Diseases of Importance of Domestic Ruminants and Free-ranging North American Cervids

Terry R. Spraker, *DVM, PhD, DACVP*
College of Veterinary Medicine and Biological Sciences, Colorado State University, Fort Collins, CO 80523

Abstract

Numerous diseases of importance in domestic ruminants and free-ranging wildlife can be transmitted between each other. This section will be limited to diseases that are known to affect both wild and domestic animals. In most cases, the disease can be transmitted in both directions and have important impacts in both domestic and free-ranging animals. In some cases the disease is primarily in domestic animals and spills over into wildlife, and in others the disease is primarily in wildlife and spills over into domestic animals. Diseases covered in this presentation include tuberculosis, brucellosis, anthrax, paratuberculosis, malignant catarrhal fever, bluetongue/epizootic hemorrhage disease, elaeophoriasis and bovine virus diarrhea.

Résumé

La maladie débilitante chronique (MDC) est une encéphalopathie spongiforme transmissible qui a été signalée chez le cerf mulet (*Odocoileus hemionus hemionus*), le cerf de Virginie (*Odocoileus virginianus*), le wapiti des Rocheuses (*Cervus elaphus nelsoni*) et l'élan du Yellowstone (*Alces alces shirasi*), chez des sujets en captivité et en liberté. La MDC a été signalée chez des cervidés en liberté de 11 états américains et de deux provinces canadiennes. La MDC a été décelée chez des cervidés en captivité dans huit états américains et deux provinces canadiennes, mais tous ces troupeaux ont été dépeuplés à l'exception de trois troupeaux du Colorado. Le mode de transmission de la MDC n'a pas été élucidé, mais on croit qu'il serait associé à l'ingestion d'aliments contaminés ou d'eau provenant d'un milieu contaminé. Les principaux signes cliniques de la MDC chez les cervidés comprennent la perte pondérale et l'hypersalivation. Les lésions macroscopiques primaires incluent l'émaciation avec perte de tissus adipeux, tandis que la lésion histologique classique de la MDC est l'encéphalopathie spongiforme. Le diagnostic ne peut être confirmé que par l'examen du cerveau et/ou par coloration immunohistochimique des tissus lymphoïdes. Les tests ELISA sont utilisés pour l'analyse d'échantillons en grand nombre, par exemple lors d'enquêtes sur les animaux tués à la chasse.

Tuberculosis

General Comments: Numerous mycobacterial diseases of animals are transmitted between wild and domestic animals. The mycobacteria are divided into two general categories: a few obligate pathogens and numerous free-living saprophytes. These saprophytes are probably the cause of many of the inconclusive or false-positive TB reactions found in wild and domestic animals. The most important mycobacterial disease in animals is *Mycobacterium bovis*.

Etiology: *Mycobacterium bovis* is a non-spore-forming, non-motile, gram-negative, acid-fast bacterium.

Susceptible Host: Most ruminants (all species of deer and elk, moose, caribou, reindeer, wood and plains bison, African buffalo, feral water buffalo), coyotes, humans, feral ferrets (New Zealand), badgers (England and Europe), Australian brushtail possum (New Zealand) are susceptible.

Distribution: Worldwide. Important hosts in US include free-ranging white-tailed deer in Michigan; captive elk, fallow deer and white-tailed deer industries.

Transmission: Routes of infection are respiratory and oral. Survival of *M. bovis* in the environment is variable. *M. bovis* can survive at least five months in cold, wet conditions; however, in dry conditions with exposure to sunlight it only can survive about a month.

Clinical Signs: Some animals never show clinical signs; others show subacute to chronic signs from six months post-infection to several years. Most common sign is emaciation; coughing and respiratory rales are rare.

Postmortem Lesions/Histopathology: Granulomas, abscesses, and enlarged retropharyngeal, mediastinal and mesenteric lymph nodes are common. Lesions in the tonsils, lungs, intestine, thoracic cavity and spleen may be found. Histopathology: proliferative granulomas with central caseation. Calcification occurs with duration of lesion.

