Importance of infectious diseases of New Zealand farmed deer

The following paper was presented by the author at a recent meeting of veterinarians in Saskatoon, Saskatchewan, Canada.

Infectious diseases may be considered important for varied reasons. To a farmer a disease is important if it causes losses through stock deaths or condemned carcasses at slaughter, or if the costs of diagnosis, treatment or prevention become significant. To the farmer's neighbours or the district, an outbreak of infectious disease becomes important when movement restrictions, or changes in the ability to sell stock, follow.

To the nation as a whole a disease assumes importance when there are costs stemming from losses of export markets, from associated surveillance and diagnostic services and directly from control or eradication schemes.

To the general public a disease assumes importance if there are zoonotic implications, or if animal welfare is compromised.

The objective of this paper is to relate these aspects of infectious diseases to the deer industry and to describe the most important diseases of deer in New Zealand.

Deer farming in New Zealand

At the time of writing, deer prices had dropped to their lowest level since deer farming started over 20 years ago. Mixed aged hinds were selling for $180-300, weaner hinds for $50-150, weaner stags for $100-170 with venison stags realising $200 (50 kg) to $320 (90 kg). These changes in values led to more emphasis being placed on farming deer for venison and velvet than for live sales, and a significant proportion of female weaners are now destined for venison production as 1 to 2-year-olds. The herd is more important than the individual and there is now more culling for temperament, infertility, conformation and old age. In other words, deer farming is settling down as a normal primary production-related system.

As profit margins are squeezed, disease prevention and planned animal health programs become even more important. The true cost of infectious diseases is difficult to assess accurately. Annual mortality due to malignant catarrhal fever (MCF), tuberculosis, yersiniosis, leptospirosis, necrobacillosis, and parasitism probably account for 3 or 4% of the population. However, there are also the costs associated with veterinary treatment of affected animals, necropsies, diagnostic services, reduced growth rates, abortions and reduced fertility in survivors. The costs of anthelmintics, vaccines and tuberculosis testing are also considerable but are economically beneficial in well planned disease prevention programs.

Important diseases

The three most important infectious diseases of farmed deer in New Zealand are MCF, tuberculosis and yersiniosis. This article will deal with these three diseases in some depth then discuss leptospirosis, necrobacillosis, and parapox infection more briefly. It is of note that five of these six diseases are zoonoses (tuberculosis, yersiniosis, leptospirosis, parapox and salmonellosis).

Malignant catarrhal fever: This is still probably the single greatest infectious cause of mortality in adult deer in New Zealand.[1] There are species differences in susceptibility, with Pere David's, white-tailed, rusa, sika and sambar deer the most susceptible (attack rates of five to 50% per annum) while red and wapiti deer are moderately susceptible (about 1% per annum) and fallow deer appear totally resistant.

The causative agent is believed to be a sheep-associated herpes virus similar to the alcelaphine herpes virus carried by wildebeest which causes bovine malignant catarrh in Africa. This sheep-associated virus has not been isolated but viral DNA has been detected in infected cells from 'normal' sheep and red deer and cattle affected with MCF. This viral DNA shares considerable homology with the alcelaphine herpes virus.[2]

Deer and cattle appear to be dead-end hosts and are not likely to pass on infection under normal conditions. The virus is strongly cell-associated and can be experimentally transmitted by injecting whole blood or homogenates of spleen or lymph node material from infected deer, cattle or laboratory rabbits.

It appears that most sheep are infected at or around birth and remain carriers for life. It is thought that the virus is shed mainly by adult ewes in late pregnancy or around the time of parturition.

The majority of clinical cases of MCF occur in winter and early spring.[3] The disease appears to be precipitated by stress and the incubation period is probably from 3 weeks to 3 months or more.

Clinically, the disease usually presents as a dramatic watery, blood stained diarrhoea with acute onset. The animal is fevered, depressed, rapidly becomes dehydrated, has purplish injected mucous membranes, usually has some buccal ulceration and dies in 24 to 48 hours. Often the farmer will simply find a dead animal with blood-stained diarrhoea around the anus. Fewer than 25% of cases survive the acute phase and develop chronic disease characterised by gradual weight loss, crustiness around mouth and anus and occasionally haemorrhages in the anterior chamber of the eye. They may survive up to 28 days. Occasionally the acute cases will show severe neurological signs and will become ataxic, progressing to recumbency and paddling.

