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## REVIEW: Epidemiology of Johne's disease in farmed red deer (*Cervus elaphus*) in New Zealand

C.G. MACKINTOSH

AgResearch Invermay, P.O. Box 50-034, Mosgiel, New Zealand

### ABSTRACT

Johne's disease, caused by *Mycobacterium avium* subsp *paratuberculosis* (*M. ptb*), has emerged as a significant cause of losses on deer farms in New Zealand. It causes outbreaks of fatal clinical disease in young deer eight to 15 months of age, sporadic deaths in adults and production losses due to poor growth rates in yearlings, reduced calving percentages in hinds, reduced velvet production in stags, interference with tuberculosis testing and tuberculosis-like lesions in gut lymph nodes at slaughter. Although bovine and ovine strains have been isolated from deer, the bovine strain is more pathogenic and is responsible for the outbreaks of serious disease in young deer. Clinical disease results from heavy early challenge of young deer with the bovine strain. Mature animals are relatively resistant to infection. Calves are generally infected via the oral route from contaminated pasture and water. Intrauterine transmission to the foetus occurs commonly in clinically and subclinically infected hinds and infected milk may also play a role in transmission. Deer-to-deer transmission appears the most common route, although cattle-to-deer and sheep-to-deer transmission may occur. The role of wildlife is not known. Resistance to Johne's disease has a genetic component, which appears to be related to resistance to bovine tuberculosis.

**Keywords:** deer; Johne's disease; *Mycobacterium avium* subsp *paratuberculosis*; epidemiology.

### INTRODUCTION

Johne's disease, also known as paratuberculosis, which is caused by *Mycobacterium avium* subsp *paratuberculosis* (*M. ptb*), has emerged as a problem on deer farms or enclosures in the United Kingdom (Fawcett *et al.*, 1995; Gilmour, 1988), Europe (Commichau, 1982; Godfroid *et al.*, 2000; Power *et al.*, 1993), North America Manning *et al.*, 1998; Starke, 1991), South America (Mereb *et al.*, 1994), Australia (Schroen *et al.*, 2003) and New Zealand (Gumbrell, 1986; Mackintosh, 1998). It was first confirmed in farmed red deer in New Zealand in the mid 1980s. Since then the incidence of Johne's disease has increased steadily. Between 1986 and 2000, *M. ptb* was confirmed by culture and/or polymerase chain reaction (PCR) from 619 farmed deer in 299 herds (de lisle *et al.*, 2003), with over 85% of cases occurring in the second half of that period. Only 5.8% of these cases were from clinically affected animals, while the majority were from tuberculosis-like lesions found in lymph nodes at meat inspection in deer slaughter premises. From 2001 to 2005, *M. ptb* was isolated from 1,141 farmed deer in over 600 deer herds in New Zealand, representing an observed prevalence of ~15% for the estimated 4,000 deer farms (de Lisle *et al.*, 2006). The prevalence of subclinical *M. ptb* infections in farmed deer is not known, but up to 10% of animals in some lines of apparently normal deer have had macroscopic lesions in mesenteric lymph nodes at slaughter (Mackintosh *et al.*, 2004). It is highly

probable that the actual prevalence is much higher and many subclinically infected animals do not have macroscopic evidence of infection. The prevalence appears to have been increasing over recent years and a nationwide case-control study in 2005 found *M. ptb* was geographically widespread on deer properties throughout both North and South Islands (Glossop *et al.*, 2006). In 2007 it was reported that 50% of New Zealand deer herds have serological evidence of *M. ptb* infection (Griffin *et al.*, 2007).

Johne's disease has emerged as a significant cause of production losses on deer farms. In addition to outbreaks of fatal clinical disease in young deer six to 15 months of age and sporadic deaths in adults, deer farmers may experience losses due poor growth rates in yearlings, reduced calving percentages in hinds, reduced velvet production in stags, interference with testing for bovine tuberculosis (tuberculosis) and tuberculosis-like lesions in gut lymph nodes at slaughter (Glossop *et al.*, 2006; Mackintosh *et al.*, 2004).

### EPIDEMIOLOGY

The epidemiology of Johne's disease in farmed deer is likely to have much in common with cattle and sheep. Research over the last 10 years has led to a better understanding of the specific features of the epidemiology of Johne's disease in farmed deer and how it differs from other domestic livestock. A nationwide case-control study has been undertaken to determine some of the risk factors of Johne's disease on deer farms in New Zealand (Glossop *et al.*, 2007b).

Recently a new database has been set up by Johne's Management Limited based on the detection of Johne's lesions at slaughter, and this should provide useful data in the future (Glossop *et al.*, 2007a).

