THE POPULATION DYNAMICS OF BOVINE TUBERCULOSIS IN POSSUMS

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ABSTRACT

With the recent outcry concerning the spread of Bovine Tuberculosis throughout the New Zealand Possum population, there is an increasing need to study some of the diverse modelling approaches to this problem. This thesis centres on modelling the epidemics of this disease using two and three-dimensional dynamical systems, which describe the change in the possum population and change in the number of individuals that are diseased.

Introductory material is covered in Chapter One, which reports on the first, through to some of the most recent research completed in the area of disease epidemics. A review of the previous model of possum tuberculosis is also introduced.

Chapter Two looks at the effects on the dynamics of the model of changing the recovery curve parameter, which measures the degree of recovery of possums following a control operation. Detailed steady-state analysis is carried out on the system and local stability determined.

In Chapter Three, a three-dimensional model is investigated that allows for a latent period following infection of disease. Instead of a possum being able to spread the disease immediately after becoming infected itself, there is a latent time until the disease becomes contagious. An in-depth description is given as to how this model originates, then steady-state analysis is explored, and finally local stability of the steady-states is examined.

Restricting the contact rate of an individual possum with the rest of the population is the model studied in Chapter Four. Rather than a possum being able to come in contact with the whole population in a set time, as was the situation in the previous models, the number of contacts is fixed at some realistic value for the given time period. Steady-state analysis is carried out for this new model, along with the local stability analysis.

Chapter Five looks at the various models and how they relate to the model in Chapter Two, as this model is the base for the subsequent ones. Computer generated plots are examined in order to display the numerical differences between the models. A brief comparison is given between these and some other models in the literature, and concludes by discussing some of the advantages and disadvantages of the various models.

Finally, Chapter Six discusses the need for implementing spatially distributed models in the future, to allow for patchiness within the population.

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Thank you all.

DEDICATION

I would like to dedicate this thesis to Mum and Dad for all their advice, encouragement and support they have given me over the years. Words cannot adequately express my gratitude, but thank you. I love you both.

CONTENTS

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§1 Introduction

1.1 The Possum Problem and Current Research in Epidemics

The population dynamics of possums in New Zealand has been of interest because of the destruction of local flora caused by high densities of this introduced marsupial. However, serious attempts to study and to control the possum population were not initiated until the possums were implicated in the late 1960s and early 1970s as a reservoir for tuberculosis (Tb), Mycobacterium bovis, and as a source of infection of dairy cattle. During the 1970s, possum surveys found tuberculous possums on or near properties with persistent cattle Tb problems and large-scale possum control operations using poison were initiated. The subsequent decline in cattle tuberculosis in the areas that were treated gave further confirmation to their implication as a primary source of this infection. A detailed description of this background material can be found in the 1986 issue of *Surveillance* [9] which is devoted to this problem.

The high number of possums in the country and their broad distribution make a general population control strategy economically impractical and possibly infeasible without unacceptable side effects. Consequently, a detailed quantitative assessment of the population dynamics of possums and of cost effective control strategies has become desirable. A model of this type has been proposed by Dr Nigel Barlow from the Ministry of Agriculture and Fisheries, who also performed several computer simulations based on this model which are being currently used as a basis for control strategies.

In this thesis we shall derive and study a number of models based around Dr Barlow's for the population and disease dynamics of possums, incorporating such things as vertical transmission, density-dependent mortality, a latency period following infection and a restriction on the contact rate per individual. All this, in an attempt to produce a model that is the most biologically realistic to the dynamics of tuberculosis in possums from which appropriate control strategies can possibly be implemented.

Some of the earliest classical work on the theory of epidemics was due to Kermack and McKendrick (1927, cited in [6]), who addressed the problem of outbreaks of disease within a population. One such model, although crude, compared rather well with data for death by plague in Bombay during an epidemic in 1906.

The interaction between epidemiology and population dynamics has been studied by Anderson and May [2] who considered models for both microparasites and macroparasites. They investigated how threshold phenomena for the persistence of epidemics were modified

when the population size was variable. They further discussed how a population could be regulated by an infective disease, and also compared theoretical predictions with some data.

Anderson *et al* [1] discuss a similar model for the spread of fox rabies in Europe, assuming a logistic growth of foxes in the absence of disease. They found that the fox population would either settle to a disease-free equilibrium density or that rabies would settle to a regulated state, (below that of the disease-free equilibrium level), which would either be a stable constant value or stable cycle; the cyclic solutions tending to arise when the carrying capacity of the habitat was relatively large and when there was a latent period of the proper length. They concluded that the model predicted patterns of population behaviour in striking agreement with observed epidemiological trends.

