risk than being pastured near hobby farms).
- Bison are dead-end hosts and do not pass the disease to other bison.

Clinical Signs:

- Incubation period can be as long as 6 months.
- Once clinical signs are observed there is a quick progression to death, generally within three days.
- Depression, weakness, and bloody diarrhea.
- Corneal opacity of one or both eyes can occur, along with conjunctivitis, nasal and ocular discharge.
- Drooling from ulcerations in the mouth, and neurological signs.

Diagnosis:

- Blood samples or nasal swabs can be submitted for molecular (PCR) testing to detect the viral DNA and for antibody tests to determine exposure.
- Multiple tissue samples sent for histopathology and molecular testing is the only validated way to establish a diagnosis

Treatment:

- Supportive care can be provided, but once clinical signs are evident this disease is most often fatal.

Prevention:

- There is no vaccine available for MCF.
- Avoid contact with sheep and sheep pastures - the farther the better due to the distance aerosolized virus particles can travel.

Above: Corneal edema and cloudiness and copious nasal discharge are often signs of MCF if the bison lives long enough for signs to appear.

Miscellaneous Diseases

Pinkeye or Infectious Keratoconjunctivitis

Causes:

- Bacteria - in cattle (presumably bison) *Moraxella bovis*.
- A culture survey for *Moraxella bovis* found this bacterium in 12.7% of normal bison eyes.
- Environment - dust, sunlight and minor injuries to the eye can also precipitate outbreaks.

Transmission

- In cattle (presumably bison) the spread of pinkeye is accomplished by face flies.
- Highly contagious within a herd.

Clinical signs:

- One or both eyes may be infected.
- Increased flow of tears, reddening of the conjunctiva, squinting, and cloudiness of the cornea, which may lead to ulceration of the cornea.
- Temporary or permanent blindness can result.

Diagnosis:

- Send cornea biopsies or swabs of the conjunctival sac to a diagnostic pathology laboratory for bacterial culture and identification.

Treatment:

- Recovery may be spontaneous in many cases.
- In cattle, small volumes of penicillin and cortisone may be injected into the tissues around the eye. Bison are unlikely to allow this.
- In cattle, third eyelid flaps or suturing the eyelids closed have been used. Bison will often remove these sutures shortly after they are placed, by rubbing their eyes and head on the ground.
- Systemic long-acting antibiotics are generally the treatment of choice.

Note: Handling bison with pinkeye can be dangerous to the handlers as well as to the animal. Bison that are blind in one or both of their eyes are unable to protect the affected eye from injury during handling and rupture of the eye can result.

Prevention:

- Reduce the local population of face flies to reduce the spread of the infective agent.
- Installing insecticide applicator wicks around feeders and water sources has been used to reduce the face fly population.
- The commercially available pinkeye vaccines on the market are designed for use in cattle. The causative agent for pinkeye in bison is unknown so the worth of these vaccines in bison is questionable.

Above: Pinkeye with corneal opacity and severe conjunctivitis

Blue Tongue Virus (BTV) / Epizootic Hemorrhagic Disease (EHD)

Causes:

- Both are viruses in the genus *Orbivirus.*

Transmission:

- Virus is transmitted by the bite of an insect vector (biting midges in the genus *Culicoides*).
- Midges (no see-ums) feed on an infected individual and transmit the virus when they feed on naive (uninfected) individual.
- It is suggested that some strains can be spread through direct animal to animal contact, and in-utero infection.
- Deer can become "reservoirs", harboring low level infections that do not cause clinical signs but can be enough to allow midges to spread the virus.
- Endemic in many Western states and is common in the Southeastern US.
- Not currently seen in Canada or Northern States—vectors can't survive.

Clinical Signs:

- Inappetance and/or difficulty eating.
- Fever, depression, lethargy, difficulty breathing or increased respiratory rate.
- Swelling of the face, throat, and joints can occur.
- Ulcerative oral lesions can be present, and muzzles show a crusty appearance.
- Spontaneous abortions are possible.
- Clinical signs in bison are rare, and can develop mild, asymptomatic infections that are not noticed.

Diagnosis:

- Take blood to test for antibodies against BTV, indicating exposure to the virus and in fatal cases some spleen, if fresh, can be collected and sent to a lab for virus isolation tests.

Treatment:

- None - Mainly supportive care (rest, palatable soft feedstuffs, and minimization of stress).

Prevention:

- Vaccination—be sure to match vaccine with the serotype of the local infection.
- Vector control can be difficult due to size and distribution of midges.

Vaccines for Bison

Vaccines prevent diseases in animals by stimulating the animal's immune system to produce the necessary antibodies and cell responses that are required to inactivate an invading bacteria or virus. Vaccines are very specific in the protection that they provide to an animal. A vaccine designed to protect against blackleg will protect an animal against blackleg and no other disease.

When designing a vaccination program for bison there are several points that must be considered:

Do bison actually get the disease that the vaccine is designed to protect against?

- Bison are more susceptible than cattle to some diseases and are less susceptible than cattle to others. There is no point in vaccinating bison with cattle vaccines to protect against a bacteria or virus that doesn't produce disease in bison.

