Bovine tuberculosis in brushtail possums: models, dogma and data

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Abstract: Three different models of bovine tuberculosis (Tb) in brushtail possums were evaluated against their stated purpose, and testable assumptions and predictions evaluated against available data where possible. Not surprisingly, two of the models may be falsified based on currently available data with respect to either important model assumptions or predictions, and the third may suffer from being right for the wrong reason. This does not mean that these models are not useful. To the contrary, I argue that all models, especially those published in the scientific literature have largely addressed their stated purpose, and have contributed to our understanding of and ability to manage bovine tuberculosis infection in brushtail possum populations. No model, however, satisfactorily explains the pronounced spatial clustering of possum Tb, and the models critiqued have provided little strong inference as to the routes of transmission of Tb among possums. This situation is not helped by the scarcity of datasets on Tb in uncontrolled possum populations that are readily available to confront competing possum/Tb models with. As time passes, there is a very real risk that these data sets will be lost. This is of particular concern, as the expansion in the area of New Zealand under active possum management means the future opportunity to collect further data on Tb in uncontrolled possum populations is severely limited.

Introduction

Models have played a central role in the understanding of the impact of parasites and pathogens on animal populations (McCallum, 1995), and through informing public policy, in guiding management strategies for the successful control of infectious diseases in humans (e.g. Anderson and May, 1991). Models have played a particularly prominent role in the understanding and management of the emerging wildlife bovine tuberculosis (*Mycobacterium bovis*, Tb) reservoirs in brushtail possums (*Trichosurus vulpecula*) and Eurasian badgers (*Meles meles*) (e.g. Anderson and Trewhella, 1985; Barlow, 2000a). This paper relates to evaluating mathematical-type models that seek to describe the dynamics of Tb infection in New Zealand brushtail possum populations, and how these dynamics would be affected by various management interventions (e.g. culling or vaccination). The models considered are the stochastic individual-based simulation model arising from the Massey University research effort at the Castlepoint site (Pfeiffer, 1994; Pfeiffer *et al.*, 1995), the mathematical and deterministic model of Roberts (1996) and the self-styled "pragmatic" mathematical models of the late Nigel Barlow (Barlow, 2000a and references therein). I do not delve into the mechanics of the different modelling approaches, as these are dealt with elsewhere (Barlow, 2000b; Smith,

2001), or the full range of published possum/Tb models such as those models that take a more mathematical treatment of the topic (e.g. Louie *et al.*, 1993). The three modelling approaches were arbitrarily selected on the basis of them being the most well known and considered by management. Since these models were developed in the early 1990s, a considerable number of field studies have been published on various aspects of Tb epidemiology in possums and other species, and it is timely to review these models in the light of what new data have become available.

Within the scientific community, modelling that involves mathematics and/or computers initially evoked great passions from the uninitiated or strongly prejudiced (Hone, 1994). The negative passions often arise from the belief that such modelling is disconnected from the real world of data collection and scientific investigation. However, McCallum (1995) argues that whatever the mode of scientific investigation, at some point a "leap of faith" is made in applying the results to the real world. Models are a case in point, where many of the leaps of faith are found in the model assumptions, whether these relate to parameter values or model constructs. However, rather than being confined to the historical record of the 1990s along with redundant behaviours such as line dancing, mathematical models of ecological systems are increasingly being viewed as tools for representing

hypotheses in a form that can be readily confronted with data (Hilborn and Mangel, 1997). Even Charlie Krebs, known for his skepticism of the usefulness of early attempts at mathematical modelling in ecology, is on the record as making a key recommendation for ecologists to "Use a mathematical model of your hypothesis to articulate your assumptions explicitly" (Krebs, 2000). Within the context of informing the management of epidemics, there has been a strong case put forward for the use of quantitative models following the recent foot-and-mouth outbreak in the United Kingdom (The Royal Society, 2002)

So how does one go about evaluating a model, mathematical or otherwise? Examining the agreement between model predictions and actual data seems reasonable, though if the model has been parameterised to these data then evaluation may not be terribly enlightening, in which case a separate, independent dataset should be used for validation. Indeed, data-based model development where one "lets the data be the judge" is only a foolproof approach for model selection providing models are derived in an entirely a priori manner. However, this is rarely the case, and although we are encouraged to keep candidate models biologically plausible (Burnham and Anderson, 2002), placing too much emphasis on finding a model that fits the data risks "model dredging" and the development of models that are right for the wrong reason. Hence, evaluating data-based models on the basis of their fit to training data, or even their predictive ability on independent data, is not definitive, despite the considerable literature on the topic and a range of techniques for seeking model parsimony.

