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A Review of Yersiniosis in Farmed Red Deer in New Zealand

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Abstract

Yersiniosis, caused by *Yersinia pseudotuberculosis*, commonly affects young farmed red deer (*Cervus elaphus*) in New Zealand. It is characterised by diarrhea, dehydration and sudden death. Lesions include severe focal enteritis with patchy ulceration and edema of the intestinal mucosa and submucosa and mesenteric lymphadenitis. Treatment with tetracyclines and fluid is usually successful if given early in the disease. Most young farmed deer experience subclinical infection in their first winter; however, concurrent stress may precipitate clinical disease in up to 40% of a group. Stressors may include inclement weather, under-feeding, and transportation. The organism is carried by a wide range of wildlife and domestic animals, and it persists in the environment, especially in cold wet conditions. Good husbandry will help prevent the disease. Natural immunity develops after initial exposure. A vaccine is being developed.

Key words: Epidemiology Yersinia pseudotuberculosis, *New Zealand, red deer, yersiniosis*

Introduction

The first recorded outbreak of yersiniosis in farmed red deer occurred in 1978 (Beatson and Hutton 1981). Since then yersiniosis has become one of the most common causes of death in young farmed red deer in New Zealand (Beatson 1984; Mackintosh and Henderson 1984a, b; Wilson 1984; Mackintosh 1990). The disease is caused by the bacterium *Yersinia pseudotuberculosis* (*Y. pstb*) serotypes I, II, and III. A related organism, *Y. enterocolitica* may occasionally cause this disease, but it is usually part of the normal intestinal flora (Henderson 1983). Clinically yersiniosis is characterized by a green or brown watery diarrhea which often contains blood, and staining may be seen around the anus, or on the tail, perineum, or hocks, but is often not very obvious. Affected animals have elevated temperatures (> 40° C) and they soon become dehydrated. The first cases are often found dead, and close inspection of the in-contact animals reveals animals with diarrhea. 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 (Mackintosh and Henderson 1984a). Sporadic cases occur throughout the year, but these usually involve recently captured or debilitated animals.

Subclinical Infection

It is believed that virtually all red deer are exposed to *Y. pstb* in the farming environment, and if they are fed and managed well the majority experience a subclinical infection. At Invermay, near Dunedin in the South Island, New Zealand, a group of 38 weaned, red deer calves were

nitored from autumn to late spring with fecal id blood samples taken at 3 to 4 weekly tervals. It was found that 11% to 20% of calves lded *Y. pstb* isolates on each sampling occaon, between May and August. A total of 20 es (53%) had positive fecal cultures on at least ne occasion and 37 (97%) seroconverted during is period. None of the animals developed inical signs of disease. A survey of fecal samples om 527 healthy deer calves in the North Island f New Zealand in late June and July yielded a % isolation rate (Hodges et al. 1984b), which is milar to the periodic point prevalences found at ermay (11% to 20%). This contrasts with the % isolation rate obtained from sampling adult s at slaughter (Mackintosh and Henderson 5). These findings, together with the very low ncidence of disease in adult red deer, suggest that oung animals develop resistance to infection bsequent to exposure in their first winter.

athogenesis

The development of clinical disease in young deer precipitated by various stressors. These may nclude underfeeding, transportation, exposure adverse weather, lack of shelter, rapid changes n diet, high stocking densities, social stresses, nd capture from the wild. It is believed that these tressors may affect the localization and multiplication of *Y. pstb* organisms in the gut and may suppress humoral and cell-mediated immunity (Mackintosh and Henderson 1984a; Griffin 989).

