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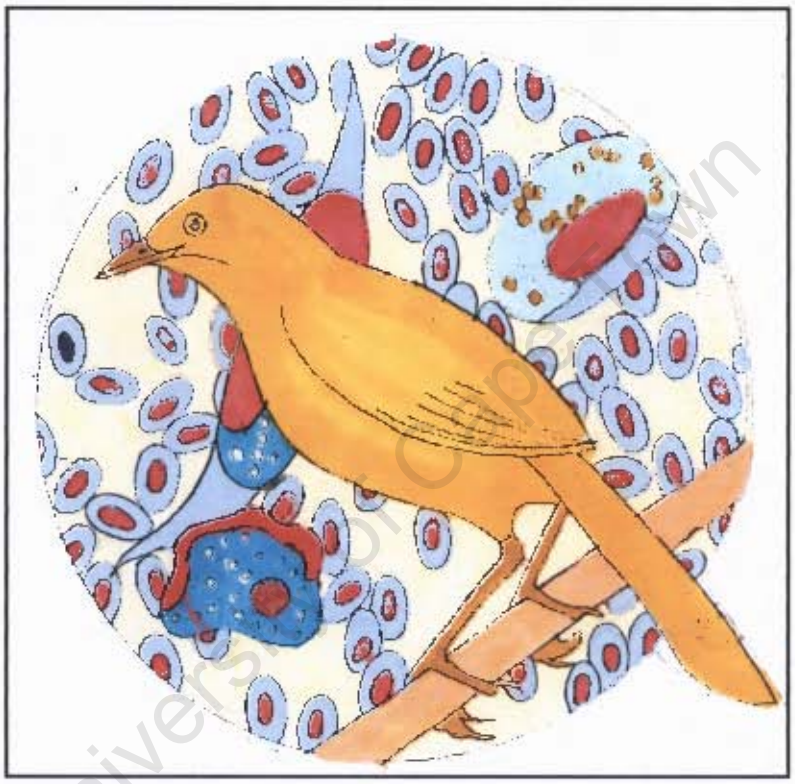
PATTERNS OF AVIAN HAEMOSPORIDIA IN BIRDS WITHIN THE GREATER CAPE TOWN AREA

Albert Schultz



University of Cape Town

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Master in Science

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Avian Demography Unit, Department of Statistical Sciences, Faculty of Science
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"Where the telescope ends, the microscope begins. Which of the two has the grander view?"

Victor Hugo
Les Miserables 1862, Book 3

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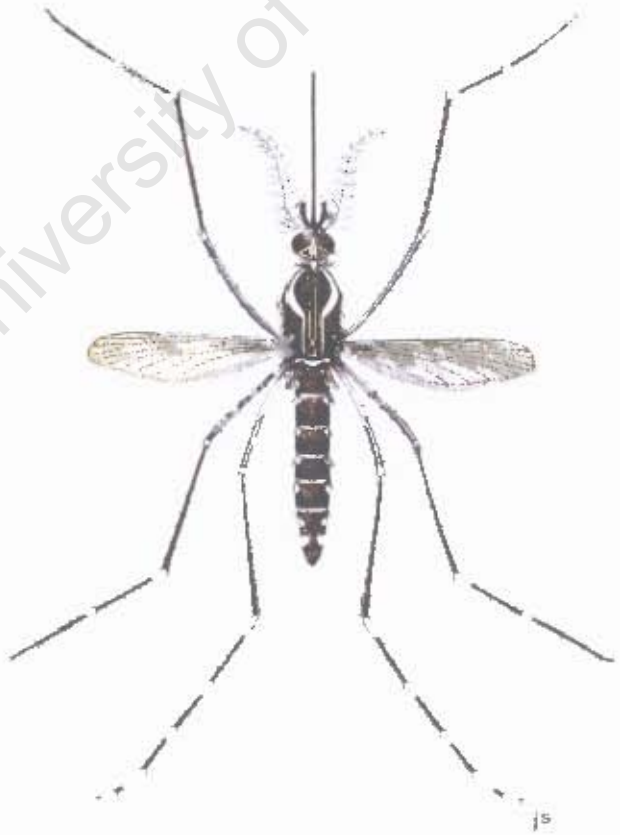

Aedes aegypti (taken from Gillett 1972)

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Abstract

Schultz, A. B. 2007. *Patterns of avian haemosporidia in birds within the Greater Cape Town Area.* M.Sc. thesis, Avian Demography Unit, Department of Statistical Sciences, Faculty of Science, University of Cape Town, Rondebosch 7701, South Africa.

Arthropods have been associated with disease and pestilence since antiquity. Only in the 19th century with the study by Patrick Manson, implicating the mosquito as vector for transmitting *Wuchereria bancrofti*, did modern medical-veterinary entomology begin. Many similar discoveries of blood-feeding arthropods transmitting pathogens followed in rapid succession, the most notable being the discovery by Sir Ronald Ross of the mosquito as vector for malaria in 1898.

Success of a vector depends on its particular location, abundance and frequency of feeding on the host, in which the circulating peripheral blood contains stages of the parasite's development. Parasite species is thus determined by vector patterns of selection and exposure to hosts. Successful transmission of infection requires the vector and parasite to have evolved through selection, enabling the survival of the vector to support parasite development and maturation for transmission. Transmission of infection is dependent on ambient temperature and humidity, occurring more rapidly in the tropics than at temperate latitudes. Where seasonal changes are slight, transmission occurs throughout the year.

The phylum Arthropodia is the most diverse group of organisms, surpassing all other species on earth, being distributed throughout all regions and having colonized all terrestrial and freshwater ecological niches. The sub-class Diptera (true flies) is the most medically and veterinary important in terms of species implicated in disease transmission, of which Culicidae (mosquitoes) are the most widely studied. Diptera, range abundantly worldwide, with the population from Afrotropical Region consisting of 16 000 species in 24 families. Of importance as vectors for transmission of avian haemosporidia parasites are the families Ceratopogonidae (biting midges), Hippoboscidae (louse flies), Simuliidae (blackflies), and Culicidae (mosquitoes).

Ceratopogonidae are the smallest of the blood sucking-flies, often unnoticed despite their ferocity. Biting midges have a worldwide distribution, with 50 genera recorded from the Afrotropical Region. Only four genera attack humans and animals, of which *Culicoides* is the most important. Species of this genus transmits *Haemoproteus* and *Leucocytozoon caulleryi* to birds—the only *Leucocytozoon* species to be transmitted by biting midges.

Hippoboscidae, known as "louse flies", are highly specialized blood-sucking ectoparasites occurring on mammals and birds. Most species have evolved to be wingless, with a number of exceptions, including species parasitizing birds. Hippoboscidae have worldwide distribution with 50 species occurring in Africa, of which the majority are avian species. Thirty-seven species in 12 genera from three subfamilies have been recorded in southern Africa. Louse flies are vectors of *Haemoproteus* species of which the most notable genera are *Pseudolynchia, Stilbometopa, Icosta* and *Ornithomyia.*

Simuliidae, known as "black flies", have worldwide distribution, being abundant at locations having suitable rivers and streams of fast-flowing water. Black flies have gained notoriety by transmitting several pathogenic organisms, which lead to grave economic consequences. Thirty-nine species have been recorded from southern Africa. The avian malarial-like disease, *Leucocytozoon* species are transmitted by black flies, of which *Simulium, Prosimulium, Cnephia,* and *Austrosimulium* are vectors and hosts.

The family Culicidae (mosquitoes) is the most studied family of the arthropods, due to its importance as a vector of human disease, namely malaria. Distribution covers nearly every region and continent of the world, comprising 3 500 described species, of which 30 are endemic to the Afrotropical Region. Thirteen genera with 220 species occur in southern Africa. The subfamily Anophelinae exclusively transmits human malaria. Thirty-eight species have been identified as transmitting avian malaria, of which *Plasmodium relictum* is the commonest.

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Every species of bird has at least one species of endoparasite or ectoparasite, if not several. Thus it cannot be taken for granted that a particular individual avian species is free from haemosporidian infections. Birds have the richest diversity of intracellular, single cell, protozoan parasites with complicated life cycles, changing between the vector as definitive host (blood-feeding arthropod) and bird as intermediate host. Avian haemosporidian parasites belong to the order Haemospororida, to which the families Haemoprotidae, Plasmodiidae, Garniidae and Leucocytozoidae belong, consisting of the genera *Haemoproteus, Plasmodium, Fallisia* and *Leucocytozoon.* Speculation exists as to the true evolutionary lineage of avian haemosporidian parasites. This controversy also extends to avian haemosporidian taxonomists with respect to the valid number of haemosporidian species, due to the principle of "one host-one parasite" conceived by G. F. Bennett. Many avian haemosporidian parasitologists still cling to this principle.

Infection rates of birds average 10% of individuals in a population, while prevalence varies between 0—100% depending on avian species, ecological factors and the different haemosporidian taxa in the corresponding host population.

The *Haemoproteus* parasite is primarily of birds and reptiles, and is the most common haemosporidian encountered in birds. There are 132 valid species worldwide, 69 of which have been identified from Afrotropical birds. They occur in 50% of birds examined worldwide and in 19.4% of those found in the Afrotropical population. The discoid form has not yet been recorded in Afrotropical birds.

The *Leucocytozoon* species are parasites solely of birds, being the second most frequently encountered group, occurring in 30% of birds examined worldwide, and in 8.1% of birds from the Afrotropical Region. Worldwide distribution consists of 35 valid species, with 26 in the Afrotropical Region. *Leucocytozoon* species have two morphological forms—round and fusiform.

The genus *Plasmodium* is set apart from *Haemoproteus* and *Leucocytozoon* due to its extensive use as an experimental laboratory model. Avian plasmodia parasites have a worldwide distribution of 38 valid species, with 13 recorded from the Afrotropical Region. Of the 45% of world avian species that have been researched, *Plasmodium* species have been found to parasitise 25% of them. Only 3.5% of birds examined from the Afrotropical Region have been found infected with *Plasmodium.*

A total of 9 304 birds representing 29 families, 55 genera and 78 species were captured in mist nets at 10 study sites within the Greater Cape Town Area between February 1993 and February 1995. Avian haemosporidian parasites were found in 20 avian families examined representing 41 species from three genera, of these, 1 978 (21.2%) individuals were found to be infected with one or more species. The highest occurrence of infection was found with species of *Leucocytozoon* (12.6% of infected birds), followed by *Haemoproteus* (9.6%) and *Plasmodium* with 0.6%. Prevalence of infection of the three genera of avian haemosporidian parasites varied from 4.5% at Patryskraal to 28.0% at Tygerberg Nature Reserve. The 10 sites can be classified into three groups: Patryskraal, Rondevlei and Mowbray having low infection rates; Koeberg, Glencairn and Durbanville having intermediate infection rates; and Tygerberg, Goedeontmoeting, Kirstenbosch and Bettys Bay with high infection rates. Avian haemosporidian parasites were found in 20 avian families consisting of 41 species from three genera. Columbidae was the avian family with the largest sample size infected by *Haemoproteus* spp. Ploceidae and Sylviidae were infected by all three avian haemosporidian genera, while Ploceidae was the most heavily infected with nine species of avian haemosporidia. The high prevalence of *Leucocytozoon* infections in birds from Bettys Bay, Kirstenbosch, Tygerberg, and Glencairn, results from the presence, at these sites, of fast-flowing water, to which Simuliidae are adapted.

Durbanville Nature Reserve, Glencairn, Goedeontmoeting, and Tygerberg Nature Reserve were selected for detailed analysis due to their adequate overall sample sizes. All these sites showed similar seasonal patterns with two seasonal peaks. The first coincided with the end of the rainy season and the second during January and February, when bird reproduction from spring has increased population size, resulting in large numbers of immature birds susceptible to infection. Prevalence of infections had two seasonal lows without a complete winter interruption. The first occurred in late autumn-early winter.

The second, known as "spring relapse", occurred during spring resulting from acute primary parasitaemia, which follows a peak in haemosporidian infection. Seasonal prevalence pattern of active avian haemosporidian transmission throughout the year as recorded here, are typically encountered at medium latitudes with a mild climate and warm seasons.

Avian haemosporidian parasites are classified according to the avian families or sub-families that they infect-this is also taken as the maximum level of specificity for the practice of avian haemosporidian taxonomy. Description of the new species is based upon morphology of gametocyte development in the peripheral blood of the avian host. This does not distinguish between morphologically identical gametocytes from different avian families, as being identical species-nor is species or family level a valid taxonomic character. Thus *Haemoproteus* and *Leucocytozoon* species encountered in this study are considered to be relegated to synonymy with previously described morphologically similar, valid species in avian hosts of other families.

A sample of 945 adult Cape Weavers *Ploceus capensis* were mist-netted at Goedeontmoeting during a two year period with a bias towards 632 male and 308 female birds. Blood smears were examined for avian haemosporidian parasites, of which 58.79% of the males and 61.90% of the females were infected. Six species of avian haemosporidian parasites from four genera were recorded. Overall, *Haemoproteus quelea* was the most commonly encountered infection, being 69.45% of the infected sample followed by *Leucocytozoon bouffardi* with 23.91%, *Plasmodium* species with 5.76% and *Trypanosoma everetti* occurring in 0.28% of the infected sample. Double infections occurred in 40 birds and 11.52% of the infections, with females having the most double infections.

Differences in infection prevalence between male and female Cape Weavers can be correlated to time spent being inactive at the nest. Also, females incubate within a nest to which a tunnel entrance is attached, providing protection from parasite transmission by vectors.

Commencement of the breeding season coincided with increased prevalence for both sexes, which correlates to energy expenditure on reproductive effort and results in reduced immunocompetence. The seasonal spring relapse was also observed, which is synchronized with the breeding season when vector intensity and activity peak due to population increase in avian species. A contributing factor to spring relapse may be related to the avian host's immunodefence system being compromised due to stress of the reproductive cycle.

In this study there was no indication that avian haemosporidian parasites impacted negatively on the avian host's mass or body condition. Short term impacts are possible when parasitemia intensity peaks over a number of days only, thereafter decreasing in intensity and not being detectable over long term investigations as undertaken in this study.

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This thesis could not have been undertaken without the faith Potchefstroomse Universiteit vir Christelike Hoër Onderwys (now Northwest University-Potchefstroom Campus) invested in me: Dr André Vosloo and Professor G. C. Loots in particular. I cannot express my sincere thanks enough to them for giving me the opportunity to pursue the scientific path, set in motion by Dr Paul Martin, through bird ringing. Thanks must go to him for his influence, which has culminated in this thesis.

My deepest appreciation and thanks must go to Professor Les Underhill who gave me this project, along with analysis of more than 6 000 blood smears taken by his late father, George Underhill. The core of the data processed in this document is in no small measure the result of George's love for birds, ringing, taking blood smears and the indomitable spirit of getting out there to ring. Benjamin Franklin said that a sundial in the shade was of no use. George was a sundial in full sun—thank you George. Acknowledgement towards the contribution of data must also go to Dr Terry Oatley and the late Colin Martin, whose blood smears also swelled the data base considerably.

Thanks with much appreciation must go to Dr Freddie de Moor and Dr Rob Palmer for their extremely valuable work on compiling an atlas of distributions of blackflies (Diptera: Simuliidae) in southern Africa. Similar work is totally lacking for biting midges, louseflies, and mosquitoes (Diptera: Ceratopogonidae, Hippoboscidae and Culicidae). It is essential for successfully studying avian haemosporidian parasites.

Appreciation and thanks to my co-supervisor, Dr Roy Earlé, who provided research material, advice and support from his faraway abode across the waters in England, leaves me without adequate words of thanks. Not to mention the hours he spent at the microscope analyzing 9 056 blood smears for the data presented here.

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Over many years, I have learnt that the writing of books, reports and treatises is not a process of non stop penning your thoughts from behind a desk. It is rather a process of living with your subject and of it growing in your thoughts and subconscious

Prof Nic Wiehahn (1929—2006)
Lewenswaardes

CHAPTER ONE

General discussion on avian haemosporidian parasite vectors and their ecology

Where'er there's a thistle to feed a linnet
And linnets are plenty, Thistles rife-
Or an acorn-cup to catch dew-drops in it
There's ample promise of further life.
Tom Hood (1835-1874)
Poets and Linnets

INTRODUCTION

Arthropods have been associated with disease and pestilence since antiquity (Service 1978). The problems caused by biting and annoying flies, mosquitoes, lice and bedbugs have been the subject of writers from Homer in the mid-8th century through Biblical times to Pliny (AD 23—79). Only in the 19th century did modern medical-veterinary entomology begin with the study by Patrick Manson (1844—1922). While working in China during 1877, he implicated the mosquito vector *Culex pipiens fatigans* with transmission of *Wuchereria bancrofti*, the cause of a highly pathogenic form of filariasis (Whitfield 2001). Many similar discovers of blood-feeding arthropods transmitting pathogens followed in rapid succession. T. Smith (1859—1934) and F. Kilbourne (1858—1936) discovered in 1891 that the tick *Boophilus annulatus* is the vector of *Babesia bigemina*, causing Texas cattle fever (Roberts & Janovy 2000) which then resulted in a successful eradication program. Sir Ronald Ross (1857—1932) in 1898, while working in India, unravelled the role mosquitoes played as vectors of avian malarial parasites from dead to healthy sparrows (Ross 1923, Harrison 1978). In the same year Giovani Grassi (1854—1925) described the cyclical development of the *Plasmodium* parasite in the anopheline mosquitoes. Other major discoveries regarding vector-parasite-host relationships continued well into the 20th century. The 50 year period of major discoveries, which started during 1877 with Patrick Manson, has been termed the "golden age of medical-veterinary entomology" (Philip & Rozeboom 1973), which has since played a major role in the development of human civilization and animal husbandry.

Numerous arthropods transmit pathogens to humans and animals. Of these, mosquitoes are the most important, followed by ticks, with viruses and bacteria the most diverse group, followed by protozoa and filarial nematodes (Durden & Mullen 2002). Transmission of pathogens by arthropod vectors is undertaken biologically or mechanically, with the biological means being the most common. Pathogens undergo development or reproduction in the arthropod vector before being biologically transmitted. Pathogens transmitted in this way include: *Plasmodium, Haemoproteus, Leucocytozoon,* African trypanosomiases and many others. Mechanical transmission takes place when pathogens are transmitted from arthropod vectors via contaminated appendages as with myxomatosis.

The word vector means "carrier" of parasites from one host to another. For a vector to be successful in a particular location it has to be abundant, and frequently feed on vertebrate hosts in which the circulating peripheral blood contains stages of the parasite's development. Efficiency of transmission is reduced when biting activity by the vector is undertaken at the wrong time, wrong location or on the wrong host.

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Thus parasite species is determined by vector patterns of selection and exposure to hosts. Anthropophagic vectors are important in the transmission of human parasites, readily entering houses to rest on interior surfaces and to selectively feed on humans. These vectors are thus termed endophilic which literally means "inside loving". Vectors which seldom enter houses are known as exophilic, meaning "outside loving". Zoophagic vectors feed primarily on blood of vertebrates other than humans, while Mammalophagic vectors feed primarily on mammals. Diversely different are ornithophagic vectors which are exophilic, feeding primarily on avian hosts, and are responsible for the maintenance of avian parasites.

Successful transmission of infection by the vector requires that the vector and parasite have evolved through selection, enabling the survival of the vector to support parasite development and maturation for transmission. Although the mosquito *Culex quinquefasciatus* becomes infected with western equine encephalomyelitis virus, development within the mosquito does not occur, thus the species is incapable of transmitting the virus (Reisen 2002). Arthropods under laboratory conditions are infected with parasites which develop, but survival in nature does not occur due to incorrect host selection. When the vector is infected, suitable hosts in adequate numbers must be available for re-feeding to ensure parasite transmission. If vectors feed on non-susceptible or dead-end hosts, transmission effectiveness is impaired. This then results in morbidity of the dead-end host as there has been no coevolution of host-parasite. This is most notable when African Penguins *Spheniscus demersus* at the Southern African Foundation for the Conservation of Coastal Birds (SANCCOB) are infected with avian malaria *Plasmodium juxtanuclearae* (Grim *et al.* 2003) or when western equine encephalomyelitis virus is transmitted to humans, being dead-end hosts (Sheals 1973, Reisen 2002). Transmission of infection, being ambient temperature and humidity dependent, occurs more rapidly in the tropics than at temperate latitudes, where transmission occurs mainly during summer months. Where seasonal changes are slight, transmission occurs throughout the year, as with the transmission of *Haemoproteus meleagridis* among turkeys in Florida (Atkinson 1988), malaria in Venezuela (Gabaldon & Ulloa 1980) and *Leucocytozoon smithi* in South Carolina (Noblet *et al.* 1975).

The phylum Arthropodia is the most diverse group of organisms, surpassing all other species on earth by 55% successfully colonizing practically all terrestrial and freshwater ecological niches. Arthropods are also the most densely distributed throughout all regions of the earth than members of any other phylum. Over one million species of insects have been described (Gullan & Cranston 2005) with true species richness probably in excess of six million remaining to be classified (Picker *et al.* 2002). When total proportions of previously unrecognized, and/or undescribed taxa are estimated by extrapolation, the derived species numbers range from four million to as many as six million species as realistic estimates (Gullan & Cranston 2005). Of all the insect orders implicated in disease transmission, Diptera (true flies) are the most medically and veterinary important species, of which Culicidae (mosquitoes) are the most widely studied. The order is divided into two suborders, consisting of the third-most species after Hymenoptera (250 000) and Lepidoptera (150 000), with 125 000 described species in 130 families (Gullen & Cranston 2005). Diptera range abundantly worldwide, from the tropics to the arctic in -10°C temperatures and from seashores to high mountains with immature stages of *Halaeomyia petrolei* occurring in North American crude oil (Harrison *et al.* 2003). The Afrotropical Region's population consists of 16 000 species in 24 families (Barraclough & Londt 1989) also occurring in as diverse and abundant habitats as the worldwide populations.

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Of importance as vectors for avian haemosporidian parasite transmission are Ceratopogonidae (biting midges), Hippoboscidae (louse flies), Simuliidae (black flies), and Culicidae (mosquitoes). The biology of each of these families is now briefly described.

