

The World's Largest Open Access Agricultural & Applied Economics Digital Library

This document is discoverable and free to researchers across the globe due to the work of AgEcon Search.

Help ensure our sustainability.

Give to AgEcon Search

AgEcon Search http://ageconsearch.umn.edu aesearch@umn.edu

Papers downloaded from **AgEcon Search** may be used for non-commercial purposes and personal study only. No other use, including posting to another Internet site, is permitted without permission from the copyright owner (not AgEcon Search), or as allowed under the provisions of Fair Use, U.S. Copyright Act, Title 17 U.S.C.

RESEARCH PAPERS AND REPORTS IN ANIMAL HEALTH ECONOMICS

AN ACIAR THAI-AUSTRALIAN PROJECT

Working Paper No. 33

Modelling to Predict Disease and Severity Using Age Specific Seroprevalence Data

by

Gavin Ramsay

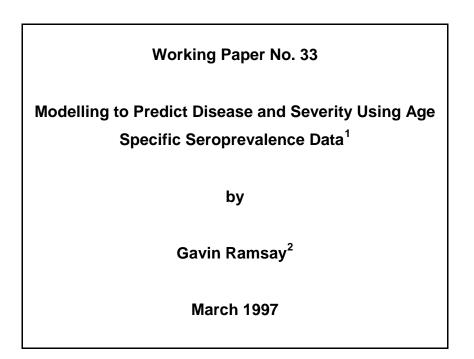
March 1997



THE UNIVERSITY OF QUEENSLAND

ISSN 1322-624X

RESEARCH PAPERS AND REPORTS IN ANIMAL HEALTH ECONOMICS



© All rights reserved

¹ This series, Research Papers and Reports in Animal Health Economics, is supported by the Australian Centre for International Agricultural Research (ACIAR) Project No. 9204 and is published by the Department of Economics, University of Queensland, 4072, Brisbane, Australia. (See next page for more information).

² School of Economics, The University of Queensland, St. Lucia Campus, Brisbane QLD 4072, Australia. Correspondence Clem Tisdell: Email: <u>c.tisdell@economics.uq.edu.au</u>

RESEARCH PAPERS AND REPORTS IN ANIMAL HEALTH ECONOMICS is published by the Department of Economics, University of Queensland, Brisbane, 4072, Australia as a part of a research project sponsored by the Australian Centre for International Agricultural Research, viz., Project No. 9204, 'Animal Health in Thailand and Australia: Improved Methods in Diagnosis, Epidemiology, Economic and Information Management'.

The Commissioned Organization is the Queensland Department of Primary Industries. Collaborating institutions in Australia are CSIRO-ANHL, Geelong, Victoria and the University of Queensland (Department of Economics; Department of Geographical Sciences and Planning). In Thailand, the collaborating institutions are the Department of Livestock Development (National Institute of Animal Health; Disease Control Division), Chiang Mai University (Department of Agricultural Economics; Department of Animal Husbandry) and Thammasat University (Faculty of Economics). The collaborating institution in Laos is the Department of Livestock and Veterinary Services. Dr F.C. Baldock, Senior Principal Epidemiologist, Queensland Department of Primary Industries is the Project Leader in Australia and Dr P. Chamnanpood, Senior Epidemiologist, Thai Department of Livestock Development is the Project Leader in Thailand. Professor Clem Tisdell and Dr Steve Harrison, Department of Economics, University of Queensland are responsible mainly for the economic component of this project.

'The overall goal of this project is to develop and evaluate the .necessary tools to provide decision-makers with reliable animal health information which is placed in context and analysed appropriately in both Thailand and Australia. This goal will be achieved by improving laboratory diagnostic procedures; undertaking research to obtain cost-effective population referenced data; integrating data sets using modern information management technology, namely a Geographical Information System (GIS); and providing a framework for the economic evaluation of the impact of animal diseases and their control.

A number of important diseases will be targeted in the project to test the systems being developed. In Thailand, the focus will be on smallholder livestock systems. In Australia, research will be directed at the northern beef industry as animal health information for this sector of livestock production is presently scarce.'

For more information on *Research Papers and Reports Animal Health Economics* write to Professor Clem Tisdell (c.tisdell@economics.uq.edu.au) or Dr Steve Harrison,(s.harrison@uq.edu.au) Department of Economics, University of Queensland, Brisbane, Australia, 4072.

Modelling To Predict Disease Incidence and Severity Using Age Specific Seroprevalence Data

ABSTRACT

This paper outlines the use of modelling in animal health with an emphasis on Markov chain models. Models that have been used to predict the incidence of disease caused by *B. bovis* are then examined. The development of a model that enables the use of age specific seroprevalence data to estimate the incidence of clinical disease is then described. This involves the use of a method to transform the seroprevalence data to incidence risk which is incorporated into a Markov chain disease prediction model. This in turn is linked to a herd model. The model predicts the proportion of animals in each age and sex class that would be affected by different severities of disease. Using the herd model, estimates of the number of animals affected are made. The model is then used to predict disease incidence and severity for *B. bovis* infection as an initial step in the determination of the effects of control of *B. bovis* by vaccination which is examined in subsequent discussion papers.

Keywords: Babesia bovis, livestock disease, Markov chain model,

JEL Classification: Q16

Modelling To Predict Disease Incidence and Severity Using Age Specific Seroprevalence Data

1. Introduction

Information on the incidence and severity of disease is needed before the effects of disease control programs can be assessed. Age specific seroprevalence is determined as part of SAHS, however, serological results alone do not provide information on the incidence or severity of clinical disease. In addition, any animals which may have died from the disease are not sampled and therefore are not counted. However, serological data can be used, in association with pre-existing knowledge of age and breed susceptibility of cattle to clinical disease, to estimate past losses, and to predict future losses, from the infection, through the use of modelling techniques.

This paper outlines the use of modelling in animal health with an emphasis on Markov chain models. Models that have been used to predict the incidence of disease caused by *B. bovis* are then examined. The development of a model that enables the use of age specific seroprevalence data to estimate the incidence of clinical disease is then described. This involves the use of a method to transform the seroprevalence data to incidence risk which is incorporated into a Markov chain disease prediction model. This in turn is linked to a herd model. The model predicts the proportion of animals in each age and sex class that would be affected by different severities of disease. Using the herd model, estimates of the number of animals affected are made. The model is then used to predict disease incidence and severity for *B. bovis* infection as an initial step in the determination of the effects of control of *B. bovis* by vaccination which is examined in subsequent discussion papers.

2. Modelling in Animal Health

Models are simplified representations of a part of the real world. The part being represented by the model can be described as a system which is defined as a set of related components which exist within a defined boundary. The definition of the boundary of the system is an important issue and will depend on the use to which the model will be put (Dent and Blackie, 1979). Therefore, models are usually designed for a specific purpose and often cannot be applied outside that area. Modelling is a useful way to investigate a disease where experiments and field observations are difficult and expensive to perform (Thrusfield, 1995). In epidemiology models are constructed for a variety of reasons including to predict patterns and levels of disease occurrence, determine the effects of alternative disease control programs and increase understanding of the lifecycles of infectious agents (Thrusfield, 1995).

