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Viral, parasitic and prion diseases of farmed deer and bison

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Summary
The most important viral disease of farmed deer and bison is malignant catarrhal fever. The other herpesviruses which have been isolated from these species are briefly described. Other viral agents that are recognised in these animals, including adenovirus, parapox, foot and mouth disease, bluetongue, epizootic haemorrhagic disease, bovine virus diarrhoea, rotavirus and coronavirus, are also discussed. Ectoparasites of importance in this group in various parts of the world include a variety of ticks, as well as lice, keds, Oestridae, mange mites and fire ants. Helminth parasites include liver flukes (*Fascioloides* and *Fasciola*), gastrointestinal nematodes of the family Trichostrongylidae, pulmonary lungworms of the genus *Dictyocaulus* and extra-pulmonary lungworms of the family Protostrongylidae. Chronic wasting disease is principally important in North America, where the disease occurs in wild cervids in a limited area and has been reported in farmed deer in a small number of states in the United States of America and one province in Canada. These diseases are summarised in terms of their classification, epidemiology, clinical signs, pathology, diagnosis, treatment and control.

Keywords
Bison – Chronic wasting disease – Control – Deer – Diagnosis – Infectious diseases – Lungworm – Malignant catarrhal fever – Ostertagia – Wildlife.

Viral diseases: DNA viruses

Herpesviruses

Malignant catarrhal fever

Introduction

Malignant catarrhal fever (MCF) is the most important viral disease of farmed or ranched bison (*Bison bison* and *Bison bonasus*) and deer (7, 9, 16, 20, 28, 53, 80, 118, 128). The disease, as a clinical entity, is known on all continents and in most countries. At least three types of the virus have been implicated in the disease.

The disease is caused by members of the gammaherpesviruses group. Alcelaphine herpesvirus-1 (AHV-1), carried asymptomatically by the wildebeest (*Connochaetes taurinus*) in Africa, has been diagnosed in disease outbreaks in zoos in North America. Ovine herpesvirus-2 (OHV-2) is carried asymptomatically by sheep (117). A third, as yet unnamed virus, has been detected in an outbreak of MCF in white-tailed deer (*Odocoileus virginianus*) (80).

There are at least 25 herpesviruses serologically related to AHV-1 that have been detected in a variety of ruminants, but only AHV-1, OHV-2 and a new virus in white-tailed deer appear to be pathogenic (80, 107, 116, 117).

Transmission

Of the three pathogenic forms of the virus, AHV-1 from wildebeest (WA-MCF or wildebeest-associated MCF) is the best understood. Sheep-associated MCF (SA-MCF) occurs when sheep are maintained together with susceptible species. Ewes experience a recrudescence of infection in late pregnancy and

shed virus in lacrimal, nasal, oral and vaginal secretions, infecting the next generation of lambs soon after birth. This may also explain the seasonal increase in cases in deer and bison in late winter and early spring. The disease has also occurred in other circumstances, and direct contact is not necessarily required. Bison have contracted MCF when sheep were grazed at some distance, as far as 3 km, upwind. Wind-borne infections have been reported and deer carried in a truck that had been used earlier for transport of affected sheep have contracted the disease.

Stress plays a major role in the development of MCF. It is well known from studies in the United Kingdom (UK), Australia and New Zealand that the incidence of the disease rises in winter/spring, at a time when conditions are harsh and deer may be in poor condition; disease incidence rises in crowded conditions. An outbreak in bison held in a saleyard that also contained sheep occurred in late 2000. Forty-three of 163 bison were affected, became sick, and died in a period of 170 days (at 8 January 2002) following the sale (9).

Malignant catarrhal fever occurs more frequently in intensively managed properties than on extensive operations (7). Injections of dexamethasone have caused adult wildebeest to excrete virus, and a sika deer (*Cervus nippon*) that had recovered from an initial infection, when exposed to dexamethasone redeveloped clinical signs.

The disease only affects a small number of susceptible stock in a group, although occasionally higher morbidity can occur (7, 9, 16, 28, 118).

Other species affected

The disease has been reported in many species of ruminant (107). Serological evidence of infection with agents related to AHV-1 has been demonstrated in 27 species in the bovid family. There is a wide variation of susceptibility among deer species. In all, MCF has been reported from 14 species of deer. Several species are highly susceptible. Among them are Père David's deer (*Elaphurus davidianus*), rusa deer (*Cervus timorensis*), axis deer (*Axis axis*) and white-tailed deer (19, 28, 53, 83).

Clinical signs

Malignant catarrhal fever has been seen in forms ranging all the way from peracute to chronic disease (7, 19, 117). The outcome is invariably fatal. In the peracute form, infected animals may die with no prior clinical signs. The most common manifestation of MCF in deer is a very rapid onset of bloody diarrhoea, dark stained urine, marked depression, and death within 48 hours. Chronic cases last from a few days up to five weeks and may develop the so-called 'head and eye' form of the disease which is the type normally seen in cattle and bison and less often in deer. Congestion of the mucous membranes occurs and excessive salivation, lachrymation and nasal discharge are seen. Bilateral corneal opacity develops, there may be haemorrhage into the anterior chamber of the eye and as cases become more chronic, the eyelids may swell and eventually become closed with a mat of catarrhal exudate. The muzzle, and often the vulva, develops a dry crusty appearance and erosions of the mucous membranes of the mouth are evident. Lymph nodes are swollen and the superficial nodes can readily be palpated. Nervous signs are also sometimes seen, particularly an initial dullness, followed by inco-ordination and hyperesthesia.

The clinical pathology most often involves an initial rise in white blood cells, followed by a marked leucopenia. A left shift also occurs, and if diarrhoea is present the ensuing dehydration gives rise to increases in packed cell volume and haemoglobin concentrations. Blood coagulation parameters characteristic of disseminated intravascular coagulation have been measured in experimentally infected red deer (*Cervus elaphus*) (133).

Pathology

The lesions seen at post-mortem vary somewhat according to the clinical course of the disease. In peracute forms, gross changes may be limited. In less rapidly fatal forms, lesions may be seen throughout the entire gastrointestinal tract. These include necrotic erosions, congestion, oedema and frank blood in the intestinal lumen (16, 19). Lymph nodes are usually swollen, and the mesenteric nodes may be grossly enlarged, with much oedematous fluid around them. Cross-section of these and other nodes shows necrosis and inflammation (16).

Congestion and reddening of the mucous membranes may be seen throughout he respiratory tract and there may be extensive ulceration and necrosis. Both the bladder and kidney are often covered in haemorrhagic foci, and in the kidney characteristic lesions may be seen under the capsule and in the cortex. These appear as raised white foci 1-4 mm in diameter, sometimes surrounded by a thin red zone of haemorrhage. The vaginal mucosa may also contain haemorrhagic lesions. Other sites where lesions may be seen are the joints, in which swelling, reddening and excess fluid may be evident, and the brain, which may be congested (16).

The most characteristic histological lesion is a lymphocytic vasculitis affecting the arteries, arterioles, veins and venules. In peracute cases, they are characterised by invasion of lymphoid cells into all levels of the vessels, and in the most severe cases they may even occlude the lumen. Vascular haemorrhages, epithelial degeneration, and lymphoid hyperplasia with invasion of lymphoid cells into non-lymphoid tissues, also occur, especially in chronic cases. These infiltrations may be visible macroscopically in the kidney.

Diagnosis

Clinical and epidemiological history and post-mortem diagnosis are important means of confirming a case of MCF (16, 133). There are two tests currently available for the diagnosis of clinical MCF. A competitive inhibition enzyme-linked immunosorbent assay (ELISA) test has been developed which detects specific antibodies to an antigenic epitope, which

is conserved across all viruses in this group. The sensitivity of this test is 95% to 100% and the specificity is 91% to 100% when the test is used to diagnose clinical cases of MCF (9, 81). Polymerase chain reaction (PCR) tests are available, but they are specific to each virus within the group. The PCR test for OHV-2 viral deoxyribonucleic acid (DNA) has a sensitivity of 95%-97% and a specificity of 94% to 100% when used on fresh tissues or peripheral blood lymphocytes (65).

Treatment and control

Treatment, even of chronic cases, is considered hopeless. There is no vaccine available. Prevention requires that sheep or wildebeest do not have direct or indirect contact with susceptible species (7).

Other herpesviruses

The two European cervid herpesviruses have been named herpesvirus of cervidae types 1 and 2 (CerHV-1 and CerHV-2) (66, 102, 121, 138). The latter is also known as rangiferine herpesvirus 1 (RanHV-1). They are alphaherpesviruses closely related to bovine herpesvirus-1 (BHV-1). Serological diagnosis of BHV-1, which causes infectious bovine rhinotracheitis (IBR) in cattle has been reported in North American cervids and bison (18, 45, 71, 135). The cervids include white-tailed deer, mule deer (*Odocoileus hemionus*), caribou (*Rangifer tarandus*) and wapiti (*Cervus elaphus* subspp.).

Experimental infection of mule deer with BHV-1 resulted in signs that included anorexia, depression, excessive salivation and signs of respiratory distress, but experimental infection of red deer produced no clinical signs (18, 119).

Nasal exudate, semen and foetal fluids are implicated in the spread of IBR. Of these, aerosol spread from coughing is considered most important, although venereal transmission can occur (115). The mode of transmission of CerHV-1 is unknown, but the virus replicates in both the upper respiratory tract and the eye and it appears that both contact and aerosol infection are important in disease transmission. All outbreaks of clinical disease so far reported have been associated with stress, such as weaning. A severe outbreak of ocular disease, with some deaths, has been reported in recently weaned farmed deer calves and has spread to several properties after a sale (101). CerHV-2 of reindeer (*Rangifer tarandus*) spreads predominantly by the venereal route (102).

The most obvious consequence of infection with CerHV-1 in red deer calves is a severe conjunctivitis (66). Excess lachrymation progresses to involve swelling and oedema of periorbital tissue, scleral injection, purulent ocular and nasal discharge, hypopyon, corneal opacity without ulceration and photophobia. In the first outbreak reported, several calves died, but their deaths were ascribed to trampling (66). In later outbreaks, very severe corneal damage was seen, and corneal rupture occurred in several calves. Recovery often took as long as two months (101).

The clinical signs of IBR in bison have not been previously reported, but are similar to those in cattle. They include fever, anorexia, reddening of the nasal mucosa, ocular discharge, nasal discharge, an increase in respiratory rate and a cough. Some animals may develop reddened eyes that may be mistaken for pinkeye. In adult cattle, abortion is a common sequel of the disease, occurring up to 90 days after infection.

Diagnosis is based upon clinical signs and epizootiology, virus isolation, and serology. Conjunctivitis has been seen in cases of bacterial infection with *Moraxella* spp., and could also be mistaken for the head and eye form of malignant catarrhal fever. Virus may be isolated in swabs taken from nasal and ocular exudate. Most commonly, diagnosis of IBR is made on post-mortem examination of aborted foetuses.

On account of the antigenic similarity of BHV-1 and herpesvirus of cervidae type 1 (HVC-1), it is possible that surveys in Europe and North America in which antibodies to the former have been detected were actually showing an incidence of the cervine agent, or even of another closely related virus (79). In Denmark, HVC-1 virus was isolated and characterised in red deer showing antibody reaction to BHV-1 (121). It is possible that the serological tests used in the North American studies failed to distinguish among the various viruses. Recently, differentiation of the various alphaherpesviruses has been accomplished using genomic techniques (123).

No specific treatment has been described, but supportive therapy with antibiotics may be used to control secondary bacterial infection. No control measures are described, but in common with other similar diseases, an avoidance of stress should be attempted.

There have been no protocols reported for controlling IBR in bison. In Canada and the United States of America (USA) there are many commercially available modified live and killed IBR vaccines marketed for use in cattle. None of them have been approved for use in bison. Most of these vaccines provide good protection against the occurrence of IBR in cattle, but their efficacy in bison has not been established. Despite this, it is common practice for bison farmers to use them, and for veterinarians to prescribe them. The same caveats for use of live or killed vaccines in cattle apply in bison.

Adenovirus

An adenovirus caused an outbreak of haemorrhagic disease, believed to have killed thousands of mule deer in California in 1993 (159). This was subsequently shown to be a new serotype of adenovirus and it was also implicated in the deaths of fourteen captive black-tailed deer (*Odocoileus hemionus*) (14, 160). One of eight white-tailed deer fawns experimentally inoculated with the mule deer virus succumbed (161).