FAMILY CERATOPOGONIDAE (biting midges)

Figure 1.1 Dorsal view of female *Culicoides nubeculosus* (modified from Freeman 1973)

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Ceratopogonidae are the smallest of the blood sucking flies, usually 1.0—2.5 mm in body length. They are commonly known as "punkies," "no-see-ums" or "biting midges" due to their small size. They often go unnoticed despite their great ferocity, which is due to the females piercing and sucking mouthparts being formed into a short proboscis. Although Diptera insects first appeared 245 million years ago during the Permian Period (Gullen & Cranston 2005), they are one of the most ancient groups of blood-feeding insects. Ceratopogonidae appeared 65 million years ago in the Cretaceous Period (Balashov 1882 in Valkiūnas 2005), by which time insect fauna had taken on a modern appearance and survived the great extinctions of the Cretaceous-Tertiary Period (Gullen & Cranston 2005). Biting midges have a worldwide distribution of 4 732 species comprising 89 genera (de Meillon & Wirth 1991); 952 species in 26 sub-genera consisting of 50 genera have been recorded from the Afrotropical Region (Segerman 1996); 15 genera of the tribe Ceratopogonini and one subgenus of *Forcipomyia* are endemic to South Africa. Four subfamilies of the biting midges are represented in southern Africa: Leptoconopinae, whose larvae are found in saline to alkaline wet sand or soil; Forcipomyiinae whose larvae are aquatic or semi-aquatic; Dasyheleinae with aquatic larvae in small temporary water bodies; and Ceratopogoninae with swimming larvae (de Meillon & Wirth 2003). Comprehensive work on worldwide distribution of biting midges is lacking, the exception being the single species *Culicoides furens* occurring in the tropical regions of the world (Roberts & Janovy 2000). This lack also extends to the family Ceratopogonidae as a whole, of which only the notorious pests have been well studied. Only four genera attack humans and animals, of which *Culicoides* is the most important genus, with over 1 000 described species (Mullen 2002). This genus requires a comprehensive taxonomic work for the Afrotropical Region (Segerman 1996).

Biting midges are characterized by their small size; stout short legs with unequal claws; narrow spotted wings, lacking veins, and which fold back over the abdomen when at rest. Mouthparts are adapted for piercing tissues, being especially well developed in the female blood-feeding species, having mandibles bearing a row of teeth along the inner edge near the tip, for lacerating the skin before feeding.

Male mouthparts are much reduced for feeding only on nectar. In conjunction with the mouthparts are a pair of five segmented maxillary palps, with sensory organs located in the third segment-the number is correlated to the host feeding species. Species feeding primarily on birds have more sensory organs than mammal feeding species (Mullen 2002). All biting midges feed during daytime on hot still days, being unable to cope with wind of any magnitude. Activity periods vary according to species, time of year, seasonal changes in temperature and light intensity. Males of all species remain near their breeding sites, while females have a tendency to disperse. Johnson (1969) states that *C. variipennis* annoyed cattle 3km from the breeding site, while *C. mississippiensis* dispersed 3 km and *Leptoconops kertezi* flew 15 km in 24 hours (Mullen 2002). Females of most species take blood-meals, although all feed on nectar flowering plants for flight activity-although it is the only food source for males. Members of the genus *Culicoides, Leptoconops* and certain *Forcipomyia* feed only on vertebrate blood (de Meillon & Wirth 1991). Of interest is that engorged mosquitoes are attacked by the Malayan species *Culicoides anophelis* for the purpose of obtaining vertebrate blood from their crops, while those of no medical importance feed mainly on other insects, especially the Chironomidae (Freeman 1973).

Developmental stages

Fertilized adult females require a blood-meal in order to develop eggs. However there are autogenous individual females which retain enough resources from the larval stage to develop eggs, during the first gonotrophic cycle, thereafter a blood-meal must be obtained for futher egg development. Eggs are laid in rows or groups of 30—450, depending upon species and blood-meal, in muddy or sandy margins of streams, pools or dams. Eggs usually incubate within 7—10 days but may be as short as 2—3 days, depending upon favourable environmental conditions. Reptile-like larvae emerge moving about the muddy substrate feeding on various organisms, including nematodes. Three larval instars occur in most species-retaining this stage of development for two weeks in the tropics and up to a year in temperate regions while over-wintering. Larval development of some arctic species may take as long as two years (Mullen 2002). On completion of the fourth moult, pupation takes place, with pupae remaining in the substrate, breathing through a syphon extending to the surface. Adult biting midges emerge from the pupa to locate a suitable host for a necessary blood-meal; thereafter the females and males copulate, resulting in female egg-laying, under favourable environmental conditions. If the species is univoltine, egg-laying occurs only once. When multivoltine females are successful at obtaining subsequent blood-meals, then a second and possibly a third batch of eggs are produced. Four generation cycles may be completed in a year. Under laboratory conditions *C. variipennis* completes seven gonotrophic cycles (Mullen 2002). Usually, each species exhibits characteristic seasonal peaks of abundance, and may be present in lower numbers outside the peak periods. Longevity of adults under natural conditions may last only a few weeks, although captive adults have survived for seven weeks under laboratory conditions.

Habitats

Breeding of Ceratopogonids occurs in a wide range of habitats, primarily in aquatic and semi-aquatic, covering all regions of the world including the Arctic tundra. The larvae of most genera are semi-aquatic, occurring in mud or sand on the margins of streams, dams and ponds. Larvae of *Leptoconops*, have been discovered along the sea shore, in desert oases and buried in soil 900 mm deep (Roberts & Janovy 2000). They may also be found in tree holes, water-retaining plants, decaying vegetation

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and cattle dung (certain species of *Culicoides*). Larvae and pupae of aquatic species occur in slow-flowing water with vegetation, in rock pools, dams and in rocks covered with moss in seepage areas. Certain species of biting midges are also known to breed in salt marshes, brackish water and mangrove swamps.

Pathogenicity

Ceratopogonids serve as vectors of a number of viruses, protozoans and nematodes. The genera *Leptoconops*, *Austroconops* and *Lasiohelea* are vicious man-biting pests, especially along beaches and mountain resorts. Certain species of *Leptoconops* from Australia cause intense swelling around human eyes, lasting three days (Freeman 1973). *Culicoides*, the largest genus in the family, has been identified as vectors for numerous diseases in man and other vertebrates. They transmit parasites of African Horse Sickness (du Toit 1944), filarial of the genus *Onchocerca* to cattle, "blue tongue" to sheep in Kenya (Walker & Boreham 1976) and Nigeria (Herniman *et al.* 1983), also causing Oropouche fever and mansonellosis in humans.

Culicoides is the only genus from which species are vectors for transmission of the single cell avian blood protozoan, *Haemoproteus*, to birds. *Leucocytozoon caulleryi* is the only *Leucocytozoon* species to be transmitted by biting midges (Mullen 2002, Valkiūnas 2005), causing serious disease in poultry. They have a pathogenic impact which is complicated and diverse, requiring much research. Garnham's (1966) studies showed that invasion by the parasite into the blood of birds, resulted in anaemia, enlargement and whitening of the liver and spleen, with pigment accumulation in the organs. *Haemoproteus* causes anaemia in many bird species resulting in weight loss and collapse, but generally has a low pathogenicity except in pigeons while *Leucocytozoon* may be pathogenic to waterfowl, young raptors (Malley & Whitbread 1996) and also poultry (Soulsby 1982).

FAMILY HIPPOBOSCIDAE (louse flies)

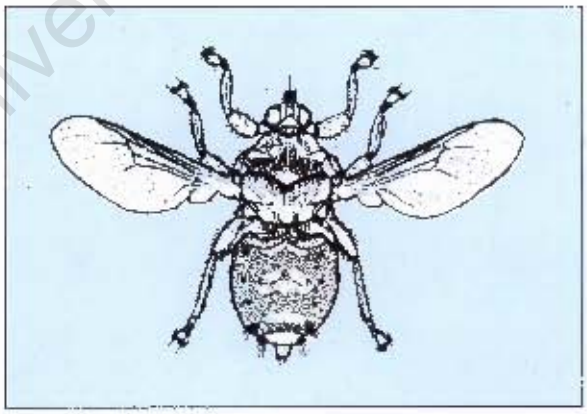

Figure 1.2 Dorsal view of *Hippobosca rufipes* (modified from Scholtz and Holm 1989)

Hippoboscidae, commonly known as "louse flies", are medium-sized, varying in length from 1.5—12.0 mm. They are highly specialized blood-sucking ectoparasites occurring upon a variety of mammals and birds. The body is tough and leathery, being dorsoventrally flattened in form, with the head fairly large, fitting into a depression on the thorax, giving the insect a louse-like appearance. Roberts & Janovy (2000) describe them as neither like lice nor flies, but rather like six-legged ticks. The forward projecting proboscis is well developed with a labium in which is encased a labrum and hypopharynx in a dorsal groove forming a food channel (Lloyd 2002). The tip of the labium has serrated teeth for cutting and piercing. During larval

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development within the female, the abdominal integument is soft and flexible, allowing for stretching and distension; these characteristics remain in adult life for feeding (Lloyd 2002). Legs are robust with strong tarsal claws and teeth. Species that infest mammals have short and strong legs with heavy tarsal claws for grasping skin and course hair. In contrast, bird infesting species have legs adapted for moving swiftly in all directions amidst soft feathers (Bequaert 1953). Wings are long and broad with well developed veins on the fore margins becoming indistinct along the posterior, with halteres representing hind wings—characteristic of dipterans. At rest wings are folded over the abdomen. Species parasitizing birds have retained wings, wereas in mammal parasitizing species they are absent as in *Melophagus ovinus*, the sheep ked. The exception is *Hippobsca rufips,* and *H. equine* on horses, and *H. camelina* on camels (Gogan 1973). In the genera *Lipoptena* and *Neolipoptena* the newly emerged adult has functional wings, but upon reaching the host they break off. After the first blood-meal physiological changes bring about histolysis of flight muscles and leg growth for adaptation of adult ecoparasitic life (Lloyd 2002).

Although Hippoboscidae have worldwide distribution with species occurring in tropical and subtropical regions, the Paleotropics have the greatest number of species (Lloyd 2002). Comprehensive overall distribution studies are lacking with species occurring in other regions due to host migratory patterns, especially birds. Fifty species of louse flies occurr in Africa, of which the majority are bird species (Skaife 1992). Thirty-seven species in 12 genera from three subfamilies have been recorded in southern Africa; Ornithomyinae, Hipposcinae and Lipoteninae, with *Struthipbosca* the only endemic genus, occurring primarly on ostriches (Barraclough & Londt 1989).

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Developmental stages

Unlike most insects, Hippoboscidae are adenotrophic viviparous, having abolished the free larval stage (Freeman 1973). A single egg is passed into the uterus where it embryonates, being nourished on yolk until hatching. Three larval instars follow, feeding orally from secretion glands, or milk glands while still in the uterus of the females reproductive system (Lloyd 2002, Gullen & Cranston 2005). When the full-grown larva emerges, its integument hardens and pupariates immediately. Puparia are normally deposited in the hosts roost, nest, bedding, or in close proximity of the host. The paparium of the sheep ked is stuck amid the sheep's wool with a sticky fluid by the female. The adult fly emerges in about three weeks to several months, depending upon species and temperature.

Habitats

All species of Hippoboscidea are permanent ectoparasites, which have morphologically evolved through selection for a permanent existence among the hairs or feathers of their hosts. The pigeon fly *Pseudolynchia canariensis* spends its entire life on pigeons and other avian species (Klei & De Giusti 1975), so too does *Melophagus ovinus* on sheep, and when removed dies within four days (Roberts & Janovy 2000).

Pathogenicity

Both sexes of the adult louse flies are blood-feeding ectoparasites of most vertebrates, including being vectors for mammalian and avian trypanosomes, filarial worms, lice, mites and haemosporidian blood parasites (Lloyd 2002). Louse flies cause irritation and painful bites to humans, while heavy infestations to animals

cause emaciation and increase susceptibility to secondary infections. Immature birds and mammals have larger infestations than older animals of the same species, and cause significant blood loss in young birds (Malley & Whitbread 1996). Several louse flies have been identified as vectors of *Haemoproteus* species of which the most notable genera are *Pseudolynchia, Stilbometopa, Icosta* and *Ornithomyia*. Of these the most common species is *P. canariensis* which is common on all Columbidae throughout most temperate regions and is the vector for *Haemoproteus columbae*. Transmission of infection to young birds results in anaemia and enlargement of the spleen (Soulsby 1982).

FAMILY SIMULIIDAE (blackflies)

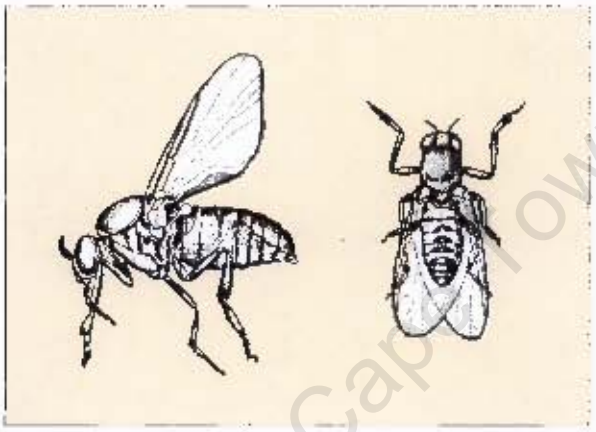

Figure 1.3 Lateral and dorsal view of *Simulium ramnosum* (modified from Scholtz and Holm 1989)

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Simuliids known as blackflies are small two winged, robust, grey flies, 1—5 mm long, commonly called "buffalo gnats", "reed smuts" or "river midges Blackflies have a worldwide distribution, being abundant at locations having suitable rivers and streams for developmental stages, especially in northern temperate and subarctic zones (Roberts & Janovy 2000). A few African species are capable of development in completely stagnant water (Crosskey 1973), the most notable being species in the *Simulium damnosum* complex (Quélennec *et al.* 1968). Blackflies, easily dispersed by the wind, have also colonized the remote oceanic islands of Crozet and St. Helena (Crosskey 1973), but are excluded from many other islands and polar regions. The species *Simulium damnosum*, which is the vector for the parasitic worm causing river blindness in the West African Region, reinvades all insecticide treated sites each year with the onset of monsoon south-westerly winds (Pedgley 1982).

Over 1 720 species have been described worldwide, comprising 25 genera, 53 sub-genera and with many new species awaiting discovery, especially in the tropics (Crosskey 1973, 1990) Southern Africa has a recorded species list of 39, of which eight are absent and eight are endemic to the southern and Western Cape (Palmer & de Moor 1998). Thirteen are distributed throughout the region, whereas 18 have restricted distributions, eight of them being rare (Palmer & de Moor 1998).

Due to morphological uniformity in blackfly species, identification creates great difficulty, requiring a holistic approach to identification: ranging from eggs, larvae, pupae, males, females, polytene chromosomes through to distributional locations. Pest and vector management requires accurate identification, which has resulted in black flies being taxonomically one of the most well researched group of arthropods, with the exception of mosquitoes, worldwide.

The notable humpbacked appearance (humped thorax) of the blackfly, results from the prescutum of their mesonotum being reduced in size. The wings are broad, clear or iridescent having strongly developed anterior veins. Antennae are thin. Female eyes are set apart, while male eyes are adjacent to each other located above the antennae. Although mouthparts are delicate, they have serrated teeth for cutting, located on the outer edge of the mandible (Roberts & Janovy 2000). Mouthparts are anchored to warm-blooded hosts during feeding by means of recurved teeth on the maxillary lacinia (Downes 1971).

Because of blackfly feeding habits, they transmit several pathogenic organisms, which lead to grave economic consequences at times thus gaining notoriety in the veterinary and medical fields. In central and southern Europe *Simulium colombaschense* killed 16 000 cattle, horses and mules in 1923 and then again 13 900 in 1934 (Harwood & James 1979). *Simulium ornatum* is the vector of the deleterious disease *Onchocerca volvulus*, prevalent in humans throughout tropical Africa and parts of the tropical Americas (Crosskey 1973, Roberts & Janovy 2000).

The avian malarial-like disease caused by *Leucocytozoon* species is transmitted by various species of Simuliidae, which are definitive hosts and vectors of the genera *Simulium, Prosimulium, Cnephia* and *Austrosimulium*. All avian families have been infected with a species of *Leucocytozoon* (Valkiūnas 2005), including domestic poultry, ducks and turkeys. *Leucocytozoon struthionis* occurring in three week old ostrich chicks caused high mortalities near Bloemfontein (Huchzermeyer 1998) having believed to be harmless (Walker 1912, 1913 quoted in Huchzermeyer 1998, Bennett *et al.* 1992b). Another severe pathogen of ducks and most anatids is *L. simondi* which is transmitted by the species *S. rugglesi* and *S. anatinum*. To date many species of simuliid vectors implicated in *Leucocytzoon* (35 species) transmission remain unidentified.

Developmental stages

Females produce 150—600 eggs, which are either laid on the surface of water or deposited below the surface, depending upon the species. Eggs of *Austrosimulium pestilens* can survive in moist soil of dry streams for two years during semi-desert droughts (Colbo & Moorhouse 1974), hatching when streams are inundated (Hunter 1978). After storage at 0.5—1.5ºC for 140 weeks, eggs of *S. venustum* hatched (Fredeen 1959), while diapause eggs of *S. rostratum* under laboratory conditions held just above freezing for two years remained viable (Adler & McCreadie 2002).

Larval development can only occur in well oxygenated running water. Black flies are thus numerous near rivers and streams. Upon hatching, the larva spins a silken mat attached to submerged objects anchoring its rear end to it, with the head pointing downstream. It filters protozoa, algae, organic detritus and small organisms through fanlike projections around the mouth, from passing water. When temperature conditions and food availability are ideal, the six to seven larval instars require 7—12 days for development before encasing itself in a spun cocoon. Upon pupation moulting takes place where after the pupa remains immobile from a few days to four weeks, before cutting itself out of the cocoon to surface. Mating occurs in flight when females fly into a massive cloud of hovering males. According to Oberholzer & Ryke (1993) it appeared that sexual organs of simuliids are not well developed due to the male tendency of mating with males or males from another species. Crosskey (1990) states that the family Simuliidae contains species which reproduce in very peculiar ways, having unisexual and bisexual species, including species showing composite body halves of the opposite sex, in the same individual blackfly. Such sexually mosaic individuals are classified as either gynandromorphs or intersexes. Gynandromorphs have varying combinations of genetically male and female cells,

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with the bilateral form of male and female being the most common (Brust 1966). Intersexes are genetically of the same sex with varying sexually mosaic body parts of the opposite sex, usually bilateral symmetry a front-to-rear transformation to the opposite sex (Crosskey 1990). Intersex specimens are commoner than gynandromorphs, although sexually less well expressed and may be more easily overlooked as by Oberholzer and Ryke (1993), and a type specimen in the Zoological Museum of the Humboldt University in Berlin (Crosskey 1990). These sexual mosaics are rare, forming a small proportion of natural wild population (Wolfe & Peterson 1959, Fredeen 1970, Dang & Peterson 1979). The most notable of the unisexual species is the boreal *Prosimulium ursinum* which is restricted to northern latitudes, particularly the Arctic. It develops unfertilized eggs without a blood-meal, producing only females, having abolished males, sexual behaviour and egg fertilization (Downes 1962, 1965).

Seasonal occurrence is dependant upon blackfly species. The tribe Simuliini contain both univoltine and multivoltine species, whereas the tribe Prosimulium are univoltine, completing a single generation annually. In mild tropical conditions *S. damnosum* can produce more than 20 generations annually (Crosskey 1990).

Habitats

Blackflies are found worldwide, with the greatest population concentrations in northern temperate and subarctic zones. Blackflies normally stay close to their emergence sites which are in rivers and streams. Daily flight activity of most species peaks in early morning and again in late afternoon and early evening, with flight activity directed towards responding to optical stimuli for finding mates, host animals and egg-laying sites (Crosskey 1990). Females of those species which undertake long migrations are wind assisted, occurring in semi-open environment and feeding on mammals. Male dispersal is restricted to within 2 km from emergence sites in rivers and streams. Both sexes of the ornithophilic species are restricted to short dispersal ranges (Crosskey 1990). Oberholzer and Ryke (1993) stated that blackflies have been found 150 km away from breeding sites in southern Africa. *Simulium damnosum* and *S. sirbanum* however disperse distances exceeding 500 km in West Africa, which is linked with southwesterly monsoon winds associated with the Intertropical Convergence Zone (Garms & Walsh 1987).

Pathogenicity

Blackflies are vectors for various diseases which kill cattle, horses, mules to human onchocerciasis. Various species of *Simulium* transmit the *Leucocytozoon* species to birds (Malley & Whitebread 1996). *Leucocytozoon simondi* which is a severe pathogen of ducks and other anatides, is transmitted by *S. rugglesi* and *S. anatinum.*

FAMILY CULICIDAE (mosquitoes)

The mosquito was the first arthropod implicated as a vector of disease, by Patrick Manson in 1877, although mosquito bites and disease were associated with humans for centuries (Harrison 1978, Garnham 1966). Studies since have established that mosquitoes are the most important insect vector of human disease and the most common blood-feeding arthropod (Roberts & Janovy 2000), thus making them the best studied families within the Diptera. Mosquito blood-feeding ranges from humans (Figures 1.5 & 1.6) to birds, mammals, reptiles and amphibians, with certain species exhibiting host specificity. "Mosquito" is a Spanish term meaning "little fly" (Skaife 1992).