Mathematical or analytical models of transmissible disease are those models that produce quantitative results (Bailey, 1975; p. 39). However, the accuracy of the results produced depends on the biological understanding of the processes on which the model is based (Medley and Anderson, 1992).

Mihram (1972) differentiates simulation models from analytical models as in the former the models are not entirely manipulated by the use of a well formed discipline such as algebra, integral calculus, numerical analysis or mathematical logic, which is the case in mathematical models. Both simulation models and analytical models are constructed using an orderly process that is an extension of the scientific method of enquiry. Morris and Marsh (1992) separate models used in animal health into differential and difference equation models, double and triple binomial models, Markov chain models, other mathematical models, Monte Carlo models, and optimising models. These authors provide a description of each type.

Early disease models were used to describe disease epidemics and predict disease occurrence (Frost, 1976). These have been more recently followed by the use of models to predict the changes in disease occurrence with different disease control procedures, the economic implications of disease control actions have been included in subsequent development (Thrusfield and Gettinby, 1984).

Irrespective of the method used to classify a model, an important characteristic of a model is the way in which it deals with chance and uncertainty. There are two ways in which disease processes can be represented in a model, they are:

- deterministic where the processes in the model are fixed and biological variation is not allowed for. These models, therefore, produce the same outcome for any given set of parameters and initial conditions, and
- stochastic (or probabilistic) in which the outcomes of some of the processes are obtained using values for factors generated at random from estimated probability distributions. These distributions may be based on observed data or estimated subjectively. Stochastic models produce different results for each simulation and treatments must be replicated several times to represent the range of likely outcomes and produce a reasonable estimate of the mean outcome (Morris and Marsh, 1992).

2.1 Markov chain models

The most commonly used disease simulation computer models have been constructed using the assumption that an infectious disease consists of a series of states, or compartments, such as susceptible, infected, recovered (with or without immunity) or dead, and that animals move from one state to another (de Jong and Diekmann, 1992; Hurd et al., 1993). Markov models have been used widely in animal disease and production studies (James, 1977; Berentsen et al., 1992; Garner and Lack 1995; Jalvingh, 1994) and are examined in this section.

A Markov model simulates the movement between states over time. In such a model the conditional probability that an individual will move from one state to another given the initial state conditional is expressed by a transition probability. The conditional probabilities for all transitions in the system make up the transition matrix. The probability that an animal moves from one state to another is independent of the states visited prior to the entry into that state, this is known as the Markovian property. The distribution of states, known as the state vector, and the changes that occur in this distribution are the primary interest in Markov analysis. The state vector at any time

depends on the previous state vector and the transition matrix. Once the current state vector is known, and the transition matrix estimated, the state vector in any future time period may be predicted. Where the transition matrix remains constant, which rarely occurs in practice, the Markov chain will reach a state vector that is not changed by transition over another time period. This is the steady state vector.

The rate of movement from one state to another can be estimated using data collected in various ways, including biological simulation and analysis of empirical data such as serological test results.

2.2 Models used to predict disease caused by Babesia bovis

Mahoney (1969) used expressions, previously developed by MacDonald (1950 a & b) for use with malaria, to model the onset of parasitaemia of *B. bovis* in calves. This model is based on the inoculation rate which was defined by Mahoney (1969) as the daily probability of an animal becoming infected with *B. bovis*. The Mahoney model assumes that the daily probability of infection is constant over time. Subsequently, Mahoney and Ross (1972) calculated the inoculation rate from the results of serological tests. The inoculation rate is the daily probability of an animal becoming infected with *B. bovis*. Smith (1983, 1991) further developed these ideas and included data on the carrier status of animals in the herd, the predicted infection rate of ticks, and the tick resistance of the host, to estimate the inoculation rate and its variation over time. However, this model is hampered by its need for detailed data on the carrier status of animals in the herd, the predicted infection rate of ticks, and the tick resistance of the host, the predicted infection rate and its variation over time.

An association between the inoculation rate and a state known as endemic stability was then devised (Mahoney and Ross, 1972; Anon, 1984). Endemic stability is defined as the state where the relationship between host, agent, vector and environment is such that clinical disease occurs rarely or not at all (Perry, 1996). Mahoney and Ross (1972) suggested that disease in susceptible *Bos taurus* cattle would be minimised if all animals were infected before they reached nine months old. In this case because almost all animals older than nine months are immune to disease, an outbreak of disease is

unlikely to occur. A similar situation was felt to apply if almost all animals in a group were not exposed to *B. bovis* as it is unlikely that disease will occur if the same low inoculation rate continues. Between the two levels of endemic stability is a gradient of disease risk that varies from low to high.

The most commonly applied model is that of Mahoney and Ross (1972). However, several authors have commented on the model's inappropriateness in predicting the disease occurrence in other situations. For example, Camus and Montenegro-James (1994) suggested that the conclusions made by Mahoney and Ross (1972) from the model did not successfully describe the relationship between the inoculation rate and disease in the Lesser Antilles. However, their findings (Camus and Montenegro-James, 1994) were based on the assumption that the inoculation rate and the disease incidence rate were the same, that is, every animal that becomes infected will show signs of disease. This is rarely the case because non-immune animals older than nine months show a high level of variation in susceptibility to disease following infection with *B. bovis*. This resistance appears to be inherited, with breeds and lines within breeds showing distinct resistance characteristics (Bock, 1996).

A more useful model than that of Mahoney and Ross (1972) could be a model that considers the variation in host susceptibility to disease following infection, in addition to the rate of infection of the host with the disease causing organism, in predicting the occurrence of disease caused by *B. bovis*. The model developed in this chapter considers both of these factors.

2.3 The use of herd models in animal health

Livestock herd models provide an insight into herd performance and have been developed for a variety of reasons, including to:

- determine the off-take and to monitor herd productivity (Baptist, 1988)
- examine the effects of disease control on herd or flock dynamics (El Shishiny et al., 1987)

- simulate the effects of changing strategies on production performance (Abassa et al., 1987; Jalvingh, 1994; Dijkhuizen et al., 1986)
- simulate the production from specified feed resources and the cattle production potential (Sanders and Cartwright, 1979)
- estimate the effect of disease control measures for East Coast fever (Nyangito et al., 1994)
- determine the change in livestock production following the implementation of a disease control program (Putt et al., 1987, p 113), and
- examine the effects of changes in herd structure and production parameters on herd profitability (Holmes, 1993).

Herd models are regarded as important in animal health programs because the implementation of a disease control program may affect not only the productivity of individual animals but also the structure of the herd population due to changes in the reproductive performance and the survival of animals (Matthewman and Perry, 1985). Herd models vary greatly in complexity; they can be large, complex simulation models such as described by Sanders and Cartwright (1979a & b) or relatively simple such as the models of Putt et al. (1987, p. 113).

