Bovine Tuberculosis infections of ferrets, stoats and feral cats in Otago, New Zealand.

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ABSTRACT

Twenty-one cattle farm properties around the Otago region in the South Island of New Zealand were surveyed for Tb prevalence in ferrets (*Mustela furo*), stoats (*M. erminea*) and feral house cats (*Felis catus*) during 1993 and 1994. As of May 1994, one thousand and eleven predators have been necropsied.

Bovine tuberculosis (*Mycobacterium bovis*) was not found in any of the predators from non-endemic areas. Tb prevalence in endemic Tb areas is 17.2% for ferrets (n=516), 4.2% for stoats (n=24), and 1.2% for cats (n=204). Peak prevalence of 66.7% occurred at one property where 18 ferrets were trapped.

There was a significant difference (c^2; $p<0.001$) between prevalences of adult (22.4%) and juvenile (2.5%) ferrets for the months of January to April. More male ferrets (22.8%) were infected with Tb than females (13.6%) (c^2; $p=0.013$).

A significant positive correlation was shown between the ratio of rabbit abundance over ferret abundance to ferret Tb prevalence (Multiple Regression: $p=0.020$). Adult ferret Tb prevalences rose 32.2% from winter (April - September) to summer (October - March) (Multiple regression: $p=0.014$).

The majority of lesions in ferrets were in lymph nodes, particularly the mesenteric nodes (57%). This site of infection suggests that ferrets may be ingesting infectious material, and once infected are capable of passing Tb organisms out into the environment through faeces. Seven percent of infected ferrets had lung lesions. "Open lesions" (capable of discharging *Mycobacterium* into the environment) were found in two ferrets and one cat, so it is conceivable that these predators are not simply "dead-end" hosts.

High prevalences and REA biotyping results from Table Hill, Milton suggest that ferrets may be capable of maintaining Tb within their own populations. There was no transmission of *M. bovis* to the ferrets from the original deer herd, or the experimentally infected herd or from the only tuberculous possum found.

Our results demonstrate widespread Tb infections of ferrets are associated with infected cattle herds. The relatively high abundance of ferrets and the high prevalence of Tb, when coupled with their ability to discharge *M. bovis* into the environment, also give cause for concern that ferrets contribute directly to the Tb problem in cattle. This suggests a multi-species management strategy is overdue for Tb containment. However, more research and especially direct experimental evidence to test for ferret involvement is still needed.

INTRODUCTION

Bovine tuberculosis (*Mycobacterium bovis*) is a bacterial disease brought to New Zealand with the first cattle in 1840 (Hickling 1991). Although cattle (*Bos bovis*) are the natural host, the disease infects most other mammalian species, including humans. Beginning in the 1940's a cattle test and cull programme achieved marked reductions in the incidence of Tb in the national herd, so that by the 1960's there was growing optimism that the disease could be eradicated (Hickling 1991). In the late 1960's, however, it became apparent that Tb had spread to wildlife populations. These wildlife reservoirs have been frustrating eradication progress ever since (Animal Health Board 1986).

Brush-tailed possums (*Trichosurus vulpecula*) have been considered the major wildlife reservoir of bovine tuberculosis in New Zealand (Efford 1991). Certainly there is overwhelming evidence that tuberculosis in possum populations is self-sustaining (O'Hara 1986), and that they contribute to cattle infection rates. Other wild animals might also spread Tb to new areas and infect domestic stock, but this has not yet been proven. Equally, there are no studies that categorically eliminate a role for wild animals other than possums in spreading and/or maintaining the disease.

Although Tb infections in cats (*Felis catus*) and ferrets (*Mustela furo*) had been noted for many years they were considered to be unimportant targets for control to achieve Tb mitigation (Allen 1991). This conclusion was based on the relatively low prevalence of the infection, and because their Tb lesions were apparently internal with no discharge (Ryan 1989, Allen 1991). Infections have been recorded in stoats (*Mustela erminea*) and weasels (*Mustela nivalis*) (Allen 1991), but little is known about the role of these animals as carriers of Tb. A ferret has been found to carry *Mycobacterium avium* in the United States (Schultheiss and Dolginow 1994).

