

# **The Role of a Wildlife Reservoir in the Epidemiology of Bovine Tuberculosis**

**A thesis presented  
in partial fulfilment of the requirements  
for the degree of  
Doctor of Philosophy  
at Massey University**

*D.U. Pfeiffer*

*1994*

## ABSTRACT

The objective of this project was to study the epidemiology of bovine tuberculosis in the presence of a wildlife reservoir species. Cross-sectional and longitudinal studies of possum populations with endemic bovine tuberculosis infection were analysed. The results were used to develop a computer simulation model of the dynamics of bovine tuberculosis infection in possum populations. A case-control study of breakdowns to tuberculosis infection in cattle herds in the Central North Island of New Zealand was conducted to identify risk factors other than exposure to tuberculosis in local possum populations.

The cross-sectional study was based on data gathered some years earlier in the Hauhungaroa Ranges from a number of traplines with a total length of 60km, hence it provided information about the epidemiology of possum tuberculosis on a large geographical scale with varying environmental conditions. The results from the study showed that disease occurrence was clustered in space with local prevalence reaching up to 20% while the overall prevalence was about 1.2%.

The longitudinal study was conducted using an area of 21 hectare of mixed pasture and bush on a sheep/beef farm. The study showed that incidence and prevalence of tuberculosis infection in possum populations has distinct spatial and temporal patterns. Environmental conditions were a major factor in determining the temporal pattern. Spatial and temporal analysis of the occurrence of different strains of *Mycobacterium bovis* allowed inferences to be made about the importance of particular transmission paths. Survival of possums depended on environmental conditions and tuberculosis disease status. Adverse weather conditions increased mortality and the incidence of clinical disease in possums. On average clinically tuberculous possums survived for about 2 to 3 months from the onset of clinical disease.

The simulation model uses a Monte-Carlo modelling approach and incorporates geographical features. Biological mechanisms which are considered important for population and infection dynamics were implemented in the model. These include mating, density-dependent and -independent mortality, pseudo-vertical transmission, transmission through spatial or temporal proximity, and transmission during mating contact. Each possum's movements and behaviour are simulated on a day-by-day basis. Simulations are conducted using a geography and possum population based on data from the longitudinal field study. For preliminary validation, model output was compared with field data from the longitudinal study. Sensitivity analyses and some initial simulation experiments were conducted to identify areas in the model structure which require the collection of additional field data. Use of the model for simulation of a possum population occupying a 400ha area in the Central North Island of New Zealand is demonstrated.

The case-control study of breakdowns to tuberculosis infection in cattle herds showed that in the Waikato area of New Zealand exposure to tuberculosis infection in local possum

populations was not the dominant cause of breakdowns when the study was conducted in 1989/90, at a time when tuberculous possums were first discovered in the region. Farmers who had breakdowns tended to follow cattle purchase and management practices which traditionally have been considered to put farms at risk of introducing tuberculosis. The results of the study indicate that there was a lack understanding among farmers about the epidemiology of tuberculosis.

## ACKNOWLEDGEMENTS

Since my arrival in New Zealand in February 1988 I have been a member of the epidemiology research group in the Department of Veterinary Clinical Sciences at Massey University. These six years have passed by very quickly and I have enjoyed every moment of the time.

I am particularly grateful to my mentor and chief supervisor, Professor Roger Morris. Almost 10 years ago when I first met with him in Colombia, South America, his optimism and enthusiasm gave me the courage to become an epidemiologist while working with him in a country which in this world could not be further away from home. None of this work would have been possible without his friendship, originality, and vision.

Thanks are also due to Associate Professor Roger Marshall and Dr. Nigel Barlow, my other supervisors, who have provided advice and offered suggestions whenever required.

There are a number of people especially from within the epidemiology research group whom I would like to thank for inspiring discussions and for their participation in the field work. I would also like to thank Mr. Anthony Harris, who selected the area for the field study and assisted me in running the field project, Mr. Ron Goile and Mr. Bill Maunsell who allowed us to work on Waio farm and Mr. Mark Stern who undertook most of the programming for the computer simulation model. I thank Dr. Ron Jackson who has always been willing to listen, discuss issues and make constructive suggestions.

A special thanks to my parents, Inge and Arnold Pfeiffer, who have provided me with the education, support and freedom which allowed me to pursue this and other projects away from Germany in the past.

My greatest appreciation and acknowledgement is to my wife, Susanne, and my son, Patrick, who have been very patient when I spent time working on this thesis which should have been spent with them. I will not forget that Susanne helped me during the early part of the field work, which at times was very hard for her. Both Patrick and Susanne were always there when I needed their support and encouragement. Without them this thesis would never have been written.

D.U. PFEIFFER,  
Department of Veterinary Clinical Sciences,  
Massey University,  
New Zealand  
March 1994

# TABLE OF CONTENTS

<b>ABSTRACT</b>	<b>I</b>
<b>ACKNOWLEDGEMENTS</b>	<b>III</b>
<b>TABLE OF CONTENTS</b>	<b>IV</b>
<b>LIST OF FIGURES</b>	<b>X</b>
<b>LIST OF TABLES</b>	<b>XVIII</b>
<b>CHAPTER 1 INTRODUCTION</b>	<b>1</b>
<b>CHAPTER 2 BACKGROUND TO THE STUDY</b>	<b>4</b>
<b>CHAPTER 3 REVIEW OF THE LITERATURE</b>	<b>8</b>
<b>EPIDEMIOLOGY OF TUBERCULOSIS IN HUMANS</b>	<b>9</b>
<b>EPIDEMIOLOGY IN DOMESTIC ANIMALS</b>	<b>14</b>
Cattle	14
Farmed Deer	16
Other Domestic Animals	17
<b>EPIDEMIOLOGY IN WILDLIFE</b>	<b>17</b>
Badger	18
Brush-Tailed Possum	19
Feral Buffalo and Bison	22
Wild Deer	23
Other Wild Animals	24
Other Species	24
<b>CHAPTER 4 A CROSS-SECTIONAL STUDY OF <i>MYCOBACTERIUM BOVIS</i> INFECTION IN POSSUMS IN THE HAUHUNGAROA RANGES, NEW ZEALAND</b>	<b>25</b>
<b>INTRODUCTION</b>	<b>26</b>
<b>MATERIALS AND METHODS</b>	<b>26</b>
Study Design and Data Collection	26
Data Analysis	28
<b>RESULTS</b>	<b>31</b>
Trapping Statistics	31
Ecological Characteristics of the Possum Population under Study	32
Characteristics of the Total Population	32
Comparison of Geographically Grouped Populations	34
Comparison of Population Density Indices	42
Possum Tuberculosis	43
Epidemiology of Tuberculosis in Possums	44
Differences in Condition, Breeding Status, Sex and Age Class	44
Comparison between Geographic Areas and Part of Summer	46

Distribution of Lesions in Tuberculous Possums	47
Spatial Patterns of Infection	51
Possum and Cattle Tuberculosis	54
<b>DISCUSSION</b>	<b>58</b>
Limitations on Interpretation	58
Ecology of Possums in the Study Area	58
Epidemiology of Bovine Tuberculosis Infection in Possums	60
<b>CHAPTER 5 A LONGITUDINAL STUDY OF BOVINE TUBERCULOSIS IN POSSUMS AND CATTLE</b>	<b>68</b>
<b>INTRODUCTION</b>	<b>69</b>
<b>MATERIALS AND METHODS</b>	<b>69</b>
Selection of Study Site	69
Field Procedures	71
Study Site and Study Design	71
General Procedure for Animal Examination	74
Details on Sample Collection	76
a) Blood collection	76
b) Collection of other specimens	76
Other Investigations	76
Data Analysis	77
<b>RESULTS</b>	<b>83</b>
Meteorological Data	83
Trapping Statistics	86
Reproduction	88
Population Dynamics	90
General Body Condition	93
Home Range	94
Denning	95
Immigration	97
Dispersal	99
Descriptive Epidemiology	101
Pathological Findings	103
Survival of Tuberculous Possums	105
Temporal Dynamics of Tuberculosis Infection	111
Spatial Dynamics of Tuberculosis Infection	113
Spatio-temporal Dynamics of Tuberculosis Infection	119
Epidemiological Analysis based on Restriction Endonuclease Analysis Types of <i>Mycobacterium bovis</i>	122
Cattle Tuberculosis	128
Catch Methods	129
<b>DISCUSSION</b>	<b>129</b>
Ecological Findings	129
Reproduction and Mortality	130
Population Size and Demographic Characteristics	131
Home Ranges and Den Use	132
Home Range and Dispersal	134
Tuberculosis Epidemiology	136
Research Design	136
Prevalence and Incidence	136
Age and Sex Distribution of Infection	137
Time to Death or Disease for Different Categories of Possums	139
Insights from Epidemiological Differentiation of Strain Variants	140
Temporal Course of the Disease Process	142
Spatial Aspects of the Disease Process	144

Evaluation of Potential Transmission Mechanisms	144
Sharing of Grazing Area	147
Transmission through Behavioural Interaction of Possums	148
Pseudo-vertical Transmission	149
Den Sharing	151
Transmission through Interactions between Males	152
A Tentative Hypothesis for Transmission of Tuberculosis on the Study Site	153

## **CHAPTER 6 A COMPUTER SIMULATION MODEL OF THE DYNAMICS OF TUBERCULOSIS INFECTION IN A WILD POSSUM POPULATION 156**

### **SIMULATION MODELLING 157**

### **EPIDEMIOLOGICAL SIMULATION MODELLING 158**

### **SIMULATION MODELLING APPROACHES 159**

### **DEVELOPMENT OF A SIMULATION MODEL 160**

Simulation, Model Verification / Validation and the Philosophy of Scientific Inquiry	162
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### **DEVELOPMENT OF A SIMULATION MODEL OF BOVINE TUBERCULOSIS IN A WILD POSSUM POPULATION 165**

Objectives of the Modelling Undertaking	165
General Model Characteristics	165
Temporal Scale	167
Spatial Scale	167
Description of Model Structure and Functionality	168
Den Site Selection	170
Reproduction	171
Infection with <i>Mycobacterium bovis</i>	172
Survival of Possums	174
Ageing Mechanisms for Possums	175
Immigration of Possums	176
Input Parameters for the Model	177
Start Population	177
Den Site Parameters	178
Reproductive Parameters	179
Infection Parameters	180
Survival Parameters	187
Ageing Parameters	189
Immigration Parameters	191
Cyclical Effects	191
Simulation Program Operation	195
Parameter Settings for Population and Disease Mechanisms used in Simulation Run	197
Random Number Generation	198
Generation of Random Variates from Non-Uniform Probability Distributions	199
Variance Reduction Techniques in Simulation Modelling	200
Verification and Validation of the Simulation Model	201
Methods of Analysis	204
Preliminary Simulation Experiment	204
Simulation of a Population with Tuberculosis Infection	210
General Population Dynamics	210
Tuberculosis Infection Dynamics	219
Seasonal Pattern of Tuberculosis Infection	221
Survival Analysis of Simulation Output	225
Spatio-Temporal Patterns of Tuberculosis Infection	227
Detailed Spatio-Temporal Patterns of Tuberculosis Infection	231
Time-Series Analysis of Simulation Output	239
Time-Series Analysis in Time Domain	240

