Reportable Diseases

Wild bison corralled for disease testing. PHOTO CREDIT: J. PEACO; COURTESY NPS

As with any species, wild or domestic, bison may carry a number of pathogens or parasites. The diseases addressed within this section are the diseases that may infect bison, are transmissible to livestock, and are "reportable" within the state of Montana. These diseases of concern are livestock diseases that could restrict trade or pose a threat to human health (Gates et al., 2010). Under Montana state code 81-2-107, "a person, including the owner or custodian, who has reason to suspect the existence of a dangerous, infectious, contagious, or communicable disease in livestock or the presence of animals exposed to the disease in this state shall immediately give notice to the department (of Livestock)". The diseases of concern for bison identified by Gates et al. (2010) that are listed as reportable diseases in Montana are anthrax, bluetongue, bovine brucellosis, bovine spongiform encephalopathy, bovine tuberculosis, and malignant catarrhal fever (sheep associated), which require immediate notification of state officials and quarantine of the infected domestic animals; and bovine anaplasmosis, bovine viral diarrhea, and Johne's disease, which require notification of state officials within 30 days (ARM 32.3.104; Gates et al., 2010; MDOL, 2010d).

Anthrax

Anthrax is a disease caused by a spore-forming bacterium, *Bacillus anthracis,* (MDOL, 2010a). The bacteria can affect all mammals, but ruminants such as cattle, sheep, bison, and goats are the most susceptible (MDOL, 2010a). Anthrax is a zoonotic disease, which means that it is possible for humans to become infected with the cutaneous form, known as Wool-sorters disease, from close contact with infected animals or their by-products, such as heads or hides (Gates et al., 2010; MDOL, 2010a; J. Rankin, Montana Department of Livestock, personal communication). Anthrax spores exist in soil, and tend to grow and contaminate the soil surface following periods of precipitation and cooler weather that are followed by extended periods of hot, dry conditions (van Ness, 1971; MDOL, 2010a). Anthrax may be spread throughout a region by streams, insects, animals and birds, animal waste, and disturbed carcasses (MDOL, 2010a). Anthrax may also be spread through wastewater effluent from water treatment plants, which can contaminate downstream sediments and pastures (Gates et al., 2010). Inadequately sterilized bone meal and fertilizers made from contaminated material may also spread the anthrax spores (Hugh-Jones and Hussaini, 1975). An animal may become infected through the ingestion of

spores in contaminated food and water, or through inhalation (Gates et al., 2010; MDOL, 2010a).

Upon infection an animal begins to express clinical signs within three to seven days, depending on the number of spores that enter the body (MDOL, 2010a). The spores germinate and replicate within the bloodstream, releasing toxins that eventually lead to death (Dragon and Rennie, 1995). The infected animal may initially have an increase in temperature and excitement, followed by a decrease in coordination, depression, loss of consciousness, difficulty breathing, and convulsions (MDOL, 2010a). Anthrax infection can be determined through testing, and may be treatable in captive bison and livestock with antimicrobials, such as penicillin and oxytetracycline (MDOL, 2010a; Gates et al., 2010). Although anthrax is not treatable in free-ranging wildlife, there are effective vaccines for captive bison and livestock (MDOL, 2010a; Gates et al., 2010). If anthrax is suspected, the healthy livestock should be removed from the affected pasture and placed in quarantine for 42 days, livestock in the surrounding region should be vaccinated, and carcasses or material contaminated with body fluids should be burned or buried at least 6 feet deep (MDOL, 2010a; ARM 32.3.1001).

Anthrax outbreaks occurred in herds of domestic cattle in Montana in Roosevelt County in 2005, and in two isolated regions of eastern Montana in 1999 (MDOL, 2010a). An outbreak of anthrax occurred in domestic bison in 2008 on a private herd in Gallatin County, killing over 287 bison (Ronnow, 2008; Person, 2010a). The ranch began a program of vaccination, and did not experience additional deaths until July 2010, when anthrax was isolated from the carcass of a bison calf that had been killed by predators (Person, 2010a; J. Rankin, personal communication). There have also been reports of anthrax within free-ranging bison in southern Saskatchewan (N. Anderson, Montana Fish, Wildlife & Parks, personal communication). A measure utilized to prevent the spread of anthrax in free-ranging bison herds is the monitoring and disposal of affected carcasses, as carcass scavenging may result in environmental contamination (Nishi et al., 2002).

Bluetongue

Bluetongue is an insect-borne, viral disease that primarily affects sheep, but can occasionally affect goats, deer, and antelope and very rarely affect cattle (APHIS, 2010a). Bison are susceptible to the virus, and infection has been observed under field and captive conditions (Dulac et al., 1988). Infection of humans has not been reported (APHIS, 2010a). The disease is caused by a virus that belongs to the Reoviridae family (APHIS, 2010a). The virus is noncontagious and cannot be transmitted between species without the presence of the insect carriers, which are various species of *Culicoides* midges (Stelljes, 1999; APHIS, 2010a).

