CANINE ZOONOSES IN ABORIGINAL COMMUNITIES:
THE EFFECTS OF A CANINE BREEDING
AND PARASITE CONTROL PROGRAM
IN THE KIMBERLEY REGION,
WESTERN AUSTRALIA

VOLUME 1

This thesis is presented for the degree of Doctor of
Philosophy of Murdoch University

by

KATHRYN MICHELLE WILKS
BSc, BVMS (Hons)
Dedicated to my parents,
Michelle, Dick and George.
I declare that this thesis is my own account of my research and contains
as its main content work which has not previously been submitted for a
degree at any tertiary education institution.

................................................

KATHRYN MICHELLE WILKS
ABSTRACT

The hypothesis central to this study is that the implementation of a canine breeding and parasite control program in Aboriginal communities results in a reduction in the reservoir of zoonotic parasites within communities. The effect of the parasite and breeding control program on the health status of dogs as well as the population characteristics of dogs in communities was also investigated.

The study was conducted in 17 Aboriginal communities of the Kimberley region of Western Australia, divided into three regions according to cultural and geographical attributes. All dogs from each community were permanently identified using a microchip system. Owners of dogs were asked the usual location of their animals, the origins of their dogs and the whereabouts of any missing animals at subsequent visits. Every three months dogs were treated with 200μg/kg ivermectin (a potent endo- and ecto-parasiticide) subcutaneously and adult female dogs were treated with an injectable contraceptive (10-30mg/kg proligestone) at the request of their owners. At the time of treatment, dogs were assessed for ecto-parasites and photographs taken for later comparison and diagnosis of alopecic skin conditions. Faecal and blood samples were collected every three to six months and skin scrapings were collected from dogs that were refractory to treatment. The samples were used to determine internal parasite prevalence (using formal ethyl acetate sedimentation), blood parameters (for anaemia status) and evidence of scabies or Demodex infestation.

A pilot study at one group of communities, involving weekly assessment of dogs after one ivermectin treatment, showed that the treatment was effective in reducing the prevalence of scabies (as determined by clinical evaluation), hookworm and ticks. The treatment resulted in improvement in animal health as evidenced by a reduction in the number of dogs with anaemia.

The long-term use of the ivermectin treatments at the other communities showed that over a period of three years, the prevalence of scabies and hookworm had reduced at most areas. The
initial scabies prevalence varied from 17 to 52% and reduced to below 10% for all communities. The hookworm infection rates were affected by seasonal factors, as was evidenced by a seasonal variance in prevalence. Animals that were treated with ivermectin, though, had lower prevalences of hookworm than those that were not.

There was a reasonable compliance rate for contraceptive treatments for female dogs (greater than 60% at each visit) and fewer puppies were born within communities when compared with rates before and after the establishment of the treatment program. High rates of acquisition of puppies from other communities continued to maintain the dog population numbers despite the reduction in breeding within communities.

The dog population was young, biased towards male dogs, and very unstable (almost 50% of dogs died or went missing in a one year period). The rate of dog ownership across the Kimberley varied according to the region investigated and always remained higher or equal to ownership rates at the town centres of the Kimberley Region (as determined by a survey conducted during the study).

Overall the canine parasite and breeding control program resulted in a reduction in scabies and hookworm prevalence in dogs (and hence a reduction in the potential zoonotic transmission), a reduction in dog breeding within communities, an improvement in dog health, and an understanding in the dynamics and health status of dogs within communities.
# TABLE OF CONTENTS

<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>DECLARATION</td>
<td>(i)</td>
</tr>
<tr>
<td>ABSTRACT</td>
<td>(ii)</td>
</tr>
<tr>
<td>TABLE OF CONTENTS</td>
<td>(iv)</td>
</tr>
<tr>
<td>LIST OF TABLES</td>
<td>(xvii)</td>
</tr>
<tr>
<td>LIST OF FIGURES</td>
<td>(xx)</td>
</tr>
<tr>
<td>ACKNOWLEDGEMENTS</td>
<td>(xxiii)</td>
</tr>
</tbody>
</table>

## CHAPTER 1  INTRODUCTION

1.1 History and Health in Aboriginal Communities of the Kimberley Region  
1.2 Dogs in Aboriginal Communities  
1.3 The Research Problem  
1.3.1 Aims and Scope of the Research  
1.3.1.1 Canine Population Control  
1.3.1.2 Parasite Control  

## CHAPTER 2  A REVIEW OF THE LITERATURE

2.1 The Association between Aboriginal People and their Dogs  
2.1.1 Health Considerations of the Association between Aboriginal People and their Dogs  
2.1.1.1 Care and Feeding of Dogs  
2.1.1.2 Aversion to Killing Dogs  
2.1.1.3 Disease  
2.1.1.4 Mythology and Education  
2.2 Health Status in Aboriginal Communities  
2.2.1 Life Expectancy of Aboriginal People  
2.2.2 Hospital Records  
2.2.3 Skin Diseases  
2.2.3.1 Pyoderma and Scabies  
2.2.3.2 Complications of Pyoderma  
2.2.4 Gastrointestinal Infections  
2.2.4.1 Causes of Gastroenteritis  
2.2.5 Reasons for Infectious Diseases in Aboriginal Communities
### 2.2.5.1 Environment  

Page No. 16

### 2.2.6 The Role of Dogs in Causing Disease in Aboriginal Communities  

Page No. 17

### 2.3 The Canine Zoonoses  

Page No. 18

#### 2.3.1 *Sarcoptes scabiei*  

Page No. 18

1. Lifecycle of *Sarcoptes scabiei*  

2. Clinical Features of Canine Infection  

3. Pathogenesis of Scabies Mite Infestation in Dogs  

4. Epidemiology in Dog Populations  

5. Skin Contact and Transmission of *Sarcoptes scabiei*  

6. Species Cross Transmission  

7. *Sarcoptes scabiei* as a Zoonosis  

- 2.3.1.1 Humans at Risk of Infection with Canine Scabies  
- 2.3.1.2 Clinical Manifestations of Human Infection with Canine Scabies  
- 2.3.1.3 Transmission of Human Scabies to Dogs  

Page No. 24

#### 2.3.2 *Ancylostoma caninum*  

Page No. 27

1. Hookworm Infection in Dogs  

2. Prevalence of Hookworm in dogs  

3. Seasonality of Hookworm Infection Rates  

4. Arrested Development of Hookworm  

5. Canine Hookworm as a Zoonosis  

- 2.3.2.1 Prevalence of Human Infection with Canine Hookworms  
- 2.3.2.2 Cutaneous Larva Migrans  
- 2.3.2.3 Visceral Larva Migrans  

Page No. 37

- 2.3.2.5.3 Eosinophilic Enteritis  

#### 2.3.3 *Toxocara canis*  

Page No. 40

1. Canine Infection with *Toxocara canis*  

2. Distribution of *Toxocara canis* throughout the World  

3. *Toxocara canis* as a Zoonosis  

- 2.3.3.1 Route of Infection  
- 2.3.3.2 Migration in the Human Host  
- 2.3.3.3 Clinical Features of Human Zoonotic Infection with *Toxocara canis*  
- 2.3.3.4 *Toxocara canis* Larvae in the Central Nervous System  
- 2.3.3.5 *Toxocara canis* as a Virus Vector  
- 2.3.3.6 Incidence of *Toxocara canis* Infection in Humans  

Page No. 47

#### 2.3.4 *Echinococcus granulosus*  

Page No. 48

1. Clinical manifestations of Infection with *Echinococcus granulosus* in Humans  

2. Prevalence of *Echinococcus granulosus*  

#### 2.3.5 Bacterial Zoonoses  

Page No. 51

1. *Salmonella* Species  

- 2.3.5.1 *Salmonella* infection in Humans  
- 2.3.5.1.2 Prevalence of *Salmonella* Infection in Humans  
- 2.3.5.1.3 Sources of *Salmonella* Infection for Humans  

Page No. 52
2.3.5.1.4 Dogs as Sources of *Salmonella* Infection for Humans 52
2.3.5.1.5 Prevalence of *Salmonella* Infection in Dogs 53
2.3.5.1.6 Sources of *Salmonella* Infection for Dogs 53

2.3.5.2 *Campylobacter* species 54

2.3.5.2.1 *Campylobacter* Enteritis in Humans 54
2.3.5.2.2 Campylobacteriosis in Dogs 55
2.3.5.2.3 Incidence of *Campylobacter* Infection in Humans 55
2.3.5.2.4 Prevalence of *Campylobacter* Infection in Dogs 56
2.3.5.2.5 Dogs as Sources of *Campylobacter* Infection for Humans 56

2.3.6 Other Canine Zoonoses 57

2.3.6.1 Nematode Zoonoses 57

2.3.6.1.1 *Strongyloides stercoralis* 57
2.3.6.1.2 *Trichuris vulpis* 58
2.3.6.1.3 *Dirofilaria immitis* 58

2.3.6.2 Cestode Zoonoses 59

2.3.6.2.1 *Dipylidium caninum* 59

2.3.6.3 Protozoal Zoonoses 60

2.3.6.3.1 *Giardia duodenalis* 60
2.3.6.3.2 *Entamoeba* species 61
2.3.6.3.3 *Cryptosporidium* species 62

2.3.6.4 Bacterial Zoonoses 62

2.3.6.4.1 *Pasteurella* species 62
2.3.6.4.2 *Yersinia* species 63
2.3.6.4.3 Other Gram Negative Bacteria 64
2.3.6.4.4 Other Gram Positive Bacteria 64

2.3.6.5 Mycotic Zoonoses 64

2.3.6.5.1 Dermatophytoses 64

2.3.6.6 Viral Zoonoses 65

2.3.6.7 Rickettsial Zoonoses 66

2.3.6.7.1 *Coxiella burnetii* 66

2.4 Control Measures to Reduce Canine Zoonoses 66

2.4.1 The World Situation 66
2.4.2 Methods to Control Zoonoses 66
2.4.3 Dog Population and Zoonoses Control Programs for Aboriginal Communities 69

2.4.3.1 Canine Population Control 70

2.4.3.1.1 Ovariohysterectomy 70
2.4.3.1.2 Hormonal Control of Oestrus 71

2.4.3.2 Canine Parasite Control 77
<table>
<thead>
<tr>
<th>Topic</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.4.3.2.1 Features of Ivermectin</td>
<td>77</td>
</tr>
<tr>
<td>2.4.3.2.2 Mode of Action of Ivermectin</td>
<td>78</td>
</tr>
<tr>
<td>2.4.3.2.3 Efficacy of Ivermectin</td>
<td>78</td>
</tr>
<tr>
<td>2.4.3.2.4 Pharmacokinetics and Efficacy</td>
<td>82</td>
</tr>
<tr>
<td>2.4.3.2.5 Side Effects of Ivermectin Treatment</td>
<td>83</td>
</tr>
</tbody>
</table>

**CHAPTER 3  GENERAL METHODOLOGY**

<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>3.1 Introduction</td>
<td>87</td>
</tr>
<tr>
<td>3.2 The Study Communities</td>
<td>87</td>
</tr>
<tr>
<td>3.2.1 Cultural considerations</td>
<td>87</td>
</tr>
<tr>
<td>3.2.1.1 Communication and Timing</td>
<td>87</td>
</tr>
<tr>
<td>3.2.1.2 Customs and Conduct</td>
<td>89</td>
</tr>
<tr>
<td>3.2.1.3 Methods Used to Encourage Continuation of the Program</td>
<td>90</td>
</tr>
<tr>
<td>3.2.2 Community Profiles</td>
<td>91</td>
</tr>
<tr>
<td>3.2.2.1 The Kimberley Region</td>
<td>91</td>
</tr>
<tr>
<td>3.2.2.1.1 Natural Physical Attributes of the Kimberley Region</td>
<td>91</td>
</tr>
<tr>
<td>3.2.2.1.2 Population of the Kimberley</td>
<td>92</td>
</tr>
<tr>
<td>3.2.2.1.2.1 The Urban Centres of the Kimberley</td>
<td>93</td>
</tr>
<tr>
<td>3.2.2.1.2.2 Aboriginal Communities of the Kimberley</td>
<td>93</td>
</tr>
<tr>
<td>3.2.2.1.3 Veterinary Services in the Kimberley Region</td>
<td>94</td>
</tr>
<tr>
<td>3.2.2.2 Communities of the Study</td>
<td>95</td>
</tr>
<tr>
<td>3.2.2.2.1 Communities of the Coastal Region</td>
<td>95</td>
</tr>
<tr>
<td>3.2.2.2.2 Communities of the Central Region</td>
<td>98</td>
</tr>
<tr>
<td>3.2.2.2.3 Communities of the Eastern Region</td>
<td>98</td>
</tr>
<tr>
<td>3.2.2.3 Conclusion</td>
<td>99</td>
</tr>
<tr>
<td>3.2.2.3.1 Similarities between Study Communities</td>
<td>99</td>
</tr>
<tr>
<td>3.2.2.3.2 Differences between Study Communities</td>
<td>100</td>
</tr>
<tr>
<td>3.3 Data Collection</td>
<td>100</td>
</tr>
<tr>
<td>3.3.1 Identification of Dogs</td>
<td>102</td>
</tr>
<tr>
<td>3.3.1.1 Sex</td>
<td>102</td>
</tr>
<tr>
<td>3.3.1.2 Age</td>
<td>102</td>
</tr>
<tr>
<td>3.3.1.3 Breed</td>
<td>102</td>
</tr>
<tr>
<td>3.3.1.4 Name of Dog</td>
<td>102</td>
</tr>
<tr>
<td>3.3.1.5 Name of Owner</td>
<td>103</td>
</tr>
<tr>
<td>3.3.1.6 House Number</td>
<td>103</td>
</tr>
<tr>
<td>3.3.1.7 Origin of Dog and Status</td>
<td>103</td>
</tr>
<tr>
<td>3.3.1.8 Microchips</td>
<td>104</td>
</tr>
<tr>
<td>3.3.2 Capture and Restraint</td>
<td>105</td>
</tr>
<tr>
<td>3.3.3 Education and Community Support</td>
<td>106</td>
</tr>
<tr>
<td>3.3.4 Drug Treatments</td>
<td>107</td>
</tr>
<tr>
<td>3.3.4.1 Daily Routine</td>
<td>108</td>
</tr>
</tbody>
</table>
CHAPTER 4 CANINE POPULATION, DISTRIBUTION AND DYNAMICS

4.1 Introduction

4.2 Methodology

4.2.1 Data Collection and Processing

4.2.1.1 Population Structure
4.2.1.2 Population Distribution
4.2.1.3 Population Dynamics

4.2.2 Survey of Dog Ownership in Kimberley Urban Communities

4.2.2.1 Problems with the Telephone Survey
4.2.2.2 Proportion of Population Reached with the Telephone Survey

4.2.3 Statistical Treatment of Data

4.3 Population Structure of Dogs

4.3.1 Results

4.3.1.1 Percentage of Dogs Subsequently Re-presented for Treatment (‘Capture Success’)
4.3.1.2 Age of Dogs
4.3.1.3 Sex of Dogs
4.3.2.4 Dog Breeds

4.3.2 Discussion

4.4 Population Distribution of Dogs

4.4.1 Results

4.4.1.1 Household Distribution
4.4.1.2 Dog Ownership

4.4.2 Discussion
4.4.2.1 Household Distribution of Dogs
4.4.2.2 Dog Ownership
4.4.2.3 Owner age
4.4.2.4 Owner Gender

4.5 Population Dynamics

4.5.1 Mortality and Outward Migration

4.5.1.1 Results

4.5.1.1.1 Age of Dogs that Died Compared with the General Population
4.5.1.1.2 Age Dependent Survival Rates
4.5.1.1.3 Sex of Dogs that Died Compared with the General Population
4.5.1.1.4 Household Distribution of Dogs that Died Compared with the General Population
4.5.1.1.5 Reasons for Death
4.5.1.1.6 Outward Migration

4.5.1.2 Discussion

4.5.2 Inward Migration and Acquisition of Dogs

4.5.2.1 Results

4.5.2.1.1 Age of New Dogs
4.5.2.1.2 Sex of New Dogs
4.5.2.1.3 Household Distribution of New Dogs

4.5.2.2 Discussion

4.5.3 Reproduction

4.5.3.1 Results
4.5.3.2 Discussion

4.5.4 Rate of Change in Population Size

4.5.4.1 Results
4.5.4.2 Discussion

4.6 Conclusions

CHAPTER 5  PRELIMINARY SURVEY OF THE ENDO- AND ECTO-PARASITES AND GASTROINTESTINAL BACTERIA AFFECTING DOGS OF THE KIMBERLEY REGION

5.1 Introduction
5.2 Methodology
5.3 Results

5.3.1 Overview of Canine Parasites of the Kimberley Region
5.3.2 Scabies
5.3.3 Hookworm (Ancylostoma caninum)
5.3.4 Roundworm
5.3.5 Other Parasites and Bacteria

5.3.5.1 Echinococcus granulosus
5.3.5.2 Seroprevalence 182
5.3.5.2 *Giardia duodenalis* 182
5.3.5.3 Heartworm 183
5.3.5.4 *Strongyloides stercoralis* 183
5.3.5.5 *Spirocercus lupi* 184
5.3.5.6 Other Arthropod Parasites 184
5.3.5.7 Other Protozoan Parasites 185
5.3.5.8 Other Cestodes 185
5.3.5.9 Gastrointestinal Bacteria 185

5.4 Discussion 187
5.4.1 Scabies 187
5.4.2 Hookworm (*Ancylostoma caninum*) 187
5.4.3 Roundworm 189
5.4.4 Other Parasites and Bacteria 191

5.4.4.1 *Echinococcus granulosus* 191
5.4.4.1.1 Seroprevalence 191
5.4.4.2 *Giardia duodenalis* 194
5.4.4.3 *Dirofilaria immitis* 195
5.4.4.4 *Strongyloides stercoralis* 197
5.4.4.5 *Spirocercus lupi* 198
5.4.4.6 Other Arthropod Parasites 199
5.4.4.7 Other Protozoan Parasites 201
5.4.4.8 Other Cestode Parasites 203
5.4.4.9 *Campylobacter* spp. and *Salmonella* spp. 204

5.5 Conclusion 205

CHAPTER 6 PRELIMINARY TESTING OF THE IVERMECTIN TREATMENT PROTOCOL FOR CONTROL OF PARASITES 206

6.1 Introduction 206
6.2 Methodology 206

6.2.1 Faecal Samples 207

6.2.1.1 The Effectiveness of Formalin Ethyl Acetate Sedimentation Technique for Recovering Hookworm Eggs and Larvae from Faecal Samples 207
6.2.1.2 Determination of Intestinal Parasite Infection 207

6.2.2 Skin Samples 208
6.2.3 Blood Samples 208

6.2.3.1 Measurement of Albumin and Total Serum Protein 208
6.2.3.2 Haematology 209

6.3.4 Side Effects to Treatments with Ivermectin 210
6.3.5 Post Mortem Examinations 211

6.3 Results 211

6.3.1 Effectiveness of Formalin Ethyl Acetate Sedimentation Technique for Recovering Hookworm Eggs and Larvae from Faeces 211
6.3.2 Parasites of the Dogs of the Kununurra Communities 213
6.3.3 Efficacy of Ivermectin in the Treatment of Scabies and Hookworm 214

6.3.3.1 Weekly Scabies Prevalence after Treatment with 200μg/kg Ivermectin 214
6.3.3.2 Hookworm Prevalence after Treatment 215

6.3.4 The Risk Factors for Canine Infection with Scabies and Hookworm 217

6.3.4.1 Sex of Host 217
6.3.4.2 Age of Host 217

6.3.5 Side Effects of Ivermectin Treatment 217
6.3.6 The Effect of a Single Treatment with 200μg/kg Ivermectin on Dog Health Parameters 217

6.3.6.1 Hydration Status 217
6.3.6.2 Haemoglobin Levels 221
6.3.6.3 Mean Corpuscular Haemoglobin Concentration (MCHC) and Mean Corpuscular Volume (MCV) 225
6.3.6.4 Globulin 225
6.3.6.5 Albumin 228
6.3.6.6 The Effect of a Single Treatment with 200μg/kg Ivermectin on Other Parasites 228

6.4 Discussion 229

6.4.1 Effectiveness of Formalin Ethyl Acetate Sedimentation (FEAS) Technique for Recovering Hookworm Eggs from Faeces 229
6.4.2 Parasites of the Dogs of the Kununurra Communities 230
6.4.3 Efficacy of Ivermectin in Treatment of Scabies and Hookworm 232

6.4.3.1 Scabies Prevalence after a Single Treatment of Dogs with 200μg/kg Ivermectin 232
6.4.3.2 Hookworm Prevalence after a Single Treatment of Dogs with 200μg/kg Ivermectin 234

6.4.4 The Risk Factors for Canine Infection with Scabies and Hookworm 236

6.4.4.1 Sex and Age 236

6.4.5 Side Effects of Ivermectin Treatment 237
6.4.6 The Effect of a Single Treatment of Dogs with 200μg/kg Ivermectin on Health Parameters 238

6.4.6.1 Hydration Status 238
6.4.6.2 Haemoglobin Levels 238
6.4.6.3 Mean Corpuscular Haemoglobin Concentration and Mean Corpuscular Volumes 241
6.4.6.4 Globulin 242
6.4.6.5 Albumin 243
6.4.6.6 The Effect of a Single Treatment with 200μg/kg ivermectin on Other Parasites 243

6.5 Conclusion 244

CHAPTER 7 EFFECTIVENESS OF IVERMECTIN IN CONTROLLING PARASITES IN DOGS IN KIMBERLEY COMMUNITIES 245

7.1 Introduction 245
7.2 Methodology 246
7.3 Results 248

7.3.1 Scabies 248
<table>
<thead>
<tr>
<th>Section</th>
<th>Page No.</th>
</tr>
</thead>
<tbody>
<tr>
<td>7.3.1.1 Prevalence of Scabies During the Ivermectin Treatment Program</td>
<td>248</td>
</tr>
<tr>
<td>7.3.1.1.1 The Prevalence of Scabies in Treated and Non-treated Dogs</td>
<td>259</td>
</tr>
<tr>
<td>7.3.1.1.2 Scabies Infection Dynamics at Kalumburu and Looma</td>
<td>259</td>
</tr>
<tr>
<td>7.3.1.2 Risk Factors for Scabies Infection</td>
<td>265</td>
</tr>
<tr>
<td>7.3.1.2.1 Age of Host</td>
<td>266</td>
</tr>
<tr>
<td>7.3.1.2.2 Sex of Host</td>
<td>266</td>
</tr>
<tr>
<td>7.3.1.2.3 The Effect of Number of Dogs per Household on Scabies Infection Rates</td>
<td>266</td>
</tr>
<tr>
<td>7.3.1.2.3.1 Association between Numbers of Dogs per Household and Scabies Infection in Dogs</td>
<td>266</td>
</tr>
<tr>
<td>7.3.1.2.3.2 Pre-treatment Correlation between Household Dog Population and Prevalence of Scabies in Dogs</td>
<td>269</td>
</tr>
<tr>
<td>7.3.1.2.3.3 Pre-treatment Distribution of Dogs with Scabies throughout Kalumburu, Looma and Warmun</td>
<td>269</td>
</tr>
<tr>
<td>7.3.1.3 Effect of Scabies Control Program on Dog Health Parameters</td>
<td>274</td>
</tr>
<tr>
<td>7.3.2 Hookworm</td>
<td>274</td>
</tr>
<tr>
<td>7.3.2.1 Prevalence of Hookworm during the Ivermectin Treatment Program</td>
<td>274</td>
</tr>
<tr>
<td>7.3.2.1.1 Prevalence of Hookworm in all Dogs from Each Region</td>
<td>274</td>
</tr>
<tr>
<td>7.3.2.1.2 Prevalence of Hookworm in Dogs that were Treated Three Months before Sampling</td>
<td>279</td>
</tr>
<tr>
<td>7.3.2.1.3 Prevalence of Hookworm in Dogs that were Not Treated Three Months before Sampling</td>
<td>285</td>
</tr>
<tr>
<td>7.3.2.1.4 Comparison of Hookworm Infection Rates in Dogs that were Treated with those that were Not</td>
<td>287</td>
</tr>
<tr>
<td>7.3.2.1.5 Hookworm Infection Dynamics at Kalumburu and Looma</td>
<td>288</td>
</tr>
<tr>
<td>7.3.2.1.5.1 Kalumburu</td>
<td>288</td>
</tr>
<tr>
<td>7.3.2.1.5.2 Looma</td>
<td>289</td>
</tr>
<tr>
<td>7.3.2.2 Risk Factors for Hookworm Infection</td>
<td>289</td>
</tr>
<tr>
<td>7.3.2.2.1 Age of Host</td>
<td>290</td>
</tr>
<tr>
<td>7.3.2.2.1.1 Age Dependency in Predisposition to Hookworm Infection after Ivermectin Treatment</td>
<td>290</td>
</tr>
<tr>
<td>7.3.2.2.2 Sex of Host</td>
<td>290</td>
</tr>
<tr>
<td>7.3.2.2.2.1 Sex Dependency in Predisposition to Hookworm Infection after Ivermectin Treatment</td>
<td>290</td>
</tr>
<tr>
<td>7.3.2.2.3 The Effect of the Number of Dogs per Household on Infection Rates</td>
<td>294</td>
</tr>
<tr>
<td>7.3.2.3 Environmental Factors Affecting Hookworm Infection Rates</td>
<td>294</td>
</tr>
<tr>
<td>7.3.2.3.1 Correlation between Rainfall and Hookworm Infection Rates</td>
<td>294</td>
</tr>
<tr>
<td>7.3.2.3.2 Temperature</td>
<td>294</td>
</tr>
<tr>
<td>7.3.2.4 Effect of Parasite Control Program on Dog Health Parameters</td>
<td>298</td>
</tr>
<tr>
<td>7.3.2.4.1 Weight Changes of Dogs during the Program</td>
<td>298</td>
</tr>
<tr>
<td>7.3.2.4.2 Packed Cell Volume Measurements</td>
<td>298</td>
</tr>
</tbody>
</table>
7.3.2.4.3 Total Plasma Protein Measurements 303

7.3.3 Roundworm 303

7.3.3.1 The Effect of Ivermectin Treatments on *Toxocara canis* Prevalence 303
7.3.3.2 Risk Factors for *Toxocara canis* Infection 307

7.3.3.2.1 Age of Host 307
7.3.3.2.2 Sex of Host 307

7.3.4 Other Parasites and Bacteria 307

7.3.4.1 *Giardia duodenalis* 307

7.3.4.1.1 The Effect of Ivermectin Treatments on *Giardia duodenalis* Prevalence 307
7.3.4.1.2 Risk Factors for *Giardia duodenalis* Infection 307

7.3.4.1.2.1 Age of Host 307
7.3.4.1.2.2 Sex of Host 310
7.3.4.1.2.3 The Effect of the Number of Dogs per Household on *Giardia* Infection Rates 310

7.3.4.2 *Dirofilaria immitis* 310

7.3.4.2.1 The Effect of Ivermectin Treatments on *Dirofilaria immitis* Prevalence 310

7.3.4.3 *Spirocerca lupi* 314

7.3.4.3.1 The Effect of Ivermectin Treatments on *Spirocerca lupi* Prevalence 314

7.3.4.4 Gastrointestinal Bacteria 314

7.3.4.4.1 Risk Factors for Infection with *Campylobacter* and *Salmonella* 314

7.3.4.4.1.1 Age and Sex of Host 314

7.4 Discussion 316

7.4.1 Scabies 316

7.4.1.1 Prevalence of Scabies during the Ivermectin Treatment Program 316

7.4.1.1.1 Scabies Infection Dynamics 320

7.4.1.2 Risk Factors for Scabies Infection 321

7.4.1.2.1 Age and Sex of Host as Risk Factors for Scabies Infection 321
7.4.1.2.2 The Effect of Household Crowding of Dogs on Scabies Infection Rates 322

7.4.1.3 Effect of Scabies Control Program on Dog Health Parameters 324

7.4.1.3.1 Weight Changes 324

7.4.2 Hookworm 325

7.4.2.1 Prevalence of Hookworm during the Ivermectin Treatment Program 325

7.4.2.1.1 Prevalence of Hookworm in All Dogs from Each Region 325
7.4.2.1.2 Prevalence of Hookworm in Treated and Non-treated Dogs 329
7.4.2.1.3 Hookworm Infection Dynamics at Looma and Kalumburu

7.4.2.2 Risk Factors for Hookworm Infection

7.4.2.2.1 Sex of Host

7.4.2.2.1.1 Sex Dependency in Predisposition to Hookworm Infection after Ivermectin Treatment at Looma

7.4.2.2.2 Age of Host

7.4.2.2.2.1 Age Dependency in Predisposition to Hookworm Infection after Ivermectin Treatment at Looma

7.4.2.2.3 The Effect of Household Crowding on Infection Rates

7.4.2.3 Environmental Factors Affecting Hookworm Infection Rates

7.4.2.3.1 Rainfall

7.4.2.3.2 Temperature

7.4.2.3.2.1 Microenvironmental Factors for Hookworm Infection

7.4.2.4 Effect of Hookworm Control Program on Dog Health Parameters

7.4.2.4.1 Packed Cell Volume Measurements

7.4.3 Roundworm

7.4.3.1 Effect of Ivermectin Treatment on *Toxocara canis* Prevalence

7.4.3.2 Risk Factors for *Toxocara canis* Infection

7.4.3.2.1 Age and Sex of Host

7.4.4 Other Parasites and Bacteria

7.4.4.1 *Giardia duodenalis*

7.4.4.1.1 Risk Factors for *Giardia duodenalis* Infection

7.4.4.1.1.1 Age and Sex of Host

7.4.4.1.1.2 The Effect of Household Crowding of Dogs on Infection Rates

7.4.4.2 *Dirofilaria immitis*

7.4.4.3 *Spirocerca lupi*

7.4.4.4 Gastrointestinal parasites

7.4.4.4.1 Risk factors for Infection with *Campylobacter* and *Salmonella*

7.4.4.4.1.1 Age of Host

7.5 Conclusion

---

CHAPTER 8 THE EFFECT OF THE CANINE PARASITE PROGRAM ON CHILD HEALTH

8.1 Introduction

8.2 Methodology
8.3 Results
  8.3.1 Cutaneous Infections
    8.3.1.1 Prevalence Study
    8.3.1.2 Retrospective Data
  8.3.2 Diarrhoeal Disease
    8.3.2.1 Prevalence Study
    8.3.2.2 Retrospective Data
  8.3.3 Dog Bites in Children

8.4 Discussion
  8.4.1 Cutaneous Infections
  8.4.2 Diarrhoeal Disease
  8.4.3 Dog Bites in Children

8.5 Conclusion

CHAPTER 9 SUMMARY
  9.1 Canine Population Control
    9.1.1 Population Structure
      9.1.1.1 Age
      9.1.1.2 Sex
      9.1.1.3 Dog Breeds
    9.1.2 Distribution
      9.1.2.1 Household Distribution of Dogs
      9.1.2.2 Owner Distribution
    9.1.3 Dynamics
      9.1.3.1 Mortality and Outward Migration of Dogs
        9.1.3.1.1 The Population Structure and Distribution of Dogs that Died or went Missing
      9.1.3.2 New Dogs and Inward Migration
      9.1.3.2.1 Population Structure and Distribution of Incoming Dogs
    9.1.3.3 Effectiveness of the Breeding Control Program
      9.1.3.3.1 Contraceptive Effectiveness

9.2 Parasite Control
  9.2.1 Parasites of the Kimberley Dog Population
  9.2.2 Risk Factors for Infection
    9.2.2.1 Age
    9.2.2.2 Sex
9.2.2.3 Numbers of Dogs per Household 380
9.2.3 Effects of Parasites on Canine Health 381
9.2.4 Effect of Parasite Control on Parasites 381
9.2.5 Effect of Canine Parasite Control on Human Health 382

APPENDICES 385
REFERENCES 401
# LIST OF TABLES

<table>
<thead>
<tr>
<th>Table No.</th>
<th>Title</th>
<th>Page No.</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.1</td>
<td>Prevalence of Canine Hookworm throughout the World</td>
<td>31</td>
</tr>
<tr>
<td>2.2</td>
<td>Prevalence of <em>Toxocara canis</em> throughout the World</td>
<td>42</td>
</tr>
<tr>
<td>3.1</td>
<td>Community Profiles – 1991</td>
<td>96</td>
</tr>
<tr>
<td>4.1</td>
<td>Dog Population Size</td>
<td>137</td>
</tr>
<tr>
<td>4.2</td>
<td>Dog Population Structure</td>
<td>138</td>
</tr>
<tr>
<td>4.3</td>
<td>Dog Population Distribution</td>
<td>146</td>
</tr>
<tr>
<td>4.4</td>
<td>Number of Dogs Per Person – September 1994</td>
<td>150</td>
</tr>
<tr>
<td>4.5</td>
<td>Mortality and Outward Migration of Dogs</td>
<td>158</td>
</tr>
<tr>
<td>4.6</td>
<td>Inward Migration and Acquisition of Dogs</td>
<td>163</td>
</tr>
<tr>
<td>4.7</td>
<td>Dog Population Dynamics – Combined Kimberley Communities</td>
<td>167</td>
</tr>
<tr>
<td>5.1</td>
<td>Pre-Treatment Prevalence of Scabies in Dogs from the Kimberley Region</td>
<td>181</td>
</tr>
<tr>
<td>5.2</td>
<td>Details of Dogs with Positive Hydatid Serology</td>
<td>182</td>
</tr>
<tr>
<td>5.3</td>
<td>Prevalence of <em>Dirofilaria immitis</em> in Dogs from the Kimberley Region</td>
<td>183</td>
</tr>
<tr>
<td>5.4</td>
<td><em>Salmonella</em> spp. and <em>Campylobacter</em> spp. Isolation from Dogs from the Kimberley Region</td>
<td>186</td>
</tr>
<tr>
<td>6.1</td>
<td>Parasitology and Haematology Methods</td>
<td>210</td>
</tr>
<tr>
<td>6.2</td>
<td>Comparison of Results of ZnSO₄ Flotation and Formalin Ethyl Acetate Sedimentation Techniques for Intestinal Hookworms in 60 Dogs from Kununurra</td>
<td>212</td>
</tr>
<tr>
<td>6.3</td>
<td>Prevalence of Hookworm in Dogs Treated with 200μg/kg Ivermectin at Kununurra on 12 April (Week 0)</td>
<td>216</td>
</tr>
<tr>
<td>6.4</td>
<td>Risk Factors Associated with Scabies Infection in Dogs – Kununurra</td>
<td>218</td>
</tr>
<tr>
<td>6.5</td>
<td>Risk Factors Associated with Hookworm Infection in Dogs – Kununurra</td>
<td>219</td>
</tr>
<tr>
<td>6.6</td>
<td>Hydration Status of Dogs – Kununurra</td>
<td>220</td>
</tr>
<tr>
<td>6.7</td>
<td>Risk Factors Associated with Low Haemoglobin Levels in Dogs – Kununurra</td>
<td>222</td>
</tr>
<tr>
<td>6.8</td>
<td>Risk Factors Associated with Low Mean Corpuscular Haemoglobin Concentrations (MCHC) in Dogs – Kununurra</td>
<td>226</td>
</tr>
<tr>
<td>6.9</td>
<td>Risk Factors Associated with High Serum Globulin Levels in Dogs - Kununurra</td>
<td>227</td>
</tr>
<tr>
<td>Table No.</td>
<td>Description</td>
<td>Page No.</td>
</tr>
<tr>
<td>----------</td>
<td>------------------------------------------------------------------------------</td>
<td>----------</td>
</tr>
<tr>
<td>7.1</td>
<td>Prevalence of Scabies in Dogs – Coastal Region</td>
<td>249</td>
</tr>
<tr>
<td>7.2</td>
<td>Prevalence of Scabies in Dogs – Central Region</td>
<td>250</td>
</tr>
<tr>
<td>7.3</td>
<td>Prevalence of Scabies in Dogs – Eastern Region</td>
<td>251</td>
</tr>
<tr>
<td>7.4</td>
<td>Prevalence of Scabies in Treated and Non-Treated Dogs – Coastal Region</td>
<td>260</td>
</tr>
<tr>
<td>7.5</td>
<td>Prevalence of Scabies in Treated and Non-Treated Dogs – Central Region</td>
<td>261</td>
</tr>
<tr>
<td>7.6</td>
<td>Prevalence of Scabies in Treated and Non-Treated Dogs – Eastern Region</td>
<td>262</td>
</tr>
<tr>
<td>7.7</td>
<td>Prevalence of Scabies in New and Previously Seen Dogs – Central Region</td>
<td>263</td>
</tr>
<tr>
<td>7.8</td>
<td>Prevalence of Scabies in New and Previously Seen Dogs – Eastern Region</td>
<td>264</td>
</tr>
<tr>
<td>7.9</td>
<td>Sex Related Prevalence of Scabies in Dogs</td>
<td>267</td>
</tr>
<tr>
<td>7.10</td>
<td>Scabies Infection and Household Density of Dogs – Coastal, Central and Eastern Regions</td>
<td>268</td>
</tr>
<tr>
<td>7.11</td>
<td>Percentage of Households and Dogs Affected by Scabies by Household Size – Mainstream Communities Pretreatment</td>
<td>270</td>
</tr>
<tr>
<td>7.12</td>
<td>Scabies Infection and Weight Changes</td>
<td>275</td>
</tr>
<tr>
<td>7.13</td>
<td>Hookworm Prevalence in Dogs</td>
<td>276</td>
</tr>
<tr>
<td>7.14</td>
<td>Prevalence of Hookworm in Treated and Non-Treated Dogs – Coastal Region</td>
<td>281</td>
</tr>
<tr>
<td>7.15</td>
<td>Prevalence of Hookworm in Treated and Non-Treated Dogs – Central Region</td>
<td>283</td>
</tr>
<tr>
<td>7.16</td>
<td>Prevalence of Hookworm in Treated and Non-Treated Dogs – Eastern Region</td>
<td>286</td>
</tr>
<tr>
<td>7.17</td>
<td>Age Related Hookworm Infection and Reinfection Rates in Dogs Three Months after Treatment – Looma</td>
<td>291</td>
</tr>
<tr>
<td>7.18</td>
<td>Sex- Prevalence of Hookworm (Pooled Data, All Regions)</td>
<td>292</td>
</tr>
<tr>
<td>7.19</td>
<td>Sex Related Hookworm Infection and Reinfection Rates in Dogs Three Months after Treatment – Looma</td>
<td>293</td>
</tr>
<tr>
<td>7.20</td>
<td>Hookworm and Numbers of Dogs per Household for Coastal, Central and Eastern Regions</td>
<td>295</td>
</tr>
<tr>
<td>Table No.</td>
<td>Description</td>
<td>Page No.</td>
</tr>
<tr>
<td>----------</td>
<td>------------------------------------------------------------------------------------------------------------------------</td>
<td>----------</td>
</tr>
<tr>
<td>7.21</td>
<td>Correlation Between Rainfall and Hookworm Prevalence</td>
<td>296</td>
</tr>
<tr>
<td>7.22</td>
<td>Correlation Between Rainfall and Hookworm Prevalence of Dogs Treated and Not Treated Previously at Looma and the Coastal Region</td>
<td>297</td>
</tr>
<tr>
<td>7.23</td>
<td>Hookworm Infection and Weight Changes</td>
<td>299</td>
</tr>
<tr>
<td>7.24</td>
<td>Packed Cell Volumes and Age – All Regions</td>
<td>300</td>
</tr>
<tr>
<td>7.25</td>
<td>Packed Cell Volumes and Hookworm Infection – Puppies and Juveniles, All Regions</td>
<td>301</td>
</tr>
<tr>
<td>7.26</td>
<td>Packed Cell Volumes and Hookworm Infection – Adults, All Regions</td>
<td>302</td>
</tr>
<tr>
<td>7.27</td>
<td>Age-Prevalence of <em>Toxocara canis</em> in Dogs – Coastal and Central Regions</td>
<td>304</td>
</tr>
<tr>
<td>7.28</td>
<td>Prevalence of <em>Toxocara canis</em> in Dogs – Kalumburu and Looma</td>
<td>305</td>
</tr>
<tr>
<td>7.29</td>
<td>Risk Factors for Infection with <em>Toxocara canis</em> in Dogs – Kalumburu</td>
<td>306</td>
</tr>
<tr>
<td>7.30</td>
<td>Prevalence of <em>Giardia</em> in Dogs – Coastal, Central and Eastern Regions</td>
<td>308</td>
</tr>
<tr>
<td>7.31</td>
<td>Prevalence of <em>Giardia</em> in Dogs – Kalumburu and Looma</td>
<td>309</td>
</tr>
<tr>
<td>7.32</td>
<td>Sex-Related Prevalence of <em>Giardia</em> in Dogs</td>
<td>311</td>
</tr>
<tr>
<td>7.33</td>
<td><em>Giardia</em> and Numbers of Dogs per Household - Coastal, Central and Eastern Regions</td>
<td>312</td>
</tr>
<tr>
<td>7.34</td>
<td><em>Dirofilaria immitis</em> Post-Treatment Prevalence</td>
<td>313</td>
</tr>
<tr>
<td>7.35</td>
<td>Prevalence of <em>Spirocerca lupi</em> in Dogs</td>
<td>315</td>
</tr>
<tr>
<td>8.1</td>
<td>June 1992 and June 1993 Survey of 5-15 Years Old Children for Skin Afflictions</td>
<td>356</td>
</tr>
<tr>
<td>8.2</td>
<td>Yearly Incidence of Scabies in 0-15 Years Old Children</td>
<td>357</td>
</tr>
<tr>
<td>8.3</td>
<td>Yearly Incidence of Skin Infections in 0-15 Years Old Children</td>
<td>359</td>
</tr>
<tr>
<td>8.4</td>
<td>Prevalence of Gastrointestinal Pathogens in 0-5 Years Old Children (n=67)</td>
<td>360</td>
</tr>
<tr>
<td>8.5</td>
<td>Yearly Incidence of Diarrhoeal Disease in 0-15 Years Old Children</td>
<td>361</td>
</tr>
<tr>
<td>8.6</td>
<td>Five Yearly Incidence of Confirmed Giardiasis in 0-15 Years Old Children</td>
<td>363</td>
</tr>
<tr>
<td>8.7</td>
<td>Five Yearly Incidence of Laboratory Confirmed Gastrointestinal Diseases in 0-15 Years Old Children</td>
<td>363</td>
</tr>
<tr>
<td>8.8</td>
<td>Five Yearly Incidence of Dog Bites in 0-15 Years Old Children</td>
<td>364</td>
</tr>
</tbody>
</table>
# LIST OF FIGURES

<table>
<thead>
<tr>
<th>Figure No.</th>
<th>Description</th>
<th>Page No.</th>
</tr>
</thead>
<tbody>
<tr>
<td>3.1</td>
<td>Study Communities and Towns in the Kimberley Region of Western Australia</td>
<td>91</td>
</tr>
<tr>
<td>3.2</td>
<td>Contrasting Facilities at Communities of the Study</td>
<td>101</td>
</tr>
<tr>
<td>3.3</td>
<td>Transponder Microchip Reader and Microchip</td>
<td>104</td>
</tr>
<tr>
<td>3.4</td>
<td>Dogs Wearing Coloured Collars</td>
<td>105</td>
</tr>
<tr>
<td>3.5</td>
<td>Owners Assisted by Gathering their Dogs</td>
<td>106</td>
</tr>
<tr>
<td>3.6</td>
<td>Examination of Dogs Generated Interest in the Community</td>
<td>107</td>
</tr>
<tr>
<td>3.7</td>
<td>Treatment of Dog with 200μg/kg Ivermectin by Subcutaneous Injection</td>
<td>111</td>
</tr>
<tr>
<td>3.8</td>
<td>Dog with Lesions Indicative of Scabies – Score 2</td>
<td>113</td>
</tr>
<tr>
<td>3.9</td>
<td>Dog with Lesions Indicative of Scabies – Score 3</td>
<td>114</td>
</tr>
<tr>
<td>3.10</td>
<td>Dog with Lesions Indicative of Scabies – Score 4</td>
<td>114</td>
</tr>
<tr>
<td>4.1</td>
<td>Percentage of Dogs Presented at One Visit that were Presented for Re-treatment at the Next Visit</td>
<td>136</td>
</tr>
<tr>
<td>4.2</td>
<td>The Seven Most Common Breeds and Crossbreeds Dogs in Kimberley Communities</td>
<td>141</td>
</tr>
<tr>
<td>4.3</td>
<td>Purebred Dog Breeds of Kimberley Communities</td>
<td>141</td>
</tr>
<tr>
<td>4.4</td>
<td>Average Number of Dogs per Dog-Owning-Household for Each Region of the Kimberley</td>
<td>147</td>
</tr>
<tr>
<td>4.5</td>
<td>The Percentage of Households in Each Region that Owned Dogs</td>
<td>148</td>
</tr>
<tr>
<td>4.6</td>
<td>Percentage of Dogs from Households with More than Four Dogs in Each Region of the Kimberley</td>
<td>148</td>
</tr>
<tr>
<td>4.7</td>
<td>Average Number of Dogs per Owner for Each Region of the Kimberley</td>
<td>149</td>
</tr>
<tr>
<td>4.8</td>
<td>The Percentage of Dogs Owned by Each Owner Age Category</td>
<td>151</td>
</tr>
<tr>
<td>4.9</td>
<td>Average Number of Dogs per Owner in Each Owner Age Category</td>
<td>152</td>
</tr>
<tr>
<td>4.10</td>
<td>Percentage of Registered Dogs that Died or Moved Away from Communities</td>
<td>157</td>
</tr>
<tr>
<td>4.11</td>
<td>Percentage of Dogs that were New or Visiting the Communities</td>
<td>164</td>
</tr>
<tr>
<td>4.12</td>
<td>Percentage of Mature Bitches that Conceived each Three Months</td>
<td>168</td>
</tr>
<tr>
<td>4.13</td>
<td>Percentage of Bitches at Each Visit that Received Contraceptive Treatment</td>
<td>169</td>
</tr>
<tr>
<td>Figure No.</td>
<td>Description</td>
<td>Page No.</td>
</tr>
<tr>
<td>-----------</td>
<td>------------------------------------------------------------------------------</td>
<td>----------</td>
</tr>
<tr>
<td>5.1</td>
<td>Pre-treatment Prevalence of Parasites in Dogs from the Kimberley Coastal Region</td>
<td>177</td>
</tr>
<tr>
<td>5.2</td>
<td>Pre-treatment Prevalence of Parasites in Dogs from the Kimberley Central Region</td>
<td>178</td>
</tr>
<tr>
<td>5.3</td>
<td>Pre-treatment Prevalence of Parasites in Dogs from the Kimberley Eastern Region</td>
<td>178</td>
</tr>
<tr>
<td>5.4</td>
<td>Pre-treatment Prevalence of Parasites in Dogs from the Kimberley Kalumburu</td>
<td>179</td>
</tr>
<tr>
<td>5.5</td>
<td>Pre-treatment Prevalence of Parasites in Dogs from the Kimberley Looma</td>
<td>179</td>
</tr>
<tr>
<td>6.1</td>
<td>Pre-treatment Prevalence of Parasites in Dogs from Kununurra</td>
<td>213</td>
</tr>
<tr>
<td>6.2</td>
<td>Scabies Infection Rates in Dogs from Kununurra before and after a Single Treatment with 200μg/kg Ivermectin</td>
<td>214</td>
</tr>
<tr>
<td>6.3</td>
<td>Prevalence of Hookworm (<em>Ancylostoma caninum</em>) in Dogs from Kununurra before and after a Single Treatment with 200μg/kg Ivermectin</td>
<td>215</td>
</tr>
<tr>
<td>6.4</td>
<td>Blood Parameters (Haemoglobin Concentration, Mean Corpuscular Haemoglobin Concentration, Mean Corpuscular Volume and Serum Globulin) of Paired Samples from Dogs before and after a Single Treatment with 200μg/kg Ivermectin</td>
<td>224</td>
</tr>
<tr>
<td>6.5</td>
<td>Prevalence of Ticks (<em>Rhipicephalus sanguineus</em>) in Dogs from Kununurra before and after a Single Treatment with 200μg/kg Ivermectin</td>
<td>229</td>
</tr>
<tr>
<td>7.1</td>
<td>Scabies Infection Rates in Dogs - Coastal Region</td>
<td>252</td>
</tr>
<tr>
<td>7.2</td>
<td>Scabies Infection Rates in Dogs - Central Region</td>
<td>252</td>
</tr>
<tr>
<td>7.3</td>
<td>Scabies Infection Rates in Dogs - Eastern Region</td>
<td>253</td>
</tr>
<tr>
<td>7.4</td>
<td>Scabies Infection Rates in Dogs – Kalumburu</td>
<td>253</td>
</tr>
<tr>
<td>7.5</td>
<td>Scabies Infection Rates in Dogs – Looma</td>
<td>254</td>
</tr>
<tr>
<td>7.6</td>
<td>Resolution of Score 2 Scabies after One Ivermectin Treatment (Three Months)</td>
<td>255</td>
</tr>
<tr>
<td>7.7</td>
<td>Resolution of Score 2 Scabies after Three Ivermectin Treatments (Nine Months)</td>
<td>256</td>
</tr>
<tr>
<td>7.8</td>
<td>Resolution of Score 3 Scabies after Two Ivermectin Treatments (Six Months)</td>
<td>257</td>
</tr>
<tr>
<td>7.9</td>
<td>Resolution of Score 4 Scabies after Three Ivermectin Treatments (Nine Months)</td>
<td>258</td>
</tr>
<tr>
<td>Figure No.</td>
<td>Description</td>
<td>Page No.</td>
</tr>
<tr>
<td>-----------</td>
<td>-----------------------------------------------------------------------------</td>
<td>----------</td>
</tr>
<tr>
<td>7.10</td>
<td>Percentage of Dogs with Improving, Worsening or No Change in Infection with Scabies – Kalumburu</td>
<td>265</td>
</tr>
<tr>
<td>7.11</td>
<td>Percentage of Dogs with Improving, Worsening or No Change in Infection with Scabies – Looma</td>
<td>265</td>
</tr>
<tr>
<td>7.12</td>
<td>Dot Map Household Distribution of Dogs with Scabies – Kalumburu</td>
<td>271</td>
</tr>
<tr>
<td>7.13</td>
<td>Dot Map Household Distribution of Dogs with Scabies - Looma</td>
<td>272</td>
</tr>
<tr>
<td>7.14</td>
<td>Dot Map Household Distribution of Dogs with Scabies - Warmun</td>
<td>273</td>
</tr>
<tr>
<td>7.15</td>
<td>Prevalence of Hookworm in All Dogs - Coastal Region</td>
<td>277</td>
</tr>
<tr>
<td>7.16</td>
<td>Prevalence of Hookworm in All Dogs - Central Region</td>
<td>277</td>
</tr>
<tr>
<td>7.17</td>
<td>Prevalence of Hookworm in All Dogs - Eastern Region</td>
<td>278</td>
</tr>
<tr>
<td>7.18</td>
<td>Prevalence of Hookworm in All Dogs – Kalumburu</td>
<td>279</td>
</tr>
<tr>
<td>7.19</td>
<td>Prevalence of Hookworm in All Dogs – Looma</td>
<td>279</td>
</tr>
<tr>
<td>7.20</td>
<td>Prevalence of Hookworm in Dogs Treated and Not Treated Three Months Prior to Sampling - Coastal Region</td>
<td>280</td>
</tr>
<tr>
<td>7.21</td>
<td>Prevalence of Hookworm in Dogs Treated and Not Treated Three Months Prior to Sampling – Kalumburu</td>
<td>282</td>
</tr>
<tr>
<td>7.22</td>
<td>Prevalence of Hookworm in Dogs Treated and Not Treated Three Months Prior to Sampling - Central Region</td>
<td>284</td>
</tr>
<tr>
<td>7.23</td>
<td>Prevalence of Hookworm in Dogs Treated and Not Treated Three Months Prior to Sampling – Looma</td>
<td>284</td>
</tr>
<tr>
<td>7.24</td>
<td>Prevalence of Hookworm in Dogs Treated and Not Treated Three Months Prior to Sampling - Eastern Region</td>
<td>285</td>
</tr>
<tr>
<td>7.25</td>
<td>Hookworm Infection Dynamics in Dogs – Kalumburu</td>
<td>288</td>
</tr>
<tr>
<td>7.26</td>
<td>Hookworm Infection Dynamics in Dogs – Looma</td>
<td>289</td>
</tr>
</tbody>
</table>
ACKNOWLEDGEMENTS

For his encouragement, support and interest I am gratefully indebted to my supervisor Peter Williamson. I am also deeply appreciative of the advise, assistance and support of Jon Dunsmore and Ian Robertson.

The excellent technical assistance of Aileen Elliot and Russ Hobbs in parasitology was essential to completion of the project and is appreciated. Gerry Goodwin is also to be thanked for her advice in production of this thesis.

I also wish to thank Chris O’Farrell and Ern Hulbert of Kimberley Health for allowing me to work with Kimberley Health and all the staff of the region. I am especially appreciative of the time provided by Michael Douglas, Alan Leckie, Anne Ward and all the Community Health staff of the region. To Howard and Shirley Saddler, I also wish to extend my appreciation for their advice and good company.

The work in the Kimberley would not have been contemplated without the support, commitment and enthusiasm of all the staff of Environmental Health, especially Bill Ellis, Iris Prouse and Helen Wright. The supervisors of Environmental Health Workers in the region, Buddy Morrison, Clayton Bell, Philip Wallaby and Flora Ah Choo, were the backbone of the project and the keepers of my sanity through the long hours spent together and deserve special mention. The Environmental Health Workers, especially Arthur Cherel, Johnny Jubadah and Freddie Timms, need to be commended for their excellent work and assistance during the program. I also owe a special debt to all the volunteers to the program, too numerous to mention, who were not only critical to the daily field work, but also a continued source of enthusiasm.
I wish to thank the chairpersons of the communities of the project for allowing me to conduct this program and I also wish to thank the people of the communities for their encouragement and agreement to participate. The work with people of the Kimberley made the project all worthwhile.

In the Pilbara, I wish to thank Faye Nelson and Julius Barker for their ongoing assistance in training and expert advice.

The Health Promotion Foundation of Western Australia and Kimberley Health provided funding for this project and I am very grateful to acknowledge their support and am thankful that they could foresee the benefit of this project.

Finally, to my family, colleagues and friends goes my deepest appreciation and gratitude for their untiring support. I am also grateful for having a Mother who is not only a never-failing support, but great telephone interviewer, dogcatcher and cook! Thanks also, to all my canine companions, especially “Julabah” and “Yorrick”, both of the Kimberley and home, without whom this would not have been half as much enjoyment.
Chapter 1

INTRODUCTION

1.1 History and Health in Aboriginal Communities of the Kimberley Region

Major differentials in terms of disease morbidity and mortality are reported between Aboriginal and non-Aboriginal people in Australia. Generally, the hospitalisation rates for Aboriginal people are much higher than for non-Aboriginal people (Waddell and Lee, 1991) and although many diseases are related to lifestyle and poor nutrition (e.g. heart disease and diabetes) infectious agents are responsible for considerable morbidity in Aboriginal communities.

The environmental health concerns in communities are considered integral in the causation of many of the lifestyle and infectious diseases suffered by Aboriginal people. These environmental factors, in turn, are related to the rapid change in lifestyle of Aboriginal people from nomadic to permanent settlements within the 200 years since European occupation of Australia.

In some areas of Australia, such as the Kimberley region of Western Australia, the transition has been even faster. The first mission in the Kimberley was established in 1872 followed in 1890 by a mission at Beagle Bay (Hunter, 1993). Through the aggressive attempts to suppress Aboriginal language, education and the widespread use of English at the missions, social change was inevitable. Within the Kimberley, Hunter (1993) identifies two different experiences for people from missions. The early coastal missions (Beagle Bay, Lombadina) contained a large mixed descent of Aboriginal people who were better educated and had greater contact with the wider society. In contrast, missions where there were predominantly “full descent, tradition-oriented” Aboriginal people (Kalumburu), control was maintained through isolation. These experiences have some consequences today, particularly in education and health provision.
Other Aboriginal people faced a transition from nomadism to working for the pastoral stations established for cattle production in the Kimberley region. Pastoralists were not welcomed warmly to the Kimberley, with many conflicts arising from the appropriation of land and resources by the settling Europeans (Bolton, 1987). A balance was struck when Aboriginal people were utilized for their labour as food was provided which relieved the need to poach the pastoralist’s animals (Basedow, 1932).

Today, Aboriginal communities have evolved from the early contacts with Europeans and the speed at which this has occurred has resulted in inadequacies in infrastructures and physical facilities provided for Aboriginal people trying to make this change (Gracey, 1992).

1.2 Dogs in Aboriginal Communities

Dogs are an integral part of the environment of Aboriginal people in remote communities. Canines have been companions, assistants and guardians of Aboriginal people since the introduction of the dingo approximately 4000 years ago (Gollan, 1984).

The pervasive presence of dogs in Aboriginal communities has led to concerns over the possibility for disease transmission from dogs to people. In an effort to address these concerns, ‘dog health programs’ have evolved to treat the most common parasitic zoonoses and control breeding. Unfortunately, the effects of these programs have mostly been anecdotal and sporadic. The largest attempt at a sustained program was in the Northern Territory from 1985-1990 (Palmer and Presson, 1990).

The major canine zoonoses known in the north of Australia include scabies and hookworm. Scabies (Sarcoptes scabiei) from canine sources is of concern for people living in close habitation with diseased dogs. Canine hookworm (Ancylostoma caninum) is known to cause cutaneous larval
migrants and another more recently discovered syndrome; eosinophilic enteritis. The degree to which these two parasites exist in people in communities is unknown, as is the distribution, prevalence and risk factors for canine infection. Likewise, the effectiveness of a parasite control program in containing these infections has not been fully investigated.

Laboratory investigations of the effect of ivermectin against hookworm and scabies in dogs have been successful in killing the parasites and abating clinical effects. Field based studies, though, have not been documented. Likewise, proligestone is a potent contraceptive with minimal side effects in bitches, but it has not been assessed in the overall control of populations where other factors, such as non-compliance for treatment, are in effect. The effect of a community-based dog health control campaign involving three monthly treatments with ivermectin and proligestone in the subtropical Kimberley Aboriginal communities forms the basis of this thesis.

1.3 The Research Problem

1.3.1 Aims and Scope of the Research

The aim here is to quantify the effects on canine and human health of a canine breeding and parasite control program in Aboriginal Communities and to make recommendations for future programs. As such, the thesis deals with two components of the program; population control and parasite control, including the effect on other types of infections.

1.3.1.1 Canine Population Control

The structure, distribution and dynamics of the populations are investigated:

*Population structure*

What are the demographic and structural characteristics of canine populations in Kimberley Aboriginal communities, including age, sex and dog breed?
Population Distribution

How is the population of dogs distributed within communities, on the basis of household distribution and owner distribution?
What are the characteristics of households and owners that own dogs?

Population Dynamics

What is the population structure and distribution of incoming dogs?
What is the population structure and distribution of dogs that die or move away from communities?
How effective are contraceptive treatments of bitches in controlling reproduction?
Does a breeding and parasite program alter the dynamics of the population?

1.3.1.2 Parasite Control

Major zoonotic infections, parasitological and bacterial, as well as those that only affect dog health are investigated:

Parasites and Zoonotic Gastrointestinal Bacteria of the Kimberley Dog Population

What are the most prevalent canine parasites in Kimberley Aboriginal Communities?

Risk Factors for Infection with Parasites and Bacteria

What are the risk factors, including sex, age and numbers of dogs per household, for infection?

Effects of Parasites on Canine Health

How do these parasites affect canine health?

Effect of Parasite Control on Parasite Prevalence

Does the parasite control program reduce the prevalence of these infections?
Effect of Canine Parasite Control Program on Human Health

What, if any, effect does a canine parasite control program have on human health?
Chapter 2
A REVIEW OF THE LITERATURE

2.1 The Association between Aboriginal People and their Dogs

Dingoes are believed to have been introduced into Australia by Asian seafarers who had strong trade and sea links with Australia (Flood, 1995; Corbett, 1995). Archaeological evidence points to the entry of the dingo in the north of Australia approximately 4000 years ago with about 500 years of lag time before the dingo colonised the southern regions of the mainland (Gollan, 1984). The dingo never reached Tasmania due to the severance of Tasmania some 8000 years ago by the rising seas of the Bass Strait (Gollan, 1984; Berndt and Berndt, 1992).

Aboriginal people are believed to have quickly adopted the dingo into their lives, although domestication of the dingo was only partial (Meggitt, 1965) with very little evidence of selective breeding. Some archaeological evidence of a physical variation from the classical uniform dingo morphology comes from the remains of dingo burials in NSW and Victoria which date back about 1000 years (Gollan, 1984), suggesting that some selection did occur, but without any continuity. The interest shown by Aboriginal people in the dingo may have been due to the difference between dingoes and any other animal in Australia. Dingoes were avid hunters and carnivores and were the only large land animals to give birth in a manner similar to humans (Tonkinson, 1984). For these and many other reasons, dingoes were often accepted as ‘members of the family’ by being given subsection (skin) names that automatically positioned dingoes into society by giving them status such as parent, grandparent and child (Kolig, 1978; Tonkinson, 1984; Ross, 1987). Dingoes were often the only animals to have special rights to attend treasured rituals and in some cases were considered to be ‘qualified’ to attend as fully-fledged lawmen (a title generally given only to men expert in religious lore) (Kolig, 1973; Kolig, 1978).

Traditionally dingoes were considered useful to Aboriginal people as hunting aides, companions, protectors and spiritual ‘necessities’. Some of these uses are evident today.
Much controversy surrounds the usefulness of dingoes (and dogs today) as hunting aides, despite this being the most obvious reason for any domestication of such an efficient killer (Meggitt, 1965). Remarks by anthropologists and observers vary from dogs being a liability during hunting due to ‘undisciplined howling and barking’ (Basedow, 1925; Gould, 1969; Hamilton, 1972; Kolig, 1973) to the hunting dogs being the primary providers of all food (White, 1972). As dingoes were the only animals on the mainland that were trained to any degree (Giles, 1889; Meggitt, 1965; White, 1972; Hayden, 1975), it is generally accepted that dingoes did serve some function in providing food for people.

The role of dingoes as companions is still evident. Great affection for dingoes and dogs has been recorded such as the observation by Lumholtz (1889, p. 179) that “the master never strikes, but merely threatens it (the dog). He caresses it like a child, eats the fleas off it, and then kisses it on the snout”.

Similarities between children and puppies may have been a reason for this closeness and “unreasonable amount of petting and pampering” (Basedow, 1925). Dingoes were often taken from the bush as puppies to be reared as ‘substitute children’ for childless women (Hamilton, 1972). Occasionally the forelegs of the puppies were broken to prevent their return to the wild (Meggitt, 1965) and women have been observed to breastfeed puppies (Berndt and Berndt, 1942; Meggitt, 1965). The dingo (and dogs’) role as a nurturing tool and emotional outlet is considered to be very important (Hamilton, 1972).

One of the most important uses of dingoes (traditionally) and dogs today is as a protector. Dingoes and dogs are considered to be expert at warning against the physical as well as the spiritual. With a keen sense of smell, acute hearing and alertness, dingoes and dogs were very important in protecting small family groups from secret revenge expeditions (Hamilton, 1972; Hayden, 1975) or supernatural threats (Hamilton, 1972; Kolig, 1978).
One of the most complex and least understood uses of the dingo is the dingoes’ role in the creation and ‘dreaming’. Roles of the dingo vary from creator on a par to the water snake (Howe, 1993) to an asocial marauder (Hamilton, 1972; Kolig, 1978; Tonkinson, 1984; Berndt and Berndt, 1992) and lethal spirit-being (Kolig, 1978). Either way, dingoes feature heavily in the dreaming for some Aboriginal groups and severe retribution by spiritual dingoes is still used as a threat to misbehaving people (personal observation).

Domestic dogs quickly replaced dingoes after the colonization of Australia by Europeans. Dogs were occasionally given as placatory gifts to Aboriginal people by the early explorers (Meggitt, 1965) and because of their relative ease to train, better assimilation to people’s living arrangements (Kolig, 1978) and perceived social stature (Meggitt, 1965) were often accepted willingly. Acceptance of the European dog did result in some problems that are evident today. Firstly, European dogs breed twice a year, whereas dingoes only breed once a year (Corbett, 1995). Also, dingoes brought from the bush often eventually returned to it (Lumholtz, 1889; Meggitt, 1965; Kolig, 1978) and very little breeding (or selection) was done in the camps. Secondly, dingoes were self sufficient and ‘economical’. Whereas dogs expected to be fed and cared for by people (a trade-off for domestication), dingoes would often hunt for the people, accept leftover food and complement their diet by foraging (Meggitt, 1965; Hamilton, 1972; Hayden, 1975).

The rapid transition of Aboriginal people from nomadic lifestyles to ‘permanent’ residence after colonisation is often considered a factor for the poor environmental health standards experienced by Aboriginal communities today (Gracey 1992). Concomitant is the perceived overpopulation and poor health of dogs, much of which is also a result of this transition. Population regulation, traditionally controlled by the capture of puppies from the wild without breeding within camps, became difficult after the introduction of the European dog. As a result, the health of dogs suffered due to the overcrowding and difficulty in feeding large numbers of dogs.
2.1.1 Health Considerations of the Association between Aboriginal People and their Dogs

The previous sections have displayed the long-standing association of people and dogs in Aboriginal communities. This section will review the pertinent aspects of this relationship with respect to both human and dog health.

2.1.1.1 Care and Feeding of Dogs

Affection and emotional attachment to dogs are well documented, but the translation of this to material wellbeing does not necessarily follow. Chewings (1936) observed, "(the dogs were)... usually emaciated and fed more on affection than food."

Some writers observed the dogs were well fed (Lane, 1928; Lumholtz, 1889; Berndt and Berndt, 1942), or fed according to the amount of food available to the owners (Nind, 1831; Buley, 1905). By contrast, other authors describe the poor condition of the dogs (Angus, 1847; Meggitt, 1965). Hamilton (1972) observed that only when food was abundant would the adult dog receive even a token amount of food, but the puppies were always fed well. More recently, Ross (1987) commented that dogs in the Halls Creek area have to find their own food and often steal meat.

Dingoes, in contrast to dogs, hunt for themselves (Meggitt, 1965; Hamilton, 1972) and require less attention. The apparent lack of feeding of dogs by owners may be a remnant of the days when dingoes roamed the camps.

2.1.1.2 Aversion to Killing Dogs

In the Halls Creek area, Ross (1987) noted that Aboriginal Law protects dogs and under Aboriginal Law, dogs can be neglected, but can not be killed. An example of this was in April 1980, when the Redhill people of Halls Creek held a ceremony lasting several days to
compensate for the culling of dogs by the shire council to reduce the excessive numbers of dogs in the town (Ross, 1987).

Meggitt (1965) also noted that no matter how many dogs a man owns, "he flatly refuses to destroy or otherwise dispose of any that are crippled, deformed or diseased". Injured dogs were often left to starve, as they could not compete with stronger dogs for the limited food supply. Kolig (1978) and Hamilton (1972) also relate stories of people's reluctance to kill dogs even if they were sick, aged or a nuisance to the camp. Berndt and Berndt (1992) go further to state that killing somebody else's dog is considered a grave offence for which retribution is sought. No other violation against people's property results in such violence.

Kolig (1978) considered that such passion relating to the killing of dogs must hark back to the dingo's place in the Dreaming. Regardless of the reasons, the strong feelings against killing (or euthanasia) of dogs means that the 'traditional methods' of population control (such as dog culling as described by Ross, 1987) are not acceptable. George Wallaby of Emu Creek, Kimberley Region, when asked about the practice of 'dogcatchers' killing dogs for bounties replied:

"Them doggers, whitefella blokes, they come here and shoot 'm in the old days, them dogs... They say gimme money or I kill the dog. I say I got no money. So they take 'm..., so I go hide 'm out bush. That's how we save 'm. Make them doggers bloody mad! Like they wanna shoot us, too!" (Arden, 1995; p. 81).

The replacement of sick dogs with healthy puppies is also unacceptable as a strategy to control disease.

2.1.1.3 Disease

Morris (1889) considered that "manginess and distemper" were principle causes of morbidity and mortality amongst the companion dingoes of the time. Matthew (1889, cited in Hamilton,
1972) also noted that Aboriginal people were frequently infected with purulent mange that was presumed to come from the dogs. Whether this was true cannot be validated, but even recent literature indicates that many dogs suffer from mange (Ross, 1987; Palmer and Presson, 1990).

Other observers point to the contribution of dogs to the overall camp hygiene. Hamilton (1972) considered the downside of keeping dogs included the attraction of flies to the campsites as well as the urination and defecation around campsites. She also referred to many of the dogs having open sores and possibly having parasites. Dogs were considered to play a positive role by their ability to dispose of any food scraps and eating faeces in the camp, usually those of babies and children (Hamilton, 1972).

The fact that many dogs sleep with their owners (Berndt and Berndt, 1942; Ross, 1987) and have mange is of particular importance when considering human health. As will be discussed, the potential for human clinical disease with canine scabies is high, particularly when there is close contact. Ross (1987) stated that “few people acknowledge any health hazard resulting from dogs, though beds are shared with dogs and the ground around some camps is strewn with faeces”.

2.1.1.4 Mythology and Education
The dingo, as mentioned, served as a medium for transmitting messages about moral truths and human behaviours in mythical tales. It may be that the modern dog too could be used as an important conveyor of health messages relating to humans by being the focus for information on disease transmission and hygiene. The dog, as a tool, subtly conveys health promotion messages without encroaching on human sensitivities.
2.2 Health Status in Aboriginal Communities

The considerable difference in health status of Aboriginal and non-Aboriginal groups in Australia has been attributed to the changes in social structure and environment impinged on Aboriginal people since European contact (Gracey 1992). Low population density and a nomadic lifestyle would have protected the people from infection and re-infection by enteric microorganisms (Gracey, 1992) and no doubt other infectious diseases during the precontact era. Indeed, Basedow (1932) in his account of the health of Aboriginal people, stated that Captain Cook, the ‘forefather’ of European settlement, had “not observed any sign of disease amongst the Aboriginal people he encountered”. Whether precontact life was as healthy as implied is difficult to determine.

2.2.1 Life Expectancy of Aboriginal People

In present times, mortality rates are higher for Aboriginal people than other Australians and the Aboriginal life expectancy remains up to 20 years less than the Australian average (Currie, 1993). Circulatory diseases, chronic respiratory conditions and trauma are the commonest causes of death, but differential mortality rates for infectious diseases between Aboriginal people and non-Aboriginal people still remain the highest (Currie, 1993).

2.2.2 Hospital Records

Determining the health status of the Aboriginal population largely relies on the hospital record system and comparisons between the Aboriginal and non-Aboriginal populations. Generally, the hospitalisation rates for Aboriginal people are much higher than for non-Aboriginal people (Waddell and Lee, 1991). When considering children under 5 years of age, the relative risk ratio for; skin infections is 18:1, infectious and parasitic diseases, 8:1, respiratory diseases, 6:1, digestive diseases, 5-8:1 and injuries and poisonings about 3:1 (Waddell and Lee, 1991). The differences are quite obvious and are supported by hospitalisation rates.
An investigation of hospital admissions from 1981 to 1986 revealed Aboriginal infants to be admitted 10 times more frequently for respiratory tract infections compared to other infants (Gracey and Anderson, 1989). As age increases, hospitalization rates decrease from a ratio of 5:1 to 2:1.

Gastroenteritis, another common complaint, was also more common and severe in Aboriginal people with Aboriginal: non-Aboriginal rates of admission for gastroenteritis being about 16-20:1 for infants and 6:1 for older children (Gracey and Anderson, 1989). In addition, Aboriginal people spend more than twice as long in hospital as other patients for gastroenteritis (Gracey, 1992) because of more serious disease and because of distance, isolation in severe weather, problems with follow-up in remote areas and environmental circumstances where reinfection is very likely (Gracey and Anderson, 1989). Overall, infectious diseases, particularly gastroenteritis and lower respiratory tract infections, are recognised as major causes of poor health in Aboriginal people (McNeilly, Cicchini, Oliver and Gracey, 1983). Skin infections are also acknowledged as causes of many clinic visits and admissions to hospitals (McNeilly et al, 1983). Skin infections and gastroenteritis and will be discussed in more detail.

2.2.3 Skin Diseases

Skin infections are considered to be particularly important in Aboriginal communities because of their high prevalence, chronic nature and extent of skin involvement that predisposes patients to rheumatic heart disease and glomerulonephritis (Waddell and Lee, 1991; Spiprakash, Gardiner, Hartas, Haase, Goodfellow, Currie, Kemp and Mathews, 1995). Scabies, for example, is endemic in many remote Aboriginal communities in Australia, where prevalences in children may be over 50% (Currie, Maguire and Wood, 1995). A health profile of an Aboriginal community in the Northern Territory found obvious scabies infestations in 17% of adults with more than a quarter of all people having skin sores (Hoy, Norman, Hayhurst and Pugsley, 1997). Data relating to other skin infections in Aboriginal communities are limited as the problems are often considered to be so common that cases are rarely recorded.
2.2.3.1 Pyoderma and Scabies

Children are particularly at risk of pyoderma, and nephritis may complicate pyoderma due to nephritogenic strains of streptococci (Burgess, 1994; Mahe, Prual, Konate and Bobin, 1995).

In a representative sample of 615 children in Mali, pyoderma was observed in 224 children (12.3%±1.6%), 52 of which were complications of scabies (Mahe et al, 1995). The prevalence of pyoderma has been reported to be higher in moist tropical climates with a possible seasonal influence (Burgess, 1994; Mahe et al, 1995).

In the Mali study, 78 patients (4.3% ±1.5%) had scabies (Mahe et al, 1995). In a study in Tanzania, 5.0% (n=1114) of the population of two rural villages had scabies (Gibbs, 1996). In 20% of the cases, secondary bacterial infections were noted. Another report from rural Tanzania found 6.0% (n=936) of one village to have scabies, but in this study, all scabies cases were complicated with secondary bacterial infections (Henderson, 1996). The skin sores associated with scabies are often multiple or confluent (Hoy et al, 1997).

Although transmissible skin diseases are often said to be associated with poor hygiene, overcrowding and poverty, the studies of Mali and Tanzania did not find any relationship between scabies infection and indicators of personal hygiene (Mahe et al, 1995; Gibbs, 1996). The high prevalence and endemicity of scabies may be due to several socio-economic factors such as; a high concentration of people per household; sleeping habits (sharing the same bed) and the absence of effective treatment of cases (Mahe et al, 1995). Mahe et al (1995) and Gibbs (1996) considered that individual treatment of scabies is ineffective in controlling the problem. Both scabies and severe pyoderma were found to be the most discomforting of all dermatoses (Mahe et al, 1995).
2.2.3.2 Complications of Pyoderma

High rates of scabies and skin sores with repeated streptococcal infection are linked to chronic renal disease (Hoy et al, 1997). Sequelae of *Streptococcus pyogenes* infections, such as acute glomerulonephritis and acute rheumatic fever, are common in some Aboriginal communities (Currie, 1993). In fact, rheumatic fever has been commonly diagnosed among the central and southern Aboriginal groups of Australia since 1860 (Basedow, 1932). In the Northern Territory, crude prevalences of rheumatic heart disease are as high as 30 per 1000 (Currie, 1993) and rates of rheumatic heart disease and renal failure are much higher in the Aboriginal population than the rest of the population (Waddell and Lee, 1991). Those cases that result in renal failure may require long-term dialysis or renal transplantation (Waddell and Lee, 1991).

2.2.4 Gastrointestinal Infections

Moodie in 1977 found that up to 25% of Aboriginal children had severe growth retardation for which diarrhoea contributed remarkably. In addition, other diseases including pneumonia, septicaemia and other serious infections often complicate diarrhoeal disease in Aboriginal children (Gracey, 1992).

2.2.4.1 Causes of Gastroenteritis

Numerous studies have been undertaken to determine the incidence, severity and cause of gastroenteritis and diarrhoea in Aboriginal children (Berry and Gracey, 1981b; Gracey, Sullivan, Burke, Wymer, Mogyorosy, Gunzburg and Iveson, 1992; Gunzburg, Gracey, Burke and Chang, 1992).

Five hundred and fifty five faecal samples were collected in a prospective study of 104 children up to 5 years of age from the Kimberley (Gunzburg et al, 1992). More than 36% of specimens from children 0-6 months old were diarrhoeic. Although *Salmonella* and *Campylobacter* species are frequently associated with diarrhoea, both were isolated from both diarrhoeic and non diarrhoeic samples. *Salmonella* was isolated from 12.7% (22) and 7.3% (28) of diarrhoeal
and non-diarrhoeal samples respectively and campylobacters from 4.6% (8) and 3.7% (14) of diarrhoeal and non-diarrhoeal samples. Neither *Hymenolepis nana* nor *Giardia duodenalis* were associated with diarrhoea although *Giardia* is highly endemic in children in Aboriginal communities with more than half infested at any one time.

In another study by Gracey *et al* (1992) involving a prospective monthly assessment of growth, nutrition and health of 49 infants to two years of age, the diarrhoea rates overall (percentage of children having one or more episodes of diarrhoea) were 6% at 0-6 months, 23% at 6-12 months, 46% at 12-18 months and 46% at 18-24 months. Isolations of *Shigella*, *Salmonella*, *Cryptosporidium* and enterotoxic *Escherishia coli* (ETEC) were strongly associated with diarrhoea, but *G. duodenale* and *H. nana* were not. *Campylobacter* was also isolated in the childrens’ first 12 months and was associated with diarrhoea episodes. Symptomless carriage of *C. jejuni*, though, is not uncommon in children living in communities where standards of living and hygiene are poor (Berry and Gracey, 1981a).

*Cryptosporidium* is also associated with poor hygiene and close person-to-person or animal-person contact and has been isolated from 10% of Aboriginal children admitted to Alice Springs hospital for diarrhoea (Gracey, 1992).

Generally there appears to be many mixed reports of the significance of the variety of pathogens usually associated with diarrhoea and ill thrift. The major pathogens isolated from children in Aboriginal communities include *Salmonella*, *Campylobacter*, *Shigella*, ETEC, *Giardia* and *Cryptosporidium*.

2.2.5 Reasons for Infectious Diseases in Aboriginal Communities

2.2.5.1 Environment

Environmental factors are considered responsible for much of the excess morbidity within Aboriginal communities (Waddell and Lee, 1991). These environmental factors include
overcrowding, unhygienic living conditions, contaminated food, inadequate rubbish disposal and “mangy dogs” (Waddell and Lee, 1991). As a consequence of these factors there is a heavy faecal contamination of the environment in which Aboriginal children live as evidenced by heavy bacterial contamination of the upper intestinal secretions found in some studies (Gracey, 1992). Many of the gastrointestinal infections, which are largely transmitted faecally- orally, are encouraged by living conditions associated with human or animal contamination (Gracey, 1992).

Some of the factors contributing to the generally poor living conditions in communities relate to the “rapidity of change from a traditional, nomadic lifestyle to living in permanent settlements and dwellings” (Waddell and Lee, 1991). Inadequacies in infrastructures and physical facilities provided for Aboriginal people trying to make this change have also been seen as factors in this problem (Waddell and Lee, 1991; Gracey, 1992).

In 1983, McNeilly et al considered environmental contamination of communities to still be a major factor causing ill health within communities despite a decline in hospital admissions for infections from 1971 to 1980.

2.2.6 The Role of Dogs in Causing Disease in Aboriginal Communities

Gracey (1992) considered that the close association of Aboriginal people and their native dogs (dingoes) and the use of hunted animal foods would have exposed them to gastrointestinal infections with such agents as *Giardia* and *Salmonella* prior to European settlement. Nowadays, dogs and other animals are still considered as potential reservoirs for some pathogens including *Salmonella* and *Campylobacter* (Gracey, 1992). In Guinea-Bissau and northern Thailand, the keeping of domestic animals and cattle has been associated with increased risk of childhood diarrhoea from cryptosporidiosis and other intestinal parasitoses (Gracey, 1992).
Likewise, Schantz (1991) considered “diseases of animal origin (to) remain of greatest importance in rural areas of developing countries... In such regions people commonly share their home environment with a variety of animal species under conditions of poor sanitation and hygiene that inevitably lead to frequent exposure to each other’s pathogens.” The situation in Aboriginal communities with poor environmental standards and close animal contact is likely to be similar.

The Aboriginal population of Australia experiences a much higher rate of morbidity and mortality than other Australians. Much of this excess morbidity has been attributed to poor standards of living and poor environmental health. Within this sphere, the potential for zoonoses from dogs is great, especially considering that many of the diseases causing morbidity may be zoonoses.

2.3 The Canine Zoonoses

The human health concerns about co-habitation with canine companions stem from the potential for disease transmission from dogs to people as well as the dogs’ ability to act as a reservoir for human pathogens. Despite the hundreds of known canine zoonoses, many do not occur in Australia (such as rabies). Of the zoonoses present in Australia, and in Aboriginal communities, many are parasites. The canine diseases that have sparked some attention for Aboriginal health include *Sarcoptes scabiei*, *Ancylostoma caninum* (dog hookworm), *Toxocara canis*, *Echinococcus granulosus*, *Salmonella* spp. and *Campylobacter jejuni*. The following section will review the features of these pathogens, particularly with respect to their zoonotic potential.

2.3.1 *Sarcoptes scabiei*

*Sarcoptes scabiei* is a skin burrowing mite capable of causing intense pruritus and resultant skin damage in many host mammals. The species is subdivided into variants, which are mostly host specific, although if close contact is maintained between host species, some cross transmission
of mites can occur. *Sarcoptes scabiei* is an important dermatological problem in Aboriginal communities for both people and dogs.

### 2.3.1.1 Lifecycle of *Sarcoptes scabiei*

*Sarcoptes scabiei* mites undergo a typical acarine development involving 4 stages; egg, larvae, nymph and (decious) adult. Most of the mite’s time is spent on or in the host’s skin although, as will be discussed, all stages are capable of living free in the environment for a short time.

The lifecycle of *Sarcoptes* may be complete in only 3 weeks (Scott and Horn, 1987; Scott, Miller and Griffin, 1995) although the time for development varies for males and females and is dependent on skin temperature and humidity (Arlian and Vyszenski-Moher, 1988). Adult mites are microscopic with females being 330 to 660μm by 250 to 200μm (Scott and Horn, 1987). Adult mites live within the stratum granulosum feeding on the cytoplasm of living skin cells that are lysed by secretions from the mites as they sink into the epidermal tissue (Arlian, Runyan, Archer and Estes, 1984a; Scott and Horn, 1987). Studies have found that it takes both *S. scabiei* var. *canis* and var. *hominis* less than 30 minutes to penetrate the stratum corneum (Arlian *et al*, 1984a).

Once submersion is achieved, the mites propel themselves forward with their appendages as the host tissue around the anterior body is dissolved (Scott and Horn, 1987). Females burrow through the stratum corneum at a rate of 0.5 to 5mm per day.

