Bacterial diseases of farmed deer and bison

C. Mackintosh [1], J.C. Haigh [2] & F. Griffin [3]

(1) AgResearch Invermay, P.O. Box 50034, Mosgiel, New Zealand
(2) Department of Large Animal Clinical Sciences, Western College of Veterinary Medicine, 52 Campus Drive, Saskatoon, S7N 5B4, Canada
(3) Disease Research Laboratory, Department of Microbiology, University of Otago, P.O. Box 56, Dunedin, New Zealand

Summary
The most important aerobic bacterial diseases of farmed deer and bison include bovine tuberculosis, Johne's disease (paratuberculosis), yersiniosis, leptospirosis, brucellosis, pasteurellosis, anthrax, salmonellosis and colibacillosis. Anaerobic bacterial infections affecting the same animals include necrobacillosis and a number of clostridial diseases such as tetanus, blackleg, malignant oedema and pulpy kidney. The relative importance of these diseases will vary throughout the world according to timing and circumstance, but bovine tuberculosis and Johne's disease are likely to present the most significant problems with respect to diagnosis, control, trade in live animals and the establishment of wildlife reservoirs of infection. The authors summarise the aetiology, the principal species of animal affected, geographical distribution, transmission, clinical signs, pathology, diagnosis, treatment and control of these diseases.

Keywords
Bacterial diseases – Bison – Control – Deer – Diagnosis – Epidemiology – Wildlife.

Anthrax

Introduction

Anthrax, caused by the sporulating aerobic bacterium, *Bacillus anthracis*, presents as an acute or peracute disease affecting multiple species. The disease has a world-wide distribution and over the centuries, severe outbreaks have occurred in red deer (*Cervus elaphus*), wapiti (*Cervus elaphus* spp.), moose (*Alces alces*) and fallow deer (*Dama dama*) in Europe and the former Union of Socialist Soviet Republics (USSR) (14). Epidemic and sporadic forms have been reported in wild bison (*Bison bison*) and white-tailed deer (*Odocoileus virginianus*), as well as domestic animals (39). Anthrax must always be considered as a potentially serious zoonotic disease.

Transmission

In herbivores, transmission is essentially by ingestion of contaminated soil or water, and in some cases, chewing infected bones. Aerosol infection at dusting sites has been postulated as a source of infection.

Clinical signs

In the acute form, the incubation period is less than two days and the animal dies rapidly from septicaemia. Affected animals may appear dull, and show evidence of abdominal pain by grinding their teeth (3). Animals may stagger or have a stiff-legged gait, and have bloody discharges from the nostrils and anus.

Pathology

If anthrax is suspected, no autopsy should be conducted until blood films have ruled out the possibility of this disease. The reason for this is that *B. anthracis* will readily sporulate once exposed to oxygen, and these spores are exceedingly resistant to environmental decontamination, remaining viable in the soil for decades. If an autopsy is mistakenly performed, copious sero-sanguineous fluid will be observed in the body cavities, the spleen will be distended and dark, and unclotted blood will be found in the heart and major vessels. These fluids contain vast numbers of the organism, which are highly infectious for humans handling the carcass. A cutaneous form of anthrax

250

Rev. sci. tech. Off. int. Epiz., **21** (2)

results in humans, involving marked local swelling and redness. *Bacillus anthracis* is one of the organisms that may be used in biological warfare, because the spores, if inhaled, are rapidly fatal for any susceptible host.

Diagnosis

Diagnosis is confirmed by detection of characteristic spores in stained blood films. Other clinical presentations which must be distinguished from anthrax include lightning strike, trauma, poisoning, yersiniosis, malignant catarrhal fever and any peracute infectious disease.

Treatment

Treatment of affected animals is unlikely to be effective. In the face of an outbreak, the prophylactic use of high doses of penicillin in animals at risk may be effective. In areas of the world in which the disease is endemic, toxoid vaccines are successfully used for protection of wildlife and cattle, and these could be considered for use in deer and bison.

Control

Control depends on limiting exposure of deer and bison to contaminated soil or water. Animals dying of anthrax should be disposed of by incineration or deep burial in areas that will not contaminate ground water.

Brucellosis

Introduction

Brucellosis is caused by various members of the genus *Brucella* and is characterised in animals by abortion, retained placenta and occasionally orchitis and infection of the accessory reproductive glands in males. The disease occurs in many countries world-wide, primarily affecting cattle, buffalo, pigs, sheep, goats, dogs and sometimes horses. The disease in humans, known as undulant fever, is a serious public health concern.

Brucellosis has been reported in wild bison and a range of wild deer species including wapiti, moose and caribou (*Rangifer tarandus*) (78). White-tailed deer and mule deer (*Odocoileus hemionus*) appear relatively resistant to infection. In North America, brucellosis due to *B. abortus* biotype 1 occurs among wapiti that congregate on winter feeding grounds in north-western Wyoming, in the United States of America (USA) (70). The disease is endemic in bison and wapiti from Yellowstone National Park in the USA and in bison from Wood Buffalo National Park in Canada. To date, brucellosis has not been reported in farmed wapiti.

Reindeer (*Rangifer tarandus*) in Alaska present with a range of disease syndromes caused by *B. suis* type 4 (21). Recently, on a small number of farms in New Zealand, *B. ovis* has been isolated from the testicles of red deer stags, in which the pathogen caused infertility (64).

Transmission

Brucella organisms are highly infectious and transmission may occur via the oral, nasal, conjunctival, vaginal or rectal mucosa. Bacteria may localise in local lymph nodes and spread to other tissues by lymphoid or haematogenous routes. Aborted foetuses, new-born infected calves, placental membranes and uterine discharges are usually highly infected. *Brucella abortus* has a predilection for the pregnant uterus, mammary gland, testicle and accessory male sex glands, lymph nodes, joint capsules and bursae. The non-pregnant uterus is generally not infected by *B. abortus. Brucella suis* type 4 causes abortions and septic arthritis. *Brucella ovis* has a predilection for the testes and epididymis.

Clinical signs

The most common signs of brucellosis due to *B. abortus* in wapiti are abortion, stillbirth and infertility in females, and orchitis and epididymitis in males. Severe lameness and swollen joints may also be observed. Similarly, *B. suis* type 4 causes abortions, infertility and severe septic arthritis in reindeer. *Brucella ovis* causes epididymitis in red deer and is associated with reduced semen quality and lowered fertility.

Pathology

The most significant lesion is a necrotising placentitis, characterised by a thickened placenta covered with purulent exudate. Lesions in male wapiti are usually milder than those seen in cattle or bison, with some testicular swelling and oedema, in addition to variable necrosis in the testicular parenchyma (70).

Diagnosis

Bacterial isolation provides the only definitive diagnosis of the disease. Recovery of *B. abortus* or *B. suis* from aborted foetuses and/or placenta is a reliable method of confirming infection (unless they are consumed by the hind or cow), although secondary bacterial overgrowth may be a problem. Gloves should be worn when handling aborted material because of the zoonotic risk. *Brucella ovis* may be recovered from affected testes.

Serological tests, although providing only a presumptive diagnosis, allow year-round testing and movement control to be applied. A number of serological tests are available in different countries. Complement fixation offers the best combination of sensitivity and specificity for many species, and remains the standard (70). Unfortunately, experience in Canada has shown that a significant number of sera from wapiti are anti-complimentary. In such cases, an enzyme-linked immunosorbent assay (ELISA) may be used. The tube agglutination test is performed to obtain a serum titre, and allows conversion of titre into international units of anti-*Brucella* antibodies. This test is often required for export purposes. The rapid card and buffered plate tests are used for

Rev. sci. tech. Off. int. Epiz., **21** (2)

251

screening in the field and laboratory, respectively. Animals reacting to these tests are retested using other techniques.

Treatment

Treatment of farmed deer is usually not attempted, as known infected animals are generally culled.

Control

The control of *B. abortus* in wild wapiti and bison is achieved by eradication of infected animals and/or by vaccination of susceptible populations (36). Control of *B. suis* type 4 in reindeer herds is difficult using test-and-slaughter, because current tests are not sufficiently sensitive to detect all infected animals. Although a killed *B. suis* type 4 vaccine is available and appears to be reasonably effective, vaccination invariably causes interference with routine serological tests used for disease surveillance. The control of *B. ovis* is achieved by serological testing of stags, killing affected animals and purchasing animals from herds with a clear herd history.

Clostridial diseases

Introduction

Clostridial organisms cause a wide range of diseases in domestic livestock and wildlife. *Clostridium* spp. have a world-wide distribution and are commonly found in soil, as well as in the gastrointestinal tract of healthy animals. The organisms are spore-forming anaerobes which are highly resistant to a wide range of environmental conditions. Disease is caused by the production of potent specific exotoxins.

