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Diseases of farmed deer in New Zealand

IN THE wild, deer are regarded as remarkably healthy species with few serious natural diseases (Mackintosh and Beatson, 1985), although contact with domestic animals has resulted in bovine tuberculosis becoming endemic in a few wild populations in New Zealand and this also appears to have occurred in Ireland, Hungary and North America. Most wild deer have internal parasite burdens but these are usually low due to their extensive pattern of grazing and browsing. The New Zealand forest proved an ideal environment for deer, and after their release over 80 years ago populations grew rapidly and from the 1930s the Government was forced to take steps to control their numbers, using professional deer cullers. In the 1960s and 1970s large-scale commercial deer shooting and recovering of venison carcasses for export reached a peak of over 4000 tonnes annually. In the late 1960s wild deer numbers were declining rapidly and the potential for farmed deer was realized. Live deer capture peaked in the late 1970s, then the cost of these operations became uneconomical and the number of farm-bred replacements increased, until today almost all the increase in numbers is from farm-bred stock.

It is estimated that by mid-1989 there were over 400 000 hinds and over 100 000 stags on New Zealand farms. Around 80–85% of these were red deer (*Cervus elaphus*) with fallow (about 8–10%), wapiti (elk), sika and various hybrids making up the other 5–10%. This chapter concentrates largely on the diseases of farmed red deer.

The farming of deer is the most recent example of the domestication of a species by man. This domestication has caused the emergence of diseases which are either uncommon or non-existent in wild deer. Many of these conditions are predisposed to by increased stocking rates, exposure to new pathogens and raised levels of stress. Some conditions, such as yersiniosis and stress-induced myopathy (McAllum, 1985) were more common in newly captured stock, but the incidence rate has declined recently as deer have become better adapted to farm conditions.

The most important diseases of farmed deer in New Zealand are malignant catarrhal fever (MCF), bovine tuberculosis (Tb), yersiniosis, lungworm infection and enzootic ataxia.

MALIGNANT CATARRHAL FEVER (MCF)

The disease in deer is characterized by either acute bloody diarrhoea, dehydration, depression, inappetence and rapid loss of condition leading to death in 1–2 days or, in a small proportion of cases, by a chronic diarrhoea, crusty exudation around eyes,

muzzle, and anus, loss of condition and death in 1–5 weeks (Beatson, 1985a). It usually affects animals six months or older and the highest incidence occurs in winter. It is the most common infectious cause of death in adult deer, affecting 1–2% annually. The infective agent is believed to be a 'sheep associated' herpes virus similar to alcelaphine herpes virus (AHV-1) that naturally infects wildebeest and causes a similar MCF in cattle in Africa (Reid *et al.*, 1986). Deer-to-deer transmission is thought not to occur under natural conditions. Fallow deer appear to be totally resistant to MCF while wapiti and red deer are moderately resistant and sika and Pere David's deer are very susceptible. Once animals show signs of disease there is no effective treatment. It is believed that good nutrition, minimal stress, isolation from sheep and avoidance of feed contamination by sheep will reduce the incidence.

BOVINE TUBERCULOSIS (Tb)

This disease is important in New Zealand, more for its potential to cause serious problems than for the actual incidence of disease, which is less than 1%. Bovine Tb is endemic in wild possums (*Trichurus vulpecula*) and feral deer and cattle in several isolated pockets throughout New Zealand (Anon, 1986) and this has led to its introduction onto deer farms.

The disease varies from longstanding subclinical infection with one or more lesions in lymph nodes to severe clinical cases with generalized Tb sometimes with sinus tracts to the skin surface (Brooks, 1984; Beatson, 1985b). The majority of lesions are found in the head lymph nodes especially the retropharyngeals. Spread within a herd can be very rapid if any deer have sinus tracts or generalized Tb and are shedding large numbers of organisms.

Control is by test and slaughter. There has been a voluntary Tb accreditation scheme operating for the last few years and this year a compulsory scheme is being implemented. Accreditation status is gained by having three clear whole herd tests in a minimum of two years, and is maintained by a clear test every two years. Any deer diagnosed as reactors are slaughtered and the herd retested every six months until clear. The standard skin test (ST) involves a single intradermal injection of bovine purified protein derivative (PPD) in the centre of a closely clipped skin patch on the side of the neck (Carter, Corrin and De Lisle, 1984). Retests may be conducted in defined circumstances using a comparative cervical test (CCT) (Carter *et al.*, 1985) with avian and bovine PPD or a blood test (BTB) which relies on a lymphocyte transformation test (Griffin and Cross, 1987). The ST, CCT and BTB have estimated sensitivities and specificities as shown in Table 1.

