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Disease transmission between and within species, and the implications for disease control

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Summary

1. Disease transmission can occur between and within species for diseases with multiple hosts. If these diseases are undesirable, for economic or health reasons, then the relative importance of each type of transmission should be determined. Bovine tuberculosis (Tb) caused by *Mycobacterium bovis* is one such undesirable disease.

2. In parts of New Zealand, *M. bovis* infection is highly prevalent in feral ferret *Mustela furo* populations, and there is concern they may be acting as a reservoir of infection for domestic livestock, similar to the role played by brushtail possums *Trichosurus vulpecula*. We undertook a manipulative large-scale field experiment to test for and quantify interspecific transmission of *M. bovis* from brushtail possums to feral ferrets, and intraspecific transmission of *M. bovis* within feral ferret populations.

3. Age-specific prevalence data obtained from cross-sectional surveys was modelled to estimate the effect of experimental reductions in possum population density on the force of *M. bovis* infection in ferrets. A simple analysis estimated the force of *M. bovis* infection in ferrets to be reduced by 88% at sites previously subjected to a possum population density reduction. A more detailed analysis, incorporating ferret survey data from both before and after possum population density reduction and controlling for the effect of ferret sampling, estimated that possum population density reduction decreased the force of *M. bovis* infection in ferrets by 29% at sites of high ferret density, and 88% at sites of low ferret density, compared with experimental control sites.

4. There is clearly substantial possum-to-ferret transmission of *M. bovis*, and controlling possum populations is the logical first step to managing *M. bovis* infection in ferret populations, especially at sites with low ferret density. Intra-specific transmission is virtually absent in low density ferret populations though evident at higher densities.

5. *Synthesis and applications.* These results have management implications for other multiple-host diseases around the world, such as bovine tuberculosis and rabies. Control of within-species transmission may not be as effective for disease control as a reduction in between-species transmission. The management decision should be based on empirical estimates of the magnitude of each form of transmission.

Key-words: bovine tuberculosis, epidemiology, modelling, *Mustela furo*, *Trichosurus vulpecula*

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Introduction

Many wildlife/pathogen systems involve multiple wildlife hosts, and determining their role is one of the main issues in the study of diseases and vertebrate pests (Hone 1994). For example, wildlife are considered of greater importance if the rate of intraspecific transmission is sufficient for the maintenance of disease in the absence of external, interspecific transmission – in which case they would be considered maintenance hosts. Quantifying and possibly controlling interspecific transmission of disease is of importance for the health of humans (e.g. bubonic plague in Europe arising from a reservoir of *Yersinia pestis* in Norway rats *Rattus norvegicus* (Berkenhout 1769; Keeling & Gilligan 2000)). It is of importance also for domestic animal

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health (e.g. bovine tuberculosis infection in New Zealand cattle arising from a reservoir of *Mycobacterium bovis* (Karlson & Lessell) in brushtail possums *Trichosurus vulpecula* (Kerr) (Caley *et al*. 1999; Ramsey *et al*. 2002) and possibly ferrets *Mustela furo* L., and diseases such as leptospirosis (Caley & Ramsey 2001)); and wildlife health (e.g. 'apparent competition' (Holt 1977) in Britain between the ring-necked pheasant *Phasianus colchicus* (L.) and the endangered grey partridge *Perdix perdix* (L.) arising from the shared nematode *Heterakis gallinarum* (Schrank) (Tompkins *et al*. 2000); cattle and wild ungulates in the Serengeti and the spread of rinderpest (Dobson 1995)).

Where sympatric wildlife species are infected with disease, quantifying intra- and interspecific transmission rates enables the disease host status of different species to be determined. Indeed, a precursor to contemplating widespread (and often expensive) pathogen control measures (e.g. culling, vaccination and non-lethal density reduction) in wildlife is to demonstrate that inter or intraspecific transmission is actually occurring. Uncertainty in estimates of disease transmission rates may result in considerable controversy surrounding management actions, as seen for transmission of *M. bovis* from Eurasian badgers *Meles meles* (L.) to cattle in Britain (Clifton-Hadley 1996; Krebs *et al*. 1998). Controversies of this type are not helped by the great challenge that estimating disease transmission rates either within or between vertebrate wildlife hosts presents (McCallum, Barlow & Hone 2001). Note however, that lack of conclusive data on rates of interspecific transmission appears to be no barrier to exploratory modelling of the system (e.g. Smith *et al*. 2001).

The simplest experimental test for interspecific transmission is to quantify the effect of controlling disease in one wildlife host on the incidence of the disease in a second wildlife host. In one such study, McInerney, Small & Caley (1995) quantified changes in the prevalence of *M. bovis* infection in feral pig *Sus scrofa* (L.) populations following the control of *M. bovis*-infected feral water buffalo *Bubalus bubalis* (L.) populations in the floodplain habitats of Northern Australia. Likewise, Caley *et al*. (1999) inferred a causative link between *M. bovis* infection in brushtail possums and livestock – reducing the density of *M. bovis*-infected brushtail possums reduced the incidence of *M. bovis* in sympatric cattle herds. In Ireland, reducing the density of Eurasian badgers reduced the incidence of *M. bovis* infection in sympatric cattle herds (O'Mairtin *et al*. 1998b). A similar, though larger-scale, manipulative experiment is presently underway to quantify interspecific transmission rates of *M. bovis* from Eurasian badgers to cattle in Britain (Krebs *et al*. 1998). Laurenson *et al*. (2003) inferred that reducing mountain hare *Lepus timidus* L. densities reduced sheep tick *Ixodes ricinus* L. burdens on red grouse *Lagopus lagopus scoticus* Lath, and eventually the prevalence of louping-ill virus.