Over the last thirty years, since the beginning of deer farming in New Zealand, Australia, North America and Europe, numerous sporadic cases of clostridial disease have been reported in deer. Most have been cases of pulpy kidney (*Clostridium perfringens* type D) in young red and fallow deer (23, 75). Blackleg or malignant oedema associated with trauma, darting or injection sites, occurs in adult red stags and wapiti. These animals are prone to injury from fighting during the breeding season and often need intramuscular injections for chemical restraint during handling and velvet antler removal (9, 38, 56). Tetanus (*C. tetani*) has also been recorded in fallow bucks in Australia after castration with rubber rings (23).

Enterotoxaemia

Enterotoxaemia due to *C. perfringens* type D has been recorded in young red and fallow deer, but is not common. The disease usually has a peracute or acute course. Animals may be found dead, or may die in convulsions within 24 h of first showing signs of anorexia, diarrhoea and depression. Few post-mortem signs of disease are observed in peracute cases. The carcass is usually in good condition, and an excess of clear straw-coloured fluid is often noted in the pericardial sac. Petechial haemorrhages may be found in the heart. In acute cases, the contents of the small intestine have a watery consistency, and areas of congestion of the abomasal and intestinal mucosa may be observed. Usually the liver is congested and the kidneys are soft and gelatinous. Confirmatory diagnosis requires identification of typical bacteria or toxin in intestinal contents.

Given the very rapid onset and short course of the disease, treatment is usually impractical.

Malignant oedema and blackleg

Malignant oedema, also known as gas gangrene, is an acute, rapidly fatal wound infection caused by several different members of the genus, including *C. septicum*, *C. chauvoei*, *C. perfringens*, *C. sordelli* and *C. novyi*. Blackleg, caused by *C. chauvoei*, develops when the spores lodge in skeletal muscle and proliferate under anaerobic conditions such as in deep bruising or wounds, either of which may be associated with handling or fighting.

Malignant oedema and blackleg are usually associated with deep puncture wounds, but have also been observed after surgery, accidental wounding and parturition. A case of clostridial myositis associated with a dart wound has been reported in a reindeer (37). Wild white-tailed deer affected with blackleg may appear dejected, unaware of surroundings, and walk with a stiff high-stepping gait (2). In most cases, few signs are present other than peracute death. At necropsy, malignant oedema is characterised by gangrene of the skin and oedema of the subcutaneous and connective tissue in the surrounding areas. Howe reported that affected wapiti had subcutaneous and intramuscular haemorrhage, with emphysema in the extremities, oedematous internal organs, tarry blood and bloody fluid exudate from body orifices (38). Swelling and crepitus of affected muscles are typical manifestations of blackleg.

Diagnosis requires the isolation of the organism from lesions or identification of the organism using a fluorescent antibody test.

Treatment is usually impractical because of the very rapid onset and short course of the disease.

Tetanus and other clostridial diseases

Tetanus, caused by *C. tetani*, has been occasionally reported in red and fallow deer. A condition closely resembling braxy has been observed in a white-tailed deer, and single cases of braxy and black disease have been reported in deer (2, 56, 75). *Clostridium haemolyticum* has been reported to cause peracute deaths in wild wapiti in areas of the Rocky Mountain foothills with high rainfall, often linked to giant liver fluke infections.

Prevention

Clostridial diseases are usually too acute to permit treatment of affected animals. Therefore, apart from good husbandry, vaccination is the only practical means of protecting animals at

252

Rev. sci. tech. Off. int. Epiz., **21** (2)

risk. Although not licensed for deer, multivalent clostridial vaccines are used in New Zealand, Australia, North America and the United Kingdom (UK) (44, 56, 63). The effectiveness of these vaccines is difficult to evaluate. Generally, pregnant females are vaccinated annually in late gestation, and fawns are vaccinated at weaning. Adult stags are often boosted annually to optimise protection against clostridial complications of wounds or bruising associated with fighting during the breeding season. Cattle 7- or 8-way clostridial vaccines are commonly used in deer in Canada (81).

Colibacillosis

Introduction

Colibacillosis is caused by *Escherichia coli*, a common pathogen of domestic livestock, and is involved in a variety of clinical conditions, especially in young animals. In wapiti and red deer calves, infection with this bacterium may cause neonatal diarrhoea, meningitis and pneumonia, sometimes in association with other pathogens (1, 22, 41, 68). The organism is ubiquitous and is a normal inhabitant of the gastrointestinal tract of all animals. Numerous serotypes are non-pathogenic, and among pathogenic serotypes some are specific for septicaemia and some for enteric disease. The organism is one of the first encountered by a new-born animal, and poor hygiene, lack of colostrum and overcrowding are important factors in development of neonatal diarrhoea. Septicaemic colibacillosis occurs when invasive strains of the bacterium enter the systemic circulation via the intestinal tract, mucosal surfaces or umbilical cord. Systemic spread may lead to localisation of the organism in almost any tissue, and arthritis and meningitis have been reported in cervids (68).

Transmission

Infection is by the faecal-oral route. Overcrowding, poor hygiene and inadequate colostral protection are important predisposing factors.

Clinical signs

Clinical signs vary according to the age of the affected animal and the strain of the bacterium. These can range from sudden death due to endotoxic shock in very young animals, to acute severe watery diarrhoea, with or without haemorrhage, to nervous signs and collapse in cases of meningitis (22, 41). A less severe form of enteritis, known as white scour, occurs in red deer in New Zealand and Australia, and is usually associated with bottle-feeding (22). This syndrome may be caused by a strain of the bacterium that is not enterotoxigenic. White scour can occur in slightly older calves up to three weeks of age and is characterised by a watery or pasty yellowish scour, occasionally streaked with blood. Affected animals are dull, anorexic, show signs of abdominal pain and become dehydrated.

Pathology

In peracute cases, few gross post-mortem signs are present. In cases of septic shock, cardiovascular collapse occurs with intravascular coagulation. Haemorrhage and necrotic changes may be found in the lungs, kidney or liver. In acute bacillosis, subserosal and submucosal petechial haemorrhages occur in association with enteritis and dehydration (22, 41).

Diagnosis

Specific diagnosis requires the isolation and typing of the bacterium. In septicaemic cases, this will involve sampling heart blood and abdominal viscera. Identification of specific pathogens in cases of enteritis is complicated by the fact that one or more pathogens may be involved. Sudden death and diarrhoea in new-born wapiti and red deer calves have several potential causes which must be distinguished. These include trauma, maternal rejection and infection by other pathogens. In the case of elk calves, a number of pathogens have been implicated, including rotavirus, coronavirus, *Cryptosporidium* spp., *Clostridium perfringens* type D and *Salmonella* spp. (33).

Treatment

Escherichia coli is generally susceptible to several different antibacterial agents including ampicillin, trimethoprim-sulphonamide, enrofloxacin and tetracyclines, but these are usually only successful if administered early in the course of the disease. Efficacy in the treatment of enteritis is the subject of debate, but such antibacterials are useful for cases of septicaemia if administered early. Appropriate electrolyte therapy must also be provided to control dehydration.

Control

Where farmed deer and bison are managed in paddocks, and not subjected to intense husbandry practices, *E. coli* is unlikely to become a serious pathogen, although hand-raised animals are at risk. Vaccination of hinds with *E. coli* vaccines to promote higher levels of specific colostral antibodies may be of some benefit. Good husbandry and minimising stress of females around parturition may improve the colostral uptake by calves. When hand-raising calves, good hygiene and frequent small feeds of colostrum during the first 24 h of life are essential components of control of neonatal enteritis including colibacillosis. In addition, the use of 20 ml-30 ml of colostrum in every feed for one to two weeks has a beneficial local immunoglobulin (IgA) effect in the gut.

Johne's disease

Introduction

Johne's disease, caused by *Mycobacterium avium* subsp. *paratuberculosis* (hereafter termed *M. paratuberculosis*), is one of the most widespread infectious conditions affecting ruminants. The disease occurs in wild and captive populations of deer and

Rev. sci. tech. Off. int. Epiz., **21** (2)

253

bison in their native habitats. Johne's disease produces chronic lymphogranulomatous enteritis affecting the mucosa of the ileocaecal valve, ileum, jejunum, and to a lesser extent, the colon. While Johne's disease normally causes chronic scouring in cattle, goats and sheep aged three to five years, the pathology in cervids is unique, with wasting and death in deer under one year of age. An outbreak of Johne's disease in the foundation deer farm in Scotland in the mid-1980s forced the cessation of stock movement and has been singularly important in restricting the growth of deer farming within the UK (27). Apart from chronic inflammation of the enteric mucosa and enlargement of the Peyer's patches, Johne's disease causes lymphadenitis and microabscessation in the gut-associated lymph nodes. The severity of disease may vary between deer breeds, with mule deer apparently the most susceptible (74). In contrast, some animals may be infected with *M. paratuberculosis* without showing any overt clinical symptoms (74).