The distribution and prevalence of the virus is dependent upon seasonal conditions and the presence of the insect vectors and susceptible animals. The midges prefer warm, moist conditions, and are most prevalent after periods of warmth and precipitation (APHIS, 2010a). Bluetongue is less prevalent in northern regions (Gates et al., 2010). The virus does

not survive outside of the host animal or the insect vector, and is not transmitted through animal carcasses or products (APHIS, 2010a).

The clinical signs of bluetongue include fever, widespread hemorrhages of the oral and nasal tissue, excessive salivation, nasal discharge, lameness, stomatitis, and occasional reproductive failure (Howerth et al., 2001; APHIS, 2010a). There are subacute, acute, and chronic cases. In acute cases the lip and tongue may become swollen, and a limited number of instances present a blue tongue (APHIS, 2010a). The virus may cause mortality within sheep, but mortality rates within the United States have been reported at around 5 percent (Stelljes, 1999). There is no known treatment for bluetongue, but the prevention of infection can be increased by using a combination of quarantine and movement control, treatment and husbandry practices to control the insect vectors, and zoning to define infected and disease-free regions (APHIS, 2010a).

Infection of bison has not been widely reported in North America (Gates et al., 2010). The testing of several public bison herds has not found seroreactors for the bluetongue virus (Gates et al., 2010). The USFWS found that bison that were located near a recent outbreak of bluetongue in deer did not show signs of infection (Gates et al., 2010).

Bovine Anaplasmosis

Bovine anaplasmosis (anaplasmosis) is a disease caused by *Anaplasma marginale,* which is a rickettsia that parasitizes the red blood cells of host animals (Davidson and Goff, 2001; Gates et al., 2010). There are multiple species of *Anaplasma* within the order Rickettsiales that infect domestic cattle, sheep, goats, and a variety of wild ruminants including deer, elk, and bison (Davidson and Goff, 2001). Anaplasmosis survives and reproduces within a host. It is not infectious for humans (Gates et al., 2010). Anaplasmosis occurs throughout tropical and subtropical regions of North and South America, Africa, Asia, Australia, and Europe (Davidson and Goff, 2001). The disease is not contagious between animals, but is transmitted primarily through blood-sucking insects (Gates et al., 2010). The most prevalent spreading of the disease occurs through ticks since the rickettsia can survive and reproduce within the tick (Davidson and Goff, 2001). Transmission from biting insects, including flies and mosquitoes, occurs less frequently because the rickettsia remains viable for only a short period of time on the insect's mouthparts and does not survive and reproduce within the insect (Davidson and Goff, 2001). The level of Rickettsemia in the carrier animal's blood may affect its transmission potential through biting insects. A biting fly with a low level of Rickettsemia does not appear to be able to transmit the disease (Davidson and Goff, 2001). Transmission has also been reported to occur through vaccination needles, or dehorning and castration equipment (Davidson and Goff, 2001).

Most infections of anaplasmosis are subclinical, and therefore the subjects do not display obvious symptoms. All naturally occurring infection among wild ruminants has been subclinical, except for two reports of acute cases in giraffes (Davidson and Goff, 2001). Domestic livestock may have acute, subacute, or chronic infection (Davidson and Goff,

2001). The clinical symptoms of the disease in bison are similar to those in domestic cattle, which include anemia, jaundice, emaciation, and debility (Gates et al., 2010). It is thought that bison may be more resistant than cattle since experimentally infected bison calves demonstrated only mild clinical signs (Zaugg and Kuttler, 1985; Gates et al., 2010).

Tests have been developed to identify anaplasmosis within domestic livestock, but serodiagnosis tests have not been as reliable for wildlife, often generating false results. Modern molecular diagnostic procedures have been more reliable for testing wildlife, but are less practical for larger samples (Davidson and Goff, 2001). Anaplasmosis has been managed within domestic livestock including bison through vector control, vaccination, and antibiotic therapy (Davidson and Goff, 2001). These management programs are not logistically feasible for free-ranging wildlife, and have not been implemented since the disease does not tend to compromise the health of wild bison.

Anaplasmosis is a disease of international regulatory concern, and therefore impacts livestock trade between Canada and the north-central and northwestern United States (Gates et al., 2010). Bison are a known host of *A. marginale* anaplasmosis (Gates et al., 2010). Naturally occurring anaplasmosis infection has occurred on the National Bison Range, where 15.7 percent of the bison have tested positive (Zaugg and Kuttler, 1985; Gates et al., 2010)