In contrast to models developed heavily in concert with observed data, models of a more theoretical nature that are developed from the bottom up without formal confrontation with data run the risk of making predictions that bear little resemblance to reality. Evaluating these models may at first appear simple— if they don't produce results that are in accord with observation then they are obviously wrong. However, this is a trivial evaluation, as we know by definition they are "wrong" anyway. Examining the model assumptions is an alternative approach for evaluating models of this type; however, falsifying a model's assumption/s does not necessarily render a model useless, as some assumptions are clearly more critical than others, as hopefully identified by a sensitivity analysis? Indeed, probably all model assumptions can be falsified given powerful enough investigation, just as it is argued that all null hypotheses can be falsified in the limit (Johnson, 1999), hence the oft-heard quote attributed to George Box that "all models are wrong though some are useful". But what is useful in the modelling sense? Mooney and Swift (1999) suggest that inherent in all models should be a sense of purpose,

and go on to define a model as a purposeful representation of reality. It is against this purpose that I consider possum/Tb models should be evaluated as being useful or otherwise. However, I additionally evaluate model assumptions and predictions, as where a model is not achieving its purpose, a review of its assumptions and ability to predict would be a first port of call in making improvements.

Methods

The models by Pfeiffer, Roberts and Barlow are evaluated against their stated purpose, and testable assumptions and data-based predictions are evaluated against available data where possible. I acknowledge that not all the assumptions are scrutinised, but rather those that I consider may have a large bearing on the model results, or sensitivity analyses indicated them to be important. I do not critique parameter estimates used in the models where these parameter estimates were taken directly from the literature.

Results

PossPop (Pfeiffer, 1994)

Purpose

PossPop was developed as part of the pioneering longitudinal study of possum Tb at the Castlepoint site. It aimed to produce a valid understanding of the epidemiological processes that influence the behaviour of the disease in the field, and to contribute to the formulation of effective control policies (Pfeiffer, 1994).

Testable assumptions

Broadly, spatial clustering is postulated to arise from a combination of pseudo-vertical transmission, aggregated mating patterns, and environmentally stressful areas (Pfeiffer, 1994). However, it appears that only the former is included in PossPop. I review the latter two assumptions for completeness.

Den sites as a limiting population factor. The search for and use of den sites plays a crucial role in driving the possum population and defining the activity areas of possums. However, it appears unlikely that den sites play a significant role in limiting possum populations. For example, in the high- density population at Pigeon Flat (Efford *et al.*, 2000) only 14% of known den sites were in use at any one time (Caley *et al.*, 1998).

Aggregated mating patterns. Whilst Australian studies suggest prolonged interactions (consort

behaviour) between pairs of mating individuals (Winter, 1976), studies in New Zealand reveal no such behaviour or any other aggregated social behaviour (Ji *et al.*, 2001; Ramsey *et al.*, 2002). Indeed, the largely polygynous mating system is argued to facilitate the spread of infectious diseases widely through the population (Sarre *et al.*, 2000).

Environmentally stressful area. This was sometimes referred to as the "slum city" hypothesis. In reality, there appears to be little difference in denning conditions in terms of protection between areas frequented by tuberculous possums and those that are not (Caley, 1996). Ironically, the alternative hypothesis (Hickling, 1995) is that spatial clusters of Tb-infected possums arise from higher population density in environmentally *favourable* areas. The original slum city hypothesis may have arisen by attributing too much importance to the environment where Tb thrives in humans in particular (slums) as opposed to the level of crowding within these areas, as the two are highly correlated. It is likely that stress promotes the development of clinical disease; however, the issue is whether environmental stress varies spatially on a scale that would cause the observed clustering of clinical cases in possums. On the balance of evidence it appears that this hypothesis is on shaky ground.