Diagnosis: Tuberculin skin testing, as done in cattle, is not as specific as in cattle. Better site preparation and care with the intradermal injection is required. Most people now use multiple tests including the intradermal skin test, a lymphocyte transformation test, ELISA and serum haptoglobin levels. In addition,

an immunohistochemical stain has been developed that can differentiate between several species of mycobacterium in formalin-fixed tissues.

Treatment: There has been no effective treatment of TB in wild animals.

Prevention and Control: Primary focus of control has been to reduce the density of the animals affected, for example the badgers in England and the white-tailed deer in Michigan. Also with the deer, elimination of winter feeding has been done. It has been found in Michigan that coyotes are susceptible, but the role in transmission is unclear.

Brucellosis

General Comments: *Brucella melitensis* is the type species, and primarily occurs in sheep and goats. *B. abortus* primarily occurs in cattle and has eight different biovars (1,2,3,4,5,6,7 and 9). *B. suis* has four biovars and usually affects swine, however, biovar 4 affects reindeer and caribou, and biovar 2 affects European rabbits. Other species of Brucella include *B. ovis* that affects sheep, *B. canis* that affects dogs, *B. neotomae* that affects desert wood rats and a *Brucella* species that has been found in marine mammals. *B. abortus* will be the primary focus of this discussion.

Etiology: *B. abortus* is a non-spore-forming, nonmotile, gram-negative, aerobic/microaerophilic coccobacillus.

Susceptible Host: Most mammals are susceptible (documented in at least 100 species).

Distribution: Worldwide

Transmission: Most via ingestion of contaminated feed (milk), licking an infected fetus, calf or placenta, or licking the genitalia of an infected cow shortly after birth. Transmission can also occur via inhalation or through the conjunctival sac.

Clinical Signs: Elk and bison: abortion, retained placenta, enlarged testicles, pendulous scrotum, hygromas.

Postmortem Lesions/Histopathology: Lesions in elk and bison are usually not dramatic or diagnostic and include placentitis, metritis, orchitis, epididymitis, bursitis and synovitis.

Diagnosis: Indirect method of diagnosis is serology. To confirm a diagnosis, the organism has to be cultured.

Treatment: No treatment for wild or domestic animals.

Prevention and Control: The primary method for prevention and control is to test and slaughter. A fairly large vaccination program which has been attempted in the greater Yellowstone elk herds with S19 does seem to show some success.

Anthrax

General Comments: Anthrax is thought to be the oldest disease recognized in ruminants. Some think one of the plagues of Egypt that killed most of the cattle, horses and camels recorded in the Book of Exodus in the Bible was anthrax. The disease and its effect on cattle and humans have also been described in ancient Greece by Homer (1000 B.C.), Hippocrates (400 B.C.) and Galen (200 B.C.). Most of the early work of Louis Pasteur and Robert Koch was done with the anthrax bacillus.

Etiology: *Bacillus anthracis* is a spore-forming, non-motile, gram-positive (rod) bacterium.

Susceptible Host: Most homoeothermic species are susceptible. Species with a high body temperature, such as birds, are usually resistant. The ostrich is the only bird confirmed with anthrax.

Distribution: Worldwide

Transmission: Spores seem to concentrate in low-lying depressions and rock-land seep areas with high moisture content, high organic content in the soil and an alkaline pH. There is no good evidence to show that spores multiply in the environment. A common misconception is that the vegetative cells sporulate in response to atmospheric oxygen, low-nutrient conditions and dehydration. Epizootics usually occur in dry seasons when animals graze close to the ground and concentrate on small, drying water holes. Scavengers (birds and carnivores) may ingest contaminated carcasses and spread the spores through their feces. Necrophilic insects feeding on contaminated carcasses can also spread the spores to vegetation. Insects feeding on the blood of animals dying of anthrax can also transmit the disease to susceptible hosts.

Clinical Signs: In herbivores, clinical signs are usually characterized by sudden onset, fever, rapidly progressing debility, respiratory distress, disorientation and death within a few hours to several days.