Transmission occurs by direct contact, and this virus is highly contagious. In experimental investigations, in-contact fawns developed the disease just as rapidly as those inoculated either intravenously or via mucous membranes and 80% of fawns developed disease within six days (160).

Many deer die of an acute haemorrhagic disease characterised by ptyalism, diarrhoea, seizures and recumbency prior to death (160). Other deer that linger may become emaciated.

The acute form of the disease is characterised by widespread haemorrhages of mucous membranes, pulmonary oedema and enteropathy. These lesions are similar to those seen in the haemorrhagic diseases caused by bluetongue (BT) and epizootic haemorrhagic disease (EHD) viruses. The more chronic forms may demonstrate severe necrotic lesions of the upper alimentary tract, including pharyngeal or gingival abscesses, starvation, verminous pneumonia, trauma, bacterial septicaemia and systemic neosporosis. Several secondary bacteria have been isolated from these lesions (159, 160).

Microscopic lesions of acute to subacute vasculitis with eosinophilic and amphophilic intranuclear inclusions in endothelial cells are seen in the majority of cases. Other lesions include non suppurative interstitial pneumonia, ulceration and abscessation of the upper alimentary tract, haemorrhagic enteritis, non-suppurative encephalitis, lymphoid depletion and centrilobular hepatic necrosis (159).

Confirmation of adenovirus diagnosis at necropsy has been achieved using virus isolation, transmission electron microscopy, fluorescent antibody tests and immunohistochemistry (159, 160).

Serum neutralisation (SN) and ELISA tests have been developed (78, 160). In SN tests, there is some degree of cross-reaction with other adenovirus types, most commonly, but not in all cases, with goat adenovirus type 1 or 2 (160). The ELISA developed after purified adenovirus was obtained from virus-infected black-tailed deer had a high degree of correlation with SN titres. As the ELISA is considered more convenient to use than the SN test, it is thought to be an adequate test for serological detection of adenovirus infection in deer (78).

The lack of any vaccine, and the highly contagious nature of this disease, make control difficult. Serological testing may provide a guide to previous infections, but there is no information on possible carrier status. Nonetheless, deer originating from herds with a history of the disease should not be transferred to other captive herds or released into the wild (14).

Poxvirus

Parapox

Introduction

A new specific parapoxvirus infection of red deer was identified in New Zealand in 1985 (62, 120). The virus is a member of the parapox genus of the *Poxviridae* family that has specific characteristics that distinguish it from other poxviruses and parapoxviruses. Restriction endonuclease analysis of viral DNA and PCR has shown it to be different from parapoxviruses of sheep or cattle (62, 67).

Parapoxvirus isolated from bighorn sheep (*Ovis canadensis*) has been experimentally transmitted to mule deer, white-tailed deer, wapiti, moose (*Alces alces*) and pronghorn antelope (*Antilocapra americana*), but the lesions did not extend beyond the inoculation site in either wapiti or white-tailed deer (76).

Poxviruses in the parapox group have a world-wide distribution, but the specific parapoxvirus of deer has not been reported outside New Zealand. The three situations where severe infections and deaths have occurred are infection in stags in velvet, hinds and calves close to calving, and recently stressed or transported animals (62, 120). As all deer in New Zealand originated from other countries, it is possible that the virus is also present elsewhere but has not been either seen or reported. A single case of natural infection has been seen in Canada, but the virus was not identified beyond the fact that it was recognised by electron microscopy as a member of the group (53).

Transmission of orf occurs by direct contact with active lesions on infected animals, or with exudates or scab material in the environment. It is probable that parapoxvirus infection in deer spreads in a similar manner. In severe outbreaks in New Zealand, the presence of thistles in paddocks was considered to play a major role in the development of lesions on the limbs (62). The parapoxviruses are extremely resistant to most environmental factors and can exist outside the body for long periods of time.

Clinical signs

In New Zealand, the most common clinical signs are scabs on the velvet antler, ears, muzzle, face and inside the lips. In more severe cases, the lesions develop over widespread parts of the body and in one outbreak 21 of 55 recently captured animals were severely affected and died (62). In a single case seen in Canada an 8-month-old wapiti calf developed ulcerative lesions at the junction of the muzzle and dental pad, which had resolved 4 weeks after onset (53).

Pathology

Lesions appear as severe proliferative dermatitis with extensive hair loss and scab formation. Removal of the scabs leaves a raw red surface. The lesions on antler velvet appear as multiple raised vesicopustules. The oral lesions are typical of a proliferative viral dermatitis and closely resemble those seen in sheep with orf.

Diagnosis

Diagnosis is based upon the clinical and epizootiological picture, but must be confirmed by histological examination as

well as electron microscopy and identification of the typical appearance of the parapoxvirus. For confirmation, DNA identification of the virus can be used and a PCR that can distinguish among the four known members of the genus has been developed (67). Secondary bacterial invasion of lesions is likely, and several different organisms could be involved

Control

Generally, severe outbreaks of disease occur when the infection is introduced to a susceptible population. There are few cases of clinical disease in subsequent years, except when new animals are introduced, suggesting that the majority of deer experience subclinical infections in the endemic state. There are suggestions that control of prickly plants may help reduce the incidence of the clinical disease (62).

The preparation of autogenous live vaccine from infected animals, which has been practised as a means of control for orf in sheep and other domesticated animals (e.g. muskoxen), has not been reported in deer.

It is difficult to be sure of the significance of parapoxvirus infection for deer species outside New Zealand. The experimental work has shown that parapoxvirus of sheep does not pose a threat to wapiti (76).

Viral diseases: RNA viruses

Picornavirus

Foot and mouth disease

Introduction

Foot and mouth disease (FMD) is caused by an aphthovirus of the family *Picornaviridae*. Within the genus *Aphthovirus* there are seven immunologically distinct strains that form two groups on the basis of ribonucleic acid (RNA) hybridisation (139). There is evidence that different strains may differ in virulence for different species of susceptible animal (56). With the exception of Australasia, the disease is known world-wide and is endemic in many areas.

Many animal species, including humans, and all artiodactyls, are susceptible. Among cervids, infection has been reported in reindeer, moose, muntjac (*Muntiacus reevesi*), white-tailed deer, Eld's deer (*Cervus eldii*), sika deer, fallow deer (*Dama dama*) and red deer (56). There do not appear to be any reports of FMD in bison. In a series of trials with British deer, type O and type C virus were used to infect animals (41, 47). Although all species can be infected, there appears to be a range of susceptibility. Red deer and fallow deer develop only mild lesions and are much less susceptible than muntjac and roe deer (*Capreolus capreolus*), which develop severe potentially fatal disease (41, 47, 48).

In the 2001 outbreak in the UK, the disease was diagnosed on a deer farm on which four species of deer were stocked. Among red deer, fallow deer, Père David's deer and sika deer in the herd, clinical signs were seen only in sika deer, in the period between initial diagnosis and herd depopulation (T.J. Fletcher, personal communication). There were deaths among wild roe deer in the same outbreak.

Transmission

The FMD virus is extremely contagious, and is readily spread via respiratory secretions, saliva, urine and faeces. Mechanical spread via contaminated animal products occurs and a variety of fomites can carry the virus, which, within a pH range of six and nine is relatively resistant to environmental conditions (139). It has been shown that infected red deer can shed amounts of virus similar to cattle and sheep, and could so infect in-contact animals. It has also been shown that fallow deer and sika deer can become carriers (48).

Clinical signs

Experimentally infected red deer, whether they were infected by exposure to infected cattle, or by inoculation of virus, developed either inapparent infection or only mild clinical signs. Vesicles developed in the mouth of only a small proportion of the infected animals. Those that did develop vesicles remained alert, did not show excess salivation, and were not lame (41). On the other hand, the closely related sika deer developed severe signs, with copious salivation, depression, lameness, and ulceration of the oral mucous membranes.

Pathology

In experimentally infected red deer, the main pathological signs are restricted to the epithelium of the mouth and hard palate, where the small vesicles mentioned above were seen. No lesions developed on the coronets. In other species that die of acute infections, degeneration of the heart muscles is a feature of the disease.

Diagnosis

Diagnosis is usually based on clinical signs, and confirmed by virus isolation and serological testing. In mildly affected animals, viral recovery from the oro-pharynx, and serological studies, are necessary (42). Serology involves either one of a variety of ELISAs, or virus neutralisation. Specific strain identification is achieved either with ELISA or PCR testing (139). If vaccination has been carried out, serological testing with cross-neutralisation must be used to differentiate between field and vaccine strains (139).

Serological evidence of vesicular stomatitis has been found in wapiti, and as this disease produces similar clinical signs to FMD in other species, it too should be considered in the differentiation of vesicular lesions in either red deer or wapiti and possibly other deer species.

Control

The old adage 'prevention is better than cure' applies particularly to FMD as control in free-ranging populations of deer or bison is likely to be impractical or impossible. In most countries, FMD is a reportable disease. Therefore, control measures are likely to be mandated under animal health regulations. In the face of an outbreak, one or more of the three basic control measures of eradication of infected stock, and sometimes nearby susceptible animals, quarantine and vaccination may be used. Eradication of farmed or ranched deer or bison may be possible, but the elimination of free-ranging animals may be almost impossible, although density reduction may be a practical measure to reduce the possibility of transmission. Since vaccination of domestic livestock is used in some countries, this may help prevent entry of the disease to an area but vaccines have not been tested in any deer species, or bison.

Reovirus

Bluetongue and epizootic haemorrhagic disease

Introduction

Both BT and EHD are vector-borne virus infections that affect deer and other ruminants in North America. The viruses are closely related members of the *Orbivirus* genus, which also includes Ibaraki virus. Bluetongue and EHD are often discussed together because they produce a similar haemorrhagic syndrome in susceptible white-tailed deer, but they are distinct diseases and can occur simultaneously in a single animal, or be detected during the same outbreak (58, 140).

Bluetongue

World-wide, there are at least 24 serotypes of BT virus and ten serotypes of EHD virus (64). In Africa, at least twenty BT types are known, while there are five BT types known in North America and four BT types in Australia. In each of these regions there is a degree of homology of the viruses, while they are distinct from group to group (58). Of all these serotypes, only four BT and two EHD types have been associated with clinical haemorrhagic diseases in natural or experimental situations (64).

The natural vertebrate host of BT is considered to be cattle, but non-clinical infections can become established in wapiti and goats, and the virus has been isolated from American bison (64). Cattle, wapiti and goats can have high concentrations of circulating virus for prolonged periods and can probably act as long-term carriers (99). Vertical transmission from a female wapiti to her calf has been demonstrated (132). Bluetongue virus was isolated between 5 and 9 days post-infection from five experimentally infected wapiti, three of which developed mild clinical signs, and also from two of them that were given steroids 105 days later (99). Sheep and deer of the genus *Odocoileus* are considered to be only short-term carriers of the disease, although they can rapidly develop clinical signs. Bluetongue (also known as soremuzzle) is also recognised as an important disease of sheep in some parts of the world.

In the carrier state, the BT virus is maintained inside vertebrate cells where it is protected from antibody and persists during periods when the vector is unavailable. It is also possible that the virus overwinters in the insect. Bluetongue virus has been found in the semen of infected domestic bulls, and infection has been transmitted by artificial insemination, so that stringent regulations concerning the testing of potential donors are an important part of protocols for testing wapiti and deer used as semen donors in endemic areas. The virus can also be transmitted transplacentally.

Although BT is principally a disease of white-tailed and mule deer, several other species can become infected. Bighorn sheep and pronghorn antelope can develop fatal infections (140), and antibodies have been found in wild carnivores and rodents, although their significance is not known (64).

There have been local incursions into the Okanagan valley of British Columbia in 1987 and 1999 that involved wapiti, cattle, mountain sheep and goats, white-tailed deer and bison (31). Bluetongue occurs in the lower 48 States of the USA and has been associated with die-offs of free-ranging wildlife. The incidence is greater in southern States than in northern and in Texas the disease is enzootic and most deer have antibodies (59).

Epizootic haemorrhagic disease

With regard to EHD, white-tailed deer are the most susceptible species, and outbreaks that have occurred are usually characterised by local die-offs of a high percentage of animals in a small area. In spontaneous outbreaks, mule deer and pronghorn antelope have also been affected, but not to the same extent as white-tailed deer. Serological evidence of infection has been found in black-tailed deer, red deer, wapiti, fallow deer and roe deer, as well as domestic cattle. Experimentally, the EHD virus has been inoculated into wapiti, red deer, fallow deer, roe deer, muntjac, cattle, sheep, goats and domestic pigs. All but the goats and pigs developed viraemias, but none showed clinical signs (49, 57).