Bovine Brucellosis

Bovine brucellosis (brucellosis) is an infectious, contagious disease caused by a bacterium of the genus *Brucella* (Thorne, 2001; MDOL, 2010c). *Brucella* has six species, each with their own principal host. Bovine brucellosis is caused by the species *Brucellosis abortus,* whose primary hosts are cattle and bison (Thorne, 2001; Gates et al., 2010). Elk (*Cervus elaphus*) are also susceptible to brucellosis, and appear to contribute to the interspecies transmission in the Greater Yellowstone Area (Davis et al., 1990; Rhyan et al., 1997; Gates et al., 2010). A study by Proffitt et al. (2010) found that despite high levels of spatial overlap between elk and bison within Yellowstone National Park, "rates of elk exposure to *B. abortus* in this population were similar to rates of exposure in other Greater Yellowstone Ecosystem free-ranging populations not in contact with bison, and lower than rates in elk populations associated with feeding programs" (pp. 287). Proffitt et al. (2010) note that it therefore "appears that the high degree of spatial overlap with bison during the period of transmission risk has little impact on elk exposure to *B. abortus*" (pp. 287). DNA genotyping has indicated that there is a relatively high genetic divergence between the *Brucellosis abortus* found in elk and that found in bison, which suggests that the disease is not extensively exchanged between the two species (Beja-Pereira et al., 2009; White et al., 2011a, pp. 17).

Brucellosis is transmittable to humans and causes undulant fever, which is treatable (Thorne, 2001; MDOL, 2010c). The disease is transmitted to humans through consumption of unpasteurized milk or, more frequently, through direct contact with infected animals during birthing, abortion, or in slaughterhouses (MDOL, 2010c). "Infection by *B. abortus* is

rarely fatal in humans, but can cause severe, reccurring, fever-like symptoms. Humans cannot pass the disease to animals or other humans" (White et al., 2011a, pp. 15–16).

There have been two reports of hunters becoming infected with brucellosis in Montana (MDOL, 2010c). The first occurred in 1986, when it is believed that a hunter had been exposed to the disease while field dressing three elk in the Madison Range (Bridgewater et al., 1997). The second report was from 1995, when a hunter was diagnosed with brucellosis three years after field dressing a bull elk in 1991 (Bridgewater et al., 1997).

Bison being hazed back into Yellowstone National Park from Montana. PHOTO CREDIT: J. PEACO; COURTESY NPS

Brucellosis does not appear to be able to replicate outside of a host, but it can survive outside the host in certain environments (Thorne, 2001). A study by Aune et al. (2009) found that soil, vegetation, and tissue at birth or abortion sites of infected bison remained infected for up to 43 days in April and 26 days in May. Brucellosis is perpetuated naturally through growth in the female reproductive tract, particularly in the membranes and fluids that surround a fetus (Cheville et al., 1998). Within cattle and bison, the disease tends to localize in the udder and reproductive organs (Cheville et al., 1998; MDOL, 2010c). Cheville et al. (1998) note that "bison with non-reproductive-tract infection rarely develop bacteremia, nor do they shed bacteria in salvia, urine, or other body secretion. Thus, even when such animals are in direct contact, bison-to-cattle transmission and even bison-to-bison transmission will occur rarely" (pp. 19). The disease is transmitted primarily though oral contact with an infected fetus, calf, or placenta; through contaminated feed or water; or through licking the genitals of an infected female after a birth or abortion (Thorne, 2001; MDOL, 2010c; Gates et al., 2010). It is possible that both cattle and bison can shed the bacteria in their fecal matter after ingesting highly infected placenta (Cheville et al., 1998). Studies have indicated that male bison who are infected with brucellosis do not appear to transmit the disease to a female through breeding (Thorne, 2001).

Incubation within the animal can be from one week to seven months, and the infection does not present many observable outward signs (Cheville et al., 1998; MDOL, 2010c). The most obvious indication of infection within a pregnant animal is abortion, birth of weak calves, and vaginal discharge (MDOL, 2010c). More than 90 percent of infected bison will abort during their first pregnancy; this rate decreases to an abortion rate of 20 percent after the second pregnancy, and to nearly zero after the third, due to naturally acquired immunity (Davis et al., 1990; Davis et al., 1991; Gates et al., 2010). Male bison experience inflammation of the testicles, seminal vessels, and epididymis, and possible sterility in advanced cases (Thorne, 2001; Gates et al., 2010). Both male and female bison can experience enlarged, arthritic joints, which can lead to lameness and possibly an increased vulnerability to predation (Tessaro, 1989; MDOL, 2010c; Gates et al., 2010).

There are tests to determine if an animal is infected with brucellosis; however, accurate testing can be difficult to achieve due to false negative cultures, which relate to the difficulties in isolating bacteria from chronically infected animals (Cheville et al., 1998; Gates et al., 2010). Brucellosis may be identified through the detection of antibodies in the blood; however, the presence of antibodies does not imply current living infection and can lead to an overestimation of the true level of infection (Cheville et al., 1998; Gates et al., 2010). New tests have been developed that do not look for the antibodies, but for the antigen/antibody complexes (K. Aune, Wildlife Conservation Society, personal communication). It is possible for a cross-reaction to occur in false positive results due to exposure to bacteria that is similar in structure to brucellosis (N. Anderson, personal communication).