2.4 Methods of model validation

It is essential that any model is validated effectively and is a realistic representation of reality. Model testing is often undertaken as the model is developed. The major steps in model validation (Mihram, 1972), are:

 verification (also known as internal validation) which is undertaken to ensure that the model behaves as the model builder intended. Verification includes examination of the model for logical soundness and debugging of the computer program

- validation (also known as external validation) which examines the wider issues of the agreement between the model and the system being modelled. This can be difficult to do as there can be considerable biological variation within the system (Bailey, 1975)
- sensitivity analysis which involves the assessment of the extent to which changes in the value of input variables affect output parameters (Thrusfield, 1995), and
- consumer satisfaction, where to be valid the model must be useful to, and used by, the intended users (Bailey, 1975).

3. Calculating Incidence Risk from Seroprevalence

This section describes the estimation of incidence risk of disease from seroprevalence. Firstly the characteristics of the serological test being used to determine the seroprevalence are considered then the way in which the model has been constructed is explained.

3.1 The effect of diagnostic test parameters on seroprevalence

Serological data collected in a cross-sectional study provide an estimate of the point prevalence of antibody positive animals within a population, known as the apparent prevalence (AP). The apparent prevalence is a function of the true prevalence and the sensitivity (Se) and specificity (Sp) of the test being used (Martin, 1984). If the sensitivity and specificity of the test are known, the true prevalence (TP) can be calculated using the following formula from Martin (1984):

$$TP = \frac{\left(AP + Sp - 1\right)}{\left(Se + Sp - 1\right)} \tag{1}$$

Provided that a test has a relatively high sensitivity and specificity and the prevalence of antibody titres is greater than about 10%, the apparent prevalence provides a useful estimate of the true prevalence (Martin, 1984).

3.2 Calculating incidence from prevalence

Because the effect of a disease on production can vary considerably with the age, physical condition and reproductive status of affected animals, it is necessary to convert the prevalence data to incidence. Incidence relates to the new cases of disease that occur in a period of time. Incidence risk (IR), also known as annual incidence, can be defined as the average probability of an animal becoming diseased within a one year time period.

Lilienfeld and Lilienfeld (1980) provide the following formula to calculate incidence risk from age specific prevalence:

$$IR = 1 - (10^{(\log(1-Pn)/n)})$$
(2)

where Pn is the seroprevalence in cattle aged n years.

This formula provides a reasonable estimate of incidence risk if: the risk of infection is constant over time and at all ages, immunity to infection is lifelong and always detectable and mortality due to infection is negligible. Houe and Meyling (1991) used this formula to estimate incidence risk from seroprevalence data in a study of the economic loss associated with bovine pestivirus infection in dairy cattle in Denmark while McGowan et al. (1992) used it for the same purpose in extensively grazed beef cattle in Queensland.

It is important to note that this formula calculates the incidence of seroconversion and where significant mortalities occur, as for example may be the case with *B. bovis* infection, additional methods must be used to estimate the number of animals that have died from the disease. In addition, if mortality due to the disease is high and the proportion of infected animals that die varies significantly between age groups this formula may not be an appropriate estimator of the incidence of seroconversion.

If the incidence risk is defined as the proportion of susceptible animals which seroconvert in a year, and the number of susceptible animals decreases each year due to the proportion of immune animals increasing, then the proportion of animals in any age group which seroconvert in relation to the total number of animals in the age group will decrease with increasing age. As indicated by Houe and Meyling (1991) and McGowan et al. (1992), the proportion of animals in an age group which seroconvert in a year can be calculated as:

Proportion seroconverting in *n* th year of life =
$$P_n - P_{n-1}$$
 (3)

or alternatively as:

Proportion seroconverting in *n* th year of life =
$$(1 - P_{n-1}) \times IR$$
 (4)

4. Determining the Amount and Severity of Disease

In order to calculate the number of cases of disease that have occurred, and the severity of the cases, it is necessary to determine the clinical syndromes which can follow infection and the probability of infected animals suffering from each of the clinical syndromes following infection. The clinical syndromes following infection with a disease causing agent were placed into five categories, namely:

- Category 1 Subclinical disease with seroconversion
- Category 2 Mild clinical disease with recovery and seroconversion
- Category 3 Severe clinical disease with recovery and seroconversion
- Category 4 Acute disease and death
- Category 5 Chronic disease and death

State probabilities are defined with respect to these categories, with p_1 being the probability of infected animals being in *Category 1*, p_2 the probability of being in *Category 2* and so on.

Due to the lack of published data on the probability of animals suffering from each different severity of disease following infection the opinions of acknowledged experts

on *B. bovis* Dr Bob Dalgliesh, Dr Bert de Vos and Dr Russell Bock were collected and are summarised in Table 1.

In the case of *B. bovis* infection, considerable variation occurs in relation to disease severity between types of cattle. The disease response was therefore estimated for three levels of susceptibility to the disease. These susceptibility classes are susceptible (*Bos taurus* breeds), intermediate (Cross bred *Bos taurus*, *Bos indicus* breeds) and resistant (*Bos indicus* breeds). Additionally, age has an important effect on susceptibility to disease with animals less than one year old less likely to show clinical signs or die following infection. Disease susceptibility was assessed for two age classes less than one year old for each of the susceptibility class.

The incidence risk of seroconversion, calculated using formula (2), therefore, estimates the incidence in Categories 1, 2 and 3 combined as animals that die from the disease that is those in Categories 4 and 5 are not sampled or counted. The incidence risk of infection with the disease agent, which also includes those animals that died, can be calculated using the above probabilities as:

Incidence risk (IR) of infection =
$$\frac{IR_of_seroconversion}{(p_1 + p_2 + p_3)}$$
(5)

The proportion of animals in any age group that have become infected in the past year can then be calculated as

$$Proportion infected = proportion susceptible x IR of infection$$
(6)

The proportion susceptible being (1- proportion seropositive)

and the proportion of the age group that are in a specific disease category, for example *Category 4* can be calculated as

Proportion of age group in Category 4 = proportion infected x proportion in Category 4 (7)

Estimates of the incidence risk and the proportion of each age group that suffer from different severities of disease in the past year for disease caused by *B. bovis* calculated

from age specific seroprevalence data are illustrated in Figures 1 and 2, and examined in Sections 4.1 and 4.2.

Table1:Probability of infected animals being in each category of disease
severity following *B. bovis* infection for cattle of different levels of
disease susceptibility

		Cattle type		
Category	Disease severity	Susceptible	Intermediate	Resistant
Cattle less t	han 1 year old			
Category 1	Subclinical	0.4500	0.6150	0.8900
Category 2	Mild clinical	0.2000	0.1625	0.0425
Category 3	Severe clinical	0.2000	0.1625	0.0425
Category 4	Acute die	0.1500	0.0600	0.0250
Category 5	Chronic die	0	0	0
Cattle greater than 1 year old				
Category 1	Subclinical	0.1500	0.4000	0.8500
Category 2	Mild clinical	0.2000	0.2000	0.0575
Category 3	Severe clinical	0.2000	0.2000	0.0575
Category 4	Acute die	0.4250	0.2000	0.0350
Category 5	Chronic die	0.0250	0	0

4.1 Model estimates of herd seroprevalence to Babesia bovis

Figure 1 illustrates the model predictions of age specific seroprevalence where the incidence risk of infection is low (seroprevalence of 0.10 in yearling animals), medium (seroprevalence of 0.50 in yearling animals) and high (seroprevalence of 0.90 in yearling animals). Where the incidence risk of infection is high, all animals in the herd are seropositive at two years old, with medium incidence risk of infection the herd becomes

seropositive at a slower rate with most animals seropositive at four years old. At the low incidence risk of infection the herd seroconverts at a much slower rate and most of the animals have not seroconverted by seven years old.