- Very little research has been done into effects of any of these diseases on bison. The lack of good information often forces producers to make guesses based on cattle about those which affect bison.

- Some common cattle pathogens and vaccines are:
  - *IBR (Infectious Bovine Rhinotracheitis)* - can cause disease in bison
  - *PI-3 (Parainfluenza 3)* - not known to cause disease in bison.
  - *BRSV (Bovine Respiratory Syncytial Virus)* - not known to cause disease in bison.
  - *BVD (Bovine Virus Diarrhea)* - will cause disease in bison.
  - *Histophilosis* - will cause disease in bison.
  - *E. coli (scours)* - not known to cause diarrhea in bison calves.
  - *Rota and Corona virus scours* - Rota virus is associated with diarrhea in bison calves.
  - *Brucella abortus* - causes disease in bison, but vaccination is controlled by federal or state government agencies.
  - *Foot rot* - occurs occasionally in bison, but the causative agent(s) is unknown.
  - *Pinkeye* - occurs in bison, but the causative agent(s) is unknown.
  - *Leptospirosis* - will cause disease in bison.
  - *Clostridium bacteria species* (7 or 8 way “multivalent” vaccines) - will cause disease in bison.
  - *Mannheimia (formerly Pasteurella) hemolytica* - causes disease in bison.

Are the bison on your farm at risk of developing the disease that the vaccine protects against?

- Some diseases are known to occur only in very specific geographic locations, whereas others, e.g. blackleg, can occur anywhere.
- Some diseases are associated with certain methods of production e.g. histophilosis which occurs in feedlot settings, but rarely in cow-calf bison operations.
- Some diseases are associated with certain environmental conditions. Calf scours in newborn bison calves most often occurs when bison cows are tightly confined in wet environments during calving.
- The best way to find out which diseases your bison herd may be at risk of getting is to consult your local veterinarian.

Is the vaccine safe for use in bison?

- Some vaccines can cause adverse reactions when they are used in a species for which they were not intended.
- Pharmaceutical companies conducting safety trials for their vaccines only in the species for which the company has been granted a license.
- There have been many instances in which vaccines have caused adverse reactions, including deaths, in their licensed species. In these cases the company is legally responsible.
- Pharmaceutical companies make no claims about the safety of a vaccine given to unlicensed species. If adverse reactions occur in these species the farmer is not legally responsible.
- Any bison producer who uses a cattle vaccine in their bison herd must be aware that they are assuming the risk for any losses that may occur from adverse vaccine reactions.
- Past experience may make us think that cattle vaccines are safe for use in bison; it does not necessarily mean that all cattle vaccines are safe to use in bison under all conditions.

Examples:

  - Modified live virus BVD vaccines have caused outbreaks of diarrhea when they have been administered to recently weaned bison.
  - Certain respiratory vaccines are not designed for use in beef calves that are very young or are under certain body weights. Similarly these vaccines may precipitate outbreaks of pneumonia when used on very young or small calves.
  - Some modified live virus respiratory vaccines are not recommended use in pregnant cows.
- For these reasons it is important to consult someone who has some knowledge of cattle vaccines before using these vaccines in your bison.

Does the vaccine you wish to use produce an immune response in bison?

- In general, bison producers use cattle vaccines on their bison with the same dose administration recommendations that are used for cattle.
- There have been no experimental trials conducted on any of these vaccines in bison so whether cattle vaccines can produce an immune response in bison is not known.
- The dose and frequency of re-administering these vaccines to bison is also not known.
- Only vaccinate your bison against those diseases that pose a definite risk to your herd.
- Vaccinating your bison against diseases for which they are not at risk is at best a waste of money, and it predisposes your bison to any adverse reactions that may be associated with the vaccine.

Does the vaccine you wish to use actually protect bison against the disease?

- Vaccinated is not the same as immunized or protected - an immune response doesn't mean the animal is actually protected from a challenge.
- The lack of clinical trials in bison means that we have no real idea about the effectiveness of any cattle vaccines when they are administered to bison.
- Using ineffective cattle vaccines on bison is a waste of money, and can place bison at risk of adverse reactions.

Creating a vaccination program for bison.

- There are many vaccines available for use in cattle. Each has its own characteristics including: type of vaccine, efficacy, safety, dose, re-administration times, cautions, restrictions, withdrawals, etc. Much of the information available for these vaccines may be of questionable value when applied to bison so designing a vaccination program for a bison herd can be difficult.

Step 1

- Identify those diseases which pose a risk to your herd.
- These diseases should include only those that are found in your geographic location, and those that are known to be associated with management practices similar to yours.

Step 2

- Select and use only those vaccines which have the potential to protect your bison from these diseases.
- Seek out as much information as you can about the risks facing your herd and the potential effects different vaccines may have on your bison.

Veterinarian:

Address:

Phone:

Fax:

Email:

Notes:

The Canadian Bison Association
200-1660 Pasqua Street
Box 3116, Regina, SK . S4P 3G7
306 522 4762

The National Bison Association
8690 Wolff Ct., Suite 200
Westminster, CO. 80031
(o) 303 292 2833
(c) 303 594 4420