Johne's disease has also been found in farmed bison herds in North America, with more than 50% of electively slaughtered animals in an infected herd showing histopathological evidence of Johne's disease (8). Apart from the direct impact of Johne's disease as a production-limiting infectious disease in ruminants, another cause for concern is the zoonotic linkage between Johne's disease in livestock and Crohn's disease (13), a disease of humans in which more than 30% of patients show evidence of *M. paratuberculosis* by deoxyribonucleic acid (DNA) hybridisation (12).

Transmission

Johne's disease is spread exclusively by the oro-faecal route from animals excreting *M. paratuberculosis* in faeces. Faecal contamination of water or animal teats may act as a source of infection for young animals. Infected dams may excrete bacteria in milk and infect the suckling offspring. Intrauterine transmission from dam to foetus has been demonstrated to occur in cattle, but whether this occurs in deer and bison is not known. Neonatal animals are considered to be most at risk from infection, and adults appear to be relatively resistant. Farmed red deer in New Zealand have been shown to be susceptible to both sheep and cattle strains of *M. paratuberculosis* (20). Infected sheep and cattle herds may act as sources of infection for deer and bison if cross-grazing or contamination of food or water occur. While Johne's disease occurs in wild populations, limited opportunities for co-mixing between wild and farmed animals suggest that infection from wildlife reservoirs is not of major significance. Nonetheless, animals captured from the wild for farming enterprises should be subjected to comprehensive diagnostic testing to exclude these diseases.

Clinical signs

Although some animals with Johne's disease may develop chronic scouring and significant malabsorption, others show few overt symptoms of disease. Scouring and gut malabsorption, resulting in wasting and death, occur in clinically-affected stock. Clinical symptoms are not normally observed in bison under two years of age. In exceptional circumstances, farmed deer may present with florid enteric disease and significant death rates (30%) in animals of eight to fifteen months of age (48, 54).

Pathology

Generalised granulomatous lesions have been found in infected deer in the caecum, ileum, jejunum and colon. Granulomatous lesions may also be found in the lymphatic organs associated with the intestines and systemically in the liver of deer (55). Organisms may be isolated from throughout the intestine and intestinal lymph nodes, tonsil, liver, lung, spleen and kidney of clinically-affected animals. Clinically-affected American bison (*Bison bison*) show non-caseating granulomatous inflammation containing acid-fast organisms, thickening of the intestinal mucosae and enlarged mesenteric lymph nodes (8). Subclinically-affected bison produce microgranulomata and multinucleate Langhans' giant cells in the mesenteric lymph nodes (7).

Diagnosis

Diagnosis of Johne's disease in ruminants presents considerable challenges because of the lack of specificity of the available immunodiagnostic tests. Although the agar gel immunodiffusion test (65) has a very high specificity and can confirm Johne's disease in clinically-affected animals, the sensitivity of the test in diagnosing subclinical Johne's disease is less than 30%. The ELISA is only of value in detecting clinically-infected deer (54, 55). The high degree of cross-reactivity between *M. paratuberculosis* and *M. avium* (a ubiquitous saprophytic mycobacterium), limits the value of the intradermal tuberculin and blood tests (27), as all have low specificity for diagnosis of Johne's disease (48). This is further complicated by the occurrence of a clinical disease identical to Johne's disease, but caused by *M. avium*, in farmed red deer in New Zealand (54).

Caseogranulomatous lesions with acid-fast organisms are often found at slaughter in mesenteric lymph nodes of farmed deer in New Zealand. The causative agent must be identified by culture or polymerase chain reaction (PCR) in order to differentiate between avian tuberculosis, bovine tuberculosis, paratuberculosis and other mycobacterial infections. Traditional microbiological culture methods have been superseded by radiometric culture tests using selective culture media, the BACTEC technique (16). Such systems, using medium enriched with 25% egg yolk, produce excellent results for the isolation of *M. paratuberculosis* from infected deer (G.W. de Lisle, personal communication). Strain typing of *M. paratuberculosis* can be performed using restriction enzyme analysis or strain-specific probes (insertion sequences IS*900* and IS*1311*) and PCR tests (73).

Treatment

Although possible in theory, the use of prophylactic or therapeutic antimicrobial therapy is impractical for Johne's disease in farmed ruminants.

Control

Rigorous control of the movement of livestock from infected properties is essential to prevent disease spread. The removal from infected mothers and hand-rearing of new-born animals can restrict disease spread within herds. Although serology can be used to eliminate late-stage infected animals, the lack of test sensitivity prevents the use of a test-and-slaughter strategy to eradicate Johne's disease from deer herds. Vaccination using proprietary live or killed Johne's disease vaccines with oil adjuvant is a potential means of control. Although these vaccines attenuate the pathology of Johne's disease and reduce clinical disease in deer, vaccination does not prevent the spread of infection (27). As significant side effects are found with oil-adjuvanted vaccines, these are normally cost-effective only in herds with a high prevalence of clinical disease (> 10%). These vaccines also cause cross-reactions with tuberculosis tests and this may preclude their use in herds at risk of tuberculosis. Proprietary Johne's disease vaccines are not approved for use in livestock or wildlife in the USA or Canada.

Leptospirosis

Introduction

Leptospirosis is caused by serovars of the bacterium *Leptospira interrogans*. Throughout the world, a large number of serovars are each carried by a restricted range of maintenance hosts, which are often rodents, but also include other wildlife species and domestic animals. Infection can spill over into a wide range of mammalian species, including deer and bison. Infection is often inapparent, but can also result in serious disease, including abortion, haemoglobinurea, hepatitis, jaundice and nephritis. Leptospirosis is also a potentially serious zoonosis.

Many serological surveys of wild and farmed deer have been undertaken, using a wide range of antigens, but the results should be interpreted with some caution because antibodies are often highly cross-reactive between serovars. Isolation and typing are the only definitive means of identifying the serovars involved. Antibodies to serovars *L. autumnalis, L. icterohaemorrhagiae* and *L. bratislava* have been found in wapiti killed by hunters in Alberta, and fluorescent antibody testing revealed *L. autumnalis* and *L. bratislava* in one of eighteen kidneys examined. Titres to *L. pomona, L. grippotyphosa, L. canicola* and *L. pyrogenes* have been found in other wild cervids in North America (40, 66). In Europe, leptospiral titres to *L. ballum, L. pomona, L. grippotyphosa* and *L. icterohaemorrhagiae* have also been detected in a variety of wild deer (45).

In New Zealand, serovars *L. pomona, L. hardjo* and *L. copenhageni* have been isolated from clinically-affected farmed red deer (25, 45, 47). In China, *L. romania*, which is carried by mice, has caused outbreaks of haematuria and death in farmed deer (29). In the former USSR, a high proportion of farmed maral deer (*Cervus elaphus* spp.) had titres to *L. grippotyphosa* and *L. pomona* (15). Naturally-occurring leptospirosis due to *L. ballum* was reported in a penned red stag in Scotland (17).

Transmission

Maintenance hosts for particular leptospiral serovars develop chronic interstitial nephritis and shed leptospires in their urine for long periods of time. Accidental or spillover hosts shed the organism in their urine for shorter periods. Red deer have been shown to be leptospiruric for as long as eight months (45). The organism can persist for long periods in the environment, especially under cool, wet conditions.

Animals are primarily infected by exposure to leptospires in urine, contaminated water or food and the organisms enter across mucous membranes, especially conjunctiva, and membranes of the mouth or nose, and through skin cuts or abrasions. Bacteraemic spread leads to localisation in various organs, especially the liver, kidneys and placenta.

Clinical signs

Young animals are generally more susceptible to clinical disease than adults, apart from pregnant females that abort. Clinical signs may range from sudden death in peracute cases to general malaise, pyrexia and anorexia, as well as haemoglobinurea in some cases. Haemolytic disease, severe nephritis, and jaundice have been observed in young red deer infected with *L. pomona* (26). In contrast, infections with *L. hardjo* are usually subclinical. A single stag infected with *L. ballum* showed dullness for several days followed by a fiery appearance of the muzzle, marked congestion of oral and conjunctival mucus membranes, nervous signs with inco-ordination and head twitching, anorexia and dullness in the six days before death (17). In white-tailed deer, *L. pomona* is thought to cause abortions in free-ranging animals, and experimental infections have produced abortion, anorexia, weakness, anaemia, haemoglobinuria, icterus, fever and death (66). Leptospirosis has been implicated in late-term abortions in farmed fallow deer in New Zealand (67).

Pathology

Post-mortem signs are highly variable, and depend upon the severity of the infection. In farm animals, the acute form is characterised by anaemia, jaundice, haemoglobinurea and widespread haemorrhages (79). Acute nephritis may also occur and most animals that recover develop chronic interstitial nephritis. A survey of wapiti killed by hunters revealed that interstitial nephritis was a feature in animals that had leptospiral antibodies. Other lesions observed in cervids include

Rev. sci. tech. Off. int. Epiz., **21** (2)

255

haemorrhages, liver enlargement, oedema and congestion in lungs and haematuria (66).