Copulation occurs in a ‘moulting pocket’ on the skin surface (Scott *et al*, 1995). The females then deposit about 50 eggs in the tunnels (Scott and Horn, 1987). Larvae hatch from eggs deposited in the tunnels of the epidermis to burrow to the surface where they wander about and feed (Scott and Horn, 1987). Larvae eventually rest in ‘moulting pockets’ before emerging as protonymphs (Arlian and Vyszenski-Moher, 1988). The protonymphs then moult to tritonymphs, which develop into either males or females (Arlian and Vyszenski-Moher, 1988).
Nymphs spend time resting in moulting pockets in the skin surface before they develop and moult into adults (Scott and Horn, 1987).

All life stages frequently surface from the burrow and wander on the skin surface. In one series of experiments, 26% of the monitored mites burrowed, 9% died in the burrows and 65% wandered from the burrow and were lost (Arlian and Vyszenski-Moher, 1988).

2.3.1.2 Clinical Features of Canine Infection

After an acute onset of intense pruritus, infected dogs develop an erythematous, nonfollicular papular dermatitis (Scott et al., 1995). Patchy alopecia results from disruption of the hair shafts by the host’s scratching, thus providing a more favourable habitat for mite growth (Shaw and Kunkle, 1990). The lesions become excoriated and thick yellow crusts and pyoderma develop (Thomsett, 1968; Scott and Horn, 1987; Sosna and Medleau, 1992a). Approximately 27% of canine scabies patients exhibit peripheral lymphadenopathy (Shaw and Kunkle, 1990).

Mites prefer thin skin with little hair (Thomsett, 1968; Shaw and Kunkle, 1990), so the lesions are usually on the pinnae, face, limb and ventrolateral trunk (Scott and Horn, 1987; Sosna and Medleau, 1992a). The disease spreads rapidly, but the dorsum is usually spared. Sometimes no lesions are present on the ear margins (Sosna and Medleau, 1992a; Scott et al., 1995) and some animals are asymptomatic carriers (Sosna and Medleau, 1992a).

Chronically and severely affected dogs may become anorectic, depressed and develop weight loss and secondary bacterial pyoderma (Scott and Horn, 1987). Malassezia pachydermatitis has been found to be a consistent associate with sarcoptic mange in red foxes, but its role as a secondary pathogen in mange infections is questionable (Pence, Windberg, Pence and Sprowls, 1983). Sarcoptic mange is also considered a major cause of mortality amongst natural red wolf (Philips and Scheck, 1991) and coyote populations (Pence et al., 1983).
Scabies 'incognito' occurs when dogs have minimal skin lesions (owing to meticulous grooming and bathing), or when dogs have unusual distributions (owing to glucocorticoid therapy) (Folz, 1984; Scott and Horn, 1987; Scott et al, 1995).

The differential diagnoses for scabies in dogs is extensive and includes; atopy, flea allergy dermatitis, food allergy, contact dermatitis, *Malassezia* dermatitis, otodectic dermatitis, infestation with other external parasites including *Demodex* (Smith and Claypoole, 1967) and *Cheyletiella*, dermatophytosis, generalized pyoderma and seborrheic dermatitis (Sosna and Medleau, 1992a).

### 2.3.1.3 Pathogenesis of Scabies Mite Infestation in Dogs

The mites cause intense pruritus by; secreting allergenic substances (Arlian, Ahmed, Vyszenski-Moher, Estes and Achar, 1988) that evoke a hypersensitivity reaction, secreting irritating byproducts, probably faecal matter, and causing mechanical irritation (Sosna and Medleau, 1992b).

The incubation period of scabies is unknown (Scott et al, 1995), but cutaneous reactions have been reported to occur within 24 hours to several weeks after infestation (Scott and Horn, 1987). Initially when mite numbers are low, the pruritus is proportional to the number of mites (Scott et al, 1995). As the number of mites increases (usually 21 to 30 days after exposure), there is a point when the pruritus explodes in severity (Scott et al, 1995). This may signify the development of hypersensitivity. The hypersensitivity reaction may persist long after the mites have been destroyed (Scott and Horn, 1987; Sosna and Medleau, 1992b; Scott et al, 1995) and often the dermatological changes are completely out of proportion to the number of mites present (Scott and Horn, 1987; Scott et al, 1995). Hypersensitivity is thought to protect the host from ectoparasitism (by either limiting numbers of mites due to toxic products from the reaction, or indirectly by evoking scratch (Dahl, 1983)) despite the pathological changes that occur. Decreased survival of *Sarcoptes* mites have been observed in humans due to
hypersensitivity (Davis and Moon, 1990). Variability in the severity of clinical scabies thus may be due to the host's immune status and the density of mites (Davis and Moon, 1990).

2.3.1.4 Epidemiology in Dog Populations

Data on the prevalence of scabies in domestic dog populations is limited. It has been suggested that 1% of British dogs are affected with *Sarcoptes scabiei* (Baxter and Leck, 1984). In wild coyote populations in southern Texas, 32% (267 of 843) were found to be infected during examinations from 1974 to 1981 (Pence *et al*., 1983).

The disease is considered non-seasonal (Scott and Horn, 1987; Sosna and Medleau, 1992a; Scott *et al*., 1995). Scabies infection is also considered by some to be without age or sex predilections (Scott and Horn, 1987), although data from coyotes in southern Texas contradicts this (Pence *et al*., 1983). Adult male coyotes had a significantly greater prevalence of mange and a greater proportion of adult than juvenile (less than or equal to 1 year old) coyotes were infected during the peak of the epizootic. Other authors believe young animals (particularly puppies) to be at a relatively high risk (Smith and Claypoole, 1967; Thomsett, 1968; Baxter and Leck, 1984). Seventy one percent of 28 infected animals in one survey were within the 1 to 48 months age group (Thomsett, 1968).

2.3.1.5 Skin Contact and Transmission of *Sarcoptes scabiei*

The most important method of transmission of *S. scabiei* is thought to be by prolonged, close, skin to skin contact (Scott and Horn, 1987), although indirect contact with various fomites (such as bedding or clothing) is also important in the spread of scabies mites between hosts (Soulsby, 1982; Folz, 1984; Scott and Horn, 1987; Arlian and Vyszenski-Moher, 1988).

Despite the requirements of mites to remain on the host, Arlian, Vyszenski-Moher and Pole (1989) have found that all stages of *Sarcoptes scabiei var. canis* are able to survive off the host for several days to several weeks depending on the relative humidity (RH) and temperature. As
mentioned, all life stages frequently leave the burrow, wander on the skin and may be dropped from the host (Arlian and Vyszenski-Moher, 1988). Dislodged mites, recovered from the environments of hosts, penetrate rapidly (9-23 minutes) once placed back on a host (Arlian et al, 1984a). Mites also respond to both host odor and thermal stimulus to reinfest a host (Arlian, Runyan, Sortie and Estes, 1984b). Partial mite dehydration and fasting results in rapid burrowing when the mites re-contact the host (Arlian et al, 1984a). All these factors are important, especially when considering the potential for cross-species transmission.

2.3.1.6 Species Cross Transmission

There is a taxonomic uncertainty of Sarcoptes strains (Arlian and Vyszenski-Moher, 1988). Mites from different host species are difficult to distinguish (Scott and Horn, 1987) but the varieties have been divided into three main groups based on variation in central dorsal field scales and ventrolateral scales near coxae III and IV viewed by SEM (Fain, 1978). Group I contains mites that parasitise humans and pigs, group II contains strains from dogs and group III contains mites that probably belong to unstable strains that are still in the process of adaptation to a new host (Fain, 1978). Morphologically the pig strain is more similar to the human strain than other domestic animal strains, which may indicate that the two may cross-infest more readily (Arlian, Runyan and Estes, 1984c). These criteria, although suggesting that morphology may be important differentials, have not been further investigated in recent times.

Physiological differences between varieties also exist (Arlian et al, 1984c). Antigenically, many strains are similar. Indirect evidence has suggested that var. canis and var. hominis may differ by only two antigens (Arlian, Vyszenski-Moher, Ahmed and Estes, 1991).

Studies involving SEM and DNA technology are essential to clarify the cross species transmission and taxonomy of S. scabiei. Recently, high levels of allelic variability have been demonstrated between individual mites by molecular techniques which will allow a DNA
fingerprinting system to be developed that should be suitable for epidemiological and taxonomic studies of *S. scabiei* (Walton, Currie and Kemp, 1997).

### 2.3.1.7 *Sarcopes scabiei* as a Zoonosis

Despite the difficulties in substantiating cross transmission, scabies is still considered one of the most common zoonotic dermatoses of dogs (Scott and Horn, 1987). There have been numerous reported cases of epidemics of canine scabies in humans (Emde, 1961; Beck, 1965; Newton and Gerrie, 1966; Schwartzman, Sauer and Koch, 1967; Smith and Claypoole, 1967; Thomsett 1968; Elgart and Higdon, 1972; Charlesworth and Johnson, 1974; Ruiz-Maldonado, 1977; Agbede, 1978; Scott and Horn, 1987; Paterson, Pike and Boydell, 1995; Burton, 1997). Overall, human involvement has been reported to occur in 30 to 50% of the canine cases (Scott and Horn, 1987). Thomsett (1968) found that out of 65 human contacts with 28 scabietic dogs, 34 people showed lesions of mite infestation. In another study of 57 cases of canine scabies by Scott and Horn (1987), one or more human contacts were affected in 23 of 45 (51%) households. Emde (1961) reported even higher rates of human disease with 10 out of 11 possible contacts with 6 scabietic puppies developing lesions.

Normaznah, Saniah, Mak, Krishmasamy and Hakim (1996) have determined the seroprevalence of *Sarcopes scabiei* var. *canis* antibodies among Malaysian Aborigines. The Aborigines of Peninsular Malaysia live in close association with dogs, similar to the situation in Australian Aboriginal communities, so the authors presumed canine origin scabies to be very common. Overall, 24.7% (n=312) of people were positive for polyvalent antibodies to canine scabies, but clinically, only 3 people were exhibiting classical scabies infection. The authors suggested that the antibody levels did not correlate to clinical symptoms and that continuous natural exposure to canine mites confers some protective immunity (to both canine and human origin scabies) in the community.
The animal reservoir of sarcoptic mange may contain a well-adapted canine strain for humans that is a source of intermittent epidemics that plague humans (Arlian et al., 1984c).

2.3.1.7.1 Humans at Risk of Infection with Canine Scabies

Information on the sex and age predilection of canine scabies in humans is varied. Charlesworth and Johnson (1974) considered no sex or age risk factors involved in the severity or prevalence of disease although children frequently had greater contact with the infested puppies of the scabies outbreak. Elgart and Higdon (1972) considered the close proximity of children to their pets as a cause of the noted increased susceptibility of children to the zoonosis. Beck (1965) and Smith and Claypoole (1967) also agreed that the frequency of contact with infected animals accounted for a high rate of transmission to humans.

Very transient contact may be sufficient to allow the transfer of the parasite from dog to human. Scott and Horn (1987) and Beck (1965) both noted cases of humans who had very little contact with infected dogs (for example, holding a dog during a bath) who subsequently developed lesions. Both clothed and unclothed areas of the body may be affected as the mites are reported to be able to migrate through clothing (Baxter and Leck, 1984; Scott and Horn, 1987). There is also no correlation between the severity or duration of the canine disease and transmission to human beings (Scott and Horn, 1987), although dogs with crusted lesions would be a greater source of infection to humans as the crusts and scales bearing mites could easily become detached in the environment (Burgess, 1994).

2.3.1.7.2 Clinical Manifestations of Human Infection with Canine Scabies

It is considered that the severe dermatitis caused by canine scabies is often misdiagnosed (Charlesworth and Johnson, 1974). Some authors believe that cutaneous infestation of dog parasites should by included in the differential diagnosis of papular and vesicular skin disease in humans (Smith and Claypoole, 1967; Thomsett, 1968). The typical lesions consist of vesicles, erythematous papules, wheals, crusts and excoriation over pet-contact areas such as the arms,
legs, abdomen and chest (Norins, 1969; Charlesworth and Johnson, 1974; Thomsett, 1990) without the interiginous lesions characteristic of human scabies (Elgart and Higdon, 1972). Although the observation of burrows in the skin usually indicates infection with the human strain, under tropical conditions clearly defined burrows are less common and may be detectable in less than 3% of patients (Burgess, 1994). This further complicates the differentiation of the origin of scabies infestation, particularly in tropical areas.

Human infestation with canine scabies has also appeared suddenly in individuals without any incubation period (Emde, 1961; Beck, 1965; Smith and Claypoole, 1967; Elgart and Higdon, 1972; Fain, 1978; Scott and Horn, 1987) indicating that the lesions are due to irritation rather than sensitisation (Smith and Claypoole, 1967; Elgart and Higdon, 1972). Other patients do not develop lesions until a period of one up to 30 days after exposure (Smith and Claypoole, 1967). This incubation period may represent a hypersensitivity reaction with patients who have been sensitised previously reacting more quickly (Scott and Horn, 1987). Charlesworth and Johnson (1974) considered this as a reason for children (with less opportunity for prior sensitization) developing less severe lesions than adults despite their frequent contact with infected dogs and increased potential for infestation with mites.

Canine scabies mites do not normally burrow into human skin (Cook, 1989). The intensity of the pruritus is enhanced by warmth - as it is in bed or after a warm shower (Emde, 1961; Thomsett, 1990; Scott et al, 1995) and self-trauma by scratching becomes habitual (Thomsett, 1990). These clinical manifestations indicate that scabies infections in warm climates would be quite severe due to the intense pruritus and potential for secondary bacterial infections of excoriated lesions.

Canine scabies in humans is generally a self limiting disease (Elgart and Higdon, 1972; Scott and Horn, 1987) with the lesions regressing in 12 to 14 days if only a few mites are transmitted and contact with affected dogs is terminated (Scott and Horn, 1987; Thomsett, 1990; Burgess,
1994). Human lesions from canine origin mites persist for long periods if there is prolonged repeated contact or if there are many mites involved (Smith and Claypoole, 1967; Scott and Horn, 1987). Lesions have been reported to remain up to 10 weeks (Smith and Claypoole, 1967). In one case, lesions continued to spread for several weeks after animal contacts were removed (Norins, 1969). Skin scrapings revealed mites and eggs, one of which hatched later. The skin lesions were typical of canine scabies in humans, but the mite was able to propagate in the human host (Norins, 1969).

There has also been one report of a child with Norwegian (crusted) scabies caused by *S. scabiei* var. *canis*. Three dogs in the household were found to have the mites and the other members of the family were also affected (Scott *et al.*, 1995).

Experimental infection of humans with *Sarcoptes* var. *canis* mites has also shown that the mites can induce severe pruritus within 24 hours, burrow, defaecate and lay eggs (Estes, Kummel and Arlian, 1983). The mites do not survive in the abnormal host more than a few days, though (Estes *et al.*, 1983). Mites retrieved from experimentally infected humans are also able to cause disease in normal dogs (Estes *et al.*, 1983). This situation is considered unusual as the disease in humans rarely results in mites establishing in the skin (and hence being retrieved by skin scraping methods).

### 2.3.1.7.3 Transmission of Human Scabies to Dogs

*S. scabiei* var. *hominis* is thought to only occasionally produce disease in dogs (Folz, 1984).

### 2.3.2 *Ancylostoma caninum*

*Ancylostoma caninum*, one of four canine hookworms, is responsible for anaemia and poor condition in dogs and also infections in humans resulting in cutaneous larva migrans, visceral larva migrans or eosinophilic enteritis. *Ancylostoma braziliense* and *Uncinaria stenocephala*
are also capable of cutaneous larva migrans in humans, but, as with *Ancylostoma ceylanicum*, are much less pathogenic in the canine host (Stevenson and Hughes, 1988; Behnke, 1990).

Hookworms are a typical geohelminth with free-living larval stages up to the third moult. Hookworms have a direct lifecycle with no intermediate host, although hookworms can utilise paratenic hosts in which there is no further development of the parasite. Infection of the definitive host is by ingestion (from environmental sources or the tissues of paratenic hosts) or cutaneous migration of the third larval stage. Third stage larvae of *Ancylostoma caninum* are also capable of infecting the offspring of infected bitches via the colostrum or placenta.

2.3.2.1 Hookworm Infection in Dogs

Oral Infection

The most direct form of infection in dogs is after ingestion of infective larvae from the environment (Bowman, 1992). There is no tissue migration with this type of infection (Dunsmore and Shaw, 1990) as the larvae continue development within the mucosa, and later, lumen of the small intestine. As adults, *A. caninum* worms attach to the second and third quarters of the small intestine where they feed on the host’s blood with the assistance of secreted anticoagulants (Behnke, 1990).

Percutaneous Infection

Third stage larvae are also capable of infecting dogs percutaneously (entering hair follicles) (Behnke, 1990) or via the oral mucosa, possibly by the use of hyaluronidase or metalloproteases enzymes (Bowman, 1992). They then undergo tracheal migration to reach the small intestine for final development to adult (Behnke, 1990; Bowman, 1992). During this migration through the blood stream to the lungs and trachea, some dogs experience severe pulmonary damage (Miller, 1971). Patency is achieved with percutaneous and direct infection after about 14 to 18 days in puppies and 15 to 26 days in older dogs (Miller, 1971; Behnke, 1990; Bowman, 1992).
Paratenic Hosts

Similar prepatent periods of 14 to 15 days are achieved if dogs ingest infected paratenic hosts (Bowman, 1992) such as mice and rats (Lee, Little and Beaver, 1975). In these hosts, the 3rd stage larvae can persist in, and move between, muscle fibres where they produce no direct inflammatory reaction (Lee et al, 1975; Bowman, 1992). This mode of infection, though, is of minor epidemiological significance for canine hookworms (Dunsmore and Shaw, 1990).

Vertical Transmission

Puppies may also be infected with *A. caninum* by trans-colostral or transplacental migration of larvae (Kelly, Thompson, Chow and Whitlock, 1976). Transcolostral transfer accounts for more than 95% of all prenatal-colostral infection (Miller, 1971; Kelly et al, 1976).

The larvae are stimulated to migrate to the mammary gland and milk by increased levels of hormones (estradiol and progesterone) in the bitch (Schad and Page, 1982). It has been suggested that the hormones of pregnancy and lactation do not act on the parasite *per se*, but rather they stimulate an increased output of corticosteroids (Schad and Page, 1982). Corticosteroids in turn compromise the host’s resistance to the parasites (Schad and Page, 1982). Bitches infected by the oral or percutaneous route may secrete larvae in the milk for more than a year (Bowman, 1992). This could have important implications for hookworm control as bitches with sequestered larvae provide an additional route of infection for puppies. Puppies can start contaminating the environment with hookworm eggs by 12 to 16 days (Bowman, 1992) and there is no tissue migration in puppies infected this way.

Clinical Effects of Canine Hookworm Infection in Dogs

Young puppies are most severely affected by hookworm infection (Miller, 1971). In one study, 11 of 17 puppies born to females experimentally infected with 10 000 to 20 000 larvae died 21 to 28 days after birth (Bowman, 1992). Deaths in puppies are due to severe anaemia (Miller, 1971).
The adult hookworms feed on mucosal tissue and change sites about six times a day (Behnke, 1990). Each time the site is changed, large amounts of blood are lost from the vacated attachment site (Crompton and Stephenson, 1990). Overall, female worms are responsible for a loss of approximately 40μL/day, and males (only one-third the size of females) cause a loss of 13μL/day (Crompton and Stephenson, 1990).

**Perpetuation of the Life cycle**

The female worms are very prolific (Kelly et al., 1976; Dunsmore and Shaw, 1990). Approximately 10 000 eggs per day are produced by a single female (Behnke, 1990), but the number of eggs per female is lower when large numbers of worms are present (density dependent depression of fecundity) (Dunsmore and Shaw, 1990; Bowman, 1992). Likewise, the egg counts are lower in longer existing infections with equivalent worm burdens (Bowman, 1992).

After passage in the faeces, the morulated eggs hatch to first stage larvae provided the environmental conditions of humidity and oxygen are favourable. The optimum temperature for hatching is 23-30°C, but they are able to hatch within a temperature range of 12-37°C (Dunsmore and Shaw, 1990). Protected by a cuticle covering, the developing larvae can survive in the environment for many months, being able to withstand both heat and cold, but not extreme dryness (Kelly et al., 1976; Nwosu and Anya, 1980). The optimum temperature of larval development has been found to be around 25 to 30°C (Bowman, 1992) at which translation to infectivity takes only one week.

**2.3.2.2 Prevalence of Hookworm in dogs**

The prevalence of hookworm in dogs varies considerably around the world depending on the local climatic and geographic conditions. Tropical regions with conditions conducive to hookworm survivability are likely to have higher prevalences as evidenced by Table 2.1. In the Kimberley, sampling done in the cooler winter months revealed 51.1% of dogs to have
### Table 2.1: Prevalence of Canine Hookworm throughout the World

<table>
<thead>
<tr>
<th>Location</th>
<th>Number</th>
<th>%</th>
<th>Study</th>
<th>Age</th>
<th>Type of Dogs</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Canada</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Montreal</td>
<td>239</td>
<td>12.5</td>
<td>Over 2 years</td>
<td>Mixed</td>
<td>Stray dogs</td>
<td>Seah, Hucal and Law (1975)</td>
</tr>
<tr>
<td>USA</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>New Orleans**</td>
<td>623</td>
<td>92</td>
<td></td>
<td>Mixed</td>
<td>Pound dogs</td>
<td>Schock (1976)</td>
</tr>
<tr>
<td>Missouri</td>
<td>1468</td>
<td>35.8</td>
<td>Retrospective</td>
<td>Mixed</td>
<td>Pet dogs</td>
<td>Visco, Corwin and Selby (1977)</td>
</tr>
<tr>
<td>Louisiana</td>
<td>4058</td>
<td>38.5</td>
<td>Retrospective</td>
<td>Mixed</td>
<td>Pet dogs</td>
<td>Hoskins, Malone, Smith and Uhl (1982)</td>
</tr>
<tr>
<td>Carolina</td>
<td>12</td>
<td>67</td>
<td></td>
<td>Mixed</td>
<td>Free range wolves</td>
<td>Philips and Scheck (1991)</td>
</tr>
<tr>
<td>Carolina</td>
<td>21</td>
<td>24</td>
<td></td>
<td>Mixed</td>
<td>Captive wolves</td>
<td>Philips and Scheck (1991)</td>
</tr>
<tr>
<td>Jamaica</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nigeria</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Zaria</td>
<td>166</td>
<td>70.5</td>
<td></td>
<td>Mixed</td>
<td>Stray dogs</td>
<td>Dada and Belino (1979)</td>
</tr>
<tr>
<td>Jordan</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Jordan *</td>
<td>756</td>
<td>5</td>
<td>June-Sept</td>
<td>Mixed</td>
<td>All types</td>
<td>Abo-Shehada and Ziyadeh (1991)</td>
</tr>
</tbody>
</table>
Table 2.1 (cont.): Prevalence of Hookworm throughout the World

<table>
<thead>
<tr>
<th>Location</th>
<th>Number</th>
<th>%</th>
<th>Study</th>
<th>Age</th>
<th>Type of Dogs</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brisbane</td>
<td>66</td>
<td>68.2</td>
<td>Over two years, post mortem</td>
<td>Mixed</td>
<td>Pet dogs</td>
<td>Setasuban and Waddell (1973)</td>
</tr>
<tr>
<td>Brisbane</td>
<td>401</td>
<td>38</td>
<td>Summer</td>
<td>Mixed</td>
<td>Stray dogs</td>
<td>Prociv, Collyer, Lim and Tang (1994)</td>
</tr>
<tr>
<td>Brisbane</td>
<td>102</td>
<td>67</td>
<td>&lt;1 year</td>
<td></td>
<td>Stray dogs</td>
<td>Boreham and Capon (1982)</td>
</tr>
<tr>
<td>Brisbane</td>
<td>105</td>
<td>87</td>
<td>&gt;1 year</td>
<td></td>
<td>Stray dogs</td>
<td>Boreham and Capon (1982)</td>
</tr>
<tr>
<td>Charleville district, Queensland</td>
<td>112</td>
<td>20.1</td>
<td>Mixed</td>
<td></td>
<td>Sheep dog</td>
<td>Cornack and O’Rouke (1991)</td>
</tr>
<tr>
<td>Sydney</td>
<td>464</td>
<td>36</td>
<td>Mixed</td>
<td></td>
<td></td>
<td>Kelly and Ng (1975) cited in Dunsmore and Shaw (1991)</td>
</tr>
<tr>
<td>New South Wales</td>
<td>15</td>
<td>100</td>
<td>Mixed</td>
<td></td>
<td>Aboriginal community</td>
<td>Jenkins and Andrew (1993)</td>
</tr>
<tr>
<td>New South Wales</td>
<td>930</td>
<td>8.3</td>
<td>Post mortem</td>
<td>Mixed</td>
<td>Feral foxes</td>
<td>Ryan (1976)</td>
</tr>
<tr>
<td>Melbourne***</td>
<td>190</td>
<td>11.6</td>
<td>Over 1 year</td>
<td>Mixed</td>
<td>Stray dogs</td>
<td>Johnston and Gasser (1993)</td>
</tr>
<tr>
<td>Melbourne***</td>
<td>152</td>
<td>4</td>
<td>Over 1 year</td>
<td>Mixed</td>
<td>Pet dogs</td>
<td>Johnston and Gasser (1993)</td>
</tr>
<tr>
<td>Northeast Victoria****</td>
<td>204</td>
<td>49.5</td>
<td>Postmortem</td>
<td>Mixed</td>
<td>Feral dogs and dingoes</td>
<td>Coman (1972)</td>
</tr>
<tr>
<td>Adelaide</td>
<td>1614</td>
<td>3.04</td>
<td>Over two years</td>
<td>Mixed</td>
<td>Suspected parasitism and routine investigations</td>
<td>Moore and O’Callaghan (1985)</td>
</tr>
<tr>
<td>Kimberley region</td>
<td>182</td>
<td>51.1</td>
<td>Winter</td>
<td>Mixed</td>
<td>Aboriginal communities</td>
<td>Thompson Meloni, Reynoldson and Hopkins (1993a)</td>
</tr>
</tbody>
</table>

All species are _Ancylostoma caninum_ unless otherwise stated.
* Genus of hookworm not specified
** _Ancylostoma_ spp.
*** 89% of hookworm isolations possibly _Uncinaria stenocephala_
**** _Uncinaria stenocephala_
*Ancylostoma caninum* (Thompson, Meloni, Hopkins, Deplazes and Reynoldson, 1993a). The prevalence of hookworm is expected to be higher during the wet season (see 2.2.2.3).

Kirkpatrick (1988) found that even pet dogs with a relatively high level of care frequently harbour intestinal parasites. Prociv, Collyer, Lim and Tang (1994), though, found *Ancylostoma caninum* to be highly prevalent in street roaming dogs as opposed to pet dogs which were more likely to be kept at home and better cared for. Prociv *et al* (1994) commented that dogs that harboured hookworm couldn’t be determined by external appearances. This has implications for client compliance in hookworm control programs, as dogs that look well are less likely to be presented for treatment.

### 2.3.2.3 Seasonality of Hookworm Infection Rates

Most studies regarding the epidemiology of hookworms and the seasonality of infection rates have been on human hookworms in field environments. The two main hookworms of humans are *Ancylostoma duodenale* and *Necator americanus*. These hookworms differ in their route of infection and in their subsequent development. *A. duodenale* is able to be transmitted percutaneously and after ingestion, whereas *N. americanus* appears to achieve infection only by percutaneous means (Behnke, 1990; Bundy and Keymer, 1991). *A. duodenale* can also undergo arrested development (see 2.2.2.3) and *in utero* or transmammary transmission is also likely with this species (Bundy and Keymer, 1991). *A. duodenale* has more possibility for infection of the host and is more resistant to extrinsic factors which makes control of this parasite in human populations more difficult than control of *N. americanus*. Of the two human species, *A. duodenale* is more closely related to *Ancylostoma caninum* in terms of transmission dynamics and control (see Appendix A), but many studies of human hookworm fail to differentiate species and hence comparison is difficult.

As a result of the strict requirements for the development of hookworm, trends have been found between season and prevalence of human hookworms. In tropical or monsoon regions (with
mixed populations of *N. americanus* and *A. duodenale*), some studies have found the greatest marked increase in human hookworm burden following the onset of the rainy season (Nwosu and Anya, 1980) with a gradual decrease in prevalence and egg counts during the later half of the dry season (Nwosu and Anya, 1980). Schad, Chowdhury, Dean, Kochar, Nwosu, Thomas and Tonascia (1973) found the greatest transmissibility of *A. duodenale* to be at the end of the wet season, whereas other studies have found the largest aggregations of larvae in the environment earlier rather than later in the rainy season (Hominick, Dean and Schad, 1987). Transmission of human hookworms generally is restricted to the rainy season (Hominick *et al.*, 1987) and in some cases, such as in Nigeria, the transmission cycle is broken during the dry season (Nwosu and Anya, 1980).

Rainfall appears to be the limiting factor to prevalence of infection and secondarily controlled intensity of infection with human hookworm (mostly *N. americanus*) (Miller, 1970). Temperature has little effect on the build-up of hookworm infection in endemic tropical areas as the minimum temperature for hookworm development (10°C) is well below the temperatures of the tropics (Nwosu and Anya, 1980).

### 2.3.2.4 Arrested Development of Hookworm

Contrary to the studies mentioned above, Schad *et al.* (1973) found that the rise in *A. duodenale* egg counts began in the dry season rather in the wet season in eastern India and Taiwan. In this case, the seasonal increase in egg counts does not reflect a rise in transmission and thus appears independent of moisture conditions in the environment (Nawalinski and Schad, 1974). In these studies, *A. duodenale* larvae acquired during the rainy season of one year remained dormant in the host (humans) until just before the monsoon season of the following year when they resumed development, matured and began to lay eggs (Nawalinski and Schad, 1974; Schad *et al.*, 1973).
This phenomenon was also demonstrated during a self-induced infection, when it was found that
the prepatent period of *A. duodenale* in humans can extend to between 22 and 38 weeks which
is 3 to 5 times normal (Nawalinski and Schad, 1974; Schad *et al.*, 1973). In another study, eggs
did not appear in the faeces until 40 weeks after percutaneous infection (Nawalinski and Schad,
1974).

Arrested development (hypobiosis) is commonly recognised amongst the trichostrongylid
nematodes of ruminants such as *Haemonchus* and *Ostertagia* (Schad *et al.*, 1973; Nawalinski
and Schad, 1974; Kelly *et al.*, 1976). Decreasing photoperiod and temperature acting on the
infective larvae of these parasites are thought to induce developmental arrest (Schad *et al,*
1973; Nawalinski and Schad, 1974). Likewise, some hookworm larvae can undergo arrested
development after exposure to degenerating environmental conditions if they are genetically
determined for this feature (Nawalinski and Schad, 1974). Kelly *et al.* (1976) (and Schad, 1973,
cited in Kelly *et al.* (1976)) found that a high proportion (65%) of *A. caninum* infective larvae in
helminth naive dogs subjected to unsuitable conditions of a sudden drop in temperature
subsequently underwent hypobiosis at the level of the small intestine in the host. Prociv *et al.*
(1994) commented on this phenomenon in their study, but were unable to determine if the strain
of *A. caninum* in Queensland was capable of this.

Arrested development appears to be an adaptation of the parasite to reduce the egg output
wasted in a hostile environment. Likewise faecal egg output is increased at times that coincide
with environmental conditions that are favourable for the development of eggs to the infective
larval stage (just before the monsoons) (Schad *et al.*, 1973).

Kelly *et al.*, (1976) have outlined several practical implications for this feature:
1. Inhibited larvae provide a ‘protected’ reservoir of infection. The intestines of dogs can be
   repopulated by development of somatically arrested larvae (Bowman, 1992).
2. Chemotherapy with conventional anthelmintics is claimed to be ineffective against the dormant tissue stage of *A. caninum* in the mammary glands of the bitch (Miller, 1971), although 'off-label' dosages with ivermectin subcutaneously have been effective in trials (Bowman, 1992). This implies that most anthelmintics may also be ineffective against arrested larvae of gut dwelling nematodes.

3. Arrested worms that have resumed development may replace susceptible adult worms removed from the small intestine by anthelmintic treatment.

4. The presence of hypobiotic forms within the host cannot be detected by faecal examination for worm eggs, which complicates diagnosis.

### 2.3.2.5 Canine Hookworm as a Zoonosis

*Ancylostoma caninum* infection in the human host can manifest in several ways; as cutaneous larva migrans, visceral larva migrans or eosinophilic enteritis.

#### 2.3.2.5.1 Prevalence of Human Infection with Canine Hookworms

Reliable data on the frequency of human infection with zoonotic hookworms are not available from the United States (Harvey, Roberts and Schantz, 1991). Likewise, in Australia, published reports of zoonotic *A. caninum* are rare although cutaneous larva migrans is not uncommon in northern Queensland (Setasuban and Waddell, 1973; Boreham and Capon, 1982). A serological study of *A. caninum* antibodies in people from a community without *A. duodenale* (which can cross-react with *A. caninum*) in coastal New South Wales revealed 58% of people to have antibodies (n=96) (Jenkins, Meek, Ardler and Hawksby, 1996). The corresponding prevalence in the dogs was 74% (n=26). Eosinophilic enteritis caused by non-patent hookworm infections was first reported from Queensland and will be discussed later.

#### 2.3.2.5.2 Cutaneous Larva Migrans

Cutaneous larva migrans (CLM), due to the cutaneous migration of *A. caninum* larvae, has been known since the 1920s (Croese, Loukas, Opdebeeck and Prociv, 1994a). Intense pruritus is the
primary symptom which is usually self-limiting within a few day or weeks (Schantz, 1991). In the typical case of CLM, a few serpiginous trails are present in the epidermis, often on the foot, as a result of the person having been in contact with damp soil on sandy beaches or in yards (Little, Halsey, Cline and Katz, 1983). Sometimes massive infection can occur in which numerous cutaneous lesions occur over a large area of the body (Little et al, 1983). In heavy infections, visceral larva migrans with pulmonary symptoms have been reported to occur within a few days after exposure (Little et al, 1983).

Cutaneous larva migrans, though, is more common in infections with another canine hookworm, *Ancylostoma braziliense*, rather than *A. caninum* (Miller, 1979; Stevenson and Hughes, 1988; Robinson, Thompson and Lindo, 1989).

2.3.2.5.3 Visceral Larva Migrans

As mentioned, in some cases *Ancylostoma caninum* larvae may penetrate into deeper tissues and produce symptoms of visceral larva migrans (Schantz, 1991). In one case of massive cutaneous larva migrans in a 20 years old man, the patient suffered pulmonary symptoms, fever and larval invasion of the skeletal muscles (Little et al, 1983). *Ancylostoma* larvae have also been recovered from the corneas of 3 people including a 25 years old man who also had a tender, painful swollen knee possibly due to migrating larvae (Little et al, 1983). Several workers have shown that third-stage *A. caninum* larvae can persist in the muscle of rodent paratenic hosts for more than one year (Matsusaki, 1939; Lee et al, 1975) and that in these hosts, the larvae are located intracellularly. This is possibly the case in humans infected with *A. caninum*.

2.3.2.5.3.1 Eosinophilic Enteritis

Adult *A. caninum* have also been isolated from human intestines in South America, Israel and the Philippines (Croese, Loukas, Opdebeeck, Fairley and Prociv, 1994b). Other occasional reports of human intestinal adult *A. caninum* infections have been from routine examination of specimens recovered from anthelmintic treatment or autopsy. In these cases, the parasites have
never been implicated in clinical disease or shown to be fully developed or producing eggs. In Townsville, Australia, an epidemic of 93 cases of eosinophilic enteritis was reported in 1990 (Prociv and Croese, 1990). Further investigation of the patients showed one patient to have an adult A. caninum attached to the ileum and another patient to have an unspeciated Ancylostoma spp. adult free in the intestines. This highlighted the possibility of A. caninum being the causative agent for the eosinophilic enteritis epidemic.

Since 1985, 11 Queensland patients have been definitively diagnosed, using laparotomy or colonoscopy, as being infected with A. caninum (Croese, 1995). A further 190 cases of obscure abdominal pain, often associated with blood eosinophilia and responsive to treatment with mebendazole, have been investigated (Croese et al, 1994b). Croese et al (1994b) have linked this syndrome of eosinophilic enteritis serologically and epidemiologically to occult infection with A. caninum. Awareness of this syndrome has led to more confirmed cases throughout north-eastern Australia. Many patients with obscure abdominal pain are now treated (or self-medicated) with anthelmintics on the basis of the typical clinical picture.

The syndrome is not restricted to the tropical north as one man from Brisbane was diagnosed with zoonotic ancylostomiasis (Sandford and Prociv, 1991). The condition has also been suspected in two cases from Darwin (Currie and Anstey, 1991). In all reports of this syndrome, patients have had higher than average dog ownership (sometimes with confirmed dog infection) (Croese et al, 1994a).

**Clinical Symptoms of Eosinophilic Enteritis**

Common clinical features of investigated patients included; pain (sometimes associated with obstruction or peritonitis), diarrhoea, abdominal distension, weight loss and rectal bleeding (Croese et al, 1994b). The symptoms coincided with transient blood eosinophilia, elevated IgE levels and a specific antibody response measured by ELISA (Croese et al, 1994a). These findings are common with other enteric parasitic infections including the human-specific
hookworms (Croese et al, 1994b), although the worms in the eosinophilic enteritis syndrome are found unusually low in the gastrointestinal tract and are immature and non-patent (Croese et al, 1994b). Patients don’t have respiratory or cutaneous symptoms as is noted with classical cases of visceral larva migrans (Prociv and Croese, 1990). The principal symptom is abdominal pain.

Cases of strongly suspected occult A. caninum infection also follow a seasonal pattern as occurs in host specific ancylostomiasis. Aestivation of larvae may also be a feature of the parasites as the lowest seasonal rates in Townsville were found during the winter (June to August) and the highest in spring (September to November) when reactivation of hypobiotic larvae is suspected (Croese, 1995).

Pathogenesis of Eosinophilic Enteritis

The pathologic finding in zoonotic eosinophilic enteritis is focal or diffuse eosinophilic inflammation caused by a type 1 hypersensitivity response to secreted antigens from the hookworms (Croese et al, 1994b).

Treatment of Eosinophilic Enteritis

Patients examined by Croese et al (1994a) were treated with mebendazole when A. caninum infection was suspected and the symptoms had not resolved spontaneously. Typically, almost complete resolution of symptoms, followed by decreasing eosinophil and IgE values, was found after treatment, although recurrent episodes after several months were common (Croese et al, 1994a). The recurrence of clinical signs may be attributed to either reinfection or mobilisation of dormant larvae from tissue sites (Prociv and Croese, 1990).

Difficulties in Diagnosis of Eosinophilic Enteritis

Owing to the difficulties in isolating non-patent adult A. caninum worms from patients, considerable scepticism has developed over the ability of such a common and widespread parasite to cause such dramatic illness (Sandford and Prociv, 1991). The use of colonoscopy
and the eradication of endemic human hookworm (the main differential diagnosis) facilitate detection of infection. The development of ELISA and Western Blot serological tests has also proven valuable in determining the epidemiology of the condition. Serological tests still have limited diagnostic application (Croese et al, 1994a) and unfortunately are unable to differentiate A. duodenale infections from A. caninum. This is important in areas where human ancylostomiasis is still prevalent (mostly Aboriginal communities) (Currie and Anstey, 1991; Hopkins, Gracey, Hobbs, Spargo, Yates and Thompson, 1997). The potential for zoonotic disease in Aboriginal communities, where there is intense contact with often heavily parasitised dogs, is considered important (Currie and Anstey, 1991) but confirmed diagnosis of zoonotic ancylostomiasis remains difficult.

2.3.3 Toxocara canis

Toxocara canis is an ascarid nematode that uses dogs as a definitive host. The parasite has a direct life cycle, but is capable of infecting various mammals as paratenic hosts. T.canis is considered one of the most important of the canine parasites because of its widespread distribution, prenatal and neonatal transmission to pups and ability to cause disease in humans (Greve, 1971).

2.3.3.1 Canine Infection with Toxocara canis

T. canis eggs require a 2-5 week maturation period in the soil for embryonation to occur (Glickman and Schantz, 1981; Baxter and Leck, 1984; Girdwood, 1986). If swallowed by the definitive host, dogs, the eggs hatch under the influence of gastric and intestinal juices to produce larvae (Lloyd, 1986) that penetrate the intestinal wall and later undergo tracheal migration (Dunsmore and Shaw, 1990). Maturation is completed in the small intestine and patency is reached at 30 - 35 days post infection (Glickman and Schantz, 1981). Adult females may produce about 200 000 eggs per day (Glickman and Schantz, 1981) and live for about 4 months.
The migratory pattern of *T. canis* varies according to the age of the host (Greve, 1971). In dogs older than 6 weeks, fewer and fewer larvae are able to complete tracheal migration and the larvae become trapped in granulomas in various tissues (somatic migration) (Greve, 1971). As long as *Toxocara* larvae remain in the granulomas, they do not develop beyond the infective stage (Greve, 1971). These larvae may survive for years (Glickman and Schantz, 1981).

In bitches, transplacental migration of larvae occurs after the 42nd day of pregnancy and has been attributed to hormonal changes (Glickman and Schantz, 1981). The location of the reservoir of larvae is not known, but infected bitches can infect prenatal pups for at least 600 days after initial infection (Webster, 1958, cited in Dunsmore and Shaw, 1990). This is the primary mode of transmission of *T. canis* in dogs (Seah, Hucal and Law, 1975) and Glickman and Schantz (1981) consider all puppies born to infected bitches to be prenatally infected with the larvae of *T. canis*. Infected puppies can be shedding eggs in about 3 weeks after birth (Seah *et al*, 1975).

Transmammary infection peaks during the second week of lactation (Glickman and Schantz, 1981), but is of minor importance (Dunsmore and Shaw, 1990). Other routes of infection to dogs include ingestion of paratenic hosts with larvae in their tissues (mostly brain) and ingestion of late stage larvae or immature adults in the vomitus or faeces of infected puppies (Glickman and Schantz, 1981).

### 2.3.3.2 Distribution of *Toxocara canis* throughout the World

*T. canis* is widely distributed and is capable of infecting canids from all tropical and temperate regions of the world (Woodruff, Bisseru and Bowe, 1966; Woodruff, 1970; Glickman and Schantz, 1981; Duwel, 1984) (Table 2.2).

*Toxocara* has not been recorded from dogs in the Kimberley region, although this may reflect the age groups of dogs examined in the only study conducted in the area (Thompson *et al*,...
Table 2.2: Prevalence of *Toxocara canis* throughout the World

<table>
<thead>
<tr>
<th>Location</th>
<th>Number</th>
<th>%</th>
<th>Study</th>
<th>Age</th>
<th>Type of Dogs</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Canada</td>
<td>Montreal</td>
<td>239</td>
<td>43.5</td>
<td>Over 2 years</td>
<td>Mixed</td>
<td>Stray dogs</td>
</tr>
<tr>
<td>USA</td>
<td>Missouri</td>
<td>1468</td>
<td>17.9</td>
<td>Retrospective</td>
<td>Mixed</td>
<td>Pet dogs</td>
</tr>
<tr>
<td></td>
<td>Pennsylvania</td>
<td>2294</td>
<td>5.5</td>
<td>Retrospective</td>
<td>Mixed</td>
<td>Pet dogs</td>
</tr>
<tr>
<td></td>
<td>Carolina</td>
<td>12</td>
<td>8</td>
<td>Mixed</td>
<td>Free range wolves</td>
<td>Phillips and Scheck (1991)</td>
</tr>
<tr>
<td>Nigeria</td>
<td>Zaria</td>
<td>166</td>
<td>38.6</td>
<td>Prevalence</td>
<td>Mixed</td>
<td>Stray dogs</td>
</tr>
<tr>
<td>Kenya</td>
<td>Nairobi</td>
<td>35</td>
<td>5.7</td>
<td>Prevalence</td>
<td>Mixed</td>
<td></td>
</tr>
<tr>
<td>Tanzania</td>
<td>Tanzania</td>
<td>50</td>
<td>28</td>
<td>Prevalence</td>
<td>Mixed</td>
<td></td>
</tr>
<tr>
<td>Ireland</td>
<td>County Cork</td>
<td>100</td>
<td>25</td>
<td>Over 3 months</td>
<td>Mixed</td>
<td>Stray dogs</td>
</tr>
</tbody>
</table>
Table 2.2 (cont.): Prevalence of *Toxocara canis* throughout the World

<table>
<thead>
<tr>
<th>Location</th>
<th>Number</th>
<th>%</th>
<th>Study</th>
<th>Age</th>
<th>Type of Dogs</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brisbane</td>
<td>102</td>
<td>35</td>
<td>&lt;1 year</td>
<td>Stray dogs</td>
<td></td>
<td>Boreham and Capon (1982)</td>
</tr>
<tr>
<td>Brisbane</td>
<td>105</td>
<td>5</td>
<td>&gt;1 year</td>
<td>Stray dogs</td>
<td></td>
<td>Boreham and Capon (1982)</td>
</tr>
<tr>
<td>Brisbane</td>
<td>108</td>
<td>0.9</td>
<td>Mixed</td>
<td>Pet dogs</td>
<td></td>
<td>Boreham and Capon (1982)</td>
</tr>
<tr>
<td>Charleville district</td>
<td>112</td>
<td>few</td>
<td>Mixed</td>
<td>Sheep dogs</td>
<td></td>
<td>Cornack and O’Rouke (1991)</td>
</tr>
<tr>
<td>Queensland</td>
<td>136</td>
<td>74</td>
<td>Mixed</td>
<td></td>
<td></td>
<td>Welsh, Dobson and Freeman (1979)</td>
</tr>
<tr>
<td>Sydney</td>
<td>63.8</td>
<td></td>
<td>&lt; 6 mo</td>
<td>Pet dogs</td>
<td></td>
<td>Kelly and Ng (1975) cited in Dunsmore and Shaw (1991)</td>
</tr>
<tr>
<td>Sydney</td>
<td>21.3</td>
<td></td>
<td>&gt; 6 mo</td>
<td>Pet dogs</td>
<td></td>
<td>Kelly and Ng (1975) cited in Dunsmore and Shaw (1991)</td>
</tr>
<tr>
<td>New South Wales</td>
<td>15</td>
<td>0</td>
<td>Mixed</td>
<td>Aboriginal community dogs.</td>
<td>Jenkins and Andrew (1993)</td>
<td></td>
</tr>
<tr>
<td>New South Wales</td>
<td>930</td>
<td>35.2</td>
<td>Post mortem</td>
<td>Mixed</td>
<td>Feral foxes</td>
<td>Ryan (1976)</td>
</tr>
<tr>
<td>Melbourne</td>
<td>190</td>
<td>17.4</td>
<td>Over 1 year</td>
<td>Mixed</td>
<td>Stray</td>
<td>Johnston and Gasser (1993)</td>
</tr>
<tr>
<td>Melbourne</td>
<td>152</td>
<td>6.6</td>
<td>Over 1 year</td>
<td>Mixed</td>
<td>Pet</td>
<td>Johnston and Gasser (1993)</td>
</tr>
<tr>
<td>Northeast Victoria</td>
<td>204</td>
<td>13.5</td>
<td>Post mortem</td>
<td>Mixed</td>
<td>Feral dogs and dingoes</td>
<td>Coman (1972)</td>
</tr>
<tr>
<td>Adelaide</td>
<td>1614</td>
<td>6.44</td>
<td>Over two years</td>
<td>Mixed</td>
<td>Suspected parasitism and routine investigation</td>
<td>Moore and O’Callaghan (1985)</td>
</tr>
<tr>
<td>Perth</td>
<td>235</td>
<td>2.6</td>
<td>Mixed</td>
<td>Clinical Pathology samples</td>
<td>Dunsmore, Thompson and Bates (1984)</td>
<td></td>
</tr>
<tr>
<td>Central Australia</td>
<td>50</td>
<td>52</td>
<td>Mixed</td>
<td></td>
<td></td>
<td>Welsh, Dobson and Freeman (1979)</td>
</tr>
<tr>
<td>Kimberley Region</td>
<td>182</td>
<td>0</td>
<td>Winter</td>
<td>Mixed</td>
<td>Aboriginal community dogs</td>
<td>Thompson, Meloni, Hopkins, Deplazes and Reynolds (1993a)</td>
</tr>
</tbody>
</table>
1993a). Welch, Dobson and Freeman (1979) attributed the relatively low infection rates in central Australia to the harsh environment as *Toxocara* eggs rapidly disintegrate when exposed to sunlight or desiccation (Glickman and Schantz, 1981). This may also be true for the Kimberley region.

Many studies of the prevalence of *T. canis* have found younger dogs to be more likely to be infected (Seah *et al*, 1975; Visco, Corwin and Selby, 1977; Kirkpatrick, 1988), which is in accordance with frequency of prenatal infection of puppies. Older dogs are also able to develop specific immunity to arrest larval development, except during pregnancy and lactation when the response is depressed (Baxter and Leck, 1984; Kirkpatrick, 1988). Puppies are consequently considered the most important sheddors of zoonotic infective eggs.

2.3.3.3 *Toxocara canis* as a Zoonosis

2.3.3.3.1 Route of Infection

Humans acquire infection by eating faecally contaminated food or by geophagia of infected soil, as is more common in children (Seah *et al*, 1975; Girdwood, 1986). Direct contact with infected dogs plays a secondary role in infection because the parasite eggs require an incubation period in the environment (Glickman and Schantz, 1981). Most infections are acquired from the soil of public places such as parks (Baxter and Leck, 1984).

2.3.3.3.2 Migration in the Human Host

In humans, ingested *Toxocara canis* larvae normally do not develop further after penetrating the intestinal wall (Baxter and Leck, 1984). Migration of the larvae through the tissues can occur and may induce hepatomegaly, pulmonary infiltration, eosinophilia, hypergammaglobulinaemia, fever and general malaise (Seah *et al*, 1975; Glickman and Schantz, 1981; Baxter and Leck, 1984). Migration is initially via the blood vessels until the larvae reach blood vessels that become small enough to impede further movement (Woodruff, 1970; Glickman and Schantz, 1981). The larvae then actively bore through the vessel walls and wander in the surrounding
tissue. Necrosis, haemorrhage and infiltration of inflammatory cells into the tissues results from the tracks made by the burrowing larvae. The main pathological manifestation is from the mechanical damage caused by an immune mediated inflammatory response to the larvae (Glickman and Schantz, 1981). Dead larvae become encapsulated in granulomas, which, depending on their location, can manifest clinically (Woodruff, 1970; Seah et al, 1975; Glickman and Schantz, 1981; Baxter and Leck, 1984).

2.3.3.3 Clinical Features of Human Zoonotic Infection with *Toxocara canis*

A wide range of tissues may be affected with wandering larvae; hence the clinical signs are often non-specific. The most common clinical features are; abdominal pain, hepatomegaly, anorexia, nausea, vomiting, lethargy, sleep and behavioural disturbances, pneumonia, cough, wheeze, pharyngitis, cervical adenitis, headache, limb pains and fever (Seah et al, 1975; Glickman and Schantz, 1981; Cook, 1989).

The two main clinical manifestations are visceral (VLM) and ocular larva migrans (OLM) (Girdwood, 1986). *Toxocara* infection rarely results in both syndromes and this may be related to the dose of the organism ingested (Glickman and Schantz, 1981). In VLM, higher doses probably invoke a large immunological response resulting in rapid destruction of larvae throughout the body. These larvae are often found encapsulated in granulomas throughout the liver (Dunsmore and Shaw, 1990). Visceral larva migrans tends to be self-limiting with symptomatic cure within the year being the most common result (Girdwood, 1986).

Lower infective doses are associated with a higher probability of OLM than VLM (Schantz, 1989). The immune response is probably not as pronounced, so the larvae are able to reach the protected site of the eye. OLM results in retinal granulomas, formed after entrapment of the larvae in the retinal capillaries. *Toxocara* chorioretinitis is difficult to clinically differentiate from malignant tumours, with the result that many patients have undergone unnecessary eye enucleation (Woodruff, 1970; Seah et al, 1975; Glickman and Schantz, 1981). Of 430 *Toxocara*
infection patients in one American study, 22% had OLM with 158 cases leading to enucleation (Glickman and Schantz, 1981). More recently, in a study of 500 patients referred by ophthalmologists to an oncology clinic in the USA for specialist eye examination, 42% of the patients had lesions which simulated retinoblastoma and 16% were identified as cases of OLM (Shields, Parsons, Shields and Shah, 1991 cited in Kenny, MacCabe, Smith and Holland, 1995).

2.3.3.3.4 *Toxocara canis* Larvae in the Central Nervous System

*Toxocara* infection may cause neurological deficits or behaviour disorders in children (Schantz, 1991). With extremely high doses of eggs, migration to the central nervous system occurs in experimentally infected animals after the larvae have overcome the filtering mechanism of the liver (Dunsmore, Thompson and Bates, 1983). This occurs in humans too, although little is known about the consequences of these parasites accumulating in the CNS (Schantz, 1991). Hyperactivity, neuropsychological deficits and lack of motor coordination have been demonstrated in infected children (Schantz, 1991).

Several studies have shown that children with epilepsy are more likely to be infected with *Toxocara canis* than those without epilepsy (Seah *et al*, 1975). Glickman and Schantz (1981), though, have questioned whether *Toxocara* infection is a major cause of neurological disease in children. In a study of 84 epileptic and 108 non-epileptic children, epileptic children were found to have a significantly higher *Toxocara* antibody titer, but the infection did not occur more frequently in children with idiopathic epilepsy compared to children with epilepsy of known cause (Glickman and Schantz, 1981). In addition, epileptic children are known to have mental retardation, hyperactivity and pica associated with their condition that would increase their likelihood of ingesting the eggs (Glickman and Schantz, 1981).

2.3.3.3.5 *Toxocara canis* as a Virus Vector

It has been suggested that *Toxocara* larvae may occasionally be responsible for carrying viruses and other micro-organisms from the intestines to the blood vessels (Woodruff *et al*, 1966;
Woodruff, 1970). *Toxocara* skin tests of people who had a history of poliomyelitis and a matched control group showed that positive reactions were much more common in those who had previously been affected with poliomyelitis (Seah *et al.*, 1975; Glickman and Schantz, 1981).

**2.3.3.3.6 Incidence of Toxocara canis Infection in Humans**

Cook (1989) considered toxocariasis to probably be a very common disease. Most diagnoses are made in childhood (especially between the ages of 1 and 3 years), although cases with eye involvement do not generally present until later (Baxter and Leck, 1984; Schantz, 1989). Of 780 well documented cases of toxocariasis in the US, 56% of the patients were less than 3 years old and only 18% were adults (Glickman and Schantz, 1981).

In England and Wales, the estimated annual number of new *Toxocara* infections is about 16 000 (Baxter and Leck, 1984). Between May 1982 and 1983, Baxter and Leck (1984) reported strongly positive ELISA results in 73 people from the *Toxocara* Reference Library in London. Another 198 showed evidence of light current or past infection. In comparison, for the 10-year period 1975-1984, only 107 clinical cases were reported in England and Wales (Cook, 1989). The disease is not notifiable in the U.K., so data relating to the number of clinical cases are probably underestimates of the true prevalence. Overall, Woodruff (1970) estimated the incidence of *Toxocara* infection in adults in Britain to be 2%. This correlated with an overall prevalence of approximately 12% in the canine population (Woodruff, 1976; cited in Baxter and Leck, 1984).

In Malaysia, by comparison, the seroprevalence of toxocariasis in humans ranges from 10.9% to 35.5% (Lokman Hakim, Thadasavanth, Raden Shamilah and Yogeswari, 1997). In Venezuela, another tropical environment, the seroprevalence of *Toxocara* infection reaches 30% for 4-5 years old children living in urban slum areas, compared with 1.8% in people from the medium-
high socioeconomic sector of the population (Lynch, Hagel, Vargas, Rotundo, Varela, Di Prisco and Hodgen, 1993).

In the U.S., there is an estimated 750 cases of ocular larval migrans each year (Harvey et al., 1991). Seroprevalence varies from 4.6 to 7.3% in different geographic regions with the rate approaching 30% among African American children aged 6-11 years of low socioeconomic status (Schantz, 1991; Harvey et al., 1991).

In Australia, one serological survey (using ELISA tests) of healthy blood donors from the Australian Capital Territory found over 7% to have elevated levels of antibodies reacting to *T. canis* antigen (Nicholas, Stewart and Walker, 1986). Thirty two percent of Australian Aboriginal people from the Northern Territory had evidence of infection, which reflects the relative increased exposure to infection in this group (Currie, 1995). In one study of a coastal New South Wales Aboriginal community, 52% of people of all ages had serological evidence of exposure to *T. canis* (Jenkins et al., 1996). Despite the increased exposure of Aboriginal people to *Toxocara*, Currie (1995) refers to only one case of confirmed *Toxocara* infection in the Northern Territory where the majority of people are Aboriginal. As the symptomatology associated with toxocariasis tends to be non-specific and diffuse, it is likely that many infections are not diagnosed (Lynch et al., 1993). *Toxocara* infection in Australia is not notifiable, so the true prevalence remains unknown.

### 2.3.4 *Echinococcus granulosus*

Hydatidosis is considered to be one of the most significant canine zoonoses in the world owing to the severity of infection in humans (Dunsmore and Shaw, 1990). *Echinococcus granulosus* is not thought to be indigenous to Australia, according to Aboriginal accounts, but instead is thought to have been introduced with imported herds of sheep (Basedow, 1932). Others believe the parasite was introduced with infected dingoes up to 4000 years ago (Schantz, Chai, Craig, Eckert, Jenkins, Macpherson and Thakur, 1995).
The taeniid parasite relies on a canid as the definitive host with numerous herbivores and omnivores acting as potential intermediate hosts in which the metacestode stage resides as a hydatid cyst. The cycle is perpetuated by canids ingesting hydatid cysts from the viscera of infected intermediate hosts. Infection of humans is commonly after handling dogs with infected faeces on their skin and hair (Cook and Clarkson, 1971; Craig, Gasser, Parada, Cabrera, Parietti, Borgues, Acuttis, Agulla, Snowden and Paolillo, 1995; Miller, 1995). Occasionally infection of humans can occur after eating food which has been contaminated by flies, cockroaches or mice, which can act as transport hosts (Cook and Clarkson, 1971; Craig et al, 1995; Miller, 1995).

*Echinococcus* worm burdens are overdispersed in the canine population (Craig et al, 1995). Up to 70,000 worms have been recovered from a dog fed a single hydatid cyst (Cook, 1989) but the mean worm burden in dogs is usually 200 – 300 per dog (Craig et al, 1995).

There are at least 7 genetically distinct populations (‘strains’) within the *Echinococcus granulosus* species complex which vary in their ability to infect intermediate hosts and hence account for local differences in patterns of transmission and public health significance (Schantz et al, 1995). In Australia, two patterns of transmission have been described; one infecting domestic dogs and sheep and another infecting dingoes and macropods (Schantz et al, 1995), although PCR/RFLP analysis (Bowles and McManus, 1993) suggests that parasites of both are identical (sheep ‘strain’). This indicates that both macropods and sheep are a potential source for human infection via canines.

**2.3.4.1 Clinical manifestations of Infection with *Echinococcus granulosus* in Humans**

Ingested oncospheres develop into hydatid cysts in humans. Human infection with hydatid cysts may manifest clinically due to; the cysts causing pressure and resultant damage to surrounding tissue; complications caused by spontaneous or traumatic rupture of cysts; or immunological reactions such as asthma and anaphylaxis (Ammann and Eckert, 1995). Clinically, up to 65% of cystic echinococcosis can be asymptomatic (Ammann and Eckert,
1995) and are only discovered during routine investigations (e.g. chest radiography) or at post mortem examination (Cook, 1989). In 60% of cases the liver is involved, followed by the lungs (25%) and other organs (Ammann and Eckert, 1995). The majority of cysts are solitary (Cook, 1989). Most presentations occur in young adults (Cook, 1989), however, morbidity due to an *E. granulosus* cyst may already occur in children less than 1 year old (Ammann and Eckert, 1995). Rupture of cysts, either associated with trauma or surgery, can produce peritonitis with seeding of protoscolices throughout the peritoneal cavity. An anaphalactoid reaction with shock and death is a rare occurrence after cyst rupture (Cook 1989).

### 2.3.4.2 Prevalence of *Echinococcus granulosus*

In Britain, the source of infection with hydatids is generally a farm dog that has been allowed to scavenge sheep carcasses (Baxter and Leck, 1984). Between 1975 and 1985, 103 laboratory reports of human echinococcosis were made in England and Wales with 42 registered deaths (Galbraith and Barrett, 1986). Craig *et al* (1995) cite a maximum incidence of 7 per 100,000 people per year in the worst affected area of South Wales which is low compared to many other countries.

In Northwest Kenya (Turkana district) the incidence in people reaches 200 per 100,000 (Craig *et al*, 1995). Uruguay (population 3 million) is also endemic for hydatids with an estimated 500 patients undergoing surgery each year (Craig *et al*, 1995).

In Australia, the incidence of hydatidosis in Aboriginal people ranges from 6.9 cases to 1.4 cases per 100,000 (Jenkins and Andrew, 1993). Hydatid infection is about 15 times more prevalent in the Aboriginal population than non-Aboriginal population of Western Australia (Moodie, 1977). As often occurs with notifiable diseases, accurate figures on the number of new cases of hydatidosis each year are limited, as the disease is notoriously under-reported (Jenkins and Andrew, 1993). As such, the present figures fail to accurately represent the actual situation.
No evidence of infection of dogs in the Kimberley region has been reported. Thompson et al (1993a) did not recover Echinococcus from 188 post mortem dogs in Fitzroy Crossing, although cattle are often infected with hydatid disease in this area. It is most likely that a wild animal cycle, probably involving dingoes and macropods, exists to maintain the cycle with cattle acting as accidental hosts (Thompson et al, 1993a).

2.3.5 Bacterial Zoonoses

2.3.5.1 Salmonella Species

There is estimated to be around 2000 Salmonella serovars (serotypes) (Baxter and Leck, 1984; Pelzer, 1989). In one report from Great Britain in 1982, 13 serotypes of Salmonella were represented amongst 30 isolations from dogs (Baxter and Leck, 1984). As in other animals, S. typhimurium accounted for approximately half of these isolations (Baxter and Leck, 1984). All Salmonella infections, except those of S. typhi, S. paratyphi A and S. paratyphi C (typhoid fever), are considered zoonoses (Pelzer, 1989).

2.3.5.1.1 Salmonella infection in Humans

Salmonella of animal origin causes an intestinal infection in humans characterised by a sudden onset (8 to 48 hours after exposure) of fever, myalgia, cephalalgia, abdominal cramps, nausea, vomiting and diarrhoea (Williams, 1980). Dehydration and electrolyte imbalances may be severe in the young and elderly, whereas in adults the disease is self-limiting in 2 to 4 days with full recovery in about a week (Pelzer, 1989).

Salmonella may also cause localized infection in any organ or tissue of the body (Pelzer, 1989). This syndrome is usually in newborns, infants or those having concurrent underlying disease (Pelzer, 1989).

Bacteraemia or septic syndrome is another manifestation of salmonellosis and may follow gastroenteritis. S. choleraesuis is the most commonly isolated serotype (Pelzer, 1989).
Infants less than 1 year old and the elderly are at higher risk of infection as are people taking antibiotics that can alter the normal enteric bacterial flora that serves as a protective mechanism against Salmonella (Pelzer, 1989). Infective dose, serotype, age, immune status and the presence or absence of other diseases all have an influence on the susceptibility to infection (Pelzer, 1989).

2.3.5.1.2 Prevalence of Salmonella Infection in Humans

The Centres for Disease Control in America estimate that only 1 to 5% of an annual 1 to 5 million Salmonella infections is reported (Potter, 1992). Baxter and Leck (1984) considered that given that dogs account for 1% of animal isolations in England and Wales, it would not be surprising if they were also the source of infection in a similar proportion of human cases. This would imply that there are about 2500 human cases/year infected from this source in the U.K. (Baxter and Leck, 1984). The risk of acquisition of Salmonella from dogs may be greater in childhood enteritis (Baxter and Leck, 1984).

2.3.5.1.3 Sources of Salmonella Infection for Humans

Infection in humans is generally by oral consumption of faeces containing Salmonella or feedstuffs contaminated with Salmonella organisms (Williams, 1980). Pork, beef, poultry, eggs and milk products are commonly involved in disease outbreak amongst people (Williams, 1980). Some surveys in America have found Salmonella contamination rates of 35.2% in poultry, 1% in beef and 12% in pork (Houston, 1987).

Oral shedding of Salmonella spp. is believed possible in dogs and could result in spread to humans that are licked in the face (Willard, Sugarman and Walker, 1987; Pelzer, 1989).

2.3.5.1.4 Dogs as Sources of Salmonella Infection for Humans

Pets that are in close contact with people are a possible source of infection (Morse and Duncan, 1975; Blaser, Cravens, Powers and Wang, 1978; Elliot, Tolle, Goldberg and Miller, 1985;
Willard *et al.*, 1987; Pelzer, 1989; Currie, 1995). Although transmission is possible, direct canine-human transmission has been reported in relatively few cases of salmonellosis (Morse and Duncan, 1975; Currie, 1995) and may be less common than in *Campylobacter* enteritis (Baxter and Leck, 1984). Even so, Pelzer (1989) believes it is difficult to assess the true risk of acquiring infection resulting in disease from pets because the number of organisms isolated from infected dogs is not reported in surveys and the infective dose to humans is difficult to define (Pelzer, 1989).

### 2.3.5.1.5 Prevalence of *Salmonella* Infection in Dogs

The average rate of *Salmonella* infections in dogs surveyed from 1947 to 1965 in the United States was 14% (Williams, 1980). Other reports from the U.S.A. cite prevalences of 0.8% to 36.5% (Morse and Duncan, 1975; Baxter and Leck, 1984; Pelzer, 1989). Often too, the serotypes isolated were similar to those affecting human beings (Elliot *et al.*, 1985). In Alaska, 16% of dogs had *Salmonella* spp. isolated from rectal swabs (Pelzer, 1989). British prevalence surveys have isolated *Salmonella* from the faeces of between 0.5 to 1% of all dogs (Baxter and Leck, 1984).

Younger dogs (less than 6 months old) may have higher prevalences than older dogs and are thus of increased transmission risk to humans (Blaser *et al.*, 1978; Pelzer, 1989). They are also more likely to develop clinical disease as are old or malnourished dogs, pregnant bitches and animals with concurrent infections (Pelzer, 1989). Surveys in the United States have also found that sick dogs are more likely to be infected with multiple serotypes (Morse and Duncan, 1975), whereas dogs with single serotypes appear symptom free (Baxter and Leck, 1984).

### 2.3.5.1.6 Sources of *Salmonella* Infection for Dogs

High stocking densities and overcrowded facilities such as kennels contribute to the spread of organisms and are usually associated with disease outbreaks in domestic animals (Pelzer, 1989). Principle sources of *Salmonella* for dogs include faecally contaminated water, undercooked or
raw contaminated meat, carrion, and saliva from other dogs that are oral shedders (Williams, 1980; Baxter and Leck, 1984; Willard et al 1987). In the past, contaminated meat was common in Britain with up to 50% of meat contaminated in 1952, but since meat regulations have been introduced, contamination levels are much lower (Baxter and Leck, 1984). Parallel studies of infection rates in dogs have not been conducted, but in Aboriginal communities, it is not uncommon for dogs to eat raw meat that has not been hygienically dressed. This would undoubtedly increase the risk of infection for dogs and potentially their owners. All meat should be cooked as if used for human consumption if it is to be fed to dogs (Williams, 1980).

### 2.3.5.2 Campylobacter species

Campylobacteriosis in humans has been known for almost 2 decades. The disease in humans equals or exceed salmonellosis as a cause of bacterial diarrhoea (Skirrow, 1990).

Campylobacters, small, spiral, microaerophilic gram negative rods (Skirrow, 1990), are capable of causing enteritis in a variety of animals. *C. jejuni* and *C. coli* are two closely related species of which there are more than 100 serotypes. A third species, *C. upsaliensis*, is also enteropathogenic and is a known cause of bacteraemia of immunocompromised children and adults (Goosens, Vlaes, De Boek, Pot, Kersters, Levy, De Mol, Butzler and Vandamme, 1990; Hanna, Enbom and Murphy, 1994). *C. jejuni*, though, is the most predominant species (80-90%) in most parts of the world (Skirrow, 1990).

#### 2.3.5.2.1 Campylobacter Enteritis in Humans

Transmission of *C. jejuni* occurs by the faecal-oral route through contaminated food and water or by direct contact with faecal material from infected animals or people (Blaser and Reller, 1981). Most campylobacters produce a cholera-like enterotoxin with one or more cytotoxins and as few as 500 organisms are capable of infecting humans (Willard et al, 1987).
In humans, campylobacteriosis is characterised by an acute enterocolitis with an abrupt onset of illness characterised by fever, abdominal pain, and diarrhoea (Blaser and Reller, 1981). In some cases there may be headache, malaise, myalgia, arthralgia, nausea or vomiting (Blaser et al, 1978; Prescott and Munroe, 1982; Skirrow, 1990). The illness is self-limiting with almost all patients recovering within a week (Blaser and Reller, 1981) although some will have relapsing or chronic illness (Willard et al, 1987; Fox, Moore and Ackerman, 1983; Skirrow, 1990). Humans may also have asymptomatic shedding (Skirrow, 1990).

Complications such as reactive arthritis are uncommon (1% of patients, 2 weeks after onset of illness). Guillain-Barre syndrome (peripheral neuropathy) is a less common, but more serious complication (Skirrow, 1990).

2.3.5.2.2 Campylobacteriosis in Dogs

Woldehiwet, Jones, Tennant and Jones (1990) considered that the role of Campylobacter spp. as enteropathogens in animals is not well established. Malik and Love (1989) concluded that campylobacters are a normal part of the intestinal flora and McOrist and Browning (1982) too believe that a proportion of healthy dogs are symptomless carriers. Campylobacters may thus have a secondary opportunistic role rather than primary enteropathogenic role in some diarrhoeic dogs. Changes to the intestinal flora, as occurs following stress such as unfamiliar housing, transport and poor diet, are considered to be triggers for increased faecal shedding of campylobacters (Malik and Love, 1989).

2.3.5.2.3 Incidence of Campylobacter Infection in Humans

The annual incidence of campylobacteriosis in humans in England and Wales is 80/100 000 (Skirrow, 1990). In Europe, Africa and North America Campylobacter has been found to be present in 3-8% of cases of diarrhoea (Blaser et al, 1978). The rate of isolation of the organism from healthy people has been 0-1.3% (Blaser et al, 1978). All of these figures are considered to be an underestimate as the frequency of testing has increased in recent years (Skirrow, 1990).
The actual number of infections is probably about 10 times the reported number (Skirrow, 1990).

In developing countries, exposure to the organism is considered sufficiently high for immunity to be gained early in childhood (Fox et al., 1983). Symptomless carriage of C. jejuni is not uncommon in children living in communities where standards of living and hygiene are poor (Berry and Gracey, 1981a; Blaser and Reller, 1981). In one isolated Kimberley community, Berry and Gracey (1981a) found a high prevalence of C. jejuni in children without diarrhoea with an overall prevalence of 23% in the dry season. Studies in South Africa and Bangladesh similarly found a high prevalence with 40% of children 9-24 months of age excreting C. jejuni (Blaser and Reller, 1981). This high rate of Campylobacter infection in clinically normal people makes it difficult to implicate the organism as cause of diarrhoea (Blaser and Reller, 1981; Prescott and Munroe, 1982; Fox et al., 1983).

2.3.5.2.4 Prevalence of Campylobacter Infection in Dogs

The prevalence of Campylobacter in normal adult dogs ranges from 1.6% (Prescott and Munroe, 1982) to 34% (Fox et al., 1983; Willard et al., 1987). Underrepresentation due to the difficulty in isolating the organism (Skirrow, 1990) and the ability for asymptomatic carriage makes the true potential for dogs to act as zoonotic hosts difficult to assess.

Generally, puppies less than 6 months old are more likely to carry C. jejuni than adult dogs (Willard et al., 1987).

2.3.5.2.5 Dogs as Sources of Campylobacter Infection for Humans

In the home, infection from contact with puppies with Campylobacter diarrhoea is well documented (Blaser et al., 1978; Blaser, Taylor and Feldman, 1983). Even so, less than 5 - 10% of human campylobacteriosis is suspected of originating from companion animals (Prescott and Munroe, 1982; Willard et al., 1987). Other sources include contaminated water and food. Wild
birds are considered the principal reservoir of infection for domestic animals and animals for food (Willard et al, 1987) which in turn infect humans.

Woldehiwet et al (1990) typed C. jejuni from 37 canine samples and found 41.1% belonging to pen 2 and 4 serotypes. Types pen 2 and 4 are also most common in humans with Campylobacter enteritis (Woldehiwet et al, 1990) which suggests that dogs are a potential source of human infections. Campylobacter from dogs are especially considered a risk for susceptible individuals, such as infants (Malik and Love, 1989). Dogs have also been cited as a probable cause of infection of humans with C. upsaliensis (Goosens, Vlaes, Butzler, Adnet, Hanicq, N’Jufom, Massart, de Schrijver and Blomme, 1991; Figura, 1991).

Hanna et al (1994) considered the close affinity between Aboriginal people and their dogs as an explanation for the high rates of Campylobacter infections in Aboriginal children. Berry and Gracey (1981) were unable to isolate campylobacters from children at a community where dogs were not allowed but found overall rates of 21% from a nearby community with dogs. Hanna et al (1994) concluded that “programs that focus on ‘dog health’ may reduce the burden of Campylobacter and some other enteric infections experienced by Aboriginal children”.

2.3.6 Other Canine Zoonoses

There are several other canine zoonoses, confirmed or potential, which can affect humans. Some of these are reviewed in the following sections.

2.3.6.1 Nematode Zoonoses

2.3.6.1.1 Strongyloides stercoralis

Strongyloides stercoralis is not strictly host specific (Genta and Grove, 1989) and opinion has been divided as to whether S. stercoralis can inhabit the small intestine of dogs (Cook, 1989). Although the parasite can infect both humans and dogs, it seems likely that different strains of
the parasite vary in both their infectivity and pathogenicity to each host (Stevenson and Hughes, 1988).

In general, dog-human or dog-environment-human transmission is considered rare (Genta and Grove, 1989) with only a few reports of dogs being the source of infection (Georgi and Sprinkle, 1974). All the same, *Strongyloides* is endemic in northern Australian Aboriginal communities (Fisher, McCarr and Currie, 1993; Prociv and Luke, 1993) and is occasionally found in dogs inhabiting these areas (Dunsmore and Shaw, 1990; Stevenson and Hughes, 1988).

### 2.3.6.12 *Trichuris vulpis*

The dog whipworm can cause human infection (Jenkins and Andrew, 1993), but its importance is unclear (Cook, 1989). In some areas of the world *T. vulpis* is very common. A prevalence of up to 52% in stray dogs in the US has been reported (Cook, 1989). In New Jersey the prevalence of *T. vulpis* in dogs was found to be 38% (n=2737) (Kenney and Yermakov, 1980).

Kenny and Yermakov (1980) documented one case of an adult *T. vulpis* isolated from histopathologic sections of an appendix in a post mortem examination in New York. There were no signs associated with the infection (Kenney and Yermakov, 1980). *T. vulpis* has also been reported in humans in Europe, but has not been reported in Australia thus far (Jenkins and Andrew, 1993). Thompson *et al* (1993a) did not find any dogs to be infected with *T. vulpis* in the Fitzroy Crossing area of the Kimberley Region although the parasite is quite ubiquitous with the eggs remaining viable at temperatures as low as 4.4°C and as high as 44.5°C (Kenney and Yermakov, 1980).

### 2.3.6.13 *Dirofilaria immitis*

*Dirofilaria immitis* infection in dogs in tropical climates is very common, although Thompson *et al* (1993a) failed to demonstrate heartworm infection in their study in Fitzroy Crossing. Transmission of L3 larvae to humans by mosquitoes (as occurs in dogs) is possible (Cook,
1989) and the exposure rate of Aboriginal people to *D. immitis* is almost 10 times higher than in Caucasians (Jenkins and Andrew, 1993). After infection in humans, partial development of the helminths occurs in the right ventricle, from which they are swept into small pulmonary arteries (Cook, 1989). The larvae do not develop any further and the consequent lesions are usually limited to the lung periphery. These ‘coin lesions’ consist of a single coiled (usually necrotic and occasionally calcified) worm in the lumen of an artery within a necrotic area (Miller, 1995). Clinically, chest discomfort, cough, haemoptysis, fever, chills and malaise may be present and the lesions are usually detected by radiography. Owing to their radiographic similarity to primary and secondary neoplasia, they are often removed at surgery (Miller, 1995).

### 2.3.6.2 Cestode Zoonoses

#### 2.3.6.2.1 *Dipylidium caninum*

*Dipylidium caninum* relies on an intermediate host (lice or fleas) to harbour the metacestode stage of its lifecycle. The definitive hosts, dogs and cats, become infected by ingesting infected fleas (*Ctenocephalides* spp.) or lice (*Trichodectes* spp.). The ingested cysticercooids develop into adult tapeworms in the small intestine. Infection in humans is by the adult tapeworm after infected fleas are accidentally ingested. People licked around the mouth by dogs that have recently nipped infected fleas are also at risk of infection. Human infections are not common and are almost confined to children (Baxter and Leck, 1984; Cook, 1989; Miller, 1995). The clinical manifestations of infection in humans are non-specific and can include anal irritation, diarrhoea and unsettled behaviour (Baxter and Leck, 1984).

*Dipylidium caninum* was not found in any dogs (or cats) in Fitzroy Crossing although *Trichodectes* was common on dogs examined (Thompson *et al*, 1993a).
2.3.6.3 Protozoal Zoonoses

2.3.6.3.1 *Giardia duodenalis*

In Australia, *Giardia* is considered to be the most important parasitic disease in terms of human morbidity and is one of the most common causes of diarrhoea in children with a failure to thrive (Swan and Thompson, 1986). Although the main features of infection with the protozoan are diarrhoea and malabsorption, some animals (and humans) may remain symptomless (Casemore, 1990).

In the Kimberley region, *Giardia duodenalis* is a very common parasite in children. In one study by Gunzburg *et al.* (1992) of 555 Kimberley children up to 5 years of age, *Giardia* was so highly endemic that more than half of the children were infested at any one time.

Meloni, Thompson, Hopkins and Reynoldson (1993) considered that the high prevalence of *Giardia* infections in Aboriginal communities, poor sanitation and hygiene levels favour cross-transmission and reinfection between individuals. This makes control of the parasite difficult.

*Prevalence of Giardia duodenalis in Canine Populations*

Thompson *et al.* (1993a) found 16.5% of 282 dogs from inland Kimberley region communities to be shedding *Giardia* cysts. Comparatively, an earlier study found the prevalence amongst dogs from Perth to be 21% (n=333) (Swan and Thompson, 1986). Even higher rates were found in dogs from refuge shelters (30%) (Swan and Thompson, 1986). Seah *et al.* (1975) only found 0.8% of dogs to be shedding cysts in their Montreal study and concluded that this was no higher than the rates found amongst humans. In Pennsylvania, 7.2% of dogs were infected, which is also lower than in Western Australia (Kirkpatrick, 1988).

*Inter Species Transmission of Giardia duodenalis*

Humans and a variety of lower animals naturally share this parasite (Schantz, 1991), but strain variation may prevent cross transmission between host species. *G. duodenalis* is
morphologically indistinguishable from *Giardia* isolates originating from several species of domestic and wild mammals (Swan and Thompson, 1986; Eckert, 1989). At least 9 different animal species, including dogs, have been successfully infected with cysts of *Giardia* isolated from different species, including humans (Swan and Thompson, 1986; Schantz, 1991). Recent studies using molecular techniques have also shown similarities of strains of *Giardia* isolated from humans and animals (Meloni, Lymbery and Thompson, 1989), although comparison of SSU-rRNA sequences of *Giardia* from dogs and people residing at the same location in the Kimberley Region have revealed differences (Hopkins, Meloni, Groth, Wetherall, Reynolds and Thompson, 1997). This latest work suggests that dogs are not a significant source of infection for humans in the Kimberley region.

2.3.6.3.2 *Entamoeba* species

*Entamoeba histolytica* is the cause of amoebic dysentery in humans (Dunsmore and Shaw, 1990) usually associated with poor hygiene and poor water quality (Casemore, 1990). Opinion is divided as to whether *E. histolytica* is of zoonotic significance. Casemore (1990) stated that there is no known animal reservoir for *E. histolytica*, although other host species can be infected, whereas Cook (1989) considered the dog to be one of several mammals that can maintain a reservoir of the protozoan. Dunsmore and Shaw (1990) suggested that dogs are not a source of infection for humans or other dogs because they pass only the trophozoites. The resistant cysts are the infective stage for both species and not the trophozoites. Regardless, all agree that the dog is of minor significance compared with human-human transmission.

*Entamoeba coli* is a common non-pathogen of Aboriginal people (Meloni et al, 1993). *Entamoeba coli* has also been isolated from dogs in Aboriginal communities of Fitzroy Crossing where they may be acting as reservoirs for infection. Overall, infection may indicate poor hygienic conditions and the potential for transmission of pathogenic species (Casemore, 1990).
### 2.3.6.3.3 Cryptosporidium species

Diarrhoea produced by *Cryptosporidium* is self-limiting in normal hosts (Schantz, 1991; Miller, 1995) and probably results in the development of protective immunity (Casemore, 1990; Current, 1994). In immunocompromised individuals the organisms can cause life-threatening disease for which there is no satisfactory chemotherapy (Schantz, 1991). Likewise, infections are more prevalent and severe in young children (Current, 1994).

There are at least 4 confirmed species of *Cryptosporidium* - 2 in mammals (*C. muris* and *C. parvum*) and 2 in birds (*C. meleagridis* and *C. baileyi*), which have been isolated from at least 40 host species (Casemore, 1990). Most human infection is probably due to *C. parvum* which is also common in livestock (Casemore, 1990). Dogs too are occasionally infected but do not seem to be an important source of the organism (Cook, 1989; Casemore, 1990). Schantz (1991) states that there is a lack of evidence of direct transmission from animals although there seems to be little host specificity. Generally it is agreed that *Cryptosporidium* may find a reservoir in dogs. This area is still being investigated by molecular techniques.

### 2.3.6.4 Bacterial Zoonoses

#### 2.3.6.4.1 Pasteurella species

*Pasteurella multocida* is a small Gram-negative coccobacillus present as a normal commensal of the oral flora of the dog (Baxter and Leck, 1984). The bacterium has been isolated from 55% of healthy London dogs (Cook, 1989) as well as 80% of dogs in a closed colony in the U.S. (Baxter and Leck, 1984).

*P. multocida* may give rise to three types of disorders in humans; local wound infections, respiratory infections and septicaemia (Baxter and Leck, 1984).

