Extending and validating the Landcare Research Possum-TB Model

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Extending and validating the Landcare Research Possum-TB Model

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Summary

Project and Client

Landcare Research was contracted by the Animal Health Board from October 2010 to June 2012 to update the Landcare Research Possum-TB Model with improved parameter estimates and additional functionality for modelling possum populations at low density.

Objectives

To improve the credibility and utility of predictions produced by the Landcare Research Possum-TB Model (PossTB Model), by:

- Extending the model to simulate possum reaggregation and habitat edge effects

- Completing a formal sensitivity analysis of how well the PossTB Model performs at low densities

- Using existing and new data on possum movement patterns and TB disease dynamics at low densities to better parameterise POF simulations

Methods

- A possum reaggregation function was developed and coded into the PossTB Model along with a new method for allocating habitat-specific maximum long-run potential possum densities (carrying capacities = K) to forest/pasture edges or patches.

- Global sensitivity of the PossTB Model to key model parameters was assessed using the Fourier Amplitude Sensitivity Test (FAST) method. TB persistence was assessed at equilibrium and under a standard possum control scenario of an initial high-intensity lethal control (95% efficacy) followed by two maintenance controls (80% efficacy each), spaced 5 years apart.

- Recently published data on possum home range sizes, dispersal rates and distances, TB survival times, and the older Castlepoint data on pseudo-vertical transmission were reviewed with a view to reparameterising the PossTB Model

Main findings

- Making the modelled possums reaggregate following control to low densities (<0.2 possums/ha) resulted in more TB infections, presumably because these surviving possums now had neighbours to infect compared with an isolated possum with no neighbours. This resulted in higher TB prevalence. However, even these enhanced local densities are too low for TB persistence and the disease continues to drop out of the system over time. In other words, it appears that at the very low densities at which possums are isolated from their conspecifics, and are therefore driven to reaggregate, the resulting clusters of possums are still too small to sustain TB long term.

- Allowing edges of native forest with pasture or patches of native forest within pasture to have enhanced possum carrying capacity did little to alter the TB dynamics within

the modelled populations. This is because we scaled the disease transmission coefficient (*β*) for each scenario to generate an equilibrium TB prevalence of 2%, in effect keeping the product *βK* constant. In addition, the structural connectivity of the modelled landscape was the same (possum habitat patches were still in the same places, but the carrying capacity within these patches was increased) so spatial effects on contact and infection rates would be similar.

- Modelled TB persistence was most sensitive to the disease mortality rate *α*, followed by the pseudo-vertical transmission rate *p*, then the disease horizontal transmission rate *β*.

- New evidence of larger home ranges at low possum densities was used to refit the home range adjustment algorithm. This algorithm appears to be a better option than using a constant value of *σ* across a heterogeneous landscape.

- New data on possum survival times following TB-infection suggest much higher disease mortality rates *α* which would increase disease turnover but, as a counter to this, there appears to be a uniform latent period before the possums become infectious and disease-related mortality occurs. The same data suggest that the length of the infectious period is longer relative to the latent period, which argues for a higher pseudo-vertical transmission rate *p*.

Conclusions

- Predictions of the PossTB Model on the probability of TB persistence were highly sensitive to changes in the model structure and parameter values. Although, in a lot of cases, while we have identified changes that do have an effect, there is still not enough empirical data to parameterise these parameter values and thus justify changing them. For example the reaggregation algorithm showed a modest effect on TB persistence, but the controlling parameters (the threshold density for reaggregation to be stimulated, the probability of a possum staying put and the effect of distance to neighbouring possums on this probability, and the resulting dispersion of the group) are unknown.

- On the other hand, the sensitivity analysis has shown that while some parameters have a large influence on TB persistence at equilibrium, once control is applied and possum numbers are dramatically reduced the disease cannot be maintained regardless.

Recommendations

The AHB should:

- Consider further exploration of disease-induced mortality as the parameter most strongly affecting model predictions.

- Consider changing the model structure to include a latent class or at least introduce some kind of delay before individuals start dying of disease. The former option would require recoding but could be combined with the next recommendation.

- Consider contracting a professional programmer to recode the model and do some formal software testing.

- With Landcare Research, develop and instigate a versioning system for model changes, and find a more permanent solution for making the model available to AHB users. In

the meantime AHB users should check the ftp site regularly to download the latest version of the PossTB Model: ftp://ftp.landcareresearch.co.nz/Spatial%20Possum%20TB%20Model/.

For AHB users specifically, I recommend the following guidelines for using the model:

- *You will need to adjust the disease transmission rate β to make TB persist in your modelled landscape; this can be done using trial and error or the 'find beta' algorithm (the latter option is very time consuming).*

- *Use the reaggregation algorithm at a threshold density of <=0.2 possums/ha if you want to make conservative predictions (but bear in mind this will slow down processing time).*

- *Modifying carrying capacity (K) maps to produce enhanced possum density at forest edges or in remnant patches is probably not worth the effort.*

- *Use the home-range-adjustment algorithm ('non-linear contact rates' check-box) in preference to using a constant value for home range size (σ), which is only appropriate when the landscape is homogeneous.*

- *For initial simulations use the parameter values in Appendix 1 . Note that the parameters for home range adjustment, horizontal disease transmission and pseudo-vertical disease transmission have been changed from earlier versions.*

1   Introduction

Landcare Research was contracted by the Animal Health Board from October 2010 to June 2012 to update the Landcare Research Possum-TB Model with improved parameter estimates and additional functionality for modelling possum populations at low density.

2   Background

The new National Pest Management Strategy proposes the eradication of TB from large areas of New Zealand and this will rely heavily on controlling possum populations, the main wildlife vector of TB. Because landscape-scale experimental manipulations are not feasible, simulation models are being used to compare various possum control strategies and make predictions on TB persistence following control.

The Landcare Research Possum-TB Model (PossTB Model) is a spatially-explicit individual-based model that simulates the demographic, movement and TB transmission processes in a possum population (Ramsey & Efford 2005, 2010). It was developed originally by Dr Mark Efford, and subsequently extended by Dr Dave Ramsey, but as both developers are no longer employed by Landcare Research 'custody' of the model has transferred to me (Dr Mandy Barron). Although developed mainly as a research tool, it has now become the 'mainstream' model guiding TB management in New Zealand. In particular, it is being used to predict the likelihood of TB extinction in an area given the history of control and/or recent data on possum densities there. These model predictions are then used as the Bayesian 'priors' for the 'Proof of Freedom' (POF) calculations (Nugent et al. 2006, 2010). However, this, and other recent applications (e.g. response options modelling for the NPMS review; Nugent et al. 2008), requires simulation of possum populations and disease dynamics at low possum densities, in patchy habitats, and/or on the boundaries of control zones – far more complex scenarios than the current PossTB Model is able to simulate.

Critical processes not currently modelled but likely to affect predictions of TB persistence under these types of scenarios are: (1) possum distribution behaviour – how do possums respond to changes or discontinuities in the distribution of habitat resources and presence of potential competitors or mates?; and (2) transmission of TB between possums – this will be a function of the contact rates and mixing between possums described by (a), but is also sensitive to the disease transmission functions assumed and their parameter values, namely the horizontal transmission rate (β), the pseudo-vertical transmission rate (p), and the disease mortality rate (α).

This report has two components. Firstly, it describes extensions to, and refinements of, the PossTB Model that aimed to enable more realistic simulation of the above scenarios, particularly in relation to possum distribution and redistribution. I use two case studies to demonstrate the effect of those changes on simulated TB persistence. Secondly, I re-evaluate those parameters that the model predictions are most sensitive to, and, in light of new data, make suggestions for updating the default parameters used for POF simulations. A copy of the latest version of the model, along with its default parameters, is available on the ftp (File Transfer Protocol) site:
ftp://ftp.landcareresearch.co.nz/Spatial%20Possum%20TB%20Model/

3   Objectives

To improve the credibility and utility of predictions produced by the Landcare Research Possum-TB Model, by:

- Extending the model to simulate possum reaggregation and habitat edge effects
- Completing a formal sensitivity analysis of how well the PossTB Model performs at low densities
- Using existing and new data on possum movement patterns and TB disease dynamics at low densities to better parameterise POF simulations.

4   Amendments to the possum model

A possum reaggregation function was developed and coded into the PossTB Model. Also, to model possum abundance at forest edges or in remnant patches, a new method was developed for allocating habitat-specific maximum long-run potential possum densities (carrying capacities; *K*) to forest/pasture edges or patches. Guidelines for using the new functionality in the model are shown in italics.

4.1   Possum reaggregation function

I have taken a phenomenological approach to modelling congregation because the biological mechanisms involved in possum 'reaggregation' or 'clumping' after control are not yet known.

The reaggregation function can be implemented in the revised model by checking 'Activate' in the 'Re-aggregate possums surviving control' box on the 'Options' tab and specifying a threshold possum density (per hectare) at which to implement the reaggregation.

If these conditions (control to or below the threshold density) are met during a model run then the model loops through each of the remaining possums in the population and allocates them to an existing or new aggregation/group with the respective probabilities:

$$P(\text{possum } j \text{ joins group } i | \text{size group } i, \text{distance to group } i) = \frac{n_i \times d_{ij}}{\mu + N}$$

$$P(\text{possum } j \text{ founds new group}) = \frac{\mu}{\mu + N}$$

Where:

n_i is the number of possums in group *i*
d_{ij} is a distance weighting factor estimated as exp (−0.05 × distance between group *i* and possum *j*)
μ describes how often a possum 'chooses' to found a new group (i.e. has other possums shift to it, rather than the reverse)
N is the number of possums in the population currently allocated to groups.

Possum *j* is randomly allocated to a group based on a random draw from a multinomial distribution with the probabilities as described above except they were normalised (so they summed to one). If a possum is allocated to an existing group then its home range centre is relocated so that it is a random direction and a random distance (drawn from an exponential distribution with a mean of 90 m) from the home range centre of the group's founder possum, with the requirement that its new home range centre must be in possum habitat (*K*>0; otherwise a new location is drawn). If a possum founds a new group, it remains in its current location and other possums shift towards it.

If you leave the 'Suppress graphics' tick box on the 'Options' tab unchecked (not normally recommended as it slows processing down considerably) you can visualise the reaggregation of possums after a control operation.