Postmortem Lesions/Histopathology: A direct blood smear should be done on all carcasses suspected of having anthrax before a necropsy is done (before the carcass is opened). If large gram-positive rods are found, the carcass should not be opened. Lesions of anthrax usually involve the reticuloendothelial and vascular systems. The blood is dark and thick, clots poorly and flows freely from the nose and rectum. Hemorrhage on serosal surfaces is common. Edema in subcutaneous tissues, trachea and lungs are common. The spleen is dark, enlarged and friable.

Diagnosis: Direct blood smear of the carcass with the finding of large, gram-positive rods with spore formation is suggestive of anthrax, but culture is needed for confirmation.

Treatment: In animals, treatment is not usually done. In humans, most broad-spectrum antibiotics are effective.

Prevention and Control: Vaccination is the primary method of preventing and controlling anthrax in domestic animals.

Paratuberculosis (Johne's Disease)

General Comments: Paratuberculosis, or Johne's Disease is a well recognized disease of both domestic and wild ruminants. It has been known in captive deer for over a hundred years.

Etiology: The etiological agent for Johne's Disease is *Mycobacterium avium paratuberculosis.* The microorganism is an acid-fast bacterium considered to be a subspecies of *M. avium.*

Susceptible Host: Many species of cervidae, bovidae and camelidae are susceptible. Johne's disease has also been found in rabbits and Stump-tailed macaques. Johne's disease is most common in domestic animals, but has been reported in Bighorn Sheep in Colorado and Wyoming, Rocky Mountain goats in Colorado, Tule elk in California and Key deer in Florida.

Distribution: Worldwide.

Transmission: The intestine and mesenteric lymph nodes are the primary tissues affected. The organism is then shed in the feces and contaminates the ground. Susceptible hosts then ingest the organism. The organism then invades the intestinal mucosa and spreads to the regional lymph nodes. It seems that the terminal ileum and adjacent lymph nodes are the locations affected first.

Clinical Signs: Johne's disease is a chronic, prolonged illness that can lasts for years. The most common clinical signs of Johne's disease are chronic diarrhea and emaciation. However, chronic diarrhea is not always present, but often intermittent in many species, especially in sheep, mouflon sheep and goats.

Postmortem Lesions/Histopathology: Typical lesions include emaciation, serous atrophy of fat, intermandibular edema, increased pericardial, thoracic and abdominal fluids. The most classic lesions are thickening of the intestinal mucosa with markedly enlarged mesenteric lymph nodes. Main histological lesions include granulomatous enteritis with the demonstration of acid-fast organisms. The lesions are most prominent in the mucosa of the lower small intestine and mesenteric lymph nodes.

Diagnosis: Antemortem diagnosis is difficult; best is postmortem examination. Gross lesions of a thickened wall of the small intestine are suggestive, but histopathology is necessary. Culture of the organism is difficult and time-consuming. Many times months are required for a positive culture, and oftentimes the organism does not grow. Immunohistochemical staining techniques have been described for Johne's disease. These techniques are most commonly used to differentiate between bovine TB and other acid-fast bacterial infections.

Treatment: There is no good treatment for Johne's disease. Extensive long-term treatment could possibility be given to endangered animals, but probably will be of little success.

Prevention and Control: There is little success in controlling Johne's disease in captive herds, much less free-ranging populations. Control and prevention are dependent on management of the herd, such as, do not move infected animals and do not bring clean animals into infected and contaminated areas. It is also important to keep the density of the animals low, whether dealing with a captive or free-ranging herd of animals

Malignant Catarrhal Fever

General Comments: Malignant catarrhal fever (MCF) is an important disease of domestic animals and bison. MCF does occur in captive Artiodactyla, but is usually rare in free-ranging animals. There are two general categories of MCF, the Wildebeest-origin form and the domestic sheep-associated form.

Etiology: MCF is caused by a highly cell-associated, lymphotrophic herpesvirus. This virus has been classified as a member of the family *Herpesviridae,* subfamily *Gammaherpesvirinae.*

Susceptible Host: Numerous species in the families *Antilocapridae*, *Bovidae*, *Camelidae, Cervidae* and *Tragulidae* are susceptible. Some species appear to be extremely sensitive to the disease such as bison, mule deer, and white-tailed deer, whereas other species appear to have more resistance and only become seropositive following exposure.