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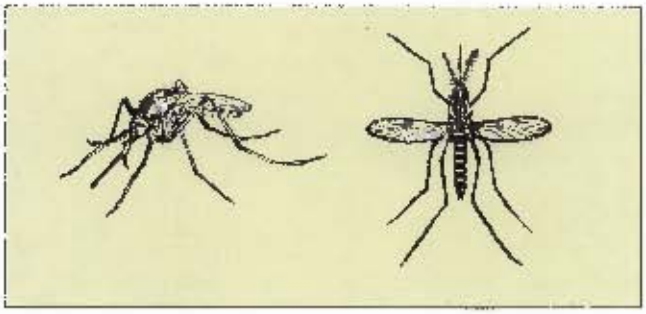

Figure 1. 4 Three dimensional and dorsal view of female *Aedes aegypti* (modified from Scholtz and Holm 1989)

As a vector for human diseases, it has impacted upon the course of human affairs with great consequences, causing misery, poverty, mortality, pathogenicity and discomfort from bites (Harrison 1978). Even the industrialized temperate countries have ongoing discomfort from mosquito bites and mosquito-borne diseases. The United States alone spends hundreds of millions of dollars annually on repellants, lotions, creams and control (Foster & Walker 2002), despite the vast amount of knowledge and improvement in insecticides. Mosquito-borne misery also extends to agriculture and wildlife, causing extensive production loss and mortality (Warner 1968, Huchzermeyer 1975, Steelman 1976, van Riper *et al.* 1986, Foster & Walker 2002, Grim *et al.* 2003). Mosquito distribution covers nearly every region and continent of the world, with the exclusion of Antarctica. They also occur in a diversity of habitats: tropical forests, salt marshes, ocean tidal zones, deserts, high mountains and savannah plains (Service 1995, Foster & Walker 2002), with the greatest species diversity occurring in tropical forests, and the highest densities in the Arctic tundra (Foster & Walker 2002).

Figure 1.5 *Aedes aegypti* female feeding on human blood (Photo by R. G. Hancock: taken from Foster & Walker 2002)

Figure 1.6 *Aedes albopictus* feeding on human blood (Photo by W. A. Foster: taken from Foster & Walker 2002)

Current culicid taxonomy shows mosquitoes belonging to the suborder Nematocrea, family Culicidae, with subdivision of three subfamilies: Anophelinae, Culicinae and Toxorhynchitinae (Knight & Stone 1977, Knight 1978, Ward 1984, 1992, Gaffigan & Ward 1985). Culicinae comprises two-thirds of the total number of species, the remaining third belonging to Anophelinae and Toxorhynchitinae (Gillett 1972). Conflicting views exist as to the taxonomic status of Anophelinae and Toxorhynchitinae being primitively grouped or being specialized derivatives from a Culicinae-type ancestor (White 1980, Besansky *et al.* 1992). Nucleotide-sequence studies by Harbach and Kitching (1998), indicate that Anophelinae are only distantly related to Culicinae and Toxorhynchitinae, and that Toxorhynchitinae does not reflect a natural relationship and shared evolutionary origin of the two subfamilies, indicating

exclusion from subfamily status, thus meriting full family status. Each family has intrinsic morphological characteristics: Anophelinae eggs have air sacs for floatation, its larvae lacking breathing siphons, and the adults having maxillary palps extending the length of the proboscis, Culicinae and Toxorhynchitinae larvae have breathing siphons, with adult females having short maxillary palps. Toxorhynchitinae larvae are predatory upon other larvae, with adults having a 90 degree curved proboscis for nectar feeding (Figure 1.8). Well studied mosquito species reveal complexes of closely related species, which are reproductively isolated in habitat niches undergoing possible speciation. Mosquito fauna is rich and diverse, with approximately 3 500 described species, having a worldwide distribution (Roberts & Janovy 2000). In southern Africa there are 13 genera to which 220 described species belong (Gillies & Coetzee 1987, Jupp 1996). There are 30 species endemic to the Afrotropical region, but none to southern Africa (Coetzee 2002).

Mosquito eggs are laid in batches of 30—350. The shape and pattern, including the manner of oviposition are species particular. The eggs of *Culex*, *Culiseta*, *Coquillettidia* and *Mansonia* are laid in egg rafts (Figure 1.7) and submerged clusters, whereas *Anopheles*, *Toxorhynchites*, *Wyeomyia*, *Aedes*, *Ochlerotatus*, *Psorophora* and *Haemagogus* lay eggs individually. Larvae often hold the key to species identification, having three distinct body parts: head, thorax and segmented abdomen (Figure 1.8).

Figure 1.7 *Culex quinquefasciatus* females laying eggs in form of floating rafts (Photo by W. A. Foster; taken from Foster & Walker 2002).

Newly hatched first-stage larvae escape from the egg thereafter undergo four larval stages, the final stage producing an active pupa, which does not feed. Larvae are suspended from the water surface by a breathing siphon, which is absent in anophelines. Exceptions are the larvae of *Coquillettidia richiardii* which remain submerged, obtaining air from plant roots and stems, and *Aedes aegypti* which inserts the breathing siphon into submerged air bubbles (Snow 1995).

Adult mosquitoes can be distinguished from the superficially similar appearing Dixidae, Chaoboridae and Chironomidae, by the forward-projecting proboscis which is two-thirds the length of the abdomen. Other features include the raised hind legs and anopheline 45° angle resting position, in conjunction with long legs and narrow wings with scales along the veins and margins (Service 1980). Each mosquito species is characterized by the markings and colours covering the body surface. These patterns consist of setae, spines and three patterns of body scales: broad and flat, narrow and curved, and erect with forking (Jupp 1996). Adults of both sexes of

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most species regularly feed on plant juices and nectar, throughout life, but only females feed on vertebrate blood between and during gonotrophic cycles, in order to obtain the nutrients necessary for development and maturation of eggs. Exceptions are *Culiseta subochrea* and *molestus* of the *Culex pipiens* complex, which develop the first egg batch, and occasionally further batches, without a blood meal (Snow 1990).

During the terrestrial life phase adults remain within a few hundred metres of breeding sites (Picker *et al.* 2002), with all flights being goal-orientated towards feeding, mating or oviposition sites, partaking only in dispersal and not migration by wind (Service 1995). *Culex tarsalis* has been recaptured 42 km from the release site (Bailey *et al.* 1965), with the salt marsh breeding *Aedes vigilax* discovered 60 km inland (MacCreary & Sterns 1937) while *Aedes taeniorhynchus* was caught 169 km on an unmanned oil rig off the Louisiana coast (Sparks *et al.* 1986). In 1930 the transportation by ship of *Anopheles gambiae* from West Africa to Brazil became a notable recording (Service 1995), so too the survival of 84% of *Culex quinquefasciatus* placed in the wheel bays of a Boeing 747B aircraft travelling from Australia to Singapore and then onto Thailand (Russell 1987).

Human malaria vectors belong exclusively to the subfamily Anophelinae, which transmit three groups of human pathogens: four species of *Plasmodium* (*P. vivax, P. falciparum, P. malariae* and *P. ovale*), one simian (*P. knowlesi*), two species filariasis and 40 arbovirsuses (Mattingly 1973). Malaria kills more than a million people every year worldwide (Roberts & Janvoy 2000), with 50 000 infected in the northern subtropical region of southern Africa during 1999 (Picker *et al.* 2002). In recent years malaria prevalence has increased as a result of resistance to antimalarial drugs and of mosquitoes to insecticides (Marshall 1991), also resulting in the reinvasion of northern KwaZulu-Natal by *Anopheles funestus.*

All avian malaria species of *Plasmodium* are transmitted by the subfamily Culicinae, tribe Culicini to which the genera *Culex, Aedes, Mansonia* and *Culiseta* belong (Bennett *et al.* 1992a, 1992b, Foster & Walker 2002). Many species of mosquitoes implicated as vectors for transmission of avian *Plasmodium* (38 species) remain unidentified, although *P. relictum* is the best studied. This pathogen completes its development in 26 species of Culicinae (Valkiūnas 2005).

Developmental stages

No exceptions are known to the standard culicid life cycle, which consists of an initial egg laying stage, taking place singly, in clusters or in the form of egg rafts (Figure 1.8), on water surfaces or soil substrates. Hatching normally occurs quickly. If eggs are laid in the soil during a drought period, they may delay hatching for a period of up to four years until the next flooding (Woodard & Chapman 1970). Larvae and pupae are adapted to an aquatic existence, where they are suspended from water surfaces by a breathing siphon. Four larval instars precede the pupation period, lasting two to three days. When fully developed, the skin splits at the thorax from where the sexually dimorphic adults emerge, and fly off. Adult females live four to five months if undergoing a period of hibernation. During hot summer months, female activity results in a shortened life span of two weeks. Male life span lasts a week to two on average, but does extend to a month when food and humidity conditions are ideal. Being solely plant juice feeders to obtain sugars for daily energy needs, males lack mandibular stylets, a basic requirement for females to obtain vertebrate blood nutrients necessary for egg development.

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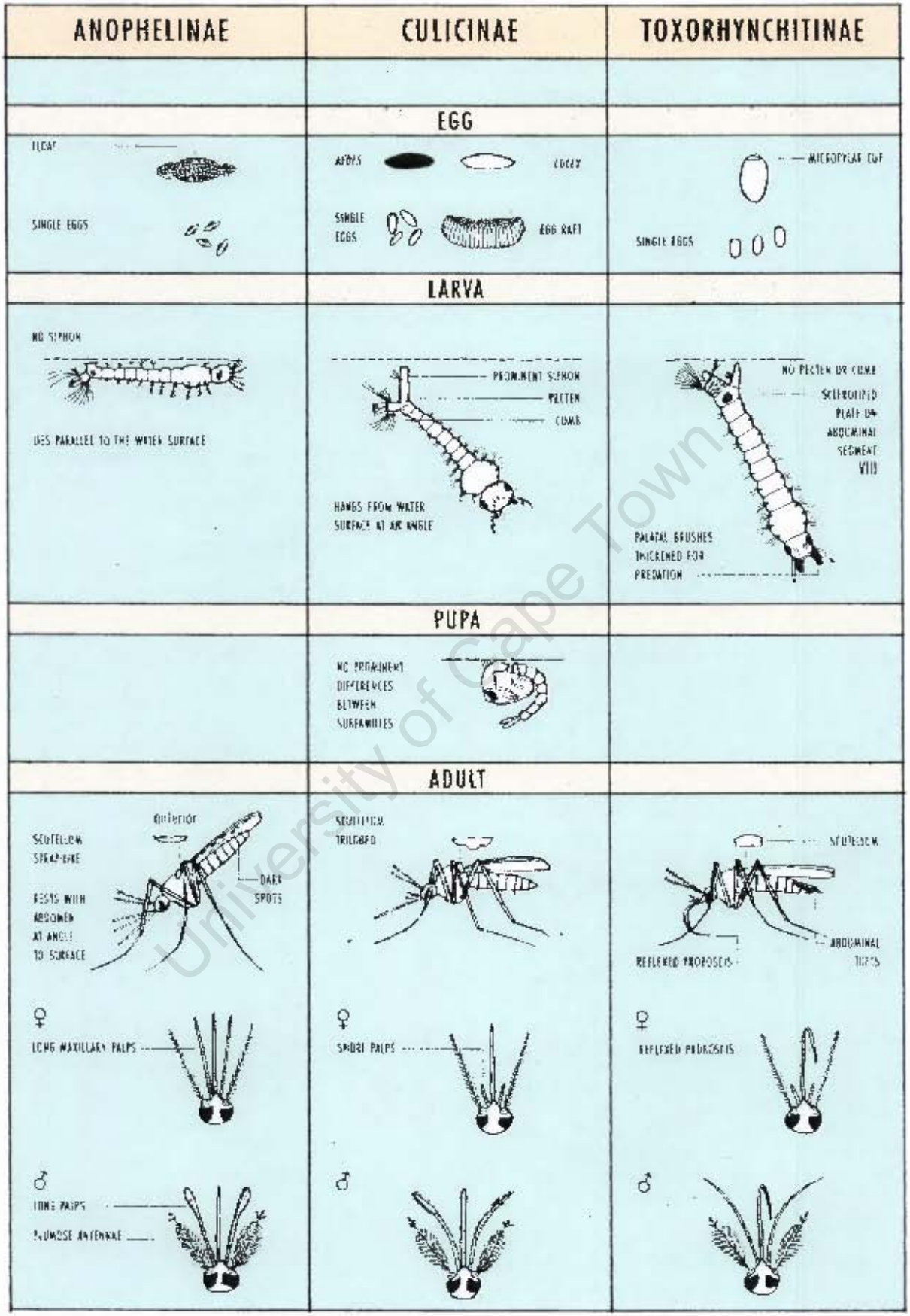

Figure 1.8 Comparison of typical life cycles of subfamilies: Anophelinae, Culicinae and Toxorhynchitinae (taken from Jupp 1996).

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Habitats

Oviposition sites have two habitat classifications; one is running-water, the other still-water, which is further subdivided into groundwater and container oviposition sites. Running water is used mainly by members of the subfamily Anophelinae which will also use permanent groundwater habitats when available with Culicines, excluding species from the genus *Aedes,* which utilizes temporary groundwater. When temporary groundwater is available in specialized situations, such as crab holes, rock holes, and water wells, then various Culicines and a few anophelines will make use of it. Container habitats are numerous and varied and include holes in trees, split bamboo, plants, made-made containers. All of these will be utilized by many groups of Culicides including species from the genera *Anopheles* and *Aedes*.

Pathogenicity

Mosquitoes are vectors for well-known human diseases such as malaria, filariasis, encephalitis, yellow fever, dengue fever and filarial worms (Gillett 1972, Mattingly 1973, Oberholzer & Ryke 1993). They are also vectors of disease agents of wildlife and domesticated animals, including Equine Encephalitides, Rift Valley Fever, Wesselbron Virus and Myxomatosis virus causing irritation, blood loss and allergic reactions (Foster & Walker 2002).

All species of birds are asymptomatic carriers of avian malaria (Garnham 1966, Valkiūnas 2005), which is widespread geographically (van Riper *et al.* 1994). Ronald Ross received the Nobel Prize (1902) for studies implicating the mosquito as vectors of avian malaria. Currently only 38 valid species of avian *Plasmodium* are recognized by taxonomists (Valkiūnas 2005). In laboratory studies, *Anopheles* mosquitoes are vectors for many avian malaria species, although the genera *Culex, Culiseta* and *Ochlerotatus* are the only natural vectors in the wild (Foster & Walker 2002). In the Afrotropical Region *Aedes aegypti* is the vector of *Plasmodium gallinaceum* to chickens (Soulsby 1982), while *Culex* mosquitoes introduced into Hawaii have caused the reduction and extinction of endemic birds (Warner 1968).

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CHAPTER TWO

General discussion on avian haemosporidia

We have reason to believe that species in a state of nature
are limited in their ranges by the competition of other organic beings
quite as much as, or more than, by adaptation to particular climates
Charles Darwin (1809—1882)
On the Origin of Species

INTRODUCTION

Not only does the human through his art express his fascination with birds, but so too do parasites. This is evident from the enormous amount of parasitological research literature published. Infectious disease has been a dynamic factor in human population selection for the past 10 000 years according to Haldane (1949) as cited by May (1988). Support for this hypothesis is found in McNeill (1976) and Lederberg (1988, 1993, 1997). But a parallel influence on avian population dynamics by protozoan parasites has been ignored (Price 1991, Holmes 1993), and suffered decades of neglect until interest was sparked by the publication of *The Evolutionary Biology of Parasites* (Price 1980), which got ecologists and parasitologists seeking ways of understanding the effects which parasites have on their hosts' life and behaviour. This was followed by Anderson & May (1982) on mathematical analyses of infectious diseases which contributed further to parasitological research. This spark of interest resulted in the Hamilton-Zuk (1982) hypothesis coming to the fore giving further impetus to avian parasitological research. This resulted in many research papers incorporating the biology, ecology and evolution of parasites and hosts alike, including a paper soley on statistical methods regarding the hypothesis (Underhill & Kalejta-Summers 1993). The Hamilton-Zuk theory stated that individuals which mated successfully and possessed genes which had bestowed them with resistance to withstand parasitic infections, produced fitter offspring. Evolutionary traits of this nature, which cannot be laboratory tested, remain controversial as shown by published papers criticizing the theory (Cox 1989, 1994, McLennan & Brooks 1991, Poulin & Vickery 1993, Yezerinac & Weatherhead 1995) as opposed to those defending it (Read 1987, Read & Harvey 1989, John 1997), including a middle of the road approach (Endler & Lyles 1989).

Most species of haemosporidia are parasites of wild animals, which appear to cause little harm, the exception being *Plasmodium,* a scourge to humanity and animal alike (Mullen & Durden 2002). Parasites may also negate conservationists' efforts to save endangered species, as manifested with avian species encountered on the archipelago of Hawaii, due to the introduction of *Culex quinquifasciatus*, the vector for *Plasmodium relictum* and avian pox. The impact of these introductions has been disastrous on the islands endemic forest birds (Warner 1968, van Riper *et al.* 1986, Atkinson *et al.* 1995), with the Hawaiian honeycreepers *Hemignathus munroi* having gone extinct, and others on the endangered list (van Riper *et al.* 1986, Pyle 1990).

Every species of bird that has been studied has at least one species of parasite, if not several (Loye & Zuk 1991, Clayton & Moore 1997). Thus when working with birds, it cannot be taken for granted that a particular individual species is free from haemosporidian infections. Although ectotherms are in the majority and do have

parasites, it is the endotherms, and particularly birds where the richest diversity of parasites and species are found. Protozoan intracellular blood parasites from the order Haemospororida are the parasites most frequently encountered in avian hosts. Belonging to this order are the families Haemoproteidae, Plasmodiidae, Garniidae and Leucocytozoidae, which in turn consist of the genera *Haemoproteus, Plasmodium, Fallisia* and *Leucocytozoon* (Figure 2.1). Speculation exists as to the possibility that avian haemosporidian parasites evolved from coccidian of the vertebrates rather than of invertebrates, with mites or other bloodsuckers initiating the cycle in arthropods (Manwell 1955, Garnham 1966, Roberts & Janovy 2005). But there are many controversial views as to the evolutionary history and in which group of vertebrates or invertebrates these parasites originated prevail (Chatton 1937, Ball 1943, Huff 1945, Grassé 1953, Garnham 1996, Valkiūnas 2005). This controversy arises from a lack of paleontological evidence, and a lack of molecular studies.

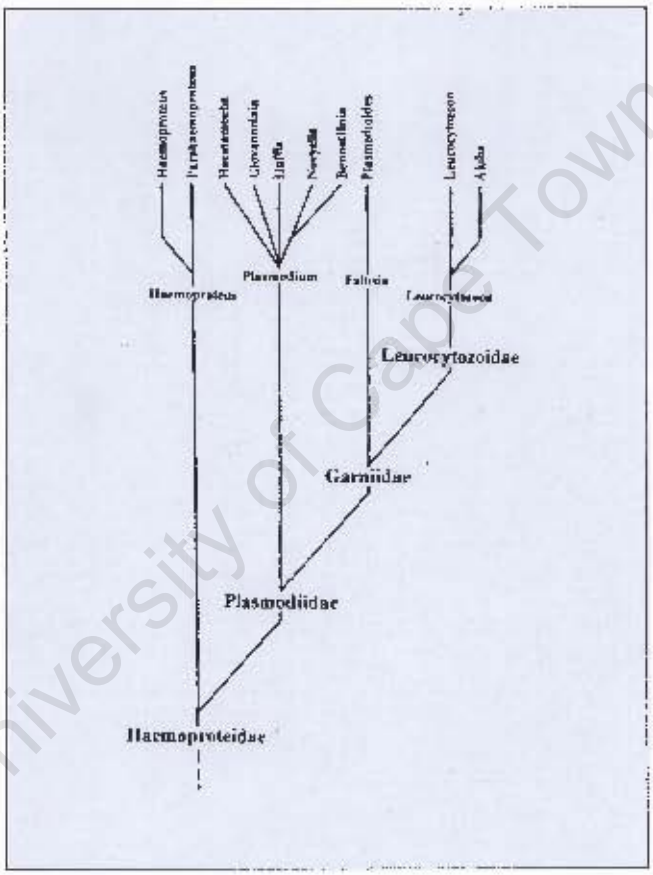

Figure 2.1 Diagrammatic representation of the possible phylogenetic relationships of haemosporidian parasites (modified from Valkiūnas 2005).

One evolutionary theory of avian haemosporidia assumes an invertebrate origin, beginning in Diptera (Huff 1945, Landau 1974). Garnham (1966) summarizes the improbability of an anisogamous union initiated in the Diptera, and ending with sporozoite production without vertebrate involvement assuming gametogony in mid-gut of the insect after ingestion of sporozoites. Garnham's opinion is that had such development formed part of evolution, some coccidian parasites of dipterous insects should have survived to the present day and not be poorly represented within other orders of insects. He sees clarity of descent of malaria parasites in numerous genera of coccidians. Occurring first in myriapods and worms, and later in other vertebrates with the logical sequence of gametocyte adaptation of a coccidian parasite to the blood stream of a vertebrate host, then taken up by a biting insect, of which the

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ectoparasitic hippoboscid fly probably was the first insect to be a vector (Manwell 1955). Most studies support the concept of avian haemosporidians being later evolutionary forms of reptilian haemosporidians (Wenyon 1926, Manwell 1955, 1965, Baker 1965, Garnham 1966), since the oldest recorded ancestral bird *Archaeopteryx* from the Upper Jurassic Period was of reptilian descent (Van Tyne & Berger 1976). Waters *et al.* (1991) sees confirmation of reptilian descent in the greater diversity of haemosporidia occurring in reptiles and birds. This is supported by several families of Diptera having only been recorded as vectors of reptilian *Plasmodium* species (Petit *et al.* 1983, Klein *et al.* 1987), which is the result of an ancient evolutionary association with vertebrate hosts. If avian haemosporidia are of reptilian descent, the absence or secondary nature of infection in all primitive species, with low phylogenetic ranking remains. Valkiūnas (2005) suggests that speciation and transmission to birds occurred in tropical rain forests soon after

Table 2.1 Classification of avian orders and haemosporidian parasites within avian orders. Modified from Valkiūnas (2005), with source material from Bennett *et al.* (1992), del Hoyo *et al.* (1992), Sinclair & Ryan (2003), Hockey *et al.* (2005) and Peirce (2005). Afrotrop.=Afrotropical; *Hae.=Haemoproteus; Pla.=Plasmodium; Leu.=Leucocytozoon*. Avian evolutionary relationships are adopted from Hockey *et al.* (2005), broadly based on Sibley and Ahlquist (1990) with modifications introduced from current taxonomic studies of DNA—DNA hybridization data (Hockey *et al.* 2005). Avian haemosporidian parasites have been classified according to Valkiūnas (2005) including Peirce (2005). Due to infection across avian orders by Plasmodiidae, totals are not given—38 valid avian Plasmodiidae species are described.