Wound infections (mostly due to dog bites) are the most common with 87% of isolations in one study from skin lesions, 8% from respiratory tract and mouth, and 4% from blood and sites that
may be infected via the blood (Baxter and Leck, 1984). Even when infections are not acquired from wounds, there is often a history of causal exposure to an animal from which the patient could have been infected by inhalation (Baxter and Leck, 1984).

It is estimated that of 209,000 dog bites that are presented to hospital each year in England and Wales, over 31,000 of these cases must have been complicated by *P. multocida* infections (Baxter and Leck, 1984). In the US, about 500,000 people (mostly children) are treated for animal bites each year by emergency departments (Olson, Nett, Bowen, Amann, Sawyer, Gorell, Niswender, Pickett and Phemister, 1986a). The actual incidence of bites is believed to be closer to 2 million (Olson et al, 1986a).

A less common organism present in dogs is *P. pneumotropica*, which seems to affect the lungs in a higher proportion of cases than *P. multocida* (Baxter and Leck, 1984; Cook, 1989).

### 2.3.6.4.2 Yersinia species

The principal reservoirs of infection of the two *Yersinia* species, *Y. pseudotuberculosis* and *Y. enterocolitica*, appear to be wild rodents, birds, and pigs (Baxter and Leck, 1984). Yersiniae have been isolated from healthy dogs (Cook, 1989) at the rates of 2% (*Y. pseudotuberculosis*) and 6% (*Y. enterocolitica*) from stray and unwanted dogs in Tokyo (Baxter and Leck, 1984).

Human yersiniosis is presumably acquired by ingestion and is primarily a gastrointestinal infection (Baxter and Leck, 1984) which may result in an ‘appendicitis-like’ abdominal pain seen in late childhood and adolescence (Cook, 1989). The main vehicle of infection has not been established, but dogs and cats are believed to play an important part in the transmission of *Y. pseudotuberculosis* to humans (Baxter and Leck, 1984; Cook, 1989).
2.3.6.4.3 Other Gram Negative Bacteria

Other Gram-negative organisms claimed to be zoonotic and which have been isolated from dogs, but from which there is only a low risk, are *Pseudomonas* spp., *Proteus* spp., *Klebsiella* spp., *Enterobacter aerogenes*, *Haemophilus* spp. and *Serratia rubidea* (Cook, 1989).

2.3.6.4.4 Other Gram Positive Bacteria

In one study from America, the most common canine zoonotic Gram-positive organism was considered to be *Staphylococcus aureus* (Cook, 1989). A case in which severe 'impetigo' in a six years old child resulted from infection by a puppy with severe facial pyoderma has been recorded (Cook, 1989).

Dogs are also considered to be a reservoir for Group A beta haemolytic *Streptococcus* (Cook, 1989). Group A beta haemolytic *Streptococcus* has been responsible for persistent or recurrent streptococcal throat infections, otitis externa, systemic bacteraemia, rheumatic fever, or post streptococcal glomerulonephritis (Miller, 1995). Humans are the natural reservoir for the bacteria, but dogs can harbour the bacteria in the oral pharynx (Miller, 1995). One report documented a family which suffered from recurrent pharyngitis until the pet dog was treated and cleared of the organism (Cook, 1989).

2.3.6.5 Mycotic Zoonoses

2.3.6.5.1 Dermatophytes

The three genera of fungi responsible for this superficial infection of keratinised tissues are *Microsporum*, *Trichophyton* and *Epidermophyton* (Baxter and Leck, 1984). These dermatophytes include species that are anthropophilic, zoophilic and geophilic (Baxter and Leck, 1984). The commonest zoophilic species found in dogs in Britain is *M. canis* (Baxter and Leck, 1984).
*M. canis* seems to occur less often and causes more severe lesions in dogs than in cats, suggesting that cats are the primary host. *T. mentagrophytes* may cause up to 36% of canine cases (Baxter and Leck, 1984). In one series of 3521 human cases that occurred in south west England in 1960-1970, the species isolated were anthropophilic in 73.3%, geophilic in 3% and zoophilic in 26.5% of the cases (Baxter and Leck, 1984). Considering that 4.7% of these cases were due to *M. canis* and that *M. canis* accounts for less than two thirds of canine cases, the overall proportion of cases of human ringworm that is acquired from dogs may be in the order of 3% (2% due to *M. canis* and 1% due to other species).

Dermatophyte spores can survive for many months or years on keratinised tissue in the environment, so infection can be spread indirectly as well as directly. Transmission between dogs and from dogs to humans can occur quite easily (11 humans and 2 dogs were infected from 3 infected dogs in one case in London) (Baxter and Leck, 1984). Children may be especially susceptible (Baxter and Leck, 1984).

As many as 30% of human ringworm cases in an urban setting in the US have been associated with direct animal contact (Cook, 1989).

### 2.3.6.6 Viral Zoonoses

Apart from rabies, few human virus infections have been traced to a canine source; this may represent a lack of recognition or under-reporting (Cook, 1989). The source of an outbreak of calicivirus-associated acute gastroenteritis affecting 17 residents of an elderly peoples’ home at Exeter, England, was suspected to be an infected dog (Cook, 1989). Caliciviruses had incidentally been well recognised as a cause of acute enteritis in animals before they were established to be human pathogens (Cook, 1989).
2.3.6.7 Rickettsial Zoonoses

2.3.6.7.1 Coxiella burnetii

In Nigeria, a prevalence rate of *Coxiella burnetii* in pet dogs of about 30% has been recorded. The relevance of this to human disease remains unclear, but might well be significant (Cook, 1989). Some authors consider the cat to be a more important source of infection to humans with potential infections occurring through handling placental and fetal tissues (Miller, 1995).

2.4 Control Measures to Reduce Canine Zoonoses

2.4.1 The World Situation

"Human health is to a considerable extent influenced by the health condition of animals,... that may be close to people in urban or village settings" (World Health Organisation, 1978).

Throughout the world, zoonoses associated with companion animals remain of importance; from industrialized countries where *Toxocara* infection is still considered a major zoonosis, to developing countries where life-threatening diseases such as rabies are of national concern. The World Health Organisation (1978) considers that the most common risks of zoonoses are from animals which often live as "members of the family". As such, a number of guidelines have been formulated to help reduce the risk of zoonoses in urban areas.

2.4.2 Methods to Control Zoonoses

(a) *Reduce the numbers of uncontrolled, ownerless dogs*

In most countries there is considered to be an overpopulation of dogs (World Health Organisation, 1978) and zoonoses are particularly difficult to control in roaming animal populations (Olson *et al*, 1986a). Approximately 3.9 to 5.9 million dogs are euthanased each year in the U.S., where there is an estimated 52 million dogs (Schantz, 1991; Nassar and Fluke, 1991). Likewise, many die from exposure, starvation or trauma (Olson *et al*, 1986a; Rollin, 1991). Most of the uncontrolled dogs are in fact stray pets (Nassar, Mosier and Williams, 1984).
Excessive measures have been taken in some countries to control the overpopulation problem. In China, private ownership of dogs was banned from 1983 in Peking for reasons of health and safety (Olson, et al, 1986a). Extermination teams in Peking drowned or clubbed to death 200 000 dogs by December 1983 (Olson et al, 1986a). Comprehensive animal legislation, enhanced public awareness of the causes of unwanted pets, and efforts by humane societies and veterinarians toward encouraging owners to neuter pets resulted in 30% fewer dogs impounded by humane organisations between 1972 and 1982 in America (Olson et al, 1986a).

Other problems associated with large, uncontrolled dog populations include; noise pollution from excessive barking, injury and death of wildlife from hunting dogs, increased potential for traffic accidents when dogs wander uncontrolled on roads and, the potential for free roaming dogs to act as reservoirs for exotic diseases, such as rabies in Australia (Robertson, Wilks and Williamson, 1996).

(b) *Construct garbage disposal facilities to keep out animals*

In Aboriginal communities this is of particular concern as refuse disposal is often the responsibility of the local community council. In areas with inadequate infrastructure, garbage disposal is lax and dogs are often reported for disturbing rubbish. This increases the potential for pathogen transmission, especially when articles such as disposable nappies are exposed.

(c) *Avoid feeding raw meat or offal to dogs.*

As mentioned, dogs are potential reservoirs for numerous gastrointestinal bacteria such as *Salmonella* and *Campylobacter* that are often acquired from eating contaminated raw meat or offal. Some parasites are also disseminated in this fashion, such as *Echinococcus*.

(d) *Keep dogs free from helminth parasites by anthelmintic treatment of pregnant females and pups*

Many developed countries have municipal bylaws enforcing the adequate treatment of dogs for endo- and ecto-parasites. As is the case with other regulations regarding animal control,
compliance is almost nonexistent with respect to endoparasitic diseases (Seah et al., 1975). The World Health Organisation (1978) also encourages regulations to prevent the fouling of pavements with dog faeces and urine. In addition, it is recommended that dogs should be excluded from playgrounds (World Health Organisation, 1978). Many city governments face the problem of inadequate removal of animal faeces (Olson et al., 1986) and again, enforcement of these laws is difficult.

(e) Educate animal owners and the general public about the risks of zoonoses and how to avoid them

This is considered to be one of the most important measures in controlling zoonoses (World Health Organisation, 1978). Unfortunately, it is probably the hardest objective to achieve. Even in developed countries with veterinary services, education is minimal. Two surveys of practicing veterinarians in the United States in 1979 and 1989 revealed that less than a third of veterinarians explained or discussed the potential zoonotic hazards of helminths with clients (Schantz, 1994).

Control of helminths in humans revolves around health education, promotion of sanitation and environmental health and mass chemotherapy of at risk populations (Pawlowski, Schad and Stott, 1991; Migasena and Gilles, 1991). Several authors have commented on the necessity for treatment in the face of education as the results of improvements in sanitation and education are not likely in the short term (Stephenson, Latham, Kurz, Kinoti and Brigham, 1989; Bradley, Chandiwana and Bundy, 1993; Chan, Guyatt, Bundy and Medley, 1994). Control by treatment campaigns has two advantages; a reduction in the worm burden in treated individuals and a reduction in further infection of all individuals due to the overall reduction in transmission (Chan et al., 1994).
2.4.3 Dog Population and Zoonoses Control Programs for Aboriginal Communities

Dog population and zoonoses control programs in Aboriginal communities present an interesting challenge for people concerned about the problems associated with canine overpopulation and zoonoses transmission. The strong association of some Aboriginal people and their dogs as well as the general aversion to killing of dogs makes most of the current population control measures unworkable. A method commonly employed for population ‘control’ involves the shooting of excess dogs. Individuals have also been known to attempt population control by selectively killing female puppies at birth (Howe, 1993). Occasionally the administration of oral anthelmintics and ‘dipping’ of dogs in organophosphates is done for ‘control’ of parasites. Generally programs have been haphazard and often met with objection. The use of organophosphates for controlling ectoparasites also presents an occupational health risk to the operators.

At times the population control measures have not been at the request of the owners or community. In 1981, one such incident occurred at Ringers Soak, a remote community in the east Kimberley, where all of the dogs were shot in the community (without owner consent) after the people were evicted from their homes (Doyle, The West Australian, January 1981). The incident reached statewide proportions and threatened to stop discussions regarding important land rights negotiations (Anon, Kimberley Land Council Newsletter, March 1981). More recently, Nyungah families of the Karramara Reserve in Moora, Western Australia, were distressed to find 3 of their dogs shot when they returned to their camp. “If they did that to the dogs they can come out anytime and do anything to anybody” (Taylor and Taylor, Press release statement, 1997). The dogs were claimed to have been killing livestock (Townsend, The West Australian, July, 1997), but the Aboriginal people considered that they had not been adequately informed of any problems relating to their dogs.
For these reasons, informative, effective, culturally aware, welfare-conscious canine breeding and parasite control programs are needed in Aboriginal communities.

2.4.3.1 Canine Population Control

The control of dog population numbers can be done by euthanasia of excess, unwanted dogs or by breeding control of males or females or both. Euthanasia is not often a viable choice as numbers continue to increase after the population is reduced. Euthanasia is a stop gap measure and offers no advantage to either dog, owner or community and as mentioned can meet with objection. Breeding control of males is effective in situations where there are small numbers of dogs and bitches can be supervised. In the situation of large numbers of dogs with minimal supervision, breeding control of the female is more suitable as there is assurance that the female will not become pregnant regardless of the number of male dogs available.

Several methods of preventing pregnancy in bitches are possible; ovariohysterectomy, oestrus control with hormonal drugs, immunisation against hormones or ovarian/oocyte tissue (Concannon and Meyers-Wallen, 1991), administration of a GnRH (gonadotropin releasing hormone)-linked cytotoxin (to achieve permanent contraception) (Concannon and Meyers-Wallen, 1991) or, administration of gonadotropin agonists such as GnRH (Concannon and Meyers-Wallen, 1991). At the commencement of the research for this thesis, ovariohysterectomy and oestrus control with hormones were the only available reliable options.

2.4.3.1.1 Ovariohysterectomy

Ovariohysterectomy is the most safe and sure method of preventing pregnancy if it is done correctly. In addition, ovariohysterectomy offers the advantages of preventing pyometra or false pregnancy in the bitch as well as reducing the risk of mammary tumours (Evans and Sutton, 1989).
Some of the potential sequelae of ovariohysterectomies, though, include; urinary incontinence due to hypoestrogenism (Salmeri, Bloomberg, Scruggs and Shille, 1991), obesity (Salmeri et al, 1991), infantile vulva, vaginitis and perivulvar dermatitis (Concannon, 1988; Salmeri et al, 1991), hair loss and, changes in coat colour and texture (Concannon, 1988). In addition, the risk of anaesthesia and suture abscesses or postoperative complications are more serious disadvantages. Post-surgical sequelae in dogs are reported to be as high as 31% depending on the surgeon’s skill (Olsen et al, 1986a). In a situation where there is no guarantee of a sterile operating environment and where gaseous anaesthesia is not available, such as at remote communities, the risks are greater.

Ovariohysterectomies are also irreversible and as such do not offer owners a choice of allowing their bitches to breed. Hormonal control of oestrus is often suggested when there is doubt about the advisability of conducting the surgery or whilst the owners are considering whether or not to breed from their bitch (Evans and Sutton, 1989). For the purposes of controlling breeding on a large scale with a potentially high rate of change in population, hormonal control of oestrus offers the best alternative.

2.4.3.1.2 Hormonal Control of Oestrus

Hormonal preparations, apart from being reversible, can be used when the bitch is already on heat (Evans and Sutton, 1989). Other features desirable in an oestrus control compound are the ability to inadvertently use the drug during pregnancy without side effects, the ability to use the drug at any time during the oestrous cycle, a relatively long period of effectiveness, minimal progestagenic side effects and minimal masculinising effects.

Several hormonal preparations are available and all have differing features and efficacy. Types of drugs include; natural steroid hormones (progesterone and testosterone), synthetic androgens (milberone) and synthetic progestagens (medroxyprogesterone acetate, megestrol acetate and proligestone).
Natural Steroid Hormones

Progesterone has not been proven to be effective even though, in theory, its negative feedback on the production of gonadotropins at high levels should control oestrus (Evans and Sutton, 1989). In addition to this, progesterone has a short duration of action and has been associated with the induction of pyometra (Evans and Sutton, 1989).

Testosterone can also control oestrus, but requires frequent injections and has the undesirable androgenic side effects such as enlarged clitoris and vulva (Evans and Sutton, 1989). Testosterone also affects the ability of bitches to breed once medication is stopped. Testosterone implants have the same effects although they do have a longer duration of activity (up to 15-20 months) (Concannon, 1988; Evans and Sutton, 1989) which precludes the need for repeated treatments. The placement of the silastic capsules, though, does require minor surgery and they are not biodegradable (Concannon, 1988; Evans and Sutton, 1989).

Synthetic Androgens

Milberone (17β-hydroxy-7α, 17-dimethylestr-4-en-3-one) exerts an antigonadotrophic effect which can control oestrus (Evans and Sutton, 1989). It is an orally active androgenic compound and has no oestrogenic, antioestrogenic or progestagenic action (Olsen, Nett, Bowen, Amann, Sawyer, Gorell, Niswender, Pickett and Phemister, 1986b) which gives it the advantage of being free of uterine side effects (Evans and Sutton, 1989). The androgenic properties of milberone can lead to hypertrophy of the clitoris (in 15 to 20% of treated bitches) (Concannon and Meyers-Wallen, 1991) and abnormal sexual behaviour, anal gland inspissation with an obnoxious body odour and obesity (Concannon, 1988; Evans and Sutton, 1989).

Milberone must be given daily commencing at least 30 days before pro-oestrus is due (Olsen et al, 1986a; Concannon and Meyers-Wallen, 1991) and will not suppress the signs of heat or prevent conception if it is given when the bitch is in pro-oestrus or oestrus (Evans and Sutton, 1989). Also, administration is recommended for only up to 2 years (Concannon, 1988; Evans
and Sutton, 1989) and treatment during pregnancy may lead to masculinisation of female foetuses (Evans and Sutton, 1989).

Milberone should also not be given to prepubertal bitches because the androgenic activity of the drug may result in early epiphyseal closure (Evans and Sutton, 1989).

**Synthetic Progestagens**

Progestagens mimic the physiological effect in the body of the natural hormone, progesterone (Evans and Sutton, 1989).

In general, progestagens are (Evans and Sutton, 1989):

1. **Antigonadotrophic.** They suppress follicular development and the production of oestrogen and prevent ovulation and the formation of corpora lutea.
2. **Antioestrogenic.** Vaginal bleeding is controlled.
3. **Antiandrogenic.** Sex drive in males is reduced.
4. **Contraceptive.** Sperm transport is interfered with and the events that need to be critically timed for pregnancy to result from mating are desynchronised.
5. **Progestagenic.** Pregnancy is maintained and a secretory endometrium is produced.

Not all progestagens are active to the same degree in all of these properties (Evans and Sutton, 1989).

**Safety and Side Effects of Progestagens**

The following side effects have mainly been documented from the use of the 1st generation injectable and oral progestagen compounds, such as medroxyprogesterone acetate (MPA) and megestrol acetate (Evans and Sutton, 1989). The duration of effect of most injectable compounds have varied considerably although continuous postponement of oestrus can be obtained with a high degree of certainty by repeated injections at 4-6 month intervals (Van Os and Oldenkamp, 1978; Evans and Sutton, 1989).
(a) Uterine Changes

Progestagens, like progesterone itself, can potentially induce changes in the uterus such as cystic endometrial hyperplasia, mucometra or pyometra (Evans and Sutton, 1989). The original first generation compounds are strongly progestagenic (Evans and Sutton, 1989). When using these compounds, it is very important to control the dose given, as repeated treatment results in accumulation. The stage of cycle that treatment is given is also important as the drug induced changes are facilitated if the uterus has been sensitized with oestrogens before dosing (as occurs in pro-oestrus) (Evans and Sutton, 1989).

MPA has been removed from the market in the U.S.A. because of the high incidence of cystic endometrial hyperplasia (Concannon and Meyers-Wallen, 1991).

(b) Mammary Tumours

Megestrol acetate has been used to control the growth of oestrogen-dependent mammary tumours, although it has subsequently been found that long term use of progestagens in bitches often results in mammary neoplasia (Evans and Sutton, 1989). MPA has also been found to stimulate the development of hyperplastic and neoplastic nodules in the mammary glands (Evans and Sutton, 1989).

(c) Induction of Growth Hormone Secretion

Recently, treatment of dogs with MPA and proligestone has been found to induce the local overproduction of growth hormone in the mammary gland (Selman, Mol, Rutterman, Vangarderen and Rijnberk, 1994a; Selman, Vangarderen, Mol and Vandeningh, 1995; Selman, Wolfswinken and Mol, 1996; Mol, Selman, Sprang and Vanneck, 1997). In dogs, there is a complex system of growth hormone and insulin-like growth hormone that is an important regulatory system of epithelial cell proliferation and differentiation (Mol et al., 1997). Administration of MPA and proligestone in dogs has resulted in a high proliferative environment in the mammary tissues that may also enhance the risk of malignant transformation
(Mol et al, 1997). In one study involving the treatment of bitches with MPA or progestone 3 weeks apart for 6 treatments, there was a high incidence of benign mammary tumours in treated dogs compared with controls (Selman et al, 1995).

**Adrenocortical Suppression**

Cats dosed with 5mg megestrol acetate daily for 14 days have been found to have adrenal suppression lasting for 2 weeks after treatment (Evans and Sutton, 1989). This could have been caused by feedback inhibition of adrenocorticotropic hormone (ACTH) storage and release or as a direct effect of the drug on the adrenal cortex. Other studies using the same dosing regime have resulted in glucose intolerance and adrenal suppression, but the effects were reversed in 4 weeks after treatment (Evans and Sutton, 1989). Megestrol acetate is also contraindicated in diabetes mellitus (Concannon and Meyers-Wallen, 1991) due to its ability to cause pancreatic changes typical of diabetes mellitus (Olsen et al, 1986a).

Treatment of bitches with progestone and MPA has also been associated with suppression of the hypothalamic-pituitary-adrenocortical (HPA) axis in dogs (Selman, Mol, Rutteman and Rijnberk, 1994b). Both of these drugs act as glucocorticoid agonists and are able to suppress the HPA axis for up to 6 months (Selman et al, 1994b). Long-term treatment with high doses of these progestins may result in an iatrogenic Cushing’s syndrome and diabetes mellitus (Selman, Mol, Rutteman and Vangarderen, 1997).

**Other Side Effects**

Progestagens have been shown to cause masculinisation of female foetuses (Olsen et al, 1986a; Evans and Sutton, 1989; Concannon and Meyers-Wallen, 1991), but the available progestagens are non-teratogenic (Evans and Sutton, 1989). Parturition may be delayed, with subsequent deaths of foetuses, if effective levels of progestagens persist for longer than the normal duration of pregnancy (Evans and Sutton, 1989).
A transient increase in weight gain and appetite may also be observed in treated animals as well as mammary hyperplasia in animals with long-term dosing (Evans and Sutton, 1989).

One preparation, proligestone, can be given during pregnancy without side effects, can be used at any time during the oestrus cycle and has a relatively long period of effectiveness. Long-term use of proligestone, though, as discussed, has been associated with excessive growth hormone production and suppression of the HPA axis.

**Proligestone**

Proligestone (14α, 17α-propylidenedioxypregn-en-3, 20-dione) is a unique second generation progestagen with the following properties that have reduced the side effects normally accompanying progestagen usage (Evans and Sutton, 1989):

1. Proligestone is antigenadotrophic, but only weakly progestagenic
2. Proligestone is weakly antiestrogenic
3. Proligestone has a medium duration of action.

Proligestone is effective at suppressing heat if given at the beginning of pro-oestrus, temporarily postponing heat if given about one month before oestrus and permanently postponing heat if given repeatedly at about 5 month intervals.

A single subcutaneous injection at the beginning of pro-oestrus was found to cause the signs of heat to disappear in about 2-5 days in 95.6% of 61 bitches (Van Os and Oldenkamp, 1978). For permanent postponement of oestrus, the first dose is given 3 months after the induction dose in ‘anoestrus’ or pro-oestrus (Evans and Sutton, 1989). The second dose is given in another 4 months and the 3rd and subsequent doses are given every 5 months thereafter (Evans and Sutton, 1989). This regimen will keep 97-98% of bitches in anoestrus permanently (Evans and Sutton, 1989). When treatments are stopped, oestrus will occur on average 6 months later, with a range of 3-9 months (Evans and Sutton, 1989).
In addition to having a long and effective period of efficacy, proligestone has some positive health advantages to bitches (Evans and Sutton, 1989). The incidence of pyometra in medicated bitches is 10-100 fold less than in bitches left to cycle normally (Van Os and Oldenkamp, 1978). The percentage of bitches with temporary discharge after treatment with one dose in pro-oestrus and anoestrus was found to be 0.7 and 0.5% respectively (Van Os and Oldenkamp, 1978). Only 0.3% of the bitches in the same trial were found to develop cystic hyperplasia/pyometra complex (Van Os and Oldenkamp, 1978). The incidence of false pregnancy in bitches with permanent postponement of heat is reduced irrespective of past history of pseudopregnancy. Only 3.9% of bitches of varied histories exhibited false pregnancy compared with 45-75% of bitches left to cycle normally (Van Os, 1980).

Proligestone administered during pregnancy may prolong the pregnancy, especially if given at a time when the effect of the drug surpasses the expected birth date (Evans and Sutton, 1989). In four bitches treated at the time of mating, one needed a caesarian section. Eighteen of 19 other bitches mated at the next oestrus after treatment had normal births (Van Os and Oldenkamp, 1978).

2.4.3.2 Canine Parasite Control

Considering the broad range of zoonotic parasites, an anthelmintic suitable for parasite control in dogs would have to have a broad range of efficacy, be easy to administer, have minimal side effects and sufficient persistence in the body without encouraging resistance. Ivermectin (a macrocyclic lactone of the avermectin family of drugs) was investigated as a potential parasiticide for dogs.

2.4.3.2.1 Features of Ivermectin

Ivermectin was first synthesised and described in 1980 (Lanusse and Prichard, 1993) as the 22, 23-dihydro-derivative of the naturally occurring fermentation product of *Streptomyces avermitilis*, avermectin B1 (Campbell, 1985). Ivermectin is a composite product of avermectin
B1a and B1b homologues (Campbell, 1985) and has a broad spectrum of high anthelmintic activity against adult and larval forms of gastrointestinal nematodes as well as against external parasites (Lanusse and Prichard, 1993).

2.4.3.2.2 Mode of Action of Ivermectin

Avermectins selectively paralyse parasites by increasing muscle chlorine ion (Cl⁻) permeability (Martin, 1997). Until recently, avermectins were thought to stimulate the release of the neurotransmitter GABA (alpha-amino butyric acid) from nerve endings as well as enhance the binding of GABA to its receptor (Campbell, 1985) in nematodes and ectoparasites. Recent evidence now suggests that the interactions of avermectins with glutamate-gated chloride ion channels is responsible for the parasiticidal action of avermectins (McKellar and Benchouai, 1996; Martin, 1997).

In Ascaris suum, the laboratory model for determining the mode of action of avermectins, avermectins inhibit pharyngeal pumping that is part of the nematode feeding process (Martin, 1997). Motility of nematodes is also affected (McKellar and Benchouai, 1996) and in ectoparasites, ivermectin is either overtly lethal or paralytic. Ivermectin is also able to inhibit oviposition or engorgement (in blood sucking arthropods) (Campbell, 1985). In ticks, altered behaviour or suppression of development has been observed (Campbell, 1985).

2.4.3.2.3 Efficacy of Ivermectin

Ivermectin is effective against nematode, insect and acarine parasites, but has no useful effect against trematodes or cestodes because they lack glutamate-gated Cl⁻ channels (Shoop, Mrozik and Fisher, 1995). Although ivermectin is similar in structure to the macrolide antibiotics, it is virtually devoid of antibacterial activity. Likewise, ivermectin is not efficacious against protozoa although in a field study 66 humans with Entamoeba coli cysts there were significant reductions in cyst loads after treatment at 200μg/kg with a cure rate of 46% (Njoo, Belling, Oosting, Vetter, Stilma and Kijlstra, 1993).
Efficacy in Dogs

When given as a single subcutaneous injection at 200μg/kg, the drug is active against the immature and adult stages of Ancylostoma caninum, A. braziliense, Uncinaria stenocephala, Toxocara canis and Strongyloides stercoralis and against immature Toxocaris leonina and adult Trichuris vulpis (Campbell, 1985).

Hookworms

Hookworms are particularly susceptible to ivermectin (Blair and Campbell, 1978; Campbell, 1989) and at doses of approximately 50μg/kg (s/c or p/o), there is maximal efficacy against both L4 and adults of A. caninum (Yazwinski, Tilley and Greenway, 1982) and U. stenocephala (Campbell, 1985). Ivermectin also affects eggs within worms (at 10μg/kg) and has lethal in vitro activity against infective L3 larvae of A. caninum at 0.025μg/mL (Campbell, 1989). No information about the efficacy of ivermectin against arrested larvae in dogs is available, although ivermectin is very efficacious against hypobiotic larvae of most gastrointestinal nematodes in cattle (McKellar and Benchouli, 1996).

Ivermectin to Prevent Transmammary Transmission of Hookworms

Ivermectin has been found to prevent transmammary migration of larvae. Administration of a single dose of 500 to 1000μg/kg ivermectin 2 to 10 days before whelping causes more than 95% reduction in the numbers of Ancylostoma caninum recovered from puppies compared to puppies born to control bitches (Bowman, 1992).

Human Hookworm and Ivermectin

Interestingly, ivermectin is reported not to have any affect on human hookworms (Whitworth, Morgan, Maude, McNicholas and Taylor, 1991; Njoo et al., 1993). It is likely, considering that A. caninum and A. duodenale are of the same genus, that ivermectin would be effective against A. duodenale.
**Toxocara canis**

Ivermectin exhibits complete anthelmintic efficacy against artificially acquired *T. canis* infections at doses greater than 50μg/kg (Yazwinski *et al.*, 1982). In one study of naturally acquired patent infections, worm burden was reduced by 91% when dogs were treated with 200μg/kg ivermectin subcutaneously (Campbell, 1989).

Both adult and intestinal fourth stage larvae are affected by ivermectin. Treatment regimes in bitches using high doses of ivermectin have shown it possible to affect somatic *T. canis* in such a way that pups born to treated bitches have a 34% to 94% reduction in adult *Toxocara* worms (Overgaauw, 1997). One such regime was 1000μg/kg subcutaneous on day 20 followed by 500μg/kg on days 42, 47 and 53 of gestation (Campbell, 1989).

**Strongyloides stercoralis**

In humans, ivermectin appears to be very effective at clearing *S. stercoralis* when administered at 200μg/kg; even refractory chronic infections (Wijesundera and Sanmuganathan, 1992). Campbell (1989) cites one trial of a dog with *S. stercoralis* being treated with ivermectin at 800μg/kg. All adults were removed, and only 2 larvae were found at post mortem 6 days after treatment.

**Trichuris vulpis**

Ivermectin was found to be 100% effective against immature *T. vulpis* in experimentally infected dogs given 100μg/kg ivermectin subcutaneously (Yazwinski *et al.*, 1982). Treatment was more than 99% effective against adults at the same dose rate.

**Dirofilaria immitis**

Ivermectin is registered for use in dogs as a chemoprophylaxis for *D. immitis* infection at 6μg/kg (McKellar and Benchaoui, 1996). Third and fourth stage larvae are exceptionally susceptible to low doses (<1μg/kg) of ivermectin, and at doses upward of 50μg/kg, ivermectin is
**Toxocara canis**

Ivermectin exhibits complete anthelmintic efficacy against artificially acquired *T. canis* infections at doses greater than 50μg/kg (Yazwinski *et al*, 1982). In one study of naturally acquired patent infections, worm burden was reduced by 91% when dogs were treated with 200μg/kg ivermectin subcutaneously (Campbell, 1989).

Both adult and intestinal fourth stage larvae are affected by ivermectin. Treatment regimes in bitches using high doses of ivermectin have shown it possible to affect somatic *T. canis* in such a way that pups born to treated bitches have a 34% to 94% reduction in adult *Toxocara* worms (Overgaauw, 1997). One such regime was 1000μg/kg subcutaneous on day 20 followed by 500μg/kg on days 42, 47 and 53 of gestation (Campbell, 1989).

**Strongyloides stercoralis**

In humans, ivermectin appears to be very effective at clearing *S. stercoralis* when administered at 200μg/kg; even refractory chronic infections (Wijesundera and Sanmuganathan, 1992). Campbell (1989) cites one trial of a dog with *S. stercoralis* being treated with ivermectin at 800μg/kg. All adults were removed, and only 2 larvae were found at post mortem 6 days after treatment.

**Trichuris vulpis**

Ivermectin was found to be 100% effective against immature *T. vulpis* in experimentally infected dogs given 100μg/kg ivermectin subcutaneously (Yazwinski *et al*, 1982). Treatment was more than 99% effective against adults at the same dose rate.

**Dirofilaria immitis**

Ivermectin is registered for use in dogs as a chemoprophylaxis for *D. immitis* infection at 6μg/kg (McKellar and Benchaooui, 1996). Third and fourth stage larvae are exceptionally susceptible to low doses (<1μg/kg) of ivermectin, and at doses upward of 50μg/kg, ivermectin is
effective against microfilariae (Campbell, 1989; Kreger, Seal, Callahan and Beckel, 1990). A single oral dose, given up to 2 months after infection prevents the development of mature infection (Campbell, 1985).

Fifth stage larvae and adults are refractory to the drug (Campbell, 1989) even at doses approaching the maximum tolerated by the host (Campbell, 1985). The drug, though, does appear to cause degeneration of embryos in adult female worms (Campbell, 1985) as well as inhibit the release of microfilariae from the uterus of adult heartworms (Campbell, 1989).

Ectoparasites

Sarcoptes scabiei

Dosages at 200μg/kg or higher have resulted in complete elimination of Sarcoptes scabiei by day 14 after treatment in single housed dogs (Yazwinski, Pote, Tilley, Rodriguez and Greenway, 1981). At 200μg/kg, ivermectin has also been found to result in complete cure of infected farmed foxes in groups of 5-10 by 10 days after treatment (Mouka, Harmannova and Konrad, 1987). Other trials have required a second treatment at 14 days for complete recovery of all animals (Thimmappa Rai and Yathira, 1988). Administration of a single dose at 400μg/kg has resulted in similar outcomes (Singh and Gill, 1987).

In dogs residing in heavily contaminated environments, two treatments (200μg/kg) at 14 day intervals have resulted in clinical improvement (Scheidt, Medleau, Seward and Schwartzman, 1984). Situations of natural infestation such as this are most closely related to the present problem faced in Aboriginal communities. Other field trials involving single treatments have not been documented.

Fleas

There appeared to be no efficacy against Ctenocephalides felis on dogs in any of the few controlled trials prior to 1989 (Campbell, 1989). No other information has since become
available, although ivermectin shows excellent efficacy against arthropods that are in contact
with or ingest host body tissues (McKellar and Benchaaoui, 1996). Limited control of fleas by
ivermectin may be related to the ability of all stages of fleas to remain off the host for long
periods of time.

Ticks
As with fleas, there have been very few trials on the effectiveness of ivermectin against
*Rhipicephalus sanguineus* or other ticks on dogs. In cattle, ‘IVOMEC’ (antiparasitic injection
for cattle, Merial, Australia, 10g/L) has been shown to be highly effective against single-host
ticks of the genus *Boophilus* and suppress the reproductive potential of many tick species
(McKellar and Benchaaoui, 1996) for up to 20 days post treatment.  Ivermectin, though, is
reported to be less efficacious against multiple-host ticks, which include *Rhipicephalus
sanguineus* (Benz, Roncalli and Gross, 1989).

2.4.3.2.4 Pharmacokinetics and Efficacy
Most work regarding the pharmacokinetics of ivermectin in dogs has been related to the oral
preparation ‘HEARTGARD’ (Merial, Australia) which is only available at 6μg/kg. As
previously demonstrated, the dose rate required for control of most parasites is 200μg/kg, but
the behaviour of the drug varies markedly with the route of administration, the formulation and
the species to be treated (Campbell, 1985).

Route of Administration and Formulation
Three main preparations for administration of ivermectin to cattle are available; topical, oral and
injectable. In cattle, the topical formulation at 500μg/kg has efficacy against nematodes similar
to parenteral or oral routes of administration at 200μg/kg (Lanusse and Prichard, 1993), but this
formulation only became available after the commencement of the present study. Some
differences in efficacy against ectoparasites have been noticed with the three routes of
administration.
Ivermectin is generally absorbed to a greater degree, but more slowly, when given subcutaneously rather than per os (McKellar and Benchaoi, 1996) and in dogs, ivermectin (regardless of formulation) is roughly equipotent against nematodes whether given orally or parenterally (Campbell, 1985). For some ectoparasites, such as Sarcoptes scabiei, subcutaneous injection is more effective than oral treatment (Campbell, 1985).

‘Pure’ ivermectin, intravenously administered as an aqueous micellar formulation, attains a half-life of 1.8 days in the dog (McKellar and Benchaoi, 1996). In cattle, the half-life of ‘IVOMEC antiparasitic injection for cattle’ (ivermectin in a non aqueous base) administered at a dose of 200μg/kg is 8.3 days which is longer than the intrinsic half life of the ‘pure’ formulation; 2.8 days (Fink and Porras, 1989). The half-life and peak plasma concentrations of the ‘IVOMEC’ formulation administered subcutaneously are not known for dogs (Paradis, de Jham and Pagé, 1997), although the half-life would probably be longer than ‘pure’ ivermectin due to the slower absorption from the injection site. In cattle, the anthelmintic activity of the drug persists for 2 weeks after treatment consistent with the plasma levels of ivermectin (McKellar and Benchaoi, 1996).

‘IVOMEC antiparasitic injection for cattle’ formulation is a 1% (w/v) solution of ivermectin in a mixed solvent vehicle of 60% (v/v) propylene glycol and 40% (v/v) glycerol formal (Fink and Porras, 1989).

2.4.3.2.5 Side Effects of Ivermectin Treatment

Acute Toxic Syndrome

Ivermectin is selectively toxic against endo- and ectoparasites. The glutamate-gated chloride channels, by which ivermectin probably derives its antiparasitic effect, have not been reported in mammals (McKellar and Benchaoi, 1996). Likewise, ivermectin distributes poorly into the brain of mammalian species which also limits ivermectin’s ability to produce toxic effects in
mammals. At sufficiently high doses of ivermectin, though, the blood brain barrier may not be able to prevent ivermectin permeation (Hopkins, 1990).

Acute ivermectin toxicosis in dogs is related to diffuse cerebral and cerebellar dysfunction (Hopkins, 1990). Ivermectin toxicosis in the acute form is manifest as mydriasis, depression, tremors, ataxia, stupor, emesis, drooling and coma (Pulliam and Preston, 1989). In Beagles, the \textit{LD}_{50} is approximately 80mg/kg and the highest single oral dose without effect has been found to be 2mg/kg (Pulliam and Preston, 1989). This dose is 10 times the effective therapeutic dose of ivermectin in dogs (200\mu g/kg), and the most instances of toxic dosing have occurred with misuse of 'off label' preparations.

The toxic effect can be reversed by picrotoxin which is believed to regulate the closing of chloride ion channels at presynaptic junctions (Campbell, 1985) and hence act as a powerful CNS stimulant (Hopkins, 1990).

\textit{Acute Ivermectin Toxicosis in Collie Dogs}

Rough-coated collies have suffered severe adverse reactions, even death, following treatment with ivermectin (Campbell, 1985; Pulliam and Preston, 1989). Collies are believed to have greater penetration of the blood brain barrier by ivermectin than other dogs leading to increased susceptibility to acute toxic reactions. Collies that die from ivermectin toxicosis show signs similar to other dogs that have acute toxic overdoses, but clinical signs are first seen at doses as low as 100\mu g/kg (compared with 2mg/kg in other dogs) (Pulliam and Preston, 1989). Breeds, such as Dobermans, may also show idiosyncratic reactions at relatively low doses (Hopkins, 1990). In Australia, no bloodlines of dog breeds have shown particular susceptibility to ivermectin, but treatment of collies at 200\mu g/kg is contraindicated.
Reproductive Side Effects

In no species is there data to suggest that the drug is a teratogen in the absence of maternal toxicity (World Health Organisation, 1993). Likewise there is no evidence of significant incidence of adverse effects on reproductive performance in treated animals; both male and female (Pulliam and Preston, 1989; World Health Organisation, 1993). Considerable excretion of ivermectin occurs via the mammary gland in lactating females (McKellar and Benchaoui, 1996) and this coupled with a poorly developed blood brain barrier in puppies may cause toxic effects in the neonate.

Adverse Effects in Microfilaraemic Dogs

Vomiting, excess salivation, blood in faeces, soft stool/diarrhoea and depression have been observed in dogs with circulating *D. immitis* microfilariae administered ivermectin (Pulliam and Preston, 1989). The shock-like reactions in heartworm infested dogs administered diethylcarbamazine have not occurred in dogs following ivermectin treatment and although deaths have been seen following ivermectin treatment, none of these reactions have been confirmed to be caused by ivermectin (Pulliam and Preston, 1989).

'IVOMEC' Formulation Side Effects in Dogs

The subcutaneous administration of 'IVOMEC' cattle preparation in dogs has been well tolerated and safe according to many previous studies (Scheidt et al, 1984; Singh and Gill, 1987). Most dogs in a scabies trial conducted by Scheidt et al (1984) displayed mild flinching or no reaction at all and no local reactions at the site of injection. In contrast, Thimmappa Rai and Yathira (1988) noticed depression and anorexia 24 hours after treatment. No explanations for this were given, although propylene glycol (the base in 'IVOMEC') can cause bradycardia and respiratory and central nervous system depression (Medleau, 1994).
**Insecticidal Effect**

Most of the ivermectin dose administered to animals is ultimately eliminated in the faeces (Lanusse and Prichard, 1993; Medleau, 1994), where it has been shown to have an insecticidal effect (Wall and Strong, 1987).

In field trials, the faeces of calves fitted with rumenal boluses delivering ivermectin at 40µg/kg per day failed to degrade in the normal way (Wall and Strong, 1987). This failure was associated with the absence of dung-degrading insects (Wall and Strong, 1987), although subsequent research shows that dung beetles are not repelled by avermectin in faeces (Wardhaugh and Mahon, 1991). Faeces not degraded by dung beetles remain as breeding foci for flies (Coe, 1987). This is particularly important in crowded community situations where flies may be responsible for the spread of many pathogens (such as hookworm (Oyerinde, 1976)).

Enough ivermectin is excreted following a single injection of 200µg/kg to eliminate nonpest Diptera in dung for at least 35 days following treatment (Wall and Strong, 1987). In another study by Mahon, Wardhuagh, Vangerwen and Whitby (1993), faeces from sheep treated with ivermectin 24 hours previously was fed to *Lucilia cuprina* adults who subsequently exhibited reduced survival, delayed ovarian development and reduced egg production. These effects were absent in dung produced 2 or more days after ivermectin treatment. Further studies have found ivermectin to inhibit survival of the larvae of *M. vetustissima* and *M. domestica* in cattle dung for up to 7 to 21 days after treatment, respectively (Wardhaugh, Holter, Whitby and Shelley, 1996; Wardhaugh and Mahon, 1998).

The overall effect on fly populations by treating community dogs is most likely transient as the fly population may be controlled for only a limited time. The effect of ivermectin residues in faeces on dung beetle populations and on the general fitness of the populations have yet to be tested (Ridsdill-Smith, 1993).
3.1 Introduction

This chapter details the methodology used to determine the effectiveness of the canine breeding and parasite control program in remote Kimberley Aboriginal communities.

Firstly, consideration of the cultural context in which the study was undertaken is examined, as the field data could not have been collected without appropriate conduct and appreciation of the cultural heritage of the communities. Descriptions of the Kimberley region and study communities are provided, including physical characteristics, location and facilities available.

Secondly, the daily routine involved in the collection of data and samples and treatment of dogs is described, including the techniques used to examine the samples.

Thirdly, methods used to collect data relating to child health are described and explained and the statistical methods used to interpret the data collected over the duration of the program are briefly examined.

Throughout the methodology chapter, the rationale for choice of techniques and procedures is given.

3.2 The Study Communities

3.2.1 Cultural considerations

3.2.1.1 Communication and Timing

The program, which was undertaken over a three and a half year period, would not have been possible without community participation. For community participation, gaining the confidence of community members was vital.
Prior to the fieldwork, two weeks were spent contacting and visiting community councils and major spokespeople of most of the communities involved in the program. With the help of well-known local Aboriginal health-workers, the program was fully explained. The contact people were then given the opportunity to discuss the matter with the wider community before acceptance. Some communities actively sought assistance from the research team and were aware of the program from communications with other communities.

The final acceptance of the program by 17 isolated communities was largely due to effective negotiations from the outset. Once the program commenced, the researcher had to communicate with almost all community members on a one-to-one basis. When communication difficulties arose, patience and respect generally resolved the problems.

“A different tradition leaves us tongueless and earless towards this other world of meaning and experience” (Stanner, 1979:230, cited in Harkins, 1994).

There are 5 language families in the Kimberley region encompassing many languages or dialects. These families are the Jarragan, the Worrorran, the Nyulnyulan, the Bunuban and the Pama-Nyungan (Ryan, 1993). Remarkably, there were people with the skill to communicate in up to four languages, but these people were few and far between and often frail with age. Consequently it was important to rely on a respected member of each community to convey messages. In the Fitzroy valley, Kriol, which is a blend of English with traditional languages, was spoken by some. Often the English used in Kriol assumes a different meaning to the original English (Harkins, 1994), so it was important not to misconstrue interpretations.

Regardless of the language barriers, all communication had to be explicit and clear without assuming literary superiority or implying that issues were straightforward and ‘cut and dried’.
In order to gain information, it was important to start up conversations by offering questions that were open-ended or suggested a variety of options for answers.

A form of communication that was often effectively used was ‘sideways talking’ where a third person is used as a buffer to convey messages to another person. This was most often used for sensitive issues or to gather information from shy people. The Aboriginal volunteers to the program were very effective for communication.

3.2.1.2 Customs and Conduct

As a result of the complex kinship systems, laws and language, there are many customs in Aboriginal communities that non-Aboriginal people may not be aware of. Breach of these customs can jeopardise a good working relation and the success of a program.

The research group was tutored at each visit on some of the local behaviours and traditions to avoid any embarrassment and to improve the chances of receiving unbiased information. An example of one sensitive area is the correct use of names for people. Sometimes the use of personal names creates tension. Pointing out individual members by name is considered impolite. Likewise using the name of someone who has died is shunned. Some Aboriginal people have also adopted ‘whitefella’ or gadiya names that may not be known to everyone, so confusion about identification is common. Some special places can not be mentioned. All of these factors were taken into consideration when collecting information.

Another impolite gesture is eye contact as is any form of physical contact with the opposite sex, as Aboriginal society is mainly regarded as genderocratic. Despite this, difficulties were not recognised in this program as the project team was composed of both sexes.
Occasionally, social discomfort amongst the Aboriginal members of the team was felt, although this was usually transitory and waned with each visit as working relations were developed. In many ways, the non-Aboriginal members acted as buffers, as any potentially embarrassing procedures (such as examination of dog’s reproductive organs or faeces collection) were conducted by the ‘whitefellas’.

Basically, sensitivity and diplomacy were very important to avoid offending people.

3.2.1.3 Methods Used to Encourage Continuation of the Program

The specific activities followed to promote self-management and continuation of the program after the research was completed involved the following:

1. Assisting the community to identify the problems clearly and with perspective. Community meetings asking for ideas and concerns were conducted prior to any intervention. Follow-up sessions to gain more ideas were conducted.

2. Acquainting the community with pertinent resources available to the community. The skills and expertise of the project team were made clear and invitation was made for the community to capitalise on them. Outside veterinary services and access to equipment and consumables to assist in maintaining dog’s health were also available.

3. Introducing stimuli to activate the community’s own dormant resources. Acknowledging assistance on a community scale encouraged participation in the program by Environmental Health Workers (community members trained in environmental health) and other people with animal handling skills. The result was an enhanced feeling of self-esteem as the community volunteers often had the role of conveying the health message to the community.

4. Creating an environment in which the community feels free to work out its own problems. At all times the group was open to suggestions and actively entered discussions on a community and council level to allow free comments on the program.
3.2.2 Community Profiles

3.2.2.1 The Kimberley Region

3.2.2.1.1 Natural Physical Attributes of the Kimberley Region

The Kimberley region is bordered by the Northern Territory to the east, the Timor Sea to the north, the Indian Ocean to the west and the Great Sandy and Tanami Deserts to the south (see Figure 3.1). The region extends from approximately 14°S to 20°S. Broome, the southwestern-most town in the region, is approximately 2000km north of Perth, the capital city of Western Australia. The region stretches 1000 km from Broome to the eastern most town of Kununurra to cover a total area of 421 000 km² (Bureau of Meteorology, 1996).

Figure 3.1: Study Communities and Towns in the Kimberley Region of Western Australia

The region has a tropical monsoonal climate with distinct wet and dry seasons separated by short transitional periods. The wet season roughly occurs from November to April, with 90% of the average yearly rainfall during this period (Bureau of Meteorology, 1996). Aboriginal people of the area believe there to be up to 10 seasons according to the bush food available.
Annual rainfall decreases southwards and is highly variable from year to year (Bureau of Meteorology, 1996). Overall there is considerable variation in the climate throughout the region.

The average maximum temperatures exceed 35ºC in November and December with the highest temperatures occurring in inland parts of the southwest Kimberley (Bureau of Meteorology, 1996). Winter maximum temperatures average about 30ºC and are highest in the north and lowest in the south (Bureau of Meteorology, 1996). Minimum temperatures in the winter (‘dry’ season) remain above 20ºC in the coastal parts, but can drop to below 5ºC in the high plateau and central regions (Bureau of Meteorology, 1996).

The land surface of the Kimberley is very ancient due to a history of geological stability and mainly consists of plains with undulating sand ridges. To the north the King Leopold and Durack ranges separate the northern basin from the drier and flatter regions in the south that extend to the desert areas. The vegetation ranges from dense eucalypt woodlands, mangrove forest and rainforest remnants in the north to Savannah woodlands in the central region and sparse acacia scrubland and spinifex Savannah in the south. The variation of vegetation reflects the distribution of rainfall and soil types.

**3.2.2.1.2 Population of the Kimberley**

The Aboriginal population of the Kimberley is far more dispersed than the non-Aboriginal population - the non-Aboriginal population being almost entirely confined within six towns. The total population of the Kimberley in 1991 was 25,208 with 64% of people residing in two western Shires.

The Aboriginal population is also far more demographically stable than the non-Aboriginal population with a committed long-term core of residents. In 1991, the Aboriginal population of
the Kimberley was 10 707 (Anon, 1993). This figure represented 45% of the estimated resident population. The Aboriginal population was also very young with 31% of people under the age of 15 (Anon, 1993).

3.2.2.1.2.1 The Urban Centres of the Kimberley

Broome, Derby, Fitzroy Crossing, Halls Creek, Kununurra and Wyndham comprise the six towns of the Kimberley that contained 75% of the population in 1991 (Anon, 1993). Each of these towns grew as service and administrative bases for various activities in the surrounding country such as pearling, ‘farming’ and mining. Broome was established as a pearling centre, Derby and Wyndham were ports for meat export, Fitzroy Crossing serviced the pastoral activities, Halls Creek developed during the gold rush era and Kununurra was another pastoralist base that developed into a major fruit and vegetable producer and service centre for the nearby Argyle diamond mine.

Each town is either on or close to the major transport spine, the Great Northern Highway. In addition to this, reasonable sized airports service Broome, Derby and Kununurra.

3.2.2.1.2.2 Aboriginal Communities of the Kimberley

In 1991, there were 16 communities of 80 or more persons in the region (Anon, 1993). Fifty three percent of Aboriginal people in the Kimberley lived in widely dispersed communities of varying origins. The smaller communities are often in very remote locations accessed by poor standard roads. Most communities have some form of social, family or economic links to one or more urban centres. The transport links between communities and urban centres are thus considered to be very important for maintaining contact (Anon, 1993).

The homeland movement gained momentum in the 1980s when smaller family groups started to leave the large communities to live in ‘outstations’. The federal government sponsored a 5
year program in WA in 1985-1990 to allow people to return to their homelands and establish communities (Anon, 1994). Although this is a more culturally appropriate lifestyle, it has made provision of services such as education and health (including this program) difficult.

In most communities there are community councils elected by the community members. Although these councils are not always working at maximum effectiveness (Crawford, 1989), it is very important for service workers not to undermine the operations and to always communicate through this official channel. As such, each community was notified of intending visits by the research team by a series of faxes and phone calls through the council which usually filtered to the Environmental Health Workers.

The councils are responsible for determining what services are required and for employing people to provide them. They have the limits of federal and state laws (including those governing Public Health) as do other local governments. Some communities also have their own by-laws supported by the Aboriginal Communities Act of 1979, WA.

3.2.2.1.3 Veterinary Services in the Kimberley Region

There are two private veterinary practices in the Kimberley region at Broome and Kununurra. Although both offer extension services to Fitzroy Crossing and Derby (from Broome) and Halls Creek (from Kununurra) these are not fully utilized by the communities and are usually for only one day a week or month.

The Kimberley division of the Agriculture Department of Western Australia and Australian Quarantine Inspection Service (AQIS) also employ veterinarians who are usually involved in cattle station work and quarantine procedures.
Several concerned veterinarians in the region have attempted to conduct ‘dog programs’ in the past.

3.2.2.2 Communities of the Study

Although grouping of communities into three broad regions does conceal their diversity and the heterogeneity in the occurrence of some infections, there were sufficient similarities (in both physical characteristics and cultural background) within the regions to warrant the grouping. For the majority of the study, communities were grouped into one of three regional study areas; coastal, central and eastern (Table 3.1).

3.2.2.2.1 Communities of the Coastal Region

The coastal communities of this study consisted of the Dampier Peninsular communities (Lombadina/Djarindjin, Beagle Bay and One Arm Point) and Kalumburu to the very far north. Kalumburu was included in the program in March 1993, one year after the others. For this reason, Kalumburu was considered as a separate community for most analyses throughout this thesis.

All of these communities were developed from mission backgrounds with management of the communities devolved to the people during the early 1980s. In 1985 Djarindjin community was formally established, but later that year a breakaway family group formed the adjacent Lombadina Aboriginal Corporation. Both Kalumburu and One Arm Point have been relocated by the state government several times over the years since occupation.

As each of these communities were well established, they are well equipped with the exception of Kalumburu which, in 1991, did not have running water or toilet facilities at many households. Overcrowding was also common at this community. In 1991, each community
<table>
<thead>
<tr>
<th>Community</th>
<th>Description</th>
<th>Land</th>
<th>Languages</th>
<th>School</th>
<th>Health Clinic</th>
<th>EHW CDEP</th>
<th>Estimated Resident Population</th>
<th>Distance from Urban Centre</th>
<th>Housing</th>
<th>Store</th>
<th>Post European Settlement History*</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>COASTAL REGION</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>One Arm Point</td>
<td>Large Community</td>
<td>ARL</td>
<td>Bardi, Jawi</td>
<td>YES</td>
<td>YES</td>
<td>CDEP</td>
<td>542</td>
<td>230km Boome</td>
<td>Steel frame asbestos</td>
<td>YES</td>
<td>Mission 1975</td>
</tr>
<tr>
<td>Lombadina/Djarindjin</td>
<td>Large Communities</td>
<td>ARL</td>
<td>Bardi</td>
<td>YES</td>
<td>YES</td>
<td>EHWCDEP</td>
<td>254</td>
<td>210km Boome</td>
<td>Metal walled, tin roof</td>
<td>YES</td>
<td>Mission 1892</td>
</tr>
<tr>
<td>Beagle Bay</td>
<td>Large Community</td>
<td>ARL</td>
<td>Nyulnyul</td>
<td>YES</td>
<td>YES</td>
<td>CDEP</td>
<td>270</td>
<td>130km Boome</td>
<td>Steel frame Colourbond/fibro/mud brick</td>
<td>YES</td>
<td>Mission 1890</td>
</tr>
<tr>
<td>Kalumburu</td>
<td>Large Community</td>
<td>ARL</td>
<td>Kwini, Wunambal, Gamberre, Miwa</td>
<td>YES</td>
<td>YES</td>
<td>CDEP</td>
<td>364</td>
<td>Tin sheds without water, steel frame asbestos</td>
<td>YES</td>
<td>Mission 1907</td>
<td></td>
</tr>
<tr>
<td><strong>CENTRAL REGION</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bayulu</td>
<td>Large Community</td>
<td></td>
<td>Gooniyandi, Walmajarri</td>
<td>NO, located at station</td>
<td>YES, operated once a fortnight</td>
<td>EHWCDEP</td>
<td>206</td>
<td>15km Fitzroy Crossing</td>
<td>Steel frame asbestos</td>
<td>YES</td>
<td>Station</td>
</tr>
<tr>
<td>Joy Springs</td>
<td>Outstation</td>
<td></td>
<td>Gooniyandi, Walmajarri</td>
<td>NO, located at station</td>
<td>NO</td>
<td>NO</td>
<td>46</td>
<td>32km Fitzroy Crossing</td>
<td>Boughsheds and tents</td>
<td>NO</td>
<td>Station</td>
</tr>
<tr>
<td>Yiyili</td>
<td>Small Community</td>
<td></td>
<td>Gooniyandi</td>
<td>YES, operated once a week</td>
<td>EHWCDEP</td>
<td>145</td>
<td>190km Fitzroy Crossing</td>
<td>Steel frame asbestos</td>
<td>YES</td>
<td>Station</td>
<td></td>
</tr>
<tr>
<td>Ngalingkadji</td>
<td>Outstation</td>
<td></td>
<td>Gooniyandi</td>
<td>NO</td>
<td>NO</td>
<td>EHW</td>
<td>24</td>
<td>65km Fitzroy Crossing</td>
<td>Steel frame asbestos</td>
<td>NO</td>
<td>Station 1990</td>
</tr>
<tr>
<td>Muludja</td>
<td>Large Outstation</td>
<td></td>
<td>Kidja, Gooniyandi, Bunuba</td>
<td>NO</td>
<td>NO</td>
<td>EHWCDEP</td>
<td>101</td>
<td>43km Fitzroy Crossing</td>
<td>Steel frame asbestos on stilts</td>
<td>NO</td>
<td>Station</td>
</tr>
<tr>
<td>Looma</td>
<td>Large Community</td>
<td>ARL</td>
<td>Mangala, Walmajarri, Nyikina, Wangkajunga, Bunuba</td>
<td>YES</td>
<td>YES</td>
<td>CDEP</td>
<td>482</td>
<td>120km Derby</td>
<td>Steel frame asbestos</td>
<td>YES</td>
<td>Station early 1970s</td>
</tr>
</tbody>
</table>
Table 3.1 (cont.): Community Profiles - 1991

<table>
<thead>
<tr>
<th>Community</th>
<th>Description</th>
<th>Land Occupancy</th>
<th>Languages</th>
<th>School</th>
<th>Health Clinic</th>
<th>EHW CDEP</th>
<th>Estimated Resident Population</th>
<th>Distance from Urban Centre</th>
<th>Housing</th>
<th>Store</th>
<th>Post European Settlement History*</th>
</tr>
</thead>
<tbody>
<tr>
<td>EASTERN REGION</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yaramun</td>
<td>Outstation</td>
<td></td>
<td>Djaru, Ngarti, Nyininy</td>
<td>YES</td>
<td>NO, clinic once a week from Halls Creek</td>
<td>NO</td>
<td>114</td>
<td>120km Halls Creek</td>
<td>Steel frame asbestos, boughsheds</td>
<td>NO</td>
<td>Station/mission 1982</td>
</tr>
<tr>
<td>Warmun</td>
<td>Large Community</td>
<td>ARL</td>
<td>Kidja, Mirwoong, Djaru</td>
<td>YES</td>
<td>EHW CDEP</td>
<td>409</td>
<td>162km Kununurra</td>
<td>Steel frame asbestos, mud brick</td>
<td>YES</td>
<td>Station early 1970s</td>
<td></td>
</tr>
</tbody>
</table>

ARL  Aboriginal Reserve Land  
CDEP  Community Development Employment Plan  
EHW  Environmental Health Worker program  
Estimated resident population figures based on 1986 census and clinic client numbers.  
* Dates of ‘community establishment’. Aboriginal people have occupied these lands for thousands of years.
considered refuse disposal and dogs to be major environmental health problems (Douglas, 1991).

Transport to these communities was via road or air. In all cases, the accessing roads were unsealed and required 4WD vehicles for most of the year. Access during the wet season was limited, if not impossible.

3.2.2.2 Communities of the Central Region

Communities in this grouping all have links to Fitzroy Crossing with the exception of Looma. Looma, as with Kalumburu, was considered as a separate case community as the program was started later at Looma, in September 1992.

Geographically, the communities of this ‘region’ are quite widely dispersed (200km between them) but most have similar cultural background and station experiences.

Facilities at each community were similar although in 1992, Joy Springs had no established houses or facilities apart from a solar power pack and bore (see Table 3.1).

3.2.2.3 Communities of the Eastern Region

Yaramun and Warmun both had influence from the Catholic Church in recent times, but were established after eviction of the people from stations, which were their traditional Homelands.

Yaramun (also known as Kundat Djaru and Ringers Soak) is situated in the Western Desert, approximately 130 km from Halls Creek. In 1981, the station manager evicted the Kundat Djaru people from their land. This caused widespread concern for Aboriginal groups throughout the Kimberley region. In 1982 an excision from the station was offered to the people and the community was established.
Access by road in the wet season is impossible and people are frequently evacuated to the town during the wet due to flooding of the nearby Lake Gregory. Housing and a school has only recently been provided. Data collected from Yaramun was rarely included in the main analyses in the present study, unless stated, because of the infrequency at which the research group could visit the community due to flooding preventing access.

Warmun community was the largest community in this study and is situated at Turkey Creek, approximately 160 km north of Halls Creek. A ration depot was established at Turkey Creek in 1901 to protect the Kija and Miriwoong people after massacres in the area (Ryan, 1993). The community, though, was not formally constructed until the early 1970s. Due to its size and the ‘political influence’ of the occupant groups, most basic amenities were provided at this community.

3.2.2.3 Conclusion

3.2.2.3.1 Similarities between Study Communities

All communities of the program are remote in the sense of distance. Communication is aided by new technologies, but sometimes technology fails during the rainy season or because of the lack of technical support. Most communities were difficult to access during the wet season.

Nearly all communities used the ‘Community Development Employment Plan’ (CDEP) to employ members in the upkeep of the communities. This was the major fund base for the Environmental Health Workers who were present in six communities at the start of the program.

Within each region, there are similarities in history since European contact. All coastal communities were missions and all inland communities had some experience with station life in the past.
All communities had access to fresh water in 1991 and most had ‘adequate housing’ (see Figure 3.2).

3.2.2.3.2 Differences between Study Communities

One of the major differences between the communities in this study was the length of establishment and contact with European ways. This has influenced the degree of European education and experience each community has had with non-Aboriginal people. Consequently, the ability of the communities to gain political power varies (e.g. coastal communities often have more amenities than remote desert communities).

Another difference is the cultural and ethnic backgrounds of each community which largely relate to the languages spoken (Anon, 1994). Within each region there are some groups with similar heritage and ways, but ultimately each community is individual.

Location and distance from major urban sites is also different. Communities with access and close ties to towns are more likely to utilize the services provided. These communities are also more likely to have less stable populations.

3.3 Data Collection

The project involved visiting 14 communities every three months for three years to collect information regarding animal population statistics and dynamics and parasite infection rates. A preliminary trial was done in 3 closely situated communities from Kununurra to test the effectiveness of a single treatment with ivermectin (200µg/kg) and methods of sample analysis prior to the wider establishment of the program at the 14 other communities. Specific methodology regarding the trial will be dealt with later (5.2). The general process of data collection is documented below.
Figure 3.2: Contrasting Facilities at Communities of the Study

Boughsheds and tents as housing

New housing

Solar pack to pump water
3.3.1 Identification of Dogs

With the incumbent difficulties in communication and abundance of dogs without ‘names’, permanent identification of dogs with microchips was necessary. Prior to identification, signalment data was collected with the owner’s consent.

3.3.1.1 Sex

Dogs were recorded as being either entire male or female unless their owners indicated that they had been ‘operated-on’ or ‘fixed up’ to produce sterility.

3.3.1.2 Age

Age and time concepts for Aboriginal and non-Aboriginal people in some cases are not equivalent. Owners were asked for their estimation of birth date of dogs. In some cases this was related to a particular event in the area, e.g. last rodeo season. Puppies’ ages were estimated based on dentition (see Springhall, 1967; Harvey, 1985) and weaning status.

Dogs were later categorized for population statistics as puppy (0-6 months), juvenile (6 months to 1 year) or adult (greater than one year) (based on Pence et al, 1983; Philips and Scheck, 1991).

3.3.1.3 Breed

The breed of dog was recorded. In the case of mixed breeds, estimation of crosses based on appearance was made. This information was most useful for locating dogs at subsequent visits. For example, dog colours and sizes often sparked people’s memory of the dog in question.

3.3.1.4 Name of Dog

Names of dogs were recorded to help identification at the following visits, but in many cases dogs were without names. It is not known if this was because of the difficulty owners had in
explaining 'Aboriginal' names of some dogs, although Howe (1993) did find that 87% of dogs did not have names in the Aboriginal communities she surveyed.

### 3.3.1.5 Name of Owner

Where appropriate, the name of the owner or 'boss' was recorded to assist in locating dogs and to determine the natural place of residence of the dog at night. While dogs in communities are often free to roam, Howe (1993) only found 4% of dogs to be stray (without owners) and 71% of dogs observed roaming were in the company of humans. Determining the owner of each dog assisted in analysing the dog distribution within communities.

### 3.3.1.6 House Number

The dog's place of residence or location was recorded as the number of the house the dog normally spent most of its time. It was noted that dogs frequently remained at their household even if the owners were absent for a short period of time.

### 3.3.1.7 Origin of Dog and Status

Where the owner was available, the origin of the dog was obtained. This was not always successful due to language barriers and memory difficulties. At subsequent visits, owners were also asked the whereabouts of missing dogs. It was assumed that after 4 missing periods (one year) the dog was either dead or had moved. These dogs were then removed from the listings after the last date they were observed.

Each dog was given a status number according to its whereabouts. Dogs were recorded as:

1. Alive and treated,
2. Alive, but not caught or treated,
3. New to community
4. Visiting community temporarily and treated,
5. Moved away to another community (usually outstation),

5. Missing/presumed dead or

6. Confirmed dead.

The total number of dogs that were recorded as alive and treated (status 1), alive but not caught or treated (status 2) and new to community (status 3) were assumed to make up the population of dogs in each community.

3.3.1.8 Microchips

Transponder microchips (‘TROVAN’ Passive Transponder System ID-100), each with an individual identification number, were inserted subcutaneously between the shoulder blades of all dogs of reasonable size (usually over 8 weeks of age). This system was accepted well by owners and was seen as a novel idea.

**Figure 3.3: Transponder Microchip Reader and Microchip**
After each dog was identified, examined and sampled, a mark of paint was placed on the rump to aid in visual identification and prevent recapture of the dog on that visit. This was also well accepted as it served as a reminder to community people which dogs had been treated on that day.

At the first visit, coloured collars were provided for all examined dogs as a promotional tool. Again, this was a novel idea even though most collars were subsequently removed by children (and used as decorative items!)

**Figure 3.4: Dogs Wearing Coloured Collars**

---

### 3.3.2 Capture and Restraint

Owner assistance was necessary for capture and restraint of most dogs. In some cases it required a household effort and help by community members comfortable with animal handling. Every dog was muzzled to prevent injury and to avoid possible ‘discrimination’ of different owner’s dogs. In difficult circumstances, ‘rabies poles’ (aluminium poles with retractable nooses) were employed.
3.3.3 Education and Community Support

Community support was not only necessary for catching dogs, but also for conveyance of the program’s health messages. Initially, ‘tailor-made’ videos featuring a well known local actor were developed to be displayed at shops, council offices or schools to notify people of the project team’s arrival and to explain the procedures. Although the videos were successful, any deaths of people featured in the videos meant that the videos had to re-edited to remove that person’s image.

In addition to videos, Environmental Health Workers (EHW) acted as facilitators and interpreters. Most of the explanation of zoonoses, population control and methods used was done in person with the assistance of the EHWs and other interested community members. Traditional methods of community education appropriate for use in western-based cultures (such as pamphlets and posters) were not as effective as person-to-person contact. Showing fixed samples of nematodes and the types of needles and syringes used as well as ‘before and after’ photos of successful cases was very beneficial in encouraging participation and disseminating the health message. Environmental Health Workers were also trained during the
program within their local environment to avoid the problems of isolating individuals and to emphasise the relevance of the training for a community setting. Taking time to explain issues and demonstrating processes are the best forms of 'education' for Aboriginal people (Anon, 1994).

**Figure 3.6: Examination of Dogs Generated Interest in the Community**

Communication with the councils was regular and on going. Half-yearly reports were sent to councils outlining the results, level of participation and appreciation for community support. Specifically, individual results of microfilariae tests for *Dirofilaria immitis* were posted at the requests of the communities so owners could make arrangements for treatment if necessary.

### 3.3.4 Drug Treatments

All methods of treatment were designed with ease of application in mind so that the program could be continued at the completion of the study.
The first visit to the (majority of) communities in March 1992 was to explain the program to community people, identify as many dogs as possible, collect baseline data and samples and to become familiarised with the region and communities.

Subsequent three monthly visits were to collect information and treat dogs.

3.3.4.1 Daily Routine

Avoidance of diurnal variations in egg counts (Nwosu and Anya, 1980; Anderson and Schad, 1985) was attempted by conducting the program during the mornings between 7.30 am and 12.00pm. Visits were timed to cause minimal disruption to community routine and were made house-to-house to encourage participation (Haswell-Elkins, Elkins, Manjula, Michael and Anderson, 1988) and aid in education.

3.3.4.2 Frequency of Treatments

Treatments were administered 3 monthly. Reasons for this included:

(a) Routine visits to communities allow adequate preparation by the community. In studies of human helminth control, rigid scheduling is considered a prerequisite (Botero and Brugmans, 1989).

(b) Previous 'dog control programs' revolved around 3 monthly visits, so by keeping the same frequency, confusion was reduced, and

(c) Three to six monthly anthelmintic treatment is most often recommended for continuing control of *Ancylostoma caninum* and *Toxocara canis* in adult dogs (Urquhart, Armour, Duncan, Dunn and Jennings, 1987). In areas were there is perennial transmission of helminths, frequent chemotherapy, such as 3 monthly, is likely to produce better control and reduction in morbidity (Migasena and Gilles, 1991). Seasonal transmission of some parasites was likely in this study, although this information had not been established prior
to the commencement of the program. As such, fewer treatments would have been sufficient.

This rigid timetabling prevented the treatment of puppies at rates required to control all worms. Control of *Toxocara*, for instance, requires treatments to be commenced at 2 (English, 1982; Lloyd, 1986; Overgaauw, 1997) to 4 weeks (Schantz, 1994) of age and continued every week or two until weaning (English, 1982; Overgaauw, 1997). Likewise, for complete treatment of scabies, two treatments 14 days apart is recommended (Thimmappa Rai and Yathira, 1988). In the scope of this program, these recommendations were not practical.

### 3.3.4.3 Mass Chemotherapy Campaign

Helminth control programs in humans are generally of 3 types; mass application of all community members, selective population chemotherapy of persons that are found to be infected at the initial or subsequent surveys (Migasena and Gilles, 1991), and targeted chemotherapy of individuals with heavy burdens or exhibiting signs of infection (Anderson and May, 1982; Upatham, Viyanant, Brockelman, Kurathong, Ardsungnoen and Chindaphol, 1992).

Selective treatment of heavily infected individuals can provide cost-effective community-wide helminth control, but additional expenses arise from identifying heavily infected individuals (Anderson and May, 1982; Schad and Anderson, 1985). Also, Upatham *et al* (1992) consider targeted chemotherapy (in human populations) only to be effective when used in conjunction with ‘targeted education’ and ‘targeted environmental intervention’.

Mass chemotherapy was chosen not only for its effectiveness against helminths and ectoparasites, but also for the easy of application and explanation to animal owners. All dogs over 6 weeks of age (post weaning) were treated with ivermectin.
Parasite treatments at most communities were commenced in June 1992, three months after the first visit to collect baseline information. Apart from the aforementioned reasons, treatments were commenced at the second visit because it was necessary to determine the heartworm status of dogs (in March 1992) prior to ivermectin administration. Definitive information regarding the effect of ivermectin treatment in microfilariae positive dogs was lacking, with most reports of reactions related to the use of diethylcarbamazine as a prophylactic treatment. Prior to treatments, owners were warned of possible reactions and asked to observe their dogs for any signs of acute toxicity or shock-like reaction.

3.3.4.4 Ivermectin treatment

Ivermectin (‘IVOMEC’ antiparasitic injection for cattle, Merial Australia, 10g/L) was administered by subcutaneous injection at the rate of 200μg/kg (see Figure 3.7). At this dose rate and route of administration, ivermectin is effective against immature and adult stages of *Ancylostoma caninum*, *A. braziliense*, *Ucinaria stenocephala*, *Toxocara canis* and against immature *Toxascaris leonina* and adult *Trichuris vulpis* (Yazwinski et al, 1982). Third and fourth stage larvae of *D. immitis* are also susceptible to ivermectin at this dose rate (Campbell, 1989). A single dose of ivermectin is also able to kill *Sarcoptes scabiei* in single housed dogs (Yazwinski et al, 1981), but at the time of the establishment of the program it was unknown what effect a single dose would have on natural infestations in dogs housed together (see 2.4.3.2.2).

3.3.4.5 Proligestone administration

Proligestone (‘COVINAN’ Intervet (Australia) Pty Ltd, 100mg/mL) was administered subcutaneously to non-pregnant bitches over 6 months of age at 10-30mg/kg at the request or consent of the owner.
As not all bitches were at the same stage of their cycle and some were just starting pro-oestrus, owners were notified of the potential for bitches to experience oestrus while receiving the contraceptive, especially during the first few treatments.

Unlike ivermectin, proligestone treatments were commenced at the first visit in March 1992. This was done to compensate for a potential increase in fecundity associated with improved health following anthelmintic treatments, as has been observed in human communities treated for hookworm (Crompton and Stephenson, 1990).

3.3.5 Physical Examination
Every captured dog was examined at each visit.

Reproduction Data
Bitches were checked for signs of pregnancy (mammary development, milk production, enlarged abdomen) and owners were questioned whether the bitches had had oestrus since the previous visit. Generally owners were quite aware of their bitches’ reproductive state.

Dog Weights
Dogs were weighed for treatment dosage and determination of the effect of treatment on weight using domestic scales. Females in the latter half of gestation were excluded from weight data because of the effects of variable fetal mass on total body mass. Likewise, growing dog’s weights were excluded from weight data calculations.

Weights for each dog were compared between visits and recorded only as increased, decreased or remaining the same, irrespective of the amount of weight gained or lost. Dogs were of different size and structure, so absolute numbers could not be used for comparison. Even so, with the sensitivity of domestic scales being low, the changes in weight would have been more
evident in dogs of larger size and alteration in weight status for smaller dogs may have been missed.

**Scabies Assessment**

Dogs exhibiting signs of erythematous, nonfollicular papular dermatitis with alopecia typical of canine scabies (Scott *et al*, 1995) were further scored on the extent of the changes. Each dog was categorised according to the 'percentage of body involved' into scores 1 (0-25%) through to 4 (75-100%) (see Appendix B; Figures 3.8-3.10). A similar method was used with success by Pence *et al* (1983) when conducting scabies assessments of coyotes in southern Texas. Correlation between prevalence of mite infestation and severity of papular dermatitis lesions of groups of pigs to monitor scabies status has also been demonstrated (Davies, Bahnson, Grass, Marsh, Garcia, Melancon and Dial, 1996).

*Figure 3.8: Dog with Lesions Indicative of Scabies – Score 2*
Figure 3.9: Dog with Lesions Indicative of Scabies – Score 3

Figure 3.10: Dog with Lesions Indicative of Scabies – Score 4

There are many differential diagnoses for Sarcopes infestation (see 2.2.1.3) although the distribution of lesions associated with pruritus in classical scabies is quite pathognomic. The
determination of the resolution of the skin lesions by means of the skin scoring system was used to monitor the efficacy of the treatment. A similar methodology was used by Paradis and colleagues (1997) in determining the efficacy of topical ivermectin in treatment of canine scabies in a clinical pen trial. Similarly, Arlian, Morgan, Rapp and Vyszenski-Moher (1996) used a clinical score system to determine the effects of reinfection of scabies mites in dogs with success.

Dogs that were refractory to treatment had their skin scraped from at least 10 locations including the periphery of early lesions and margins of ears to determine whether *Sarcoptes scabiei* mites, eggs or nymphs were present. Diagnosis by skin scrapings is reported to be only 20-50% successful (Dunsmore and Shaw, 1990; Burton, 1997). In addition, mites are often removed with the scurf as dogs scratch (Schwartzman *et al.*, 1967). For these reasons, diagnosis by treatment trials is often used (Dunsmore and Shaw, 1990; Burton, 1997; including human surveys, see Blumenthal, Taplin and Schultz, 1976). Deep skin scrapings were collected and preserved in 70% ethyl alcohol with 5% glycerin for further light microscope examination.

**General Examination**

The general physical examination was completed with the reporting of any orthopaedic or traumatic wounds including fractures, scalding, and dog bites. Other miscellaneous conditions such as otitis externa and abscesses were recorded and treated if possible. Special considerations of the reproductive organs were made. Signs of endometrial infection (suppuration or staining) were noted.

**3.3.6 Sample Collection**

Samples were usually collected before treatments. As both ‘IVOMEC’ and ‘COVINAN’ caused some skin irritation and mild pain (personal observation), it was more conducive to collect samples first to reduce disruption to the dogs.
3.3.6.1 Ectoparasite sampling

Skin scrapings were taken from refractory cases. Ticks, lice and fleas were also collected periodically from randomly selected dogs for identification. Ticks that were of morphological difference to the majority of ticks found on dogs were also collected for species identification. All the arthropod samples were preserved in 70% ethyl alcohol with 5% glycerin (Soulsby, 1982; Dunsmore and Shaw, 1990) to prevent evaporation and distortion of the structures.

3.3.6.2 Faecal Samples

Faecal samples for parasite identification were collected 6 monthly initially and then 3 monthly for the majority of communities. As the prevalence of helminths (hookworm in particular) was expected to decrease slowly, samples were only collected six monthly initially. A greater seasonal variation than expected was noticed, so collection was continued 3 monthly from September 1992. Dogs from Kalumburu and Looma were sampled 3 monthly from the beginning of the program at these communities (March 1993 and September 1992, respectively). As a result, Kalumburu and Looma are considered as special case studies separate from the ‘mainstream’ communities of the coastal, central and eastern regions.

Approximately 5g of faeces was collected per rectum to ensure freshness and correct identification of samples. All samples from dogs of reasonable size (usually over 4 months) were collected in this manner. Smaller dogs were swabbed or observed for defaecation and samples collected.

All faecal samples were completely mixed and preserved in 10% buffered formalin within 4 hours of collection (Dunsmore and Shaw, 1990) at the ratio of 2-5 parts faeces to 1 part solution (Bartlett, Harper, Smith, Verbanae and Smith, 1978; Urquhart et al, 1987; Dunsmore and Shaw, 1990) to minimise development and hatching of eggs (Urquhart et al, 1987). As
access to temperature controlled rooms for immediate processing of samples was not possible, preservation for further examination was necessary. Formalin fixation also prevents distortion commonly associated with salt solutions of high specific gravity used in many helminthological tests (Bartlett et al, 1978).

Samples were sent to the main laboratory at Murdoch University, Perth, by road.

In June 1992, faecal swabs collected per rectum were preserved in transport media for culture and identification of Campylobacter and Salmonella bacteria.

### 3.3.6.3 Blood Samples

Blood for hydatid serology, Dirofilaria immitis microfilariae tests, serum protein determination and packed cell volume readings was collected 3 monthly (once) and then 6 monthly. Five to 10mL of whole blood collected via cephalic and jugular veins was preserved in dipotassium ethylene-diamine-tetra-acetate (EDTA) or plain tubes ('VACUTAINER' Becton Dickinson, 10mL and 5mL) and refrigerated. Serum was frozen in sterile tubes for transport to Perth.

Overall the difficulties in sample collection were related to capture of animals. Controlling temperatures of storage samples was also difficult with unexpected events such as the freezing over of refrigerators in the dry season which were set for wet season conditions.

### 3.3.7 Treatment of Samples

#### 3.3.7.1 Skin Scraping Samples

Preserved samples were pooled for each dog and lysed with potassium hydroxide (KOH) solution (10 volumes KOH: 1 volume of skin scraping) at 90°C for about 15 minutes (modified method from Soulsby, 1982). The sediment was centrifuged at 3000rpm for 3 minutes. The supernatant was poured off, and the sediment re-suspended in distilled water to remove any
soap (developed from KOH and fat). The samples were re-centrifuged and the sediment examined under a light microscope for mites. Evidence of eggs, nymphs or adults was considered diagnostic for *Sarcoptes scabiei*. *Demodex* mites and products were also examined and considered diagnostic if there were large numbers isolated, and the animals were refractory to treatment with ivermectin, demonstrated pustular skin lesions indicative of demodecosis and were not excessively pruritic.

Sections of whole skin samples from euthanased dogs exhibiting mange were also examined. Formalin preserved skin samples were scraped to the level of the dermis in the laboratory and the sediment transferred to a beaker containing 50mL of KOH at 90°C and digested for 15 minutes until the epidermal scale and hair was dissolved (modified method from Soulsby, 1982). The resultant suspension was poured through 0.1mm filter and flushed with distilled water. The flushings were examined on a petri dish with a binocular dissection microscope (40x).

### 3.3.7.2 Faecal Samples

**Parasite Examination**

Due to the lack of laboratory space, refrigeration to preserve faecal samples and time to do parasite examination of faeces immediately after collection, a method was sought that could use preserved samples. After a preliminary trial at Kununurra (see 5.4.1, methodology discussed below) testing the effectiveness of formalin ethyl acetate sedimentation technique (FEAS) (Young, Bullock, Melvin and Spruill, 1979) against ZnSO₄ flotation (described in Dunsmore and Shaw, 1990), to recover helminth eggs, larvae and protozoa, the FEAS method was chosen (see Appendix C).

Preserved faecal samples were examined between one to two months after collection. A single sample of sediment was examined for each sample and each slide was examined under a light
microscope in its entirety using overlapping fields under 10x objective. Identification of specific structures was performed with 40x objectives. Single counting procedures plus low sensitivity of faecal egg concentration can make detection difficult, but multiple sampling was very time consuming for the added benefit. Ideally, worm recovery techniques are preferred as egg counting using formalin ether (or formalin ethyl acetate) sedimentation have been found to be 33% less sensitive (Haswell-Elkins et al, 1988). As all samples were treated the same, errors of this nature were consistent.

Reasons for Not Doing Egg Counts

In many studies of helminths in humans and herbivores, egg-counting procedures are used. This technique was not employed in this study for many reasons.

The relationship between egg output and worm burden does not appear to be linear and is characterised by high variability (Haswell-Elkins et al, 1988). This may be due to sampling heterogeneity or variability due to biological processes such as density dependent depression of parasite fecundity (Anderson and Schad, 1985). As such, egg counts are only considered to be qualitative measures that give an approximation of those suffering high helminth or low helminth burdens.

Stools of most dogs also contained abundant grit and other foreign matter that made determining the consistency difficult and precluded use of tests such as the Kato technique (Anderson and Schad, 1985). Usually varied stool consistencies can be taken into account for determining egg counts in most species (Soulsby, 1982; Anderson and Schad, 1985), but the variety of foreign objects in the stools made this very difficult. Examples of contaminants included plastic bags, cotton wool, fishhooks and splintered bones.
The greatest drawback of only monitoring prevalence rather than intensity (egg counts) of infection is that it may lead to the incorrect conclusion that control measures have little effect on parasite abundance (Anderson and May, 1982).

