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Ferrets as vectors of bovine Tb in New Zealand: a review

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ABSTRACT

Feral ferrets (*Mustela furo*) are a serious pest of agricultural importance because they transmit bovine tuberculosis (*Mycobacterium bovis*; Tb) to domestic livestock. They therefore pose a significant risk to New Zealand's international trade in dairy, beef and venison products. The most likely route of infection to ferrets is oral, through killing and eating infected prey or scavenging infected carrion (particularly possums). Indeed, the prevalence of Tb in ferrets is positively correlated with possum abundance and infected possums. Male ferrets have a higher prevalence of Tb than females, and adult ferrets have higher prevalence than juveniles. It is still not clear whether Tb can cycle independently in feral ferrets in the absence of external (non-ferret) sources of infection (i.e., "maintenance hosts") or whether the disease is simply "spillover" infection from other wildlife populations (predominantly possums). Available evidence suggests that ferrets are more likely to be spillover than maintenance hosts in most habitats in New Zealand. However, several aspects of the epidemiology of Tb in feral ferrets, and the ecology of feral ferrets, need to be investigated in order to resolve this debate.

Keywords: bovine tuberculosis; ferret; maintenance host; *Mustela furo*; *Mycobacterium bovis*; spillover host; vector.

FERRETS: A BRIEF OVERVIEW

Feral ferrets (*Mustela furo*; Mustelidae) weigh up to 2.5 kg but are one of the larger carnivores in New Zealand. Historically, feral ferrets were found throughout the central North Island as well as the Bay of Plenty and Hawke's Bay and in the drier eastern and southern areas of the South Island. Current evidence suggests that their range is expanding into Northland and the wetter western areas of both islands (Clapperton, 2001). However, ferrets are found in particular where rabbits, their primary prey, occur in high numbers (Ragg & Walker, 1996). Hence they are found predominantly in pasture, rough grassland, and scrubland, and are still relatively rare in forests and wetter areas (Lavers & Clapperton, 1990). In semi-arid grasslands and braided riverbeds of the South Island, their population densities range from 3 to 8/km^2 (Cross *et al.*, 1998; Caley & Morriss, 2001). The diet of feral ferrets consists primarily of small mammals (lagomorphs, rodents, and possums), and part of their diet consists of scavenged material (Smith *et al.*, 1995; Ragg, 1998). They also eat a variety of birds, lizards, and invertebrates (Lavers & Clapperton, 1990). Ferrets therefore pose a threat to conservation values in New Zealand because of their predation impacts on indigenous wildlife, particularly ground-nesting birds (Pierce, 1987).

There is now growing evidence in New Zealand that feral ferrets may transmit *Mycobacterium bovis* (bovine tuberculosis; Tb) to domestic livestock, and Sauter & Morris (1995) showed a plausible mechanism whereby Tb could be transmitted from ferrets to livestock. Terminally ill ferrets attract the attention and curiosity of cattle and farmed deer, which may inspect, sniff, and mouth atypically-behaving ferrets (Sauter & Morris, 1995). Ferrets may therefore pose a significant risk to New Zealand's international trade in dairy, beef and venison products. In this review I discuss the history of Tb in feral ferrets in New Zealand and examine aspects of the ecology of ferrets that make them susceptible to the disease (with emphasis on diet and dispersal movements). I also summarise the latest research efforts to determine whether ferrets are "maintenance" hosts of *M. bovis* (i.e., whether *M. bovis* is cycling independently in feral ferrets in the absence of external [non-ferret] sources of infection) or "spillover" hosts (i.e., whether the disease is sourced from other wildlife populations, predominantly possums).

HISTORY OF BOVINE TB IN FERRETS IN NEW ZEALAND

The first record of a feral ferret infected with bovine Tb in New Zealand was from Taumarunui in 1982 (de Lisle *et al.*, 1993). In the late 1980s, Tb was recorded in ferrets from Marlborough (de Lisle *et al.*, 1993) and in 1990 in the Mackenzie Basin (Walker *et al.*, 1993). A survey undertaken in the Mackenzie Basin in 1991/92 indicated that 15.5% of ferrets were tuberculous (Walker *et al.*, 1993).