Order	Aves. World Species	Haemosporidian species				Aves. Afrotrop. Species	Haemosporidian species			
		Hae.	*Pla.*	*Leu.*	Total		*Hae.*	*Pla.*	*Leu.*	Total
Struthioniformes	10	0	0	1	**1**	2	0	0	1	1
Tinamiformes	47	0	3	0	**3**	—	—	—	—	—
Craciformes	69	3	0	0	**3**	—	—	—	—	—
Galliformes	212	7	17	7	**31**	50	3	5	4	**12**
Anseriformes	161	2	1	9	**12**	30	1	0	0	1
Turniciformes	17	0	0	1	**1**	4	0	0	0	**0**
Piciformes	355	8	8	1	**17**	112	4	1	1	**6**
Galbuliformes	51	1	0	0	1	—	—	—	—	—
Bucerotiformes	56	1	3	1	**5**	29	1	0	1	**2**
Upupiformes	11	1	1	1	**3**	11	1	1	1	**3**
Trogoniformes	39	1	0	0	1	3	0	0	0	**0**
Coraciiformes	152	9	3	3	**15**	45	6	1	1	**8**
Coliiformes	6	1	1	0	**2**	6	1	0	1	**2**
Cuculiformes	143	1	1	2	**4**	29	1	1	1	**3**
Psittaciformes	358	2	5	0	**7**	21	1	0	1	**2**
Apodiformes	113	1	0	0	**1**	22	0	0	0	**0**
Trochiliformes	319	3	3	0	**6**	—	—	—	—	—
Musophagiformes	23	1	1	1	**3**	23	1	1	1	**3**
Strigiformes	291	2	5	1	**8**	59	3	3	2	**7**
Columbiformes	310	4	12	1	**17**	32	3	3	1	**7**
Gruiformes	196	5	8	1	**14**	55	3	1	1	**5**
Charadriiformes	366	7	2	2	**11**	134	3	1	1	**5**
Falconiformes	304	6	5	1	**12**	77	4	1	1	6
Ciconiiformes	392	5	5	4	**14**	115	4	1	3	**8**
Passeriformes	5 827	63	16	7	**86**	1 255	29	14	4	**47**
Total	**9 828**	—	—	—	—	**2 114**	—	—	—	—

birds first appeared during the Eocene and Oligocene Periods, being relatively recent in geographical time scale. Valkiūnas (2005) sees confirmation for his hypothesis in the maximum species diversity of all haemosporidians occurring in the tropics and subtropics, where the avian order Passeriformes occurs with the largest number of

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species in conjunction with the largest number of avian haemosporidian species (Table 2.1). This does not explain the absence of primitive birds from the loci of haemosporidian parasites and their vectors. The Neotropical Region also contains the greatest number of primitive birds that acquire secondary infections, for example, Sphenisciformes acquiring *Plasmodium relictum, P. elongatum* and *P. cathemerium* in zoos within the temperate zone of the Northern Hemisphere (Cranfield *et al.* 1990), and *Leucocytozoon tawaki* in Australian and Afrotropical zoogeographical regions (Graczyk *et al.* 1995). Valkiūnas overlooks the natural distribution of haemosporidian vectors occurring abundantly in the tropics and subtropics (Knight & Stone 1977) in which Passeriformes occur, and that Sphenisciformes move into distribution areas of haemosporidian vectors, acquiring pathogens from local avifauna (Grim *et al.* 2003).

The average infection rate of avifauna by haemosporidian parasites is 10% of individuals in a population, while prevalence varies between 0—100% depending on avian species and ecological circumstances, which vary between different haemosporidian taxa in the corresponding host population (Janovy 1997). An example of this was found in a study of Tengmalm's Owl *Aegolius funereus.* Only 10% of the population was infected with *Haemoproteus noctuae, H. symii* or *Plasmodium circumflexum* whereas the prevalence of *Leucocytozoon ziemanni* was greater than 90% (Korpimäki *et al.* 1993). Distribution and prevalence varies with host species and geographic area (Bennett 1960). When studying waterfowl in Canada, Bennett *et al.* (1975) found that each habitat had unique environmental characteristics which affected vector populations and transmission. Atkinson (1988) also found differences, in the time of onset of transmission, duration, and intensity of *H. meleagridis* at sites 1 km apart in Florida.

Avian haemosporidians are intracellular, single cell, protozoan parasites with complicated life cycles in which they change between two hosts, passing through various stages of development. The first stage is the vector as definitive host (blood-feeding arthropod) then the bird as intermediate host. Avian haemosporidian life cycles are obligate heteroxenous, commencing when transmission occurs with sporozoite injection during blood-feeding upon the bird. Haemosporidian parasites undergo generations of asexual multiplication (merogony) within the liver, kidney, spleen and lungs, known as exoerythrocytic meronts, forming uninuclear merozoites. Exoerythrocytic merozoites induce gametocyte development in blood cells and cells of blood forming organs in the bird. When inside the bird's cells, *Plasmodium* and *Haemoproteus* species produce a pigment called haemozoin from the bird's haemoglobin, which distinguishes them from *Leucocytozoon* species. These parasites resemble coccidian, but lack conoids (truncated cone of spiral fibrils located within the polar rings) and syzygy (the stage during sexual reproduction in which two or more sporadins connect end to end). The macrogametocyte (female parasite) and microgametocyte (male parasite) develop independently, with the later producing eight flagellated gametes. The final stage of the sexual cycle with gametogenesis occurs in the vector's gut with fertilization of the gametocytes occurring extracellularly. The motile zygote is transformed by meiosis into an elongated ookinete, which then penetrates the peritrophic membrane into the epithelial layer of the vector's midgut, developing into an oocyst by sporogony between muscle layers. During development the zygote has a diploid set of chromosomes, while all other developmental stages are haploid. Reproduction within the cysts in the gut wall produces many heteroxenous sporozoites. The cysts eventually break, releasing sporozoites that migrate through the haemocoel entering the salivary glands of the vector.

Hippoboscid flies and biting midges remain infected throughout their life transmitting the infection to the squabs (Roberts & Janovy 2005). Infected birds also maintain parasites as a source of infection for vectors throughout their lives

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(Valkiūnas 2005). Studies of *Haemoproteus*, *Leucocytozoon* and *Plasmodium* in temperate northern hemisphere climates have shown that transmission occurs mainly during the breeding season, when immature birds fledge, and adult birds are a source of infection for a resurgent vector population resulting from the onset of warmer weather (Herman 1938, Bennett & Fallis 1960, Janovy 1966, Bennett & Cameron 1974, Baker 1975, Butterworth & Harcourt-Brown 1996). This contrasts with studies by Earlé *et al.* (1991), which showed that rainfall resulted in increased transmission in northern South Africa. At present only 7% of avian haemosporidian life cycles have been researched (Valkiūnas 2005).

GENUS *Haemoproteus*

The *Haemoproteus* parasite is a blood protozoa which is primarily found in birds and reptiles, and is the most common haemosporidian encountered in birds (Atkinson & Van Riper 1991). Exoerythrocytic merogony occurs in endothelial cells, with merozoites penetrating erythrocytes and maturing to pigmented gametocytes in the bird's circulating blood (Figure 2.2). There are currently 132 valid species which have been identified world wide (Valkiūnas 2005), of which 69 are from Afrotropical birds (Bennett *et al.* 1992, Peirce 2005). This might not be a true reflection of numbers, since life cycles of most of the *Haemoproteus* are unknown. Study shows that *Haemoproteus* is benign to all orders of birds except Columbiformes (for which it is highly pathogenic) suggests that *Haemoproteus* species have a long evolutionary history with their avian hosts. Their sexual cycle takes place in biting midges (*Culicoides*) and louse flies (Hippoboscidae), but not in mosquitoes.

Figure 2.2 *Haemoproteus zosteropis* gametocytes taken from blood of *Zosterops palpebrosa* **1—2**, immature gametocytes; **3—6**, macrogametocytes, **7—9**, microgametocytes (modified from Valkiūnas 2005).

Life cycle in the vector

Exflagellation in the stomach of the hippoboscid flies or biting midges produces four to eight microgametes. The ookinete is similar to that of *Plasmodium* except there is a mass of pigment at the posterior end. It penetrates the intestinal epithelium and encycts between the muscle layers, where the oocyst grows to maturity within nine

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days, measuring 40 µm in diameter. Great numbers of sporozoites are released when the oocyst ruptures, taking a day to reach the salivary glands, from where they are injected into the bird when having a blood-meal (Figure 2.3). Development of the parasite within *Culicoides* species usually takes 6—10 days (Mullen 2002).

Life cycle in avian host

Sporozoites injected into the circulating blood of the bird initiate exoerythrocytic merogony in the endothelium of the lung capillaries, and less often in other organs. Generally each meront takes 25 days to produce numerous merozoites (Roberts & Janovy 2005), but is also capable of rupturing to release many multinucleate cytomeres, which penetrate the capillary lumen, releasing thousands of merozoites upon bursting, then penetrate erythrocytes and develop into gametocytes (Figure 2.3). At least two generations of exoerythrocytic development occur before gametocyte penetration. The first generation of development takes five days after sporozoite injection, whereas the second generation matures in 17 days after development in the skeletal and heart muscles (Atkinson *et al.* 1986).

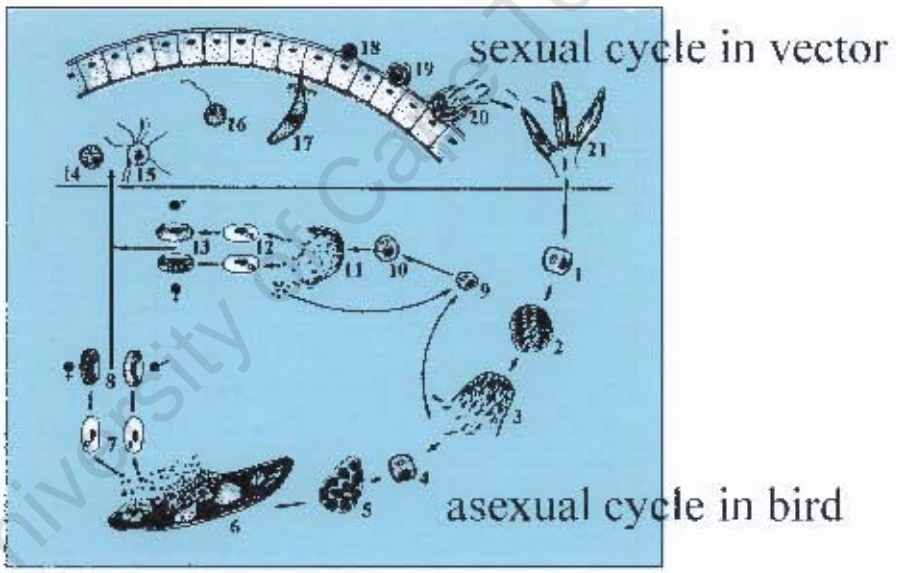

Figure 2.3 Life cycle of *Haemoproteus mansoni* **1** sporozoite in endothelial cell; **2—3** exoerythrocytic meronts of the first with elongated merozoites; **4** merozoite in endothelial cell; **5-6** growing and mature megalomeronts in skeletal muscles; **7** merozoites in erythrocytes; **8** mature gametocytes; **9** merozoites in reticuloendothelial cell in spleen; **10—11** growing and mature meronts in spleen; **12** merozoites in erythrocytes; **13** mature gametocytes; **14** macrogamete; **15** exflagellation of microgametes: **16** fertilization of macrogamete; **17** ookinete penetrating the peritrophic membrane; **18** young oocyst; **19—20** sporogony; **21** sporozoites in the salivary glands of vector. (modified from Valkiūnas 2005).

During the first generation, merozoites induce secondary merogony in the endothelial and reticular cells of the spleen, resulting in the maintenance of chronic parasitemia and relapses (Valkiūnas 2005). The early stages of *Haemoproteus* development in erythrocytes resembles *Plasmodium*, but matures into gametocytes within five to six days, curving around the nucleus (Figure 2.2).

Most macrogametocytes measure 14 µm with a granular cytoplasm and a small nucleus staining a deep blue colour. Microgametocytes are 13 µm having a pale blue cytoplasm and a diffused nucleus. Dark-brown, pigmented granules vary in number from 2—45 within the parasite in the Afrotropical avian species.

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The discoid form has not yet been recorded in Afrotropical birds, occurring only in avian species from the Nearctic and Neotropical Regions.

Pathogenicity

The genus *Haemoproteus* generally has a low pathogenicity, but is pathogenic in Columbiformes (Markus & Oosthuizen 1972). No sign of disease is discernible with slight infections, but changes occur when infection is high. Birds appear restless, lose appetite, and their lungs may become congested. Anaemia can result from erythrocyte malfunction, resulting in spleen and liver enlargement with dark pigmentation (Butterworth & Harcourt-Brown 1996, Roberts & Janovy 2005). Warm weather in the United Kingdom increases the incidence of *Haemoproteus*, particularly in captive bred Snowy Owls *Nyctea scandiaca* and Harris's Hawk *Parabuteo unicinctus* (Butterworth and Harcourt-Brown 1996).

GENUS *Leucocytozoon*

The *Leucocytozoon* species parasitize only birds, being the second most frequently encountered group, occurring in 8.1% of birds examined and in 24.7% of infected birds (Bennett *et al.* 1992). Exoerythrocytic merogony occurs in fixed tissues, with gametogony in leukocytes and immature erythrocytes. Life cycles of 30% of leucocytozoids have been recorded, in which malarial pigment (hemozoin) is absent from all phases. World wide distribution consists of 35 valid species (Valkiūnas 2005), of which 26 occur in the Afrotropical Region. Sporogony occurs in the black flies (Diptera: Simuliidae), which are vectors of these parasites.

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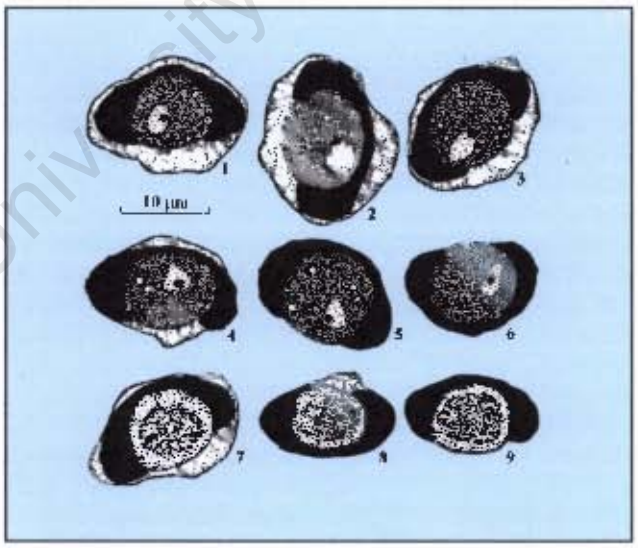

Figure 2.4 *Leucocytozoon dubreuili* gametocytes from the blood of *Turdus philomelos*. 1—6 macrogametocytes; 7—9, microgametocytes (taken from Valkiūnas 2005)

Leucocytozoon fit into eight morphological forms, which can be divided into two groups—the round or ovoid and the fusiform shape which is characteristic only of the leucocytozoids (Figure 2.5). Species with gametocytes which are round or ovoid are restricted to the order Passeriformes, while lower avian orders have both forms. Deformation from host cells to young fusiform gametocytes occur only during the

growth stages through the production of merogony in the kidneys, spleen and reticulo-endothelial system, while round forms are produced by hepatic merogony (Fallis & Desser 1977). The growth of leucocytozoid gametocyte impacts upon the host cell nucleus relocating to the periphery as well as hypertrophy of its nucleus. Common to most species are azurophilic inclusions called valutin which weakly refract light, unlike hemozoin (malarial pigment) which occurs in *Haemoproteus* and *Plasmodium*, but does not occur in *Leucocytozoon* due to the parasite digesting the haemoglobin which is present in the host cell (erythrocyte).

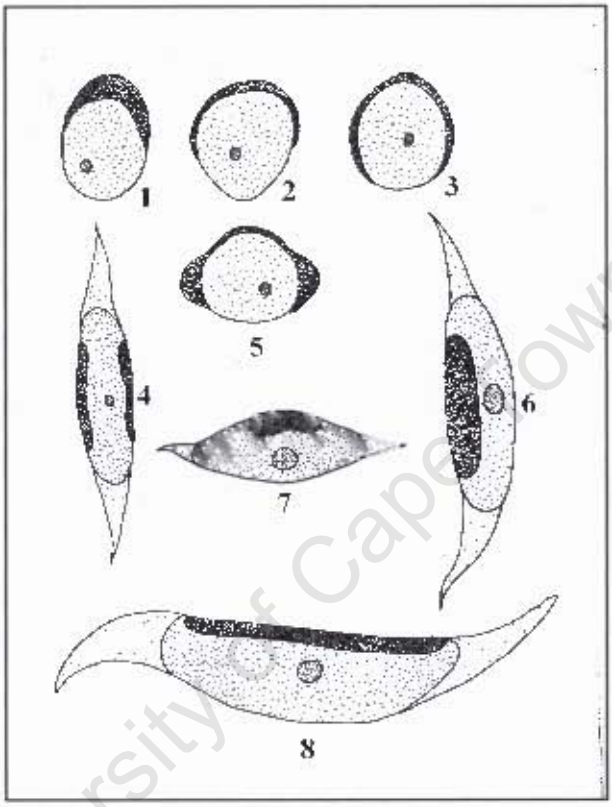

Figure 2.5 Morphological forms of *Leucocytozoon*. **1** Round form with host cell nucleus as a pronounced cap; **2** Round form with host cell nucleus as a thin band covering approximately 50% of the circumference of the parasite; **3** Round form with host cell nucleus as a thin band covering approximately 80% of the circumference of the parasite; **4** Fusiform parasite with host cell nucleus split into two halves; **5** Round form with host cell nucleus forming a pronounced double cap; **6** Fusiform parasite with host cell nucleus covering most of one side of the parasite; **7** Fusiform parasite with host cell nucleus covering only small part of one side of the parasite; **8** Large fusiform parasite with host cell nucleus covering entire side of parasite (modified from Bennett *et al.* 1992).

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Life cycle in the vector

Only after forming gametocytes and circulating in the blood of the avian host can *Leucocytozoon* enter its simuliid host. Once the female blackfly has ingested blood containing gametocytes development in the fly's mid-gut begins by transforming into male and female gametes, which sexually fuse into a motile zygote. The zygotes transform into elongate motile ookinetes which penetrate the mid-gut where fertilization, ookinete development and sporogony take place growing into large spherical oocysts which then project into the haemocoel within three days or less (Crosskey 1990). Only 50 to 60 sporozoites are released from a ruptured oocyst, into the hemocoel to make their way to the salivary glands (Figure 2.6). Unlike biting midges or mosquitoes, sporozoites do not enter the salivary glands of the black fly, but enter the proboscis instead. They are then transmitted to the avian host through contamination or washed into an open wound by saliva.

Development of the parasite within Simuliidae species usually takes 4—7 days (Fallis & Bennett 1961, 1962, Baker 1970, Fallis *et al.* 1973).

Life cycle in avian host

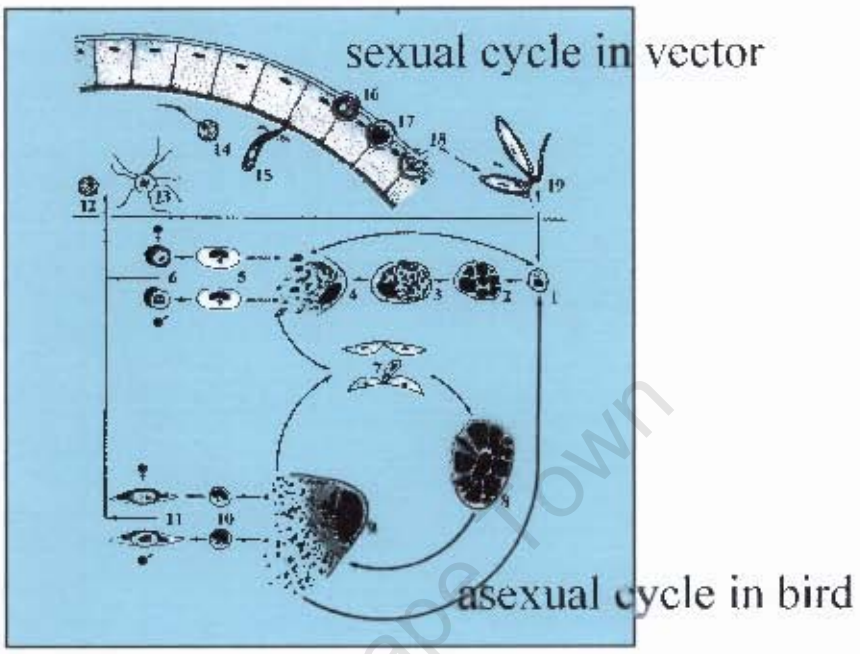

Figure 2.6 Life cycle of *Leucocytozoon simondi* 1 sporozoite in the liver cell; **2—4** hepatic meronts; **5** merozoites in erythrocytes; **6** gametocytes in host cells; **7** merozoite in reticulo-endothelial cell; **8—9** megalomeronts; **10** merozoites in leukocytes; **11** fusiform gametocytes in host cells; **12** macrogamete; 13 exflagellation of microgametes; **14** fertilization of macrogamete; **15** ookinete penetrating intestinal epithelium of vector; **16** mature oocyst; **17—18** sporogony; **19** sporozoites in the salivary glands of vector (modified from Valkiūnas 2005).

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After a black fly transmits sporozoites into its avian host, they enter hepatocytes in the liver, developing into small schizonts of 11 µm to 18 µm and producing merozoites in four to six days (Roberts & Janovy 2005). Merozoites then enter the circulating blood, penetrating immature erythrocytes and becoming round gametocytes in two days, after deforming the host cell and its nuclei (Figure 2.4, 2.5, 2.7). Secondary exoerythrocytic merogony is induced when merozoites are ingested by a macrophage (developing erythrocytes) in the brain, heart, liver, kidneys, lymphoid tissues or other organs. These second generation meronts develop into megalomeronts 50 µm to 400 µm or greater in diameter (Valkiūnas 2005), which are more abundant than the small hepatic merozoites. Megalomeronts divide successively internally three times into primary cytomeres, which become smaller, until the final multiplication by merogony into merozoites. When ruptured, a million merozoites are released from a single megaloschizont, which then penetrate leukocytes or macrophages to become elongated gametocytes 12 µm to 14 µm long in both sexes (Figures 2.6, 2.7).

