

**Inference on the host status of feral ferrets (*Mustela furo*) in New Zealand for *Mycobacterium bovis* infection**

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## Abstract

This thesis is about making inference on the host status of feral ferrets in New Zealand for *Mycobacterium bovis*, the aetiological agent of bovine tuberculosis. The central question addressed is whether the rate of intra-specific transmission of *M. bovis* among ferrets is sufficient for the disease to persist in ferret populations in the absence of external, non-ferret sources of infection (inter-specific transmission). The question is tackled in three parts—firstly using model selection to identify suitable models for estimating the force of *M. bovis* infection in ferret populations; secondly applying statistical hypothesis testing to the results of planned manipulative field experiments to test the relationship between *M. bovis* infection in brushtail possums and that in ferrets; and thirdly using modelling to estimate intra-specific disease transmission rates and the basic reproductive rate ( $R_o$ ) of *M. bovis* infection in ferrets.

The model selection approach clearly identified the hypothesis of oral infection related to diet was, as modelled by a constant force of infection from the age of weaning, the best approximation of how *M. bovis* infection was transmitted to ferrets. No other form of transmission (e.g., during fighting, mating, or routine social interaction) was supported in comparison. The force of infection ( $\lambda$ ) ranged from 0.14 yr<sup>-1</sup> to 5.77 yr<sup>-1</sup>, and was significantly higher (2.2 times) in male than female ferrets.

Statistical hypothesis testing revealed transmission of *M. bovis* to ferrets occurred from both brushtail possums and ferrets. The force of *M. bovis* infection in ferrets was reduced by 88% ( $\lambda=0.3$  yr<sup>-1</sup> vs.  $\lambda=2.5$  yr<sup>-1</sup>) at sites with reductions in the population density of sympatric brushtail possum populations. A smaller decline in the force of infection resulting from the lethal cross-sectional sampling of the ferret populations was also demonstrated.

The modelling approach estimated the basic reproductive rate ( $R_o$ ) of *M. bovis* infection in ferrets in New Zealand to vary from 0.17 at the lowest population density (0.5 km<sup>-2</sup>) recorded to 1.6 at the highest population density (3.4 km<sup>-2</sup>) recorded. The estimates of  $R_o$  were moderately imprecise, with a coefficient of variation of 76%. Despite this imprecision, the  $R_o$  for *M. bovis* infection in ferrets was significantly less than unity for all North Island sites surveyed. Hence it is inferred ferrets are spillover hosts ( $0 < R_o < 1$ ) for *M. bovis* infection in these environments. That is, *M. bovis* infection will progressively disappear from these ferret populations if the source of inter-specific transmission is eliminated. The estimates of  $R_o$  for *M. bovis* infection in South Island ferret populations were above one (the level required for disease establishment) for a

number (5/10) of populations, though the imprecision made it impossible to ascertain whether  $R_o$  was significantly greater than one. The estimated threshold population density ( $K_T$ ) for disease establishment was 2.9 ferrets  $\text{km}^{-2}$ . It is inferred that, given sufficient population density ( $>K_T$ ), the rate of intra-specific transmission of *M. bovis* among ferrets is sufficient for the disease to establish in ferrets in the absence of inter-specific transmission. In these areas, ferrets would be considered maintenance hosts for the disease. Active management (e.g., density reduction or vaccination) of ferrets would be required to eradicate *M. bovis* from ferret populations in these areas, in addition to the elimination of sources of inter-specific transmission, particularly brushtail possums.

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