Subsequent observations indicated that Tb-infected ferrets were found wherever Tb had been recorded in cattle or possums (Ragg *et al.*, 1995b; Ragg & Walker, 1996; Caley 1998). Visible lesions in ferrets are usually characterised by enlargement of the lymph nodes (primarily the mesenteric lymph nodes; Lugton *et al.*, 1997a). Prevalence of visible lesions in ferret populations varies from zero to 66% (Ragg *et al.*, 1995b; Caley, 1998; Caley, 2000), and Ragg *et al.*, (1995a) commonly recorded figures of 15% and 18% in some ferret populations in Tb-endemic areas of Otago and the Mackenzie Basin. In North Canterbury, between 0% and 33% of ferrets have been recorded with visible lesions (Caley *et al.*, 1998). Estimates of prevalence obtained from visible gross lesions can be increased by up to 50% upon bacterial culture of tissue from necropsied ferrets (e.g., Caley *et al.*, 1998). This finding is in accordance with the finding by Lugton *et al.* (1997a) that 27% of infected tuberculous ferrets did not have visible lesions.

ROUTES OF INFECTION IN FERAL FERRETS

By far the most significant route of infection for Tb in feral ferrets in New Zealand appears to be via the alimentary tract. The lymph nodes associated with the alimentary tract are the most common site of gross lesions, which suggests

that ferrets contract the disease either by scavenging infected carrion or by killing infected prey (Lugton *et al*., 1997b; Ragg *et al.*, 2000). One of the most likely sources of Tb in ferrets is the scavenging of infected possum carcasses (Smith *et al.*, 1995; Ragg, 1998; Ragg *et al.*, 2000). Surveys from various parts of New Zealand have shown that the prevalence of Tb in ferrets is positively correlated with possum abundance, but not with ferret abundance (Caley, 1998). This suggests that tuberculous possums are a major source of Tb infection for feral ferrets. However, high densities of ferrets and often high prevalences of Tb are found in Otago and the Mackenzie Basin, where infected possums are found at relatively low abundance (Ragg *et al.*, 1995b; Lugton *et al.*, 1997b). This has prompted some authors to conclude that ferrets are becoming infected with Tb from other wildlife sources such as hedgehogs (a species that carry Tb; Lugton *et al.*, 1995), and indeed ferrets are known to eat hedgehogs (Pierce 1987; Smith *et al.*, 1995). Other sources of Tb infection in ferrets would be the scavenging of carcasses of domestic livestock, or cannibalism (de Lisle *et al.*, 1995; Ragg *et al.*, 2000).

There are several other possible routes by which feral ferrets could be infected with bovine Tb. Approximately 20% of visible lesions have been found in peripheral lymph nodes (Ragg *et al.*, 1995b), suggesting that infection could occur through social contact with other ferrets, such as fighting and mating. These infections are particularly common among males, and appear to reflect the higher rate at which males interact with other ferrets in a population (Lugton *et al.*, 1997a; Ragg *et al.* 1995b). Ferret-to-ferret contact through den-sharing is another possible way in which ferrets could become infected with Tb. However, Norbury *et al.* (1998b) suggested that den-sharing among adult ferrets is relatively rare in tussock grassland habitat and that if transmission of Tb from ferret to ferret occurs, it is unlikely to occur by this means. Respiratory infections are relatively uncommon in ferrets (Lugton *et al.* 1997a). About a quarter of tuberculous ferrets in Lugton *et al.*'s (1997b) study provided evidence of oral shedding of Tb bacilli (through coughing or sneezing), apparently in ferrets where the disease is well advanced. Likewise, shedding of potentially infectious bacilli through faeces and urine is probably relatively rare (Lugton *et al.*, 1997b). Bacilli in milk from infected female ferrets could transmit disease to suckling young (Lugton *et al.*, 1997b) but again, appears to be uncommon (Caley, 2000).

SEX AND AGE OF FERRETS INFECTED WITH BOVINE TB

Male ferrets on average have a higher prevalence of Tb than females, possibly because they are more likely to scavenge carrion (Lugton *et al.*, 1997b). Adults appear to have a higher prevalence than juveniles (Lugton *et al.*, 1997b; Caley *et al.*, 1998). Female ferrets eat more invertebrates and birds, whereas males focus on possums and lagomorphs (Ragg, 1998), which also may account for the lower observed prevalence of the disease in female ferrets, although observed sex-specific differences were small.