4.2   Modelling edge effects

There is some evidence that possum densities in areas where native forest adjoins pasture, or in patches of woody vegetation surrounded by pasture, are higher than those in large tracts of native forest alone (Efford 2000). This is thought to represent, to some degree, use of the pasture by possums, but until now flat pasture was assumed in the model to have a near zero possum carrying capacity, including right up to forest edges. To capture this effect in the maps of possum carrying capacity (*K*-maps) used to drive the PossTB Model I modified the method used to generate these maps. This was done using the GIS software ERDAS Imagine v9.1, although it could probably also be accomplished in ESRI ArcMap.

Firstly, a new field was added to the LCDB2 raster attribute table (pixel size = 15 ×15 m) and was populated with values of '0' if the pixels represented exotic pasture (LCDB2 classes 40 & 41), '1' if they were native forest or deciduous hardwoods (LCDB2 classes 68, 69, 72–80), and '2' for all other classes. Secondly, a focal analysis was used to define edges between the three habitat types by passing a 5-pixel by 5-pixel moving window over the raster and assigning the minimum value to the focal pixel. Then native forest/pasture edges (1/0) were selected out of all of the edges identified in the previous step, to create a forest/pasture 'edge' raster. Lastly, an adjusted *K*-map raster was generated by overlaying the original raster with the edge raster and multiplying the default carrying capacity values by 2 if the pixels were on a forest/pasture edge or leaving them as the default value if they were not. The LCDB2 raster has a resolution of 15 m so the modified raster was degraded by a scalar of 3 to give a 45-m-resolution raster to make it comparable with the 50-m-resolution *K*-maps originally distributed with the PossTB Model. The result was an 'edge-enhanced' *K*-map with higher possum densities at edges than predicted in the original *K*-maps.

4.3   Model simulations

To assess the effects of these changes on model-predicted TB dynamics and the probability of TB persistence, the amended model was tested using two case studies: the Ashley Buffer Vector Control Zone and the West Karamea VCZ. Five hundred replicate simulations were run for each case study/aggregation/edge combination. For each replicate the model was run for 50 years with a burn-in period of 30 years, an initial 95% population reduction control in year 31, followed by 80% maintenance controls every 5 years thereafter (years 36, 41 & 46).

The Ashley Buffer VCZ comprised an area of 27 812 ha, of which 27 582 ha were considered to be possum habitat and the mean possum carrying capacity assessed over the entire habitable area was 2.3 possums/ha for the unadjusted-K scenario and 2.4 possums/ha for the edge-enhanced scenario (Figure 1).

The West Karamea VCZ was smaller at 15 403 ha (with 14 204 ha of possum habitat) but was characterised by a higher possum carrying capacity due to the greater prevalence of native forest, averaging 3.5 possums/ha over the entire habitable area for the unadjusted K scenario and 3.9 possums/ha for the edge-enhanced scenario (Figure 2).

Initial simulations without possum control were run to determine the value of the disease transmission coefficient (β) required to generate 2% disease prevalence at equilibrium possum density. These were $\beta = 0.760$ and $\beta = 0.755$ for the Ashley Buffer unadjusted and edge-enhanced scenarios respectively and $\beta = 0.390$ and $\beta = 0.383$ for the West Karamea scenarios respectively. All other model parameters were set to their usual default values. The threshold density for reaggregation was set to 0.2 possums/ha approximately equivalent to a 1.5–2.5% RTCI. The probability of TB freedom was estimated as the proportion of simulations where TB became extinct.
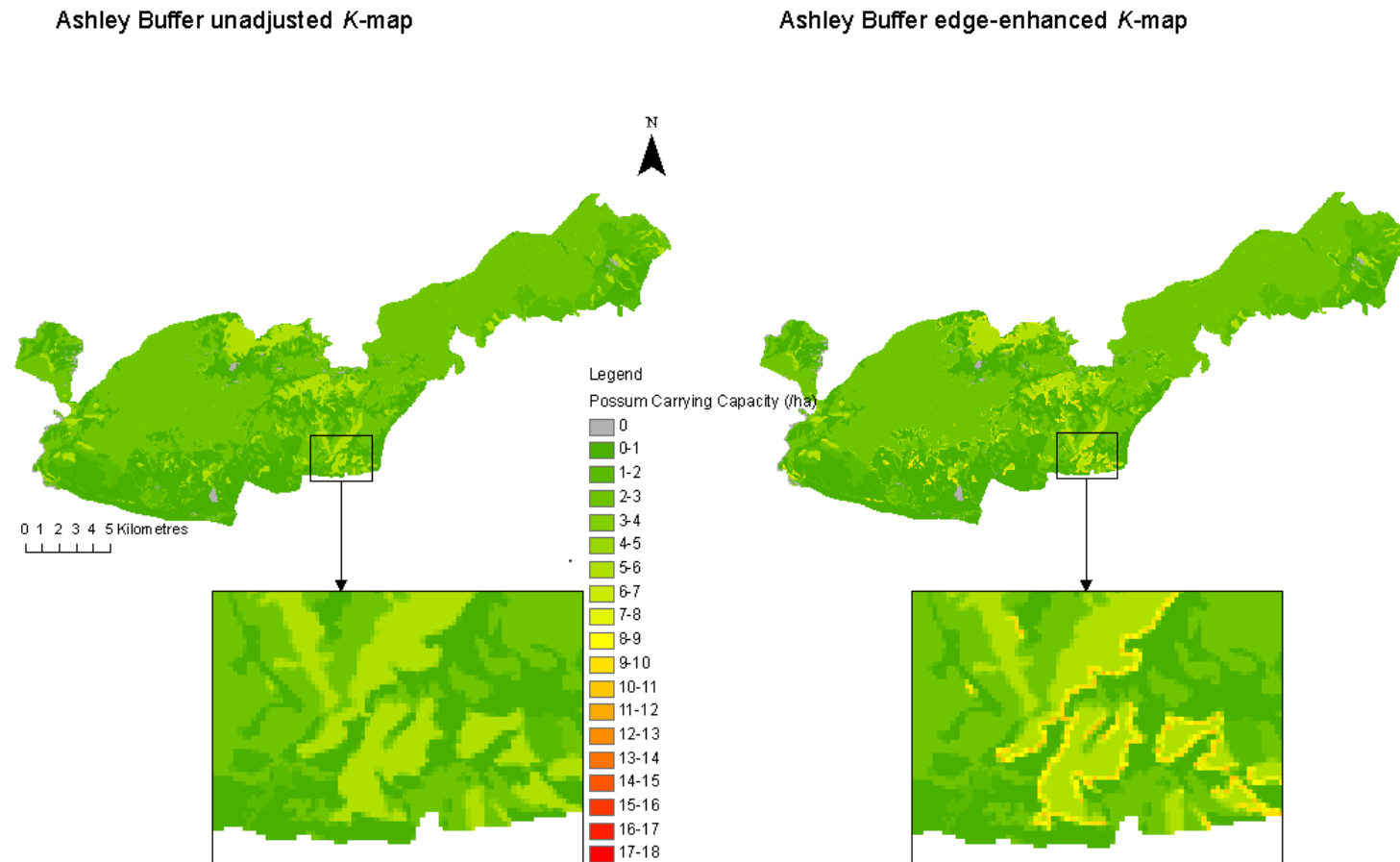
Figure 1 Possum carrying capacity maps (*K*-maps) for the Ashley Buffer Vector Control Zone (27 812 ha). Insets show close-up of habitat boundaries, where the map on the right has had the carrying capacity on the pasture/forest edge enhanced, shown by the brighter yellow around the palest green areas representing forest.
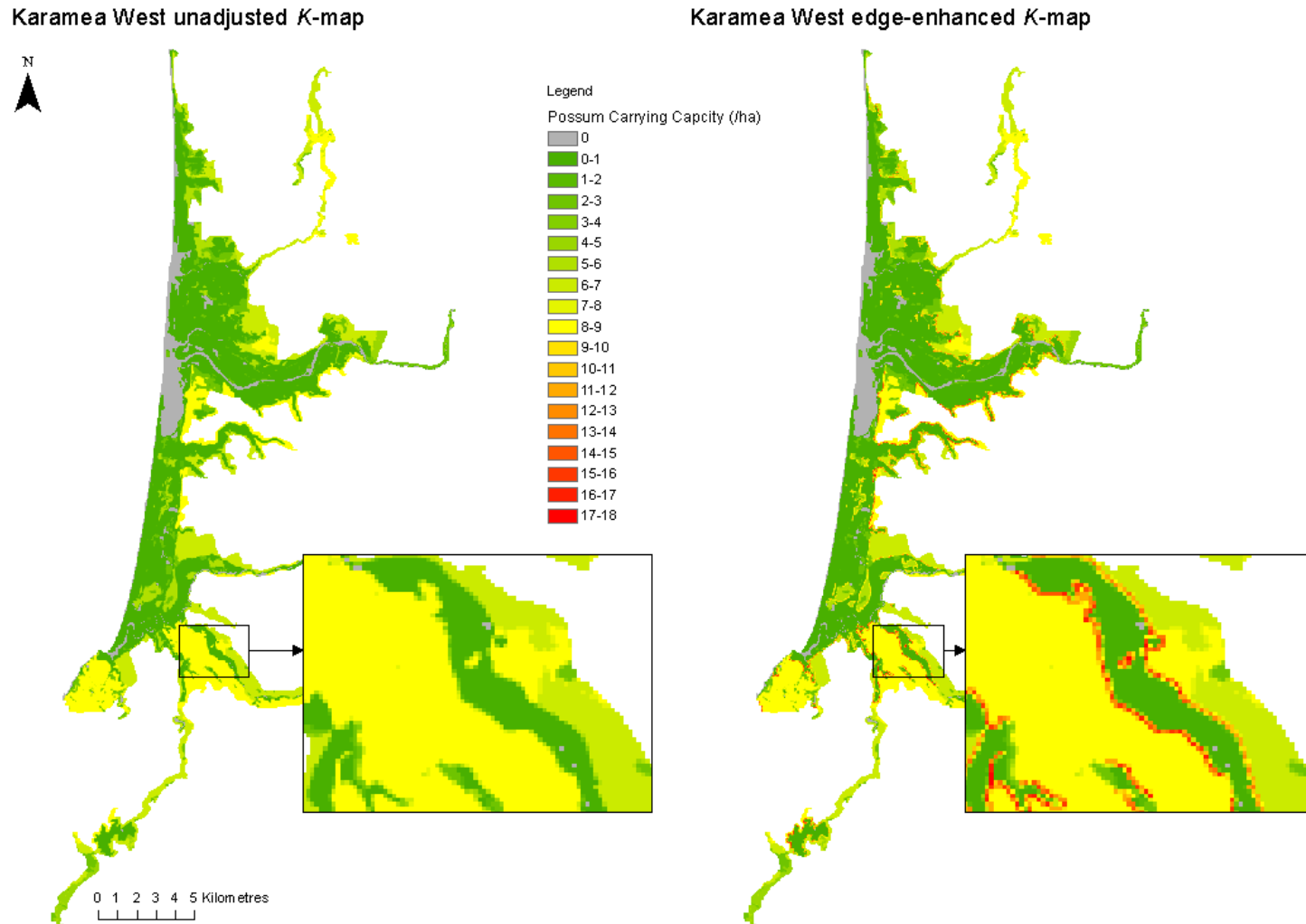
Figure 2 Possum carrying capacity maps (*K*-maps) for the West Karamea Vector Control Zone (15 403 ha). Insets show close-up of habitat boundaries, where the map on the right has had the carrying capacity on the pasture/forest edge enhanced, shown by the red around the yellow areas representing forest.
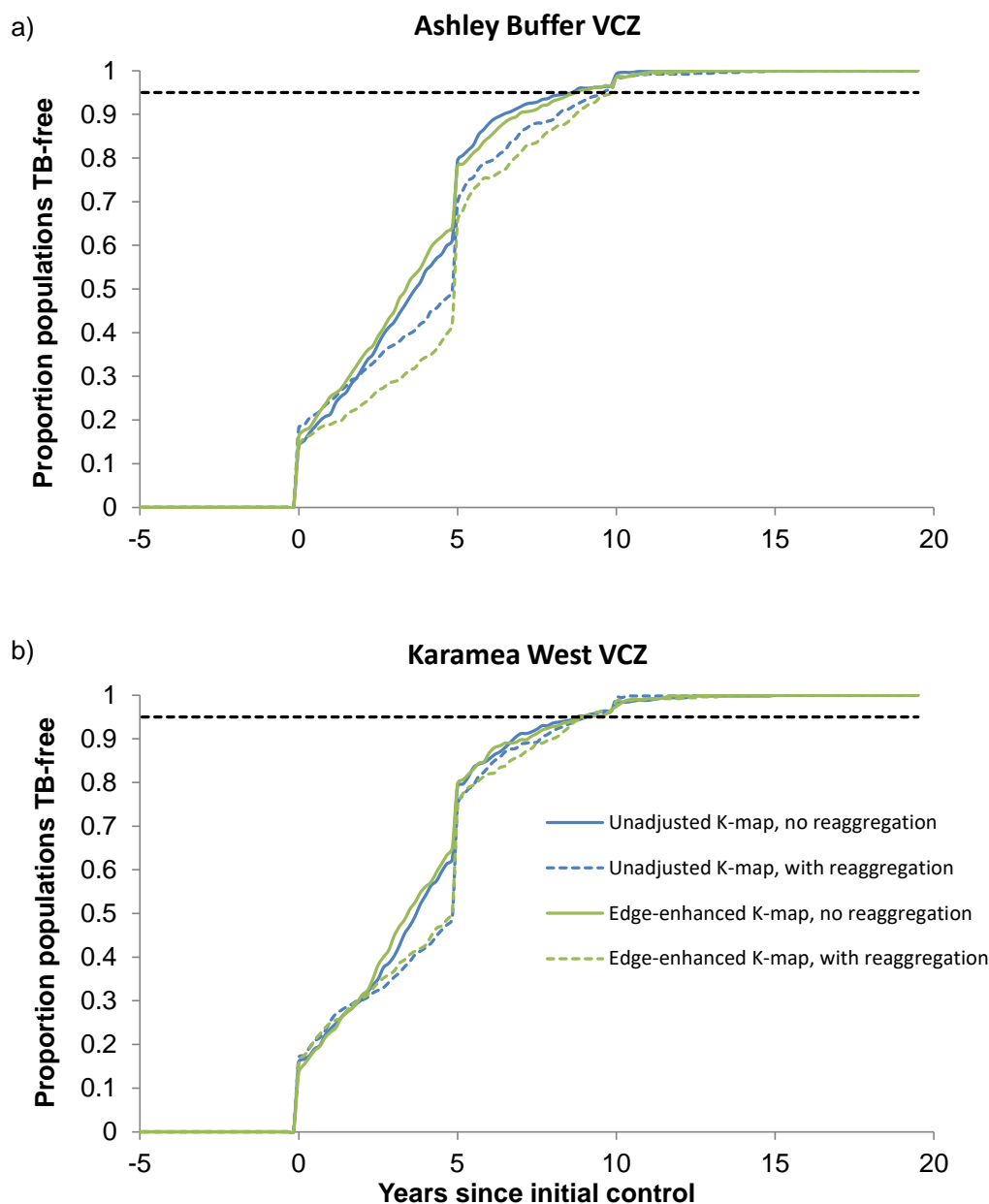
4.4 Sensitivity of predicted TB persistence to the model changes