Transmission

Transmission of both diseases is primarily via biting gnats or sand-flies of the genus *Culicoides*. In North America, the main vector of BT is *C. sonorensis*, the range of which is limited. This may explain the distribution pattern of the disease, its virtual absence from Canada and its low to nil seroprevalence in the upper mid-west and northeastern USA (60). However, many other members of this insect genus have been incriminated as potential vectors (64). Bluetongue infection is most commonly seen in late summer and early autumn, especially during wet seasons. Wind-borne movement of vectors over long distances has been incriminated in new outbreaks of BT in some parts of the world.

Epizootic haemorrhagic disease has been reported in most of the States of the USA as well as in western Canada. The disease occurs less frequently in northern areas, which is no doubt related to the lack of vectors at these latitudes for much of the year (31, 43, 59). *Culicoides variipennis* is considered to be the principal biological vector of EHD (44, 59). The incubation period is usually from 10 to 20 days, and outbreaks are seen in late summer and early autumn, often associated with wet weather. Cattle develop chronic viraemia and may be reservoirs of infection.

Clinical signs

The clinical signs of bluetongue vary widely according to the species affected, as well as the strain of the virus and the immune state of the animals (59, 140). The most severely affected species are sheep and white-tailed deer. In susceptible sheep, the incubation period is usually less than a week. White-tailed deer and sheep can die in less than 24 hours, after suddenly developing a high fever, and showing signs of respiratory distress. There may be a bloody nasal discharge and marked reddening of the buccal and nasal mucosa. Oedema, especially of the head and neck, is often evident. Diarrhoea and dysentery may occur, and inflammation of the coronary band may cause severe lameness. Congenital deformities of new-born animals, or abortion, have been seen in cattle, sheep and deer. Mortality in white-tailed deer herds may reach 50%, but in enzootic foci outbreaks of clinical disease are rare.

Bluetongue causes production losses following infection, that may be manifested as impaired reproduction, poor weight gain and general unthriftiness. Foot lesions may show in recovered animals or those that develop a chronic form of the disease.

In white-tailed deer, EHD manifests as an acute or peracute haemorrhagic disease (59, 141). Mortality rates may be high, and up to 90% of infected deer usually die within 8 to 36 hours of the onset of signs after an incubation period of 5 to 10 days (59).

Pathology

The gross pathological picture varies according to the severity and the two diseases cannot be distinguished from one another by either gross or histopathological examination. In peracute forms, severe oedema of the head, neck, tongue and lungs may be evident. In the acute form, the oedema may be accompanied by haemorrhages in many parts of the body including the heart, and the entire gastrointestinal tract. There may be areas of necrosis or ulceration on the dental pads, tongue, hard palate, rumen and abomasum (59).

Histologically, disseminated vasculitis and thrombosis, associated with haemorrhages, degenerative changes and necrosis are seen in many organs, but the lesions can be very subtle if the animals die early in the course of the disease (64).

Diagnosis

Haemorrhagic diseases present with such a diverse range of signs that field diagnosis is often very difficult, except in the more severe forms. The occurrence of a haemorrhagic disease during vector seasons should increase the index of suspicion. For confirmation, virus isolation and serological tests must be performed. Presence of antibody alone is not enough to confirm diagnosis, and may only indicate that exposure to the agent has occurred.

There are a number of serological tests available to assist in BT and EHD diagnosis. Agar gel immunodiffusion (AGID) tests have the advantage that they can be performed with minimal laboratory equipment. However, they are not as sensitive as some other tests and they do not provide adequate indication about a specific serotype. The only test that can provide indications of a previous exposure to a specific virus serotype is the SN, but cross-reactions between serotypes are reported. Several ELISAs have been developed. The competitive ELISA (cELISA) for BT is available and reliable, but there is no cELISA available for EHD (64).

The only means of confirming the causative agent of haemorrhagic disease is virus isolation. Tissues for viral isolation (blood collected in anticoagulant, lung, liver, lymph node or spleen), should be collected as soon as possible after death and carried to a laboratory experienced with the disease within a few hours, or frozen and shipped. Tissues from suspect cases can be inoculated into embryonated chicken eggs or a variety of tissue culture cell lines for virus isolation and identification. If febrile animals are found, blood inoculation into susceptible sheep is a useful diagnostic technique (59, 141).

There are also several molecular techniques used for BT and EHD diagnosis (64, 124). These include dot blot, *in situ* hybridisation and PCR. Of these, PCR provides much quicker results than does virus isolation and has the advantage that it can be applied to tissues that may not be suitable for virus isolation. Moreover, viral RNA may remain in a recovered deer host for up to 160 days, long after virus isolation techniques can detect the pathogen or animal inoculation can show its presence (64).

Differential diagnosis

There are a number of diseases that can be confused with either BT or EHD. The picture is further complicated by the wide range of clinical signs that are seen with BT, and the differing susceptibility among species. In white-tailed deer that die of haemorrhagic disease, the two diseases must be distinguished from one another. A haemorrhagic disease of mule deer caused by an adenovirus has been described in California (159).

Control

Prevention is the keyword in the control of these diseases. Stringent testing is already in place for the detection of

antibodies, and as carrier states are known to exist, the finding of a positive result is usually enough to preclude the issuing of movement permits. Quarantine, and the movement of animals outside vector seasons, are also important considerations.

When susceptible white-tailed deer are moved to areas in which the disease is endemic, they stand the risk of becoming infected and suffering high mortality rates.

There are no vaccines approved for use in deer or bison.

Flavivirus

Bovine viral diarrhoea

Introduction

Bovine viral diarrhoea (BVD) virus, a *Pestivirus* in the family *Flaviviridae* is recognised world-wide as an important disease of cattle and has been detected in numerous species of bovid and cervid (70, 115, 145). It has been diagnosed, either through virus isolation or the presence of antibody, in fourteen species of deer as well as in both North American and European bison (145) (Table I). Clinical signs or lesions typical of BVD have been found in Père David's deer, axis deer, fallow deer, reindeer and muntjac. Pestiviruses have been isolated from necropsy material from several species of deer, but in no instance has the role of the virus been clearly linked to the cause of death. Experimental infections have been established in wapiti, mule deer and white-tailed deer. New pestiviruses that have different DNA genotypes have been found in several deer species, but it is not known if they cause disease.

In wapiti, experimentally infected animals, as well as one in-contact non-inoculated animal developed viraemia, nasal shedding and/or seroconversion to either type 1 or a virulent cattle type 2 strain of the virus, although none showed any clinical signs (137). Similarly, in experimentally infected white-tailed and mule deer fawns, no clinical signs were observed but virus was isolated from white blood cells of four of five fawns from day 2 through to day 15 post inoculation, and nasal swabs of three fawns from day 2 through to day 15 (144). It is therefore possible that these species can transmit the disease.

Clinical signs

None of the range of clinical signs described in cattle has been reported in deer species. Little is known about the behaviour of the virus in bison herds, other than that it is present and that it can cause disease (8, 135). The Yellowstone National Park survey of 1991-1992 demonstrated antibody titres in 31% of bison tested (135). Acute BVD in bison has not been reported in the literature, but can cause mortalities in bison at any age (8). However, neither foetal infection nor mucosal disease have been confirmed in bison, although cytopathic BVD virus (BVDV) has been repeatedly isolated from bison with clinical disease and pathology consistent with mucosal disease as described in cattle (K. West, personal communication).

Table I
Virus agents or viral antibodies confirmed in deer and bison
(adapted and updated from 53)

Viridae family	Disease	Species
DNA viruses		
Herpesviridae	Malignant catarrhal fever	A, BA, MD, R, Re, S, W, WT, O
	Infectious bovine rhinotracheitis	R, W
	Herpesvirus of cervidae (HVC-1)	R, O
	Rangiferine herpesvirus-1	Re
Adenoviridae	Adenovirus	MD, R, WT
	Bovine adenovirus group A	R
Poxviridae	Parapox	R, W
RNA viruses		
Picornaviridae	Foot and mouth disease	F, R, Re, S, WT, O
Reoviridae	Bluetongue	BA, BE, R, S, W, O
	Epizootic haemorrhagic disease	MD, R, W, WT
	Rotavirus	R, W
	Reovirus	R
Togaviridae	Cache Valley virus	W
	California group viruses	WT
	California encephalitis	W
	Jamestown Canyon virus	W, O
	La Crosse/snowshoe hare virus	W, O
Flaviviridae	Bovine virus diarrhoea	A, BA, F, MD, R, Re, S, W, WT, O
	Louping ill	R
Paramyxoviridae	Parainfluenza 3	BA, W
	Influenza (Bangkok)	R
	Influenza (USSR)	R
	Rinderpest	WT
Rhabdoviridae	Rabies	R
	Vesicular stomatitis (New Jersey serotype)	W
	Brest AN/219	R
Coronaviridae	Unclassified or uncertain	BA, W, R
Astroviridae	Eyach virus	R
	Sicilian sand-fly fever	R

A : Axis deer
BA : *Bison bison*
BE : *Bison bonasus*
F : fallow deer
MD : mule and black-tailed deer
O : other deer
R : red deer
Re : reindeer
S : sika deer
W : wapiti
WT : white-tailed deer

Pathology

At post-mortem examination in cattle, the basic lesion of mucosal disease and peracute BVD is small vesicle ulceration that occurs anywhere in the gastrointestinal tract from the oral cavity to the colon. In mucosal diseases, cases with short courses or peracute BVD, there may be widespread haemorrhage. There may also be lesions on the feet (115).

Diagnosis

Ante-mortem diagnosis of BVDV infection can be performed by virus isolation or PCR on buffy coat. Serum can also be used to detect persistently infected animals. The SN test has been the standard test to detect the occurrence of rising BVDV titres and ELISAs are a rapid and economical alternative, although ELISAs have not gained wide acceptance (115).

Most commonly, a diagnosis of BVD is made on post-mortem examination and submission of samples to a diagnostic laboratory. Confirmation of diagnosis should include findings of gross and histological lesions with positive virus isolation or detection of BVDV by PCR or immunohistochemistry. Virus can be isolated from numerous tissues, particularly lymphoid tissues associated with the gastrointestinal tract. Skin samples from dead or live infected cattle can be used to identify the virus even if the carcass is decomposed or partly eaten by scavengers.

Control

There are no protocols established for controlling BVD in deer or bison. In cattle, control of BVD has proven to be difficult for some forms of the disease. Vaccination may not provide adequate protection against foetal infection and the formation of persistently infected cattle. Vaccination may, however, provide adequate protection against acute BVD infection and the thrombocytopenic form of the disease in cattle.

Despite this, many bison producers use both live and killed cattle BVD vaccines although none of them have been registered for use in bison and their effectiveness has not been established. The use of these vaccines in bison should be approached with caution. There are variations in the constituents of both modified live and killed virus vaccines and the adjuvants and immune stimulants found in killed BVD vaccines may have unfavorable affects on bison calves (8). Modified live BVD vaccines have been observed to cause diarrhoea in recently weaned bison calves on bison ranches, but older animals on bison ranches have not been observed to be as susceptible to the harmful effects of some of these vaccines (8). In the past, modified live virus BVD vaccines have been associated with abortion when administered to pregnant beef cattle. These vaccines therefore should probably not be administered to pregnant bison cows.

Rotavirus and coronavirus

Introduction

These two viruses, which are from different viral families, have been associated with scouring in young deer and bison. They are amongst the most commonly isolated organisms in such cases. Rotavirus has been isolated from outbreaks in red deer under 10 days of age (15, 143) and scouring bison calves that were kept in overcrowded conditions (8).

Coronavirus has been isolated in scouring wapiti neonates as well as sambar deer (*Cervus unicolor*) and white-tailed deer (130, 142). A coronavirus that has a close molecular relationship to bovine coronavirus has been isolated from 10-month-old clinically affected wapiti calves and grown in cell culture (88).

Clinical signs

The most consistent clinical sign in bison calves and young deer is a watery diarrhoea. As the disease progresses, the calves with scours may become dehydrated, weak and depressed. They may stagger and will often become recumbent before death. In some cases, the calves show minimal clinical signs and may be identified as being sick only when the disease has progressed to the point where treatment is unrewarding, and death is impending. Since bison calves have the ability to mask clinical signs of disease, it is important to constantly monitor the faecal excretion. This involves observing the calves while they nurse, observing the perineal area of calves, and observing the bedding areas of calves for diarrhoea. In some instances, the first indication of scours is the occurrence of mortalities in the herd (8).