In many circumstances controlled experiments are unworkable (Tompkins *et al*. 2000), especially those of a manipulative nature, and rates of interspecific transmission may be estimated by modelling data collected from unmanipulated systems or observational experiments. This could be statistical modelling, whereby models of disease transmission are fitted to observed incidence or prevalence data to estimate interspecific transmission parameters. For example, Begon *et al*. (1999) estimated the rate of transmission of cowpox virus between sympatric populations of bank voles *Clethrionomys glareolus* (Schreb.) and wood mouse *Apodemus sylvaticus* (L.) populations in Britain by fitting transmission models to longitudinal data on disease prevalence and population density of both species. Although Begon *et al*. (1999) did not attempt to test critically whether interspecific transmission was in fact occurring, model selection procedures (e.g. Akaike's Information Criterion, Burnham & Anderson 1998) could be used to compare the fitted models with and without interspecific transmission, as a way of making inference on whether interspecific transmission occurs. This approach is considered strong inference by some (e.g. Burnham & Anderson 2001), particularly for ecological studies. Alternatively, mathematical modelling may be used to estimate inter- and intraspecific transmission rates. For example, Rhodes *et al*. (1998) used a mathematical model to argue that rabies would not occur in side-striped jackals *Canis adustus* (Sundevall) in Zimbabawe without significant interspecific transmission from domestic dogs associated with human settlements.

This study aimed to test experimentally whether there is evidence of interspecific transmission of *M. bovis* from possums to feral ferrets *Mustela furo* (L.) in New Zealand. Infection of livestock and wildlife with *M. bovis* is a major economic issue in New Zealand. Many wildlife species, including possums and ferrets, are hosts of *M. bovis* in New Zealand, and there is concern that feral ferrets are acting as a source of *M. bovis* infection for cattle. Wildlife managers want to know if population control of ferrets is necessary and useful. Oral consumption of infected carrion/prey is the most strongly supported hypothesis for the transmission of *M. bovis* infection to feral ferrets (Caley & Hone 2002). However, it is not clear what the source of this infection is, and recent work (Ragg, Mackintosh & Moller 2000) has demonstrated that ferrets will readily scavenge the carcasses of ferrets, identifying the potential for intraspecific transmission. The most parsimonious working hypothesis, however, is that the force of *M. bovis* infection in ferrets is influenced by the density of sympatric brushtail possum populations (Caley & Hone 2002). That is, there is a significant amount of interspecific transmission of *M. bovis* from possum populations. Here we explore the most parsimonious working model, namely a two-host one-pathogen model (possum/ferret/*M. bovis*); with emphasis on manipulating the population density of sympatric possum populations (cf. other possible prey items of ferrets). A

manipulative experiment is described where the population density of possums was reduced, and changes in the force of *M. bovis* infection (λ) in ferrets were quantified and compared with sites where possum population density was left unchanged. Transmission of *M. bovis* from ferrets to possums is unlikely, as possums are predominantly infected via the aerosol route (Jackson *et al*. 1995) and the pathogenesis of *M. bovis* infection in ferrets shows they are unlikely to excrete aerosols (Lugton *et al*. 1997). The analysis tests for an experimental effect of reducing the population density of possums on the force of *M. bovis* infection in feral ferret populations, thus demonstrating interspecific transmission. It also addresses whether there is intraspecific transmission of *M. bovis* in ferret populations by estimating the effect of lethal ferret sampling (akin to culling) on the force of *M. bovis* infection in feral ferret populations. Similar time-dependent epidemiological models have previously been fitted to age-specific disease data of humans to look for time-dependent changes in the force of infection, arising from changed epidemiological circumstances (treatments), for example hepatitis A in Europe (Schenzle, Dietz & Frosner 1979), and toxoplasmosis in England (Ades & Nokes 1993). Anderson & Trewhella (1985) modelled changes in the force of *M. bovis* infection in badgers arising from badger removal operations in the Gloucestershire area. The force of *M. bovis* infection in badgers was estimated to decrease from 0·28 year^{-1} to 0·12 year^{-1} following badger removal operations, though the statistical significance of this decrease was not presented (Anderson & Trewhella 1985). The study also examines the general implications for disease control of such results.

Materials and methods

STUDY SITES

Cross-sectional surveys of *M. bovis* infection in feral ferrets were undertaken at nine sites in New Zealand (Fig. 1). Effective trapping area ranged from 15·5 km^2 to 61·2 km^2 ($\bar{x}$ = 38·7 km^2). All sites lie within areas where wildlife are considered to be infected with *M. bovis*, with *M. bovis*-infected possums recorded from seven of the nine sites, and nearby for the remaining two sites. It is therefore reasonable to assume that *M. bovis*-infected possum occurred in all sites. At all sites, DNA fingerprinting (Collins & de Lisle 1985) has revealed a REA (restriction endonuclease analysis) match between at least one of the strains of *M. bovis* found in ferrets and that found in possums. These data provide clear evidence of interspecific transmission between wildlife, though give no clue to what species are involved in the transmission (e.g. is transmission from possum-to-ferret or from possum-to-deer-to-ferret) or the direction of transmission (e.g. possum-to-ferret or ferret-to-possum). The domestic cattle testing regime at all sites is such that where cattle do occur, cattle-to-wildlife transmission of *M. bovis* can be considered negligible.

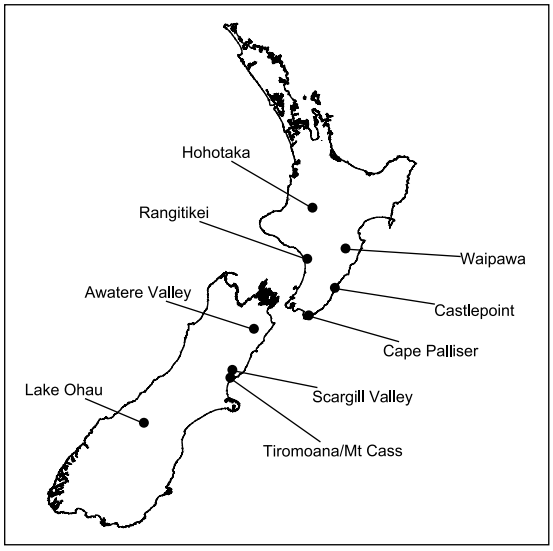

Fig. 1. Sites of cross-sectional surveys of *M. bovis* infection in feral ferrets in New Zealand.