In both case studies, the median time until TB extinction was less than 4 years after the initial control in the simulations with no possum reaggregation compared with 5 years for the simulations with reaggregation (Table 1). This was also apparent in the probability of TB freedom over time where the simulations with reaggregation lagged behind those without it, although this difference was reduced by subsequent controls so that the time taken for 95% of the simulated populations to reach TB freedom was only delayed by a maximum of one year in the Ashley Buffer simulations and half a year in West Karamea (Figure 3). Under all scenarios, 95% of simulations reached TB freedom less than 10 years after the initial possum control operation, i.e. after the second control in year 36 but before the third control in year 41, implying that a third control would have been unnecessary to achieve TB freedom in these areas if control was applied evenly and consistently. There was limited (Ashley) or no (Karamea) difference in TB dynamics apparent between the simulations with or without edge-enhanced K-maps.

Table 1 Median years to TB extinction under different model scenarios. The numbers in brackets are the 95th percentiles of time to TB extinction for 500 replicate simulations

Case study	K-maps	No reaggregation	With reaggregation
Ashley Buffer VCZ	Unadjusted	3.8 (0–10.0)	5.0 (0–10.0)
	Edge-enhanced	3.5 (0–10.0)	5.0 (0–10.0)
Karamea West VCZ	Unadjusted	3.8 (0–10.0)	5.0 (0–10.0)
	Edge-enhanced	3.5 (0–10.1)	4.8 (0–10.0)

a)

Ashley Buffer VCZ

b)

Karamea West VCZ

Legend:
- Unadjusted K-map, no reaggregation
- Unadjusted K-map, with reaggregation
- Edge-enhanced K-map, no reaggregation
- Edge-enhanced K-map, with reaggregation

Figure 3 Probability of TB freedom estimated from 500 replicate model simulations for possum populations in the two case-study areas. The grey vertical lines indicate timing of control operations.

The number of TB infections occurring was higher in the simulations with possum reaggregation compared with without reaggregation and this difference was more pronounced in the Ashley Buffer than in the Karamea West simulations (Table 2). This was also reflected in higher TB prevalence following initial possum control in the simulations that had reaggregation implemented (Figures 4 & 5). However, this higher prevalence is not maintained and, with time (and the implementation of the second possum control), TB rapidly drops out from the modelled population. Possum density was slightly lower following control in the simulations that implemented possum reaggregation (Figures 4 & 5).

Table 2 Mean number of TB infection events per annum occurring under different model scenarios, for edge-enhanced scenarios only

Case study	Assessment period	No reaggregation	With reaggregation
Ashley Buffer VCZ	Post - 1st control	22.47	68.28
	Post - 2nd control	0.44	1.73
	Post - 3rd control	0.01	0.01
Karamea West VCZ	Post - 1st control	20.49	29.37
	Post - 2nd control	0.39	0.91
	Post - 3rd control	0.02	0.02

## 4.5 Assessment of model changes

Allowing native forest/pasture edges or patches of native forest within pasture to have enhanced possum carrying capacity did little to alter TB dynamics within the modelled populations. This is because I scaled the disease transmission coefficient (β) for each scenario to ensure TB persistence and generate an equilibrium TB prevalence of 2%, in effect keeping βK constant. In addition, the structural connectivity of the modelled landscape was the same (possum habitat patches were still in the same places, but the carrying capacity within these patches was increased) so that spatial effects on contact and infection rates would be similar.

*Modifying carrying capacity (*K*) maps to produce enhanced possum density at forest edges or in remnant patches is probably not worth the effort involved.*

Making the modelled possums reaggregate following control to low densities (<0.2 possums/ha) resulted in more TB infections, presumably because these surviving possums now had neighbours to infect compared with an isolated possum with no neighbours. This resulted in higher TB prevalence, but even these enhanced local densities were too low for TB persistence and the disease continues to drop out of the system over time in face of continued control that drove densities (on average) ever lower. In other words, it appears that at the very low densities as which possums are isolated from their conspecifics (and are therefore driven to reaggregate) the resulting clusters of possums are still too small to sustain TB long term. The slightly lower overall (landscape-wide) density in the simulations with reaggregation could possibly be explained by greater density-dependent effects (higher death rates, lower birth rates) due to the higher local possum density when possums form aggregations.