Time-Series Analysis in Frequency Domain	241
Comparison of Antithetic Pairs Approach and Mean of Random Runs Approach	244
Effect on Population Size	245
Effect on Prevalence of Clinical Tuberculosis Infection	248
Effect on Time to Extinction of Clinical Tuberculosis	251
Sensitivity Analysis	253
Baseline Simulation without Clinical TB in Immigrants	253
Effect of Spatial Parameters on Infection Dynamics	254
Radius of Area around an Infected Den with Increased Risk of Infection	254
Maximum Mating Travel Distance	256
Maximum Search Distance for a Den	258
Transmission Mechanisms for <u>Mycobacterium bovis</u> infection	260
Statistical Comparison of Simulations Runs	264
Model Experimentation	267
Simulation Run using Base Parameter Files without Immigration	267
Single Reduction in Population Size without Immigration	268
Single Reduction in Population Size in the Presence of Immigration Free from Clinical Tuberculosis	270
Single Reduction in Population Size with Clinical Tuberculosis in Immigrants	272
Permanent Reduction in Den Site Density in the Absence of Immigration	273
Permanent Reduction in Den Site Density in the Presence of Immigrants free from Clinical Tuberculosis	275
Permanent Reduction in Den Site Density with Clinical Tuberculosis in Immigrants	276
Repeated Population Reduction at Three - Yearly Intervals in the Presence of Clinical Tuberculosis in Immigrants	278
Repeated Control Operations at Six - Yearly Intervals in the Presence of Clinical Tuberculosis in Immigrants	279
Simulation over a 400 Hectare Area	282
Model Performance	288
<b>DISCUSSION</b>	<b>289</b>
Comparison of Model Output and Field Data	289
Evaluation of Disease Control Options with the Current Model	291
Improvements on the Current Model	293
Next Stage of Model Development	295
<b>CHAPTER 7 A CASE-CONTROL STUDY OF TUBERCULOSIS BREAKDOWN IN CATTLE HERDS IN THE WAIKATO REGION, NEW ZEALAND</b>	<b>296</b>
<b>INTRODUCTION</b>	<b>297</b>
<b>MATERIALS AND METHODS</b>	<b>298</b>
Study Design	298
Data Collection	301
Data Analysis	302
General Outline of Approach to Data Analysis	304
Methods used in Multivariate Analysis	305
Stepwise Multiple Logistic Regression	305
Path Analysis	306
Path analysis using regression techniques	307
Path analysis using LISREL	308
Classification Tree Analysis	311
<b>RESULTS</b>	<b>314</b>
Descriptive Analysis	314
General Management and Farm Characteristics	314
Farmer's Interest in Disease Control and Knowledge about the Epidemiology of TB	314
Herd Characteristics	315
Purchase Patterns of Farmers in Study Area	316

Stock Management	316
Univariate Analysis	316
Multivariate Analysis	328
Multidimensional Preference Mapping	328
Stepwise Logistic Regression Models	329
Path Analysis using Standard Regression Procedures	338
Path Analysis using LISREL	344
Classification Tree Analysis	349
<b>DISCUSSION</b>	<b>353</b>
Likelihood of Involvement of Infection from Wildlife Reservoir Species	354
Farmer's Self Concept	355
Farmers' Views about Disease Control and Knowledge about Tuberculosis Infection	356
General Management and Farm Characteristics	357
Patterns of Stock Purchase	358
Herd Characteristics	359
Stock Management	359
Synthesis	360
<b>CHAPTER 8 GENERAL DISCUSSION - TOWARDS A STRATEGIC APPROACH TO WILDLIFE DISEASE CONTROL</b>	<b>362</b>
<b>INTRODUCTION</b>	<b>363</b>
<b>WILDLIFE RESERVOIRS OF DISEASE</b>	<b>363</b>
Rinderpest	364
Rabies	365
Fox Rabies	365
Rabies in Other Species	367
African Swine Fever	370
<b>BOVINE TUBERCULOSIS</b>	<b>371</b>
<b>EPIDEMIOLOGICAL COMPARISONS OF TUBERCULOSIS AND OTHER WILDLIFE DISEASES</b>	<b>374</b>
<b>EPIDEMIOLOGICAL STUDY METHODS FOR DISEASES IN WILDLIFE</b>	<b>377</b>
<b>EVALUATION OF THE STUDY METHODS ADOPTED FOR TUBERCULOSIS</b>	<b>379</b>
Longitudinal Study of Bovine Tuberculosis in Possums	379
Cross-sectional Study of Bovine Tuberculosis in Possums	380
Case-control Study of Tuberculosis Breakdowns in Cattle Herds	381
<b>RESEARCH TECHNIQUES</b>	<b>382</b>
Analysis of Difficult Longitudinal Data	383
Analysis of Data including a Temporal Component	383
Analysis of Data including a Spatial Component	383
Analysis of Data including Both a Temporal and Spatial Component	384
Analysis of Multivariate Data	384
<b>COMPUTER SIMULATION MODELLING AS A TOOL IN DISEASE CONTROL</b>	<b>385</b>
Simulation and Disease Control in Animal Populations	386
Strategies for Pest Management	387
Pest Control and Animal Population Dynamics	388
Modelling of Infectious Diseases	389
Modelling and Planning for the Control of Bovine Tuberculosis in New Zealand	391
Simulation Models of Infectious Diseases in Epidemiological Research	394
<b>BOVINE TUBERCULOSIS DISEASE CONTROL</b>	<b>396</b>

	IX
Strategies available in the Short-Term	398
Strategies available in the Medium-Term	399
Strategies available in the Long-Term	400
<b>DIRECTIONS FOR FUTURE RESEARCH</b>	<b>401</b>
<b>SYNTHESIS</b>	<b>402</b>
<b>BIBLIOGRAPHY</b>	<b>403</b>
<b>APPENDIX</b>	<b>427</b>
<b>APPENDIX I: FORM FOR RECORDING OF DATA COLLECTED DURING CLINICAL EXAMINATION OF POSSUMS</b>	<b>428</b>
<b>APPENDIX II: FORM FOR RECORDING TRAP CATCH DATA</b>	<b>429</b>
<b>APPENDIX III: FORM FOR RECORDING OF POSSUM NECROPSY DATA</b>	<b>430</b>
<b>APPENDIX IV: FORM FOR RECORDING OF DEN SITE TRACKING DATA</b>	<b>431</b>
<b>APPENDIX V: QUESTIONNAIRE FOR CASE FARMS INCLUDED IN TUBERCULOSIS CASE-CONTROL STUDY</b>	<b>432</b>
<b>APPENDIX VI: TURBO PASCAL FOR WINDOWS PROGRAM CODE FOR SIMULATION MODEL</b>	<b>456</b>

## LIST OF FIGURES

Figure 1: Vegetation map of the Hauhungaroa Ranges, Central North Island, with location of farms and base trap lines	27
Figure 2: Possum catch stratified by geographic zone and month	31
Figure 3: Kidney fat index and breeding status in adult female possums	33
Figure 4: Weight-length relationship in possums	34
Figure 5: Possum catch stratified by geographic area and part of summer	35
Figure 6: Average weights and lengths of adult possums stratified by geographic area	36
Figure 7: Proportion of breeding females in total adult females and proportion of female in adult possums stratified by geographic area	39
Figure 8: Proportion of breeding females in total adult females stratified by geographic area and part of summer	40
Figure 9a: Possum population density indices based on PELLET and BATCHELER	42
Figure 9b: Possum population density indices based on OTIS, LESLIE and CATCH	43
Figure 10: Tuberculosis infection status and kidney fat index	45
Figure 11: Tuberculosis infection and weight-length relationship in adult possums	46
Figure 12: Spread of disease within body in tuberculous possums	47
Figure 13a: Spread of disease within body in tuberculous possums stratified by age class	48
Figure 13b: Proportion of single site lesions stratified by sex class	49
Figure 14: Proportion of possums with "open" lesions stratified by month	50
Figure 15: Histogram of size of clusters of tuberculosis infection	51
Figure 16: Histogram of tuberculosis prevalence within clusters of infection	52
Figure 17: Tuberculosis prevalence within clusters of infection and size of cluster	53
Figure 18a: Contour map of the Hauhungaroa Ranges with cattle and possum tuberculosis information, farm and trap line locations	55
Figure 18b: Correlation between cattle tuberculosis incidence and possum tuberculosis prevalence	56
Figure 19: Possum tuberculosis prevalence and cattle tuberculosis incidence by subzone	57
Figure 20: Location of all traps which were used during the three project phases draped over digital terrain model of the area	71
Figure 21: Paddock boundaries for Waio farm draped over a contour map	72
Figure 22a: Photograph of the study area	73
Figure 22b: Trapgrid draped over digital terrain model of the study area	73
Figure 22c: Possum captured in most commonly used trap model	74
Figure 23: Possum with ear notches and ear tag	75
Figure 24a: Distribution of monthly total rainfall during the period 1972 - 1990	84
Figure 24b: Distribution of monthly average ratio between minimum and maximum daily temperature during the period 1972 - 1990	85
Figure 24c: Distribution of monthly average daily temperature during the period 1972 - 1990	85
Figure 25a: Trapcatch statistics	86
Figure 25b: Individual possum captures and clinical examinations	87
Figure 26: Distribution of ages in possums at post-mortem examination stratified by sex class	88

Figure 27: Fertility distribution of births and rearing periods in possums	89
Figure 28a: Temporal dynamics of Jolly-Seber population parameters for the possum population	90
Figure 28b: Temporal dynamics of Jolly-Seber population parameters for the possum population emphasising relationship immigration/births and disappearance	91
Figure 28c: Comparison of population size estimates based on jackknife (with 95% confidence limits) and Jolly-Seber estimator	91
Figure 29a: Temporal pattern of average body weights of possums	93
Figure 29b: Seasonal pattern of average body weights of adult possums stratified by sex class	94
Figure 30: Distribution of number of different possums captured at individual trap locations stratified by sex class	95
Figure 31a: Scatter plot of den site tracking effort and number of different den locations per possum	96
Figure 31b: Histogram of maximum distance between den sites stratified by sex class	97
Figure 32a: Captures of new possums stratified by age groups	97
Figure 32b: Captures of new juvenile possums stratified into immigrants and locally recruited animals	98
Figure 32c: Number of months for which possums were recaptured after initial capture depending on month of first capture	98
Figure 33a: Distances of possum dispersal movements (dots represent trap site and den site locations)	99
Figure 33b: Location data available for possum no. D3565	100
Figure 34: Temporal distribution of new tuberculosis cases in possums stratified by age and sex class	101
Figure 35: Temporal distribution of incident tuberculosis cases in adult female possums stratified by pregnancy status	102
Figure 36a: Possum with a draining lesion in axillary lymph center	104
Figure 36b: Tuberculous lesion in axillary lymph node of a possum	104
Figure 36c: Tuberculous lesions in the lung of a possum	105
Figure 37: Kaplan-Meier survival curves for infected and uninfected possums	106
Figure 38: Kaplan-Meier survival curve for tuberculous possums, by age group	108
Figure 39: Kaplan-Meier curve of cumulative probability of not developing tuberculous lesions	110
Figure 40: Incidence and prevalence of tuberculosis in possums and cattle	111
Figure 41: Tuberculosis incidence in possums and weather conditions	112
Figure 42a: Distribution of distances to the nearest den site utilized by tuberculous possums	113
Figure 42b: Distribution of distances to the tuberculous possum with nearest arithmetic center of activity	114
Figure 43: Contour map of the spatial distribution of proportion tuberculous possums in total catch	115
Figure 44: Contour map of the spatial distribution of total captures	116
Figure 45: Spatial distribution of den sites used by infected and non-infected possums	117
Figure 46a: Histogram of the distribution of slope aspect for den sites which had been used by tuberculous possum and which had only been used by non-tuberculous possums	118
Figure 46b: Histogram of the distribution of height above sea level for den sites which had been used by tuberculous possum and which had only been used by non-tuberculous possums	118
Figure 47a: Time-space interaction between tuberculosis cases stratified by sex class	120
Figure 47b: Temporal and spatial distribution of tuberculosis infected possums stratified by season and year	121
Figure 48a: Details from case histories and post mortem examinations of tuberculous possums with REA type data	123