In further research completed by Anderson and May [3], they looked at several different models which described the population dynamics of any microparasitic infection that was directly transmitted among invertebrate hosts, and explored the circumstances under which the parasitic infection would regulate the host populations for the various models. This regulated state was either a stable constant value or a stable cyclic oscillation; the latter case occurring when highly pathogenic microparasites produced large numbers of relatively long-lived infective stages with relatively low intrinsic growth rates.

Badger tuberculosis was the next piece of research discussed in depth by Anderson and Trewhella [4]. They reviewed the various processes which influenced badger abundance and presented available data. Using this information, they formulated simple models for the dynamics of badger populations in the absence of disease and decided that non-linear functions were required so that density-dependent constraints began to operate as the density of badgers approached the carrying capacity of the habitat. Local stability analysis revealed either a non-oscillatory return to equilibrium, damped oscillation or instability depending on threshold values.

Next they examined the information on the epidemiology of Tb and presented data relevant to the dynamics of disease transmission, then constructed simple models of disease spread and examined the factors determining infection prevalence. From the analysis, the models suggested the interaction between badger and disease was stable and that oscillatory fluctuations would rapidly damp to a steady state. The models also suggested that the disease acted to depress significantly badger density below disease-free levels. The level of disease prevalence predicted from the models broadly matched those observed in the field.

Finally, they concluded that the persistence of bovine Tb in badger populations was enhanced by the probable involvement of pseudo-vertical transmission and the presence of carriers; both factors acting to suppress the critical density of susceptible animals necessary for the maintenance of the infection within the host population.

More from a mathematical approach to the problem of epidemics within a population was a paper studied by Pugliese [11], in which he used a two equation system to depict the change in a total population and change in the number of diseased subjects within that

population. (As Pugliese's work is very similar to the models we investigate in the subsequent analysis, a little more depth will be added in reviewing his papers.)

A generalised mortality function was implemented along with a non-specific function to describe the contact rate between diseased and non-diseased individuals. Infected individuals were expected to reproduce at a lesser rate than those of non-infected, while a specified proportion of the offspring of infecteds were born diseased. Thus, the possibility of vertical transmission was considered in the model.

The results showed that above a certain threshold, the population would tend to either a diseased steady-state (which was proved to be globally stable) or a steady-state in which there existed infected individuals but no susceptible individuals. Below this threshold, the population tended to a disease-free equilibrium. No limit cycles were found, so all trajectories would converge to an equilibrium. The final piece of analysis involved a small extension to the model to allow for a vaccinated class of individuals, for which similar equilibria and stability results were obtain as above, except that the stability of the endemic (or diseased) equilibrium was only proved locally.

Lastly was a further paper studied by Pugliese [10] in which he introduced a latent period, i.e. the diseased individuals from [11] were divided into exposed individuals (infected but not contagious) and infectious individuals (contagious), giving a three equation dynamical system.

Again he utilised a generalised function to describe the population mortality and a general function for the contact rate. Exposed and infective individuals reproduced at differing rates, with two specific cases being examined. Firstly, the fertility of exposed individuals was at the same rate as that of susceptibles with infectives reproducing at some lesser rate and secondly, neither exposed nor infective individuals contributed to the reproductive effort of the population. For this study, it was assumed that all newborns were susceptibles, eliminating vertical transmission. This was done since the exclusion of vertical transmission did not change the results in [11] but made the mathematics easier.

The results showed that when the fertility of exposed individuals was equal to that of susceptible individuals, the model appeared to be respondent to the biology and gave rise to a picture similar to that of most epidemic models with the possibility of oscillations. Namely, there was a clear threshold below which there was a global convergence to a disease-free equilibrium, and above to a diseased equilibrium.

When the fertility of exposed and infective individuals was zero, the threshold phenomenon was much weaker. Below this threshold the disease-free equilibrium was stable; above, it was unstable and there existed a diseased equilibrium. However, this diseased equilibrium was not necessarily unique and it could exist below the threshold. Multiple equilibria would not arise if mortality and contact rate were linear with population size but could arise if one of these was relaxed. The diseased equilibrium only existed in an

intermediate range of values and was always unstable. From almost all initial values, there was a convergence either to the disease-free equilibrium or to a zero population.

1.2 Review of a Previous Model Studied on Tb in Possums

This now brings us up to the research which I completed in 1989, [13], where I used a two-equation system to model the spread of bovine tuberculosis in possums. I shall introduce the equations now so that the reader can familiarise themself with the basic model and how it was derived.

