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Improved modelling of TB persistence in possum populations

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Improved modelling of TB persistence in possum populations

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Contents

Summary

Project and client

- Landcare Research, Lincoln, was contracted by OSPRI to improve (and make more credible) the way in which TB persistence in possum populations is modelled using a spatially explicit model of TB epidemiology in possums (the Spatial Possum-TB model, SPM). This is the final project report and it describes all work undertaken in the project.

Objectives

- To identify ways in which TB transmission in possums could be more credibly modelled than previously.
- To re-program the SPM to incorporate a new approach to emulating TB transmission and, at the same time, add new functionality, enabling the likelihood of TB persistence and/or re-establishment in recovering possum populations to be readily and realistically modelled.
- To explore the effect of changing SPM assumptions on predicted probabilities of TB freedom.
- To demonstrate how the SPM can be used to inform a new approach to declaring TB freedom based on simultaneous conduct of surveillance and control.

Methods

- A workshop and literature review were conducted to identify potential mechanisms for increasing TB persistence in the SPM.
- An individual-based simulation model was constructed to explore how metrics of possum home range overlap/contacts are affected by possum density, possum aggregation and density-dependent home range sizes.
- The SPM was revised to calculate possum-to-possum contact rates based on Bhattacharyya's affinity' (BA) metric. An algorithm to seed the modelled possum population with TB-infected possum(s) was also included in the revised SPM.
- The effects of changes to the SPM on TB dynamics and model-predicted prior probabilities of TB freedom were explored using four vector control zones (VCZs) as case studies.
- The revised SPM was used to predict the distribution of the number of TB-infected possums remaining after population control.

Results

- Two potential mechanisms for maintaining disease in the SPM were identified as promising:

  - individual heterogeneity in TB progression, which involves allowing a small proportion of infected individuals to survive for longer periods due to lesion resolution
  - maintaining a minimum level of contact with conspecifics, even when possum density is very low.
- The individual-based simulation model showed that using the BA metric for calculating possum contact rates enabled maintenance of contacts at low possum density, and that aggregation of possum home range centres increased the overall number of contacts.
- When the new contact rate was coded into the SPM, the effects on TB dynamics were only apparent when control was inadequate, as illustrated by the Waihoki VCZ simulations. In this case, the higher post-control density of possums and the maintenance of contacts at this density led to TB persistence in approximately 20% of simulations ($P_{Free} < 0.80$), in contrast to the original version, which predicted $P_{Free} \geq 0.95$, 12 years post-control.
- Increasing the disease mortality rate from $\alpha = 1$ to $\alpha = 2.4$ increased disease turnover and meant that TB dropped out of the possum population at a faster rate following simulated control because infected possums were dying sooner.
- The revised SPM has already been used to predict residual distributions of TB-infected possums for input to the survey-then-control methodology. It is also being used for other current projects: one to identify the characteristics of habitat patches (size, isolation) that can be excluded from control and surveillance (R-10801), and another looking at how spatial heterogeneity in possum carrying capacity and control can influence residual possum abundance and TB persistence (R-10804).

Conclusions

- A mechanism for TB persistence based on empirical evidence showing possums maintain contact with a minimum number of neighbouring possums over a range of possum densities has been incorporated into the SPM by calculating contact rates as a function of joint space use between a susceptible and an infectious possum (BA's metric).
- The revised SPM is less sensitive to the value of the disease transmission rate (β) compared with the old version and generates TB persistence over a wider range of possum-carrying capacity landscapes for a fixed β.

Recommendations

- Area disease managers should use the revised SPM when developing their cases for declaring VCZs free in 2018.
- When running simulations, the default parameter values and standard adjustments should be used. These are described here and in the guidelines document.

1 Introduction

Landcare Research, Lincoln, was contracted by OSPRI to improve (and make more credible) the way in which TB persistence in possum populations is modelled using a spatially explicit model of TB epidemiology in possums (the Spatial Possum-TB model, SPM). The research involved an initial exploration of potential alternative mechanisms by which TB might persist in possum populations, then progressed to fundamentally changing the way in which contact between possums (and therefore TB transmission) is simulated with the SPM. The effect of those changes on model predictions was then explored. The work was conducted between May 2015 and July 2017.

2 Background

A spatially explicit model of the epidemiological dynamics of bovine tuberculosis (TB) in possums (SPM; Ramsey & Efford 2010) is used by OSPRI to predict the effect of possum control on TB prevalence in possums. Since 2011 it has become central to the Proof of Freedom (POF) framework (Anderson et al. 2013) used by OSPRI to declare areas free of TB in possums. However, area disease managers (ADMs) and others have progressively become concerned that the model predictions do not always fit well with their empirical observations of where TB is known to have occurred (and appeared to persist) in low-density possum populations.

Initially this concern appeared to mostly relate to model predictions that TB would disappear far more rapidly from possum populations after the implementation of possum control than ADMs considered was credible, thus generating overly optimistic estimates of the prior probability of TB freedom. However, a workshop with the ADMs in May 2015 clarified that the primary issue they had with the SPM was not the SPM predicting unrealistically rapid disappearance of TB in the face of control, but the lack of TB persistence at low density generally, including at possum carrying capacity (before control is applied). This broad concern emerged from the ADMs' experiences of running model simulations for vector control zones (VCZs) being considered for declaration of freedom and finding they could not get the disease to persist at the 'burn-in' (pre-control) phase, even though the ADMs had strong historical evidence of TB presence in possums in those VCZs.

Part of this apparent inability of the SPM to predict TB persistence at low possum densities is simply a consequence of OSPRI POF protocols specifying an upper limit to the value of the disease transmission coefficient (β) for each landscape run. One of the central assumptions of the SPM, and indeed of all Barlow's possum-TB models (1991, 1993, 2001), is that β is inversely proportional to carrying capacity (K), so that the product βK is constant between landscapes. This means that in very patchy, generally low-density habitats (such as farmland), the value of β must be increased substantially to predict TB persistence at carrying capacity. However, an increase in the value of β to greater than 1 was often required to predict TB persistence, which seems unrealistically high given that TB typically occurs at only low prevalences (2-5%) even in good possum habitat.

Based on those concerns, this project focused on making the SPM prediction less sensitive to the value of the disease transmission coefficient (β) used, so that for a given value of β it predicts persistence over a greater range of possum carrying capacity. As the project progressed, we identified that this could be achieved by fundamentally altering the way contact (and therefore transmission of TB) between individual possums is modelled in the SPM.

This major change in how the disease transmission process is simulated largely obviated the need to identify (and incorporate) alternative mechanisms by which TB could persist in possums at low density, but we report on some of these investigations where relevant. We also describe how predictions from the revised and recoded SPM affect predicted probabilities, and how the predictions can be integrated into the survey-then-control (StC) framework (Nugent et al 2014; Nugent, Sweetapple, Yockney, & Morriss 2017) that has recently been developed as an alternative approach to declaring TB freedom.

Given the multifaceted nature of the project, this report is structured as a series of more-or-less stand-alone sections, with each (to varying degrees) having its own background and methods, results and discussion subsections, as required.

3 Objectives

To improve the credibility and utility of the SPM in predicting TB persistence at low possum density, particularly in relation to real-world examples of TB persistence, by:

- identifying ways in which TB transmission in possums could be more credibly modelled than previously
- re-programming the SPM to incorporate a new approach to emulating TB transmission and, at the same time, adding new functionality, enabling the likelihood of TB persistence and/or re-establishment in recovering possum populations to be readily and realistically modelled
- exploring the effect of changing SPM assumptions on predicted probabilities of TB freedom
- demonstrating how the SPM can be used to inform a new approach to declaring TB freedom based on simultaneously conducting surveillance and control.

4 Assumptions of the Spatial Possum-TB Model that affect TB persistence

The SPM assumes that the maximum disease transmission rate βK is constant. This means that a different value for β (the TB transmission co-efficient) has to be assumed for each VCZ, because each VCZ has a unique landscape (and therefore possum carrying capacity, K).

The disease transmission coefficient is an *instantaneous rate* (as are the birth and death rates in the SPM),. Further, the transmission rate is not applied to the number or density of hosts in the SPM, as it would be in a non-spatial model. The SPM calculates contact rates between infectious and susceptible possums based on the height of their home range

kernels, so it deals in units of home range overlap rather than individuals, which makes it difficult to conceptualise.

Another key assumption of the SPM is that disease transmission is density-dependent (i.e. it scales with the density of infected possums). This means there is a threshold possum density for TB persistence. This threshold is relative to carrying capacity (i.e. is expressed as N_T/K) because of the earlier assumption that βK is a constant. The density dependence in disease transmission is non-linear because the home range kernels used to calculate contact rates are inversely related to possum density, whereby home range kernels expand at low possum density and contract with high possum density.

There is good empirical evidence that possum home range size varies inversely with possum density (Cowan & Clout 2000; Rouco et al. 2013; Whyte et al. 2014; Efford et al. 2016, and Barlow (1991) identified this mechanism as one that could maintain constant contact (and thus transmission rates) across landscapes with different possum densities.