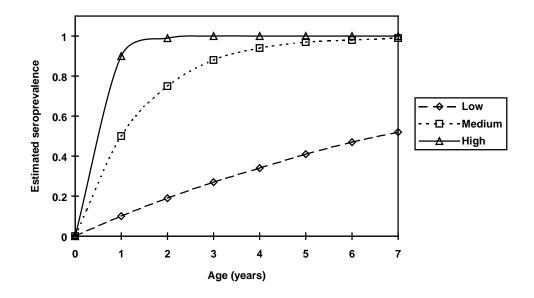


Figure 1: Estimates of seroprevalence for various age groups for low, medium and high incidence risk of infection

4.2 Model predictions of the incidence of disease due to Babesia bovis

Model predictions of the proportion of each age group affected by clinical disease with recovery, due to *B. bovis* infection are illustrated in Figure 2. Where the incidence risk of infection is low the proportion of the herd likely to be affected by clinical disease and recover remains at a low level for cattle in all age groups. An initial small increase is predicted in animals one year old. This could be because these animals are more susceptible to disease following infection than animals less than a year old.

With medium incidence risk of infection the proportion of the herd predicted to clinical disease and recover decreases with increasing age. This decrease in the proportion of animals predicted to show clinical disease is associated with the increasing level of

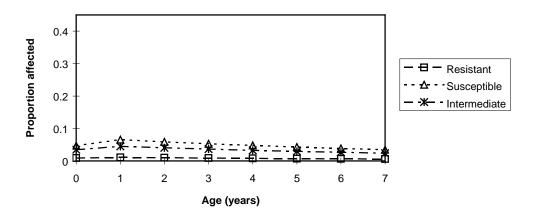
immunity in the herd, and consequent decrease in the proportion of animals susceptible to disease.

Where the incidence risk of infection is high, model predictions of the proportion of the herd affected by clinical disease from which they recover is high in animals up to one year old but decreases rapidly and reaches low levels when animals are three years old irrespective of the level of disease resistance of the herd. The patterns are similar for cattle of each level of disease resistance.

5. Development of the Disease Prediction Model

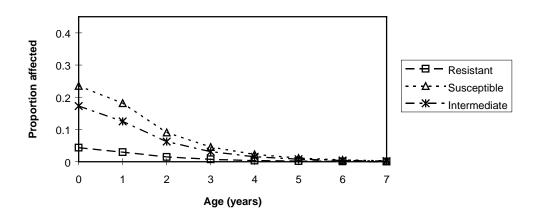
The above method is useful for determining the incidence risk of infection and for estimating the amount of disease that occurred in the past year. However, this model is static and deterministic and greater flexibility is needed to simulate the disease occurrence over several years. A model that can be used to predict the number and severity of cases of disease that may occur over a period of years, and can be further developed to predict the effects of different vaccination programmes is now developed. This section outlines the development of a Markov model that can be used to predict the number of cases of a disease that will occur in each year. Predictions are made for eight age classes from zero years to seven years old for male and female sex classes. The planning horizon is eight years.

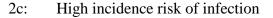
Figure 2: Model predictions of the proportion of each age group that will suffer from clinical disease due to *B. bovis* and recover, for herds containing cattle that are either disease resistant, susceptible or of intermediate susceptibility

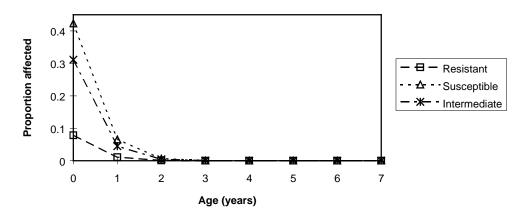


2a: Low incidence risk of infection

2b: Medium incidence risk of infection







The model simplifies the natural history of disease and includes only disease states which it is thought will have an effect on production. The eight states in this model are susceptible, infected, subclinical disease, mild disease with recovery, severe disease with recovery, acute disease with death, chronic disease with death, and immune. The relationships between the states are illustrated in the transition diagram in Figure 3.

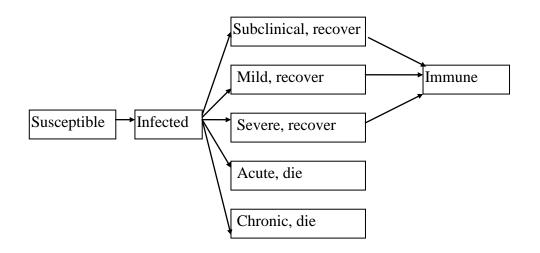


Figure 3: Transition diagram for disease prediction model

5.1 Assumptions in the disease prediction model

The following assumptions are made in the disease prediction model:

• immunity following natural infection does not wane

- the carrier state is ignored as it is assumed that the carrier state does not affect production
- recovery from the disease is permanent and there is no recurrence of the disease in that animal
- sex does not affect the probability of an animal's transition into each of the five categories of disease severity outlined in Section 4.
- an animal that is seropositive for a disease is immune to that disease and an animal that is not seropositive is susceptible to that disease, and
- the incidence risk is constant between age groups and years.
- The model is not being used to determine the natural history of the disease. For diseases where a chronic carrier state occurs which has an impact on production or affects the animal's chance of survival, as occurs with bovine pestivirus infection, the model would need to be modified to include such a state.

5.2 Transition probabilities

The conditional probability of progression from susceptible to infected in one year is the incidence risk of infection, which was calculated from the serological data.

The conditional probability of progression from infected to each of the five severities of disease are those estimated by experts in association with experimental data and are the same as those used in Section 4 to estimate the number of animals in each category from the serological data. These are presented in Table 1.

5.3 Variations in transition probabilities over time

For many diseases, especially vector transmitted diseases, which are affected by environmental conditions, the incidence risk of infection often varies from one year to the next. No attempt has been made to estimate what these changes may be but instead the model is constructed so that incidence risk (that is the transition probability from susceptible to infected) can be varied between years and age groups. To enable variation in the incidence risk of infection the steady state vector is determined and then, using the steady state vector as the initial state vector, the incidence risk is varied. Alternatively, the incidence risk can be kept constant at the level estimated from the serological data.