YNP bison being gathered for disease testing.
PHOTO CREDIT: J. PEACO; COURTESY NPS

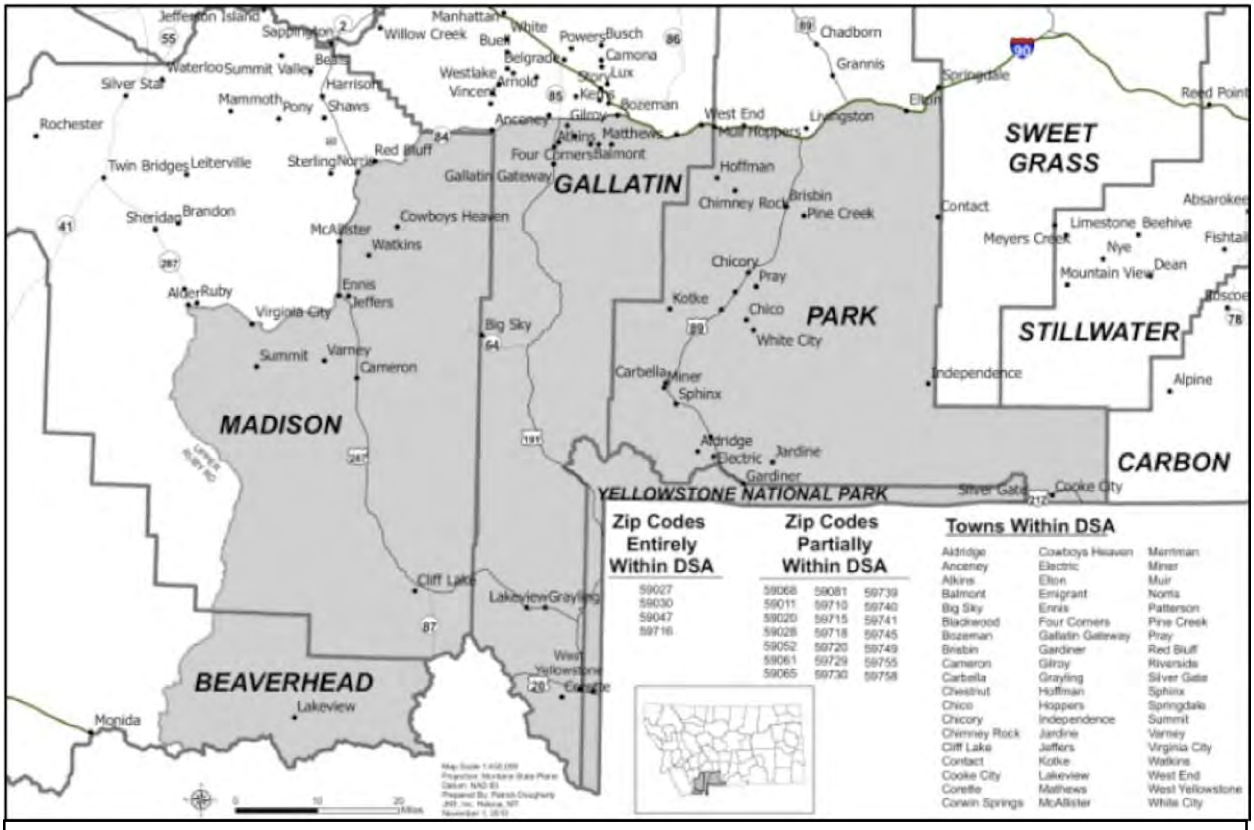
Currently there is no treatment for animals that have been infected with brucellosis (MDOL, 2010c). Many bison develop immune responses, but do not become free of the bacteria (Cheville et al., 1998). The MDOL (2010c) encourages the testing, vaccinating, and isolation of replacement stock, separation of domestic livestock from wild herds that are infected, maintenance of clean calving environments, and use of gloves when assisting in calving or abortions to reduce the transmission of brucellosis.

Through the vaccinating, testing, and culling of infected cattle, Montana was certified brucellosis free in 1985. The first cattle herd to test positive after 1985 was in May 2007 when seven cows that originated from a ranch near Bridger tested positive for brucellosis (Brown, 2007; J. Rankin, personal communication). A second herd tested positive for brucellosis following the detection of an elk serovar in a Paradise Valley cow (Brown, 2008; J. Rankin, personal communication). The detection of a second infected herd caused the state to lose its brucellosis-free status in 2008 (MDOL, 2010c; J. Rankin, personal communication). The state regained brucellosis-free status in July 2009 (J. Rankin, personal communication).