Identification of Parasites

Morphometric identification of eggs (Dunsmore and Shaw, 1990) was used rather than the specific identification of L3 from faecal cultures (see Appendix D). This method for identification of parasites (including differentiation of hookworm species) has been evaluated and considered to be reliable. In a study comparing known hookworm species (Uncinaria stenocephala and Ancylostoma caninum), even with an overlap in egg sizes, the method was suitable in more than 95% of cases (Moore and O’Callaghan, 1985).

Bacterial Isolation

Per rectal swabs from dogs were collected in June 1992 (from most communities) and September 1992 (from Looma) for the isolation of enteric bacteria. The faecal sample swabs were placed into Campylobacter broth transport media and faeces transport media for the growth and isolation of Campylobacter jejuni and Salmonella typhimurium, respectively. The samples were then sent to the Western Australia State Health Laboratories in Perth by airplane and always within two days.

At Perth, the Campylobacter broth samples were incubated at 42°C and plated onto Campy-BAP medium and re-incubated for 48 hours in a microaerophilic environment. The Salmonella samples were inoculated into a selective enrichment medium (strontium chloride B broth) then incubated for 24 hours at 42°C before plating onto a selective medium (desoxycholate agar, DCA).
Colonies isolated from the incubated selective medium plates were then biochemically and serologically tested for the identification of *Campylobacter jejuni* and *Salmonella typhimurium*, as described elsewhere (Buckle, Davey, Eyles, Hocking, Newton and Stuttard, 1989).

**3.3.7.3 Blood Samples**

**Heartworm Testing**

Preserved blood was examined for the presence of microfilariae by filtration using the Wylie method (described in Dunsmore and Shaw, 1990) (Wylie, 1970). *D. immitis* microfilariae were differentiated from *Dipetalonema reconditum* microfilariae by size and morphology (Kelly, 1973; Dunsmore and Shaw, 1990). Once a sample was found positive for *D. immitis*, no further examination for *D. reconditum* was carried out. In March 1992, blood was collected from 468 juvenile or adult dogs at 12 locations and in March 1993, blood was collected from a further 71 from Kalumburu prior to treatment. At intervals between 6 and 12 months after the initial sampling, repeat sampling was performed at 12 communities. As mentioned, the objective of identifying microfilariae (rather than using serological tests) was to determine if any reactions to treatment could be correlated to microfilariae status.

**Total Plasma Protein and Packed Cell Volume**

Total plasma protein and packed cell volume were measured from 308 blood samples in September 1992 to give an indication of the hydration status of each animal. The hydration status of the sampled dogs was estimated because a decreased blood volume (dehydration) results in spurious increases in haematological concentrations. Dehydration is indicated by hyperproteinaemia with an elevated albumin level, but a normal albumin to globulin ratio. For the survey of September 1992, albumin and globulin levels could not be measured, so dehydration was suspected based on elevated plasma protein (total solids) readings.
EDTA preserved blood samples in micro-capillary tubes were centrifuged at 12 000 revolutions/minute for 3 minutes. The haematocrit reading was determined by reading the packed red cell volume up to the level of the plasma in each tube. The microcapillary tubes were then snapped immediately above the buffy coat and the plasma poured off onto the glass surface of a refractometer. The total solids reading was then taken to give a rough estimate of the plasma protein. Haemolysed samples were not examined to avoid false results.

The normal values for packed cell volume are between 0.37-0.55L/L (Duncan and Prasse, 1986) and the normal values for plasma protein, based on refractometry, are between 60 and 75g/L (Duncan and Prasse, 1986) (see Table 6.1). Haemolysed samples were not used as haemolysis causes mild elevations in plasma protein refractometer readings (Duncan and Prasse, 1986).

**Hydatid Serology**

Diagnosis of *Echinococcus granulosus* by egg or proglottid detection in faeces is difficult because the eggs are sparsely shed (Urquhart et al., 1987). The eggs of *Echinococcus* (which can be liberated from the proglottid prior to exit from the host) are also morphologically indistinguishable at the light microscope level from various *Taenia* species (Soulsby, 1982; Thompson, 1995). For these reasons, hydatid serum antibody ELISA tests to detect IgG, IgM, IgA and IgE were conducted on 430 sera samples.

The samples were randomly chosen from dogs sampled during the three-year period. The ELISA test has been previously described by Gasser, Jenkins, Paolillo, Parada, Cabrera and Craig (1993) and was developed from protoscoleces antigen preparations to detect adult antibodies (IgG). The sensitivity ranges between 73% and 84% (Gasser et al., 1993; Thompson, Robertson, Gasser and Constantine, 1993b) and the specificity is approximately 98% when using post mortem and arecoline purgation confirmation. In other field trials in Uruguay (Craig
et al, 1995) serum IgG-ELISA had a sensitivity of 34.6% when used alone. When used in combination with IgE and IgA, the sensitivity increased to 69.2%. A degree of geographic variability in sensitivity has been observed and this may be due to differing worm burdens (Gasser et al, 1993).

The alternative ante-mortem diagnosis of hydatidosis currently relies on arecoline dosing and detailed examination of the purge for adult worms. Disadvantages of purgation include; variable sensitivity, logistically difficult to carry out en masse, very time consuming, requires trained personnel, biohazards, may cause distress to some dogs, and has a high failure rate (27% did not successfully purge in one study) (Craig et al, 1995). Purgation is always 100% specific for Echinococcus (Craig et al, 1995).

3.4 Child Health Data Collection

The conclusion of an integral approach to studying the effects of a population and parasite control program in dogs in communities required information on the health status of the people. As most information regarding Aboriginal health is based around child health and many zoonoses are more common in children, a variety of data were collected from 0-15 years old community members.

Clinical surveys of skin diseases were conducted at four communities during June in 1992 and 1993. Medical practitioners were requested to complete forms inquiring the nature of skin infections and distribution (see Appendix E). These surveys were often conducted as part of the normal health screening tests for otitis or trachoma in 5-15 years old children. Examples of skin diagnoses include scabies, impetigo, boils and tinea.

In March 1992, faeces from 62 children under 5 years of age from 5 communities were collected for general parasitological examination using the formalin ethyl acetate sedimentation
technique. The samples were also tested for *Campylobacter* spp. and *Salmonella* spp. as described (3.2.7.2)

At the end of active intervention in the communities a mass retrospective study of hospital and clinic records was undertaken to record any information related to environmental or dog health parameters.

Skin infections and afflictions that were recorded included; scabies, impetigo, ringworm, boils, 'sores', blisters, abscesses, burns, allergic dermatitis, cellulitis, lacerations, ulcers, urticaria and dog bites. Yearly incidence data was compiled for scabies infections and general skin infections (pyoderma). The pyoderma group included all cases of impetigo, abscess, cellulitis and infected abrasions and bites, but did not include any cases of scabies, which may have shown secondary pyoderma.

For gastrointestinal conditions, all presentations and diagnoses of diarrhoea, vomiting, *Giardia*, *Salmonella*, *Campylobacter*, *Strongyloides stercoralis*, *Hymenolepis nana*, *Blastocystis hominis*, *Entamoeba histolytica* or *E. coli*, *Cryptosporidium*, *Shigella*, *Ancylostoma duodenale*, enteropathogenic *Escherishia coli*, *Trichuris trichiura*, *Aeromonas hydrophilia*, *Edwardsiella tarda*, *Isospora belli*, *Enterobius vermicularis* and rotavirus were recorded. The category of 'diarrhoeal disease' included those cases for which a definitive cause was found as well as those of undetermined cause.

Data was compiled for all 0-15 years old children in four communities from 1990 to 1995. A control community was also examined. This community had similar health services and history and was within the same location and of the same size as another intervened community. Overall 985 records were examined for 693 children from five communities.
Clinical and hospital data is notorious for biases including differing diagnosticians and diagnoses, attendance rates and health programs. In the mid 1980s, a maternal and child health program was established in many communities which resulted in nurses visiting communities on a weekly basis to monitor health and provide treatments. This drastically increased the amount of data recorded. In addition, compliance rates of mothers and their children vary between individuals and communities. Other variations include the difference in those communities close to towns and hospitals and those in remote areas. In each case data was collected from all possible health outposts to minimise discrepancies.

In all cases the major problem encountered was the frequent changing of medical staff in the region. This prevented consistency in data collection and diagnosis.

3.5 Statistical Analyses

A variety of statistical analyses were used throughout the thesis. Statistical analyses were used to:

(a) Describe individual data

Descriptive statistics such as mean, standard error and proportion were used to describe individual groups of data.

(b) Compare two unpaired groups

Chi squared tests were used to test the null hypothesis that two (or more) portions were equal in the overall population. The use of Chi squared tests is based on several assumptions (Motulsky, 1995) which were met in the present study:

1. The data must be randomly selected from, or at least representative of, a larger population.
2. The data must form a contingency table,
3. The values must not be too small. Where expected values were less than 5, Fisher’s test was used.
4. The samples must be independent without any matching.

Data from 2x2 contingency tables were sometimes summarised as odds ratios. An odds ratio is the ratio of the odds in the at-risk group to the odds in the not-at-risk group. Odds are defined as the probability that the event will occur divided by the probability that the event will not occur.

(c) Compare two paired groups

The paired t test was used to test whether the means of two matched groups of parametric data were different.

(d) Compare three or more unmatched groups

When measurements were made from Gaussian distributions (symmetrical bell shaped distribution, parametric data), the one-way ANOVA test was used to test if population group means differed significantly from each other. For data expressed as ranks or scores (non-Gaussian populations), the Kruskal-Wallis one-way ANOVA test was used. For proportions (binomial data), the Chi-squared test was used to determine statistical difference between data sets expressed in contingency tables.

(e) Quantify the association between two variables

For measurements from Gaussian populations, the Pearson correlation test was used to quantify the association between two variables.

Interpretation of P Values

P values were generated for each of the comparison and association tests described. The level of significance was set at 0.05. Data were pooled when there was no statistically significant difference between two populations (i.e. P> 0.05).
Confidence intervals

Ninety five percent confidence intervals were calculated for odds ratios and proportions (including those expressed in graphs). The 95% confidence intervals displayed as bars on the graphs and in brackets ([ ] ) throughout this thesis represent the range of proportions in which there was a 95% probability that the range included the true population value (Motulsky, 1995).
Chapter 4
CANINE POPULATION STRUCTURE, DISTRIBUTION AND DYNAMICS

4.1 Introduction
Population data from many dog populations across the world have been collated for many important uses (Franti and Kraus, 1974):

(a) Effective population control programs require knowledge of the distribution and dynamics of existing populations (Nassar and Fluke, 1991).

(b) Zoonotic diseases are also best controlled if population information is available, as susceptible groups within society can be targeted for education on the prevention of these diseases.

(c) The planning of provisions of veterinary services also relies on information relating to human and pet populations.

(d) Knowledge of pet populations can also be useful in studies of human behavior and health because of the intimate relationship between humans and pets.

Dog population information in remote Aboriginal communities is equally important, to enable the development of effective breeding and parasite control programs.

4.2 Methodology
Estimates of dog population structure, density and dynamics in remote Aboriginal communities are complicated by many factors. As the dog populations in Aboriginal communities are neither entirely domesticated, nor wild, the methodology used in the present study needed to consider aspects of both types of sampling techniques. For estimations of wild animal populations, the Lincoln Index technique, based on capture-mark-recapture, is commonly used (Overton and Davis, 1969).
The formula is;

\[
\frac{N_p}{N_{mp}} = \frac{N_r}{N_{mr}}
\]

where

\(N_p\) is the number in the population

\(N_{mp}\) is the number marked in the population

\(N_r\) is the number in recaptured sample

\(N_{mr}\) is the number of marked animals in the recaptured sample.

Unfortunately this method (and others like this) must meet certain assumptions to allow unbiased estimates. The population must be closed to migrations of dogs in (births and immigration) and out (deaths and emigration) of the area and all animals (marked and unmarked) must be equally likely to be captured in each sample (Seber, 1973, cited in Childs, Robinson, Sadek, Madden, Miranda and Miranda, 1998). In the present study, these assumptions could not be made because the animals were owned. Owners influence the migrations of animals in and out of communities and the success of recapture is reliant on the owners catching the dogs, i.e. some dogs will be captured more often than others because of the ease of restraint.

In most non-Aboriginal communities, techniques for surveys of owned dog populations are based on telephone, mail or personal interview of a randomly selected proportion of the population and are well developed and effective (Franti and Kraus, 1974; Schneider and Vaida, 1975; Griffiths and Brenner, 1977; Nassar and Mosier, 1980; Nassar et al, 1984; Nassar and Mosier, 1984; Robertson, Edwards, Shaw and Clark, 1990; Ticman and Carlos, 1992; Odendaal, 1994; Leslie, Meek, Kawah and McKeon, 1994; Patronek, Beck and Glickman, 1997). In the present study, the objective was to treat as many dogs as possible, and as such, randomly selected households for interview would not have been effective. The small size of communities and the difficulties in communication also preclude such surveying techniques.
To overcome the problems with determining population characteristics, a combination of detailed non-structured interview of owners (and other community members) along with capture of as many dogs as possible at each three monthly visit was used.

Data collected from the communities were pooled according to location, structure, ethnicity and cultural association of the communities to provide three broad regions; coastal, central and eastern (see 3.2.2.2). Within each region, data collected over the duration of the program was pooled if no statistical difference was found between each visit’s data.

4.2.1 Data Collection and Processing

Data regarding population structure, distribution and dynamics were collected every three months from March 1992 to June 1995. Data from March 1992 was excluded from most calculations, as many younger dogs were not presented for examination.

At each visit, dogs were assigned a ‘status number’ indicating the fate of each dog. Owners and other community members were specifically asked the whereabouts of individual dogs (which had been permanently identified with microchips). Dog names and physical characteristics were noted for each dog to help identification of the dogs during the interviews. As described previously (3.3.1.7), the states assigned to each dog at each visit were; alive and treated (status 1), alive but not caught or treated (status 2), new to community (status 3), visitor (status 4), moved to another community (usually outstation) (status 5), missing/presumed dead (status 6), and confirmed dead (status 7). Data collected in March 1995 and June 1995 was used to confirm the status of dogs that were alive or missing at the last treatment in September 1994. That is, if the dogs were seen after September 1994, they were then considered alive but not treated at the last data collection (status 2). Likewise, if any dogs were not treated or seen for 12 months, they were presumed dead (status 6).
4.2.1.1 Population Structure

The structure of the population included the population size and the sex and age characteristics. Population size at each visit was determined from the estimated numbers of dogs that were alive at each visit, including those that missed treatment or were new to the community (status 1, 2 and 3).

4.2.1.2 Population Distribution

Population distribution information included the numbers of dogs per household, the percentage of households with dogs, the number of dogs per dog-owning-household and the percentage of dogs living in single habitat or in ‘multiple-dog’ households. Multiple-dog households, in the present study, were described as households owning more than 4 dogs. In determining the household distribution of dogs, vacated households were not included in the calculations. It is customary in some communities for houses to be vacated if someone from that household has died. In this case, houses may be vacant for months up to years.

Characteristics about the owners of dogs (such as their gender and age) were also included in this section. Estimation of the number of people living at each community was done to determine the ratio of dogs to people. Government census data was available for 1991, but specific information regarding place of abode and dates of birth of people in communities had to be determined from hospital and clinic records. A complete census was made for September 1994 after examination of government census data, community health centre records, hospital records and lists of owners as determined by the project.

4.2.1.3 Population Dynamics

Overall population dynamics addressed the outward (emigration and deaths) and inward (immigration and births) movement of dogs in communities.
Mortality rates were calculated for each three monthly visit from the number of dogs alive at each visit that had subsequently died or gone missing before the next visit. The result is presented as a percentage of the dog population at the original visit. The cause of death was also ascertained where possible and the population structure and household distribution characteristics of dogs that died were also calculated.

The inward migration rate at each visit was determined from calculating the percentage of all dogs that were new to the program (including those born in the communities) within the three month period between visits. The percentage of dogs born within the communities was also calculated separately. Other calculations referred to the structure and distribution of new dogs to the communities at each visit.

The overall effect of the population control was expressed as the rate of change of population every three months. The rate of change of population was calculated as the ratio of the population size at one visit to the population size of the previous visit. That is;

\[ N_t = N_0 \lambda_t \]

where

- \( N_0 \) is the initial population size,
- \( N_t \) is the size of the population at time \( t \) and
- \( \lambda_t \) is the rate of change of population over timespan \( t \) (Nassar and Mosier, 1980).

Estimates of reproduction rates of bitches and determination of pregnancy were achieved through questioning of owners about the bitches' reproductive activities such as oestrus and mating and clinical examination for abdominal swelling and mammary development. Owners were also asked about any litters of puppies being born between visits. All females were also examined for signs of pro-oestrus or oestrus and mammary development. Estimates of reproduction rate based only on the numbers of litters born would not have been sufficient for this exercise as some litters may have died between visits.
This information was compiled and approximate dates of conception were extrapolated to give an indication of the effectiveness of the contraceptive treatment. In the case of puppies, the estimated date of birth was determined and then taken back approximately 2 months (63 day gestation period) to the nearest visit to determine whether the bitch had been treated with proligestone prior to conception. As such, the month of mating or conception is an estimate within approximately one month of the actual date. Treatment histories could then be compiled for each of the bitches that had conceived during the program.

4.2.2 Survey of Dog Ownership in Kimberley Urban Communities

In March 1995, 175 householders in major population centres through the Kimberley region were telephoned as part of a survey to determine the dog ownership in the townships (Appendix F). The information from the survey was used as a comparison for the information collected from the project communities and as such was analysed as described for the data from the communities.

Households were selected randomly from the northwest region telephone directory (1995) using computer-generated numbers for the page, column (1-4) and position of the number down the page (approximately 1-100). The next household down the list was chosen if there were no answers to calls on three consecutive days or nights, if the phone was disconnected or if the telephone number was a business. New numbers were selected to replace those where the household did not own a dog or the people declined to answer any questions.

4.2.2.1 Problems with the Telephone Survey

Many problems with the survey were encountered. As the population of the Kimberley was relatively small and the telephone book included all people in the Pilbara, Kimberley and Midwest regions, relevant telephone numbers were difficult to find at times. Frequent thunderstorms at towns many kilometers away interfered with telephone calls. Telephone calls in the region are also expensive and had to be conducted locally where possible.
Biases of the selected population were also probable as the households with telephones were likely to represent a different socioeconomic group than those without telephones and therefore reflect differing pet ownership (Schneider and Vaida, 1975; Robertson et al., 1990). Survey by means of personal interview can eliminate some of these problems, but the vast size of the region precluded this method. Postal surveys also present problems, as many people do not have postal addresses due to the use of post office boxes rather than home delivery in the Kimberley. Response rates by this means are also low and discriminate against those who are not able to read or write.

4.2.2.2 Proportion of Population Reached with the Telephone Survey.

The estimated resident population of the Kimberley region in 1991 was 23 375 with an estimated 17 739 living in the urban centres. The estimated occupied private dwellings (including remote communities) was 7 695 of which 1 846 were caravans or equivalent without telephones (Anon, 1993). In 1995, there were an estimated 4 175 connected residential telephones (Telstra, Perth, Western Australia, personal communication). It can be assumed that only a very small proportion of these telephones was in communities as most communities only have communal telephone services or business connections. Assuming that 90% of urban households were connected (Telstra estimate, personal communication), there were probably around 4 600 households in the urban centres of the Kimberley.

Overall, 175 (out of 4 175) telephone subscribers in the region were surveyed (see Table 4.2). This extrapolates to approximately 3.8% (of 4 600) households in the urban centres. This is similar to the proportions of households surveyed in other studies (Schneider and Vaida, 1975; Franti and Kraus, 1974) (see Appendix G)

Seven people declined to answer questions and a further six could not be contacted because of disconnected lines or absence for more than 3 days.
4.2.3 Statistical Treatment of Data

The statistical methods used in this chapter have been described elsewhere (3.5).

When no statistical difference could be demonstrated between data collected for each visit or region, the data was pooled and is shown in the tables.

The level of significance was set at $P=0.05$ or confidence intervals of 95%, unless stated. Ninety five percent confidence interval bars are shown on graphs expressing proportions and standard error bars are displayed on graphs of other types. Ninety five percent confidence intervals are displayed in brackets ([ ] ) throughout this chapter.

4.3 Population Structure of Dogs

Population structure consists of population size and age and sex characteristics. Population structure information is vital as a basis for information on the dynamics and behaviour of a population.

4.3.1 Results

4.3.1.1 Percentage of Dogs Subsequently Re-presented for Treatment ('Capture Success')

Overall, the percentage of dogs presented at one visit that was presented for re-treatment at the next visit ('capture success') was high for each month. Initially 99% of dogs were re-presented, but this decreased over time to 81% at June 1994 (Figure 4.1).

Three percent of all registered dogs that were missing for one year were subsequently re-presented for treatment (35/1162). This indicates that assuming that dogs that were missing for one year were dead was satisfactory for estimations of population size in the present study.
Population Size

In the mainstream communities, the collective number of dogs seen at each visit ranged from 421 (June 92) to 625 (September 93). Overall, the maximum total estimated population of the entire study brief was 871 in September 1993 (see Table 4.1)

Warmun always had the maximum number of dogs with a range of 155 (J92) to 173 (D93). Joy Springs usually had the lowest number of dogs with a range of 9 to 27 at any visit.

Human population numbers also varied for each community with Warmun having the greatest number (507) and Joy Springs having the smallest (30) in September 1994.

4.3.1.2 Age of Dogs

The dog population was young with the average age of all dogs at each visit being 2 years. Average ages could be determined from only approximately 61% of all dogs as 55% of owners could not provide one or more dog’s ages and some mature dogs’ ages could not be determined by dental examination (Table 4.2).
Table 4.1: Dog Population Size

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>COASTAL</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Beagle Bay*</td>
<td>27</td>
<td>39</td>
<td>34</td>
<td>31</td>
<td>36</td>
<td>41</td>
<td>49</td>
<td>43</td>
<td>35</td>
<td>47</td>
<td>32</td>
</tr>
<tr>
<td></td>
<td>Lombadina/Djarindjin</td>
<td>30</td>
<td>50</td>
<td>50</td>
<td>56</td>
<td>53</td>
<td>44</td>
<td>55</td>
<td>48</td>
<td>52</td>
<td>46</td>
<td>42</td>
</tr>
<tr>
<td></td>
<td>One Arm Point</td>
<td>29</td>
<td>26</td>
<td>32</td>
<td>33</td>
<td>42</td>
<td>54</td>
<td>58</td>
<td>61</td>
<td>61</td>
<td>53</td>
<td>57</td>
</tr>
<tr>
<td></td>
<td>Kalumburu*</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>104</td>
<td>101</td>
<td>102</td>
<td>95</td>
<td>91</td>
<td>82</td>
<td>84</td>
</tr>
<tr>
<td>CENTRAL</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Looma*</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Joy Springs</td>
<td>10</td>
<td>9</td>
<td>15</td>
<td>21</td>
<td>15</td>
<td>15</td>
<td>27</td>
<td>23</td>
<td>22</td>
<td>24</td>
<td>20</td>
</tr>
<tr>
<td></td>
<td>Bayulu</td>
<td>31</td>
<td>42</td>
<td>37</td>
<td>39</td>
<td>41</td>
<td>37</td>
<td>45</td>
<td>43</td>
<td>36</td>
<td>44</td>
<td>45</td>
</tr>
<tr>
<td></td>
<td>Muludja</td>
<td>38</td>
<td>50</td>
<td>61</td>
<td>57</td>
<td>51</td>
<td>47</td>
<td>57</td>
<td>61</td>
<td>57</td>
<td>51</td>
<td>55</td>
</tr>
<tr>
<td></td>
<td>Ngalingkadji</td>
<td>18</td>
<td>18</td>
<td>19</td>
<td>19</td>
<td>24</td>
<td>34</td>
<td>38</td>
<td>38</td>
<td>28</td>
<td>28</td>
<td>27</td>
</tr>
<tr>
<td></td>
<td>Yiyili</td>
<td>22</td>
<td>32</td>
<td>43</td>
<td>31</td>
<td>21</td>
<td>27</td>
<td>31</td>
<td>27</td>
<td>37</td>
<td>25</td>
<td>17</td>
</tr>
<tr>
<td>EASTERN</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Warmun</td>
<td>104</td>
<td>155</td>
<td>153</td>
<td>154</td>
<td>143</td>
<td>143</td>
<td>163</td>
<td>173</td>
<td>172</td>
<td>157</td>
<td>153</td>
</tr>
<tr>
<td></td>
<td>Yaramun*</td>
<td>29</td>
<td>51</td>
<td>59</td>
<td>48</td>
<td>51</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>92</td>
</tr>
</tbody>
</table>

*Not included in most calculations
### Table 4.2: Dog Population Structure

<table>
<thead>
<tr>
<th></th>
<th>Combined Kimberley Communities</th>
<th>Kimberley Urban Centres</th>
<th>Statistical Method*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Data collection</td>
<td>Personal interview</td>
<td>Telephone survey</td>
<td></td>
</tr>
<tr>
<td>Proportion of population interviewed</td>
<td>Up to 100%</td>
<td>4.2%</td>
<td></td>
</tr>
<tr>
<td><strong>SEX</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Males (%)</td>
<td>59</td>
<td>45</td>
<td>$\chi^2$ test</td>
</tr>
<tr>
<td>Females (%)</td>
<td>41</td>
<td>55</td>
<td>$\chi^2$ test</td>
</tr>
<tr>
<td><strong>AGE</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Average age (years)</td>
<td>2.0*</td>
<td>5.0*</td>
<td>One Way ANOVA</td>
</tr>
<tr>
<td>Percentage of owners not knowing one or more dog’s ages</td>
<td>55</td>
<td>0</td>
<td>$\chi^2$ test</td>
</tr>
<tr>
<td>Percentage of dogs that were puppies</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group A (J92 S92 S93 J94 S94)</td>
<td>18.4*</td>
<td>9.2*</td>
<td>$\chi^2$ test</td>
</tr>
<tr>
<td>Odds ratio (compared with urban centres)</td>
<td>2.2 [1.2, 4.3]</td>
<td>9.2*</td>
<td>$\chi^2$ test</td>
</tr>
<tr>
<td>Group B (others)</td>
<td>12.2*</td>
<td>9.2*</td>
<td>$\chi^2$ test</td>
</tr>
<tr>
<td>Odds ratio (compared with urban centres)</td>
<td>1.5 [0.7, 2.7]</td>
<td>9.2*</td>
<td>$\chi^2$ test</td>
</tr>
<tr>
<td>Percentage of dogs that were juveniles (ex S93)</td>
<td>15.9*</td>
<td>3.7*</td>
<td>$\chi^2$ test</td>
</tr>
<tr>
<td>Odds ratio (compared with urban centres)</td>
<td>4.2 [1.5, 11.4]</td>
<td>3.7*</td>
<td>$\chi^2$ test</td>
</tr>
<tr>
<td>Percentage of dogs that were adults (ex J93)</td>
<td>70.7*</td>
<td>78.0*</td>
<td>$\chi^2$ test</td>
</tr>
<tr>
<td>Odds ratio (compared with urban centres)</td>
<td>2.7 [1.5, 4.8]</td>
<td>78.0*</td>
<td>$\chi^2$ test</td>
</tr>
</tbody>
</table>

*Statistical method used to analyse data for pooling to determine combined Kimberley communities’ results and to determine statistical difference between combined communities and Kimberley urban centres

*Statistical difference between combined Kimberley communities and Kimberley urban centres
The average age of dogs in the urban centres in the present study was 5 years and all owners could provide an age.

There were proportionately more puppies in the communities during the dry season than the wet season. A larger proportion of puppies was seen in June 1992 and 1994 and September 1992, 1993 and 1994 (dry season months) for each region. At these times, 18% of the population were puppies. At the other times (wet season months), the average proportion of puppies was 12%.

The survey of Kimberley urban centres showed that the proportion of dogs that were puppies was 9%. When the urban centres were compared with the remote Kimberley communities, the Kimberley communities were found to be 2 [1.1, 4.3] times more likely to have puppies during the dry season that the urban centres at the time of the survey. For the wet season months, there was no difference in the proportion of puppies in the Kimberley communities when compared with the urban centres.

The percentage of juvenile dogs was much more consistent for each visit with 16% of all dogs being aged between 6 months and one year old at every visit except September 1993, where there were fewer (9%). The Kimberley remote communities were 4 times more likely to have juveniles dogs that the townships ([11.4, 1.5]).

In June 1993 there were statistically more adults (80%) than compared with other months (71%) for the Kimberley communities. The townships were 3 times more likely to have adult dogs than the communities ([1.5, 4.8]).
4.3.1.3 Sex of Dogs

There was a bias toward the ownership of male dogs rather than females across all dog age groups, with an increased tendency to own males in the adult population. Overall there were 59% [57.6, 60.4] males and 41% [39.6, 42.4] females (Table 4.2).

Surgical sterilization of dogs was rare, but owners were keen for contraceptive treatments for females. Very small numbers of each sex were neutered with 0.8% [0.5, 1.0] of males castrated and 0.7% [0.5, 0.9] of females spayed. Between 62 and 100% of bitches presented for examination were treated with the contraceptive, which extrapolates to between 62 and 82% of all mature bitches receiving proligestone at each visit.

In the Kimberley urban centres, female dogs were more popular than males (55% [46, 55] vs. 45% [35, 55], respectively). Most females were spayed (63% [51, 75]) and a third of the males were castrated (33% [20, 46]).

4.3.2.4 Dog Breeds

Ninety one percent of dogs from all communities (assessed at the pre-treatment visit) were of mixed breed [88, 94]. The most common crossbreed was Australian Cattle Dog (22% [17, 25] of all dogs), followed by Bull Terrier, Kelpie, undefined Terrier, Dingo and Staffordshire Bull Terrier crossbreeds (see Figure 4.2).

Of the purebred dogs, by far the most common were Australian Cattle Dogs (58% [44, 72]) (Figure 4.3). The other breeds encountered at the communities, in order of decreasing popularity, were Kelpies, Chihuahuas, Bull Terriers, German Shepherds, Collies, Rhodesian Ridgebacks and Staffordshire Bull Terriers.
4.3.2 Discussion

Community size and dog populations varied considerably across the Kimberley region and had effects on many parameters of dog ownership. Each region had large communities of greater than 80 people, but the central region was over-represented with small communities that were outstations (break-away groups) from the larger communities. Warmun, traditionally a gathering point for many Aboriginal people during the station days, had the largest population of both people and dogs.
The largest numbers of dogs across all communities were seen in September 1993. This was due to increased reproduction (see 4.5.3.2).

The dog population of the study was young (average 2 years) and is comparable with another study from the small villages of the Philippines (1.9 years average). Other communities in North America, Canada and Australia have considerably older populations of dogs (4.6 to 6.1 years average) (Nassar andMosier, 1980; Nassar et al, 1984; Nassar and Mosier, 1984; Leslie et al, 1994; Robertson et al, 1990). This different age profile may reflect differing pet ownership and values as the Philippines (Ticman and Carlos, 1992) and Kimberley populations are largely uncontrolled with potentially higher mortality rates and have low levels of sterilization.

In the present study, only 61% of dogs were included in the calculation of average age, due to the inability of all owners to provide their dog’s ages, hence the necessity to categorise dogs as puppies, juveniles and adults for further calculations.

The times at which larger proportions of puppies were in communities followed a seasonal trend with the greatest proportion during the months of June and September. These puppies were up to 3 months of age which indicated that births had occurred somewhere between March and September. Reproductive data from the present study helps to explain some of these trends. The proportion of all dogs that were juveniles in September 1993 was lower than at other times due to the disproportionately high numbers of dogs that were less than 6 months of age.

By comparison with the Kimberley urban townships, the communities had higher proportions of puppies and juvenile dogs, which is reflected by the average age of the populations (2 years vs. 5 years).

Overall, there was a greater proportion of males (59%) in remote communities in the present study. Bias toward male dog ownership has occurred in other populations such as those of Yolo
county in California (56%) and South Africa (54%), but not to such an extent as in the present study. Explanations for male dog popularity in these overseas reports have not been provided, but one possible reason for the bias in the Kimberley is the selective killing of female newborn puppies to help control dog population numbers (personal observation; Howe, 1993). It is often commented that males are more favorable than females for protection, hunting and lack of reproduction.

Sex ratios for litters of wild dingoes in central Australia are biased toward male offspring (1.1 males to 1 female) as are domestic dogs, as determined from a survey in Alice Springs (1.7:1) (Corbett, 1995). The average ratio of the present study (1.44:1) is similar to these figures which may indicate that the ratios are a natural phenomenon rather than a result of human intervention. The older age groups of dogs tend to have even more bias toward males which may be due to a slightly higher mortality rate of females or increased acquisition of male adult dogs (see 4.5.2.1.2, 4.1.1.1), although the data do not support this.

Sterilization of dogs by surgery was rare in the present study. Other studies have found sterilization to be relatively common (particularly in females) (Nassar and Mosier, 1980; Nassar and Mosier, 1984; Nassar et al, 1984; Robertson et al, 1990; Patronek et al, 1997) with the exception of the study of the Yolo (Franti and Kraus, 1974) and Contra Costa (Schneider and Vaida, 1975) counties of California where only 4 and 7.4% of males were castrated, respectively. The Kimberley urban townships had dog sterilization rates (33.3% of males and 66.3% of females) similar to those of Perth (33.8% of males and 69.4% of females) (Robertson et al, 1990). This increased rate compared with the remote communities may, among other factors, indicate the greater availability of veterinary services, a higher disposable income and increased education on dog population control in the urban centres.

Aversion to permanent sterilization by some owners may also explain the low sterilization rates. The acceptability of sterilization of male dogs was not determined in the present study although
some owners indicated that male behavioural and physical traits were important in their dogs. This information may be necessary if contraceptives interfering with testosterone production are to be used to lower male fertility.

Unfortunately data from the Philippines or South Africa were lacking which would have served as interesting comparisons as these countries contain dog populations under similar conditions to the Kimberley communities.

In the present study, when the contraceptive treatments were provided with the program, the contraceptive treatment rate was high (see Figure 4.13). This may indicate the effects of education of dog owners through the program and the previous lack of suitable, low cost veterinary services for provision of breeding control mechanisms.

The types of breeds and crossbreeds found in communities generally reflect the types expected in rural Australia with Australian Cattle Dogs and Kelpies being common. Bull Terrier dogs as well as Dingo crossbreeds were also common. In urban Australia, the most popular breed is German Shepherd, followed by Kelpie, Australian Cattle Dog and Chihuahua (Murray and Penridge, 1997). This is in contrast to urban America and Canada where the most common breeds tend to be Poodles, German Shepherds, Dachshunds and Labrador Retrievers (Franti and Kraus, 1974; Schneider and Vaida, 1975; Leslie et al, 1994). The proportion of crossbreds is much higher in the Kimberley than in other countries surveyed, as only 52.8% (Schneider and Vaida, 1975) and 36.6% (Franti and Kraus, 1974) of dogs from counties of California were mixed breed, compared with 91% in the present survey. This is most likely due to the difficulty and expense in acquiring purebred dogs in remote communities.

The rates of dog bites to children in Aboriginal communities is much higher than elsewhere (see 8.3.3) which may be related to the choice of dogs in communities. Of the six most common crossbreeds in the Kimberley communities, two are crossbreeds of those ranked amongst the six
breeds with the highest attack rates in Australia (Murray and Penridge, 1992). Australian Cattle Dogs, the most common choice of purebred in the Kimberley, are also the second most common dog breed responsible for bites that require hospital treatment, according to one hospital survey (Thomas and Buntine, 1989). Dingo crossbreds, although not amongst the dangerous six, are also renowned for aggressiveness and territoriality (Philip Wallaby, personal communication) and are considered to be dangerous as pets (Corbett, 1995).

4.4 Population Distribution of Dogs

The distribution of dogs within communities is centred on the households where dogs spend their time and the owners with which they associate and who are ‘responsible’ for them. Household distribution data includes the number of dogs per household, number of dogs per dog-owning-household and the proportion of households within a community that contain dogs. Owner distribution data includes age and gender demographics of the owners as well as the numbers of dogs per owner and dogs per person. This information helps to determine if there is a dog overpopulation problem and if the dog population is uniform within the community.

4.4.1 Results

4.4.1.1 Household Distribution

The average number of dogs per household and dogs per dog-owning-household varied for each region (Chi squared test, P<0.05) (Table 4.3). The eastern communities had the largest number of dogs per household (4.1) and dogs per dog-owning-household (5.2) (Figure 4.4). By comparison, the coastal communities had 1.0 and 1.9 dogs respectively and the central region was intermediate with 1.6 and 3.3, respectively. The urban centres were similar to the coastal region with 0.6 dogs per household and 1.3 dogs per dog owning household.
<table>
<thead>
<tr>
<th></th>
<th>Combined Kimberley Communities</th>
<th>Coastal Region</th>
<th>Central Region</th>
<th>Eastern Region</th>
<th>Kimberley Urban Centres</th>
<th>Statistical Method*</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>HOUSEHOLDS</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dogs/household</td>
<td></td>
<td>0.96</td>
<td>1.58</td>
<td>4.06</td>
<td>0.62</td>
<td>K-W One Way ANOVA</td>
</tr>
<tr>
<td>Households with dogs (%)</td>
<td></td>
<td>49.4²</td>
<td>47.5²</td>
<td>78.7³</td>
<td>48.7³</td>
<td>χ² test</td>
</tr>
<tr>
<td>Dogs/dog-owning-household (DOH)</td>
<td></td>
<td>1.94²</td>
<td>3.34</td>
<td>5.20</td>
<td>1.28³</td>
<td>K-W One Way ANOVA</td>
</tr>
<tr>
<td>Percentage of DOH with one dog</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>χ² test</td>
</tr>
<tr>
<td>Percentage of DOH with &gt; 4 dogs (multiple DOH)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Percentage of dogs in single dog houses</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Percentage of dogs in multiple dog houses</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>OWNERS</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dogs to people ratio</td>
<td>1:4.9 (ex. Ngalingkadji)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>K-W One Way ANOVA</td>
</tr>
<tr>
<td>Number of dogs per owner</td>
<td></td>
<td>1.44*</td>
<td>2.14²</td>
<td>2.70²</td>
<td>1.28*</td>
<td>K-W One Way ANOVA</td>
</tr>
<tr>
<td>Owner age group which kept the most dogs</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>K-W One Way ANOVA</td>
</tr>
<tr>
<td>Owner age group with the largest number of dogs per owner</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>K-W One Way ANOVA</td>
</tr>
<tr>
<td>Percentage of dogs owned by men</td>
<td></td>
<td>64.9%</td>
<td></td>
<td></td>
<td>44.0</td>
<td></td>
</tr>
</tbody>
</table>

* Statistical method used to analyse data for pooling to determine combined Kimberley communities’ results and to determine statistical difference between combined communities and Kimberley urban centres

² No statistical difference between data (P>0.05)

* No statistical difference between data (P>0.05)
Overall, the largest number of dogs per household was found at Kalumburu with one household containing 28 dogs.

The percentage of households with dogs was also statistically higher in the eastern region than the other regions with 78% [63, 93] of households having at least one dog present (see Figure 4.5). The coastal and central regions had much lower percentages (49% [35, 63] and 47% [34, 61] respectively), which were similar to the Kimberley urban communities (48% [37, 59]).

Only 14% [10, 18] of households in the eastern region had only one dog, which represented 2.6% of the total dog population i.e. 97% of dogs were kept in households with more than one dog. By comparison, 51% [46, 55] of dog-owning-households in the coastal region had only one dog and these dogs represented 27% of the population (comparison between coastal and eastern, P<0.00001).
The situation was reversed when multiple dog-owning-households (over 4 dogs per household) were considered. Seventy eight percent of dogs in the eastern region lived in households with more than 4 dogs [73, 84] whereas only 13% [6, 20] of dogs in the coastal region lived this way (Figure 4.6).

**Figure 4.6: Percentage of Dogs from Households with More than Four Dogs in Each Region of the Kimberley**
4.4.1.2 Dog Ownership

There was no statistical difference in the dogs-to-people ratio between each community (Kruskal-Wallis ANOVA, P>0.05, average 4.9:1), except Ngalingkadji (see Table 4.4). The number of dogs per owner varied for each region, with the eastern region having the greatest number of dogs per owner (2.7) (see Figure 4.7; Table 4.3).

**Figure 4.7: Average Number of Dogs per Owner for Each Region of the Kimberley**

The number of dogs per owner for the eastern region (2.7) was much lower than the number of dogs per household (4.1) (see Table 4.3) suggesting that there were several dog owners living at the same household.

For the central and eastern region, 40 to 60 years old people kept the most dogs, whereas the 20 to 40 years old groups of the coastal region and Kimberley urban centres kept the largest proportion of dogs (Table 4.3, Figure 4.8).
Table 4.4: Number of Dogs per Person – September 1994

<table>
<thead>
<tr>
<th>Location</th>
<th>Number of People</th>
<th>Number of Dogs</th>
<th>Ratio of Dogs to People</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lombadina/Djarindjin</td>
<td>326</td>
<td>42</td>
<td>1:7.8†</td>
</tr>
<tr>
<td>One Arm Point</td>
<td>336</td>
<td>57</td>
<td>1:5.9‡</td>
</tr>
<tr>
<td>Kalumburu</td>
<td>251 (adults)</td>
<td>84</td>
<td>1:3 (adults)</td>
</tr>
<tr>
<td>Looma</td>
<td>428</td>
<td>85</td>
<td>1:5‡</td>
</tr>
<tr>
<td>Joy Springs</td>
<td>30</td>
<td>20</td>
<td>1:1.5‡</td>
</tr>
<tr>
<td>Bayulu</td>
<td>309</td>
<td>45</td>
<td>1:6.9‡</td>
</tr>
<tr>
<td>Muludja</td>
<td>130</td>
<td>55</td>
<td>1:2.4‡</td>
</tr>
<tr>
<td>Ngalingkadji</td>
<td>32</td>
<td>27</td>
<td>1:1.2</td>
</tr>
<tr>
<td>Yiyili</td>
<td>266</td>
<td>17</td>
<td>1:15.6‡</td>
</tr>
<tr>
<td>Warmun</td>
<td>507</td>
<td>153</td>
<td>1:3.3†</td>
</tr>
<tr>
<td>Yaramun</td>
<td>113</td>
<td>92</td>
<td>1:1.2</td>
</tr>
</tbody>
</table>

† No statistical difference between data (P>0.05)
Although these owner-age groups owned the greatest numbers of dogs, these groups did not necessarily have the largest number of dogs per owner. Dog owners older than 80 years had the most dogs per person than other age groups in the eastern and central region (5.5 and 4.0 dogs per owner, respectively) (Figure 4.9). For the coastal region, there was no significant difference in the average number of dogs per owner for each age category (average 1.6) except the 20-40 years group where the number of dogs per owner was lower (1.2 dogs per owner).

For each region (and urban centre), there was no statistical difference in the number of dogs per owner in the 0-20 years group, which had the lowest number of dogs per owner (Figure 4.9). There was no statistical difference in the numbers of dogs per owner between the coastal communities and the urban centres for each age category.

Overall, 65% [60, 70] of dogs belonged to men in the remote communities (September 1994, P>0.05 between communities). Dogs were 2.3 times more likely to be owned by men than women in remote communities compared with the Kimberley townships [1.5, 3.6] (Table 4.3).
Figure 4.9: Average Number of Dogs per Owner in Each Owner Age Category

4.4.2 Discussion

4.4.2.1 Household Distribution of Dogs

Regional differences for household distribution were quite marked and may reflect the relative difference in numbers of people per household for each region. Studies in other countries have found household family structure to be one of the most important determinants of household dog distribution. Generally as the number of persons in a household increase, so too does the probability of the household owning a dog (or perhaps owning more than one dog in this case) (Nassar and Mosier, 1986; Nassar and Mosier, 1991).

Community history and development may also explain the differences for each region. The average number of dogs per household (1.0) and dogs per dog-owning-household (1.9) were quite low in the coastal region and were almost in accordance with other non-Aboriginal community studies (0.42 to 0.74 dogs per household and 1.24 to 1.7 dogs per dog-owning-household) (Franti and Kraus, 1974; Schneider and Vaida, 1975; Griffiths and Brenner, 1977; Nassar and Mosier, 1980; Nassar et al, 1984; Nassar and Mosier, 1984; Robertson et al, 1990;
Odendaal, 1994; Patronek et al., 1997). The communities of the coastal region are of mission background and have had a relatively long period of European contact, which may have altered previous canine ownership patterns. These communities are also sea based and may not have had the necessity for dingo assistance in hunting in the past, compared with desert communities. Also, it appears that many dogs at these communities have companionship roles as small dogs, such as Chihuahuas, were popular.

In contrast, the eastern (desert) communities of Warmun and Yaramun have had less European contact and have recently evolved from station backgrounds where larger working dog breeds, such as Australian Cattle Dogs, would be necessary.

In the Yolo County of California, similar trends to the present study have been found with rural districts having larger numbers of dogs per household than suburban areas (Franti and Kraus, 1974). Similarly in Ontario, Leslie et al. (1994) found 81% of rural households to own dogs, which is more than found in the present study. The Kimberley communities again show diversion in the percentage of households owning dogs with the central and coastal showing similarity to the urban communities (approximately 50%) but differences to the eastern region (78%).

The variability between rural and suburban dog ownership in the U.S. and Canada have been attributed to the larger size of rural properties, but differences in socioeconomic status, family structure, and employment of the head of the household may also contribute to the differences. These factors are unlikely to be the reasons for differences between Aboriginal communities in the present study.

The high percentage of dog-owning-households that only kept one dog in the coastal region (51%) is similar to findings from a survey in the Philippines where 49.3% of dog-owning-households kept only one dog. In Perth, by contrast, 79.1% of dog-owning households only
kept one dog (Robertson *et al.*, 1990), which demonstrates that although the coastal communities have some similarity to Australian urban communities in dog ownership characteristics, they still have a higher density of dogs per household.

### 4.4.2.2 Dog Ownership

In most non-Aboriginal communities, the number of dogs per owner and dogs per household are much the same, as only one person per household takes the role of owner. In the Kimberley communities, many owners may live at the same household due to the crowded living conditions. The average number of Aboriginal people per private community dwelling in 1991 was 7.7 compared with 2.6 per dwelling for non-Aboriginal people (Anon, 1993).

In the present study, the number of dogs per household was much higher than the number of dogs per owner indicating that households are shared between several dog owners. This has implications for animal management programs as workers requiring owner co-operation must ensure they address all owners at each household. The difference between dogs per household and dogs per owner was more marked in the east, demonstrating the more crowded nature of these communities.

Overall the ratio of dogs to people was found to be an average of 1 to 4.9 which is within the range found in many other studies (range of 1:3.9 to 1:7.3) (Franti and Kraus, 1974; Schneider and Vaida, 1975; Griffiths and Brenner, 1977; Nassar and Mosier, 1980; Nassar *et al.*, 1984; Nassar and Mosier, 1984; Robertson *et al.*, 1990; Ticman and Carlos, 1992; Odendaal, 1994; Leslie *et al.*, 1994; Patronek *et al.*, 1997). Since the number of dogs per household and owner was high, but the number of dogs per person was not, this indicates that the perceived dog overpopulation may not be due to too many dogs, but not enough houses. The same number of people, owners and dogs located in another community with more houses could result in figures similar for non-Aboriginal communities of other studies.
There also was a disproportionately high number of dogs per owner for older age groups of the human population although 31% of the Aboriginal population was under the age of 15 at the 1991 census (Anon, 1993). That is, there was an overdispersion of dog ownership in the older age groups although these groups contributed less to the human population census. To outsiders, there may appear to be an overpopulation of dogs in Aboriginal communities, but this was really only the case for certain owner-age groups in the present study (see 4.4.2.3).

In some communities there was a disproportionately high number of dogs per person compared to other communities (see Table 4.4). The communities with high numbers of dogs per person were often small (Ngalingkadji and Yaramun) and/or led a very ‘traditional’ lifestyle. Typically these communities are very isolated and consider dogs to be important, possibly for reasons such as assistance with hunting and protection in the spiritual and physical sense. Due to the small size of these communities, introduction of relatively few new dogs to the community or small numbers of bitches breeding can lead to disproportionate increases in canine distribution and population characteristics.

### 4.4.2.3 Owner age

As mentioned, it was often the older members of communities that owned dogs. This was especially so in the eastern and central region where the greatest number of dogs was owned by 40 to 60 years old people. People over 80 years of age in these regions, though, were the ones with the highest density of dogs (although they owned a small proportion of the total population of dogs) and this has many implications for targeted education campaigns. Education regarding immediate population control needs to be focused at the 40 to 60 years old group who own the greatest number of dogs. Programs aimed at prevention of zoonoses need to be targeted at the people at greatest risk from contact with many dogs; the 80-plus years olds who had an average of 1.8 to 5.5 dogs per owner for each region. Obviously for long term canine control, children are the most receptive, absorptive group for education campaigns.
In South Africa, respondents between 55 and 64 years of age kept the most dogs, which is similar to the eastern and central communities in the present study (Odendaal, 1994). Explanations for the 55 to 64 years old people keeping the most dogs included the better financial status of this group, larger living spaces and the need for child substitution by adults during the "empty nest" phase of life (Odendaal, 1994). In Aboriginal communities, it is unlikely the former two would be of importance, but companionship and the holding on to traditional beliefs surrounding dogs may be appropriate reasons for the overdispersion of dog ownership in older age groups.

4.4.2.4 Owner Gender

Odendaal’s (1994) study in South Africa did not reveal any differences in male or female ownership of dogs. In the present study, 65% of dogs belonged to men (and boys), which was higher than found at the Kimberley townships where 56% of dogs were the responsibility of women (and girls). Given the genderocratic nature of Aboriginal society, this data may have implications in the form of education used as part of a control program. Education programs may need to be targeted at men, particularly those relating to population control of dogs.

4.5 Population Dynamics

The dynamics of the dog population within the study region during the period of the project were largely dependent on the effect of the breeding control. This in turn was dependent on the response of owners to the administration of proligestone to bitches, which was favourable (see Figure 4.13). Some factors independent of the contraceptive program, such as the inward migration of dogs from other communities, influenced the overall success of the dog population control program. Mortality was to some degree dependent on the success of the ivermectin treatments in reducing deaths, but was also dependent on extraneous factors such as season and disease epidemics. Likewise, outward migration was independent of the program. All these factors combined to alter the population structure and distribution and affect the effectiveness of the population control program.
Population dynamics studies concerning the birth, death and migration rates are essential if one is to understand the behaviour of a population and devise effective methods to control or regulate its growth (Nassar and Mosier, 1980).

4.5.1 Mortality and Outward Migration

4.5.1.1 Results

The average 3 monthly mortality rate was 15% [12, 19], except in March 1993 (23% [19, 28]) (Table 4.5; Figure 4.10). There was no statistical difference in mortality rate between regions and visits. One year after the start of the program (June 1993), only 53% [48, 58] of the original dogs were still present in the communities, i.e. 47% of the original dogs had died or gone missing in one year.

Figure 4.10: Percentage of Registered Dogs that Died or Moved Away from Communities

Likewise, overall, 55% [46, 63] of dog owning households had dogs that died in the previous 12 months. By contrast, only 12% [5, 19] of dog owning households of the Kimberley urban centres had lost a dog in the same period.
Table 4.5: Mortality and Outward Migration of Dogs

<table>
<thead>
<tr>
<th></th>
<th>Combined Kimberley Communities</th>
<th>Coastal Region</th>
<th>Central Region</th>
<th>Eastern Region</th>
<th>Kimberley Urban Centres</th>
<th>Statistical Method*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Three monthly mortality rate (%)</td>
<td>15.2 (ex M93)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>$\chi^2$ test</td>
</tr>
<tr>
<td><strong>STRUCTURE</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Proportion of dogs that died that as puppies or juveniles A</td>
<td>30.45 (ex S92, D92 and D93)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>$\chi^2$ test</td>
</tr>
<tr>
<td>B</td>
<td>58.65 (S92, D92, D93)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>$\chi^2$ test</td>
</tr>
<tr>
<td>Odds ratio (compared with living dogs) B</td>
<td>0.56 [0.4,0.8]</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>$\chi^2$ test</td>
</tr>
<tr>
<td>Percentage of dogs that died that were male</td>
<td>58.6</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>HOUSEHOLD DISTRIBUTION</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Percentage of houses with dogs that died in previous 12 months</td>
<td>54.9</td>
<td>19.2</td>
<td></td>
<td></td>
<td></td>
<td>$\chi^2$ test</td>
</tr>
<tr>
<td>Percentage of dogs that died that were from multiple DOH</td>
<td></td>
<td></td>
<td>68.9</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Odds ratio (compared with living dogs)</td>
<td></td>
<td>1.58 [1.0,2.5]</td>
<td></td>
<td></td>
<td></td>
<td>$\chi^2$ test</td>
</tr>
<tr>
<td>Percentage of dogs from single DOH that died</td>
<td></td>
<td></td>
<td></td>
<td>0.63 [0.5,0.8]</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Percentage of dogs from multiple DOH that died</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>$\chi^2$ test</td>
</tr>
<tr>
<td><strong>CAUSE</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Percentage of deaths of known cause (apart from euthanasia)</td>
<td>9.2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>$\chi^2$ test</td>
</tr>
<tr>
<td>Proportion of deaths caused by euthanasia</td>
<td>8.4% (ex M93)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>$\chi^2$ test</td>
</tr>
</tbody>
</table>

* Statistical method used to analyse data for pooling to determine combined Kimberley communities’ results and to determine statistical difference between combined communities and Kimberley urban centres
4.5.1.1.1 Age of Dogs that Died Compared with the General Population

During the dry season months of September 1992 and December 1992 and 1993, the proportion of dogs that died as puppies or juveniles was greater than the proportion found during the other months of the year (59% vs. 30%, P=0.004). Puppies were over-represented in the groups that died during these dry season months when compared with the remaining living population (P=0.001, 59% compared with an average 29% in the living population).

4.5.1.1.2 Age Dependent Survival Rates

The probability of puppies of 0-6 months of age surviving from one visit to the next was 0.74 for each visit, excluding the start (June to September 1992) and the months of the wet season in 1992 (December 1992 to March 1993) (Chi Squared test, P=0.53). At these times, the puppy survival rates were much lower (0.5).

4.5.1.1.3 Sex of Dogs that Died Compared with the General Population

Males constituted 58.6% of the dogs that had died at any time indicating that there was no difference in the sex distribution of dogs that died compared to those that were living (59.0%, Chi Squared test, P=0.83).

4.5.1.1.4 Household Distribution of Dogs that Died Compared with the General Population

The percentage of dogs that died which had lived in multiple-dog households varied for each region. The central region had no regular pattern of household distribution of dog deaths for each visit, but the other regions did. Nineteen percent of dead and missing dogs from the coastal region had resided in multiple-dog households (Table 4.5). This was significantly greater than the proportion of dogs in the general population that resided in multiple-dog households (P=0.01). Overall, dogs that died were 1.6 times more likely to come from multiple-dog households than the general population [1.0, 2.5]. In the east, the opposite trend occurred with dogs in the general population being 1.6 times more likely to come from multiple-dog households than those that died [1.2, 2.2].
4.5.1.1.5 Reasons for Death

Only 9.2% [6.0, 12.3] of the dog deaths were of a known cause when voluntary euthanasia was excluded (Table 4.5). The causes of death ranged from drowning to snake envenomation. At all visits an average of 8.4% of deaths were from voluntary euthanasia except March 1993 when the requests were much higher (20.7%).

4.5.1.1.6 Outward Migration

The percentage of dogs seen at each visit that subsequently left the communities before the next visit varied between 0.7% [0, 1.6] and 5.9% [3.5, 8.3] for the duration of the program.

4.5.1.2 Discussion

The three monthly mortality rate was high across all months (average 15.22% excluding March 1993). Crude comparisons from North America indicate that the turnover rate in the Kimberley communities was much higher, as only 6.4% of dogs were found to die in a 12-month period in Las Vegas (Nassar et al., 1984). One year after the commencement of the Kimberley program, 47% of the original dogs had died or gone missing. Consequently, over 50% of dog-owning-households had lost a dog in the same period which was 4 times the rate of similar households in the urban communities of the Kimberley region.

Although there was no statistical difference in mortality figures for most months, greater than normal losses were evident in March 1993 (23.3%). These deaths occurred between December 1992 and March 1993 at the time of the wet season. Many of the reported deaths were season related as 6 (33.3% of deaths of known cause, including euthanasia) were due to drowning. The voluntary euthanasia rate at March was also higher than normal (20% of deaths) due to the poor condition of some dogs after widespread flooding across the region. The wet season of that year was particularly severe. In the Fitzroy Valley, many communities (including 4 of the program’s 12 communities) had to be evacuated after major flooding of the Fitzroy River. Dogs were left
at the communities and many owners reported dogs killing pet livestock (pigs and cattle) for food.

Although not statistically different to the overall mortality rate, there is also a slightly higher mortality rate during the wet season of 1994 (March 1994) (see Figure 4.10).

The spread of age of dogs that died was generally the same as that of the surviving population, except during the months surrounding the wet seasons. Much higher proportions of dogs less than or equal to one year died in the three months of the wet season indicating that puppy survival at these times was difficult. Age dependent survival rates supported this as the rates were lower for the period of December 1992 to March 1993 (0.5 compared to 0.75).

There was no sex predilection for death at any time, implying that biases in population sex (that is, more males than females) were not due to higher mortality of females. Even so, the ratio of males to females for most months was higher for the adult age group implying that sometime between birth and one year of age (adulthood), a greater proportion of females left the communities or died. Conversely, more males may have been born or entered the communities, although the data do not support this (4.5.2.1.2).

Higher density of dogs per household would seem to be an important factor for high mortality as disease is more likely and food more scarce. In the coastal region, dogs that died were 1.6 times more likely to have come from multiple-dog households than those that were living. On the contrary, crowded conditions did not cause increased death rates, but acted inversely for the eastern region. The number of dogs per household in the east does not play a major role in mortality, but the number of dogs per owner may do so. As mentioned, many people live at one household that may be able to provide food for the dogs. Alternatively, in regions were fewer people look after many dogs, there is less chance that all dogs will receive adequate nutrition or supervision.
Very few people could provide explanations for missing or dead dogs, which is in accordance with other difficulties encountered regarding information retrieval in communities. As many dogs resided at each household, it was often difficult for owners to provide information on the whereabouts of their dogs, especially puppies without names. Disease epidemics occurred at the coastal communities between March 1992 and June 1992 when distemper claimed 12 dogs. Poison baits (strychnine usually) were also common in areas with cattle raising. Reasons for voluntary euthanasia included overpopulation, aggressive behaviour and disease or poor condition.

The percentage of dogs that left the communities between visits was similar to the percentage of dogs that were visiting at any one time (3.8%) (see 4.5.2). The number of dogs leaving did not contribute greatly to the reduction in population, but did indicate that the population of dogs at communities is mobile between communities.

4.5.2 Inward Migration and Acquisition of Dogs

4.5.2.1 Results

After the program had stabilized (after December 1992), 13% [9.1, 14.9] (inward migration rate) of dogs at each visit were new, either just born or introduced, to the community in the previous three months (Table 4.6; Figure 4.11). The percentages of dog-owning-households that acquired new dogs in the previous 12 months were 86% [80, 92] and 94% [90, 98] for June 1993 and June 1994, respectively.

Of the new dogs to the communities at each visit (excluding September 1993 and 1994), 26% [17, 35] of them were born in the community. That is, 74% of new dogs were acquired from other sources; most often other Kimberley communities not on the program. The percentage of all dogs that were visitors to the communities or only transitory at each visit was 3.8% [2.0, 5.6].
Table 4.6: Inward Migration and Acquisition of Dogs

<table>
<thead>
<tr>
<th></th>
<th>Kimberley Communities</th>
<th>Coastal Region</th>
<th>Central Region</th>
<th>Eastern Region</th>
<th>Statistical Method*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inward migration rate (from D92)</td>
<td>13.1%</td>
<td></td>
<td></td>
<td></td>
<td>$\chi^2$ test</td>
</tr>
<tr>
<td>Percentage of transient dogs</td>
<td>3.8</td>
<td></td>
<td></td>
<td></td>
<td>$\chi^2$ test</td>
</tr>
<tr>
<td>Percentage of dogs born in community</td>
<td>3.4 (ex S93, S94)</td>
<td></td>
<td></td>
<td></td>
<td>$\chi^2$ test</td>
</tr>
</tbody>
</table>

**STRUCTURE**

|                                | 65.1% (ex S93, s94) |                |                |                | $\chi^2$ test       |
| Odds ratio (compared with other living dogs) | 0.12 [0.10, 0.14] |                |                |                | $\chi^2$ test       |
| Percentage of new dogs that were male | 57.5 (ex D92) |                |                |                | $\chi^2$ test       |

**HOUSEHOLD DISTRIBUTION**

|                                |                  |                  | 76.9            |                | $\chi^2$ test       |
| Odds ratio (compared with other living dogs) |                |                  | 0.9 [0.7, 1.2]  |                | $\chi^2$ test       |
| Percentage of new dogs in single DOH |                  |                  | 15.3            | 5.5            | $\chi^2$ test       |
| Odds ratio (compared with other living dogs) |                |                  | 0.4 [0.3,0.5]   | 0.4 [0.3,0.7]  | $\chi^2$ test       |
| Percentage of households that acquired a dog in previous 12 months | J93 86.5 | J94 93.9 |                |                | $\chi^2$ test       |

*Statistical method used to analyse data for pooling to determine combined Kimberley communities' results and to determine statistical difference between combined communities and Kimberley urban centres.
Figure 4.11: Percentage of Dogs that were New or Visiting the Communities

4.5.2.1.1 Age of New Dogs

The dogs that were acquired by people in the communities (either after births in the community or from elsewhere) were generally younger than the established dogs. Excluding September 1993 and 1994 (when the percentages were higher), the percentage of new dogs that were puppies and juveniles was 65% [55, 75] indicating that young dogs were 8.3 times more likely to be new to the program than already registered [7.1, 10.0].

4.5.2.1.2 Sex of New Dogs

As with the dogs that had died between each visit, the sex distribution for new dogs was not remarkable. Fifty seven percent [46, 67] of new dogs (born in the community or acquired) were males indicating that there was no exceptional preference for males or females compared to the dogs already established at the communities (P=0.1, 59% males). In December 1992, though, only 44% of new dogs were male which was different to the other months (P=0.008).
4.5.2.1.3 Household Distribution of New Dogs

The percentage of new dogs that moved into multiple-dog households varied considerably for each visit for the coastal and central communities (P<0.05). The eastern region experienced much more stability with an average of 77% [62, 92] of new dogs entering multiple-dog households (P=0.4), which was no different to the percentage of established dogs living in multiple-dog households (P=0.6). Overall, people in households with many dogs (>4 dogs per household) were just as likely to acquire new dogs as those in households with fewer dogs.

The proportion of new dogs in single-dog households was much less than the proportion of established dogs in single-dog households for the coastal and central region. At the coastal region, 15.3% of new dogs went to households that had no other dogs compared with 47.4% of established dogs living without other dogs. Dogs living alone were therefore 2.6 [0.5, 0.3] and 2.3 [0.3, 0.7] times (respectively) more likely to be dogs already existing in the communities than newly born or introduced dogs for the coastal and central region. There were too few dogs living alone at the eastern region for similar comparisons to be made.

The percentage of households acquiring new dogs over a 12 months period increased during the program from 86% [80, 92] (June 1993) to 94% [90, 98] (June 1994).

4.5.2.2 Discussion

Coupled with a very high mortality rate was a very high inward migration rate of dogs. Overall, between 87 and 94% of dog owning households had acquired a new dog within a 12 month period, adding to the high turnover rate of dogs in the communities. Approximately 74% of these dogs were acquired from other sources such as outstations, other communities and local townships. The inward migration was not always coupled to inward migration of people, although on three occasions new community members were noted to have imported pregnant bitches. Puppies were presented to owners as gifts.
The majority of dogs that were new to the communities in any three month period were less than one year old (average 65%). Old dogs were rarely imported unless with their owners. In September 1993 and 1994, the proportion of new dogs that were puppies or juveniles increased remarkably.

Owner preferences for the gender of new dogs did not seem to differ from the established dogs. That there is a bias towards males amongst new dogs from other regions implies that owners still have a slight preference for new dogs to be male, or that other communities not involved in the study also have more males than females. Those born in the community were also of the same sex distribution, but, as mentioned, there was an even greater tendency toward males as the population aged.

The number of new dogs that moved into households with many dogs (multiple-dog households) did not show any particular trend. In the eastern region, the percentage of new dogs in multiple-dog households was the same as the rest of the population which implies that having many dogs at a household did not preclude the occupants from acquiring more dogs.

In the coastal region, new dogs were not as likely to live on their own as other dogs. The same situation occurred in the central region. Puppies born within the communities were unlikely to be living on their own for the first few months until weaning, so this contributed to this finding. This also further implies that households with dogs were more likely to acquire new dogs than those that did not have dogs.

4.5.3 Reproduction

4.5.3.1 Results

The average ratio of new dogs born in the communities to those acquired from other places was 1:3.9 (see Table 4.7). This was similar for all months except September 1993 and 1994 when the average ratio was 1:1.4. During these months there was a distinct increase in the number of
### Table 4.7: Dog Population Dynamics – Combined Kimberley Communities

<table>
<thead>
<tr>
<th><strong>REPRODUCTION</strong></th>
<th><strong>Statistical Method</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Ratio of dogs acquired outside to those born in community</td>
<td>3.9:1 (ex S93 and S94)</td>
</tr>
<tr>
<td>Percentage of entire females that reproduced in previous 12 months</td>
<td>11.1</td>
</tr>
</tbody>
</table>

**POPULATION**

| Rate of change of population per three months | 0.99 (ex. J93-S93) | $\chi^2$ test |
| Age dependent survival rates from one visit to next 0-6 months old puppies | 0.75 (ex. J92-S92 and D92-M93) | $\chi^2$ test |
| Ratio of dogs 0-6 months old to 6 m.o. -1 yr | |
| Group A (J92, S92, S93, J94 S94) | 1.3 | $\chi^2$ test |
| Group B (others) | 0.9 | $\chi^2$ test |

*Statistical method used to analyse individual communities and regions for pooling of data*
puppies born within the communities.

The (three monthly) conception rates of mature bitches during the treatment program (from June 1992 to September 1994) remained below the initial baseline rate of 10.5% [4.6, 16.4] in March 1992 (Figure 4.12). Although there was no statistical difference in the conception rates from June 1992 to June 1994 (average 4.1%, P=0.18), except for March 1994, a slight increase in conception rate (compared with surrounding months) occurred in June 1993 and June 1994. No bitches had conceived at the last treatment month in September 1994. After contraceptive treatments were stopped in September 1994, the conception rate returned to over 15% [9, 21].

**Figure 4.12: Percentage of Mature Bitches that Conceived Each Three Months**

Overall, the percentage of entire females (including those that were treated) reproducing per year between June 1992 and June 1994 was 11% [6, 16]. This was significantly lower than found in both the Kimberley urban centres and Perth (54%, P<0.00001 and 19%, P=0.1 respectively). Entire bitches in Perth were 2 times more likely to reproduce than the bitches in the communities of the program. Similarly, entire bitches of the urban centres of the Kimberley were 10 times more likely to reproduce in one year.
Entire, mature bitches that had never received proligestone treatment were 5 times more likely to reproduce than those that had received at least one treatment [3, 8]. Of all the pregnancies in bitches between June 1992 and June 1994, 12% [3, 21] of them were planned by their owners. Fifty one percent of the pregnant bitches had never been treated with proligestone [37, 65]. Only 14% [4, 24] of the pregnant bitches had been treated the visit before their estimated conception date. Only one bitch was pregnant twice during this time, but both pregnancies were intentional.

Between 62% [53, 71] and 100% of mature bitches presented for examination were treated with the contraceptive at each visit (Figure 4.13). This rate is equivalent to treatment of between 62% [52, 71] and 82% [76, 88] of all bitches assumed to be alive at each visit. Owners elected not to treat if they intended to breed the bitch or if the bitch was already pregnant. If the owner was not available, the consenting people also occasionally elected not to treat. Despite this, these rates indicate a high acceptance of the contraceptive.

**Figure 4.13: Percentage of Bitches at Each Visit that Received Contraceptive Treatment**

![Graph showing percentage of bitches treated with contraceptive over time.](image-url)
4.5.3.2 Discussion

The contraception program was effective. The conception rates before and after the treatment period (between March 1992 and September 1994) were well above those during this period. The reproductive rate for bitches during the program was between approximately 40% and 80% less than Perth and the Kimberley urban centres, respectively.

Although some bitches did breed during the program, this was not due to ineffective contraception treatments, as bitches that had not received any treatment were 5 times more likely to conceive than those that had received one or more treatments. In addition, 51% of pregnant bitches had not been treated with proligestone before their pregnancy. The pregnancies throughout the program were either due to bitches entering the communities already pregnant or because they missed the treatments. Twelve percent of the pregnant bitches had intentional pregnancies and a further 14% missed treatments just before the estimated conception date, thus leaving a period of over 6 months between treatment and conception. The remaining pregnant bitches had histories of inconsistent treatments.

It is possible that breeding control could have been more effective if the capture success of mature bitches was greater and the capture rate of individuals more consistent. As is seen in Figure 4.13, between 18 to 38% of the estimated population of mature bitches in communities missed treatment at each visit although 82 to 100% of bitches presented for examination at each visit were treated. Even at times when conception rates were relatively high (during the dry seasons), the percentage of bitches presented for examination that were treated was also high indicating that people were keen to treat the dogs, but not all reproducing bitches were caught.

The reason for the rise in number of puppies born in the study communities during September 1993 could not be completely explained by reproductive patterns, although a slight increase in conception rate during June 1993 did occur (Figure 4.12). Although domestic dogs are claimed
to breed continually and have two litters per year, the present study suggests there may be a seasonal breeding pattern with an annual peak in births, between June and September.

Purebred dingoes are known to be seasonal breeders, but their hybrids often revert to the domestic dog pattern (Corbett, 1995). The seasonality is largely due to the environment as is evident in the differences between dingoes in tropical, arid or temperate climates.

In the tropical regions of Australia, dingoes are capable of breeding all year round, although maximal litter numbers are usually expected during the cool, dry months (Corbett, 1995). Corbett (1995) reports that the peak litter births occur in July in the northern areas of Australia (13°S) with 33% of all litters being born in that month.

Dingoes from the central arid regions of Australia, where the climate is hot and dry, exhibit a seasonal pattern in breeding for both males and females. Males have maximum sperm production at the same time as the females come into oestrus, which is around April/May. Litter rates reach a peak in July (Corbett, 1995). In the temperate southern regions, female dingoes come into oestrus about a month later than their counterparts in the arid regions (about June or July) (Corbett, 1995).

Generally, the maximum litters born to dingoes in Australia are between the months of June and September (Corbett, 1995). This is similar to the pattern found in the Kimberley dogs.

Corbett (1995) also notes that female dingo/dog hybrids will often only have one litter a year due to the expense of energy required to produce the one litter. This may be the case in dogs in communities, particularly in areas where many dogs compete for few resources. This may also account for a peak in reproduction during the dry season.
4.5.4 Rate of Change in Population Size

4.5.4.1 Results

Excluding the period between June 1993 and September 1993, the average rate of change in population for every three months was 0.99 (Table 4.7). This extrapolates to an annual rate of change in population of 0.94 indicating a very low decline in population. This is not entirely correct as the boost in population during June to September 1993, with a rate of change of population of 1.19, led to numbers in excess of the original census.

4.5.4.2 Discussion

The overall annual rate of change of population (excluding June to September 1993) was similar to those found in Manhattan and Las Vegas (0.975 and 0.96) (Nasser and Mosier, 1984; Nassar et al, 1984) where there are higher reproductive rates (17 and 13%), but much lower ratios of dogs acquired from outside the community to those born within (0.5 to 1.26:1). The dynamics of dog populations in these communities is different to those found in the present study, as they are more stable, with fewer inward migrating dogs. For adequate canine population control in the Kimberley communities, breeding control must therefore be coupled with reductions in importation of new dogs from other areas. This will bring the dynamics more in accord with centres where population control of dogs is considered to be more adequate.

4.6 Conclusions

Overall, the Kimberley community dog populations varied from other studies of dog populations throughout the world in structure, distribution and dynamics. The population was young and the rate of sterilisation very low compared with western, industrialised communities. The number of dogs per household was also greater than found in other studies and often reflected the number of owners (or people) living at each household, particularly in the more ‘traditional’ communities. Middle aged people (40-60 years old) were also more likely to own the greatest proportion of dogs, although the very old people (>80 years of age) were likely to own the most dogs per person.
The annual rate of change of dog population (excluding September 1993) was similar in the present study to other studies in North America. Despite this, the reproduction rate in the present study was lower and the inward migration of dogs from other communities greater than the comparative studies of North America.

(a) Strategies for Improvement in Population Control

The contraceptive drug treatments were effective at preventing reproduction on an individual scale, but overall population numbers did not reduce considerably during the three years of the program. Analysis of the population dynamics indicates that a large proportion of the dogs contributing to the population size stability were imported from other (non-program) communities. Overall the mortality rates were high (compared to other studies) and reproduction had decreased, but the numbers of dogs entering communities remained high or increased. Population control, therefore, needs to include breeding control and inward migration control. Inward migration control requires community-wide support for education of community members about the need to resist importation of puppies from other communities. Widespread adoption of the breeding control program by all communities in the Kimberley region is also likely to reduce the availability of puppies.

Contraceptive drugs that have a longer period of action and require fewer administrations would have more success in a combined treatment/education based program. Reduced treatment frequency would be more convenient for workers conducting the program and greater compliance rates by owners would be expected. Long-acting drugs are more likely to combat any potential seasonal increase in reproduction.

Although permanent surgical sterilisation appears to address these factors, the high turnover rate in dogs in remote communities (47% of dogs not seen one year after last encountered) suggests that this method would not be cost effective. Further research into the demand for permanent sterilisation would be necessary to determine the feasibility of providing this service to the
Kimberley region. Despite this, compliance with the 3 monthly treatment regimen was adequate in the present study, appeared to be preferable to permanent sterilisation to some owners and without detrimental health side effects (for the duration of the program).

Given the dog ownership characteristics of the present study, education campaigns addressing the ‘overpopulation’ of dogs in communities would need to be targeted at the groups owning the largest numbers of dogs; the 40-60 years olds of the central and eastern regions and the 20-40 years olds of the coastal communities. The present study has shown that owners living in households with many dogs are just as likely to acquire new dogs as those in households with fewer dogs. Further research into the reasons for dog ownership by people in these age groups would assist workers in determining how to reduce the importation of dogs into communities. Involvement of these key age categories in the ongoing contraceptive treatments for dogs is also important for immediate control of reproduction.

(b) Population Control, Zoonoses and Education Programs

Zoonotic disease transfer is often considered a greater risk for people in close habitation with diseased animals, particularly for scabies infection. In the present study, people over the age of 80 years were found to own the most dogs per owner. Although the present study does not describe the behavioural association of owners with their dogs, by nature of the living conditions of the people in this age group and the numbers of dogs sharing their living space, the possibility for transmission of disease is high. Health professionals involved in reducing disease transmission are therefore advised to target efforts at this age group. The age of these people and the possibility that they would have traditional beliefs centred around dingoes/dogs would make removal of dogs from their environment difficult from an emotional and social perspective. Efforts are best centred on improving the health status of the dogs by effective parasite control to limit the most common potential zoonoses. Education of health professionals of the importance of dog ownership to older members of the community would also help avoid clashes between community people and the health providers.
(c) Veterinary Services and Dog Health Programs

In the overall approach to education and control campaigns, the variability in dog ownership patterns between communities that are well established with a longer history of contact with Europeans (such as the coastal communities) and communities that are younger and more ‘traditional’ in beliefs and lifestyle (such as the central and eastern communities) needs to be considered. In the present study, the coastal communities exhibited dog ownership characteristics that were very close to the situation in the urban Kimberley centres, but much removed from the ownership patterns at the other areas of the Kimberley. From this, it is assumed that coastal community members are more likely to respond to campaigns that would be effective in the wider non-Aboriginal communities. Introduction of veterinary services, including vaccination for communicable diseases (distemper, parvovirus, hepatitis) and prevention of heartworm, are possibly more likely to be successful in these communities than others.
Chapter 5

PRELIMINARY SURVEY OF THE ENDO- AND ECTO-PARASITES AND GASTROINTESTINAL BACTERIA AFFECTING DOGS OF THE KIMBERLEY REGION

5.1 Introduction

This chapter investigates the pre-treatment prevalence of endo- and ecto-parasites and gastrointestinal bacteria in dogs from the communities in which the parasite control program was conducted. The effect of the control program on ivermectin sensitive parasites is dealt with in the following chapters.

5.2 Methodology

The pre-treatment surveys were conducted in June 1992 for the majority of the communities that were grouped as belonging to the coastal, central or eastern region. At Looma and Kalumburu, the program was started later. The surveys at these two communities were done in September 1992 and March 1993, respectively. Looma and Kalumburu are treated as separate communities from the mainstream communities throughout this chapter because the prevalence of some of the parasites investigated in the present survey are affected by season (see 7.3.2.1).

The routine involved in collecting samples is described in the general methodology chapter. After collection of blood and faecal samples at the first visit, treatments with ivermectin and proligestone were given.

Gastrointestinal parasites were detected in faecal samples using the formalin ethyl acetate sedimentation technique (see Appendix C). Blood samples were used to identify dogs with circulating D. immitis microfilariae. Likewise, sera were collected for serological testing for antibodies to E. granulosus (see 3.3.7.3).
5.3 Results

5.3.1 Overview of Canine Parasites of the Kimberley Region

The parasites found in dogs from the Kimberley Region during the pretreatment testing of dogs were; *Sarcoptes scabiei*, *Rhipicephalus sanguineus*, *Ancylostoma caninum*, *Giardia duodenalis*, *Sarcocystis* spp., *Spirocerca lupi*, *Toxocara canis*, *Hymenolepis nana*, *Strongyloides stercoralis*, *Spirometra erinaceieuropai*, *Hammondia heydorni*, *Isospora ohioensis*, *Trichodectes canis*, *Ctenocephalides* spp. and *Dirofilaria immitis* (see Figures 5.1-5.5). Serological testing from a sample of dogs from all communities suggested that *Echinococcus granulosus* was also present in the Kimberley region.

The following results and discussion examine the prevalence of these parasites.

**Figure 5.1: Pre-treatment Prevalence of Parasites* in Dogs from the Kimberley Coastal Region**

*Percentages of dogs with lesions indicative of *Sarcoptes scabiei* are shown on graphs.
Chart error bars indicate 95% confidence intervals.
Figure 5.2: Pre-treatment Prevalence of Parasites in Dogs from the Kimberley Central Region

Figure 5.3: Pre-treatment Prevalence of Parasites in Dogs from the Kimberley Eastern Region
Figure 5.4: Pre-treatment Prevalence of Parasites in Dogs from the Kimberley Kalumburu

Figure 5.5: Pre-treatment Prevalence of Parasites in Dogs from the Kimberley Looma
5.3.2 Scabies

The percentage of dogs with lesions indicative of scabies varied from 16.7% [6.6, 21.0] to 52.3% [41.7, 62.9] across each of the regions (see Table 5.1). The dogs from the coastal and central communities shared similar overall infection rates (average 16.9% [12.5, 21.3]) as did Looma and the eastern region dogs (average 46.2% [42.7, 56.1]). The infection rate at Kalumburu was intermediate in prevalence (27.4% [18.9, 35.9]).

Dogs with more than 75% of hair lost were evident at all locations except the coastal region prior to treatment. Looma and the eastern region had the highest proportion of dogs with more than 25% of hair lost.

5.3.3 Hookworm (*Ancylostoma caninum*)

Hookworm was the most common parasite infecting dogs from each region and community (see Figures 5.1-5.5). No statistical difference was found in the pretreatment prevalence of hookworm at each of the regions. Overall, the average prevalence was 64.7% (n=201 [60.9, 73.9]).

The pretreatment prevalence of hookworm at Kalumburu was considerable higher at 90.7% (n=43 [82.0, 99.4]), but this sampling was done in March 1993. At Looma, where the pretreatment samples were collected in September 1992, the prevalence was 72.7% (n=44 [59.5, 85.9]).

5.3.4 Roundworm

Dogs shedding *Toxocara canis* eggs were found at Kalumburu and the communities of the Fitzroy Valley region, including Looma, in the preliminary survey. The prevalence of *Toxocara* at these regions and communities varied from 2.3% to 3.6% (average 3.5%, P>0.05 [27.9, 42.1]). At subsequent surveys during the program, *Toxocara* was found at all regions except the eastern region.
Table 5.1: Pre-Treatment Prevalence of Scabies in Dogs from the Kimberley Region

<table>
<thead>
<tr>
<th>Location*</th>
<th>Number Examined</th>
<th>Percentage Displaying Alopecia</th>
<th>Mean Prevalence for Locations with no Statistical Difference in Scabies Prevalence</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Total</td>
<td>Score 1</td>
<td>Score 2</td>
</tr>
<tr>
<td>Central Region</td>
<td>154</td>
<td>16.7</td>
<td>8.4</td>
</tr>
<tr>
<td>Coastal Region</td>
<td>123</td>
<td>17.1</td>
<td>12.2</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kalumburu</td>
<td>106</td>
<td>27.4</td>
<td>3.8</td>
</tr>
<tr>
<td>Eastern Region</td>
<td>127</td>
<td>40.2</td>
<td>15</td>
</tr>
<tr>
<td>Looma</td>
<td>86</td>
<td>52.3</td>
<td>18.6</td>
</tr>
</tbody>
</table>

* Groupings indicate no statistical difference between communities or regions (P=0.05)
5.3.5 Other Parasites and Bacteria

5.3.5.1 *Echinococcus granulosus*

5.3.5.2 Seroprevalence

The ELISA test used to detect anti-*E. granulosus* protoscolex serum antibody (IgG, IgA and IgE) had a sensitivity between 73 and 84% and specificity of approximately 98%, according to other studies (Gasser et al., 1993; Thompson et al., 1993b). Serological evidence of hydatid infection occurred in 4.2% [2.3. 6.1] of 430 random samples collected over the period of the program (see Table 5.2). At no time were taeniid eggs recovered by faecal examination.