EXPERIMENTAL DESIGN: CONTROL/INTERVENTION

The timing of experimental interventions (reduction in possum population density) and observations (lethal cross-sectional surveys of ferrets) at the study sites are given in Table 1. The predominant methods used to reduce possum population density included leg-hold trapping, poisoning using tree-mounted bait stations (compound 1080, encapsulated cyanide, broadifacoum), and aerially delivered cereal and/or carrot baits containing 1080. Further details are given below, including possible effects of possum control on ferret populations. These data may be analysed in many ways. Two approaches are used here. The first and simplest is a CI (control, intervention) design that compares estimates of λ from sites with no history of possum population reduction (experimental control treatment) with those from sites following a sustained reduction in possum population density (experimental intervention treatment). The Castlepoint, Cape Palliser, Awatere Valley, Scargill Valley and Lake Ohau sites made up the experimental control treatment, whilst Hohotaka, Rangitikei, Tiromoana/Mt Cass and Waipawa sites made up the experimental intervention treatment. The Castlepoint and Scargill Valley sites included in the experimental control treatment (Table 1) were subsequently subjected to the experimental intervention treatment, and so also form part of the BACI design (see below). For analysis of the CI design, only survey data collected from these two sites before the experimental intervention were included in the analysis.

EXPERIMENTAL DESIGN: BEFORE/AFTER CONTROL/INTERVENTION

The second approach follows a BACI (before vs. after, control vs. intervention) design (Green 1979), which

Table 1. Summary of the application of experimental interventions (X) and observations (O) of *M. bovis* infection in feral ferrets (following the notation of Manly 1992). The experimental intervention is the sustained reduction of possum density by lethal control ('press' cf. 'pulse'). Observations are cross-sectional surveys of the ferret population. Numbers in parentheses are sample sizes

	Site*								
Year	AWA	CP	CPR	HH	LO	RA	SCAR	TIRO	WAIP
Pre-94				X				X	X
1994		O (15)							
1995		O (2)		O (22)			O (78)†	O (19)†	
1996							†	†	
1997					O (72)	X	O (50)†	O (50)†	O (28)
1998		O (31)	O (19)	O (55)			O (33)†	O (39)†	
		X					X		
1999		O (27)	O (14)				O (58)		O (4)
2000	O (47)	O (14)	O (7)		O (40)	O (30)	O (62)		
2001	O (42)	O (8)	O (1)				O (85)		
2002	O (32)								

*HH, Hohotaka; RA, Rangitikei; CP, Castlepoint; CPR, Cape Palliser; AWA, Awatere Valley; SCAR, Scargill Valley; TIRO, Tiromoana/Mt Cass; LO, Lake Ohau; WAIP, Waipawa.
†Both the Scargill Valley and Tiromoana/Mt Cass sites were subjected to intensive ferret control during this period (1995–98), with 779 and 753 ferret removed, respectively (including those shown here). Further details are given by Caley, Morley & Thomas (1998).

inferentially is considerably stronger than a simple CI design. Four sites were used in the BACI design, these being Castlepoint (experimental intervention), Cape Palliser (experimental control), Scargill Valley (experimental intervention) and Awatere Valley (experimental control) (Table 1). These sites were originally chosen to be matched as practicably as possible for possum density (in the absence of experimental intervention), ferret density, and the force of *M. bovis* in fection.

Possum control over a 6400-ha area encompassing the Scargill Valley survey area started in winter/spring of 1998 using leg-hold traps, cyanide paste and encapsulated cyanide (Feratox®). Maintenance control to maintain the possum population at the lowered post-control population density was undertaken using encapsulated cyanide in 1999 and 2000. Possum control over a 6510-ha area encompassing the Castlepoint survey area started during the summer/autumn of 1998 using leg-hold traps and encapsulated cyanide, with further maintenance control in 1999. *Mycobacterium bovis*-infected possums had been found at the Awatere Valley, Cape Palliser, Scargill Valley and Castlepoint sites, and reducing the population density of possums at the latter two sites can reasonably be assumed to reduce the density of *M. bovis* infected possums (Caley *et al*. 1999), and hence density of *M. bovis*-infected possum carcasses. Indeed, possums macroscopically infected with *M. bovis* were removed during trapping at Castlepoint during 1998.

ESTIMATING POSSUM POPULATION DENSITY

Two indices of possum population density were obtained. The first was based on the number of possums caught incidentally in traps targeted at catching ferrets, using a modified version of Leslie's Removal Method (Seber 1982) modified to account for unequal sampling effort. The measure of abundance was the estimated number of possums per trap (rather than density). This was done as home-ranges of possums are in general small compared with the distance between traps. These data were collected from all sites, and provide a standardized index of possum population density enabling comparisons between surveys at all sites.

The second index of possum population density was based on the nationally recognized residual trap-catch (*RTC*) methodology (NPCA 2001). At the time of the study, the *RTC* method for monitoring changes in possum population density involved catching possums on lines of 20 soft-catch leg-hold traps, with the starting point of each line randomly selected within available possum habitat (stratified random sampling). For repeated yearly surveys, a new random sample of starting points was selected each year. The 20 traps in each line were spaced at 20-m intervals along transects which ran in a north–south direction. Traps were lured with a mix of flour (5 parts) and icing sugar (1 part), and set for 3 fine nights. The trap-catch statistic for each line was calculated as the average number of possums caught per trap per night. The *RTC* method was used to monitor changes in the population density of possums at Scargill Valley and Castlepoint resulting from possum control and to monitor natural fluctuations in the population density of possums at Cape Palliser and Awatere Valley. Possums captured at Scargill Valley and Castlepoint point during *RTC* monitoring were killed, whereas those captured at Cape Palliser and Awatere Valley were released. Possums captured during ferret trapping (see below) were treated similarly (humanely killed at experimental intervention sites and released at experimental control sites).

