LEPTOSPIROSIS IN DEER

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Epidemiology of leptospirosis in New Zealand

Six serovars of *Leptospira interrogans* are commonly isolated from animals in New Zealand; *hardjo*, *pomona*, *tarassovi*, *ballum*, *copenhageni*, and *balcanica*. Each serovar has one or more maintenance hosts which can carry the infection in their kidneys for relatively long periods of time, maintaining endemic infection in those populations of animals. Usually the maintenance host is unaffected by the infection. Other non-maintenance species may be susceptible to infection and usually show some clinical signs of disease and are regarded as accidental hosts.

Some of these accidental hosts may shed the organism in their urine for weeks or even months, thus propagating an epidemic within the flock or herd, although infection will not become endemic in a stable group of animals. For example, the pig is the maintenance host for *pomona* and can shed the organism for over a year (Ryan, 1978). Cattle are accidental hosts for *pomona* with infection resulting in abortions and haemoglobinuria and leptospiruria can persist for three to four months (Hellstrom, 1978; Mackintosh, 1981). However, *pomona* infection does not appear to become endemic in cattle herds in New Zealand. Conversely, cattle are maintenance hosts for *hardjo* while other ruminants (sheep, goats and deer), although susceptible to infection, are probably accidental hosts for this serovar (Schollum and Blackmore, 1981; Blackmore *et al.*, 1982; Fairley *et al.*, 1984).

In summary, *hardjo*, *pomona* and *tarassovi* are maintained by domestic animals while *ballum*, *copenhageni* and *balcanica* are maintained by wildlife in New Zealand. A seventh serovar, *australis* has been isolated on only one occasion from a man in Northland and no animal reservoir has been identified.

Feral deer in New Zealand: There is no evidence to suggest that leptospirosis is endemic in feral deer in New Zealand. A survey of 279 deer of various species from throughout New Zealand (Daniel, 1966; 1967) only detected one titre ≥1:200 to *pomona*. All samples were seronegative to *canicola*, *icterohaemorrhagiae*, *grippotyphosa*, *australis* A, *australis* B, *hyos* and *andoman*. Hathaway *et al.* (1981) found 27 red and 4 sika deer seronegative (<1:24) to *ballum*, *copenhageni*, *hardjo*, *pomona*, *tarassovi*, *australis*, *autumnalis*, *bataviae*, *biflexa*, *canicola*, *grippotyphosa* and *pyrogenes*. The kidneys from 5 deer were also negative on culture.

In the past, when feral deer frequently grazed near bush margins and on pasture they were likely to have been exposed to *hardjo*, *pomona* and *tarassovi* carried by domestic livestock. The single *pomona* titre in Daniel's deer survey probably originated from such exposure, but the infection did not become established in the feral population. However, with the recent hunting pressure on deer in accessible areas such exposure is now even less likely to occur in feral deer. Their exposure to wildlife reservoirs of leptospirosis is probably not great either, due to the low stock density of feral deer and their extensive grazing and browsing habits.

Farmed deer in New Zealand: The majority of deer farms in New Zealand have been developed on existing farmland and deer are usually managed in conjunction with traditional livestock. Sheep and cattle are often grazed inside the deer fence to assist with pasture management, and many farmers still keep a few pigs on their property. Consequently farmed deer are likely to be exposed to *hardjo*, *pomona* and *tarassovi*.

Farms are also synanthropic foci for rodents which are a potential source of *ballum* and *copenhageni*. Such increased exposure is bound to result in an increased incidence of infection in farmed deer compared with feral deer. The wallowing behaviour of deer make them especially liable to exposure since leptospires need water in which to survive in the environment. Any upstream contamination may lead to an outbreak of infection which would be propagated rapidly by deer-to-deer transmission via pasture and water.

Hardjo is endemic in the majority of dairy herds in New Zealand and infection appears to cycle in the spring when young heifers joining the herd for the first time and the environmental conditions are most suitable for leptospiral survival (Hellstrom, 1978). *Pomona* causes sporadic outbreaks in cattle herds at any time of the year when they are exposed to infected pigs or cattle, or contaminated waterways.