Testable predictions

Relative importance of transmission mechanisms. Simulation runs of PossPop determined that the most important transmission mechanism was "spatial proximity", followed by pseudo-vertical then transmission during mating. Indirect transmission via den sites was deemed to be relatively unimportant. There remains no empirical published data on the relative importance of these routes of transmission. Additional cases of pseudo-vertical transmission continue to be recorded (Jackson *et al.*, 1995a), though the importance of mating remains equivocal (D. Ramsey, Landcare Research, Palmerston North, N.Z., unpublished data). The postulated unimportance of indirect transmission fitted with the respiratory (aerosol) paradigm of Tb transmission, which is applied to both intra-species (Morris *et al.*, 1994) or inter-species (Sauter and Morris, 1995) transmission. A corollary of this paradigm is that contamination of feed and pasture is unimportant to the transmission of *M. bovis* (Morris *et al.*, 1994; Morris and Pfeiffer, 1995). Indeed, when reports of indirect transmission of *M. bovis* among white-tailed deer via contaminated feed first surfaced (Palmer *et al.*, 2001), considerable unpublished skepticism was directed at the results, although more recent studies (Palmer *et al.*, 2004) have validated the initial result. The argument behind the postulated unimportance of pasture contamination as a source of infection for livestock is largely based on logic—a combination of the relatively short survival time of the organism on pasture (Jackson *et al.*, 1995b) with the high infective dose required for reliable infection via the alimentary route (Morris *et al.*, 1994)—which seems logical. However, applying logic to the badger/cattle situation in the United Kingdom reveals that the infectious probability per grazing contact with *M. bovis* would only need to be of the order 10^{-7} to account for the observed prevalence of Tb in British cattle, which seems logically feasible. The first application of logic reveals that the event has a very low probability of occurring, whereas the second reveals that the experiment (cows ingesting a mouthful of grass) is repeated so many times as to logically only require a very low event probability for the transmission route to be plausible. We have no idea who is right, and that is often the problem with logic, in that it does not rank far above anecdote in terms of strength of inference (McArdle, 1996). It seems clear that aerosol transmission occurs in possums housed under experimental conditions (Corner and Presidente, 1981), though it does not necessarily follow that this is the major route of transmission in free-living populations. Adherence to the aerosol paradigm makes it a little tricky to explain how possum-to-possum transmission occurs via the spatial proximity of den sites (apart from during mating or by mother-to-offspring) as possums live largely solitary lives (Day *et al.*, 2000), and although they frequently share dens sequentially (Cowan, 1989), they simultaneously share dens only rarely and choose not to given the chance (Caley *et al.*, 1998). Indeed, how PossPop infers spatial proximity to be the major form of transmission and the sequential use of dens as unimportant is a little unclear given that both are driven by den proximity. Being close spatially allows more opportunity for direct or indirect contact, should this occur, but also heightens the likelihood of sharing dens sequentially. It appears that it is the choice of parameters used for describing the deposition and survival of *M. bovis* in the den site that drives the inference. However, apart from the survival time of *M. bovis* within a den (based on Jackson *et al.*, 1995b), empirical estimates of the probability of depositing and/or acquiring infection from a contaminated den are non-existent and it is unclear how the values for these parameters were arrived at. So although there is strong evidence that pasture contamination is an insignificant route of possum-to-possum transmission (Pfeiffer, 1994), the possible role of infected den sites remains somewhat unresolved, and it would be fair to say that the aerosol transmission paradigm could be considered slightly dogmatic. Note however that Corner *et al.* (2003) argues for the inability of infected den sites to initiate and maintain infection over long periods of time (months), with the reemergence of infection on the Castlepoint site caused by infected immigrants

following depopulation.

Substantial pool of sub-clinically infected individuals. A key finding or emergent property of PossPop was that a substantial pool of sub-clinically infected individuals were needed to reproduce observed clinical disease patterns, with a prevalence in the order of 5–20%. These individuals are postulated to become clinically infected through the onset of stressful weather conditions interacting with the failure of an individual to find a suitable unoccupied den site. However, this postulated high prevalence of sub-clinically infected individuals has not been observed, either at the Castlepoint site where the model was developed (Lugton, 1997) or elsewhere (Caley *unpubl. data*) based on the culture of pooled tissue from the postulated sites of predilection (lungs, axillary lymph nodes etc.). Either the culture technique used was not sufficiently sensitive to detect such low numbers or organisms, or this rather important model assumption/prediction is incorrect.