The effects of reaggregation were not as pronounced for the Karamea West as for the Ashley Buffer simulations probably because the reaggregation algorithm was called less often following the first possum control in the Karamea West compared with the Ashley Buffer simulations (approximately 75% vs 83% of the simulations, respectively). This was because the Karamea West VCZ had a higher overall carrying capacity and the initial control

operation with a mean percentage kill of 95% did not always reduce the population to below the 0.2 possums/ha threshold required for the reaggregation algorithm to run.
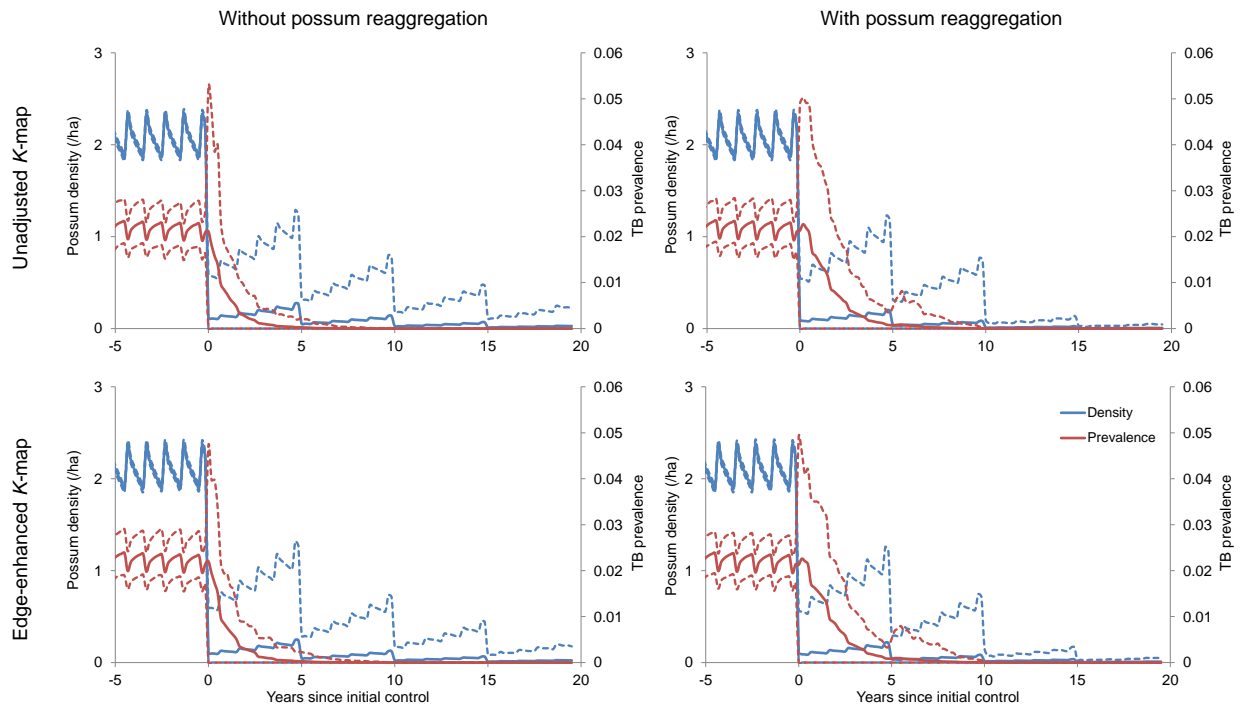
Figure 4 Mean possum density (blue line) and mean TB prevalence (red line) under the four scenarios for the Ashley Buffer Vector Control Zone simulations. Dashed lines are the 95th percentiles for the density or prevalence.
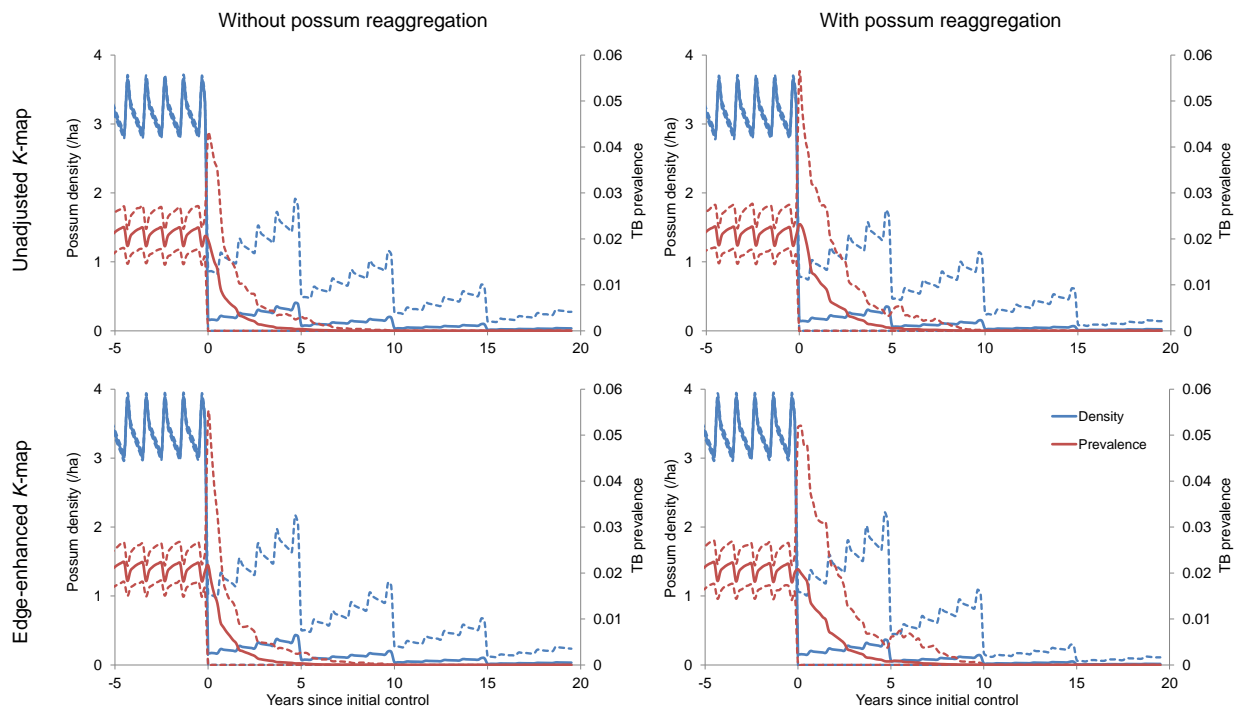
Figure 5 Mean possum density (blue line) and mean TB prevalence (red line) under the four scenarios for the Karamea West Vector Control Zone simulations. Dashed lines are the 95th percentiles for the density or prevalence.

5 Re-evaluation of model structure and parameter values

5.1 Sensitivity analysis methods

Global sensitivity analysis of PossTB Model predictions to key model parameters was assessed using the Fourier Amplitude Sensitivity Test (FAST) method implemented in the R package 'FAST' (R Development Core Team 2012). This method estimates the expected value and variance of the output, and the contribution of individual inputs to the variance of the output using Fourier coefficients (Frey & Patil 2002). For example a sensitivity value of 0.25 for a particular parameter would mean that 25% of the variation in the model output can be attributed to varying that parameter. An advantage of the FAST method is that it makes no assumptions of model structure and can thus be used on complex models like the PossTB Model.

The FAST method is the same as that used by Ramsey and Efford (2010) for their sensitivity analysis of the PossTB Model. Indeed that is how I identified which key parameters to vary for the current sensitivity analysis. In their analysis, they varied the home-range-scaling parameter and found it explained a lot of the variation in the output, whereas here I have used a modified (see 5.2) home-range-adjustment algorithm (where home range size increases in response to reduced local possum density) and examined the sensitivity of other key parameters under the assumption of home range adjustment. Also I have assumed that the product βK remains more or less constant (see 5.3) within the range 4–6, so that the random values of β used for the sensitivity analysis were obtained by drawing a random variable from $\beta K \sim U(4,6)$ and a random variable from $K \sim U(1,10)$ with $\beta = \beta K \div K$.

The model parameters listed in Table 3 were varied within the specified ranges and two types of model output were assessed: TB prevalence over time and the probability of TB persistence over time.

Table 3 Model parameters varied in the sensitivity analysis

Parameter	Description	Range
b	Instantaneous recruitment rate per year	0.25–0.69
Maledispersal	Male breeding dispersal rate per year	0–0.5
Femaledispersal	Female breeding dispersal rate per year	0–0.3
βK	Product of TB transmission rate × carrying capacity	4–6
p	Proportion of offspring infected via pseudo-vertical transmission	0.1–1
α	Disease mortality rate per year	0.8–3
K	Carrying capacity of habitat (possums/ha)	1–10

The probability of TB persistence was estimated as the number of replicates where TB persisted divided by the number of replicates done, which was 100 replicates for each combination of parameters. Each replicate was run for 50 years with a burn-in period of 30 years (i.e. model output was collected from years 31–50) with an initial 95% kill possum control simulated in year 36 followed by two maintenance controls (80% kill) in years 41 and 46.

5.2 Sensitivity analysis results

TB prevalence at possum equilibrium abundance was most sensitive to the disease-induced mortality rate *α*, followed by (at a much lower level) the pseudo-vertical disease transmission rate (*p*), then the horizontal disease transmission rate (*β*) (years 31–35 in Figure 6). Once possum abundance was lowered through simulated lethal control, the sensitivity to *α* declined although it still explained more of the variation in TB prevalence than any other of the parameters tested (years 36–50 in Figure 6). The importance of *β* declined initially with simulated possum control then increased again after the third simulated control (Figure 6) although sensitivities estimated at this stage should be viewed with caution because they were based on very few data – after the third control only 19 out of the 167 parameter sets tested had TB persisting by this stage, i.e. the sensitivity data were zero-inflated.
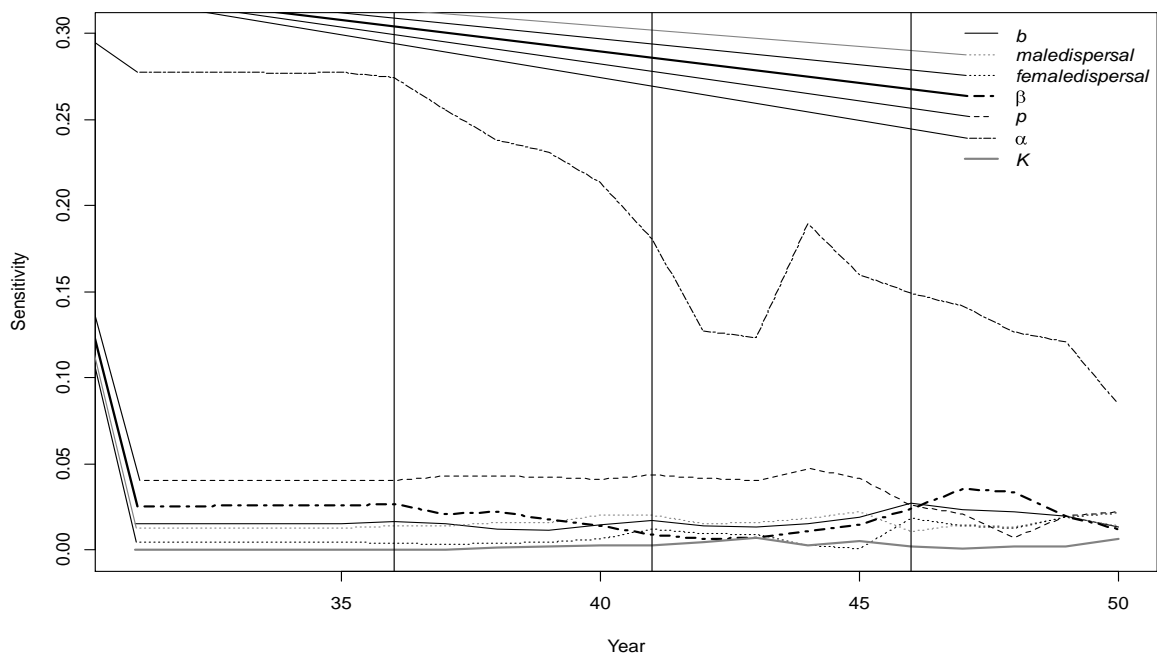

Figure 6 Sensitivity of TB prevalence to variation in model parameter values with time. Vertical lines indicate timing of simulated possum population control.