Management patterns for deer are different to those for cattle. Much of the mixing of different age groups occurs in the late summer-autumn period before the rut, and in the winter after the rut. However, the management varies greatly and is influenced mostly by the herd size and the number of paddocks available. In addition, the market for deer is very fluid at the moment with all classes of stock being bought and sold. Consequently, the epidemiology of leptospiral infections in farmed deer is likely to vary greatly from farm to farm. Clinical cases of leptospirosis associated with *pomona* have been reported from the Waikato, Rotorua and Southland areas and case histories of two of these outbreaks will be reported in the following paper. *Pomona* and *hardjo* were isolated from the urine of deer in these outbreaks and it is assumed that domestic animals were the original sources of infection, although it is very likely that the outbreaks were propagated by deer-to-deer transmission. Although some deer have been shown to excrete *pomona* for up to eight months and *hardjo* for up to three months they are unlikely to act as maintenance hosts. This would require leptospiruria to last for longer than the generation interval of the host to ensure transmission from one generation to the next. (Fairley *et al.*, 1984). In a recent survey of yearlings on 10 farms in the Nelson area (V.B. Williams, pers. comm.) only one farm had evidence of *hardjo* infection and in this case seven out of 12 animals had titres ≧1:50. None of these animals had displayed any clinical signs of disease leading one to suspect that, as in cattle, sheep and goats, *hardjo* infections in deer are subclinical. The survey also showed that 6 of the farms had a high prevalence of low titres to *copenhageni*, while a few deer had titres to *pomona*. If these *copenhageni* titres truly reflect previous infection with this serovar then it indicates that the deer are being exposed to infected rat urine and that these infections in deer were subclinical.

Leptospirosis in deer overseas

Australia: Wild populations of deer in Tasmania, Victoria and New South Wales appear to have a low serological prevalence of leptospiral titres to *pomona* and *grippotyphosa* (Munday, 1972; Milner *et al.*, 1981; English, 1982). Surveys of farmed deer in Victoria have shown that 11% of fallow deer and 79% of rusa deer in one area had titres to *hardjo* ≧1:32 and one of the rusa

which had a titre of 1:128 was found to be shedding *hardjo* in its urine (Durfee and Presidente, 1979; Presidente and Westbury, 1979; Milner *et al.*, 1981; English, 1982). These infections are thought to have originated from cattle infected with *hardjo* grazing paddocks adjacent to the deer enclosures.

United Kindgom: Surveys of wild deer in the U.K. have found a very low prevalence of leptospiral titres. Twigg *et al.* (1973) found 1/94 fallow deer seropositive (1:30 to *icterohaemorrhagiae*) and 1/210 red deer seropositive (1:30 to *bataviae*). In a survey of red deer farmed in Scotland, Blaxter *et al.* (1974) reported 2/94 seropositive (1:30 to *icterohaemorrhagiae* and 1:300 to *ballum*) both presumably resulting from wildlife reservoirs. In 1978, Corrigall reported a fatal *ballum* infection in a penned deer at the Rowett Institute in Scotland. The increasing number of deer being farmed in the U.K. may lead to an increased incidence of leptospirosis in deer, due to their greater exposure to domestic livestock and rodents and to the increased stock density, especially when deer are kept indoors over the winter.

United States: Leptospirosis due to *pomona* in white-tailed deer was first recorded in 1956 (Wedman and Driver, 1957). Since then numerous surveys of wild white tailed and black tailed deer in the U.S. have demonstrated high serological prevalences of leptospiral titres to *pomona*, *grippotyphosa*, *canicola* and *pyrogenes* (Ferris *et al.*, 1961; Reilly *et al.*, 1962b; Trainer *et al.*, 1963; Shotts and Hayes, 1970; Cirone *et al.*, 1978). The infections originate from both wildlife and domestic livestock reservoirs.

U.S.S.R.: During a 10 year survey of farm animals in Western Siberia 16% of 191 reindeer and 56% of 32 maral (wapiti-type) deer had leptospiral titres ≥1:100, especially to *grippotyphosa* and *pomona* (Chulovskii, 1971). A survey of red deer on a deer farm in Western U.S.S.R. found a high proportion with leptospiral titres (Korenberg *et al.*, 1975).

France: A zero seroprevalence of leptospiral titres in 189 wild roe deer has been reported (Blancou, 1983).

Yugoslavia: 7% of 270 wild deer had titres, especially to *ballum* and *pomona* (Kovacic *et al.*, 1983).

German Democratic Republic and Federal Republic of Germany: Surveys of wild deer have demonstrated low serological prevalences from 1.8 to 5.7%, with titres to *icterohaemorrhagiae*, *ballum* and *grippotyphosa* predominating, in various deer species (Hubner and Horsch, 1977; Weber and Christoph, 1981).