A field study that looked at possum distribution following control (Sweetapple et al. 2016) also supports the theory that contact rates are maintained over a range of possum densities. This work showed that possums appear to be in contact (assessed by trapped location proximity) with the same number of neighbouring possums regardless of possum density. In theory, the density-dependent home range kernels already implemented in the SPM should maintain contact rates at low possum densities, and therefore make disease transmission less sensitive to the choice of β. However, contact rates were not maintained at low possum density in initial versions of the SPM due to the way kernel overlap was calculated (explained in detail in section 5.3).

The assumptions of constant βK and density-dependent transmission mean that *between* SPM simulations done at large spatial scales (i.e. VCZs), TB prevalence is kept constant, but *within* simulations at smaller spatial scales, TB prevalence varies with K, creating temporally persistent local 'hotspots' or focuses of disease. There is evidence of hotspots of TB infection in the field. For example, Hickling (1995) found that at the small scale (trap sites spaced 25 m apart), higher TB prevalence was found at sites where higher numbers of possums were caught. But when summarised at the spatial scale of transects/trap-lines (0.5–2 km in length), no such relationship was found (Barlow 1991; Coleman 1988; Hickling 1995). Likewise Nugent (2005) found no evidence for a positive relationship between TB prevalence and possum abundance at the trap-line scale in North Island indigenous forest.

The preceding supports the belief that the assumptions of constant βK and density-dependent transmission are reasonable (Barlow 1991). However, Nugent's (2005) data, evaluated at the larger, landscape scale (>5,000 ha), showed a positive relationship between TB prevalence and possum abundance. This relationship, at the scale at which most SPM simulations are run, potentially supports the counterargument for a constant β (and variable βK) between SPM simulations. On the other hand, the relationship used realised possum density (with variable control histories) rather than potential density (K), and all areas had broadly similar vegetation (mixed podocarp-broadleaved indigenous forest), and so we would expect carrying capacity to be similar. Another confounding influence on the patterns observed in the field is the effect of other wildlife hosts of TB (e.g. pigs, red deer, ferrets) on

TB dynamics in possums; these factors cannot be accounted for in single host species models such as the SPM.

Other probably unrealistic assumptions of the SPM that affect modelled TB persistence are that there is no individual heterogeneity in disease susceptibility or transmission – there is only a single 'infected' class – and 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. Together these assumptions mean that upon infection all individuals are immediately infectious and immediately have a chance of dying from the disease. Mechanisms to produce a more distributed waiting time (to delay progression through the stage) include making the disease mortality rate (α) dependent on time since infection, subdividing the infected class into multiple stages (e.g. a pre-clinical and clinical phase), or allowing individual heterogeneity in the infected stage (e.g. by drawing an individual's disease mortality rate from a distribution rather than it being the same for all).

A variation of the latter option (individual variation in disease progression) was identified by Tompkins and Holland (2015) as an important determinant of modelled TB persistence. They explored the potential for spatial clustering, correlation of disease and social parameters, and lesion resolution to drive disease persistence in wild animal populations. Their model was parameterised to approximate TB in possums and was based on empirical data from experimental TB transmission trials in Orongorongo Valley possum populations. This and previous studies had shown that a few individuals can resolve TB lesions, surviving in excess of 2.5 years (Corner & Norton 2003; Nugent et al. 2013; Ramsey and Cowan 2003). They found that modelled TB persistence was significantly increased by the inclusion of lesion (but not disease) resolution but was relatively unaffected by both the spatial clustering of individuals and the correlation of disease and social parameters in their simulations. They recommended that

> the current project, already commissioned by OSPRI, to update the individual-based possum spatial model used to guide proof-of-freedom and control applications (R-10791 Identifying potential mechanisms of TB persistence) should consider including the potential for lesion resolution (and thus the much longer survival of some infected individuals) at field observed rates. If the level of lesion resolution is as high as we suggest it could be, this will likely provide a more accurate reflection of disease dynamics , and (we expect) prediction of TB persistence in more low-possum-carrying areas or for longer after possum control.

Note, however, that their models differed from the SPM in several key ways: possum population dynamics were not included; infected possums were divided into latent and infectious stages; and contact rates were modelled as the area of home range overlap, then normalised to give the same daily probability of contact with neighbours (akin to frequency- rather than density-dependent transmission). Also, they assumed a higher proportion of infected possums survived long term (more than a year) than has been observed in field studies of the fate of naturally or artificially infected possums. Corner and Norton (2003) monitored 90 infected possums, of which all but two died. Importantly, they argued that the possums were no longer infected (i.e. had resolved the disease, not just the lesions), in which case they would not have shown a cycle of infectiousness. We conclude that although their modelling identified that lesion resolution followed by a subsequent return to

infectiousness could plausibly increase the likelihood of TB persistence at low density, the available epidemiological evidence does not strongly support this.

5 Changes made to the Spatial Possum-TB Model

Based on Tompkins & Holland 2015, simulation of alternative mechanisms based on spatial clustering or differences in sociality between individual possums seemed unwarranted. The disease resolution mechanism they identify was also rejected, partly for the reasons discussed above. Instead, the main change made to the SPM was to alter the way contact rates are calculated in the model so that they are maintained at low possum density. This mechanism was preferred because:

- it was simpler to incorporate into the existing model structure (cf. incorporating four new classes and calculating four times as many transitions between classes needed for lesion-resolution modelling)
- no new parameter value estimates were required (cf. estimating the proportion of population resolving lesions and transition rates through infection classes)
- a mechanism that maintains possum contacts at low density produces a model that is less sensitive to the choice of β, which is closer to the preferred model construct of a constant β value across all landscapes
- user feedback from the 5-year POF review meeting in November 2016, where the new contact rate function was presented, was against introducing further complexity to the model.

In addition, we identified a need to update the default value for the disease mortality rate parameter (based on new empirical data), and to add a function to re-introduce TB-infected possums into modelled populations in order to enable easier exploration of disease recovery in post-control populations.

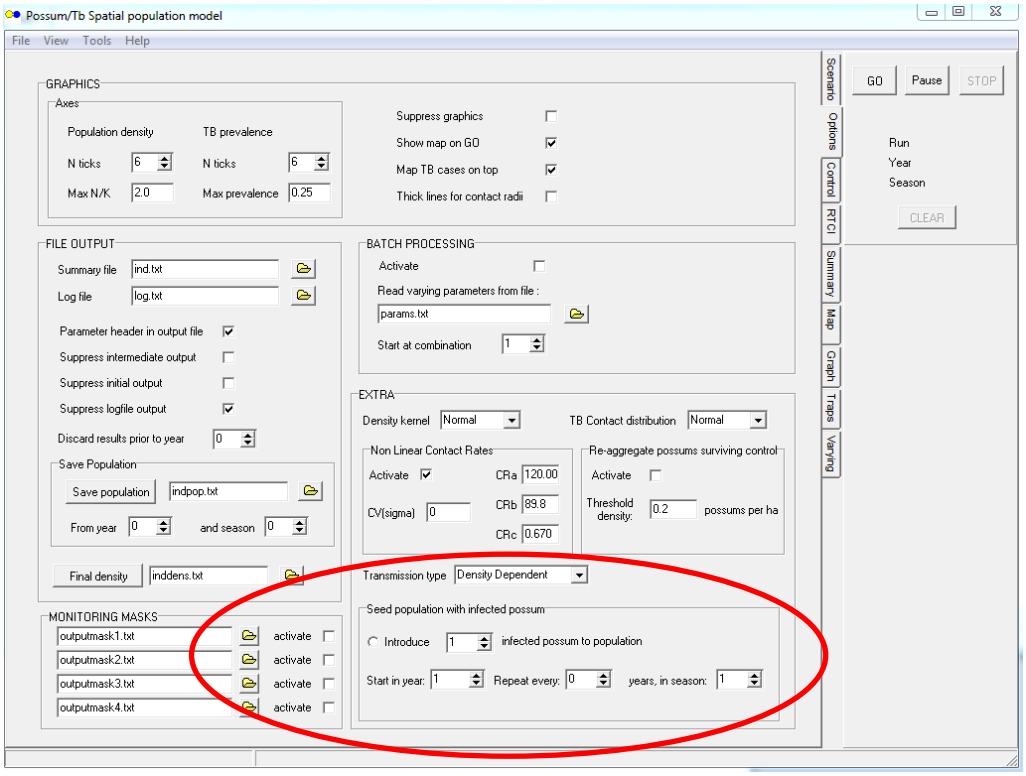
5.1 Ability to 'seed' low numbers of TB-infected possums incorporated into the SPM

This functionality gives the user the ability to explore the consequences of re-introduction of the disease (e.g. by the dispersal of infected possums from surrounding uncontrolled areas, or from infection by sympatric host species such as ferrets), and to evaluate what number of infected possums is sufficient to compromise TB eradication in a given landscape. Note that although this seeding *is* a method of inducing TB persistence, it should not be used by default when trying to model endemic disease and generate prior probabilities of TB freedom for the POF process. Instead, it should be used to evaluate relative risks to TB elimination on a case-by-case (VCZ) basis if the subjective assessment phase of the POF process indicates re-introduction is a possibility.