The overall low amount of variance explained by the modelled parameters (54% at equilibrium/year 31) combined with the low numbers of parameter combinations that gave TB persistence at equilibrium (51/167 = 31% at year 31) highlights the difficulty in getting the model to generate endemic disease – which can, in part, be explained by stochastic extinction of the disease. Figure 7 shows the probability of TB persisting in the sensitivity simulations with varying carrying capacity K and an approximation of the reproductive rate of the disease R' as:

$$R' = \frac{\beta K + pB(K)}{\alpha + D(K)} \; ;$$

where *B(K)* and *D(K)* are the realised birth and death rates respectively at equilibrium possum density. Note this is not a calculation of the actual reproductive rate of the disease R_0 because the contact-rates component of β in the PossTB Model is partitioned out and modelled explicitly as home range overlap. Whilst R' largely explains whether the disease persists for that particular combination of parameter values (with a threshold at $R'{\approx}4$), the chances of persistence are higher for simulations where there are larger numbers of possums (high K), indicating the importance of stochastic (chance) events on simulated TB persistence (Figure 7).
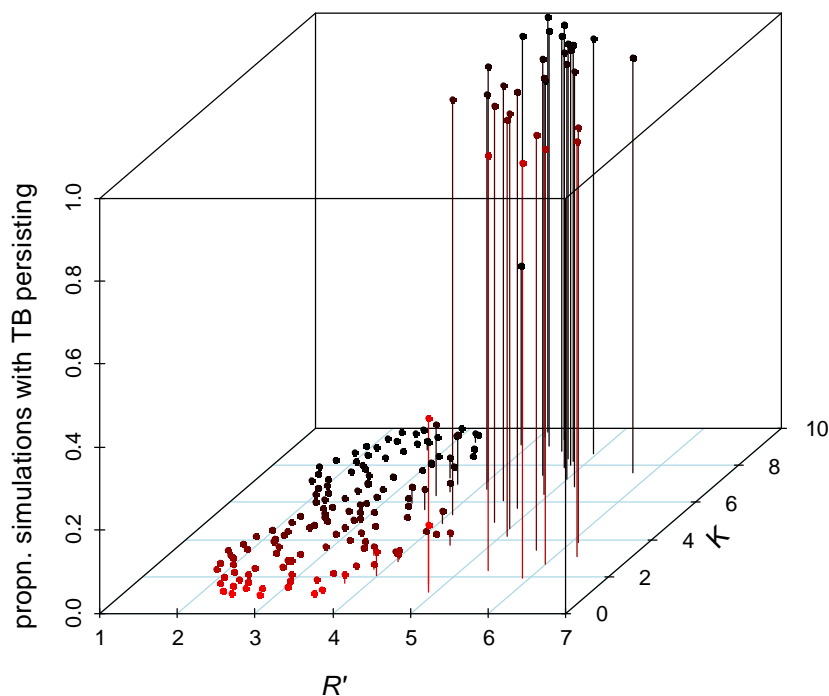
Figure 7 TB persistence at equilibrium in the sensitivity simulations with possum carrying capacity (K) and an approximation of the reproductive rate of the disease (R'). The colour scale of the points from red to black represents increasing possum carrying capacities from 1 to 10 possums/ha.

## 5.3   Home range size (σ) and home range adjustment

The PossTB Model assumes circular normal home ranges with a scaling parameter of $\sigma =$ 30 m equating to a 95% range area of approximately 1.7 ha. This value was derived using the inverse prediction method (Efford 2004) applied to mark–recapture data from seven different possum populations (Ramsey et al. 2005). This estimate agrees with home range estimates of 1–4 ha in forested habitats from telemetry studies (table 3.1 in Cowan & Clout 2000) and is appropriate for continuous forested sites but likely not for farmland or scrubby sites, which were not well represented in the Ramsey et al. (2005) study. Early studies on possums living in low-carrying-capacity habitats such as beech forest (Clout & Glaze 1984) or farmland (Brockie et al. 1997) documented large home ranges and more recent work in dryland tussock grassland/shrub habitats also suggests possums in low-density habitats have much larger home ranges than those in forested habitats. Using GPS fixes from collared free-ranging possums on two high-country stations (Molesworth and Muzzle) in Marlborough, Yockney (unpublished data) estimated an average home range area of 21 ha (range: 7–42 ha, $n = 20$).

Likewise, home range estimates from GPS-collared possums in typical drylands tussock grassland/shrub habitats in Central Otago averaged 22 ha (Rouco & Glen 2011). Possum density estimates were also derived in this study, using mark–recapture methods, and they confirmed low possum density in these habitats of 0.5–1 per hectare. Possum-home-range estimates from another drylands site, Molesworth Station, were intermediate at 5.1 ha (Glen et al. 2012) but these were estimated from daytime fixes from den sites; if the relationship of a 10-fold difference between den-site- and night-time-revealed home range estimates observed in the Central Otago study holds true, this suggests a 51-ha home range. The cause of differences in home range size between high-density populations in mixed forest and other habitats with low carrying capacity is presumably due to the much more sparsely distributed food resources with possums having to forage further afield in low-quality habitats to meet their energetic needs.

These large differences suggests that when modelling areas of low-quality habitat ($K{<}3$) using the PossTB default of $\sigma = 30$ m, we are greatly underestimating possum home range sizes and thus the extent of contact/overlap between distant neighbours. One solution to this would be to use a different value of σ for model simulations depending on the predominant habitat type. However, when modelling a mixed landscape, such as a forested buffer (high $K)$ adjacent to farmland, what value of σ would be appropriate? An alternative solution is to activate the home-range-adjustment algorithm (which is already built into the PossTB Model). This algorithm calculates, for each time step, for each individual possum, a unique home range scalar (σ) based on local possum density using the relationship illustrated in Figure 8. This relationship was fitted by Ramsey and Efford (2005; solid line in Figure 8) using estimates of σ and density derived from mark–recapture data and using the inverse prediction method. Note the fewer data at the lower end of the density scale, which is probably a consequence of the difficulty in obtaining enough recaptures to fit the mark–recapture models when possum numbers are low. It is important to note that the fitted relationship and its implementation in the model are based on the effects of actual/current density (N) not potential density or carrying capacity (K) on home range size, which is subtly different to the effect of habitat on home ranges described above. This could be due to the same postulated mechanism, i.e. the amount of resources that may become available when a neighbouring possum is removed, or it could be due to unknown social factors. There is some evidence from a recent study that possums do adjust their home range size in response to

removal of neighbours. Pech et al. (2010) found that GPS-collared possums adjacent to a poisoned area had larger home ranges and moved over greater distances than possums within untreated (but otherwise similar) forest, suggesting some effect of neighbours on possum movement behaviour over and above the effects of habitat. Likewise, surviving radio-collared possums in a forested area subject to control were found to move greater distances compared with possums in an area where the possums were not controlled (Nugent & Whitford 2011).
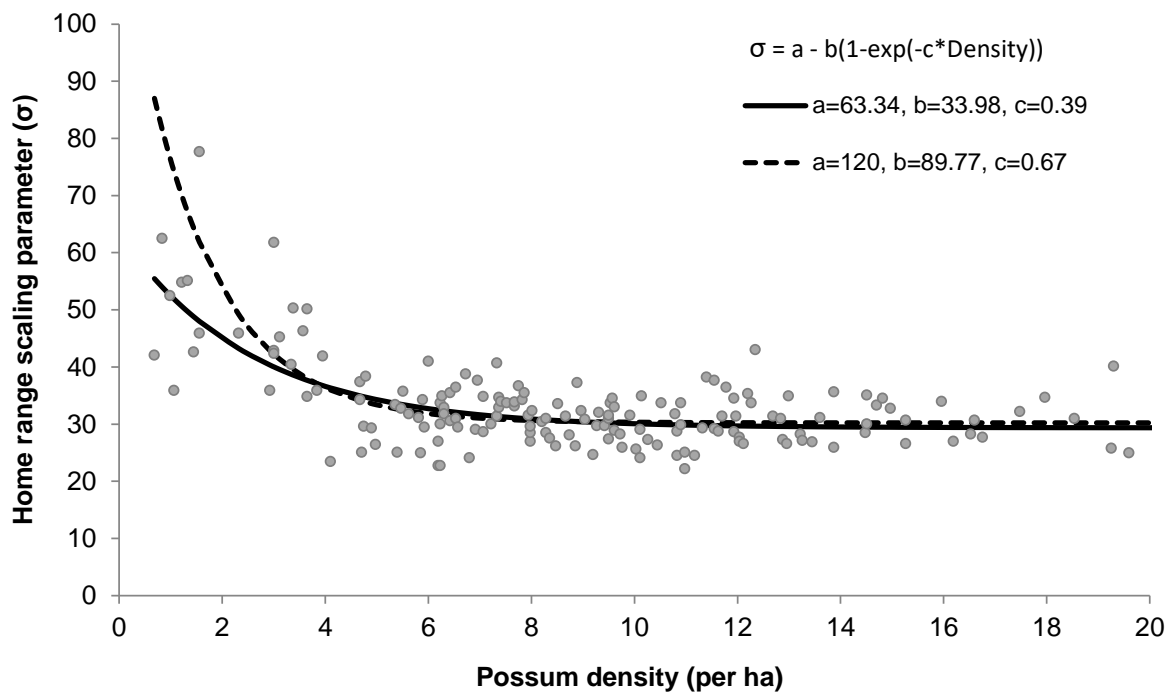
Figure 8 Relationship between possum home range scalar (σ) and possum density. The grey points and the solid line are the original data and fitted relationship derived by Ramsey and Efford (2005), the dashed line is the proposed alternative relationship.