Diagnosis

Diagnosis on clinical grounds may be difficult unless obvious signs such as jaundice or haemoglobinuria are present. A combination of dark ground microscopy of urine, culture of urine or kidneys, and fluorescent antibody techniques, especially on kidney tissue, is considered important for confirmation. In freshly dead deer, including aborted late-term foetuses, dark ground examination of aqueous humour may reveal leptospires. Serology on a herd basis, particularly of paired samples taken at an interval of fourteen days or more, can also help establish evidence of infection. The microscopic agglutination test is the most common serological test used traditionally, but ELISAs have been developed recently and may be more accurate. However, serology on individuals is not completely reliable because some infected animals can have negligible titres.

Treatment

Streptomycin at 25 mg/kg daily for four days appears to be effective at treating clinically-affected animals and eliminating the carrier state. Tetracyclines may also be used during the acute phase, but may not prevent kidney colonisation. Young deer suffering from a haemolytic crisis may be treated by blood transfusion. As foetal erythrocytes are less resistant to haemolysis, adult deer should be used as donors. Electrolyte therapy should be considered, especially if the kidneys are damaged (45).

Control

A number of practical measures can be taken to reduce the risk of exposure of deer and bison to infection. Vaccination of all herd animals and replacement stock against the prevailing serovars is the most common (76). Purchased animals may be treated with streptomycin before entry into a herd and then vaccinated. Carrier animals may be detected by examination or culture of urine, and treated. Wildlife sources of infection should be controlled or eliminated. Clean drinking water should be supplied and natural water sources fenced off. Deer and bison should not be grazed with other livestock, especially cattle and pigs, unless known to be free of infection. Farm dogs should be vaccinated against relevant serovars (e.g. *L. canicola* and *L. copenhageni*).

Necrobacillosis

Introduction

Necrobacillosis, caused by *Fusobacterium necrophorum*, commonly affects wild and farmed deer and bison, causing purulent necrotic lesions of the mouth, throat, umbilicus, feet, liver and lungs. The organism is a pleomorphic Gram-negative filamentous rod, a strict anaerobe with a world-wide distribution. Infection commonly causes foot abscesses in farmed red, wapiti and fallow deer, and mouth lesions in fallow deer in New Zealand, Australia, Europe and North America, and has also been associated with die-offs of wapiti on winter feeding grounds in the USA, and white-tailed deer in Canada (23, 24, 33, 41, 59).

Transmission

Fusobacterium necrophorum is an element of normal intestinal flora and is an opportunistic pathogen. The organism is present in faeces and survives well in soil and mud. Entry is usually through damaged skin or mucous membranes and infection often occurs in animals that are debilitated or suffering stress such as overcrowding. Cold, wet conditions can predispose to interdigital infection; abrasions or injuries of the oral mucosa due to thistles, foreign objects or teething can lead to infections of the mouth and throat, while cuts and abrasions to the feet or lower legs can lead to infected limbs, especially in cold, muddy conditions or in animals kept in feedlots. Mixed infections involving other pyogenic organisms are common. Grain overload may predispose to necrobacillosis of the wall of the rumen or reticulum. Haematogenous spread can lead to abscesses in the liver, lungs and brain (33). Toxins produced by the bacterium kill cells in the surrounding tissue, disrupting the blood supply and providing the anaerobic conditions suitable for multiplication.

Clinical signs

Infections of the oral mucosa, which usually affect young deer under three months of age, result in swelling of the head and/or tongue, bottle-jaw, salivation, pyrexia and an inability to suckle or feed.

Infections of feet and lower limbs lead to interdigital dermatitis, swollen joints and lameness. Systemic spread to the liver and lungs leads to pyrexia, inappetence, respiratory signs and death.

Pathology

Oral lesions result in destruction of tissue of the tongue, gingiva and the roof of the mouth. Liver and lung abscesses are purulent, foul smelling and often have a greenish tinge. Ulcerative rumenitis is often accompanied by peritonitis and adhesions. In cases of foot abscess, the digit above the claw is swollen, and there may be a sinus that discharges pus.

Diagnosis

The history and appearance of the lesions are fairly characteristic, but culture and isolation of the organism may be attempted. As the bacterium is difficult to grow, a fluorescent antibody technique may be used for confirmation (59). An ELISA may be used to detect antibodies to *F. necrophorum*.

Treatment

Early cases of foot abscess may be treated with parenteral antibiotics together with cleansing and dressing of the affected

256

Rev. sci. tech. Off. int. Epiz., **21** (2)

foot. Hydrogen peroxide is useful for irrigating wounds and abscesses, and the application of oily antibiotic mastitis preparation has been successful in treating deep-seated abscesses in red deer feet. In severe or chronic cases, digital amputation may be required. Treatment of early cases of necrotic stomatitis involves extensive débridement of lesions and vigorous antibiotic therapy. High doses of penicillin, tetracyclines and sulphonamides have been used successfully. If an outbreak is occurring in a herd, mass treatment may be useful. Concurrent vaccination against tetanus and clostridial diseases may be indicated. Once generalised lesions have developed, treatment is usually futile (33).

Control

Prevention of necrobacillosis depends on the removal of predisposing factors such as sharp or protruding surfaces, which may lead to abrasion of the lower limbs. Access to thistles or other plants with prickles, which may injure the oral mucosa, should be prevented. Grain overload should be avoided, and in feedlots, clean dry bedding should be provided.

Animals should be immunised against *F. necrophorum* if a vaccine is available. Veterinarians and deer farmers in New Zealand and Australia claim that vaccination of fallow does with a killed *F. necrophorum* vaccine significantly reduces the incidence of necrotic stomatitis in young fawns. Weaners that are at risk should be vaccinated twice in the autumn and pregnant does should be given an annual booster three weeks prior to fawning.

Pasteurellosis

Introduction

Sporadic outbreaks of pasteurellosis due to *Pasteurella multocida* have been reported in bison and farmed, park, zoo and wild deer. Deer affected include axis (*Axis axis*), red, sambar (*Cervus unicolor*), wapiti, fallow, white-tailed, black-tailed (*Odocoileus hemionus*), mule and caribou deer (11, 60, 71). High mortality in fallow deer as a result of *Pasteurella septica* has also been reported in a park in the UK (57).

Transmission

Pasteurellosis, also known as haemorrhagic septicaemia or shipping fever, is an infectious disease usually caused by *P. multocida*, which is thought to be part of the normal flora of the upper respiratory tract of a wide range of animals and has a world-wide distribution. Transmission is by aerosol, droplets and faeces. Disease appears to be precipitated by a number of stressors including transport, underfeeding, overcrowding, intercurrent viral infections, inclement weather (especially high temperatures, heavy rain or strong winds), and high parasite burdens, especially lungworms. Disease may also be caused by *P. haemolytica* and *P. septica*.

Clinical signs

During an outbreak of pasteurellosis at the National Elk Refuge in the USA, most wapiti were found dead or showed severe depression for less than 24 h before death. The animals held their heads low with the ears drooping, and showed excess salivation. Tremors were seen in 2 of 120 animals that died in 1986 (28). In the pneumonic form of the disease, acute death may occur, but signs of severe respiratory distress are often observed (28, 60, 71).

Pathology

The septicaemic form of pasteurellosis results in acute haemorrhagic disease with engorged blood vessels and widespread petechial haemorrhages. Gross post-mortem signs typical of haemorrhagic septicaemia were observed during the outbreak at the National Elk Refuge. These signs included enlarged congested lymph nodes, enlarged spleen, and haemorrhages in the endocardium, lungs, coronary fat and on the surface of the diaphragm. Swelling of the head has been reported in fallow deer. Fibrinoid degeneration in the glomerular capillaries was observed in reindeer that had died of pasteurellosis. All of the above are characteristic symptoms of endotoxic shock.

The thoracic or respiratory syndrome results in pneumonia with haemorrhages in the lungs, trachea, and on occasions, the nasal mucosa. Cases in wapiti have shown inflamed nasal turbinates and trachea, retropharyngeal oedema, excess thoracic fluid containing fibrin, fibrinous adhesions in the thorax and red consolidated lungs. Affected bison had haemorrhagic lungs, pleura and pericardium, the musculature of the body was pink and oedematous and the lymph nodes were swollen and haemorrhagic (60).

Diagnosis

The diagnosis of pasteurellosis is based upon a combination of post-mortem signs, and the isolation and identification of the bacteria involved.

Treatment

Antibiotic treatment may be attempted if infection is detected before the animal becomes moribund and if the animal can be handled. Oxytetracycline (either 10 mg/kg intravenously for three days, or 20 mg/kg of the long-acting preparation intramuscularly), trimethoprim/sulphonamide (3-5 ml/45 kg daily for three days), and penicillin (20,000-30,000 international units [IU] daily for three days) have been shown to be effective. In an outbreak situation, treatment of all in-contact animals may be worthwhile. Sulphamethazine in water, oxytetracycline in feed or long-acting injection may be used.