SAMPLING FERRET POPULATIONS

Ferrets were captured in Victor Soft-Catch® leg-hold traps (size 1.5) baited with fresh rabbit *Oryctolagus*

cuniculus L., hare *Lepus europaeus occidentalis* de Winton or domestic chicken meat. Traps were set at approximately 200-m intervals, usually over 5–10 nights. Suffering of animals was minimized by using rubber jawed (cf. steel jawed) traps, and checking traps as soon as practically possible each morning following setting. The use of leg-hold traps increases the efficiency of trapping, and enables traps to be set down burrows out of the way of livestock. Alternative methods of obtaining large samples, such as shooting, were not feasible logistically. Any non-target animals captured were released, and ferret and possums [if the experimental treatment so required] were humanely killed at the trap site where they were captured. All fieldwork procedures were approved by the Landcare Research Animal Ethics Committee (Approval Project no. 98/10/4), conforming to the legal requirements of New Zealand. Methods used to diagnose *M. bovis* infection and estimate ferret age are presented in Caley & Hone (2002). All ferret and possum carcasses were either incinerated or disposed of in covered offal pits.

Lethal cross-sectional sampling of ferret populations is essentially a form of population control. There has been no examination of the effect of ferret control on the force of *M. bovis* infection in feral ferrets. Lethal sampling of ferret populations infected with *M. bovis* should decrease the force of *M. bovis* infection in ferret populations by reducing the density of *M. bovis*-infected carcasses available to be scavenged by ferrets – assuming this is a significant mechanism of transmission. Ferret population density was estimated in each trapping session at each site using Leslie's Removal Method (Seber 1982), modified to account for unequal sampling effort. Possible changes in the population density of ferrets caused by sampling were assessed by regressing the natural logarithm of population density on time, and testing using a *t*-test (Sokal & Rohlf 1995) whether the instantaneous rate of increase (r) estimated as the slope of this regression (Caughley & Sinclair 1994) was significantly less than zero. The test is one-tailed, as we expected *a priori* that lethal ferret sampling should decrease ferret population density. This approach to hypothesis testing is in line with one of the recommendations of Krebs (2000), who argued there should be much more use of one-tailed tests in ecology, especially in the case of planned experiments.

ANALYSIS OF CONTROL/INTERVENTION DESIGN

For the experimental control sites, λ was estimated from age-specific prevalence data assuming a step-exponential model (Lee 1992), with λ modelled as being zero up until the age of weaning (at 1·75 months), and a constant thereafter (though allowed to differ between sites). The rate of disease-induced mortality (α), and the effect of gender were estimated during the model fitting procedure ($\alpha = 1{\cdot}4$ year^{-1}; 2·2 increased hazard for males), assuming these effects to be constant across all sites. This particular model was chosen during the model selection exercise of Caley & Hone (2002). For the experimental intervention sites, estimates of λ were made using the same model, although with the effect of sex and disease-induced mortality fixed at estimates from the experimental control sites. To avoid any effect of ferret sampling, only data collected during the first survey from each site was used for estimating λ used in this analysis. Differences in the means of λ (denoted $\bar{\lambda}$) and possum density between the treatments were compared using a *t*-test (Sokal & Rohlf 1995). Again the *t*-tests were one-tailed, as we hypothesized *a priori* that the possum control intervention treatment would reduce both possum population density and λ.

An important assumption of the analysis is that the experiment was not confounded (e.g. ferret population density reduced) by the method used to control possums. Two studies have reported on the effect of possum control on the population density of ferrets. Ground-laid, 1080-poisoned jam baits (note this is not a currently approved control method) resulted in significant mortality of resident ferrets (Moller, Showers & Wright 1996). In contrast, Caley *et al*. (1999) recorded no change in the year-to-year population density of ferrets at a site subjected to possum control using a variety of means, including 1080-jam baits (above ground), cyanide baits (above ground), aerially sown 1080-cereal baits, 1080-cereal baits in bait stations, broadifacoum cereal bait in above-ground bait stations, and leg-hold trapping. High ferret mortality was recorded following a rabbit poisoning operation using brodifacoum to target rabbits (Alterio 1996). Hence, ferrets appear highly susceptible to secondary poisoning from a chronic anticoagulant like brodifacoum. Five of the sites reported on here had been subject to possum control before being surveyed (Hohotaka, Scargill Valley, Waipawa, Rangitikei, Tiromoana/Mt Cass). Other than Hohotaka (for which no change in ferret population density was observed, see Caley *et al*. 1999), none of these sites used anticoagulants as the method for either initial or maintenance control of possums, before our ferret surveys. Hence, we assumed that possum control had not greatly influenced ferret population density at these sites, and tested this by comparing the population density of ferrets between the treatments using a *t*-test (two-tailed this time).