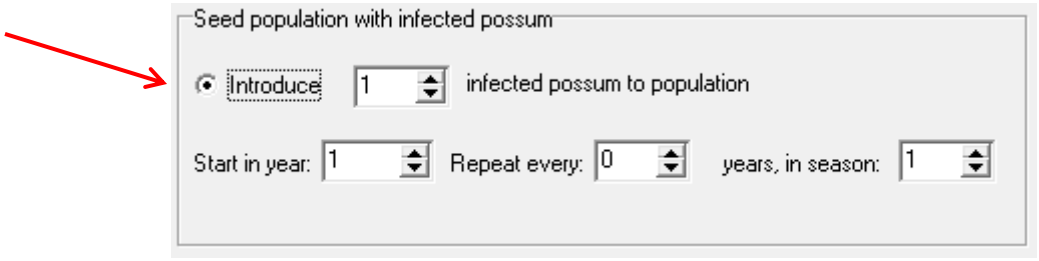
The ability to seed low numbers of TB-infected possums has been incorporated into the SPM, and the amended code has been uploaded to the TBfree bitbucket repository:

https://bitbucket.org/ospritbfreeproofoffreedom/spatial-possum-model

This functionality can be accessed on the 'Options' tab of the SPM interface in a box titled 'Seed population with infected possums':



To implement, click the 'Introduce' radio button:



Then enter the per annum number of TB-infected possums you wish to add to the simulated population and the year of the first introduction, 'Start in year'. In the input above 'Repeat every' is set to zero, which results in a one-off introduction; you can schedule regular introductions by increasing this value. The season of introduction can also be altered (but is the same for each year); by default it is set to season 1, which is the same season that juveniles are recruited and disperse within the population.

5.2 Increasing the default value of the disease mortality rate (α)

The default disease mortality rate was increased from $\alpha = 1$ to $\alpha = 2.4$, corresponding to a decrease in the assumed mean infected period from about 1 year to about 5 months. This increase was in light of new data on the survival times of infected possums (Nugent et al. 2013) and to ensure compatibility with parameter values assumed in the rest of the POF process (specifically the duration of sentinel exposure to TB-infected possums).

Nugent, Sweetapple, Yockney, & Whitford (2017) in a discussion document regarding POF parameter values argue that

> However, the duration of infection in possums is now considered to average only 5 months rather than the 8–12 months formerly assumed. This is based on combining the average of 3.5 months for survival times for clinical stage possums with detectable lesions (Ramsey & Cowan 2003; Norton et al. 2005; Nugent et al. 2013) and a direct estimate of 1.7 months for the time to reach clinical stage infection (Nugent et al. 2013).

Increasing the disease mortality rate had a substantial negative effect on TB persistence in the SPM, which is not surprising given that this is the parameter value to which the SPM is most sensitive (Barron 2012). An increased disease mortality rate results in a shorter infectious lifetime for the possum. Possum control reduces the supply of susceptible possums, reducing transmission/input to the infected cohort. At the same time, losses from the cohort are occurring faster through increased disease mortality, and possums are dying before they get an opportunity to transmit the disease to their neighbours, resulting in rapid TB drop out and faster progression to TB freedom.

This outcome of even faster disappearance of TB after the imposition of possum control than under previous assumptions obviously exacerbates rather than alleviates the perceived concern that originally prompted this project. However, there is little recent evidence that actually contradicts rapid eradication. Neither Latham and Nugent (2017) nor Nugent,

Sweetapple, Yockney and Whitford (2017) found any evidence of TB persisting anywhere in face of sustained control. The only areas in which TB was found in possums occurred where control was poor (and possum density high), or where there was an infected uncontrolled possum population nearby.

5.3 Changing the way contact rates are modelled

The SPM has modelled possum home range use by using a bivariate Gaussian kernel function (Ramsey & Efford 2010) to compute a utilisation distribution (UD) on a discrete grid at a 50 × 50 m cell resolution:

$$h(|x' - x|) = \frac{1}{H} \exp\left(\frac{|x'-x|^2}{2\sigma^2}\right)$$

where the parameter σ is a scalar for home range size ($2.45\sigma \approx$ 95% home range radius) and the numerator in the exponent is the squared distance between the home range centre grid cell (x') and the grid cell being evaluated (x). The parameter H is a normalisation constant so that the area under the curve or the sum of the UDs across the home range is equal to 1 (i.e. the influence of one possum sums to 1).

For each grid cell in the modelled landscape the UDs are summed across the population to give a density surface (including all possums) or an infection surface (counting only infected possums), which is used to estimate density-dependent population growth rates and disease transmission rates, respectively. However, for disease transmission, the height of the infection surface is only assessed in the central home range cell (x') of the susceptible possum, *not* across the susceptible possum's entire home range.

In the original model, in which home range scalars were constant, the infection surface was calculated from an un-normalised kernel. This meant the height of the infection surface when two possums' home ranges overlapped completely was 1 ($x'–x = 0$ and $exp(0) = 1$). However, when density-dependent home range estimators (i.e. the 'Non-linear contact rates' box was clicked, resulting in σ being based on density) were becoming routinely used for simulations, the infection surface was changed to use normalised kernels. This was considered necessary because the large σ values predicted at low possum density meant that, unrealistically, the potential force of infection (area under the infection kernel) from a single infected possum was 13 times as much for a possum with σ = 115 (home range size = 26 ha) compared to a possum with σ = 30 (home range = 1.7 ha). Assessing the infection surface only at the susceptible possum's home range centre means that possums that overlap at the margins of their home ranges have a very low rate of contact, and using normalised kernels makes this value smaller still so that the chances of contact (and transmission) are remote (Figure 1).

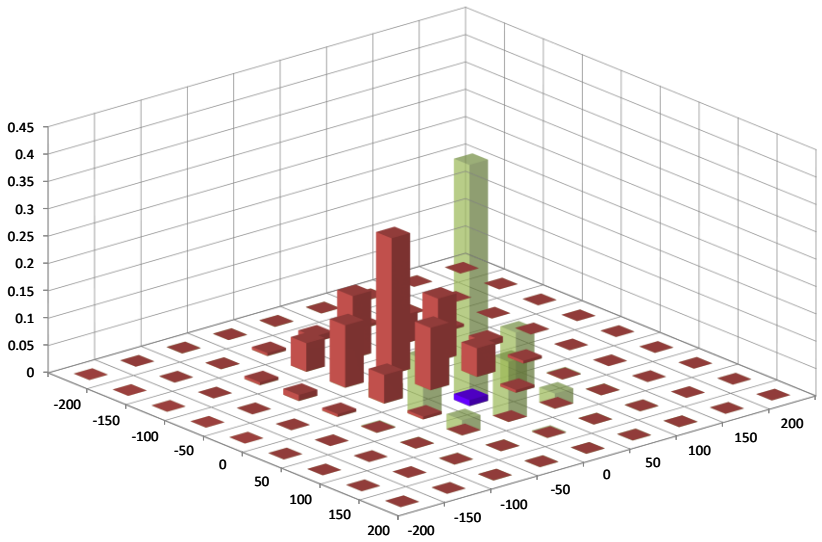


Figure 1 Schematic of the current SPM method for calculating possum contact rates (*cr*). The red bars represent the UD for an infected possum, the green bars the UD for a susceptible possum. The infection risk experienced by the susceptible possum is estimated from the height/value of the infected possum's UD at the susceptible possum's home range centre (here at $x = 0$, $y = 50$), indicated by the purple bar, $cr = 0.01$. The proposed metric (BA) is based on the summed mean height of all cells that the infected and susceptible possums overlap, $cr = 0.35$.

To remedy this, other methods of quantifying home range overlap were investigated, using the overlap metrics detailed in Fieberg et al. 2005, and compared with the current metric used by the SPM (Table 1). Of these metrics, the volume of intersection (VI) and Bhattacharyya's affinity (BA) metric looked most appropriate because they were based on the *joint* UDs of the two individuals potentially in contact (under the assumption that they use space independently of one another) and they scale from 0 (no overlap) to 1 (complete overlap).

