THE EPIDEMIOLOGY OF YERSINIOSIS IN DEER

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Introduction

Yersiniosis is one of the most important infectious diseases of farmed deer in New Zealand (N. Gladden, 1981; McAllum, 1981) and has caused a number of deaths among British farmed deer (Fletcher, 1982; Anon, 1983a; Anon, 1983b; Anon, 1984). In this paper yersiniosis refers to the primary condition in deer caused by *Yersinia pseudotuberculosis (Y. pstb.)*. A related organism *Y. enterocolitica* does not appear to be a common cause of primary disease in deer and can be isolated from 30-80% of normal deer faeces. When isolated from dead deer *Y. enterocolitica* is frequently associated with other conditions such as trauma and malignant catarrhal fever (Henderson, 1983) and therefore it is probably an opportunist or secondary invader. Clinically, yersiniosis presents as a profuse, green, watery, smelly diarrhoea, anorexia, rapid loss of condition, dehydration, recumbency and death. Often animals are found dead with no obvious premonitory clinical signs, although when examined *post mortem* the animals usually have evidence of diarrhoea, which is often blood stained, around the tail and on the hocks. The disease is characterised by a very severe haemorrhagic enteritis, enlarged mesenteric lymph nodes and a terminal septicaemia.

The objective of this paper is to review previously published information on the epidemiology of yersiniosis in deer and to describe recent epidemiological investigations carried out at Invermay.

Incidence and Prevalence

True incidence or prevalence data on yersiniosis are not available, although subjective assessments and reports have been made in various areas based on clinical observations, small surveys and Animal Health Laboratory records.

There is a general view that yersiniosis was more common in the early days of deer farming in New Zealand (i.e. mid to late '70s) when many deer were live-captured, transportation was common and deer were frequently underfed in winter. These cases were often sporadic but occasionally large outbreaks occurred (Hunter, 1981). One such outbreak in 1978 (Beatson and Hutton, 1981) resulted in the death of 60 deer from a herd of 863 with all ages and classes of stock involved. The Lincoln Animal Health Laboratory reported that this property and 4 others in Canterbury had losses of up to 5% in that season (Anon, 1978).

For the 4 years, 1980 to 1983, annual telephone surveys of deer farmers in the Canterbury area (N.S. Beatson, pers. comm.) have recorded yersiniosis as responsible for between 3.5% and 12% of deer losses, with the peak occurring in 1981. Farmers reported yersiniosis and tuberculosis as the two most common infectious causes of deer deaths after M.C.F. which accounted for 30-40% of deaths.

The prevalence of yersiniosis varies from year to year, with 1978 being particularly bad (Anon, 1978). During the winter of 1983 there was an

apparent increased incidence, not only in deer (Anon, 1983b) but also in goats and sheep in Canterbury (Hutton, 1983). From the Waikato district there was a report that 50 deer on a property died from yersiniosis (Anon, 1983c). A subsequent telephone survey (Anon, 1983e) indicated that over a 6 week period 621 deer developed signs of the disease and 141 of these died. The total number of deer at risk in these herds was about 2100. During the same season the Whangarei Animal Health Laboratory reported increasing numbers of cases in cattle, sheep, goats and deer (Anon, 1983f).

Of the total number of deer samples submitted to the Invermay Animal Health Laboratory between 1979 and 1982 16.3% were diagnosed as cases of yersiniosis, and this percentage exceeds that found for any other domestic mammal submitted to the laboratory (Henderson, 1983). Similarly, 14.3% of deer specimens submitted for bacteriological examination to the Ruakura Animal Health Laboratory in the 5 month period June-October 1979 yielded *Y. pstb.* (Hodges *et al.*, 1980). Over the last 5 years (1979-83) there were respectively 75, 56, 64, 53 and 92 recorded cases of *Y. pstb.* diagnosed from deer samples submitted to the Animal Health Laboratories throughout New Zealand (N.S. Beatson, pers. comm.) and these are assumed to reflect annual variations in the numbers of cases occurring in the field.

