

## Scientific Article

## Habitat-related prevalence of macroscopic *Mycobacterium bovis* infection in brushtail possums (*Trichosurus vulpecula*), Hohonu Range, Westland, New Zealand

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### Abstract

**AIM:** To identify broadscale habitat factors influencing the prevalence of macroscopic *Mycobacterium bovis* infection in brushtail possums (*Trichosurus vulpecula*) at a site in Westland, New Zealand.

**METHODS:** During 1973/74, 1989/90 and 1997, we undertook repeated cross-sectional surveys of *M. bovis* infection in a possum population on the Hohonu Range, Westland. Data were analysed to determine the influence of site-specific habitat characteristics (land form, aspect, slope, altitude), distance from forest-pasture margin and time since infection on the spatial and temporal prevalence of macroscopic *M. bovis* infection.

**RESULTS:** The prevalence of *M. bovis* infection was highest in 1973/74 (13.4%), compared with 1989/90 (3.1%) and 1997 (9.4%). The prevalence of macroscopic *M. bovis* infection was significantly influenced by habitat, as indexed by altitude and slope in this study site. Every 100 m increase in elevation was associated with a 29% decrease in the odds of infection, and every 10° increase in slope was associated with a 20% decrease in the odds of infection. For possums caught in the lowland podocarp forest (altitude 100–200 m, average slope=5.7°), the odds of infection were nearly 30-fold higher than those for possums caught in high-altitude hardwood forest near the tree line (altitude 900–1000 m, average slope=28°). Whilst the prevalence of disease fluctuated markedly between surveys, its broadscale spatial distribution changed little over time. Proximity to the forest-pasture margin had no significant influence on the prevalence of disease, once the effect of habitat was taken into account.

**CONCLUSION:** The prevalence of macroscopic *M. bovis* infection in possums was strongly influenced by habitat type, being highest in habitats that supported the highest density of possums, and lowest in habitats where population density was low. There was no evidence of progressive spread of *M. bovis* infection in possums into forest away from pasture-forest margins over the 24-year period of these surveys.

**KEY WORDS:** *Mycobacterium bovis*, brushtail possum, *Trichosurus vulpecula*, epidemiology, modelling, bovine tuberculosis

### Introduction

Brushtail possums are considered to be the most significant wild-life reservoir host of *M. bovis* (bovine tuberculosis, Tb) in New Zealand (Morris and Pfeiffer, 1995; Coleman and Caley, 2000). Continuing transmission of *M. bovis* infection from possums to cattle is the single greatest barrier to New Zealand achieving a bovine Tb-free status for its cattle and deer herds (O'Neil and Pharo, 1995). To date, non-selective culling has been the main tactic for controlling *M. bovis* infection in possums to reduce, or in some areas eliminate, the incidence of Tb in livestock (Caley et al, 1999). An understanding of the mechanisms by which *M. bovis* is transmitted among possums is needed for targeted control of the disease, rather than simply targeting possums *per se*.

Historical data suggested that the prevalence and hence per capita transmission of *M. bovis* infection in possums was highest in possum populations foraging on pasture adjacent to the forest edge. For example, a survey in the Hohonu Range in Westland in 1973/74 recorded 75% of all possums with macroscopic lesions within 50 m of pasture, a prevalence of nearly 20% (Coleman, 1988). Only 19% of infected possums were more than 1 km from pasture, the farthest being 4 km into the forest. Several hypotheses were proposed to explain the observed pattern of disease, namely: (a) possums interacted with and continued to acquire *M. bovis* infection from tuberculous cattle; (b) *M. bovis* infection had recently established in the possum population and was spreading progressively further into the forest, or; (c) the higher density and particular foraging behaviour of possums at the forest edge maintained a higher per-capita rate of disease transmission in this population compared with possums living further into the forest (the 'forest-pasture margin' effect) (Coleman, 1988).

The apparent forest-pasture margin effect could have a biological explanation unrelated to the forest edge *per se*, but resulting from a correlation between distance to forest-pasture margins and increasing elevation and changing habitat, as pasture clearance often stops at the edge of the more productive lowlands. Increasing elevation and changing habitat usually correspond to decreasing possum density (Coleman et al, 1980; Clout and Gaze, 1984), presumably as the physical environment becomes harsher (lowered temperatures, increased rainfall) and forest productivity decreases (shallower soils and lower soil nutrient levels).