Control

Given that *P. multocida* is ubiquitous and carried by a wide range of animals in the respiratory tract, disease prevention

Rev. sci. tech. Off. int. Epiz., **21** (2)

257

should be aimed at improving management as well as minimising stressors and predisposing factors.

Salmonellosis

Introduction

Many different species of *Salmonella* are known to cause disease in a wide variety of animals, including humans, throughout the world. *Salmonella* spp. have occasionally been isolated from dead or diseased wapiti and red deer, but have not been reported to cause epidemic outbreaks.

Transmission

Transmission is usually via the faecal-oral route, and carrier animals are considered to be important in the epidemiology of the infection. In a number of hosts, the gall bladder is an important site in which *Salmonella* organisms are maintained, and the lack of this organ in deer may explain why salmonellosis is a less prominent disease in deer, compared to other domestic species (33).

Clinical signs

Peracute septicaemia and acute enteritis have been observed in red deer and wapiti (35, 43). The peracute form occurs in young animals, often under two weeks of age, which may be found dead or comatose and showing nervous symptoms. Other signs include anorexia, depression, recumbency and opisthotonos.

Pathology

Peracute cases may show non-specific widespread petechial haemorrhage. Signs in animals that develop acute enteritis are those of haemorrhagic enteropathy and include gas distension, haemorrhages and erosions of the intestinal mucosa. Histologically, widespread evidence of coagulative necrosis is present.

Diagnosis

In the live animal, faecal culture and fluorescent antibody testing may yield results. At necropsy, samples of abdominal lymph nodes, spleen, liver, kidney and lung should be obtained for culture and histopathological examination.

The differential diagnosis should include both toxic and infectious agents. Yersiniosis, enterotoxaemia and colibacillosis should be considered in young animals. Malignant catarrhal fever and acute haemorrhagic enteropathy associated with severe stress should be considered in older animals.

Treatment

Acutely ill animals should be treated with appropriate antibiotics and fluid therapy. Potential antibiotics include ampicillin, gentamicin and trimethoprim/sulphonamide. If possible, the sensitivity of the organism to antibacterial agents should be determined, as many instances of antibiotic resistance have been reported in *Salmonella*.

Control

Vaccination has not been used for prophylactic control, but stress reduction and optimal hygiene are important when deer are being hand-raised. In the face of clinical cases, the isolation of affected animals and strict attention to hygiene is essential. When a positive diagnosis of salmonellosis is made in farmed animals, enhanced hygiene should be implemented for humans associated with the premises.

Tuberculosis

Introduction

Tuberculosis, caused by infection with *Mycobacterium bovis*, has been found in deer and bison herds living in natural habitats world-wide. The antiquity of this disease is witnessed by the recent isolation of tuberculosis complex DNA from 'tuberculosis lesions' in fossilised bone from extinct long horned bison, considered to be at least 17,000 years old (61). Tuberculosis presents as a granulomatous lymphadenitis, with the lymph nodes of the head and thorax most commonly affected. Tuberculosis has been diagnosed in farmed deer in almost every country that has taken up deer farming (in Europe, North America and Australasia) (32). Classically, the index herds in any country in which tuberculosis is diagnosed have shown widespread and severe pathology. In the first half of the last century, over 50% of bison slaughtered from Buffalo Park, near Wainwright, Alberta, were diagnosed as tuberculous. This herd was formed by aggregating bison raised on dairy farms throughout the country in 1906 and 1907. Animals culled from this herd biennially up to 1929 showed a lesion prevalence of over 70%. When the herd was depopulated in 1939, 30% of animals had visible tuberculosis lesions (34). Between 1983 and 1985, 21% of 72 bison found dead in and around Wood Buffalo Park had tuberculosis confirmed at necropsy (69). Tuberculosis has been diagnosed in a farmed bison herd in North America, with disease affecting over 20% of adults (72). The advanced progress towards eradication of tuberculosis from cattle in most developed countries has led to the introduction of compulsory tuberculosis surveillance programmes for exotic farmed ruminants, including deer and bison.

Transmission

Tuberculosis may be spread directly via aerosols passing from animal to animal by the respiratory route, thereby producing lung and pulmonary lymph node lesions. Spread by the oropharyngeal route may also occur in animals sharing the same food source, through grooming or from exposure to contaminated secretions, fomites or dust particles, resulting in head and intestinal lymph node lesions (32). The route of

transmission may vary, depending on population density, environment and feeding patterns of animals. A recent outbreak of tuberculosis in wild white-tailed deer in Michigan (62) has been associated with the congregation of animals at feed stations during winter.

Clinical signs

Animals usually show little clinical evidence of tuberculosis, except in the terminal stages of disease. This explains why tuberculosis remains undetected within herds for long periods in the absence of routine tuberculosis surveillance protocols. Many animals may have a single caseo-calcified lesion of a lymph node and show no overt symptoms throughout their life. Animals with bronchial disease may cough and develop cachexia in the terminal phase of the disease. Susceptible animals that develop fulminating generalised tuberculosis may only show clinical signs in the late stages of disease. These may include peripheral lymph node enlargement, the development of sinuses tracking to the skin surface from lymph nodes (especially around the throat), loss of condition, coughing and exercise intolerance. The interval between the development of overt clinical signs and death is usually short.

Pathology

Tuberculosis in Cervidae may present pathologically in many forms. The infection may produce liquefactive necrosis with a zone of granulomatous reactivity containing giant cells, surrounded by a fibrous capsule. Fibrous capsules may be thin and the degree of mineralisation may vary between different species of deer (58). Acute abscessation, involving neutrophils and large numbers of acid-fast organisms may be found in acutely infected animals in severe outbreaks of tuberculosis in deer herds (32). Post-mortem studies of the bison in Wood Buffalo Park showed some animals with generalised tuberculosis and others with thoracic or head lymph node lesions (69).

Diagnosis

The intradermal tuberculin skin test, developed for the diagnosis of tuberculosis in cattle has been adapted for use in exotic ruminants. The mid-cervical skin test (MCST) has a sensitivity of approximately 80%, if applied carefully and interpreted conservatively (18). The MCST has limited specificity, especially when used in an environment in which high levels of saprophytic mycobacteria are present. This has required the introduction of the comparative cervical skin test (19) and ancillary blood tests have been developed to improve the precision of the MCST for tuberculosis diagnosis in deer. A composite immune cell and antibody test has been developed for tuberculosis diagnosis in deer (30). The blood test for tuberculosis measures lymphocyte transformation of circulating T cells and antibody levels using an ELISA, and has consistently high levels of sensitivity (>90%) and specificity (>98%). A modified ELISA, which measures IgG1 antibodies, has been evaluated recently for tuberculosis diagnosis in deer. The test

has high sensitivity (>90%) for tuberculosis diagnosis, and acceptable levels of specificity (97%) (J.F.T. Griffin, unpublished findings). Trials are currently underway to evaluate the potential of the interferon-γ test for tuberculosis diagnosis in deer and buffalo (80).

Histopathological examination of suspect lesions in a range of tissues is routinely used to confirm a diagnosis of tuberculosis *post mortem*. The spectrum of pathology seen in exotic ruminants may be more extensive than that seen traditionally in cattle and sheep. Microbiological culturing and strain typing of *M. bovis* is performed using restriction enzyme analysis or strain-specific PCR probes (6).

Treatment

Although possible in theory, the use of prophylactic or therapeutic antimicrobial therapy is impractical for tuberculosis in farmed ruminants.

Control

The control and eradication of tuberculosis in farmed deer and bison is strongly influenced by the presence or absence of wildlife vectors of tuberculosis. In the absence of infected wildlife, the most effective strategy for eradication is to use a comprehensive test-and-slaughter policy, surveillance of animals sent for slaughter and rigorous control of the movement of livestock from infected properties. Herd depopulation is usually invoked to control tuberculosis outbreaks in farmed deer and bison in countries that have a low prevalence of tuberculosis in cattle herds. However, the precision of currently available diagnostic tests for tuberculosis allows eradication from infected herds cost-effectively in a short period, using the available test protocols (32), combined with selective slaughter.

The presence of infected wildlife vectors, such as badgers (*Meles meles*) in the UK, brush-tailed possums (*Trichosurus vulpecula*) in New Zealand and white-tailed deer in the USA, necessitates additional control measures (32). Obviously, the elimination of the infected vectors is the best option, but this is usually expensive, difficult or impractical. However, reduction of wildlife vector numbers may greatly assist in reducing the number of herd breakdowns (42). Vaccination, although demonstrated to be efficacious (31), may interfere with routine diagnostic methods and compromise disease surveillance programmes.