Post-mortem findings in acute cases usually include dramatic haemorrhagic gastroenteritis, swollen lymph nodes, haemorrhagic oedematous lungs, excess pericardial fluid and pericardial haemorrhage. There is often 'tiger-striping' in the colon and erosions in the mouth, oesophagus and abomasum. In chronic cases there are often a few large haemorrhages and characteristic enlarged tortuous blood vessels in the intestinal mesentery and in the kidney due to chronic arteritis and vasculitis.

The diagnosis of MCF is based on the histological demonstration of vasculitis. This is usually most dramatic in the brain, especially the cerebellum, where the media and perivascular space of medium sized arterioles show acute necrotising change or are infiltrated by mononuclear lymphoid cells. Similar lesions are usually also found in the lung, liver, kidney and lymph nodes.[2]

There is no treatment which has proven effective. Sometimes treatment will result in an acute case becoming a chronic case with inevitable death in 2 to 4 weeks. There is a saying in New Zealand: 'If the animal gets better then it didn't have MCF.'

Prevention is by excluding or minimising sheep contact and by adequately feeding and sheltering the animals in winter and minimising stress.

Tuberculosis

As deer farming expanded rapidly, and the demand for deer increased, many deer were moved extensively throughout the country. Many wild deer were captured from areas now known to have endemic tuberculosis infection in other wild or feral animals, and an appreciable number of infected deer were introduced into the farmed population in this manner. Deer farmed in these endemically-infected areas are also exposed on occasions to infection principally from tuberculous possums. A voluntary tuberculosis control scheme was introduced in 1985[4], and a compulsory scheme came into force in January 1990.[5]

Despite all of this, tuberculosis in farmed deer is not a big problem, with an annual incidence of 0.33% in 1991.[6] Over half the herds in the country are now accredited tuberculosis free. This situation is due largely to the combined efforts of the Deer Farmers Association, Ministry of Agriculture and Fisheries and the Deer Branch of the New Zealand Veterinary Association, in jointly developing and implementing the compulsory tuberculosis control scheme.

Tuberculosis is taken seriously by deer farmers. There is no compensation paid by the state for reactors, and all the costs of testing are carried by the farmer. Some seriously affected herds have had to be

depopulated as the only economic means of solving the problem. However, most infected herds have overcome the problem in 1 to 2 years by skin testing, and with strategic use of the lymphocyte transformation test. Proximity to endemic areas also increases the frequency of testing, and can preclude farms from exporting deer to some countries.

Tuberculosis needs to be put into perspective. It is potentially very damaging if introduced and allowed to go unchecked for a few years. Therefore, it is essential to prevent its introduction and to test regularly in order to detect any breakdown.

Tuberculosis in deer can be manifest clinically as emaciation, cough, subcutaneous abscessation or simply as sudden death in an apparently normal animal.[7] Grossly lesions can be quite varied in appearance, but the tendency is towards an abscess rather than the granuloma seen typically in cattle. These can vary in size from 1 mm to 6 cm and may replace an entire lymph node. Lesions are most often seen in the head lymph nodes with the medial retropharyngeal node being the most common site. Histological diagnosis of the abscess relies on the presence of giant cells in the thin outer inflammatory zone and the presence of acid fast organisms in varying numbers.[8]

Colin Mackintosh (centre) with **Surveillance** *editor Stuart MacDiarmid (left) and MAF Quality Management epidemiologist Terry Ryan (right).*

Yersiniosis

Yersiniosis is one of the most common causes of death in young farmed red deer in New Zealand[1] and has also been reported to occur in deer in Australia, Canada and the United Kingdom. The disease is caused by the bacterium *Yersinia pseudotuberculosis* serotypes I, II and III. A related organism, *Y. enterocolitica*, may occasionally cause disease but is usually part of the normal intestinal flora. Clinically, yersiniosis is characterised by a green or brown watery diarrhoea which often contains blood, and staining may be seen around the anus, on the tail, perineum or hocks but is often not very obvious. Affected animals have elevated temperatures (greater than 40 °C) and soon become dehydrated. The first cases are often found dead and close inspection of the in-contact animals reveals animals with diarrhoea. Outbreaks of the disease can affect up to 40% of a group, although usually 5 to 20% are affected.

