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The aetiology of tuberculosis and mycobacterial diseases in farmed deer

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Mycobacterial diseases are of paramount importance among the infectious processes that cause disease in farmed deer. Deer may be infected by *Mycobacterium bovis*, *M. tuberculosis*, *M. paratuberculosis* or *M. avium-intracellulare*. Tuberculosis in farmed deer is caused predominantly by *M. bovis*, though in a zoological park deer may be infected by *M. tuberculosis*. Tuberculosis may present as small encapsulated caseo-calcified lesions within the lungs or lymph nodes or it may produce large liquefactive lesions within the lymphoid organs. Tuberculous abscesses within the lymphoid tissues may produce sinuses that drain onto the epithelial surfaces of the body, facilitating spread of infection. Clinical symptoms of tuberculosis in deer are usually evident only in the last few weeks of life, and infected animals may harbour large volumes of liquefactive material without any overt clinical evidence of disease. Significant levels of tuberculous infection may occur in young animals that show no detectable macroscopic lesions. Infection of deer with *M. paratuberculosis* may produce the classical symptoms of Johne's disease. This disease usually occurs sporadically in New Zealand, though widespread infection of young animals has been observed in one herd in Scotland. Infection of compromised animals with *M. avium* may produce caseo-calcified lesions within the lymph nodes that are indistinguishable microscopically from *M. bovis* lesions.

Key Words: Deer, Tuberculosis, Paratuberculosis, Avian Tuberculosis, Pathology, Microbiology.

Epidemiology

Tuberculosis that results from the infection with *Mycobacterium bovis* is known to occur in cervidae in the wild, in zoological parks and, more recently, in farmed deer. Infection has been diagnosed in a number of species that include fallow deer (*Dama dama*) (Kollias, 1978; Quinn and Towar, 1963), roe deer (*Capreolus capreolus*) (Gunning, 1985), axis deer (*Axis axis*) (Jones, Manton and Cavanagh, 1976), sika deer (*Cervus nippon*) (Dodd, 1984), red deer (*Cervus elaphus*) (Livingstone, 1980) and antelope (Clancey, 1977).

Since tuberculosis (Tb) was recognised as a disease in feral deer within New Zealand (NZ) the prospect that the infection could be an important health issue in farmed deer has been given the most serious consideration. For a considerable time (1950-1980) Tb was not perceived as a significant disease in wild NZ deer, because tuberculous lesions had not been found within meat processing plants. Subsequent studies have shown that deer have a propensity to develop lesions within the lymph nodes of the head, thorax and abdomen. This may mean that the true incidence of Tb in feral deer has been underestimated because routine meat inspections were carried out on headless, eviscerated carcases of culled feral deer. Since 1978, when Tb was first diagnosed in farmed NZ deer (Beatson and Hutton, 1981), there has been an increasing awareness of possible problems due to tuberculosis within the local deer farming industry. It is now recognised (de Lisle and Havill, 1985) that tuberculosis is the most important bacterial disease affecting farmed deer in New Zealand, and there is a comprehensive voluntary control scheme in place with the aim of eradicating tuberculosis from farmed NZ deer.

The reasons why this disease is being given special consideration are as follows:

1. Deer appear to be particularly susceptible to infection by *M. bovis*, which may spread quickly within an infected herd.
2. A reservoir of Tb infection exists and is endemic within NZ in cattle herds and is present in feral possums (Ekdahl, Smith and Money, 1970), which are recognised to be particularly susceptible to Tb infection (Corner and Presidente, 1980), producing acutely fulminating disease with massive levels of infection.
3. Other mycobacterial species may infect and sensitize deer to tuberculin antigens, causing complications due to false positive reactivity to routine diagnostic skin tests that have been used in tuberculosis eradication schemes.

Experience gained in the management of tuberculosis in deer herds (Griffin and Cross, 1986) suggests that should Tb remain undiagnosed within a deer herd for three to five years, it may become so widespread that depopulation of the herd is the most likely prospect. Recently, a herd under study by our laboratory progressed from the initial identification of Tb in a young animal that died unexpectedly to 90 *per cent* infection in the adult stock within six months, when the herd was depopulated. Strikingly, none of 80 young stock (< four months) running with infected adults on this farm had a detectable lesion on slaughter at six months of age. Such extreme cases of disease spread are in contrast to other herds where a small number of Tb-infected animals may not cause widespread infection. Inadequate diagnosis of infection, due to the absence of disease management, coupled with the special susceptibility of some deer herds to infectious Tb spread, may produce rapid and widespread infection within a herd. Infectious spread may also relate to genetic resistance factors and it may be exacerbated by stress associated with

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capture of feral animals, or with transport and intensified management practices involved with farmed animals (Griffin, 1987).