Concerns developed in 1991-1992 over the role and importance of small mammalian carnivores (ferrets, stoats, and cats) in contributing to the Tb cycle because:-

- possum control operations were not achieving a reduction of Tb in domestic stock in some areas,
- possums were at low abundances, or sometimes absent, in some Tb trouble spots (therefore suggesting another feral animal was involved).
- tuberculous infections in cats and ferrets were detected in various sites around New Zealand.

Prior to this study, high Tb prevalences had been recorded amongst ferrets in two areas.
Three out of the five ferrets caught in 1992 during a possum maintenance control operation at a research farm at Table Hill, Milton (20 km south of Dunedin) were infected with Tb (K. Waldrup *pers. comm.*). *M. bovis* was cultured from four out of 109 ferrets caught in the McKenzie Basin in 1991-1992 (Walker *et al* 1992).

Our research set out to investigate Tb prevalence in a much larger sample of predators from throughout the Otago region. This was considered to be the first step to evaluate the need, or otherwise, for experimental tests of whether or not predator control might assist Tb containment. Our studies alone could not prove whether predator control is necessary or cost effective. However if further research and experimental management seems justified, we sought to advise when and where such control efforts might provide the most useful return, and to advise how best to perform such investigations.

If these predators are important Tb transmitters, then we need to know a lot more about determinants of their distribution and density. A wider understanding of predator/prey relationships will be invaluable in order to predict effects of control operations and any subsequent effect on Tb prevalence.

Our studies are still in progress, so this paper can only provide preliminary results to speed guidance of managers and researchers combating the Tb problem. From the present study, approximately 95% of the collected animals have been necropsied. We are still waiting on culture results from the Central Animal Health Laboratory at Wallaceville. More detailed analysis and interpretation of complete data set will be published elsewhere as soon as possible.

RESEARCH OBJECTIVES

Our specific research objectives were to:-

1. survey the prevalence of bovine tuberculosis in feral/wild populations of feral house cats, ferrets and stoats (to establish the status of these animals as carriers of Tb, and to target the species that we most need to research and control).

2. determine whether there is a correlation between cattle reactor rate and the prevalence and/or abundance of Tb infected ferrets (any such correlation would provide circumstantial evidence for transmission of Tb between cattle and predators).

3. compare the prevalence of Tb in different age, sex, and reproductive segments of the populations (to suggest routes of Tb spread, optimum times for control etc).

4. determine ecological predictors (rabbit abundance, altitude, vegetation characteristics) of predator abundance, predator guild, and Tb prevalence in predators (these would allow us to suggest where predators may most be contributing to the Tb problem, and so where preliminary control experiments should be directed).

5. determine whether these predators discharge Tb organisms back into the environment (and so potentially might spread the disease to other wild animal vectors and/or to domestic cattle and deer).

METHODS

A stratified random choice of sample areas was used, with the primary stratification being MAF Quality Control's cattle reactor rates for each property. Some properties were also chosen that are considered to be 'Tb free' from non endemic*[1] areas. Such properties had no history of Tb, and were at least 10 km from any property that had Tb.

Twenty-one properties were sampled in this Tb prevalence survey. The aim was to have 5 traplines per property. Each trapline was approximately two kilometres in length (~20 traps per trapline). The minimum number of traps was 80 but the aim was for approximately 100 traps. Traps were set for at least 10 consecutive days. The Tb survey commenced in late January 1993 and ended February 1994.

Study animals were caught by leg-hold 1/2 inch Soft-jaw Victor in open sets. The trap was buried under a fine layer of soil, and was secured by a large pin or waratah. Each trap was baited by rabbit meat, which was changed at least every three days. Bait was hung from a hook on a trap surround (a triangular-shaped tin structure that was used to standardise each trap and to direct the animal onto the trigger plate of the trap).