Figure 48b: Joint plots of the second and third against the first dimension of the result of multiple correspondence analysis describing the association between REA type, sex class, age group and season	124
Figure 48c: Temporal distribution of restriction endonuclease types of <i>Mycobacterium bovis</i> isolates	125
Figure 48d: Spatial distribution of restriction endonuclease types of <i>Mycobacterium bovis</i> isolates based on capture and den site locations used by tuberculous possums	126
Figure 48e: Spatial distribution of restriction endonuclease types of <i>Mycobacterium bovis</i> isolates based on den site locations used by tuberculous possums	127
Figure 48f: Time-space interaction between tuberculosis cases stratified by combination of major REA types	128
Figure 49: Structural steps of the simulation modelling process	162
Figure 50: Important factors in the epidemiology of possum tuberculosis	166
Figure 51: Overview of the model structure	169
Figure 52: Structure of the den site selection module	171
Figure 53: Structure of the reproduction module	172
Figure 54: Structure of the tuberculosis infection module	174
Figure 55: Structure of the survival module	175
Figure 56: Structure of the ageing module	176
Figure 57: Structure of the immigration module	177
Figure 58a: Distribution of distances to the tuberculous possum with nearest arithmetic center of activity	183
Figure 58b: Variogram of difference in prevalence between trap locations on longitudinal study site	183
Figure 58c: Frequency histogram of geographical distances between centres of activity for tuberculous possums detected within 3 month intervals from each other stratified by REA type	184
Figure 59a: Contour map of the spatial distribution of proportion tuberculous possums in total catch	186
Figure 59b: Histogram of period prevalence per trap location	186
Figure 59c: Histogram of period prevalence for locations with tuberculosis based on a 20m grid cell size	187
Figure 60: Temporal pattern of possum disappearance in the longitudinal study	189
Figure 61a: Distribution for sampling age of independence in possums	190
Figure 61b: Distribution used for sampling age of sexual maturity	190
Figure 62: Example of a poisson probability distribution with a mean of 7 expected immigrants per month	191
Figure 63a: Parameter settings for the <i>bad</i> year simulation scenario	193
Figure 63b: Parameter settings for the <i>average</i> year simulation scenario	193
Figure 63c: Parameter settings for the <i>good</i> year simulation scenario	194
Figure 64a: Startup screen after program execution	195
Figure 64b: First screen for defining simulation run parameters	196
Figure 65: Second entry screen for defining simulation run parameters	196
Figure 66: Screen display during computer simulation run	197
Figure 67: Worksheet model for creation and editing of parameter files	198
Figure 68a: Time series plot of monthly data for population size from the original and antithetic run of the preliminary simulation experiment	206
Figure 68b: Time series plot of monthly data of the proportion of sexually mature animals in the population based on data from the original and antithetic run of the preliminary simulation experiment	206
Figure 68b: Time series plot of monthly data of the proportion of female animals in the population based on data from the original and antithetic run of the preliminary simulation experiment	207

Figure 68d: Average age structure in simulated population of preliminary simulation experiment	207
Figure 68e: Error bar chart for population size for each month of the year based on data from the original and antithetic run of the preliminary simulation experiment	208
Figure 68f: Error bar chart for proportion of female possums in total population for each month of the year based on data from the original and antithetic run of the preliminary simulation experiment	208
Figure 68g: Error bar chart for proportion of immature possums in the total population for each month of the year based on data from the original and antithetic run of the preliminary simulation experiment	209
Figure 69a: Time series plot of monthly data for population size, proportion of females and immature animals in the population for the <i>original</i> simulation run using <i>base</i> parameter files	214
Figure 69b: Time series plot of monthly data for population size, proportion of females and immature animals in the population for the <i>antithetic</i> simulation run using <i>base</i> parameter files	214
Figure 69c: Graphical comparison of average population size (incl. standard deviation bars) during the course of a year between output from the simulation model for <i>base</i> parameter files and data obtained from the longitudinal study	215
Figure 69d: Graphical comparison of average proportion females in total population (incl. standard deviation bars) during the course of a year between output from the simulation model using <i>base</i> parameter files and data obtained from the longitudinal study	216
Figure 69e: Graphical comparison of average proportion immatures in total population (incl. standard deviation bars) during the course of a year between output from the simulation model using <i>base</i> parameter files and data obtained from the longitudinal study	216
Figure 69f: Proportion of adult female possums with a dependent young present for each month of the calendar year based on summarized simulation output ( <i>base</i> parameter files; incl. standard deviation bars) and data from the longitudinal study	217
Figure 69g: Average and cumulative number of possums represented as bars using different number of den sites during the period of a month summarized over the whole simulation period ( <i>base</i> parameter files)	217
Figure 69h: Average number of possums (y-axis) using different numbers of den sites categorized into three groups (shaded areas) during the months of a year summarized over the whole simulation period ( <i>base</i> parameter files)	218
Figure 69i: Average number of possums (as vertical bars) spending a given number of days per month without finding a suitable den site summarized over the whole simulation period ( <i>base</i> parameter files)	218
Figure 69j: Average number of days (y-axis) spent by possums without finding a suitable den site over the course of a year summarized over the whole simulation period ( <i>base</i> parameter files)	219
Figure 70a: Time series plot of monthly data for population size, prevalence and incidence of clinical tuberculosis in the population for a simulation run using <i>base</i> parameter files based on simulation output from <i>original</i> run	220
Figure 70b: Time series plot of monthly data for population size, prevalence and incidence of clinical tuberculosis in the population for a simulation run using <i>base</i> parameter files based on simulation output from <i>antithetic</i> run	220
Figure 71a: Average monthly clinical tuberculosis prevalence (including standard deviation bars) over the course of a year for simulation output and data points obtained during the longitudinal study ( <i>base</i> parameter files)	222
Figure 71b: Average monthly clinical tuberculosis incidence (including standard deviation bars) over the course of a year for simulation output and data points obtained during the longitudinal study ( <i>base</i> parameter files)	222
Figure 71c: Average monthly clinical tuberculosis prevalence (including standard deviation bars) in immature possums over the course of a year for simulation output and data points obtained during the longitudinal study ( <i>base</i> parameter files)	223

Figure 71d: Average monthly clinical tuberculosis prevalence (including standard deviation bars) in mature possums over the course of a year for simulation output and data points obtained during the longitudinal study ( <i>base</i> parameter files)	223
Figure 71e: Average monthly clinical tuberculosis prevalence (including standard deviation bars) in male possums over the course of a year for simulation output and data points obtained during the longitudinal study ( <i>base</i> parameter files)	224
Figure 71f: Average monthly clinical tuberculosis prevalence (including standard deviation bars) in female possums over the course of a year for simulation output and data points obtained during the longitudinal study ( <i>base</i> parameter files)	224
Figure 71g: Average monthly tuberculosis infection prevalence (including standard deviation bars) in dependent young possums over the course of a year for simulation output ( <i>base</i> parameter files)	225
Figure 72a: Survival of possums with subclinical and clinical tuberculosis ( <i>base</i> parameter files)	227
Figure 72b: Transition from subclinical to clinical tuberculosis ( <i>base</i> parameter files)	227
Figure 73a: Cumulative spatial distribution of den sites used by possums with clinical tuberculosis infection during the simulation run ( <i>base</i> parameter files; <i>original</i> and <i>antithetic</i> run) overlaid on map of total den site locations (circles represent TB den sites and dashes non-TB den sites)	229
Figure 73b: Cumulative spatial distribution of den sites used by possums with clinical tuberculosis infection during the two simulation runs ( <i>original</i> and <i>antithetic</i> run) with height representing cumulative distribution of TB den sites	230
Figure 73c: Density of all mapped den sites per 20m <sup>2</sup> used for simulation runs	231
Figure 74a: Spatio-temporal pattern of clinical tuberculosis during year 1 to 5 of a simulation run ( <i>original</i> run)	233
Figure 74b: Spatio-temporal pattern of clinical tuberculosis for years 6 to 10 of a simulation ( <i>original</i> run)	234
Figure 74d: Spatio-temporal pattern of clinical tuberculosis for years 11 to 15 of a simulation ( <i>original</i> run)	235
Figure 74e: Spatio-temporal pattern of clinical tuberculosis for years 16 to 20 of a simulation ( <i>original</i> run)	236
Figure 74f: Spatio-temporal pattern of clinical tuberculosis for years 21 to 25 of a simulation ( <i>original</i> run)	237
Figure 74f: Spatio-temporal pattern of clinical tuberculosis for years 26 to 28 of a simulation ( <i>original</i> run)	238
Figure 75: Time series decomposition plot of clinical tuberculosis prevalence ( <i>base</i> parameter files)	241
Figure 76a: Spectrogram for subclinical and clinical TB prevalence	243
Figure 76b: Spectrogram for subclinical and clinical TB incidence	243
Figure 76c: Spectrogram for population size	244
Figure 77a: Error bar chart for population size (incl. standard deviation) based on 10 <i>original</i> and 10 <i>antithetic</i> runs and 10 averages of antithetic pairs from the 10 simulation runs over the period of a simulation year	246
Figure 77b: Box-and-whisker plots for population size based on 10 independent runs, 10 antithetic runs and averages of antithetic pairs from 10 simulation runs for the whole simulation period of 10000 days	246
Figure 77c: Box-and-whisker plots for population size based on 5 independent runs, 5 antithetic runs and 5 averages of antithetic pairs for the whole simulation period of 10000 days	247
Figure 78a: Error bar chart for clinical tuberculosis prevalence (incl. standard deviation) based on 10 independent runs and 10 averages of antithetic pairs over the period of a simulation year	249
Figure 78b: Error bar chart for clinical tuberculosis prevalence (incl. standard deviation) based on 10 independent runs and 10 average of antithetic pairs during the first 24 months of the simulation runs	249
Figure 78c: Box-and-whisker plots for clinical tuberculosis prevalence based on 10 independent runs, 10 antithetic runs and 10 averages of antithetic pairs for the whole simulation period of 10000 days	250
Figure 78d: Box-and-whisker plots for clinical tuberculosis prevalence based on 5 independent runs, 5 antithetic runs and 5 averages of antithetic pairs for the whole simulation period of 10000 days	250

Figure 79a: Survival plot of time to extinction of clinical tuberculosis based on 10 independent runs, 10 antithetic runs and 10 averages of antithetic pairs for the whole simulation period of 328 months	251
Figure 79b: Survival plot of time to extinction of clinical tuberculosis based on <i>first</i> set of 5 independent runs, 5 antithetic runs and 5 averages of antithetic pairs for the whole simulation period of 328 months	252
Figure 80: Time plots of prevalence and incidence of clinical tuberculosis and population size for simulation output from <i>original</i> and <i>antithetic</i> runs used as <i>baseline</i> simulation scenario for sensitivity analysis	253
Figure 81a: Time plots of prevalence and incidence of clinical tuberculosis and population size for simulation output from <i>original</i> and <i>antithetic</i> runs testing the effect of a 25m radius of increased risk of infection around infected dens	255
Figure 81b: Time plots of prevalence and incidence of clinical tuberculosis and population size for simulation output from <i>original</i> and <i>antithetic</i> runs testing the effect of a 75m radius of increased risk of infection around infected dens	256
Figure 82a: Time plots of prevalence and incidence of clinical tuberculosis and population size for simulation output from <i>original</i> and <i>antithetic</i> runs testing the effect of a 75m mating travel distance	257
Figure 82b: Time plots of prevalence and incidence of clinical tuberculosis and population size for simulation output from <i>original</i> and <i>antithetic</i> runs testing the effect of a 125m mating travel distance	258
Figure 83a: Time plots of prevalence and incidence of clinical tuberculosis and population size for simulation output from <i>original</i> and <i>antithetic</i> runs testing the effect of a 75m den search distance	259
Figure 83b: Time plots of prevalence and incidence of clinical tuberculosis and population size for simulation output from <i>original</i> and <i>antithetic</i> runs testing the effect of a 125m den search distance	260
Figure 84a: Time plots of prevalence and incidence of clinical tuberculosis and population size for simulation output from <i>original</i> and <i>antithetic</i> runs with the transmission mechanism in den sites disabled	261
Figure 84b: Time plots of prevalence and incidence of clinical tuberculosis and population size for simulation output from <i>original</i> and <i>antithetic</i> runs with transmission through spatial proximity disabled	262
Figure 84c: Time plots of prevalence and incidence of clinical tuberculosis and population size for simulation output from <i>original</i> and <i>antithetic</i> runs with transmission through mating contact disabled	263
Figure 84d: Time plots of prevalence and incidence of clinical tuberculosis and population size for simulation output from <i>original</i> and <i>antithetic</i> runs with pseudo-vertical transmission disabled	264
Figure 85a: Box-and-whisker plots for population size based on combined simulation output from <i>original</i> and <i>antithetic</i> runs for each of the sensitivity analysis scenarios	266
Figure 85b: Box-and-whisker plots for clinical tuberculosis prevalence based on combined simulation output from <i>original</i> and <i>antithetic</i> runs for each of the sensitivity analysis scenarios	266
Figure 86: Time plot of prevalence and incidence of clinical tuberculosis and population size for simulation output from <i>original</i> and <i>antithetic</i> run testing the effect of removing immigration	268
Figure 87: Time plot of prevalence and incidence of clinical tuberculosis and population size for simulation output from <i>original</i> and <i>antithetic</i> run testing the effect of a single reduction in population density to 25% without immigration	270
Figure 88: Time plot of results of <i>original</i> and <i>antithetic</i> run for prevalence and incidence of clinical tuberculosis and population size for a simulation scenario testing the effect of a single reduction in population density to 25% in the presence of immigration free from clinical tuberculosis	271
Figure 89: Time plot of results of <i>original</i> and <i>antithetic</i> run for prevalence and incidence of clinical tuberculosis and population size for a simulation scenario testing the effect of a single reduction in population density to 25% in the presence of 5% clinical tuberculosis prevalence in the immigrants	273
Figure 90: Time plot of prevalence and incidence of clinical tuberculosis and population size based on <i>original</i> and <i>antithetic</i> run for simulation output showing the effects of a permanent reduction in den site density in the absence of immigration	274
Figure 91: Time plot of prevalence and incidence of clinical tuberculosis and population size based on <i>original</i> and <i>antithetic</i> run for simulation output showing the effects of a permanent reduction in den site density in the presence of immigration free from clinical tuberculosis	276