Yersiniosis is primarily a disease of young red deer calves in their first winter when they are 5 to 9 months old, with a peak of clinical cases in June, July, and August. Sporadic cases occur throughout the year, but these usually involve recently captured or debilitated animals.

It is believed that virtually all red deer are exposed to *Y. pseudotuberculosis* in the farming environment but, if they are fed and managed well, the majority experience a subclinical infection only.

The development of clinical disease in young deer is precipitated by various stresses. These may include underfeeding, transportation, exposure to adverse weather, lack of shelter, rapid changes in diet, high stocking densities, social stresses, and capture from the wild. It is believed that these stresses may affect the localization and multiplication of *Y. pseudotuberculosis* organisms in the gut and may suppress humoral and cell-mediated immunity.

Y. pseudotuberculosis organisms can survive for long periods in the environment in cold, wet winter conditions, whereas they do not persist in hot, dry environments. This property of cold tolerance is utilized in the laboratory to enhance the recovery of organisms from samples which are sub-cultured into media and kept at 4°C for up to 3 weeks, during which time *Y. pseudotuberculosis* multiplies and most other bacteria remain dormant or die.

Enteric yersiniosis causes profound damage to the gastrointestinal tract. Usually the ileum, caecum and colon are most severely affected, and occasionally the changes extend to the upper small intestine and abomasum. On necropsy, there is usually severe reddening of the intestinal mucosa with blood-stained contents in the lumen, and sometimes focal ulceration with pseudomembranes. Mesenteric lymph nodes are often swollen and oedematous. The abomasal wall may be reddened and there may be petechial haemorrhages. Histopathological changes in the intestine can include acute enteritis with patchy ulceration and oedema of the mucosa and submucosa. This may be accompanied by mesenteric lymphadenitis with focal necrosis. This profound damage to the mucosa causes dehydration and protein loss and allows *Y. pseudotuberculosis* organisms to invade the body via the lymphatic system.

Treatment with parenteral antibiotics, proprietary scour medicines, and fluid replacement therapy for 3 or 4 days is usually successful if instituted early in the disease. The antibiotics of choice are oxytetracycline or trimethoprim and sulphonamide combinations to which the majority of isolates are sensitive. To curtail an outbreak, in-contact deer may be treated prophylactically.

Good management practices, adequate feeding, provision of shelter from bad weather and minimising stress will help to prevent yersiniosis. The stress of weaning can be reduced by weaning prior to the rut when feed is more plentiful, environmental temperatures are higher and severe storms less likely. Once weaned, the calves can be brought into the yards regularly for anthelmintic dosing in autumn and given preferential grazing over the winter. Indoor wintering of weaners is proving popular in the colder areas in the south of New Zealand and can reduce the likelihood of yersiniosis if the level of feeding is adequate and if the accommodation is warm and dry.[9]

Invermay Agricultural Research Centre has, for a number of years, been investigating the immune response of deer to *Y. pseudotuberculosis* infection and killed bacterins. A killed vaccine has undergone two years of experimental challenge trials and has given significant protection. In 1992 the vaccine underwent field trials involving 5,000 calves on farms throughout New Zealand and on the basis of good results of the trial the vaccine is now marketed commercially under the name *Yersiniavax*.

Leptospirosis

Over the last decade there have been several reports of outbreaks of haematuria and deaths in farmed red and fallow weaners caused by *Leptospira pomona*. This has been especially so in dairy farming areas. In the autumn of 1989 six outbreaks in weaners were reported in *Surveillance*.[10] One outbreak involved 1,000 yearling red deer in the early winter.[11] After yersiniosis, leptospirosis is regarded as the most important disease of weaned red deer in the central and

northern North Island. Morbidity and mortality rates have reached 50% and 10% respectively. The maintenance host for *L. pomona* in New Zealand is the pig, but cattle may remain carriers and spread infection for some time, probably up to a year. One young red deer has been shown to be leptospiruric for at least 8 months.[12] *L. hardjo* (cattle host) and *L. balcanica* (possum host) have also been isolated from deer but no clinical disease has been associated with these infections. *L. copenhageni* (rat host) has been implicated in some deaths in weaners on a few occasions. Serological evidence of *L. ballum* (mouse host) and *L. tarassovi* (pig host) infections has also been recorded but has not been associated with disease.