Captured animals were immobilised by an intramuscular injection of ketamine with xylazine. One part of 10% xylazine was mixed with 10 parts ketamine (100mg/ml), and the dosage based on ketamine concentration. The dose rate was approximately 0.2ml for a stoat (approximately 67mg/kg of ketamine), 0.4-0.5ml for a ferret (approximately 50mg/kg of ketamine) and 1-1.5ml for a cat (approximately 30mg/kg of ketamine). While immobilised a blood sample was obtained by cardiac puncture. Blood was allowed to clot overnight, and then serum was separated and frozen. If there were any external signs of Tb lesions, a swab was taken for culturing. The animal was euthanased using an overdose of ether, and transported back to the lab for necropsy. A Tb necropsy was performed on all animals. The Tb necropsy involved removing and weighing each lymph node and scoring condition. Body organs were also examined by palpation. All lesions were submitted to the Animal Health Laboratory at the Invermay Agricultural Centre for histopathological examination. All samples suggestive of tuberculosis were submitted for culture at the Central Animal Health Laboratory at Wallaceville.

A general necropsy was also performed on each animal which involved measurements of condition, fat deposits and reproductive organs. Skulls, baculums (penis bone), testes, ovaries and digestive tracts were removed. In the future, this information will be written up into papers on the general ecology of mammalian predators in New Zealand.

Rabbit abundance was assessed at all trapsites using 'Gibb scores' that measured pellet abundance (Gibb *et al* 1969). Mouse and rat abundance were assessed by standard rodent traplines (Fitzgerald 1978) of 3 days duration at all trapsites. Predator abundance was indexed from the "number trapped per 100 trap nights".

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* An Tb endemic area is an area where Tb has entered the wildlife population (Hickling 1991).

This research project was also assisted by the collection of animals through other means. Anita Middlemiss and Hiltrun Ratz of the Zoology Dept, University of Otago, trapped predators for their conservation-orientated research projects and provided the bodies for necropsy. AgResearch, MAF livestock officers, and other Zoology students also contributed to the sample sizes (mainly with collection of animals killed on roads). DoC officers contributed animals trapped for conservation purposes, especially stoats from the Otago Peninsula. These additional samples helped the prevalence survey but were chosen for reasons and in places that do not relate to the Tb issue. Similarly we do not have the associated measures of habitat and prey abundance to explore the predictors of predator abundance in these additional sites.

RESULTS

1. Overall prevalences of bovine Tuberculosis infections.

A total of 1011 predators have been necropsied (Table 1), but no Tb infections were found in predators from non endemic areas (Table 2). This difference in prevalence from endemic (17.2%) and non-endemic areas (0%) is highly significant (Mann Whitney U test on prevalences for different properties; p= 0.0107).

The following results will exclude those samples from non endemic areas.

There were no significant differences between ferret abundance and whether the Tb status was endemic or Tb free using a Mann-Whitney U test.

Table 1. The number of ferrets, cats and stoats examined in the Otago region in March 1994, and their Tb status according to histopathological (histo) examination and culture.

Species	CAT	FERRET	STOAT
Total # necropsied	204	715	92
# histo +ve	2	91	1
% histo +ve	1.0	12.7	3.2
# culture +ve	2	74	1
# culture pending	0	14	0
# culture -ve	0	3	2

Table 2. The number of ferrets, cats and stoats necropsied from endemic and Tb free areas, and their Tb status according to histopathological (histo) examination and culture. Animals were excluded that did not have exact location information.

	CATS		FERRETS		STOATS	
	endemic	Tb free	endemic	Tb free	endemic	Tb free
# necropsied	140	56	516	175	24	46
# histo +ve	2	0	89	0	1	0
% histo +ve	1.4	0	17.2	0	4.2	0
# culture +ve	2	0	73	0	1	0
# culture pending	0	0	13	0	0	0
# culture -ve	0	0	3	0	0	2

Ferrets had a much higher prevalence(17.2%) than did stoats (4.2%) and cats (1.4%) caught in endemic areas.