Figure 92: Time plot of prevalence and incidence of clinical tuberculosis and population size based on <i>original</i> and <i>antithetic</i> run for simulation output showing the effects of a permanent reduction in den site density, in the presence of immigration with 5% clinical tuberculosis prevalence	277
Figure 93: Time plot of prevalence and incidence of clinical tuberculosis and population size based on <i>original</i> and <i>antithetic</i> run for simulation output showing the effects of repeated population control operations at 3 yearly intervals in the presence of 5% clinical tuberculosis infection in immigrants	279
Figure 94: Time plot of prevalence and incidence of clinical tuberculosis and population size based on <i>original</i> and <i>antithetic</i> run for simulation output showing the effects of repeated population control operations at 6 yearly intervals in the presence of 5% clinical tuberculosis infection in immigrants	280
Figure 95: Rasterized map of major vegetation cover classes in simulation area and locations of random den sites	283
Figure 96a: Time plots of incidence/prevalence of clinical tuberculosis and population size for both simulation runs over 400 hectare area	284
Figure 96b: Locations of den sites used by possums with clinical tuberculosis for years 1 to 12 for <i>original</i> run over 400 hectare area	285
Figure 96c: Locations of den sites used by possums with clinical tuberculosis for years 13 to 24 for <i>original</i> run over 400 hectare area	286
Figure 96d: Locations of den sites used by possums with clinical tuberculosis for years 25 to 28 for <i>original</i> run over 400 hectare area	287
Figure 96e: Digital elevation model with height representing frequency with which clinically tuberculous possums use den sites and shades of grey representing vegetation type based on cumulative TB den site locations used between years 10 and 28 of the <i>original</i> run of the simulation over 400 hectare area	287
Figure 97: Histogram of time required to simulate one year of simulation time based on data from simulation runs over the 21ha longitudinal study area	288
Figure 98a: Map of farm locations	299
Figure 98b: Vegetation map of study area with farm locations	300
Figure 98c: Digital terrain model of study area with farm locations	301
Figure 99: Farm size distribution of properties included in study	314
Figure 100: Cattle herd size distribution of properties included in study in livestock units	315
Figure 101a: Beef component per cattle livestock unit stratified by case-control status	319
Figure 101b: Total beef cattle in livestock units stratified by case-control status	319
Figure 101c: Total cattle in livestock units stratified by case-control status	320
Figure 101d: Total cattle livestock units purchased stratified by case-control status	320
Figure 101e: Proportion of heifers/steers per cattle livestock unit stratified by case-control status	321
Figure 101f: Distance to next case farm stratified by case-control status	321
Figure 101g: Distance to next endemic TB area stratified by case-control status	322
Figure 101h: Total area pasture stratified by case-control status	322
Figure 101i: Permanent labour units stratified by case-control status	323
Figure 101j: Total labour units stratified by case-control status	323
Figure 101k: Total livestock units stratified by case-control status	324
Figure 101l: Proportion of weaners/yearlings per cattle livestock unit stratified by case-control status	324
Figure 101m: Proportion of weaners/heifers/steers per cattle livestock unit stratified by case-control status	325
Figure 101n: Scores for knowledge about possible mechanisms of tuberculosis transmission between cattle and humans stratified by case-control status	325
Figure 101o: Purchase of replacements and number of different sources stratified by case-control status	326

Figure 101p: Scores for knowledge about MAF TB control methods stratified by case-control status	326
Figure 101q: Scores for knowledge about species involved in epidemiology of tuberculosis stratified by case-control status	327
Figure 102: Personality trait means for interviewees by case-control status	328
Figure 103: Biplot of multidimensional preference mapping of study farms within the preference space describing their self concept	329
Figure 104: Diagnostic plot of difference chi-square versus predicted probability with plot symbol proportional to standardized influence measure for final logistic regression model comparing cases and random controls	333
Figure 105a: Null hypothesis path diagram for comparison of cases with random controls	340
Figure 105b: Final path diagram for comparison of cases with random controls	340
Figure 106a: Null hypothesis path diagram for comparison of cases with matched controls	343
Figure 106b: Final path diagram for comparison of cases with matched controls	343
Figure 107a: Q-plots of normalized residuals for final path models	347
Figure 107b: Path diagram for final path model comparing cases and matched controls	348
Figure 107c: Path diagram for final path model comparing cases and random controls	348
Figure 108a: Classification tree for comparison of cases and matched controls	352
Figure 108b: Classification tree for comparison of cases and random controls	352

## LIST OF TABLES

Table 1: Possum catch stratified by sex and age	32
Table 2: Possum catch stratified by sex and geographic area	37
Table 3a: Possum catch stratified by age class and geographic area	37
Table 3b: Catch of male possums stratified on age class and geographic area	37
Table 4: Catch of adult possums stratified on sex and geographic area	38
Table 5: Breeding status of female adult possums stratified by geographic area	39
Table 6: Average body length and tuberculosis infection status	45
Table 7: Distribution of single lesion sites	48
Table 8: Bovine tuberculosis history of cattle herd on study farm from 1979 until 1989	70
Table 9a: Total monthly rainfall summarized over the period 1972 until 1990	83
Table 9b: Average ratio of minimum and maximum daily temperature summarized over the period 1972 until 1990	84
Table 10: Statistical tests of the assumptions for Jackknife estimator	92
Table 11: Home range estimates for adult males and female possums	95
Table 12: Summary of stepwise selection procedure for the 'best' discrete hazard rate regression model of time until death or disappearance	107
Table 13: Summary of stepwise selection procedure for the 'best' discrete hazard rate regression model of time until development of detectable tuberculous lesions	109
Table 14: Distance to nearest tuberculous possum or den site utilized by infected possums	113
Table 15: Results of multivariate analysis of den site utilization by tuberculous possums	119
Table 16: Structure of the start population	178
Table 17: Monthly probabilities of a successful mating	179
Table 18: Worksheet for modelling of parameters for probability of transition from subclinical to clinical tuberculosis	181
Table 18: Worksheet model for estimation of infection probabilities for the three variable transmission mechanisms in the model	187
Table 19: Worksheet for calculation of monthly survival in dependent possums	188
Table 20: Worksheet for estimation of monthly survival probabilities which are not density-dependent	188
Table 21: Monthly distribution of average number of immigrants per year	191
Table 22: Characteristics of the three types of years ( <i>good</i> , <i>average</i> , <i>bad</i> ) based on results of <i>k</i> -means cluster analysis	192
Table 23: Parameters used to estimate different probability arrays for the three types of years ( <i>good</i> , <i>average</i> , <i>bad</i> )	194
Table 24a: General characteristics of preliminary simulation experiment	205
Table 24b: Results of descriptive analysis of output from preliminary simulation experiment	205
Table 25: General characteristics of the simulation experiment	210
Table 26a: Summary statistics of population parameters for simulation run using <i>base</i> parameter files	213
Table 26b: Summary statistics of population parameters by simulation run and type of year using <i>base</i> parameter files	213
Table 27: Summary statistics of tuberculosis infection dynamics by simulation run and type of year using <i>base</i> parameter files	225

Table 28: Classical decomposition model for clinical tuberculosis prevalence based on simulation output from <i>original</i> run using <i>base</i> parameter set files	241
Table 29: Summary statistics for population size based on simulation output for the three different methods of treatment of random numbers (using 5 and 10 runs)	245
Table 30: Summary statistics for clinical tuberculosis prevalence based on simulation output for the three different methods of treatment of random numbers (using 5 and 10 runs)	248
Table 31: Summary of simulation output used as <i>baseline</i> simulation scenario for sensitivity analysis	254
Table 32a: Summary of simulation output for simulation scenario testing the effect of a 25m radius of increased risk of infection around infected dens	255
Table 32b: Summary of simulation output for simulation scenario testing the effect of a 75m radius of increased risk of infection around infected dens	256
Table 33a: Summary of simulation output for simulation scenario testing the effect of a 75m mating travel distance	257
Table 33b: Summary of simulation output for simulation scenario testing the effect of a 125m mating travel distance	258
Table 34a: Summary of simulation output for simulation scenario testing the effect of a 75m den search distance	259
Table 34b: Summary of simulation output for simulation scenario testing the effect of a 125m den search distance	260
Table 35a: Summary of simulation output for simulation scenario testing the effect of disabling transmission through infected den sites	261
Table 35b: Summary of simulation output for simulation scenario testing the effect of disabling transmission through spatial proximity	262
Table 35c: Summary of simulation output for simulation scenario testing the effect of disabling transmission through mating contact	263
Table 35d: Summary of simulation output for simulation scenario testing the effect of disabling pseudo-vertical transmission	264
Table 36: Statistical comparison of sensitivity analysis scenarios with the <i>baseline</i> simulation scenario using Scheffé's test combining data from <i>original</i> and <i>antithetic</i> run	265
Table 37: Summary of simulation output for model testing the effect of removing immigration	268
Table 38: Summary of simulation output for model testing the effect of a single reduction in population density to 25% without immigration	270
Table 39: Summary of simulation output for model testing the effect of a single reduction in population density to 25% in the presence of immigration free from clinical tuberculosis	272
Table 40: Summary of simulation output for model testing the effect of a single reduction in population density to 25% in the presence of 5% clinical tuberculosis prevalence in immigrants	273
Table 41: Summary of simulation output for model testing the effect of a permanent reduction in den site density in the absence of immigration	275
Table 42: Summary of simulation output for model testing effect of permanent reduction in den site density in the presence of immigration free from clinical tuberculosis	276
Table 43: Summary of simulation output for model testing effect of permanent reduction in den site density, in the presence of immigration with 5% clinical tuberculosis prevalence	278
Table 44: Summary of simulation output for model testing effect of repeated population control operations at 3 yearly intervals in the presence of 5% clinical tuberculosis in immigrants	279
Table 45: Summary of simulation output for model testing effect of repeated population control operations at 6 yearly intervals in the presence of 5% clinical tuberculosis in immigrants	281
Table 46: Codes and descriptions of variables used in the multivariate analysis	313
Table 47a: Some results of univariate analysis for random controls using logistic regression	317

Table 47b: Some results of univariate analysis for matched controls using logistic regression	318
Table 48a: Stepwise logistic regression analysis for cases compared with random controls	330
Table 48b: Summary of the results of stepwise logistic regression analysis for cases compared with random controls	331
Table 49: Final logistic regression model comparing cases and random controls	332
Table 50a: Stepwise logistic regression analysis for cases compared with matched controls using the unconditional approach	334
Table 50b: Summary of the results of unconditional stepwise logistic regression analysis for cases compared with matched controls	335
Table 50c: Stepwise logistic regression analysis for cases compared with matched controls using the conditional approach	336
Table 50d: Summary of the results of conditional stepwise logistic regression analysis for cases compared with matched controls	337
Table 51: Comparison of coefficients of logistic regression models for cases and matched controls using the unconditional and the conditional approach	337
Table 52: Final conditional logistic regression model comparing cases and matched controls	338
Table 53: Results of regression analyses for final path model comparing cases with random controls	339
Table 54: Results of regression analyses for final path model comparing cases with matched controls	342
Table 55a: Goodness of fit of the final path models	345
Table 55b: Total and direct effects on case-control status in the final path models	346
Table 56a: Summary of information about the final classification trees	350
Table 56b: Variable rankings according to relative importance	351

# **CHAPTER 1**

## **INTRODUCTION**

For a brief period after the advent of such advances in infectious disease control as antibiotics and modern vaccines, it was possible for people to envision a future with total control over most infectious human and animal diseases. This view changed dramatically with the advent of a growing range of apparently new diseases such as acquired immunodeficiency disease (AIDS) and the re-emergence of 'old' diseases such as human and bovine tuberculosis. It became recognized that traditional purely laboratory-based scientific approaches have difficulty in providing adequate solutions for complex disease problems. In keeping with a similar development in physics, this positivistic and reductionistic epistemology has had to be replaced by a more comprehensive scientific method which allows for uncertainty and offers a more holistic view of the nature of problems (van Gigch 1991).