Summary

To soundly contribute to the scientific understanding and effective management requires that the model be not only publicised but also published, and unfortunately PossPop, along with the longitudinal field study of the dynamics of Tb in an uncontrolled possum population on which it is based, is contained largely within unpublished PhD theses (Pfeiffer, 1994; Jackson, 1995; Lugton, 1997) and hence not easily accessible or subject to genuine peer review. Pfeiffer (1994) acknowledged the limitation of validating the simulation model with only the first 22 months of field data, and foreshadowed more extensive model validation using the longer run of data from the Castlepoint study. These extremely valuable data and the associated collective knowledge of the scientists who gathered it are deserving of a better fate. In contrast, data on the dynamics of Tb in the recovering possum population at the Castlepoint site is published (Corner *et al.*, 2003), along with enlightening individual case studies (Corner and Norton, 2003) and responses to management intervention (Corner *et al.*, 2002a). Returning to the PossPop model, some of the key assumptions and predictions do not seem to be supported by data. Likewise, the postulated mechanisms by which spatial clustering is maintained are not fully supported. In terms of increasing scientific understanding, the model-derived inference that indirect transmission is unimportant as a route of transmission has been quite influential, though given the lack of support for so many of the model predictions and assumptions, must be open to question also. As with all simulation-type models, there is always a challenge to identify what is truly an emergent property of the model, as opposed to simply the retrieval of an

initial assumption. My efforts to review it here based largely on the description in Pfeiffer (1994) may well not do it justice—publication is the obvious course of action to rectify this.

Roberts (1996)

Purpose

Roberts (1996) clearly states the aim of using a simple model possum/tuberculosis system with explicitly stated assumptions to investigate the feasibility of eradicating bovine tuberculosis from possum populations.

Testable assumptions

Homogeneous mixing. Roberts (1996) notes that adding spatial complexity to the model could change some of the conclusions, and it was an area of investigation. This is wise, as there remains little or no support for homogeneous mixing of infectious and susceptible possums at the population level. A longitudinal study (Corner *et al.*, 2003) and repeated surveys of the same site (Coleman, 1988; Caley *et al.*, 1999; Caley *et al.*, 2001) suggests that disease remains spatially aggregated over considerable time periods, effectively ruling out homogeneous mixing. Hickling (1995), when analyzing some of these aforementioned surveys, estimated that tuberculous possums were 4–16 times more crowded than would be expected if they were randomly distributed, and that 7–61% of the habitat contained infected possums. The survey of Coleman *et al.* (1994) comes closest to population-wide mixing, reporting an exceptionally high prevalence with infection occurring nearly throughout the population.

Convex-up contact rate function. There is some behavioural support for the convex-up contact rate function (Ramsey *et al.*, 2002), assuming that contacts associated with mating behaviour are important to Tb transmission. In addition, there is considerable anecdotal support from wildlife disease managers that suggest that disease eradication is more difficult than would be predicted assuming a linear contact rate function.

Population intrinsic rate of increase (r_m). As would be expected, Roberts (1996) reported that the model results were quite sensitive to changes in the population rate of increase (equal to the maximum birth rate (a) minus the minimum death rate (b)). The default values of a and b correspond to $r_m = 0.1$. This is undoubtedly a substantial underestimate for reasons discussed by Barlow (2000a).

Testable predictions

Culling rate to achieve disease eradication. Whilst field managers of Tb would to a person offer the

opinion that 12% per year was far too low a culling rate to achieve a 90% reduction in possum Tb in 5 years, published data to support this are lacking. But their collective actions speak loudly, with the majority seeking residual-trap-catch indices of possums to be reduced to 2–3%, which would correspond to >90% reductions below pre-control levels, and only achievable with culling rates substantially higher than 12% per year.