Age and Sex

In an analysis of samples submitted from the Otago-Southland region, 64% of yersiniosis cases were from animals less than one year old (Henderson, 1983). However, this age group comprises only 20 to 25% of the total population. Therefore, it appears that the attack rate for this age group is much higher than for any other. Henderson (1983) also reported that there were no significant differences in prevalence between sexes of the cases submitted to the Invermay Animal Health Laboratory. However, in the surveys carried out in the Canterbury area (N.S. Beatson, pers. comm.) farmers reported more males dying of yersiniosis than females. The attack rate (number of cases of yersiniosis in males divided by the total number of males) for males in 1982 was 9 times as high as that for females while in 1983 it was 4 times as high.

The duration of the immunological response to infection and the consequent resistance to reinfection are not known, nor is the degree of cross-protection between serotypes. These factors influence the age susceptibility of animals. The fact that few cases develop in calves less than 3 months of age suggests there is some colostral protection, although summer conditions do not favour infection (see below). However, calves in their first winter when 4-9 months of age, appear to be most susceptible to infection. Similarly newly captured animals which presumably have not been exposed to *Y. pstb.* serotypes commonly found in the farming environment may also appear very susceptible. The longer animals survive on farms the more likely they are to develop some immunity and this could be why the attack rate is higher in young animals (Henderson, 1983). The greater susceptibility of young deer to infection with *Y. pstb.* is demonstrated by the high isolation rates (10%) obtained from normal weaner calves in their first winter (Hodges *et al.*, 1984b; Mackintosh and Henderson, unpub.) compared with the low isolation rate (1%) from clinically normal adult stags samples at the same time of year in a deer slaughter premise (Mackintosh and Henderson, 1984a). A large proportion of these latter animals had low serological titres indicating that they had been previously exposed to infection and were unlikely to be susceptible to reinfection.

Seasonal Pattern

Clinical yersiniosis has a distinctly seasonal pattern of occurrence with a peak in winter (see Fig. 1). The seasonal distribution of cases diagnosed by Animal Health Laboratories between 1975 and 1983 is shown in Figure 1. Over 74% of cases occurred in June, July and August. Similarly, in an analysis of cases occurring in the Otago/Southland region the peak of cases occurred from May to August (Henderson, 1983). Large outbreaks have been recorded only in the winter months. Cases at other times of the year appear to be sporadic and some of these are associated with recent capture (Mackintosh and Henderson, unpub.). Years in which increased numbers of reported cases have occurred, such as 1978 and 1983, have had wet, cold winters following dry autumns when feed shortages have occurred (Anon, 1978; Hutton, 1983).

Predisposing Factors

It is well recognised that stressors of various kinds are essential predisposing factors to clinical yersiniosis. *Y. pstb.* has been isolated from apparently normal deer on a number of occasions (Henderson and Hemmingson, 1981; Hodges *et al.*, 1984b; Mackintosh and Henderson, 1984a) including studies at Invermay (see below). However, it appears that clinical disease is manifested only when an animal is weakened by intercurrent disease or is exposed to external stressors. These can include feed shortage, capture, transportation, frequent yarding and handling, social stress such as frequent introductions of new animals into a group or high stocking densities, rapid changes of diet and extreme climatic conditions. Deer, especially young animals and stags after the rut, enter the winter with low fat reserves, they have relatively sparse coats, and they have reduced voluntary food intakes. If shelter is not provided during periods of bad weather in winter it can result in animals going into a negative energy balance. Hunter (1981), when commenting on yersiniosis outbreaks stated that "losses invariably occurred within 24-48 hours of wet southerly weather" and "body condition of dead animals was always poor".