With regard to tuberculosis infection, after a period in which the disease was seen to have declined to negligible proportions, quite pessimistic visions of the future have been drawn more recently both for human disease in most countries and for animal disease in countries where wildlife reservoirs exist. In the case of human tuberculosis, an alarming increase in tuberculosis incidence in humans has been seen in the United States (Glassroth 1992) and elsewhere. Discussing the rise of drug-resistance in *Mycobacterium tuberculosis*, Bloom points out that we may be working our way back to a frightening future (Bloom 1992). Recently, reports have come from the United States about deaths in HIV-infected prisoners caused by multi-drug resistant strains of *Mycobacterium tuberculosis*, which would pose a very major health hazard if they become established in the wider community. Stanford *et al* (1991) suggest that we are facing one of the greatest public-health disasters since the bubonic plague.

Although bovine tuberculosis does not represent a significant human health hazard now in the way it did in the nineteenth century, and the disease in animals has responded very favourably to classical test and slaughter control methods in the absence of a wildlife reservoir, such reservoirs have emerged in various countries over recent decades, and have undermined control efforts under these circumstances. Although various wild and feral species can become infected with *Mycobacterium bovis* (Allen 1991), the most important reservoir hosts so far discovered are the European badger (*Meles meles*), the Australian brushtail possum (*Trichosurus vulpecula* Kerr) and various species of deer.

In New Zealand, Australian brushtail possums had been introduced to the country on multiple occasions between about 1840 and 1940, and wild populations grew and expanded from initial colonization sites to the point where they occur in most of the country and total numbers have been estimated at about 70 million. In 1967 the first tuberculous possum was found in an area of the West Coast of the South Island where it had proved difficult to eradicate bovine tuberculosis using methods which had been successful in other parts of the country. Over the following 25 years it has gradually become clear that possums are a major reservoir species for *Mycobacterium bovis* infection in New Zealand, although not in their native Australia. In the presence of this source of infection eradication of bovine tuberculosis from the cattle population seems unlikely to succeed. Traditional methods of disease control

such as test and slaughter of cattle and culling of possums have not proved adequate to achieve effective control of tuberculosis in either the cattle or the possums.

It was concluded from an examination of the situation that it was essential to better define the epidemiology of the disease in possums in New Zealand. Using an epidemiological approach, it would become possible to adopt a structured systems approach towards identifying and implementing effective control of tuberculosis infection in domestic cattle populations which are exposed to tuberculous possums. This study forms the first stage of a long term research program to achieve these goals.

## **CHAPTER 2**

### **BACKGROUND TO THE STUDY**

Since the start of the tuberculosis eradication campaign in 1945, tuberculosis incidence in New Zealand cattle has been successfully reduced from about 8.6% to 0.11% in dairy cattle and from 0.8 % to 0.29% in beef cattle, using figures derived for the testing year 1990/91 (Hennessy 1986, O'Hara 1992). In 1991 2.7% of herds in the national population (n=47000) were classified as infected or under movement control restrictions. However, despite continuing "test and slaughter" efforts, in 18 areas (in 1991) of the country reactor incidence remained significantly higher than in the rest of the country. These 'endemic areas' are now considered to be continuously infected with tuberculosis because of the existence of a reservoir of infection in possums and possibly in other wild animals. Some have expressed the view that eradication of the disease in cattle may be impossible (O'Hara 1992). After the first possum with tuberculosis infection was discovered in 1967, it took a few years before the first surveys for non-bovine sources of tuberculosis infection were conducted. It was then found that in most 'endemic' areas tuberculosis was present in the possum population. Introduction of extensive possum control operations resulted in a prolonged reduction in cattle reactor rates (Hennessy 1986).

An experiment to determine the risk from a tuberculosis - infected possum population to cattle grazing adjacent areas was conducted on the West Coast of the South Island in 1970/71. Twenty nine tuberculosis-free calves were introduced into an area where a tuberculosis prevalence of 12% had been found previously in the possum population. After 6 months 26 animals were tuberculin positive, and lesions were found at slaughter in 16 (Davidson 1976).

In a summary of experiences with *Mycobacterium bovis* infection in cattle on the West Coast Stockdale (1975) states that the likelihood of recurrence of cattle tuberculosis on individual farms appeared to be linked to the prevalence of tuberculosis in possums. Properties with a persistent problem seemed to have in common a topography and vegetation which made it difficult for possum control to be successful. Tuberculosis "break downs" in cattle herds usually coincided with the presence of only one tuberculous possum, which was found during subsequent investigations on the property. Often, possum tuberculosis was found in local clusters and it was then possible to demonstrate that only animals which had grazed the associated areas were infected. Other potential wildlife reservoirs of tuberculosis infection including deer, pigs, ferrets and hedgehogs were considered to be a minor risk of infection for cattle. Stockdale concluded that there was ample circumstantial evidence that *Mycobacterium bovis* infection cycling in possum populations was the major factor contributing to persistent tuberculosis problems in cattle in New Zealand.

In the Wairarapa the first tuberculous possum was detected in 1969. From then until 1981 tuberculous possums were found on 114 farms all over the Masterton district. Most of these farms, and very often their neighbouring properties, had a persistent tuberculosis problem in cattle (Shortridge 1981).

A number of reports have found that in problem areas cattle reactor rates decreased significantly over the years following a reduction in the possum population density as a result

of a control operation (Hennessy 1986). After recognizing that the disease situation did not respond to normal testing procedures a 3 monthly whole herd testing scheme was introduced in Buller County. This resulted in a reduction of reactor incidence from 12.5% of 4306 cattle tested in January 1970 to an average incidence of 4.97% (s.d. 1.02) in April 1972. Extensive aerial poisoning operations to kill possums were conducted in June-July 1972 and were followed by ground control work over the following years. Cattle reactor incidence dropped to an average of 1.36% (s.d. 0.46) over the period from January 1973 to July 1975 (Stockdale 1975). In the Wairarapa, cattle tuberculosis incidence averaged 2 - 2.3% per year over four years before possum population control was implemented. After two years reactor incidence reached a minimum of 0.3 and started to increase steadily over the following 4 years to about 0.7 (Hennessy 1986). Recurrence of tuberculosis in cattle has been observed in several areas of the country a number of years after initial reduction following possum control operations (Batcheler and Cowan 1988).

In the Central North Island between 1975 and 1987 the size of the area with endemic tuberculosis infection in possums was increasing by a multiplication factor of 1.104 every year. Assuming that spatial spread follows a model of logistic growth, tuberculosis infection would be present in all North Island possum populations by the year 2024 and in all South Island populations by 2031 (Batcheler and Cowan 1988).

Following recognition of tuberculosis-infected possums as a significant source of persistent infection in cattle herds in the early seventies it became the policy of the Ministry of Agriculture and Fisheries to conduct possum population control operations in areas where there was a persistent cattle tuberculosis problem and tuberculous possums were found on or near the property. In the early eighties it was concluded that despite the huge financial investment incurred in reducing possum population density, total eradication of tuberculosis in the major areas with endemic tuberculosis infection in possums, was neither technically feasible nor cost-effective. The modified objective therefore became to achieve a break-even point between the costs of reactor compensation and possum population control (Anon. 1984). In 1985 the objectives of the tuberculosis control scheme were redefined and included the cost-effective prevention of spread of tuberculosis infection from endemic areas (Anon. 1985). According to the Chief Veterinary Officer's annual reports for the period it was decided in 1986 in consultation with ecologists to establish low possum density buffers of about 3-5 km width on the edge of a major endemic area in Central North Island to contain the spread of tuberculosis infection in possum populations (Anon. 1986, Livingstone 1988). This policy was revised in 1989 when tuberculous possums were found outside the buffer zone. The current objective is to restrict the spread of tuberculous possums from the major endemic areas and to reduce cattle tuberculosis incidence by localised cost-effective possum population control within these areas. Based on a results from a deterministic simulation model, management plans for eradication of tuberculosis from possum populations were developed for a number of small- to medium-sized endemic areas (Anon. 1989).

At the end of the tuberculosis testing year 1991/92 there were 20 areas in New Zealand where tuberculosis infection was present in possum populations. In 1991/92 about 83% of cattle reactors to the tuberculin test and herds under movement control restrictions were found in these areas (TB endemic and tuberculosis investigation areas; O'Hara 1993). The predicted cost of the New Zealand bovine tuberculosis control programme for the financial year 1992/93 was NZ\$ 21.82 million. Of this sum a total of NZ\$ 5.1 million was to be spent on possum population control (Anon. 1992).

## **CHAPTER 3**

### **REVIEW OF THE LITERATURE**

In this review special emphasis will be given to the epidemiology of bovine tuberculosis in species of potential importance as disease reservoirs. A section on human tuberculosis is included to take advantage of the wealth of historical information available on the epidemiology of tuberculosis in humans.

### EPIDEMIOLOGY OF TUBERCULOSIS IN HUMANS

Tuberculosis infection occurs throughout the world. A recent report by the World Health Organization (Anon. 1992) stated that about a third of the world population is harbouring the pathogen. Tuberculosis in man is caused primarily by *Mycobacterium tuberculosis*, but *Mycobacterium bovis* remains a contributor to the total disease, even though it is now much less important than it was in the nineteenth century before control of transmission from cattle. The World Health Organization reports that in humans there are about 20 million active cases of tuberculosis in the world, who probably infect 50 to 100 million people (mainly children) annually. It is estimated that about 3 million die due to the disease every year, at least 80% in developing countries (Stead and Dutt 1988). As species identification is not carried out routinely, it is difficult to estimate the present contribution of *Mycobacterium bovis* to total tuberculosis morbidity and mortality in humans (Pritchard 1988).

Since the beginning of this century the epidemiology of human tuberculosis has been studied extensively. This review looks at parallels between mycobacterial infection in humans and animals. Rich (1951) has reviewed reports of the occurrence of tuberculosis caused by *Mycobacterium bovis* in humans. He writes that after Koch's insistence that this bacillus would not be pathogenic in man, it took a number of years before it was generally accepted that *Mycobacterium bovis* can cause all of the forms of tuberculosis which the human-type bacillus is able to produce. Milk turned out to be the most important source of *Mycobacterium bovis* infection for humans. Hence, the incidence of infection in any area where the bacillus was prevalent in cattle was determined by the extent to which raw milk and milk products were consumed, which in turn was determined by local habits. Rich quotes a number of studies from the beginning of this century where up to 60% of cases with bone and joint tuberculosis and 90% of cases with tuberculous cervical adenitis in Scottish children were found to be caused by infection with *Mycobacterium bovis*. At that time human tuberculosis of bovine origin was proportionately more common in the British Isles than in any other industrialised country in the world. It was estimated that around the beginning of this century at least 2000 children died annually from bovine tuberculosis in Great Britain, because powerful dairy interests had prevented the introduction of legislation requiring the pasteurization of milk sold to the public. Rich writes that infection and progressive disease caused by *Mycobacterium bovis* is more likely to occur in children than in adults. He attributes this to a combination of factors. These include, that children are more susceptible, that they are more likely to be exposed and that infection through the alimentary tract (which is the main portal of entry for the bovine bacillus) is easier to achieve. Rich extensively discusses the relative importance of the alimentary and respiratory tract as portals of entry with particular reference to *Mycobacterium tuberculosis*

infection in humans. He concludes that the respiratory tract is the most common portal of entry, and that to achieve infection via the alimentary tract far greater quantities of bacteria are needed. Rich writes that intrauterine infection occurs infrequently. He also mentions that infection can be induced by dropping tubercle bacilli into the conjunctival sac, which has led to cervical adenitis and generalized tuberculosis in the absence of a macroscopic local conjunctival lesion. Rich believes that there is no greater susceptibility in humans towards infection with *Mycobacterium tuberculosis* than there is to *Mycobacterium bovis*.

Yates and Grange (1988) suggest that despite virtual eradication of bovine tuberculosis infection in cattle in the region, about 1% of bacteriologically proven tuberculosis cases in South-East England are still caused by bovine tubercle bacilli. In the period 1954-1957 in areas in Germany with high cattle reactor rates, up to 25% of tuberculosis cases in humans were caused by *Mycobacterium bovis*. By comparison only 2.7% of human tuberculosis cases in an area with low cattle tuberculosis incidence were related to infection with *Mycobacterium bovis* (Schliesser 1982).