There are two vaccines for brucellosis in domestic cattle, and officials recommend the vaccination of female cattle in areas with heavy infection rates (APHIS, n.d.). Strain 19 was used from the 1930s until 1996, and was shown to be 65 to 67 percent effective in the prevention of infection and abortion in cattle (Cheville et al., 1998). Strain 19 was not as effective in bison and caused a high frequency of abortions (Davis et al., 1991; Gates et al., 2010). A new vaccine, Strain RB51, is now preferred to Strain 19 and does not have as many adverse effects on cattle as Strain 19, nor does it interfere with the accuracy of diagnostic tests as Strain 19 did (Cheville et al., 1998). Strain RB51 is approximately 50

percent effective in bison (Olsen et al., 2009). It has been shown to induce endometritis, placentitis, and abortion in adult bison, though it is believed that this may be related to the timing and location of the injection (Palmer et al., 1996; N. Anderson, personal communication). Strain RB51 does not appear to have significant adverse effects on bison calves (Roffe et al., 1999), and it has been provisionally approved for use in bison, though its safety and efficacy still remain unclear (Gates et al., 2010).

Montana Department of Livestock, Official Order No. 10-01-D



Map 8: Designated Surveillance Area from Official Order No. 10-01-D. Courtesy Montana Department of Livestock.

Official Order No. 10-01-D outlines the requirements for brucellosis vaccination and testing within Montana by establishing the surveillance requirements for brucellosis and a Designated Surveillance Area (DSA). Livestock owners within Beaverhead, Madison, Gallatin, Park, Sweet Grass, and Carbon Counties who have not submitted an Operation Specific Risk Survey to the MDOL, or completed a whole herd brucellosis test since January 1, 2009, must brucellosis-test cattle within 30 days prior to ownership with the following exceptions: Steers and spayed heifers and animals moving directly to slaughter do not need to be tested, and cattle that are being transferred to an approved Montana Livestock Market may be tested upon arrival.

Livestock producers operating within any part of Beaverhead, Gallatin, Madison, and Park Counties shall Officially Calfhood Vaccinate (OCV) all eligible animals, to include female cattle and domestic bison that are 4 to 12 months of age and sexually intact, prior to change of ownership. Female cattle or domestic bison that are not OCV eligible may become official adult vaccinates after completing a negative brucellosis test. This is by written permission of the Montana State Veterinarian.

In addition to requiring OCV of all eligible animals, livestock producers operating within the DSA are required to conduct annual whole herd testing of all sexually intact cattle or domestic bison. The current DSA boundaries, consisting of limited sections of Park, Gallatin, Madison, and Beaverhead Counties (See Map 8), were established based on the movement of elk herds that have exhibited some level of brucellosis seroprevalence. These boundaries are subject to change as more information is gathered on the extent of brucellosis in Montana elk herds (E. Liska, Montana Department of Livestock, personal communication). Producers within the DSA are required to test all age-eligible animals, which includes sexually intact male and female cattle over 12 months old, within 30 days prior to movement out of the DSA or within 30 days prior to a change of ownership. The following exceptions apply to the above testing requirements: Steers and spayed heifers or animals moving directly to slaughter do not have to be tested, and animals being transferred to an approved Montana Livestock Market may be tested upon arrival.

Brucellosis Management

In November 2010 the first report of a brucellosis infection in domestic bison occurred in Gallatin County. A seven-year-old bison cow from a closed herd showed signs of infection (Person, 2010b). The wild bison of Yellowstone National Park are considered to be chronically infected with brucellosis (Cheville et al., 1998), and quarantine protocols have been developed to eliminate all of the bison that have been exposed to brucellosis. A quarantine program such as this was successful in eliminating brucellosis in wood bison during the Hook Lake project (Nishi et al., 2002; Gates et al., 2010). The practice of vaccinating, testing, and slaughter has been successfully used to eliminate brucellosis in the Henry Mountains, Wind Cave National Park, and Elk Island National Park herds (Gates et al., 2010). The quarantine program that has been established for the Greater Yellowstone area is a pilot project, with a goal to produce bison that are free of brucellosis for use in establishing new herds or for the augmentation of existing herds (N. Anderson, personal communication).

As the prevalence and distribution of brucellosis within the United States has been greatly reduced due to effective eradication and testing programs, the U.S. Department of Agriculture's Division of Animal and Plant Health Inspection Service (APHIS) has proposed a new strategy that will allow a more effective and efficient application of limited resources toward minimizing disease risk (APHIS Veterinary Services, 2009). The proposed strategy would shift from state-by-state sampling to a national surveillance strategy that would allow a greater focus on regions of known reservoirs of the disease, such as the Greater Yellowstone Ecosystem (APHIS Veterinary Services, 2009). There would be a shift from

herd depopulation to the development of risk-based affected-herd management plans (APHIS Veterinary Services, 2009).

This strategy would move away from a whole state approach to one using established disease management areas that would be collaboratively managed by the state and federal government, thus minimizing the burden on individual states (APHIS Veterinary Services, 2009). This would also allow for a more effective approach to disease management and would minimize the economic impact on producers (APHIS Veterinary Services, 2009). Under this new strategy the status of the entire state would not be affected based on the infection of individual herds (APHIS Veterinary Services, 2009). There was a series of public meetings throughout the United States to obtain stakeholder input on the new strategy from January to March 2011. The projected time line is to have a published proposed rule by July 2011 and a published final rule by April 2012.