Leucocytozoids have species with round forms only (e.g. *L. dubreulli, L. fringillinarum*) and others with fusiform gametocytes (e.g. *L. neavei, L. sousadiasi*), and some species in which both forms occur, such as *L. danilewskyi* and *L. simondi*. It is in the maturing phase that gametocytes distort their host cells into elongated and spindle shaped forms.

Macrogametocytes have a nucleus which stains red in contrast to the pale-staining diffused nucleus of the microgametocytes. Gametocytes of many

Leucocytozoon species contain volutin, which weakly reflects light, unlike the malarial pigment (hemozoin). This pigment results from complete digestion of haemoglobin by the parasite and is absent in leucocytozoids (Valkiūnas 2005).

Figure 2.7 *Leucocytozoon simondi* showing both fusiform and round gametocytes from the blood of *Anas perrelope*; **1**—**4**, **6**, macrogametocytes; **5**, **7** microgametocytes; **Che**—cytoplasm of host cell; **Nc**—nucleolus; **Nhc**—nucleus of host cell; **Np**—nucleus of parasite, **Vc**—vacuole; **Vg**—valutin granule. An uninfected erythrocyte is shown for comparison (taken from Valkiūnas 2005).

Ratios of macrogametocytes and microgametocytes in the peripheral blood differ, although gametocytes in small numbers remain after infection. According to Valkiūnas (2005) synchronized relapses occur in the avian hosts during the breeding season, stimulated by sexual hormones, resulting in a renewal of exoerythrocytic merogony within the internal organs. Such relapses are essential to the vector's survival, so that infections of new vector populations and host offspring can occur.

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Pathogenicity

Infection by leucocytozoonosis is highly pathogenic in domestic and zoo birds (Bennett *et al.* 1982) and may be pathogenic in young raptors (Jennings 1996). In young birds the death rate can exceed 85% whereas older birds are more resistant (Roberts & Janovy 2005). Studies have shown that *Leucocytozoon caulleryi* infections are devasting to poultry (Noblett *et al.* 1975), *L. smithi* to domestic turkeys (Stoddard *et al.* 1952, Kissam *et al.* 1973, 1975) and *L. simondi* to domestic ducks (Roberts & Janovy 2005), while also being a limiting factor in waterfowl populations (Herman *et al.* 1975, Herman & Bennett 1976). High mortalities in domestic and zoo birds may result from the absence of parasitic resistance, which may be present in wild birds of the same species (Bennett *et al.* 1992). Anaemia is a typical symptom of leucocytozoonosis which coincides with increased leukocytes. Birds with chronic infections have weakened immune systems and reduced reproductive capability. With severe infections, pathological changes lead to emaciation, dehydration, convulsions, and eventually to death (Adler & McCreadie 2002). With severe infections, certain pathological changes, such as internal organ transformation take place. These lead to liver enlargement which becomes necrotic, the spleen increases

up to 20 times in size, heart muscles become pale, and lungs become conjested, finally leading to death (O'Roke 1934). An outstanding feature of an outbreak of leukocytozoonosis is the suddenness of onset.

GENUS *Plasmodium*

All avian plasmodia are taxonomically diverse yet closely related protozoans (Figure 2.1) which undergo development in the circulating blood and tissues of the avian host. Common to all avian plasmodia species is transmission by mosquito vectors, variation in life cycles and pathogenesis—not unlike human malarias. These variations can be related to intrinsic qualities of the species and to variation in susceptibility among host species, including age and general health status. What sets avian *Plasmodium* apart from *Haemoproteus* and *Leucocytozoon* as an important protozoan parasite, was its extensive use as an experimental laboratory model to study human malaria, host-parasite interactions, and ecological modelling (Garnham 1966, Hamiliton & Zuk 1982, Read 1988, Atkinson & van Riper 1991). Danilewsky (1885) was the first to study the morphology of malaria and its effects on avian hosts. He also described the process of exflagellation in fresh blood. Starting with Danilewsky's studies and continuing with subsequent workers, confusion existed for many years regarding the identity between true plasmodia and *Haemoproteus*. Gametocytes of certain species of avian *Plasmodium* and *Haemoproteus* are almost indistinguishable. Complicating the identity between the two genuses further is the appearance of mixed infections, which can occur in many avian species. Order came about with the development of Romanovsky (Giemsa) stains for clear visibility of the parasites in methanol-fixed blood smears and when MacCullum (1898) clarified microgametogenesis, coupled with the discovery by Ross (1898) of the development of *Plasmodium relictum* in mosquitoes, and its transmission to sparrows. The futher unraveling of the plasmodial life cycle (Huff 1935, Raffaele 1936, James & Tate 1937) exposed the close relationship between *Haemoproteus, Leucocytozoon* and *Plasmodium*.

Avian malaria knowledge which accumulated mainly in the 1930—1950s as a result of being studied as an experimental model for human malaria had lead to the life cycle of the species *Plasmodium relictum* (Figure 2.8) from the subgenus *Haemanoeba* being comprehensively studied—being the most widely occurring species in its avian host. This line of research ceased when it was discovered that rodent malaria was biological akin to human malaria and more convenient for experimental research (Valkiūnas 2006). Thus the life cycles of many malaria species remain fragmentarily studied, while species from the subgenus *Novyella* remain uninvestigated.

The 38 valid specific species of avian plasmodia parasites are distributed worldwide with the exception of the Antarctic and sub-Antarctic islands. Most species of avian *Plasmodium* occur over a range of several orders, families and species, especially *P. relictum, P. circumflexum* and *P. vaughani* (Bennett *et al.* 1982). Over 45% of world bird species have been studied for avian haemosporidians of which approximately 50% of bird species researched having been parasitized by avian *Plasmodium* (Valkiūnas 2005). In the African tropics the situation is rather similar, with avian species infected with malaria reaching 20—50% (Crewe 1975). Thirteen avian plasmodia species have been recorded in 3.5% of host species which represented 10.3% of infected birds in the Afrotropical Region (Bennett *et al.* 1992). This contrasts markedly with the 30% infection rate encountered worldwide or in the African tropics (Valkiūnas 2005).

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Avian plasmodia are transmitted by most species of mosquitoes from the genus *Anopheles*, which is responsible for human plasmodia transmission. Avian plasmodia are not host or order specific (Janovy 1997) whereas *Haemoproteus* and *Leucocytozoon* are order specific (Valkiūnas 2005). Certain avian families have a high incidence of infection (Fringillidae, Emberizinae, Sturnidae, Ploceidae and Zosteropidae), while some families are rarely infected with plasmodia (Laridae, Scolopacidae, Alcidae) (Valkiūnas 2005). Factors involved in the dynamics of infection and prevalence remain unclear, although one or a combination of factors, such as host susceptibility, avoidance behaviour, habitat selection, environmental conditions and coevolution of host and parasite, all appear to play a significant role (van Riper *et al.* 1994). Studies undertaken with rodent malaria *Plasmodium chabaudi* have found that mixed clone infections are detrimental to mosquitoes (Taylor & Read 1997), contrasting with conventional selection-that parasites evolve reduced virulence because their fitness depends on the survival of the host. Thus a plasmodia protozoa transmitted by vectors may not suffer a reduction in fitness by harming its host. These parasites can thus evolve a higher efficiency of host exploitation (Tompkins *et al.* 2002), which could account for the host's immune response being genotype specific (Wilson *et al.* 2002).

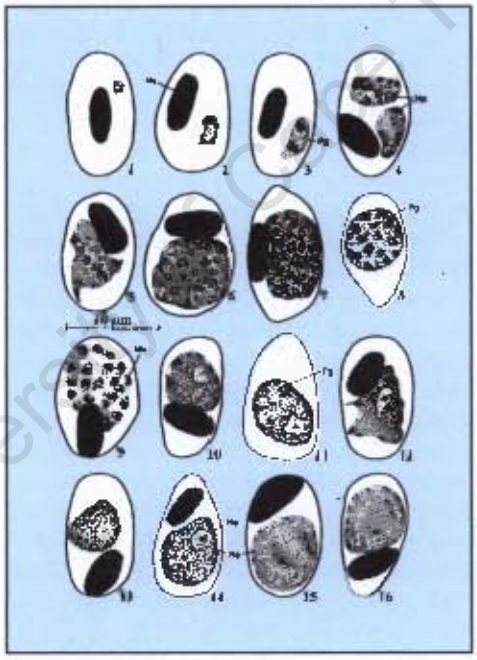

Figure 2.8 *Plasmodium relictum* from the blood of *Passer hispaniolensis*. 1—2, trophozoites; 3—9 erythrocytic meronts; 10—14, macrogametocytes; 15—16,microgametocytes; **Me**—merozoite; **Nc**—nucleus; **Ne**—nucleus of erythrocyte; **Np**—nucleus of parasite; **Pg**—pigment granule. (taken from Valkiūnas 2005).

Huff (1931) also demonstrated that haemosporidia are restricted to a particular vector according to genetics. His studies found that susceptibility of *Culex pipiens* to *P. cathemerium* was dependent on a single recessive gene. Thus a mosquito receptive to one species of avian malaria will not be receptive to infection from other malaria species. Irrespective of such specifics, van Riper *et al.* (1994) caution researchers working in the field of avian plasmodia against assuming that only culicine mosquitoes are responsible for the transmission of avian malaria. They foresee the possibility that blood sucking arthopods such as Ceratopogonidae, Simuliidae, Tabanidae and Psycodidae could also be implicated in transmission of avian plasmodia.

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This may well be true among the penguin population on Bird Island, Algoa Bay, where according to microscopy and PCR analyses a high prevalence of avian plasmodia occurs (pers. obs., D.U. Bellstedt pers. comm.) in the absence of mosquitoes (N.T.W. Klages & R.M. Randall pers. comm.).

Life cycle in the vector (invertebrate host)

When suitable erythrocytes containing gametocytes are ingested by the mosquito, they develop into gametes to be released from the disintegrating erythrocyte membrane to mature from a macrogametocyte into a macrogamete, with few morphological changes. In contrast, a microgametocyte transforms through exflagellation, becoming extracellular, with its nucleus dividing repeatedly to form six to eight nuclei within 10 to 12 minutes (Roberts & Janovy 2005). Gametogenesis is controlled by falling temperature and rising pH caused by escaping dissolved carbon dioxide from the blood with the transfer of erythrocytes from the vertebrate host to the mosquito midgut (Nijhout & Carter 1978, Carter & Graves 1988). During the process of gametogenesis the life span of microgametes is short, since it swims about until it finds a macrogamete, penetrating the cytoplasm and fertilizing it. Once together, the two nuclei fuse into a diploid zygote which elongates, developing into a motile ookinete (Figure 2.9) resembling a merozoite in morphology. It is 10 µm to 12 µm in length and has a large centrally positioned nucleus with a distinct nucleolus, cristate mitochondria with electron-dense matrix. It has polar rings and microtubules which are located beneath the inner membrane of the pellicle for motility (Sinden 1984).

Ookinetes migrate intracellularly and intercellularly (Torii *et al.* 1992) through peritrophic membrane to the hemocoel side of the mosquito's midgut. Oocyst development then occurs, resulting in the disappearance of the subpellicular microtubules and the ookinete pellicle, including the oocyst growth projecting into the hemocoel. As with all other coccidians, meiosis takes place immediately after zygote formation (Sinden & Hartley 1985). Internally the formation of a number of haploid nucleated masses of the cytoplasm into sporoblasts occur, which in turn divide repeatedly to form thousands of sporozoites (Rosenberg & Rungsiwongse 1991) within 10 days to two weeks, depending on *Plasmodium* species and temperature (Roberts & Janovy 2005, Valkiūnas 2005). When the oocyst ruptures, sporozoites migrate throughout the mosquito's tissue towards the salivary gland, from where they are injected into a new host at the next blood-meal. Infected mosquitoes remain infective for life, capable of transmitting avian plasmodia to every bird it feeds on (Roberts & Janovy 2005). The parasite's presence within the mosquito, comes the stimulus for more frequent feeds to increase transmission rates (Morell 1997).

Life cycle in avian host (vertebrate host)

Plasmodia parasite development within avian hosts occurs in four phases. The first phase is primary exoerythrocytic merogony, in which two minor developmental stages occur, consisting of primary (pre-erythrocytic) and secondary (posterythrocytic) stages. Two generations of meronts take place within the primary stage consisting of cryptozoites and metacryptozoites, with numerous generations of meronts consisting of phanerozoites in the secondary stage. Thereafter erythrocytic merogony and the formation of gametocytes complete the vertebrate developmental cycle (Figure 2.9).

Transfer to vertebrate

Only female mosquitoes are blood-feeding and vectors for spreading avian plasmodia. The males lack mandibular stylets, thus feeding only on plant juices. Female mosquitoes start blood-feeding on vertebrate hosts at least 1—3 days after adult emergence. This can be postponed until after mating and sugar feeding (Foster & Walker 2002). The female cuts into a blood vessel with stylets, while the labium serves as its guide while being bent backwards without penetrating the skin. Saliva flows from the tip of the hypopharynx as the fascicle bends probing for a small arteriole or venule. The saliva contains apyrase, an inhibiting agent for platelet aggregation, causing punctured vessels to bleed freely, including anticoagulants which prevent blood clotting, making its ingestion easier. When the tip of the fascicle is inserted into the lumen, accumulated sporozoites in the salivary glands are transmitted via the saliva to the bird host. At the same time, also drawing blood up through the food canal by pumps in the cibarium and pharynx-ingesting erythrocytes containing possible gametocytes. The blood accumulates in the midgut while the mosquito engorges fully in 1—4 min (Foster & Walker 2002). If the gametocytes are unsuitable to the mosquito, they are then digested along with the blood meal (Roberts & Janovy 2005).

Primary exoerythrocytic merogony

After the injection of sporozoites with salivary secretion into the peripheral blood, they migrate to tissues of the host bird. The mechanisms employed by parasites for penetration into host cells remain unclear (Perkins 1992). Avian plasmodia sporozoites resemble those of mammalian species by being elongate, spindle-shaped and 9 µm in length. Sporozoites have a polar ring at their apical ends to which 12 subpellicular microtubules are attached for motility (Vanderberg 1974, Vanderberg *et al.* 1990). Internal organelles of sporozoites include a nucleus, mitochondrion, cystostome composed of a pair of concentric electron-dense rings, rhoptries and variable numbers of micronomes. The initial asexual reproduction cycle starts in the fibroblasts and cells of the lymphoid-macrophage system in the spleen, including similar cells immediately around the site of the mosquito bite (Huff 1969). Upon invasion of host tissue, the worm-like sporozoites transform into 30 µm diameter exoerythrocytic meronts or cryptozoites, losing the specialized organelles of host penetration, which results in the first generation of primary exoerythrocytic meronts. This developmental phase is termed merogony. The parasite obtains its nutrients for growth and differentiation by diffusion across the membrane of the parasitophorous vacuole and by phagocytosis of the host cell cytoplasm or of material within the parasitophorous vacuole (van Riper *et al.* 1994). Ingestion occurs through a cytosome composed of two concentric electron-dense rings (Beaudoin & Strome 1972). The internal diameter of a developing cytosome growing exoerythrocytic, ranges from 40—50 µm in meronts to 80—50 µm in merozoites. Material ingested through the cystosome is degraded within membrane-bounded food vacuoles. Merozoites which develop in cryptozoites stimulate a second generation of primary exoerythrocytic meronts, which results in metacryptozoites, which usually encapsulate less than 1 000 merozoites (Valkiūnas 2005).
Merozoites produced by exoerythrocytic meronts are 3—4 µm in length and 1—2 µm in width (Hepler *et al.* 1966), which is 2—3 times longer than erythrocytic produced meronts. When the metacryptozoite ruptures, releasing the merozoites, it also induces simultaneous development of the next generation.

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Part of the developed merozoites transform into metacryotozoites and phanerozoites, while the remainder are capable of invading erythrocytes and stimulating agamic development in the circulating blood.

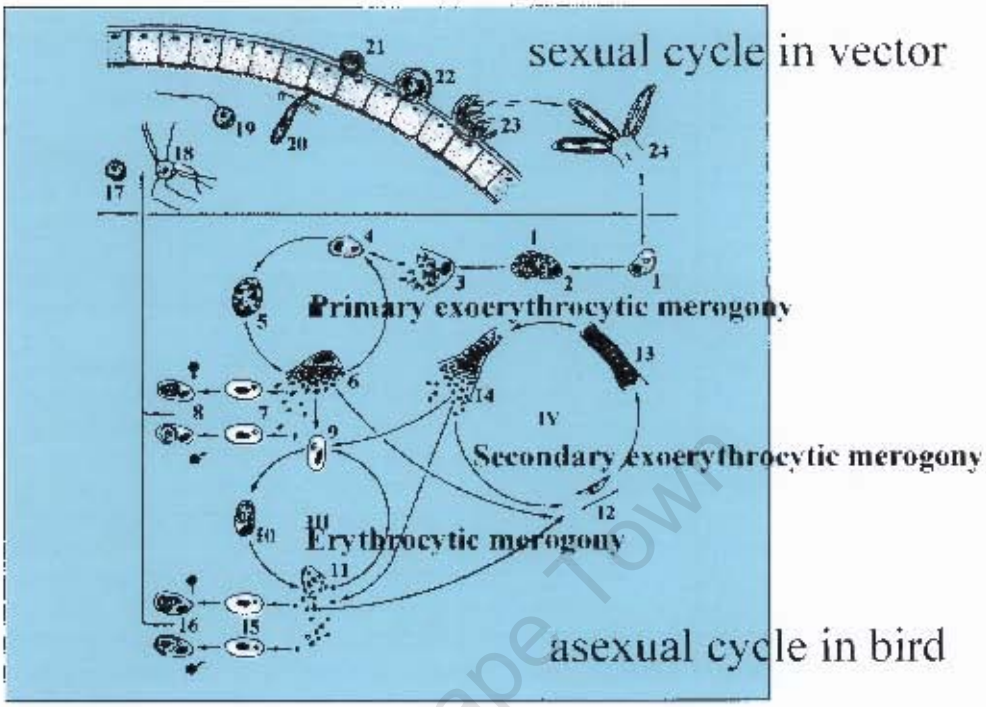

Figure 2.9 Life cycle of *Plasmodium relictum*. **1** sporozoite in reticuloendothelial cell; **2—3** cryptozoites; **4** merozoites in macrophage; **5—6** metacryptozoites; **7** merozoites in erythrocytes; **8** gametocytes; **9** merozoites in erythrocyte; **10-11** erythrocytic meronts; **12** merozoites in endothelial cell of capillaries; **13—14** phanerozoites; **15** merozoites in erythrocytes; **16** gametocytes; **17** macrogamete; **18** exflagellation of microgametes; **19** fertilization of macrogamete; **20** ookinete penetrating the peritrophic membrane; **21** young oocyst; **22—23** sporogony; **24** sporozoites in the salivary glands of vector (modified from Valkiūnas 2005).

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Secondary exoerthrocytic merogony

The panerozoites, part of the merozoite development, penetrate tissues of many organs, including the brain, inducing secondary exoerythrocytic merogony in conjunction with the merozoites developing into metacryptozoites. Mature first generation phanerozoites induce relapses (Valkiūnas 2005), and together with erythrocytic meronts maintain parasitemia during chronic stages of infection.

Erythrocytic merogony

Certain merozoites are phagocytized by Kupffer cells within the liver as a host defence system (Terzakis *et al.* 1979), while others penetrate into erythrocytes. Cell penetration studies of *Plasmodium elongatum* (Ladda *et al.* 1969) and later studies by Aikawa *et al.* (1978) on primate plasmodia, confirmed the mechanisms of penetration. The merozoite anterior is pressed into the plasma membrane of the erythrocyte, forming an ever increasing depression until entry is gained and the membrane seals. Once inside the merozoite metamorphoses into round trophozoites in which its nucleus and cytoplasm increase as the parasite grows within the erythrocyte cytoplasm. Trophozoites feed on host cell cytoplasm by way of cytostomes, accumulating malarial pigment (hemozoin), an iron containing by-

CHAPTER THREE

Study sites in the Greater Cape Town area

We keep in view these facts—that the minor features of the earth's surface are everywhere slowly changing; that the forms, and structure, and habits of all living things are also slowly changing; while the great features of the earth, the continents, and oceans, and loftiest mountain ranges, only change after very long intervals and with extreme slowness, we must see that the present distribution of animals upon the several parts of the earth's surface is the final product of all these wonderful revolutions in organic and inorganic Nature...

Alfred Russel Wallace (1823-1913)
The Geographical Distribution of Animals

University of Cape Town

INTRODUCTION

The collection of blood smears for analyzing avian haemosporidian parasites started with a request from Earlé & Bennett (1991) for African material, representing the Afrotropical region. This effort by ringers in Southern Africa to supply samples on a regular basis gathered momentum country wide slowly, with renewed requests to the ringing community (Earlé & Bennett 1993a, 1993b). The making of blood smears became an extension of the established bird ringing procedure, taking place at existing ringing sites. Due to logistical and practical constraints, no new ringing sites were added to the 10 which had been operational prior to the study period in the greater Cape Town area, within the Western Cape Province, South Africa (Figure 3.1).

Each sampling site has varying environmental and ecological conditions, with varying dominant vegetation types within meso and microclimates, situated within the Cape Floristic Region of the Fynbos biome (Low & Rebelo 1996). The following paragraph describes the region in general, followed by sections describing each of the 10 study sites at which blood smears were collected for analyses of avian haemosporidian parasites.

This is the smallest of the six floristic kingdoms of the world, and with 8 550 plant species is one of the most diverse (Acocks 1988, Cowling *et al.* 1986). The natural vegetation is typically low, species–rich heathland, comprising varying proportions of woody Proteaceae, grassy Restionaceae and ericacious heaths (Cowling *et al* 1997). Most indigenous vegetation has been replaced or modified by development with remnants existing on high lying slopes too steep for development. Study sites confined to the coastal strip (Koeberg, Bettys Bay and Glencairn) have dune thicket and flat areas. Durbanville, Tygerberg and Rondevlei have Sand Plain Fynbos, with patches of Afromontane Forest on the protected eastern slopes of mountains (Kirstenbosch) (Cowling *et al.* 1986, Low & Rebelo 1996).