Table 1 Comparison of home range overlap metrics between two possums (i and j) over a range of home range sizes (scalar σ and 95% kernel area a), and at increasing distance apart $d_{i,j}$. The old SPM metric is based on the height of the infected possum's (i) home range kernel at the susceptible possum's (j) home range centre (x_j = 0,y_j = 0). Note that with old SPM metric, potential contacts decline to zero if the possums are located 150 m apart, whereas potential contacts are maintained at this distance for the other home range overlap metrics

Home range scalar σ and 95% kernel area a (ha)		Distance between possums (m)	Old SPM metric		Home range overlap metrics described in Fieberg & Kochanny 2005					
σ_i (= σ_j)	a^i (=a^j)	di,j	$P(i \mid x_j=0, y_j=0)$	$P(i \mid x_j=0, y_j=0)$ std.	$HR_{i,j}$	$PHR_{j,i}$	PHRi,j	VI	BA	UDOI
30	1.7	0	1.00	0.44	1.00	1.00	1.00	1.00	1.00	24.30
30	1.7	50	0.25	0.11	0.65	1.00	1.00	0.34	0.70	9.84
30	1.7	100	0.00	0.00	0.34	1.00	1.00	0.17	0.25	1.24
30	1.7	150	0.00	0.00	0.09	1.00	1.00	0.01	0.04	0.03
50	4.7	0	1.00	0.16	1.00	1.00	1.00	1.00	1.00	7.88
50	4.7	50	0.61	0.10	0.79	1.00	1.00	0.60	0.88	5.58
50	4.7	100	0.14	0.02	0.59	1.00	1.00	0.36	0.61	2.37
50	4.7	150	0.01	0.00	0.40	1.00	1.00	0.12	0.32	0.60
70	9.2	0	1.00	0.08	1.00	1.00	1.00	1.00	1.00	4.03
70	9.2	50	0.77	0.06	0.85	1.00	1.00	0.72	0.94	3.22
70	9.2	100	0.36	0.03	0.70	0.99	0.99	0.49	0.77	1.98
70	9.2	150	0.10	0.01	0.56	0.97	0.97	0.27	0.56	0.93

We also explored the consequences of density-dependent home range size and the distribution of possum home range centres (random versus aggregated) on home range overlap metrics using a simplified model coded in R programming language (R Core Team 2016). This model did not include any possum population or disease dynamics, but simply modelled the home range kernels of individuals across a range of possum density (1 to 10 possums per hectare), for Poisson (random) or negative binomial (aggregated) distributed home range centre locations, and with or without density-dependent home range areas. Home range overlap statistics, the number of neighbours within 100 m, and the mean distance to nearest six neighbours were calculated from 10 randomly selected individuals. Two hundred replicate simulations were run for each parameter combination.

These simulations showed that having density-dependent home range areas had only a minor positive influence on home range overlap metrics at low possum density (<5 possums per hectare), narrowing to no difference at higher possum density (Figure 2b). However if possums' home range centres were aggregated/clustered in space, this resulted in a large increase in overlap metrics consistent across a range of possum densities (Figure 2b), and this was due to individuals having a greater number of neighbours in close proximity (<100 m between home range centres) to them (Figure 2a).

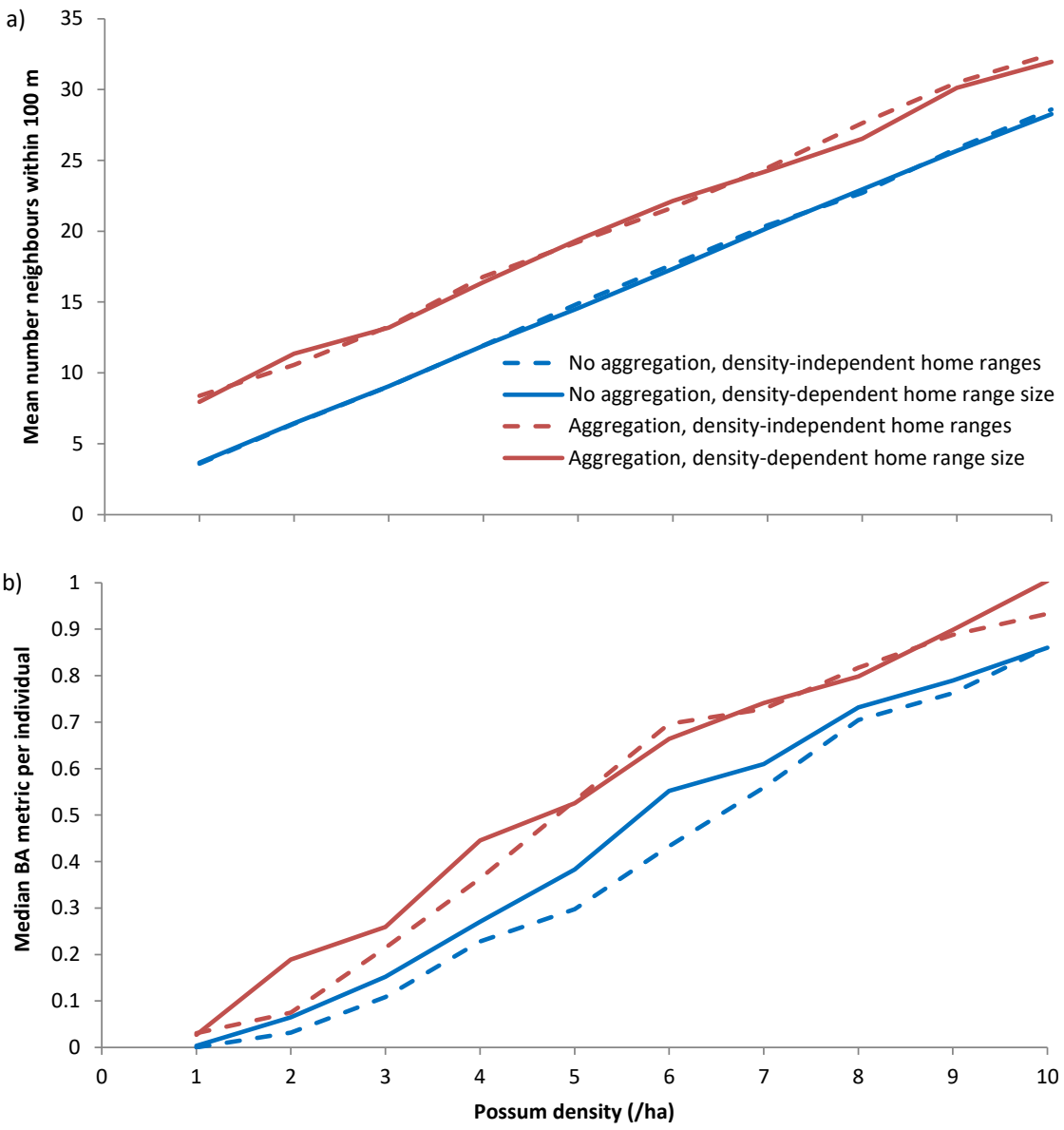


Figure 2 Relationship between a) the mean number of neighbours with home range centres less than 100 m from a focal individual, and b) the median index of home range overlap (BA) with a focal individual and possum density. Dashed lines show the relationship under the assumption that home range size is density independent, solid lines for density-dependent home range size. Blue lines show the relationship when home range centres are randomly located in space, red lines if they are aggregated in space.

The VI and BA home range overlap metrics showed a clear advantage over the original SPM metrics, in terms of both the overall magnitude of potential overlap/contact and maintenance of overlap at low possum density (Figure 3). Using the original SPM home range overlap metric, there appeared to be a threshold density of four possums per hectare before significant contact occurred, whereas for the BA metric potential contacts occurred over the range of possum densities tested and showed a linear relationship with increasing possum density (Figure 3). The BA method of calculating home range overlap was subsequently coded into an updated version of the SPM. The amended code has been uploaded to the TBfree bitbucket repository:

https://bitbucket.org/ospritbfreeproofoffreedom/spatial-possum-model
The BA metric was chosen over the VI metric simply because it was easier to integrate into the existing code. The extra calculations result in the updated SPM taking approximately 1.5 times longer to run a simulation compared to the original version.

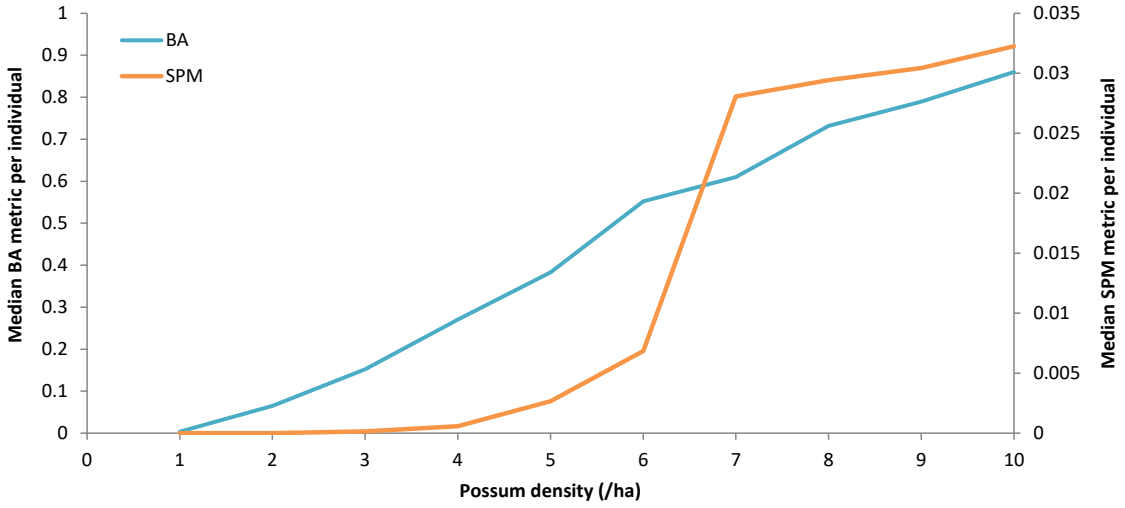


Figure 3 The median BA (blue line) and old SPM (orange line) overlap metric estimated across a range of possum densities and assuming density-dependent home range size and no aggregation of home range centre locations.