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Tb           Bovine tuberculosis  
OR           Odds ratio

This study used the results of repeated cross-sectional surveys of the prevalence of *M. bovis* infection in a possum population in Westland, New Zealand, to examine these hypotheses about the epidemiology of the disease in possums. The area was selected in 1973 for the first major survey of the spatial patterns of *M. bovis* infection in a possum population (Coleman, 1988). At that stage, the site was chosen for its isolation, the presence of *M. bovis* infection in possums and cattle, and the absence of local possum control. The site was resurveyed in 1989/90 (Hickling, 1991) and again in 1997 (reported here). Since the 1973/74 survey, 2 factors have changed significantly: all cattle in the area have been subjected to yearly tuberculin testing, effectively removing them as a potential source of *M. bovis* infection for possums (Cousins et al, 1998) and; areas originally covered with pasture have become overgrown by regenerating scrub, such that the forest-pasture margin has moved substantially. This combination of circumstances enables re-examination of the influence of forest-pasture margin, *M. bovis*-infected livestock, and time since *M. bovis* infection became established in the possum population, along with other habitat and possum demographic variables, on the prevalence of *M. bovis* infection in possums and the distribution of infected possums within the study site. The identification of broadscale habitat correlates of *M. bovis* infection will help target management efforts to control *M. bovis* infection in possum populations more effectively.

## Materials and Methods

### Study area

The study area is located on the northern flanks of the Hohonu Range, immediately south of Lake Brunner, in central Westland. It is described in detail by Coleman (1988). To the north and north-west, the area is bounded by 2 beef-fattening farms on improved and rough pasture; in the centre is a formerly grazed clearing (Beehive Clearing) now covered with revegetating scrub. Both areas are bordered by mixed-hardwood forests on steeply dissected uplands varying from 100 to 1000 m in altitude. Forest canopy dominants include *Dacrydium cupressinum* and *Weinmannia racemosa* at low altitudes, *Metrosideros umbellata*, *W. racemosa*, and *Quintinia acutifolia* at mid-altitudes, and *M. umbellata*, *Libocedrus bidwillii*, *W. racemosa*, and *Griselinia littoralis* at high altitudes. The forest understorey also varies with altitude, and comprises roughly equal proportions of woody shrubs, pteridophytes, and organic litter.

Bovine Tb has been known in cattle in the area since the early 1970s. Official possum control began in 1974 when the entire study area was aerially sown with toxic bait (1080) in an attempt to eliminate transmission of *M. bovis* to livestock. Since then, the farm/forest margins have been ground baited with toxic bait in most years (D Rowling, West Coast Regional Council, pers. comm.), but no control has been carried out in the forest. Commercial possum hunting using cyanide and/or leg-hold traps occurred throughout the area in the 1970s to early 1980s, but has probably been uncommon since then because of persistent low pelt prices.

### Possum trapping

Possums were collected on the flanks of the Hohonu Range using cyanide poison bait stations or leg-hold traps lured with flour

and cinnamon essence. Poisoning or trapping sites were permanently located at 50 m intervals beginning in 1973/74. Trap lines began principally at the edge of the pasture grazed by cattle (then considered the likely source of the disease in possums) and ran up leading spurs (ridges) to end in alpine grassland. Possums were also collected daily along the edge of the road that dissects the study site, using leg-hold traps set at variable intervals at sites of recent possum activity (runs, faeces, or recent browse).

In 1973/74, all hill lines were baited with cyanide paste for 2 nights every 2 months, while all road-edge lines were trapped with leg-hold traps for 4–5 nights. In 1989/90 and 1997, the same sites at low-mid altitude on the hill and all sites along the road edge were trapped using leg-hold traps for 2–6 nights in each survey in spring. Sites at higher altitudes were again poisoned with cyanide paste. Sites further into the Hohonu Range were only re-trapped partially in 1989, and not trapped at all in 1997. The lines trapped in all surveys are numbered 1, 2, 3, 4, 5, 7, 8, 9 in Figure 1 in Coleman (1988).

All trapped possums were killed humanely. They, along with all poisoned possums, were tagged, their trap/poison site noted, and then removed to a central necropsy site. There, each animal's sex, maturity (determined from the size of the testes of males and the presence of a pouch in females), and bodyweight were recorded.