ANALYSIS OF BEFORE/AFTER CONTROL/INTERVENTION DESIGN

The first problem encountered when analysing this type of observation-intervention-observation data is that some animals spend time in both treatments. During the first sampling session, all animals captured have been subject to one treatment only, making estimation of λ up to this point relatively easy (Caley & Hone 2002). However, in subsequent sampling sessions, some individuals have been subject to either both treatments, or only the second treatment (estimation of λ is again

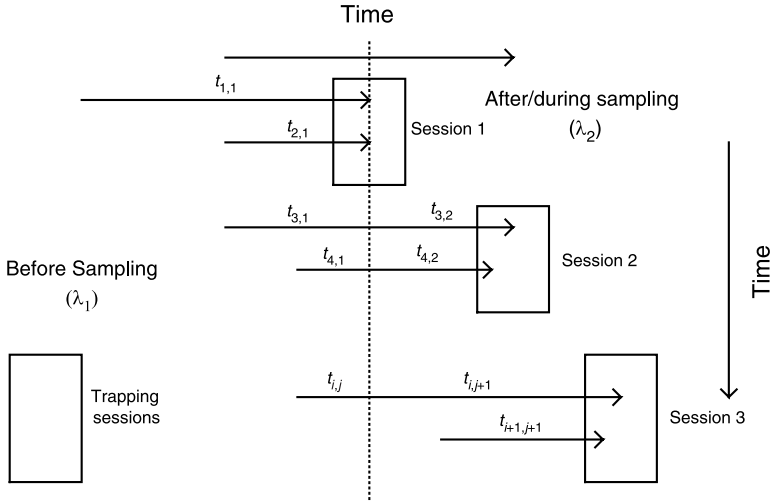

Fig. 2. A schematic representation of how sampled ferrets have spent different times in the 'sampling' treatments (demarcated by vertical dotted line) with force of infection λ_1 before sampling and λ_2 after sampling. For example, during Session 1, ferret number 1 spends a period $t_{1,1}$ during Treatment 1 (before sampling), whilst during Session 2, ferret number 3 spends a period $t_{3,1}$ during Treatment 1, and $t_{3,2}$ during Treatment 2 before capture. In general, $t_{i,j}$ refers to the time spent by the ith ferret in the jth treatment.

straightforward for these animals), as shown schematically in Fig. 2. Dealing with animals that have spent time in more than one treatment is problematical. One way around this problem is to exclude these individuals from the analysis. This approach is undesirable as it wastes information.

Alternatively, if the time period an animal has spent before capture is divided into two treatment periods, no ferret sampling (Treatment 1), and after the start of ferret sampling (Treatment 2), the prevalence of infection can be expressed as a function of the respective forces of infection in each treatment and the time spent by each individual in each treatment. More specifically, if the times spent in Treatment 1 and Treatment 2 are t_1 and t_2, respectively, then the probability (Pr) of being infected at capture can be expressed as:

Pr(infected at capture) = 1 – Pr(not infected during t_1)Pr(not infected during t_2).

An exponential model ignoring disease-induced mortality (setting $\alpha = 0$) is adequate for modelling the force of *M. bovis* infection in feral ferrets, and is much more tractable than the exponential model including disease-induced mortality (Caley & Hone 2002). This is the approach taken here. To avoid confusion, from now on we denote the force of infection estimated assuming no disease-induced mortality as λ'. Assuming a constant force of infection during each treatment period (λ_1' during t_1, λ_2' during t_2), the prevalence of infection at capture for ferrets that spend time in both treatment periods can be modelled as:

$$p(t_1,t_2) = 1 - e^{-\lambda_1' t_1} e^{-\lambda_2' t_2} \qquad \text{eqn 1}$$

Combining these results gives a model of the prevalence of infection in a system where the force of infection takes on two time-dependent values (Model 1).

$$p(t_1,t_2) = 1 - e^{-\lambda_1' t_1} \quad t_1 > 0,\ t_2 = 0$$

$$p(t_1,t_2) = 1 - e^{-\lambda_1' t_1} e^{-\lambda_2' t_2} \quad t_1 > 0,\ t_2 > 0 \qquad \text{(Model 1)}$$

$$p(t_1,t_2) = 1 - e^{-\lambda_2' t_2} \quad t_1 = 0,\ t_2 > 0$$

Expressions for age-specific prevalence in Model 1 are all nested within eqn 1, which makes calculations simple. Rearranging eqn 1 gives the prevalence of *M. bovis* infection as a function of the λs in the different treatments, and the time spent in each treatment (eqn 2).

$$\ln(1-p) = -\lambda_1' t_1 - \lambda_2' t_2 \qquad \text{eqn 2}$$

The aims of this study are to test whether λ_1' differs from λ_2', and to estimate the size of the effect. If sampling ferrets reduces the force of infection by an amount τ, then $\lambda_2' = \lambda_1' - \tau$. Substituting for λ_2' in eqn 2 yields the prevalence as a function of the unknown parameters λ_1' and τ (eqn 3).

$$\begin{aligned} \ln(1-p) &= -\lambda_1' t_1 - (\lambda_1' - \tau) t_2 \\ &= -\lambda_1'(t_1 + t_2) + \tau t_2 \\ &= -\lambda_1' a + \tau t_2 \end{aligned} \qquad \text{eqn 3}$$

Here, $a = t_1 + t_2$ is the age of the animal at capture and subsequent necropsy. This equation may be fitted to the data using a generalized linear model (GLM) with the response variable $q = (1 - p)$ distributed binomially with a logarithmic link function (Crawley 1993). An estimate of λ_1' is made by adding the term a to the model and estimating its regression coefficient. The magnitude and significance of τ is then estimated by adding t_2 to the model and estimating its regression coefficient. Testing whether τ differs from zero determines if $\hat{\lambda}_2'$ differs from $\hat{\lambda}_1'$. The appropriate test is one-tailed, as we expect τ to be positive. That is, we are testing the null hypothesis $\tau = 0$ against the working

hypothesis $\tau > 0$. To remain consistent with dietary-related transmission requires that the 'guarantee time', denoted g, when ferrets are suckling and hence not exposed to infection ($g = 1{\cdot}75$ months) is subtracted from either t_1 or t_2 (as determined by the individual circumstances of each individual).