Yersiniosis

Introduction

Yersiniosis, caused by *Yersinia pseudotuberculosis*, was first diagnosed in outbreaks of diarrhoea among deer herds in 1978 (4). The disease rapidly became regarded as one of the most serious and common infectious diseases of farmed deer in New

Rev. sci. tech. Off. int. Epiz., **21** (2)

259

Zealand (49). Yersiniosis is common in young farmed deer during their first autumn/winter or in newly captured deer; predisposing factors include stressors such as weaning, transport, bad weather and underfeeding. The disease also occurs in farmed deer in Australia, North America and Europe. The organism has been implicated in a die-off of free-ranging musk oxen (*Ovibos moschatus*) (5), and causes high mortality rates in zoological collections of birds and primates. *Yersinia enterocolitica* is commonly isolated from domestic livestock including deer, but the significance of this species as a primary pathogen in deer is not proven. Both *Y. pseudotuberculosis* and *Y. enterocolitica* cause zoonotic disease.

Transmission

The epidemiology of yersiniosis in New Zealand has been reviewed by Mackintosh and Henderson (49) and Mackintosh (46). Although at least six serotypes of *Y. pseudotuberculosis* exist world-wide, only serotypes I, II and III are found in New Zealand. *Yersinia pseudotuberculosis* is carried in the intestines by a wide range of wild and domestic animals including birds, rodents, rabbits, hares, pigs, cattle, sheep and household pets. Carrier animals tend to excrete more *Y. pseudotuberculosis* in the faeces at times of stress, especially in winter, and the organisms survive in the environment for long periods in cold, wet conditions. The majority of farmed deer are exposed to contaminated food and water during their first autumn/winter and experience subclinical infections. Outbreaks of disease occur when animals are stressed and experience a heavy challenge. Genetic factors also appear to influence susceptibility (50, 51, 77).

Clinical signs

Affected deer, which are usually four to eight months old, tend to be depressed, anorexic, to separate off from the herd and stand back from feed. Signs of staining of the hocks and tail hairs by green watery diarrhoea are usually present. The diarrhoea generally turns dark or bloody. Pyrexia is frequently noted early in the course of the disease. Affected deer rapidly become dehydrated and weak. Acute cases may be found dead without any obvious clinical signs.

Pathology

Yersiniosis is characterised by an acute haemorrhagic enteropathy, especially involving the lower jejunum and terminal ileum. The caecum and colon are sometimes affected, and occasionally changes extend to the upper small intestine and abomasum. Oedema of the serosal surface of the alimentary tract frequently occurs. The contents of the intestines are usually watery and bloody. In some cases, the intestinal wall is thickened and is covered by a fibrino-necrotic pseudomembrane. The mesenteric lymph nodes are frequently swollen, oedematous, haemorrhagic and necrotic. The miliary abscess lesions of liver, spleen and mesenteric lymph nodes seen in other species are seldom seen in deer (4, 10).

Diagnosis

A number of causes of death in weaners during the autumn/winter period can be confused with yersiniosis, including malignant catarrhal fever, leptospirosis, salmonellosis, colibacillosis, pasteurellosis, stress enteropathy, stress/duodenal ulceration (and subsequent fungal infection), lungworm, fusobacterial abscesses of rumen/liver/lung as well as misadventure, poisoning and polioencephalomalacia. As the majority of healthy deer experience subclinical infection with *Y. pseudotuberculosis* and can carry and excrete the organism for some weeks or months, the organism may be isolated, with *Yersinia*-specific media, from faeces and intestinal contents of normal animals and of animals dying from other diseases. In cases of clinical yersiniosis, *Y. pseudotuberculosis* organisms cross the wall of the intestine and spread to the mesenteric lymph nodes via the lymphatic drainage, and isolation of *Y. pseudotuberculosis* from the mesenteric lymph nodes should be possible in these cases. Diseased animals suffering from other conditions may also experience a recrudescence of *Y. pseudotuberculosis* infection when terminally ill, or overgrowth of *Y. pseudotuberculosis* after death.

A confirmed diagnosis of yersiniosis should include typical clinical signs, the isolation of *Y. pseudotuberculosis* from mesenteric lymph nodes, and a characteristic histopathological picture of pseudomembranous enteritis with colonies of Gram-negative coccobacilli.

Treatment

Intensive treatment with antibiotics, such as tetracyclines or potentiated sulphonamides, and oral or parenteral fluids is usually effective if administered early in the course of the disease. During an outbreak, treatment of all in-contact animals with parenteral tetracyclines is usually worthwhile.

Control

The recommended strategy for prevention of yersiniosis in farmed deer in New Zealand involves the use of good management, optimal feeding, provision of shelter and minimising stress. These are combined with the use of a locally developed killed vaccine. The vaccine contains serotypes I, II and III together with diethylaminoethyl (DEAE) dextran adjuvant and has proved a valuable adjunct to good management for disease prevention. The vaccine was shown to have significant efficacy for protection against yersiniosis in experimental challenge trials and in field trials (50, 51, 52, 53). The vaccine has been used widely in New Zealand since 1994 and has demonstrated good protection against outbreaks of disease, although vaccination does not prevent clinical disease in every individual (77).

Maladies bactériennes des cerfs et des bisons d'élevage

C. Mackintosh, J.C. Haigh & F. Griffin

Résumé
Les principales maladies des cerfs et des bisons d'élevage dues à des bactéries aérobies sont la tuberculose bovine, la maladie de Johne (paratuberculose), la yersiniose, la leptospirose, la brucellose, la pasteurellose, le charbon bactéridien, la salmonellose et la colibacillose. Les infections dues à des bactéries anaérobies affectant les mêmes animaux comprennent la nécrobacillose et un certain nombre de clostridioses telles que le tétanos, le charbon symptomatique, l'œdème malin et l'entérotoxémie. L'importance relative de ces maladies dans le monde varie selon le moment et les circonstances, mais la tuberculose bovine et la maladie de Johne vont probablement poser les problèmes les plus graves en termes de diagnostic, de prophylaxie, d'échanges d'animaux vivants et de détermination des réservoirs d'infection dans la faune sauvage. Les auteurs présentent un résumé de l'étiologie, des principales espèces d'animaux atteints, de la répartition géographique, de la transmission, des signes cliniques, de la pathologie, du diagnostic, du traitement et de la prophylaxie de ces maladies.

Mots-clés
Bisons – Cerfs – Diagnostic – Épidémiologie – Faune sauvage – Maladies bactériennes – Prophylaxie.

■

Enfermedades bacterianas de ciervos y bisontes de granja

C. Mackintosh, J.C. Haigh & F. Griffin

Resumen
Las principales enfermedades causadas por bacterias aerobias que afectan a los ciervos y bisontes de granja son la tuberculosis bovina, la enfermedad de Johne (o paratuberculosis), la yersiniosis, la leptospirosis, la brucelosis, la pasteurelosis, el carbunco bacteridiano, la salmonelosis y la colibacilosis. En cuanto a las infecciones por bacterias anaerobias, destacan la necrobacilosis y una serie de clostridiosis como el tétanos, el carbunco sintomático, el edema maligno o la enterotoxemia. Aunque la importancia relativa de estas enfermedades en diversas zonas del mundo puede variar según el momento y las circunstancias, la tuberculosis bovina y la enfermedad de Johne resultan seguramente las más problemáticas en términos de diagnóstico y control, comercio de animales vivos y aparición de reservorios infecciosos entre la fauna salvaje. Los autores resumen la etiología, las principales especies afectadas, la distribución geográfica, la transmisión, los síntomas clínicos, la patología, el diagnóstico y el tratamiento y control de estas enfermedades.

Palabras clave
Bisontes – Ciervos – Control – Diagnóstico – Enfermedades bacterianas – Epidemiología – Fauna salvaje.

■

Rev. sci. tech. Off. int. Epiz., **21** (2)

261

References

1. Angus K.W. (1994). – Neonatal enteritis. *In* Management and diseases of deer (T.L. Alexander & D. Buxton, eds). Veterinary Deer Society, London, 136-139.

2. Armstrong H.L. & MacNamee J.K. (1950). – Blackleg in deer. *J. Am. vet. med. Assoc.*, **117**, 212-214.

3. Barlow R.M. & Buxton D. (1988). – Anthrax. *In* Management and diseases of deer (T.L. Alexander, ed.). Veterinary Deer Society, London, 160-161.

4. Beatson N.S. & Hutton J.B. (1981). – An outbreak of yersiniosis in farmed red deer. *In* Proc. Deer seminar for veterinarians, Queenstown, February. Deer Advisory Panel, New Zealand Veterinary Association, Wellington, 136-139.

5. Blake J.E., McLean B.D. & Gunn A. (1989). – Yersiniosis in free-ranging muskoxen on Banks Island, N.W.T., Canada. *Can. J. Zool.*, **67**, A58.