Remaining analyses in this paper will be for ferrets only.

2. Relationship between ferret Tb prevalence and cattle reactor rates.
Cattle Tb prevalence was calculated by the number of reactors in 1992 and 1993 and dividing by the herd size. No relationship was found between cattle Tb prevalence and ferret Tb prevalence on different properties (Table 3). Nor was there a relationship between cattle reactor rates and the abundance of Tb infected ferrets.

3. Tb prevalences in different age and sex segments of the ferret population.
Male ferrets were aged using baculum weights. The cut-off point for discriminating between juveniles and adults was determined as 0.8 gm by examining frequency distributions of baculum size in each month and discerning modal shifts as young of the year are recruited to the population. For female ferrets, during the months of juvenile emergence (Dec-March), an adult female was one that was lactating. After April, this method was not effective, so all females were considered to be adult if they were caught after April. These aging criteria are crude and preliminary. A Chi-Square test found that there was a significant difference ($p<0.001$) between prevalences of adult (22.4%) and juvenile (2.5%) ferrets for the months of January to April.

A significantly higher proportion of male ferrets (14.8%) were infected with Tb than with females (9.5%) infected (Chi-Square, $p=0.029$).

4. Ecological predictors of ferret abundance and Tb prevalence.
Ferret abundance increased significantly between the months of February and May (Fig 1; one factor ANOVA p= 0.0081).

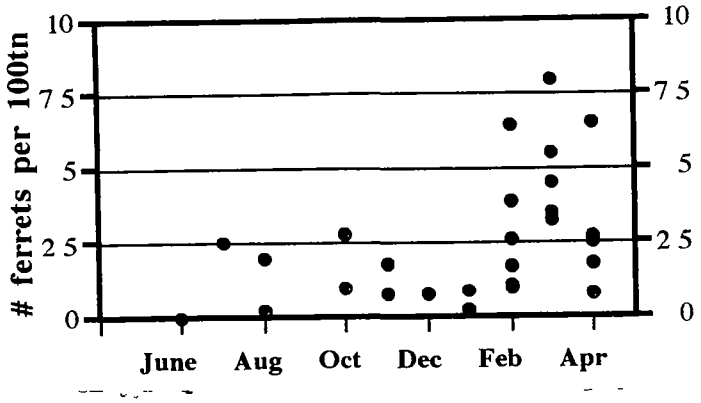

Fig 1. Seasonal changes in relative abundance of ferrets with juveniles included. Ferret abundance is expressed as the number of ferrets caught per 100 trapnights (tn). Each dot represents the combined results for all traplines on a single property.

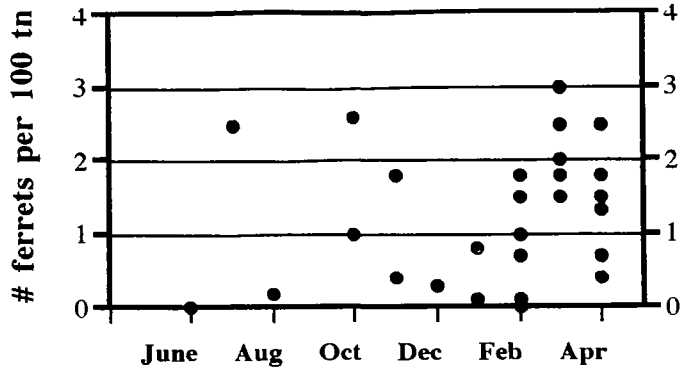

Fig 2. Seasonal changes in ferret abundance excluding juveniles. Key as for Fig 1.

When the juvenile ferrets were removed from the analyses (Fig 2.) there was no significant seasonal change in the observed ferret abundance.