The model used previously for two treatments (Model 1) can be extended to three treatments, to estimate the additional effect of possum control on the force of infection:

$$p(t_1,t_2,t_3) = 1 - e^{-\lambda_1' t_1} e^{-\lambda_2' t_2} e^{-\lambda_3' t_3} \qquad \text{eqn 4}$$

where λ_3' is the force of infection during the period t_3 that the animal is subjected to treatment 3 (here, a reduction in possum population density in combination with lethal ferret sampling). Let Δ be the reduction in λ' over and above that observed after the start of ferret sampling, hence:

$$\begin{aligned} \lambda_3' &= \lambda_2' - \Delta \\ &= \lambda_1' - \tau - \Delta \end{aligned} \qquad \text{eqn 5}$$

Substituting for λ_2' and λ_3' into eqn 4 and rearranging yields:

$$\begin{aligned} \ln(1-p) &= -\lambda_1'(t_1 + t_2 + t_3) + \tau t_2 + (\tau + \Delta) t_3 \\ &= -\lambda_1' a + \tau t_2 + (\tau + \Delta) t_3 \end{aligned} \qquad \text{eqn 6}$$

Here, a denotes the age of ferrets and eqn 6 can be used to estimate χ_1', τ and $(\tau + \Delta)$ using a GLM (again subtracting g from either a, t_2 or t_3 as appropriate). Where τ is estimated to be significantly different from zero, estimates of Δ and its standard error (assuming Δ and τ are independent) are then calculated as:

$$\hat{\Delta} = \widehat{(\tau+\Delta)} - \hat{\tau}, \text{ and} \qquad \text{eqn 7}$$

$$\text{SE}(\hat{\Delta}) = \sqrt{\text{var}\widehat{(\tau+\Delta)} + \text{var}(\hat{\tau})} \qquad \text{eqn 8}$$

Otherwise, estimates of Δ and its standard error were obtained by refitting eqn 6 without the t_2 term.

For each site, testing whether $\hat{\Delta}$ or $\hat{\tau}$ differed from zero was undertaken using a one-tailed t-test. Initial analyses of this data set (Caley 2001) ignored any sex effects on λ', however, subsequent analyses revealed that the sex ratio was unlikely to be independent of the time spent in different treatments, with the proportion of males in cross-sectional survey samples decreasing over time ($\chi^2 = 4{\cdot}3$, d.f. = 2, $P = 0{\cdot}12$). We accommodated for this by increasing the time spent by males in all treatments by a factor of 2·2, in line with the results of Caley & Hone (2002). Hence, the results here differ slightly to those presented by Caley (2001). A meta-analysis approach was used to combine the results from the different sites within the North Island (Castlepoint and Cape Palliser) and South Island (Awatere Valley and Scargill Valley). The probabilities arising from the t-tests (examining whether the treatments 'ferret sampling' or 'possum control' influenced λ') from the different sites were combined using the formulae presented by Fisher (1935) (cited by Underwood (1997)):

$$C = -2\sum_{i=1}^{k} \log_e P_i \qquad \text{eqn 9}$$

Here, P_i is the probability associated with the i^{th} site, and k is the number of sites. C is distributed as χ^2 with $2k$ degrees of freedom. Analyses were undertaken using the software R (Ihaka & Gentleman 1996) and GLIM4 (Francis, Green & Payne 1993).

Results

CONTROL/INTERVENTION ANALYSIS

The population density of possums was significantly ($t = -2{\cdot}2$, d.f. = 7, $P = 0{\cdot}013$, one-tailed test) and substantially (89% reduction) lower at experimental intervention sites ($\bar{x} = 0{\cdot}10$ possums trap^{-1}) than experimental control sites ($\bar{x} = 0{\cdot}89$ possums trap^{-1}). Likewise, the estimated force of *M. bovis* infection (with nonzero α) in ferrets was significantly ($t = -1{\cdot}9$, d.f. = 7, $P = 0{\cdot}049$, one-tailed test) and substantially (88% reduction) lower at experimental intervention sites ($\bar{\lambda} = 0{\cdot}30$ year^{-1}) than experimental control sites ($\bar{\lambda} = 2{\cdot}50$ year^{-1}) (Fig. 3). Ferret population density did not differ ($t = 0{\cdot}16$, d.f. = 7, $P = 0{\cdot}88$, two-tailed test) between experimental intervention sites (2·4 ferrets km^{-2}) and experimental control sites (2·2 ferrets km^{-2}).

BEFORE/AFTER CONTROL/INTERVENTION ANALYSIS: CHANGES IN POSSUM DENSITY

Possum control at Scargill Valley significantly reduced the possum trap-catch from 13·1% (95% CI 9·9–16·3%) pre-control (1998) to 1·2% (95% CI 0·5–1·9%) in post-control year 1 (1999), and 0·12% (95% CI 0·0–0·24%) in post-control year 2 (2000). Likewise, possum control

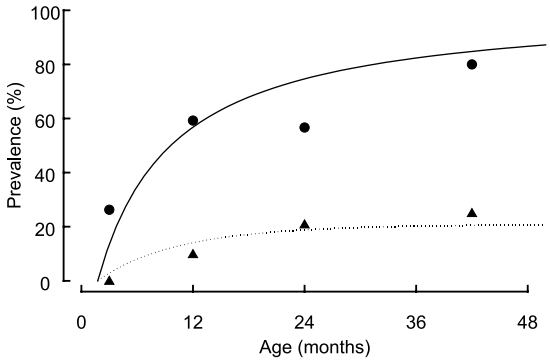

Fig. 3. Age-specific prevalence of *M. bovis* in ferrets from experimental control (no possum control) sites (solid circles and solid line) compared with experimental intervention (possum control) sites (triangles and dotted lines). Data have been pooled over sites, sexes and ages, hence fitted curves differ in values of λ from those presented in text.