On December 27, 2010, APHIS adopted an interim rule in order to refocus resources to the control and prevention of brucellosis and to protect and maintain the economic viability of the livestock industry (75 FR 81090). The rule was open to public comment until February 25, 2011. This interim rule will reduce the amount of testing required to maintain Class Free status for states that have been Class Free for five years or more and do not have wildlife populations that are known to carry brucellosis (75 FR 81090). The interim rule also removes the provision for automatic reclassification of any Class Free State or area to a lower status if two or more herds have tested positive for brucellosis within a two-year period, or if a single herd that has shown signs of infection is not depopulated within 60 days (75 FR 81090). The interim rule lowers the age at which cattle are included in herd blood tests and requires any regions with brucellosis-infected wildlife to develop and implement a brucellosis management plan in order to maintain Class Free status. The rule also provides alternative testing protocol for dairy cattle herds to allow producers more flexibility for herd certification processes (75 FR 81090).

Bovine Spongiform Encephalopathy

Bovine spongiform encephalopathy (BSE), which is also referred to as "Mad Cow Disease," is one of the transmissible spongiform encephalopathies, which is caused by rogue, misfolded protein agents called prions that are lacking nucleic acids (Prusiner, 1982; Gates et al., 2010). BSE was identified in ten species of Bovidae and Felidae in zoological collections in the British Isles, including bison, though there has not been a case of BSE reported in bison in North America (Kirkwood and Cunningham, 1994; Gates et al., 2010). There have also been no reported wildlife cases of BSE in North America. There has been only one report of an infected cattle herd in Washington, and two cases of infection of atypical BSE in Alabama and Texas (Gates et al., 2010; J. Rankin, personal communication).

Research has shown that an animal becomes infected with BSE through the consumption of feed that is derived from infected animals (MDOL, 2010b). In order to prevent BSE in Montana, the MDOL issued an official order in March 2001, which states that "animal protein derived from mammalian tissues shall be prohibited in ruminant feeds

in Montana" (Official Order No. 02-01-001). "Protein derived from mammalian tissues means any protein portion of mammalian animals, excluding blood and blood products; gelatin; inspected meat products which have been cooked and offered for human food and/or further heat-processed for feed (such as plate waste and used cellulosic food casings); milk products (milk and milk proteins); and any product whose only mammalian protein consists entirely of porcine or equine protein" (Official Order No. 02-01-001).

BSE is a chronic degenerative disease that affects the central nervous system of the infected animal. The signs of BSE include abnormal posture, difficulty rising, a decrease in coordination, progressive weight loss, decreased milk production, low-level tremors, and possibly changes in temperament, such as nervousness or aggression (Gates et al., 2010; MDOL, 2010b). Though there are tests for BSE, there is not a treatment, and death tends to occur within six months of infection (MDOL, 2010b). The human consumption of BSE-contaminated food causes the new variant Creutzfeldt-Jakob disease, which is fatal in humans (Gates et al., 2010).

Bovine Tuberculosis

Bovine tuberculosis (BTB) is a chronic and progressively debilitating contagious disease caused by a bacterium that is part of the Mycobacterium group (MDOL, 2010e). There are three types of bacteria in the Mycobacterium group: *Mycobacterium bovis, Mycobacterium avium,* and *Mycobacterium tuberculosis* (MDOL, 2010e; Gates et al., 2010). BTB is caused by the bacterium *Mycobacterium bovis* and has the largest range of hosts of the Mycobacterium group with the ability to infect all warm-blooded vertebrates (MDOL, 2010e). Cattle and bison are the primary hosts for BTB, making them susceptible to infection and allowing the bacteria to grow and spread within them under natural conditions (Gates et al., 2010). BTB is zoonotic and therefore can be transmitted from livestock and wildlife to humans (MDOL, 2010e). Human infection is rare, and treatment requires six to nine months of antimicrobial drugs, but the treatment success rate is more then 95 percent (Gates et al., 2010). BTB replicates and grows within a host, and can only survive outside a host for a few weeks (MDOL, 2010e). The bacteria cannot tolerate prolonged exposure to heat, direct sunlight, or dry conditions, but under cold, dark, and moist conditions it may survive longer outside a host (MDOL, 2010e).

