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 (λ) ranged from 0.14 yr^{-1} to 5.77 yr^{-1}, 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% (λ=0.3 yr^{-1} vs. λ=2.5 yr^{-1}) 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^{-2}) recorded to 1.6 at the highest population density (3.4 km^{-2}) 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 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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