### Possum necropsies

The Tb status of possums was determined from the presence of macroscopic lesions resembling *M. bovis* infection in superficial axillary, inguinal, deep axillary, bronchial, and mesenteric lymph nodes, and in all major thoracic and abdominal organs (lung, liver, kidney, spleen and adrenal glands). Tb status was classified as suspicious if lymph nodes were enlarged, caseous or discharging, if there was consolidation in the lungs, or discrete white nodules were evident in abdominal organs. Tissue samples from suspect lesions were stored either frozen, for later bacteriology which was the primary form of diagnosis used in all surveys, or in 10% formalin for histopathology. For comparison among the 1973/74, 1989/90, and 1997 surveys, we calculated the prevalence of macroscopically *M. bovis*-infected possums from the number of possums with *M. bovis* infection evident macroscopically that were later confirmed by either bacteriology or histopathology, using only data taken from lines and sites trapped or poisoned in all 3 surveys. Our analyses ignored possums with non-visible lesions and hence undoubtedly underestimated the true prevalence of infection (Morris and Pfeiffer, 1995).

### Trap-site habitat data

At each trap or poison site, the altitude (m above sea level), distance to forest-pasture margin (m), angular aspect (direction of slope in degrees), landform (face, terrace, ridge, or gully) and slope (degrees above horizontal) were recorded in 1973. Distances from the forest-pasture margin were measured. Distances of some (though not all) trap sites from the forest-pasture margin changed between 1973 and 1997, as areas of pasture became overgrown with non-pasture species (e.g. bracken, *Pteridium esculentum*). The average increase in distance for all traps was 582 m (range 0–1750 m), though for many trap sites, distance increased substantially between surveys, especially for those traps around the original Beehive Clearing (mean=1600 m). The altitude of each trap site was calculated by marking the trap locations on a 1:50,000 topographic map and interpolating between the marked elevation contour levels. For the purposes of analysing whether

the disease was progressively moving further into the forest, the distance of trap sites from the forest-pasture margin defined in 1973 (distance73) was recorded against each trap site for all surveys

Trapping in this project in 1997 was undertaken with the approval of the Landcare Research Animal Ethics Committee.

### Statistical analysis

The data set comprised 1273 possums caught from 307 out of a possible 353 trap sites (Table 1); possums were not caught at all trap sites. Data from trap sites not trapped in all 3 surveys were excluded from analyses. Possums were trapped at sites ranging from 100–933 m in altitude, and on slopes ranging from 0–80° (most slopes were <35°). Distances from the original forest-pasture margin ranged from 0–3300 m, and from the current (1997) forest-pasture margin ranged from 0–3950 m.

The prevalence of macroscopic *M. bovis* infection was modelled as a function of the site habitat variables using logistic regression (Ahlbom, 1993). Data were initially modelled as binary, as the relatively small numbers of individual possums caught per site per survey (see Results) meant that very little grouping could be undertaken within demographic factors (see below). Hence model deviance could not initially be used as a measure of the goodness of fit of the model, or to test for over-dispersion (Collet, 1991). Subsequent to fitting the model to the data in binary form, the model was fitted to the data grouped over non-significant factors (see Results), enabling assessment of model fit. Analyses were undertaken using SPI US (MathSoft, Seattle, Washington, USA) and GLIM4 (Numerical Algorithms Group, Oxford, UK).

The demographic factors, sex (male or female), age (immature or adult), and survey (1973, 1989, 1997) were included in the model from the outset, as was the interaction between sex and age, as *a priori* information suggested their potential importance (Coleman, 1988; Pfeiffer et al, 1995). This study did not set out to examine demographic factors influencing *M. bovis* infection in possums *per se*, but has included them as a precursor to investigating other habitat variables of interest. For example, aspect has been hypothesized as influencing the abundance of possums, abundance postulated to be higher on warm sunny faces (Cook, 1975). The angular aspect data (aspect) was classified as northerly (316–45°), easterly (46–135°), southerly (136–225°), and westerly (226–315°). Land form was postulated to influence possum abundance through the preference of possums for dry ridgetops over damp gullies (Cook, 1975), and altitude was included as a proxy for habitat type and population density.

The changing forest composition with increasing altitude has a profound effect on possum density in this area of New Zealand, densities of possums in low altitude forest being an order of magnitude higher than those in high altitude forests (Coleman et al, 1980). To test the hypothesis that *M. bovis* infection was spreading progressively further into the forest, the interaction term time\*distance73 was included, where time was the time (in years) since the 1973/74 survey. The purpose of the variable 'time' was to index the time since *M. bovis* became established in the local possum population; 1973 was used as an estimate of establishment date, as the exact date at which infection became established was unknown. Interactions between other variables were not examined as there were no *a priori* reasons for doing so.