Note that we are making the assumption that contact rates (and therefore direct disease transmission) scale with joint space use. Studies on white-tailed deer (Schauber et al. 2007) and racoons (Robert et al. 2012) have shown that measures of spatial overlap and contact rates are positively correlated, although the former study showed that direct contact rates are also strongly influenced by social group membership. In the absence of any contrary information (and low sociality in possums) we have assumed that possum-to-possum contact rates *are* positively correlated with home-range overlap and that the model mechanisms of expanding home ranges at low possum density combined with contact rates derived from the BA metric of home range overlap will enable TB persistence at low possum abundance in the SPM.

6 The effect of changes to the SPM on predictions of the prior probability of TB freedom

6.1 Introduction

Predictions of TB prevalence and the prior probability of TB freedom following simulated possum control were compared using the old and new versions of the SPM for four VCZs. The only differences between the SPM developed here and that routinely used by ADMs are that:

- the latest version has contact rates between possums (used for calculating TB transmission) estimated from BA, which calculates joint space use based on the overlap between an infected and susceptible possum's home range kernels (cf. just in the susceptible individual's home range centre for the old version)
- the default disease mortality rate (α) has been increased from α = 1 to α = 2.4 based on an updated estimate of the duration of TB infection in possums (around 5 months, cf. 12 months).

The VCZs chosen for the comparison were identified by ADMs as ones in which they had conducted simulations using the old SPM (subject to the constraints imposed by OSPRI) but found that it failed to predict persistence of TB in uncontrolled populations despite historical records of TB in wildlife vectors at that location, a moderately high amount of possum habitat (>40% of area), and a modest possum carrying capacity (over two possums per habitat hectare). The four VCZs were:

- Mangakino (north and central North Island)
- Waihoki (southern and eastern North Island)
- Ure-Medway East (northern South Island)
- Bare Hill (southern South Island).

6.2 Methods

Possum carrying capacity maps (*K*-maps) for these VCZs were supplied by OSPRI staff, and in two cases (Mangakino and Waihoki) the estimated possum reduction estimates (%Kill) were also supplied. Default parameter values were used for all simulations (see Appendix 1), apart from the disease mortality rate (α), which was set at either 1 or 2.4 per annum, and the disease transmission coefficient (β), which was adjusted to generate approximately 2% TB prevalence at equilibrium possum abundance (i.e. in the absence of possum control). The probability of TB freedom (P_{Free}) was estimated as the proportion of replicates in which TB went extinct (out of 1,000 replicates per simulation).

6.3 Results

6.3.1 Mangakino

Mangakino is a large VCZ of 23,246 ha with possum habitat covering 42% of the area at an average carrying capacity of 2.2 possums per hectare within possum habitat. There is a historical presence of TB possums in the area. A %Kill file detailing possum control was supplied, and it assumed a 90% initial cull followed by five maintenance controls of 50–70% over the following 8 years. The old version of the SPM required β = 1.85 (i.e. above the maximum of 1 permitted under OSPRI protocols) to generate 2% TB prevalence in the absence of control, whereas the new version required β = 0.134 or β = 0.286, with α = 1 or α = 2.4, respectively. Predicted TB dynamics were very similar between the old and new versions of the SPM with the same disease mortality rate. TB was predicted to decline faster following control in the model version, with the higher disease mortality rate (α = 2.4) compared with the other scenarios, resulting in faster progression to TB freedom.

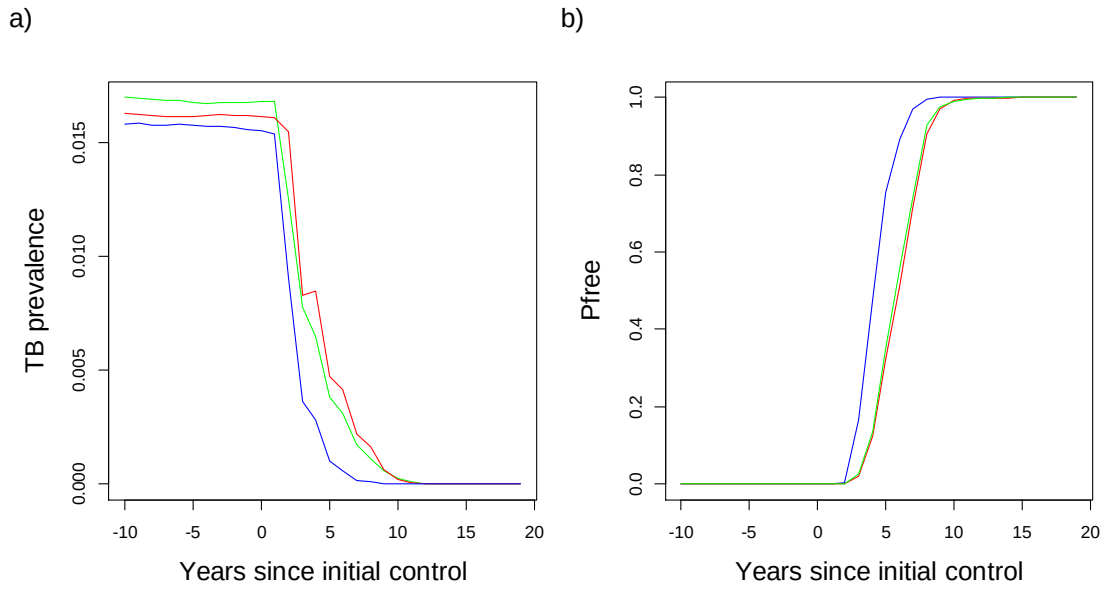


Figure 4 SPM predictions of a) TB prevalence and b) probability of TB freedom for Mangakino VCZ under different model versions and parameter settings. Red line = old contact rates calculated from the home range centre of the susceptible possum and α = 1.0; green line = new contact rates calculated from the joint distribution of susceptible and infectious possums and α = 1.0; and blue line = new contact rates and α = 2.4.

6.3.2 Waihoki

Waihoki is a relatively large VCZ of 12,475 ha with possum habitat covering 44% of the VCZ with a carrying capacity of 2.6 possums per habitat hectare. There are historical records of TB in possum populations as well as other vectors (deer and pigs). The old version of the SPM required β = 1.73 to generate 2% TB prevalence in the absence of control, and the new version β = 0.135 or β = 0.29, with α = 1 or α = 2.4 respectively. With only moderate control (percentage kills of 60, 75 and 18% in three successive years), TB freedom ($P_{free} \geq 0.95$) was predicted by the old model version at year 12 post-control and in the new version with α =

2.4 even faster: by year 9 post-control. However, in the new SPM version with lower disease mortality rates, TB freedom was never achieved (green line: Figure 5b).

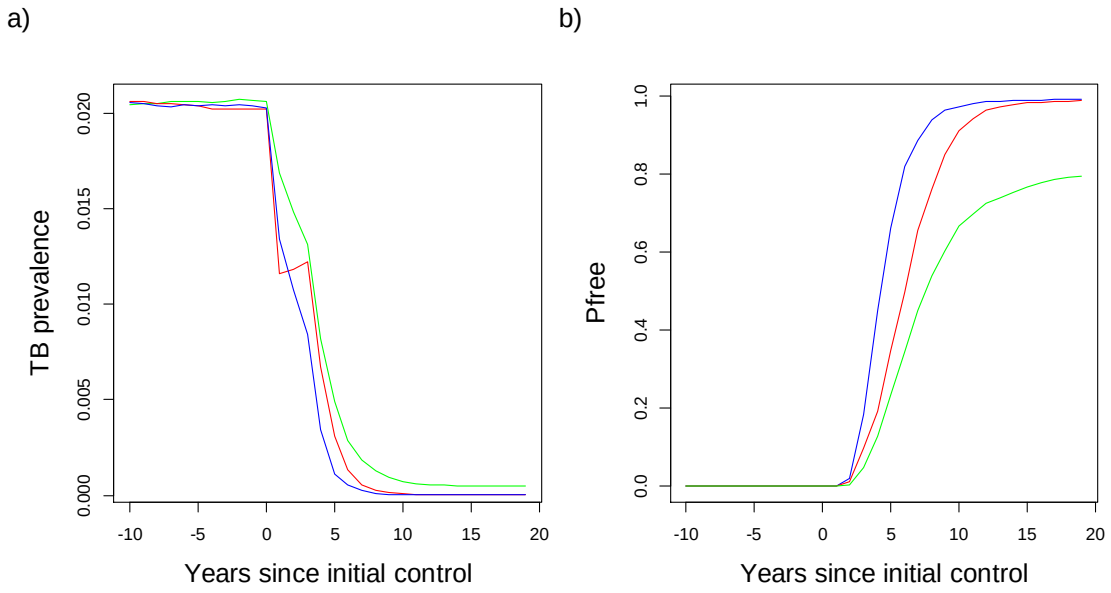


Figure 5 SPM predictions of a) TB prevalence and b) probability of TB freedom for Waihoki VCZ under different model versions and parameter settings. Red line = old contact rates calculated from the home range centre of the susceptible possum and α = 1.0; green line = new contact rates calculated from the joint distribution of susceptible and infectious possums and α = 1.0; and blue line = new contact rates and α = 2.4.