6. Bolske G., Englund L., Wahlstrom H., de Lisle G.W., Collins D.M. & Croston P.S. (1995). – Bovine tuberculosis in Swedish deer farms: epidemiological investigations and tracing using restriction fragment analysis. *Vet. Rec.*, **136**, 414-417.

7. Buergelt C.D. & Ginn P.E. (2000). – The histopathologic diagnosis of subclinical Johne's disease in North American bison (*Bison bison*). *Vet. Microbiol.*, **77**, 325-331.

8. Buergelt C.D., Layton A.W., Ginn P.E., Taylor M., King J.M., Habecker P.L., Mauldin E., Whitlock R. & Rossiter C. (2000). – The pathology of spontaneous paratuberculosis in the North American bison (*Bison bison*). *Vet. Pathol.*, **37**, 428-438.

9. Buxton D. (1994). – Clostridial diseases. *In* Management and diseases of deer (T.L. Alexander & D. Buxton, eds). Veterinary Deer Society, London, 121-123.

10. Buxton D. & Mackintosh C.G. (1994). – Yersiniosis. *In* Management and diseases of deer (T.L. Alexander & D. Buxton, eds). Veterinary Deer Society, London, 117-119.

11. Carrigan M.J., Dawkins H.J.S., Cockram F.A. & Hansen A.T. (1991). – *Pasteurella multocida* septicaemia in fallow deer. *Aust. vet. J.*, **68**, 201-203.

12. Chamberlain W., Graham D.Y., Hulten K., El-Zimaity H.M.T., Schwartz M.R., Naser S., Shafran I. & El-Zaatari F.A.K. (2001). – *Mycobacterium avium* subsp. *paratuberculosis* as one cause of Crohn's disease. *Aliment. Pharmacol. Ther.*, **15**, 337-346.

13. Chiodini R.J. (1989). – Crohn's disease and the mycobacterioses: a review and comparison of two disease entities. *Clin. Microbiol. Rev.*, **2**, 90-117.

14. Choquette L.P.E. & Broughton E. (1981). – Anthrax. *In* Infectious diseases of wild mammals, 2nd Ed. (J.W. Davis, L.H. Karstad & D.O. Trainer, eds). Iowa State University Press, Ames, Iowa, 288-296.

15. Chulovskii I.K. (1971). – Leptospirosis in Western Siberia. *Veterinariya (Moscow)*, **2**, 66-68.

16. Cook W.E., Cornish T.E., Shideler S., Lasley B. & Collins M.T. (1997). – Radiometric culture of *Mycobacterium avium paratuberculosis* from feces of Tule elk. *J. Wildl. Dis.*, **33**, 635-637.

17. Corrigall W. (1978). – Naturally occurring leptospirosis (*Leptospira ballum*) in a red deer (*Cervus elaphus*). *Vet. Rec.*, **103**, 75-76.

18. Corrin K.C., Carter C.E., Kissling R.C. & de Lisle G.W. (1987). – Short interval skin testing in farmed red deer (*Cervus elaphus*) inoculated with *Mycobacterium bovis*. *N.Z. vet. J.*, **35**, 204-207.

19. Corrin K.C., Carter C.E., Kissling R.C. & de Lisle G.W. (1993). – An evaluation of the comparative tuberculin for detecting tuberculosis in deer. *N.Z. vet. J.*, **41**, 1-12.

20. De Lisle G.W. & Collins D.M. (1993). – Johne's disease in farmed deer. *Surveillance*, **20**, 18-19.

21. Dieterich R.A. (1985). – An overview of Alaskan reindeer diseases and current control methods. *In* Biology of deer production (P. F. Fennessy & K. R. Drew, eds). *Roy. Soc. N.Z. Bull.*, **22**, 97-100.

22. English A.W. (1984). – Veterinary aspects of deer farming in New South Wales. *In* Deer refresher course. Proc. No. 72, Refresher course for veterinarians. University of Sydney, Sydney, 127-202.

23. English A.W. (1993). – Fallow deer – animal health and disease prevention. *In* Proc. 1st World Forum on fallow deer farming, 10-13 March, Mudgee, New South Wales. Australian and New Zealand Fallow Deer Societies, Hamilton, 83-86.

24. Evans P. (1987). – Infected injuries. *Proc. N.Z. vet. Assoc. Deer Branch*, **4**, 186-188.

25. Fairley R.A., Schollum L.M. & Blackmore D.K. (1984). – Leptospirosis associated with serovars *hardjo* and *pomona* in red deer calves (*Cervus elaphus*). *N.Z. vet J.*, **32**, 76-78.

26. Fairley R.A., Cooper D.R., Guilford W.G. & Schollum L.M. (1986). – Haemolytic disease associated with *Leptospira interrogans* serovar *pomona* in red deer calves (*Cervus elaphus*). *N.Z. vet. J.*, **34**, 116-117.

27. Fawcett A.R., Goddard P.J., McKelvey W.A., Buxton D., Reid H.W., Greig A. & MacDonald A.J. (1995). – Johne's disease in a herd of farmed red deer. *Vet. Rec.*, **136**, 165-169.

28. Franson J.C. & Smith B.L. (1988). – Septicemic pasteurellosis in elk (*Cervus elaphus*) on the United States National Elk Refuge, Wyoming. *J. Wildl. Dis.*, **24**, 715-717.

29. Gou Y. (1981). – Research on haematuria disease of deer. *In* Wildlife diseases of the Pacific Basin and other countries. Proc. 4th International Conference of the Wildlife Disease Association (WDA) (M.E. Fowler, ed.), 25-28 August, Sydney. WDA, Sacramento, 207-208.

30. Griffin J.F.T., Cross J.P., Chinn D.N., Rodgers C.R. & Buchan G.S. (1994). – Diagnosis of tuberculosis due to *M. bovis* in New Zealand red deer (*Cervus elaphus*) using a composite blood test (BTB) and antibody (ELISA) assays. *N.Z. vet. J.*, **42**, 173-179.

262

Rev. sci. tech. Off. int. Epiz., **21** (2)

31. Griffin J.F.T., Mackintosh C.G., Slobbe L., Thomson A.J. & Buchan G.S. (1999). – Vaccine protocols to optimise protective efficacy of BCG. *Tubercle Lung Dis.*, **79**, 135-143.

32. Griffin J.F.T. & Mackintosh C.G. (2000). – Tuberculosis in deer: perceptions, problems and progress. *Vet. J.*, **160**, 202-219.

33. Haigh J.C. & Hudson J.R. (1993). – Farming wapiti and red deer. Mosby, St Louis, 369 pp.

34. Hawden S. (1942). – Tuberculosis in buffalo. *J. Am. vet. med. Assoc.*, **100**, 19-22.

35. Henderson T.G. & Hemmingsen P. (1983). – Faecal survey of deer for *Yersinia pseudotuberculosis* and *Salmonella* spp. *N.Z. vet. J.*, **31**, 225-226

36. Herriges J.D., Thorne E.T., Anderson S.L. & Dawson H.A. (1989). – Vaccination of elk in Wyoming with reduced dose strain 19 *Brucella*: controlled studies and ballistic implant trials. *Proc. U.S. Anim. Hlth Assoc.*, **93**, 640-655.

37. Herron A.J., Garman R.H., Baitchman R. & Kraus A.L. (1979). – Clostridial myositis in a reindeer (*Rangifer tarandus*): a case report. *J. Zoo Anim. Med.*, **10**, 31-34.

38. Howe D.L. (1981). – Miscellaneous bacterial diseases. *In* Infectious diseases of wild mammals, 2nd Ed. (J.W. Davis, L.H. Karstad & D.O. Trainer, eds). Iowa State University Press, Ames, Iowa, 418-422.

39. Kellog F.E. & Prestwood A.K. (1970). – Anthrax epizootic in white-tailed deer. *J. Wildl. Dis.*, **6**, 226-228.

40. Kingscote B.F., Yates W.D.G. & Tiffin G.B. (1987). – Diseases of wapiti utilizing cattle range in southwestern Alberta. *J. Wildl. Dis.*, **23**, 86-91.

41. Korner E. & Winkelmann J. (1988). – Health aspects of deer farming. *In* The management and health of farmed deer (H.W. Reid, ed.). Kluwer Academic Publishers, Dordrecht, 89-99.

42. Livingstone L. (1995). – Developing a national pest management strategy for the control of bovine tuberculosis. *In* Tuberculosis in wildlife and domestic animals (F. Griffin & G.W. de Lisle, eds). Otago Conference Series No. 3. University of Otago Press, Dunedin, New Zealand, 337-341.

43. McCallum H.J.F., Familton A.S., Brown R.A. & Hemmingsen P. (1978). – Salmonellosis in red deer calves. *N.Z. vet. J.*, **26**, 130-131.

44. McKenzie A.R. (1984). – Diseases of deer in Queensland. *In* Deer refresher course. *Postgrad. Comm. vet. Sci. Proc.*, **72**, 595-602.