Initially, the full model, including all factors and interactions was fitted. The significance of each term (factor or interaction)

was examined using the deletion test (Crawley, 1993). Starting with the least significant, interactions between factors, then factors, were removed from the model one at a time. If the removal of the variable caused a significant increase in the model deviance, it was returned to the model, otherwise it was left out. The process was repeated until the model contained only significant terms (a minimal adequate model). A probability value of 0.10 was arbitrarily chosen for retaining terms in the final model.

We acknowledge some problems in our experimental approach, particularly as some of the hypotheses we are testing were generated as a result of the 1973/74 survey results (Cook, 1975; Coleman, 1988). Hence not all the hypotheses we have proposed are truly *a priori*, which potentially increases the chance of identifying spurious associations (Burnham and Anderson, 1998). We have tried to minimise the risk of this by only analysing those factors we consider to have biological plausibility.

## Results

### Non-significant model terms

The prevalence of infection was not influenced by distance from the forest-pasture margin ( $\chi^2=0.002$ ,  $df=1$ ,  $p=0.96$ ). Neither did the effect of distance from the forest-pasture margin vary significantly over time (time\*distance73,  $\chi^2=0.002$ ,  $df=1$ ,  $p=0.96$ ). However, distance was highly collinear with altitude for all surveys (1973/74,  $r=0.85$ ; 1989/90,  $r=0.84$ ; 1997,  $r=0.83$ ). Prevalence of infection was somewhat higher in males than in females (see below), regardless of age class (sex\*age,  $\chi^2=0.03$ ,  $df=1$ ,  $p=0.86$ ). Age had little influence on the prevalence of infection ( $\chi^2=1.4$ ,  $df=1$ ,  $p=0.24$ ), and the same was true for land form ( $\chi^2=1.4$ ,  $df=3$ ,  $p=0.7$ ) and aspect ( $\chi^2=4.8$ ,  $df=4$ ,  $p=0.3$ ).

### Significant model terms

Compared with the 1973/74 survey, the prevalence of macroscopic *M. bovis* infection was significantly lower in both 1989/90 and 1997 (Table 2, raw data are presented in Table 1). This was particularly so in 1989/90, when the odds of trapping infected possums were 80% lower than in 1973/74. However, the prevalence of disease had increased by 1997, the odds of infection then being only 30% lower than in 1973/74 (Table 2). The prevalence of infection was slightly greater for males compared with females (OR=1.34,  $Z=1.4$ ,  $p=0.09$ ). Each 100 m increase in elevation was associated with a 29% decrease in the odds of *M. bovis* infection, whilst each 10° increase in slope was associated with a 20% decrease in the odds of infection (Table 2). There was a moderate correlation between slope and altitude

Table 1. Summary of macroscopic *M. bovis* infection in possums from surveys of the Hohonu Range, Westland, New Zealand.

Year	Number necropsied	Number infected (%)
1973/74 <sup>a</sup>	499	67 (13.4)
1989/90 <sup>a</sup>	542	17 (3.1)
1997	233	22 (9.4)
<b>Total</b>	<b>1274</b>	<b>106 (8.3)</b>

<sup>a</sup> These data are a subset of the total survey extracted for comparison with the 1997 survey

Table 2. Odds Ratios (OR), 95% confidence intervals (CI), Z-statistics and probabilities from the logistic regression model<sup>a</sup> relating the prevalence of macroscopic *M. bovis* infection in possums from the Hohonu Range to temporal and environmental parameters.

Parameter	OR	95% CI	Z	p-value
SURVEY (1989/90)	0.2	0.1–0.3	-6	<0.001
SURVEY (1997)	0.7	0.4–1.1	-1.5	0.07
SEX	1.3	0.9–2.0	1.4	0.09
ALTITUDE <sup>b</sup>	0.7	0.6–0.9	-3.5	<0.001
SLOPE <sup>c</sup>	0.8	0.6–1.0	-1.7	0.048

<sup>a</sup> The full model is:  $\text{logit}(p) = -0.86 - 1.72 \cdot \text{survey}(1989/90) - 0.39 \cdot \text{survey}(1997) + 0.29 \cdot \text{sex}(\text{male}) - 0.23 \cdot \text{slope} - 0.35 \cdot \text{altitude}$  where  $\text{logit}(p)$  is the natural logarithm of the odds of being macroscopically infected with *M. bovis*

<sup>b</sup> Altitude is measured in 100's of metres above sea level

<sup>c</sup> Slope is measured in 10's of degrees

( $r=0.45$ ). Macroscopic infection was found in a possum at an altitude of 760 m in 1997; the next highest altitude at which an infected possum was caught was 701 m, in 1973/74. The observed prevalence of macroscopic *M. bovis* infection as a function of altitude (pooled over 100 m increments) is shown in Figure 1.