5.4 Construction of the spreadsheet model

The model was then entered into a computer spread sheet using the package Microsoft Excel. A spread sheet was selected because spreadsheets provide a flexible and powerful tool for model construction. Complex formulae entered into the sheet can be cross referenced to other cells within the spreadsheet or to cells in other spreadsheets. Spreadsheets also provide powerful graphics capabilities that enable data to be easily graphed.

The spreadsheet model developed in this chapter consists of a series of sheets linked to each other within a workbook.

The spreadsheet has been constructed using the age specific seroprevalence from the model described in Section 4 as the initial state vector for the disease prediction model.

6. Linking the Disease Prediction Model to the Herd Model

The disease prediction model is linked to a herd model which estimates the number of animals that suffer from each category of disease severity for each age and sex group in the herd. The herd model maintains a constant herd size and structure.

A single herd size is examined. The herd selected is a breeding herd with 400 females. This herd size and type was selected because it approximates the most common herd size in Central Queensland. Thirty four of the 46 properties sampled in Structured Animal Health Surveillance in 1994 bred and fattened cattle (Black, 1995). Most herds in the Central Queensland region have less than 500 breeding females (74.9%), 12.4% have 500 to 1,000 breeding females, 9% have 1,000 to 2,000 breeding females while few herds (0.5%) have more than 5,000 breeding females (Black, 1995).

As part of Structured Animal Health Surveillance producers provided data on the age structure of their herds. These data are summarised in the Structured Animal Health Surveillance report (Black, 1995) and are presented in Table 2.

Class of cattle	Number in class
Females less than 1 year old	5163
Females 1 to 2 years old	4646
Females more than 2 years old	16137
Total females	25946
Males/steers less than 1 year old	5243
Males/steers 1 to 2 years old	5452
Males/steers greater than 2 years old	6031
Total males/steers	16726
Total cattle	42672

Table 2: Number of cattle in each class in herds sampled in Central Queenslandfor Structured Animal Health Surveillance in 1994

Source: Black (1995).

The age categories in Table 2 are limited with all female and males animals over two years old being placed in the single age category of older than two years. It was therefore necessary to estimate from these data the number of animals in yearly age groups. To do this the mortality rate (5.1% per year) and culling rate (12.5% per year) for these animals were taken from the Structured Animal Health Surveillance report. Using these and assuming that the culling and mortality rates are the same for each age group it is possible to estimate the proportion of the herd in each age class (X_c) using the following formula:

$$T = X_1 + X_2 + X_3 + X_4 + X_5 + X_6$$

If we let $X_c = X_{c-1} L$, then

$$T = X_I + X_I L + X_I L^2 + X_I L^3 + X_I L^4 + X_I L^5$$

which can be simplified to

$$T = X_1(1 + L + L^2 + L^3 + L^4 + L^5)$$

where: *T* is the number in the combined age group,

 X_1 is the number in age category 2-3 years X_2 the number in age category 3-4 years and so on,

X_c is the number in an age class, and

L is one minus the proportion of animals that are removed from the herd each year (the proportion removed is made up of the sum of the proportion that die and the proportion that are culled) and is constant from year to year.

This formula can then be solved for X_1 with X_2 to X_6 calculated from X_1 . The resultant herd structure is shown in Table 3. In order to maintain the herd size and structure, rules relating to the sale and culling of animals and the calving rate are applied. These are:

- all cows are culled at 8 years old with 12.5% of females in each age group culled in each younger age group,
- 18% of males are sold at 2 years old, 50% of the remainder are sold at 3 years old with the remainder sold at 4 years old. No males are kept beyond 4 years of age,
- 40% of all breeding females calve each year.

Because of the small number of bulls in the herd they are not considered in the herd model.

Age and sex class	Proportion of sex	Number in a herd of 400 females		
Females				
0-1 years	0.199	80		
1-2 years	0.179	72		
2-3 years	0.159	64		
3-4 years	0.131	53		
4-5 years	0.108	43		
5-6 years	0.089	36		
6-7 years	0.073	29		
7-8 years	0.061	23		
Castrated males				
0-1 years	0.325	84		
1-2 years	0.313	81		
2-3 years	0.245	63		
3-4 years	0.115	30		

 Table 3:
 Estimated structure for a herd of 400 females in Central Queensland

7. Model Predictions of Number of Cases and Severity of Disease

The results of simulation experiments using the model developed in this chapter carried out to determine the number of cases of disease and their severity are presented and examined in Sections 7.1 and 7.2. Factors varied in these simulation experiments are the:

- incidence risk of infection, and
- level of disease resistance of cattle in the herd.

The number of cases of clinical disease from which animals recover and the number of animals that die for each age group are determined. Three herds with different levels of disease resistance are examined. These are resistant, intermediate and susceptible as defined in Section 4. To simplify the presentation of the results and to show the effect of variation in the incidence risk of infection on predicted disease occurrence three levels of incidence of infection have been selected for comparison. The three levels of incidence risk of infection are:

- low, which is the incidence calculated when the seroprevalence in yearling animals is 10%
- medium, which is the incidence risk calculated when the seroprevalence in yearling animals is 50%, and
- high, which is the incidence risk calculated when the seroprevalence in yearling animals is 90%.

The transition matrix is held constant for these simulations and the results presented are those predicted for the steady state vector. The number of cases of clinical disease from which animals recover (which is the sum of mild disease and recovery and severe disease and recovery defined as Categories 2 and 3 in Section 4) and the number of deaths due to the disease are determined for each age group.

Model predictions of the number of animals that will suffer from clinical disease and recover are presented in Section 7.1 and the number of deaths predicted in Section 7.2.

These experiments do not attempt to determine the incidence risk of infection at which the largest number of cases of disease occur but rather to demonstrate the variation in the number of cases of clinical disease that occur at different levels of incidence of infection and provide an explanation for the variation in the number of cases of disease in different age groups.

7.1 Model predictions of the number of animals that will suffer from clinical disease and recover

Simulations demonstrate the number of cases of clinical disease, with recovery varies with the level of disease resistance in the herd and level of incidence risk of infection as illustrated in Figure 4. Most cases of disease from which animals recover are predicted to occur in young animals.

Resistant cattle are predicted to rarely suffer clinical disease from which they recover. As illustrated in Figure 4.4a less than seven cases are predicted to occur in animals less than one year old at medium and high levels of incidence risk of infection. At medium incidence risk of infection a small number of cases are predicted for animals one year old but in older age groups no cases are predicted to occur. Where the incidence risk of infection is low almost no cases are predicted in all age groups.

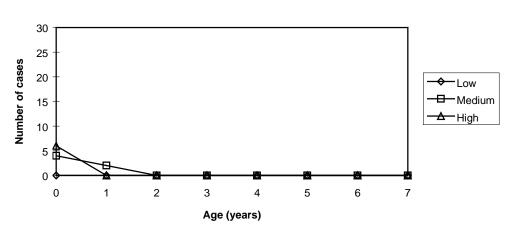
Many more cases of clinical disease with recovery are predicted in the herds of intermediate and susceptible cattle than in the herd of resistant cattle. The intermediate and susceptible herds show similar levels and patterns in each age group of clinical disease with recovery. These predictions are illustrated in Figure 4b and 4c. Most cases are seen in animals less than one year old with a peak where the incidence of infection is high. The age distribution of the cases is complex and is explained in relation to the level of immunity in each age group.