The spread of invasive alien vegetation, which comprises mostly pines, eucalypts and acacias, as well as urban and peri-urban sprawl, has modified many of these habitats (Glencairn and Mowbray), including many of Greater Cape Town's catchment and river system. Transformation of the river fauna of the Cape Floristic Region's rivers from 1890 onwards has been caused through stocking of European fish species such as Brown Trout *Salmo trutta*, Carp *Cyprinus carpio*, and North American species such as Largemouth Bass *Micropterus salmoides* and Rainbow

product of haemoglobin digestion, which the parasite cannot metabolize (Yamada and Sherman 1979, van Riper *et al.* 1994). On maturing, trophozoites undergo nuclear division to form meronts. Within meronts, asexual multiplication and division occurs, forming uninuclear merozoites. The development process within erythrocytes is identical to exoerythrocytic tissue development, only with fewer merozoites (Aikawa 1971). The number of merozoites within a meront aids identification of plasmodia species (Valkiūnas 2005).

When merozoite development is completed, meronts rupture releasing parasitic metabolic wastes, body residuals and hemozoin, all responsible for typical malaria symptoms. Hemozoin is toxic to macrophages depressing their effectiveness as phagocytes (Turrini *et al.* 1993). Erythrocyte merogony development varies from 24 to 36 hours, according to plasmodia species, with synchronization of all organisms maturing within the blood during early morning. Although the cause of periodicity remains unclear (van Riper *et al.* 1994), there are species which have well defined periodicity (*Plasmodium cathemerium, P. gallinaceum, P. matutinum*) and species that do not (*P. relictum, P. rouxi,* and *P. vaughani*) (Valkiūnas 2005).

Gametocyte formation

Merozoites formed within erythrocytic meronts stimulate further cycles of erythrocytic merogony (Valkiūnas 2005), resulting in merozoites entering uninfected erythrocytes to become macrogametocytes (female) or microgametocytes (male) as depicted in Figure 2.8. Studies by Bruce *et al.* (1990) using *Plasmodium falciparium* cultures have shown that merozoites from an individual meront become either a gametocyte or a new generation of asexual parasites.

Gametocytes not ingested by the vector die and are phagocytized by the reticuloendothelial cells and leukocytes (Roberts & Janovy 2005). If destroyed and infected erythrocytes are not balanced by synthesis and the release of immature erythroblasts, severe anaemia results.

Pathogenicity

Avian plasmodia is geographically widespread varying considerably in pathogenicity among parasites and strains (van Riper *et al.* 1994). Parasites of the same species also differ in pathogenicity in different hosts (Grim *et al.* 2003). Reasons for such differences remain unclear, although variation may be due to intrinsic qualities and susceptibility among host species, including age and health status (Foster & Walker 2002). Studies indicate that acute malaria and death are caused by *P. cathemerium, P. relictum* and *P. circumflexum* to wild birds (Garnham 1966, van Riper *et al.* 1994), while *P. gallinaceum, P. durae* and *P. juxtanucleare* affect young and adult domestic fowl (Garnham 1980, Huchzermeyer 1993), and *P. hermansi* affects both wild and domestic turkeys (Foster & Walker 2002). *Plasmodium relictum* is the most common species of avian plasmodia occurring in birds worldwide. It is considered the most pathogenic of all avian species (Garnham 1966, Cranfield *et al.* 1990), especially to penguins in zoos, in conjunction with *P. elongatum* (Fleischman *et al.* 1968, Sladen *et al.* 1979, Stoskopf & Beier 1979, Beier & Stoskopf 1980, Beier & Trpis 1981, Fix *et al.* 1988, Cranfield *et al.* 1990, Brossy 1992, Graczyk *et al.* 1994b). When penguins are exposed to avian plasmodia infections, or have recrudescence and relapses under stressful nutritional and environmental conditions, prevalence and morbidity are severe (Brossy 1992, van Riper *et al.* 1994). This could result from exposure to

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new populations of vectors absent from the cold and windy conditions which dominate their natural habitats (Brossy 1992, van Riper *et al.* 1994).
Young birds are more susceptible to avian plasmodia than adult birds (Herman 1938, van Riper *et al* 1986, Foster & Walker 2002), although studies on *Spheniscus demersus* by Graczyk *et al.* (1994a) show that maternal antibody transfer of *Plasmodium* species occurs, detectable up to eight weeks after hatching.

Plasmodia infection to birds after sporozoite transmission from an infective mosquito may develop into acute infection. This results in anaemia, enlargement and whitening of the spleen with accumulation of pigment in the liver (Foster & Walker 2002, Valkiūnas 2005). These may well be related to tissue necrosis (Garnham 1966) due to effects of blockages of the brain, capillaries and other organs and tissues (Atkinson & van Riper 1991).

RELAPSES

Relapse mechanisms in bird haemosporidians remain unclear and poorly studied (Valkiūnas 2005). Studies of primary exoerythrocytic merogony by Short and Garnham (1948) seemed to have solved the mystery. It was assumed that merozoites simply reinfected hepatocytes, with subsequent reinvasion of erythrocytes, triggering a relapse. But not all species of *Plasmodium* cause relapses. Appelgate (1971) and Yang *et al.* (1971) have demonstrated that merozoites from exoerythrocytic merogony and erythrocytic meronts do contribute to a relapse. Atkinson *et al.* (1988) believe that meronts of *H. meleagridis* cause and maintain chronic parasitemia in turkeys, when developing in reticular cells of the spleen, while Valkiūnas (2005) is of the opinion that phanerozoites induce relapses.

Due to differences in life cycles of various genera of avian haemosporidian parasites a relapse occurs as secondary parasitemia after a latent stage of infection or as an increase in intensity during chronic parasitemia. The process which activities a relapse are exoerythrocytic merogony, characterized by high intensification of merogony. Exoerythrocytic merogony only occurs in Haemoproteidae and Leucocytozoidae families, but erythrocytic merogony in addition to exoerythrocytic merogony occur in Plasmodiidae. The occurrence of exoerythrocytic merogony is associated with a relapse whereas erythrocytic merogony is a recrudescence of high parasitemia, but not a relapse. Thus difficult to distinguish between recrudescence and a relapse in avian species infected with *Plasmodium* species, especially when employing the blood stained-smear and microscopy method. This results from parasites not always being present in the peripheral blood as intensity of chronic parasitemia may be low, or because of a latent period of infection which precedes the recurrence of high parasitemia.

Two relapses occur annually with most avian haemosporidia: non-seasonal and spring (Valkiūnas 2005), with spring being the most important in terms of parasite transmission. Non-seasonal relapse occurs without connection to season or known triggers (Barrow 1963), but could be stress related as with penguin cleaning during oil spillage events. Spring relapses have been well studied (Huff 1942, Applegate 1971, Alverson & Noblett 1977, Allan & Mahrt 1989), showing increased infectivity of gametocytes during this period, contrasting with a period of severe parasitemia but less severe infectivity. This has been demonstrated in studies with *Plasmodium relictum* (Applegate and Beaudoin 1969).

Increased infectivity during spring relapses is influenced by vertebrate hosts (Applegate 1971), resulting from increased sexual hormones in the blood during the breeding season. Sexual hormone and corticosterone injections have activated relapses (Haberkorn 1968, Applegate 1970).

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The same hormones are probably responsible for stimulating relapses of *Leucocytozoon tawaki* with the onset of the moult cycle in penguins (Fallis *et al.* 1976). Increased infectivity is also synchronized with increased vector activity in spring for maximum parasite ingestion and transmission (Morell 1997).

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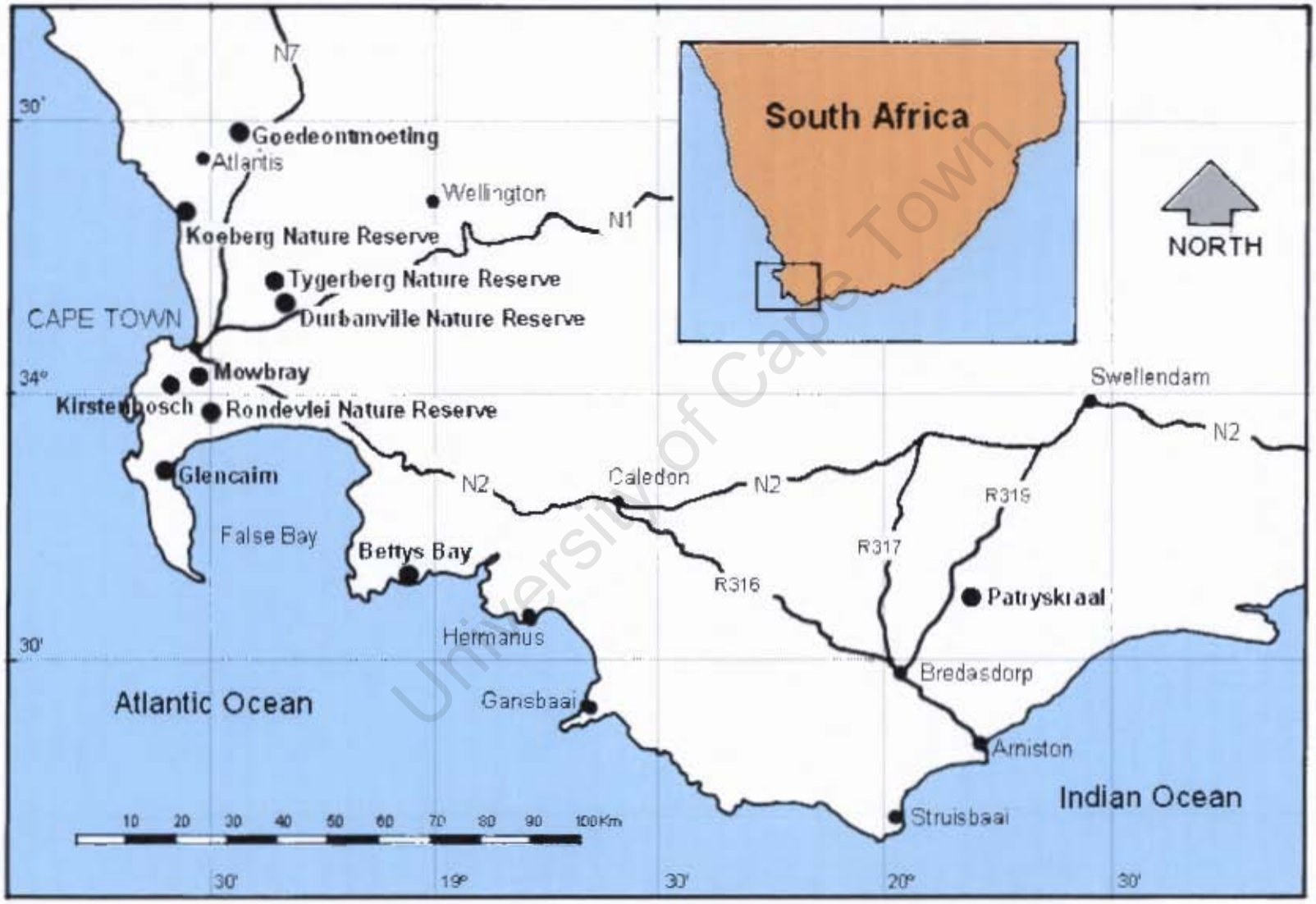

Figure 3.1 Location of 10 study sites within the Greater Cape Town Area

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Trout *Oncorhynchus mykiss*. Then from 1960, widespread domination by alien fish resulted from the introduction of Tilapia *Tilapia sparmanni*, Mozambique Tilapia *Oreochromis mossambicus* and Catfish *Clarias gariepinus*, all from Southern Africa but beyond the rivers of the Western Cape. Competition with indigenous freshwater fish for food, habitat, and predation has caused the near disappearance of Cape Galaxies *Galaxias zebratus* and Cape Kurper *Sandelia capensis* (River Health Programme 2005). This has led to an abundant increase in many invertebrate taxa (Lowe *et al.* 2005) including Simuliidae (blackflies), Ceratopogonidae (biting midges) and Culiciade (mosquitoes), which were heavily predated upon by indigenous fish (Lowe *et al.* 2005). This has resulted in a possible increase in prevalence of avian haemosporidian parasites. The invasion of the alien aquatic weed *Pistia stratiotes* water hyacinth on many of the regions water bodies has resulted in clogging water surfaces to rivers and dams, depleting oxygen levels, restricting aquatic life and fostering mosquitoes which are vectors of plasmodia.

Climate

All study sites are situated in the southwestern corner of South Africa and below the 1 000 m escarpments within the Winter Rainfall Region. The region has a warm, temperate, Mediterranean macroclimate, characterized by cool wet winters and warm dry summers, where extremes of temperatures are unusual. Due to ecological and vegetation resemblance between other Mediterranean regions this region is frequently compared with California, Chile, southwestern Australia and the Mediterranean Basin (Hockey *et al.* 1989). The warm Mozambique-Agulhas Current (22°C) of the Indian Ocean, flowing southwest ward along the east coast, meets the cold Benguela Current (15°C) which results from Antarctic Drift, at Cape Agulhas. These two ocean currents in conjunction with climatic gradients running from north to south, west to east, and the topography of coast to interior, and from lowland to mountains, create meso-and microclimates which result in a wide range of climatic variations. The combined influence has a marked impact on the climate and vegetation of the region. Although frost occurs in the high mountain valleys during winter months, it is not as severe as experienced on the Highveld, in the interior of the country, and a rare occurrence on the coastal flats. Rainfall is mainly cyclonic and orographic with occasional thunderstorms occurring about five times a year, with hail a rare phenomenon. Sunshine duration varies from about 60% during July in mid-winter, to over 70% in January. During winter and early spring, mountain peaks are occasionally snow-covered, but snow persists for short durations only (Schulze 1965, Slingsby & Coombe 2001).

Temperature

Summers are hot and very dry, with an average daily maximum air temperature of 28°C between January and March, the hottest months of the year. Although a maximum summer temperature of 40°C have been recorded the probability of getting temperatures higher than 34°C in February are infrequent (Agricultural Metereological Section: Elsenburg Agricultural Development Institute 1989). Winter months are cool, with an average daily minimum air temperature of 17°C recorded during July, while lower-lying areas have recorded 4°C, with -5°C at altitude in mountain valleys (Schulze 1965).

Rainfall

Summers are generally dry, with 80% of the annual rainfall occurring during winter from April to September. Annual rainfall varies from 382 mm to 500 mm on the Cape Flats to over 3 000 mm in mountain kloofs (Schulze 1965, Agricultural Metereological Section: Elsenburg Agricultural Development Institute 1989). Maximum days of rainfall during winter vary from 12—15 days per month, while the dry summer months experience 4—5 rain days per month (Schulze 1965). Rainfall over the winter rainfall region is mainly cyclonic due to frontal systems moving in from the southern Atlantic Ocean. A rapid decrease in rainfall is experienced with northwards movement along the coast, resulting from the cold Benguela Current which counteracts cloud development. Seasonal shift of the earth's wind zones causes unstable contact between the warm temperate wind zone and the cool temperate one, which generates the frontal action to move across southern Africa in winter and south of the land mass in summer.

Fog

A typical occurrence in this region during summer months from December to March is fog, which increases in frequency from south to north (Marshall & Mommsen 1994). This results from the strong southeasterly winds which causes upwelling of cold Atlantic Ocean water along the coast. Fog then drifts in from the sea covering areas of up to 3—5 km inland, which in conjunction with dew compensates for the lack of summer rain, as well as moderating summer temperatures.

Wind

The region is windy, which impacts on the coast, the coastal vegetation, invertebrates and vertebrates. Hot, dry offshore summer winds cause plants to dry out, if sufficient moisture is not supplied by fog, while salt-laden, on-shore winds stunt coastal shrub growth. Prevailing winter winds (20—40 mh^{-1}) are northwesterly rain-bearing and frequently reach gale force conditions, especially along the coast. Summer winds (25—50 kmh^{-1}) are mainly southerly to reasonably strong south-easterlies which produce the "table-cloth" (orographic cloud) on Table Mountain (Schulze 1965), also known as the "Cape Doctor."

1. BETTYS BAY

Site description

Bettys Bay (34°22'S 18°56'E) is situated east of Cape Hangklip on a narrow coastal plain between the Kogelberg Mountain range to the north and the coastline to the south, 46 km southeast of Gordon's Bay and 3 km west of the Palmiet River. Bettys Bay is a scenic coastal and holiday village, consisting of weekend and holiday cottages sprawling along the coast from Silversands in the west, to Dewetsbaai in the east. The Kogelberg Mountain has many deeply incised kloofs from which numerous streams run strongly after winter rains. Two of these, Disa Kloof with its reservoirs and Leopard Gorge with numerous pools and waterfalls, lie within the Harold Porter National Botanical Garden, situated north of Bettys Bay. An abundance of flowing fresh water especially in the perennially waterlogged areas on the south slopes of the

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Kogelberg Mountain, creates ideal habitats for biting midges, blackflies and mosquitoes, which are vectors for avian haemosporidian parasites.

Simulium dentulosum, S. hessei, S. merops and *S. harrisoni* are blackflies restricted mostly to the Western Cape, with *S. dentulosum* occurring near sea level. The other species prefer mountain torrents, waterfalls and cascades, where water quality is good to excellent. *S. hessei* occurs in mountain trickles while *S. rutherfoordi* and *S. nigritartse* can be found in slow-flowing mountain streams. *S. adersi* and *S. medusaeforme* are also found in the area in varying water-quality conditions (Palmer & de Moor 1998). A great diversity of oviposition sites exist in the area for mosquitoes, which would resume activity during summer with increased sunlight to the southern mountain side.

The Harold Porter Botanical Garden on the lower mountain slopes is habitat to fynbos and forest birds such as Cape Siskin *Serinus totus*, Cape Grassbird *Sphenoeacus afer*, Victorin's Warbler *Bradypterus victorini*, and Olive Woodpecker *Mesopicos griseocephalus*. From the cultivated gardens and beyond onto the narrow coastal plain, localized birds occurring are all common Fynbos species, including Black Saw-wing Swallow *Psalidoprocne holomelas*, Blue-mantled Crested-Flycatcher *Trochocercus cyanomelas*, African Dusky Flycatcher *Muscicapa adusta*, and Swee Waxbill *Estrilda melanotis* (Cohen *et al.* 2006).

Climate

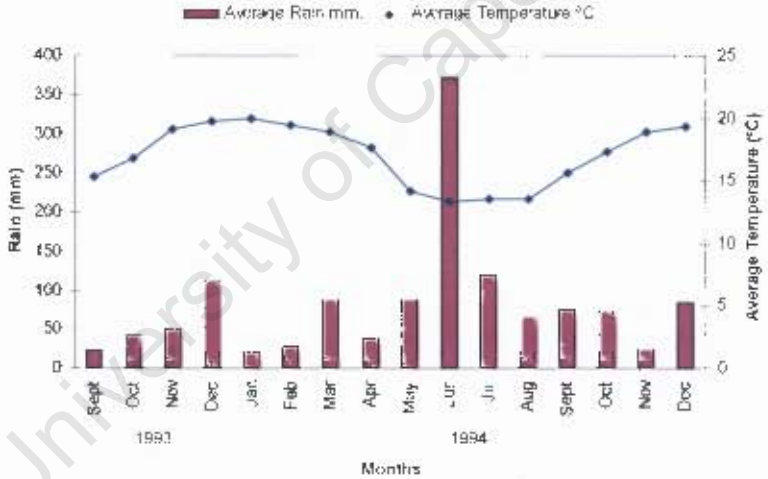

Figure 3.2 Average daily ambient temperature recorded at Hermanus (34°25'S 19°15'E); and monthly rainfall recorded at Harold Porter National Botanical Garden (34°52'S 18°58'E), from September 1993 to December 1994. (South African Weather Service)

Vegetation

The mesic Mountain Fynbos occurring along the coast from sea level to the mountain tops is dominated by a diversity of plant communities, of which Proteaceae, Ericaceae and Restionaceae are the primary constituents. The dominant plant species occurring here are *Protea grandiceps, P. repens, P. eximia, P. nitida, P. neriifolia, Leucadendron spissifolium, L. salignum, Leucospermum* species, *Erica hispidula, E. vestita, E. melanthera, Metalasia* species, *Anthochortis crinalis, Cliffortia serpyllifolia, Widdringtonia nodiflora*. Natural vegetation has been invaded in parts, particularly in areas of development, by exotic *Acacia cyclops* and *A. saligna* (Barnes 1998).

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2. DURBANVILLE NATURE RESERVE

Site description

Durbanville Nature Reserve (33°50'S 18°38'E) is situated 21 km from Cape Town, northeast of the Tygerberg Nature Reserve, surrounded by suburban development and the Durbanville Race Course to the northwest. Being only 6 ha in size, its main importance is that of a transition zone between Renosterveld and Sand Plain Fynbos. This is due to being situated on Malmesbury shale containing relatively nutrient rich clay soils, nutrient poor sands and alluvium (du Plessis 1997).

The area is dominated by the Tygerberg Hills which form a complex landscape, consisting of a range of hills running north to south. These vary with altitudes from 414 m above sea-level at Tierberg to 458 m at Kanonkop, linked with prominent valleys in between. The topography changes from being hilly in the south to less indented rolling hills at the northern and western boundaries of the Tygerberg. The eastern side towards Durbanville Nature Reserve is drained by the seasonal Elsieskraal River, while many seasonal streams in the north spring from Kanonkop, Hoogekraal and Humeklip, draining north and east into the perennial Diep River. Durbanville Race Course adjacent to the Nature Reserve, although sandy and well-drained, may occasionally become waterlogged which also results in streams. Blackflies of the species *S. medusaeforme* and *S. nigritarse* may occur in the area due to their adaptation to slow flowing water with a wide range of water-quality conditions, even tolerating pollution, with *Simulium medusaeforme* commonly found at stormwater outlets (Palmer & de Moor 1998). Aquatic and semi-aquatic conditions exist in the area, consisting of ponds, streams, algae-covered soil, decomposing plant material, leaf compost and litter—all oviposition sites for mosquitoes, including biting midges.