From [13], we have:

$$\frac{dH}{dt} = H(a-b-sH^{\theta})-\alpha D \qquad (\mathbf{A})$$

$$\frac{dD}{dt} = \beta D(H-D)-D(\alpha+b+sH^{\theta})+\frac{aD}{2} \qquad (\mathbf{B})$$

where $\theta=3$.

The first equation states that the total population of possums (H) increases at a per capita rate equal to the birth rate minus the death rate in the absence of disease, where $b+sH^{\theta}$ is the combined density-dependent and independent per capita mortality rate. In the presence of disease, there is an additional loss of αD possums each year, where D is the population of diseased possums; α being the age-independent death rate due to disease.

The second equation describes the change in the number of diseased individuals which increases in proportion to the number of contacts between the susceptibles and diseased, with each diseased animal infecting a proportion β of the susceptibles each year. An additional $\frac{aD}{2}$ become diseased each year by pseudo-vertical transmission i.e. transmission of disease from parent to offspring. Death of diseased animals occurs at a rate $\alpha+b+sH^{\theta}$, α being the excess over the usual natural mortality rate due to the presence of disease.

In all four equilibria were found for this system of equations, with the most trivial being a zero equilibrium which was always unstable. Further analysis showed a Disease-free equilibrium whose stability depended on parameter values and an Endemic equilibrium (i.e. an equilibrium with non-zero disease and population numbers), whose value *and* stability was determined by parameter values. The remaining equilibrium corresponded to negative population size, so because of the irrelevance of such a solution in the study no further analysis was carried out on this solution.

The most significant finding from the analysis was that at a particular parameter threshold, the Endemic equilibrium entered the feasible region by bifurcating with the Disease-free equilibrium. (The feasible region is the region in phase-space for which population and disease numbers are positive and the number of diseased individuals is at most equal to the total population size). At this bifurcation threshold there was an *exchange of stability* between the two equilibria. Below this threshold, the Endemic equilibrium was outside the feasible region and unstable with the Disease-free equilibrium stable, while above this threshold the Endemic equilibrium was inside the feasible region and stable with the Disease-free equilibrium unstable. This can be represented by the following bifurcation diagram:

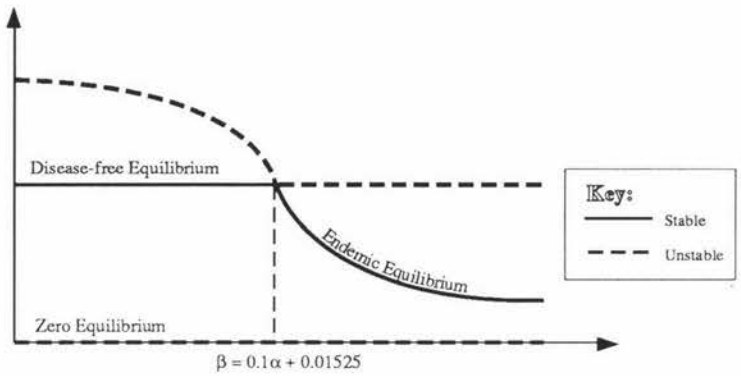

Figure 1.1

Figure 1.2 (taken from [13]) depicts the graph of the equations (A) and (B) at equilibrium ($\frac{dH}{dt} = \frac{dD}{dt} = 0$), with the positive intersection point of (A) and (B) representing the Endemic equilibrium. As parameters vary, this intersection point varies and thus represents the path or *track* of the Endemic equilibrium.

The final piece of analysis showed that solutions to the system remained bounded once they entered the feasible region.

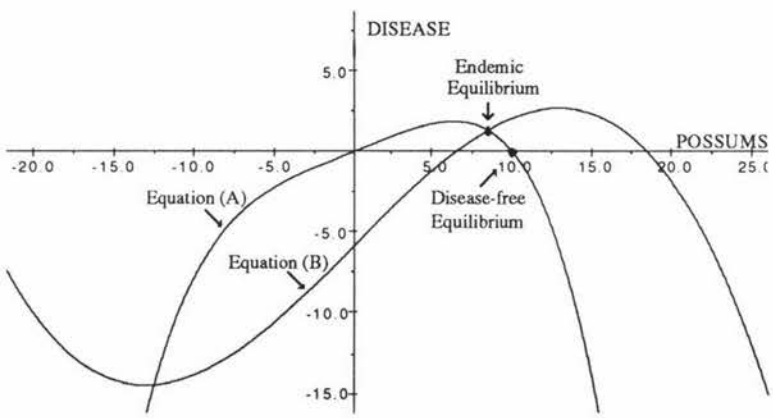

Figure 1.2