In summary, using the home-range-adjustment algorithm in PossTB Model simulations could be used to deal with both home range expansion in response to reductions in possum density as a result of control, and differences in home range size at carrying capacity (including heterogeneity in habitat type, e.g. a mixture of farmland and forest that is typical of VCZ simulations). However, with the current default parameters it does not generate large enough home ranges at very low population densities. To remedy this I refitted the equation to the data shown in Ramsey & Efford (2005), arbitrarily fixing the intercept to $a = 120$ and weighting the data by density (effectively placing more importance on higher density estimates of σ). This produced a curve very similar to the original, asymptoting to $\sigma = 30$ at high possum densities (>3 possums/ha) yet generating larger home ranges at low possum densities (dashed line in Figure 8). For example a possum density of 0.5 per hectare would give $\sigma = 94$ m, which equates to a 95% home range kernel of 17 ha similar to that observed by Rouco and Glen (2011) in dryland habitats.

Use of the home-range-adjustment algorithm (with the new parameter values) is recommended when modelling heterogeneous landscapes.

5.4 Interaction between transmission rates and carrying capacity *βK*

Model predictions of TB persistence are sensitive to the value of *β*, the rate at which susceptible possums become infected with TB, yet this is the disease parameter we know least about. The typical approach to estimating *β* for model simulations is to work backwards from the assumption that TB was endemic in a population at some level of prevalence, say 2%, then find the value of *β* that generates 2% prevalence when the possum population is uncontrolled or at equilibrium. This is the approach taken using analytical methods by Barlow (1991b, 1993) and more recently by Ramsey & Efford (2010) using numerical methods to find a solution for *β*. Essentially this assumes *β* is inversely proportional to *K* so that *βK* is a constant. In other words, a higher *β* value is needed to generate the same level of prevalence at low compared with high possum density. Barlow (1991b) argued that *β* might be expected to vary inversely with *K* because the contact rate component of *β* is enhanced at lower possum densities due to larger home range sizes, more home range overlap and thus more potentially infectious contacts.

In the PossTB Model contact rates are modelled explicitly by home range overlap, so using the home range adjustment algorithm (see 5.2) models the effect described by Barlow, but we still need a higher value of *β* to generate a given level of prevalence in low-density populations because the contact rates are not as high between distant neighbours as they are for close ones. Also while the larger home ranges allow some contact between distant neighbours they also result in relatively lower contact rates between close neighbours because an individual's home range use is spread more thinly across the landscape in a larger home range compared with a smaller one where home range use is concentrated near the home range centre. This is because the home range utilisation function uses a normalised kernel so that the area under the curve or the sum of the home range utilisation kernel is 1, equivalent to one individual's use of the landscape. This is a change from the previous version of the model, which used non-standardised home range kernels for the infected possums. The area under the curve (and thus the maximum number of contacts as possum can make) in a non-standardised kernel scales with σ^2 so that a single infected possum with $\sigma = 80$ has, unrealistically,7 times the influence of a possum with $\sigma = 30$. Using standardised kernels means the maximum number of contacts of an infected possum can make at any point on the landscape is proportional to their use of that space.

With no new data available on transmission rates since the current model was developed, it is recommended that the status quo be retained of 'tuning' the transmission rates to the habitat carrying capacity and connectivity of the specific landscape being modelled. A new Marsden-funded study looking at social connectivity between free-living possums and the number of secondary TB infections arising from targeted artificial infection of individuals within these social networks promises to provide new data on rates of infection. A pilot trial to test the concept, using percutaneous injection of *M. bovis* into possum paws to initiate TB infection, has proved successful, with evidence that at least one secondary infection has been generated within the surrounding possum population (J. Whitford pers. comm., R-10738: Detection of TB in possums by possums).

## 5.5 Disease mortality rate α and infected residence time

The current value assumed for the additional-disease-induced mortality rate is 1 per annum, which is consistent with the estimate of $\alpha = 1.08$–1.33 provided by Ramsey & Cowan (2003). This, combined with a natural mortality rate of $d = 0.1$ per annum, gives an average longevity of an infected possum of $1/1.1 = 0.91$ years = 11 months. Note that this is the average longevity of an infected possum so includes both the pre-clinical and clinical stages of TB, where clinical infection was defined as the presence of externally detectable (visible or palpatable) lesions. The pre-clinical and clinical phases have been (not always explicitly) assumed to be equivalent to latent and infectious stages respectively, but there is no evidence to support this equivalency. Because of that, Ramsey and Efford (2010) followed the example of Barlow (2000) and Roberts (1996) in combining the latent and infectious stages into a single class, so the PossTB Model assumes possums are infectious from as soon as they are infected. Another critical assumption is that this mortality rate is constant with respect to time since infection, so that the time possums spend in this infected stage (called the 'waiting time') is exponentially distributed. In practice this means that more individuals have a much shorter or much longer waiting time, i.e. there is greater variability, in the infected stage than you would expect from the mean (Figure 9b). Compare this with waiting times predicted from a much less dispersed distribution, the gamma, where the chance of dying increases with time since infection (Figure 9d), times spent in the infected stage are closer to the mean (Figure 9e), and it takes longer for a cohort to start dying off from the disease (Figure 9f).

Recent data (Figure 10) documents, for the first time, the survival probability of a cohort of 38 free-living possums live-caught and artificially-infected with *M. bovis* using inter-digital percutaneous injection, then released back into the wild. Firstly note the extended period of survival to about 7 weeks before individuals start dying. This equates to the length of the preclinical phase identified using this challenge model (Nugent et al. 2012) during which there are no visible lesions, making it unlikely that the disease is having any effect on possum survival during this phase. The exponential model (fitted to derive an estimate for α) is a poor fit to the data because of this apparent preclinical stage where the risk of dying is negligible followed by the development of clinical disease with an increased risk of dying. Comparing the fitted models (Figure 10) suggests that a gamma function would provide a more realistic description of the *pattern* of mortality than an exponential one.

Secondly, the mean survival time was about 19 weeks, much shorter than the 47 weeks assumed by the current default values of α and d. This difference was apparent in both the preclinical and clinical stages, i.e. a preclinical phase of <2 months (Nugent et al. 2012) vs 3.6 months (Corner et al. 2002) and a clinical phase of 2.5 months (Nugent et al. submitted) vs 4.7 months (Ramsey & Cowan 2003) was estimated from possums infected by percutaneous injection and naturally infected, respectively. Disease progression in possums that were infected via percutaneous injection may have been unnaturally fast because each possum was inoculated twice (to guarantee that infection established), therefore I would hesitate to recommend changing the α value just yet. However, the low-dose percutaneous *M. bovis* infection model does appear to reliably mimic natural disease in possums, and as results from further studies with this method come to hand, the value of α should be reassessed.
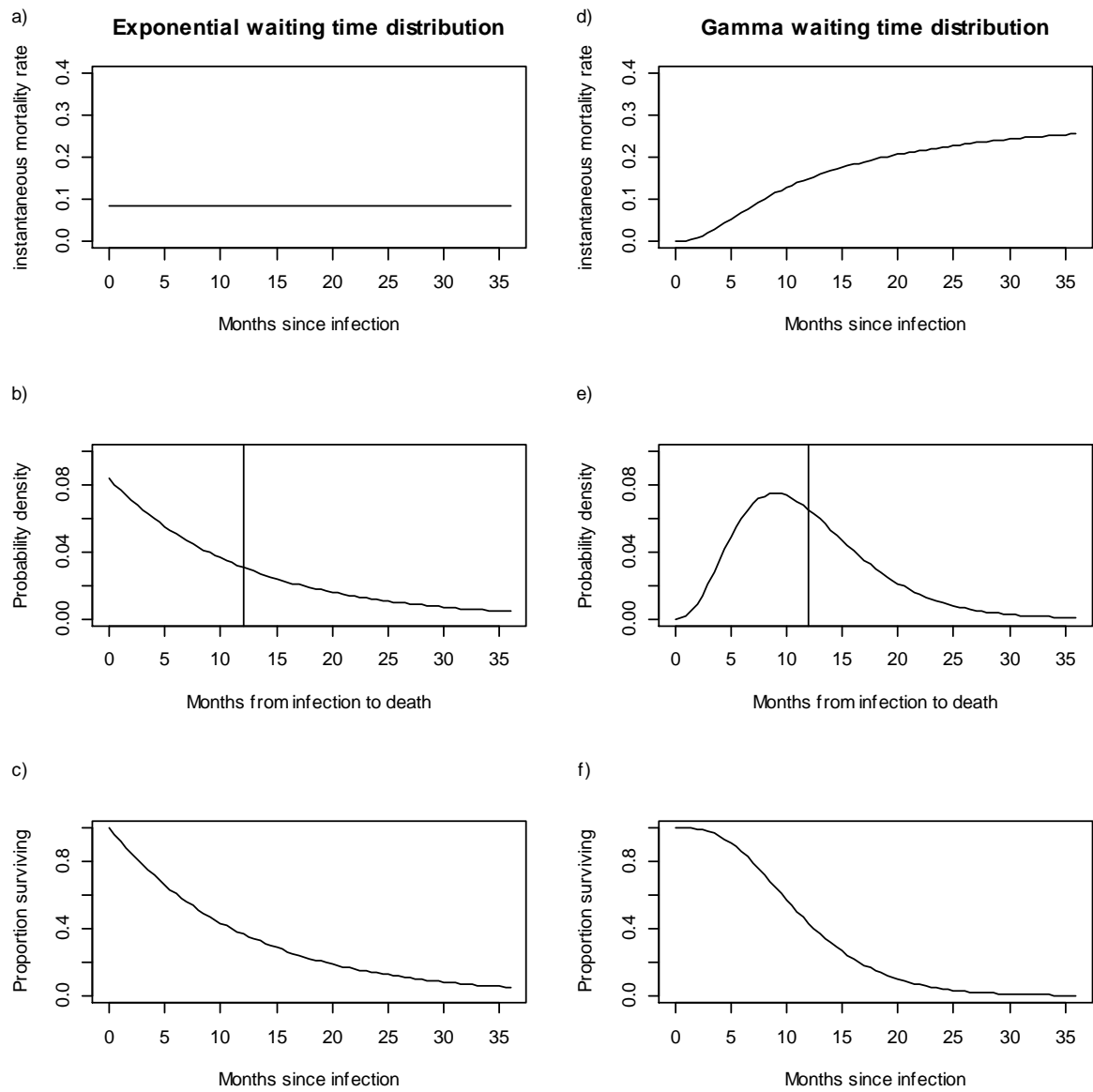
Figure 9 Theoretical hazard, density and survival functions for two different waiting-time distributions.
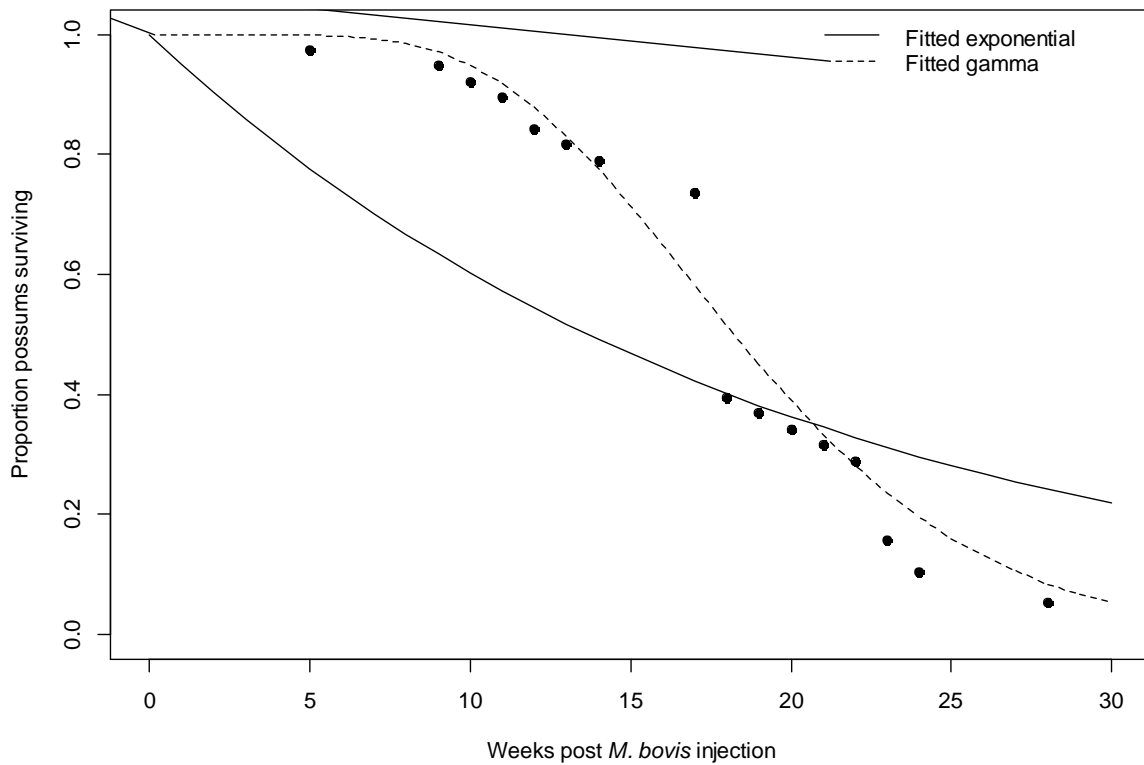
Figure 10 Survival curves of possums artificially-infected with *M. bovis* (from Nugent et al. submitted). Data are indicated with black dots, fitted curves for waiting times that are exponentially- and gamma-distributed are indicated with solid and dashed lines respectively.