Table 1 SENSITIVITY AND SPECIFICITY OF TESTS FOR Tb

	Sensitivity	*Specificity*	*Comment*
ST	≈ 85%	< 90%	Cheap, quick screening test
CCT	≈ 75%	> 90%	More expensive, slower retest
BTB	> 95%	> 95%	Expensive, but very accurate retest

YERSINIOSIS

This is one of the most common causes of death in young deer in their first winter and is caused by *Yersinia pseudotuberculosis* (*Y. pstb*) serotypes I, II and III. It is characterized by watery, smelly diarrhoea which quickly becomes blood-stained and affected calves may die in 1–2 days if left untreated. The majority of young deer appear to experience a subclinical infection and develop natural immunity to subsequent infection. However, if the calves are stressed while infected with *Y. pstb*, they may succumb to an acute fulminating enteritis (Mackintosh and Henderson, 1984).

Treatment with long-acting tetracyclines or trimethoprim/sulphonamide mixtures plus fluids and gut protectives is usually effective if given early enough in the disease. Prevention is by minimizing stress, feeding well and providing adequate shelter. In an outbreak, mass injection with long-acting tetracylines is effective. A vaccine is currently being investigated at Invermay.

LUNGWORM INFESTATION

Verminous pneumonia caused many deaths in the early days of deer farming but now the routine use of effective anthelmintics has eliminated most clinical disease. The parasite involved, *Dictyocaulus viviparus*, appears to be very similar to the cattle strain and has a prepatent period in red deer of around 21 days. Burdens start developing in late summer and warm moist autumn conditions quickly result in heavy burdens if young deer are not treated. New generation benzimidozoles at three-weekly intervals, or ivermectin at five-weekly intervals, should be given for optimal control in young deer from late summer to early winter (Mackintosh *et al.*, 1984). Lungworms are usually not a clinical problem in adult deer (Mason, 1985; Watson and Charleston, 1985). However, pre-calving treatment of hinds will reduce contamination of paddocks on which the next crop of calves will be grazed. Alternatively, faecal samples may be monitored and treatment instituted if necessary.

ENZOOTIC ATAXIA

This condition occurs in many areas of New Zealand where the pastures are copper deficient and/or high in molybdenum and sulphur. It is analogous to 'swayback' in lambs, but because deer are born with moderate levels of copper stored in their liver (unlike lambs) the condition does not usually develop until the animals are yearlings or older. Pasture copper levels tend to peak in summer and autumn during which time liver levels usually rise. However, low pasture copper levels in winter and spring result in a decline in liver levels and, especially in times of feed shortage in dry seasons, clinical cases of enzootic ataxia may develop in late spring and early summer. Liver copper levels of less than 100 μmol/kg (wet weight) and serum copper levels of less than 9 μmol/l indicate deficiency. Liver copper levels below 60 μmol/kg are likely to result in enzootic ataxia in some deer (Mackintosh *et al.*, 1986). Clinically, the first sign is a slight unsteadiness in the hind legs, especially at a fast walk. This progresses to marked ataxia and eventually loss of strength in the hind limbs so that the animal assumes a 'dog-sitting' posture (Wilson, Orr and Key,

1979). Once the clinical signs develop, treatment will only arrest the condition and not reverse it. North American elk or wapiti appear to be particularly susceptible to copper deficiency (Mackintosh, Orr and Turner, 1986). Other clinical signs attributed to copper deficiency include poor growth rates, pale coat colour, swollen limbs, joints and poor or misshapen antlers. Supplementation may take the form of copper top-dressing of pastures, oral dosing with copper needles or injection with copper compounds, although the latter are resented by deer and often lead to abscess formation.

OTHER DISEASES

Other diseases which occur less commonly include necrobacillosis (*Sphaerophorus necrophorus*) causing foot, liver and lung abscesses, leptospirosis, tick infestation (*Haemaphysalis longicornis*), Johnes disease, avian tuberculosis, perennial ryegrass staggers, parapox virus disease, facial eczema, liver fluke, cryptosporidiosis and clostridial disease. Wapiti have a relatively high incidence of a wasting syndrome which may be related to one or a number of factors which include ostertagiosis, copper deficiency, mycotoxicosis and unknown feed factors. It does not appear to be related to the wasting in wapiti in North America caused by spongiform encephalopathy.

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