Caley (2000) proposed five possible hypotheses explaining the underlying mechanisms for transmission of *M. bovis* among ferrets:

1. Transmission occurs during suckling until the age of weaning at age 1.75 months.
2. Transmission occurs during mating, and the fighting activities associated with it, starting at age 10 months.
3. Transmission occurs during routine social activities from the age of independence at 2.5 months.
4. Transmission occurs through scavenging tuberculous carrion or killing tuberculous prey from the age of weaning at 1.75 months.
5. Transmission occurs through environmental contamination from birth.

These hypotheses are not necessarily mutually exclusive. For example, in any given population of ferrets, some ferrets may come into contact with the disease at weaning, whereas others may not come into contact with the disease until they have started routine social activities at 2.5 months (a combination of hypotheses 4 and 3). On average, however, the prevalence of Tb in the ferret population increases sharply in ferrets from the age of 1.75 months. Available data would indicate that hypothesis 4 explains how most feral ferrets in New Zealand acquire the infection (Caley, 2000), although more work needs to be done on changes in the rate of infection with age, i.e., the instantaneous rate of infection.

DISPERSAL MOVEMENTS OF FERRETS AND THE IMPLICATIONS FOR TRANSPORTING TB

The reliance of ferrets on rabbits as primary prey in the South Island high country means that, when rabbits are removed (either through large-scale control operations or by an RHD [Rabbit Haemorrhagic Disease] epidemic), concomitant large-scale dispersal occurs involving potentially infected adult and juvenile ferrets in search of food. This has important implications for transporting the disease throughout the landscape. For example, large (up to 3-fold) home range expansions of adult ferrets were observed on Grays Hills Station in the Mackenzie Basin following a 99% reduction in rabbit population densities (Norbury *et al.*, 1998a). Norbury *et al.* (1998a) suggested that, depending on the extent that ferrets are capable of spreading bovine Tb among themselves and to other wildlife and domestic stock, these dispersal events could increase the rate of spread of the disease. Two recent studies have examined dispersal movements in juvenile ferrets, and observed maximum movements of 45 km (Byrom, 2001) and 21 km (Caley & Morriss, 2001) respectively. Both studies indicate that juvenile ferrets already infected with bovine Tb may transport the infection to new areas, with the potential to create new localised sources of infection. Median dispersal distances of juvenile ferrets were 5.0 km and 1.2 km in the respective studies, which suggests that from a management perspective, buffer zones of up to 5 km wide may be required to reduce the spread of the disease to domestic stock and to other wildlife by 50% of dispersing ferrets.

FERRETS AS MAINTENANCE OR SPILLOVER HOSTS OF BOVINE TB

The question of whether *M. bovis* is cycling independently in feral ferrets in the absence of external (non-

ferret) sources of infection (i.e., whether ferrets are "maintenance" hosts), or whether ferrets are simply infected as a "spillover" from other wildlife populations (predominantly possums) is the subject of considerable debate in New Zealand (Morris & Pfeiffer, 1995; Ragg *et al.*, 1995b; Lugton *et al.*, 1997b; Qureshi *et al.*, 2000).

Available evidence suggests that most Tb infection in ferrets is derived from non-ferret sources of infection, primarily possums (Caley, 1998; Caley, 2000). However, work on captive ferrets has shown that ferret-to-ferret transmission of Tb can occur in some situations, and raises the possibility that horizontal transmission might occur in areas with high population densities of ferrets (Qureshi *et al.*, 2000). For example, ferret scavenging on ferret carcasses is particularly common (Ragg, 1998; Ragg *et al.*, 2000), which could result in horizontal transmission. In addition, it is still not known whether there is a "threshold" population density of ferrets above which the population can maintain Tb infection independent of other wildlife sources. Determining whether such a threshold exists is critical from a management perspective, because such localised "hot spots" of ferrets would be a top priority in any control operation.