Table 3. Results of Multiple Regression models of adult ferret Tb prevalences (calculated from sample sizes of over eight ferrets as predicted by ferrets per 100 trapnights, Gibbs score, % cattle reactors and season (as a dummy variable ie. 0= winter, 1= summer).

VARIABLE	Coefficient ± SE	P value
Ferret abundance	-4.99 ± 3.00	0.1307
Rabbit abundance	-9.01 ± 6.22	0.1811
Cattle reactor rates	28.11 ± 14.55	0.0854
Season (winter vs summer)	32.23 ± 10.63	0.0142

Adult ferret abundance was not significantly related to rabbit abundance (Table 3). There was no significant relationship between ferret abundance and ferret Tb prevalence (Table 3)

Tb prevalences of adult ferrets increased 32.3% from winter (April - September) to summer (October - March) (Table 3; Multiple regression; p=0.0142). Juvenile ferrets were excluded from the population, as well as prevalences that were calculated from less than eight adult ferrets. No seasonal relationship was found between Tb prevalences including juveniles.

A multiple regression model showed that there was a significant positive relationship between the ratio of rabbit abundance over ferret abundance and the Tb prevalence of ferrets (Table 4; p= 0.0292).

Table 4. Results of a Multiple Regression model of ferret Tb prevalences (calculated from sample sizes of over eight ferrets) as predicted by the ratio of ferrets per 100 trapnights/Gibbs scores, cattle reactor rates and season (as a dummy variable ie. 0= winter, 1= summer).

VARIABLE	Coefficient ± SE	P value
Season (winter vs summer)	0.0647 ± 9.0548	0.8337
Cattle reactor rates	1.9344 ± 7.4637	0.9932
Rabbit abundance/ferret abundance	9.954 ± 4.1296	0.0292

5. Bovine Tuberculosis lesions in ferrets.

Of the 91 histo-positive ferrets, 57% of the infected ferrets had lesions in the mesenteric lymph node (not necessarily the only node or body organ infected);43% were in nodes other than the mesenteric. Forty-seven percent of lesions were single node infections, 34% involved lesions in two lymph nodes, and 18% of infections could be called generalised (three or more lesions in lymph nodes and body organs). Lung infections were detected in six ferrets, and one liver infection was found. "Open lesions" were detected in two ferrets; in the inguinal node and the other in the popliteal node. Another ferret was found to have a pus-filled sinus associated with a ruptured popliteal node.

6. DNA restriction endonuclease (REA).
REA biotyping was performed on eight ferrets, one cat, one stoat, and one red deer (*Cervus elaphus scoticus*) from Table Hill, Milton. The results found that there were two very closely related strains of *M. bovis* present. Seven ferrets, one cat and the stoat shared the same strain; one ferret, one deer, one possum and the other cat carried the other. The deer from which Tb was biotyped was from the original herd that was on the property before the Infected Deer Farm was set up in August 1992. This original herd was subsequently removed from the property, and now the only stock remaining since August 1992 are the experimentally infected deer that are confined within a possum-proof enclosure, and which carried a third strain.

Therefore seven out of the eight ferrets that have been REA biotyped shared the same strain of *M. bovis*, and this was a different strain to that found in the original deer herd, the experimentally infected deer herd and the only tuberculous possum found.

From regular trapping sessions at Table Hill, Milton, it was found that eleven out of forty one ferrets (27%) were infected with Tb. Regular possum control operations in the same area found that comparable prevalence levels in possums were extremely low at 0.55% (1/240).

DISCUSSION

Feral cats, ferrets and stoats all carry bovine Tb in the survey area, but ferrets have by far the highest prevalence. Sample sizes are low for stoats in endemic areas so their prevalence estimates should be treated cautiously. The results from the Tb survey indicate that ferrets are carrying Tb throughout the endemic area in Otago. A good coverage was achieved and it seems that the ferret Tb problem is widespread albeit accentuated in certain areas.