Stress results in increased plasma levels of glucocorticoids which inhibit leucocyte response to infection, reduce hormonal immunity to suppressing B and T lymphocytes and alter other non-specific cell-mediated immune responses (Fowler, 1978; Trindle *et al.*, 1978). For example it has been shown in cattle that stress caused by transportation and parturition or the administration of exogenous corticosteroids can cause the reactivation of latent IBR virus infections (Narita *et al.*, 1981). Prolonged stress can lead to adrenal exhaustion, and manipulation of an animal in a state of incipient adrenal insufficiency is likely to cause fatal shock (Fowler, 1978). Adverse dietary and environmental conditions profoundly affect the factors that regulate the localisation and population levels of micro-organisms in the gastro-intestinal tract, such as gut motility, mucous secretion and antibody production and they appear to precipitate overt disease in domestic animals and experimental mouse models infected with *Salmonella* spp. (Tannock and Savage, 1974). The pathogenesis of yersiniosis in stressed deer is likely to be similar.

Transmission

Infected animals shed *Y. pstb.* in their faeces and it is assumed that natural infection follows ingestion of faecally contaminated food or water. *Y. pstb.* infection is endemic in some species of animals which act as long term reservoirs and faecal excretors, while other species which are

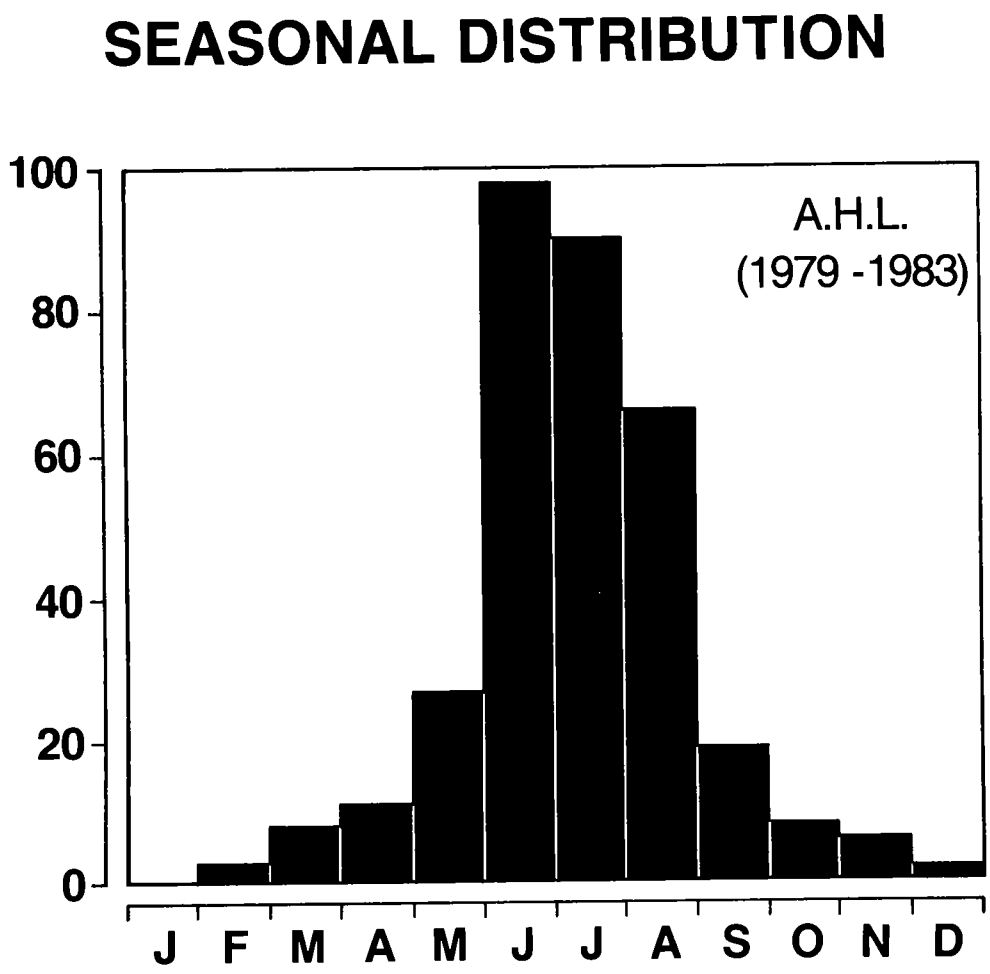

Fig. 1: The total number of cases per month of *Yersinia pseudotuberculosis* in deer diagnosed by the Animal Health Laboratories from 1979 to 1983.