Blood and Radostits (1989) write that complete eradication of bovine tuberculosis has not been achieved in any country of the world, but in many a state of virtual eradication with minor recrudescences can be claimed. The complex epidemiology of this organism, which has one of the broadest host ranges of all pathogens, complicates control efforts (Grange and Collins 1987).

The transmission dynamics of *Mycobacterium bovis* infection changed considerably from prehistoric times to the present. This was mainly associated with the rise of civilization. There are some reports which consider the possibility that after the domestication of cattle a mutation occurred in the bovine bacillus to create the human type (Lancaster 1990, Wadsworth 1984). Due to its chronicity, tuberculosis would have been able to perpetuate itself in small communities. But, it has not been detected in many modern day isolated communities, which leads Lancaster to the conclusion that it can be thought of as a common or even true human disease only after the development of agricultural societies. There is some evidence from prehistoric skeletons that it existed in simple early human societies, even though it was uncommon among such groups (Cohen 1989, Clark *et al* 1987).

Stead and Bates (1980) describe *Mycobacterium tuberculosis* as having the potential to cause epidemics when introduced into any susceptible populations. They write that early skin-testing data shows that most people in Europe eventually became infected, but only some of these developed disease. One possible explanation of this is that there are variations in natural resistance to tuberculosis, which over several generations resulted in the elimination of the relatively more susceptible population, although a simpler explanation would be that not all individuals were exposed to sufficient risk factors to precipitate the conversion of their subclinical infection into clinical disease.

First reports on the current tuberculosis pandemic are from 16<sup>th</sup> century England. Stead and Bates estimate that the disease requires about 300 years to complete its course in one geographic area. There is some data which suggests that the present pandemic began in 16<sup>th</sup> century England, reaching a peak in 1750 during the early beginnings of the industrial revolution. During this period of urbanization the life-style of people changed significantly and as a result the risk of person-to-person spread was considerably higher. From England it spread all over the world, reaching high incidence in western Europe in the early 1800s, in the late 1800s in North America and still has not reached its peak in some developing countries. Stead and Bates suggest that the downward slope of tuberculosis mortality was only little affected by tuberculin testing, BCG vaccination and early chemotherapeutic efforts until in 1952 isoniazid was introduced as a chemotherapeutic agent.

Mercer (1990) reviewed changes in tuberculosis incidence and mortality since the 18<sup>th</sup> century in England. He writes that the disease had become endemic in the towns of England by the 18<sup>th</sup> century and 'consumption', as the disease in humans was called, contributed about 15-20% of deaths in the London Bills of Mortality. Sharing of airspaces with infected individuals along with under-nutrition were likely to have affected the risk of clinical disease establishing from initial contact, while other living conditions affected the risk of mortality on re-activation of initially dormant disease later in life. During both centuries people in towns were likely to have contracted primary infection at an early age. Its destructiveness has to be seen in the context of the other epidemic diseases to which everyone was exposed in overcrowded living conditions. Mercer writes that suppression of tuberculin reaction has been found during scarlet fever, glandular fever, measles infection and probably smallpox. He points out that the actual physical disruption produced by the respiratory complications of such infections was probably even more important. During the second half of the 19<sup>th</sup> century there was a decline in mortality, but the population continued to have 100% contact with the tubercle bacillus. Mercer suggests that this decline could have been an indirect benefit of vaccination against smallpox, since this disease probably re-activated latent infection or weakened the resistance of the survivors. He does not believe that any immuno-genetic changes can account for the decline in tuberculosis death rate, particularly as the trends for non-respiratory forms of tuberculosis did not fall much in the second half of the 19<sup>th</sup> century. Mercer thinks that changes in transmission were rather unlikely to account for the decline in mortality from tuberculosis, as during this time the population in towns was increasing most rapidly but there were only slight improvements in general living conditions. The first countries to industrialise were also the first to experience the downturn in tuberculosis mortality. But even in the 1940s tuberculosis case-fatality was about 50%. Mercer notes that in addition to preventive measures used against tuberculosis such as vaccination and chemotherapy, changes in family size (initially among the middle classes), and in housing conditions among the less well-off, could have been involved with a reduction of transmission rates and severity of air-borne infectious disease.

Stead and Bates (1980) reviewed factors of importance in the epidemiology of *Mycobacterium tuberculosis* infection in humans. They consider industrialization and urbanization resulting in crowded living conditions as more important than any other single factor. Low socio-economic status appears to be associated with increased tuberculosis mortality. Poor nutrition is probably less important, as are racial differences. Stead and Bates emphasize that individual infection is more likely, given a sustained and intimate contact with infectious persons resulting in the probability of multiple exposures. Since infection with *Mycobacterium bovis* has become a rarity in cattle and humans, infection through inhalation is considered the most important transmission path. Size of the inhaled particle is more important than the quantity of organisms. Stead and Bates quote a landmark study by Riley and colleagues (1962) which demonstrated that infectious particles having the aerodynamic properties of droplet nuclei were capable of passing through the duct system from a hospital ward occupied by sputum smear-positive patients and infecting guinea pigs kept on the roof. Droplet nuclei are so small that they can stay constantly airborne and can rapidly disperse throughout an enclosed atmosphere. These particles are not filtered by simple gauze masks. It was shown that in guinea pigs and mice almost all tubercle bacilli that were inhaled as single organisms, reached the lung alveoli and produced a tubercle. Survival of aerosolised bacilli is brief, with only about 1 percent surviving for several hours. It was therefore concluded that effective prevention of transmission has to be targeted at these aerosolised particles. In a recent official statement the American Thoracic Society (Anon. 1990) points out that because of the extraordinary significance of airborne droplet nuclei for successful transmission of *Mycobacterium tuberculosis* any of the other methods which were once thought to be important for preventing infection such as disposing of clothes and bedding, sterilizing fomites, using caps and gowns and gauze or paper masks, boiling dishes, and washing walls are unnecessary because they have no bearing on airborne transmission.

Schliesser (1985) reviewed the epidemiology of bovine tuberculosis in humans. He emphasizes the importance of aerogenous as well as alimentary transmission from cattle to humans. Traditionally infection was attributed mainly to the consumption of milk from tuberculous cattle. Schliesser quotes work by Schmiedel (1970) which suggests that due to its high fat content which increases absorption into the lymph system, milk represents an ideal medium for alimentary transmission. Schliesser notes the accepted fact that pasteurization kills pathogenic bacteria in milk if the correct procedures are followed. In the absence of pasteurization, bacteria contained in milk products can remain infectious for extended periods of time. This can be up to 100 days in butter and 322 days in certain types of cheese. Schliesser states that meat from tuberculous animals may constitute a significant risk of infection if available for consumption. *Mycobacterium bovis* has even been isolated from mince meat. Schliesser quotes Jensen (1937) who considered the risk of aerogenous infection as being higher in a shed with tuberculous cattle than in a hospital with tuberculosis cases. Infection by direct contact would be a risk factor for certain occupations such as farmers, abattoir workers and veterinarians.

In Australia human tuberculosis caused by *Mycobacterium bovis* is now rare. About 10 cases are recorded every year. Most of them show pulmonary lesions and are usually older people, but in earlier times a number of younger abattoir workers probably did become infected by aerosol transmission from cattle (Patel and Streeton 1990). In New Zealand the annual incidence of tuberculosis infection (including *Mycobacterium tuberculosis* and *Mycobacterium bovis*) averaged 4.2 cases per 100,000 population during 1985-90. The risk of infection was higher in Maori and Pacific Islanders compared with people of European ethnic background (Stehr-Green 1992). During 1988 1.8% of a total of 228 *Mycobacterium* isolates from human tuberculosis cases were typed as *Mycobacterium bovis*. All 4 cases were between 41 and 60 years of age. In 3 cases it was possible to isolate *Mycobacterium bovis* from sputum, in all cases from pleural fluid and in 1 of the cases from a urine sample. One case each was reported from Hamilton and Wellington Area Health Board and 2 cases were identified in the Palmerston North area (Anon. 1989). During 1989 a total of 15 cases with specimens yielding *Mycobacterium bovis* isolates were reported by the New Zealand Communicable Disease Centre (Anon. 1989, Anon. 1990). Amongst these were three infants, 8 people older than 41 years of age, and 4 of intermediate age. 13 of the 15 individuals were males.

Concurrently with the recent epidemic of the human immunodeficiency virus (HIV) a resurgence of tuberculosis has been reported from many areas of the world where HIV infection is widespread. One example for which data are available is New York City (Brudney *et al* 1991). The authors discuss the possible reasons for this unexpected development. They found that the disease is a growing problem in impoverished populations, particularly in homeless people living in mass shelters. In this group non-compliance with the treatment regimen was found to be particularly common, further augmenting the disease problem. It was noted that despite the current belief that most tuberculosis cases in HIV patients are related to reactivation of old tuberculosis infection, there is also a substantial amount of primary tuberculosis. This is becoming more likely as there are more inadequately treated individuals with active tuberculosis crowded together with highly susceptible HIV-infected homeless people. In New Zealand an outbreak of tuberculosis in a ward of 30 psychogeriatric patients was reported, which was probably caused by a single undiagnosed fatal index case (Taylor *et al* 1991). The incident probably resulted in three deaths and the infection of 43.3% of the patients accommodated in the closed geriatric ward. Non-compliance with treatment instructions is also likely to result in the appearance of drug-resistant strains of *Mycobacterium tuberculosis*. This has recently been reported from New York prisons, where 13 HIV-infected inmates died due to tuberculosis treatment failure (Purvis 1991).

The occurrence of AIDS and tuberculosis has been termed 'the cursed duet' (Chretien 1990). Tuberculosis is considered the only AIDS-related opportunist disease that can infect healthy members of the community. Tuberculosis infection may hasten the onset of AIDS in otherwise asymptomatic HIV-infected individuals, possibly by several years (Festenstein and Grange 1991). Contrary to the general belief that tuberculosis follows immunosuppression

caused by HIV infection, it has been suggested that immune suppressant conditions such as tuberculosis, malnutrition and others may have preceded HIV infection and facilitated its transmission (Packard and Epstein 1991). The recent developments in human tuberculosis point to the continued importance of this disease, which was once considered to be nearly conquered (Rich 1951).

## **EPIDEMIOLOGY IN DOMESTIC ANIMALS**

### **Cattle**

The first confirmed description of *Mycobacterium bovis* infection in cattle is based on a report by Columella in the year 40 A.D.. In the 17<sup>th</sup> and 18<sup>th</sup> century in Germany "Perlsucht" (a term referring to the grape-like lesions) was considered a symptom of syphilis, which resulted in strict procedures for the disposal of the affected animals. When this misconception was corrected, the control measures were removed. It was considered possible to eat meat from tuberculous animals and there were then no obstacles in the way of the spread of the disease. It was not until Robert Koch identified *Mycobacterium bovis* that the relationship between "Perlsucht" and lung tuberculosis in cattle was established (Schliesser (1982).

Based on the tuberculin test, the first disease control programme which was successfully implemented was Bang's eradication scheme in Denmark in 1892. In Germany such a programme based on tuberculin testing every animal was not considered economically feasible. Ostertag's eradication scheme which relied on the detection of animals with open lesions was introduced in Germany in 1912 on a voluntary basis. In 1939 the scheme was discontinued, because it was found that reactor rates were higher in herds which took part in the programme. In 1952 a compulsory tuberculosis control scheme was introduced in West Germany which was based on identification of infected cattle using the tuberculin test. The number of tuberculosis-free herds increased from 9.9% in 1952 to 99.7% in 1961. Schliesser (1982) discussed the major factors associated with maintaining reactor rates in cattle at a low level. Major problem areas included the specificity of the tuberculin test and the risk of reintroduction into tuberculosis free herds. The latter, Schliesser attributes mainly to international trade of animals and their products and to transmission from *Mycobacterium bovis* infected humans. He also points out the differences between the epidemiology of tuberculosis in humans and domestic animals.

During the last 100 years in industrialized countries human living conditions have improved and infection rates have decreased despite the absence of a disease eradication programme. On the other hand, during the same period of time changes in domestic animal husbandry occurred (such as increased herd sizes, higher stocking densities and greater numbers of animal movements) which facilitated transmission and spread of infectious diseases. In Germany by 1975 such good progress had been made that it was possible to reduce herd testing to once every three years. Since then, the number of infected herds has steadily decreased, but the number of reactors within infected herds has increased (Schliesser 1985).