6.3.3 Ure-Medway East

This is a large VCZ of 19,073 ha with patchy possum habitat covering 47% of the VCZ with a carrying capacity of 2.2 possums per habitat hectare. TB has never been found in possums in this VCZ, but it has been found in ferrets and cattle herds historically. The old version of the SPM needed β = 1.85 to generate 2% TB prevalence in the absence of control, and the new version β = 0.139 or β = 0.301 with α = 1 or α = 2.4 respectively. The simulation of three good (90, 80 and 80% kills) control operations spaced 5 years apart resulted in rapid elimination of TB under all scenarios, with the shortest time to $P_{free} \geq 0.95$ in 6 years predicted by the model with the new contact rate function and the highest disease mortality rate.

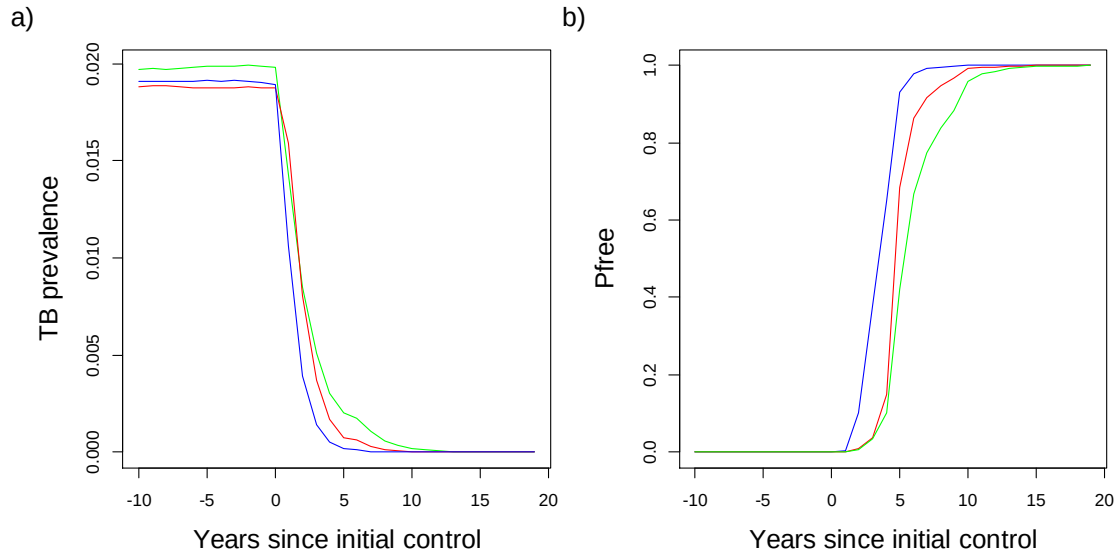


Figure 6 SPM predictions of a) TB prevalence and b) probability of TB freedom for Ure-Medway East VCZ under different model versions and parameter settings. Red line = old contact rates calculated from the home range centre of the susceptible possum and α = 1.0; green line = new contact rates calculated from the joint distribution of susceptible and infectious possums and α = 1.0; and blue line = new contact rates and α = 2.4.

6.3.4 Bare Hill

The Bare Hill VCZ is a moderate-sized VCZ at 8,703 ha, with areas of significant possum habitat covering 68% of the VCZ with an average carrying capacity of 3.6 possums per habitat hectare. TB has never been found in possums in the area but it has been found in ferrets there. The old version of the SPM needed β = 1.29 to generate 2% TB prevalence in the absence of control, and the new version β = 0.120 or β = 0.259 with α = 1 or α = 2.4, respectively. TB freedom (P_{free} > 0.95) was predicted under all model versions given three good control operations (with 90, 80 and 80% kills) spaced 5 years apart. Assuming a high disease mortality rate (α = 2.4) resulted in predictions of TB freedom 4 years earlier than that predicted under a low disease mortality rate (α = 1).

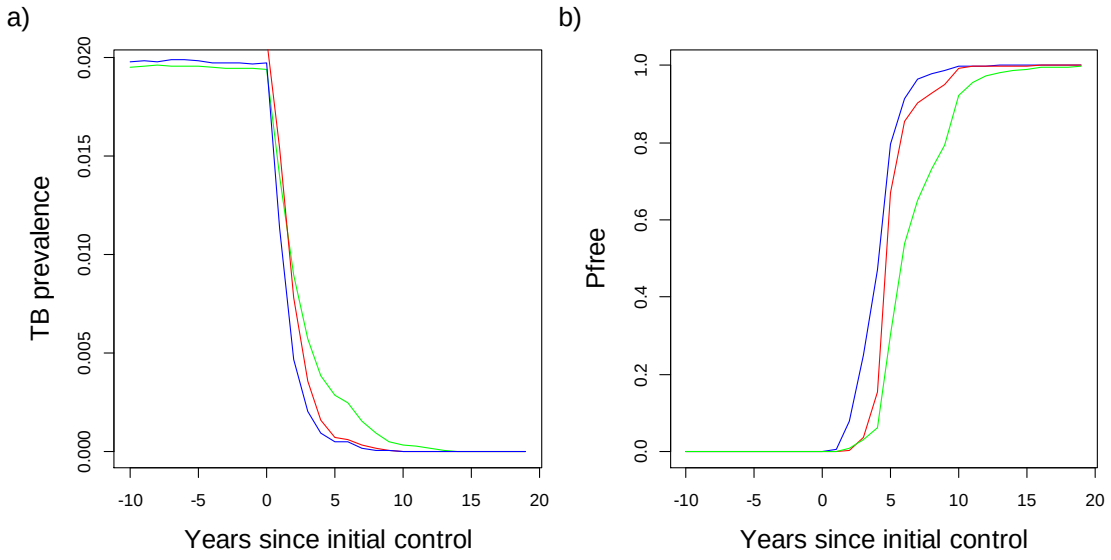


Figure 7 SPM predictions of a) TB prevalence and b) probability of TB freedom for Bare Hill VCZ under different model versions and parameter settings. Red line = old contact rates calculated from the home range centre of the susceptible possum and α = 1.0; green line = new contact rates calculated from the joint distribution of susceptible and infectious possums and α = 1.0; and blue line = new contact rates and α = 2.4.

Table 2 Summary of SPM input and output for the four VCZs

VCZ	Area (ha)	Prop. habitat[1]	Mean K[2]	Contact rate function	β	α	TB prev.[3]	Yrs to $P_{Free} \geq 0.95$
Mangakino	23,246	0.42	2.2	old	1.850	1.0	0.016	9
				new	0.134	1.0	0.017	9
				new	0.286	2.4	0.016	7
Waihoki	12,475	0.44	2.6	old	1.730	1.0	0.020	12
				new	0.135	1.0	0.021	NA
				new	0.290	2.4	0.020	9
Ure-Medway East	19,073	0.47	2.2	old	1.850	1.0	0.019	9
				new	0.139	1.0	0.020	10
				new	0.301	2.4	0.019	6
Bare Hill	8,703	0.68	3.6	old	1.290	1.0	0.021	9
				new	0.120	1.0	0.020	11
				new	0.259	2.4	0.020	7

[1] Proportion of the VCZ area classified as possum habitat ($K > 0$)

[2] Possum carrying capacity per hectare, assessed over possum habitat only

[3] TB prevalence assessed at equilibrium (in absence of possum control)

6.4 Discussion

In all four VCZs, TB persistence could be achieved by changing the disease transmission parameter value (β). For the old SPM, however, the β required to predict TB persistence in the absence of control was substantially higher than the maximum value permitted under OSPRI's POF protocols for using the SPM, particularly when possum density was only about two possums per hectare. We conclude that the failure of the SPM to predict persistence during simulations conducted by the ADMs was simply because of the OSPRI constraint.

The SPM version with the new contact rate function was much less sensitive to the value of β required to get TB to persist; i.e. a single value of β = 0.29 predicted TB persistence over all four VCZs simulated, although we further adjusted the value to give the desired 2% TB prevalence at equilibrium.

Changing the possum contact rate function had very little effect on TB dynamics in response to control, at least when control efficacy was high. In all four scenarios possum numbers were reduced to below the threshold for disease persistence and the few infected possums remaining post-control did not have enough neighbours to infect and re-establish disease, which eventually died out when they did. The effect of the altered contact function was only apparent in the Waihoki VCZ simulations where control efficacy was poor. Here disease was maintained and TB freedom was not achieved due to the combination of maintenance of contact rates at low possum density when it was assumed possums had longer infectious lifetimes (α = 1) than now appears to be the case.

Increasing the disease mortality rate had a far greater effect on TB dynamics than the altered contact rate function. An increased disease mortality rate results in a shorter infectious lifetime for the possum. Possum control reduces the supply of susceptible possums, reducing transmission/input to the infected cohort. At the same time, losses from this cohort are occurring faster through increased disease mortality and possums are dying before they get an opportunity to transmit the disease to their neighbours, resulting in rapid TB drop-out and faster progression to TB freedom.