Despite the association of infected ferrets with infected cattle herds (Ragg *et al.*, 1995a), no strong causal relationships have yet been found between the incidence of Tb reactors in cattle and either the prevalence of Tb in ferrets or the abundance of infected ferrets (Morris and Pfeiffer, 1995). However Caley *et al.* (1998) found that reducing total ferret density resulted in a decline in the reactor incidence in cattle in North Canterbury. Admittedly, the frequency of close contact between ferrets and livestock is likely to be lower than that between livestock and possums, as livestock are less attracted to ferrets than to possums (Sauter & Morris, 1995). In addition, open-draining lesions are less common in ferrets than in possums, making transmission from ferrets to livestock less likely. However, the discovery of *M. bovis* infection in ferrets in southern areas of New Zealand, such as Otago and the Mackenzie Basin (where possums are uncommon overall, and the incidence of Tb in possum populations is also low), suggests that more information is needed on the link between Tb infection in ferrets and Tb infection in livestock, particularly since Tb is apparently declining from these areas without much vector control (R. Walker, pers. comm.).

Morris & Pfeiffer (1995) argued that the persistence of specific strains of Tb in possum populations for prolonged periods of time could be taken as evidence that the disease was being maintained in those populations independent of reinfection from domestic livestock or from other wildlife hosts. However, unlike in possum populations, little work has been done on specific strains of Tb in ferret populations in New Zealand, and ferret populations have not been investigated over a wide enough geographic area or through time to determine whether specific strains of Tb can be maintained in ferret populations without reinfection from other wildlife sources. DNA fingerprinting has identified Tb isolates of both the West Coast Group and the Central Otago Group from ferrets in the Mackenzie Basin (de Lisle *et el.*, 1995), which suggests that ferret populations in those areas have been infected (and possibly reinfected) from multiple sources. Given that the most common source of infection in ferrets is from non-ferret sources, it is highly likely that ferrets would pick up a mixture of strains of Tb from whatever other sources of wildlife were present in a particular area.

In summary, evidence to date suggests that ferrets are primarily spillover rather than maintenance hosts of *M. bovis* in most New Zealand habitats (Caley, 1998, 2000). The obvious management implication of this conclusion is that a reduction in possum abundance may substantially reduce the prevalence of the disease in ferret populations (Caley, 2000; Caley *et al.*, 2001;), which can lead to strategic decisions about which species to target first if resources for control operations are limited. No clear-cut case has been found where Tb is persisting in a ferret population in the absence of a possum population, so ferrets are probably spillover hosts at all population densities of ferrets, but they may also be maintenance hosts where their densities exceed some unknown threshold that appears likely to be above population densities found in most areas of New Zealand.

WHAT RESEARCH STILL NEEDS TO BE DONE?

It would be fruitful to explore several avenues of research into the epidemiology of Tb in ferret populations. First, the maintenance/spillover debate needs to be resolved definitively. From a management perspective this is critical, because if ferrets are simply spillover hosts, management of other wildlife sources of infection (particularly possums) would be the most cost-effective way of managing the disease in ferrets also. This can only be done by examining the host status of ferrets after sustained control of all other potential wildlife hosts, including possums, for a considerable period of time. Second, the instantaneous rate at which ferrets acquire *M. bovis* infection (and the way in which this changes with ferret population density) should be determined. This is important because it would explain the most likely means by which a young ferret in any given population is likely to acquire the disease. This can only be done through longitudinal studies of ferret populations, again for a considerable period of time. Finally, there are few data on the rate of disease-induced mortality in ferret populations in New Zealand, yet understanding when and how quickly ferrets die from the disease could lead to better management of ferret populations, and better models of disease transmission in this species.

Several aspects of the ecology of ferrets also warrant further investigation. For example, is it possible to maintain an intact but Tb-free resident population of adult ferrets, thereby repelling invading juvenile ferrets (who may be transporting Tb) during the crucial autumn dispersal period? Do dispersing juvenile ferrets use "low population density" as a criterion for settling in a new location and, if so, how do current control operations affect ferret settlement patterns (and transport of Tb)? Do ferrets remaining in an area after ferret control operations have higher reproductive rates and, if so, does the consequent rapid increase in population density provide a means for ferret-to-ferret transmission of bovine Tb? A better understanding of these aspects of ferret ecology may help to reduce rates of disease transmission among ferrets and from ferrets to other wildlife and to domestic stock.

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