In deer, affected animals also soon become dehydrated and up to 50% mortality may occur. Bacterial agents, especially *Escherichia coli* may play an important role in exacerbating the disease (15, 130). *Cryptosporidium* has also been implicated in outbreaks of neonatal diarrhoea in both wapiti and red deer (15). A high prevalence of antibodies to rotavirus has been found in adult red deer (4).

Pathology

Affected animals develop degrees of dehydration, emaciation and fluid faeces in the intestinal tract. Faecal samples taken from calves affected with scours should be submitted to a diagnostic pathology laboratory for bacterial culture and virus isolation. Post-mortem tissue samples should be sent to a pathology laboratory for histopathology, bacterial culture and virus isolation.

Many bison calves with scours recover spontaneously (8). Treatment of scouring deer and bison calves is especially challenging, as the decision to treat may often mean a concomitant decision to proceed to hand rearing. Dams of both types of calf may refuse to accept a calf that has been taken away for periods longer than a few hours. If treatment is attempted, fluid replacement is of paramount importance, but the use of appropriate fluids is essential. Hypernatremia has been identified as an important mortality risk factor in wapiti calves treated with oral replacement fluids designed for bovines (17). Scouring wapiti calves rapidly become severely hypoglycaemic and immediate attention must be given to this situation. Fresh drinking water should also be supplied to calves, even if they are receiving intravenous therapy (122).

Control

Many deer and bison farmers use combined cattle scour vaccines that incorporate one or both viruses and sometimes

E. coli antigen as well, despite the lack of any objective data on efficacy. Better results may be achieved by altering environmental conditions and management programmes, than by vaccinating the stock.

The occurrence of neonatal diarrhoea may be associated with many determinants other than bacterial, parasitic or viral agents, such as wet environmental conditions, overcrowding and poor nutrition of the dam. If a herd problem arises it would be prudent to try to identify not only a causative agent specific to the herd, but also other environmental factors that may be significant contributors to the development of the disease.

Parasitic diseases: ectoparasites

Introduction

Arthropod parasites may be involved in the transmission of other organisms, or they may have a direct effect upon the host. They may act as true vectors, in which case the organism undergoes life-cycle changes in the arthropod, or they may act as mechanical transmitters of infection. Direct effects include bloodsucking leading to anaemia, irritation, and annoyance or physical damage leading to losses of production and reduced weight gain. Most arthropod parasites are ectoparasites, but some may affect other organs.

Ticks

Introduction

Ticks can act both as pathogen vectors and as severe irritants. Direct effects include trauma, pruritis and local inflammation. Several species of tick can cause tick paralysis. A large number of tick species have been reported from deer. Presidente has provided a list of 38 species of tick found on sambar deer (108). Only two species, *Dermacentor albipictus* and *Haemaphysalis longicornis* are known to be the direct cause of significant problems. In North America, the white-tailed deer acts as a physical vector of *Ixodes* ticks that transmit Lyme borreliosis, although the deer itself is not considered to be a reservoir host. A recent extensive review of ticks that affect wild mammals of North America is available (1).

Dermacentor albipictus, the winter tick, is one of the most widely distributed ticks in North America and it causes the most severe lesions of irritation. It is generally recognised more as a serious parasite of moose than wapiti, and it has also been found in the hides of reindeer from a zoo (94). Up to 178,000 ticks have been recovered from a single moose hide, but one of the reindeer had over 400,000 ticks on it (154). It has also been found in large numbers on mule deer and can cause serious irritation and hair damage in wapiti. *Dermacentor albipictus* has also been found on white-tailed deer and bison (95).

The winter tick completes the three stages of its life-cycle on a single animal during the winter months. Long warm autumns and early spring snowmelts enhance the survival and transmission of this parasite. The larval ticks climb up vegetation in the autumn, and clumps of them can be found on the tips of plants. When a host walks past, the larvae leave the plants and attach to the animal. After attaching to the host, the larva feeds for a short while, and then molts into a nymphal stage. It goes through a rest period before feeding again, and again molting, this time into the adult stage. In spring, the adults take another meal, and the engorged females, after mating, drop off onto the ground. It has been shown that adult females that drop from the host before snowmelt have a poor chance of surviving. In Alberta, egg laying takes place in May and June and a single female can produce over three thousand eggs. The eggs hatch into larvae in August and September and climb nearby vegetation upon which they wait to attach to a passing host that brushes past. The duration of the larval inactivity varies markedly with location and climate (29, 30).

Heavily infested cervids may lose much of the hair on their bodies, due to frequent rubbing, licking, biting and scratching with the hind feet stimulated by intense irritation caused by the ticks, especially in late winter and early spring. The parasite can and has created such severe hair loss and debility that population die-offs, associated with late winter stress, have occurred. Experimentally infested moose show chronic weight loss, and changes in complete blood cell counts (50).

Treatment

A satisfactory treatment of animals infested with winter tick has not thus far been developed. Unfortunately, topical acaricides with sufficient residual action have not been licensed in North America, although several have been used by farmers.

Control

Paddock management has been tried as a method of tick control on wapiti farms. If the larvae are not picked up in the autumn they will not survive the prolonged period of inactivity and cold on vegetation over the winter, and so the cycle could be broken in a single year. Controlled burning has also been suggested as a means of eradication of winter tick (153).

Haemaphysalis longicornis is recognised as a significant problem of farmed deer in northern areas of New Zealand. Infestations can affect fawn survival and growth and production. The tick also causes direct damage to hides and velvet antler (158).

Attempts at control include animal sprays, pour-on medications, impregnated ear tags, modification of tick life-cycles, and pasture treatment. A topically applied 1% flumethrin solution has been shown to both be directly effective against the tick and to have a residual action of at least four weeks (55, 158).

Other ticks

In other areas of the world, different species of tick may play significant roles in deer farming (108, 158). The trading of deer

from one region to another brings a risk of transfer of their parasites. Effective control of ticks on any deer due to be transported is essential.

Feeding ticks may induce paralysis in a variety of animals. Tick paralysis has been reported in white-tailed deer, wapiti and bison, and it has been produced experimentally in mule deer (1, 53, 73).

Ticks may transmit fatal babesiosis in wapiti (61) but *Babesia* in other cervids does occur and ticks of the genera *Amblyomma*, *Dermacentor* and *Boophilus* have been implicated in North America. Babesiosis, transmitted by two species of the genus *Boophilus*, has been seen in white-tailed deer (1).

Ixodes ricinus, a three-host tick common in Europe, is ubiquitous on wild deer and is an efficient vector of *Babesia capreoli*, the virus of louping-ill and tick-borne fever (98).

Lice

Louse infestations are common world-wide. However, they are not a major problem in deer or bison. Lice infestations of deer have been recorded in the UK, New Zealand, Australia and North America.

Topical application of agents used to treat other external parasites is effective, as is the use of ivermectin for sucking lice, which controls louse infestations in other species. Lice of domestic species are known to rapidly develop resistance to many of these agents.

Biting flies

Apart from their role as vectors of specific parasites or pathogens, biting flies have been noted for their ability to cause annoyance to ruminants. Tabanid flies may drive wapiti from summer range, and in one instance tabanids and mosquitoes caused death in wapiti and bison through a probable combination of blood sucking and prevention of access to food (53). In areas where black flies or midges (*Simulium*) are a recognised pest, antler damage due to fly bites has been seen. Treatment, either of fly breeding areas, or of animals, with effective topical agents, may be difficult. The provision of shade may help by placing the animals out of reach of the tabanids.

Keds

Keds (louse flies), are found as wingless parasites on deer. Two species, *Lipoptena cervi* and *L. depressa* have been reported. The flies make only one flight in their life times, the adult emerging from the pupa on the ground, and flying to its final host where the wings break off. The adults feed on the blood of their host, but are not important as parasites.

Throat bot

Several throat bots of the genera *Pharyngomyia* and *Cephenemyia* are seen in the upper airways of cervids in the northern hemisphere (21). Their life-cycles involve the deposition of first stage larvae in the nasal cavity. The most common site where large numbers of third stage larvae may be found is the retro-pharyngeal pouch where they may grow to about 20 mm in length. The hatched larvae migrate to the pouches where they may either complete their development in 30-35 days or remain until spring. They then leave the host through the nares, fall to the ground, pupate and go on to complete the life-cycle. No treatment has been described.

Warble flies

Warble flies of the genus *Hypoderma* do not appear important as a parasite in wapiti or red deer in North America (72). *Hypoderma tarandi* occurs as an important parasite of reindeer and has a Holarctic distribution. It causes severe irritation and damage to hides, but there is a degree of age-related increased resistance (21). *Hypoderma bovis* infection occurs in bison. *Hypoderma diana* and *H. actaeon* are specific warble flies of deer species which occur in red deer and roe deer in several parts of Europe and are not transmissible to cattle (21). Caruncles are found on the back of the animal. Serological tests have been developed for cattle that can be applied to deer and bison (21). As *H. diana* and *H. actaeon* are not known in North America, deer species being imported should be treated with an effective compound during quarantine. Pour-on organophosphates or ivermectin are effective, but reinfection of farmed red deer from wild red deer may occur (21, 37).

Mange

The mange mite *Psoroptes cervinus* is probably the most important mite to affect wapiti (22). It does not appear to have been reported in red deer. *Demodex* spp. mites have been seen in New Zealand in red deer and in bison in Canada, but their significance is minimal or unknown (53, 147). The chorioptic mange mite, *Chorioptes bovis*, has a wide host range that includes cervids (115). Sarcoptic mange due to *Sarcoptes* spp. mites has been described in European red deer, moose, roe deer, sambar and reindeer (13, 134).

The modes of transmission are likely to be by either direct contact, or by contact at mutual rubbing points. The diseases occur predominantly in winter, in debilitated mature males and occasionally old females. Animals on poor range, under nutritional stress, are most susceptible.

Initially psoroptic mites cause irritation, wet eczema and hair loss. The skin surface is covered with dense, thick, moist scabs, and the lesions may be approximately symmetrically bilateral (126). The irritation can become so severe that animals lose condition and spend much time rubbing. In the worst cases of mange animals can die from exposure and heat loss due to hair loss (134). Demodecosis in bison appears as pus filled nodules around the eyes, perineum and ventral aspect of the tail. It has also been associated with small but palpable lesions on the neck, flank and shoulders (147).

Histological evidence shows that mites cause direct damage to the skin of the host, producing local inflammation and exudation of lymph. As many as 6.5 million psoroptic mites may occur on a severely affected animal (126).

A presumptive diagnosis in severely affected animals, seen most often in winter, shows them to be so debilitated that they will allow close approach. For diagnosis, microscopic examination of lesions, and skin scrapings are required, in order to identify the mites.

Severe cases of psoroptic mange are obvious, but milder cases, or those seen early in the development of the condition, must be differentiated from a variety of fungal, bacterial and parasitic conditions that could affect the host animal. The potential for mixed parasitic infestations to affect an animal should not be overlooked.

Demodectic nodules occur around the eyes and the perineal region in bison. The pus from nodules may be examined under a microscope for the presence of the mite.

Treatment

Improvements in range conditions and nutritional status of the national elk (*Cervus elaphus nelsoni*) herd in Wyoming, as well as destocking, have markedly reduced the incidence of mange in wapiti, and the disease no longer poses the problem that it once did (72). Well-managed farmed and ranched animals should rarely, if ever, develop this disease.

Fire ants

The fire ant (*Solenopsis invicta*) is a parasite that is seen most frequently in young white-tailed deer in Texas and other southern States. The ants originally entered the USA from Argentina in the 1930s. They have since spread to at least eleven States in the southeast and appear only to be limited by climatic conditions, being unable to survive below –12°C (53).

The venom produced by the ants is comprised of piperidine alkaloids which have been shown to be cytotoxic, hemolytic, bactericidal and insecticidal, killing insects other than fire ants (53).

Very young (<4 days old) fawns are found 'covered with fire ants'. They may lick the ants, and many parasites are found in the stomach. The ants grasp with their mandibles and then rotate their bodies and inflict numerous stings. Clinically, many fawns are found with facial injuries. The eyelids, nose and mouth may have multiple stings, and there may be severe corneal damage, with ulcerative and infiltrative keratitis. There may be lesions in many other parts of the body, particularly hairless areas such as the teats, axilla, groin, umbilical and peri-anal regions.

Fawns may be presented 'in extremis'. As many ants should be removed as possible. Fluid therapy should be initiated early and vigorously. Maintenance of body temperature is also crucial. Other symptomatic treatments such as antibiotic therapy, ophthalmic ointments and atropine where indicated, gastric lavage to remove ants, and energy replacement via milk replacer are all important.