This summary suggests we may have got both the value and the structure wrong for the disease mortality rate. The consequences of changing these assumptions in the model are, as yet, unknown. All else being equal, a higher α would lead to a more acute disease with faster disease turnover in the population reducing TB persistence. Countering that, simulating a delay before disease starts affecting mortality could enhance disease persistence if infected possums had greater opportunity to infect others before dying of the disease.

It has often been noted that it is difficult to get disease to establish and persist using the PossTB Model. I suggest that this reflects a number of interacting factors. Firstly, the combination of low TB prevalence at equilibrium and demographic stochasticity means the modelled disease is more prone to chance extinction compared with a deterministic model. Secondly, the spatial nature of the model means that mixing/contacts between possums are heterogeneous and highly localised so that once an infected possum infects all of its neighbours it has 'used up' its supply of susceptible possums, effectively putting a limit on the reproductive rate of the disease. Compare this with a simple homogeneous mixing model like Barlow (1991a) where each and every possum in the modelled population is susceptible to infection, i.e. there is (unrealistically) no notion of spatial location. Finally, I suggest that the lumping together of the latent and infectious stages and the exponential loss from the infected stage results in many possums dying quickly before they get much of a chance to infect another, i.e. their reproductive rate $R_0 = 0$. To remedy this would require a change in the PossTB Model by directly drawing the time to death from a less dispersed distribution

than the exponential (which would require recording an individual's time of infection) or more usually (e.g. Conlan et al. 2010) by subdividing the infected class into multiple stages effectively creating a gamma distribution of waiting times where the number of subdivisions is the shape parameter to the gamma distribution. If using the latter method it would make sense to also divide the infected class into a latent and infectious stage although this method would have the disadvantage of greatly increasing the number of events processed each time step and thus the processing time. Given this, I would recommend initially trying the first approach to see what effect less dispersed waiting times have on disease dynamics.

5.6 Pseudo-vertical transmission, *p*

Pseudo-vertical transmission is the transmission of TB from mother to dependent offspring. Barlow (2000) used a value of $p = 0.25$ in his model, reasoning that if a mother was infectious, 100% of her offspring would become infected, but since an individual is only infectious for a quarter of the time it is infected (has a latent period 3 times the length of the infectious period) then on average only a quarter of the offspring produced while the mother was infected would acquire TB. Ramsey and Efford (2010) followed the same logic and also used a pseudo-vertical transmission rate of $p = 0.25$, and that assumption remains the default in the current model.

The idea that 100% of offspring of infectious mothers become infected originated from the longitudinal study of possums with TB at Castlepoint (Pfeiffer & Morris 1991). Jackson et al. (1995) argue that given the long and close association between mothers and their dependent young, in the order of 6–9 months, transmission must occur if the mother is infectious. Pfeiffer (1994) and Morris et al. (1994) concluded that pseudo-vertical transmission must be very high to be able to maintain localised TB infection. The best data available were the detailed case histories of eight infected females that reared young in the Castlepoint study (Jackson 1995). They were based on identification of TB infection in both mothers and their joeys through a mixture of methods (serological tests, clinical examination, necropsy, cultural examination). In the five cases where lesions from both mother and offspring were cultured the strain types (identified by restriction endonuclease analysis) were identical, consistent with a common source. While this is compelling evidence of pseudo-vertical transmission and there is no doubt it does occur, these case studies cannot give us a proportional estimate because in most cases the temporal sequence of TB identification (e.g. the joey being diagnosed before the mother) means it is impossible to differentiate pseudo-vertical transmission from concurrent transmission from the same source. In the three cases where the mother was observed to be clinically infected before the joey and the fate of the joey was known, the offspring did all contract TB.

Assuming that clinically detectable TB with palpable lesions is roughly equivalent to the infectious stage and therefore that the preclinical phase approximates the non-infectious phase, information on the relative lengths of these stages is available from the recent work of Nugent et al. (submitted) who found a median survival period of 18 weeks and a preclinical period of 7 weeks post-infection, corresponding to an infectious period of around 0.6 of the total infected period. This proportion is similar to that estimated from Ramsey & Cowan (2003) as 4.7 months/8.3 months = 0.57, although their survival times were longer, and their preclinical period was estimated from another study using naturally-infected possums in a laboratory rather than a field setting. Based on the above and, for want of a better estimate,

assuming 100% of offspring of *infectious* mothers become infected, a higher pseudo-vertical transmission rate of around $p = 0.6$ should be considered.

The PossTB Model takes a retrospective approach in modelling pseudo-vertical transmission since offspring are not added into the possum population until they are independent at around 9 months. Therefore the model may underestimate the number of infected juveniles being recruited, as TB-infected mothers that may have given birth, passed on infection, then subsequently died do not produce any offspring in the model. However, if you accept that most of the still-dependent offspring would also have died after their mother did, this is not a big oversight. A related problem is that juveniles only become infected upon independence so their survival times will be overestimated since mortality only begins to affect them once they are recruited, when in theory mortality should occur from the time they became infected (prior to independence). This may overstate the likelihood of an infected juvenile establishing a new disease focus since all juveniles are assumed to disperse when they become independent. Of course the two biases might cancel each other out, but without rebuilding the model and comparing with the current we have no way of knowing what the effect of this delay might be.

5.7 Possum dispersal

Ten parameters control possum dispersal in the PossTB model; these are the proportion of adult male and female possums dispersing each year (10% and 5% respectively) and the shape and scale parameters for a gamma distribution of dispersal distances for male and female, juvenile and adult classes. All juvenile possums are assumed to disperse upon independence and the shape and scale parameters for females and males respectively are assumed to be the same whether they are juveniles or adults, giving a mean dispersal distance of 1 km for female possums and 4 km for male possums (Figure 11).
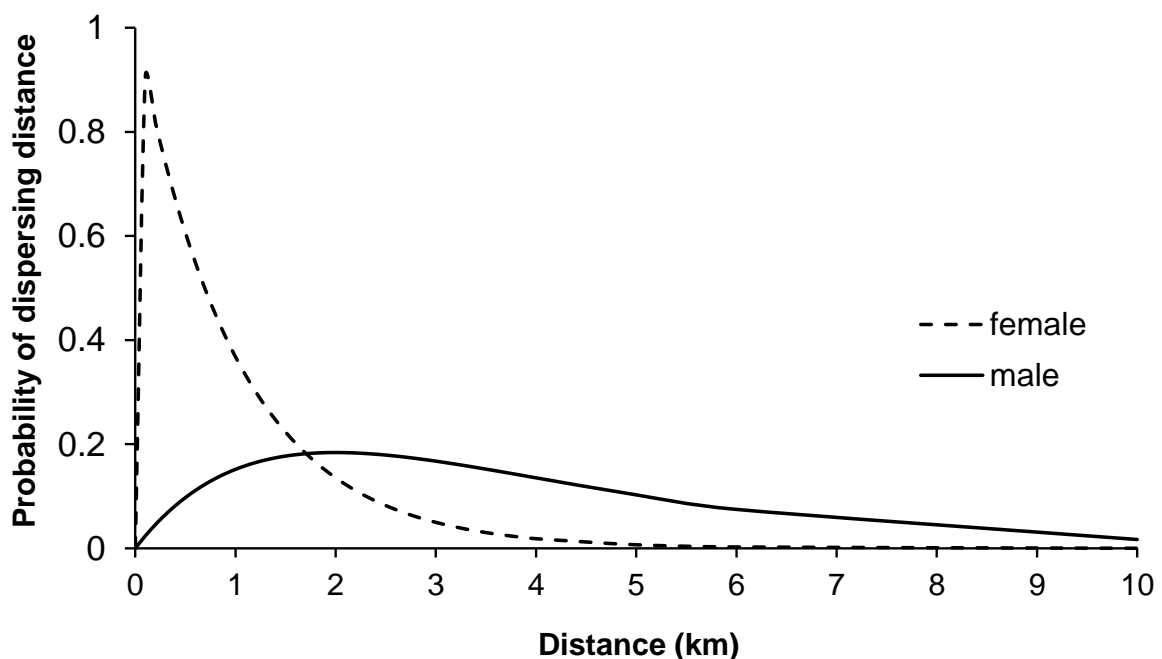
Figure 11 Distribution of dispersal distances assumed in the PossTB model, applies to both juvenile and adult dispersal.