### Strain of *M. ptb*

In New Zealand and Australia, sheep are generally infected with the "ovine" or "S" strain and cattle are infected with the "bovine" or "C" strain of *M. ptb* (Collins *et al.*, 2002; Whittington *et al.*, 2000a). Both strains have been isolated from deer (de Lisle *et al.*, 1993; de Lisle *et al.*, 2006), although the bovine strain was found on >95% of the 95 infected farms studied. Evidence from the field suggests that the "bovine" strain is responsible for the majority of serious outbreaks of clinical disease in young red deer (O'Brien *et al.*, 2006). An oral challenge study was carried out to compare the virulence of "bovine" and "ovine" strains of *M. ptb* in young red deer, and the results showed that the "ovine" strain, which had been isolated from a clinical case of Johne's disease in a sheep, was considerably less virulent for red deer than the "bovine" strain, which had been isolated from a clinical case of Johne's disease in a deer (Mackintosh *et al.*, 2007). Thus there is a potential risk of cross-species infection with grazing deer with other livestock, especially cattle (Mackintosh & Wilson, 2005). A recent study showed that the prevalence of Johne's disease in weaner deer was negatively associated with the proportion of days grazed by sheep on the deer unit compared with all stock units, but was positively correlated with grazing beef yearlings on the deer farm (Glossop *et al.*, 2007b). As with sheep and cattle, one of the greatest risk factors is the introduction of infected deer from other herds.

### Dose

It has been shown in sheep and cattle that heavy exposure to *M. ptb* early in life is likely to lead to severe clinical Johne's disease (Nisbeth *et al.*, 1962; Reddacliff & Whittington, 2003; Sweeney *et al.*, 2006). A challenge study in young red deer showed that there was a direct correlation between the dose rate of the "bovine" strain and severity of disease in young red deer (Mackintosh *et al.*, 2007). The minimal oral infective dose was close to  $10^3$  organisms for this "bovine" strain, while clinical disease resulted from an oral dose  $>10^9$  organisms. Clinical Johne's disease in yearling red deer is likely to result from high early challenge with the "bovine" strain of *M. ptb*.

### Age susceptibility

There is evidence from studies in sheep and cattle that there is increasing resistance to Johne's disease with age. Although sheep and cattle may be infected as adults they are much less likely to

develop serious lesions or clinical disease (Clarke, 1997; Larsen *et al.*, 1975; Rankin, 1962). Sheep and cattle generally do not develop clinical disease until they are two to four-years-old, although under experimental conditions young lambs <14 days-old exposed to very heavy challenge of  $>10^9$  colony forming units, developed severe disease in less than six months (Nisbeth *et al.*, 1962). It is assumed that deer exposed to early challenge are more likely to develop clinical disease, and the development of clinical signs of Johne's disease in deer as young as eight months of age suggests very heavy early challenge.

### Genetic resistance/susceptibility

Genetic resistance/susceptibility is thought to play a role in Johne's disease in dairy cattle (Gonda *et al.*, 2006; Gonda *et al.*, 2007; Koets *et al.*, 2000) and sheep (Hickey *et al.* 2003; Reddacliff *et al.*, 2005) and it is likely to be important in deer as well. Estimates of the heritability of resistance to Johne's disease range from 0.06 in cattle in Holland (Koets *et al.*, 2000) to 0.1 in cattle in Denmark (Mortensen *et al.*, 2004) and to 0.18 in Merino sheep (Hickey *et al.*, 2003). There has also been a recent report of a quantitative trait loci (QTL) on BTA20 affecting susceptibility to *M. ptb* infection in United States Holsteins (Gonda *et al.*, 2007). A study of transmission of Johne's disease in dairy cattle in Denmark concluded that the parental contribution was significant, and both heritability of susceptibility and vertical transmission should be considered in any control programs (Nielsen *et al.*, 2000).

Over the last 10 years, considerable insight into genetic resistance of red deer to tuberculosis has been gained (Mackintosh *et al.*, 2000) and semen has been collected from some stags that showed resistance or susceptibility to tuberculosis in order to breed offspring with these attributes for further research. A recent *M. ptb* challenge trial showed a strong correlation between resistance to bovine tuberculosis and resistance to *M. ptb* (C.G. Mackintosh, Unpublished data).

### Routes of transmission

Traditionally, the faecal-oral route is regarded as the most important route of transmission of Johne's disease, but transmission can also occur across the placenta and via colostrum or milk (Sweeney, 1996).