Only samples that were judged to be 'typical of tuberculosis' by histopathological examination were sent to Wallaceville for culturing. No non-lesion animals were cultured for Tb, so additional infected ferrets with no clinical signs of the disease were probably present in the area. Morris *et al* (1993) found possums free of visible lesions to harbour *M. bovis*. The prevalence levels detected in this study are probably underestimated. Upcoming analysis of samples by an ELISA blood test may pick up additional early infections and those infections with no clinical signs.

The abundance of ferrets is also generally higher than that of cats in the Otago region (H. Moller *et al*, unpublished data). This conclusion suggests that amongst the small mammalian carnivores, it is the ferrets that pose the greatest potential threat to Tb control in the Otago region. Other behavioural and epidemiological differences may intervene in ways that mean that risk to cattle is not directly proportional to disease prevalence and/or abundance of the different predator species. However, our results suggest that ferrets should be targeted as the first priority for management and research for Tb control.

Seven out of sixteen properties surveyed had Tb prevalences of over 30% in ferrets. This may suggest that ferrets may cycle the disease quite readily within their own population, or that infected ferrets live for a long time.

There were no infected predators found in non endemic areas which indicates that there is a broad scale spatial correlation between Tb infections in predators and cattle reactor rates. Questions arise whether this relationship is a cause or effect, the result of an interaction with infected possums, or a correlation driven by some unknown factor. Had we found Tb in predators outside endemic areas, we could have inferred that the disease was cycling independently in cattle and predators. However, the spatial correlation observed does not necessarily mean that Tb infections in ferrets promote Tb infections in cattle within endemic areas.

Ferret abundance is not greatly different between endemic areas and non-endemic areas, so the observed absence of Tb in non-endemic areas is unlikely to result from density dependant factors.

The fact that there was no correlation between ferret Tb prevalences and cattle Tb prevalences amongst properties within endemic areas supports a hypothesis that Tb is self sustaining in ferret populations. Once Tb enters the ferret population it may cycle quite independently to cattle reactor rates. However, cattle reactor rates may be too crude and coarse a measure to give an accurate estimation of cattle Tb prevalence, or the continued culling of infected cattle may mean that the recent reactor rates do not estimate risk of giving Tb to the ferrets (or vice versa). Accordingly, our preliminary analyses should not be taken to infer that flow-rates of the disease in cattle and ferrets are not linked.

It is not surprising to find that adult ferrets have a higher prevalence than juvenile ferrets, but it is interesting to find that more male ferrets have Tb. Hickling (1991) found that Tb was more prevalent in male possums, especially immature males. Male mustelids greatly expand home ranges in the breeding season and can travel large distances to seek mates (King 1989). These social and spatial changes may facilitate the spread of Tb, and could be responsible for the seasonal rise in infection levels we observed in spring and summer.

Autopsy records showed that around the breeding season, the majority of ferrets had open wounds - presumably inflicted through fighting and mating. Mating in mustelids is prolonged and vigorous, and often involves males biting females - usually in the neck region. This time of contact may be an obvious opportunity for disease transmission to take place. Seasonal patterns in prevalence may be due to stress; improved survival of infected ferrets; or delays before earlier infections are expressed and detectable. Another explanation for the rise in adult Tb prevalences from winter to summer could be that there were young adult ferrets present that have not had time to develop clinical signs of infection. More sophisticated aging techniques and the application of a blood test may help address this issue in the future. Replication and a more detailed study of these seasonal fluctuations may provide valuable leads on the rates of transmission and survival, etc.

Ferret abundance measures showed the expected increase in late spring when juvenile emergence occurs, but adult ferret abundance did not show any seasonal trend. Ferret abundance was not correlated with rabbit abundance. The measures of rabbit abundance may not accurately represent the numbers present, especially if rabbit control operations have been carried out. The relatively short breeding season of rabbits in rabbit prone districts may hinder the development of stable predator-prey relationships (Gibb and Williams 1990), so for Central Otago properties a direct correlation between ferret abundance and rabbit abundance may not be expected.