Distribution: Worldwide. The Wildebeest-origin is most common in Africa. The sheep-associated form is most common in North America and Europe.

Transmission: Several means of transmission have been shown, including transplacental and inhalation of the virus by the calf from the dam during the first week of life. Transmission can also occur via ingestion of contaminated forage from nasal secretions, tears and feces from infected animals. The transmission of sheep-associated herpes virus, especially to bison, is not as well known, but similar means of transmission probably occurs.

Clinical Signs: Clinical MCF has been arbitrarily separated into four categories: peracute, intestinal, head and eye form, and mild forms. The peracute form is characterized by severe inflammation of the oral and nasal mucosa, hemorrhagic gastroenteritis and death

usually within several days. The intestinal form is characterized by pyrexia, diarrhea, hyperemia of the oral and nasal mucosa with nasal discharge, and moderate to severe lymphadenopathy. Animals often live about a week with this form. The head and eye form is characterized by pyrexia and nasal and ocular discharges which progress from serous to mucopurulent. This often causes encrustations around the nostrils, causing dyspnea, open-mouth breathing and drooling. There is intense hyperemia and multifocal areas of necrosis of the oral mucosa. Ocular signs include lacrimation progressing to purulent exudation, photophobia, hyperemia, edema of the palpebral conjunctiva and injection of the scleral vessels. A well recognized sign is corneal opacity starting peripherally and progressing centripetally, resulting in partial or complete blindness. Pyrexia is commonly high until the animal becomes moribund. Clinical signs of the mild forms are syndromes produced by experimental infections of cattle with attenuated viruses, are usually not fatal and probably occur in the most resistant hosts. This form of MCF is often missed.

Postmortem Lesions/Histopathology: The postmortem lesions vary considerably and are somewhat consistent with the form of MCF. With the peracute form, few lesions other than hemorrhagic enteritis are found. There is often severe ulceration of the oral mucosa and opacity of the eyes. The mucosa of the frontal sinuses is usually congested. Generalized lymphadenopathy is typical. Hemorrhages within the mucosa of the urinary bladder are common. Vasculitis with fibrinoid necrosis is the hallmark histological lesion associated with MCF. This lesion is not pathognomonic, but highly suggestive of MCF. This vasculitis may be mild to severe. The best location to identify vasculitis is in the vessels of the choroid plexus of the brain; other tissues of importance are the kidney, liver, adrenal glands and brain.

Diagnosis: The diagnosis of MCF is primarily based on clinical signs, postmortem lesions and the finding of vasculitis with fibrinoid necrosis on histopathology. A real-time PCR test has been developed for the sheep-associated herpesvirus and is commonly used in the US.

Treatment: The only treatment is supportive.

Prevention and Control: The primary prevention/control technique used with MCF is to limit exposure to domestic sheep in North America.

Bluetongue/Epizootic Hemorrhage Disease

General Comments: Bluetongue (BT) and epizootic hemorrhage disease (EHD) are an *Orbivirus* infection of wildlife and domestic animals. Since they produce similar lesions in affected animals, these two diseases, although caused by several different strains of virus, are collectively referred to as hemorrhage disease (HE). Bluetongue is usually mild in sheep and cattle, but at times can cause high mortality in sheep, whereas EHD can cause high mortality in free-ranging mule deer, white-tailed deer and Bighorn sheep. First cases similar to HD were noted in the late 1800s and early 1900s in deer. HD is now observed annually in the US in white-tailed deer.

Etiology: The etiology of HD is several strains of the genus *Orbivirus* in the family *Reoviridae*. These viruses are vector-borne, double-stranded RNA viruses. There are 24 serotypes of BT virus and 10 serotypes of EHD virus worldwide. However, BT serotypes 10, 11, 13 and 17 and EHD serotypes 1 and 2 are important in the US.