**Table 5.2: Details of Dogs with Positive Hydatid Serology**

<table>
<thead>
<tr>
<th>Serologically Positive Dogs*</th>
<th>Immunoglobulin class</th>
<th>Community</th>
<th>Age (years)</th>
<th>Sex</th>
<th>Total Number of Samples from each Community</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>IgG, IgA, IgE</td>
<td>Warmun</td>
<td>&gt;1</td>
<td>F</td>
<td>73</td>
</tr>
<tr>
<td>2</td>
<td>IgG, IgE</td>
<td>Warmun</td>
<td>&gt;1</td>
<td>F</td>
<td>73</td>
</tr>
<tr>
<td>3</td>
<td>IgA, IgE</td>
<td>Warmun</td>
<td>&gt;1</td>
<td>F</td>
<td>73</td>
</tr>
</tbody>
</table>

| 4 | IgG                             | Yiyili    | >1          | F   | 29                                       |
| 5 |                                 | Kupingarri| >1          | F   | 30                                       |
| 6 |                                 | Yaramun   | >1          | F   | 32                                       |
| 7 |                                 | Looma     | 0.33        | M   | 42                                       |
| 8 |                                 | Muludja   | >1          | M   | 36                                       |

| 9 | IgA                             | Wamum   | 0.67        | M   | 73                                       |
| 10|                                 | Warmun  | >1          | M   | 73                                       |
| 11|                                 | Warmun  | >1          | M   | 73                                       |
| 12|                                 | Yiyili  | 1           | M   | 29                                       |
| 13|                                 | Kalumburu| 0.33        | M   | 47                                       |
| 14|                                 | Kupingarri| >1        | M   | 30                                       |
| 15|                                 | Joy Springs| 5         | M   | 8                                        |
| 16|                                 | Looma   | >1          | F   | 42                                       |
| 17|                                 | Djarindjin| >1        | M   | 28                                       |
| 18|                                 | Muludja | >1          | M   | 36                                       |

*Total number of samples was 430

5.3.5.2 *Giardia duodenalis*

The prevalence of *Giardia* ranged from 2.3% at Looma to 18.1% at the central region. Overall, there was no statistical difference in prevalence for the three regions at the pre-treatment survey (P=0.40), with the average prevalence being 14.9% [10.0, 19.8].
5.3.5.3 Heartworm

The results of the microfilariae tests are shown in Table 5.3. The highest prevalence was found at Yaramun (65.5% [48.2, 82.8]), a remote inland community in semiarid country. There was no statistical difference between this prevalence and the prevalence at two other locations, Kalumburu and Kununurra. An average of 16.2% [12.2, 20.2] prevalence was found for the central region, Kupingarri and Warmun where there was no statistical difference in prevalence of heartworm. The coastal communities (excluding Kalumburu) had a prevalence intermediate to the other regions (39.8% [30.3, 49.2]).

Table 5.3: Prevalence of *Dirofilaria immitis* in Dogs from the Kimberley Region

<table>
<thead>
<tr>
<th>Location</th>
<th>Number Sampled</th>
<th>Number with Microfilariae</th>
<th>Prevalence (%)</th>
<th>Mean Prevalence for Locations with no Statistical Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yaramun</td>
<td>29</td>
<td>19</td>
<td>65.5</td>
<td></td>
</tr>
<tr>
<td>Kalumburu</td>
<td>74</td>
<td>45</td>
<td>60.8</td>
<td>58.6 [51.3, 66.0]</td>
</tr>
<tr>
<td>Kununurra</td>
<td>71</td>
<td>38</td>
<td>53.5</td>
<td></td>
</tr>
</tbody>
</table>

| Coastal Region | 103 | 41 | 39.8 | 39.8 [30.3, 49.2] |

| Eastern Region | 102 | 28 | 27.4 | 16.2 [12.2, 20.2] |
| Kupingarri    | 30  | 6  | 20   |                                |
| Central Region | 196 | 19 | 9.7  |                                |

*D. reconditum* was not detected in any samples and 4 out of 6 dogs from Kununurra examined by post mortem had adult heartworms in the right ventricle and pulmonary arteries.

5.3.5.4 *Strongyloides stercoralis*

Larvae of *Strongyloides stercoralis* were isolated from dogs from the central and eastern regions during the preliminary survey. Only 1.5% [0, 4.1] to 2.4% [0, 6.0] of dogs were infected. *Strongyloides stercoralis* was never isolated from dogs from the coastal region communities at any of the subsequent surveys.
5.3.5.5 *Spirocerca lupi*

Six [0,14] and 14 [6.0, 22.2] percent of dogs from the eastern region and 36% [22.0, 50] of dogs from Kalumburu were found to be shedding spirurid eggs in their faeces. Examination of 42 eggs from 10 samples revealed a mean size of 33.3μm (± 0.2 SEM) x 13.7μm (± 0.19 SEM). On the basis of egg size and morphology, they were identified as *Spirocerca lupi* eggs. Other spirurid nematodes known to occur in dogs and cats in Australia, *Gnathostoma spinigerum*, *Physaloptera praeputialis* (Barton and McEwan, 1993) and *Cyathospirura dasyuridis* (Ryan, 1976) have larger eggs (Soulsby, 1982; Mawson, 1966). Even the closest sized eggs from *C. dasyuridis* (33-35 x 18μm) are wider than *S. lupi* eggs (11-15 μm) and those examined.

5.3.5.6 Other Arthropod Parasites

Ticks (*Rhipicephalus sanguineus*) were found on dogs from all communities and regions during the survey, excluding one eastern Kimberley community; Yaramun. The prevalence of infection with ticks was 18.7% [11.8, 25.6] for the coastal region, 12.3% [7.1, 17.5] for the central region and 23.6% [16.2, 31.0] for the eastern region. The prevalence attained at the two communities representing the coastal and central region (Kalumburu and Looma, respectively) was much higher (43.4% [34.0, 52.8] and 47.7% [37.1, 58.3], respectively). No other species of ticks were detected on the dogs throughout the program.

Dogs from the coastal region communities (including Kalumburu) were infested with fleas (*Ctenocephalides* spp.) to varying degrees. No attempts to determine the prevalence were conducted as fleas, being primarily ‘nest parasites’, are only on the host intermittently. Fleas were never detected on dogs from the eastern or central region (including Looma). Lice (*Trichodectes*), though, were found at all regions, mostly on puppies.
5.3.5.7 Other Protozoan Parasites

*Sarcocystis* spp. was found at the eastern and central region during the pre-treatment survey at rates of 5.9% [3.0, 11.5] to 7.2% [1.6, 12.8], respectively. The protozoan was also isolated from dogs in the coastal group, but only from 2% [0, 5.9] of dogs.

*Isospora ohiensis* was only isolated from 1.2% [0, 3.5] of dogs from the central region prior to treatment. During the treatment program, *Hammondia heydorni* and *Isospora canis* were also sporadically detected at similar rates from the central region. *Cryptosporidium* oocysts were not isolated, but selective staining (such as the modified Ziehl-Neelsen method (Casemore, Armstrong and Sands, 1985)) was not used.

5.3.5.8 Other Cestodes

Oncospheres of *Hymenolepis nana*, a parasite primarily of humans, were detected in the faeces of between 1.2% [0, 3.5] to 2.9% [0, 6.9] of dogs from Kalumburu, the central region and the eastern region. Subsequent faecal examinations during the program also showed the parasite to be present at the other areas, also at low rates.

Dogs shedding eggs of *Spirometra erinaceieuropaei* were only found at the central region during the pre-treatment survey (3.6% [0, 7.6]), but at later surveys the tapeworm was also detected in dogs from the eastern and coastal region (including Looma and Kalumburu).

5.3.5.9 Gastrointestinal Bacteria

The prevalence of *Campylobacter* ranged from 2.9% [0, 8.5] (coastal communities) to 6.9% [1.0, 12.8] (central region). *Salmonella* isolates were more common with the prevalence ranging from 2.9% [0.8.5] to 65% [49.6, 80.4] (see Table 5.4).
Table 5.4: *Salmonella* spp. and *Campylobacter* spp. Isolation from Dogs from the Kimberley Region*

<table>
<thead>
<tr>
<th>Organism</th>
<th>Coastal Region (n = 34)</th>
<th>Central Region (n = 72)</th>
<th>Looma (n = 37)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Percentage Infected</td>
<td>Percentage Infected</td>
<td>Odds Ratio**</td>
</tr>
<tr>
<td></td>
<td></td>
<td>% Infected that were</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Adults</td>
<td>Odds Ratio**</td>
</tr>
<tr>
<td>Salmonella spp.</td>
<td>2.9</td>
<td>20.8</td>
<td>53.3</td>
</tr>
<tr>
<td>Campylobacter spp.</td>
<td>2.9</td>
<td>6.9</td>
<td></td>
</tr>
</tbody>
</table>

*No significant difference between sexes at each region

** Odds ratio for infection in adults
5.4 Discussion

5.4.1 Scabies

Prior to treatments, scabies was present at every community in varying degrees of severity. Data on the prevalence of sarcoptic mange in canids is limited, but surveys on wild canids have been conducted in North America. In southern Texas, 32% of the wild coyote population was found to have mange (determined by visual appraisal and quantified by a mange scoring system) during an epizootic (Pence et al, 1983). In other regions of North America, scabies is enzootic in wild canids, but at a much lower prevalence. In fact, prior to the epizootic discussed by Pence et al (1983), the rate of infection was 1% or less. Despite the similarities in prevalence to the present study, the dynamics of the infestations in wild animals is different to that of domesticated dogs and the scabies documented in communities is most likely enzootic.

In Britain, the prevalence of scabies is estimated to be 1% (Baxter and Leck, 1984). In Australia no comparisons exist, except that scabies has been seen in dogs from Aboriginal communities (Thompson et al, 1993a). A report of the effects of ivermectin treatments in 4 communities of northern Queensland found scabies infestation in 21 out of 39 (53.8%) dogs (as determined by clinical presentation) (Presson, Palmer and Leach, 1989). Another report from Palmer and Presson (1990) describes signs of mite infestation in 42% (n=106) of dogs from selected communities in the Northern Territory and 52% (n=62) of dogs from communities in Cape York. No differentiation from Demodex canis mites was done, either by skin scraping, response to treatment or clinical appraisal of distribution or type of skin lesions.

5.4.2 Hookworm (Ancylostoma caninum)

The results from faecal examination may underestimate the number of dogs with intestinal helminths (Kirkpatrick, 1988; O'Sullivan, 1997), by up to 25 to 35% in some cases (O'Sullivan, 1997). Single stool examination lacks sensitivity in lightly infected dogs (O'Sullivan, 1997) and prepatent infections are not detected by helminth egg examination. The prevalence of hookworm in the Kimberley Region may thus be greater than found in the present survey.
Given the variability in climatic and geographical conditions throughout the Kimberley, the pretreatment prevalence of hookworm was expected to vary for each region, but didn't (57.8% to 70.6%, average 64.7%, p>0.05). The only area that had considerable variation from the main regions was Kalumburu, but this sampling was conducted during the wet season, which often has an increased prevalence (see 7.3.2.1).

Comparison of the present study's prevalence of hookworm with reports from other locations is confounded by the differences in sampling and diagnostic testing. Post mortem and faecal flotation investigations of mixed age dogs from Aboriginal communities in the central region of the Kimberley in another study found the prevalence to be 52.7% (n=188) (Thompson et al, 1993a). This is significantly lower than the present study (57.8%, p=0.001), which may be partly due to the differences in sample collection and analysis (Thompson et al's (1993a) study was undertaken over a 5 year period).

Results of similar studies in Brisbane, Queensland, have shown that the prevalence of hookworm infection ranges from 17% in summer sampling of pet animals (Prociv et al, 1994) to 87% of stray dogs over 1 year of age (Boreham and Capon, 1982). In southern Australia, the most common hookworm species is Uncinaria stenocephala (Dunsmore and Shaw, 1990; Johnston and Gasser, 1993), which was not isolated from samples from the Kimberley (identification based on egg morphometrics and examinations of whole worms from post mortems at Kununurra).

Other countries have recorded prevalence of canine hookworms varying from 5% in Jordan (Abo-Shehada and Ziyadeh, 1991) to 92% in New Orleans (Schock, 1976) (see Table 2.1). These prevalences reflect differing climatic conditions and also the types of dogs examined; their age, sex and living conditions. The risk factors for infection with hookworm will be discussed in more detail later (7.4.2.2).
5.4.3 Roundworm

The low rate of infection with *T. canis* is in contrast to most studies of this parasite throughout the world (see Table 2.2). Even within Australia, the prevalence of roundworm for most studies is much higher than found in the present study. Only two studies failed to recover roundworm, one in New South Wales (Jenkins and Andrew, 1993) and the other in the Fitzroy Valley region of the Kimberley (Thompson *et al*, 1993a), both studies from Aboriginal communities. The prevalence of *T. canis* varies with the age of the host (see 7.4.3.1.1), so assessment of data must take into account this difference. Thompson *et al* (1993a) did comment on the lack of *T. canis* infection in the dogs of their survey, despite post mortem examination of 25 puppies.

A possible reason for the low prevalence is the effect of climate on survivability of roundworm eggs. Nicholas *et al* (1986) found that *T. canis* eggs will develop to the infective stage, becoming spontaneously motile, at a relative humidity between 35 and 100%. When exposed to direct sunlight for eight hours, though, both undeveloped and infective eggs are killed. Even when the eggs in sealed moist chambers are protected from direct sunlight by a leaf, the eggs are killed. This situation with exposure to sunlight is probable in the Kimberley, although Nicholas *et al* (1986) did not determine the effect of sunlight on eggs found naturally and possibly protected in faeces. Temperatures above 37°C kill eggs (Levine, 1968), so this in combination with sunlight may explain the absence of *T. canis* at some of the inland communities where temperatures above 37°C are frequently attained.

Sunlight and high temperatures, though, are plentiful in arid central Australia where Welch *et al* (1979) found 52% (*n*=52) of mixed age dogs to be infected. Welch *et al* (1979) commented that this prevalence was considerably lower than coastal regions of northern eastern Australia (prevalence of 75%) and was most likely due to the harsh environment on infective stages.

One of the few reports of the effect of climate on naturally occurring canine alimentary tract parasites (Becker, Selby, Hutcheson and Hacker, 1977) did not find any climatic associations
with the prevalence of ascarids. Becker et al (1977) attributed this to the life cycle of *T. canis* including environmentally resistant eggs, fairly resistant larvae and the possibility of paratenic hosts. Hookworm, by comparison, is a parasite that is very reliant on environmental factors and much more susceptible to climate than roundworm and yet is endemic and highly prevalent in the Kimberley.

Glickman and Schantz (1981), in a review of zoonotic toxocariasis, suggest that the character of the top soil is perhaps the most important factor favouring endemcity of geohelminths. Survival of ascarid eggs is enhanced in moisture-retaining clay soils, where they are “sedimented in a layer of fine silt between a thin protective blanket of colloidal clay and a deeper layer of coarser particles” (Glickman and Schantz, 1981). Here, the eggs are protected from sunlight and drying and remain as a reservoir for infection, for both dogs and children practicing geophagia.

*Ascaris lumbricoides* (an ascarid of humans), is similar in biology to *Toxocara canis* and is also extremely resistant to chemicals and other environmental factors (Mango, 1970). *A. lumbricoides* infection is much more common in the tropics and subtropics than in the temperate zone (Levine, 1968), and is found in areas around the world which have similar or more severe climatic conditions than the Kimberley (Upatham et al, 1992; Bundy, Wong, Lewis and Horton, 1990; Asaolu, Holland, Jegede, Fraser, Stoddard and Crompton, 1992). *Ascaris lumbricoides*, though, has not been isolated during any gastrointestinal parasite surveys of children and adults of the Kimberley region (Gracey et al, 1992; Meloni et al, 1993; Reynoldson, Behnke, Pallant, Macnish, Gilbert, Giles Spargo, Thompson, 1997; Reynoldson, Behnke, Gracey, Horton, Spargo, Hopkins, Constatine, Gilbert, Stead, Hobbs and Thompson, 1998).

Welch et al (1979) suggested that apart from the harsh environment in central Australia, the lower prevalence of *Toxocara canis* in Alice Springs compared with the eastern coast of Australia might have been due to the recent introduction of the parasite to the area. The data
from the present survey may further substantiate this suggestion as many infections in the central region of the Kimberley were in dogs that had been introduced from coastal areas. Likewise, even in the tropical coastal community of Kalumburu, 12 of the infections were from puppies born to one dam. In the Kimberley, it may be that the parasite fails to become endemic because of the low frequency at which it is introduced and when it is introduced, it is not sustained because of the harsh environment. Very rarely did dogs outside of the Kimberley enter the relatively remote communities of the region (see 4.5.2). Studies on dingoes also suggest that *T. canis* was not endemic in Australia before colonisation (Welch *et al*, 1979).

### 5.4.4 Other Parasites and Bacteria

#### 5.4.4.1 *Echinococcus granulosus*

**5.4.4.1.1 Seroprevalence**

As the ELISA test available at the time was only 73-84% sensitive (a potential for 16 to 27% false negatives), the number of dogs who had been exposed to *Echinococcus granulosus* could have been higher. Likewise, the specificity of the test could account for 3% of false positive errors due to cross-reaction to other taeniids.

Variability in the test has been attributed to variations in immune responses to *E. granulosus* infection. These variations are related to the influence of host genotype on antibody responses as well as the effect of difference in the nutritional status of individual dogs (Gasser *et al*, 1993).

IgA serum antibodies have been readily detected in dogs with *E. granulosus* infection (Gasser *et al*, 1993). In the present study, 67% (12/18) of dogs with seropositivity (4.3%, 18/420) showed IgA antibody activity. This class of immunoglobulin is specific for *E. granulosus* and in similar infections with other taeniids, the presence of serum IgA has been closely associated with egg hatching and oncospheral penetration of the gut epithelium (Gasser *et al*, 1993), indicating an active infection. Past infections are also detected with this test as serum antibodies can persist
for several weeks after removal of worms (Gasser et al, 1993). Likewise, positive antibody tests do not always reflect the number of worms per host.

The results of the present study, therefore, provide some evidence for the exposure of some of the dogs from selected communities to *E. granulosus*. The location of the dogs throughout the Kimberley gives some suspicion for the source of infection. Lyembery, Thompson, Constantine and Kruger (1995) described the distribution of hydatid infection in cattle in Western Australia and 14 communities of the Kimberley region were adjacent to stations from which 80% of infected cattle in the survey had come from. Prior to this report, the Kimberley had always been considered virtually free of hydatid disease (Kumaratilake and Thompson, 1982). Most of the dogs of the present survey had come from communities situated on or nearby the stations from which infected cattle had been detected. The exact foci of hydatid disease is not revealed by the report by Lyembery et al (1995) as the birthplace (origin) of the hydatid-infected Kimberley cattle was not determined. Kimberley cattle (Brahman herd bulls and cows) slaughtered at the Katherine (Northern Territory) meat works have been detected with hydatid disease, but of those infected animals whose origins could be traced, all came from south eastern Queensland (Jubb T, Agriculture Department of Western Australia, Kununurra, personal communication), where *Echinococcus* is endemic (Baldock, Thompson, Kumaratilake and Shield, 1985). As most cattle hydatid cysts in Australia are without protoscoleces, and are therefore non-infective (Schantz et al, 1995), this does not explain how the dogs of the present survey have been exposed to *Echinococcus*, unless there is another source of infection for dogs (and possibly cattle) in the Kimberley region.

Hydatid disease was not suspected in the Kimberley mainly because of the climatic requirements for survival of the oncospheres. Temperatures above 27°C and rainfall less than 25mm for 6 months, which is common in the Kimberley, hinder transmission of the parasite (Gemmell, 1959). Lyembery et al (1995), consider that if an endemic cycle is established in the area, then it may be centred around areas of permanent water and/or maintained by a highly
susceptible definitive host population. Wild or domestic dogs may be acting as definitive hosts, but with such a low prevalence in dogs from Aboriginal communities, it is more likely to be a wild canid cycle.

Potential intermediate hosts in the area include wallabies, donkeys, horses and camels, but no data has been collated on the prevalence (or infection) of these animals, although veterinarians of the region have never seen hydatid disease in animals culled during population control campaigns (Jubb T, personal communication).

The serological evidence of hydatids in the Kimberley region has great implications for public health and control programs such as the present study. E. granulosus seems highly vulnerable to the implementation of control measures as the cycle can be interrupted by preventing dogs from eating raw viscera of intermediate hosts (Schantz et al, 1995). The difficulty lies in educating owners about the risks of infection and how to break the cycle (Thompson et al, 1993b). A control program in Aboriginal communities requires adequate surveillance of the disease, determination of the relevant intermediate hosts and targeted education on the problems with feeding dogs raw offal. Owners would also have to take responsibility to not let their dogs forage and roam on their own, which would be very difficult to institute in communities which often have no physical boundaries.

Echinococcus control, based on strict control of home slaughter of sheep, with quarantine of infected dogs or sheep and routine treatment of dogs with echinococcocidal drugs, has been successful in New Zealand and Tasmania (Schantz et al, 1995). Prevalence rates of echinococcosis in dogs, sheep and humans have also been decreased in Argentina, Cyprus, Iceland and the Falkland Islands through the implementation of control programs (Walters, 1986). Other proven methods to control hydatids include health education, stray dog control, registration of owned dogs, diagnostic testing of dogs coupled with treatment and enactment and enforcement of legislation supporting the other measures (Schantz et al, 1995). The possible
development of an effective vaccine for intermediate hosts (Heath and Lightowlers, 1993, cited in Schantz et al., 1995) may be of benefit in the present situation, but only if the intermediate hosts of this cycle are domestic animals. Given the extensive nature of the cattle rearing in the Kimberley, vaccination is unlikely to be feasible.

With appropriate parasite and breeding control programs in place, as is the case in the Kimberley as a result of the present study, education and institution of hydatid treatments would not be as difficult to start as it would be without established programs. A study of *Echinococcus* in dogs from the rural areas of Uruguay, found that dogs become reinfected with *Echinococcus* between 2 to 4 months after complete purgation with arecoline hydrobromide (which is analogous to treatment with an echinococcosidal drug) (Cabrera, Parietti, Haran, Benavidez, Lloyd, Perera, Valledor, Gemmell and Botto, 1996). Although the numbers of dogs that were examined were too small to draw firm conclusions about the rate of reinfection, the authors did suggest that control of *Echinococcus* could be based on the actual reinfection rates rather than the prepatent period of the cestode. Treatment intervals of 3-4 months may be adequate to control *Echinococcus* in Uruguay. Studies to determine the potential reinfection rates of dogs may be useful in the Kimberley, but the level of infection is possibly too low to effectively determine this. Conversely, with low infections rates in the Kimberley compared with Uruguay (13.2%, n=303), the reinfection rate will probably be much slower.

Despite the potential problems, a detailed survey of potential hosts and location of endemic foci is necessary before the commencement of targeted control and education campaigns.

**5.4.4.2 *Giardia duodenalis***

The average prevalence of 14.9% for the three regions is likely to be an underestimate, since only one faecal sample was examined for each dog and *Giardia* is intermittently shed (Swan and Thompson, 1986). Despite this, these results can be compared to some other studies in Australia, also based on single faecal sample testing.
Thompson et al (1993a) found 16.3% of 282 dogs from inland Kimberley (central) region communities to be excreting Giardia cysts, which is similar to the present study (P=0.68). Comparatively, other studies have found the prevalence amongst dogs from a variety of backgrounds in Perth to be 21% (Swan and Thompson, 1986) and 22.1% (Bugg, 1996), with even higher rates amongst dogs from refuge shelters (30%) (Swan and Thompson, 1986). These rates are higher than those of the Kimberley communities with the risk of infection in the general population being 1.8 times more likely in Swan and Thompson’s (1986) study than the present study (P=0.0001). Another survey in the south west of Australia by Savini, Dunsmore and Robertson (1993) revealed similar findings to Swan and Thompson (1986) with dogs in kennels having a higher prevalence than dogs from farms or urban environments (46.7% vs. 10.0% and 3.0%, respectively P=0.001). The relative differences between the prevalence in the south of Western Australia and the Kimberley Region may indicate that environmental factors, such as temperature and humidity, are affecting cyst survival in the warmer Kimberley. Although the majority of dogs in the comparative studies in the south were in crowded conditions (kennels and breeding establishments) with a continuous source of infection for susceptible animals, a similar situation occurs in Kimberley communities.

Johnston and Gasser (1993) found Giardia to vary between 2.3% for dogs from kennels to 14.2% for strays in Melbourne, Victoria. Extrapolation of the data shows that stray dogs were 4 times more likely to have infections than dogs visiting veterinary hospitals (3.9%). These rates are much less than found in Perth and are similar to the Kimberley Region.

5.4.4.3 Dirofilaria immitis

Variations in the prevalence of Dirofilaria immitis between individual communities in the regions led to some communities being examined separately to the others (see Table 5.3).

Heartworm is most prevalent in the coastal regions of Australia, but is also common in the tablelands of Queensland. The prevalence of heartworm in northeastern coastal Australia,
determined by circulating microfilariae, varies from 37% (Carlisle, 1969) to 68% (Aubrey and Copeman, 1972). In Brisbane, 36% of dogs were found with microfilariae with a further 12% having occult infections (Welch et al, 1979). These results are similar to those found in the present study (10.3% to 65.5%) and reflect the tropical climate and abundance of mosquitoes. Reasons for the slightly lower prevalence in the Kimberley relate to the lower numbers of dogs in fairly remote and isolated communities. Most of the infected dogs were born within the communities and did not travel to other areas indicating that heartworm is endemic, but at a low level in some areas (Fitzroy Crossing).

The true prevalence of heartworm could not be determined from this survey, as adequate numbers of dogs examined by post mortem were not obtained. The prevalence of *D. immitis* attained in the present survey is likely to be underestimated because of the inability to detect occult infections with filter techniques. The absence of microfilariae in heartworm infections may be due to immature worms, single sex worms, dead adult parasites, or treatment of the dog with a microfilaricide before sampling (Kelly, 1973). Jackson (1969) found that out of 98 dogs examined by post mortem, 8 of the dogs with adult *D. immitis* did not have microfilariae. Carlisle (1969) only found 50% (91/183) of dogs with patent infections to have circulating microfilariae. Despite this, post mortem examination of 58 dogs by Thompson et al (1993a) did not show any signs of adult parasites whereas in the present survey, 10.3% (21/204) of dogs from nearby communities were found to have circulating microfilariae.

Warmun, Kupingarri and the central Kimberley communities all had prevalences considerably lower than at the other regions. Reasons for the differing prevalence are not obvious as all areas (Liewne, 1991) are inhabited by the most efficient mosquito vector for *D. immitis*, *Culex annulirostris* (Russell and Geary, 1996). Yaramun, with a prevalence of 65.5%, is very close to a large permanent lake (Lake Gregory) which is prone to flooding during the wet season. Likewise, though, the Fitzroy valley locations are in the flood plains for the Fitzroy River and also have abundant mosquito activity. *Culex annulirostris* is the most common mosquito
inhabiting the more permanent wetlands of the Kimberley (Lichne, 1991) and is also able to take advantage of the more permanent urban sites such as drains and containers.

_C. annulirostris_ also acts as the major vector for Murray Valley Encephalitis virus and Kunjin virus, both of which are known to cause Australian Encephalitis in humans (Mackenzie and Broom, 1995). The distribution of Australian encephalitis cases in the Kimberley is remarkably similar to the relative prevalence of microfilaraemic dogs. Between 1978 and 1991, 8 out of 18 human cases in the Kimberley were from the south-east compared with 1 case from the Fitzroy Crossing area (Mackenzie, Smith, Broom and Bucens, 1993).

5.4.4.4 _Strongyloides stercoralis_

_Strongyloides stercoralis_ is well established in the Aboriginal population of northern Australia (Prociv and Luke, 1993; Fisher _et al_., 1993). In Western Australia, an overall prevalence of 4.0% (33/817) was found in people from 7 northern Aboriginal communities (Jones, 1980). The findings of the present survey also suggest that _Strongyloides_ is well established, albeit at a low prevalence, in dogs from some communities of the Kimberley.

The significance of canine infection in Aboriginal communities is questionable. Dogs have been implicated as potential infection reservoirs for _Strongyloides_ (Fisher _et al_., 1993), but experimental evidence indicates that dogs are either refractory to infection with _S. stercoralis_ of human origin, or remain infected for only short periods, unless immunosuppressed (Genta and Grove, 1989). In Vietnam, where human _Strongyloides_ is relatively common, _Strongyloides_ has been reported in less than 1% of dogs (Genta and Grove, 1989).

Natural human infection from contact with infected dog faeces with the canine strain has occurred (Georgi and Sprinkle, 1974), but the epidemiological significance of canine infection in communities, with so few dogs infected, remains slight.
Considerable regional variation in human *Strongyloides* occurs in the north of Australia. Similarly, the infection rates varied in the present study with the nematode only being recovered from the central and eastern communities. The recovery of the larvae from dogs of the semi-arid regions, rather than the coastal communities, is unusual as the environmental requirements for the transit of the larvae through the soil include high rainfall, temperature and humidity (Prociv and Luke, 1993). This is similar to hookworm larval requirements and, as will be discussed, may indicate that microenvironmental conditions in communities are aiding the survival of these geohelminths.

### 5.4.4.5 *Spirocerca lupi*

*Spirocerca lupi*, a spiruroid nematode commonly found in the walls of the oesophagus, stomach and aorta of dogs from tropical and subtropical countries, has been reported only occasionally in dogs in Australia (Hamir 1984, Barton and McEwan, 1993). *S. lupi* was detected from 3 communities in the present study.

The intermediate hosts of *S. lupi* can be any one of several genera of coprophagous beetle that ingest embryonated eggs passed in the faeces or vomitus of dogs (Soulsby 1982; Dunsmore and Shaw 1990). The larvae develop to infectivity and encyst within the beetle which may be ingested by the definitive host, dogs (and less commonly cats) (Hamir 1984; Dunsmore and Shaw 1990; Johnson 1992). Larvae may encyst in a number of paratenic hosts, which include many small mammals, birds, reptiles and amphibians (Wandera 1976; Soulsby 1982). Because of their indiscriminate eating habits, the dogs in the present study were suitable candidates for *S. lupi* infection.

It is possible that infection was acquired by either ingestion of the beetles or a paratenic host such as lizards or frogs. Many genera of coprophagous beetles are present in the Kimberley Region (Britton 1970; Mathews 1974). Given that one or more of these relatively ubiquitous species may be capable of acting as intermediate host and the versatility of the parasite to
survive in many paratenic hosts, it is difficult to explain why the nematode was not found at more sites. One possibility is that, because of the relatively long prepatent period of 5 to 6 months (Soulsby 1982) and the isolation of some of the communities, the spread of infection has been slow. *S. lupi* is not able to survive in the dog for more than a few years (Bailey 1972), which further decreases the likelihood of introducing a new infection into a different community. The community in which 36% of the dogs were infected is very isolated and all dogs examined were born within the community. It is likely that the infection has been endemic within the community for a long time.

*Spirocerca lupi* has emerged as an important parasite in Papua New Guinea where Hamir and Onaga (1986) found that 60% of 133 dogs examined at necropsy had adult worms present and an additional 16% of dogs had lesions consistent with a past infection. The prevalence of *S. lupi* in other countries is variable and is between 0 to 3% in North America (Bailey 1972), 23% in Malaysia (Hamir and Onaga 1986), 24% in India (Chandrasekharon Sastry and Menon 1958) and 78% in Kenya (Brodey Thomson Sayer and Eugster 1977). In Australia, 35% of 31 dogs from central Australia in a recent study were found to be infected (Barton and McEwan 1993). It is likely that the prevalence in the Kimberley Region is higher than found by faecal analysis, because eggs are not passed in the faeces unless the granulomas, in which the nematodes reside, are open to the lumen of the oesophagus (Chandrasekharon et al 1958; Soulsby 1982). Eggs are intermittently deposited (Chandrasekharon et al 1958; Bailey 1972) which also precludes detection of all positive cases with a single faecal test.

### 5.4.4.6 Other Arthropod Parasites

The prevalence of tick (*Rhipicephalus sanguineus*) infestation varied across the Kimberley with the central region having the lowest prevalence (12%). Ticks were not found at one inland eastern region community and the highest prevalences were attained in Kalumburu (43%) and Looma (48%).
The variation in distribution of ticks is most likely due to differences in climate across the Kimberley region. Ticks are not seen in the colder climates of Australia and are generally considered to exist in tropical/subtropical climates (Dunsmore and Shaw, 1990). Seasonal occurrences of ticks have been observed in Japan where it was found that ticks were not seen on dogs when the mean temperature was below 15°C (Inokuma, Tamura and Onishi, 1995). During laboratory trials, Inokuma et al (1995) found that the larval and nymphaal post parasitic period, the pre-oviposition period and the oviposition period of *Rhipicephalus* were prolonged when the temperature was decreased from 37 to 23°C and development was halted at 14°C. Although the Kimberley region does not experience temperatures below 14°C continuously, temperatures in some areas of the desert regions can fall below 5°C in the dry season evenings (Bureau of Meteorology, 1996). The lower temperatures of the dry season coupled with a low relative humidity may explain the differences in tick prevalence across the Kimberley. The higher prevalences attained in Kalumburu and Looma may be due to the warmer times of the year when the surveys were conducted at these two communities (March and September, respectively).

Fleas are dependent on humidity and temperature for development (Dunsmore and Shaw, 1990) which may explain the absence of fleas from the eastern and central region where it is very dry during the ‘winter’ months. Fleas, while not of major zoonotic concern, are responsible for flea allergy dermatitis in some dogs which can be severe.

Lice, being permanent parasites, are less dependent on environmental factors and hence were found at all regions in the Kimberley. Lice numbers are reported to be highest in the cooler months of the year, but this has not been demonstrated with *Trichodectes canis* infections in dogs (Dunsmore and Shaw, 1990). The main clinical presentation of heavy infestation with the chewing louse, *Trichodectes*, is pruritus and self-trauma (Dunsmore and Shaw, 1990). Lice populations can multiply fast in dogs that are sick or debilitated (Dunsmore and Shaw, 1990),
which may explain the occurrence of lice in puppies in the present study, many of which were anaemic (see 7.3.2.4.2; Table 7.26).

5.4.4.7 Other Protozoan Parasites

The finding of Sarcocystis spp. protozoans was expected considering the raw meat diet of many dogs in the survey. Sarcocystis has a ‘predator-prey’ life cycle with herbivorous intermediate hosts harbouring ‘sarcocysts’ in the muscles. The finding of higher infection rates at the central and eastern region (5.9% and 7.2%) compared with coastal communities (2%) was also expected since the infection rate is likely to relate directly to the amount of raw meat dogs are fed (Dunsmore and Shaw, 1990; Savini et al, 1993). At the central and eastern regions, it is a common practice for the hindquarters of slaughtered beef to be left for dogs to feed upon (personal observation).

Despite the raw meat diet, the overall rates were much lower than found elsewhere in Australia. Savini et al (1993) found an average of 31.1% (n=132) of stray urban and farm dogs from the south west of Australia to be shedding Sarcocystis sporocysts. A survey of the dogs of the Sydney residential area revealed 20.9% of dogs to be shedding sporocysts (Collins Emslie, Farrow and Watson, 1983). Wet climates are more likely to allow survival of oocysts in the environment than dry climates. This may explain the comparatively low rates of infection in dogs from the Kimberley.

Sarcocystis spp., while of no clinical significance to dogs, do have the potential to cause severe loss in intermediate host species due to myositis. Fortunately, the dry conditions of the Kimberley may reduce survival of the oocysts in the environment. Also, wild dogs and dingoes are more likely to perpetuate the life cycle than community dogs, although unrestricted wandering of community dogs could be a concern for graziers.
*Isospora ohioensis* has a simple direct life cycle that is dependent on adequate temperature and moisture for sporulation in the environment. *Isospora canis* can also have a direct life cycle, although infection of mice, which act as a reservoir host, is also possible. *Hammondia heydorni*, in contrast, requires cattle as an intermediate host.

The finding of *I. ohioensis* in the preliminary survey indicates that this protozoan is more common than the other two coccidian species. In other surveys of dogs from Sydney and New Zealand (Callow, 1984), this was also the case. *Hammondia* infection patterns across the Kimberley region followed that of *Sarcocystis* spp., which was expected since both can utilise cattle as an intermediate host. The infection rates for *Hammondia* and *Isospora* were very low (, which may reflect the ability of coccidians to evoke immunity in the host (Dunsmore and Shaw, 1990) or it may reflect the effect of the dry climate on the environmental cyst stages.

Similarly to *Sarcocystis*, the coccidians rarely cause clinical disease, although young dogs under certain environmental and management stresses may suffer severe enteritis from heavy infections (Johnston and Gasser, 1993).

*Cryptosporidium* oocysts were not recovered using the sedimentation technique without selective staining in the present survey. Other surveys of dogs in Western Australia have also failed to isolate *Cryptosporidium* (Savini *et al.*, 1993; Meloni *et al.*, 1993), but Johnston and Gasser (1993) found 11% of dogs from varying environments in southern Victoria to be shedding oocysts. The highest infection rates in the Victorian survey were in stray dogs (15.3%, n=190) and dogs that had defaecated in parklands (19.6%, n=107). The stray dogs were maintained at an animal shelter for one to two weeks which is within the prepatent period for many intestinal protozoa, thus allowing continual transmission of the parasites. Dogs visiting parks were assumed to gain infection from ingestion of contaminated lake water. The authors considered that this was of concern because the dogs may have been acting as reservoirs for human infection as evidence suggests that *Cryptosporidium* is zoonotic. *Cryptosporidium*
infection does occur in the humans of the Kimberley, but considering that no dog faecal samples had oocysts recovered throughout the duration of the program, dogs are unlikely to be heavily infected in the present study area. Molecular techniques are being employed to determine the potential of risk of infection in humans with Cryptosporidium from canine sources (Morgan, Murdoch University, Perth, personal communication).

5.4.4.8 Other Cestode Parasites

Infection of dogs with Hymenolepis nana and Spirometra erinaceieuropaei is unlikely to be of any clinical significance to dogs, but may indicate that dogs can act as reservoirs or 'vectors' for human infection. Similar findings to the present study, with similar infection rates (2.1% and 3.7% n=188, respectively), were found at the Fitzroy river communities in an earlier survey (Thompson et al, 1993a). Thompson et al (1993a) found dogs were passing morphologically normal eggs, but were unable to isolate adult tapeworms from necropsied animals. The parasite eggs were most likely the result of coprophagy of cat and human faeces, rather than an indication of true infection of the dogs (Thompson et al, 1993a).

Both of these parasites are of significance to humans. The findings of low level shedding of the parasites from dogs indicate that dogs may enhance transmission, but treatment of dogs with ivermectin is unlikely to curtail this feature. Ivermectin has no effect against cestodes or their eggs (Campbell 1985).

Other cestodes were not found during the pre-treatment survey, but later in the program, Dipylidium caninum eggs were found in samples from dogs from the coastal region. Although Trichodectes can act as intermediate host for Dipylidium, as well as fleas (Ctenocephalides), D. caninum was not found at areas where only lice were found (central and eastern regions). Tapeworm eggs are found usually after a mature segment has disintegrated, thus formalin ethyl acetate sedimentation of faeces, used in the present study, will only rarely reveal their presence.
5.4.4.9 Campylobacter spp. and Salmonella spp.

Surveys of normal adult dogs have found the prevalence of Campylobacter isolation to range from 1.6% (Prescott & Munroe, 1982) to 34% (Fox et al., 1983; Willard et al., 1987). This is similar to the findings in the present study.

At least 30% of asymptomatic dogs may have Campylobacter in their faeces (Willard et al., 1987). In Australia, McOrist and Browning (1982) found 9.5% of healthy dogs and 16% of diarrhoeic dogs to have C. jejuni. Likewise, a study in Sydney found only 15% and 0.8% of pound and hospitalized dogs with Campylobacter to have diarrhoea (Malik and Love, 1989). Under-representation due to the difficulty in isolating the organism (Skirrow, 1990) and the ability for asymptomatic carriage makes the true potential for dogs to act as zoonotic hosts difficult to assess. As such, it is likely that the true prevalence in communities is higher than determined here.

Similarly to Campylobacter, the true prevalence of Salmonella is difficult to determine. The sensitivity of identifying these Salmonellae is only about 75% and the organism readily colonises the mesenteric lymph nodes, surviving there for many months and re-entering the intestinal lumen at times of stress or viral infection (Baxter and Leck. 1984). As an example, one British study in which salmonellae were isolated from the faeces of 0.5% of dogs that had been euthanased, the lymph nodes were infected in 4.5% (Baxter and Leck, 1984).

Carrier animals, too, only shed organisms periodically (Pelzer, 1989). Dogs typically shed Salmonella spp. for 20 to 40 days, but may shed for as long as 100 days. Shedding may be re-induced by stress or infection (Pelzer, 1989).

Despite this, 65% of dogs from Looma were shedding Salmonella organisms, which is much higher than found in other surveys. The average rate of Salmonella infections in dogs surveyed from 1947 to 1965 in the United States was 14% (Williams, 1980). Other reports from the
U.S.A. cite prevalences of 0.8% to 36.5% (Morse & Duncan, 1975; Baxter and Leck, 1984; Pelzer, 1989). In Alaska, 16% of dogs had Salmonella spp. isolated from rectal swabs (Pelzer, 1989) and British prevalence surveys have isolated Salmonella from the faeces of between 0.5 to 1% of all dogs (Baxter and Leck, 1984). Given the relatively high rate of Salmonella carriage in the Kimberley (possibly from feeding dogs contaminated meat), and the poor standards of domestic hygiene (Gracey, 1992) the risk for infection of humans from canine sources would be expected to be of significance.

5.5 Conclusion

Overall, scabies and hookworm were the most important canine parasites found in the present survey because of their high prevalence, clinical effects in the canine host and potential zoonotic significance. Roundworm, although not common, is also a parasite of zoonotic importance that is potentially controlled by ivermectin treatment. Heartworm and Spirocerca lupi, although not major zoonotic agents, are capable of disease in dogs and were found at high rates of infection in the present study.

Based on the prevalence and relative importance of each parasite, the thesis will continue to investigate the effect of the breeding and parasite control program on the infection rates and risk factors for infection with scabies, hookworm, Toxocara canis, Spirocerca lupi, Dirofilaria immitis and Giardia duodenalis.

Although hydatid disease is one of the most serious zoonoses in terms of clinical effects in infected humans, Echinococcus was not investigated further because of the low seroprevalence and lack of effect of ivermectin against cestodes. As mentioned, surveys for potential intermediate hosts need to be addressed before a major control campaign for hydatids is considered.
CANINE ZOONOSES IN ABORIGINAL COMMUNITIES:
THE EFFECTS OF A CANINE BREEDING
AND PARASITE CONTROL PROGRAM
IN THE KIMBERLEY REGION,
WESTERN AUSTRALIA

VOLUME 2

This thesis is presented for the degree of Doctor of
Philosophy of Murdoch University

by

KATHRYN MICHELLE WILKS

BSc, BVMS (Hons)
Chapter 6

PRELIMINARY TESTING OF THE IVERMECTIN TREATMENT PROTOCOL FOR CONTROL OF PARASITES

6.1 Introduction

The objectives of the preliminary trial conducted at Kununurra were to;

(a) assess the effectiveness of ivermectin in killing endo and ecto-parasites in a field situation,
(b) assess the effects of a single treatment on health status of the dogs,
(c) determine likely risk factors for the major parasites, and
(d) monitor any side effects of treatments with ivermectin.

The results of the trial were used to determine the regime to be used at the communities of the coastal, central and eastern regions for the three monthly treatments.

6.2 Methodology

The testing of the treatment and sampling regime took place at three communities in Kununurra. Kununurra is the eastern most town of the Kimberley region which supported a population of approximately 3,000 people (550 Aboriginal) in 1991 (Anon, 1993). The preliminary trial was conducted at the beginning of the dry season during which the mean daily maximum temperatures were between 30°C and 35°C and the mean daily minimum temperatures were between 12°C and 15°C (Bureau of Meteorology, 1996). Although the communities of the trial are separate, they share common borders. For this reason, the data from the communities was pooled.

Identification, sampling and treatment of dogs were as described for the general program (see 3.3), but faecal sampling was conducted weekly for 5 weeks after treatment (in April) and again at 3 months (13 weeks, July) after initial treatment.
6.2.1 Faecal Samples

6.2.1.1 The Effectiveness of Formalin Ethyl Acetate Sedimentation Technique for Recovering Hookworm Eggs and Larvae from Faecal Samples

The effectiveness of formalin ethyl acetate as a concentration technique for examination of preserved faecal samples for hookworm eggs and larvae was tested and compared with zinc sulphate flotation treatment of fresh samples (Appendix C). Pre-treatment samples were collected from 60 dogs. Of the 60 samples, 49 samples were of adequate size (approximately 10 grams each) to be equally divided into fresh and formalin preserved sub samples to be tested by both methods. The remaining 11 smaller (approximately 5 gram) samples were kept fresh and tested using only the ZnSO₄ method (Table 5.2). The fresh samples were refrigerated within 4 hours of collection and examined within 24 hours. The formalin fixed samples were stored for up to 2 months before examination. The whole procedure was repeated approximately 3 months later (after the July treatment) with another 49 samples. The samples collected at weekly intervals for 5 weeks after the first treatment were analysed without preservation using only the ZnSO₄ flotation technique. This method was more appropriate than the sedimentation technique to use at the temporary laboratory that did not have fume hoods and adequate disposal facilities for ethyl acetate.

6.2.1.2 Determination of Intestinal Parasite Infection

Crude approximations of intestinal hookworm intensities were made by categorising samples as light, moderate or heavy according to the number of eggs per coverslip, as recovered from the ZnSO₄ flotation technique. Light infections were those with 0-5 eggs per slip, moderate were those with 6-20 eggs per slip and heavy were those with more than 20 eggs per coverslip. This was only done on samples of similar quantity and consistency. As most owners of dogs at these communities were feeding their dogs commercial diets, the faeces were probably more consistent and egg counts more accurate than at other communities. Other intestinal parasite infections were recorded as being present or absent depending on the recovery of eggs, cysts or larvae of each parasite. The same technologist examined all individual specimens eliminating the possibility of inconsistent technical ability in examining the samples.
Identification of gastrointestinal parasites was based on morphology and size of eggs, larvae or cysts (Soulsby, 1982; Dunsmore and Shaw, 1990) (see Appendix D).

6.2.2 Skin Samples
Skin scrapings from dogs with clinically suspected *Sarcoptes scabiei* infection were taken pretreatment and processed as described (see 3.3.7.1). Post treatment scrapings were also taken from dogs that were not recovering as quickly as others after treatment.

6.2.3 Blood Samples
Blood samples were collected from 71 dogs before treatment to measure total serum protein, albumin, total red cell counts, haemoglobin and to perform microfilariae tests. Three months (13 weeks) after a single treatment with ivermectin (200µg/kg), 54 dogs were re-sampled.

For each dog, 5 to 10mL of blood were collected into plain (serum) tubes (‘VACUTAINER’ Becton Dickinson, 10mL and 5mL) and a further 2 mL were collected into EDTA coated tubes (‘VACUTAINER’ Becton Dickinson, 5mL) for microfilariae tests. Serum was decanted from the samples after 6 to 12 hours and frozen immediately for transport to Perth. The blood samples collected into EDTA tubes were examined within 24 hours of collection.

6.2.3.1 Measurement of Albumin and Total Serum Protein
A “Cobas Mira” biochemistry analyser (ROCHE, Switzerland, 1986 model) was used to measure albumin and total serum protein.

Albumin concentrations were determined by bromocresol green dye binding. This procedure may give false high results with low albumin levels as a result of attachment of the dye to other proteins. Alternatively, false low readings may occur when bilirubin, anticonvulsants and certain antibiotics compete with albumin for the dye and cause a color shift different from that caused by albumin (Duncan and Prasse, 1986). None of the sampled dogs were receiving any
antibiotics and all samples appeared clear (free of bilirubin). The normal range for serum albumin is 230 to 430 g/L for dogs (Duncan and Prasse, 1986).

The colorimetric biuret method was used to determine total serum protein. Accuracy is lost with low serum protein levels, but the procedure is very precise for the normal range (Duncan and Prasse, 1986). Using this method, values for normal dogs are within 55 to 75 g/L (see Table 6.1). Serum globulin levels were determined by subtracting albumin levels from total serum protein. The normal range for serum globulins is between 25 and 45 g/L (Mills and Sutherland, 1994).

The hydration status of the sampled dogs was calculated because a decreased blood volume results in spurious increases in haematological and biochemical concentrations. Dehydration (or absolute reduced blood volume) was indicated by hyperproteinaemia with an elevated albumin level, but a normal albumin to globulin ratio (see normal serum protein levels in Table 6.1).

### 6.2.3.2 Haematology

Packed Cell Volumes (PCV) were used to identify anaemic dogs during the survey of September 1992 (see 7.3.2.4.2), but haemoglobin levels were determined in the preliminary trial at Kununurra. Although haemoglobin levels provide no clinical advantage over PCV readings, they do allow the determination of mean corpuscular haemoglobin content (MCHC) and provide the most direct indication of oxygen carrying capacity.

The haematological tests (haemoglobin, red blood cell counts, mean corpuscular volume and mean corpuscular haemoglobin content) in this survey were performed on a “Cobas Minos Vet” (ROCHE, France, 1993 model) haematology analyser.
The haemoglobin concentration was colorimetrically determined by the cyanmethaemoglobin method, which usually gives an accuracy of ±5% (Duncan and Prasse, 1986). Normal haematological values are shown in Table 6.1.

**Table 6.1 Parasitology and Haematology Methods**

<table>
<thead>
<tr>
<th>PARASITOLOGY</th>
<th>METHOD</th>
<th>TEST RANGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gastrointestinal Parasites</td>
<td>ZnSO₄ flotation (Dunsmore and Shaw, 1990)</td>
<td>Score 1: 1-5 eggs or cysts per slide</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Score 2: 6-20 eggs</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Score 3: &gt;20 eggs</td>
</tr>
<tr>
<td>Gastrointestinal parasites</td>
<td>Formalin ethyl acetate sedimentation</td>
<td>Positive: One or more eggs or cysts per slide</td>
</tr>
<tr>
<td></td>
<td>(Young et al., 1979)</td>
<td></td>
</tr>
<tr>
<td>Scabies</td>
<td>Skin scraping (based on Soulsby, 1982)</td>
<td>Positive: One or more eggs or mites recovered from up to 10 skin scrapings.</td>
</tr>
<tr>
<td>Scabies</td>
<td>Visual inspection (based on Pence et al., 1983)</td>
<td>Score 1: Up to 25% alopecia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Score 2: 25%-50%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Score 3: 50%-75%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Score 4: 75%-100%</td>
</tr>
<tr>
<td>Dirofilaria immitis</td>
<td>Wylie Method (Wylie, 1970)</td>
<td>Positive: One or more microfilariae per filter.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>HAEMATOLOGY</th>
<th>METHOD</th>
<th>NORMAL VALUES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Haemoglobin</td>
<td>Cyanmethaemoglobin method</td>
<td>120 to 180g/L (Jain, 1993)</td>
</tr>
<tr>
<td>Mean Corpuscular Volume</td>
<td>Automatic counts – haematology analyser</td>
<td>60-77fL (Jain, 1993)</td>
</tr>
<tr>
<td>Mean Corpuscular Haemoglobin Concentration</td>
<td>Haematology analyser</td>
<td>320-360g/L (Jain, 1993)</td>
</tr>
<tr>
<td>Total Serum Protein (TSP)</td>
<td>Colorimetric biuret method</td>
<td>55-75g/L (Mills and Sutherland, 1994)</td>
</tr>
<tr>
<td>Albumin</td>
<td>Bromocresol green dye binding</td>
<td>20-40g/L (Duncan and Prasse, 1986)</td>
</tr>
<tr>
<td>Globulin</td>
<td>TSP value subtract Albumin level</td>
<td>25-45g/L (Mills and Sutherland, 1994)</td>
</tr>
</tbody>
</table>

**6.3.4 Side Effects to Treatments with Ivermectin**

Prior to treatment, owners were warned of possible side effects to treatment with 200μg/kg ivermectin, especially in those dogs that had heartworm. Owners were told to contact the research group if they noticed any of the following signs after treatment; vomiting, excess salivation, diarrhoea, depression, inappetence (Pulliam and Preston, 1989), prolonged pain at the site of injection or change in behaviour.
As the treatments were conducted over two day’s duration, the research team was in close contact to deal with any immediate reactions.

During the treatment, dogs exhibiting pain to the subcutaneous injections were noted. Over the course of sampling every week, owners were continually questioned about any disagreeable reactions. A general physical examination was made of each dog prior to sampling to check for abnormalities such as skin discoloration or swelling at the site of injection.

6.3.5 Post Mortem Examinations

Six dogs were euthanased at the request of their owners and complete post mortems were conducted. Gross examinations were made of the entire length of the opened alimentary tract, trachea, bronchi, lungs, heart and pulmonary vessels. Worms recovered from the viscera were washed in 0.9% saline and preserved in 10% buffered formalin for later examination to determine species. Detailed observations of the coat of each dog were undertaken immediately after euthanasia to detect the presence of ectoparasitic arthropods. Visible ectoparasites were collected into 70% ethyl alcohol with 5% glycerin (Soulsby, 1982; Dunsmore and Shaw, 1990).

Skin samples from dogs exhibiting mange were also preserved and later examined for Sarcopes mites as described (see 3.3.7.1).

6.3 Results

6.3.1 Effectiveness of Formalin Ethyl Acetate Sedimentation Technique for Recovering Hookworm Eggs and Larvae from Faeces

The formalin ethyl acetate sedimentation (FEAS) technique was more sensitive than the ZnSO₄ flotation method in detecting hookworm eggs (average sensitivity of 88% compared with 61% for ZnSO₄ flotation) (see Table 6.2). The recovery of eggs from the samples taken after treatment was almost twice as effective when using the formalin ethyl acetate technique rather than the flotation method.
Table 6.2: Comparison of Results of ZnSO₄ Flotation and Formalin Ethyl Acetate Sedimentation Techniques for Intestinal Hookworms in 60 Dogs from Kununurra

<table>
<thead>
<tr>
<th></th>
<th>ZnSO₄ Flotation</th>
<th>Formalin Ethyl Acetate Sedimentation</th>
<th>Total Number of Samples with Hookworm (Determined from Both Tests)</th>
<th>Average Sensitivity*** for Both Test Dates</th>
</tr>
</thead>
<tbody>
<tr>
<td>Week 0</td>
<td>37 (80%)*</td>
<td>41 (89%)*</td>
<td>46</td>
<td>88%</td>
</tr>
<tr>
<td>Week 13</td>
<td>8 (47%)**</td>
<td>15 (88%)**</td>
<td>17</td>
<td>61%</td>
</tr>
</tbody>
</table>

* No statistical difference (P>0.05)
** Statistically different P=0.01
*** Sensitivity defined as the proportion of samples that have hookworm eggs that are positive when subjected to each of the tests (proportion of ‘true positive’ samples).
6.3.2 Parasites of the Dogs of the Kununurra Communities

The initial survey by skin appraisal, faecal examination and Wylie microfilariae testing of the blood samples revealed the most prevalent parasite to be *Ancylostoma caninum* (75% [64.1, 85.9]) followed by heartworm (53.5% [42.0, 65.1]) (see Figure 6.1). External parasites were also common with 53.4% [42.0, 64.8] and 50.7% [39.2, 62.2] of dogs having ticks (*Rhipicephalus sanguineus*) and scabies lesions, respectively. Skin scrapings from 10 dogs with severe lesions indicative of scabies were taken and scabies mites and eggs were recovered from 7 of the dogs.

**Figure 6.1: Pre-treatment Prevalence of Parasites in Dogs from Kununurra**

Of much lower prevalence were *Giardia duodenalis* (8.2% [1.3, 15.1]), *Sarcocystis* spp. (3.3% [0, 7.8]) and *Isospora canis* (1.6% [0, 4.7]). Two important zoonotic parasites, *Toxocara canis* and *Echinococcus granulosus*, were not found, although *Echinococcus* spp. eggs or proglottids are rarely detected by faecal examination without purgation (Urquhart et al, 1987).

As the two most prevalent zoonotic parasites were hookworm and scabies, further analyses of the effect of treatment, risk factors for infection and associations of infection with health parameters were undertaken in the present survey.
6.3.3 Efficacy of Ivermectin in the Treatment of Scabies and Hookworm

It is estimated that 86% of the dogs in the communities were treated (n=73) at the first visit in April as 12 additional new dogs were seen during the subsequent 5 visits. These 12 dogs were treated with 200μg/kg ivermectin when they were first examined.

6.3.3.1 Weekly Scabies Prevalence after Treatment with 200μg/kg Ivermectin

After an initial pre-treatment prevalence of 50.7% [43.5, 56.1], the percentage of dogs with alopecia dropped to an average of 35.7% [30.5, 40.9] for the following 5 weeks (P>0.05) (see Figure 6.2). By 13 weeks after treatment, the rate was 10.1% [3.0, 17.2] indicating that one treatment every 3 months was effective at reducing the prevalence of mange. In addition, there was no evidence of secondary infections or active lesions after treatment.

Seven dogs did not show complete recovery after the treatment, but six of these dogs had moderate to severe mange (scores higher than one) prior to the ivermectin treatment. No scabies mites were isolated from multiple skin scrapings taken from each of the seven dogs 13 weeks after treatment (July).

Figure 6.2: Scabies Infection Rates in Dogs from Kununurra before and after a Single Treatment with 200μg/kg Ivermectin
6.3.3.2 Hookworm Prevalence after Treatment

The prevalence of hookworm in dogs prior to treatment was 75% (see Table 6.3). One week after treatment, 3 out of 45 sampled dogs that had hookworm before treatment (6.7% [0, 10.4]) still had *A. caninum* eggs recovered from the faeces.

The percentage of dogs shedding eggs increased to 19% [6.4, 31.6] by 5 weeks post treatment.

The measurements of intensity of infection (ova/coverslip) revealed that initially, 23.4% [1.2, 45.6] (n=14) had infections of score 2 and 3 (see Figure 6.3). Only at week 13 were scores higher than 1 recorded again (8.2% [0.5, 15.6] of dogs sampled). At week 13, there was no difference in the proportion of infected dogs with each score compared to pretreatment data (P>0.05).

Figure 6.3: Prevalence of Hookworm (*Ancylostoma caninum*) in Dogs from Kununurra before and after a Single Treatment with 200µg/kg Ivermectin
### Table 6.3: Prevalence of Hookworm* in Dogs Treated with 200μg/kg Ivermectin at Kununurra on 12 April (Week 0)

<table>
<thead>
<tr>
<th>Week</th>
<th>Total Sampled</th>
<th>Number Positive by Faecal Examination</th>
<th>Percentage Positive</th>
<th>Actual Number Positive**</th>
<th>Percentage Positive</th>
<th>% Difference***</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 (Pre-treatment)</td>
<td>60</td>
<td>45</td>
<td>75</td>
<td>45</td>
<td>75</td>
<td>0</td>
</tr>
<tr>
<td>Week 1</td>
<td>51</td>
<td>3</td>
<td>5.9</td>
<td>3</td>
<td>5.9</td>
<td>0</td>
</tr>
<tr>
<td>Week 2</td>
<td>50</td>
<td>5</td>
<td>10</td>
<td>5</td>
<td>10</td>
<td>0</td>
</tr>
<tr>
<td>Week 3</td>
<td>50</td>
<td>3</td>
<td>6</td>
<td>4</td>
<td>8</td>
<td>2</td>
</tr>
<tr>
<td>Week 4</td>
<td>49</td>
<td>3</td>
<td>6.1</td>
<td>6</td>
<td>12.2</td>
<td>6.1</td>
</tr>
<tr>
<td>Week 5</td>
<td>37</td>
<td>4</td>
<td>11</td>
<td>7</td>
<td>18.9</td>
<td>8.1</td>
</tr>
<tr>
<td>Week 13</td>
<td>49</td>
<td>10</td>
<td>20</td>
<td>14</td>
<td>28.6</td>
<td>8.2</td>
</tr>
</tbody>
</table>

* Based on ZnSO₄ flotation tests only
** Includes results from samples from all dogs that had converted from negative to positive hookworm status and were later sampled
*** Difference between true prevalence and prevalence determined from single faecal examination
6.3.4 The Risk Factors for Canine Infection with Scabies and Hookworm

6.3.4.1 Sex of Host

For both scabies and hookworm, there was no statistical difference between infection rates for both male and females (Tables 6.4 and 6.5). When age was controlled and only adults examined, there still was no sex predilection for scabies or hookworm infection.

6.3.4.2 Age of Host

There was no age predilection for scabies or hookworm infection found for the dogs of Kununurra (see Tables 6.4 and 6.5).

6.3.5 Side Effects of Ivermectin Treatment

Some dogs did vocalise during the ivermectin treatments (as well as the implantation of the microchips) but no other adverse reactions were noticed immediately after treatment. Likewise, during the 5 weeks of weekly examinations of dogs, no side effects to ivermectin administration were noted, including the 53.5% of dogs with circulating Dirofilaria immitis microfilariae.

6.3.6 The Effect of a Single Treatment with 200µg/kg Ivermectin on Dog Health Parameters

6.3.6.1 Hydration Status

Hydration status of the dogs had to be determined to interpret the haematology values (see 3.3.7.3). A decreased blood volume can lead to a false increase in blood cell and protein values. From the combined samples (n=99) before and after treatment, 23% ([14.8, 31.2]) had hyperproteinaemia, but only 13% of these had normal albumin to globulin ratios indicative of dehydration (see Table 6.6). Normal albumin to globulin ratios are expected with loss of water as both protein concentrations increase to the same degree. All of the dehydrated dogs were adults and two of the samples from dehydrated dogs were collected in April (before treatment).
Table 6.4: Risk Factors Associated with Scabies Infection in Dogs – Kununurra

<table>
<thead>
<tr>
<th>Factor Tested</th>
<th>Category</th>
<th>Scabies Positive</th>
<th>Scabies Negative</th>
<th>Percentage</th>
<th>Odds Ratio</th>
<th>95% Confidence Intervals</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>≤ 1 year</td>
<td>13</td>
<td>19</td>
<td>40.6</td>
<td>1.7</td>
<td>0.8, 3.9</td>
</tr>
<tr>
<td></td>
<td>&gt; 1 year</td>
<td>31</td>
<td>79</td>
<td>28.2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sex</td>
<td>male entire</td>
<td>22</td>
<td>47</td>
<td>31.9</td>
<td>1.3</td>
<td>0.6, 2.7</td>
</tr>
<tr>
<td></td>
<td>female entire</td>
<td>22</td>
<td>62</td>
<td>26.2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Treatment</td>
<td>Pre-treatment April</td>
<td>37</td>
<td>36</td>
<td>50.7</td>
<td>9.1</td>
<td>3.7, 22.5</td>
</tr>
<tr>
<td></td>
<td>Post-treatment July</td>
<td>7</td>
<td>62</td>
<td>10.1</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Table 6.5: Risk Factors Associated with Hookworm Infection in Dogs - Kununurra

<table>
<thead>
<tr>
<th>Factor Tested</th>
<th>Category</th>
<th>Hookworm Positive</th>
<th>Hookworm Negative</th>
<th>Percentage</th>
<th>Odds Ratio</th>
<th>95% Confidence Intervals</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex (Adult Dogs Only)</td>
<td>male entire</td>
<td>26</td>
<td>17</td>
<td>60</td>
<td>1.9</td>
<td>0.8, 4.2</td>
</tr>
<tr>
<td></td>
<td>female entire</td>
<td>23</td>
<td>28</td>
<td>45</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>≤ 1 year</td>
<td>12</td>
<td>9</td>
<td>57</td>
<td>1.2</td>
<td>0.5, 3.2</td>
</tr>
<tr>
<td></td>
<td>&gt; 1 year</td>
<td>49</td>
<td>45</td>
<td>48</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Treatment</td>
<td>Pre-treatment April</td>
<td>44</td>
<td>17</td>
<td>72</td>
<td>5.6</td>
<td>2.5, 12.7*</td>
</tr>
<tr>
<td></td>
<td>Post-treatment July</td>
<td>17</td>
<td>37</td>
<td>31</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Hookworm infection determined from combined results of ZnSO₄ and FEAS tests
*Statistically significant
Table 6.6: Hydration Status of Dogs - Kununurra

<table>
<thead>
<tr>
<th></th>
<th>Total Number Sampled</th>
<th>Number of Dogs with Total Serum Proteins &gt;75 g/L</th>
<th>Number and Percentage of Hyperproteinaemic Dogs with Low Albumin: Globulin (A/G Ratios)</th>
<th>Number and Percentage with Clinical Dehydration (Normal A/G Ratios)</th>
<th>PCV Range (L/L)</th>
<th>Mean PCV (L/L)</th>
<th>Number with Hyperglobulinaemia (&gt;45g/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total</td>
<td>98</td>
<td>23</td>
<td>20 (20.4%)</td>
<td>3 (3.1%)</td>
<td>21-54</td>
<td>41.5± SD 8.8</td>
<td>20</td>
</tr>
<tr>
<td>April</td>
<td>68</td>
<td>21</td>
<td>19 (28.0%)</td>
<td>2 (3.0%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>July</td>
<td>30</td>
<td>2</td>
<td>1 (3.3%)</td>
<td>1 (3.3%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Puppies and Juveniles</td>
<td>20</td>
<td>3</td>
<td>3 (15.0%)</td>
<td>0</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adults</td>
<td>68</td>
<td>20</td>
<td>17 (22%)</td>
<td>3 (4.4%)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
6.3.6.2 Haemoglobin Levels

The Effect of Sex and Age on Haemoglobin Levels

Dogs less than one year of age were found to be 5 times more likely than adult dogs to have low haemoglobin values [1.9, 14.4] (see Table 6.7). The sex of the dogs did not affect haemoglobin levels.

The Effect of a Single Treatment with Ivermectin on Haemoglobin Levels

The mean haemoglobin concentration of matched samples (n=54) from the same dogs prior to treatment (114.73 g/L SEM ± 3.85) was significantly lower than the mean three months after treatment (144.33 g/L SEM ± 3.41; paired t test, P<0.00001) (see Figure 6.4). The mean of the difference between the paired samples was 29.60 g/L. When only adult dogs were examined (n=40), paired t tests of matched samples also showed a statistical difference in haemoglobin content before and after treatment (116.95 g/L SEM ± 4.53 vs. 146.95 g/L SEM ± 3.99, P<0.00001).

Prior to treatment, the dogs were found to be approximately 6 times more likely to have anaemia (haemoglobin < 120 g/L) than three months after treatment [2.5, 13.0] (see Table 6.7).

The Effect of Scabies Infection on Haemoglobin Levels

High scabies infection rates were found to be associated with low haemoglobin concentration when pretreatment and post-treatment data from Kununurra were examined (see Table 6.7). As the prevalence of scabies and hookworm were high in this investigation, the effect of hookworm was controlled by analysing data from samples from dogs without hookworm. An association between scabies infection and low haemoglobin was found. When age and hookworm status was controlled, no association between scabies infection and mange infection rate was found. This demonstrates the effect of confounding variables on data.
Table 6.7: Risk Factors Associated with Low Haemoglobin Levels in Dogs - Kununurra

<table>
<thead>
<tr>
<th>Factor Tested</th>
<th>Category</th>
<th>Haemoglobin (&lt;120g/L)</th>
<th>Haemoglobin (≥120g/L)</th>
<th>Percentage</th>
<th>Odds Ratio</th>
<th>95% Confidence Intervals</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td>male entire</td>
<td>24</td>
<td>37</td>
<td>39</td>
<td>0.9</td>
<td>0.5, 1.9</td>
</tr>
<tr>
<td></td>
<td>female entire</td>
<td>26</td>
<td>38</td>
<td>41</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>≤ 1 year</td>
<td>16</td>
<td>6</td>
<td>73</td>
<td>5.2</td>
<td>1.9, 14.4*</td>
</tr>
<tr>
<td></td>
<td>&gt; 1 year</td>
<td>35</td>
<td>68</td>
<td>34</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Treatment</td>
<td>Pre-treatments April</td>
<td>40</td>
<td>31</td>
<td>56</td>
<td>5.7</td>
<td>2.5, 13.0*</td>
</tr>
<tr>
<td></td>
<td>Post-treatment July</td>
<td>10</td>
<td>44</td>
<td>18</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Statistically significant
Table 6.7 (Cont.): Risk Factors Associated with Low Haemoglobin Levels in Dogs - Kununurra

<table>
<thead>
<tr>
<th>Factor Tested</th>
<th>Category</th>
<th>Haemoglobin (&lt;120 g/L)</th>
<th>Haemoglobin (≥ 120 g/L)</th>
<th>Percentage</th>
<th>Odds Ratio</th>
<th>95% Confidence Intervals</th>
</tr>
</thead>
<tbody>
<tr>
<td>Scabies</td>
<td>Positive</td>
<td>25</td>
<td>15</td>
<td>62.5</td>
<td>4.0</td>
<td>1.81, 8.83</td>
</tr>
<tr>
<td></td>
<td>Negative</td>
<td>25</td>
<td>60</td>
<td>29.4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Scabies (Hookworm Negative Dogs Only)</td>
<td>Positive</td>
<td>7</td>
<td>6</td>
<td>53.8</td>
<td>4.8</td>
<td>1.2, 19.0</td>
</tr>
<tr>
<td></td>
<td>Negative</td>
<td>7</td>
<td>29</td>
<td>19.4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Scabies (Hookworm Negative Adult Dogs Only)</td>
<td>Positive</td>
<td>4</td>
<td>5</td>
<td>44.4</td>
<td>3.5</td>
<td>0.7, 16.9</td>
</tr>
<tr>
<td></td>
<td>Negative</td>
<td>6</td>
<td>26</td>
<td>18.8</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Scabies (Treated Adult Dogs Only)</td>
<td>Positive</td>
<td>17</td>
<td>13</td>
<td>56.7</td>
<td>1.4</td>
<td>0.5, 4.1</td>
</tr>
<tr>
<td></td>
<td>Negative</td>
<td>12</td>
<td>13</td>
<td>48</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hookworm (Adult Dogs Only)</td>
<td>Positive</td>
<td>20</td>
<td>10</td>
<td>67</td>
<td>3.3</td>
<td>1.3, 8.5</td>
</tr>
<tr>
<td></td>
<td>Negative</td>
<td>21</td>
<td>35</td>
<td>38</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hookworm (Pre-Treatment Only)</td>
<td>Positive</td>
<td>26</td>
<td>18</td>
<td>59.1</td>
<td>1.4</td>
<td>0.5, 4.6</td>
</tr>
<tr>
<td></td>
<td>Negative</td>
<td>8</td>
<td>8</td>
<td>50</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hookworm (Treated Adult Dogs Only)</td>
<td>Positive</td>
<td>18</td>
<td>16</td>
<td>52.9</td>
<td>1.1</td>
<td>0.3, 4.2</td>
</tr>
<tr>
<td></td>
<td>Negative</td>
<td>6</td>
<td>6</td>
<td>50</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hookworm Burden</td>
<td>&gt; Score 1</td>
<td>13</td>
<td>5</td>
<td>72.2</td>
<td>3.9</td>
<td>1.2, 13.1</td>
</tr>
<tr>
<td></td>
<td>Score 1</td>
<td>16</td>
<td>24</td>
<td>40</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ticks</td>
<td>Positive</td>
<td>26</td>
<td>44</td>
<td>37.1</td>
<td>1.0</td>
<td>0.5, 2.0</td>
</tr>
<tr>
<td></td>
<td>Negative</td>
<td>25</td>
<td>43</td>
<td>36.8</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Statistically significant
Figure 6.4: Blood Parameters (Haemoglobin Concentration, Mean Corpuscular Haemoglobin Concentration, Mean Corpuscular Volume and Serum Globulin) of Paired Samples from Dogs before and after a Single Treatment with 200μg/kg Ivermectin

The Effect of Hookworm Status on Haemoglobin Levels

When all of the samples from adult dogs before and after treatment were pooled, hookworm infection was found to be associated with low haemoglobin levels (OR 3.3 [1.3,8.5] (Table 6.7). When only adult dogs before treatment were considered, low haemoglobin values were not associated with hookworm infection (P=0.86). High hookworm burden, though, was associated with low haemoglobin concentration as dogs with greater than ‘score 1’ hookworm were 4 times more likely to have below normal haemoglobin levels than score 1 dogs.
6.3.6.3 Mean Corpuscular Haemoglobin Concentration (MCHC) and Mean Corpuscular Volume (MCV)

The MCHC was not found to be statistically associated with any of the risk factors (see Table 6.8), although all of the dogs less than one year of age had below normal MCHC. There was no statistical difference in the mean MCHC of matched samples (n=54) taken prior to treatment (309.79 ± 2.92 g/L) and three months after treatment (313.08 ± SEM 1.88 g/L)(paired t test, p=0.18) (see Figure 6.4). Abnormally low concentrations (<320g/L) were found in 72% of all samples.

Before treatment, nine dogs (12.7%) had MCV less than normal (<60fL). All of these dogs were adults, anaemic and had low MCHC and MCV readings indicating that they had microcytic and hypochromic anaemia. Matched samples from the same dogs (n=54) did not reveal any statistical difference in values before and after treatment (see Figure 6.4), although only 3 dogs still had abnormally low MCV after treatment. No samples had a MCV greater than normal (>77fL).

6.3.6.4 Globulin

The age, sex or hookworm status of the dogs were not found to be associated with hyperglobulinaemia (see Table 6.9).

The Effect of Treatment with Ivermectin on Globulin Levels

The mean of the globulin levels of matched samples (n=30) before treatment (42.79 ± SEM 1.64) was significantly greater than three months after treatment (32.77 ± SEM 1.42; paired t test, P=0.0002)(see Figure 6.4). Overall, dogs after treatment were 8 times more likely to have normal globulin levels than before treatment [1.8, 37.1] (see Table 6.9).
Table 6.8: Risk Factors Associated with Low Mean Corpuscular Haemoglobin Concentrations (MCHC) in Dogs - Kununurra

<table>
<thead>
<tr>
<th>Factor Tested</th>
<th>Category</th>
<th>MCHC (&lt;320 g/L)</th>
<th>MCHC (≥ 320 g/L)</th>
<th>Percentage</th>
<th>Odds Ratio</th>
<th>95% Confidence Intervals</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td>male entire</td>
<td>42</td>
<td>19</td>
<td>68.9</td>
<td>0.7</td>
<td>0.3, 1.6</td>
</tr>
<tr>
<td></td>
<td>female entire</td>
<td>48</td>
<td>16</td>
<td>75</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>≤ 1 year</td>
<td>22</td>
<td>0</td>
<td>100</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>&gt; 1 year</td>
<td>71</td>
<td>32</td>
<td>76.3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hookworm (Adult Dogs Only)</td>
<td>Positive</td>
<td>27</td>
<td>16</td>
<td>62.8</td>
<td>0.9</td>
<td>0.4, 2.1</td>
</tr>
<tr>
<td></td>
<td>Negative</td>
<td>33</td>
<td>18</td>
<td>64.7</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Treatment</td>
<td>Pre-treatment April</td>
<td>49</td>
<td>22</td>
<td>69</td>
<td>0.7</td>
<td>0.3, 1.6</td>
</tr>
<tr>
<td></td>
<td>Post-treatment July</td>
<td>41</td>
<td>13</td>
<td>75.9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Factor Tested</td>
<td>Category</td>
<td>Globulin (&gt; 45 g/L)</td>
<td>Globulin (≤ 45 g/L)</td>
<td>Percentage</td>
<td>Odds Ratio</td>
<td>95% Confidence Intervals</td>
</tr>
<tr>
<td>----------------</td>
<td>------------------------</td>
<td>---------------------</td>
<td>---------------------</td>
<td>------------</td>
<td>------------</td>
<td>--------------------------</td>
</tr>
<tr>
<td>Age</td>
<td>≤ 1 year</td>
<td>3</td>
<td>17</td>
<td>15</td>
<td>0.4</td>
<td>0.1, 1.5</td>
</tr>
<tr>
<td></td>
<td>&gt; 1 year</td>
<td>24</td>
<td>54</td>
<td>31</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sex</td>
<td>male entire</td>
<td>17</td>
<td>32</td>
<td>35</td>
<td>2.1</td>
<td>0.8, 5.2</td>
</tr>
<tr>
<td></td>
<td>female entire</td>
<td>10</td>
<td>39</td>
<td>20</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Scabies</td>
<td>positive</td>
<td>18</td>
<td>19</td>
<td>49</td>
<td>4.3</td>
<td>0.4, 2.9*</td>
</tr>
<tr>
<td></td>
<td>negative</td>
<td>11</td>
<td>50</td>
<td>19</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Scabies (Pre-Treatment Only)</td>
<td>positive</td>
<td>16</td>
<td>18</td>
<td>47.1</td>
<td>2.5</td>
<td>1.7, 9.8*</td>
</tr>
<tr>
<td></td>
<td>negative</td>
<td>9</td>
<td>25</td>
<td>26.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hookworm</td>
<td>positive</td>
<td>15</td>
<td>37</td>
<td>29</td>
<td>1.1</td>
<td>0.9, 6.8</td>
</tr>
<tr>
<td></td>
<td>negative</td>
<td>8</td>
<td>21</td>
<td>28</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Treatment</td>
<td>Pre-treatment April</td>
<td>25</td>
<td>43</td>
<td>37</td>
<td>8.1</td>
<td>1.8, 37.1*</td>
</tr>
<tr>
<td></td>
<td>Post-treatment July</td>
<td>2</td>
<td>28</td>
<td>6.7</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Statistically significant
The Effect of Scabies Infection on Globulin Levels

With no factors controlled, dogs with scabies were 4 times more likely to have hyperglobulinaemia than those without (see Table 6.9). As dogs were overall found to have higher globulin levels before treatment than after, pretreatment data was examined to control the effect of treatment. In this case, only marginal association between scabies infection rates and hyperglobulinaemia was found (P=0.08).

6.3.6.5 Albumin

Only one sample (n=99) from Kununurra was hypoalbuminaemic. All the other samples were within the normal limits for serum albumin (20-40g/L).

6.3.6.6 The Effect of a Single Treatment with 200µg/kg Ivermectin on Other Parasites

Ticks (*Rhipicephalus sanguineus*)

Ticks were the only parasites (apart from scabies and hookworm) where a single treatment with 200µg/kg of ivermectin resulted in any measurable effect on the prevalence of infection in the present study (Figure 6.5). Prior to treatment, 53.4% [42.0, 64.8] of dogs were infected with ticks. One week after treatment, the prevalence of tick infestation dropped to 17.4% [8.5, 26.3]. By 5 weeks after treatment, there was no statistical difference in prevalence of tick infestation (47.1% [33.4, 60.8]) compared with the pre-treatment prevalence.
6.4 Discussion

6.4.1 Effectiveness of Formalin Ethyl Acetate Sedimentation (FEAS) Technique for Recovering Hookworm Eggs from Faeces

Comparisons between formalin ether sedimentation (which is similar to formalin ethyl acetate sedimentation) and modified zinc sulphate flotation tests (using formalinised samples) have shown the sedimentation technique to be more sensitive at detecting a wide variety of parasite eggs, larvae and cysts (Bartlett et al., 1978). Before the treatments, in the present study, the sensitivity of FEAS technique and ZnSO₄ flotation to detect hookworm eggs were equivalent with an average of 88%. After treatments, though, when low egg counts were likely, the FEAS technique was almost twice as effective at detecting hookworm eggs. Bartlett et al (1978) found hookworm eggs to be recovered more frequently with formalin ether sedimentation than ZnSO₄ flotation (92% c.f. 71%) especially in situations with low egg counts (Bartlett et al., 1978). Truant, Elliott, Kelly and Smith (1981), by contrast, found ZnSO₄ flotation of formalinised samples to be more effective than FEAS technique for the detection of hookworm eggs.
Although the effectiveness of the FEAS technique to detect parasites in long stored samples or fresher formalinised samples was not verified in the present study, Bartlett et al (1978), found storage of samples for over a month did not adversely affect the recovery of hookworm (91% of samples were detected compared with 95% for fresher samples).

The prevalence of other parasites in the present study was too low to compare between the two methods (see Figure 6.1). Despite this, Giardia, Sarcocystis and Isospora cysts were detected by both techniques, although background detritus in the formalin ethyl acetate sedimentation test did make identification of these protozoan cysts more difficult. The flotation technique is considered to be better at detecting light infections with protozoa (Seah et al, 1975; Truant et al, 1981). Eggs of other parasites were not found in the present survey, but the formalin ethyl acetate sedimentation technique is better for detecting heavier eggs (such as Toxocara canis and Trichuris spp.) and larvae than flotation (Seah et al, 1975; Truant, 1981).

Based on the results of this survey, the formalin ethyl acetate sedimentation procedure was used for the rest of the program, although ZnSO₄ flotation was used to examine faeces in the survey of this chapter due to limited laboratory facilities available at Kununurra (see 6.2.1.1).

6.4.2 Parasites of the Dogs of the Kununurra Communities

Ancylostoma caninum was the most frequently encountered parasite in the present survey, which was also the case of the only other survey of canine parasites in the Kimberley region, by Thompson et al (1993a). In view of the warm climate and lack of anthelmintic treatment of dogs, this finding was expected.

Dirofilaria immitis was also common at Kununurra (53.5%) which was also expected considering the available breeding locations (including a large lake adjacent to the town) for the mosquito intermediate hosts. It is likely that the true prevalence of heartworm is greater than found here due to undetected occult infections (see 5.4.4.3). It is surprising, though, that
Thompson et al (1993a) did not find any heartworm-infected dogs amongst the 21 samples examined by the Wylie method and 188 dogs examined at post mortem from Fitzroy River communities.

*Sarcoptes scabiei* affected about 50% of the dogs in the present study to varying degrees. Thompson et al (1993a) also found lesions indicative of sarcoptic mange in most of the dogs examined from communities of the Fitzroy River Valley area. Surveys of other Aboriginal communities in the Northern Territory have also shown scabies to be a common skin complaint of dogs (Presson et al, 1989). Ticks were another common parasite in the present survey. Although (*Rhipicephalus sanguineus*) ticks are not of zoonotic importance, they are capable of causing dermatitis and anaemia in heavy infestations and are also an intermediate host for *Babesia canis* (not yet detected in the Kimberley region) (Dunsmore and Shaw, 1990).

*Giardia duodenalis* cysts were recovered from 8.2% of dogs in the present survey. This is less than found at the Fitzroy Valley communities where 17% of dogs had cysts in their faeces. It is also much less than found in a study based in the Perth metropolitan area (21% (Swan and Thompson, 1986). Although serial testing of the same dogs would reveal a higher prevalence, the compared studies from Perth and the Fitzroy Valley were both based on a single faecal sample from each dog.

*Toxocara canis* and *Echinococcus granulosus* were not found during the present survey and subsequent samplings during the test period at Kununurra. *Toxocara canis* has been found at Fitzroy Crossing, but only at a low prevalence (2/188), even though puppies were sampled (Thompson et al, 1993a). No serology to check for *E. granulosus* antibodies was done at Kununurra, so the true prevalence remains unknown.
Of the parasites found at Kununurra, *Sarcoptes scabiei* and *Ancylostoma caninum* were the most common of clinical and zoonotic importance that are also susceptible to ivermectin treatment. For these reasons, most of the present trial focused on control of these parasites.

### 6.4.3 Efficacy of Ivermectin in Treatment of Scabies and Hookworm

#### 6.4.3.1 Scabies Prevalence after a Single Treatment of Dogs with 200μg/kg Ivermectin

A single treatment with 200μg/kg subcutaneous ivermectin was sufficient to improve the skin condition of the dogs at Kununurra. Within 5 weeks, approximately 30% of scabietic dogs had shown complete recovery. After 3 months only 10% of the original dogs still had alopecia, although signs of hair regrowth were evident.