at Castlepoint significantly reduced the trap-catch from 31·2% (95% CI 20·6–41·8%) pre-control (1998) to 0·7% (95% CI 0·0–1·4%) in post-control year 1 (1999), and 1·2% (95% CI 0·1–2·3%) in post-control year 2 (2000). At the Cape Palliser site where there was no possum control over the same period, the incidental catch rate of possums in traps targeted at ferrets was 9·9% in 1998, 8·4% in 1999 and 6·5% in 2000, indicating a slight decline in population density. Standard trap-catch monitoring of possums at Cape Palliser estimated the trap-catch to be 23·8% (95% CI 18·3–29·3%) in 1999 and 20·0% (95% CI 13·6–26·4%) in 2000. At the Awatere Valley site, the possum trap-catch decreased from 16% in 2000–9% in 2001, but the index based on possums caught in traps targeted at ferrets (using a much larger sample size collected over a larger area than the *RTC* estimate) increased from 1·5 possums trap^{-1} to 1·9 possums trap^{-1} over the same period. We conclude that no significant change in possum density occurred over this period.

BEFORE/AFTER CONTROL/INTERVENTION ANALYSIS: CHANGES IN FERRET POPULATION DENSITY

While ferret population density was reduced at the Scargill Valley site in response to the intensive control from 1995 to 1998, when the intensity of sampling was eased after 1998, the population density increased (Fig. 4). The estimated rate of increase (0·01 year^{-1}) over the duration of the study did not differ from zero (t = 0·01, d.f. = 5, P = 0·95). At the other South Island site used in the BACI analysis (Awatere Valley), no change in ferret population density was evident following the start of sampling (Fig. 4), though there were too few data points for regression analysis. In contrast, at the two North Island sites used in the BACI analysis, sampling led to a decline in ferret population density (Cape Palliser: r = −1·4 year^{-1}, t = −4·8, d.f. = 2, P = 0·02, one-tailed test; Castlepoint: r = −0·4 year^{-1}, t = −2·2, d.f. = 2, P = 0·08, one-tailed test). This was particularly so at the Cape Palliser site, where the ferret population steadily declined to near extinction as a result of the sampling (Fig. 4). There was no evidence that ferret population density was affected by possum control (Fig. 4).

BEFORE/AFTER CONTROL/INTERVENTION ANALYSIS: CHANGES IN λ′

At the two North Island sites (Castlepoint and Cape Palliser), λ′ was unaffected by ferret sampling, with $\hat{\tau}$ small biologically and statistically non-significant (Table 2; combining probabilities; χ^2 = 3·0, d.f. = 4, P = 0·55). In contrast, the effect of reducing possum population density ($\hat{\Delta}$) was both large (88·4% reduction) and statistically significant (P < 0·001; Table 2). These results demonstrate negligible intraspecific transmission but substantial interspecific (possum-to-ferret) transmission in these ferret populations.

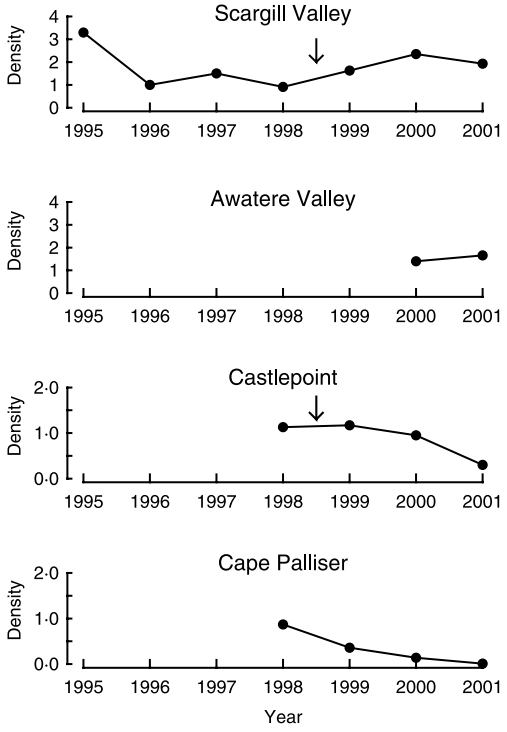

Fig. 4. Trends in the population density (km^{-2}) of ferrets at experimental intervention sites (Scargill Valley and Castlepoint) and experimental control sites (Awatere Valley and Cape Palliser). Arrows indicate when the experimental intervention (possum control) started. Note the difference in scale of the y axes.

At the two South Island sites (Awatere Valley and Scargill Valley), lethal sampling of ferret populations reduced $\hat{\tau}$ by biological meaningful amounts (31% and 51%, respectively). Combining the probability values using eqn 9 leads us to reject the hypothesis of sampling having no effect on λ (χ^2 = 11·0, d.f. = 4, P = 0·026). Again, the effect of reducing possum population density was statistically significant (Table 2), though the effect size was not as large (28% reduction) as for the North Island sites. It is inferred that both intraspecific and interspecific transmission were occurring in these populations.

Discussion

Estimating the extent of disease transmission between and within species is a prerequisite to the effective control of undesirable diseases, such as bovine Tb. A low level of within-species transmission and high between-species transmission may indicate that host population control needs to target the source of the between-species transmission to achieve effective disease control. Reductions in population density of a host species that is acquiring infection predominantly from another host species may produce little if any results. Alternatively, control of the source of between-species transmission will produce a greater reduction in disease prevalence. In the specific example in this study it is concluded that ferrets acquired more *M. bovis* from

Table 2. Estimates of the parameters λ_1' (force of infection before any treatment interventions), τ (additive effect [reduction] of ferret sampling) and Δ (additive effect [reduction] of possum control) from fitting the model $\ln(1-p) = -\lambda_1' a + \tau t_2 + (\tau + \Delta)t_3$, where a is the age of ferrets, t_2 is the time spent by ferrets in the ferret sampling treatment, and t_3 is the time spent in combined ferret sampling and possum control treatments. Note that figures are rounded