Clinically, *L. pomona* infections usually present as haematuria, weakness and sudden death. Necropsy examination usually shows signs of an acute haemolytic episode with severe nephritis and dark red urine, although sometimes only acute nephritis is found without signs of haemolytic disease. Occasionally hepatopathy and icterus are found. Silver stains may reveal leptospires on histological examination. *L. pomona* is relatively easy to culture from kidneys and urine.

The drug of choice for treatment is streptomycin (25 mg/kg daily for 3 days). This usually stops the leptospiruria. Tetracyclines are useful clinically but may not prevent leptospiruria. Blood transfusions from adult deer may be worthwhile, together with fluid replacement and anti-inflammatory therapy.

Annual vaccination of hinds is recommended for herds at risk. This will provide passive immunity until the weaners can be vaccinated in the autumn.

Necrobacillosis

Fusobacterium necrophorum causes foot abscesses, necrotic stomatitis, navel ill, liver abscesses and lung abscesses in red and fallow deer.[13] These infections are predisposed to by injuries and abrasions to the lower legs, feet and mouth. They occur especially in weaners in autumn and winter. Fallow weaners are particularly susceptible to necrotic stomatitis and the condition often appears to be associated with thistle-infested paddocks. Yarding of weaners and indoor wintering can lead to foot abscesses if injuries occur to the lower legs, especially the skin around the coronary band. Foot abscess infections may spread, resulting in liver and lung abscesses.

Treatment of foot abscesses involves flushing with dilute disinfectant or hydrogen peroxide (*F. necrophorum* is a strict anaerobe) to remove necrotic debris, infusing local antibiotics (especially oily intramammary cerates which appear to diffuse through the wound well) and giving high levels of parenteral antibiotics, especially short-acting penicillin to achieve peak blood levels to penetrate into necrotic areas.

If acute treatment of foot abscess is unsuccessful, chronic infective arthritis results, necessitating amputation of the digit or even the whole lower limb in extreme cases.

Necrotic stomatitis and abscessation of liver and lung often respond to high levels of parenteral antibiotics if treated early enough. Severe cases usually die.

Preventive measures involve minimising any trauma to feet and mouths. Leadin races to yards should be free from damaging projections and stones. Indoor wintering pens should be free of projections and have frequently renewed or freshened deep litter. Weaners and fawning fallow does should be kept away from paddocks with thistles. Some veterinarians and deer farmers have used a killed *F. necrophorum* bacterin and believe it gives significant protection.

Deer parapox

This disease was reported first in a number of places simultaneously in spring of 1985.[14,15] Previously, a few isolated cases of scabby velvet antler in stags were noticed by farmers and practitioners and put down as 'scabby mouth' (orf), 'ringworm' and 'dermatophilus' infections. However, a number of large outbreaks of scabby lesions on the velvet antler, head and/or body of affected deer led to the discovery of a unique parapox virus not previously described.[16] The mystery remains; where did it come from and how long has it been in New Zealand? The parapox virus shares some DNA homology with the sheep parapox virus which causes orf or 'scabby mouth' but is distinctly different.[17]

Outbreaks tend to occur in stags when in velvet antler and to be associated with grazing in paddocks with thistles or prickly hedges. However, cases also occur in hinds and weaners, affecting primarily the head. Most cases occur in late spring and summer. The morbidity rate in an affected group can be very high (approaching 100%) but mortality is very low. The lesions usually heal and the scabs that drop off over 2 or 3 weeks contain large amounts of virus.

Considerable financial loss can result from outbreaks involving velveting stags because the velvet is unsaleable. The virus has been shown to survive the drying process.

Fortunately, there appears to be some degree of immunity and few cases are experienced in years following a large outbreak when the majority of animals are affected. It is thought that the virus is fairly persistent in the environment and it is likely that on affected properties most animals will be mildly affected as young animals.

Other diseases

Sporadic cases of salmonellosis occur in farmed deer but it is not common.

Similarly sporadic cases of 'pulpy kidney', tetanus and blackleg have occurred but vaccination is now quite widespread and losses are minimal.

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