Most other studies into the effects of treatment of scabietic dogs with ivermectin have been laboratory based. Arlian, Morgan, Rapp and Vyszenski-Moher (1995) found dogs that had been artificially infected with *Sarcoptes* mites for 8 weeks had their clinical signs abating 2 weeks after treatment. Yazwinski *et al* (1981) found treatment of single housed dogs to be completely effective by 2 weeks, as determined by lack of mites from skin scrapings. Likewise, dogs were showing marked cutaneous improvement by 3 days after treatment, including relief from infestation. Administration of topical ‘IVOMEC’ (500μg/kg) in a clinical study was also found to be very effective (Paradis *et al*, 1997). Paradis *et al* (1997) found a substantial improvement in clinical presentation by day 15 with evidence of hair growth and resolution of erythema and crusting at day 30. Pruritus was also markedly decreased by 7 to 10 days after treatment.

Naturally infested dogs living in heavily contaminated environments have also shown clinical improvement after treatment (Scheidt *et al*, 1984). Scheidt *et al* (1984) recovered mites from one skin scraping (1/20 dogs) and found a decrease in the overall degree of pruritus by two weeks after a single treatment. A second treatment was given to ensure complete recovery (Scheidt *et al*, 1984). No other studies of the effect of treatment with ivermectin in a field situation have been conducted.
In humans, ivermectin has also been successful in treating scabies (Marty, Gari-Toussaint, Le Fichoux and Gaxotte, 1994), including patients with HIV (delGiudice, Carles Couppie, Bernard, Lacour, Marty, Pradinaud, Ortonne, Dellamonica and LeFichoux, 1996; Meinking, Taplin, Hermida, Pardo, Kerdel, 1995). Single oral doses at 200μg/kg have resulted in resolution of pruritus in two days and complete healing within one week (Meinking et al, 1995; del Giudice et al, 1996). In some patients with thick skin crusting, repeat treatments have been necessary, possibly due to the inability of the ivermectin to penetrate through to the keratotic crusts (Meinking et al, 1995). In dogs with severe and crusted scabies, this is also a potential problem, although all dogs with crusted scabies did show improvement in the Kununurra trial. Another problem includes reinfection, which occurred in two, three and nine months in 5 people with thick crusting after one treatment (Meinking et al, 1995). The authors noted that complete resolution of scabies in some cases may take one month and that in these types of cases, a second treatment may be necessary because “ivermectin may have no residual activity against scabies two months after a single dose” (Meinking et al, 1995). Despite the pharmacokinetics of the drug, Meinking and associates (1995) did not mention environmental contamination as a possible source of reinfection.

Since the dogs that still had alopecia 3 months after treatment in the present study (7/69) were dogs that were severely affected before treatment, and did not have any mites isolated from skin scrapings, it is most likely that these dogs were still recovering from the initial scabies infection. Reinfestation, it seems, did not occur in this situation, despite the dogs remaining in environments that potentially were heavily infested. Arlian et al (1995) noted that all dogs in their survey started to display localised hyperkeratosis on some body parts by 6 weeks after infection. Assuming no resistance to reinfection, the dogs at Kununurra would have been expected to start showing some lesions by week 13 if they became reinfected after treatment.

Protective immunity has been demonstrated in reinfected dogs (Arlian et al, 1996), and this may partly explain why the dogs did not redevelop scabies. Arlian et al (1996) challenged 8 dogs
with _Sarcoptes scabiei_ mites 47 days after ivermectin treatment of a previous infection. Clinical signs of the challenge infection peaked by 24 days and then waned, as the skin scrapings became negative by 64 days (for 7 of the dogs). Likewise, in a previous study, 65% of rabbits infested with _S. scabiei_ var. _canis_ mites exhibited resistance to subsequent parasite challenge (Arlian, Morgan, Vyszenski-Moher and Stemmer, 1994). Although they still became infected, they exhibited significant reductions in mite burden. The exact mechanism for the resistance to challenge infection has not been determined, but oxidative bursts by neutrophils in challenge infection may play a role (Arlian _et al_ 1996).

### 6.4.3.2 Hookworm Prevalence after a Single Treatment of Dogs with 200μg/kg Ivermectin

Based on the assumption that dogs that started to shed eggs after treatment were infected with hookworm, then the error of testing was up to 8.2% (see Table 6.3). This assumption does not consider those dogs that were positive one week after treatment but had no eggs recovered. That is, the number of dogs with hookworm immediately after treatment may have been higher, but were not detected.

Assuming that dogs that did not shed eggs were free of adult hookworms, the ivermectin treatments, at 200μg/Kg subcutaneous, were about 93% effective in removing the intestinal hookworms in the present study. _Ancylostoma_ is among the most susceptible of parasites to the action of avermectins, and a pen trial involving artificial infections has found doses greater than 50μg/kg subcutaneous to be 100% effective (Yazwinski _et al_, 1982). Even at concentrations of 5μg/kg per os, avermectin B2a is fully effective (Blair and Campbell, 1978).

One possibility for the recovery of eggs one week after treatment may be the reactivation of gut-dwelling hypobiotic larvae. Yazwinski _et al_ (1982) and Blair and Campbell (1978) were unable to demonstrate the effectiveness of ivermectin against arrested larvae (L3). Kelly _et al_ (1976) proposed that such inhibited larvae provide a “protected” reservoir of infection, which may reactivate after removal of adults from the intestines. Later studies into the stimuli for
reactivation of larvae found that removal of previously established adults is not an important trigger of resumption of larval development (Schad and Page, 1982). Furthermore, repopulation, reactivation and sexual maturity (patency) is likely to take over one week to occur, as demonstrated by the prepatent period of 12-16 days for puppies receiving transcolostral larvae (L3) from infected bitches (Smith and Hooke, 1975; Bowman, 1992).

Over a 13 week period in the present trial, the number of dogs again shedding hookworm eggs gradually increased. At two weeks, there were an additional two dogs (2/50, 4%) shedding eggs. As ivermectin is equally potent against intestinal hookworm larvae as adults, the eggs were not likely to be due to development of larvae already present in the intestine. These dogs were not suckling and hence did not gain infection through milk. Infection through percutaneous, oral or paratenic host ingestion is also unlikely to result in patent infection so quickly. A possible reason for the dogs shedding eggs is that the treatments were not fully effective at killing the intestinal worms and these dogs' infections were not detected one week after treatment.

After week 3, 9 (18%) extra dogs began shedding eggs up to the time of re-treatment at week 13. It is quite feasible that these dogs acquired their infection from the environment or paratenic hosts, although the latter is less likely (Dunsmore and Shaw, 1990). This suggests that even during the dry season, when larval availability is reduced (see 7.4.2.1.3), infection of dogs is possible.

Carroll and Grove (1985) found hookworm eggs (A. ceylanicum) in the faeces of dogs three weeks after challenge infection and concluded that dogs with chronic hookworm infection are considerably resistant to reinfection for one month after treatment. Similar results have also been found with A. caninum following natural primary infection or after exposure to attenuated, irradiated larvae (Carroll and Grove, 1985). The most likely reason for this is the acquisition of immunity to migrating larvae or adult worms in the gut (Carroll and Grove, 1985).
Three months after treatment there was a similar proportion of dogs in each hookworm intensity score category compared with the proportion of dogs in each score category before treatment. Challenge infections of *A. ceylanicum* in dogs by Carroll and Grove (1985) resulted in lower intensity levels when measured 3 weeks after infection, but the experiment was terminated too soon to determine when pre-treatment values would have been reached. Workers studying the intensity of helminth infections in humans have found that pre treatment intensity levels are generally achieved within a year (Anderson and May, 1982, Anderson and Medley, 1985; Haswell-Elkins, Elkins and Anderson, 1987; Upatham et al, 1992). Bradley et al (1993) however, found that treated communities in Zimbabwe did not have significant rises in intensity or prevalence until 24 months after cessation of treatment. Certainly in the present study, the prevalence was significantly lower 3 months after treatment than before treatment.

Unfortunately there were insufficient numbers in the present study to test for predisposition of initially heavily infected animals to regaining heavy intensity of infection.

### 6.4.4 The Risk Factors for Canine Infection with Scabies and Hookworm

#### 6.4.4.1 Sex and Age

There was no evidence in the present survey that sex or age of the dogs affected the likelihood of infection with either scabies or hookworm.

No consistent associations between scabies and sex or age of the host have been found in other studies, although individual studies of dogs and humans have found differences amongst differing age and sex groups (Pence et al, 1983; Green, 1989).

Several field and laboratory based studies have found male sex (Kirkpatrick, 1988; Hoskins, Malone, Smith and Uhl, 1982; Miller, 1965; Miller, 1971) and older age (Miller, 1965; Behnke, 1990; Prociv et al, 1994; Visco et al, 1977) to be important risk factors for the development of hookworm in dogs. This was not the case with the present preliminary survey, although later
data from the coastal, central and eastern regions did find sex and age affected hookworm status (see 7.4.2.2).

As the parasite control program was based on mass chemotherapy of all age and sex classes, rather than targeted treatments, the potential findings of age and sex as risk factors did not affect the program procedure.

6.4.5 Side Effects of Ivermectin Treatment

Acute toxicosis due to ivermectin treatment was not expected, nor observed, in the present study as the toxic dose is at least 10 times the effective therapeutic dose (200μg/kg) of ivermectin in dogs (Pulliam and Preston, 1989).

The subcutaneous administration of cattle preparation ‘IVOMEC’ in dogs has been well tolerated and safe according to previous studies (Scheidt et al, 1984; Singh and Gill, 1987). In the present study, some dogs did vocalise and flinch during and after treatments with ivermectin, but no local reactions were left at the site of injection. Scheidt et al, (1984) also found similar reactions in a scabies treatment trial, but Thimmappa Rai and Yathira (1988) reported depression and anorexia 24 hours after treatment in a similar trial. No explanations for this were given.

Of concern was the potential for reactions to ivermectin treatment in dogs with circulating heartworm microfilariae, but no reactions either immediately after treatment or during the 5 weeks of sampling after treatment were observed or brought to our attention. Vomiting, excess salivation, blood in faeces, soft stool/diarrhoea and depression have been observed in dogs with circulating D. immitis microfilariae administered ivermectin (Pulliam and Preston, 1989). The shock-like reactions in heartworm infested dogs administered diethylcarbamazine have not occurred in dogs following ivermectin treatment and although deaths of dogs have been recorded following ivermectin treatment, none of these reactions have been confirmed to be
caused by ivermectin (Pulliam and Preston, 1989). A trial by McCall, McTier, Ryan, Gross and Soll (1996) to evaluate the effectiveness of ivermectin in dogs with heartworm infections of three and four month’s duration found no adverse reactions to the heartworm tablets (ivermectin 6μg/kg) nor the infections themselves. As the infections in McCall et al’s (1996) trial were only of 3 and 4 month’s duration, they were within the period before patency (6 months) and hence there were no circulating microfilariae.

Despite the lack of adverse reactions, dogs from the subsequent trials in the present study, were tested for heartworm before treatment to forewarn owners of any possible, but unlikely, reactions.

6.4.6 The Effect of a Single Treatment of Dogs with 200μg/kg Ivermectin on Health Parameters.

6.4.6.1 Hydration Status

Hydration status was calculated (see 5.2.3.2) to ensure that there were no changes in plasma volume to affect the determination of anaemia status. Only 3 dogs (3% of samples) had concomitant increases in Total Serum Protein with a normal albumin to globulin ratio. As the albumin to globulin ratio was normal, this indicates that the hyperproteinaemia was due to an overall decrease in plasma volume (dehydration) rather than increase in either protein fraction (Duncan and Prasse, 1987). All other samples (97%, 96/99 samples) from dogs with hyperproteinaemia had low albumin to globulin ratios, which was consistent with the findings of high serum globulin concentrations (see Table 5.9). These dogs were not clinically dehydrated, but were anaemic.

6.4.6.2 Haemoglobin Levels

Dogs tested 3 months after treatment were found to be 6 times more likely to have normal or above normal haemoglobin concentrations than before the treatment. Likewise, the mean haemoglobin levels of matched samples were statistically higher at the testing 3 months after
treatment. This indicates that the treatment is likely to be responsible for the recovery of previously anaemic dogs.

The finding of an association between haemoglobin levels and scabies prevalence was considered spurious, as haemoglobin was not associated with scabies when the important confounding variables of hookworm status and age were controlled. Lowenstein, Loupal, Baumgartner and Kutzer (1996) did not find any difference in red blood cell parameters after the treatment (and cure) of calves infected with *Sarcoptes scabiei* var. *bovis*. In contrast, other studies have found prolonged and heavy infections with scabies to induce changes in blood cytology and have effects on organs other than the skin in a variety of species (Burgess, 1994), including dogs (Pence *et al.*, 1983).

In rabbits and pigs, a severe anaemia with reduced total haemoglobin, haematocrit and cellular haemoglobin can develop (Burgess, 1994), but in a controlled laboratory study of dogs, this was not evident (Arlian *et al.*, 1995). In their study, Arlian *et al* (1995) found dogs did have significantly lower average haemoglobin and haematocrit concentrations, after 8 weeks of infestation, compared with the controls or the dogs prior to infestation, but the blood parameters were within normal limits. The authors suggested that the gradual decline over time indicated that had the infestation continued there would have been a progression toward anaemia.

Sheahan (1974) explained the anaemia demonstrated in pigs with heavy infestations of scabies as a reduced ability of the animals to absorb iron from food and increased loss of iron in desquamated skin. Arlian *et al’s* (1988) work with rabbits failed to show any significant reductions in iron levels compared with controls. Iron deprivation from an incomplete diet coupled with infection, rather than inability to absorb iron, may contribute considerably to loss of weight gain and anaemia (Burgess, 1994).
Dogs with evidence of hookworm infection in the present study were 3 times more likely to have anaemia than those without. When only adult dogs were considered, though, hookworm infection alone did not significantly affect the anaemia status of the dogs. This indicates that other confounding variables, such as infections with scabies or ticks (although anaemia was not associated with tick infestation in the present study, see Table 6.7), may also be affecting haemoglobin levels in dogs.

Overall, the degree of anaemia and clinical signs associated with hookworm are related to the intensity of infection, age of the dog, nutritional status, iron reserves and the presence of acquired and age resistance (Miller, 1971). In the present study, egg count scores provided a rough estimate of hookworm burden. High scores (over 1) were found to be associated with low haemoglobin levels. In laboratory trials, Miller (1971) found that an infection of 50-75 worms per pound of body weight (of puppies) approximates an LD₅₀, while lower burdens induce severe anaemia. Low burdens, though can lead to increased volume of blood loss per worm, possibly due to more movement and reattachments of adults in the intestines whilst in search of mating partners (Miller, 1968).

Hookworm disease (as opposed to infection) in dogs, is almost always acute and restricted to puppies (Miller, 1968). The most important form of acute ancylostomiasis occurs in nursing puppies of 2-4 weeks as a result of prenatal and neonatal infection (Miller, 1971). Within a few days, almost all of a litter may die of acute haemorrhagic anaemia. Hookworm disease and hookworm burden is also more severe in puppies because of the lack of age and acquired resistance (as discussed). Large burdens of worms coupled with a low plane of nutrition can result in severe anaemia. Further more, iron-lack anaemia occurs more readily in young, rapidly growing animals because they have less storage iron and are usually on a milk diet low in iron (Duncan and Prasse, 1986; Jain, 1993). If puppies are fed well with good iron reserves, blood loss can be compensated for, as was found by Miller (1966). By the 23rd-25th day of heavy infection, puppies were losing blood at a rate equivalent to almost one-quarter of their total
circulating erythrocyte volume. In spite of their loss, their packed cell volumes were increasing, indicating that their erythropoietic activity was compensating for the blood loss (Miller, 1966).

In the present study, puppies and juveniles were approximately 5 times more likely to have low haemoglobin levels than adults. When haemoglobin figures were adjusted for young animals (20-30g/L haemoglobin less than adults (Mills and Sutherland, 1994)), only 4 dogs were classed as anaemic. The dogs most likely to be affected by acute disease, the suckling puppies, were not examined in the present study.

The marked improvement in mean haemoglobin levels, from below normal (114.73 g/L ± SEM 3.85) to normal (144.33g/L ± SEM 3.41), within 13 weeks of a single treatment with ivermectin, indicates that the treatments were effective in improving an important aspect of the dogs' health status; blood oxygen carrying capacity. Anaemia is characteristically associated with reduced exercise tolerance and associated with heartworm infection places a considerable load on the cardiovascular function of the dogs.

6.4.6.3 Mean Corpuscular Haemoglobin Concentration and Mean Corpuscular Volumes

Approximately 13% of dogs in the present survey were found to have microcytic, hypochromic anaemia which is consistent with iron deficiency anaemia. It is possible that the anaemia was due to hookworm infection, especially since 67% of these dogs returned to normal haematological values after treatment with 200µg/kg ivermectin.

Chronic hookworm disease, which commonly results in iron deficiency anaemia in humans, is reported to be rare in dogs (Miller, 1968). Rare, that is, in dogs that are well fed and regularly treated. Chronic disease may be more common than thought in dogs that survive under “abysmal nutritional conditions” in the tropics (Miller, 1968).
6.4.6.4 Globulin

Scabies infection and hyperglobulinaemia were associated in the pretreatment data, but globulin levels decreased after treatment, irrespective of previous scabies infection. This may indicate that other factors that are dependent on treatment (such as inflammation from other parasites) are responsible for hyperglobulinaemia apart from scabies.

Arlian et al (1995) did not find any changes in serum total protein, albumin or globulin during the 8 week infestation and subsequent cure of 15 laboratory dogs. Pence et al (1983), on the other hand found that albumin levels were lower in chronic, heavily Sarcopes infested wild coyotes, possibly due to loss of lower molecular weight components of serum through the skin. Gamma globulin measurements were also above normal level in their study and were correlated with progressing severity of infection. This was attributed to the development of a humoral antibody response. It is possible that this is why the dogs in the present study had high total globulin levels. Inexplicably, though, alpha globulins decreased in the coyote study, which resulted in normal overall globulin levels.

The relatively common hyperglobulinaemia in the present study was not found to be associated with hookworm infection. As electrophoresis was not performed, the various globulin fractions could not be determined. Generally, the increased globulin levels are suspected to be due to inflammation and initiation of an immune response.

Views differ on the relative importance of gastrointestinal parasites in affecting immunoglobulin production. Jarrett and Bazin (1977) found intestinal parasites to act as stimulants to IgE production and are effective, to a lesser extent, in the production of other immunoglobulin classes. Alternatively, Carswell, Hughes, Palmer, Higginson, Harland and Meakins (1981) failed to demonstrate any association between the presence of gut parasite eggs in the faeces and elevated serum IgE (apart from A. lumbricoides) in children from Tanzania.
The other globulin classes of that study were in accordance with normal developed world standards, irrespective of parasitism.

6.4.6.5 Albumin

Except for one dog, all of the sampled animals had serum albumin levels within the normal limits, irrespective of scabies or hookworm infection or age. Studies in humans have shown that serum albumin concentration declines during hookworm disease and that the overall amounts lost can not be accounted for on the basis of blood loss alone (Crompton and Stephenson, 1990). Furthermore, experimental studies in dogs suggest that a protein losing enteropathy can be a feature of hookworm disease in addition to frank blood loss into the lumen (Miller, 1971). Nutritional intake needs to be increased or maintained at an adequate level to compensate for the strain on the dog’s protein metabolism and to restore serum albumin levels. Since anaemia was present, but hypoalbuminaemia was not a feature, this may indicate that there was sufficient protein available to compensate for loss of plasma proteins, but not enough iron to revive haemoglobin levels.

6.4.6.6 The Effect of a Single Treatment with 200µg/kg ivermectin on Other Parasites

Ticks (Rhipicephalus sanguineus)

The prevalence of infestation with ticks in dogs in the present study was reduced one week after treatment, but returned to pretreatment levels by week 2. It is likely that ivermectin was able to kill the preexisting ticks on dogs, but unable to prevent reinestation from environmental life cycle stages by two weeks after treatment. As half-life of ivermectin in dogs is about 1.8 days (McKellar and Benchaoui, 1996), there would be a low residual concentration of ivermectin in the dogs’ blood by 14 days after treatment with 200µg/kg ‘IVOMEC’ (even though ‘IVOMEC’ sustains a longer half-life than ‘pure’ ivermectin (Fink and Porras, 1989)). Dogs also do not become immune to Rhipicephalus sanguineus (Kettle, 1984).
'IVOMEC' is very effective in the control of *Boophilus* spp. for up tp 20 days post treatment in cattle (McKellar and Benchaoui, 1996). *Boophilus* is a one-host tick which parasitises a single host for its entire life span. *Rhipicephalus sanguineus*, in contrast, is a three-host tick, which utilises different hosts between moulting stages and is thus harder to control because of the number of stages that may be in the environment when the animals are being treated. Treatment of dogs with an acaricide and use of environmental insecticides or steam cleaning of heavily infested areas are often recommended to control *Rhipicephalus* in dogs (Dunsmore and Shaw, 1990).

### 6.5 Conclusion

The sampling and treatment methods used during the preliminary testing at Kununurra were found to be effective and were used for the remainder of the present study in the other Kimberley communities. Dogs from the other communities were also affected by a wide variety of parasites, with hookworm, heartworm, scabies and ticks being the most common (see 5.3.1).

A single treatment with 200μg/kg ivermectin was effective in reducing the prevalence of hookworm and scabies when assessed on a weekly basis for 5 weeks. Ivermectin was also effective in reducing the prevalence of infestation with ticks for one week after treatment.

The result of reducing the prevalence (and intensity) of parasitic disease was an improvement in blood parameters. Prior to treatment, 56% of dogs were anaemic, but three months after a single treatment, only 18% of dogs had below normal haemoglobin levels. Scabies infestation was associated with hyperglobulinaemia and after a single treatment, dogs were 8 times less likely to have hyperglobulinaemia.

Ivermectin treatment did not result in any noticeable side effects and hence was used for the long-term parasite control program.
Chapter 7

EFFECTIVENESS OF IVERMECTIN IN CONTROLLING PARASITES IN DOGS IN KIMBERLEY COMMUNITIES

7.1 Introduction

Despite the extensive list of publications concerning the epidemiology of hookworm and scabies in human populations before and after treatment with parasiticides, there have been few reports of the effects of mass treatment campaigns for scabies and hookworm in dogs. Likewise, there are few reports on the effects of climatic factors on parasite control programs in dogs, the risk factors for reinfection with parasites and the effect of parasite control programs on health parameters.

The aims of the parasite control program detailed in this thesis were to reduce the disease in dogs caused by these parasites and to reduce environmental contamination with infective stages of zoonotic parasites.

Both of these aims were addressed by using mass chemotherapy of dogs in the communities being studied, without targeting specific groups. In the literature search and analysis of results, specific groups of animals can be seen to be at greater risk of acquiring infection (and possibly disease) and contributing to environmental contamination with infective stages of parasites. Future control campaigns specifically targeting these higher risk groups may help to reduce the reservoir of zoonotic parasites in communities.

This chapter details the effectiveness of the parasite control program in reducing the prevalence of parasites, the dynamics of parasite infection throughout the program, the risk factors for parasite infection and reinfection, the effect of environmental factors on parasite infection and the overall effect of controlling parasites on the health status of dogs.
7.2 Methodology

The methods used for the treatment program are detailed in the general methodology chapter (3.3).

Timing of Treatment with Ivermectin and Sampling

Treatments with ivermectin and proligestone were given every three months. For the ‘mainstream communities’, of the coastal, central and eastern regions, pretreatment faecal samples were collected before the first treatment in June 1992. Post-treatment faecal sampling was then started 6 months after the commencement of treatment and then every three months. The program was started later in Kalumburu and Looma and faecal sampling in these communities was conducted every three months from the outset. General examinations and skin assessments were conducted at every 3 monthly visit from the commencement of the program at all communities. Skin assessments were continued 6 months after the completion of the treatment program (until March 1995) at Looma, Kalumburu and the eastern region.

General Health Indicators for Determining the Effectiveness of Ivermectin Treatments in Dogs

Weights for each adult and non-pregnant dog were compared between visits and recorded only as increased, decreased or remaining the same, irrespective of the amount of weight gained or lost (see 3.3.5).

The weight information was compared with hookworm and scabies infection at the time of the second and subsequent weighings. There were not enough subjects at each visit for analyses to compare individual weight changes with changes in infection status.

A survey to determine anaemia status (packed cell volume) and total plasma protein of dogs was undertaken three months after treatments started (September 1992) for the mainstream
communities of each region. Other blood samples for establishing heartworm status were taken at varying intervals before and after the commencement of the program (see Table 7.37).

**Determination of the Dynamics of Scabies and Hookworm Infection during the Program**

The dynamics of parasitic infection were determined for scabies and hookworm infection in dogs from Kalumburu and Looma. These communities were chosen as representative communities because the sampling and assessment was conducted every three months from the beginning of the program at these communities. The percentage of dogs converting from positive infection status to negative infection status or an improvement in skin score (negative conversion) or *vice versa* (positive conversion) was determined for each sampling period as well as the percentage of dogs that remained without change.

**Climatic Data for Hookworm Prevalence**

The relationship between hookworm prevalence and total (3 monthly) rainfall, and prevalence and temperature data (mean minimum and mean maximum temperatures) were determined using climatic data for the region provided by the Bureau of Meteorology, Perth, Western Australia.

The specific climatic data for correlation with hookworm prevalence at each visit was accumulated over a 3 month period before the date of each sampling. This was done for 3 reasons:

1. At temperatures of 25-30°C, *A. caninum* requires approximately one week in the environment before infectivity (Dunsmore and Shaw, 1990). The prepatent period is about 14-21 days after ingestion of eggs (Behnke, 1990), which adds up to about 4 weeks (one month) for development from egg to egg. To allow two generations of hookworms to develop to produce sufficient eggs, 2 months of climatic data needed to be collated (Miller, 1970).
2. Treatments were given every three months. Climatic data collected before the previous treatment date would not correlate to hookworm prevalence because L4 and L5 stages in the hosts would have been killed by the ivermectin treatments.

3. Ideally, the climatic data should have been collected from one week before treatment (see above) up to 14-21 days before the next treatment to ensure that all stages that were susceptible to climatic factors were accounted for. As there is quite a variation in prepatent period, all three months’ data was collated to ensure that all climatic factors potentially affecting the environmental stages of hookworm were included.

7.3 Results

7.3.1 Scabies

7.3.1.1 Prevalence of Scabies During the Ivermectin Treatment Program

The effect of treatments every 3 months over 28 months was evidenced by the reduction in percentage of dogs with scabietic lesions (Tables 7.1-7.3).

For the coastal and eastern regions, the prevalence remained at pretreatment levels for 3 months after the first treatment (until September 1992) (14.3% [10.4, 18.2] and 43.3% [38.3, 48.3], respectively) then declined, whereas the central region did not have a significant reduction in infection rates until after 6 months (March 1993). The central region had a rebound in the proportion of dogs with scabies 16 months after treatments began (September 1993), whereas the other regions showed a decline in infection rates until the end of the program.

Overall, each region, including Kalumburu and Looma, had statistically lower infection rates at the end of the program compared to the beginning. The coastal region changed from an average of 14.3% of dogs infected to 1.5% [0.7, 2.3], the central communities declined from 16.6% [13.6, 19.6] of dogs infected to 4.3% [2.7, 5.9] and the eastern region fell from 43.3% of dogs
Table 7.1: Prevalence of Scabies in Dogs – Coastal Region

<table>
<thead>
<tr>
<th></th>
<th>Number Examined</th>
<th>Percentage of Dogs Exhibiting Clinical Signs of Scabies in Each Score Category</th>
<th>Mean Prevalence for Months with No Statistical Difference in Prevalence*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Score 1</td>
<td>Score 2</td>
</tr>
<tr>
<td>Mar-92</td>
<td>87</td>
<td>9.2</td>
<td>2.3</td>
</tr>
<tr>
<td>Jun-92</td>
<td>123</td>
<td>12.2</td>
<td>4.1</td>
</tr>
<tr>
<td>Sep-92</td>
<td>104</td>
<td>9.6</td>
<td>1.9</td>
</tr>
<tr>
<td>Dec-92</td>
<td>115</td>
<td>3.5</td>
<td>0</td>
</tr>
<tr>
<td>Mar-93</td>
<td>109</td>
<td>2.8</td>
<td>0</td>
</tr>
<tr>
<td>Jun-93</td>
<td>116</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Sep-93</td>
<td>131</td>
<td>0.8</td>
<td>0</td>
</tr>
<tr>
<td>Dec-93</td>
<td>118</td>
<td>1.7</td>
<td>1.7</td>
</tr>
<tr>
<td>Mar-94</td>
<td>123</td>
<td>0.8</td>
<td>0</td>
</tr>
<tr>
<td>Jun-94</td>
<td>124</td>
<td>0.8</td>
<td>0</td>
</tr>
<tr>
<td>Sep-94</td>
<td>109</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

*Chi Squared Test, 95% confidence intervals in brackets.
Table 7.2: Prevalence of Scabies in Dogs – Central Region

<table>
<thead>
<tr>
<th></th>
<th>Number Examined</th>
<th>Percentage of Dogs Exhibiting Clinical Signs of Scabies in Each Score Category</th>
<th>Mean Prevalence for Months with No Statistical Difference in Prevalence*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Score 1</td>
<td>Score 2</td>
</tr>
<tr>
<td>Mar-92</td>
<td>126</td>
<td>7.9</td>
<td>2.3</td>
</tr>
<tr>
<td>Jun-92</td>
<td>154</td>
<td>8.4</td>
<td>4.5</td>
</tr>
<tr>
<td>Sep-92</td>
<td>172</td>
<td>12.2</td>
<td>3.5</td>
</tr>
<tr>
<td>Dec-92</td>
<td>142</td>
<td>15.5</td>
<td>2.1</td>
</tr>
<tr>
<td>Mar-93</td>
<td>115</td>
<td>1.7</td>
<td>0.9</td>
</tr>
<tr>
<td>Jun-93</td>
<td>133</td>
<td>4.5</td>
<td>1.5</td>
</tr>
<tr>
<td>Sep-93</td>
<td>163</td>
<td>10.4</td>
<td>2.5</td>
</tr>
<tr>
<td>Dec-93</td>
<td>172</td>
<td>1.2</td>
<td>0.6</td>
</tr>
<tr>
<td>Mar-94</td>
<td>157</td>
<td>2.5</td>
<td>1.3</td>
</tr>
<tr>
<td>Jun-94</td>
<td>142</td>
<td>4.2</td>
<td>0.7</td>
</tr>
<tr>
<td>Sep-94</td>
<td>144</td>
<td>5.6</td>
<td>1.4</td>
</tr>
</tbody>
</table>

* Chi Squared Test, 95% Confidence Intervals in brackets.
<table>
<thead>
<tr>
<th>Number Examined</th>
<th>Percentage of Dogs Exhibiting Clinical Signs of Scabies in Each Score Category</th>
<th>Mean Prevalence for Months with No Statistical Difference in Prevalence*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Score 1</td>
<td>Score 2</td>
</tr>
<tr>
<td>Mar-92</td>
<td>106</td>
<td>15.1</td>
</tr>
<tr>
<td>Jun-92</td>
<td>127</td>
<td>15.1</td>
</tr>
<tr>
<td>Sep-92</td>
<td>139</td>
<td>18.7</td>
</tr>
<tr>
<td>Dec-92</td>
<td>129</td>
<td>21.7</td>
</tr>
<tr>
<td></td>
<td>26.4% [18.8, 34.0]</td>
<td></td>
</tr>
<tr>
<td>Mar-93</td>
<td>117</td>
<td>6.8</td>
</tr>
<tr>
<td>Jun-93</td>
<td>109</td>
<td>8.3</td>
</tr>
<tr>
<td>Sep-93</td>
<td>129</td>
<td>5.4</td>
</tr>
<tr>
<td>Dec-93</td>
<td>140</td>
<td>5</td>
</tr>
<tr>
<td>Mar-94</td>
<td>146</td>
<td>4.8</td>
</tr>
<tr>
<td>Jun-94</td>
<td>129</td>
<td>7</td>
</tr>
<tr>
<td>Sep-94</td>
<td>132</td>
<td>7.6</td>
</tr>
<tr>
<td>Mar-95</td>
<td>121</td>
<td>8.4</td>
</tr>
<tr>
<td></td>
<td>9.0% [7.2, 10.8]</td>
<td></td>
</tr>
</tbody>
</table>

* Chi Squared Test, 95% Confidence Intervals in brackets.
infected to 9.0% [7.2, 10.8]. The greatest reduction was at the coastal region with a 90% decline in prevalence, followed by the eastern region (80%) and the central communities (74%) (see Figures 7.1-7.3, 7.6-7.9).

Figure 7.1: Scabies Infection Rates in Dogs
Coastal Region

Figure 7.2: Scabies Infection Rates in Dogs
Central Region
The scabies infection rates in dogs at Kalumburu decreased 78% (from 27.4% to 6%) within 3 months of the initial treatment (Figure 7.4).

The overall prevalence at Looma was slower to fall, but the initial infection rate was never regained throughout the program (Figure 7.5).
Figure 7.5: Scabies Infection Rates in Dogs
Looma

Although the prevalence of clinically affected dogs declined, occasionally dogs of score 2 (25-50% alopecic) were encountered after the initial reduction. Multiple skin scrapings of 19 dogs with refractory alopecia of scores 2, 3 and 4 (from all regions and communities) in March 1994 were negative for *Sarcoptes scabiei* mites, eggs or nymphs. These dogs were considered to be affected by other conditions rather than scabies. Four of these cases were exhibiting generalised demodecosis rather than sarcoptic mange, as judged by the widespread distribution of lesions with secondary infections and recovery of large numbers of *Demodex canis* mites from the skin scrapings. Of the dogs from all the regions and communities that were examined every three months for over one year (n=420), only six were showing generalised alopecia (score 3 or 4) with secondary skin infection for the entire time. Two of these were the dogs with demodecosis, whereas a definitive diagnosis was not established for the others. None of the dogs with severe generalised dermatitis (score 2, 3 or 4) progressed in their clinical signs (i.e. increased in score).

There was no statistical difference in the prevalence of scabies 6 months after the completion of treatments compared to the recordings at the last treatment in September 1994 at Looma,
Figure 7.6: Resolution of Score 2 Scabies after One Ivermectin Treatment (Three Months)
Figure 7.7: Resolution of Score 2 Scabies after Three Ivermectin Treatments
(Nine Months)
Figure 7.8: Resolution of Score 3 Scabies after Two Ivermectin Treatments (Six Months)
Figure 7.9: Resolution of Score 4 Scabies after Three Ivermectin Treatments (Nine Months)
Kalumburu and the eastern region (Figures 7.3, 7.4 and 7.5). Scabies prevalence was not recorded in March 1995 for the other regions.

7.3.1.1.1 The Prevalence of Scabies in Treated and Non-treated Dogs

There was no statistical difference between the prevalence of scabies in treated dogs compared with non-treated dogs for each visit for every region (see Tables 7.4-7.6). Treatment not only resulted in a reduction in scabies prevalence in treated dogs, but also their untreated counterparts.

The infection rates in new dogs, that had never been treated, in the central and eastern regions (where there were adequate numbers of dogs for analyses), were similar to the rates reported in dogs already registered in the program (Tables 7.7-7.8). Registered dogs had received at least one treatment during the program.

7.3.1.1.2 Scabies Infection Dynamics at Kalumburu and Looma

The prevalence data indicate that the percentage of dogs that underwent an improvement in skin condition score (negative conversion) declined as the number of dogs that remained without change increased (Figures 7.10 and 7.11). At Looma in September 1993, a rise in the number of dogs that had become clinically affected with scabies in the previous 3 months (positive conversion) was mirrored by an increase in those that improved in their condition by December 1993.
Table 7.4: Prevalence of Scabies in Treated and Non-Treated Dogs – Coastal Region

<table>
<thead>
<tr>
<th></th>
<th>Treated 3 Months Prior to Examination</th>
<th>Not Treated</th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number Examined</td>
<td>Percentage with Scabies</td>
<td>Number Examined</td>
<td>Percentage with Scabies</td>
<td>Odds Ratio</td>
<td>95% Confidence Intervals</td>
</tr>
<tr>
<td>Jun-92</td>
<td></td>
<td></td>
<td>87</td>
<td>13.8</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sep-92</td>
<td>69</td>
<td>11.6</td>
<td>28</td>
<td>14.3</td>
<td>0.8</td>
<td>0.2, 2.9</td>
</tr>
<tr>
<td>Dec-92</td>
<td>66</td>
<td>1.5</td>
<td>31</td>
<td>9.7</td>
<td>0.1</td>
<td>0.01, 1.4</td>
</tr>
<tr>
<td>Mar-93</td>
<td>51</td>
<td>3.9</td>
<td>53</td>
<td>1.9</td>
<td>2.1</td>
<td>0.2, 24.2</td>
</tr>
<tr>
<td>Jun-93</td>
<td>56</td>
<td>0</td>
<td>55</td>
<td>0</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Sep-93</td>
<td>65</td>
<td>1.5</td>
<td>64</td>
<td>0</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Dec-93</td>
<td>77</td>
<td>0</td>
<td>36</td>
<td>11.1</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Mar-94</td>
<td>63</td>
<td>1.6</td>
<td>56</td>
<td>0</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Jun-94</td>
<td>76</td>
<td>1.3</td>
<td>48</td>
<td>0</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Sep-94</td>
<td>65</td>
<td>0</td>
<td>43</td>
<td>0</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Treated 3 Months Prior to Examination</td>
<td>Not Treated</td>
<td>Treated and Positive</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>----------------</td>
<td>--------------------------------------</td>
<td>-------------</td>
<td>----------------------</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Number Examined</td>
<td>Percentage with Scabies</td>
<td>Number Examined</td>
<td>Percentage with Scabies</td>
<td>Odds Ratio</td>
<td>95% Confidence Intervals</td>
</tr>
<tr>
<td>Jun-92</td>
<td>126</td>
<td>18.3</td>
<td>68</td>
<td>14.7</td>
<td>1.7</td>
<td>0.7, 3.9</td>
</tr>
<tr>
<td>Sep-92</td>
<td>97</td>
<td>22.7</td>
<td>68</td>
<td>14.7</td>
<td>1.7</td>
<td>0.7, 3.9</td>
</tr>
<tr>
<td>Dec-92</td>
<td>103</td>
<td>20.4</td>
<td>35</td>
<td>11.4</td>
<td>2.0</td>
<td>0.6, 6.2</td>
</tr>
<tr>
<td>Mar-93</td>
<td>71</td>
<td>2.8</td>
<td>40</td>
<td>2.5</td>
<td>1.1</td>
<td>0.1, 12.9</td>
</tr>
<tr>
<td>Jun-93</td>
<td>67</td>
<td>4.5</td>
<td>58</td>
<td>8.6</td>
<td>0.5</td>
<td>0.1, 2.2</td>
</tr>
<tr>
<td>Sep-93</td>
<td>82</td>
<td>14.6</td>
<td>80</td>
<td>11.2</td>
<td>1.4</td>
<td>0.6, 3.6</td>
</tr>
<tr>
<td>Dec-93</td>
<td>119</td>
<td>0.8</td>
<td>43</td>
<td>4.7</td>
<td>0.2</td>
<td>0.02, 2.0</td>
</tr>
<tr>
<td>Mar-94</td>
<td>97</td>
<td>5.2</td>
<td>52</td>
<td>1.9</td>
<td>2.8</td>
<td>0.3, 24.4</td>
</tr>
<tr>
<td>Jun-94</td>
<td>93</td>
<td>2.2</td>
<td>49</td>
<td>10.2</td>
<td>0.2</td>
<td>0.04, 1.0</td>
</tr>
<tr>
<td>Sep-94</td>
<td>83</td>
<td>2.4</td>
<td>60</td>
<td>5</td>
<td>0.5</td>
<td>0.08, 2.9</td>
</tr>
</tbody>
</table>
Table 7.6: Prevalence of Scabies in Treated and Non-Treated Dogs – Eastern Region

<table>
<thead>
<tr>
<th>Month</th>
<th>Number Examined</th>
<th>Percentage with Scabies</th>
<th>Number Examined</th>
<th>Percentage with Scabies</th>
<th>Odds Ratio</th>
<th>95% Confidence Intervals</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jun-92</td>
<td>106</td>
<td>46.2</td>
<td>60</td>
<td>40</td>
<td>0.8</td>
<td>0.4, 1.6</td>
</tr>
<tr>
<td>Sep-92</td>
<td>80</td>
<td>35</td>
<td>60</td>
<td>40</td>
<td>0.7</td>
<td>0.3, 1.7</td>
</tr>
<tr>
<td>Dec-92</td>
<td>94</td>
<td>24.5</td>
<td>35</td>
<td>31.4</td>
<td>0.8</td>
<td>0.2, 2.9</td>
</tr>
<tr>
<td>Mar-93</td>
<td>87</td>
<td>11.5</td>
<td>30</td>
<td>13.3</td>
<td>0.8</td>
<td>0.2, 2.9</td>
</tr>
<tr>
<td>Jun-93</td>
<td>74</td>
<td>9.5</td>
<td>33</td>
<td>6.1</td>
<td>0.8</td>
<td>0.3, 8.2</td>
</tr>
<tr>
<td>Sep-93</td>
<td>81</td>
<td>7.4</td>
<td>59</td>
<td>3.4</td>
<td>1.6</td>
<td>0.4, 11.7</td>
</tr>
<tr>
<td>Dec-93</td>
<td>87</td>
<td>9.2</td>
<td>55</td>
<td>7.3</td>
<td>0.3</td>
<td>0.4, 4.5</td>
</tr>
<tr>
<td>Mar-94</td>
<td>87</td>
<td>8</td>
<td>57</td>
<td>3.5</td>
<td>2.4</td>
<td>0.5, 12.0</td>
</tr>
<tr>
<td>Jun-94</td>
<td>93</td>
<td>12.9</td>
<td>36</td>
<td>5.6</td>
<td>2.5</td>
<td>0.5, 11.9</td>
</tr>
<tr>
<td>Sep-94</td>
<td>79</td>
<td>13.9</td>
<td>53</td>
<td>3.8</td>
<td>0.9</td>
<td>0.9, 19.4</td>
</tr>
</tbody>
</table>
### Table 7.7: Prevalence of Scabies in New and Previously Seen Dogs – Central Region

<table>
<thead>
<tr>
<th></th>
<th>Previously Seen</th>
<th></th>
<th>New Dogs</th>
<th></th>
<th>Previously Seen and Positive</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number Examined</td>
<td>Percentage with Scabies</td>
<td>Number Examined</td>
<td>Percentage with Scabies</td>
<td>Odds Ratio</td>
<td>95% Confidence Intervals</td>
</tr>
<tr>
<td>Jun-92</td>
<td>102</td>
<td>11.8%</td>
<td>52</td>
<td>26.9%</td>
<td>2.8</td>
<td>1.2, 6.5*</td>
</tr>
<tr>
<td>Sep-92</td>
<td>112</td>
<td>21.4%</td>
<td>60</td>
<td>13.3%</td>
<td>0.6</td>
<td>0.2, 1.4</td>
</tr>
<tr>
<td>Dec-92</td>
<td>111</td>
<td>21.6%</td>
<td>31</td>
<td>3.2%</td>
<td>0.1</td>
<td>0.02, 0.9*</td>
</tr>
<tr>
<td>Mar-93</td>
<td>87</td>
<td>2.3%</td>
<td>28</td>
<td>3.6%</td>
<td>1.6</td>
<td>0.1, 18.0</td>
</tr>
<tr>
<td>Jun-93</td>
<td>96</td>
<td>5.2%</td>
<td>37</td>
<td>8.1%</td>
<td>1.6</td>
<td>0.4, 7.1</td>
</tr>
<tr>
<td>Sep-93</td>
<td>106</td>
<td>12.3%</td>
<td>57</td>
<td>14.0%</td>
<td>1.2</td>
<td>0.4, 3.0</td>
</tr>
<tr>
<td>Dec-93</td>
<td>149</td>
<td>1.3%</td>
<td>23</td>
<td>4.3%</td>
<td>1.2</td>
<td>0.3, 38.4</td>
</tr>
<tr>
<td>Mar-94</td>
<td>130</td>
<td>3.8%</td>
<td>27</td>
<td>3.7%</td>
<td>1.0</td>
<td>0.1, 8.6</td>
</tr>
<tr>
<td>Jun-94</td>
<td>111</td>
<td>1.8%</td>
<td>31</td>
<td>16.1%</td>
<td>0.8</td>
<td>1.9, 57.1</td>
</tr>
<tr>
<td>Sep-94</td>
<td>111</td>
<td>6.3%</td>
<td>33</td>
<td>9.1%</td>
<td>1.5</td>
<td>0.4, 6.1</td>
</tr>
</tbody>
</table>

* Statistically significant
<table>
<thead>
<tr>
<th></th>
<th>Previously Seen</th>
<th>New Dogs</th>
<th>Previously Seen and Positive</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number Examined</td>
<td>Percentage with Scabies</td>
<td>Number Examined</td>
</tr>
<tr>
<td>Jun-92</td>
<td>59</td>
<td>16.9</td>
<td>68</td>
</tr>
<tr>
<td>Sep-92</td>
<td>98</td>
<td>37.8</td>
<td>41</td>
</tr>
<tr>
<td>Dec-92</td>
<td>103</td>
<td>27.2</td>
<td>26</td>
</tr>
<tr>
<td>Mar-93</td>
<td>99</td>
<td>13.1</td>
<td>18</td>
</tr>
<tr>
<td>Jun-93</td>
<td>90</td>
<td>8.9</td>
<td>19</td>
</tr>
<tr>
<td>Sep-93</td>
<td>96</td>
<td>7.3</td>
<td>33</td>
</tr>
<tr>
<td>Dec-93</td>
<td>111</td>
<td>9.9</td>
<td>29</td>
</tr>
<tr>
<td>Mar-94</td>
<td>115</td>
<td>7.8</td>
<td>31</td>
</tr>
<tr>
<td>Jun-94</td>
<td>110</td>
<td>12.7</td>
<td>19</td>
</tr>
<tr>
<td>Sep-94</td>
<td>106</td>
<td>12.3</td>
<td>26</td>
</tr>
</tbody>
</table>
7.3.1.2 Risk Factors for Scabies Infection

Data relating to risk factors for scabies infection were examined for the pretreatment survey as well as each of the subsequent three monthly examinations during the program.
7.3.1.2.1 Age of Host

No statistical evidence of an age predilection for puppies (less than 6 months old) to be infected with scabies was found at any of the 3 regions for any of the examination dates (see Appendix H). Dogs less than 1 year of age, though, were found to be 2.6 [1.1, 6.5] and 7.7 [1.4, 50] times more likely to have scabies than adults in the central region at two visits. Analysis of data from other visits to all regions showed no statistical difference in age predilection for scabies.

7.3.1.2.2 Sex of Host

Only on two occasions sex predilection for scabies was demonstrated. Male dogs appeared to be more susceptible to scabies than females. Males were 2.9 [1.0, 9.0] and 3.4 [1.2, 10.0] times more likely to have scabies than females (Table 7.9).

7.3.1.2.3 The Effect of Number of Dogs per Household on Scabies Infection Rates

The average number of dogs per household at each region were; 0.96 at the coastal region, 1.58 at the central region and 4.06 at the eastern region (see 4.4.1.1; Table 4.3). From this data, the average number of dogs per household that was considered to represent ‘crowded’ conditions in the present study was more than 4 dogs.

7.3.1.2.3.1 Association between Numbers of Dogs per Household and Scabies Infection in Dogs

There was a higher prevalence of scabies in dogs from households with more than four dogs (multiple-dog households) than those from households with 4 or less dogs. For the central and eastern communities, dogs from households with more than 4 dogs were 1.7 [1.2, 2.4] and 1.6 [1.2, 2.2] times more likely to have scabies than dogs from households with 4 dogs or less (Table 7.10). Likewise, at a significance level of P=0.1, dogs from multiple-dog-households at the coastal region were 2.2 times more likely to have scabies than those from less densely populated households.
### Table 7.9: Sex Related Prevalence of Scabies in Dogs*

<table>
<thead>
<tr>
<th></th>
<th>Dogs with Scabies</th>
<th>Dogs without Scabies</th>
<th>Odds Ratio**</th>
<th>95% Confidence Intervals</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of Males</td>
<td>Number of Females</td>
<td>Number of Males</td>
<td>Number of Females</td>
<td></td>
</tr>
<tr>
<td>Mar-92*</td>
<td>48</td>
<td>32</td>
<td>122</td>
<td>80</td>
</tr>
<tr>
<td>Jun-92</td>
<td>56</td>
<td>34</td>
<td>162</td>
<td>92</td>
</tr>
<tr>
<td>Sep-92</td>
<td>59</td>
<td>39</td>
<td>168</td>
<td>106</td>
</tr>
<tr>
<td>Dec-92</td>
<td>32</td>
<td>29</td>
<td>170</td>
<td>117</td>
</tr>
<tr>
<td>Mar-93</td>
<td>16</td>
<td>4</td>
<td>165</td>
<td>121</td>
</tr>
<tr>
<td>Jun-93</td>
<td>12</td>
<td>5</td>
<td>161</td>
<td>231</td>
</tr>
<tr>
<td>Sep-93</td>
<td>17</td>
<td>11</td>
<td>205</td>
<td>143</td>
</tr>
<tr>
<td>Dec-93</td>
<td>12</td>
<td>12</td>
<td>217</td>
<td>158</td>
</tr>
<tr>
<td>Mar-94</td>
<td>10</td>
<td>7</td>
<td>218</td>
<td>152</td>
</tr>
<tr>
<td>Jun-94</td>
<td>12</td>
<td>10</td>
<td>205</td>
<td>129</td>
</tr>
<tr>
<td>Sep-94</td>
<td>16</td>
<td>6</td>
<td>194</td>
<td>145</td>
</tr>
</tbody>
</table>

* No statistical difference between regions for each visit [P>0.05]

**Odds ratio for infection with scabies in males compared with females

* Statistically significant difference
Table 7.10: Scabies Infection and Household Density of Dogs - Coastal, Central and Eastern Regions

<table>
<thead>
<tr>
<th>Coastal Region</th>
<th>Number with Scabies</th>
<th>Total Number of Examinations</th>
<th>Percentage with Scabies</th>
<th>Odds Ratio</th>
<th>95% Confidence Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt; 4 Dogs per house</td>
<td>8</td>
<td>83</td>
<td>9.6</td>
<td>2.2</td>
<td>0.9, 5.2</td>
</tr>
<tr>
<td>≤ 4 Dogs per house</td>
<td>18</td>
<td>383</td>
<td>4.7</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 Dog per house</td>
<td>7</td>
<td>91</td>
<td>7.7</td>
<td>1.6</td>
<td>0.6, 3.8</td>
</tr>
<tr>
<td>&gt; 1 Dog per house</td>
<td>19</td>
<td>375</td>
<td>5.1</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Central Region</th>
<th>Number with Scabies</th>
<th>Total Number of Examinations</th>
<th>Percentage with Scabies</th>
<th>Odds Ratio</th>
<th>95% Confidence Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt; 4 Dogs per house</td>
<td>114</td>
<td>931</td>
<td>12.2</td>
<td>1.7</td>
<td>1.2, 2.4*</td>
</tr>
<tr>
<td>≤ 4 Dogs per house</td>
<td>53</td>
<td>713</td>
<td>7.4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 Dog per house</td>
<td>10</td>
<td>100</td>
<td>10</td>
<td>1.0</td>
<td>0.5, 1.9</td>
</tr>
<tr>
<td>&gt; 1 Dog per house</td>
<td>157</td>
<td>1554</td>
<td>10.2</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Eastern Region</th>
<th>Number with Scabies</th>
<th>Total Number of Examinations</th>
<th>Percentage with Scabies</th>
<th>Odds Ratio</th>
<th>95% Confidence Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt; 4 Dogs per house</td>
<td>221</td>
<td>1074</td>
<td>20.6</td>
<td>1.6</td>
<td>1.2, 2.2*</td>
</tr>
<tr>
<td>≤ 4 Dogs per house</td>
<td>65</td>
<td>466</td>
<td>13.9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 Dog per house</td>
<td>4</td>
<td>19</td>
<td>21.1</td>
<td>1.17</td>
<td>0.4, 3.6</td>
</tr>
<tr>
<td>&gt; 1 Dog per house</td>
<td>282</td>
<td>1521</td>
<td>18.5</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Data pooled from pre-and post-treatment examinations
* Statistically significant
There was no statistical difference in scabies infection rates in dogs from single-dog households compared with dogs that lived with other dogs (>1 dog per household).

7.3.1.2.3.2 Pre-treatment Correlation between Household Dog Population and Prevalence of Scabies in Dogs

There was a strong correlation between the number of dogs per household and the proportion of households with infected dogs \((r=0.84, P=0.001)\) (Table 7.11). There was a weaker correlation between the number of dogs infected per household and the number of dogs per household \((r=0.69, P=0.02)\).

7.3.1.2.3.3 Pre-treatment Distribution of Dogs with Scabies throughout Kalumburu, Looma and Warmun

Dot map distributions of dogs with scabies before treatment showed that dogs with scabies tended to be clustered at households with more than one dog (Figures 7.12 – 7.14). This was especially evident at Kalumburu where 84% (27/32) of dogs with scabies were at one household.

At Looma (see Figure 7.13), 86% of households with more than one dog had scabies and dogs with scabies were 3 \([1.0, 9.8]\) times more likely to come from households with more than one dog. Only 34% (10/29 households) \([16.8,51.2]\) of households with dogs did not have mange dogs.

Dog-owning-households at Warmun (see Figure 7.14) rarely had only one dog per household (3 out of 21 dog-owning-households), so scabies infection rates in dogs from single-dog households and others could not be compared. Overall 76.2% (16 out of 21) \([58.7, 94.3]\) of dog-owning-households had dogs with scabies.
Table 7.11: Percentage of Households and Dogs Affected by Scabies According to Household Size
– Mainstream Communities Pretreatment

<table>
<thead>
<tr>
<th>Number of Dogs per Household (Household Size)</th>
<th>Number Surveyed</th>
<th>Percentage of Households with Infected Dogs</th>
<th>Number of Dogs</th>
<th>Percentage of Dogs Affected</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>42</td>
<td>26.2</td>
<td>42</td>
<td>26.2</td>
</tr>
<tr>
<td>2</td>
<td>23</td>
<td>8.7</td>
<td>46</td>
<td>4.3</td>
</tr>
<tr>
<td>3</td>
<td>10</td>
<td>40</td>
<td>30</td>
<td>30</td>
</tr>
<tr>
<td>4</td>
<td>10</td>
<td>80</td>
<td>40</td>
<td>37.5</td>
</tr>
<tr>
<td>5</td>
<td>6</td>
<td>50</td>
<td>30</td>
<td>13.3</td>
</tr>
<tr>
<td>6</td>
<td>3</td>
<td>100</td>
<td>18</td>
<td>44.4</td>
</tr>
<tr>
<td>7</td>
<td>10</td>
<td>100</td>
<td>21</td>
<td>23.8</td>
</tr>
<tr>
<td>8</td>
<td>1</td>
<td>100</td>
<td>8</td>
<td>75</td>
</tr>
<tr>
<td>9</td>
<td>1</td>
<td>100</td>
<td>9</td>
<td>22.2</td>
</tr>
<tr>
<td>10</td>
<td>1</td>
<td>100</td>
<td>10</td>
<td>50</td>
</tr>
<tr>
<td>12</td>
<td>1</td>
<td>100</td>
<td>12</td>
<td>91.7</td>
</tr>
<tr>
<td>Pearson’s Correlation</td>
<td>0.84</td>
<td></td>
<td>0.69</td>
<td></td>
</tr>
<tr>
<td>P</td>
<td>0.001</td>
<td></td>
<td>0.02</td>
<td></td>
</tr>
</tbody>
</table>
Figure 7.12: Dot Map Household Distribution of Dogs with Scabies - Kalumburu
Figure 7.13: Dot Map Household Distribution of Dogs with Scabies - Looma

Not to scale
Only houses shown

House
Road
Dogs with scabies
Dogs without scabies
Figure 7.14: Dot Map Household Distribution of Dogs with Scabies - Warmun

Not to scale
Only houses shown
- House
- Road
- Dogs with scabies
- Dogs without scabies
7.3.1.3 Effect of Scabies Control Program on Dog Health Parameters

It was possible to analyse weight data on a community basis in the three largest communities. Only at one community, Looma, was there an association between scabies infection and weight loss (OR 2.4 [1.2, 5.2]). At Warmun and Kalumburu, there was no association found between either weight loss or gain and scabies infection rates (see Table 7.12).

Pooled haematocrit data from all ‘mainstream’ communities, analysed from samples collected in September 1992, showed similar findings to the results of haemoglobin levels of dogs at Kununurra. There was no association between anaemia and scabies infection when hookworm infection was taken into account. Non-controlled data demonstrated an association between low haematocrit values and scabies infection.

7.3.2 Hookworm

7.3.2.1 Prevalence of Hookworm during the Ivermectin Treatment Program

7.3.2.1.1 Prevalence of Hookworm in all Dogs from Each Region

The initial prevalence of *Ancylostoma caninum* in dogs was high for each region of the program (57.8% to 70.6%, average 64.7% [60.9, 73.9]) (Table 7.13). After 3 monthly treatments with 200μg/kg ivermectin, the prevalence reduced across all regions to an average of 22.6% [17.8, 27.4] at the last sampling in September 1994.

A seasonal fluctuation in hookworm prevalence was found at all regions during the treatment program (Figures 7.15 – 7.17). Pre-treatment prevalences were reached during the wet season (March) in 1993 and 1994 at the coastal (average prevalence 60% [51.2, 68.8]) and central regions (average prevalence 47.1% [39.6, 54.6]) (Table 7.13). At the eastern region, pre-treatment prevalence was only regained in March 1993 (average 66.8% [58.9, 74.4]).
Table 7.12: Scabies Infection and Weight Changes*

<table>
<thead>
<tr>
<th></th>
<th>Kalumburu</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Infected with Scabies</td>
<td>%</td>
<td>Odds Ratio</td>
<td>95% Confidence Interval</td>
<td>Infected with Scabies</td>
<td>%</td>
<td>Odds Ratio</td>
<td>95% Confidence Interval</td>
</tr>
<tr>
<td>Weight Increase</td>
<td>0</td>
<td>0</td>
<td>-</td>
<td>13</td>
<td>9.9</td>
<td>0.92</td>
<td>0.4, 2.0</td>
<td>24</td>
</tr>
<tr>
<td>Weight Decrease</td>
<td>2</td>
<td>3.8</td>
<td>0.6</td>
<td>0.1, 3.2</td>
<td>15</td>
<td>18.5</td>
<td>2.43</td>
<td>1.2, 5.2</td>
</tr>
<tr>
<td>No Change</td>
<td>5</td>
<td>6.1</td>
<td></td>
<td>4</td>
<td>5.9</td>
<td>0.69</td>
<td>0.2, 2.1</td>
<td>20</td>
</tr>
</tbody>
</table>

* Data pooled for each visit (P>0.05)
### Table 7.13: Hookworm Prevalence in Dogs

<table>
<thead>
<tr>
<th></th>
<th>Coastal Region</th>
<th></th>
<th>Central Region</th>
<th></th>
<th>Eastern Region</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number Sampled</td>
<td>Percentage Positive</td>
<td>Number Sampled</td>
<td>Percentage Positive</td>
<td>Number Sampled</td>
<td>Percentage Positive</td>
</tr>
<tr>
<td>Jun-92*</td>
<td>50</td>
<td>68.0</td>
<td>83</td>
<td>57.8</td>
<td>68</td>
<td>70.6</td>
</tr>
<tr>
<td>Sep-92</td>
<td>45</td>
<td>20.0</td>
<td>109</td>
<td>39.4</td>
<td>76</td>
<td>40.8</td>
</tr>
<tr>
<td>Mar-93</td>
<td>44</td>
<td>52.3</td>
<td>68</td>
<td>45.6</td>
<td>73</td>
<td>63.0</td>
</tr>
<tr>
<td>Sep-93</td>
<td>85</td>
<td>28.2</td>
<td>118</td>
<td>33.0</td>
<td>82</td>
<td>18.3</td>
</tr>
<tr>
<td>Dec-93</td>
<td>93</td>
<td>24.7</td>
<td>111</td>
<td>30.6</td>
<td>103</td>
<td>40.8</td>
</tr>
<tr>
<td>Mar-94</td>
<td>76</td>
<td>64.5</td>
<td>104</td>
<td>48.1</td>
<td>98</td>
<td>41.8</td>
</tr>
<tr>
<td>Jun-94</td>
<td>79</td>
<td>24.1</td>
<td>110</td>
<td>16.4</td>
<td>91</td>
<td>30.8</td>
</tr>
<tr>
<td>Sep-94*</td>
<td>93</td>
<td>16.1</td>
<td>111</td>
<td>23.4</td>
<td>92</td>
<td>28.3</td>
</tr>
</tbody>
</table>

**Average Prevalence**

<table>
<thead>
<tr>
<th></th>
<th>Coastal Region</th>
<th></th>
<th>Central Region</th>
<th></th>
<th>Eastern Region</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Pretreatment and Wet Season months</td>
<td>61.6% P (J92, M93 and M94) = 0.26</td>
<td>50.5% P (J92, M93 and M94) = 0.26</td>
<td>66.8% P (J92 and M93) = 0.34</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dry Season months</td>
<td>22.6% P (S92, S93, D93, J94 and S94) = 0.37</td>
<td>31.6% P (S92, S93, D93 and S94) = 0.08</td>
<td>36.5% P (S92, D93, M94, J94 and S94) = 0.16</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*No significant difference in prevalence between regions for visit
Figure 7.15: Prevalence of Hookworm in All Dogs
Central Region

Figure 7.16: Prevalence of Hookworm in All Dogs
Central Region
For most of the other months of sampling (dry season), there was no significant difference in prevalence between samplings (P>0.05). At the coastal communities the average prevalence for the post-treatment dry season months was 22.6% [18.5, 26.7]. The average prevalence at the central communities was 31.6% [28.0, 35.2] except in June 1994 when the prevalence was lower (16.4% [9.5, 23.3]). In the east, the average was 36.5% [32.4, 40.6], except for September 1993 (18.3% [9.9, 26.7]).

The hookworm infection rates at Kalumburu, in contrast to other coastal communities, did not show any seasonal variation. The prevalence of hookworm at this community declined from 90.7% [82.0, 99.4] (pre-treatment) to an average of 27.2% [22.8, 31.6] (Figure 7.18).

The seasonal pattern of hookworm infection in dogs from Looma was similar to that of the other communities of the central region (see Figure 7.19). The initial prevalence of hookworm was higher at Looma (72.7% [59.5, 85.9]), but samples were collected in September 1992 rather than June 1992.
7.3.2.1.2 Prevalence of Hookworm in Dogs that were Treated Three Months before Sampling

The prevalence of hookworm in dogs that were treated 3 months prior to sampling was found to be the same or lower than in non-treated dogs for all months at each region.
Coastal Region (Table 7.14)

Samples that were taken during the dry season months from dogs that had not been treated three months prior to sampling showed that there was no significant difference between prevalence for each of the months (average 13.8% [9.3, 18.3], P=0.08) (Figure 7.20). For the two wet season (March) samplings, the average prevalence was greater than the dry season months and was not statistically different to the pre-treatment prevalence of June 1992 (average prevalence 39.8% [31.3, 48.3], P=0.19).

Figure 7.20: Prevalence of Hookworm in Dogs Treated and Not Treated Three Months Prior to Sampling - Coastal Region

The seasonal pattern observed at most coastal communities was not apparent at Kalumburu where there was no statistical difference in prevalence of hookworm in treated dogs for all months (P>0.05), excluding June 1993 (prevalence 31.6% [19.5, 43.7] vs. 15.4% [10.7, 20.1]) (Figure 7.21).
Table 7.14: Prevalence of Hookworm in Treated and Non-Treated Dogs – Coastal Region*

<table>
<thead>
<tr>
<th></th>
<th>Treated 3 Months Prior to Sampling</th>
<th>Not Treated</th>
<th>95% Confidence Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number Sampled</td>
<td>Percentage Infected</td>
<td>Number Sampled</td>
</tr>
<tr>
<td>Jun-92</td>
<td>41</td>
<td>21.2</td>
<td>54</td>
</tr>
<tr>
<td>Sep-92</td>
<td>25</td>
<td>48.0</td>
<td>9</td>
</tr>
<tr>
<td>Mar-93</td>
<td>53</td>
<td>15.1</td>
<td>36</td>
</tr>
<tr>
<td>Dec-93</td>
<td>75</td>
<td>20.0</td>
<td>23</td>
</tr>
<tr>
<td>Mar-94</td>
<td>49</td>
<td>57.1</td>
<td>28</td>
</tr>
<tr>
<td>Jun-94</td>
<td>56</td>
<td>8.9</td>
<td>27</td>
</tr>
<tr>
<td>Sep-94</td>
<td>61</td>
<td>4.9</td>
<td>33</td>
</tr>
</tbody>
</table>

Average Prevalence

- Pretreatment and Wet Season Months: 39.8% (P (J92, M93 and M94) = 0.19) vs. 71.2% (P (J92, M93 and M94) = 0.81)
- Dry Season Months: 13.8% (P (S92, S93, D93, J94 and S94) = 0.10) vs. 43.2% (P (S92, S93, D93, J94 and S94) = 0.93)

*Includes data from dogs that were visitors
*Statistically significant
Central Region (Table 7.15)

As occurred at the coastal region, the prevalence of infection during the wet season months (P=0.88) was significantly higher than the non wet season months (P=0.12) for dogs that had been treated three months prior to sampling (average 44.9% [36.4, 53.4] vs. 22.7% [18.5, 26.9]) (Figure 7.22). Although the prevalence was higher during the wet season, the prevalence was statistically lower than that found during the pretreatment survey (June 1992, 57.8%, P=0.006).

A similar pattern was found in Lomca except that there was no statistical difference between the prevalence of hookworm in treated dogs during the wet season (March 1993, 78.6%) and the pretreatment prevalence (68.2%) (Figure 7.23).
Table 7.15: Prevalence of Hookworm in Treated and Non-Treated Dogs – Central Region*

<table>
<thead>
<tr>
<th></th>
<th>Treated 3 Months Prior to Sampling</th>
<th>Not Treated</th>
<th>Not Treated and Positive</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number Sampled</td>
<td>Percentage Infected</td>
<td>Number Sampled</td>
</tr>
<tr>
<td>Jun-92</td>
<td></td>
<td></td>
<td>83</td>
</tr>
<tr>
<td>Sep-92</td>
<td>65</td>
<td>32.3</td>
<td>39</td>
</tr>
<tr>
<td>Mar-93</td>
<td>52</td>
<td>44.2</td>
<td>23</td>
</tr>
<tr>
<td>Sep-93</td>
<td>71</td>
<td>21.1</td>
<td>52</td>
</tr>
<tr>
<td>Dec-93</td>
<td>85</td>
<td>25.9</td>
<td>39</td>
</tr>
<tr>
<td>Mar-94</td>
<td>79</td>
<td>54.4</td>
<td>27</td>
</tr>
<tr>
<td>Jun-94</td>
<td>94</td>
<td>20.2</td>
<td>33</td>
</tr>
<tr>
<td>Sep-94</td>
<td>76</td>
<td>14.5</td>
<td>42</td>
</tr>
</tbody>
</table>

**Average Prevalence**

<table>
<thead>
<tr>
<th></th>
<th>Wet Season Months</th>
<th>Dry Season Months</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>44.9% P (M93 and M94) = 0.88 P (J92, S92, M93, S93, D93, M94, J94 and S94) = 0.53</td>
<td>48.1% P (J92, S92, M93, S93, D93, M94, J94 and S94) = 0.53</td>
</tr>
</tbody>
</table>

*Includes data from dogs that were visitors
*Statistically significant
Figure 7.22: Prevalence of Hookworm in Dogs Treated and Not Treated Three Months Prior to Sampling - Central Region

![Graph showing the prevalence of hookworm in dogs treated and not treated three months prior to sampling in the Central Region.]

Figure 7.23: Prevalence of Hookworm in Dogs Treated and Not Treated Three Months Prior to Sampling - Looma

![Graph showing the prevalence of hookworm in dogs treated and not treated three months prior to sampling in Looma.]

Eastern Region (Table 7.16)

An average prevalence of 27.6% [23.0, 32.2](P= 0.12) was attained for all dogs previously treated excluding those sampled in March 1993 (Figure 7.24). In March 1993, the hookworm prevalence was significantly higher and there was no difference in prevalence compared with the pretreatment prevalence in June 1992 (average 61% [52.7, 69.3], P=0.28).

Figure 7.24: Prevalence of Hookworm in Dogs Treated and Not Treated Three Months Prior to Sampling - Eastern Region

7.3.2.1.3 Prevalence of Hookworm in Dogs that were Not Treated Three Months before Sampling

The prevalence of hookworm in untreated dogs was always equal to or lower than the pre-treatment prevalence. In addition, seasonal variations in hookworm prevalence were noticed at the coastal and eastern regions, but not at the central region.

Coastal Region (Table 7.14)

The pre-treatment prevalence (June 1992) was regained for both March (wet season) samplings (average 71.2% [62.4, 80.0], P=0.81). For all the other (dry season) months, there was no significant difference in hookworm infection rates (average 43.2% [34.6, 51.8], P=0.93).
<table>
<thead>
<tr>
<th></th>
<th>Treated 3 Months Prior to Sampling</th>
<th>Not Treated</th>
<th>Not Treated and Positive</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number Sampled</td>
<td>Percentage Infected</td>
<td>Number Sampled</td>
</tr>
<tr>
<td>Jun-92</td>
<td></td>
<td></td>
<td>74</td>
</tr>
<tr>
<td>Sep-92</td>
<td>45</td>
<td>31.1</td>
<td>21</td>
</tr>
<tr>
<td>Mar-93</td>
<td>59</td>
<td>23.7</td>
<td>15</td>
</tr>
<tr>
<td>Sep-93</td>
<td>60</td>
<td>18.3</td>
<td>23</td>
</tr>
<tr>
<td>Dec-93</td>
<td>66</td>
<td>33.3</td>
<td>37</td>
</tr>
<tr>
<td>Mar-94</td>
<td>72</td>
<td>37.5</td>
<td>30</td>
</tr>
<tr>
<td>Jun-94</td>
<td>70</td>
<td>24.3</td>
<td>23</td>
</tr>
<tr>
<td>Sep-94</td>
<td>52</td>
<td>21.1</td>
<td>40</td>
</tr>
</tbody>
</table>

Average Prevalence

- Pretreatment and Mar-93:
  - Pretreatment: 61.0%
  - Mar-93: 72.4%
  - \( P(J92 \text{ and } M93) = 0.28 \)
  - \( P(J92 \text{ and } M93) = 0.83 \)

- Other Months:
  - P (S92, S93, D93, M94, J94 and S94) = 27.6%
  - P (S92, D93, M94, J94 and S94) = 48.2%

*Includes data from dogs that were visitors

*Statistically significant
Central Region (Table 7.15)
No seasonal variation was noted at the central region. The prevalence of hookworm in untreated dogs for each sampling did not differ from the pre-treatment prevalence (average 48.1% [42.8, 53.4], P=0.53).

Eastern Region (Table 7.16)
The pretreatment prevalence of hookworm was regained in untreated dogs at the first wet month sampling after the treatment program commenced (March 1993; average 72.4% [63.1, 81.7] P=0.83). For each of the other post-treatment months there was no statistical difference in prevalence of hookworm in untreated dogs (average 48.2% [40.8, 55.6], p=0.17), excluding September 1993 when the prevalence of hookworm was considerably lower (17.4% [1.6, 32.4]).

7.3.2.1.4 Comparison of Hookworm Infection Rates in Dogs that were Treated with those that were Not

Coastal Region (Table 7.14)
There was no difference in prevalence of hookworm between treated and non-treated dogs for the wet season months of March 1993 and March 1994. During the dry season months, the non-treated groups were between 3 to 11 times more likely to be positive than the treated groups.

Central Region (Table 7.15)
There was no difference between the infection rates of hookworm in dogs that were treated and those that weren’t for most sampling periods, excluding the dry season months of September (1993 and 1994) and December (1993), when the non-treated dogs were 3 to 4 times more likely to have hookworm than treated dogs.

Eastern Region (Table 7.16)
Overall, statistical differences between treated and untreated dogs were only found for the dry season sampling months of September 1992, December 1993 and June 1994. For each of these
months, the prevalence of hookworm in non-treated dogs was higher than previously treated dogs (OR 2.3 to 3.4).

7.3.2.1.5 Hookworm Infection Dynamics at Kalumburu and Looma

The percentage of dogs whose hookworm status changed from negative to positive and vice versa (see 7.2) was considered for Kalumburu and Looma where data was collected every three months.

Generally, more dogs were likely lose their infection or remain negative during the dry season months of June and September.

7.3.2.1.5.1 Kalumburu

The majority of negative conversions occurred in June 1993 and June 1994 (dry season) (average 30.1% [22.1, 38.1], P=0.91) (Figure 7.25).

There was no difference in the rates of acquiring infection (positive conversion) for all months (average 6.4% [3.9, 8.9], P=0.13).

Figure 7.25: Hookworm Infection Dynamics in Dogs
Kalumburu
7.3.2.1.5.2 Looma

Similar to Kalumburu, the majority of conversions of dogs from positive hookworm status to negative hookworm status (negative conversion) occurred in June 1993 and June 1994 (dry season) (average percentage 33.4% \([25.2, 41.6], \ p=0.31\) (Figure 7.26), one and two years after the commencement of ivermectin treatments, respectively. For all other months the average percentage of dogs losing their hookworm infection was 15.5% \([11.8, 19.2]\).

![Figure 7.26: Hookworm Infection Dynamics in Dogs Looma](image)

The percentage of dogs changing from negative hookworm state to positive hookworm state (becoming infected) varied between 11.5% and 18.5% (average 14.02% \([9.2, 18.8], \ P=0.06\) for all months excluding December 1992, June 1994 and September 1994, when the average rate of infection was lower (5.0% \([2.0, 8.1], \ P=0.16\)).

7.3.2.2 Risk Factors for Hookworm Infection

Data relating to risk factors for hookworm infection were considered for all samplings (including the pretreatment survey) and all communities.
7.3.2.2.1 Age of Host

Puppies were the most susceptible to infection with *A. caninum*. For the sampling visits where there was a statistically demonstrable age predilection for infection in puppies, puppies were between 4 [1.3, 10.0] and 28 [7.0, 111.2] times more likely to be infected than the other age groups (see Appendix H).

When only the adults were considered, the odds ratios were predictably lower than one; around 0.2 [0.1, 0.7] to 0.4 [0.1, 0.8]. This indicates that puppies and juveniles (all dogs apart from adults) were 3 to 5 times more likely to be infected with hookworm than adults.

7.3.2.2.1.1 Age Dependency in Predisposition to Hookworm Infection after Ivermectin Treatment

The effect of age of the host on the likelihood of reinfection with hookworm after treatment was examined for Looma where data was collected on a three monthly basis.

There was no age dependency in predisposition to reinfection demonstrated for any of the months (*P* > 0.05) (Table 7.17).

7.3.2.2 Sex of Host

Pooled data from the three regions showed a sex predilection for hookworm infection for all sampling periods with males being one and a half times more likely to be shedding hookworm eggs than females [1.27, 1.84] (see Table 7.18).

7.3.2.2.1 Sex Dependency in Predisposition to Hookworm Infection after Ivermectin Treatment

Dogs from Looma that were treated with ivermectin, and assumed to be cleared of infection, did not show any sex-dependency in predisposition to hookworm infection for all months of sampling, excluding December 1992 and 1993 (see Table 7.19). During these months, males were 3 [1.0, 9.2] and 7 [1.7, 31.0] times more likely to acquire infection than females, respectively.
<table>
<thead>
<tr>
<th></th>
<th>Hookworm Positive Dogs</th>
<th>Hookworm Negative Dogs</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number of Positive Dogs</td>
<td>Number of Negative Dogs</td>
</tr>
<tr>
<td>Dec-92</td>
<td>26</td>
<td>30</td>
</tr>
<tr>
<td>Mar-93</td>
<td>33</td>
<td>9</td>
</tr>
<tr>
<td>Jun-93</td>
<td>20</td>
<td>29</td>
</tr>
<tr>
<td>Sep-93</td>
<td>17</td>
<td>27</td>
</tr>
<tr>
<td>Dec-93</td>
<td>29</td>
<td>25</td>
</tr>
<tr>
<td>Mar-94</td>
<td>22</td>
<td>21</td>
</tr>
<tr>
<td>Jun-94</td>
<td>11</td>
<td>41</td>
</tr>
<tr>
<td>Sep-94</td>
<td>6</td>
<td>38</td>
</tr>
</tbody>
</table>
Table 7.18: Sex-Prevalence of Hookworm (Pooled Data, All Regions)

<table>
<thead>
<tr>
<th></th>
<th>Male</th>
<th></th>
<th>Female</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Positive</td>
<td>Negative</td>
<td>Percentage</td>
<td>Positive</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Positive</td>
<td></td>
</tr>
<tr>
<td>Jun-92</td>
<td>88</td>
<td>43</td>
<td>67</td>
<td>45</td>
</tr>
<tr>
<td>Sep-92</td>
<td>53</td>
<td>73</td>
<td>44</td>
<td>26</td>
</tr>
<tr>
<td>Mar-93</td>
<td>68</td>
<td>43</td>
<td>61</td>
<td>37</td>
</tr>
<tr>
<td>Sep-93</td>
<td>43</td>
<td>117</td>
<td>29</td>
<td>38</td>
</tr>
<tr>
<td>Dec-93</td>
<td>46</td>
<td>112</td>
<td>29</td>
<td>35</td>
</tr>
<tr>
<td>Mar-94</td>
<td>95</td>
<td>66</td>
<td>59</td>
<td>46</td>
</tr>
<tr>
<td>Jun-94</td>
<td>44</td>
<td>110</td>
<td>29</td>
<td>21</td>
</tr>
<tr>
<td>Sep-94</td>
<td>43</td>
<td>127</td>
<td>25</td>
<td>26</td>
</tr>
<tr>
<td>Total*</td>
<td>480</td>
<td>691</td>
<td>41</td>
<td>274</td>
</tr>
</tbody>
</table>

Odds Ratio** 1.5 [1.3, 1.8]

* No significant difference between regions or visits [Chi squared P>0.05]
** Odds ratio for infection with hookworm in males compared with females
Table 7.19: Sex Related Hookworm Infection and Reinfection Rates in Dogs
Three Months after Treatment - Looma

<table>
<thead>
<tr>
<th></th>
<th>Hookworm Positive Dogs</th>
<th>Hookworm Negative Dogs</th>
<th>Positive and Male</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number of Positive Dogs</td>
<td>Number of Negative Dogs</td>
<td>Percentage of Positive Dogs that were Male</td>
</tr>
<tr>
<td>Dec-92</td>
<td>26</td>
<td>30</td>
<td>76.9</td>
</tr>
<tr>
<td>Mar-93</td>
<td>33</td>
<td>9</td>
<td>57.6</td>
</tr>
<tr>
<td>Jun-93</td>
<td>20</td>
<td>29</td>
<td>55</td>
</tr>
<tr>
<td>Sep-93</td>
<td>17</td>
<td>27</td>
<td>64.7</td>
</tr>
<tr>
<td>Dec-93</td>
<td>29</td>
<td>25</td>
<td>89.7</td>
</tr>
<tr>
<td>Mar-94</td>
<td>22</td>
<td>21</td>
<td>59.1</td>
</tr>
<tr>
<td>Jun-94</td>
<td>11</td>
<td>41</td>
<td>27.3</td>
</tr>
<tr>
<td>Sep-94</td>
<td>6</td>
<td>38</td>
<td>50</td>
</tr>
</tbody>
</table>

*Statistically significant
7.3.2.3 The Effect of the Number of Dogs per Household on Infection Rates

No statistical association was found between multiple-dog households or single-dog households and hookworm status, except for the central region, when all pre- and post-treatment data were pooled (Table 7.20). Dogs from single-dog households in the central region were found to be 2 times more likely to be infected with hookworm than dogs from households with more than one dog [1.1, 3.8].

7.3.2.3 Environmental Factors Affecting Hookworm Infection Rates

7.3.2.3.1 Correlation between Rainfall and Hookworm Infection Rates

The only area to show strong statistically significant correlation between overall hookworm prevalence and accumulated 3 monthly rainfall was the coastal region ($r=0.94$, $P=0.002$) (Table 7.21). As the accumulated 3 monthly rainfall volumes increased, so did the prevalence of hookworm at these communities.

The prevalence of hookworm in dogs treated three months before sampling and those not treated was also strongly correlated to the total rainfall attained in the three months before sampling at the coastal region ($r=0.9$, $P=0.006$ and $r=0.97$, $P=0.0003$, respectively) (Table 7.22). This indicated that increased rainfall was an important environmental factor for acquiring hookworm infection for both treated and untreated dogs. Data from Looma also showed a positive correlation between rainfall and prevalence in previously treated dogs ($r=0.7$, $P=0.05$), but not in dogs that were untreated.