Where the incidence risk of infection is high most animals in the herd are exposed to infection in the first year of life and become immune as illustrated in Figure 1. In subsequent years the number of susceptible animals is low and therefore few cases of disease occur in animals older than one year.

The situation where the incidence risk of infection is low is different. In this case disease is seen at a low level in animals up to five years old. This is because the level of herd immunity increases at a slower rate, as illustrated in Figure 1, and many animals in older age groups are susceptible to disease, but as incidence risk of infection is low only a small number of animals are infected each year.

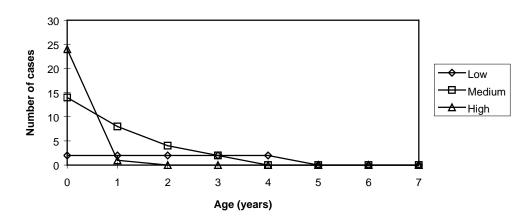
At a medium incidence risk of infection the number and age distribution of cases of disease is again different. In this case disease occurs at a lower level than for the high incidence risk of infection in animals less than one year old but more cases are seen in one and two year old animals than are seen where the incidence risk of infection is low or high. The number of cases of clinical disease with recovery decreases to zero in animals four years old and older. In this situation the proportion of the herd immune increases at a slower rate than that where the incidence risk of infection is high and more rapid rate than where the incidence risk of infection is low (Figure 1). Thus in this case many susceptible animals are present in the herd in the older age groups and are therefore exposed to infection and disease at older ages.

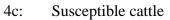
Figure 4: Predictions of the number of animals in each age group that suffer clinical disease due to *B. bovis* and recover

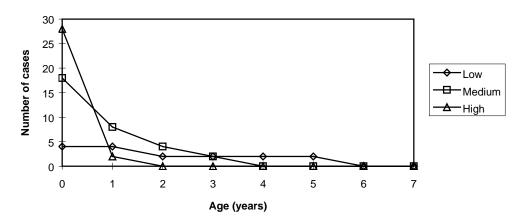


4a: Resistant cattle

4b: Intermediate cattle







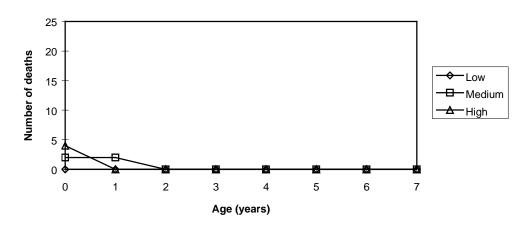
7.2 Model predictions of the number of animals that will die due to Babesia bovis infection

As is the situation for the number of cases of clinical disease with recovery the predicted number of deaths due to *B. bovis* is low in resistant cattle at all ages (Figure 5a). The most deaths are predicted to occur in animals less than one year old where the incidence risk of infection is high. Deaths are predicted to only occur in the herd of resistant animals where animals are younger than two years.

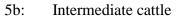
In the case of the herd of intermediate cattle more deaths are predicted to occur at all levels of incidence risk of infection and in older animals as illustrated in Figure 5b. Because animals older than one year are more susceptible to disease than animals less than one year an increase in the number of deaths that occur is seen between the first and second year of life where the incidence risk of infection is medium and low. Where the incidence risk of infection is high this increase is not seen because few animals are infected for the first time in the second year of life.

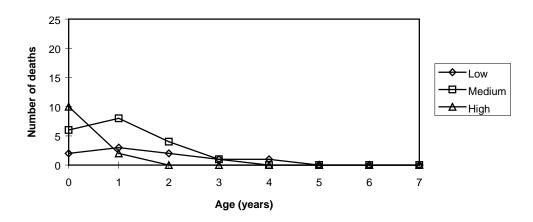
Many deaths are predicted to occur at all levels of incidence risk of infection in the herd of susceptible cattle as illustrated in Figure 5c. An increase in the number of deaths between the first and second years of life is also predicted to occur where the incidence risk of infection is low or medium. In addition to the increase in the number of deaths animals older animals are also predicted to die. Deaths are predicted to occur in animals in each age category where the incidence risk of infection is low and in animals up to three years old with a medium incidence risk of infection.

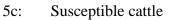
Figure 5: Predictions of the number of animals in each age group that die due to disease caused by *B. bovis*

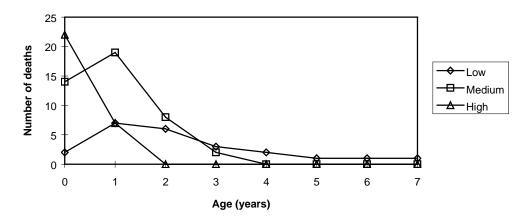


5a: Resistant cattle









7.3 Validation of the disease prediction model

While the model was being constructed input data were used that produced the same outputs for all ages and years of the program so that any errors in the formulae could be easily detected. The logical framework used in the construction of the model was examined.

Comparison of outputs generated by the model against empirical data could not be performed because of a lack of empirical data. However, the model predictions for the age distribution of cases of disease agree with the findings of Rogers (1971) and with the age distribution found for cases of *B. bovis* in the LOIS database as reported in Section 3.2. In both Rogers (1971) and the LOIS database, most cases of disease caused by *B. bovis* occurred in animals less than two years old with a small number of cases diagnosed in cattle older than four years.

The model predictions were examined by experts on disease caused by *B. bovis namely* Dr Bob Dalgliesh, Dr Bert de Vos, Dr Russell Bock and Dr Wayne Jorgensen of the QDPI. These experts indicated that the disease prediction model provided an accurate estimate of the field disease situation. In addition further examination of the disease prediction model developed in this chapter is being carried out by the Tick Fever Research Centre of QDPI in the project "Assessment of the risk of tick fever mortalities in the northern Queensland beef industry" (de Vos, 1996).

8. Summary

This paper examines modelling in animal health then describes the development of a disease prediction model that uses age specific seroprevalence, in association with knowledge of the level of disease susceptibility of the host following infection, to predict the number and severity of cases. This model is then used to predict the number and severity of cases of disease caused by *B. bovis* in a standard beef herd in Central Queensland.

The model developed converts age specific seroprevalence data collected in SAHS into information on incidence and severity of disease. In doing so it provides a sound basis for the studies in subsequent chapters to determine the benefits of vaccination on herd production. The model predictions are most accurate where serological data used is age specific seroprevalence in yearling animals. This is because the data provides information on a single year of life and therefore enables an accurate estimate of the incidence risk of infection to be made.

Simulations demonstrate that disease occurs mostly in young animals. Few cases of disease are predicted to occur in animals older than four years where the incidence of infection is medium. Where the incidence risk of infection is low cases of disease are predicted to occur in all age groups, while with a high incidence risk of infection disease is rare in animals older than two years. The number of cases of disease predicted are, as expected, highest in the susceptible herd. The results of the simulations agree with the limited information available from other sources on the occurrence of disease due to *B*. *bovis*.