Climate

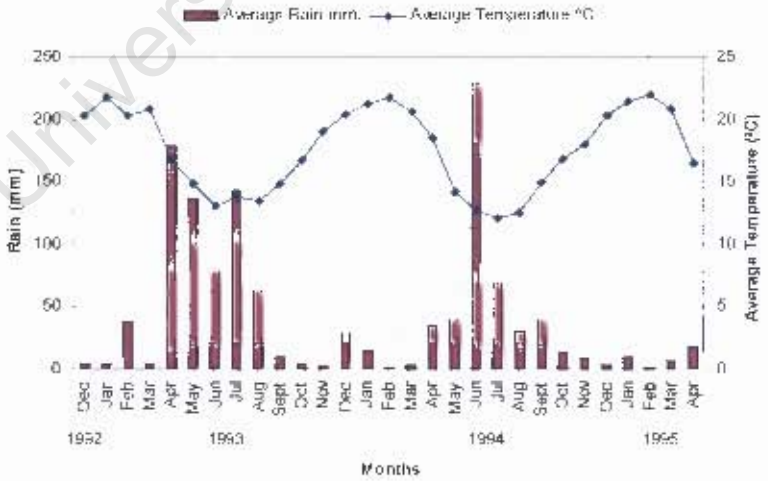

Figure 3.3 Average daily ambient temperature and monthly rainfall recorded at the Cape Town Weather Office, Cape Town International Airport (33°58'S 18°36'E), 17km south of Durbanville Nature Reserve, from March 1993 to April 1995 (South African Weather Service)

Vegetation

The Reserve is an ecotone where transition between Renosterveld and Sand Plain Fynbos occurs, resulting in high species diversity (Wood & Low 1993). Some transformation has taken place, making it a semi-natural reserve. Where undisturbed

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a mixture of species occurs, so that Renosterveld species such as *Anthospermum* species and *Elytropappus rhinocerotis* exist together with fynbos species such as *Metalasia muricata, Phylica plumosa* and *Protea repens*. A number of Red Data plant species occur in the Reserve (du Plessis 1997).

3. GLENCAIRN

Site description

The study site is a residential erf within Glencairn (34°09'S 18°25'E) situated 4 km north of Simon's Town, on the False Bay coast, 6 km from Fish Hoek and 37 km from Cape Town. This small, predominantly residential area is situated in the Elserivier Valley around the slopes of Else Peak, below the water supply dam on the upper Else River. It straddles the wetland which runs along the valley bottom to drain into Elsebaai within False Bay. The water supply dam has reduced the flow to the lower reaches of the river, although numerous small streams drain into the river and wetland below the dam. Water treatment residue occasionally released from the Brooklands Water Treatment Plant in the upper Else River reduces water quality down river (River Health Programme 2005). The blackfly species *Simulium dentulosum*, *S. merops* and *S. rutherfoordi* which is found in the Western Cape, could also occur in the foothill streams on the lower slopes of Else Peak where water quality is good to excellent. *S. nigritarse* would be found in the slow-flowing downstream Else River and wetlands, where it tolerates polluted water. Numerous habitats exist for both biting midges and mosquitoes. The predominately southwesterly winds from False Bay could have an impact on the flight of mosquitoes thereby inhibiting biting activity and affecting population density.

Climate

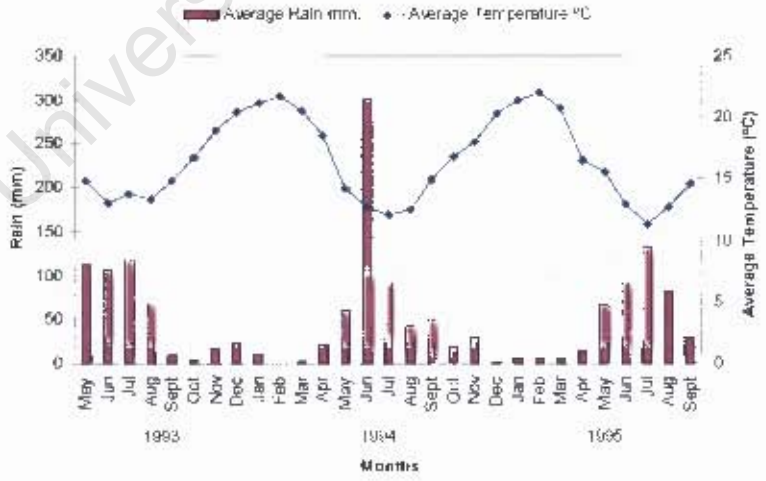

Figure 3.4 Average daily ambient temperature was recorded at Cape Town Weather Office situated at Cape Town International Airport (33°58'S 18°38'E), 26 km north of Glencairn. Monthly rainfall was recorded 3km north at Fish Hoek (34°08'S 18°26'), from May 1993 to September 1995. Both sites of recording have typical temperature and rainfall patterns common to Glencairn. (South African Weather Service)

Vegetation

Natural terrestrial strandveld surrounds the wetland and suburban development, consisting of scattered perennial overstorey of spinescent species, succulents and moderately tall evergreen thickets including *Metalasia muricata, Phylica* species and

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Chrysanthemoides monolifera. Areas of the strandveld are heavily invaded by an alien woody overstorey, consisting mainly of *Acacia cyclops* and *A. saligna*.

Typical aquatic vegetation associated with the wetland comprises reed, rush and sedge beds, with kikuyu-grassed banks. *Typha capensis* and *Phragmites* spp. dominate the reed beds. The reed marsh consists of *Phragmites australis*, with the sedge marsh dominated by *Bolboschoenus maritimus* and *Juncus kraussi* (Barnes 1998).

4. GOEDEONTMOETING

Site description

The farm Goedeontmoeting (33°41'S 18°36'E) is situated in the Swartland agricultural region 30 km north of Cape Town, which includes the hilly regions in the north, from Darling, Mamre, Paardeberg and Riebeeck to the rolling plains in the south at Tygerberg Hills. Farming is mixed, consisting in the main of wine-growing, small grains and animal husbandry, of which the latter two are the main sources of agricultural income of the farm Goedeontmoeting.

The surrounding farmland is drained by a moderately polluted Mosselbank River (River Health Programme 2005), which runs north of the farm building complex —which was also the ringing site. At Philadelphia to the northwest, the Mosselbank River turns southwards from where it continues as the Diep River. During winter, high rainfall results in the river flowing strongly, but drains quickly. During the hot dry summer months the water course consists of a series of pools.

Mosquito populations could be stable throughout the year due to conditions of a low wind factor across the Swartland and numerous oviposit sites, consisting of open water surfaces, mud debris and leaf-litter, habitats also suitable for biting midges. *Simulium adersi, S. bovis, S. medusaeforme*, and *S. nigritarse* occurs in slow-flowing water having a wide range of water-quality conditions, with *S. nigritarse* and *S. ruficorne* tolerating polluted water. *S. vorax* is restricted to fast-flowing rivers after the winter rains when water quality is moderate to excellent, so too *S. rutherfoordi*, but then only in the slow-flowing foothill streams (Palmer 2002).

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Climate

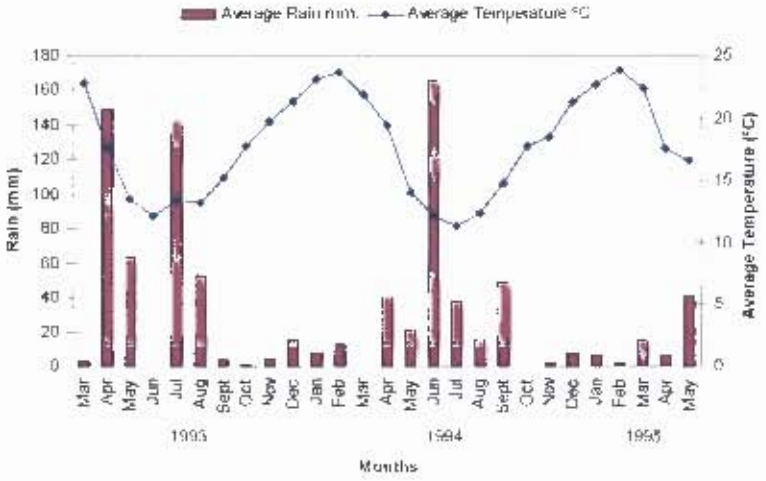

Figure 3.5 Average daily ambient temperature and monthly rainfall was recorded at Malmesbury (33°28'S 18°44'E) 27km north of Goedeontmoeting, from March 1993 to May 1995 (South African Weather Service)

Vegetation

Large-scale agriculture has greatly transformed the Renosterveld, reducing the original extent of the natural vegetation. What remains is confined to the small kloofs and watercourses too steep or rocky to plough. Fragmented Renosterveld patches found throughout the area are dominated by *Elytropappus rhionceroti*, while aquatic vegetation is dominated by *Chondropetalum tectorum* and other restioids, such as *C. rectum* and *Thamnochortus erectus*, dominate this community. Alien shrubs and plants, including clumps of *Eucalyptus camaldulensis*, *Schinus molle* and indigenous *Phoenix recinata* dominate vegetation surrounding the farm building complex.

5. KOEBERG NATURE RESERVE

Site description

The Koeberg Nature Reserve (33°40'S 18°26'E) was proclaimed in 1992 (Marshall & Mommsen 1994, Greeff *et al.* 2001) when the farms Duynefontein and Kleine Springfontein, were bought by Eskom to surround the Eskom Koeberg Nuclear Power Station of which the first phase became operational in 1982. This 3 000 ha Reserve is situated on the West Coast, 30 km north of Cape Town, with Melkbosstrand to the south and Atlantis to the north east, bounded by the Atlantic Ocean to the west and the West Coast Road to the east.

The Reserve is characterized by long sandy beaches with extensive dune plumes. Strandveld vegetation ranges from sprawling species growing close to the ground and short open scrub to dense tall thicket growing to over 2 m in areas protected from the prevailing winds. A bird list of 209 species has been recorded in the reserve (Marshall & Mommsen 1994). With the eradication of alien plants (e.g. acacias) and the return of the natural vegetation to the area, a number of species is evident in the thicker vegetation such as White-backed Mousebird *Colius colius,* Cape Pendline-Tit *Anthoscopus minutus,* Cape Bulbul *Pycnonotus capensis,* Cape Robin-Chat *Cossypha caffra,* Karoo Scrub-Robin *Erythropygia coryphaeus,* Layard's Tit Babbler *Parisoma layardi,* Grey-backed Cisticola *Cisticola subruficapilla,* and Long-billed Crombec *Sylvietta ruficapilla.*

Numerous and extensive wetlands including a salt pan occur on the reserve, which are typically formed in Strandveld depressions (Greeff *et al.* 2001), above deep underlaying calcareous coastal Quaternary sands (Marshall & Mommsen 1994). The wetlands are filled during winter rains, normally retaining significant levels throughout summer. Three reclamation dams resembling natural vleis were built in the reserve due to the high water table, to contain recycled waste water pumped from the Atlantis Industrial Area (G. Greeff pers. comm.) Ideal habitats for mosquito and biting midges are created in these water bodies, including the seepage water from the sand dunes along the high water margin of the beach, although the prevailing strong southeasterlies and northwesterlies could impact upon mosquito population densities. The blackfly species *S. merops* found in the Okavango Swamps (de Meillon 1955), southern and Western Cape and particularly Kirstenbosch Gardens, may occur in the water bodies of Koeberg. *Simulium medusaeforme* occurs in a wide range of stream types and water-quality conditions, such as trout hatcheries, stormwater outlets (Palmer & de Moor 1998), and may be present in the Koeberg area, together with *S. nigritarse* which is usually found in slow-flowing water.

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Climate

The reserve falls within the Mediterranean Climatic Zone of South Africa, situated within the Winter Rainfall Region. It has hot, dry summers and cool rainy winters, when 80% of the annual rainfall occurs between April to September. The reserve receives a mean annual rainfall of 422 mm (Marshall & Mommsen 1994). Average daily temperatures between summer and winter range from 28°C—17°C are recorded on the reserve.

Summer months are characterized by prevailing strong south to southeasterly winds, persisting for several days, deforming vegetation into a stunted growth pattern where exposed. The rain bearing northwesterly winds predominate during winter months.

Vegetation

The reserve has Sandveld, Coastal fynbos, Renosterveld and Strandveld which is the dominant vegetation type and of which only 1% is protected along the West Coast (Greeff 2001). Common Strandveld species found in the reserve include *Chrysanthemoides monilifera, Ehrharta villosa, Euclea racemosa, Pelargonium capitatum* and *Rhus* species. Wetland vegetation consist mainly of *Scirpus nodosus, Juncus kraussii, Chondropetalum tectorum* and *Typha capensis.* Large, transformed open areas are dominated by a mixture of grasses: *Pennisetum clandestinum, Stenotaphrum secundatum, Paspalum distichum, Lolium perenne* and *Cynodon dactylon* (Greeff 2001, Marshall & Mommsen 1994). Almost 25% of the Reserve has been invaded by alien *Acacia cyclops* and *A. saligna* (Greeff 2001), which has since been cleared and replaced by well established indigenous vegetation.

6. KIRSTENBOSCH NATIONAL BOTANICAL GARDENS

Site description

Kirstenbosch (33°58'S 18°26'E) is one of the major botanical gardens in the world, and the first to be devoted to indigenous plants. The gardens also houses the National Biodiversity Institute of South Africa, situated 12 km from Cape Town on the eastern slopes of Table Mountain. The gardens were established in 1913, now cover 530 ha of which 478 ha are natural forest and fynbos, 36 ha are cultivated and 16 ha are utilized for buildings and service facilities. Kirstenbosch is zoned into different floral areas devoted to indigenous plants covering southern Africa, of which nearly 6 000 species grow in the cultivated area of the garden. The Cape Flora is represented by 900 plant species in the natural areas (Paterson-Jones 1993). A diversity of fynbos and forest birds totaling 112 species occur in the garden which include a number of Cape endemics such as Cape Sugarbird *Promerops cafer,* Orangebreasted Sunbird *Nectarinia violacea,* and Cape Francolin *Francolinus adspersus* (Cohen & Winter 1995).

The mountain drains numerous natural perennial streams (River Health Programme 2005) into the upper Liesbeck River, mostly through the Kirstenbosch Garden. Water conditions varying from cool clear, swiftly-flowing mountain streams with excellent to good water quality are ideal habitat for *Simulium hessei* and the widespread *loutetense* group of the subgenus *Nevermannia*, especially *S. loutetense, S. narcaeum* and *S. rutherfoordi* in slower-flowing stretches (Palmer & de Moor 1998, de Moor 2002). *S. dentulosum* prefers waterfalls and cascades on

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mountain slopes with good to excellent water quality (Palmer & de Moor 1998). *S. merops* is common in Kirstenbosch Garden, occurring in forested foothill streams, including black-water streams which are cool and acidic (Palmer & de Moor 1998).

Conditions for biting midges in Kirstenbosch Gardens are also ideal, due to the numerous semi-aquatic habitats, comprising damp places, decomposing plant material, mud or wet sand along margins of ponds including the (irrigation dam at the top gate), streams, rock pools and slow-flowing streams with vegetation (de Meillon & Wirth 2003).

Climate

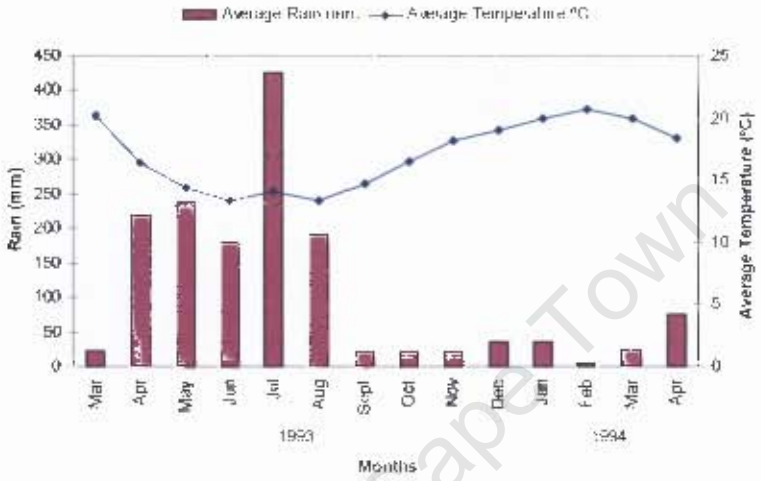

Figure 3.6 Average daily ambient temperature and monthly rainfall recorded at Kirstenbosch National Botanical Gardens, from March 1993 to April 1994. (South African Weather Service)

Vegetation

Ringing activity was carried out near the irrigation dam situated in the fynbos area at the south end of the gardens, near Rycroft Gate. Vegetation in this area is extremely diverse reflecting the richness of the Fynbos Biome. Protea species, which dominate, consist of *Protea rubropilosa*, *P. repens*, *P. neriifolia* and *P. aristata*, with many *Leucospermum* species. Ericas are represented by over 600 southern African species, of which *Erica regia* is the most notable, while *Elegia capensis* is the most prominent of the restio species. The aquatic vegetation is dominated by *Scirpus nodosus*, *Typhus capensis*, *Juncus kraussii*, with *Paspalum vaginatum* as ground cover in the open areas.

7. MOWBRAY

Site description

The study site is an erf located in a transformed urban setting of Mowbray (33°56'S 18°28'E), a residential suburb of Cape Town, 5.6 kilometers south of the city center beyond Observatory.

The middle Liesbeek River flows through densely populated urban areas including Mowbray. Forty percent of the river's length has been canalized resulting in poor water quality from wastewater discharges, stormwater runoff and litter disposal (River Health Programme 2005). Water conditions of this nature create habitats for biting midges in damp sand, wet places, mud, rock pools along margins of ponds,

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slow-flowing water and algal mats for the tribe Ceratopogonini of biting midges (de Meillon & Wirth 1991). Polluted water runoff and slow-running water from urban areas creates habitats for *Simulium nigritarse*, particularly trailing vegetation. Fuller (1899, cited by Palmer & de Moor 1998) reported unknown simuliids near Wynberg, which were pests to poultry. Palmer and de Moor (1998) identified the species as *S. nigritarse* from an illustration which accompanied the report at the time. Species which also tolerate polluted conditions and which have been recorded in the Western Cape include *S. adersi* and *S. ruficorne*, while *S. nigritarse* and *S. medusaeforme* commonly occur at stormwater outlets (Palmer & de Moor 1998). *S. ruficorne* also occurs in industrial polluted waters (Palmer 1991).

Climate

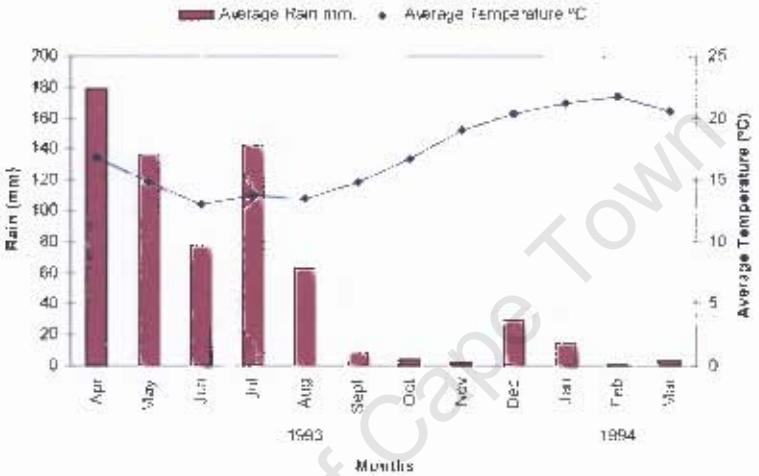

Figure 3.7 Average daily ambient temperature and monthly rainfall recorded at Cape Town Weather Office weather station, situated at Cape Town International Airport (33°58'S 18°36'E), 11km east of Mowbray, from April 1993 to March 1994 (South African Weather Service).

Vegetation

Typical urban and suburban gardens have been transformed and modified to be dominated by alien invasive herbaceous and exotic plants (nasturtium, kikuyu and wild ginger) with alien *Pinus radiata* and *P. pinaster* dominant in public spaces and parks.

8. PATRYSKRAAL

Site description

Patryskraal (34°26'S 20°11'E) is a farm situated on the low-lying, rolling Agulhas Plain of the Overberg wheatbelt, stretching from Caledon to Riversdale in the north, and between the two coastal towns of Hermanus and Stilbaai in the south. The farm consists primarily of cultivated wheat fields.

Numerous streams drain the Agulhas Plain and Overberg region, with their sources in the Riviersonderend and Langeberg Mountains. Many of these streams supply the area with run-off water; thus the numerous earth-dams scattered across the region. Soutpansvlei, 2 km from the ringing site, is a dynamic system which is transformed during high winter rainfall, when the pan is flooded by muddy fresh

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water. As the dry season commences in summer the salinity increases with the receding water level.

The widespread *Simulium ruficorne* occurs in the drier regions of the world including oases of the Sahara Desert, Middle East and Iberian Peninsula (Crosskey & Büttiker 1982), also recorded in the Western Cape, can tolerate high water temperatures. It is particularly adapted to small, temporary, mineralized streams, agricultural seepage waters and stagnant pools (Palmer & de Moor 1998), conditions which exist in the Overberg wheatbelt, especially during summer. Widespread species which have been recorded in the area, occurring in a diversity of conditions, include *S. adersi*, *S. medusaeforme* and *S. nigritarse*.

Biting midges including mosquitoes are found in nearly every aquatic or semi-aquatic habitat in every region of the world (de Meillon & Wirth 2002, Coetzee 2002). Water conditions in the area comprising earth and cement-dams, reservoirs, slow-moving streams and artificial water containers; all create habitats for biting midges and mosquitoes.