An animal or human becomes infected with BTB primarily though inhalation or ingestion of unpasteurized milk (J. Rankin, personal communication). Microscopic droplets, or aerosols, containing the BTB bacteria are expelled from the infected animal through exhaling or coughing, and then may be inhaled by a susceptible animal or human (MDOL, 2010e). Enclosed spaces, such as barns, increase the risk of infection from inhalation (MDOL, 2010e). Humans may also be infected if they have cuts or abrasions and come into contact with infected animals or their meat (Clifton-Hadley et al., 2001). Infection of offspring may occur through ingestion of contaminated milk, and humans may become infected through the consumption of unpasteurized milk from infected cows (MDOL, 2010e). Infection may also occur through communal water sources contaminated with saliva or other discharges from infected animals (MDOL, 2010e). Once the bacterium has

entered a new host, it may take many months to develop due to its slow growth rate. It is also possible for the bacteria to remain dormant within a host without causing the disease (MDOL, 2010e).

The signs and symptoms of BTB often do not become apparent until the advanced stages of the disease (MDOL, 2010e). Upon inhalation the bacteria enters the terminal bronchi and from there moves to the blood and lymph system, which allows it to spread throughout the body, causing chronic lesions that may become necrotic, calcified, and caseous (Clifton-Hadley et al., 2001; Gates et al., 2010). The clinical signs of the disease depend on which organs are affected, which can include respiratory, digestive, urinary, nervous, skeletal, and reproductive systems (Clifton-Hadley et al., 2001; Gates et al., 2010). As infection progresses it leads to a generalized stage, which causes weakness, debility, a reduction in fertility, and eventually death (Clifton-Hadley et al., 2001; Gates et al., 2010).

The immune response that results from a BTB infection allows detection of the disease through tuberculin skin tests, though this is not an effective test for wildlife as it requires a three-day waiting period for the results and produces false positives that require further testing (MDOL, 2010e; N. Anderson, personal communication). There is not an effective vaccine for BTB (Gates et al., 2010). There is evidence that individual domestic animals can be treated though the long-term use of antibiotics (Gates et al., 2010). This treatment is not practical for wildlife due to the need for long-term containment and the high cost of therapy (Gates et al., 2010). The MDOL (2010e) recommends controlling BTB within a domestic herd through repeated testing and culling of infected individuals; however, since this method is not guaranteed, they then recommend herd depopulation. Maintaining a closed herd may also reduce infection.

Current endemic infection within bison has only been documented in and around Wood Buffalo National Park in Canada (Clifton-Hadley et al., 2001; Gates et al., 2010). There have not been reported cases of BTB within bison herds in Montana. BTB has previously been detected in a few game farms within Montana. In the mid-1990s a mule deer and a coyote located near a BTB-infected game farm near Hardin tested positive for BTB (N. Anderson, personal communication).

Bovine Viral Diarrhea

Bovine viral diarrhea (BVD) is a disease caused by a virus that is a member of the *Pestivirus* genus (Van Campen et al., 2001). BVD infects a variety of domestic and wild ruminants (Loken, 1995; Van Campen et al., 2001). The virus is very common in cattle in North America, but there are few confirmed cases of pestivirus-caused disease in free-ranging ruminants, and no evidence that these viruses have significant population impact (Van Campen et al., 2001; Gates et al., 2010). BVD poses no known threat to humans (Gates et al., 2010). The virus is mainly transmitted to wildlife through interactions with domestic livestock, as the principal reservoirs of BVD are persistently infected cattle and sheep (Van Campen et al., 2001; Gates et al., 2010). BVD is transmitted from persistently infected animals to susceptible animals through direct contact, aborted fetuses, fetal membranes,

secretions, and shared food and water sources (Van Campen et al., 2001). It is believed that transmission of pestiviruses by acutely infected animals is inefficient (Van Campen et al., 2001). The factors that influence the persistence of BVD in a population are size and density, herd behavior, the timing of reproduction, and the survivorship of offspring (Van Campen et al., 2001). There is evidence of BVD in bison in the Greater Yellowstone area, and positive antibodies were detected in 31 percent of tested bison within Yellowstone National Park (Williams et al., 1993; Taylor et al., 1997). Pastoret et al. (1988) suggest that wildlife do not play a determinant role in the transmission of BVD to domestic livestock.

Infection within wild ruminants and cattle depends upon the immune status of the animal, the route of transmission, and the virulence of the isolate (Van Campen et al., 2001). The infection in cattle and sheep is usually subclinical (Van Campen et al., 2001). In cattle, acute infection typically occurs in younger animals and tends to be associated with diarrhea (World Organization for Animal Health, 2009). In an acute infection cows may suffer from infertility, and bulls may show a temporary decrease in fertility with the ability to shed the virus in their semen (World Organization for Animal Health, 2009). Some infections may cause fetal malformations, weakened offspring, death, or abortion in pregnant animals (Van Campen et al., 2001). Though rare, persistently infected cattle may eventually develop mucosal disease, which results in severe diarrhea, dehydration, fever, and loss of appetite, and often leads to death (Van Campen et al., 2001; World Organization for Animal Health, 2009).