Parasitic diseases: helminth parasites

Trematodes

Liver flukes

Introduction

A detailed and extensive review of these parasites is available in Pybus (111) and only a brief summary is offered here. Readers are encouraged to examine this review with its comprehensive bibliography.

On a world-wide basis, there are two liver flukes of importance to the deer farming industry. They are the so-called common, or sheep liver fluke *Fasciola hepatica* and the giant liver fluke, *Fascioloides magna* (5, 12, 34, 36, 82, 110, 114). Other flukes of the liver, rumen and caecum, have been detected in deer species, but have not been incriminated in any disease situations. Giant liver fluke has been detected in bison, but has seldom been reported to be associated with any clinical condition or severe pathological lesion (8).

Transmission

Liver flukes have indirect life-cycles which involve transmission through aquatic snails, usually of the genus *Lymnaea*.

Transmission of *F. magna* generally involves a wetland area, lake, pond or marsh. Moisture is an essential component of the life-cycle of the flukes as it is essential for egg development, and the movement of some of the larval stages, such as cercariae and miracidia (36, 40). In order for the intermediate stages of either species to develop, environmental temperatures must be high enough. Development is arrested in winter in most countries where temperatures are below 10°C. *Fascioloides magna* may produce eggs for up to five years. The eggs can overwinter, but are susceptible to desiccation.

Infection with either organism is therefore likely only where snails can become established. Snails can also establish themselves on irrigated ground that remains dry for long periods. At the time of irrigation they can rapidly emerge and spread large numbers of infective stages of the parasite. Infection with *F. hepatica* has been seen in stock drinking at troughs located as far as 3 km from dug-out dams in arid areas. *Lymnaea* snails were found in the water pipes supplying the troughs (J.C. Haigh, unpublished findings).

In terms of their effects upon deer species, the giant liver fluke is probably the more important of these two flukes. Pybus has

classified the species susceptible to infection (111). These are definitive hosts, all of which are cervids, dead-end hosts, which include moose, sika deer and bison, and aberrant hosts, which include roe deer. The definitive hosts listed are white-tailed deer, wapiti, caribou, black-tailed deer, mule deer, red deer and fallow deer.

Fascioloides magna was once confined to North America, but a translocation of infected wapiti to Italy in the late 19th Century led to the spread of the parasite in eastern Europe, where it established itself as an important problem of domestic sheep and other ungulates, and it appears to have become well established in red deer populations in some parts of Europe (36, 111).

Although wapiti and white-tailed deer are considered definitive hosts, they can develop heavy, sometimes fatal, natural infections, as can black-tailed deer and red deer (110, 111). There is one instance in which a rancher failed to rear any wapiti calves for consecutive years and at necropsy the attending veterinarian noted large numbers of immature flukes (*Fascioloides*) in the peritoneal fluid (D. Yarborough, personal communication). It has been suggested that heavy infestations with *F. magna*, accompanied by winter stresses and malnutrition, may contribute significantly to mortality of wild white-tailed deer. Chronic infection associated with hepatic fibrosis is seen in wapiti, red deer, fallow deer and caribou (111).

Fascioloides magna infections are abnormal in aberrant hosts. In moose, the life-cycle is rarely completed and it is thought that infection with this fluke will not persist in moose populations in the absence of white-tailed deer or wapiti. Extensive liver fibrosis commonly occurs, loss of condition and mortality have been reported (111). In cattle and bison, *F. magna* flukes are trapped in a heavy fibrous capsule within the liver and apparently do not complete their life-cycle. Clinical signs vary very markedly not only according to the species infected, but also to the level of infection.

Fascioloides magna causes a fatal disease in both domestic and bighorn sheep, and goats, which are aberrant hosts, because it never encapsulates and continues to migrate through the liver parenchyma and abdominal cavity so that one or two flukes can prove fatal.

Fasciola hepatica is distributed world-wide. Cervid species in which the infection has been reported include moose, sika deer, white-tailed deer, wapiti, black-tailed deer, mule deer, red deer, fallow deer and roe deer. Bison infections have also been reported from North America (111).

There are differing levels of susceptibility to infection with the common liver fluke among deer species, as well as sheep and cattle. *Fasciola hepatica* is said to be almost indiscriminate in its range of mammalian host, and the parasite can complete its life-cycle in several species including donkeys and humans (34). European roe deer are very susceptible to infection with either species of fluke and even a few of them are able to cause severe disease. The red deer is somewhat tolerant of *F. hepatica*, and has been reported to exhibit few signs even when pastured on the same ground as sheep that are dying of fluke-induced disease. Fallow deer are also tolerant of infection with *F. hepatica* and rarely exhibit any clinical signs (5, 34). In experimental infections there are also differences between white-tailed and black-tailed deer. In the former, the migrating larvae are destroyed before they cause any extensive damage. In the latter, an acute fascioliasis associated with the migrating larvae occurs (111).

It has been suggested that because the common liver fluke is often absent in cervids infected with the giant liver fluke or in areas where it occurs in traditional domestic animals, it may be that wild cervids are not significant reservoirs of *F. hepatica*. Most reports are of incidental cases, although prevalence and intensity appear to be higher in Europe than in North America (111).

Clinical signs

There is little other information on the effects of giant liver fluke infection upon farm productivity, but it is likely that both fluke species can cause subclinical disease, and anemia has been associated with the early prepatent period of giant liver fluke infection in white-tailed deer. Similarly, cattle infected with the giant liver fluke are 'poor doers' and fail to gain weight as expected (115).

No clinical signs associated with liver flukes have been reported in bison.

Pathology

The lesions caused by *F. magna* vary according to the type of infected host, as well as the level of infection. The primary lesions are principally found in the liver, and are either associated with mechanical damage caused by the migrating immature flukes or fibrous encapsulation of adult flukes (111). In definitive hosts, the fibrous reaction around the flukes may cause distortion of the liver shape (110). The surface of the liver is often pitted and contains fibrous scars. In many cases, no distortion is evident, but when infection is heavy, the liver may take on a bizarre form, and there may be adhesions to adjacent tissues. In heavy infections, adhesions may also be seen in the pleural cavity. Disruption of liver parenchyma, along with increased fibrous tissue, results in blockage of normal drainage patterns within the liver. Large pockets of blood and parasite eggs, seen as viscid black fluids, are formed in most infected livers. Infected livers are larger than normal. Adult flukes, as much as 80 × 35 mm in size, are often found encapsulated within these pockets which, in wapiti and caribou, have walls from 2-5 mm in thickness. Immature *F. magna* continue to migrate within the liver and thus are not encapsulated. They are most often found within the tracks of fresh haemorrhage and

tissue damage and can be persuaded to leave the liver tissue by soaking sections cut at 1 cm intervals in warm water. Mature flukes are readily visible. The pathological picture varies considerably according to the host species and its reaction to the flukes.

In aberrant hosts, the lesions are associated with the wandering of immature flukes, principally in the liver, although other abdominal and pleural organs have been affected. Haemorrhage, trauma, necrosis and peritonitis have all been observed (111).

Liver damage is rarely severe in red deer infected with *F. hepatica*. Red deer are easily infected experimentally, but development of the adult flukes is somewhat arrested. In contrast to roe deer, in which virtually all the flukes are adult by 100 days post infection, one third of the flukes in red deer are still immature in the liver parenchyma at this stage. The flukes do not cause the severe fibrous reaction seen in sheep, and the very heavy burdens seen in sheep and cattle are not seen in red deer. The main lesion is portal cirrhosis (5, 34, 115).

In a single instance reported from Wyoming, *F. hepatica* were found in many large liver abscesses and a partially abscessed bile duct in a bison (10).

Diagnosis

In the living animal, the most reliable diagnostic tool for diagnosis of mature flukes is examination of faeces for the presence of the characteristic operculate eggs. A reliable ELISA has been developed for the diagnosis of *F. hepatica* in sheep and cattle (5, 34, 115). In neither case is the test able to detect early infection; in the former because the flukes are immature and migrating, in the latter, because the rise in antibody titre cannot be diagnosed until 6 to 8 weeks post infection. The prepatent period of *F. magna* may be as long as 30 to 32 weeks, and eggs would not be detected in faeces of infected animals before this time. Special flotation or sedimentation techniques are required to detect the eggs, which will not usually be found upon routine faecal flotation. Giant liver fluke infections do not usually become patent in cattle, bison and moose.

Treatment

Pybus provides a table showing both doses and efficacy of medications used to treat giant liver fluke infections in white-tailed deer, wapiti and red deer (111). In natural infections, albendazole, clorsulon, diamphenethidine, oxyclozanide, rafoxanide and triclabendazole have proven effective at varying dose rates in some studies, while clioxanide, diamphenethidine, hexachlorophene and nitroxynil have been either ineffective or minimally useful.

Triclabendazole has been tested in wapiti infected with *F. magna* and found to be effective against all adult flukes and a high proportion of immatures. It was given as a 24% drench at the rate of 50 mg/kg with a recommended repeat drench after 7 days (112). Similar high efficiency has been reported against *F. magna* in white-tailed deer. Albendazole, given on treated feed for seven days, has also been reported to be 82%-84% effective in white-tailed deer (114). Rafoxanide has been shown to be effective against *F. hepatica* in red and roe deer. When given in medicated feed at 10 mg/kg it eliminated over 90% of adult, and 60% of immature flukes. The recommended programme involves two treatments on consecutive days, and a repeat in 4 weeks (5, 34).

Tightening restrictions on off-label use of drugs may create problems in the treatment of many diseases. One instance of this problem occurs in the USA, where the use of triclabendazole in cervids is forbidden.

Control

Neither parasite can be directly controlled except by treatment of animals with effective anthelmintics. However, there is some possibility of preventing the problem by controlling the snail intermediate hosts. The cheapest and easiest control method is prescribed burning of contaminated wetlands, which will also help to eliminate larval stages on vegetation. However this approach has obvious environmental implications. What is likely to be effective is management of the animals so that they do not come into contact with the snails or their moist environment except during cold parts of the year when water bodies are frozen and transmission is unlikely to occur. Fencing and paddock management, together with appropriate anthelmintic regimens is probably the most practical approach.

In the spring, infected animals that carry adult flukes in their bile ducts can contaminate pastures. If deer or bison were to be treated with flukicides, the treatment should be administered early in the spring, to reduce the number of flukes that infected animals pass out onto pasture during the summer.

The major impact of either parasite upon farmed deer and wapiti is condemnation of livers at the time of slaughter. Heavily infected animals may be unthrifty and experience reduced reproductive success. There is evidence that wapiti dying of redwater caused by *Clostridium haemolyticum* in Oregon and the eastern slopes on the Rocky Mountains in Alberta may have concurrent infections with *Fascioloides*, although no definite link has been established.

Gastrointestinal nematodiasis

Introduction

Most intestinal worm parasites of deer and bison are closely related to those of other ruminants and occupy the same regions of the gastrointestinal tract as their equivalent species (8, 23, 32, 33, 37, 52, 74, 91, 100, 125, 146, 152). The important genera of parasite that have been associated with clinical disease in bison and deer from several countries are members of the family Trichostrongylidae and include *Ostertagia*, *Haemonchus* and *Spiculopteragia*. Most abomasal

parasite species that infect deer will not infect cattle and sheep. It is known that many intestinal nematode parasites of cattle and sheep differ in their infectivity and pathogenicity for these two species, and it would be unreasonable to assume that similar differences do not occur among deer and bison (146, 152). The most important are probably the abomasal worms, four species of which make up 95% of the total count in the UK (32, 152). There are similar intestinal nematodes of deer in Europe, New Zealand and Australia, not all of them of the same species as those found in North America. Free-ranging bison were compared to cattle in one study and found to have a higher prevalence of nematode parasites (146). However, in bison no disease syndrome has been associated with any intestinal parasite other than *Ostertagia*.

Type II ostertagiasis is recognised in bison, white-tailed deer, red deer and wapiti (23, 24, 148, 149, 150). It has been incriminated as a direct cause of death in bison (148) and as a major contributing cause of poor performance and death in wapiti in New Zealand, where a condition named 'elk fading syndrome' is associated with it (25, 151). It has also been recognised in wapiti in North America (53).

These parasites are known to cause disease in much the same way as they do in traditional domesticated livestock. Haemonchosis in white-tailed deer has been associated with weakness, emaciation and anaemia (27). However, infections that do occur are often overshadowed by the presence of the lungworm *Dictyocaulus* spp. (152).

