A new perspective on the epidemiology of tuberculosis in farmed deer

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Introduction

It is accepted that the chief route of infection for tuberculosis in both man and cattle is via the respiratory tract (Francis, 1958). Sneezing and coughing generate a range of droplets sizes, with only the smallest going directly to the lungs which are very susceptible to infection by small numbers of organisms. Larger droplets and dust particles lodge in the upper respiratory passages which are more resistant to infection. Thus close contact, especially in enclosed environments, facilitates the spread of Tb. The incidence of Tb is considerably higher in dairy than beef cattle in most countries, especially in Europe where dairy cattle spend much of their time housed. Surveys of these dairy animals have shown that up to 80% of lesions were in the lung, bronchial or mediastinal lymph nodes (LNs), clearly suggesting an aerogenous spread. However, 10% were in the mesenteric LN suggesting some alimentary spread. The origin of the 10% of lesions in the retropharyngeal LNs has always been uncertain. In New Zealand, thoracic lesions account for only half of the single lesions in cattle and the head nodes are the second most common area affected. This suggests that the epidemiology of Tb in cattle in New Zealand differs from elsewhere, and it is likely that dairy cattle and beef cattle also differ within this country. The epidemiology of Tb in deer also appears to be different to that in cattle.

Route of transmission: A number of surveys of deer have all shown that 50-60% of single Tb lesions in farmed red deer are in the head LNs, especially the medial retropharyngeal, and tonsils. Thoracic lesions are usually 10-20% of the total and similar to abdominal lesions. These figures suggest that the majority of infections result from exposure of the nasal and oral cavities by contaminated droplets, fomites, dust or discharges. Relatively few are infected by true aerosols. The medial retropharyngeal LN receives lymphatic drainage from the pharyngeal and palatine tonsils, the tongue, floor of the mouth, hard and soft palate, gums, caudal nasal passage and sinuses.

Experimental infection studies (Mackintosh *et al.* 1993) have shown that intra-nasal spray inoculation with 200 colony forming units (cfu) of virulent *M. bovis* resulted in only one of five animals becoming infected, and this animal had thoracic lesions. Intra-nasal inoculation with 20,000 cfu resulted in disease in all five deer, with three animals having submandibular and retropharyngeal LN abscesses and extensive lung and thoracic LN involvement when necropsied 8

months after inoculation. The other two had abscessation of either retropharyngeal and anterior mediastinal or retropharyngeal and ileocaecal LNs. By contrast intratonsil inoculation in over 100 deer in a number of trials has resulted in Tb in 50-100% of deer with doses as low as 8 cfu (Mackintosh *et al.* 1995b). In almost all cases the primary lesion is in the medial retropharyngeal LN. These findings strongly point to the tonsils as common routes of primary infection in farmed deer. Infected tonsils may also act as a source of secondary spread to the lungs and alimentary tract, and to other animals via infected saliva and coughed droplets. The majority of early cases of progressive Tb in deer are confined to the retropharyngeal LN (and tonsils). Later in the course of disease infection may spread to thoracic and abdominal organs, and a small number of head LN abscesses burst out onto the skin surface, leaving an open draining sinus which can discharge highly contaminated material.

Source of Infection for Farmed Deer: Within farms, transmission is either deer-to-deer or wildlife-to-deer. Ten to twenty years ago much of the tuberculosis on farms was due to the purchase of infected deer, which were either infected when live-captured, or infected on-farm from other deer. Today, there is little or no live capture of wild deer, and the National Tb Control Programme has severely curtailed movement of infected stock. There are still some Tb infected herds where deer-to-deer transmission occurs, especially if a herd breakdown is not detected for one to two years.

From observations on the rate of lesion development in naturally and experimentally infected animals deer in the first 6 to 8 months of disease are not very infectious under normal pasture conditions. Factors that affect the degree of exposure include stocking density, social interactions between animals (e.g. hind/calf, hind/hind and hind/stag interactions) and management procedures such as yarding, supplementary feeding, indoor wintering, transport and oral drenching.

As the Control Scheme continues to reduce the prevalence of Tb in deer, the relative importance of wildlife-to-deer transmission increases. Risk factors associated with wildlife are poorly understood, but it has been suggested that the curiosity and aggressiveness of deer towards wildlife, especially terminally ill possums which behave abnormally, may put them at higher risk than cattle (Morris *et al.* 1994).

Susceptibility: Although the degree of exposure is probably the most important predetermining factor in Tb infection, the susceptibility of the host is also a significant component. Two of the most important factors are stress and genetics. Their recent domestication and the physiological stresses of their annual hormonal cycles probably make deer more susceptible than other domestic ruminants. Simulated chronic stress, using dexamthasone implants, reduces their response to vaccination and increases the severity of Tb in red deer (Thomson *et al.* 1994). Recent experimental challenge studies suggest that there is a genetic component to resistance/susceptibility (Mackintosh *et al.* this series).

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