There are tests to determine the presence of the antibodies that occur during exposure to BVD, and domestic livestock can be vaccinated (Van Campen et al., 2001). Animals can develop immunity to the virus, though there does not appear to be a proven treatment (Van Campen et al., 2010). Maintaining a closed herd and quarantining replacement stock is recommended to reduce the chance of infection. Maintaining a clean environment and preventing contact with biological waste and birthing fluids can also reduce the risk of infection (Van Campen et al., 2001).

Johne's Disease

Johne's disease, or paratuberculosis, is caused by the bacterium *Mycobacterium avium,* subspecies *paratuberculosis,* which occurs worldwide in a variety of wild and domestic ruminants including bison, cattle, and sheep (Buergelt et al., 2000; Williams, 2001; Gates et al., 2010). Johne's disease typically enters a herd when a healthy but infected animal is introduced. An animal is most susceptible to the disease during the first year of life. Infection occurs when the newborn swallows a small amount of infected manure from the birthing environment or the udder of an infected mother. Infection may also occur while the animal is in the uterus or through the milk and colostrum (APHIS, 2010b). The bacterium that causes the disease may survive in the soil or water after fecal contamination for over a year, but will not grow and multiply outside the host species (APHIS, 2010b). Humans are not at risk of Johne's disease from either livestock or wildlife (Gates et al., 2010).

The clinical signs of the disease rarely present themselves until two or more years after the initial infection. Johne's disease primarily affects the small intestine of ruminants, and signs of the disease include weight loss and diarrhea with a normal appetite, decreased milk production, and mortality (Gates et al., 2010; APHIS, 2010b). Once an animal has begun to show signs of the disease, it can shed the bacteria within its feces (Gates et al., 2010). There is no known treatment for Johne's disease, and the disease typically leads to mortality. Certain cattle herds are enrolled in the Voluntary Johne's Disease Herd Status Program, which identifies herds of low risk through repeated testing. The best ways to prevent the spread of Johne's disease are the maintenance of a clean birthing environment, the removal of females that test positive from the herd, the removal or culling of offspring born to infected females, implementation of practices to prevent manure contamination of feed, and replacement of stock from low-risk herds (Gates et al., 2010; APHIS, 2010b). Within bison, there have not been reports of Johne's disease within conservation herds, though some commercial herds have had cases (Gates et al., 2010).

Malignant Catarrhal Fever (sheep associated)

Malignant catarrhal fever (sheep associated)(MCF) is a disease caused by a virus of the genus *Rhadinovirus* (Gates et al., 2010). There have been at least ten MCF viruses recognized worldwide, and five have been linked to disease within sheep, goats, cattle, and pigs (Gates et al., 2010). Within bison, MCF is caused by infection of the ovine herpes virus type two. Ovine herpes virus type two's natural host is domestic sheep, and though domestic sheep carry the virus they do not express the disease (Heuschele and Reid, 2001; Gates et al., 2010). Testing has indicated that the virus is common in the United States in domestic goats (61 percent) and sheep (53 percent) (Li et al., 1996; Gates et al., 2010). Non-natural host animals that develop MCF are not considered contagious and may be dead-end hosts (Heuschele and Reid, 2001). MCF infection in bison is highly lethal, with almost 100 percent mortality within an infected herd (Schultheiss et al., 2001). Studies have shown that bison herds that are not associated with domestic sheep do not show evidence of MCF (Gates et al., 2010). There is no evidence that MCF is infectious in humans (Heuschele and Reid, 2001; Gates et al., 2010).

After initial infection, sheep experience periodic reactivation episodes in which they can transmit the virus (Heuschele and Reid, 2001). Inhalation of aerosol droplets and ingestion of food contaminated with the virus through feces, nasal secretions, and tears are the most common modes of transmission (Heuschele and Reid, 2001). Bison mostly become infected through direct contact with domestic sheep, though MCF was reported in bison herds that were located 3 miles from a lamb feedlot (Schultheiss et al., 2001; Gates et al., 2010).

MCF is expressed in two forms in bison, acute and chronic. In acute cases the bison typically dies within seven to ten days of infection, or within 48 hours of showing symptoms. In some cases death may not occur for as long as two months following infection (Schultheiss et al., 2001; Gates et al., 2010). It is also possible for an infected animal to survive, but remain persistently infected (Schultheiss et al., 2001). Clinical signs

of MCF within bison include hemorrhagic cystitis, colitis, conjunctivitis, ocular discharge, nasal discharge, excess salivation, loss of appetite, diarrhea, melaena, haematuria, multifocal ulceration of the oral mucosa, fever, circling, ataxia, blindness, lameness, and difficultly urinating (Liggitt et al., 1980; Schultheiss et al., 1998; Heuschele and Reid, 2001; Gates et al., 2010).

It is possible to test for the presence of infection, though there is currently no vaccine or effective treatment for MCF (Heuschele and Reid, 2001). In order to reduce the spread of MCF from domestic livestock to bison, bison should not be grazed in the same pastures or adjacent to sheep pastures, especially during lambing periods (Heuschele and Reid, 2001; Gates et al., 2010).