These parasites have a direct life-cycle involving the shedding of eggs in the faeces, their hatching and development through larval stages to an infective larva and the ingestion of larvae while grazing. The larvae either continue to develop in the gut or may have a period of arrested development associated with winter. The spring egg rise known in other domestic stock, associated with this winter inhibition phase, has been observed in deer.

As the larval stages of these parasites require high humidity and warmth in the microclimates in forage to complete their development in optimum time, areas where high faecal egg counts occur, or in which large numbers of adult worms have been found at post-mortem examination tend to be warm, for at least part of the year, and moist. For example, intestinal parasitism is a major management concern in many areas of New Zealand, but much less so in the prairie regions of North America.

Clinical signs

When clinical signs are observed, they are similar to those of parasitic gastroenteritis in other stock. They include weight loss, or failure to thrive, staring coat, soft faeces or frank diarrhoea, and soiled tail and perineum. Type II ostertagiasis has been reported to cause anorexia, weight loss, weakness, dull hair coat, severe diarrhoea, anaemia, hypoproteinemia, lymphocytopenia, neutrophilia and death in bison (148).

Pathology

Pathology is similar in deer to that in other ruminants. Generalised signs of debility or condition loss may be evident and the characteristic 'Morocco leather' appearance of the mucosa has been seen in *Ostertagia* infections (33). Worm counts are usually modest, but figures as high as 90,000 parasites have been reported in individual animals, with fourth stage larvae being approximately 80% of the total (97).

Diagnosis

Attempts to correlate nematode egg numbers in faecal samples with numbers of adult worms in the gut have been frustrated by generally low numbers of eggs, as well as low numbers of parasites and the fact that in a high proportion of cases no eggs were found even when adult worms were found at post-mortem examination. The interpretation of faecal egg counts, and their relevance as indicators of potential clinical disease have not been adequately evaluated in deer species (152).

Although faecal egg counts should be interpreted with some caution, they may serve as indicators of worm burdens. Loss of condition and other clinical signs, combined with positive faecal samples, may be taken as positive in the absence of evidence of other specific conditions. Plasma pepsinogen measurement has been used to confirm abomasal parasitism but may not be reliable in red deer or wapiti (69, 162). The heavy winter coat of wapiti and red deer can readily mask the fact that they are in poor body condition, and suffering severe weight loss.

Treatment

Most modern anthelmintics appear to be effective in the treatment of gastrointestinal nematodiasis in both bison and deer. There are concerns about drug licensing that must be met in different countries. In some countries, no products are licensed, while in New Zealand, which has the most advanced deer farming industry in the world, 31 different anthelmintic formulations have been licensed specifically for deer.

There are some differences in response to anthelmintic treatment, at least among wapiti and red deer. Wapiti require more frequent treatment and respond differently to the use of benzimidazoles than do the con-specific red deer.

Some of the different products have been compared for efficacy. For instance in red deer, it has been found that pour-on moxidectin at 500 µg/kg and ivermectin injection formulations at 400 µg/kg have similar effectiveness (>99%) against abomasal parasites. However, ivermectin used at the standard cattle dose of 200 µg/kg has only an 84% effectiveness against hypobiotic abomasal larvae (85). Duration of effectiveness has also been compared among products. Moxidectin pour-on has been shown to persist from 5 to 6 weeks, where pour-on ivermectin persisted for only 3 to 4 weeks (87).

Anthelmintics have been much less studied in bison. However, ivermectin pour-on has been shown to be highly effective

against *Ostertagia ostertagi* in a controlled trial. In the same study, the authors considered that the established bovine withdrawal time of 48 days appears adequate to ensure that unacceptable residues do not occur in bison (89).

The potential for treating extensively farmed or ranched stock by adding suitable anthelmintics to formulated rations or drinking water has not been adequately tested. There are commercially available worming pellets, but if they are used great care must be taken to ensure that all of the animals are able to access them. The social order of deer and bison, and the fact that stags may hardly eat at all during the rut, mean that many animals may not get a sufficient dose to be effective. Furthermore, it is virtually impossible to treat young bison during the first summer of their lives, when they may be most susceptible to intestinal parasitism, as handling at that time inevitably leads to trauma in the chute systems. Bison are normally treated in autumn.

Control

Control measures involve good husbandry. Where these parasites have been shown to be a problem, regular faecal sampling and anthelmintic treatment should be combined with pasture management to ensure that the animals have adequate nutrition. Strategic rotation of animals on pasture will also ensure that infective larvae are not available to them (33, 75). The use of pasture containing plants high in dietary tannins has recently been investigated as a means of reducing intestinal worm burdens and shows promise (63).

Lungworms

Introduction

Several species of helminth that infect deer and bison are classified under the general heading of lungworms. They fall into two categories, as follows: those that have a direct life-cycle, principally *Dictyocaulus* spp., and those whose life-cycle involves transmission through a gastropod. The latter are members of the family Protostrongilidae, and include three important members that cause pathological lesions other than in the lungs. These are *Parelahostrongylus tenuis* and different species of the genus *Elaphostrongylus* that affect reindeer and red deer. One lungworm that has been seen in post-mortem examination but not implicated in any disease process is *Muellerius capillaris*.

Dictyocauliasis

Introduction

Lungworm infection caused by *Dictyocaulus* is considered by several authors to be the most important parasitic disease of farmed deer and is also seen in bison (53, 90, 136). It has also been recorded in a wide range of other deer species (3).

The specific taxonomic name of the *Dictyocaulus* affecting various deer species or bison has not been fully established. Cross-infection trials have shown that the cattle parasite can infect red deer and wapiti, and instances of infection of a wapiti and red deer calves with the cattle strain has been reported (26, 109). Cattle should therefore be considered as a potential source for infection of these deer species. In one trial, only a low-grade infection of bovine calves was established with red deer strain lungworm larvae and wapiti strain larvae administered to Holstein calves which did not cause clinical signs or develop patent infections (109). Similarly, cross-infection trials with infective third stage larvae of a *Dictyocaulus* collected from white-tailed deer failed to produce either patent infections or parasites in the lungs of bull calves (6).

PCR evaluation of *Dictyocaulus* isolates from wild deer in Europe has shown that in fallow deer, the parasite is most likely *D. eckerti* (35). Studies involving DNA typing and electron microscopy in New Zealand have shown that the lungworms of cattle and red deer are different and that *D. eckerti* is the parasite of red deer (68).

Epizootiology

Young animals of 3 to 5 months of age are the most susceptible to infection. Adult deer are somewhat resistant, and have lower faecal larval counts than calves, some of which appear to develop some degree of resistance by 5 to 6 months of age, and most of them by 9 months of age. Seasonal variations in larval outputs have been recorded (11).

Dictyocaulus is found world-wide, but is most important in warm moist climates, where microclimatic conditions on pasture are suitable for development of the larvae. The potential for lungworm disease of farmed deer is of major importance in New Zealand and the UK (90). In the dry prairie regions of North America it has not been considered a major problem in deer, but is known to seriously affect wapiti and red deer in North America wherever or whenever the climate is suitable (11, 38, 53). *Dictyocaulus* spp. have been isolated from bison in several American states and Canadian provinces (8).

Adult worms live in the bronchial tree and lay their eggs there. The eggs either hatch in the bronchi, or in the gastrointestinal tract after they have been coughed up and swallowed. Only first-stage larvae are passed in the faeces. If the right climatic conditions (18°C-21°C) prevail, the larvae may go through their molts to the infective third stage in 3 to 7 days. There is a prepatent period of about 20 days in deer (90). Cold weather arrests their development, but they can overwinter in cold climates, and can withstand a temperature of 4.5°C for a year.

The infective larvae are quite inactive and studies of them in cattle faeces show that they remain within 5 cm of the pad. Fungi (*Pilobolus* spp.) in the faeces have been shown to be important disseminators of the larvae as the exploding sporangium may throw them as much as 3 metres. These fungi have been found in the faeces of wapiti in Yellowstone National Park in association with a high prevalence of lungworm infection. The fungi may also serve to protect the larvae from desiccation (39).

To infect the final host, the third-stage larvae must be swallowed. They penetrate the intestinal wall, then migrate to the mesenteric lymph nodes where they undergo a moult to fourth-stage larvae. They travel via the lymphatic drainage and pulmonary artery to the lungs, where they migrate from the capillaries into the alveoli and thence to the bronchi. In red deer, the prepatent period is approximately 24 days (86). There appears to be no information on the duration of patency in red deer, but continued shedding of larvae has been recorded for at least ten months. Low-grade infections in overwintered animals have the potential to be important in the dissemination of the parasite to susceptible calves in the following summer. Increasing burdens of parasites can readily build up in these animals in late summer and autumn as the parasites cycle through the deer. Calves over two months old shed more larvae than their dams and thus become the major source of infection.

Clinical signs

Clinical signs in deer include decreased appetite, gradual loss of weight and retarded growth rate, roughened coat and unexpected deaths in a herd that may continue over several weeks (96). The hacking cough, or the acute syndrome seen in cattle is not a feature of the disease in deer species, although a soft bronchial cough has been noted (53).

The clinical signs associated with lungworm in bison include increased respiratory rate, cough, slight nasal discharge, increased heart rate and mild fever (8). These clinical signs are difficult to recognise in bison on pasture and often the first indication of lungworm infection is dead bison (8). The disease is most commonly seen in late summer (8).

Pathology

In some deer with heavy infections, hyperaemia, failure of the lungs to collapse upon opening and dark red areas of consolidation may be seen. In deer, the principal gross pathological signs are seen in the lumen of the bronchial tree. Adult worms, which are often coated with a foamy mucoid exudate, may completely occlude the bronchi or bronchioles. Post-mortem findings in bison include pulmonary oedema, emphysema and large quantities of bloody froth in the trachea and bronchi containing adult lungworms (8). As lungworm infection is often associated with loss of condition, other parasite infections, particularly of the gastrointestinal tract, may be evident, as well as other general signs of debility. A few adult lungworms may be seen as an incidental finding in bison and deer dying of other causes.

The important difference between cattle and deer species infected with *Dictyocaulus* lungworms is that deer do not develop the severe lesions of alveolar epithelialisation seen in cattle (96). Hyaline membrane formation and interstitial emphysema have not been seen in deer in either experimental or natural infections (96). In natural infections, most of the reaction is seen close to the airways. In mild cases, some degree of eosinophil infiltration of the walls of the bronchi and bronchioles may be seen, and lymphoid follicles may develop adjacent to the bronchioles. More severe responses show irregular ulceration of bronchial and bronchiolar epithelium with eosinophils being evident in the lumen, lamina propria, to some extent in the epithelium, the alveoli adjacent to the airways and the inter-lobular septa. Lymphoid follicles are also seen near the airways (96). The histologial character of the lesions in bison has not been described.

Diagnosis

Diagnosis in the live animal depends upon examination of faecal samples using the Baermann technique and the finding of larvae (26). More specific (and expensive) diagnosis of the actual species of *Dictyocaulus* may not be warranted for routine herd management purposes. Bronchiolar-alveolar lavage techniques may also be used to isolate larvae from lungs and trachea. The larvae must be differentiated from other larvae that may be found in faeces, particularly those of *P. tenuis*.

Treatment

Several anthelmintics have been tested for efficacy against *Dictyocaulus* (91). It has been shown that diethylcarbamazine and levamisole are not as effective in deer as they are in cattle, but several other products including avermectins and benzimidazoles have been shown to be effective in both deer and bison, although none are licensed anywhere for use in the latter (8, 53, 84, 86, 92).

Treatment of bison during the summer months, when calves are at greatest risk of infection, is a challenge, because any attempt to handle them for direct anthelmintic administration will almost certainly lead to significant losses among the calves due to trampling, horning and/or capture myopathy. Other management options for prevention, such as pasture management and grazing rotation should be explored.

If treatment of bison is needed it should be instituted in late spring, just before the herd goes out to pasture, in order to reduce the worm load in the adult bison and so result in a reduction in the number of eggs that are deposited on the pasture over the course of the summer. However, pregnant cows may be difficult to handle during the spring. If lungworm is suspected, oral fenbendazole can be provided in the feed, or in the salt (8).

Control

The principal method practised to control this parasite is a combination of good stockmanship and the strategic use of anthelmintics (26, 37, 53, 90, 96). If there is sufficient land available, it may be possible to rotate animals through paddocks at intervals great enough to ensure that any larvae that have been passed onto pasture are no longer infective by the time the animals return. This strategy is employed in some extensively managed bison herds. Routine monitoring of faecal samples by the Baermann technique should be carried out in early, middle and late summer, especially where the climate is moist.