Major variations in the prevalence of Tb in possums were evident in relation to the combined influences of elevation and slope across the study site. For example, the modelled odds of infection in possums captured on the lowland terraces at an altitude of 100 m was nearly 16-fold higher than that of possums captured on terrace equivalents (benches) near the tree line at an altitude of 1000 m. Incorporating the effect of slope in the model increased these odds substantially, as the slope of sites in general increased with increasing altitude. For example, for possums caught in the lowland podocarp forest (100–200 m altitude, average slope=5.7°), the modelled odds of infection were nearly 30-fold higher than those for possums caught near the upper treeline (900–1000 m altitude, average slope=28°). However, ignoring differences between surveys, fitting the effect of altitude alone provided a good description of the change in prevalence (Figure 1).

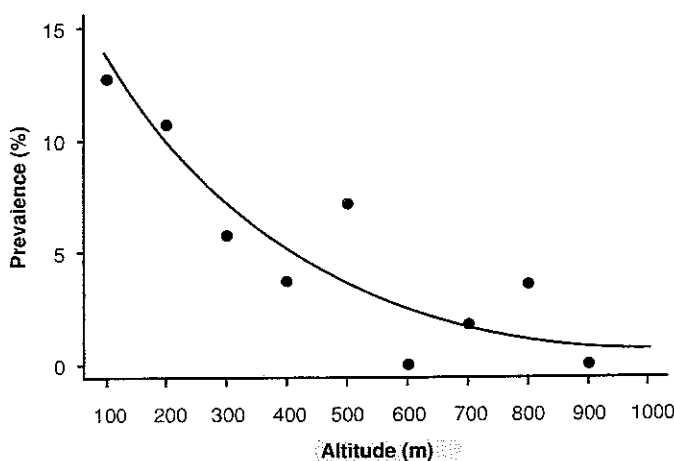


Figure 1. Mean prevalence of macroscopic *M. bovis* infection in possum populations on the Hohonu Range, Westland, as a function of altitude. Data from 1973/74, 1989/90 and 1997 surveys combined ( $n=1274$ ). Fitted line is  $\text{logit}(p) = -1.45 - 0.37 \cdot \text{altitude}$ , where  $\text{logit}(p)$  is the natural logarithm of the odds of being macroscopically infected with *M. bovis* and altitude is measured in 100's of metres above sea level.

If the slightly increased prevalence of infection for males was ignored and the data pooled across ages and sexes for each trap site, the model could be refitted (with factors: survey, altitude, and slope) with the response variable being binomial (rather than binary, although after pooling, many trap sites still only caught a solitary possum in any 1 survey). The resulting model revealed no evidence of over-dispersion (residual deviance=359.7,  $df=575$ ). A plot of standardised Pearson residuals against altitude revealed no major trend, although many of the residuals were  $>2$ , suggesting that at a trap-site level, significant lack of fit existed. Pooling the data into 100 m altitudinal increments revealed that a simple model could describe the data reasonably well (Figure 1). The most obvious deviation between the observed and fitted prevalence occurred at an altitude spanning 450–550 m, caused by a persistent cluster of infection on the ridge-top immediately behind Beehive Clearing.

In summary, the prevalence of *M. bovis* infection was highest in possums captured from low altitudes in areas of the flattest topography.

## Discussion

This study has identified possum habitat, as indexed by elevation and slope, and time of survey, as the major influences on prevalence of macroscopic *M. bovis* infection in possums in this study site. At the comparable Mt Bryan O'Lynn study site, about 25 km from Hohonu and with similar plant associations, possum densities were estimated to range from 25.4 per ha in low altitude forest, to 1.9 per ha in high altitude forests, in an apparently uninfected possum population (Coleman et al, 1980). Density was not directly measured in the current study, but a similar gradient of density with increasing elevation exists on the Hohonu Range. Based on catch rate (from poisoning) undertaken during the 1973/74 survey, possum density was 6 to 8 times greater in habitats of lowest altitude compared with those at highest altitude (Coleman, 1988). Hence, disease transmission appears to occur most frequently where local possum numbers are greatest, as identified by Coleman (1988).