Substantial population depression by Tb. A key prediction is that *M. bovis* infection depresses population density by 43% below the disease-free carrying capacity. The best field estimates of the effect of *M. bovis* infection on possums at a population density level (Arthur *et al.*, 2004) suggest the size of the population depression is negligible, possibly due to the combined effects of disease clustering and immigration. The most likely explanation for this overestimation of disease-induced population depression is the inappropriate choices of birth and death rates, as pointed out by Barlow (2000a), in combination with the assumption of homogeneous mixing.

Summary

The modelling work of Roberts (1996) achieves its purpose of explicitly presenting a simple possum/Tb model. The model generated debate, which from the scientific viewpoint should be viewed as healthy, and contributed to ongoing model development. For example, Barlow (2000a) incorporated the non-linear contact rate function into his final model. As stated, the model is explicit in its assumptions and formulation, which is commendable and a necessary prerequisite for any kind of evaluation. Whether the system is totally reasonable is open to some debate, particularly the assumption of homogeneous mixing. Perhaps the model is most appropriate for modelling within clusters of disease rather than the population as a whole. A subsequent possum/Tb model by the group (Fulford *et al.*, 2002) incorporates non-homogenous mixing at a broad scale (movement between patches occurs by dispersal) which still does not accommodate the finer scale at which non-homogeneous mixing occurs. The choice of parameters, notably the maximum birth rate and minimum death rate, have been rightly questioned (Barlow, 2000a). However, Roberts (1996) clearly acknowledges that the culling rate needed to achieve eradication is sensitive to the maximum population growth rate. Fortunately, the sensitivity analysis provides estimates of required culling rates for disease eradication etc. under the scenario of a realistically higher intrinsic rate of increase, so a user of the model who believed in a higher rate of increase could get the appropriate model output. So in terms of meeting its purpose of estimating required culling rates for disease

eradication, it does so conditional on the parameters chosen.

Barlow (1991a, 1991b, 2000a, 2000b)

Purpose

In his final contribution to the topic, Barlow (2000a) aimed to present an updated parsimonious possum/Tb model that differed from previous models, most notably Roberts (1996) but also Barlow (1991a, 1991b, 1993). He wanted to demonstrate how the predictions of the updated model were significantly different to past models, and to argue the relevance of heterogeneous mixing and non-linear transmission to wildlife disease models in general.

Testable assumptions

Negative binomial mixing term. The outcome of heterogeneous mixing is treated in a phenomenological way using a negative binomial mixing term. This is probably the most important assumption, and there simply isn't the data to test its validity. The data of Corner *et al.* (2003) may be the best place to start.

Testable predictions

More so than the models of Roberts and Pfeiffer, Barlow placed more emphasis on reproducing observed disease phenomena. In particular, Barlow made it clear he wanted his model to reproduce key epidemiological features of Tb infection in free-living possums, namely: (1) low overall prevalence; (2) rapid disease recovery after control (never mind that these data were based on Tb testing in cattle); and (3) little host suppression. His final model (Barlow, 2000a) largely achieved this, which is a significant achievement. However, evaluating the model based on its ability to produce these phenomena is not terribly rigorous as they are model constructs rather than predictions. The result of Barlow's commitment to reproducing what data there are available is that it is a challenge to find a model prediction that the model has not been "tuned" to reproducing for which there are actually good data on. The response to culling is broadly in line with that reported in the single trial of Caley *et al.* (1999). However, we don't know how representative this single study is of the dynamics of possum Tb in populations subject to culling.

Summary

The Barlow possum/Tb models, like Roberts (1996), do not attempt to address the basic issue of making inference on the mode of transmission of *M. bovis* between possums (other than modelling the contribution of pseudo-vertical transmission). His approach was successful in that he produced a model that achieved its

purpose of reproducing the epidemiological features of possum Tb mentioned previously. The lack of data on disease transmission rates necessitated that the key transmission parameters were estimated *conditionally* on a model. In the words of Barlow (2000a), "Disease transmission coefficients are notoriously hard to measure in the field, and the most robust way of estimating them is probably by tuning models with repeated trial values to mimic observed disease behaviour." This approach is essentially analogous to estimating a parameter using (conditional on) a statistical model, only it escapes being subjected to standard statistical tools such as model parsimony, variance estimates etc. The model stands up to the "tests" that we can throw at it based on our current empirical knowledge of the dynamics of possum Tb. As such it has some respect, somewhat analogous to a null hypothesis that has withstood several critical tests. The nagging doubt remains that it may be producing the right results for the wrong reasons.