Trials with the bovine irradiated lungworm vaccine were carried out in a small number of red deer and showed that vaccination had a limited positive effect upon feed intake and weight change, but the use of this product has not become established (26).

Extra-pulmonary lungworms

These parasites are all members of the family Protostrongylidae and are important in farmed cervids because they not only cause clinical disease in one or more species, but they may infect some species without causing any sign of disease and have the potential to be translocated to new geographic regions and establish new foci of infection, potentially even in free-ranging wildlife. They have not been associated with any disease in bison, nor have bison been reported to be suitable hosts for transmission of the parasites. A detailed and extensive review of these parasites has been published by Lankester (77) and only a brief summary is offered here.

There are three species in two genera that are of economic and health significance for domestic and semi-domesticated cervids. These are *Parelaphostrongylus tenuis*, also known as meningeal worm, *Elaphostrongylus rangiferi*, known among other names as muscle worm, and *Elaphostrongylus cervi*, commonly known as tissue worm. The principal wild hosts of these three parasites are white-tailed deer, reindeer or caribou and red deer, respectively.

Parelaphostrongylus tenuis is a parasite of white-tailed deer and other species that reside in the eastern half of North America. A detailed distribution map is provided by Lankester (77). *Elaphostrongylus rangiferi* occurs throughout the range of reindeer in northern Fennoscandia and Russia above 62°N. In North America, its range is restricted to the island of Newfoundland where it arrived with reindeer imported from Norway in 1908, and has become established in the native caribou population. *Elaphostrongylus cervi* occurs naturally in many parts of Europe, ranging from Great Britain through to Fennoscandia into Russia and the Far East. It was introduced into New Zealand with red deer importations around 1900, but incursions into Australia and Canada are believed to have been prevented at the point of quarantine (77).

The members of this family all have indirect life-cycles that involve gastropod intermediate hosts. These differ in detail according to the species of the parasite, but the fundamental cycle involves the presence of adult worms in the tissue of the cervid host. *Parelaphostrongylus tenuis* adults are found in the meninges of the white-tailed deer, while adults of the two members of the genus *Elaphostrongylus* reside principally in the skeletal muscles of the shoulders, chest and abdomen of their host, as well as the arachnoid and subdural spaces of the central nervous system.

Gravid female worms are believed to lay their eggs into the venous system, and the eggs are carried to the lungs where they complete development. The first-stage larvae (L_1s) hatch, invade the air spaces, ascend the tracheal mucociliary escalator and are swallowed and passed out in the faeces.

The L_1s are ingested by, or enter the foot of, one of several species of gastropod, which may vary according to geographic locale. Further development of the larvae is temperature-dependent and may be delayed if the gastropod estivates in arid conditions, or at the approach of winter. In the gastropod intermediate host, the L_1s go through two moults to second- and third-stage larvae (L_2 and L_3 respectively) at which point they are infective if the gastropod is ingested by the mammalian definitive host.

In the mammal, the L_3s leave the gastropod and penetrate the gastrointestinal wall, finding their way to the spinal cord. In the case of *P. tenuis*, this takes about 10 days, and is a direct migration. The route taken by *Elaphostrongylus* is debated. The L_3s undergo two moults in the spinal cord and make their way to the central nervous system (CNS) from where they either move on to the muscular tissues, or remain in the CNS.

Other hosts

All three of these parasites can infect species other than those already mentioned. If a non-traditional host ingests an infected gastropod the nematode larva can undergo development and often cause pathological changes.

In addition to the white-tailed deer, *P. tenuis* can infect several other species that share range with the deer, or live in close proximity with them in zoos or game farms. They include moose, wapiti, red deer, woodland caribou, mule deer, black-tailed deer, and hybrids of these two with white-tailed deer, fallow deer and a number of non-cervids. The non-cervids include bighorn sheep, domestic sheep and goats, llamoids, domestic cattle and several exotic bovids. The guinea-pig has proven to be a useful experimental model of the infection.

The natural hosts of *E. rangiferi* are reindeer and caribou, but moose are also naturally infected. Fallow deer have been experimentally infected, although the life-cycle was not completed in these animals. Guinea-pigs and some domestic animals living on reindeer range have also been infected.

Other than red deer, *E. cervi* has been reported in sika deer, roe deer and experimentally in mule deer. Other older reports in reindeer and moose need confirmation.

Clinical signs

The clinical signs depend upon which parasite is involved, how many larvae infect the host, where it is located in that host, and most important, which species of mammalian host is involved.

Meningeal worm

In some situations, such as infections in goat kids and fallow deer fawns, experimentally infected subjects have succumbed

to *P. tenuis* with an acute colitis and peritonitis and died within a short space of time (2, 113).

Species that survive this initial effect may develop no clinical signs, as is the case with almost all white-tailed deer, or may develop neurological signs of varying degrees of severity, which may or may not progress to severe ataxia, blindness, collapse and death. There are a few reported cases of circling and loss of motor function in white-tailed deer, but experimental infections may cause only transient lameness or limb weakness, if anything at all. The predominant clinical signs in other species are neurological.

It is considered that in abnormal hosts the parasite takes longer to complete development in the CNS than it does in white-tailed deer, becoming larger and engaging in more coiling behaviour as it does so. It is this fact, together with the possible host reactions to the foreign material that may explain the severity of the infections and the resulting clinical signs in these hosts.

Clinical signs have most often been described in moose, in which the disease is known as moose sickness. Wild moose may progressively show any or all of the following signs: ataxia, torticollis, knuckling, circling, fearlessness, depression, nystagmus, apparent blindness, paresis, inability to stand and weight loss. Similar signs have been noted in wapiti. Although there are no specific reports of clinical signs in reindeer or caribou, there is good evidence that these cervids are particularly susceptible to meningeal worm, which has been implicated in the failure of numerous re-introduction and translocation efforts in areas with white-tailed deer.

The appearance and severity of clinical signs in wapiti and moose is dose-dependent. In an experimental trial, wapiti given 15 L_3s developed no signs, although two animals had two and three adult worms in the cranium. All but two of eight animals given 25 L_3s developed signs (two died), and all of those given 75 L_3s died after developing neurological signs (127). It is evident that some wapiti given larval doses in excess of those likely to be found in nature can survive, although both wild and experimentally infected animals can develop patent infections. Similarly, the effect of *P. tenuis* infection in moose appears to be dose- and age-dependent, and the effects of low doses of larvae may not appear clinically or be lethal (77).

The only report of meningeal worm infections in red deer concerned a captive population of mixed red deer and wapiti in which an overall prevalence between 26.6% and 63.4% over a four-year period was reported (105).

Muscle worm

There are two clinical manifestations of *E. rangiferi* infection in reindeer. The most common is a subacute verminous pneumonia that may lead to inanition and death over winter or in early spring in young animals. Affected animals are weak, dyspneic and sometimes cough. They may also experience delayed antler development. More serious cases in reindeer develop neurological signs similar to those seen in meningeal worm infection in aberrant hosts.

Tissue worm

There appear to be differences in the clinical manifestation of infections with *E. cervi* according to geographic locale. In New Zealand, it is rare to find any clinical evidence of disease, while in Europe, neurological signs are rarely seen and the principal clinical sign is interstitial pneumonia. Further east, differences are reported between species of deer. In Siberian maral deer (*Cervus elaphus maral*), neurological disease is common in heavily infected animals, but may otherwise be subclinical, whereas for sika deer the parasite appears to be less pathogenic, and most adult worms are found in the musculature.

Pathology

Verminous pneumonias can occur with any of the three parasites described. In heavy infections of *P. tenuis* in white-tailed deer there may be lung lesions as eggs and larvae may cause vascular congestion, collapsed alveoli, fibrosis and petechial haemorrhage.

For all three species of parasite discussed here, gross pathological changes in the CNS may range from almost no visible signs to slight oedema and yellowish discolouration to firm adhesions between the dura and the pia over much of the brain surface. The degree of change may depend upon the species of mammal and the parasite. When the cord or brain is sectioned, lesions may be visible to the naked eye as discoloured patches. The thread-like adult worms may be visible in the meninges. Histologically, the lesions in the CNS vary somewhat according to the mammalian species. They range from mild degenerative and inflammatory lesions to meningitis with focal, disseminated areas of lymphocytes, macrophages, eosinophils and some giant cell infiltration (77). Sections of eggs or parasites may be evident in histological preparations.

Discoloration of meat in *E. cervi* and *E. rangiferi* infections may be evident and is of concern to venison producers.

Diagnosis

Diagnosis of any of these three parasitic conditions in the live animal has, until recently, only been based upon clinical signs and the finding of spine-tailed larvae in the faeces. However, there are promising developments in the field of serological and nucleic acid-based detection methods (46, 103, 104). The presence of white-tailed deer in regions of known meningeal worm range may provide strong indicators if susceptible species are showing clinical signs. However, this is not sufficient to provide a definitive diagnosis.

While the characteristic spine-tailed L_1s may be collected from faeces and examined, the appearance of the larvae of specific members of the family cannot be differentiated on morphology alone.

The Baermann funnel technique cannot be relied upon to collect all larvae present in a faecal sample. A more reliable technique using screen envelopes immersed in straight-sided beakers has been developed, but even this will not help when animals are infected with single-sex meningeal worms or when infected animals only shed larvae intermittently (77).

Confirmation of meningeal worm infection can be obtained after larvae collected from faeces of the affected host are cultured in gastropod hosts, passaged through white-tailed deer and, after the appropriate delay, are collected from the meninges when adult male worms can be identified by the appearance of the spicules in their bursae.

Parelaphostrongylus species can be differentiated from one another and from closely related genera by PCR techniques that can be used on L_1s, L_3s and adult worms (46). Progress towards a reliably sensitive and specific serological test for *P. tenuis* in live cervids has been made, but will require rigorous field evaluation before it can be routinely used (77).

Treatment

The only time at which treatment with anthelmintics may be effective in killing these parasites is when any of their life-cycle stages in the mammalian host are outside the CNS, as the blood-brain barrier prevents therapeutic doses from reaching the parasites. However, treatment with a variety of anthelmintics does suppress larval output in all three of the parasites discussed here.

Clinicians have tried a variety of treatments in cases of suspected meningeal worm infection. These have included several different anthelmintics of the avermectin and benzimidazole classes, together with one or more anti-inflammatory drugs, but critical trials are lacking. To be effective in preventing establishment of meningeal worm in the CNS, anthelmintic treatment of white-tailed deer must take place less than 10 days after infection.

Some successes in treatment of *E. rangiferi* and *E. cervi* infections have been reported, using either injectable ivermectin or mebendazole in medicated feed, but even these have met with mixed results which Lankester ascribes to possible variations in time of treatment and whether the worms are in the CNS or not (77).

Control

Control of the spread of infections of these parasites depends on two main factors: prevention of movement of infected animals into areas where suitable intermediate hosts exist and where susceptible mammalian hosts are found, and reliable screening of animals to be moved. Control of gastropod intermediate hosts may assist in these efforts.

In areas where meningeal worm exists in the white-tailed deer population, the only sure way to prevent infection of new hosts, or movement of the parasite to new areas, is to remove juvenile stock that have not yet started to graze. The use of molluscicides, and/or establishment of a *cordon sanitaire* around paddocks may prevent incursion of gastropods across fences. Dry sandy or vegetation-free soil should aid in such efforts. When sensitive serological tests become available they will further assist in control programmes.

Pasture management, with three-year rotations has been recommended for control of *E. rangiferi*. Pasture management has also been reported to be the most effective means of controlling *E. cervi*. The technique involved leaving animals on summer pasture for no more than 30 days, which was aimed at ensuring that completion of development of the parasite larvae to infective L_3s in the gastropod had not occurred before the deer left the paddock, and the non-use of those pastures for at least two years.

Prion diseases

Chronic wasting disease

Introduction

Chronic wasting disease (CWD) is one of a group of fatal mammalian neurodegenerative diseases known as transmissible spongiform encephalopathies (TSEs) that is characterised by accumulations of abnormal proteinaceous material that is a protease resistant (PrP^{res}) form of cellular protein (PrP^{c}) normally produced in the CNS. PrP^{res} is a transmissible agent and catalyses the conversion of PrP^{c} to PrP^{res} in susceptible hosts.

When first reported, CWD had occurred in 53 of 67 captive mule deer in wildlife facilities in Colorado and Wyoming, where the first clinical case occurred in 1967 (155). It has since been reported in mule deer, white-tailed deer (and hybrids thereof) and wapiti. Cases in wild cervids of these species were first reported in 1981 (131) but it is likely that the disease was present before then (93). Of the three species in which the disease has been reported, mule deer appear to be the most susceptible. A recent comprehensive review is available and readers are encouraged to obtain this for a more complete discussion and bibliography (157).