China: Outbreaks of "haematuria disease" have occurred on some deer farms affecting up to 75% of animals and causing 90% mortality in those affected. *L. romania*, which is carried by mice, was isolated from affected animals and a homologous vaccine has controlled the disease (Gou, 1981).

Clinical Signs

There have been a small number of outbreaks of leptospirosis on deer farms in New Zealand, some of which have resulted in deaths. On one reported case a four year old red hind collapsed and died after 18 hours of general malaise. Post mortem examination showed signs of an acute haemolytic episode with haemoglobinuric nephrosis and dard red urine. The deer had a high *pomona* titre and leptospiruria just prior to death. Five out of 12 in contact deer had titres to *pomona*, *hardjo*, *tarassovi* and *ballum* (Anon, 1980). Bertram and Howell (1981) reported that deer calves infected with

pomona became weak, unthrifty and exhibit redwater before they die. Fairley *et al.* (1984) reported on the death of four 3 to 4 month old calves in the Rotorua district which, on post mortem, showed enlarged kidneys but no sign of red water or icterus. This and one other case history will be presented in the following paper.

Generally it appears that infections with *hardjo* are subclinical while *pomona* causes clinical disease in a proportion of those infected, especially younger animals. These features are similar to those seen in cattle infected by these two serovars. Similarly it is possible that they may cause abortions in pregnant hinds as they do in cattle. Experimental infections of five pregnant white-tailed deer with *pomona* in the U.S. caused four to abort while non-pregnant hinds showed few clinical signs of infection (Trainer *et al.*, 1961; Ferris *et al.*, 1960). However, experimental infection of one immature deer with *pomona* resulted in haemoglobinuria and death (Reilly *et al.*, 1962a). Abortions of wild white-tailed deer due to *pomona* have been shown to occur occasionally under natural conditions (Shotts, 1981).

In Scotland, a 22 month old red stag infected with *ballum* was inappetant and dull for several days before a 6 day period of severe illness when it had congested mucous membranes, severe inco-ordination and colic before it died (Corrigall, 1978). It had a elevated leucocyte count (11,000 per mm^3).

"Haematuria disease" in farmed deer in China caused by *romania* was characterised by pyrexia, haemolytic anaemia, jaundice and haemoglobinuria, with an acute course of 10 days and over 90% mortality (Gou, 1981). Necropsy showed ecchymotic haemorrhages in the lungs and necrotic, haemorrhagic lesions in the liver and kidney.

Treatment

As with other domestic livestock the drug of choice is streptomycin (25 mg/kg daily for four days by injection) or penicillin/streptomycin mixtures. Tetracycline may be useful during the acute phase but may not eliminate kidney colonisation and leptospiruria.

Young deer may suffer a haemolytic crisis and a blood transfusion from an adult deer could be considered. Adult erythrocytes are probably more resistant to haemolysis than foetal erythrocytes present in young deer. In severe cases where there is kidney damage, electrolyte therapy such as lactated Ringer solution should be administered to compensate for impaired renal function.

If a leptospiral abortion storm appears to be in progress it may be prudent to vaccinate the hinds and inject them with streptomycin, in the hope that the streptomycin will eliminate infection during the incubation phase and eliminate leptospiruria. However, if the leptospires are already in the foetus the antibiotic is unlikely to cross the placenta in high enough concentrations to prevent foetal death.

Prevention

The prevention of leptospirosis is a more satisfactory strategy than treating clinically affected animals, especially since deer are so valuable and also because of the zoonotic risks. The most reliable prevention strategy is to institute a vaccination programme for all livestock on the property. In most areas a *hardjo/pomona* bivalent vaccine should suffice

but a trivalent vaccine containing an additional *copenhageni* component may be necessary in areas where rats carry this serovar, such as in the Waikato district. The following speaker will deal with the subject of vaccination more fully.

If vaccination is not practical then deer should not be exposed to other domestic livestock or water that may have been contaminated by them.

Zoonotic Aspects

Hardjo and *pomona* have both been isolated from the urine of clinically normal deer as well as obviously diseased animals. All deer urine should be considered as potentially hazardous. Likewise aborted foetuses, placenta membranes and kidneys. It is the veterinarian's responsibility to warn his clients of the potential dangers, especially if leptospirosis is diagnosed on the property.

Veterinarians working in Deer Slaughter Premises should also be aware of the potential dangers to them as well as meat workers and meat inspectors associated with urine splash from ruptured bladders and with handling kidneys.

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