**Faecal-oral:** A proportion of infected animals shed large numbers of *M. ptb* in their faeces (Collins, 2003), resulting in contamination of pasture, water, concentrate feed and teats, and ingestion is the most likely route of infection (Valentin-Weigand & Goethe, 1999). It has been shown that M cells in the epithelium of intestinal tract are targeted by *M. ptb* for attachment and

provide a means of gaining entry into the animal (Sigurdardottir *et al.*, 2004).

The oral route has been used in many experimental infection trials in sheep, cattle and deer (Hines, *et al.*, 2007). Most trials have successfully infected the animals, but many have failed to produce clinical Johne's disease and have attributed this failure to a number of factors including the strain of *M. ptb*, the use of cultured organisms that may have become attenuated and older animals being less susceptible.

**Transplacental:** Intra-uterine transmission in clinically affected animals has been demonstrated in cattle (Kopecky *et al.*, 1967; Lawrence, 1956; McQueen D, 1979; Pearson & McClelland, 1955; Seitz *et al.*, 1989) and sheep (Lambeth *et al.*, 2004), and recently in deer (Deutz *et al.*, 2003; van Kooten *et al.*, 2006). There is a high risk of transmission of *M. ptb* from clinically affected red deer hinds to their foetuses during pregnancy, with 90% of hinds having an infected foetus (van Kooten *et al.*, 2006). In a second study, *M. ptb* was isolated from 58% of the foetuses of a group of 18 subclinically infected red deer hinds (Thompson *et al.*, 2007). In sheep and cattle, by contrast, it has been reported that transmission to the foetus occurs in <10% of subclinically affected animals (Kopecky *et al.*, 1967; Lambeth *et al.*, 2004; Sweeney *et al.*, 1992). A recent meta-analysis showed that 9% (95% c.i. 6-14%) of foetuses from subclinically infected cows and 39% (20-60%) from clinically affected cows were infected with *M. ptb* (Whittington & Windsor, 2007). The true rates of infection are thought to be higher than these figures suggest due to the sensitivity of culture methods being less than 100%. The incidence of cattle-calf infection derived via the *in utero* route was estimated to be in the range 0.44 to 1.2 infected calves per 100 cows per annum. in herds with within-herd prevalence of 5% and 3.5 to 9.3 in herds with 40% prevalence.

**Via milk:** *M. ptb* is shed in the milk of infected cattle and sheep (Collins, 2003) and it is very likely that infected hinds can transmit infection to their fawns via this route since *M. ptb* was isolated from 67% (12/18) of subclinically infected hinds (Thompson *et al.*, 2007).

### Wildlife

*M. ptb* has been isolated from rabbits (*Oryctolagus cuniculus cuniculus*), foxes and stoats (*Mustela erminea*) in the UK (Beard *et al.*, 1999; Greig *et al.*, 1997), a variety of wildlife in Australia (Cleland *et al.*, 2001) and ferrets in New Zealand (de Lisle *et al.*, 2002), suggesting that wildlife may act as a source of infection for farmed deer, as well as sheep and cattle. Recently a survey of wildlife was conducted in the vicinity of three Johne's affected deer farms in Canterbury, and *M. ptb* was

isolated from a wide range of small animals and birds (Nugent *et al.*, 2007). The significance of interspecies transmission of *M. ptb* is open to much speculation because it is relatively easy to show associations, but it is difficult to prove causal relationships. One factor that may facilitate interspecies transmission is that animals tend not to avoid the faeces of other grazing species (Greig, 2005). For example cattle calves have been shown to ingest rabbit faecal pellets (Daniels *et al.*, 2001; Judge *et al.*, 2005).

### Rates of shedding and survival of *M. ptb* in the environment

Clinically affected animals generally excrete large numbers of *M. ptb* organisms and represent the most obvious source of infection. However, it has been shown in cattle (Bolton *et al.*, 2005) and sheep (Whittington *et al.*, 2000b) some subclinically infected individuals may also excrete significant numbers of organisms. Stress and pregnancy may increase the rate of shedding.

In deer the rate of excretion appears to be related to the severity of disease and the strain of *M. ptb* (Mackintosh *et al.*, 2007). Deer that received the highest dose of cattle strain *M. ptb* developed the most severe lesions, had the highest levels of antibody and were the heaviest excretors.

*M. ptb* organisms are able to survive for up to 11 months in soil (Collins, 2003), although there are a wide range of environmental factors that can affect the survival rate (Norby *et al.*, 2007).

### CONCLUSION

In farmed red deer, outbreaks of clinical disease generally occur in red deer eight to 12 months of age, suggesting high early challenge with the bovine strain of *M. ptb*. A proportion of these cases may be due to intra-uterine infection because of the high rate of transmission in clinically and subclinically affected hinds. Early exposure may also occur via milk or by grazing in close contact with infected hinds.

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