Discussion

All three models of possum/Tb evaluated have achieved their stated purpose to various degrees. Despite their being small in number, possum/Tb modellers and their models have achieved much, in what will be historically considered a short time period. It is disappointing that the models have not been more successful in illuminating the mode of transmission among possums, as other inferential approaches such as pathological data from cross-sectional studies provide inconclusive explanations of routes of infection (Jackson *et al.*, 1995a). The PossPop model of Pfeiffer set out with the clearest purpose of addressing this issue, though has subsequently been subject to the least ongoing development, in the published literature at least, which is unfortunate. It is an example of a complex model driven by a large number of parameters, which ultimately may have been too many to make strong model-based inferences given the limited data and knowledge available at the time. The model of Roberts is arguably the most parsimonious of the three, hence its inability to reproduce all observed disease phenomena. Peer review is an essential component of model evaluation, improvement and ultimately scientific progress, and the efforts of Barlow and Roberts in publishing their models is commendable. Identifying spatial determinants of persistent disease clusters was identified by Morris & Pfeiffer (1995) as a particularly important issue in possum/Tb models and this probably remains the case. The approach of Barlow (2000a) leaves us none the wiser as to the mechanism(s) that drive spatial clustering of possum Tb. The experience with Tb infection in white-tailed

deer in Michigan (Palmer *et al.*, 2004) indicates we can no longer be so confident that the respiratory paradigm applies to possum Tb transmission. Indeed, the discovery of *M. bovis* infection in European hares (*Lepus europaeus*) (Cooke *et al.*, 1993; Coleman and Cooke, 2001) is more suggestive of indirect transmission via environmental contamination than direct aerosol transmission. Likewise the observation, albeit rare, of *M. bovis* infection in a rabbit possibly cannot be passed off as the result of a non-fatal bite from an infected predator (Gill and Jackson, 1993). Of course such logical conclusions may not be inferentially sound. What I am arguing for is that the critical experiments needed to confirm the respiratory route of transmission as the major route simply have not been undertaken, and to date the models have failed to provide sound model-based inference either.

All three modelling approaches include a degree of pseudo-vertical transmission to various degrees, though rate its importance as a route of transmission from high (Pfeiffer, 1994) to unimportant (Roberts, 1996). Although studies continue to identify clear cases of pseudo-vertical transmission in very young possums (e.g. Jackson *et al.*, 1995a), the largely unknown incubation period in free-living possums makes it difficult to quantify the contribution of this form of transmission to observed disease cases in older animals. It now appears that brushtail possums exhibit a wide spectrum of responses to infection with *M. bovis*, ranging from rapid disease fulmination and death (Ramsey and Cowan, 2003), to complete resolution of disease (Corner and Norton, 2003) as foreshadowed by Morris & Pfeiffer (1995). Notably, the resolution of infection observed by Corner & Norton demonstrates that possums can mount a protective immune response to virulent infection. This observation is in conflict with the paradigm of "once infected always infected" that is so commonly heard with respect to *M. bovis* and *M. tuberculosis* infections. Hence the mean field approach is probably inadequate, and could possibly lead to underestimation of the contribution of pseudo-vertical transmission—both from the point of view of the number of offspring an infected female can produce (i.e. secondary infections through pseudo-vertical transmission) and the probability of the survival of infected offspring. Modelling the rate of disease-induced mortality as a distribution with a heavy tail rather than a constant rate (the expected value assuming an exponential hazard) could better model the temporal persistence of infection. As is often stated, it is the tails of the distribution that drive the system, and long-lived Tb-infected possum can conceivably contribute to the persistence of clusters of disease. This is more easily implemented using a stochastic approach, of which PossPop showed such early promise.