Within New Zealand, Tb has been found predominantly in the wild in three areas, which include the Central North Island, the West Coast of the South Island and the Wairarapa area. Endemic deer infection in these areas may be the result of Tb infection in wild possums or cattle herds. There is now evidence (Livingstone, 1988) that farmed deer are acting as a reservoir for infection of possums in non-endemic tuberculosis areas of New Zealand. It is recognised that farmed deer add a new dimension to the complexity of tuberculosis control in National Eradication programmes for Tb in cattle.

Tb has been found predominantly in mature breeding hinds: this may relate, in part, to current deer farming practices, which have involved retention of the majority of females as breeding stock to expand the National herd. It is also possible that higher rates of infection in breeding hinds may be associated with impaired diagnosis due to false negative skin test reactivity in those animals where there is reduced sensitivity of pregnant females to tuberculin (Outteridge, 1985), possibly with an increased susceptibility to spread of infection. Tuberculosis (Rich, 1951) and leprosy (Duncan, Pearson, Ridley, Melson and Bjune, 1982), have been found to be exacerbated during pregnancy in women and there has been a demonstrable pregnancy-associated depression of cellular immunity in ruminants (Griffin and Davis, 1985), which may compound the problem of mycobacterial infection and diagnosis in farmed deer hinds.

Infection usually spreads directly by the respiratory or oral route so the primary concern must be to accurately diagnose infection within a deer herd before entertaining ideas about possible reinfection from feral animals such as possums. Because seriously infected animals may produce open lesions that drain as sinuses to the skin or to the epithelial surfaces of the lungs or gastrointestinal tract, early and accurate diagnosis of infected stock is of paramount importance in control of tuberculosis spread within a deer herd. Because animals may produce enlarged superficial lesions it is important that surgical lancing of lymph node abscesses is not carried out indiscriminately in a herd with an unknown Tb history. Spread of Tb infection within one of the herds under study in our research programme has been traced to the surgical discharge of a pyogenic abscess on the jaw of an affected hind. An unsuspecting veterinarian could justifiably have diagnosed such a lesion as a classical *Actinomyces pyogenes* abscess, found commonly in ruminants.

Clinical presentation of tuberculosis in deer

Severely infected animals may show signs of emaciation, though the low toxigenicity of the infection produces disease in some animals that is clinically undetectable even though they may harbour large abscesses within the thorax, or in lymph nodes of the head or mesenteric area. Gross enlargement of superficial lymph nodes may be evident by palpation. Following clinical presentation animals usually show rapid deterioration and may become moribund within weeks.

Tb lesions in adult animals are often characterised by the formation of abscesses within the lymph nodes. On incision these lesions usually discharge yellow liquefactive pus more typical of acute pyogenic infection. Such lesions are more common than the classical caseous or calcified lesions typical of tubercle formation in Tb infection of cattle or humans. Lesions within lymph nodes may vary in size from 1 to 2 mm to large space occupying lesions up to 10 cm in diameter, which may involve total replacement of the lymphoid tissue with an encapsulated necrotic mass.

In feral deer lesions are found predominantly in the thorax (75 *per cent*) or as generalised tuberculosis (GTb) (13 per *cent*), which involves more than one site within the lymphoid tissue Lisle and Havill, 1985). Farmed deer usually present with le in the head (especially retro-pharyngeal lymph nodes) o mesenteric lymph nodes. Within a single infected farmed herd there is usually a characteristic type of lesion found prec nantly in the head, thorax or abdomen (Griffin - unpublis Where a significant level of infection is present within a generalised infection may be found in up to 20 *per cent* affected animals. Usual combinations of infection involve and thorax or head and abdomen (Brooks, 1984; de Lisl Havill, 1985). However, there is a need for a comprehe necropsy of all suspect animals. There have been herd his in New Zealand where Tb was spreading from deer to deer a herd, and yet the only lesions found in animals at necropsy classical tubercles in the retropharyngeal lymph nodes (I gomery, 1986). Similarly, approximately 90 *per cent* of re cattle with confirmed infection have tuberculous lesions in l nodes associated with the respiratory tract but lung lesion found in only 1 to 2 *per cent* of such cases during routine at examination; however, careful dissection, supplemented by logical examination, revealed tuberculous lesions in over cent of such lungs (McIlroy, Neill and McCracken, 198 should not be assumed that the detection of a single encaps lesion in a lymph node of a deer indicates a small risk of spr Tb within a herd.