Susceptible Host: HD primarily affects white-tailed deer and mule deer, but has been reported in pronghorn antelope, desert bighorn sheep and Rocky Mountain bighorn sheep. Rarely, HD has been found in bison and mountain goats. The BT virus has affected zoological ruminants including greater kudu, muntjac, Grant's gazelle, gemsbok, sable antelope, African buffalo, ibex, hartebeest and addax.

Distribution: BT and EHD are primarily diseases of North America.

Transmission: HD is known to be vector-borne. The primary vector in North America is *Culicoides* midges. The two most important midges are *C. variipenis* and *C. insignis.*

Clinical Signs: Infection does not always result is disease and often is asymptomatic. If signs occur, they are similar with BT and EHD. HD can be either acute with severe signs to chronic with mild signs, and the disease may or may not be fatal. Signs are often severe: hyperemia of the skin and mucous membranes; swelling of the face, conjunctiva, and neck; anorexia; lethargy; weakness with blood-tinged saliva; nasal discharge; hemorrhagic diarrhea; lameness; hemorrhage at the coronary band, skin, buccal papillae, and oral ulceration. Most animals only show a few of these signs, which are at first mild, then rapidly progress.

Postmortem Lesions/Histopathology: Gross lesions vary and include edema, multifocal hemorrhages and ulcerations. Mild to severe pleural, peritoneal and pericardial effusions are common. Pulmonary edema, edema of subcutaneous tissues and fascial planes are common. Petechial and ecchymotic hemorrhages are a prominent feature, especially of the digestive system and heart. Hemorrhages can be seen in any organ system. Petechia of the tunica media of the pulmonary artery and serosal surface of the pyloric region of the abomasum are highly suggestive of HD. Erosions and ulcerations are common throughout the entire digestive system. Primary histological lesions include congestion, hemorrhage, thrombosis and necrosis.

Diagnosis: Serological tests including agar gel immunodiffusion, serum neutralization and competitive enzyme-linked immunosorbent assays have been used to detect exposure of the animal to HD. Diagnosis primarily involves gross and histological lesions. Confirmation of HD is by virus isolation.

Treatment: The only treatment is supportive. Treatment of wild animals is not done.

Prevention and Control: Management strategies are not available to prevent, predict, or minimize HD in free-ranging populations.

Elaeophoriasis

General Comments: Elaeophoriasis is a disease caused by a filarial nematode that normally infects mule deer, but can be fatal should it infect Rocky Mountain elk, moose, domestic sheep and goats, and llamas. An old name for this disease in elk was "clear-eyed blindness". Due to management changes in sheep and goats, elaeophoriasis is not as common as it was 20 to 30 years ago.

Etiology: *Elaeophora schneideri*, a filarial nematode.

Susceptible Host: *E. schneideri* can infect and cause death in Rocky Mountain elk, domestic sheep and goats, white-tailed deer and llamas. *E. schneideri* has been found in moose, but has not been shown to be fatal in this species.

Distribution: Current information suggests that elaeophoriasis is most common in animals higher that 6,000 feet in elevation; however, there are a few exceptions. Elaeophora has been documented in New Mexico, Arizona, California, Colorado, Wyoming, Oregon, Utah and Montana.

Transmission: *E. schneideri* is transmitted by species of horse flies of the genera *Hybomitra* and *Tabanus*. These horse flies primarily feed on the face and legs of the host. Flies pick up third-stage larvae that are located within the subdermal tissues. The microfilariae are too large to circulate in the blood. When ingested by the horse fly, the microfilariae migrate to the fat bodies lining the fly's abdominal wall. Within two weeks, the microfilariae reach the infective stage. These third-stage infective larvae then migrate to the head and mouth parts of the horse fly, and when it feeds again, these infective third-stage larvae enter the host. In the natural definitive host (mule deer), these larvae travel to the leptomeninges for further development into mature fourth and early fifth-stage larvae. The early fifth-stage larvae then migrate retrograde to the carotid artery, mature, mate and produce microfilariae. The microfilariae are released into the bloodstream and are trapped in the capillaries of the head. Horse flies then feed in these areas, acquire an infection and the cycle starts over again. In the abnormal host (elk, sheep, goats, etc.) many of the microfilariae go to the leptomeningeal arteries, but some go to small arteries of the eyes, ears, nose, kidneys and even legs. Here, they grow and lead to fibromuscular intimal proliferation and thrombosis, which leads to infarction and necrosis.