Gibb, Ward and Ward (1978) observed a predator-prey cycle; in which predator numbers lagged behind rabbit numbers. Such lags mean that at any one time, there will not be a direct relationship between rabbit numbers and ferret numbers, and that the relative abundance measures based on relatively slow decaying pellets may be particularly misleading.

There was also no relationship between ferret abundance and Tb prevalence in ferrets but a significant relationship was found between ferret Tb prevalence and the ratio of rabbit abundance to ferret abundance. These correlations could indicate the degree of stability of the predator-prey relationship, diet shifts, condition and susceptibility to disease, survival time of diseased ferrets, the emergence of juveniles that cause low prevalences, or several other important parameters in the Tb epidemiology and/or ecology. Tracking the reason for this relationship may now provide valuable leads on factors influencing Tb spread and control. Preliminary research and predator control for Tb containment should target rabbit prone areas since this is where prevalence was highest in our study.

Ferrets have been found with open lesions that indicate that they are capable of shedding *Mycobacteria* into the environment. A feature of the pathology of infected ferrets has been the very large numbers of acid-fast bacilli in the lesions (de Lisle *et al* 1993). These facts suggest that ferrets could transmit Tb onwards to other animals and therefore cannot be assumed to be dead-end hosts. The crucial and unanswered question, however, is how often does transmission occur from ferrets to domestic stock or to other potential wildlife vectors. Fifty seven percent of ferrets had lesions in the mesenteric lymph node and so it could be that these animals are discharging Tb from their bodies through faeces, as has been found to occur with badgers (Wilesmith 1991). A small proportion of ferrets were found to have lung infections, so aerosol contamination is also possible.

The presence of open lesions and very high prevalences of Tb suggests that ferrets could be capable of spreading Tb onwards to other animals and domestic stock. Unless further REA typing show that generally ferrets carry different Tb from those appearing in stock, we recommend that ferrets be assumed to be capable of spreading Tb to stock and/or other wildlife vectors. Further research is required on Tb epidemiology in ferrets, ferret ecology, and control are needed to test this assumption. The risk of assuming ferrets to be dead-end hosts may be much greater than the risk of wasting expenditure on unnecessary control programmes and research.

Whether ferrets are "spillover hosts" or "maintenance hosts" is very much in question. As defined by Morris *et al* (1993) a maintenance host is one that can maintain infection in an area in the absence of cross-transmission from other species of domestic or wild animals. A spillover host is one that becomes infected when the challenge level is relatively high, but are not capable of maintaining infection within their own population. The only information we have available to address this question is from the infected deer farm at Table Hill, Milton. Seven out of eight ferrets whose infection were REA biotyped had a different strain of *M. bovis* to the original deer herd, the experimental deer herd and the only infected possum found. It seems that the ferrets there are maintaining the infection within their own population without cross-transmission from domestic stock, or possums immediately present around them. We cannot draw any conclusions from this data about whether ferrets are spreading Tb back to stock because the only stock at Table Hill are the experimentally infected deer. They are not reliable indicators for a variety of reasons

(ie. access to stock is restricted, the deer are already experimentally infected and predators are continually culled from the vicinity, etc).

Even if ferrets are proven not to be maintenance hosts, they might still spread Tb to cattle. Their control would therefore would be warranted.

In conclusion, our research has shown several causes for concern that ferrets may be contributing to Tb infections in domestic stock. Tb was widespread, and was associated with infected cattle herds within the endemic area of Otago. Ferret abundance is relatively high compared to other feral animal vectors in some places, and the Tb prevalence in ferrets was much higher than previously envisaged. Open lesions and mesenteric node lesions suggest that ferrets are capable of discharging Tb into the environment. However, more research, and especially direct experimental evidence for the value of ferret control for Tb containment is warranted (before there is large scale and persistent expenditure on ferret control). Experimental ferret control programmes, better and cheaper control techniques, and more research on predator ecology are urgently needed.

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