7 Adaption of revised SPM for use in a survey-then-control framework

7.1.1 Introduction

The current POF framework uses a Bayesian belief-updating approach in which the predictions of the SPM are used to generate a prior belief about the probability that TB is absent from an area, given the ability of the area to support possums (i.e. K) and the amount and duration of control that has been applied, as per the simulations in the preceding section. That belief is then updated using empirical surveillance data. Major weaknesses with this approach are that actual possum carrying capacities are not known for most areas, and the extent to which carrying capacity varies within major habitat type is rarely if ever known. In addition, assessment of the efficacy of historical control is often difficult because percentage kill is rarely measured, and even residual post-control abundance is often not assessed (especially after large-scale aerial poisoning). For these

reasons, OSPRI protocols require that a minimum amount of empirical surveillance is conducted to support a case for declaring possums free of TB. However, the cost of such surveillance can be prohibitively high in mountainous forested areas.

A modified 'survey-then-control' (StC) approach has therefore been developed and field tested (Nugent et al 2014; Nugent, Sweetapple, Yockney, & Morriss 2017). This primarily aims to reduce the amount of surveillance required by measuring control efficacy (%Kill) just after measuring surveillance sensitivity and then imposing control. This StC approach can still require an assessment of the prior probability of TB freedom, so SPM modelling is still needed.

The current formulation of the POF Bayesian updating framework used to combine empirical surveillance data with prior beliefs/estimates of the probability of TB freedom (P_{free}) is dichotomous. It assumes that TB is either absent or present in one and only one possum (or sometimes two, and only two, possums). The assumption that TB can only be present in just one (or just two) possums is unrealistic, so a more realistic formulation would use a probability distribution for the number of TB-infected possums present (i.e. the respective probabilities that there are 0, 1, 2, 3, 4… *n* TB-infected possums present in a population of *N* possums). That distribution would then be updated by empirical surveillance data, as usual. Because this formulation assumes that unless TB has been eradicated there will be a non-zero probability of there being more than one TB possum present, and because it is substantially easier to detect TB when two or more TB possums are present, empirical surveillance in which TB is not detected has a greater effect on increasing P_{free} than under the current formulation when the design prevalence is set at 1.

In addition to this change in the underlying theoretical basis of the POF framework, the new StC approach further adjusts the probability distribution for the number of TB possums for the probability that TB possums will have been killed during a subsequent control operation for which an estimate of the percentage kill is available.

The StC approach therefore requires a way of generating the prior probability *distribution* for the number of TB-infected possums remaining in a population. Unfortunately, there are no empirical data from which such a distribution could be derived. It would require a large amount of surveillance data from a range of areas in which possum control had reduced possum numbers to a low level but in which TB was still present and in which TB possums were detected in various numbers. No such data set currently exists.

The alternative is to choose an appropriate theoretical distribution, but again the empirical evidence is too sparse to determine which distribution might be appropriate. We therefore used the SPM as the best available summary of what is known and assumed about TB epidemiology and used simulation to predict numbers of residual (post-control) TB-infected possum numbers to generate 'post-declaration' probabilities of TB persistence. We show how these can be generated using the revised SPM (with its updated contact function and disease mortality rate).

7.1.2 Methods

The revised SPM was used to simulate the Hokonui Hills VCZ possum population with the standard *K*-map, default parameters, a transmission rate of β = 0.2 (to give approximately 2% TB prevalence at equilibrium), and a control history of: 80, 95 and 10% possum kill in years 1, 9 and 10, respectively. The number of simulations where TB went extinct was divided by the number of replicates (n = 1,000) to estimate a probability of TB freedom (P_{free}) for each year since control. For each of these annual P_{free} estimates, the frequency distribution of the number of TB-infected possums was calculated. This predicted distribution of TB-infected possums was subsequently used to calculate the joint probability (distribution) of an infected possum evading detection in the possum survey *and* surviving control (Nugent et al. 2014; Nugent, Sweetapple, Yockney, & Morriss 2017). The same process was repeated for two other VCZs, Milltown South and Scargill, which represent a mostly forested and a patchy farmland VCZ, respectively.

Standard distributions, the Poisson and the negative binomial were fitted to these residual possum distributions using the 'fitdistrplus' package in the R statistical environment (Delignette-Muller & Dutang 2015).

7.1.3 Results

Examples of the residual TB-infected possum distributions generated by the SPM are shown below. These distributions are for the Hokonui Hills VCZ, assessed at years 9–14 (where the last of three controls was simulated in year 10).

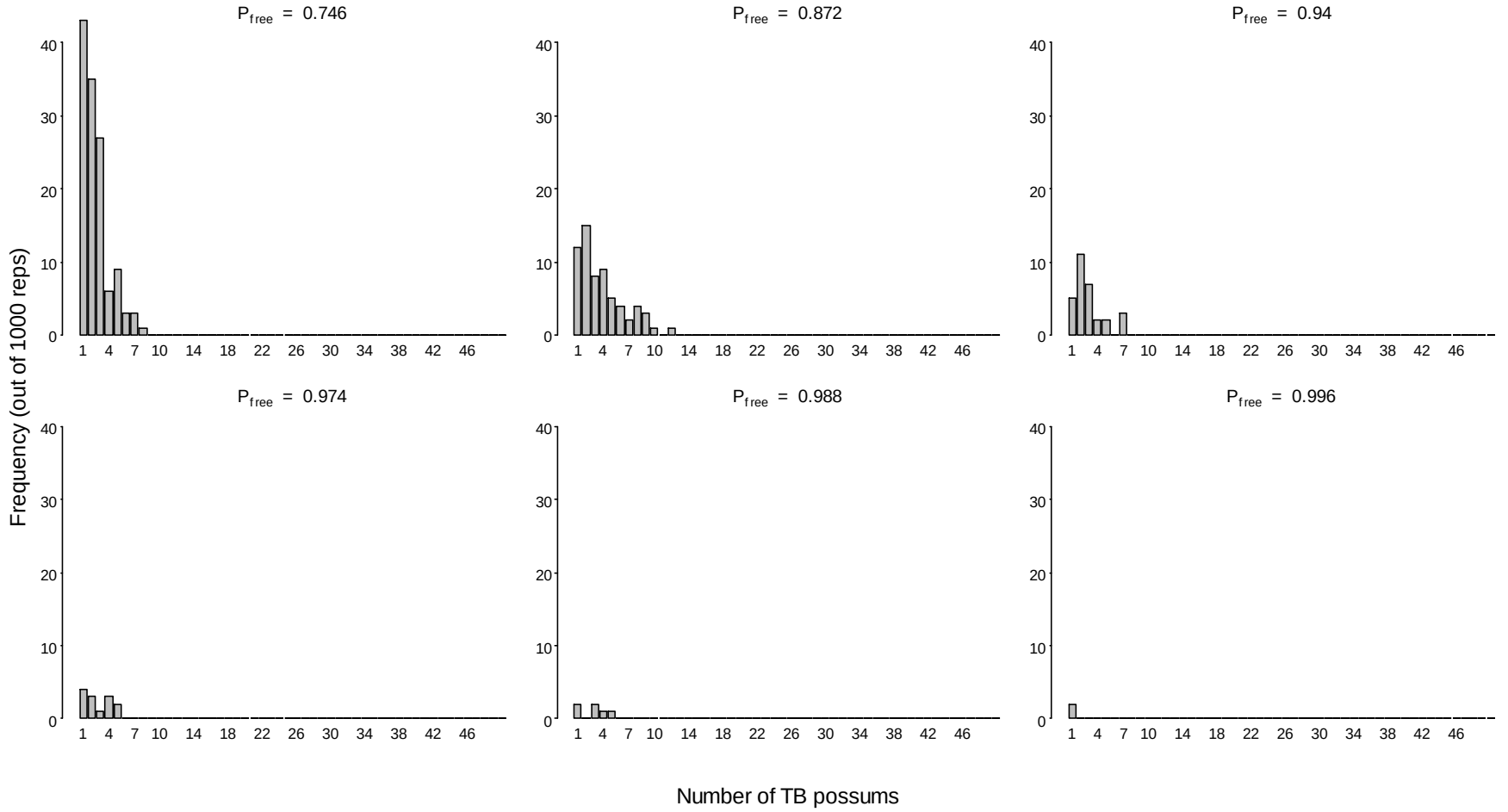


Figure 8 Frequency distribution of the number of TB-infected possums predicted to persist in a population (despite previous possum control) from 1,000 SPM simulations of the Hokonui Hills VCZ. The P_{free} estimate is the proportion of the 1,000 simulations in which there were no TB-infected survivors.