accidental hosts may be clinically affected but are probably not long term excretors. The organisms can survive for long periods in the environment in cold, wet winter conditions, whereas they are susceptible to drying and sunlight and therefore do not persist in the environment during the summer months (Borg, 1968). This was demonstrated by Barre *et al.* (1979a) during an environment survey for *Y. pstb.* in France. They obtained isolates from the soil only during winter months (November-March) and none at other times of the year. Similarly, isolates of *Y. pstb.* have been made during the winter from soil and pasture samples from the Invermay deer farm (Mackintosh and Henderson, unpub.). This property of cold tolerance is utilised in the laboratory to enhance the recovery of the organisms from samples which are sub-cultured into media and kept at 4°C for up to 3 weeks, during which time the *Y. pstb.* organisms multiply and most of the other bacteria remain dormant or die. Thus the transmission of infection is facilitated by cold, wet winter conditions.

Potential Reservoirs of Infection

In northern hemisphere countries, rodents, lagomorphs and various birds are regarded as the principal reservoirs of infection (Mair, 1968; Hubbert, 1972; Mair, 1973; Mair, 1975; Obwolo, 1976; Stovell, 1979). Predators such as cats which may be directly infected by eating rodents and birds may also act as carriers (Mair, 1973).

During an investigation of potential wildlife sources of *Y. pstb.* from deer on the Invermay farm during 1982 and 1983 22 strains of *Y. pstb.* were isolated from 675 apparently normal small mammals and birds with the following prevalence rates: feral cats 27.8%, Norway rats 8.6%, mice 5.5%, hares 3.8%, rabbits 1.9%, ducks 5.3%, sparrows 2.3%, seagulls 2.3% and starlings 1.7% (Mackintosh and Henderson, 1984b). It thus appears that the Invermay farm and presumably the rest of New Zealand has wildlife reservoirs of infection similar to those found overseas. This is not unexpected since most of these species were introduced to New Zealand from the northern hemisphere.

It is not known to what extent domestic livestock act as reservoirs of *Y. pstb.* Sporadic cases of yersiniosis have been diagnosed in sheep, goats, cattle and pigs in New Zealand (Anon, 1979; Anon, 1983e; Anon, 1983f; Hutton, 1983; Hodges *et al.*, 1984a) and overseas (Watson and Hunter, 1960; Mair and Harbourne, 1963; Langford, 1969; Hubbert, 1972; Mair *et al.*, 1979; Delia *et al.*, 1981; Jones and Mair, 1982) and have usually been associated with enteritis or abortions. As in deer most cases occur in winter and stress is a predisposing factor. *Y. pstb.* has also been isolated from apparently normal domestic animals including cattle (Hodges *et al.*, 1984b), pigs (Zen-Yoji *et al.*, 1974; Toma and Deidrick, 1975; Mair *et al.*, 1979; Mackintosh and Henderson, unpub.), dogs and cats (Yanagawa *et al.*, 1978).

Serotypes

To determine the sources of outbreaks and reservoirs of infection it is necessary to identify accurately the strains of *Y. pstb.* At present *Y. pstb.* strains are classified as serotypes I (A and B), II (A and B), III, IV, V and VI. To date only strains belonging to serotypes I, II and III have been recovered from animals in New Zealand (Hodges *et al.*, 1984a; Henderson, 1983; Mackintosh and Henderson, 1984b).