7.3.2.3.2 Temperature

Mean minimum and maximum daily temperature for the months preceding hookworm infection was not shown to correlate with overall prevalence of hookworm or the infection rate in treated or non-treated groups. This probably indicates that the mean temperature was always adequate for larval development.
Table 7.20: Hookworm and Numbers of Dogs per Household for Coastal, Central and Eastern Regions *

<table>
<thead>
<tr>
<th>Coastal Region</th>
<th>Number Positive for Hookworm</th>
<th>Total Number Sampled</th>
<th>Percentage Infected</th>
<th>Odds Ratio</th>
<th>95% Confidence Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt; 4 Dogs per house</td>
<td>20</td>
<td>66</td>
<td>30.3</td>
<td>1.0</td>
<td>0.6, 17.8</td>
</tr>
<tr>
<td>&lt;= 4 Dogs per house</td>
<td>106</td>
<td>351</td>
<td>30.2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 Dog per house</td>
<td>23</td>
<td>75</td>
<td>30.7</td>
<td>1.0</td>
<td>0.6, 1.8</td>
</tr>
<tr>
<td>&gt; 1 Dog per house</td>
<td>103</td>
<td>343</td>
<td>30.0</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Central Region</th>
<th>Number Positive for hookworm</th>
<th>Total Number Sampled</th>
<th>Percentage Infected</th>
<th>Odds Ratio</th>
<th>95% Confidence Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt; 4 Dogs per house</td>
<td>169</td>
<td>500</td>
<td>33.8</td>
<td>0.8</td>
<td>0.6, 1.1</td>
</tr>
<tr>
<td>&lt;= 4 Dogs per house</td>
<td>126</td>
<td>323</td>
<td>39</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 Dog per house</td>
<td>23</td>
<td>44</td>
<td>52.3</td>
<td>2.0</td>
<td>1.1, 3.8</td>
</tr>
<tr>
<td>&gt; 1 Dog per house</td>
<td>272</td>
<td>781</td>
<td>34.8</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Eastern Region</th>
<th>Number Positive for Hookworm</th>
<th>Total Number Sampled</th>
<th>Percentage Infected</th>
<th>Odds Ratio</th>
<th>95% Confidence Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt; 4 Dogs per house</td>
<td>219</td>
<td>544</td>
<td>40.3</td>
<td>0.4</td>
<td>0.6, 12</td>
</tr>
<tr>
<td>&lt;= 4 Dogs per house</td>
<td>67</td>
<td>153</td>
<td>43.8</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 Dog per house</td>
<td>4</td>
<td>7</td>
<td>57.1</td>
<td>1.9</td>
<td>0.4, 8.7</td>
</tr>
<tr>
<td>&gt; 1 Dog per house</td>
<td>282</td>
<td>690</td>
<td>40.9</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Data pooled from pre-and post- treatment samplings
### Table 7.21: Correlation Between Rainfall and Hookworm Prevalence

<table>
<thead>
<tr>
<th>Coastal Region</th>
<th>Central Region</th>
<th>Eastern Region</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rainfall (mm)</td>
<td>Hookworm Prevalence</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sep-92</td>
<td>0.4</td>
<td>20</td>
</tr>
<tr>
<td>Mar-93</td>
<td>560.6</td>
<td>52.3</td>
</tr>
<tr>
<td>Sep-93</td>
<td>27.2</td>
<td>28.2</td>
</tr>
<tr>
<td>Dec-93</td>
<td>0.1</td>
<td>24.7</td>
</tr>
<tr>
<td>Mar-94</td>
<td>753.4</td>
<td>64.5</td>
</tr>
<tr>
<td>Jun-94</td>
<td>121</td>
<td>24.1</td>
</tr>
<tr>
<td>Sep-94</td>
<td>0</td>
<td>16.1</td>
</tr>
<tr>
<td>Correlation (Pearsons)</td>
<td>0.94</td>
<td>0.46</td>
</tr>
<tr>
<td>P value</td>
<td>0.0017</td>
<td>0.3</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Looma</th>
<th>Kalumburu</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rainfall (mm)</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>Dec-92</td>
<td>2.8</td>
</tr>
<tr>
<td>Mar-93</td>
<td>505</td>
</tr>
<tr>
<td>Jun-93</td>
<td>14.6</td>
</tr>
<tr>
<td>Sep-93</td>
<td>67.4</td>
</tr>
<tr>
<td>Dec-93</td>
<td>16.0</td>
</tr>
<tr>
<td>Mar-94</td>
<td>489.9</td>
</tr>
<tr>
<td>Jun-94</td>
<td>54.0</td>
</tr>
<tr>
<td>Sep-94</td>
<td>0</td>
</tr>
<tr>
<td>Correlation (Pearsons)</td>
<td>0.64</td>
</tr>
<tr>
<td>P value</td>
<td>0.09</td>
</tr>
</tbody>
</table>
Table 7.22: Correlation Between Rainfall and Hookworm Prevalence of Dogs Treated and Not Treated Previously at Looma and the Coastal Region*

<table>
<thead>
<tr>
<th>Coastal Region</th>
<th>Hookworm prevalence – Treated Previously</th>
<th>Hookworm prevalence – Not Treated Previously</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sep-92</td>
<td>21.2</td>
<td>44.4</td>
</tr>
<tr>
<td>Mar-93</td>
<td>48</td>
<td>70</td>
</tr>
<tr>
<td>Sep-93</td>
<td>15.1</td>
<td>47.2</td>
</tr>
<tr>
<td>Dec-93</td>
<td>20</td>
<td>43.5</td>
</tr>
<tr>
<td>Mar-94</td>
<td>57.1</td>
<td>75</td>
</tr>
<tr>
<td>Jun-94</td>
<td>8.9</td>
<td>44.4</td>
</tr>
<tr>
<td>Sep-94</td>
<td>4.9</td>
<td>36.4</td>
</tr>
<tr>
<td>Correlation (Pearsons)</td>
<td>0.90</td>
<td>0.97</td>
</tr>
<tr>
<td>P value</td>
<td>0.006</td>
<td>0.0003</td>
</tr>
</tbody>
</table>

*Correlation not statistically significant at other regions.

<table>
<thead>
<tr>
<th>Looma</th>
<th>Hookworm prevalence – Treated Previously</th>
<th>Hookworm prevalence – Not Treated Previously</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dec-92</td>
<td>48</td>
<td>75</td>
</tr>
<tr>
<td>Mar-93</td>
<td>79</td>
<td>75</td>
</tr>
<tr>
<td>Jun-93</td>
<td>39</td>
<td>80</td>
</tr>
<tr>
<td>Sep-93</td>
<td>39</td>
<td>100</td>
</tr>
<tr>
<td>Dec-93</td>
<td>53</td>
<td>75</td>
</tr>
<tr>
<td>Mar-94</td>
<td>53</td>
<td>50</td>
</tr>
<tr>
<td>Jun-94</td>
<td>21</td>
<td>38.5</td>
</tr>
<tr>
<td>Sep-94</td>
<td>14</td>
<td>86.7</td>
</tr>
<tr>
<td>Correlation [Pearsons]</td>
<td>0.70</td>
<td>-0.32</td>
</tr>
<tr>
<td>P value</td>
<td>0.05</td>
<td>0.44</td>
</tr>
</tbody>
</table>
7.3.2.4 Effect of Parasite Control Program on Dog Health Parameters

7.3.2.4.1 Weight Changes of Dogs during the Program

For pooled data for each month of sampling from three representative communities of each region (Kalumburu, Looma and Warmun), Kalumburu was the only location where an association between weight changes and infection with hookworm was noted (Table 7.23). Overall, dogs with hookworm were two and a half times more likely to have a weight increase between sampling periods [1.5, 7.2].

7.3.2.4.2 Packed Cell Volume Measurements

The survey in September 1992 (three months after the commencement of the treatments) to determine anaemia status (see 7.2) included dogs from each of the mainstream communities of each region. There was no statistical difference in the mean packed cell volume (PCV) in this survey for each region, so the data from each region were pooled (Student t tests, P>0.05).

The mean PCV for the population sampled three months after the first treatment was 45.00 ± SEM 0.57 L/L. Dogs less than one year of age had a mean PCV of 35.29 ± SEM 0.89 L/L and adult dogs had a mean PCV of 48.57 ± SEM 0.55 L/L.

Dogs less than 1 year of age were found to be 23 [11.3, 46.4] times more likely to have below normal PCV levels (0.37L/L-0.55L/L; Jain, 1993) than older dogs, irrespective of hookworm status (Table 7.24).

The low haematocrit readings from samples from puppies and juveniles were associated with both age and hookworm status as puppies and juveniles with hookworm were found to be 5 times more likely to have low packed cell volumes than those without hookworm [1.4, 17.7] Table 7.25). Adult dogs with hookworm were also found to be 4 times more likely to have below normal haematocrit than dogs without [1.1, 14.7] (Table 7.26).
Table 7.23: Hookworm Infection and Weight Changes*

<table>
<thead>
<tr>
<th></th>
<th>Kalumburu</th>
<th></th>
<th></th>
<th>Looma</th>
<th></th>
<th></th>
<th>Warmun</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Hookworm Positive</td>
<td>%</td>
<td>Odds Ratio**</td>
<td>95% Confidence Interval</td>
<td>Hookworm Positive</td>
<td>%</td>
<td>Odds Ratio**</td>
<td>95% Confidence Interval</td>
<td>Hookworm Positive</td>
</tr>
<tr>
<td>Weight Increase</td>
<td>21</td>
<td>31.8</td>
<td>3.2</td>
<td>1.7, 8.7</td>
<td>36</td>
<td>34.3</td>
<td>0.7</td>
<td>0.4, 1.3</td>
<td>23</td>
</tr>
<tr>
<td>Weight Decrease</td>
<td>6</td>
<td>18.8</td>
<td></td>
<td></td>
<td>28</td>
<td>49.1</td>
<td></td>
<td></td>
<td>10</td>
</tr>
<tr>
<td>No Change</td>
<td>6</td>
<td>9.5</td>
<td></td>
<td></td>
<td>16</td>
<td>33.3</td>
<td></td>
<td></td>
<td>17</td>
</tr>
</tbody>
</table>

* Data pooled for each visit

** Odds ratio for hookworm infection and weight increase compared with no change in weight or weight loss
Table 7.24: Packed Cell Volumes and Age – All Regions*

<table>
<thead>
<tr>
<th>Packed Cell Volume [L/L]</th>
<th>Number of Puppies and Juveniles</th>
<th>Number of Adults</th>
<th>%</th>
<th>Odds Ratio</th>
<th>95% Confidence Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;0.37</td>
<td>50</td>
<td>13</td>
<td>79.4</td>
<td>22.9</td>
<td>11.3, 46.4</td>
</tr>
<tr>
<td>0.37 - 0.55</td>
<td>35</td>
<td>172</td>
<td>16.9</td>
<td>0.2</td>
<td>0.1, 0.3</td>
</tr>
<tr>
<td>&gt;0.55</td>
<td>0</td>
<td>36</td>
<td>0</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Range</td>
<td>18-55</td>
<td></td>
<td></td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Mean</td>
<td>35.9</td>
<td></td>
<td></td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>
Table 7.25: Packed Cell Volumes and Hookworm Infection – Puppies and Juveniles, All Regions*

<table>
<thead>
<tr>
<th>Packed Cell Volume [L/L]</th>
<th>Number Positive</th>
<th>Number Negative</th>
<th>% Positive</th>
<th>Odds Ratio</th>
<th>95% Confidence Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;0.37</td>
<td>20</td>
<td>17</td>
<td>54</td>
<td>5</td>
<td>1.4, 17.7</td>
</tr>
<tr>
<td>0.37 - 0.55</td>
<td>4</td>
<td>16</td>
<td>20</td>
<td>0.2</td>
<td>0.1, 0.8</td>
</tr>
<tr>
<td>&gt;0.55</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>
Table 7.26: Packed Cell Volumes and Hookworm Infection – Adults, All Regions*

<table>
<thead>
<tr>
<th>Packed Cell Volume [L/L]</th>
<th>Number Positive</th>
<th>Number Negative</th>
<th>% Positive</th>
<th>Odds Ratio</th>
<th>95% Confidence Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;0.37</td>
<td>5</td>
<td>5</td>
<td>50</td>
<td>4</td>
<td>1.1, 14.7</td>
</tr>
<tr>
<td>0.37 - 0.55</td>
<td>25</td>
<td>115</td>
<td>18</td>
<td>0.4</td>
<td>0.2, 0.9</td>
</tr>
<tr>
<td>&gt;0.55</td>
<td>8</td>
<td>17</td>
<td>32</td>
<td>1.9</td>
<td>0.7, 1.9</td>
</tr>
</tbody>
</table>

* No statistical difference between regions [Chi squared test, P>0.05]
7.3.2.4.3 Total Plasma Protein Measurements

For each of the PCV samples, total plasma protein (total solid - TS) levels were calculated to give an indication of the hydration status of each animal (see 3.3.7.3). Out of 308 samples, 167 were hyperproteinaemic (TS >75g/L). Of these samples from suspected dehydrated animals, 28 were polycythaemic (PCV>0.55L/L) and 11 were anaemic (PCV<0.37L/L) according to packed cell volume readings. The latter 11 dogs were most likely severely anaemic.

7.3.3 Roundworm

7.3.3.1 The Effect of Ivermectin Treatments on Toxocara canis Prevalence

Toxocara canis was uncommon at the central region with the prevalence varying at each visit from 0 to 3.6% [0, 7.6] (Table 7.27-7.28). Although Looma is in close vicinity to the other central region communities, the prevalence was higher with 1.5% [0, 4.4] to 9.3% [2.2, 16.2] being infected at each visit. Five of 18 infected dogs from this community were just acquired from coastal communities.

The coastal region had an overall higher prevalence with a range of 0 to 8.6% [2.9, 14.3] (Table 7.27). Kalumburu had the greatest proportion of dogs shedding eggs with an average of 9.6% [6.7, 12.5] for each visit (Table 7.28). Twelve dogs from Kalumburu with Toxocara were born from the same dam.

Although 50% of dogs with roundworm from the coastal and central region communities were new to the program and subsequently did not maintain infection after treatment, no statistical association between treatment and roundworm infection could be demonstrated for Kalumburu (where there were adequate numbers for statistical analyses) (Table 7.29). When age was controlled and only adults were considered, there still was no demonstrable association between treatment and a reduction in Toxocara prevalence.
Table 7.27: Age-Prevalence of *Toxocara canis* in Dogs – Coastal and Central Regions*

<table>
<thead>
<tr>
<th></th>
<th>Coastal Region</th>
<th></th>
<th>Central Region</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number Infected/Total Number in Age Group</td>
<td>TOTAL</td>
<td>Overall Prevalence (%)</td>
<td>Number Infected/Total Number in Age Group</td>
</tr>
<tr>
<td></td>
<td>Puppy</td>
<td>Juvenile</td>
<td>Adult</td>
<td>Puppy</td>
</tr>
<tr>
<td>Jun-92</td>
<td>0/8</td>
<td>0/8</td>
<td>0/34</td>
<td>0</td>
</tr>
<tr>
<td>Sep-92</td>
<td>1/3</td>
<td>2/13</td>
<td>0/29</td>
<td>3</td>
</tr>
<tr>
<td>Mar-93</td>
<td>1/2</td>
<td>0/3</td>
<td>0/39</td>
<td>1</td>
</tr>
<tr>
<td>Sep-93</td>
<td>0/17</td>
<td>0/10</td>
<td>1/58</td>
<td>1</td>
</tr>
<tr>
<td>Dec-93</td>
<td>4/16</td>
<td>0/9</td>
<td>4/68</td>
<td>8</td>
</tr>
<tr>
<td>Mar-94</td>
<td>0/11</td>
<td>0/11</td>
<td>0/54</td>
<td>0</td>
</tr>
<tr>
<td>Jun-94</td>
<td>0/8</td>
<td>1/16</td>
<td>0/55</td>
<td>1</td>
</tr>
<tr>
<td>Sep-94</td>
<td>2/18</td>
<td>1/12</td>
<td>0/63</td>
<td>3</td>
</tr>
</tbody>
</table>

*Toxocara canis* not found at eastern region

* *Toxocara canis* not found at eastern region
Table 7.28: Prevalence of *Toxocara canis* in Dogs – Kalumburu and Looma

<table>
<thead>
<tr>
<th></th>
<th>Kalumburu</th>
<th></th>
<th></th>
<th>Looma</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number</td>
<td>Overall Prevalence (%)</td>
<td>Number</td>
<td>Overall Prevalence</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Sampled</td>
<td></td>
<td>Sampled</td>
<td>(%)</td>
<td></td>
</tr>
<tr>
<td>Mar-93</td>
<td>43</td>
<td>4.7</td>
<td></td>
<td>Sep-92</td>
<td>2.3</td>
</tr>
<tr>
<td>Jun-93</td>
<td>72</td>
<td>11.1</td>
<td></td>
<td>Dec-92</td>
<td>2.9</td>
</tr>
<tr>
<td>Sep-93</td>
<td>61</td>
<td>6.6</td>
<td></td>
<td>Mar-93</td>
<td>2</td>
</tr>
<tr>
<td>Dec-93</td>
<td>53</td>
<td>7.5</td>
<td></td>
<td>Jun-93</td>
<td>6.6</td>
</tr>
<tr>
<td>Mar-94</td>
<td>60</td>
<td>10</td>
<td></td>
<td>Sep-93</td>
<td>1.6</td>
</tr>
<tr>
<td>Jun-94</td>
<td>54</td>
<td>11.1</td>
<td></td>
<td>Dec-93</td>
<td>1.5</td>
</tr>
<tr>
<td>Sep-94</td>
<td>61</td>
<td>14.8</td>
<td></td>
<td>Mar-94</td>
<td>1.6</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Jun-94</td>
<td>9.2</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Sep-94</td>
<td>1.5</td>
</tr>
<tr>
<td>Average (Pooled Data)</td>
<td>9.6 [6.7, 12.5]</td>
<td></td>
<td>Average (Pooled Data)</td>
<td>3.3 [1.9, 4.7]</td>
<td></td>
</tr>
</tbody>
</table>
Table 7.29: Risk Factors for Infection with *Toxocara canis* in Dogs - Kalumburu

<table>
<thead>
<tr>
<th>Factor Tested</th>
<th>Category</th>
<th>T. canis Positive</th>
<th>T. canis Negative</th>
<th>Percentage</th>
<th>Odds Ratio</th>
<th>95% Confidence Intervals</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td>male entire</td>
<td>26</td>
<td>189</td>
<td>12.1</td>
<td>1.3</td>
<td>0.6, 2.6</td>
</tr>
<tr>
<td></td>
<td>female entire</td>
<td>13</td>
<td>120</td>
<td>9.8</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>≤ 1 year</td>
<td>16</td>
<td>24</td>
<td>40</td>
<td>9.1</td>
<td>4.2, 19.8</td>
</tr>
<tr>
<td></td>
<td>&gt; 1 year</td>
<td>21</td>
<td>288</td>
<td>6.8</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Treatment</td>
<td>Treated 3 months previously</td>
<td>26</td>
<td>218</td>
<td>10.6</td>
<td>0.7</td>
<td>0.2, 1.9</td>
</tr>
<tr>
<td></td>
<td>Not treated</td>
<td>5</td>
<td>29</td>
<td>14.7</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
7.3.3.2 Risk Factors for *Toxocara canis* Infection

7.3.3.2.1 Age of Host

*Toxocara canis* infection was much more common in young dogs than adults (Table 7.27). About a third of the infections at the coastal region were in adults, and at the central region, all dogs with *T. canis* were under 1 year old. At Kalumburu, puppies were 9 [4.2, 19.8] times more likely to be infected with roundworm than juveniles or adults (Table 7.29).

7.3.3.2.2 Sex of Host

There was no demonstrable sex predilection for roundworm infection. Even when the confounding variable of age was controlled, sex of the hosts still did not affect the *T. canis* infection rate in the present study.

7.3.4 Other Parasites and Bacteria

7.3.4.1 *Giardia duodenalis*

7.3.4.1.1 The Effect of Ivermectin Treatments on *Giardia duodenalis* Prevalence

The average prevalence of *Giardia* for each region for the entire program was 13% [11.6, 14.4] (P>0.05) (Table 7.30-7.31). The treatments with ivermectin did not affect the prevalence of *Giardia* at any region or community.

7.3.4.1.2 Risk Factors for *Giardia duodenalis* Infection

7.3.4.1.2.1 Age of Host

When statistical association was evident, younger dogs were more likely to be infected with *Giardia* than older dogs (see Appendix H). Puppies from the coastal region were 4 [1.1, 17.7] to 28 [3.1, 263] times more likely to have *Giardia* than juveniles and adults. Similarly, puppies from the eastern region were 3 [1.1, 10.2] to 14 [3.5, 55.4] times more at risk than adults.
<table>
<thead>
<tr>
<th></th>
<th>Coastal Region</th>
<th></th>
<th>Central Region</th>
<th></th>
<th>Eastern Region</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number Sampled</td>
<td>Percentage Positive</td>
<td>Number Sampled</td>
<td>Percentage Positive</td>
<td>Number Sampled</td>
<td>Percentage Positive</td>
</tr>
<tr>
<td>Jun-92</td>
<td>50</td>
<td>16</td>
<td>83</td>
<td>18.1</td>
<td>68</td>
<td>10.3</td>
</tr>
<tr>
<td>Sep-92</td>
<td>45</td>
<td>8.9</td>
<td>109</td>
<td>11</td>
<td>76</td>
<td>23.7*</td>
</tr>
<tr>
<td>Mar-93</td>
<td>44</td>
<td>13.6</td>
<td>68</td>
<td>20.6</td>
<td>73</td>
<td>15.1</td>
</tr>
<tr>
<td>Sep-93</td>
<td>85</td>
<td>11.8</td>
<td>118</td>
<td>10.2</td>
<td>82</td>
<td>20.7</td>
</tr>
<tr>
<td>Dec-93</td>
<td>93</td>
<td>15.1</td>
<td>111</td>
<td>9.9</td>
<td>103</td>
<td>11.7</td>
</tr>
<tr>
<td>Mar-94</td>
<td>76</td>
<td>15.8</td>
<td>104</td>
<td>14.4</td>
<td>98</td>
<td>8.2</td>
</tr>
<tr>
<td>Jun-94</td>
<td>79</td>
<td>13.9</td>
<td>110</td>
<td>16.4</td>
<td>91</td>
<td>19.8</td>
</tr>
<tr>
<td>Sep-94</td>
<td>93</td>
<td>6.5</td>
<td>111</td>
<td>9</td>
<td>92</td>
<td>20.7</td>
</tr>
<tr>
<td>Average Prevalence***</td>
<td>12.3 [9.6, 15.0]</td>
<td></td>
<td>13.3 [11.0, 15.6]</td>
<td></td>
<td>15.0** [12.4, 17.6]</td>
<td></td>
</tr>
</tbody>
</table>

* Statistically different to other visits (P<0.05)
** Excluding September 1992
***No statistical difference between regions; average percentage = 13.0% [CI: 11.6, 14.4]
Table 7.31: Prevalence of *Giardia* in Dogs – Kalumburu and Looma

<table>
<thead>
<tr>
<th></th>
<th>Kalumburu</th>
<th>Looma</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number Sampled</td>
<td>Percentage Positive</td>
</tr>
<tr>
<td>Mar-93</td>
<td>43</td>
<td>8.9</td>
</tr>
<tr>
<td>Jun-93</td>
<td>72</td>
<td>5.5</td>
</tr>
<tr>
<td>Sep-93</td>
<td>61</td>
<td>11.5</td>
</tr>
<tr>
<td>Dec-93</td>
<td>53</td>
<td>17.0</td>
</tr>
<tr>
<td>Mar-94</td>
<td>60</td>
<td>5.0</td>
</tr>
<tr>
<td>Jun-94</td>
<td>54</td>
<td>13.0</td>
</tr>
<tr>
<td>Sep-94</td>
<td>61</td>
<td>14.8</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Average</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prevalence *</td>
<td>10.6 [7.6, 13.6]</td>
<td></td>
</tr>
</tbody>
</table>

*No statistical difference between visits*
7.3.4.1.2.2 Sex of Host

No statistical association between *Giardia* infection and sex of host was demonstrated in the present study (Table 7.32).

7.3.4.1.2.3 The Effect of the Number of Dogs per Household on *Giardia* Infection Rates

Dogs from the central region living in households with four or more others were 2 [1.2, 3.0] times at risk of attaining *Giardia* than other dogs. Dogs from households with two or more dogs were 7 times more likely to be infected with *Giardia* than dogs living without others in the eastern region (Table 7.33).

7.3.4.2 *Dirofilaria immitis*

7.3.4.2.1 The Effect of Ivermectin Treatments on *Dirofilaria immitis* Prevalence

Repeat testing of dogs (from Kalumburu, Warmun and the coastal and central regions), that had been treated with ivermectin 3 months earlier, revealed a statistically significant reduction in prevalence of microfilariae circulation (Table 7.34). After 3 treatments (9 months), the percentage of dogs with microfilariae reduced from 60.8% [49.7, 71.9] to 4.6% [0, 9.7] at Kalumburu. At the coastal region a significant reduction was seen after one treatment (reduction from 39.8% [30.3, 49.2] to 19.4% [9.6, 29.2]). At Warmun and the central region, there were almost no dogs with circulating microfilariae (0 and 0.8%, respectively) after 7 treatments.

Of the 103 dogs originally tested from the coastal region, the mortality rate over the duration of the program was 24.3% [16.0, 32.6] for dogs with heartworm compared with 45.6% [31.1, 60.1] for those without. There was no statistical difference between the two (P=1.0000, Fishers Exact test).
Table 7.32: Sex-Related Prevalence of *Giardia* in Dogs

<table>
<thead>
<tr>
<th></th>
<th>Male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number Sampled</td>
<td>Percentage Positive</td>
</tr>
<tr>
<td>Jun-92</td>
<td>131</td>
<td>13.0</td>
</tr>
<tr>
<td>Sep-92</td>
<td>126</td>
<td>13.5</td>
</tr>
<tr>
<td>Mar-93</td>
<td>111</td>
<td>15.3</td>
</tr>
<tr>
<td>Sep-93</td>
<td>160</td>
<td>13.1</td>
</tr>
<tr>
<td>Dec-93</td>
<td>158</td>
<td>14.6</td>
</tr>
<tr>
<td>Mar-94</td>
<td>161</td>
<td>11.8</td>
</tr>
<tr>
<td>Jun-94</td>
<td>154</td>
<td>17.5</td>
</tr>
<tr>
<td>Sep-94</td>
<td>170</td>
<td>13.5</td>
</tr>
<tr>
<td>Average Percentage (Pooled Data*)</td>
<td>14.0</td>
<td>13.5</td>
</tr>
</tbody>
</table>
| Odds Ratio** | 1.0 [0.8, 1.3] | **No statistical difference between visits and communities**
|         |              |              |

**Odds ratio for infection with *Giardia* in males compared with females**
Table 7.33: *Giardia* and Numbers of Dogs per Household - Coastal, Central and Eastern Regions

<table>
<thead>
<tr>
<th>Coastal Region</th>
<th>Number Positive for <em>Giardia</em></th>
<th>Total Number Sampled</th>
<th>Percentage</th>
<th>Odds Ratio</th>
<th>95% Confidence Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt; 4 Dogs per house</td>
<td>11</td>
<td>66</td>
<td>16.7</td>
<td>1.4</td>
<td>0.7, 3.0</td>
</tr>
<tr>
<td>&lt;= 4 Dogs per house</td>
<td>42</td>
<td>351</td>
<td>12.0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 Dog per house</td>
<td>10</td>
<td>75</td>
<td>13.3</td>
<td>0.9</td>
<td>0.5, 1.9</td>
</tr>
<tr>
<td>&gt; 1 Dog per house</td>
<td>43</td>
<td>343</td>
<td>13.5</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Central Region</th>
<th>Number Positive for <em>Giardia</em></th>
<th>Total Number Sampled</th>
<th>Percentage</th>
<th>Odds Ratio</th>
<th>95% Confidence Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt; 4 Dogs per house</td>
<td>78</td>
<td>500</td>
<td>15.6</td>
<td>1.9</td>
<td>1.2, 3.0*</td>
</tr>
<tr>
<td>&lt;= 4 Dogs per house</td>
<td>29</td>
<td>323</td>
<td>9.0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 Dog per house</td>
<td>4</td>
<td>44</td>
<td>9.1</td>
<td>1.6</td>
<td>0.6, 4.5</td>
</tr>
<tr>
<td>&gt; 1 Dog per house</td>
<td>106</td>
<td>781</td>
<td>13.6</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Eastern Region</th>
<th>Number Positive for <em>Giardia</em></th>
<th>Total Number Sampled</th>
<th>Percentage</th>
<th>Odds Ratio</th>
<th>95% Confidence Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt; 4 Dogs per house</td>
<td>83</td>
<td>544</td>
<td>15.3</td>
<td>0.8</td>
<td>0.5, 1.4</td>
</tr>
<tr>
<td>&lt;= 4 Dogs per house</td>
<td>27</td>
<td>153</td>
<td>17.6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 Dog per house</td>
<td>4</td>
<td>7</td>
<td>57.1</td>
<td>0.1</td>
<td>0.03, 0.6*</td>
</tr>
<tr>
<td>&gt; 1 Dog per house</td>
<td>106</td>
<td>690</td>
<td>15.4</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Statistically significant*
Table 7.34: *Dirofilaria immitis* Post-Treatment Prevalence

<table>
<thead>
<tr>
<th>Region or Community Sampled</th>
<th>Number of Treatments</th>
<th>Number Sampled</th>
<th>Number with Microfilariae</th>
<th>Percentage of Sampled Dogs with Microfilariae</th>
<th>95% Confidence Intervals</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kalumburu</td>
<td>0</td>
<td>74</td>
<td>45</td>
<td>60.8</td>
<td>49.7, 71.9</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>65</td>
<td>3</td>
<td>4.6</td>
<td>0, 10.1</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>64</td>
<td>2</td>
<td>3.1</td>
<td>-0.1, 7.3</td>
</tr>
<tr>
<td>Coastal Region</td>
<td>0</td>
<td>103</td>
<td>41</td>
<td>39.8</td>
<td>30.3, 49.2</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>62</td>
<td>12</td>
<td>19.4</td>
<td>9.6, 29.2</td>
</tr>
<tr>
<td></td>
<td>7</td>
<td>85</td>
<td>4</td>
<td>4.7</td>
<td>0.9, 9.2</td>
</tr>
<tr>
<td>Warmun</td>
<td>0</td>
<td>102</td>
<td>28</td>
<td>27.4*</td>
<td>8.7, 36.1</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>82</td>
<td>14</td>
<td>17.1*</td>
<td>9.0, 25.2</td>
</tr>
<tr>
<td></td>
<td>7</td>
<td>97</td>
<td>0</td>
<td>0</td>
<td>-</td>
</tr>
<tr>
<td>Central Region</td>
<td>0</td>
<td>196</td>
<td>19</td>
<td>9.7*</td>
<td>5.6, 13.8</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>67</td>
<td>10</td>
<td>14.9*</td>
<td>6.4, 12.4</td>
</tr>
<tr>
<td></td>
<td>7</td>
<td>120</td>
<td>1</td>
<td>0.8</td>
<td>-0.8, 2.4</td>
</tr>
</tbody>
</table>

*No statistical difference between samplings (P<0.05)*
7.3.4.3 *Spirocerca lupi*

*Spirocerca lupi* was only isolated from dogs from three communities; Kalumburu, Yaramun and Warmun (the latter two are communities of the eastern region). Male and female dogs with *S. lupi* infection were equally represented (P>0.05) with age ranges from approximately 9 months to 5 years. Twenty of the 29 infected dogs that were re-examined throughout the program were found to be in good condition. The mortality rate for dogs with *S. lupi* did not differ from those without (P>0.05).

7.3.4.3.1 The Effect of Ivermectin Treatments on *Spirocerca lupi* Prevalence

After the pretreatment survey, a further 38 dogs (in addition to the original 28) were found to be shedding *S. lupi* eggs. Nine dogs (13.6% [5.3, 21.9], 11/66) were still shedding eggs at a second sampling, 3 months after treatment (see Table 7.35). Repeated positive samples preclude the possibility of ‘pseudoparasitism’ by spirurids for which dogs are not normally a host (such as *Abbreviata hastaspicula* (Barton and McEwan, 1993)).

On two occasions, in June 1993 and June 1994, at Kalumburu, there was a statistically significant decrease in *S. lupi* prevalence in dogs that had been previously treated. Dogs that had been treated were 7 [1.7, 31.9] and 25 [1.4, 35.2] times more likely to be negative than dogs that had not been treated.

7.3.4.4 Gastrointestinal Bacteria

7.3.4.4.1 Risk Factors for Infection with *Campylobacter* and *Salmonella*

7.3.4.4.1.1 Age and Sex of Host

No statistical associations could be found for sex of host and bacterial infections, but adults were found to be 5 [1.1, 20.5] times more likely to have *Salmonella* than younger dogs at Looma. Age predilections were not demonstrated for the other locations.
Table 7.35: Prevalence of *Spirocerca lupi* in Dogs

<table>
<thead>
<tr>
<th>Month of Sampling</th>
<th>Number Sampled</th>
<th>Number Positive</th>
<th>%</th>
<th>Prevalence in Dogs Treated 3 Months Previously</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Kalumburu</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mar-93</td>
<td>45</td>
<td>16</td>
<td>35.6</td>
<td></td>
</tr>
<tr>
<td>Jun-93</td>
<td>69</td>
<td>13</td>
<td>18.8</td>
<td>12.7* OR 7 [1.7, 31.9]</td>
</tr>
<tr>
<td>Sep-93</td>
<td>62</td>
<td>3</td>
<td>4.8</td>
<td>5.8</td>
</tr>
<tr>
<td>Dec-93</td>
<td>50</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Mar-94</td>
<td>59</td>
<td>3</td>
<td>5.1</td>
<td>2.4</td>
</tr>
<tr>
<td>Jun-94</td>
<td>54</td>
<td>5</td>
<td>9.3</td>
<td>2.1* OR 25 [1.4, 35.2]</td>
</tr>
<tr>
<td>Sep-94</td>
<td>61</td>
<td>1</td>
<td>1.6</td>
<td>0</td>
</tr>
</tbody>
</table>

5 out of 37 infections were repeat infections

| **Yaramun**       |                |                 |     |                                               |
| Sep-92            | 26             | 2               | 7.7 |                                               |
| Dec-92            | 28             | 3               | 10.7| 0                                             |
| Jun-93            | 22             | 3               | 13.6| 9.1                                           |
| Sep-93            | 26             | 3               | 11.5| 18.2                                          |
| Sep-94            | 50             | 3               | 6   | 13.3                                          |

2 out of 14 infections were repeat infections

| **Warmun**        |                |                 |     |                                               |
| Jun-92            | 68             | 10              | 14.7|                                               |
| Sep-92            | 76             | 5               | 6.6 | 4.4                                           |
| Mar-93            | 74             | 3               | 4.1 | 3.4                                           |

All other treatments were negative

2 out of 19 infections were repeat infections

*Statistically significant difference to those not treated (P<0.05), OR = odds ratio for lack of infection and treatment compared to no treatment previously*
7.4 Discussion

7.4.1 Scabies

7.4.1.1 Prevalence of Scabies during the Ivermectin Treatment Program

Although the pilot treatment at Kununurra resulted in a decrease in prevalence of scabies within 3 months (see 5.4.3.1), this was not so obvious in the mainstream communities. The only other community to show such a rapid decline was Kalumburu where the prevalence decreased by 78% after one treatment. Resolution of scabies induced skin lesions may take at least 30 days (Paradis et al., 1997). It is likely that much longer than this is required if secondary skin infections and extensive hyperkeratotic lesions are evident initially, as was the case for approximately 10% of pretreatment dogs (score 4) at the mainstream communities. Conceivably, these dogs may also require a second dose for the ivermectin to reach the mites in the crusts.

Kalumburu was unique in that 84% of the dogs with scabies were at one household, and all dogs in the household were treated. The effect of the number of dogs per household (household density) is important in the treatment of scabies (see 7.4.1.2.2).

There is only one published report of data relating to field treatment trials for scabies control in dogs (Presson et al., 1990). Presson et al. (1990) found that there was a statistically significant reduction in the number of dogs exhibiting signs of scabies in 2 out of 3 Northern Territory communities after monthly treatments with avermectin. The initial prevalence varied from 78% (n=9) to 28% (n=5), but after (an undocumented number of) treatments, there were no dogs with scabies remaining at any of the three communities. The only other information about field treatments for scabies relates to treatment trials in humans. As the scabies mites in humans behave similarly to those in dogs, the effectiveness of treatment studies in humans can be compared with the present study.
A scabies treatment program in people from Minjilang community in the Northern Territory using topical 5% permethrin cream showed similar results to those from the dogs at Kalumburu (Carapetis, Connors, Yarmirr, Krause and Currie, 1997). People were treated and then re-examined every 1-3 months over a 25 month period. Any new cases or contacts were treated at each follow-up visit. The initial prevalence was approximately 29%, which decreased to about 8% one month after treatment (72% reduction). Although the actual compliance rate was unknown, families that had high infestation rates were encouraged to apply the treatment to all members. Overall the post treatment prevalence varied between approximately 2% and 10%. This study demonstrated that a one-off treatment could be successful in controlling scabies, provided that new cases are treated, along with their contacts. A likely reason why the prevalence rate did not decrease as rapidly after one treatment in the present study (apart from the dogs at Kalumburu) is the difficulty in treating all contacts.

The prevalence of scabies in treated and non-treated dogs remained similar throughout the program. This could indicate that; 1) the treated dogs were being reinfected between visits, or 2) the non-treated dogs were benefiting from a reduction in the reservoir of mites, both from scabietic dogs and the environment, or 3) the dogs that were not treated 3 months previously may have been treated beforehand and were still refractory to reinfection due to protective immunity.

The possibility for some reinfection from infected household-partners is high considering the
density of dogs per household (see 7.4.1.2.2). There is potential for contracting mites from the
environment, especially when dogs with crusted lesions are sharing living space. In human
epidemics fomites have been found to be significant in the transmission of mites from infected
to uninfected household members (Blumenthal et al, 1976; Gulati, Singh and Braganza, 1977).

Untreated dogs seem to benefit from the treatment of other dogs because of the reduction in
reservoir of mites. Infected new dogs to the program would serve as a source of infection to the
others, but new dogs without infection are saved from infection if they are introduced to
households without mites. Even if new infected dogs are introduced to dogs that have been
infected, but have subsequently been cured, there is likely to be some protective immunity to
prevent these dogs from severe reinfection (Arlan et al, 1996). This may explain why there
was an equilibrium in infection rates between those that had just been treated and those that
hadn’t.

Misdiagnosis of scabies infection is a possible source of error in these findings. Scott et al
(1995) stated that when scabies is a differential diagnosis, it can be excluded only by the
animal’s failure to respond to treatment. In pigs, the scoring system for papular dermatitis is
effective when groups of pigs are examined, rather than individuals. Davies et al (1996)
considered that reinfection in pen situations is very common and can lead to severe papular
dermatitis in hypersensitive pigs which is not proportional to the number of mites being
harboured. Hence, they devised a group scoring system. On an individual scale, the specificity
of grade 1 lesions was estimated to be 0.79, but generalised lesions (grades 2 and 3) were highly
specific for *Sarcoptes* infection (Davies, Moore and Pointon, 1991). This would also be true of
the present scoring system, but it is also compounded by the extensive list of differentials for
clinically similar skin infections, such as *Demodex*. Score 1 and 2 lesions, when not covering
many areas pathognomonic for *Sarcoptes*, could be confused for localised *Demodex*, in a field
situation. Despite this, most of these lesions would be expected to resolve within 4 to 8 weeks
(Sosna and Medleau, 1992c), irrespective of treatment, if the dog is immunologically normal
(Dunsmore and Shaw, 1990). If the dog is immunosuppressed, has an underlying disease or a
familial predisposition (for juveniles), then the lesions can spread to become generalised. Very
rarely did the severity of lesions progress to become generalised. Generalised demodecosis, on
the other hand, would have remained severe throughout the program and would have been
refractory to treatment. Of 19 dogs with refractory severe dermatitis in the present study, 4
were subsequently diagnosed as having generalised demodecosis. Dogs of the present survey
were good candidates for adult onset generalised demodecosis considering the high prevalence
of intestinal parasitism and heartworm, both possible predisposing factors for the disease (Sosna and Medleau, 1992b).

Six months (March 1995) after the treatment program was completed (in September 1994), the prevalence of scabies at Looma and Kalumburu remained at the same low level as it had at the last treatment visit (September 1994). Even though the dogs were not being treated during the 6 month period, the prevalence had not increased. This complements the previous arguments about protective immunity from previous infection and the reduction of mite reservoirs in treated dogs. It may also be a reflection of increased understanding of owners about scabies, with fewer people introducing infected new dogs to the communities compared with those already in the communities (see Tables 7.7-7.8). Once _Sarcoptes_ is stabilised at a low prevalence, treatments every 3 months may be no more beneficial than treatments every 6 months.

As the treatments were successful in reducing the prevalence of scabies, no effect of season on the prevalence of scabies could be seen. The pretreatment prevalence of scabies at March and June 1992 was statistically similar for each of the mainstream communities, indicating that the prevalence does not differ between the wet season (March) and dry season (June). _Sarcoptes_ infection in canids is generally considered non-seasonal (Scott and Horn, 1987; Sosna and Medleau, 1992b; Scott _et al._, 1995), but Pence and colleagues (1983) did find some changes in prevalence in coyotes during autumn and spring. This was explained by the over-winter survival, during one epizootic, of adult coyotes with scabies, which subsequently infected younger naïve coyotes in the following spring. As such, the trend noticed was not an effect of season directly, rather the effect of season on the survivability of adult coyote hosts harbouring the mites.

Seasonality of infection has been observed in humans (Blumenthal _et al._, 1976; Green, 1989; Burgess, 1994) and pigs (Davies _et al._, 1991; Davies _et al._, 1996). In humans, it is generally
accepted that the incidence of scabies increases through the autumn into the winter, with a subsequent decline in spring (Burgess, 1994). Some believe this is due to the increased huddling of people during the cold months, but the same seasonality has been observed in regions with monsoon weather patterns where the ‘winter’ months are actually warmer (Burgess, 1994). Sokolova, Radchenko and Lange (1989) (cited in Burgess, 1994), found some female mites entered a latent phase during December (winter) and July (summer) when oogenesis was suspended. This was coupled with an overall reduction in the number of eggs in burrows during the late winter to early summer period.

Similar seasonal patterns are found with scabies infection in pigs, except that the spring months also have an increase in severity (Davies et al, 1991). It has not been shown that low environmental temperatures per se affect the severity of scabies in pigs (Davies et al, 1991), but high environmental temperatures (coupled with low humidity) may be responsible. Certainly, the survivability of Sarcoptes scabiei var. canis and var. hominis off the host is reduced with low relative humidity and high temperatures (Arlian et al, 1984a; Arlian et al, 1989).

7.4.1.1 Scabies Infection Dynamics

The dynamics of scabies prevalence within Looma and Kalumburu was without any trend or pattern and reflected the effectiveness of the treatments.

Pence et al (1983) documented an epizootic of scabies in coyotes in southern Texas. The dynamics of Sarcoptes in wild populations is quite different to those in capture or domestic situations. The cause of the epizootic was considered to be a combination of increased density of coyotes in the region and mutation of an existing Sarcoptes strain of low virulence. Similar patterns are found in human scabies epidemiology, with some suggestions that periodic pandemic cycling of scabies (occurring every 30 years and lasting 15 years (Green, 1989)) is a reflection of herd immunity (Church and Knowelden, 1978; Burgess, 1994). The cycles are considered a result of a steady increase in the proportion of the population becoming infected
leading to a peak incidence. After the peak is reached, the proportion of ‘immune’ people becomes so large that there is a reduction in transmission, with a subsequent significant decrease in prevalence (Burgess, 1994). The disease may then disappear for a time. Once a new susceptible population has grown up, a new cycle can begin. Green (1989) considered that this may be an oversimplification of the epidemiology of the disease, but the prospect of herd immunity may have some local effects on epidemics.

In the situation of mass chemotherapy of dogs in communities, herd immunity may also play a part in keeping the incidence of scabies low. If the dogs continue to be treated without any exposure to scabies, then introduction of infected dogs to these communities may result in an epizootic because the herd immunity is reduced.

7.4.1.2 Risk Factors for Scabies Infection

7.4.1.2.1 Age and Sex of Host as Risk Factors for Scabies Infection

No consistent findings on the age and sex predilection of scabies were found in the present study. Adult male coyotes were infected more often that other age and sex groups in Pence and colleagues’ study (1983). This is most likely a reflection of the greater contact these canids have with other coyotes and hence greater potential to become infected. In domestic situations, where there are plenty of opportunities for cross transmission due to close contact, this may not be as important in the epidemiology of the disease. Adult coyotes were also more likely to be infected than juveniles and this was possibly related to the relative time of exposure and time of development of an infection (Pence et al, 1983). Localised hyperkeratotic lesions in dogs infected for the first time, though, can develop in 6 weeks (Arlian et al, 1995).

Similar inconsistent reports on sex and age differences are documented for human infections. Most of the associations have been related to behaviour and crowding of differing groups of people (Green, 1989) rather than the physiological differences of age or sex.
7.4.1.2.2 The Effect of Household Crowding of Dogs on Scabies Infection Rates

Transmission of scabies between animals (including humans) is facilitated by close contact, which is expected to be more frequent in crowded living conditions. Correlation between household size, proportion of households affected and number of dogs infected was demonstrated in the present study. A causal association could not be proven in the present case because the larger households were more likely to have a larger number of susceptible dogs at risk. When crowded households with over 4 dogs were considered, an association was found with scabies, although it was not very strong (OR 1.7 and 1.6).

Burgess (1994) considered that the relationship between scabies and relative levels of crowding in human communities is likely to be complex and is probably confounded by other exposure risks. Although infra family spread is common, in one study, the role of the use of fomites, such as towels and linen, was a risk, whereas family size wasn't (Gulati et al, 1977). Likewise, the practice of exchanging clothes with family or friends and spending nights at friends' houses was more important than household size in a community outbreak in North America (Blumenthal et al, 1976).

Determining the relative importance of fomites versus household size was not possible in the present study, due to the difficulties in tracing dogs' activities. Nonetheless, it is assumed that some dogs will sleep in their owners' beds during the night. Likewise, the hierarchy of dogs at each household will determine which dogs are more likely to have contact with each other and it is possible that dogs do move from house to house with some frequency and thus the distribution of dogs is not stable. Despite this, prolonged close contact is said to be the most important method of disease transmission (Scott and Horn, 1987), for both dogs and humans. No association between scabies infection rates and sex or age was determined in the present study. If behavioural factors are important in canine scabies transmission, then sex and age related differences in the infection rates would be expected.
The number of dogs per household may explain the differences in initial prevalence between the coastal/central regions (average 15.5%) and the eastern region (43.3%). The coastal and central regions had 1.94 and 3.34 dogs per dog-owning-household, respectively, whereas Warmun had an average of 5.20 dogs per household.

Another possible suggestion for the differences in prevalence between regions is proximity of communities to water and frequency of washing and grooming of dogs. Dogs from the coastal communities were noted to spend much time swimming in the ocean (with the exception of Kalumburu and Beagle Bay, which are not immediately on the coast). Also, dogs from some of the central region accompanied their owners to the rivers for fishing and hunting. Personal hygiene practices and availability of water, though, have been discredited as controlling factors for infection in humans (Blumenthal et al, 1976; Ryder, Reeves, Singh, Hall, Kapikian, Gomez and Sack, 1985). Continual washing or excessive washing may increase the susceptibility of humans to scabies possibly due to the effect of soap decreasing the bacterial flora (Green, 1989). The opposite has been reported in meticulously groomed infected dogs that have minimal skin lesions (scabies 'incognito') (Scott and Horn, 1987).

Rather than access to water or grooming practices, owner awareness of scabies infection may play an important part in scabies infection rates. On several occasions, owners (particularly older people) commented that lack of hair was normal for their dogs. Owners with more contact with health providers and access to veterinary advice are possibly more educated on the disease, its transmission and control and are less likely to tolerate infected animals. This may explain why very few infected dogs were introduced to the communities after the commencement of the program (see Tables 7.7-7.8).

The widespread distribution of scabies at some communities is of some concern for control of zoonotic transmission. At Looma, Warmun and Kununurra, between 62 and 76% of households had dogs with scabies. This places the human occupants at risk if they are in contact
with infected dogs, particularly if the dogs share their sleeping places. Again, the household epidemiology of scabies is complex and dependent on many factors apart from mere household size.

The wild coyote epizootic of southern Texas was facilitated by movement of animals radiating out from a central focus of infection (Pence et al, 1983). In this case, there was a minimum threshold density necessary for transmission. This is not so important in the present study, despite the seemingly semi-wild characteristics of the community dog populations.

7.4.1.3 Effect of Scabies Control Program on Dog Health Parameters

7.4.1.3.1 Weight Changes

As very few dogs became infected during the program it was not possible to analyse the data for an association between weight loss and infection rate in most communities. At Looma (where some dogs did become infected), such an association was demonstrated. Weight gain, on the other hand, if associated with loss of infection, should have been demonstrable as many dogs did lose their infections. No association between scabies prevalence and weight gain was shown.

Chronic infestations with scabies mites can cause progressive deterioration of the host’s health which is evident by weight loss (Pence et al, 1983; Scott and Horn, 1987), reduced weight gain and increased food intake (in pigs, Arends, Stanislaw and Gerdon, 1990). In laboratory experiments with heavily parasitised rabbits, loss of condition and failure to gain weight were not specifically due to energy loss resulting from ingestion of host skin and serous fluids by the mites (Artlian et al, 1988). Host responses such as hyperplasia and sloughing of the stratum corneum attribute to indirect energy loss and resultant loss of weight. Increased scratching may also lead to energy depletion (Burgess, 1994).
7.4.2 Hookworm

Most studies into the biology, ecology and control of hookworm have centred around the effects of hookworm on human communities. As such, field studies, similar to the present study, have been on *Ancylostoma duodenale* or *Necator americanus*, two human hookworm species that show some differences to *A. caninum* and each other (such as route of transmission, duration of infection, pre-patent period, fecundity and blood consumption) (see Appendix A). Despite this, most of the knowledge about the host-parasite relationships of hookworms is based on *A. caninum* in dogs (Behnke, 1990).

7.4.2.1 Prevalence of Hookworm during the Ivermectin Treatment Program

7.4.2.1.1 Prevalence of Hookworm in All Dogs from Each Region

The data reflected the changes in infection status of the population in general and demonstrated the effect of seasonality, which overrode whether or not individuals were treated. For the coastal and central regions, the initial pretreatment prevalence was regained for the two wet season months (March 1993 and 1994) but not for the dry months. The eastern region was similar except that the March 1994 prevalence was similar to the dry months.

Most studies investigating reinfection with hookworms (mostly *Necator americanus*) in people after treatment have not taken season into consideration (Haswell-Elkins *et al.*, 1988; Bradley *et al.*, 1993; Quinnell, Slater, Tighe, Walsh, Keymer and Pritchard, 1993), but in those studies that have investigated seasonality, without treatment of the host, most have found that transmission of infection occurs during the wet season (Nwosu and Anya, 1980; Schad *et al.*, 1973; Hominick *et al.*, 1987).

In Nigeria, where most human hookworm infections are due to *N. americanus*, Nwosu and Anya (1980) found hookworm prevalence to vary with season. During the wet or monsoon season, the hookworm prevalence was 78.1% one year, followed by 67.6% in the dry and 81.0% in the next wet season. Intensity of hookworm infection, as indicated by egg output, also varied
with increased burden in the wet months. Recovery of infective larvae from defaecation sites during the dry season was so low that effective transmission was zero at this time of the year.

Croese (1995), in investigating suspected seasonality in cases of eosinophilic enteritis in humans after infection with *A. caninum*, reported that infected dogs produced less ova during the winter months (dry season). This conclusion was largely deduced from a previous hookworm prevalence survey in Brisbane (Prociv *et al*, 1994), where lower prevalences (rather than intensity or numbers of eggs per gram) were detected in winter. Similar observations were made in the present study and further support the evidence for variation in prevalence of canine hookworm, similar to human hookworm, throughout the year.

In West Bengal, where weather patterns are similar to the Kimberley (a wet and dry season), transmission of human hookworm only occurs during the wet season (Hominick *et al*, 1987). Furthermore, large aggregations of hookworm larvae in the soil are encountered earlier rather than later in the rainy season. Contrary to this, Schad *et al* (1973), using rates of conversion from negative to positive *A. duodenale* infection status for Bengali children, found transmission to be maximal at the end of the monsoon season. Further studies into the effect of alternate wetting and drying of the soil, at the break of the wet season, on the survivability of larvae at defaecation sites (Hominick *et al*, 1987) is necessary to resolve when maximum transmission occurs during the wet season. In either case, transmission of hookworm did not occur during the dry season. In the Kimberley, the high prevalence of infection of dogs in March, which is toward the end of the rainy season, supports the argument for increased transmission of hookworm through the wet, although data from Kalumburu and Looma do not show any increase (see 7.4.2.1.3). The ability of *Ancylostoma caninum* to be transmitted transcolostally further complicates the epidemiological picture, as this type of transmission is not dependent on season.
Not all bimodal rainfall regions show association between season and prevalence or intensity of hookworm infection. In Kenya, no seasonal pattern was found for *Necator americanus* in lactating mothers (Griffin, 1981). This may be due to the greater longevity of this species (780 weeks (Behnke, 1990)) which ensures survival of the adults in the host throughout the dry seasons and hence less dependence on environmental hookworm stages for maintenance of infection. In *A. caninum*, adults only survive up to 100 weeks (Behnke, 1990), which means that aged worms need to be regularly replaced by larvae to maintain the numbers. Even so, in the present study, longevity is irrelevant as L5 and adult worms were removed by the ivermectin treatment and would be expected to have been replaced with environmental larvae or hypobiotic larvae (if this is a feature of the hookworm population) when conditions were suitable.

Interestingly, the prevalence of hookworm at Kalumburu did not exhibit any significant seasonal pattern. Sampling was conducted 3 monthly over an 18 month period. There was no statistical association between the same months of each year, despite similar weather patterns for 1993 and 1994 (see Figure 7.18). This may be due to microclimatic factors, such as watering of lawns and shading around houses, ensuring egg and L3 survival in the environment throughout the year, irrespective of the macroclimate.

The effect of repeated cycles of mass treatment was to reduce the average prevalence (at each region) from 65.5% to 22.6% at the end of the study in September 1994. Although the effects on intensity would have been more dramatic than on prevalence (Anderson and May, 1982), the reduction achieved by 3 monthly treatments shows the effect of a cumulative reduction in the rate of reinfection. This is possibly due to a reduced rate of environmental contamination with infective stages (Bundy *et al*, 1990).

Eradication of hookworm was not practical in the circumstances of the present study. To eradicate hookworm from the population, the proportion of hosts treated per unit of time must be greater than a critical value related to the basic reproductive rate and degree of aggregation of
the worms within the host population (Anderson and May, 1982). The treatments must also be continued for a period of time greater than the maximum life span of the adult worm (up to 100 weeks, Behnke (1990)) to ensure that those hosts that are not treated are still not contaminating the environment by the pre-existing hookworm infection (Anderson and May, 1982). If treatment ceases before the average worm burden is below the breakpoint (which is close to zero worms per host), the parasite population will rapidly return to its pre-control level (Anderson and May, 1982). Hence the importance of continual treatment in a mass chemotherapy campaign.

Seasonal fluctuations in the prevalence of hookworm are considered to be of limited significance to the long term stability of the parasitic population in either treated or untreated animals (Anderson and May, 1982). This is because the lifespan of the longest lived stage of hookworm (the adults) is greater than one year. Only if the reproductive rate approaches zero and the life span is markedly less than one year, will the endemicity of the parasite be challenged. Although the seasonal changes may result in the reproductive rate falling below unity at certain times of the year (e.g. during the dry season), if the average remains above this, then the parasites will survive (Anderson, 1982). To capitalize on this factor, control plans need to be applied intensively at times of the year when the reproductive rate is at its minimum (see 7.5).

Limiting the degree of environmental contamination by zoonotic hookworm eggs was a major objective of the program. By reducing the number of dogs shedding eggs (prevalence) as well as the number of eggs shed by each host (intensity), potential for human infection by zoonotic infective stages is reduced. Selective treatment of high risk groups (see 7.4.2.2) may achieve this, as the worms are probably highly clumped in their distribution in the host population (Anderson and May, 1982), but the effect on overall prevalence in the communities would be very slow.
7.4.2.1.2 Prevalence of Hookworm in Treated and Non-treated Dogs.

Epidemiological studies of helminth control in humans often report the prevalence and intensity of infection but rarely report the compliance rates for treatment (Nokes and Bundy, 1993). Generally it is accepted that individuals that miss treatment or sampling are a random portion of the population and that their absenteeism does not affect the representative nature of the sample (Nokes and Bundy, 1993). If basic epidemiological data relating to the helminth population is available, then mathematical models can be used to determine the efficacy of the control program with a given compliance rate (Anderson and May, 1982). Even so, in humans, individuals that do not comply with surveys may not be random and may represent individuals in which hookworms are aggregated.

In the present study, potential biases in determining treatment efficacy were reduced by examining data from dogs that were treated and not treated during each visit (3 months) before sampling. Generally, the pattern of prevalence of hookworm in dogs that were treated and those that weren't revealed a seasonal trend with increases in hookworm infection during the wet season months. An exception to this was in the central region. Examination of prevalence in dogs that did not receive treatment (whether new to the program or absent), exhibited hookworm infection rates statistically similar to the pretreatment prevalence (June 1992). Examination of prevalence in dogs that did not receive treatment gives some approximation of the natural prevalence expected without a treatment program, although an overall reduction in infective stages as a 'spill-over' from treatment of other dogs is a possibility.

Sometimes the prevalence values obtained for non-treated dogs were similar to the treated dogs. This may be due to the effect of season on the overall prevalence or there may have been some 'spill-over' effect from treating dogs that had resulted in less infective stages in the environment and hence reduced prevalence in untreated dogs.
In treated dogs in the present study, the seasonality, with increased prevalence toward the end of the wet season, was a reflection of the availability of larvae according to the climate. It is highly unlikely that it was due to hypobiotic larvae reactivating after treatment as reactivation is not usually triggered by loss of adults (in a related species; A. ceylanicum, Carroll and Grove, 1985), and the prevalence was not significantly higher in the pre-wet season (December samplings) as would be expected with this phenomenon (Nawalinski, Schad and Chowdhury, 1978b).

The data in the present study do not distinguish between dogs that originally had infections and then became reinfected and dogs that were uninfected before treatment, so it is difficult to determine the effect of resistance to reinfection. Some of the dogs that did become infected three months after treatment must have been infected before treatment. This would indicate that complete immunity to reinfection was not occurring.

Miller (1971) states that immunity to infection is not an appropriate term as “complete resistance to the establishment and development of all larvae from challenge infections is a rare phenomenon”. Indeed, this is the case in the present study, which has shown that some re-establishment must have occurred. ‘Resistance to reinfection’ is more appropriate as there is some reinfection, but possibly at a lower intensity. In original studies into vaccination against hookworm, challenge infections resulted in a reduction in the morbidity and/or mortality of hosts and a reduction in establishment of worm populations (Miller, 1971). The output of hookworm eggs was also diminished in some cases (Miller, 1971). Vaccination trials with normal hookworm larvae (rather than attenuated or irradiated larvae) required at least 100 days (approximately 3 months) between vaccination and administration of the challenge larvae to allow for the development of a satisfactory resistance.
7.4.2.1.3 Hookworm Infection Dynamics at Looma and Kalumburu

Conversions from positive to negative hookworm status at Looma and Kalumburu occurred at a greater rate during the dry season than the wet season, even though compliance rates for treatment remained the same for both seasons. Conversions from negative to positive, though, were essentially the same for all months, excluding 3 dry season months at Looma.

Both Looma and Kalumburu showed little seasonality in overall prevalence and in the prevalence of treated and non-treated dogs compared with data from the three regions. The trends for conversion rates are in accordance to this.

During the dry season, dogs that lose their infection (either due to treatments or natural attrition) are less likely to regain infection over the three months between testing. This is probably due to the hot dry climatic conditions adversely affecting the survival of L3 infective larvae. In the wet season, no increase in transmissibility (positive conversion, see Figure 7.25-7.26) was evidenced in the present study. In another study, an increase in transmissibility of human hookworm was noted for the monsoon months (Hominick et al, 1987), but in that study population, the people were not receiving treatments. The overall effect of the treatment at these two communities in the present study was a reduction of available larvae during the wet season, due to treatments of dogs before the commencement of increased rainfall. Since weather conditions during the wet were appropriate for transmission and survival of larvae, the reduced transmission is likely to be due to reduced larval numbers. If hypobiosis is a feature of this strain of hookworm, then increased conversions would be expected before the wet season (Schad et al, 1973).

At all testing periods, transmission of infection was possible. In several human hookworm (A. duodenale and N. americanus) studies, as mentioned, transmission is reported to be restricted to the rainy season (Schad et al, 1973) and completely stopped during the dry season (Hominick et
al., 1987). In the present study, microenvironmental factors may be acting to preserve hookworm larvae during the dry periods (see 7.4.2.3.2.1).

7.4.2.2 Risk Factors for Hookworm Infection

Predisposition to geohelminths is largely a result of ecological, genetic, social or behavioural factors acting singly or in combination (Schad and Anderson, 1985).

7.4.2.2.1 Sex of Host

Male dogs at the mainstream communities were found to be 1.5 times more likely to be infected than female dogs. Unfortunately, there was inadequate data to analyse the sex predilection for each age category.

Kirkpatrick (1988) and Hoskins et al (1982) also found male sex to be a risk factor for canine hookworm parasitism. Males have also been shown to have greater susceptibility to A. caninum in experimental infections (Miller, 1965; Miller, 1971; Hoskins et al, 1982). Likewise, in surveys of human hookworm infections, males have been found to be more often and more heavily infected than females (Nawalinski, Schad and Chowdhury, 1978a; Asaolu et al, 1992).

The number of surgically sterilised animals in the present survey was too low to examine the difference between intact and neutered dogs. Spayed females in other studies have been found to be less likely parasitised with hookworms than intact females. Visco et al (1977) attributes this to the likelihood of spayed females being better-cared for and less inclined to roam.

7.4.2.2.1.1 Sex Dependency in Predisposition to Hookworm Infection after Ivermectin Treatment at Looma

Reinfection with hookworm following anthelmintic treatment of people has been found to be influenced by the sex of the host (Haswell-Elkins et al, 1988). Both prevalence and intensity of infection after treatment was greatest in females in a community in South India, although the burden was only 28% of pretreatment level, 11 months after treatment (Haswell-Elkins et al,
1988). A strong correlation was also demonstrated between initial burden and reinfection burden, which has been demonstrated elsewhere (Schad and Anderson, 1985; Upatham et al, 1992; Quinnell et al, 1993).

In the present study, male dogs examined at Looma were significantly more likely to regain hookworm infection after treatment or to develop new infection than females (see Table 7.21). In humans, defaecation behaviour, including choice of site and exposure to contaminated areas with viable hookworm larvae, have been found to be important in acquisition of hookworm and these are in turn dependent on the activities of each gender. In dogs, it may be that male dogs are more likely to roam over greater areas, thereby increasing their chances of acquiring hookworm.

Although behaviour of each sex is important in acquisition of infection, hormonal, immunological and mechanical factors may also play a part in limiting infections in females (Nawalinski et al, 1978a). In laboratory studies, where many behavioural factors are controlled for, resistance to *A. caninum* infection is greatest in female dogs and is age dependent with resistance increasing after 8 months of age (Miller, 1971). The earlier and stronger development of immunological resistance in females compared to males may be due to the sequestration of dormant larvae in the tissues of the bitch in anticipation of prenatal-colostral transfer to puppies (Miller, 1971).

The infection rate in pregnant bitches would be expected to be higher than in non-pregnant bitches because of the reactivation of these larvae. Also, pregnant bitches may be more prone to infection by hookworm larvae because of the depression of the immune response that occurs during pregnancy and lactation (Griffin, 1981). This sector of the population is particularly important in the transmission and epidemiology of the disease because of the transcolostral transfer of larvae. In the present study, reproduction was controlled such that very few pregnant bitches were sampled during the survey.
7.4.2.2.2 Age of Host

For most of the communities in the present study, when statistical significance was demonstrated, puppies were found to be more commonly infected than adults.

In laboratory studies, investigating the establishment of adult worms in dogs, similar findings of susceptibility in puppies have been found, with natural age resistance to infection occurring by 11 months of age (Miller, 1965; Behnke, 1990). The mechanism of age resistance has not been analysed in any detail (Kirkpatrick, 1988) and the site of expression of resistance remains unknown (Behnke, 1990).

Field-based studies have also found evidence of reduced infection rates in older animals. Prociv et al (1994) found hookworm infections to be less common in dogs over 3 years of age and Visco et al (1977) found the same in dogs over 6 month of age. Extrapolated data from Visco et al (1977), showed dogs under 1 year of age to be 1.4 times more likely to have hookworm than those over (P=0.002). With each of these field studies, though, it is difficult to ascertain how much of the observed ‘resistance’ in older groups is due to age per se and how much is due to prior exposure to infection with *A. caninum*.

Kirkpatrick (1988), however, found pet dogs in Pennsylvania had a significantly higher prevalence of hookworm in those greater than or equal to 2 years of age. Boreham and Capon (1982) also found hookworm ova more often from stray dogs over one year of age compared with younger dogs. Older dogs suffering from chronic hookworm disease have been demonstrated to have a reduced resistance to reinfection (Kelly et al, 1976) which may explain the increase in prevalence in older stray dogs which are more likely to have sustained chronic hookworm infections than young dogs.

Other authors believe that hookworm infections are sustained in the older populations, long after transmammary infection, due to the opportunity for infection by cutaneous or oral routes (Visco
et al, 1977; Boreham and Capon, 1982; Hoskins et al, 1982). As has been shown by the seasonal prevalence data, there is abundant opportunity for continual reinfection in the Kimberley communities.

7.4.2.2.1 Age Dependency in Predisposition to Hookworm Infection after Ivermectin Treatment at Looma

Predisposition to helminth infection in humans has been demonstrated to be age-dependent with children and adolescents having higher rates and intensity of reinfection after treatment (Bradley and Chndiwana, 1990). Infections with hookworms in humans have also shown the reverse, with adults, who usually have higher initial burdens (Haswell et al, 1988), being predisposed to reinfection (Haswell-Elkins et al, 1988; Bradley and Chndiwana, 1990; Bradley et al, 1993).

In the present study, prevalence data did not show any age predisposition to hookworm reinfection for the dogs at Looma, which is similar to findings by Quinnell et al (1993) in New Guinea human populations. Examination of intensity of hookworm infection in Quinnell et al’s (1993) study, showed some correlation between initial burdens and post-treatment burdens, as has been demonstrated by Bradley and Chndiwana (1990) in Zimbabwe. Less heavily infected, younger hosts were not as predisposed to infection as older, more heavily infected people (Bradley and Chndiwana, 1990). As older people have had greater exposure to hookworm and hence opportunity for developing immunity, this relationship shows that acquisition of immunity is not as important as the behavioural factors which influence the degree of exposure to areas contaminated with infected larvae (Bradley and Chndiwana, 1990). Hominick et al (1987) also found that heavily infected individuals were not necessarily those who were the greatest source of infection for others because of the differences in sociobehavioural factors in exposing individuals to infective larvae.

Lack of age predisposition to reinfection in dogs in the present study may reflect age-independent exposure to infective stages or an increase in both exposure and immunity with age (Quinnell et al, 1993).
In hookworm control programs for people, child targeting is less effective than targeting adults for control of hookworm infections (Bundy et al., 1990) because hookworm infections are generally more prevalent and intense in adults than children (Bradley, Chandiwana, Bundy and Medley, 1992; Chan et al., 1994). The data presented here, though, suggests that targeting young dogs, males and pregnant or lactating females could contribute to overall control of infection. This, coupled with a possible ‘spill-over’ of effect to other groups, may result in less necessity to treat all dogs.

7.4.2.2.3 The Effect of Household Crowding on Infection Rates

Crowding of dogs at each household did not result in a greater risk for infection. In fact, in the central region, dogs belonging to single dog households were at a greater risk of infection with hookworm than those living with other dogs. Although the dogs are attached to specific households, they probably do not aggregate at the households to which they belong, and defaecation is likely to occur elsewhere. This indicates that when attempting to reduce worm egg loads in the environment, reducing the intensity of infection in heavily burdened dogs may help the entire community and not just the occupants of the households to which the infected dogs are attached. Density dependent depression of fecundity is a feature of *A. caninum* (Miller, 1971; Anderson and Schad, 1985), so control of dogs with high egg output is more important than controlling dogs with high burdens in this case.

7.4.2.3 Environmental Factors Affecting Hookworm Infection Rates

The most important environmental factors that influence larval survival are relative humidity, temperature and exposure to direct sunshine (Smith, 1990).

7.4.2.3.1 Rainfall

A strong positive correlation between hookworm prevalence and rainfall in the months leading up to infection was demonstrated for the coastal region. The coastal region was also the region that showed the greatest seasonality in prevalence (as determined by the differences in
prevalence for wet and dry seasons). Rainfall, it appears, is a very important determinant in prevalence in both treated and non-treated animals.

Miller (1970) found rainfall to be the limiting factor to incidence of *Necator americanus* infection when correlating incidence against rainfall and minimum and maximum temperatures for human samples from East Africa. A field study by Nwosu and Anya (1980) in Nigeria supported these findings with a significant correlation between a wetness index (derived from the product of monthly precipitation and number of rainy days) and a measure of infection pressure (based on the availability of *N. americanus* larvae during the sampling period). Similarly, Hominick *et al* (1987) considers moisture to be the most important factor in larval development and survival, both as rainfall and soil moisture content.

The results of the above field studies may be due to an increase in larval survival at recognised transmission foci (where humans defaecate) or an increase in the number of sites that are suitable for larval development (Smith, 1990). Behavioural change in human activity as a result of the variations in weather is also a factor (Smith, 1990), but this is less likely to be the case when investigating field canine infections.

Becker *et al*, (1977) demonstrated a strong climatic dependency for the rate of new hookworm infections when looking at well cared for dogs at a military facility in Missouri. Despite the study not addressing potential confounding factors (such as altered microclimate from lawn watering), there still was a strong association of hookworm infection rates and rainfall. Dogs that had been treated with anthelmintics were excluded from the study, so comparisons with rates of reinfection and climate are not available.

Despite the importance of rainfall, no correlation between rainfall and hookworm prevalence was found for the other regions of the Kimberley that did not show as defined seasonality (as determined by the changes in prevalence throughout the program).
7.4.2.3.2 Temperature

As the minimum daily temperatures for the Kimberley are above the temperatures required to restrict development of hookworm larvae (10°C), temperature did not correlate with prevalence of infection. In the dry ‘build-up’ months prior to the rainy season, soil temperatures could exceed those in which larvae can survive (>37°C, Dunsmore and Shaw, 1990) which should result in a break in transmission. As already noted, this did not occur for Kalumburu and Looma, which allowed transmission, albeit at a lower rate, to continue throughout the dry seasons (see 7.4.2.1.3).

Becker et al (1977) found temperature variables affected the rate of new hookworm infection in Missouri. This is to be expected, as Missouri temperatures occasionally fall below the threshold for maintenance of hookworm enzyme activity and infectivity.

7.4.2.3.2.1 Microenvironmental Factors for Hookworm Infection

The microenvironmental factors that influence the survivability of hookworm larvae relate to the major determinants of survival, namely humidity and temperature. These factors include vegetative cover, degree of shading and soil type.

It has been suggested that higher than expected prevalence for relatively dry regions (and hence dry seasons) of the Kimberley is due, in part, to the humidity provided by the watering of lawns where dogs defaecate (Thompson et al, 1993a). Dog faeces was noticed on lawns as well as non-grassed areas throughout the communities, but it is probable that infected faeces on lawns are the main source of infection because of the protection from direct sunlight, when the eggs dissipate under the lawn, and the moisture from watering. In addition to protection from sunlight, up to 80% of the solar radiant flux density may be absorbed by the upper layers of vegetation and at night, heat losses from radiation and convection are reduced by the vegetation covering (Smith, 1990). Despite this, a definite seasonal pattern, irrespective of defaecation
site, was shown at some regions. Likewise, hookworm was still prevalent at areas where there is little lawn, such as at Yaramun in the eastern region.

Shaded areas are also considered to be more conducive to hookworm survival (Schad and Anderson, 1985; Croese, 1995). Studies elsewhere, though, have suggested that dung beetles, through providing eggs and larvae in faecal matter with a means for escaping lethal temperatures on the soil surface, are more important in hookworm survival (Hominick et al, 1987).

Other factors which affect hookworm development include the soil type, which should be loose and friable to promote larval survival (Hominick et al, 1987). In most communities in the present study, the soils are generally compacted clay, which allows water to build up and wash away infective stages.

7.4.2.4 Effect of Hookworm Control Program on Dog Health Parameters

Bradley et al (1993) considered that a significant reduction in prevalence of hookworm in people resulting from a mass chemotherapy campaign indicates immediate short-term improvement in human community health. Certainly in the present study, basic indicators of health status in dogs in communities show that ivermectin treatment did result in improved health (see 6.4.6).

Weight changes for each individual animal were determined rather than finding the average weight for the whole community before and during treatments. This was because of the great variation in dog sizes and inability to correlate size with age as can be done with humans. This method, though, was less than satisfactory because, apart from the errors of weight measurement in the field, the proportion of weight change according to average weight for each breed or type could not be determined. Needless to say, a weight change of one kilogram for a large dog is less significant than for a small dog. The weight data can not be correlated with the
intensity of infection, because only prevalence was recorded (see 3.3.7.2.1.1). It is likely that as the treatments continued, the intensity of infection would have decreased even if the prevalence of infection had remained high (Anderson and May, 1982). Morbidity has been found to be more closely related to the intensity of infection than prevalence (Miller, 1971).

Hookworm infections, in human field trials and laboratory studies of animal models, have been linked with an alteration in nutritional status (Crompton, Walters and Arnold, 1981; Crompton and Stephenson, 1990) and hence body weight (Carswell et al, 1981). Hookworm infections have been suggested to depress growth by decreasing both food intake (Crompton et al, 1981) and the efficiency of nutrient utilisation (Stephenson et al, 1989). Nutrients are also excreted through blood loss, vomiting and diarrhoea (Crompton and Stephenson, 1990). Although gut parasites are more likely to produce malnutrition, gut parasites are also more likely to parasitise the malnourished (Carswell et al, 1981) and cause worse effects than they would in well fed individuals (Crompton et al, 1981).

The overall clinical effect of hookworm disease in dogs is dependent on the intensity of infection, nutritional status and iron reserves (Miller, 1971). The intensity of infection, in turn, may be related to the age of the host and the presence of acquired resistance.

7.4.2.4.1 Packed Cell Volume Measurements

Measurement of Packed Cell Volume (PCV) gives a basic indication of anaemia status of an animal, if the animal is adequately hydrated. Dogs in the hot climate of the Kimberley were likely to be dehydrated resulting in low blood volumes with a spurious increase in PCV. Likewise, spurious polycythaemia is possible in stressed animals that undergo splenic contraction (Duncan and Prasse, 1986). As anaemic packed cell volumes were more common than polycythaemic packed cell volumes, there either was no error associated with dehydration and stress, or the actual number of dogs with anaemia and the extent of the anaemia was underestimated. Total serum protein levels for the sampled dogs revealed that 54% of the dogs
were hyperproteinaemic, although only 17% of those dogs were polycythaemic according to PCV readings. It is likely that the hyperproteinaemia may not be due to dehydration alone and may be due to hyperglobulinaemia, which was common amongst the population tested at Kununurra (see 5.4.6.4). Albumin and globulin ratios were not determined, so the true extent of dehydration amongst these dogs is unknown. In either case, adults, puppies and juvenile age groups all showed association between hookworm infection and decreased PCV.

The primary pathogenic mechanism in the induction of hookworm anaemia in dogs and humans is intestinal haemorrhage from the feeding habits and activities of the immature and mature hookworms (Miller, 1966; Miller, 1968; Miller, 1971; Crompton and Stephenson, 1990). Initially the anaemia is acute haemorrhagic with normochromic and normocytic erythrocytes. As the animal becomes iron deficient with chronic infection, the erythrocytes show increasing microcytosis and hypochromia (Miller, 1971). Infection with at least 10,000 larvae is necessary to produce marked microcytic anaemia in adults (Carroll and Grove, 1985).

The effect of treatment on the health of the dog population extends beyond an immediate post treatment improvement as dogs that later become reinfected are likely to be resistant to reinfection, and hence are expected to suffer lower intensity of hookworm infection (Miller, 1971).

7.4.3 Roundworm

7.4.3.1 Effect of Ivermectin Treatment on Toxocara canis Prevalence

Dogs with artificial infections with Toxocara canis can be cleared of infection with ivermectin treatment doses of greater than 50μg/kg subcutaneously (Yazwinski et al, 1982). In the present field situation when there were sufficient numbers of infected dogs for analysis, treatment of puppies or adults did not significantly affect infection rates at Kalumburu.
It is possible that reinfection after treatment occurred from roundworm eggs persistent in the environment. As most of the infections were isolated to a few households with puppies, the environments at these locations would have been heavily infected. A study of dog breeding establishments in Perth found 2 of 5 kennels to have *T. canis* eggs in every soil sample taken (Dunsmore *et al.*, 1984). This is in contrast to the general public areas (such as beaches and parks) of Perth, which did not have any eggs recovered from any soil samples (Dunsmore *et al.*, 1984). The distribution of *Toxocara* eggs, thus, seems to be restricted to where there are many puppies and conducive environmental conditions such as at breeding households. In addition to this, if any puppies had not been treated, they would have served as a reservoir for future infection of the other housemates.

In addition to environmental contamination, any puppies that are still suckling are capable of developing patent infection after treatment and hence contributing to the overall infection rates. Milk transmission can occur continuously for up to 3-4 weeks post-partum (Lloyd, 1986). Hence, if puppies are treated within this 3-4 week period, they are still receiving *Toxocara* larvae from the dam that are capable of maturation, regardless of the treatment killing any adult roundworms in the puppies’ intestines. Larvae that reach the intestine need at least 2 weeks to mature and start passing eggs, so treatment is recommended every two weeks to kill the continual supply of worms during the suckling period.

Ivermectin needs to be given at large doses, frequently, to affect somatically arrested larvae of bitches. Even so, egg output is still high in the offspring (Overgaauw, 1997), which contributes considerably to the contamination of their environment. In a control program with treatment every 3 months, prenatal infection could not be controlled because the dams would retain their arrested larvae up to the last trimester. Even when bitches are treated before mating and two weeks before anticipated whelping date, with approved anthelmintics at recommended doses, there is no useful effect on prenatal transmission (Fisher, Jacobs, Hutchinson and Dick, 1994).
Environmental contamination can be controlled by ensuring treatment of puppies and adult male dogs, which contribute significant numbers of eggs to the environment (Overgaauw, 1997). Puppies in particular are capable of infecting bitches that ingest the faeces of young pups that have larvae passed in the faeces (Lloyd, 1986). This in turn results in infection of the next litter of puppies.

The results of the present study show the importance of puppies in the epidemiology of *Toxocara canis* infection. Although only a few dogs were infected, the likelihood of heavy contamination of the household environment by puppies with small children living at many households suggests a potential for zoonotic infection. Surveys by the State Health Laboratory Services of Western Australia in November 1990 found only 4% of people from the central region communities (n=98) to be seropositive to *Toxocara*. This figure is much lower than the annual rate of 13.6% for the general Western Australian population (Thompson et al, 1993a). It is not known whether there is a higher seroprevalence in children of the coastal region.

In the Turkana district of Kenya, where the nomadic people show close attachment to their dogs, the serological evidence (using an ELISA test) of infection in people is about 7.5% (Kenny et al, 1995). By comparison, studies of the canine parasites of the district have not shown any evidence of *T. canis*, although it is likely that very few puppies were tested in the study (Kenny et al, 1995). The clinical significance of humans showing evidence of exposure to *Toxocara* needs to be established for both the Turkana district and the people of the present study.

**7.4.3.2 Risk Factors for Toxocara canis Infection**

**7.4.3.2.1 Age and Sex of Host**

The finding of *T. canis* infection being almost exclusive to puppies in the present study is in accordance with the accepted effect of age-related resistance to infection (Greve, 1971). Extrapolated data from stray dogs from Brisbane shows that dogs under one year of age were 10
times more likely to have infection than their older counterparts (P<0.00001) (Boreham and Capon, 1982). Similarly, dogs less than 6 months old from Sydney had a prevalence 3 times greater than those over 6 months (Kelly and Ng, 1975, cited in Dunsmore and Shaw, 1990). Studies overseas have also found younger dogs to be more likely to be infected (Seah et al, 1975; Visco et al, 1977; Kirkpatrick, 1988).

Most infections with *Toxocara canis* are acquired prenatally or immediately postnatally from the dams (Burke and Roberson, 1983). This in part explains the higher prevalence in younger dogs. The larvae in the livers of prenatally infected pups undergo tracheal migration before reappearing in the intestine to grow to adulthood (Dunsmore and Shaw, 1990; Abbott and Dent, 1998). In weaned dogs (older than 6 weeks), fewer and fewer parasites are able to complete this migration. The larvae, instead, undergo somatic migration and become ‘trapped’ in granulomas in various tissues where they do not develop beyond the second larval stage. This ‘age resistance’ is not dependent on previous exposure to *Toxocara* (Greve, 1971). Acquired resistance from previous exposure, though, is a significant contribution to this phenomenon. The mechanism of acquired immunity in mature dogs may operate partly within the lungs, perhaps as a delayed-type hypersensitivity response against the infective stage (Overgaauw, 1997).

As demonstrated in the present study, a proportion of the adult population of dogs can also be infected. Adult dogs may harbour *Toxocara* worms as a result of suppressed immune response, ingestion of low numbers of infective eggs (Dubey, 1978), or following ingestion of infected paratenic hosts (Warren, 1969). Maizels and Meghji (1984) also describe a situation in which adult dogs are fully susceptible, despite repeated egg exposure and development of antibodies. This, though, may be due to the exposure of the experimental dogs to low doses of *T. canis* eggs. In the Kimberley, where environmental conditions are difficult for *Toxocara* stages to survive, it is likely that most dogs are only exposed to low doses in any case. This may explain why infections were found in adults of both sexes.
Although no sex predilection was demonstrated in the present study (even when the effects of age were controlled), this is unusual as patent *T. canis* infection tends to be found more often in adult male dogs than female dogs, excluding post weaning bitches (Dunsmore and Shaw, 1990). It seems that the age resistance in males is weaker than females.

Adult females are capable of developing patent infections during the peri-parturient period when the resistance of the bitch is diminished and exposure to *T. canis* larvae passed out by their puppies is high (Lloyd, 1986). The duration of infection, though, is usually only a few weeks (Dunsmore and Shaw, 1990).

### 7.4.4 Other Parasites and Bacteria

#### 7.4.4.1 *Giardia duodenalis*

##### 7.4.4.1.1 Risk Factors for *Giardia duodenalis* Infection

**Age and Sex of Host**

Puppies younger than 6 months of age were found to be at greater risk of acquiring *Giardia* than juveniles or adults in the present study. Swan and Thompson (1986) also found *Giardia* in dogs to be more common among the younger animals and suggested that this may be due to either the development of acquired resistance with exposure to *Giardia* (Hoskins et al, 1982) or decreased susceptibility with age. Kirkpatrick (1988) and Hoskins *et al* (1982) also found *Giardia* most often in dogs less than 2 and 0.5 years of age, respectively. Likewise, older animals may be more susceptible if their natural resistance is compromised such as from a low plane of nutrition, stress or impending parturition or lactation.

The sex of host did not appear to affect *Giardia* infection in the present study. This has also been reported elsewhere (Swan and Thompson, 1986).
7.4.4.1.2 The Effect of Household Crowding of Dogs on Infection Rates

Surveys of dogs housed in enclosed spaces with other dogs have revealed higher infection rates for these dogs compared with others in less crowded conditions (Swan and Thompson, 1986; Savini et al, 1993). This is generally because *Giardia* is faecal-orally spread, which is facilitated by close contact of susceptible hosts. Other factors such as possible stress and low plane of nutrition compound the effect of crowding (Swan and Thompson, 1986). In the present study (for the central region), dogs living in high density situations were more likely to be infected with *Giardia* than those living with fewer dogs. Crowding is likely to be a risk factor if it is coupled with poor domestic hygiene (Savini et al, 1993) that facilitates dogs coming in contact with infected faeces. Owners in households that support many dogs may also have less to feed their dogs and the stress of many dogs living together may also play a role. Intra-household transmission is also greater if there are many young dogs in the household.