Weber *et al* (1988) report that the proportion of reactors in infected herds increased with the time elapsed since the last tuberculin test.

In intensive production systems bovine tuberculosis is more common in dairy cattle than in beef cattle. This is related to the fact that dairy cattle are usually kept in sheds in close contact with one another whereas beef cattle are usually kept on pasture. The different age structure of the two major cattle production systems may also be of importance. Prevalence increases with age and dairy cattle are usually kept longer than beef cattle (Schliesser 1985). Francis (1958) quotes Villemin (1868) who observed higher prevalences of tuberculosis in those cattle which were kept indoors. In extensive cattle production systems in North and South America, in Asia and Africa tuberculosis usually is less prevalent. But in some situations (Africa, Australia) high incidences have been reported in range cattle. This may be related to aggregation of animals around watering points. Shortridge (1981) analysed the patterns of tuberculosis infection in a problem area in New Zealand. He considered congregation of animals around water ponds and dams as conducive to the spread of infection within herds.

Recent studies in Northern Ireland suggest that within- and between-herd transmission through aerosolised secretions may be of continued importance in the epidemiology of bovine tuberculosis. It was found that in cattle infected with *Mycobacterium bovis*, lung lesions and nasal excretion occurred frequently and could be diagnosed from 2 months after the last clear tuberculin test (McIlroy *et al* 1986, Neill *et al* 1988). Dunnet *et al* (1986) also mention in their report that analysis of breakdown data from Great Britain shows that tuberculosis is rarely transmitted to neighbouring herds. They conclude that the presence of non-reacting excretors is more likely to affect within- herd transmission. Dunnet *et al* mention the importance of the introduction of infection through purchases of tuberculous cattle. Especially in the early 1970s imported Irish cattle were related to a significant number of breakdowns in Great Britain. In Ireland in areas with a high incidence, transmission between cattle is considered the principal means of spread. In some areas other transmission paths are also thought to be of relevance (O'Connor and O'Malley 1989). Downey (1990) outlined the main reasons for failure of the Irish tuberculosis eradication programme in containing spread of infection. He mentions that one of the factors involved was residual infection within herds which had not been detected by tuberculin testing. A major reason for failure was spread from infected cattle to other cattle by movement and contact, especially under stressful conditions.

There have been few attempts to model the epidemiology of bovine tuberculosis infection within cattle herds. As part of the Australian Brucellosis and Tuberculosis Eradication Campaign (BTEC) a deterministic simulation of the interaction of bovine tuberculosis in range cattle was developed to evaluate the effect of different disease control strategies in northern Australia (Stoneham and Johnston 1987). In these areas in many large herds it had not been possible to eliminate infection despite immense testing and culling pressure. Herds appeared to clear up, but sometimes the disease re-emerged at even higher incidence than the original level. The results of several simulation model runs suggest that the disease can be maintained at low

levels in small matriarchal breeder groups (9 to 15 head) and is spread by wandering infected males. Disease build-up is generally slow except in situations where cattle congregate in very large numbers. Efficient mustering to ensure that all groups are represented in the yards for testing would probably be more important than test efficiency. It was recommended that when an infected female was detected all members of its social group should be culled to reduce the risk of undiscovered residual infection. The model was used to estimate the management pressure which had to be exerted for particular geographic locations to achieve tuberculosis eradication, depending on cattle density and water availability.

Livingstone (1985) developed a computer simulation model of tuberculosis in a cattle herd. He tested the hypothesis that tuberculosis can persist due to factors such as incomplete mustering, presence of anergic infectious animals in the herd and infrequent testing. Modelling results were compared with actual cattle TB testing information from the West Coast of the South Island, New Zealand. Progression of the disease after the addition of one infected animal was significantly slower in extensively farmed herds compared with those intensively farmed. It was concluded that infection cannot be maintained, given the current testing and culling pressure in this area, without an external source of infection.

### **Farmed Deer**

In 1978, tuberculosis infection was reported from farmed red deer in New Zealand (Beatson 1985). It is now considered the most important bacterial disease in farmed deer in New Zealand and in the United Kingdom (de Lisle and Havill 1985).

It has been suggested that deer kept under farm conditions may be more susceptible to *Mycobacterium bovis* infection than are cattle. Given the circumstances with regard to behavioural and environmental factors, extensive lesions can develop rapidly which results in increased probability of spread within a herd (Clifton-Hadley *et al* 1991). As is the case with cattle, the pathogenesis of the disease is mainly dependent on the size of the infecting dose and the susceptibility of the host. The latter depending on variables such as genetic constitution, previous exposure to *Mycobacterium spp.*, nutrition, social status in the herd, handling stress and sex hormone levels (de Lisle *et al* 1985).

Recognizing the threat of tuberculosis to the deer farming industry, New Zealand, Denmark and Great Britain have all embarked on tuberculosis control or eradication programmes. They are mainly based on tuberculin skin testing and subsequent slaughter of reactors. Clifton-Hadley and Wilesmith (1991) write that due to the presence of endemic infection in wildlife in New Zealand and in the United Kingdom the eradication of tuberculosis in farmed deer may be difficult to achieve. Tuberculous captive deer have been implicated in New Zealand as a source of infection for possums which are an important wildlife reservoir for bovine tuberculosis (Livingstone 1988).

### **Other Domestic Animals**

Prevalence of tuberculosis infection in small ruminants is believed to be linked to the disease frequency in other hosts such as cattle. In industrialized countries detection of tuberculous lesions in small ruminants is less common when disease levels within the cattle population decreases. In extensive animal husbandry systems transmission probabilities are low resulting in low overall prevalences (Schliesser 1985). In some instances prevalences of up to 5% have been observed in sheep flocks in New Zealand (Davidson *et al* 1981).

Disease levels in pigs also usually reflect the incidence in local cattle populations. Myers and Steele (1969) report that in 1921 in the U.S. 12% of hogs slaughtered under federal inspection were found to have tuberculous lesions. They note that in the mid west of the U.S.A. it was possible to trace 96% of swine carcass condemnations for tuberculosis to feeding of unsterilized skim milk or other dairy products or to keeping them together with cattle. Prevalence in pigs is thought to increase with age. The principal route of infection in the pig is the digestive tract, by consumption of milk or milk products, kitchen and abattoir scraps, and excreta from tuberculous cattle (Acha and Szyfres 1989). Transmission between pigs is considered epidemiologically insignificant, as lesions usually remain localized and pigs are slaughtered at an early age. High disease levels in cattle can result in prevalences of up to 20% in local pigs (Blood and Radostits 1989).

Lepper and Corner (1983) quote work by Snider and Cohen who found tuberculous lesions in four dogs and 24 cats out of 61 contacts on farms with *Mycobacterium bovis* infection. The authors concluded that on premises with tuberculosis infection surveillance of these species is recommended. Yet, it is unlikely that domestic dogs and cats represent a epidemiologically significant factor in the dynamics of tuberculosis infection.

### **EPIDEMIOLOGY IN WILDLIFE**

Tuberculosis has been known as a serious clinical disease in wild mammals in captivity for more than a century (Thoen and Himes 1981). These authors write that it is widely distributed in wild mammal populations in the United States, where outbreaks caused by infection with *Mycobacterium bovis* have been reported mainly from zoos, game parks and primate colonies. Schliesser (1985) notes that in European countries sporadic incidents of bovine tuberculosis in wild mammals were mainly reported before eradication of cattle tuberculosis was achieved. Evidence from various countries in the world shows that given specific epidemiological circumstances significant levels of tuberculosis infection can be found in wild species such as buffalo, goats, pigs, deer, badgers and brush-tailed possums (Lepper *et al* 1983). The risk which these reservoirs of infection constitute for infection in domestic animals and man is difficult to estimate.

#### **Badger**

Badgers are an important reservoir of reinfection for cattle in the United Kingdom and Ireland (Zuckerman 1980, O'Connor and O'Malley 1989). The disease is considered to be endemic

throughout badger populations in both countries (Wilesmith 1991, Morris and Pfeiffer 1990). In Great Britain, local surveys of badger populations around areas with recently infected cattle herds revealed tuberculosis prevalences of up to 50% in localized areas (Wilesmith 1991). In the Republic of Ireland, during the period 1980-1989 17.4% of 3915 badgers examined showed evidence of tuberculosis infection. Infection was present in every county of the country (Dolan 1990). In a local post-mortem survey in an area with a continuing TB problem in the cattle population 48% of 30 badgers showed tuberculous lesions (McAleer 1990). Wilesmith (1991) reviewed the epidemiology of bovine tuberculosis infection in badgers based on the results from a longitudinal study in Gloucestershire, in the southwest of England. Most badgers get infected via the respiratory route. Submandibular abscesses are often found as the first clinical sign of infection. Transmission through bite wounds from territorial fights could account for a higher prevalence in males. Mother to cub transmission could be a major epidemiological factor. It is suggested that adult females are responsible for maintenance of infection within a population. A large proportion of excreting (ie infectious) tuberculous badgers survived for more than 12 months. Mortality induced by tuberculosis does not seem to have an effect on population density. Single badgers with advanced tuberculous lesions shedding large numbers of bacteria in their urine are thought to be the primary source of infection in cattle. Hence, the probability of transmission of infection from badgers to cattle is low and probably occurs mainly as a sporadic incident. The risk increases with increasing badger density (Wilesmith 1991).

In contrast to the findings from the above field study, Mahmood *et al* (1987) challenged badgers with bovine tubercle bacilli in an experimental study and found that intradermal inoculation was almost always successful and intratracheal challenge always failed. The badgers did show immune responses which enabled them to hold the disease in check for up to 22 months. They begin to shed organisms only in the late stages of the disease. Disease is found in local "pockets" of infection, which do not necessarily equate with individual social groups. They may involve a number of contiguous groups (Dunnet *et al* 1986).

Benham and Broom (1989) studied the interactions between badgers and cattle on pasture. They found that badgers were approached by cattle of all ages. Badgers preferred not to use pastures occupied by cattle and avoided approaching closer than 10-15m from cattle. They concluded that tuberculosis transmission would be more likely through badger products such as urine or faeces than direct contact. Other researchers found that badgers did not always avoid cattle (Kruuk 1989). Kruuk also found that badgers need short-grazed pastures to feed efficiently, which could bring them into contact with cattle. Badgers defend their territories ferociously. They form social groups which use the same territory and setts. Female offspring tend to stay with their mother's group. Young males stay for extended periods of time within their mother's social group. They eventually may emigrate into a vacancy in a neighbouring group. This tendency to stay close to their mother's home range could explain why infection has not spread evenly through areas with endemic infection (Cheeseman *et al*

1988). Stuart and Wilesmith (1988) consider that the social structure of the population, their subterranean existence, social grooming and group sleeping are all important factors for maintenance of infection in a badger population.

An attempt has been made to develop a simulation model of the population dynamics of the badger and the epidemiology of bovine tuberculosis (Anderson and Trehwella 1985). A simple mathematical model was developed which was mainly aimed at identifying areas where current knowledge was inadequate. It was recognized at that time that the understanding about the epidemiology of the disease was only limited. The modelling results suggest that average prevalence is positively correlated with badger density. The disease should be able to persist endemically at low badger population densities due to factors such as frequent pseudo-vertical transmission and long survival periods of infectious animals. Anderson and Trehwella (1985) suggest that a high risk of cattle TB breakdowns is probably associated with high tuberculosis prevalence in infected badgers, high cattle herd density on pastures close to good badger habitat and farm management practices such as allowing badgers access to cattle sheds, salt licks and water troughs. The authors predict that very substantial reductions in badger abundance are necessary to induce marked changes in prevalence within the badger population. However, moderate reductions in badger density would significantly reduce, but not eliminate the risk of cattle herd infections.

### **Brush-Tailed Possum**

Between the beginning of the nineteenth century and the start of this century the Australian common brushtailed possum (*Trichosurus vulpecula* Kerr) has been introduced and liberated at numerous locations in New Zealand (Pracy 1962). The susceptibility of possums to *Mycobacterium bovis* infection was first experimentally demonstrated in 1948 by Bolliger and Bolliger (1948). In 1967 the first possum with tuberculous lesions to be identified in New Zealand was found by a trapper on a farm with a persistent tuberculosis problem. In this and later cases it was reported that in all tissues examined, large numbers of *Mycobacterium bovis* organisms were present and the animals were discharging organisms through open sinuses or the respiratory system (Ekdahl 1970).