9. References

- Abassa, K.P., Conrad, J.H., Smerage, G.H., 1987. Simulated effects of changing strategies on production performance of Gobra zebu cattle. *Agricultural Systems*, 23: 277-286.
- Anon., 1984. *Ticks And Tick-Borne Disease Control A Practical Field Manual*. Food and Agriculture Organisation of the United Nations, Rome.
- Anon., 1995. Structured Animal Health Surveillance Workshop, 15-16 February, 1995 Rockhampton, Queensland, Queensland Department of Primary Industries.
- Bailey, N.T.J., 1975. *The Mathematical Theory of Infectious Diseases and Its Applications*. Charles Griffin and Company, London.
- Baptist, R., 1988. Herd and flock productivity assessment using the standard off-take and the demogram. *Agricultural Systems*, **28**: 67-78.
- Berentsen, P.B.M., Dijkhuizen, A.A., Oskam, A.J., 1992. A dynamic model for costbenefit analyses of foot-and-mouth disease control strategies. *Preventive Veterinary Medicine*, **12**: 229-243.

- Black, P., 1996. *Structured Animal Health Surveillance 1995*. Queensland Department of Primary Industries, Rockhampton.
- Bock, R., 1996. unpublished data.
- Camus, E., Montenegro-James, S., 1994. Bovine anaplasmosis and babesiosis in the Lesser Antilles: risk assessment of an unstable epidemiologic situation. Veterinary Research, 25: 313-317.
- de Jong, M.C.M., Diekmann, O., 1992. A method to calculate for computer simulated infections - the threshold value, Ro, that predicts whether or not the infection will spread. *Preventive Veterinary Medicine*, **12**: 269-285.
- Dent, J.B., Blackie, M.J., 1979. Systems Simulation in Agriculture. Applied Science Publishers, London.
- de Vos, A.J., 1996. personal communication.
- Dijkhuizen, A.A., Stelwagen, J., Renkema, J.A., 1986. A stochastic model for the simulation of management decisions in dairy herds, with special reference to production, reproduction, culling and income. *Preventive Veterinary Medicine*, 4: 273-289.
- El-Shishiny, H., Mansour, H., Galal, E.S.E., 1987. Modelling the dynamics of sheep flocks in Egypt. *Agricultural Systems*, **23**: 301-309.
- Frost, W.H., 1976. Some conceptions of epidemics in general. American Journal of Epidemiology, 103: 141-151.
- Garner, M.G., Lack, M.B., 1995. An evaluation of alternate control strategies for footand-mouth disease in Australia: a regional approach. *Preventive Veterinary Medicine*, 23: 9-32.
- Holmes, W.E., 1993. Breedcow and Dynama herd budgeting models Version 2.00, Queensland Department of Primary Industries, Townsville.
- Houe, H., Meyling, A., 1991. Prevalence of bovine virus diarrhoea (BVD) in 19 Danish dairy herds and estimation of incidence of infection in early pregnancy. *Preventive Veterinary Medicine*, **11**: 9-16.

- Hurd, H.S., Kaneene, J.B., Lloyd, J.W., 1993. A stochastic distributed-delay model of disease processes in dynamic populations. *Preventive Veterinary Medicine*, 16: 21-29.
- Jalvingh, A.W., 1994. *Dynamic livestock modelling for on-farm decision support*. Ph.D. Dissertation, Wageningen Agricultural University.
- James, A.D., 1977. Models of animal health problems. *Agricultural Systems*, **2**: 183-187.
- Lilienfeld, A.M., Lilienfeld, D.E., 1980. *Foundations of Epidemiology*. Oxford University Press, New York.
- MacDonald, G., 1950a. The analysis of malaria parasite rates in infants. *Tropical Diseases Bulletin*, **47**: 915-938.
- MacDonald, G., 1950b. The analysis of infection rates in diseases in which superinfection occurs. *Tropical Diseases Bulletin*, **47**: 907-915.
- Mahoney, D.F., 1969. Bovine babesiasis: a study of factors concerned in transmission. Annals of Tropical Medicine and Parasitology, 63: 1-14.
- Mahoney, D.F., Ross, D.R., 1972. Epizootiological factors in the control of *bovine* babesiosis. Australian Veterinary Journal, 48: 292-298.
- Martin, S.W., 1984. Estimating disease prevalence and the interpretation of screening test results. *Preventive Veterinary Medicine*, **2**: 463-472.
- Matthewman, R.W., Perry, B.D., 1985. Measuring the benefits of disease control: relationship between herd structure, productivity and health. *Tropical Animal Health and Production*, 17: 39-51.
- McGowan, M.R., Baldock, F.C., Kirkland, P.D., Ward, M.P., Holroyd, R.G., 1992. A preliminary estimate of the economic impact of bovine pestivirus infection in beef cattle herds in Central Queensland. Proceedings of the Australian Association of Cattle Veterinarians, *New Horizons*: 129-132.
- Medley, G.F., Anderson, R.M., 1992. The contribution of modelling to our understanding of infectious disease, Modelling vector-borne and other diseases,

Proceedings of a workshop held at ILRAD, Nairobi, 23-27 November 1992, 3-12.

- Mihram, G.Q., 1972. Some practical aspects of the verification and validation of simulation models. *Operational Research Quarterly*, **23**: 17-29.
- Morris, R.S., Marsh, W.E., 1992. The impact of modelling on animal disease control, Modelling vector-borne and other parasitic diseases, Proceedings of a workshop held at ILRAD, Nairobi, 23-27 November 1992, 15-22.
- Nyangito, H.O., Richardson, J.W., Mukhebi, A.W., Mundy, D.S., Zimmel, P., Namken, J., Perry, B.D., 1994. Whole farm economic analysis of East Coast fever immunization strategies in Kilifi District, Kenya. *Preventive Veterinary Medicine*, 21: 215-235.
- Perry, B.D., 1996. Epidemiological indicators and their application to the control of tick-borne diseases. In: (Editors), *Manual on tick and tick-borne disease control*. Food and Agricultural Organisation of the United Nations, Rome. In press.
- Putt, S.N.H., Shaw, A.P.M., Woods, A.J., Tyler, L., James, A.D., 1987. Veterinary Epidemiology And Economics In Africa: A Manual For Use In The Design And Appraisal Of Livestock Health Policy. International Livestock Centre for Africa, Addis Ababa.
- Rogers, R.J., 1971. An evaluation of tick fever outbreaks in northern Queensland in recent years. *Australian Veterinary Journal*, **47**: 415-417.
- Sanders, J.O., Cartwright, T.C., 1979a. A general cattle production systems model. I: Structure of the model. *Agricultural Systems*, **4**: 217-227.
- Sanders, J.O., Cartwright, T.C., 1979b. A general cattle production systems model. Part
 2 Procedures used for simulating animal performance. *Agricultural Systems*, 4: 289-309.
- Smith, R.D., 1983. Babesia bovis: computer simulation of the relationship between the tick vector, parasite and bovine host. Experimental Parasitology, 56: 27-40.