In free-ranging animals, CWD is reported from a few adjacent counties in Colorado and Wyoming, as well as a neighbouring county in Nebraska. Cases have recently been reported from free-ranging mule deer in the Canadian province of Saskatchewan. In captive environments, the disease has been recognised in a few zoological parks in Canada and the USA,

and on game farms in Colorado, Nebraska, Oklahoma, South Dakota and Saskatchewan.

Transmission

The mode of transmission is unknown, but modelling has demonstrated that maternal transmission alone is inadequate to maintain the disease at current levels, and may be relatively rare (93). Horizontal transmission appears to be the most common route, but detailed information is lacking and indirect transmission through environmental contamination may also be important (93). Miller and his group have described the CWD situation in north-eastern Colorado and south-eastern Wyoming as an epizootic with a protracted time scale.

The youngest animal diagnosed with CWD to date has been 17 months of age, but the date of infection was not known, so that a true incubation period in natural infections is undetermined (157). However, under experimental conditions, PrP^{res} has been detected in alimentary lymphoid tissue of mule deer fawns within a few weeks of the oral administration of a brain homogenate prepared from a naturally occurring case of the disease in mule deer (129). Maximum incubation periods are not known.

The role of genetic resistance to CWD has come under investigation. Polymorphisms in the PrP gene are associated with variations in incubation times, clinical course, susceptibility and pathological lesions patterns in TSEs in mice, sheep and humans. In wapiti, individuals homozygous for PrP codon at the methionine/methionine132 were over-represented in CWD cases. Heterozygous methionine/leucine132 animals have also been diagnosed in captive wapiti, but no cases have been detected in leucine/leucine132 individuals. As these make up only a small proportion of the overall population, no firm conclusions can yet be drawn (106).

There is no evidence of natural transmission of CWD to species other than the three cervids already mentioned. In deer-to-cattle transmission trials, ten calves that were inoculated intracerebrally with brain suspension from a naturally infected mule deer had shown no signs of the disease three years after treatment, while three other calves became recumbent between 24 and 27 months post inoculation. Despite the microscopic lesions being subtle in two calves, and absent in the third, all three were positive for PrP^{res} by immunohistochemistry (54).

Clinical signs

Initially, clinical signs may involve subtle changes in behaviour, observable only by persons with whom the animals are familiar, and vice versa. This is because farmed deer may mask clinical signs when approached by unfamiliar people. Signs include hyperexcitability and changes in personality, including changes in response towards handlers. Clinical signs may be more subtle and take longer to develop in wapiti than in mule deer (157). As the disease progresses, the signs become more obvious, the animals lose weight steadily and eventually become markedly emaciated. Polydypsia, polyuria, increased salivation, drooling, inco-ordination, posterior ataxia, and fine head tremors may all be seen. Death is inevitable if the animal is not euthanised on welfare grounds (157).

The course of the disease may last from a few days to as much as a year, with most cases surviving for a few weeks to 3 or 4 months, although Miller has reported acute death in white-tailed deer (cited in 157).

Pathology

Gross pathological findings are non-specific and attributable to emaciation and include the dry rough coat and the loss of body fat in most cases. However, aspiration pneumonia, possibly due to faulty swallowing, is not an uncommon finding in cases of CWD and may cause death before emaciation becomes marked (157). If pneumonia is diagnosed at necropsy, the brain should routinely be examined for CWD. Polydipsic animals may have excessively watery rumen contents, and sand and gravel are often present in the forestomachs. An incidental finding by Williams and Young (156) in two of six affected wapiti was traumatic reticulitis. This is an unusual finding in wapiti, which are normally fastidious eaters, and the possibility exists that loss of brain function prevented them from noticing the foreign material as they picked up their food.

The microscopic lesions that have been described in wapiti and mule deer are typical of the TSEs. Spongiform changes, with varying degrees of severity, are present and are most readily appreciated in the vagal nucleus in the dorsal portion of the medulla oblongata at the obex, and the obex should be routinely sectioned when CWD is a possible diagnosis. Spongiform changes are also seen in the thalamus and cerebellum, but are usually mild in the cerebral cortex, hippocampus and basal ganglia (157).

PrP^{res} plaques can readily be appreciated in hematoxylin-eosin stained brain tissues of CWD-affected white-tailed deer, and some mule deer, but are not obvious in wapiti (157). The plaques in white-tailed deer are often surrounded by vacuoles in the neuropil, which allows easy visualisation. Scrapie-associated fibrils are found in brains and spleen of CWD-affected wapiti and deer (157).

Diagnosis

Clinical signs and gross necropsy findings of pneumonia are not diagnostic, although they may provide useful pointers to a diagnosis of CWD. The histological appearance of brain tissue typical of spongiform encephalopathies increases the likelihood of a positive diagnosis, but for confirmation, one of several immunohistochemical tests that have been developed for CWD is required (157). The demonstration of PrP^{res} in lymphoid tissues, which has been successfully employed in sheep scrapie, has not thus far been achieved in cervids, and the possibility of developing a test for live animals is being investigated (157).

There is no known treatment for CWD, although antibiotic treatment may prolong the lives of CWD-affected animals with pneumonia.

Control

a) Wild populations

Control of CWD in wild populations is a problem, especially in the face of the lack of an adequate understanding of modes of transmission. Long incubation periods, subtle early signs, an exceedingly resistant infectious agent and the lack of reliable ante-mortem diagnostic tests compound the difficulties. Models predict population declines once the disease prevalence exceeds about 5% (93). Further modelling has predicted that for control programmes to succeed, it may be necessary to instigate culling when prevalence rates are low (<0.1%), and that at these levels culling of <20% of infected populations may eliminate CWD (51). However, the authors cautioned that in their model the likelihood of control diminished rapidly as prevalence increased and that sustained efforts over many months would be required to achieve the goal of elimination (51).

b) Captive herds

Control and elimination in farmed and other captive cervids suffer from the same limitations as does control in free-ranging cervids, but the tool of entire herd culling can be employed. In Canada, there is a national control programme regulated by the Canadian Food Inspection Agency (CFIA), and CWD is a reportable disease.

The fundamental components in control programmes are based upon the following principles:

– an animal exposed to infection will develop the disease within 36 months

– clinically affected animals could be infectious as much as 18 months prior to death

– environmental contamination with infective prions by CWD-affected animals is possible where the disease has been established for a considerable period of time

– decontamination is assumed to be possible using the following procedures

– feed and manure is scraped off until undisturbed soil is reached (or deeper if clinically affected animals have spent considerable time at the site)

– the material removed is buried

– new uncontaminated material is added to form a barrier

– all facilities and equipment exposed to clinically affected animals are cleaned of organic material and disinfected by soaking for a least one hour with either sodium hydroxide or sodium hypochlorite

– wild cervids are not permitted on any infected premises; if found, they are destroyed and tested for CWD.

If a positive CWD case is found on premises, the following compulsory actions are taken:

– incineration and/or deep burial of the carcass of the affected animal in an approved site

– quarantine and inventory of all animals on the farm

– depopulation of all cervids in the herd that have had contact with the positive animal and proper disposal in an approved site

– collection of samples from all slaughtered animals and testing for CWD

– clean-up and disinfection of contaminated areas

– evaluation and compensation to the owner

– tracing out, identification, slaughter and testing of all animals that have left the herd in the last three years

– entire herd depopulation of any such trace-out herd where positive animals are found

– monitoring of animals that left any affected herd in the last 36-60 months.

In Canada, there is also a voluntary control programme for producers who wish to have their herds identified as CWD-free. It is a joint programme between the Canadian Cervid Council (CCC), the provincial governments and the CFIA. There is a similar proposal involving a co-operative Federal-State-Private Sector programme being discussed in the USA. Further discussion of control is based on the Canadian programme.

There are six levels proposed in the Canadian programme, from the entry level (level E), to the highest level, 'certified'. Under certain circumstances, an owner may apply for advancement of certification status. A minimum of five years is necessary for an enrolled herd to reach the 'certified' level. Due to the lack of a diagnostic test for the disease on live animals, 'freedom' from CWD is determined on a herd basis by testing the animals that die or are slaughtered, absence of clinical signs and lack of exposure to CWD over a designated period of time.

Any elk or deer owner with an established, negative status herd, is eligible to apply for membership and to do so must contact the CFIA district veterinarian where the herd is located and fulfil requirements related to records, surveillance, facilities and specimen submission. Membership can be revoked for failure to comply.

Procedures that must be followed include identification with a minimum of two devices, detailed record-keeping of all stock, herd monitoring, reporting to the district veterinarian of any

animals that show any one of a number of signs associated with CWD and the submission to an approved laboratory for testing of brains of any animal over 12 months of age that die for any reason.

In order to maintain the membership status, the herd will have to meet stringent requirements for diagnostic specimens from the herd. These include not only the laboratory testing of animals that die, but proof of status of any animals that have left the herd.

Producers who wish to import animals, semen or embryos into a herd, and maintain their certification standard, may only do so if the incoming material is for a herd of the same or higher status.

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Maladies virales, maladies parasitaires et infections à prion affectant les cerfs et les bisons d'élevage

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Résumé

La maladie virale la plus importante affectant les cerfs et les bisons d'élevage est le coryza gangreneux. Les auteurs présentent succinctement les autres herpèsvirus isolés chez ces espèces. Ils décrivent également d'autres virus identifiés chez ces animaux, notamment les adénovirus, les parapoxvirus, les virus de la fièvre aphteuse, de la fièvre catarrhale, de la maladie hémorragique épizootique et de la diarrhée virale, les rotavirus et les coronavirus. Les ectoparasites d'intérêt sanitaire pour ces espèces, dans plusieurs régions du monde, sont les tiques, les poux, les mélophages, les œstridés, les sarcoptoïdés et les fourmis rouges. Les helminthes affectant les cerfs et les bisons d'élevage sont la douve du foie (*Fascioloides et Fasciola*), les nématodes gastro-intestinaux de la famille des Trichostrongylidés, le strongle pulmonaire du genre *Dictyocaulus* et le strongle extrapulmonaire de la famille des Protostrongylidés. La cachexie chronique est surtout répandue en Amérique du Nord, où elle affecte les cervidés sauvages dans une région bien délimitée ; la maladie a été signalée également chez des cervidés domestiques dans quelques États nord-américains ainsi que dans une province du Canada. Les auteurs présentent une synthèse sur chacune de ces maladies : classification, épidémiologie, signes cliniques, pathologie, diagnostic, traitement et prophylaxie.

Mots-clés

Bison – Cachexie chronique – Cerfs – Coryza gangreneux – Diagnostic – Faune sauvage – Maladies infectieuses – Ostertagia – Prophylaxie – Strongle respiratoire.

Enfermedades víricas, parasitarias y priónicas de los ciervos y bisontes de granja

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

Resumen

Tras describir la fiebre catarral maligna, que es la enfermedad vírica más importante que afecta a los ciervos y bisontes de granja, los autores pasan revista someramente a otros herpesvirus y demás agentes víricos detectados en esos animales: adenovirus, parapoxvirus, rotavirus, coronavirus y los agentes etiológicos de la fiebre aftosa, la lengua azul, la enfermedad hemorrágica epizoótica y la diarrea vírica. Entre los principales ectoparásitos que afectan a esos animales en varias zonas del mundo se cuentan diversos tipos de garrapatas, además de piojos, moscas melófagas, éstridos, ácaros de la sarna y hormigas rojas. Entre los parásitos helmínticos destacan los tremátodos hepáticos (*Fascioloides y Fasciola*), los nemátodos gastrointestinales de la familia Trichostrongylidae, los vermes pulmonares del género *Dictyocaulus* y los extrapulmonares de la familia Protostrongylidae. La caquexia crónica es importante sobre todo en Norteamérica, donde afecta a los cérvidos salvajes de un área determinada y se ha descrito en ciervos de granja de unos pocos estados de los Estados Unidos y de una provincia canadiense. Los autores hacen un resumen de esas enfermedades, indicando en cada caso su clasificación, epidemiología, signos clínicos, patología, diagnóstico, tratamiento y control.

Palabras clave

Bisonte – Caquexia crónica – Ciervos – Control – Diagnóstico – Enfermedades infecciosas – Fauna salvaje – Fiebre catarral maligna – Nematodiosis pulmonar – Ostertagia.

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