Clinical Signs: Clinical signs are not observed in mule deer, but have been primarily described in calf elk, lambs and kid goats. The primary clinical sign in sheep and goats is poll dermatitis. If infection is high in young lambs and goats, sudden unexpected death can occur usually due to severe cerebral infarctions. Signs in elk include bilateral blindness, circling when the animal moves, poor coordination, nystagmus, necrosis of the muzzle and nostrils, dry gangrene of the ear tips, abnormal antler growth and emaciation.

Postmortem Lesions/Histopathology: Granulomatous dermatitis of the skin of the poll is the primary gross and histological lesions found in sheep and goats. Rarely, parasites are observed on histopathology. In some cases, infarctions of the brain and fibromuscular intimal proliferation of cerebral vessels can be found. The cephalic arterial system is the primary target in elk. Primary lesions include thrombosis, fibromuscular intimal proliferation, ischemic necrosis of numerous tissues, and fragments of viable and nonviable parasites.

Diagnosis: Clinical signs with postmortem and histological lesions are highly suggestive of elaeophoriasis. It is important to find the parasites in the carotid arteries and identify them.

Treatment: There is no treatment for deer and elk. Little to no literature has been published for treatment of elaeophoriasis in sheep and goats. Some have treated these animals with various drugs, but killing the parasites may lead to thrombosis of cerebral vessels and death of the host.

Prevention and Control: There is no available management tool to control *E. schneideri* in the wild. Avoiding grazing of sheep and goats at high elevations will control the disease in these species.

Bovine Virus Diarrhea

General Comments: *Pestivirus* are a group of viruses that infect at least 173 ruminants and 11 species of pigs. The three primary conditions in this family of viruses include bovine virus diarrhea virus (BVDV) of cattle, border disease virus (BDV) of sheep and classic swine fever virus (CSFV). At present, there is no evidence that these viruses have a significant impact in free-ranging populations of ruminants. However, they do have a significant economic impact on livestock production worldwide and could serve as carrier hosts.

Etiology: Bovine viral diarrhea virus is a single-stranded RNA virus with a single open reading frame

encoding for four structural and six non-structural proteins. The virions are pleomorphic, spherical structures 50-60 nm in diameter, with a bilaminar envelope of cellular origin surrounding a semidense core of 20-25 mm diameter. Virions mature within intracytoplasmic membranes, and virus is liberated by exocytosis of virus-containing membrane vesicles. Bovine viral diarrhea virus and BDV have two biotypes: noncytopathic (ncp) and cytopathic (cp). Disease is usually caused by the cytopathic biotypes, while the noncytopathic viruses are not usually pathogenic.

Susceptible Host: Primary evidence for BVD infection in wild ruminants is serological evidence and virus isolation. Serological surveys in free-ranging and captive populations have confirmed prior infection with BVDV in over 40 species in North America, Africa, Europe and Australia. Some of these reported species are not associated with virus isolation, and therefore skeptically viewed.

Distribution: Probably worldwide, but has only been reported in North America, Africa, Europe and Australia.

Transmission: Primary reservoirs of BVDV and BDV are persistently infected cattle and sheep. Because these reservoirs have such high titers of virus, they shed it in many secretions, such as nasal and ocular. Virus is also present in high titers in aborted fetuses, fetal membranes and uterocervical fluids, and can spread to other animals by close contact with these infected tissues. The role wildlife play as reservoirs is not known, but at present it is believed that domestic livestock are the reservoirs for wildlife, and not visa-versa. However, some believe there are independent cycles of these viruses in wild populations that do not involve cattle or sheep.