A recent study of possum movements in dryland habitat on Molesworth Station showed similar mean dispersal distances for the sexes of 2.4 km for females and 2.4 km for males (Glen et al. 2012) although sample sizes were small ($n = 9$) and definitions of dispersal differed between this study and in the model. The model assumes dispersal is a shift in a possum's home range centre with female dispersal distances skewed to the left (Figure 11) to simulate female offspring settling in or close to their natal range while allowing for occasional very long (>4 km) dispersal movements. The Molesworth study looked at the distance between first and last locations of radio-collared possums and considered dispersal movements to be only those movements ≥1 km. Despite these differences it does appear that there were fewer long-distance movements recorded for male possums compared with previous studies (Cowan & Clout 2000) and those assumed in the PossTB model. Information on the proportion of different age and sex classes dispersing can be gleaned from Pech et al. (2012) who analysed the movements over the main dispersal period (February to August) of 79 GPS-collared possums in forest habitat and classified their movement into long-distance dispersal, exploratory movements, home-range displacement, and settled home range. If we take home-range displacement and long-distance dispersal to be analogous to the dispersal in the PossTB Model and assume there was not much more movement for the rest of the year then the adults showed similar proportions dispersing to those assumed in the model at 4% for males and 7% for females. For the juveniles, however, only 35% of males and 6% of females dispersed, a lot less than the 100% assumed in the model. All three of the long-distance-dispersal events recorded were by juveniles and the distances moved were within the range expected at ~1.3–2.5 km for the two males and 1.1 km for the female. While these two studies suggest male dispersal distances and the proportion of juveniles dispersing may be overestimated in the current model, the small sample sizes, different definitions of dispersal, and a general desire to err on the side of caution advocate for keeping the current dispersal parameters of the PossTB model.

5.8 Model construction

In undertaking this extension and re-evaluation of the PossTB model, I identified a number of coding errors and inconsistencies between the described model and the model code. This is inevitable with 'inheritance' of the model from one researcher to another, the overall complexity of the model, the adaption and adding of code onto the original framework, and the DIY nature of the programming. Because it is not commercial software, there is no bug checking or code validation before the model is released to the users. This can obviously create problems, particularly if different users are using different versions.

I therefore suggest that a versioning and change-tracking system be developed for the model, so at least if bugs occur we will know what changes they are due to. Further, a more permanent site needs to be obtained to house the most recent model and for AHB staff to download the executable model from, because the current mechanism of making the model available via the Landcare Research public ftp site provides only short-term availability. Finally, I suggest that a professional programmer is hired to recode the model and conduct formal software testing.

6   Conclusions

The modifications to the PossTB Model structure, and my re-evaluation of the parameter values that influence the probability of TB persistence, indicate, not surprisingly, that the predictions of the model can change greatly as the basic structure and assumptions are changed. Although I have identified changes that do have an effect, there is, almost always, still not enough empirical data to accurately parameterise or validate the model. For example, the reaggregation algorithm showed a modest effect on TB persistence but the controlling parameters (the threshold density for reaggregation to be stimulated, the probability of a possum staying put, and the effect of distance to neighbouring possums on this probability, and the resulting dispersion of the group) are unknown. On the other hand, the sensitivity analysis has shown that while some parameters have a large influence on TB persistence at equilibrium, the model predicts that once control is applied and possum numbers are dramatically reduced, the disease cannot be maintained regardless.

Taking a pragmatic approach to decide on how to further refine the PossTB Model, we need to bear in mind firstly what the predictions are going to be used for, and secondly the consequences of being wildly out with the model predictions. For the purposes of providing prior probabilities of TB freedom for the Proof of Freedom software, the output is arguably not so critical because empirical surveillance data from the field are used to validate the predicted probability of freedom and the current practice of creating a distribution for the PossTB Model prior allows for some uncertainty in prior knowledge.

If, however, the PossTB Model is to be used to make decisions about whether another VCZ-wide aerial control is required to be able to move into the eradication phase, then the costs of being wrong are higher, particularly the opportunity costs of potentially letting possum populations and the TB infection within them recover. In this case a conservative approach is warranted in running model simulations, and the fall-back approach is to assume that the TB-transmission rate (β) *is* high enough to generate endemic TB in the possum population of concern when they are at equilibrium abundance. Contact, and thus transmission rates, is inextricably linked to carrying capacity (K) through differences in home range areas with possum density, and using the home-range-adjustment algorithm provides a way of simulating this relationship and maintaining contacts at low density. The disease transmission rate will have to be altered for each landscape simulated because the default value presented corresponds to a continuous tract of high-quality habitat ($K = 10$ possums/ha) – a situation unlikely for most VCZs.

The re-evaluation and extension of the model inevitably highlight the vast amount of knowledge and information that are needed (but are not yet available) to accurately characterise the combined complexity of possum population dynamics and TB epidemiology in diverse landscapes and under complex management scenarios. Nonetheless, the model appeared to be remarkably robust despite this shortcoming, in the sense that for any set of assumptions that are able to predict long-run persistence of TB in the absence of possum control, the instigation of intensive control invariably results in the model predicting TB disappearance within 10–15 years, if not sooner.

7   Recommendations

The AHB should:

- Consider further exploration of disease-induced mortality as the parameter most strongly affecting model predictions.

- Consider changing the model structure to include a latent class or at least introduce some kind of delay before individuals start dying of disease. The former option would require recoding but could be combined with the next recommendation.

- Consider contracting a professional programmer to recode the model and do some formal software testing.

- With Landcare Research, develop and instigate a versioning system for model changes, and find a more permanent solution for making the model available to AHB users. In the meantime AHB users should check the ftp site regularly to download the latest version of the PossTB Model: ftp://ftp.landcareresearch.co.nz/Spatial%20Possum%20TB%20Model/.

For AHB users specifically, I recommend the following guidelines for using the model:

- *You will need to adjust the disease transmission rate β to make TB persist in your modelled landscape; this can be done using trial and error or the 'find beta' algorithm (the latter option is very time consuming).*

- *Use the reaggregation algorithm at a threshold density of <=0.2 possum/ha if you want to make conservative predictions (but bear in mind this will slow down processing time).*

- *Modifying carrying capacity (K) maps to produce enhanced possum density at forest edges or in remnant patches is probably not worth the effort.*

- *Use the home-range-adjustment algorithm ('non-linear contact rates' check-box) in preference to using a constant value for home range size (σ), which is only appropriate when the landscape is homogeneous.*

- *For initial simulations use the parameter values in Appendix 1. Note that the parameters for home range adjustment, horizontal disease transmission and pseudo-vertical disease transmission have been changed from earlier versions.*

8   Acknowledgements

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Appendix 1 – Recommended default parameters for PossTB Model simulations

Symbol	Description	Default value
Cell size	Resolution of *K*-map (m)	50
Neighbourhood radius	Limit to home range calculations (m)	250
K	Carrying capacity of habitat (possums/ha)	10*
MaleSus	Initial proportion of population male and susceptible	0.49
FemaleSus	Initial proportion of population female and susceptible	0.49
MaleInf	Initial proportion of population male and infected	0.01
FemaleInf	Initial proportion of population female and infected	0.01
Seasons per year	1/time step of model in years	6
b	Instantaneous birth rate / year	0.50
d	Instantaneous death rate / year	0.10
ϑ	Asymmetry of density dependence	3
δ	Proportion of density dependence in breeding	0.5
sexratio	Proportion of offspring female at independence	0.5
γ	Proportion of births in first pulse	1.0
br1	Julian day of first birth pulse	1
br2	Julian day of second birth pulse	160
σ	Sigma, home range distribution scalar (m)	30[#]
Maledistance	Scale parameter male natal dispersal km	2.0
Femaledistance	Scale parameter female natal dispersal km	1.0
Maleshape	Shape parameter male natal dispersal	2
Femaleshape	Shape parameter female natal dispersal	1
Maledispersal	Male breeding dispersal rate / year	0.10
Femaledispersal	Female breeding dispersal rate / year	0.05
maledistanceB	Scale parameter male breeding dispersal (km)	2.0
femaledistanceB	Scale parameter female breeding dispersal (km)	1.0
MaleshapeB	Shape parameter male breeding dispersal	2
femaleshapeB	Shape parameter female breeding dispersal	1
TB σ	TB contact distribution scalar (m)	30[$]
β	TB transmission rate	0.500*
p	Prop. offspring infected via pseudo-vertical transmission	0.6
α	Disease mortality rate / year	1
Density kernel	Shape of home range distribution	Normal[$]
TB Contact distribution	Shape of TB contact distribution	normal[$]

Symbol	Description	Default value
Non-linear contact rates	Adjust sigma (home range scalar) with possum density	on[#]
CRa	Parameter to home range adjustment function	120
CRb	Parameter to home range adjustment function	89.8
CRc	Parameter to home range adjustment function	0.670
CV(σ)	Coefficient of variation for variable home ranges	0
Transmission type	Type of TB transmission rate assumed	Density dependent
Reaggregate possums surviving control	Implement the reaggregation algorithm	on
Threshold density	Possum density (/ha) at or below which possums reaggregate	0.2

[*]If a K-map is imported with anything other than $K = 10$ then $β$ will also have to be adjusted to achieve TB persistence.

[#]If the Non-linear contact rates function is checked, this overrides the constant $σ$ value.

[$]Not currently used in model – TB contact distributions are set to the same as the possum home range distributions.