The ratios of serotypes found in clinical cases and reservoir hosts may give clues to the possible origins of infections and demonstrate differences in the epidemiology of yersiniosis in Otago/Southland and the South Auckland Region. In Otago-Southland the most common serotype isolated from deer is serotype I. From 61 clinical cases of yersiniosis in deer submitted to the Invermay Animal Health Laboratory (Henderson, 1983) *Y. pstb.* isolates had serotype I:II:III ratios of 4:2:1. Clinically normal young deer in their first winter on the Invermay deer farm yielded 20 isolates with ratios of 8:1:1. Conducted in parallel with this, the wildlife survey on the Invermay deer farm yielded 22 isolates with ratios of 15:6:1 (Mackintosh and Henderson, in press). These results are similar to those found in the United Kingdom (Mair, 1965) and France (Barre *et al.*, 1979b) where I is the predominant serotype isolated from wildlife. Therefore it is likely that the majority of deer infections in the Otago-Southland area originate from wildlife.

In the South Auckland Region serotype III is the most common serotype isolated from both clinically affected and normal deer (Hodges *et al.*, 1984a; Hodges *et al.*, 1984b). The serotype ratios reported were 4:1:8 and 4:1:43 for clinically affected and normal deer respectively. These authors also reported III as the most common serotype isolated from cattle, sheep, goats and pigs, while rabbits, guinea-pigs and aviary birds yielded only serotypes I and II. This suggests that in the South Auckland Region domestic animals are an important source of serotype III infection. Overseas, serotype III is rarely isolated from wildlife but has been commonly isolated from aborted foetuses and clinically normal calves and pigs (Zen-Yoji *et al.*, 1974; Toma and Deidrick, 1975; Mair *et al.*, 1979).

Subclinical Infections in Deer

The widespread distribution of *Y. pstb.* in wildlife, domestic animals and the environment is likely to result in the exposure of the majority of farmed deer in winter. Faecal samples from 38 red deer calves on the Invermay deer farm were cultured at 3 to 4 weekly intervals from March to October, 1983 and 11 to 20% of deer yielded *Y. pstb.* isolates on each sampling occasion between May and August. A total of 20 (53%) animals had positive faecal cultures on at least one occasion and 37 (97%) developed serological macroscopic agglutination titres ≥1:80 over this period. None of these animals showed any clinical signs of disease. A survey of faeces from 527 weaner deer conducted in late June and July 1983 on 10 farms near Hamilton yielded isolates from 47 animals (9%) (Hodges *et al.*, 1984b). This point prevalence is similar to that found at each sampling at Invermay, suggesting that Hodge's survey detected current outbreaks of *Y. pstb.* infection. Therefore it appears likely that the majority of young deer experience a subclinical *Y. pstb.* infection in their first winter. Clinical disease is probably precipitated by concurrent stress.

Summary and Conclusions

Yersiniosis caused by *Y. pstb.* is one of the most important infectious diseases of farmed deer in N.Z. The majority of clinical cases occur in young deer under stress in their first winter. Wild animals and possibly some domestic animals act as reservoirs of infection and contaminate the farming environment. *Y. pstb.* organisms survive well in the environment under cold wet winter conditions. Young deer entering their first winter and recently captured adults are exposed to these organisms which colonise and multiply in their intestines. Once infected the deer shed the organisms in their faeces, thus propagating an outbreak within the group by

deer-to-deer transmission. If deer are stressed by climatic conditions, underfeeding or transport, then an outbreak of clinical disease may result in a proportion of infected animals. Yersiniosis is characterised by severe enteritis, septicaemia and death. If deer are healthy and relatively stress free then the infection is likely to be subclinical, with the development of an immunological response and transient faecal shedding. The longevity and the degree of cross-protection of the immunological response is not known. However, adult deer appear to be much less susceptible to yersiniosis. Warn farmers that yersiniosis is a zoonosis and causes lymphadenitis with appendicitis-like symptoms.

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