Negative binomial distributions were fitted to the residual TB-infected possum distributions generated by the SPM for the Milltown South and Scargill VCZs. The negative binomial distribution was a superior fit compared to the Poisson distribution due to the large number of zeroes in the data. At very high P_{free} values (>0.99), even the negative binomial could not satisfactorily fit the empirical distribution as there were only 10 or fewer non-zero values, but this lack of fit is inconsequential because OSPRI will be using the distributions only when P_{free} is below 0.95

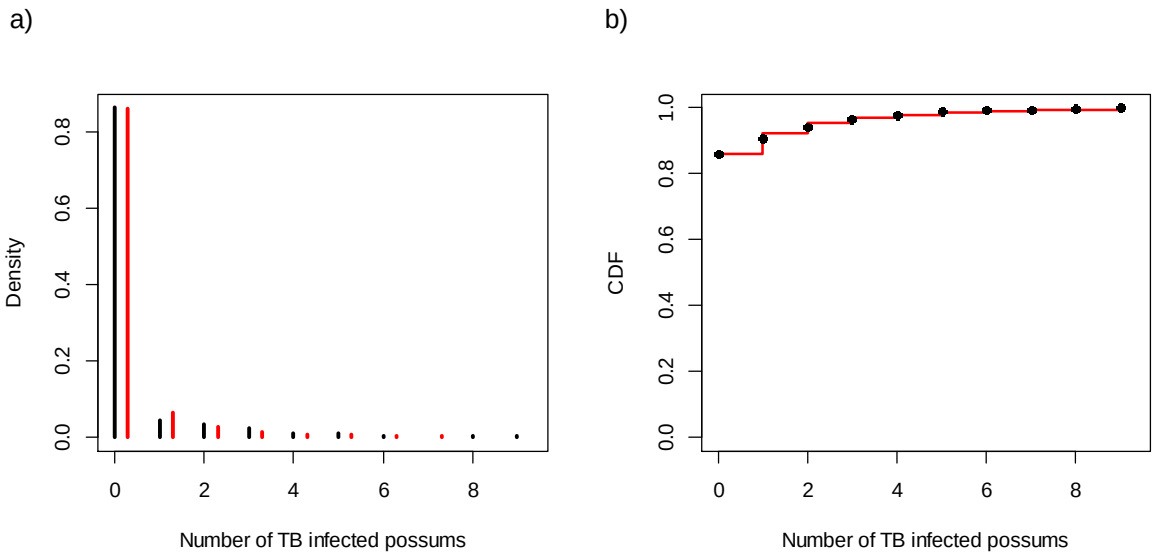


Figure 9 Example of an empirical (black bars and points) and fitted (red bars and line) a) probability density function and b) cumulative distribution function (CDF). The empirical data were SPM output of the number of residual TB-infected possums in the Milltown South VCZ estimated at 7 years post-control (P_{free} = 0.862).

7.1.4 Discussion

The revised SPM was readily able to generate predicted distributions for the number of residual TB-infected possums at any time point after the last possum control operation. Such distributions will facilitate the application and uptake of the StC framework. The standard model outputs can be readily summarised in a way that enables characterisation of the required probability distribution for the number of TB possums.

The distributions generated for the Hokonui Hills are being used to provide an updated POF estimate for the publication of that case study. The same Hokonui distributions were used in the Hauhungaroa case study (Nugent, Sweetapple, Yockney, & Morriss 2017) simply because we found that the computing resources required to model control outcomes over 80,000 ha greatly exceeded the capabilities of desk-top computing. That is an issue that will need to be resolved when the StC approach is operationalised.

However, it is not currently an urgent problem because OSPRI is not yet using the StC approach: the first operational use by an ADM is tentatively scheduled to take place in the Catlins area in 2018 . Presuming that it will eventually become standard practice, we expect

to progressively develop a much fuller library of negative binomial distributions spanning the 0.60–0.95 range of P_{free} values, in which ADMs will almost always be operating.

8 Conclusions

The project has successfully identified and implemented a way of using the SPM to predict TB persistence in areas with naturally low possum density, even in the absence of control. This was achieved primarily by re-evaluating and amending the way in which home range overlap between individual possums is modelled, rather than by moving towards alternative mechanisms for predicting TB persistence.

Changing the possum contact rate function in the SPM resulted in higher rates of possum contacts, because now the total area of overlap between possums' home ranges is considered in the calculations rather than just at the susceptible possum's home range centre. This change, in combination with the modelled expansion of possum home ranges at low possum density, means that for a given disease transmission coefficient TB can persist over a wider range of possum carrying capacity, because contacts between possums are maintained at low density (i.e. they are still able to infect their neighbours). In practice this means that large adjustments of the disease transmission coefficient (β) will not be necessary to generate persistent disease in a given landscape. Nonetheless, the model will still probably not predict TB persistence in areas with very low carrying capacity (<1 possum ha; Figure 3).

Increasing the disease mortality rate from $\alpha = 1$ to $\alpha = 2.4$ had a large effect on modelled TB dynamics, making it much harder (all else being equal) to predict TB persistence at any given density. Because of that, there has been some previous reluctance to change the mortality rate because doing so would have (in the old SPM) required increasing β to high levels to compensate, often to above the maximum OSPRI-permitted level of 1. Changing the contact-rate function has removed that impediment, allowing more realistic simulation of the rate of TB-induced mortality recorded in recent studies.

The major changes in contact rate function and mortality rate appeared to have largely cancelled each other out in relation to predicting the effect of control on TB prevalence, resulting in only modest increases in the speed with which TB disappears after control is imposed. The expected outcomes for ADMs are therefore that (i) the model will predict TB persistence in the absence of control in many more places than previously, and (ii) the new model will predict faster eradication than the old model. As already noted, the latter outcome is supported by recent empirical evidence that TB is never found in areas with a history of effective landscape-scale possum control.

Both the old and the new SPMs can be used to predict the frequency distributions for the number of TB possums required by the StC approach, but we consider the new model to be the most appropriate.

9 Recommendations

- OSPRI should shift to assuming the much higher but more realistic rate of disease mortality of $\alpha = 2.4$ than that used previously, even though this results in much more rapid turnover of disease.
- Users of the SPM should preferentially use the revised SPM for assessing the prior probability of TB freedom (or any other purpose). A set of recommended default parameter values and settings is provided in Appendix 2 (and also in the *User Guide to the Spatial Possum-TB Model*).

10 Acknowledgements

We thank the OSPRI ADMs for their input to this project, and Andrew Gormley for his input and review.

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Appendix 1 – Parameter values used for simulations to assess effects of changes to SPM on estimates of P(free)

INDIVIDUAL-BASED POSSUM/TB MODEL – BASE PARAMETER VALUES

b	0.5	initialPMS	0.475
d	0.1	initialPFS	0.475
theta	3	initialPMI	0.025
delta	0.5	initialPFI	0.025
sexratio	0.5	initialPMR	0
gamma	1	initialPFR	0
br1	1	initialK	10
br2	160	patcharea	500
sigma	30	initPopProp	1
maledistance	2	pctkillI	90
femaledistance	1	cvpctkillI	5
maleshape	2	pctkillM	90
femaleshape	1	cvpctkillM	15
maledispersal	0.1	pctvacc	50
femaledispersal	0.05	controldensity	1
maledistanceB	2	targetdensity	1
femaledistanceB	1	targetRTCI	5
maleshapeB	2	targetRTCImax	10
femaleshapeB	1	meang0	0.1
TBsigma	30	meansigma	41
beta	varying	CVg0	0.3
p	0.6	CVsigma	0.3
alpha	1.0 / 2.4	CVNLsigma	0
vaccineeff	0.9	CRa	120
vaccineimm	1	CRb	89.8
vaccinedecay	1	CRc	0.67

Density dependent Tb transmission

Possum re-aggregation implemented following control to less than 0.6000 possums per hectare.

Appendix 2 – Default parameter values and settings to use for SPM simulations.

Note: these values and settings will load automatically when the SPM is run. Changes can be reset by selecting: Scenario | GENERAL | Default parameter values

Guidelines

- Adjust the disease transmission coefficient (β) to generate the desired level of TB prevalence. The values β = 0.25 and uniform K = 5 loaded when the SPM is run generate a 2% TB prevalence. As a general rule of thumb, if the K-map loaded has a mean K < 3 possums per hectare, increase β up to maximum of 0.35; if mean K > 7, decrease β down to a minimum of 0.150.
- Run at least 200 replicate simulations for each landscape K-map.
- Reduce the burn-in period to 20 years (due to the faster dynamics with α = 2.4)
- Use the 'Seed new infections' functionality if spillback infection or immigration of infected possums is suspected.

Table A1 Default parameter values

Tab	Symbol	Description	Default value
Scenario	*Cell size*	Resolution of *K*-map (m)	50
	Neighbourhood radius	Limit to home range calculations (m)	250
	K	Carrying capacity of habitat (possums/ha)	5[a]
	Random Fill	Initialise the population at random according to proportions in adjacent table	on
	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
	initPopProp	Relative density (*N*/*K*) to initialise population at	1
	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[b]
	Maledistance	Scale parameter male natal dispersal km	2.0

Tab	Symbol	Description	Default value
Scenario, con't	*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[c]
	β	Instantaneous TB transmission rate	0.250[a]
	p	Prop. offspring infected via pseudo-vertical transmission	0.6
	α	Disease mortality rate / year	2.4
Options	*Density kernel*	Shape of home range distribution	Normal[c]
	TB Contact distribution	Shape of TB contact distribution	Normal[c]
	Non-linear contact rates	Adjust sigma (home range scalar) with possum density	on[b]
	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	0.6
Control	*Kill % Initial*	Mean percentage kill for first control operation	90
	CV Kill % Initial	Coefficient of variation for first control operation % kill	8
	Kill % Maintenance	Mean percentage kill for maintenance control operations	80
	CV Kill % Maintenance	Coefficient of variation for maintenance control operations % kill	18

[a] If a K-map is imported with anything other than $K = 5$, then β will also have to be adjusted to generate the desired TB prevalence.

[b] If the non-linear contact rates function is checked, this overrides the constant σ value.

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