As a result of these findings, in 1970/71 the New Zealand Ministry of Agriculture and Fisheries conducted an epidemiological survey of possum tuberculosis in the Buller and Inangahua Counties. The study had a duration of one year. Possum kill operations were done at monthly intervals. 4.9% of 5908 possums captured showed tuberculous lesions on post mortem examination. Monthly prevalence ranged from 2.5% in August to 9.35% in December. In mature possums there was a trend towards higher prevalences in summer than in the winter months, although the difference was not statistically significant. Cattle tuberculosis incidence rates declined from the beginning to the end of the survey. 65% (N=100) of tuberculous possums had lung lesions and 52.6% (N=81) had lesions in the axillary and pre-scapular lymph nodes. It was 2.3 times as likely that infection would be found in a possum population if infected cattle were grazing in the same area, although the difference was not statistically

significant ( $p > 0.1$ ). A "spring rise" in cattle tuberculosis incidence was attributed to the fact that on the New Zealand West Coast it is common practice to graze herds in bush areas during winter months (Cook 1975).

In 1973/74 a one year study of *Mycobacterium bovis* infection in possums involving two-day trapping surveys at two-monthly intervals was conducted in the Hohonu Range, New Zealand, in a cooperative project between the New Zealand Forest Research Institute, Christchurch and the Ministry of Agriculture and Fisheries. An overall tuberculosis prevalence of 7.7% in a total of 1486 possums was found. Both tuberculosis prevalence and relative density of possums were highest on pasture and decreased from the bush/pasture margin to the remote forest. Tuberculous possums were clustered in small foci of 2-5 animals. It was suggested that indirect transmission through sequential or simultaneous den sharing might be an important infection route. Seasonal variation in prevalence was observed, with highest levels in autumn and winter along the pasture margin and in spring within the forest. It was suggested that possums were at higher risk of infection during summer and autumn months due to increased foraging and social activities and that the rise in detectable prevalence in winter reflects the length of the incubation period. It was found that infection was more likely in immature male possums than in immature female possums. The body condition of the possums appeared to be worse in infected than in uninfected possums. 29.5% of tuberculous animals showed lesions in the axillary lymph nodes and 55.4% in the lung (Cook undated, Coleman 1988).

In an experimental study on the course and pathology of the disease in possums it was possible to demonstrate initiation of infection by subcutaneous and intranasal inoculation and transmission through direct and close contact, both by aerosol and by mother - joey contact (pseudo-vertical). Disease developed rapidly to a fatal stage and lesions were typically widely disseminated. However, it was realized that the disease process might have been accelerated by reduced body defenses in captive possums and that the infection dose was very high (O'Hara *et al* 1976).

In 1981 the pathology and histopathology of *Mycobacterium bovis* infection in possums was reviewed by Julian (Julian 1981). Distribution of lesions was summarised, including data on 327 tuberculous possums necropsied in three different areas. In 62% (N=203) of the cases, superficial lymph nodes and in 61.8% (N=202) the lungs were involved in the disease process. The pathological appearance of affected somatic lymph nodes is typically a soft fluctuating abscess with a diameter of up to 4 cm containing semi-liquid lime green pus, which on occasion may form open sinuses to the exterior. In visceral organs and lymph nodes white to yellow nodules of up to 2 cm in diameter are most commonly seen. These lesions may be multilobular with a more caseous centre than is seen in typical somatic lesions. Miliary white lesions occur in lungs, liver, spleen or kidneys. In lungs, generalised grey-white consolidation of lobes or part of a lobe has been observed. Histopathologically, lesions consist typically of granulomatous tissue, with no distinctive fibrous capsule and with varying amounts of

amorphous eosinophilic debris centrally. Lymphocytes, plasma cells, macrophages, giant cells and many neutrophils in great concentration around the caseous material were observed in the reaction process. Large numbers of acid-fast organisms were present. Typical tubercles (including epithelioid cells, Langhans' giant cells and capsule formation) were rarely seen.

Results of a preliminary analysis of the data from the possum tuberculosis survey in the Hauhungaroa Ranges have been published previously (Hennessy 1986), but are analyzed more fully in this thesis. A mean prevalence of 1.4% was reported. There was no association between relative density estimates (derived from trapping and faecal pellet count data) and tuberculosis prevalence in possums. No statistically significant association between infection and sex, breeding ability and condition was found. Sexually mature animals were more likely to be infected than immature animals ( $p < 0.05$ ). A correlation coefficient of 0.9 was observed between cattle tuberculosis incidence (averaged estimates for 11 zones) and possum tuberculosis prevalence. It was concluded that level of infection in cattle represents a good indicator for level of infection in adjacent possum populations. Hence, in areas with endemic tuberculosis infection in possums direct inference from presence of infection in cattle to presence of tuberculous possums would be possible.

Based on current knowledge of possum ecology and the epidemiology of tuberculosis in possum populations a deterministic simulation model was developed to aid the understanding of the disease problem, evaluate different options for control strategies and identify future directions of research. The model suggested that disease in possums has to be aggregated locally at high prevalence levels to persist at the relatively low overall prevalence which has been observed in possum population surveys all over the country. It showed that assuming immigration is insignificant, eradication of tuberculosis infection within a possum population is theoretically possible if 16% of the possum population is removed every year. The model predicts that control of tuberculosis in endemic areas is most likely to be successful using repeated single possum population control operations or an initial single control operation, followed by sustained cropping or vaccination. In the light of the high cost involved in intensive possum control operations it was suggested that separation of cattle from margins of high-density possum habitats, or ground control operations targeted at clusters of disease aggregation may be more cost-effective (Barlow 1989).

### **Feral Buffalo and Bison**

Bovine tuberculosis is known to be endemic in feral swamp buffalo populations of the Australian Northern Territory. Hein and Tomasovic (1981) found a prevalence of 0.017 in 11322 buffalo examined during routine post-mortem examination at 2 abattoirs during 1979. This was a significant reduction compared with a prevalence of 0.16, which was reported by Letts (1964) in 1964. This reduction in prevalence was explained by Hein and Tomasovic (1981) as due to selective harvesting of mature animals for meat, resulting in removal of many tuberculous animals from the population. But as prevalence was highest along the relatively narrow coastal strip of land, which was mainly sampled during the earlier survey, differences in

habitat may provide another explanation for the decrease in prevalence. The authors suggest that lower population density of feral buffalo may affect transmission probability. The large proportion of cases with sole or predominant involvement of the thoracic organs suggests that as in cattle the respiratory route is the most important transmission path in feral buffalo. McCool and Newton-Tabrett (1979) indicate that 97% of buffalo tuberculosis in northern Australia is contracted via the respiratory route. They considered this finding rather surprising in view of the work by Tulloch (1978) on buffalo behaviour which showed that there was ample opportunity for both alimentary and respiratory infection. In fact, Tulloch was reported to have estimated buffaloes were spending on average 4 hours per day in intimate contact in communal wallows. McCool and Newton-Tabrett write that this extended period of close contact is not seen in northern Australian cattle when resting around watering points, only during the few minutes when they drink from crowded water troughs. They concluded with respiratory transmission being the main method of spread, sources of mycobacteria in the environment are probably unimportant. Hence, control of tuberculosis in feral buffalo populations should be possible through standard tuberculosis control measures.

Freeland and Boulton (in preparation) developed a model of the epidemiology of bovine tuberculosis in swamp buffalo in the Northern Territory of Australia. They included the effects of social groups into their model. Modelling results suggested that the mode of group size regulation has a major effect on prevalence levels. The importance of matriarchal breeder groups was stressed for maintenance of infection. Spread of disease would depend on the mode of group size regulation (emigration of matriarchal family clans). Small group sizes which resulted from migration of clans were likely to result in low prevalences. Infected males could potentially spread the disease during emigration or when visiting females from other social groups during breeding season.

Woodford (1982) studied the occurrence of tuberculosis in wild Cape buffalo in Ruwenzori National Park, Uganda. He found tuberculous lesions in 10% of 52 buffaloes from a random sample and 38% of 64 animals which were selected based on being in poor condition. *Mycobacterium bovis* was identified in 12 of 14 cases. Most cases appeared to be infected by respiratory transmission and no lesions were seen which could be ascribed to alimentary infection. The author explains this finding with the close herding habits of wild buffaloes and their propensity for wallowing in tight groups in small mud holes which facilitates droplet transmission. Woodford concludes that bovine tuberculosis could cause an annual mortality of about 1% in this particular area.

In 1961 Choquette *et al* (1961) reported that they found tuberculous lesions in 50% of 436 bison from Wood Buffalo National Park, Canada, which had reacted to the tuberculin test. They quote other work by Fuller who wrote that tuberculous lesions had been found in 39% of 1508 bison slaughtered between 1952 and 1956. The same authors refer to a report by Hadwen who during the period between 1923 and 1939 found tuberculous lesions in 53.4% of 12,005 bison slaughtered at Wainwright. Choquette *et al* examined another 500 bison from Elk

Island National Park, which were not tuberculin tested and did not show any lesions on necropsy. They conclude that in Wood Buffalo National Park current tuberculosis control based on tuberculin testing and slaughter of reactors would be at best a disease reduction program. More recently concern has been expressed again about the levels of tuberculosis infection in bison in Canada. Between 1983 and 1985 during a survey in and around Wood Buffalo National Park, Canada, 21% of 72 bison found dead showed tuberculous lesions on post-mortem. The results suggest that infection occurred primarily via the respiratory route. It was concluded that the disease was endemic within the population and that therefore there was a growing risk of spread to uninfected bison and cattle (Tessaro *et al* 1990).

### **Wild Deer**

Infection with *Mycobacterium bovis* has been reported from a number of free-ranging deer species, as pointed out in a recent review by Clifton-Hadley and Wilesmith (1991). These authors and others report that there have been some incidents where infected wild deer were suspected of introducing infection into captive deer populations (Mackintosh and Beatson 1985). Mackintosh and Beatson write that in New Zealand a high proportion of wild deer captured or shot was found to be infected with *Mycobacterium bovis*. It has been suggested that in New Zealand where deer had been live-captured in many parts of the country and then traded for breeding purposes, infected animals probably provided a means for introducing tuberculosis infection into areas which previously had been free of tuberculosis.

### **Other Wild Animals**

Wild pigs have been found to be infected with *Mycobacterium bovis* at significant levels in a number of countries. Letts (1964) confirmed *Mycobacterium bovis* infection in 54% of 149 tuberculosis-like lesions from a total of 260 wild pigs in Australia's Northern Territory which were autopsied. He associated this relatively high prevalence with the pigs living in close association with swamp buffalo. At the end of each dry season hundreds of old buffalo die, thereby providing food and a potential source of infection with *Mycobacterium bovis* taking into account the high incidence of tuberculosis in wild buffalo in the Northern Territory. Corner *et al* examined 751 wild pigs in Australia's Northern Territory and found a infection prevalence of about 19%. No pulmonary lesions were found. Corner *et al* concluded that the wild pig is probably an end host for *Mycobacterium bovis* and not a significant source of infection for cattle. Recently a survey was conducted in New Zealand, where 251 wild pigs were post-mortemed and 31% were found to have tuberculous lesions (Wakelin and Churchman 1991). The authors suggested that the disease possibly had spread between pigs by aerogenous transmission because 33% of infected pigs had either lung or bronchial lymph node lesions. This finding suggests a possible difference in the epidemiology of *Mycobacterium bovis* in Central Otago, New Zealand, because in general the importance of infection in pigs through the digestive tract is emphasized in the literature.

### **Other Species**

Feral goats were found with tuberculosis prevalences of up to 31% within individual groups in areas with endemic tuberculosis in New Zealand (Sansom 1988). The epidemiological significance of bovine tuberculosis in goats is generally considered as minimal. In most cases it is related to the presence of a reservoir of infection in another species, such as the brushtail possum in New Zealand.

Woodford (1982) found that bovine tuberculosis infection was endemic in the wart hog population of Ruwenzori National Park, Uganda. He concluded that the disease must have been introduced with domestic cattle.

Infection in wild carnivorous species has to be expected in areas with endemic tuberculosis in important infection reservoir species such as for example the brush-tail possum, cattle and deer in New Zealand. Allen (1991) reviewed the occurrence of bovine tuberculosis in feral species other than cattle, possums and deer in New Zealand. He suggests that most of the few cases with bovine tuberculosis in feral cats and ferrets which were found in New Zealand were related to infection in at least one of the major reservoir species mentioned above. Cats and ferrets are unlikely to contribute significantly to the maintenance and spread of bovine tuberculosis infection.