Regardless of the prevalence and risk factors associated with *Giardia* in communities, if the genotype of *Giardia* from this area is zoonotic (or if dogs are able to act as reservoirs for human ‘strains’), the degree of transmission to humans is dependent on the contact and hygiene practices of humans. Dogs have the potential to contract human ‘strains’ and possibly act as reservoirs due to coprophagy of human faeces, which was frequently observed. Since *Giardia* is transferred by faecal-oral route, children who play intimately with puppies (especially those with diarrhoea) are at a greater risk of acquiring infection.

7.4.4.2 *Dirofilaria immitis*

Interestingly, no statistical difference in microfilariae diagnosis was found between dogs that had been treated and those that hadn’t, despite the known sensitivity of microfilariae to ivermectin treatment doses higher than 50μg/kg (Campbell and Benz, 1984). Despite this, the prevalence of microfilariae positive dogs had decreased significantly after several treatments over several months. It is likely that the untreated dogs that were tested were younger than the treated counterparts, and therefore were less likely to have heartworm. Also, the effect of the
treatment on those dogs that were infected would have resulted in reduced microfilaraemia, if not complete removal.

Although the treatments remove the circulating microfilariae within 24 hours, the established adult worms are able to resume production of microfilariae. Hence, complete elimination of microfilariae does not occur without concurrent removal of the adults (Campbell and Benz, 1984). This also accounts for the apparent non-association of treatment and removal of microfilariae, as indicated by only 8% of dogs becoming negative 6 months after the initial survey.

The long-term effect of treatment with ivermectin is likely to be a reduction in the reservoir of microfilaraemic dogs and hence heartworm infection in the communities. It is still unknown exactly at which age D. immitis is completely refractory to ivermectin prophylaxis (McCall et al., 1996). Ivermectin is very effective against 3rd and 4th stage larvae (up to 70 days of infection), so prophylactic administration of ivermectin every one to two months is considered necessary to prevent maturation of any circulating heartworm larvae. Treatment every 3 months may be too sparse to completely ensure that larvae do not mature.

McCall et al., (1996) conducted a trial to assess the efficacy of ivermectin against heartworm of 3 and 4 month’s duration. Commencement of one monthly heartworm prophylaxis (ivermectin PO 6 μg/kg) in dogs that had established infections of 3 months duration resulted in 97.7% less total worm burden than in the controls. Likewise, microfilaraemia was prevented in all cases when ivermectin was given 3 months after infection. The results indicate that ivermectin at 3 monthly intervals may be of some benefit in preventing further infection.

The most important aspect of the present survey was to determine if any reactions to ivermectin (given at 200μg/kg) occur in dogs with heartworm infection. Owners noticed no reactions within 1-2 days of treatment. Comparative mortality rates for dogs with microfilariae and those
without were not statistically different (P=1.000 F-E test) for the coastal region (where there were adequate numbers for analysis) indicating that the treatments did not affect those dogs with microfilariae differently to those without. The result also suggests that heartworm infection does not affect the death rate in the dogs of the present study.

7.4.4.3 *Spirocera lupi*

The average estimated age of the *S. lupi* infected dogs in the present survey was about 2 years, which does not differ considerably from that of the uninfected dogs sampled in the study (2.3 years). The mean age of the native dogs examined by necropsy in Kenya was 2.9 years in comparison to 5 years for pet dogs in the same regions (Brodey et al, 1977). The native dogs in Kenya appeared to have similar lifestyles to the dogs in the present investigation, with many of them supplementing their diets by hunting birds and small mammals. The lowest age at which a patent infection was found in the present survey was 9 months indicating that infection must have occurred at about 3 months (pre-patent period 6 months). Studies in other countries have found immature worms and associated aortic lesions in dogs as young as 4 months (Chandrasekharon et al, 1958) and 7 months (Hamir, 1984) of age. Death due to haemothorax, as a result of the recent migration of worms in the aorta, was also more commonly reported in younger dogs (less than 3 years) in studies in Papua New Guinea (Hamir and Onaga, 1986). Because the infected dogs in the present survey had patent, mature infections, they were past the stage of developing aortic aneurisms and rupture due to migrating immature worms. *S. lupi* may be one causal factor for the low average age of dogs in these communities.

These findings demonstrate the sporadic distribution of spirurid parasites in areas previously not investigated within Australia. Although it is presumed that these parasites are endemic in these areas and in Australia, surveillance studies of this nature are of assistance in alerting a possible introduction of other canine parasites from countries to the north of Australia.
Although no studies have been documented on the efficacy of ivermectin against *S. lupi*, ivermectin has been found to effective against a closely related nematode, *Physaloptera preputialis*, in cats. In two cases with vomiting and melena due to the adults residing in the stomach, ivermectin at 200μg/kg SC was successful in abating clinical signs in the cats (Gustafson, 1995). This may also be the case with *Spirocerca lupi* as dogs that were treated in the present study were more likely to stop egg shedding than those not treated. More trial studies are needed to confirm this.

### 7.4.4.4 Gastrointestinal parasites

#### 7.4.4.4.1 Risk factors for Infection with *Campylobacter* and *Salmonella*

**7.4.4.4.1.1 Age of Host**

Although there were too few isolations in the present survey to determine any sex predilection for *Campylobacter*, generally, puppies less than 6 months old are more likely to carry *C. jejuni* than adult dogs (Willard et al, 1987). This may reflect increased exposure of young animals to faecal excrement through grooming and eating habits and confinement to a limited space (Willard et al, 1987).

Likewise, younger dogs are more likely to harbour *Salmonella* organisms and are thus of increased transmission risk to humans (Blaser et al, 1978; Pelzer, 1989). Despite this, adult dogs in the present survey were more likely to excrete the organisms. This may be due to other risk factors, such as malnourishment and concurrent infections, rather than age alone.

As there were only small numbers of infected dogs, only one survey of gastrointestinal bacterial infection was performed in the present study. The effect of treatment of concurrent parasite infections on the prevalence of *Campylobacter* and *Salmonella* remains unknown.
7.5 Conclusion

Mass treatment of dogs in remote communities with 200μg/kg ivermectin every three months resulted in the reduction in prevalence of scabies and hookworm. The prevalence of scabies dropped to below 10% by about 9 months after the commencement of treatments and the severity of infection was also reduced. Seasonal factors (rain especially) affected the prevalence of hookworm with prevalences during the wet season reaching the values attained before the treatments began. Ivermectin treatments did not affect the prevalence of *Toxocara canis* (which was below 10% for each region and community) or *Giardia duodenalis*. The prevalence of heartworm microfilaraemia reduced after treatment (and hence the reservoir for infection of other dogs) and ivermectin treatments did result in preventing *Spirocerca lupi* egg shedding in infected dogs.

The effect of the program on scabies and hookworm infection was not limited to those animals that were treated as untreated dogs also showed reduced prevalence in infection. This effect may be due to a ‘spill-over’ effect where there are fewer animal and environmental reservoirs for infection due to a reduction in infective stages because of treatments.

Transmission of scabies and hookworm was possible throughout the program although the prevalence of hookworm was affected by season. Although transmission of hookworm was possible through the dry season when the environment was less conducive to larval survival, the transmission rate was higher during the wet season. Concentration of treatments during the dry season to complement the effects of the season in breaking the transmission cycle may result in better control of geohelminths.

The host risk factors for infection with parasites provided some indication that targeted therapy rather than mass chemotherapy of all animals may be preferable. Both male and female dogs were equally susceptible to scabies infection although older dogs were more likely to show signs of infection. Hookworm infection was more common in male dogs and male dogs were
more likely to become reinfected after treatment. Likewise, puppies were more likely to be infected than older dogs suggesting that treatment of male dogs and puppies is important in the control of environmental contamination with hookworm eggs. Pregnant female dogs are also important in the epidemiology of hookworm, but with controlled breeding, the reservoir of infection is reduced. Similar to hookworm, roundworm infection was more likely in male dogs and puppies, but the significance of this is questionable in the present study because of the low prevalence of roundworm. In communities with low prevalence of scabies the targeting of male dogs and younger animals may be effective for parasite control.

The number of dogs per household was only found to be a factor for parasite infection for scabies and *Giardia* (which is not affected by ivermectin treatment). Both of these parasites rely on close contact for transmission, which explains this phenomenon. Population control of dogs to reduce the number of dogs per household may ultimately help reduce the potential for cross transmission of scabies, although continual treatment of dogs for scabies is effective in reducing the prevalence to an acceptable level. Once the prevalence of scabies is below 10%, 6 monthly treatments are likely to be effective in controlling scabies as demonstrated in the present study at Looma and Kalumburu.

Infection with scabies was found to be associated with weight loss in the present study, but hookworm infection was not considered to affect the host’s weight status. Hookworm infection, though, was associated with anaemia. The overall effect of control of hookworm and scabies in the present study is considered to be a general increase in health status with resolution of anaemia and improved weight gain (or reduced loss).

The treatment of dogs with ivermectin every three months at the compliance rates attained in the present study is expected to reduce the reservoir of zoonotic parasitic infection in communities and improve the health status of dogs. After the establishment of the program and control of scabies infection rates to low levels, strategic treatment of dogs (particularly male dogs and
puppies) coupled with a breeding control program at intervals less than six months would also be effective if treatments are concentrated during the dry season.
Chapter 8

THE EFFECT OF THE CANINE PARASITE PROGRAM
ON CHILD HEALTH

8.1 Introduction

One of the major aims of the project was to reduce the likelihood for zoonotic transmission in Aboriginal communities. Measurement of improvement in human health due to a canine parasite control campaign is fraught with problems, mainly because of the non-specific nature of many zoonoses. Other environmental health studies have also faced problems in methodology in determining the impact of environmental intervention. Pholeros, Rainow and Torzillo (1993) implemented improved housing and environmental strategies for communities of the Anangu Pitjantjatjara Freehold Lands of South Australia. They subsequently concluded “the most important outcome measure is to demonstrate the effective implementation of sustainable health hardware programs”, rather than try to demonstrate improved health status in communities of small numbers. A similar situation occurs in the communities of the Kimberley, where the effects of one program interrelate with other programs also trying to achieve similar objectives.

The most sensitive indicator of any improvement in human health related to environmental health intervention over a short period of time is skin disease (Pholeros et al, 1993). Although proof of direct relationships between canine skin disease and human skin disease is beyond the scope of this thesis, a reduction in morbidity due to skin disease may, in part, indicate the impact of the ivermectin treatment program in dogs on people in the communities.

It is not likely that the effect of ivermectin sensitive zoonoses on general gastrointestinal disease presenting as diarrhoea will be major. Gastrointestinal disease, though, is a good indicator of general hygiene and environmental health as many of the common pathogens, such as Campylobacter and Giardia, are dependent on faecal-oral transmission including food and water as vehicles. Any general effect of improved water and sewage or domestic hygiene may
be expected, in part, to be exhibited by a reduction in diarrhoeal disease. Diarrhoeal disease acts as an appropriate morbidity control factor.

8.2 Methodology

Data relating to the health of children in the present study communities was collected from several sources. The general methodology regarding the surveys is described elsewhere (see 3.4).

Clinical surveys of skin diseases of 5-15 years old children, conducted by medical practitioners, were undertaken at three of the communities of the present study in June 1992 and June 1993.

A gastrointestinal pathogen survey (parasites and bacteria) was conducted on samples collected from 0-5 years old children from 5 of the study communities in March 1992.

In addition to the two prevalence studies, a retrospective study of hospital and clinic data from 0-15 years old children over a 5-year period (1990-1995) was undertaken in June 1995. Information collected from the hospital and clinic records included all presentations for skin and gastrointestinal conditions. All cases of dog bites were also investigated. The information is presented in the thesis as yearly (or 5 yearly) incidence of each affliction.

The five communities from which retrospective data were collected are labeled as communities A, B, C, D and control community. Communities A and B are within the same ‘region’ as the control community (central region) whereas the others are in a separate region to the control community.
8.3 Results

8.3.1 Cutaneous Infections

8.3.1.1 Prevalence Study

Repeat skin surveys of children from communities A, B and D in June 1992 (before the program started) and June 1993 showed impetigo to be the most common skin complaint, followed by scabies (Table 8.1). Only at community D was a reduction in scabies and impetigo noted, whereas at community B, scabies was not seen.

8.3.1.2 Retrospective Data

Scabies

The yearly incidence of scabies at the control community increased over the five-year period from 6.3% [1.4, 11.1] to 20.4% [13.1, 27.7], the projected value for 1995 (Table 8.2). Community A followed very closely with every year’s incidence being similar to the control community. Communities B and C, on the other hand, showed rapid declines in scabies infection rates over the period of the dog parasite control program.

At community D, the trend for infection was not as linear, as the incidence rebounded in 1991 (24.4% [18.5, 30.2]) and 1994 (18.4% [13.3, 23.4]). Despite this, the rate was always below that of the control community apart from the first two years, which were before intervention.

Skin Infections

Because of the ambiguity in diagnosis of scabies infection in humans, all skin infections, excluding definitive scabies diagnoses, were compiled to determine if any changes had occurred over the six-year period. Impetigo was included, as it is a common sequel of scabies if the mite infection is left untreated for any length of time (Sharma, Mishra, Pal, Gupta, Dutta and Dutta, 1984; Burgess, 1994).
### Table 8.1: June 1992 and June 1993 Survey of 5-15 Years Old Children for Skin Afflictions

<table>
<thead>
<tr>
<th></th>
<th>Community A</th>
<th></th>
<th>Community B</th>
<th></th>
<th>Community D</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Scabies</td>
<td>13.8</td>
<td>13.5</td>
<td>0</td>
<td>0</td>
<td>12.5</td>
<td>6.2</td>
</tr>
<tr>
<td>Impetigo</td>
<td>27.0</td>
<td>27.0</td>
<td>6.6</td>
<td>21.7</td>
<td>32.5</td>
<td>25</td>
</tr>
<tr>
<td>Boils</td>
<td>8.3</td>
<td>2.7</td>
<td>0</td>
<td>13.0</td>
<td>3.7</td>
<td>0</td>
</tr>
<tr>
<td>Tinea</td>
<td>0</td>
<td>0</td>
<td>2.2</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Scalp Sores</td>
<td>2.7</td>
<td>0</td>
<td>2.2</td>
<td>0</td>
<td>1.2</td>
<td>1.2</td>
</tr>
<tr>
<td>Undefined</td>
<td>8.3</td>
<td>13.5</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>No Abnormalities Detected</td>
<td>52.7</td>
<td>54.0</td>
<td>88.8</td>
<td>67.4</td>
<td>65</td>
<td>61.7</td>
</tr>
</tbody>
</table>
Table 8.2: Yearly Incidence of Scabies in 0-15 Years Old Children

<table>
<thead>
<tr>
<th>Year</th>
<th>Community A</th>
<th>Community B</th>
<th>Community C</th>
<th>Community D</th>
<th>Control Community</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number of Children</td>
<td>% Infected</td>
<td>Number of Children</td>
<td>% Infected</td>
<td>Number of Children</td>
</tr>
<tr>
<td>1990</td>
<td>57</td>
<td>1.8*</td>
<td>98</td>
<td>17.3</td>
<td>167</td>
</tr>
<tr>
<td>1991</td>
<td>56</td>
<td>14.3*</td>
<td>109</td>
<td>18.3*</td>
<td>173</td>
</tr>
<tr>
<td>1992</td>
<td>58</td>
<td>25.9*</td>
<td>113</td>
<td>8</td>
<td>168</td>
</tr>
<tr>
<td>1993</td>
<td>60</td>
<td>21.7*</td>
<td>111</td>
<td>6.3</td>
<td>161</td>
</tr>
<tr>
<td>1994</td>
<td>57</td>
<td>24.6*</td>
<td>114</td>
<td>7</td>
<td>171</td>
</tr>
<tr>
<td>1995 (up to July)</td>
<td>56</td>
<td>12.5*</td>
<td>110</td>
<td>0.9</td>
<td>168</td>
</tr>
<tr>
<td>1995 (projected)</td>
<td>56</td>
<td>25*</td>
<td>110</td>
<td>1.8</td>
<td>168</td>
</tr>
</tbody>
</table>

* No significant difference to corresponding values from control community at P<0.05
There was no statistical difference between the yearly incidence of skin infections at the control community for any of the years examined (P=0.517) (Table 8.3). The average incidence was 23.7%. At communities A and B, the incidence of skin infections fell although at some years the incidences were similar to the control community. At community C, the incidence also decreased to a level similar to the control community.

The incidence of skin infections at community D did not decrease, but always remained similar to or below the control community.

### 8.3.2 Diarrhoeal Disease

#### 8.3.2.1 Prevalence Study

The prevalence study of 0 to 5 years old children from 4 communities prior to commencement of the program showed the most common parasite of the children was *Entamoeba coli*, followed by *Giardia duodenalis*. *Hymenolepis nana* was also common (16.4%) (Table 8.4). Overall, only 28.4% of children had no parasites. *Salmonella* and *Campylobacter* isolations were much less common, with 91% of children having no bacteria cultured. *Salmonella* was the most commonly isolated bacterium, but still only in 4.5% of samples.

#### 8.3.2.2 Retrospective Data

The number of new cases per year was determined by examining hospital and clinic records of all children in the 0-15 years age group. The incidence of diarrhoeal disease was then calculated based on the population census of children in the community at the time. Approximately 38% of all presentations to clinics or hospitals were for a complaint of diarrhoea (Table 8.5). For all communities, except community C, the incidence of diarrhoeal disease decreased over the five years of retrospective assessment. At community C, the situation had neither improved nor become worse.
<table>
<thead>
<tr>
<th>Year</th>
<th>Community A</th>
<th></th>
<th>Community B</th>
<th></th>
<th>Community C</th>
<th></th>
<th>Community D</th>
<th></th>
<th>Control Community</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number of Children</td>
<td>% Infected</td>
<td>Number of Children</td>
<td>% Infected</td>
<td>Number of Children</td>
<td>% Infected</td>
<td>Number of Children</td>
<td>% Infected</td>
<td>Number of Children</td>
<td>% Infected</td>
</tr>
<tr>
<td>1990</td>
<td>57</td>
<td>35.1*</td>
<td>98</td>
<td>45.9</td>
<td>167</td>
<td>44.3</td>
<td>198</td>
<td>12.6</td>
<td>96</td>
<td>23.9</td>
</tr>
<tr>
<td>1991</td>
<td>56</td>
<td>33.9*</td>
<td>109</td>
<td>32.1*</td>
<td>173</td>
<td>35.8</td>
<td>209</td>
<td>16.7*</td>
<td>106</td>
<td>21.7</td>
</tr>
<tr>
<td>1992</td>
<td>58</td>
<td>12.1</td>
<td>113</td>
<td>11.5</td>
<td>168</td>
<td>38.7</td>
<td>209</td>
<td>14.6</td>
<td>108</td>
<td>25.9</td>
</tr>
<tr>
<td>1993</td>
<td>60</td>
<td>15*</td>
<td>111</td>
<td>5.4</td>
<td>161</td>
<td>28.6</td>
<td>212</td>
<td>21.7*</td>
<td>118</td>
<td>17.8</td>
</tr>
<tr>
<td>1994</td>
<td>57</td>
<td>7</td>
<td>114</td>
<td>14</td>
<td>171</td>
<td>16.9*</td>
<td>228</td>
<td>24.1*</td>
<td>118</td>
<td>23.7</td>
</tr>
<tr>
<td>1995 (up to July)</td>
<td>56</td>
<td>8.9*</td>
<td>110</td>
<td>4.6</td>
<td>168</td>
<td>16.7*</td>
<td>219</td>
<td>6.4</td>
<td>118</td>
<td>14.4</td>
</tr>
<tr>
<td>1995 (projected)</td>
<td>56</td>
<td>17.9*</td>
<td>110</td>
<td>9.1</td>
<td>168</td>
<td>33.3*</td>
<td>219</td>
<td>12.8</td>
<td>118</td>
<td>28.8</td>
</tr>
</tbody>
</table>

* No significant difference to corresponding values from control community (Junjuwa) at P<0.05
<table>
<thead>
<tr>
<th>Parasites</th>
<th>Number</th>
<th>Prevalence (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Entamoeba coli</td>
<td>24</td>
<td>35.8</td>
</tr>
<tr>
<td>Giardia duodenalis</td>
<td>17</td>
<td>25.4</td>
</tr>
<tr>
<td>Hymenolepis nana</td>
<td>11</td>
<td>16.4</td>
</tr>
<tr>
<td>Entamoeba sp.</td>
<td>2</td>
<td>3.0</td>
</tr>
<tr>
<td>Blastocystis hominis</td>
<td>2</td>
<td>3.0</td>
</tr>
<tr>
<td>Strongyloides stercoralis</td>
<td>2</td>
<td>3.0</td>
</tr>
<tr>
<td>Ancylostoma duodenale</td>
<td>2</td>
<td>3.0</td>
</tr>
<tr>
<td>Balantidium coli</td>
<td>1</td>
<td>1.5</td>
</tr>
<tr>
<td>Iodamoeba spp</td>
<td>1</td>
<td>1.5</td>
</tr>
<tr>
<td>No parasites detected</td>
<td>19</td>
<td>28.4</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Bacteria</th>
<th>Number</th>
<th>Prevalence (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Salmonella</td>
<td>3</td>
<td>4.5</td>
</tr>
<tr>
<td>Shigella flexneri 6</td>
<td>2</td>
<td>3.0</td>
</tr>
<tr>
<td>Campylobacter jejuni</td>
<td>1</td>
<td>1.5</td>
</tr>
<tr>
<td>No bacteria detected</td>
<td>61</td>
<td>91</td>
</tr>
</tbody>
</table>
### Table 8.5: Yearly Incidence of Diarrhoeal Disease in 0-15 Years Old Children

<table>
<thead>
<tr>
<th></th>
<th>Community A</th>
<th>Community B</th>
<th>Community C</th>
<th>Community D</th>
<th>Control Community</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number of Children</td>
<td>% Infected</td>
<td>Number of Children</td>
<td>% Infected</td>
<td>Number of Children</td>
</tr>
<tr>
<td>1990</td>
<td>57</td>
<td>57.9†</td>
<td>98</td>
<td>46.9‡</td>
<td>167</td>
</tr>
<tr>
<td>1991</td>
<td>56</td>
<td>46.4‡</td>
<td>109</td>
<td>48.6‡</td>
<td>173</td>
</tr>
<tr>
<td>1992</td>
<td>58</td>
<td>29.3‡</td>
<td>113</td>
<td>60.2</td>
<td>168</td>
</tr>
<tr>
<td>1993</td>
<td>60</td>
<td>13.3†</td>
<td>111</td>
<td>42.3</td>
<td>161</td>
</tr>
<tr>
<td>1994</td>
<td>57</td>
<td>12.3†</td>
<td>114</td>
<td>8.8†</td>
<td>171</td>
</tr>
<tr>
<td>1995 (up to July)</td>
<td>56</td>
<td>7.1†</td>
<td>110</td>
<td>6.4†</td>
<td>168</td>
</tr>
<tr>
<td>1995 (projected)</td>
<td>56</td>
<td>14.3†</td>
<td>110</td>
<td>13.7‡</td>
<td>168</td>
</tr>
</tbody>
</table>

Average percentage of children presented to clinic or hospital in a one year period that have diarrhoea (pooled data)

<table>
<thead>
<tr>
<th></th>
<th>Average Percentage</th>
<th>Community A</th>
<th>Community B</th>
<th>Community C</th>
<th>Community D</th>
<th>Control Community</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>36.9**</td>
<td>33.0**</td>
<td>40.7**</td>
<td>40.9**</td>
<td>37.4**</td>
<td></td>
</tr>
</tbody>
</table>

† No statistical difference to control community (Chi squared test P<0.05, Fisher’s Exact test used if expected cell value less than 5)

** No statistical difference between communities
Interestingly, the communities of the central region were all very similar in incidence. There was no statistical difference in incidence of diarrhoea between communities A and B and the control community for most years indicating that either the actual rate of diarrhoea had decreased for all communities, or the rate of reporting had decreased. Diarrhoea of unknown cause accounted for between 38.9% [31,46] to 67.6% [61,74] of all new diarrhoea cases.

Laboratory confirmed cases of *Giardia duodenalis* had decreased for most communities over the five-year period (Table 8.6). The community with the highest five yearly incidence was community B with 38.5% of children being diagnosed with a new infection in a five year period. The three communities of the central region recorded the highest incidences, but this may have been due to a *Giardia* education/awareness campaign in these communities which may have resulted in increased reporting and investigation rates by medical staff.

Other gastrointestinal pathogens were diagnosed less often with *Hymenolepis nana* being the next most frequently diagnosed organism (Table 8.7). *Salmonella* and *Cryptosporidium* were also diagnosed occasionally. *Ancylostoma duodenale* was infrequently diagnosed at the communities investigated.

### 8.3.3 Dog Bites in Children

The five yearly incidence of dog bites in 0-15 years old children varied for each community from 0.3 at community A to 7.3 at the control community (Table 8.8). The total number of dog attacks for each year during the five year period for the project communities did not vary significantly (Chi squared test, P=0.27) indicating that the incidence of dog bites during the parasite and breeding control program did not change.
Table 8.6: Five Yearly Incidence of Confirmed Giardiasis in 0-15 Years Old Children

<table>
<thead>
<tr>
<th></th>
<th>Community A (n=57)</th>
<th>Community B (n=109)</th>
<th>Community C (n=168)</th>
<th>Community D (n=211)</th>
<th>Control Community (n=109)</th>
<th>TOTAL</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number Infected</td>
<td>Number Infected</td>
<td>Number Infected</td>
<td>Number Infected</td>
<td>Number Infected</td>
<td>Number Infected</td>
</tr>
<tr>
<td>1990</td>
<td>6</td>
<td>17</td>
<td>0</td>
<td>3</td>
<td>5</td>
<td>31</td>
</tr>
<tr>
<td>1991</td>
<td>8</td>
<td>6</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>23</td>
</tr>
<tr>
<td>1992</td>
<td>0</td>
<td>13</td>
<td>6</td>
<td>5</td>
<td>5</td>
<td>29</td>
</tr>
<tr>
<td>1993</td>
<td>1</td>
<td>5</td>
<td>2</td>
<td>2</td>
<td>0</td>
<td>10</td>
</tr>
<tr>
<td>1994</td>
<td>0</td>
<td>1</td>
<td>3</td>
<td>0</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>Five Yearly Incidence (%)</td>
<td>26.0</td>
<td>38.5</td>
<td>7.7</td>
<td>6.2</td>
<td>14.7</td>
<td></td>
</tr>
</tbody>
</table>

Table 8.7: Five Yearly Incidence of Laboratory Confirmed Gastrointestinal Diseases in 0-15 Years Old Children

<table>
<thead>
<tr>
<th></th>
<th>Community A (n=57)</th>
<th>Community B (n=109)</th>
<th>Community C (n=168)</th>
<th>Community D (n=211)</th>
<th>Control Community (n=109)</th>
<th>AVERAGE</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Hymenolepis nana</td>
<td>13.9</td>
<td>10.1</td>
<td>10.7</td>
<td>1.9</td>
<td>12.8</td>
</tr>
<tr>
<td></td>
<td>Salmonella spp</td>
<td>10.4</td>
<td>12.8</td>
<td>6.5</td>
<td>5.7</td>
<td>6.4</td>
</tr>
<tr>
<td></td>
<td>Cryptosporidium parvum</td>
<td>6.9</td>
<td>13.8</td>
<td>4.2</td>
<td>2.8</td>
<td>6.4</td>
</tr>
<tr>
<td></td>
<td>Shigella spp</td>
<td>1.7</td>
<td>6.4</td>
<td>10.1</td>
<td>1.4</td>
<td>9.2</td>
</tr>
<tr>
<td></td>
<td>Blastocystis hominis</td>
<td>8.7</td>
<td>6.4</td>
<td>1.8</td>
<td>1.4</td>
<td>4.6</td>
</tr>
<tr>
<td></td>
<td>Strongyloides stercoralis</td>
<td>1.7</td>
<td>1.8</td>
<td>6.5</td>
<td>4.7</td>
<td>7.3</td>
</tr>
<tr>
<td></td>
<td>Campylobacter spp</td>
<td>0</td>
<td>6.4</td>
<td>5.4</td>
<td>2.8</td>
<td>2.8</td>
</tr>
<tr>
<td></td>
<td>Enteropathogenic E. coli</td>
<td>1.7</td>
<td>6.4</td>
<td>2.4</td>
<td>0</td>
<td>2.8</td>
</tr>
<tr>
<td></td>
<td>Hookworm</td>
<td>5.2</td>
<td>1.8</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Aeromonas hydrophila</td>
<td>0</td>
<td>1</td>
<td>1.2</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Trichuris trichiura</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0.5</td>
<td>0</td>
</tr>
</tbody>
</table>
Table 8.8: Five Yearly Incidence of Dog Bites in 0-15 Years Old Children

<table>
<thead>
<tr>
<th>Year</th>
<th>Community A (n=57)</th>
<th>Community B (n=109)</th>
<th>Community C (n=168)</th>
<th>Community D (n=211)</th>
<th>Control Community (n=109)</th>
<th>TOTAL</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number of Reported Bites</td>
<td>Number of Reported Bites</td>
<td>Number of Reported Bites</td>
<td>Number of Reported Bites</td>
<td>Number of Reported Bites</td>
<td>Number of Reported Bites</td>
</tr>
<tr>
<td>1990</td>
<td>1</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>1</td>
<td>4*</td>
</tr>
<tr>
<td>1991</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>2*</td>
</tr>
<tr>
<td>1992</td>
<td>0</td>
<td>0</td>
<td>5</td>
<td>2</td>
<td>1</td>
<td>8*</td>
</tr>
<tr>
<td>1993</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>5</td>
<td>3</td>
<td>9*</td>
</tr>
<tr>
<td>1994</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>7*</td>
</tr>
<tr>
<td>Five Yearly Incidence (%)*</td>
<td>0.3</td>
<td>1</td>
<td>6.5</td>
<td>4.3</td>
<td>7.3</td>
<td></td>
</tr>
</tbody>
</table>

*No statistical difference between years
8.4 Discussion

Most reports of disease in Aboriginal communities have been based on hospital admission rates or surveys of a single cause or aspect of diarrhoea (Berry and Gracey, 1981b; McNeilly et al, 1983; Gracey and Anderson, 1989; Waddell and Lee, 1991; Gracey, 1992). Few studies have concentrated on prospective, longitudinal assessment of diarrhoeal risk factors in communities (Gunzburg et al, 1992; Gracey et al, 1992). Attempted prospective studies of environmental program impacts are complicated by factors such as; lack of adequate control and resultant necessity to do one to one comparison between communities, differences in health indicator definitions, failure to analyse health data by age of subjects and seasonality of impact variables (Blum and Feachem, 1983). In the present study, retrospective analysis of clinic and hospital data was necessary due to the difficulties in implementing a well-constructed prospective assessment of health. Despite the flaws in examining incidence data from attendance at health facilities, this type of data collection does give some indication of the disease in the population (Gibbs, 1996).

8.4.1 Cutaneous Infections

It is not in the scope of this thesis to determine a direct link between scabies infection rates in humans and animals. However, the numerous reports of cross-transmission of scabies mites (Emde, 1961; Beck, 1965; Newton and Gerrie, 1966; Schwartzman et al, 1967; Smith and Claypoole, 1967; Thomsett 1968; Elgart and Higdon, 1972; Charlesworth and Johnson, 1974; Agbede, 1978; Scott and Horn, 1987; Paterson et al, 1995; Burton, 1997) suggest that canine scabies may contribute to some cases of pruritus and secondary bacterial infection in humans. As such, a reduction in infection of dogs may reduce cutaneous infections in humans, but is unlikely to result in a decrease in diagnosis of classical scabies.

The diagnosis of scabies in humans is as difficult as it is in dogs, hence scabies has been termed the "great imitator" (Burgess, 1994). A definitive diagnosis of classical scabies is made when the burrows of the mites are found, however, under tropical conditions, the development of
clearly defined burrows is less common and may be detected in as few as 3% of infected patients (Taplin, Arrue, Walker, Roth and Rivera, 1983). Burrows are also particularly hard to find in children and even more so if excoriation, eczematization or pyoderma have obscured the presentation (Burgess, 1994). In the present study, diagnosis was mostly conducted by nursing staff and occasionally medical practitioners. Overall the reliability of diagnosis was dependent on the experience of the staff and diligence in examining the children. Scabies distribution and presentation in children differ from adults with papular eruptions and involvement of the scalp being more common in children (Burgess, 1994). However, the probable incidence of scabies at community A and the control community were similar and did not decrease. This was in contrast with communities B and C where there was an obvious improvement over the five-year period.

Most studies documenting scabies infection in Aboriginal communities provide point prevalence rates. At one island community of the Northern Territory, approximately 32% of children had evidence of scabies prior to treatment (Carapetis et al, 1997). Seventeen percent of all people from another community in the Northern Territory had scabies, but age analysis was not done (Hoy et al, 1997). As under 15 years old children often have higher rates of scabies infection than adults, the prevalence in children is expected to be higher (Porter, 1979).

At the three communities involved in the present study, where prevalence surveys were conducted in June 1992 and 1993, the prevalences (between 0 to 13.8%) were well below the rates reported from the Northern Territory. Scabies infestations have shown seasonality in some studies (Blumenthal et al, 1976; Green, 1989; Burgess, 1994); the prevalence is increased in ‘winter’ months. If this is the case in the present study, then the prevalence was at its peak when the surveys were conducted, in June. However, Porter (1979) did not demonstrate seasonality at a Gambian village where the prevalence of scabies was unexpectedly low at 2%. Porter (1979) suggested that the low prevalence might be due to the village being in a period of high group resistance, as occurs with the well documented cycling of scabies in human
populations. To determine if there is any cyclical activity in the present study, the data would have to extend back further, but adequate records for the child and maternal health initiative only started in the mid 1980s.

The prevalence data in the present study followed the incidence patterns found from the hospital and clinic records. Community A children were experiencing similar, high incidences for scabies in 1992 and 1993 and likewise, had the same prevalence of infection for both years. At community B, where the incidence was 8 and 6.3% respectively, no children of the survey in June of each year had scabies. At community D, where the incidence decreased for 1992 and 1993, so did the prevalence.

The reasons for the differences between and within communities are not evident. It is likely that where improvements have occurred, there has been heightened awareness with treatment of cases, possibly coupled with improved domestic hygiene. Carapetis et al (1997) did not institute any environmental measures, such as washing and airing of linen and clothing, during a treatment control campaign using 5% permethrin cream at an Aboriginal Community in the Northern Territory, but still had success in reducing the prevalence over a 25 month period. Other studies have found fomites to be of prime importance in transmission of infection (Gulati et al., 1977; Blumenthal et al., 1976). Gibbs (1996) considered that treatment of individuals without improvement in socioeconomic conditions in rural Tanzanian villages was unlikely to provide lasting change in the community because early re-infection with scabies is possible.

In any case, the treatment of household dogs is not likely to have any impact on definitive scabies infection rates, as Carapetis et al (1997) had suggested, because zoonotic infection with scabies is likely to result in clinical presentations very dissimilar to human scabies, due to the lack of burrows. Zoonotic scabies are more likely to be intensely pruritic and with complications of excoriations and secondary pyoderma and hence would not necessarily be diagnosed as classic scabies. The clinical effects of canine scabies are reported to be transitory
yet symptomatically severe (Charlesworth and Johnson, 1974), but do recur quickly or persist if the infected contact animals are not treated (Elgart and Higdon, 1972). Likewise, the secondary infections are expected to persist after the mites have disappeared.

The prevalence surveys conducted in June 1992 and 1993 revealed impetigo to be the most important skin affliction of the children. Interestingly, 8.3 and 13.5% of skin conditions at community A could not be defined by the medical staff, indicating that diagnosis of skin complaints can be complicated. The prevalence data do not follow the incidence data closely as found with scabies. It is likely that there is under-reporting of some types of skin infections such as skin sores and impetigo because they are relatively common conditions. This would result in lower reported incidence figures.

Overall, the recorded rates of pyoderma in the children of the Kimberley are lower than in Queensland or the Northern Territory. Nimmo, Tissiswood, Nuttal, Baker and McDonald (1992) found 42% of children from a community of Cape York Peninsula to have pyoderma from which 76% of skin cultures grew group A streptococci (GAS). Approximately 69% of children in the treatment program conducted by Carapetis et al (1997) had impetigo. More than a quarter of all people in Hoy et al’s (1997) Northern Territory survey had skin sores, ranging up to 20 sores per person. In addition to this, Hoy et al (1997) found a substantial prevalence of acute glomerulonephritis associated with skin sores.

Pyoderma, which is frequently caused by scabies infections, is considered to be the major reservoir of group A streptococcal infection in Aboriginal communities (Van Buyneder, Gaggin, Martin, Pusgley and Mathews, 1992; Carapetis et al, 1997). Post-streptococcal infection sequelae include acute rheumatic fever and acute glomerulonephritis (Bisno, 1995), both of which are very common in Aboriginal persons (Streeton and Hanna, 1995). In one survey of Yarrabah community of North Queensland, the prevalence rate of acute rheumatic fever was 21.6 per thousand (Neilson, Streatfield, West, Johnson, Glavin and Baird, 1993) which is well
above developed world rates (0.23 to 1.88/100 000 school children in the USA during the 1970’s) (Bisno, 1995).

Chronic renal disease is also being recognised and treated with increased frequency in Aboriginal communities (Van Buyneder et al, 1992). One possible cause of Aboriginal people being 8 times more likely than Caucasians to have end stage renal disease, is delayed effects of post-streptococcal glomerulonephritis (Van Buyneder et al, 1992). These two major complications of pyoderma are the reason for attempts at community based and treatment based control of scabies infections in people.

Dogs are not considered significant reservoirs of Group A Streptococci as about 80% of canine origin beta haemolytic streptococci belong to Lancefield group G (Biberstein, Brown and Smith, 1980). Group A Streptococci, though, are sometimes cultured from canine sources (Biberstein et al, 1980). Despite the lack of reservoir, canine scabies may provide a source of pruritus and papular rash, which is sufficient to start pyodermic lesions that may contain human derived Group A Streptococci.

The reduction in incidence of pyoderma at most communities in the present study can not be directly attributed to the canine scabies control program undertaken, but do indicate that health conditions may be improving. More rapid improvements would be made by community based treatment programs for human scabies, such as at Minjilang in the Northern Territory, where the whole community was treated once and monitored every few months over a 25 month period (Carapetis et al, 1997). Environmental health based programs (involving education about the role of fomites in scabies infections) take much longer to achieve similar results, but have the advantage of being education based to change people’s behaviour and promote long term prevention of disease.
8.4.2 Diarrhoeal Disease

The findings of the parasitology survey of children in the present study were very similar to other studies, although the prevalence of *Entamoeba coli* was much higher than in other studies (Meloni *et al.*, 1993; Gunzburg *et al.*, 1992; Gracey *et al.*, 1992). Meloni *et al.* (1993), found *Giardia duodenalis* to be the most common parasite amongst 0-15 years old children with an infection rate of 32.1% (compared with 25.4% in 0-5 years olds in the present survey), based on direct stool microscopy and flotration methods. In fact, Meloni *et al.* (1993) considered the prevalence to be an underestimate because of examination of single samples only. Although sedimentation techniques were used in the present study, with good recovery rates, it is likely that some infections would have gone undetected. The sampling of children in the dry season also results in lower recovery rates because the prevalence of *Giardia* is considered to be lowest at this time of the year (Meloni *et al.*, 1993).

The high prevalence rates of faecal-orally transmitted parasites, as well as those that are potentially zoonotic, indicates poor living conditions and low standards of hygiene in the communities (Meloni *et al.*, 1993). Despite this, the rates of gastrointestinal bacterial isolation were much lower than in previous studies even though these pathogens also rely on faecal-oral transmission. Gracey *et al.* (1992), isolated *Salmonella* from 37.5% (n=48) of children (with and without diarrhoea) in their second year of life. This is in comparison with only 4.5% of 0-5 years old children in the present study having *Salmonella* infections. *Campylobacter jejuni* was cultured in even fewer children (1.5%), compared with Gracey *et al*’s (1992) study (12.5%). In contrast to most parasite recoveries, bacterial pathogens have been detected more frequently in both asymptomatic and symptomatic faecal samples during the dry season (Gunzburg *et al.*, 1992). As the faecal samples in the present study were collected during June 1992 (dry season), the low rates are even more extraordinary.

Very few studies document the incidence of diarrhoeal disease as determined by both hospital and clinic records. The majority of retrospective studies show hospitalisation rates, rather than
regular morbidity patterns in communities. Unfortunately, hospital and clinic records do have flaws mostly related to the variation in personnel recording the data. Hospitalisation data document cases that are of sufficient severity to warrant extra attention, but cases which are less severe, that contribute to overall morbidity, are excluded. By examination of all recorded cases from hospital and clinic sources, a general indication of the common, endemic diarrhoeal disease incidence rates is provided, rather than just those cases that are severe enough to warrant hospitalisation.

Hospital and clinical data from the five communities showed *Giardia* and *H. nana* to be the most frequently diagnosed gastrointestinal pathogens, followed by *Salmonella*. As opposed to the prevalence data, the diagnoses were from children clinically affected, so the actual rate of infection with these pathogens is likely to be much higher than found in the present retrospective study. Even in children that had diarrhoea, not all cases were followed through with laboratory work-ups, so, again, the true incidence of infection with these pathogens is likely to be higher. Whether cases were worked-up or not depended on the clinician. Despite these problems, the reported rate of diarrhoeal disease in all communities of the central region (communities A, B and control) was very similar.

Overall, the finding that the rate of diarrhoeal disease decreased in incidence for all communities, apart from community C, indicates that there have been some positive changes in the communities. These may include improvement in the provision of safe water and sanitation facilities, medical intervention and changes to personal and community behaviour. Education about the hazards of canine related zoonoses and improvement in dog health may have contributed to the changes in people’s behaviour, even through the treatments did not specifically target any diarrhoea producing pathogens.
8.4.3 Dog Bites in Children

Medical staff from some communities in the present study were concerned about perceived higher rates of dog attacks after the commencement of the program, as the dogs were less lethargic after treatments. Overall, no significant changes in dog bite incidence for the duration of the program were determined from the records, although community D did experience some increase. It is likely that the program brought increased awareness of dog associated problems in the communities, and hence the concern for possible increased aggression in dogs as a result of the intervention.

Although no increases in dog bite incidence were determined, the overall incidence rate for dog bites is much higher in the present study population than the national average. There are very few reports on dog attacks in Australia (Thomas and Buntine, 1987; Podberscek, Blackshaw and Nixon 1990) with only one citing annual incidence figures (Nixon, Pearn and McGarn, 1980). Nixon et al (1980) estimated an average dog-bite incidence for the total population of Canberra, Australia, to be 184 per 100,000 people per year. The figures from the present survey range from 300 to 7300 per 100,000 children per year for reported dog bites. The actual incidence of dog bites including those that are not reported is likely to be much higher than found in the present study as Nixon et al (1980) found that only 1 dog bite in 38 results in an injury that requires medical attention and is reported. If the total human population were considered in the present study (rather than just children), the incidence would be less, as most bites occur to children (Thomas and Buntine, 1987; Gershman, Sacks and Wright, 1994; Sinclair and Zhou, 1995).

After experience in handling dogs for treatments it is apparent that the dogs in the study communities were rarely handled and if so, usually by only one person. Children also do not receive any instruction or training in appropriate behaviour towards dogs. The choice of dogs in communities is also a major factor for dog attacks, as many are Australian Cattle Dogs or crossbreeds from these dogs (see 4.3.2.5). Australian Cattle Dogs have been listed as the second
most common dogs to cause bites that require hospital treatment (Thomas and Buntine, 1987). They are also ranked 5th for the dog breeds that cause the highest attack rates (quoted in Murray and Penridge, 1992). Bull Terriers (and their crosses) are also relatively common in communities and are regarded as the most dangerous breed in Australia (Murray and Penridge, 1992; Podbersek and Blackshaw, 1993). Other dogs that are among the top six dangerous breeds include German Shepherds, Dobermans and Rottweilers, which are also represented in communities.

In addition to breed types, a survey in Denver (USA) found male, unsterilised dogs residing in houses with one or more children are more likely to bite than others (Gershman et al, 1994). These factors are also very common in Aboriginal communities as shown previously (see 4.3.1.3). Development of contraceptive drugs that block testosterone production or castration of male dogs are likely to be of benefit in curtailing unwanted aggressive behaviour in dogs in the future.

8.5 Conclusion

The effect of the canine parasite control program on human health remains unclear. As with most programs that indirectly address health improvement, a significant change is likely to take a long time and be difficult to measure. The incidence and prevalence of skin disease in the present study were lower than found at other Aboriginal communities. Had the baseline rate of cutaneous infections been higher in the present communities, then improvements in skin conditions due to the intervention may have been more dramatic and assessable.

In some study communities, the incidence of skin infections decreased during the program, and in others the rates remained static. In those communities where there was an improvement in skin infections, the improvement can not be directly attributed to the canine parasite control program. Likewise, no scabies treatment programs for people were conducted in the communities during the time of the program, so the improvement was also not related to
treatment alone. It is likely that a combination of factors were acting at the intervened communities during the five years of assessment which all resulted in a reduction in skin-related conditions.

As *S. scabiei* var. *canis* is unlikely to be able to maintain itself in the absence of re-infection from dogs, the diagnosis of classical scabies (from human adapted mites) in people does not indicate that canine scabies have not caused some skin conditions. Clinically affected people are unlikely to be harbouring canine adapted scabies mites. Active cross-transmission clinical trials and may assist in determining the true extent to which canine scabies cause clinical disease in humans.

The incidence of diarrhoeal disease for the intervened communities was similar to the control communities and was decreasing at most of the communities for the duration of the study period. This indicates that environmental health conditions (leading to diarrhoeal disease) were similar for each of the communities and possibly improving. High rates of diarrhoea in Aboriginal children are often associated with inadequate living conditions and personal and community hygiene practices that facilitate the spread of gastrointestinal infections (Gracey, 1992).

The finding that dog bites to children are very common in Aboriginal communities and are possibly much higher than found in other non-Aboriginal communities warrants further investigation and establishment of education programs to encourage safe dog ownership. The introduction of a contraceptive to curtail testosterone production in male dogs may also be of some assistance in controlling dog attacks in communities.

The establishment of the canine breeding and parasite control program has provided the infrastructure for the establishment of other environmental health programs in communities. Environmental health staff providing treatments to dogs have access to communities in a
positive manner as the improvement in dog health and potential improvement in human health gives a firm platform for the acceptance of other environmental health programs. The most effective outcome of the present study was the effective implementation of a sustainable environmental health program.
Chapter 9

SUMMARY

9.1 Canine Population Control

9.1.1 Population Structure

The population of dogs in remote communities of the Kimberley region had the following structural characteristics:

9.1.1.1 Age

The dog population in the Kimberley communities was young (average age 2 years) and there were more puppies in communities during the dry season than wet season. This was most likely due to an increase in breeding during the dry season months.

9.1.1.2 Sex

There was a bias towards male dogs in communities across all ages, but with increased tendency in the adult population. Surgical sterilization of dogs was rare, but owners were keen for contraceptive treatments for females. It is unknown whether owners would be as keen for permanent sterilization or use of other contraceptive products in both male and female dogs.

9.1.1.3 Dog Breeds

Ninety one percent of dogs were of mixed breed and the most common crossbreed was Australian Cattle Dog cross. The most common purebred dog was the Australian Cattle Dog, which is ranked as the second most common dog breed responsible for dog bites that require hospitalisation.

9.1.2 Distribution

9.1.2.1 Household Distribution of Dogs

Between approximately 48% and 78% of households had at least one dog. The eastern region experienced the densest population of dogs per household and owner, with the central region
intermediate and the coastal region least and sharing many parameters similar to urban centres in the Kimberley and the Philippines.

The number of dogs per dog owning household was greater than the number of dogs per owner as many owners lived at each household.

9.1.2.2 Owner Distribution

Smaller communities in desert regions were more likely to have a higher density of dogs per person.

For the central and eastern communities, most dogs were kept by 40 to 60 years olds. The 20 to 40 years old groups of the coastal communities kept the most dogs which was the same as the Kimberley townships.

Dog owners older than 80 owned the most dogs per owner in the central and eastern communities. This has important implications for education and zoonoses control programs, as many diseases are in higher prevalence in crowded conditions.

The number of dogs per person (rather than owner) was similar to other countries and non-Aboriginal communities of Australia and indicates that the overpopulation perceived in communities may be due to large numbers of dogs being owned by older owners, rather than an absolute overpopulation. There was also a high number of dogs per household in the present study compared with other studies which also indicates that the perceived dog ‘overpopulation’ may be due to the large numbers of owners and dogs living at each household due to inadequate housing in remote communities.

Men owned most of the dogs.
Education programs and veterinary services need to be targeted at people (particularly men) in the 40-60 years old category for population control and the over 80 years old people for control of potential zoonoses.

9.1.3 Dynamics

There is a very high turnover rate of dogs in communities with 47% dying or going missing in a 12 month period.

9.1.3.1 Mortality and Outward Migration of Dogs

The mortality rate increased during the wet season compared with nearby months. This may have been due to flooding in the wet season and the resultant shortage of food available for dogs.

Only 9.2% of non-euthanasia deaths had a defined cause and the rate of voluntary euthanasia increased during March 1993 after a severe wet season.

9.1.3.1.1 The Population Structure and Distribution of Dogs that Died or went Missing

During the months of the wet season, the proportion of dogs that died as puppies increased indicating reduced survival of these dogs at this time.

9.1.3.2 New Dogs and Inward Migration

After the program was stabilized, 13% of dogs at each three monthly visit were new to the community. Seventy four percent of new dogs were not born within the communities, but acquired from other communities (usually of the Kimberley).
9.1.3.2.1 Population Structure and Distribution of Incoming Dogs

Most of the new dogs were puppies or juveniles. The ratio of male dogs in the new dog population was the same as the established population (except for one visit) indicating that there was a preference for acquiring male dogs.

People in households with many dogs were just as likely to acquire new dogs as those in households with fewer dogs. The percentage of households acquiring new dogs over a 12 month period increased during the program and was approximately 94%.

9.1.3.3 Effectiveness of the Breeding Control Program

9.1.3.3.1 Contraceptive Effectiveness

Between 62 and 82% of bitches at each region were treated at each visit. Entire bitches that had never received progestrone treatment were 5 times more likely to breed than those that had received at least one treatment. The (three monthly) conception rates during the program (4%) were well below those before the treatments commenced (10%) and after the completion of the treatment program (15%).

The annual rate of change of dog population (excluding September 1993) was similar to other areas in North America although the reproduction rate was lower in the present study and the inward migration from other communities was greater.

Better control of population numbers in Kimberley communities may be achieved through a reduction in importation of new dogs coupled with a higher contraceptive treatment rate of female dogs. The use of longer acting contraceptives would reduce the need to catch female dogs as often and possibly improve compliance rates.
9.2 Parasite Control

9.2.1 Parasites of the Kimberley Dog Population

The parasites found in dogs from the Kimberley Region during the pretreatment testing of dogs were; *Sarcoptes scabiei*, *Rhipicephalus sanguineus*, *Ancylostoma caninum*, *Giardia duodenalis*, *Sarcocystis* spp., *Spirocerca lupi*, *Toxocara canis*, *Hymenolepis nana*, *Strongyloides stercoralis*, *Spirometra erinaceieuropai*, *Hammondia heydorni*, *Isospora ohiensis*, *Trichodectes canis*, *Ctenocephalides* spp. and *Dirofilaria immitis*. Serological testing from a random sample of dogs from all communities suggested the dogs may have been exposed to *Echinococcus granulosus*.

Hookworm was the most common parasite infecting dogs and the percentage of dogs with lesions indicative of scabies varied from 17% to 52% across each of the regions.

9.2.2 Risk Factors for Infection

9.2.2.1 Age

Dogs over the age of one year were more likely to be infected with scabies than younger dogs, whereas puppies were found to be at greater risk of infection with hookworm, *Toxocara canis* and *Giardia* than older dogs. These results were in accordance with other studies.

9.2.2.2 Sex

The only parasite that showed sex predilection for infection of dogs in the present study was hookworm where males were found to be one and a half times more likely to be infected with hookworm than females.

9.2.2.3 Numbers of Dogs per Household

Only scabies and *Giardia* infections were associated with high numbers of dogs per household (crowding) in the present study. Dogs with scabies were more likely to live at houses with more
than one dog and there was a strong correlation between the number of dogs infected per household and the number of dogs per household.

9.2.3 Effects of Parasites on Canine Health

At one community, scabies infection was associated with a loss in weight of the dogs. There was no association between scabies infection and anaemia when hookworm infection was accounted for at any of the communities.

Hookworm infection was associated with anaemia in puppies, juveniles and adults.

Treatment of dogs with ivermectin resulted in improved haematology values; dogs were 6 times less likely to have anaemia after treatment than before. *Sarcoptes scabiei* was associated with high serum globulin levels (most likely due to inflammation) which were reduced to normal levels after one treatment with ivermectin.

9.2.4 Effect of Parasite Control on Parasites

A single treatment with 200μg/kg ivermectin was found to reduce the prevalence of scabies and hookworm. Scabies was reduced by 70% by 5 weeks after treatment. The hookworm prevalence was reduced by 93% within one week of treatment, but reached 25% of the original prevalence by 5 weeks after treatment.

Ivermectin was also effective against ticks with a reduction in prevalence of 32% by one week after treatment. The original prevalence was reached by 5 weeks after treatment.

Long term three monthly treatment with ivermectin resulted in a reduction in the rate of scabies infection which was not directly related to treatment only as non-treated dogs also showed a reduction in infection rates. Generally by the third treatment (9 months after commencement) the prevalence was reduced from 17-52% to 10% or less in each region.
A seasonal fluctuation in hookworm prevalence was found during the treatment program. Wet season months had hookworm prevalences similar to pretreatment levels. For each visit at each region, the prevalence of hookworm in treated dogs was the same or lower than non-treated dogs indicating that the ivermectin treatments were effective. Dogs were more likely to lose their hookworm infections or remain negative during the dry season months.

Treatment did not affect the prevalence of *T. canis*, although the prevalence of *T. canis* was very low to begin with (<10%).

The percentage of dogs with circulating microfilariae decreased over the duration of the program suggesting that ivermectin treatment may reduce the reservoir of heartworm in communities.

Treated dogs were less likely to be shedding *Spirocerca lupi* eggs than those not treated. It is unknown whether ivermectin kills adult *S. lupi*.

**9.2.5 Effect of Canine Parasite Control on Human Health**

The direct effect of the canine parasite control program on child health was not determined in the present study, but at each of the communities in which the program was conducted, the rate of skin infections either decreased or remained the same for the duration of the program. The rates were also always lower or equivalent to the rates found at a control community.

Rates of diarrhoeal disease, an indicator of environmental health standards, in the program communities was similar to the control community indicating that environmental health factors had not changed during the program. As the rates of skin disease decreased, but the rates of diarrhoeal disease did not, the improvement in skin diseases was probably not related to environmental health alone, but a number of factors.
Recommendations for Future Canine Parasite and Breeding Control Programs

Recommendations for future canine parasite and breeding control programs are:

1. Time treatments of dogs with ivermectin to take advantage of the reduction in environmental stages of parasites due to seasonal effects. Concentration of treatments during the dry season would coincide with reduced environmental stages of hookworm due to the climate. This would also help reduce the potential for helminth resistance to ivermectin by removing all stages at once.

2. Reduce the number of ivermectin treatments given to dogs per year after establishment of the program. Once scabies rates have reduced (after 3 treatments at the present compliance rates), the number of treatments can be reduced to 2-3 per year if given at appropriate times.

3. Once the program is established and scabies rates have reduced, concentrate ivermectin treatments in male dogs and puppies if not all dogs are able to be caught.

4. Improve rate of capture of female dogs for proligestone treatments. In communities where there are fewer dogs, this may be easier. When compliance rates are high, the frequency of treatments can be reduced to suit the ivermectin treatments.

5. Discourage people from importing dogs (particularly puppies) from other areas. If more communities are involved in the program, there are less puppies available for importing/importing due to the breeding control.

Recommendations for Further Study

Recommendations for further study are:

2. Further clarification of the taxonomic and zoonotic status of canine parasites (such as *Sarcoptes scabiei* and *Giardia*) in Aboriginal communities using molecular techniques.

3. Correlation of molecular studies of zoonotic pathogens in Aboriginal communities with clinical presentations in people and outcomes of canine parasite treatment programs. For example, determining the true extent of lesions due to *Sarcoptes scabiei* var. *canis* rather than *Sarcoptes scabiei* var. *hominis*, which present different clinically.
### Appendix A

Comparison of *Ancylostoma caninum* and Human Hookworms*

<table>
<thead>
<tr>
<th>Parasite</th>
<th>Route of Transmission</th>
<th>Primary Infection</th>
<th>Duration of Infection</th>
<th>Fecundity (Eggs per Female Worm per Day)</th>
<th>Blood Loss (mL per Worm per Day)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Percutaneous</td>
<td>Oral</td>
<td>Transmammary or Transplacental</td>
<td>Pre-Patent Infection (Days)</td>
<td>(Weeks)</td>
</tr>
<tr>
<td><em>Ancylostoma caninum</em></td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>14</td>
<td>62 (up to 100)</td>
</tr>
<tr>
<td><em>Ancylostoma duodenale</em></td>
<td>Yes</td>
<td>Yes</td>
<td>Possibly</td>
<td>43</td>
<td>312</td>
</tr>
<tr>
<td><em>Necator americanus</em></td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>47</td>
<td>780</td>
</tr>
</tbody>
</table>

* Modified from Behnke (1990) and Bundy and Keymer (1991)
Appendix B

Scabies Scoring System

Score 1: 0-25% alopecia

Score 2: 25-50% alopecia

Score 3: 50-71% alopecia

Score 4: 75-100% alopecia
Appendix C

Techniques for the Recovery of Parasite Eggs Larvae and Cysts from Preserved and Fresh Faeces


The formalin ethyl acetate sedimentation technique used in the present study is based on the method described by Young, Bullock, Melvin and Spruill (1979):

- Strain approximately 10mL of well-mixed formalinised faecal suspension through gauze into a 15mL conical centrifuge tube and centrifuge for 2 minutes at 2000 to 2500 rpm.
- Decant supernatant and retrieve approximately 0.5mL of sedimented faeces (Adjust amount of sediment to this amount if more or less is left after centrifuging).
- Add 9mL of neutral buffered 10% formalin to the tube and thoroughly mix with the sediment.
- Add 4mL of ethyl acetate to each tube, stopper tube and shake in an inverted position for 30 seconds.
- Centrifuge each tube for 2 minutes at 1800 to 2000 rpm.
- Four layers appear in each tube; solvent, a plug of debris, fromalin and sediment. Loosen plug of debris with an applicator stick and decant the top three layers.
- Prepare an iodine-stained mount of the resultant sediment by adding a drop of Lugols iodine to one drop of sediment on a slide. Place a coverslip on slide and examine the entire slide with a light microscope.

Zinc Sulphate Centrifugal Flotation Technique for the Recovery of Parasite Eggs and Cysts in Fresh Faecal Specimens.

The zinc sulphate centrifugal flotation technique used in the present thesis is described in Dunsmore and Shaw (1990):

- Emulsify 0.5g of fresh faeces in 9-10mL of water in a centrifuge tube and centrifuge at 2000 rpm for 2 minutes.
- Pour off supernatant and re-emulsify in 9-10mL of ZnSO₄ (33.1g in 100mL distilled water, SG 1.2).
- Centrifuge at 1800 rpm for 2 minutes with no caps on the tubes.
- After centrifuging, touch the meniscus with a platinum wire loop and transfer 2-3 loopsful to a slide.
- Stain with lugols Iodine and apply a coverslip. Examine promptly with a light microscope.
### Appendix D

**Morphometric Characteristics for Differentiation of Canine Parasites**

<table>
<thead>
<tr>
<th>Parasite</th>
<th>Morphometric Characteristics</th>
<th>Distinguishing Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Faecal Specimens</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Ancylostoma caninum</em></td>
<td>Eggs containing morula; smooth thin shelled. Occasionally identify L1 larvae in faecal specimens that have not been preserved immediately; larvae have long tubular buccal canals; genital primordium cannot be seen (Orihel and Ash, 1997); about 400 μm long.</td>
<td></td>
</tr>
<tr>
<td><em>Ancylostoma braziliense</em></td>
<td>Eggs containing morula; smooth thin shelled.</td>
<td>Larvae similar to <em>A. caninum</em>.</td>
</tr>
<tr>
<td><em>Uncinaria stenocephala</em></td>
<td>Eggs containing morula; smooth thin shelled.</td>
<td></td>
</tr>
<tr>
<td><em>Toxocara canis</em></td>
<td>Unembryonated eggs; thick pitted shell.</td>
<td></td>
</tr>
<tr>
<td><em>Strongyloides stercoralis</em></td>
<td>Embryonated eggs; thin shelled. Most often identified as L1 larvae; larvae have short tubular buccal canals; genital primordium is prominent (Orihel and Ash, 1997); about 400 μm long.</td>
<td></td>
</tr>
<tr>
<td><em>Spirocerca lupi</em></td>
<td>Embryonated eggs; thick shelled; ‘folded’ larvae.</td>
<td></td>
</tr>
<tr>
<td><em>Trichuris vulpis</em></td>
<td>Unembryonated eggs; bipolar plugs.</td>
<td></td>
</tr>
<tr>
<td><em>Echinococcus/Taenia</em></td>
<td>Embryonated eggs with striated embryophore; may be shed in proglottids.</td>
<td></td>
</tr>
<tr>
<td><em>Dipylidium caninum</em></td>
<td>Embryonated eggs in an egg capsule containing about 30 eggs; contained in proglottids.</td>
<td></td>
</tr>
<tr>
<td><em>Spirometra erinaceieuropai</em></td>
<td>Unembryonated eggs; operculate; pointed at each end.</td>
<td></td>
</tr>
<tr>
<td><em>Giardia duodenalis</em></td>
<td>Thin walled cysts; axostyle present.</td>
<td></td>
</tr>
<tr>
<td><em>Isospora canis</em></td>
<td>Unsporulated oocysts; smooth; pale yellow.</td>
<td></td>
</tr>
<tr>
<td><em>Isospora ohiensis</em></td>
<td>Unsporulated oocysts.</td>
<td></td>
</tr>
<tr>
<td><em>Sarcocystis spp</em></td>
<td>Sporocysts containing 4 sporozoites.</td>
<td></td>
</tr>
<tr>
<td><em>Hammondia heydorni</em></td>
<td>Unsporulated oocysts; smooth; colourless.</td>
<td></td>
</tr>
<tr>
<td><strong>Blood Specimens</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Dirofilaria immitis</em></td>
<td>Microfilariae; asymmetrical notch at anterior end usually present; cephalic hook absent.</td>
<td></td>
</tr>
<tr>
<td><em>Dipetalonema reconditum</em></td>
<td>Microfilariae; asymmetrical notch at anterior end absent; cephalic hook present.</td>
<td></td>
</tr>
</tbody>
</table>

* Adapted from Dunsmore and Shaw (1990), Soulsby (1982)
Appendix E
Skin Disease Survey Form for 0-15 years old Children

DOG HEALTH PROGRAMMES
COMMUNITY SCREENING
SKIN DISEASES

NAME: ________________________________

COMMUNITY: __________________________

DATE OF BIRTH: ______________________

DATE OF SCREEN: 5-06-92

SKIN DISEASE (mark site of figures)
A. SCALP SORES .................................................. [ ]
B. IMPETIGO .................................................. [ ]
C. SCABIES (BURROWS, NODULES, VESICLES SECONDARY INFECTION) ........................................ [ ]
D. BOILS .................................................. [ ]
E. TINEA .................................................. [ ]
F. UNDEFINED .................................................. [ ]

FORM/27052DL1
Appendix F

Telephone Survey

DOG OWNERSHIP IN KIMBERLEY TOWNS

<table>
<thead>
<tr>
<th>Town</th>
<th>Telephone number</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Kununurra</td>
<td></td>
<td>(1)</td>
</tr>
<tr>
<td>Halls Creek</td>
<td></td>
<td>(2)</td>
</tr>
<tr>
<td>Fitzroy Crossing</td>
<td></td>
<td>(3)</td>
</tr>
<tr>
<td>Derby</td>
<td></td>
<td>(4)</td>
</tr>
<tr>
<td>Broome</td>
<td></td>
<td>(5)</td>
</tr>
</tbody>
</table>

**Record of interviewer contacts**

<table>
<thead>
<tr>
<th>Date</th>
<th>Time</th>
<th>Action</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Checklist**

- Questionnaire completed
- Questionnaire data entered
- Questionnaire data checked
Day
Month
Phone number ________________

1. Hello, this is Kathryn Wilks from the School of Veterinary Studies at Murdoch University. We are conducting a random telephone survey of dog ownership in the Kimberley and I would like to know if you keep any dogs at your household?

Yes = 1
No = 2
Don’t know = 8

2. Are you the owner of the dog?
Yes = question 4
No = question 3

3. Could I please speak to the owner to ask them a few questions about their dog?

Hello, this is Kathryn Wilks from the School of Veterinary Studies at Murdoch University. We are conducting a random telephone survey of dog ownership in the Kimberley.

4. Can you spare me 5-10 minutes to answer some questions about you and your dog?

5. Firstly, would you mind telling me what year you were born in?

Now about your dogs.

1. How many dogs, including puppies, do you have at your household?

2. What is the name of your (oldest) dog?

   __________  __________  ________  ________

3. How old is ______? (years)

   . . . . . . . .
4. Is ______ a purebred dog?

Yes  purebred  Go to Question 5
No   mixed   Go to Question 6
Don’t know  Go to Question 6

5. What breed is ______?
   Fill in Breed Code

6. What sex is ______?

Female = 1
Male = 2
Don’t know = 8

7. Has ______ been desexed?

Yes  = 1    go to question
No   = 2
No and female = question 8
Don’t know = 8

8. Has ______ had any puppies in the last year?

9. How many puppies did she have?

10. Of the live puppies born, how many were female?
11. Where did you get ______?

other  ________  ________  ________  ________

Knx =1
Halls Ck =2
FX =3
Derby =4
Broome =5

12. How old was ______ when you got him/her?

. . . .

13. Have you lost any dogs in the last year?

Yes = question 11
No = question 13

14. How many dogs have you lost?

15. What happened to them?

__________

__________

Thank you very much for answering these questions and for your time.
Appendix G

Dog Population Structure, Distribution and Dynamics – Comparative Studies

Dog Population Structure – Comparative Studies

<table>
<thead>
<tr>
<th></th>
<th>Greater Las Vegas Area</th>
<th>Manhattan, Kansas</th>
<th>Salina, Kansas</th>
<th>Yolo county California</th>
<th>Alameda and Contra Costa Counties, California</th>
<th>Champaign County, Illinois</th>
<th>St Joseph County, Indiana</th>
<th>Ontario, Canada</th>
<th>South Africa</th>
<th>Philippines</th>
<th>Perth</th>
</tr>
</thead>
<tbody>
<tr>
<td>Data collection</td>
<td>postal questionnaire</td>
<td>postal questionnaire</td>
<td>household interview</td>
<td>postal questionnaire telephone survey</td>
<td>telephone survey</td>
<td>telephone survey</td>
<td>postal questionnaire household interview</td>
<td>household interview telephone survey</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Proportion of population interviewed</td>
<td>1500 houses</td>
<td>1500 houses</td>
<td>4% of population 3% to 7% of regions</td>
<td>5% of households</td>
<td>1.3% of households</td>
<td>3.6% of households</td>
<td>455 houses, population 83500</td>
<td>2000 houses</td>
<td>5789 houses</td>
<td>0.2% of population</td>
<td></td>
</tr>
<tr>
<td>Percentage respondents</td>
<td>30%</td>
<td>40%</td>
<td>82.9%</td>
<td>86.9%</td>
<td>62.3%</td>
<td>75.3%</td>
<td>65%</td>
<td>N/R</td>
<td>N/R</td>
<td>62%</td>
<td></td>
</tr>
</tbody>
</table>

**SEX**

<table>
<thead>
<tr>
<th></th>
<th>Males (%)</th>
<th>N/R</th>
<th>Females (%)</th>
<th>44.1</th>
<th>48.7</th>
<th>49.1</th>
<th>51.7</th>
<th>46.1</th>
<th>53.9</th>
<th>N/R</th>
<th>47</th>
</tr>
</thead>
<tbody>
<tr>
<td>Percentage of males castrated</td>
<td>26.5</td>
<td>12.4</td>
<td>26.5</td>
<td>4.0</td>
<td>7.4</td>
<td>32.04 (all dogs)</td>
<td>52.9</td>
<td>&lt;10</td>
<td>N/R</td>
<td>N/R</td>
<td>33.8</td>
</tr>
<tr>
<td>Percentage of females spayed</td>
<td>77</td>
<td>66.7</td>
<td>69.6</td>
<td>31.4</td>
<td>47.8</td>
<td>63.1</td>
<td>33</td>
<td>N/R</td>
<td>N/R</td>
<td>N/R</td>
<td>69.4</td>
</tr>
</tbody>
</table>

**AGE**

|                  | Average age (years) | 5.32 | 4.6 | 5.9 | N/R | N/R | 4.64 | 5 | 6.12 | N/R | 1.89 | 6.1 |

394
Dog Population Distribution – Comparative Studies

<table>
<thead>
<tr>
<th>Greater Las Vegas Area</th>
<th>Manhattan, Kansas</th>
<th>Salina, Kansas</th>
<th>Yolo county California</th>
<th>Alameda and Contra Costa Counties, California</th>
<th>Champaign County, Illinois</th>
<th>St Joseph County, Indiana</th>
<th>Ontario, Canada</th>
<th>South Africa</th>
<th>Philippines</th>
<th>Perth</th>
</tr>
</thead>
</table>

**HOUSEHOLDS**

<table>
<thead>
<tr>
<th>Dogs/household</th>
<th>0.69</th>
<th>0.58</th>
<th>0.61</th>
<th>0.73*</th>
<th>0.42*</th>
<th>0.467</th>
<th>0.59*</th>
<th>N/R</th>
<th>0.74</th>
<th>0.43*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Households with dogs (%)</td>
<td>46</td>
<td>43</td>
<td>45.2</td>
<td>50</td>
<td>77 (rural)</td>
<td>35.8</td>
<td>37.3</td>
<td>37.9*</td>
<td>65 overall</td>
<td>37</td>
</tr>
<tr>
<td>Dogs/dog-owning Household (DOH)</td>
<td>1.49</td>
<td>1.36</td>
<td>1.34</td>
<td>1.3 to 1.7</td>
<td>1.17*</td>
<td>1.24</td>
<td>1.56*</td>
<td>N/R</td>
<td>2.0*</td>
<td>N/R</td>
</tr>
</tbody>
</table>

**OWNERS**

<table>
<thead>
<tr>
<th>Dogs to people ratio</th>
<th>1:3.9</th>
<th>1:4.1</th>
<th>1:4.6</th>
<th>N/R</th>
<th>1:7.3</th>
<th>1:7.4</th>
<th>N/R</th>
<th>N/R</th>
<th>N/R</th>
<th>1.7</th>
<th>1.7</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex of owners</td>
<td>N/R</td>
<td>N/R</td>
<td>N/R</td>
<td>no significance</td>
<td>N/R</td>
<td>32.9% males</td>
<td>N/R</td>
<td>N/R</td>
<td>N/R</td>
<td>N/R</td>
<td></td>
</tr>
<tr>
<td>Age of owners who keep most dogs</td>
<td>N/R</td>
<td>N/R</td>
<td>N/R</td>
<td>N/R</td>
<td>18 to 29 years (27.9%)</td>
<td>N/R</td>
<td>N/R</td>
<td>55 to 64</td>
<td>N/R</td>
<td>N/R</td>
<td></td>
</tr>
</tbody>
</table>

* Extrapolated from data presented.
### Dog Population Dynamics – Comparative Studies

<table>
<thead>
<tr>
<th></th>
<th>Greater Las Vegas Area</th>
<th>Manhattan, Kansas</th>
<th>Salina, Kansas</th>
<th>Yolo county California</th>
<th>Alameda and Contra Costa Counties, California</th>
<th>Champaign County, Illinois</th>
<th>St Joseph County, Indiana</th>
<th>Ontario, Canada</th>
<th>South Africa</th>
<th>Perth</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rate of change of population per year</td>
<td>0.96</td>
<td>0.975</td>
<td>0.87</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age dependent survival rates</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0–weaning</td>
<td>0.87</td>
<td>0.73</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Weaning-1 yr</td>
<td>0.95</td>
<td>0.87</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ratio of puppies to juveniles</td>
<td>2.4</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Percentage of new (acquired) dogs to households</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ratio of dogs acquired outside to those born in community</td>
<td>0.5:1</td>
<td>1.26:1</td>
<td>1.16:1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>14.1%</td>
</tr>
<tr>
<td>Percentage of entire females reproducing in previous 12 months</td>
<td>17</td>
<td>12.9</td>
<td>6.2</td>
<td>20*</td>
<td>10.5</td>
<td>4.6 (all dogs)</td>
<td>14 (all dogs)</td>
<td>19.2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Average age at death (yrs)</td>
<td>9.57</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mortality rate (%)</td>
<td>6.4</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Extrapolated from data presented.
Appendix H

Age Prevalence of Scabies, Hookworm and *Giardia* in Kimberley Community Dogs

Age Prevalence of Scabies in Puppies

<table>
<thead>
<tr>
<th>Puppies</th>
<th>Coastal Region</th>
<th>Central Region</th>
<th>Eastern Region</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number of Puppies</td>
<td>Percentage with Scabies</td>
<td>Odds Ratio*</td>
</tr>
<tr>
<td>Mar-92</td>
<td>11</td>
<td>18.2</td>
<td>1.5</td>
</tr>
<tr>
<td>Jun-92</td>
<td>22</td>
<td>18.2</td>
<td>1.1</td>
</tr>
<tr>
<td>Sep-92</td>
<td>15</td>
<td>6.7</td>
<td>0.5</td>
</tr>
<tr>
<td>Dec-92</td>
<td>11</td>
<td>0</td>
<td>-</td>
</tr>
<tr>
<td>Mar-93</td>
<td>18</td>
<td>5.6</td>
<td>2.6</td>
</tr>
<tr>
<td>Jun-93</td>
<td>13</td>
<td>0</td>
<td>-</td>
</tr>
<tr>
<td>Sep-93</td>
<td>36</td>
<td>0</td>
<td>-</td>
</tr>
<tr>
<td>Dec-93</td>
<td>21</td>
<td>4.8</td>
<td>1.6</td>
</tr>
<tr>
<td>Mar-94</td>
<td>18</td>
<td>0</td>
<td>-</td>
</tr>
<tr>
<td>Jun-94</td>
<td>16</td>
<td>0</td>
<td>-</td>
</tr>
<tr>
<td>Sep-94</td>
<td>23</td>
<td>0</td>
<td>-</td>
</tr>
</tbody>
</table>

*Odds ratio for infection with scabies in puppies compared with all other age groups*
### Age Prevalence of Scabies in Adult Dogs

<table>
<thead>
<tr>
<th>Adults</th>
<th>Coastal Region</th>
<th>Central Region</th>
<th>Eastern Region</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number of Adults</td>
<td>Percentage with Scabies</td>
<td>Odds Ratio*</td>
</tr>
<tr>
<td></td>
<td>65</td>
<td>15.4</td>
<td>1.8</td>
</tr>
<tr>
<td></td>
<td>85</td>
<td>14.1</td>
<td>0.5</td>
</tr>
<tr>
<td></td>
<td>71</td>
<td>9.9</td>
<td>0.6</td>
</tr>
<tr>
<td></td>
<td>89</td>
<td>2.2</td>
<td>0.3</td>
</tr>
<tr>
<td></td>
<td>81</td>
<td>2.5</td>
<td>0.6</td>
</tr>
<tr>
<td></td>
<td>120</td>
<td>0</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>83</td>
<td>1.2</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>84</td>
<td>3.4</td>
<td>1.2</td>
</tr>
<tr>
<td></td>
<td>90</td>
<td>1.1</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>90</td>
<td>1.1</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>73</td>
<td>0</td>
<td>-</td>
</tr>
</tbody>
</table>

*Odds ratio for infection with scabies in adults compared with all other age groups

* Statistically significant [95% Confidence Intervals in brackets]
### Age-Prevalence of Hookworm in Puppies and Adults

<table>
<thead>
<tr>
<th>Puppies</th>
<th>Coastal Region</th>
<th>Central Region</th>
<th>Eastern Region</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number of Puppies</td>
<td>Positive (%)</td>
<td>Odds Ratio (Puppy and Positive)</td>
</tr>
<tr>
<td>Jun-92</td>
<td>8</td>
<td>75</td>
<td>1.5</td>
</tr>
<tr>
<td>Sep-92</td>
<td>3</td>
<td>33.3</td>
<td>2.2</td>
</tr>
<tr>
<td>Mar-93</td>
<td>2</td>
<td>100</td>
<td>-</td>
</tr>
<tr>
<td>Sep-93</td>
<td>17</td>
<td>58.8</td>
<td>4.1* [1.3, 12.8]</td>
</tr>
<tr>
<td>Dec-93</td>
<td>16</td>
<td>43.7</td>
<td>3.0* [1.0, 9.2]</td>
</tr>
<tr>
<td>Mar-94</td>
<td>11</td>
<td>47.6</td>
<td>6.6</td>
</tr>
<tr>
<td>Jun-94</td>
<td>8</td>
<td>20</td>
<td>1.1</td>
</tr>
<tr>
<td>Sep-94</td>
<td>18</td>
<td>61.1</td>
<td>27.9* [7.0, 111.2]</td>
</tr>
</tbody>
</table>

*Statistically significant [95% Confidence Intervals in brackets]

<table>
<thead>
<tr>
<th>Adults</th>
<th>Coastal Region</th>
<th>Central Region</th>
<th>Eastern Region</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number of Adults</td>
<td>Positive (%)</td>
<td>Odds Ratio (Puppy and Positive)</td>
</tr>
<tr>
<td>Jun-92</td>
<td>34</td>
<td>64.7</td>
<td>0.6</td>
</tr>
<tr>
<td>Sep-92</td>
<td>29</td>
<td>66.7</td>
<td>1.1</td>
</tr>
<tr>
<td>Mar-93</td>
<td>39</td>
<td>84.6</td>
<td>0.3</td>
</tr>
<tr>
<td>Sep-93</td>
<td>58</td>
<td>45.8</td>
<td>0.2* [0.10, 0.7]</td>
</tr>
<tr>
<td>Dec-93</td>
<td>68</td>
<td>52.2</td>
<td>0.2* [0.10, 0.54]</td>
</tr>
<tr>
<td>Mar-94</td>
<td>54</td>
<td>61.2</td>
<td>0.2* [0.1, 0.7]</td>
</tr>
<tr>
<td>Jun-94</td>
<td>55</td>
<td>63.2</td>
<td>0.7</td>
</tr>
<tr>
<td>Sep-94</td>
<td>63</td>
<td>26.7</td>
<td>0.2* [0.1, 0.6]</td>
</tr>
</tbody>
</table>
### Age Prevalence of *Giardia* in Puppies

<table>
<thead>
<tr>
<th>Puppies</th>
<th>Coastal Region</th>
<th>Central Region</th>
<th>Eastern Region</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number Sampled</td>
<td>Positive (%)</td>
<td>Odds ratio</td>
</tr>
<tr>
<td>Jun-92</td>
<td>8</td>
<td>12.5</td>
<td>0.71</td>
</tr>
<tr>
<td>Sep-92</td>
<td>3</td>
<td>33.3</td>
<td>-</td>
</tr>
<tr>
<td>Mar-93</td>
<td>2</td>
<td>100</td>
<td>-</td>
</tr>
<tr>
<td>Sep-93</td>
<td>17</td>
<td>29.4</td>
<td>4.42* [1.1, 17.7]</td>
</tr>
<tr>
<td>Dec-93</td>
<td>16</td>
<td>37.5</td>
<td>5.18* [1.5, 18.0]</td>
</tr>
<tr>
<td>Mar-94</td>
<td>11</td>
<td>36.4</td>
<td>6.9* [1.7, 28.6]</td>
</tr>
<tr>
<td>Jun-94</td>
<td>8</td>
<td>37.5</td>
<td>4.72* [1.0, 23.6]</td>
</tr>
<tr>
<td>Sep-94</td>
<td>18</td>
<td>27.8</td>
<td>28.5* [3.1, 263.8]</td>
</tr>
<tr>
<td>Pooled data***</td>
<td>39.4</td>
<td>5.27* [3.0, 9.2]</td>
<td></td>
</tr>
</tbody>
</table>

*Statistically significant [95% Confidence Intervals in brackets]

***Data pooled for visits where no statistical difference was found [P>0.05]
REFERENCES


Giles WE (1889): *Australia Twice Traversed: The romance of exploration, being a narrative compiled from the journal of five exploring expeditions into and through central South Australia.* Libraries Board of South Australia. Adelaide. pp. 109.


Mawson PM (1968): Two species of Nematoda (Spirurida: Spiruridae) from Australian dasyurids. *Parasitology.* **58:** 75-78.


Nassar R, Moiser JE and Williams LW (1984): Study of the feline and canine populations in the

Nawalinski TA and Schad GA (1974): Arrested development in *Ancylostoma duodenale*: course of

Nawalinski TA, Schad GA and Chowdhury AB (1978a): Population biology of hookworms in
children in rural West Bengal. I. General parasitological observations. *American Journal
of Tropical Medicine and Hygiene*. **27**: 1152-1161.

Nawalinski TA, Schad GA and Chowdhury AB (1978b): Population biology of hookworms in
children in rural West Bengal. II. Acquisition and loss of hookworms. *American Journal
of Tropical Medicine and Hygiene*. **27**: 1162-1173.

and chronic rheumatic heart disease in Yarrabah Aboriginal community, north Queensland.

*Canadian Veterinary Journal*. **7**: 43.

Nicholas WL, Stewart AC and Walker JC (1986): Toxocariasis: a serological survey of blood
donors in the Australian Capital Territory together with observations on the risks of
infection. *Transactions of the Royal Society of Tropical Medicine and Hygiene*. **80**: 217-
221.

Nimmo GR, Tinniswood RD, Nuttall N, Baker GM and McDonald B (1992): Group A

Nind S (1831): Description of the natives of King George’s Sound (Swan River Colony) and


parasitic infections in onchocerciasis and the occurrence of adverse reactions after

Nokes C and Bundy DAP (1993): Compliance and absenteeism in school children: implications
for helminth control. *Transactions of the Royal Society of Tropical Medicine and Hygiene*. **87**: 148-152.


Russel RC and Geary MJ (1996): The influence of microfilarial density of dog heartworm, Dirofilaria immitis, on infection rate and survival of Aedes notoscriptus and Culex annulorostris from Australia. Medical and Veterinary Entomology. 10: 29-34.