Treatment (site)	Parameter	Estimate (mth^{-1})	Standard error	τ	Probability (one-tailed)
Experimental Control	λ_1'	0·035	0·014	–*	–*
(Cape Palliser)	τ	0·003	0·026	0·117	0·45
Possum Control	λ_1'	0·22	0·05	–	–
(Castlepoint)	τ	0·02	0·45	0·05	0·48
	Δ	0·18	0·05	3·97	< 0·001
Experimental Control	λ_1'	0·091	0·023	–	–
(Awatere Valley)	τ	0·029	0·025	1·07	0·13
Possum Control	λ_1'	0·032	0·007	–	–
(Scargill Valley)	τ	0·016	0·009	1·9	0·03
	Δ	0·009	0·002	4·0	< 0·001

*Testing whether λ_1' is significantly greater than zero would be trivial, as by definition λ_1' must be greater than zero, given that infection has already been observed.

possums than from other ferrets. Indeed, most of the force of *M. bovis* infection observed in ferrets can be explained by their association with possum populations. This supports the contention of Lugton *et al.* (1997), and is consistent with the pattern in the prevalence of *M. bovis* in ferrets reported by Caley, Hone & Cowan (2001). As such, ferret control may be less effective than possum control in reducing the prevalence of *M. bovis* in livestock in New Zealand.

The experimental result supporting intraspecific transmission, at least in high density habitats, has major implications for our understanding of the epizootiology of *M. bovis* infection in ferrets. At low ferret population density there was little support for intraspecific transmission. Indeed, at the Cape Palliser site, the force of infection remained constant despite the ferret population being trapped nearly to extinction. In contrast, there appeared to be a significant level of intraspecific transmission at sites of higher ferret density. This indicates that intraspecific transmission is likely to be density-dependent, which fits with the classical prediction (Kermack & McKendrick 1927). Alternative explanations do exist for the observed reduction in the force of infection arising from lethal ferret sampling, including that the reduction in ferret population density arising from sampling resulted in higher *per capita* food availability of preferred prey (rabbits) and lower rates of scavenging. The data do not allow the relative support for these two hypotheses to be compared. A critical experiment could involve reducing the population density of ferrets by removing non-diseased ferrets only. This would require a highly sensitive and specific non-lethal diagnostic test, which currently does not exist.

We now return to issues of inference for interspecific transmission in host/pathogen systems mentioned in the Introduction. McInerney *et al.* (1995) inferred that a major reduction in the prevalence of *M. bovis* infection in feral pigs following a major reduction in the population density of *M. bovis*-infected feral water buffalo in the floodplains of the Northern Territory indicated that feral pigs were spillover hosts for *M. bovis* in this habitat. Their inference has been accepted without question, despite the experimental design used (simply a 'before' vs. 'after' comparison) being generally considered weak in terms of strength (reliability) of inference (Caughley & Sinclair 1994). Why this is so deserves further consideration. First, the experimental result was hypothesized *a priori* by Corner *et al.* (1981), based on a biological understanding of the host/pathogen system in question. This is deductive logic (most powerful). Second, the size of the effect was very large (there was no doubt that the response variable 'after' was different from the response variable 'before'). Third, the scale of the experiment was large, with considerable replication. Finally, there was considerable supporting evidence. For example, the density of buffalo had indeed declined following control (Freeland & Boulton 1990), demonstrating an experimental treatment (here the reduction in population density of *M. bovis*-infected buffalo) had been applied in nature as well as name. Hone (1999) provides further discussion on the need for supporting evidence in experimental studies. Caley *et al.* (1999) essentially used a BACI design to test the effect of reducing the population density of possums on the incidence of *M. bovis* in sympatric domestic livestock at one study site in New Zealand. Again the reported experimental result (of a causative link between *M. bovis* in possums and *M. bovis* in cattle) has received little or no opposition.

In contrast to the water buffalo/feral pig/*M. bovis* system or the possum/cattle/*M. bovis* system, the inference on whether *M. bovis* in British cattle is a spill over from infected badgers, and hence whether reducing the population density of badgers results in a lowered incidence of *M. bovis* infection in cattle has been subjected to considerable scrutiny and scepticism (Krebs *et al.* 1998). This is despite the experimental design of studies purporting to infer a link (e.g. Clifton-Hadley *et al.* 1995) being similar to the feral pig and possum studies. There was supporting evidence as to why badger culling could

potentially reduce the incidence of *M. bovis* in cattle, as Anderson & Trewhella (1985) described (though did not statistically test) a reduction in the estimated force of *M. bovis* infection in badgers following badger removal operations in the Gloucestershire area. Subsequently, stronger inference has come to light from Ireland on the role of badgers in transmitting *M. bovis* to cattle (O'Mairtin *et al*. 1998a,b). The intense scrutiny of the evidence supporting interspecific transmission of *M. bovis* from badgers to cattle, and hence management of badger populations is understandable, given the public affection for badgers (Neal & Cheeseman 1996). In contrast, affection for possums in New Zealand, or feral pigs in the Northern Territory is near to non-existent (P. Caley, personal observations). Clearly the level of scepticism given to the results of studies varies depending on the species involved.

In summary, this study has examined disease transmission between and within species using a combination of modelling and experimental manipulation. Evidence is provided of substantial between species transmission of *M. bovis*, and variable levels of within-species transmission. The main implication for disease control is that it may be more useful to reduce between-species transmission than within-species transmission, to reduce disease spread. The ferret-possum-Tb system may be similar to the ungulates-cattle-rinderpest system (Dobson 1995) and jackal-dog-rabies system (Rhodes *et al*. 1998), in that ferrets, like ungulates and jackals, need substantial between-species transmission to maintain disease in their populations.

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