45. Mackintosh C.G. (1984). – Leptospirosis in deer. *Proc. N.Z. vet. Assoc. Deer Branch*, **1**, 100-106.

46. Mackintosh C.G. (1992). – A review of yersiniosis in farmed red deer in New Zealand. *In* The biology of deer (R.D. Brown, ed.). Springer-Verlag, New York, 126-129.

47. Mackintosh C.G. (1992). – Vaccines for control, prevention and eradication of disease in farmed deer. *Proc. N.Z. vet. Assoc. Deer Branch*, **9**, 92-97.

48. Mackintosh C.G. (1999). – Update on investigations into diagnostic tests for Johne's disease in red deer. *Proc. N.Z. vet. Assoc. Deer Branch*, **16**, 145-150.

49. Mackintosh C.G. & Henderson T.G. (1984). – The epidemiology of yersiniosis in deer. *Proc. N.Z. vet. Assoc. Deer Branch*, **1**, 34-42.

50. Mackintosh C.G., Buddle B.M. & Griffin J.F.T. (1990). – Yersiniosis vaccine research. *Proc. N.Z. vet. Assoc. Deer Branch*, **7**, 200-203.

51. Mackintosh C.G., Buddle B.M., Griffin J.F.T. & Cross J.P. (1991). – Yersiniosis vaccine update. *Proc. N.Z. vet. Assoc. Deer Branch*, **8**, 206-209.

52. Mackintosh C.G., Buddle B.M. & Griffin J.F.T. (1992). – Yersiniavax efficacy under field conditions in weaner red deer. *Proc. N.Z. vet. Assoc. Deer Branch*, **9**, 107-110.

53. Mackintosh C.G., Buddle B.M. & Griffin J.F.T. (1998). – Vaccination of farmed red deer (*Cervus elaphus*) against yersiniosis. *In* Recent developments in deer biology (J.A. Milne, ed.). Macaulay Land Use Research Institute, Craigiebuckler, Aberdeen, 318.

54. Mackintosh C.G., Reichel M.P., Griffin J.F.T., Montgomery H. & de Lisle G.W. (1999). – Paratuberculosis and avian tuberculosis in red deer in New Zealand: clinical syndromes and diagnostic tests. *In* Proc. 6th International Colloquium on paratuberculosis (E.J.B. Manning & M.T. Collins, eds), 14-18 February, Melbourne. International Association for Paratuberculosis, Madison, Wisconsin, 449-457.

55. Manning E.J., Steinberg H., Rossow K., Ruth G.R. & Collins M.T. (1998). – Epizootic of paratuberculosis in farmed elk. *J. Am. vet. med. Assoc.*, **213**, 1280-1281.

56. Mulvaney C. (1981). – Clostridial diseases: clinical occurrence, control and prevention. *In* Proc. Deer seminar for veterinarians. Deer Advisory Panel, New Zealand Veterinary Association, Queenstown, 113-116.

57. Putman P., Davidson H. & Goldspink C.R. (1992). – *Pasteurella*-related mortality in park fallow deer. *In* The biology of deer (R.D. Brown, ed.). Springer-Verlag, New York, 152.

58. Rhyan J.C. & Saari D.A. (1995). – A comparative study of the histopathological features of bovine tuberculosis in cattle, fallow deer (*Dama dama*), sika deer (*Cervus nippon*), and red deer and elk (*Cervus elaphus*). *Vet. Pathol.*, **32**, 215-220.

59. Rosen M.N. (1981). – Necrobacillosis. *In* Infectious diseases of wild mammals, 2nd Ed. (J.W. Davis, L.H. Karstad & D.O. Trainer, eds). Iowa State University Press, Ames, Iowa, 332-338.

60. Rosen M.N. (1981). – Pasteurellosis. *In* Infectious diseases of wild mammals, 2nd Ed. (J.W. Davis, L.H. Karstad & D.O. Trainer, eds). Iowa State University Press, Ames, Iowa, 244-252.

61. Rothschild B.M., Martin L.V., Lev G., Bercovier H., Bar-Gal G.K., Greenblatt C., Donoghue H., Spiegelman M. & Brittain D. (2001). – *Mycobacterium tuberculosis* complex DNA from an extinct bison dated 17,000 years before the present. *Clin. infect. Dis.*, **33**, 305-311.

62. Schmitt S.M., Fitzgerald S.D. & Cooley T.M. (1997). – Bovine tuberculosis in free-ranging white-tailed deer from Michigan. *J. Wildl. Dis.*, **33**, 749-758.

Rev. sci. tech. Off. int. Epiz., **21** (2)

263

63. Scott I. (1987). – Deer health and production programmes. *Proc. N.Z. vet. Assoc. Deer Branch*, **4**, 109-115.

64. Scott I. (1998). – *Brucella ovis* in deer. *Proc. N.Z. vet. Assoc. Deer Branch*, **15**, 87-91.

65. Sherman D.M., Markham R.J.F. & Bates F. (1984). – Agar gel immunodiffusion test for diagnosis of paratuberculosis in cattle. *J. Am. vet. med. Assoc.*, **185**, 179-182.

66. Shotts E.B. (1982). – Leptospirosis. *In* Infectious diseases of wild mammals, 2nd Ed. (J.W. Davis, L.H. Karstad & D.O. Trainer, eds). Iowa State University Press, Ames, Iowa, 323-331.

67. Smith B.L. (1993). – Fallow deer – animal health and disease prevention. *In* Proc. 1st World Forum on fallow deer farming, 10-13 March, Mudgee, New South Wales, Australia. Australian and New Zealand Fallow Deer Societies, Hamilton, 87-98.

68. Smits J.E.G. (1992). – Elk disease survey in western Canada and northwestern United States. *In* The biology of deer (R.D. Brown, ed.) Springer Verlag, New York, 101-106.

69. Tessaro S.V., Forbes L.B. & Turcotte C.A. (1990). – A survey of brucellosis and tuberculosis in bison in and around Wood Buffalo National Park. *Can. vet. J.*, **31**, 174-180.

70. Thorne E.T. (1982). – Brucellosis. *In* Diseases of wildlife in Wyoming (E.T. Thorne, N. Kingston, W.R. Jolley & R.C. Bergstrom, eds). Wyoming Game and Fish Department, Laramie, 54-63.

71. Thorne E.T. (1982). – Pasteurellosis. *In* Diseases of wildlife in Wyoming (E.T. Thorne, N. Kingston, W.R. Jolley & R.C. Bergstrom, eds). Wyoming Game and Fish Department, Laramie, 72-77.

72. Van Tiem J.S. (1994). – Bovine tuberculosis. *In* DxMonitor Animal Health Report, Winter. United States Department of Agriculture, Animal and Plant Health Inspection Service, Veterinary Services, Washington, DC, 9.

73. Whittington R.J., Marsh I.B. & Whitlock R.H. (2001). – Typing of IS*1311* polymorphisms confirm that bison (*Bison bison*) with paratuberculosis in Montana are infected with a strain of *Mycobacterium avium* subsp. *paratuberculosis* distinct from that occurring in cattle and domesticated livestock. *Molec. cell. Probes*, **15**, 139-145.

74. Williams E.S., Snyder S.P. & Martin K.L. (1983). – Pathology of spontaneous and experimental infection of North American wild ruminants with *Mycobacterium paratuberculosis*. *Vet. Pathol.*, **20**, 274-290.

75. Wilson P. (1984). – Diseases relevant to deer farming. *In* Deer refresher course. *Postgrad. Comm. vet. Sci. Proc.*, **72**, 105-125.

76. Wilson P.R. & Schollum L.M. (1984). – Serological responses of farmed red deer to vaccine against *Leptospira interrogans* serovars *hardjo* and *pomona. N.Z. vet. J.*, **32**, 117-119.

77. Wilson P.R., Mackintosh C.G. & Griffin J.F.T. (1999). – Yersiniosis: resistance, susceptibility and vaccination. *Proc. N.Z. vet. Assoc. Deer Branch*, **16**, 145-150.

78. Witter J.F. (1981). – Brucellosis. *In* Infectious diseases of wild mammals, 2nd Ed. (J.W. Davis, L.H. Karstad & D.O. Trainer, eds). Iowa State University Press, Ames, Iowa, 280-287.

79. Wood D.A. (1988). – Leptospirosis. *In* Management and diseases of deer (T.L. Alexander, ed.). Veterinary Deer Society, London, 101-104.

80. Wood P.R., Corner L.A. & Plackett P. (1990). – Development of a simple, rapid *in vitro* cellular assay for bovine tuberculosis based on the production of γ-interferon. *Res. vet. Sci.*, **49**, 46-49.

81. Wright R.G. (2002). – Medicines and withdrawal times for farmed deer. *In* Proc. 4th International Wildlife Ranching Symposium, 8-12 November 1999, Toronto (in press).