Some of the constructs of the early possum/Tb models were clearly more conceptual than data-based—they described how researchers conceptualized the possum/Tb system operated. That some of these assumptions were made is largely a reflection of the lack of data to refute them. Making assumptions is a form of scientific risk taking. In the words of McArdle (1996): "But that's the nature of science, perceived truth changes: today's dogma, tomorrow's bad joke." None of the possum/Tb modelling efforts constitute bad jokes, although some of the assumptions are in hindsight a little far-fetched, particularly those related to the mechanism(s) underlying disease clustering. However, inferentially sound data-based explanations for such a possum/Tb phenomenon remain elusive. Our attempts (mine included) to generate a data-based mechanistic explanation for the spatial clustering of disease remain far from convincing. Corner *et al.* (2002b) provides evidence for the role of social structure (albeit in highly artificial housing) for influencing transmission at an individual level, though this would not be expected to generate persistent clusters in free-living possums as the social protagonists will succumb to disease. In fact, free-living possums have no tendency to form social aggregations, and it is considered that possums seek to actively space out from each other and any unevenness in population density reflects environmental heterogeneity (Efford, 2000). Corner *et al.* (2003) also highlighted the huge differences in infectiousness of individual possums, a phenomenon incorporated in the badger/Tb models using different infectious classes such as super-excretors (Smith *et al.*, 2001). Perhaps the combined effects of environmental heterogeneity in concert with density-dependent transmission (Ramsey *et al.*, 2002), disease-induced mortality (Ramsey and Cowan, 2003) and the local movements in response to removal (disease-induced death) (Efford *et al.*, 2000) provide a mechanism of maintaining the spatially clustered nature of *M. bovis* infection in possums.

Possum/Tb modellers can be largely forgiven for writing models that may not predict the actual dynamics of Tb in possum populations, as there are precious few data sets available for confronting models with. Indeed, some of the key parameters for which models were tuned (e.g. time to recovery of disease used by Barlow) are based on data from *M. bovis* infection in cattle rather than possums. There are now data available on the recovery of a possum population and concomitantly the recovery of Tb-infected possums (Corner *et al.*, 2003) though this recovery was aided by a cluster of infected possums contiguous to the study site, which may complicate its interpretation. Critically, there is no published empirical estimate of disease transmission parameters. Many estimates of disease prevalence have been published (e.g. Coleman, 1988); however,

the prevalence of disease alone is insufficient to inform management of likely disease dynamics and their response to control. Age-prevalence data may be used to estimate disease incidence (e.g. Caley and Hone, 2002) and this may be converted into transmission coefficients (Caley and Hone, 2005), although the age-prevalence models will have the added complexity of accounting for the pronounced spatial clustering of disease. Knowing rates of disease transmission is fundamental to determining what level of control is necessary for disease eradication. An analogy from managing overpopulated wildlife would be trying to control/eradicate a pest when the intrinsic rate of population increase is unknown, or trying to determine what level of payments will be required to pay off your mortgage without knowing the interest rate. Worse still for the progression of models, much of what is arguably the best data collected to date from the Castlepoint longitudinal study remain largely unpublished, and as time passes there is a very real risk that these datasets will be lost to science. As the proportion of New Zealand under active possum management increases these data become all the more valuable, as the ability to collect similar data will be diminished considerably. I suggest that as a matter of some urgency, existing data sets on *M. bovis* in brushtail possums should be documented, and stored in such a manner as to facilitate retrieval, analysis and modelling by interested parties—the original players have all moved on in one way or another, and the time for data hoarding has long gone. An example would be the Global Population Digital Database (NERC Centre for Population Biology Imperial College, 1999), which contains nearly 5000 time series in one database.

It is argued that the strength of selection for ecological theory/models depends largely on the importance of a particular field (Ginzburg and Jensen, 2004). With this in mind, special mention should be made of the substantial contribution the late Nigel Barlow made to the world of possum/Tb models, despite this only being a fraction of his wide-ranging scientific interests. In an address to the New Zealand Ecological Society, McArdle (1996) suggested "The primary aim of a scientific investigation is to find the most likely model for a situation out of a host of alternative explanations." Given the data available, Barlow largely achieved this aim, although his phenomenological treatment of heterogeneous mixing falls short of providing full insight into the epidemiology of Tb in possums (Smith, 2005). This is a challenge from which we should not shirk. Indeed, when the current "shock and awe" component of Tb management in New Zealand is inevitably scaled back, the need for good possum/Tb models to give insight into the disease epidemiology and facilitate cost-effective management solutions will never be greater.

Acknowledgements

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