- Smith, R.D., 1991. Computer simulation of bovine babesiosis using a spreadsheet ageclass model with weekly updates. *Journal of Agricultural Entomology*, 8: 297-308.
- Thrusfield, M., 1995. Veterinary Epidemiology. Blackwell Science, Oxford.
- Thrusfield, M.V., Gettinby, G., 1984. An introduction to techniques of veterinary modelling, Proceedings, Society for Veterinary Epidemiology and Preventive Medicine, Edinburgh 10-11 July 1984, pp. 114-138.

ANIMAL HEALTH ECONOMICS

WORKING PAPERS IN THIS SERIES

- 1. Assessing Public Programmes for Control of Animal Diseases in Developing Countries: General Economic Issues with Thai Examples – An extended ISVEE Paper by C. A. Tisdell, S. Harrison and F. C. Baldock, August 2004.
- 2. Animal Health and the Control of Diseases: Economic Issues with Particular Reference to a Developing Country by C. A. Tisdell, September 1994.
- 3. Assessing the Approach to Cost-Benefit Analysis of Controlling Livestock Diseases of McInerney and Others by C. A. Tisdell, May 1995.
- 4. The Economics of Vaccinating or Dosing Cattle Against Disease: A Simple Linear Cost Benefit Model with Modifications by C. A. Tisdell and G. Ramsay, May 1995.
- 5. Livestock, The Environment and Sustainable Development with Illustrations of Issues from Thailand by C. A. Tisdell and S. Harrison, June 1995.
- 6. The Use of Serology to Produce Disease Information for the Economics Analysis of Disease in Extensively Grazed Cattle by G. C. Ramsay, R. J. Dalgliesh, F. C. Baldock and C. A. Tisdell, September 1995.
- 7. The Role of Animal Health Programmes in Economic Development by S. Harrison and C. A. Tisdell, October 1995.
- 8. Trends in the Thai Livestock Industry, Animal Health Implications and Thailand's Development: An Introduction by T. Murphy and C. A. Tisdell, October 1995.
- 9. Specific Livestock Industries, Livestock Diseases and Policies in Thailand: An Overview of Bovines (Buffalo/Cattle) by T. Murphy and C. A. Tisdell, October 1995.
- 10. Overview of Pigs and Poultry: Specific Livestock Industries, Livestock Diseases and Policies in Thailand by T. Murphy and C. A. Tisdell, December 1995.
- 11. Livestock and Livestock Health in Northern Thailand: A Socio-Economic Analysis of a Cross-Sectional Survey of Villages by T. Murphy and C. A. Tisdell, March 1996.
- 12. A Review and Extension of Economic Pest Control Model Incorporating Multi-Pest Species and Insect Resistance by R. Davis, April 1996.
- 13. Foot and Mouth Disease: An Overview of its Global Status, Control Policies and Thai Case by T. Murphy, August 1996.
- 14. An Overview of the Status of Cattle Tick *Boophilus microplus* in Queensland by R. Davis, August 1996.
- 15. A Review of the Economics of Controlling Diseases in Livestock and the Modelling of Control Policies by T. Murphy, August 1996.
- 16. Optimal Livestock Disease Control Models and Their Possible Application to Thailand by T. Murphy, August 1996.
- 17. An Overview of Trends in Development in the Thai Dairy Industry by T. Murphy and C. Tisdell, September 1996.
- 18. Cost-Benefit Analysis with Applications to Animal Health Programmes: Basics of CBA by S. Harrison, September 1996.

- 19. Cost-Benefit Analysis with Applications to Animal Health Programmes: Complexities of CBA by S. Harrison, September 1996.
- Cost-Benefit Analysis with Applications to Animal Health Programmes: Spreadsheet Implementation of Discounted Cash Flow and Risk Analysis by S. R. Harrison, September 1996.
- 21. Cost-Benefit Analysis with Applications to Animal Health Programmes: Allowing for Project Risk in CBA in S. R. Harrison, October 1996.
- 22. Cost-Benefit Analysis with Applications to Animal health Programmes: Valuation of Non-Market Costs and Benefits by S. R. Harrison, October 1996.
- 23. Cost-Benefit Analysis with Applications to Animal Health Programmes: Animal Health Programmes and Information Systems by S. R. Harrison, October 1996.
- 24. Women and Common Property Resources in the Management and Health of Livestock in Thai Villages by T. Kehren and C. A. Tisdell, November 1996.
- 25. Animal Health Information Systems by G. Ramsay, November 1996.
- 26. Collecting Animal Health Data for Cattle Properties in Extensive Grazing System by G. Ramsay, November 1996.
- 27. Sampling Considerations for Active Surveillance of Livestock Diseases in Developing Countries, November 1996.
- 28. On the Economics of Maintaining the Health of Livestock with Thai Examples by C. A. Tisdell, November 1996.
- 29. Economics of Investing in the Health of Livestock: New Insights? by Clem Tisdell, November 1996.
- Macroeconomic Effects of Disease Control in the Thailand Livestock Sector A CGE Analysis by T. Purcell, N. Karunaratne and C. Tisdell, January 1997.
- 31. The Thai Dairy Industry: Its Economic Evolution and Problems Raised by Land Rights and Cattle Diseases by T. Kehren and C. A. Tisdell, February 1997.
- 32. A Review of Economic Evaluations of Government Policies for the Control of Cattle Tick by R. Davis, February 1997.
- 33. Modelling to Predict Disease Incidence and Severity Using Age Specific Seroprevalence Data by G. Ramsay, March 1997.
- 34. Assessing the Effect of Vaccination on Disease Incidence and Severity by G. Ramsay, March 1997.
- 35. Calculating the Production Loss A Voided by Disease Control by G. Ramsay, March 1997.
- 36. Discounted Cash Flow Analysis of Diseases Control Programmes by G. Ramsay, C. A. Tisdell and S. R. Harrison, April 1997.
- 37. Private Decisions in Livestock Disease Control and the Value of Additional Information About Animal Health by G. Ramsay, S. R. Harrison and C. A. Tisdell, April 1997.
- 38. The Distribution of Benefits from Improved Animal Health Decision Making as a Result of the Collection of Additional Animal Health Information by G. Ramsay, C. A. Tisdell and S. R. Harrison, April 1997.
- 39. An Overview of the Occurrence of FMD in Thailand and Policies for its Control, by T. Kehren and C. A. Tisdell, September 1997.
- 40. Socio-Economic Status and Animal Disease Prevention and Treatment by P. Thani, T. Aphichatsarangkoon, B. Aekasing, L. Worachai, November 1997.

- 41. Production and Trade of Cattle, Buffalo and Pigs in Thailand by A. Smith and S. R. Harrison, December 1997.
- 42. Termination Report, Improved methods in diagnosis, epidemiology, economics and information management in Australia and Thailand by P. Black, December 1997.