Clinical Signs: While relatively little is known about the clinical signs of *Pestivirus* infections in free-ranging ruminants, the clinical signs in cattle and sheep have been well documented and will be covered here. Acute infection is often asymptomatic, but can result in peracute death or a hemorrhagic disease. *Pestivirus* infections can also lead to abortion, fetal malformations, stillbirths and weakened neonates, as well as infertility and prolonged breeding and birthing intervals. Transplacental infection has not been documented in free-ranging wildlife, but mummified fetuses, stillbirths and normal healthy fawns have been seen in white-tailed deer experimentally infected with cytopathic BVDV. BVDV has also been isolated from Rocky Mountain bighorn sheep that died with a hemorrhagic pneumonia. Persistently infected cattle often develop mucosal disease characterized by severe diarrhea, dehydration and fever. The morbidity of mucosal disease is low, but the mortality is high. Clinical signs experimentally produced in reindeer include transient mild diarrhea, coronitis and laminitis. Clinical signs observed in free-ranging roe deer include weakness, lack of fear of man, impaired hearing and vision, dehydration and emaciation. Pyrexia, anorexia, salivation and nasal discharge have been observed.

Postmortem Lesions/Histopathology: Gross and histopathological lesions have been experimentally produced in reindeer and roe deer. These include emaciation, erosion and ulceration of the oral mucosa, hemorrhagic enteritis, interdigital ulceration and inflammation of the coronary band.

Diagnosis: Diagnosis using clinical signs and gross and histological lesions has to be used with caution, because they are similar to BT and EHD. Tests that must be used include virus isolation, demonstration of viral antigen in tissues by immunofluorescent antibody staining or immunohistochemistry, or detection of viral RNA. Virus is best isolated from the buffy coat cells, plasma, serum or nasal secretions collected early in the disease from clinical cases. Tissues that should be collected at postmortem examination include thymus, spleen, lung, liver, mesenteric lymph nodes, tonsils, intestines and kidney.

Treatment: Treatment is supportive only.

Prevention and Control: The primary strategy for control of BVDV in free-ranging and captive populations of wild ruminants is to keep them separated from cattle and sheep.

Suggested Readings

Clifton-Hadley RS, Sauter-Louis CM, Lugton IW, Jackson R, Durr PA, Wilesmith JW: Mycobacterial disease, in Williams ES, Barker IK (eds): *Infectious Diseases of Wildlife*, ed 3. Ames, Iowa State University Press, 2001, pp 340-355.

Gates CC, Brett E, Dragon D: Anthrax, in Williams ES, Barker IK (eds): *Infectious Diseases of Wildlife*, ed 3. Ames, Iowa State University Press, 2001, pp 396-409.

Hibler CP, Adcock JL: Elaeophoriasis, in Davis JW, Anderson RC (eds): *Parasitic Diseases of Wild Mammals*, ed 3. Ames, Iowa State University Press, 1971, pp 157-163.

Heuschele WP, Reid HW: Malignant Catarrhal, in Williams ES, Barker IK (eds): *Infectious Diseases of Wildlife* ed 3. Ames, Iowa State University Press, 2001, pp 157-163.

Howerth EW, Stallknecht DE, Kirkland PD: Bluetongue, epizootic hemorrhagic disease, and other Orbivirus-related diseases, in Williams ES, Barker IK (eds): *Infectious Diseases of Wildlife*, ed 3. Ames, Iowa State University Press, 2001, pp 77-91.

Thorne ET: Brucellosis, in Williams ES, Barker IK (eds): *Infectious Diseases of Wildlife*, ed 3. Ames, Iowa State University Press, 2001, pp 372-389.

Van Campen H, Frolich K, Hofmann M: Pestivirus infections, in Williams ES, Barker IK (eds): *Infectious Diseases of Wildlife*, ed 3. Ames, Iowa State University Press, 2001, pp 232-244.

Williams ES: Paratuberculosis and other mycobacterial diseases, in Williams ES, Barker IK (eds): *Infectious Diseases of Wildlife*, ed 3. Ames, Iowa State University Press, 2001, pp 361-396.