otential Reservoirs of Infection

nfected animals shed *Y. pstb* in their feces, and ransmission appears to be via contaminated ood and water. *Y. pstb* nfection is endemic in me species of animals which act as long-term eservoirs and excretors, whereas others are ccidental hosts and short-term excretors. In the orthern hemisphere, rodents, lagomorphs, and arious birds are regarded as the principal reervoirs of infection (Mair 1968, 1973, 1975; bbert 1972; Obwolo 1976; Stovell 1979). Investigations of New Zealand wildlife showed lation rates of 27.8% in feral cats (*Felis omesticus*), 8.6% in Norway rats (*Rattus norvegicus*), 5.5% in mice (*Mus musculus*), 3.8% in hares (*Lepus europeaus*), 1.9% in rabbits (*Oryctolagus cuniculus*), 5.3% in ducks (*Anas platyrhynchos*), 2.3% in sparrows (*Passer domesticus*), 2.3% in seagulls (*Larus dominicanus*), and 1.7% in starlings (*Sturnus vulgaris*) (Mackintosh and Henderson 1984b). It is not known to what extent domestic animals act as reservoirs of *Y. pstb*, but in New Zealand it has also been isolated on many occasions from domestic livestock which have died or shown signs of disease, especially diarrhea (Hodges et al. 1984a). These included isolates from 56 cattle, 8 sheep, 13 goats, 7 pigs, 6 rabbits, 5 guinea pigs, and 22 aviary species of birds, in addition to 117 deer. *Y. pstb* has also been isolated from apparently healthy domestic animals including cattle (Hodges et al. 1984b) and pigs (C.G. Mackintosh, unpublished data).

Y. pstb organisms can survive for long periods in the environment in cold, wet winter conditions, whereas they do not persist in hot, dry environments (Borg 1968). Environmental surveys in France (Barre et al. 1979) and New Zealand (C.G. Mackintosh and T.G. Henderson, unpublished data) have yielded *Y. pstb* isolates from pasture and water samples in winter months. This property of cold tolerance is utilized in the laboratory to enhance the recovery of organisms from samples that are subcultured into media and kept at 4° C for up to 3 weeks, during which time *Y. pstb* multiplies, and most other bacteria remain dormant or die.

Pathology

Enteric yersiniosis causes profound damage to the gastrointestinal tract in deer. Usually the ileum, cecum, 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 edematous. The abomasal wall may be reddened, and there may be petechial hemorrhages. Histopathological changes in the intestine can include acute enteritis with patchy ulceration and edema of the mucosa and submucosa (Beatson and Hutton 1981). This may be

accompanied by mesenteric lymphadenitis with focal necrosis. This profound damage to the mucosa causes dehydration and protein loss and allows *Y. pstb* organisms to invade the body via the lymphatic system.

Treatment

Treatment with parenteral antibiotics, proprietary scour medicines, and fluid therapy for 3 or 4 days is usually successful if instituted early in the disease (Mackintosh 1984). The antibiotics of choice are oxytetracycline or trimethoprim/sulphonamide combinations to which the majority of isolates are sensitive (Hodges et al. 1980). To curtail an outbreak, in-contact deer may be treated en masse with long-acting tetracyclines or neomycin or they may be fed concentrate pellets medicated with tetracyclines.

Prevention

Good management practices, adequate feeding, provision of shelter from bad weather, and minimizing stress will help to prevent yersiniosis. The stress of weaning can be minimized 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. Alternatively, 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.

Immunity and Vaccination

Investigation of the immune response of deer to *Y. pstb* infection and killed bacterins has been carried out at Invermay for a number of years. A previous paper (Mackintosh et al. 1986) reviewed the literature and presented the results of field studies, experimental infections, and vaccination trials. In essence, these studies showed that preliminary trials using killed *Y. pstb* organisms with aluminum hydroxide adjuvant gave promising results, but future vaccines should have a higher antigenic load, especially of the "virulence" antigens and have a better adjuvant to ensure a higher and more prolonged immunological response. Subsequent work has led to the development of an ELISA test for *Y. pstb* in deer which can measure the humoral response of deer to both somatic and virulence antigens (S. Hook, unpublished data) based on methods developed by Hibma and Griffin (1978) and modified specifically for deer (Hibma and Griffin 1988). Studies have also shown that there is a cell-mediated immune (CMI) component in the response of deer to infection and immunization with *Y. pstb* (Mackintosh et al. 1986). Recent vaccination studies have confirmed that deer produce a good humoral and CMI response to an oil adjuvanted killed *Y. pstb* vaccine which gave significant protection against a live virulent *Y. pstb* experimental challenge compared with unvaccinated controls (C. Mackintosh, unpublished data). Interestingly, this experimental challenge also showed that some sires had a significantly higher proportion of their calves affected with yersiniosis than others, suggesting that there is a genetic component to susceptibility to this disease.

The vaccination studies are continuing, and it is hoped that a commercial vaccine will be available in the near future. This, along with good management practices, should minimize losses due to yersiniosis in young weaned deer.

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