Our analysis shows that the high prevalence of macroscopic *M. bovis* infection recorded in the possum population at Hohonu, occurred on or away from the forest-pasture margin, and with or without the presence of *M. bovis*-infected cattle. The reason the forest-pasture margin effect was rendered unimportant in our

present study was simple — a high prevalence of infected possums persisted at trap sites even after the forest-pasture margin (and the cattle that grazed on it) had moved several kilometres away. Hence, despite the high correlation between the distance to the forest-pasture margin and altitude (with the clear possibility for confounding), we are confident that the analysis is correct in identifying altitude (and associated changes to habitat and possum abundance) as the most appropriate proxy variable describing broad-scale variation in the prevalence of macroscopic *M. bovis* infection in possums at this site.

Our analysis also shows that macroscopic *M. bovis* infection has not spread further into the forest with the passing of time. Indeed, the broadscale spatial distribution of prevalence has remained reasonably constant since 1973/74, although the prevalence varied considerably between surveys. The factor(s) underlying the major fluctuation in the prevalence in macroscopic *M. bovis* infection between surveys is unknown. Control of the possum population undertaken between the 1973/74 and 1989/90 surveys may have contributed, as possum control decreases the prevalence of macroscopic infection (Caley et al, 1999). However, major fluctuations in the prevalence of *M. bovis* infection have been observed in another Westland possum population not subject to control (Coleman et al, 1999).

The highest prevalences of infection correlated with the habitats capable of supporting the highest densities of possums, which partially supports one of the hypotheses made after the original 1973/74 survey (Coleman, 1988). This is at odds with previous studies that have found no relationship between prevalence and possum density (Barlow, 1991), though Hickling (1995) since found local density to be an important predictor of *M. bovis* prevalence. General epidemiological theory predicts that for directly transmitted infectious diseases, the per capita rate of disease transmission and, hence, disease prevalence, should increase with increasing population density; indeed this appears to be the case for many infectious diseases of humans (Anderson and May, 1992). However, data for *M. bovis* infection in another wildlife species (the badger, *Meles meles*) are equivocal. For example, Cheeseman et al (1989) reported no correlation between the prevalence of *M. bovis* infection in badgers and the abundance of badgers, whereas Gallagher and Nelson (1979) had previously reported a significant relationship between density and prevalence of *M. bovis* infection in badgers from the same general area. *M. bovis* infection in badgers is most prevalent in southwest England where average badger densities are generally higher than elsewhere in Britain (Anderson and Trewhella, 1985), consistent with a relationship between host density and prevalence (Zuckerman, 1981). Crude regression analyses (such as those presented by Barlow in 1991) that examined relationships between disease prevalence and density may have been clouded by variation in prevalence arising from natural fluctuations, age of the local epidemic in relation to equilibrium prevalence, local variation in carrying capacity, and/or disease-induced mortality reducing local abundance, as described by Coleman et al (1999).

Based on the relationship we postulate between the prevalence of *M. bovis* infection and local population densities of possums, we suggest further studies are warranted that look, empirically, for threshold densities necessary for disease persistence within possum populations, as described for disease hosts in general by Anderson and May (1979), and May and Anderson (1979). Although we recorded several *M. bovis*-infected possums at reason-

ably high altitudes (>700 m) on the flanks of the Hohonu Range, where the possum population density was low, this does not necessarily demonstrate disease persistence at these densities or sites. Recent analysis of possum demography reveals that a high proportion of resident possums (particularly males) are immigrants, originating from areas up to 40 km distant (Cowan et al, 1996; Efford, 1998). Such animals may be responsible for the ongoing introduction of *M. bovis* infection from sites of high disease prevalence in the lowland forests to sites higher up the mountain. This is akin to the source-sink hypothesis (Dias, 1996).

An explicitly spatial model of *M. bovis* infection in possums, fitted to prevalence data along the density gradient, could provide empirical estimates of a threshold density, complimenting estimates of density thresholds from strategic modelling (such as that described by Barlow, 1996). The potential benefits of having threshold population density targets for disease eradication are substantial.

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