Histopathology of cervine tuberculosis

Characteristic Tb lesions in deer are encapsulated and surro by macrophages and epithelioid cells. In the outer zone mac ages coalesce to form Langhan's giant cells (Beatson, 198 have a large central zone of acidophilic cytoplasm with ar ring of nuclei. Lesions may have a central necrotic are rounded by neutrophils and containing invading bacteria may be surrounded by Langhan's cells which often engulf isms. When lesions are found with a caseous or granular c tency they usually have many Langhan's giant cells prese an outer zone of lymphocytes and fibrous granulation tis wall off the lesion. Acid-fast bacilli, identified by Ziehl N staining, may be found in numbers that vary from plent extremely sparse (i.e., they are evident only after detailed nation of many histological fields). Because of the propen Tb to form abscesses in deer it has been recommended (B 1985) that any abscess found in a deer from a herd with defined herd history must be suspected as a possible Tb unless proven otherwise by histological or bacteriological nation. In addition, the absence of macroscopic lesions ca taken as unequivocal evidence of freedom from Tb in ind animals necropsied in herds with known infection, as it ha shown that, in heavily infected herds, microbial culture of lymph nodes may result in the isolation of *M. bovis* from *per cent* of animals showing no evidence of tuberculous (Beatson, Hutton and de Lisle, 1984). Necropsy examina young deer (up to four months old) from seriously infecte must also be treated with special caution as there may be n levels of infection with acid-fast bacilli in lesions that are detectable macroscopically. There appears to be an inver tionship between the cellularity of the mononuclear cell re mounted by the host and the level of proliferation of a bacilli within the animal. These observations suggest th care is necessary when carrying our necropsies on deer l be infected by *M. bovis*. Detailed macroscopic examinatic be followed by histological and bacteriological studies accurate diagnosis or exclusion of Tb. Recent studies of M isolates from feral animals within a small area of New Z (Collins, Gabric and de Lisle, 1988), suggest that many d

biotypes of *M. bovis* may be present within a small geographic area.

Diseases caused by Mycobacteria of the avium-intracellulare complex (MAIC)

Johne's disease in deer

This disease was first described in roe deer in 1905, six years before the first laboratory isolation of the causative organism, *Mycobacterium paratuberculosis*. Evidence of exposure of free-living or farmed deer to *M. paratuberculosis* is seen from isolation of the organism or seroconversion in animals. The infection has been reported to occur in fallow and axis deer (Reiman, Zaman, Ruppanner, Jorgensen, Worsaae and Behymer, 1979), white tailed deer (Chiodini and Van Kruiningen, 1983), elk (Jessup, Abbas and Behymer, 1981) and red deer (McKelvey, 1987; Buxton, 1987). A serological survey of white-tailed deer (*Odocoilcus virginianus*) (Shulaw, Gordon, Beech-Nielsen, Pretzman and Hoffsis, 1986) in Ohio showed that 2.5 *per cent* of 954 feral animals sampled had serologic titres to *M. paratuberculosis*.

Transmission of *M. paratuberculosis* has been shown to occur between Rocky mountain goats and domestic sheep (Williams, Snyder and Martin, 1983) so it is likely that *M. paratuberculosis* in feral or domesticated ruminants can provide a reservoir of infection that allows deer to contract Johne's disease. Although widespread in sheep and cattle, Johne's disease has been observed to occur only sporadically (12 cases) in NZ farmed deer. A significant outbreak of Johne's disease has been documented in a major deer herd in the UK with fatal clinical disease being found especially in young stock (McKelvey, 1987). Management of infection in this herd (using microbiological culture and serologic testing) failed to control the spread of disease and vaccination has been introduced in an attempt to control disease spread within young stock.

Together, these data suggest that deer can become infected by *M. paratuberculosis* and under certain conditions the disease can produce a relatively acute syndrome, especially in young stock, with dramatic consequences for herd management.

M. avium infection of farmed deer.

After *M. bovis*, *M. avium* is the second most improtant mycobacterial species isolated from deer specimens at necropsy. De Lisle and Havill (1985) found 35 isolates of *M. avium-intracellulare* from deer in New Zealand between 1979 and 1983. Twenty-two of the isolates were from skin test reactor deer but only five of these had gross lesions detectable at necropsy. Spread of *M. avium* within a deer herd has never been documented in New Zealand. *M. avium* has also been isolated from lesions found in axis deer in a zoological park in the UK (Jones *et al.*, 1976).

While this disease is usually found to occur sporadically, widespread infection has been reported in a red deer herd in Scotland (H.W. Reid - personal communication). Munro (1986) noted that the experience with *M. avium* in the UK suggested that fallow deer and sika deer appear to be the most susceptible to *M. avium* infection. However, different levels of infection may be found even within a species in different geographic areas, as seen by a fourfold higher level of *M. avium* infection of roe deer in Southern England compared with that found in Northern Scotland. Avian tuberculosis in deer usually produces lesions in the intestinal wall and mesenteric lymph nodes. Lesions may be purulent, caseous or granulomatous. Haematogenous spread to the liver and lungs may occur to produce miliary lesions and a terminal septicaemia.

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