Climate

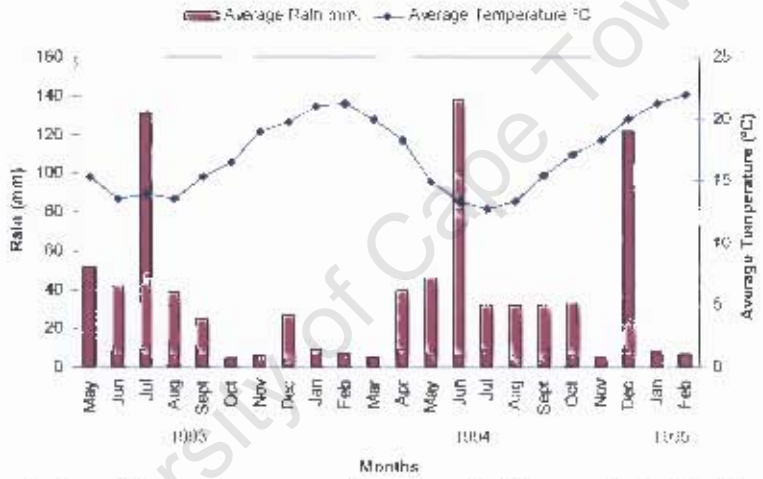

Figure 3.8 Average daily ambient temperature and monthly rainfall, recorded at Struisbaai (33°43'S 20°07'E), 80km south of Patryskraal, from May 1993 to February 1995. Although Patryskraal is situated inland from the coast, rainfall isohyets extend well inland showing similar rainfall patterns over Patryskraal (South African Weather Service)

Vegetation

The Overberg wheatbelt consists of cereal and cultivated wheat croplands, with marginal vegetation remaining along the coast and fragmented Renosterveld patches, inland. Milkwood *Sideroxylon inerme* dominates thicket along the coast, and forest fragments are characterized by consisting mostly of *Elytropappus rhinocerotis*, *Celtis africana*, *Olinia ventosa*, *Apodytes dimidiata*, *Rhus* spp. and *Euclea racemosa*.

The region has a number of fynbos communities. *Leucadendron elimense*, *L. modestum*, *L. laxum*, *Phylica ericoides* and *Disparago anomala* dominate Asteraceous fynbos with rich endemism. *Calopsis fruticosus* and *Ficinia lateralis*, with ericoid shrubs such as *Passerina paleacea* are dominant in dune Asteraceous fynbos along the coast. Widespread along the Agulhas Plain are three proteoid fynbos communities, which are dominated by *Protea refens*, *P. obtusifolia*, *P. susannae*, *P. compacta*, *Leucadendron meridianum* and *L. xanthoconus* (Acocks 1988, Low & Rebelo, 1996 Cowling *et al.* 1997). Shrubs and exotic plants surround farm buildings, with many patches of *Eucalyptus camaldulensis* dominating the landscape of the ringing site.

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9. RONDEVLEI NATURE RESERVE

Site description

Rondevlei Nature Reserve (34°04'S 18°30'E) is a shallow coastal lake situated at the southwestern corner of the Cape Flats next to Zeekoevlei, 24 km south of Cape Town and 5 km from False Bay. Southeasterly winds originally covered the area with wind blown sand from the False Bay coastline, cutting off the western end of a much larger Zeekoevlei in the 1800s, according to early maps (Berruti 1989), resulting in the vlei laying between two low sand dunes. With the planting of *Acacia saligna* and *A. cyclops* from Australia in the late 1800s to stabilize the dunes, the link between Zeekoevlei and Rondevlei was finally severed. Rondevlei forms part of an a wetland complex, which includes Zeekoevlei, Princess Vlei, and the Strandfontein Sewerage Works. The whole complex, covers an area of 217 ha with Rondevlei being 105 ha in size, of which 50 ha consists of open water when the vlei is full (Berruti 1989, Gibbs 1995).

Rapid urbanization of the surrounding land since 1952 resulted in canal building to drain stormwater runoff into the vlei. This resulted in pollution from the densely populated sub-economic housing schemes surrounding the vlei. In 1958, as a flood prevention measure, a weir was constructed at the southeast end of the vlei. The construction of the weir has resulted in stabilizing the water level within the vlei and reducing high fluctuations after heavy rainfalls (Berruti 1989). Before the weir was constructed, the water level ranged between 4.6—5.2 m above mean sea level (MSL). Since construction, the maximum level has been maintained at 4.9 m above MSL, fluctuating between this high and an average summer low of 4.1 m above MSL (Berruti 1989). After construction the slight drop in water level exposing a large seasonal shoreline was soon colonized by plants such as sedge, bulrush and vleigrass. This resulted in a large reduction in wading birds as the shoreline became less suitable for foraging. When hippos were returned in 1981 after 300 year absence (Wheeler 1992), they successfully eradicated two invasive species of vegetation, vlei grass *Paspalum vaginatum* and bulrush *Typha latifolia*, also opening up channels and improving access for birds (Berruti 1989). When levels do subside during dry summer spells (D. Gibbs 2007 pers. comm.) the exposed sandy and muddy banks below the reedbeds attract Little Stint *Calidris minuta,* Curlew Sandpiper *Calidris ferruginea,* Wood Sandpiper *Tringa glareola,* and Ruff *Philomachus pugnax* (Wheeler 1992, Gibbs 1995, Cohen & Spottiswoode 2000, Cohen *et al.* 2006, D. Gibbs 2007 pers. comm.). There are a number of islands and large reedbeds in the vlei, which are used for nesting and roosting. Of the 218 bird species recorded in the reserve, 84 are resident species (Wheeler 1992).

Rondevlei nearly surrounded by human settlement forms an oasis for the near depleted Cape Flats fauna and flora. The increasing human population on the Cape Flats presents a threat to the survival of Rondevlei, especially since a large portion of the remaining dune area to the south of the vlei was nearly lost to development for the Lavender Hill Extension. Only through intervention by environmentally concerned Capetonians was development averted (Berruti 1989). Although the reserve is small, many avian species find sanctuary within the reserve for breeding while foraging further afield. As the remaining natural open spaces on the Cape Flats are lost to development, it is possible that Rondevlei will become an important breeding area and habitat for many avian species. Competition with nature for living space and the associated problems of pollution and invasive of alien plants are pervasive, that only through extraordinary human input can the nature reserve survive.

Water in the shallow saucer-shaped vlei starts receding from October to an average summer low of 4.1 m above MSL in April, attaining a of low of 3.6 m MSL during

exceptionally dry spells (Berutti 1989, D. Gibbs 2007 pers. comm.). The exposed shoreline of mud and sand during these months, including algal mats, wet decomposing plant material and the vlei itself, create conditions for breeding biting midges and mosquitoes. The predominately southeasterly wind blowing during summer at an average wind speed of 23.9 ms^{-1} off the False Bay coast might impact on mosquito population densities. Active mosquito numbers are reduced by 80% in wind speeds >2.3 ms^{-1} (Service 1995). *Simulium medusaeforme* and *S. nigritarse* are two species of blackfly likely to be found at Rondevlei. *S. medusaeforme* occurs in a wide range of conditions, particularly artificial waterways and near stormwater outlets, including polluted water, and on a thick moss carpet at Buffelsjags Dam spillway (Palmer & de Moor 1998). The most common and widespread blackfly in southern Africa, *S. nigritarse* is usually found in slow-flowing water under rocks and trailing vegetation, and can tolerate polluted water.

Climate

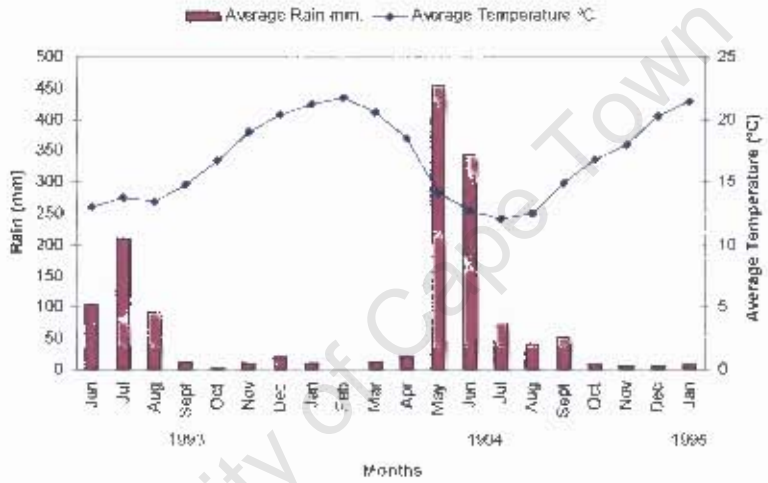

Figure 3.9 Average daily ambient temperature recorded at Cape Town Weather Office, situated at Cape Town International Airport (33°58'S 18°36'E), while monthly rainfall was recorded at Rondevlei, from January 1993 to January 1995. (South African Weather Service)

Vegetation

The land surrounding the vlei has been invaded by acacia thickets on the northern and eastern sides consisting mainly of *Acacia cyclops* and *A. saligna*. Although reserve staff have eradicted much of the invading alien vegetation (Ashwell & Allen 2001, Ashwell & Younge 2002), the threat remains. The southern sections consist of indigenous dune, marsh vegetation and reedbeds, with semi-natural habitats, deep and shallow open water, seasonal open ponds and canals. The perennial wetland is characterised by aquatic vegetation dominated by *Typha capensis*, *Phragmities* spp. *Potamogeton pectinatus, Lemma gibba, Myriophyllum aquaticum* and *Nasturtium* spp., especially in the water canals. *Typha capensis* and *Phragmites* spp. dominate the reedbeds. The reed marsh consists of *Phragmites australis*, invaded in parts by *Typha capensis*. The sedge marsh is diverse, and is dominated by *Bolboschoenus maritimus* and *Juncus kraussii* (Barnes 1998).

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10. TYGERBERG NATURE RESERVE

Site description

The Tygerberg Nature Reserve (33º52'S 18º36'E) is part of the Tygerberg Hills which form a complex landscape, consisting of a range of hills running north to south. The topography changes from being hilly in the south to less indented rolling hills at the northern and western boundaries of the Tygerberg. These range in height from 230 m to 460 m above sea level at Kanonkop, with steep western and gentle eastern slopes. Tygerberg Hill is particularly noticeable, with its southern tip bordering the low-laying Cape Flats. The first section of 76 ha of Strandveld was proclaimed as a Local Authority Nature Reserve in 1973. In 1996 additional areas were included to cover 278 ha in extent. The nature reserve is situated north of the N1 national highway and south of the cultivated farmland to the north, and between the suburbs of Parow to the west and Durbanville to the east.

Bird species totalling 158 have been recorded on the last small remnants of the West Coast Renosterveld (Wood & Low 1993). Three Red Data species occur in the reserve although only as vagrants: White Pelican *Pelecanus onocrotalus*, Peregrine Falcon *Falco peregrinus* and Caspian Tern *Hydroprogne caspia*. Sixteen of the bird species found in the study area are endemic to southern Africa, eight to South Africa and three species are restricted to the Western Cape: Cape Sugarbird *Promerops cafer*, Orangebreasted Sunbird *Nectarinia violacea* and Protea Canary *Serinus leucopterus* (Wood & Low 1993). Two valleys extend down and around a projection of suburbia, with the northern valley being the most prolific for birds on the reserve. This valley also proved ideal for bird ringing because of a small reed-fringed dam, which accounts for a number of species that would not otherwise be present. These aquatic related species are Little Bittern *Ixobrychus minutus,* Common Moorhen *Gallinula chloropus,* Giant Kingfisher *Megaceryle maximus,* Malachite Kingfisher *Alcedo cristata,* Lesser Swamp Warbler *Acrocephalus gracilirostris* and a number of Anatidae species.

Tygerberg Hills act as a catchment area for four rivers. The Elsieskraal and Kuils rivers drain the east and south slopes. The Mosselbank River drains the eastern section of the Tygerberg hills towards Durbanville, running northwards to Klipheuwel, changing direction west, then again southwards at Philadelphia to become the Diep River, draining the western slopes.

The hills north of Tygerberg (Kanonkop, Hoogekraal and Humeklip) have various seasonal streams, which drain north into the Diep River, being the only perennial watercourse. Several man-made dams built along the Elsieskraal River provide storm water control and recreation opportunities. The existence of numerous water bodies including the disused stone quarry on the southern slope of the Tygerberg Reserve, provide numerous oviposition sites for breeding mosquitoes. Mosquito population densities could be found on the wind protected east side of the reserve during winter due to northwesterlies, but could shift to the protected west side of the reserve in summer as a result of southerly to reasonably strong southeasterly winds. Blackfly species which could be found on the reserve include *Simulium dentulosum* occurring in the southwestern Cape near sea level while *S. merops,* is found in temporary and foothill streams, having good to excellent water-quality. *Simulium rutherfoordi, S. nigritarse* and *S. alcocki* occurs in slow-flowing foothill streams while *S. vorax* occurs in fast-flowing streams, and would probably be encountered during winter months.

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Climate

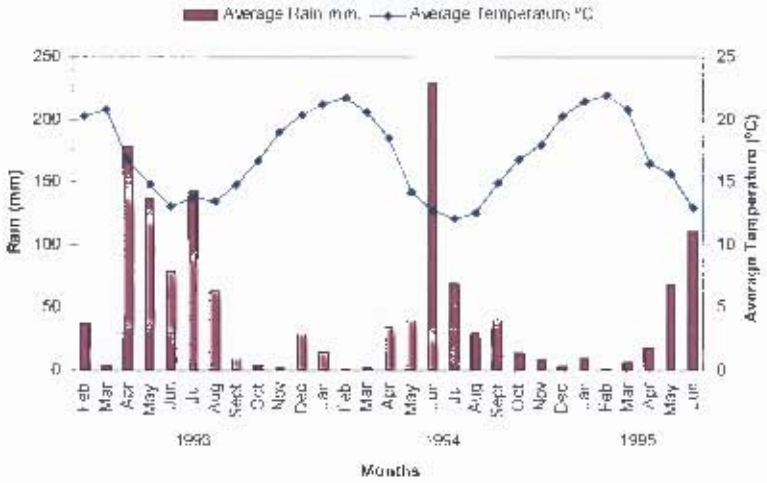

Figure 3.10 Average daily ambient temperature and monthly rainfall recorded at the Cape Town Weather Office, at Cape Town International Airport (33°58'S 18°36'E), 11km south of Tygerberg Nature Reserve, from February 1993 to June 1995. (South African Weather Service)

Vegetation

The proclamation of the reserve has secured the preservation of threatened West Coast Renosterveld, which occurs on nutrient rich soil. Only 3% of the original area remains (Wood & Low 1998), as original vegetation that occurred beyond the reserve boundaries has been transformed into suburbia, wheat fields and vineyards. Vegetation is characterized by mid-dense to closed cupressoid and small-leaf, mid-high evergreen, regular clumps of broad-leaved shrubs. Dominant plant species are *Elytropappus rhinocerotis*, *Eriocephalus africanus*, *Anthospermum aethiopicum*, *Athanasia trifurcata*, *Felicia filifolia*, *Metalasia muricata* and *Stoebe spiralis*. Grass cover is dominated by the genera *Ehrharta*, *Pentaschistis*, *Merxmuellera*, *Tribolium* and *Eragrostis*. Bush clumps are dominated by *Olea europaea*, *Rhus laevigata* and *Euclea racemosa* (Cowling *et al.* 1997).

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CHAPTER FOUR

Seasonal and regional distribution between sites within the Greater Cape Town area of avian haemosporidian parasites and their taxonomic status

...that being rare gives an organism an advantage, because parasites are more adapted to the more common hosts.
Carl Zimmer, *Parasite Rex*

INTRODUCTION

Zoogeographical and seasonal prevalence of avian haemosporidian parasites lacks adequate study throughout the six zoogeographical regions of the world, with a particular lack of published papers on subregions and smaller subordinate regions within these zoogeographical regions (Valkiūnas 2005). The seasonality of transmission of infection by vectors is dependent upon suitable environmental conditions, consisting of favourable temperature, humidity and successful completion of the haemosporidian's life cycle in both vertebrate and invertebrate hosts. At high latitudes, the short warm season is the active time for transmission (Bennett & Cameron 1974, Baker 1975, Herman & Bennett 1976, Atkinson *et al.* 1988). At low latitudes, the humidity that follows the rainy season is the ideal period for transmission (Bennett & Warren 1966, Greiner & Mundy 1979, Young *et al.* 1993), especially in countries with warm climates where prevalence of infection is recorded in the range 90—100% during this season (Markus & Oosthuizen 1972, Ayala *et al.* 1977). Transmission of *Haemoproteus meleagridis* occurs throughout the year in those parts of the tropics and subtropics with a mild humid climate (Atkinson 1988, Atkinson *et al.* 1988). For example there is transmission throughout the year of avian plasmodia in Venezuela (Gabaldon & Ulloa 1980) and of *Leucocytozoon smithi* in regions of South Carolina (Noblet *et al.* 1975).

The zoogeographical distribution of haemosporidian parasites is worldwide, from mountain ranges 3 000 m above sea level in Hawaii to *Leucocytozoon* species being transmitted far beyond the North Polar Circle (Valkiūnas 2006), but absent from the Antarctic (Valkiūnas 2005). The Holarctic contains the greatest number of species (1 260), while the Australian region is the poorest with 22 species. This may be ascribed to the relatively low number of avian haemosporidia occurring in that particular region, or due to the relative low level of research undertaken. This contrasts significantly with the Palearctic and Nearctic which have been well studied with a combined total of 123 species.

Haemosporidia are widespread in the tropics and subtropics where they parasitize birds, amphibians, reptiles and mammals. In central and northern Palearctic Region, they are absent from all groups of vertebrate animals with the exception of birds and a few species of bats (Valkiūnas 2006). The study of avian haemosporidian parasite distribution in certain zoogeographical regions has been recorded despite certain impeding factors. First, infrequent research is undertaken leading to a lack of adequate data covering avian hosts and vectors (Bennett *et al.* 1992a). Second, there is a lack of adequate distribution studies in certain orders and families of birds (Valkiūnas 2005). Third, there is inadequate study of migrating birds

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transporting parasites to other regions of the world (Hoogstraal *et al.* 1961, Valkiūnas 2005). Fourth, there are haemosporidians which have only been recorded once in certain avian species and which require review (Peirce 2005). Fifth, anthropogenic activity which has changed distribution ranges of birds, with possible corresponding impact on changing avian haemosporidia and vectors, needs constant study. Nevertheless catalogues of avian haemosporidian prevalence have been complied for the various regions. The Nearctic was complied by Greiner *et al.* (1975), the Neotropical by White *et al.* (1978), the western Palearctic by Peirce (1981), the Afrotropical (sub-Saharan Africa) by Bennett *et al.* (1992a) and the Oriental by McClure *et al.* (1978). The Afrotropical, Oriental and Holarctic regions contain the majority of avian haemosporidian species, with all known current *Leucocytozoon* species occurring in these three zoogeographical regions. The Holarctic has the highest overall infection rates of *Leucocytozoon* and *Haemoproteus* species, while the highest overall prevalence of *Plasmodium* infections occurs in the Afrotropical region (Valkiūnas 2005).

Although microscopical examinations of blood smears underestimate the prevalence of haemosporidian infections, it remains the most reliable method to quantify the diversity of haemosporidia present in a particular host species (Valkiūnas *et al.* 2006), as encountered in this study. The possibilities of researching avian haemosporidia by means of molecular genetics are inexhaustible, but at present remain in its infancy due to lack of funding for zoological research in systematics. The PCR procedure underestimates simultaneous infections in a host, as specific primers still need development. Thus to study the ecology and evolutionary biology of avian haemosporidian parasites requires the combined resources of microscopy and PCR-based methods (Valkiūnas *et al.* 2006).

A taxonomic re-appraisal of *Leucocytozoon* and *Haemoproteus* species in this study has been undertaken, following the recent review of avian haemosporidia by (Valkiūnas 2005) and subsequently by Peirce (2005). Accepted current taxonomy was put forward by Bennett *et al.* (1972, 1991a, 1991b) at a time when early researchers were naming species according to the host's identity. This resulted in a proliferation of invalid names and much confusion (Valkiūnas & Ashford 2002). Bennett *et al.* (1972, 1991a, 1991b) and Bennett & Peirce (1988) believed that avian haemosporidia were family and subfamily specific from the morphology of avian haemosporidia in blood smears. They proposed that morphologically indistinguishable *Leucocytozoon* and *Haemoproteus* species should be described and named as separate species when occurring in hosts of different avian families or subfamilies (Bennett & Peirce 1988, Bennett *et al.* 1994, Peirce 2005). Although aware of the implications of such a concept (Bennett & Campbell 1975), it did bring about order were none existed (Valkiūnas & Ashford 2002), but also resulted in the "one host-one parasite" paradigm (Bennett & Laird 1973, Bennett *et al.* 1994, Valkiūnas 2005).

The "one host-one parasite" approach to the systematics of the genera *Leucocytozoon* and *Haemoproteus* became the perceived general rule among researchers (Bennett *et al.* 1972, 1986, 1991b, 1992a, 1993, Bennett 1989, Bishop & Bennett 1990, Bennett & Peirce 1991, 1992, Bennett & Squires-Parsons 1992, Burry-Caines & Bennett 1992, Adlard *et al.* 2002). This despite being contrary to the fundamental principals of valid taxonomy, as the natural host range or geographical distribution of a parasite, at species or family level is not a valid taxonomic characteristic as applied by Adlard *et al.* (2004), nor is it the sole reflection of genotype. Thus the fundamental principal in taxonomy must apply, of the "single-species null hypothesis"—organisms belong to a single species unless proved different. Now that PCR techniques are available to examine DNA of avian haemosporidia in blood smears (Ribeiro *et al.* 2005), Bennett's device should be

discontinued as it has served its purpose. Thus the current checklist of avian species of *Babesia, Haemoproteus, Hepatozoon,* and *Leucocytozoon* (Peirce 2005), which contains species morphologically similar but described as new species are invalid and should become junior synonyms of earlier described similar species. This would be a first step in bringing about the modern application of systematics for avian haemosporidian taxonomy which has been lacking at species level.

Beside implementing present avian haemosporidian parasite taxonomy in this study as reviewed by Valkiūnas (2005) and rejected by Peirce (2005), it also examines over a three year period the spatial and seasonal distribution of avian haemosporidian parasites within the winter rainfall region of South Africa, situated in the Afrotropical region. The results are contrasted with those obtained from northern South Africa by Earlé *et al.* (1991).

MATERIALS AND METHODS

Birds were trapped in mist-nets at 10 study sites in the greater Cape Town area: Bettys Bay (34°22'S 18°56'E), Durbanville Nature Reserve (33°50'S 18°38'E), Goedeontmoeting (33°41'S 18°63'E), Glencairn (34°09'S 18°25'E), Koeberg Nature Reserve (33°40'S 18°26'E), Kirstenbosch National Botanical Gardens (33°58'S 18°26'E), Mowbray (33°56'S 18°28'E), Patryskraal (34°26'S 20°11'E), Rondevlei (34º04'S 18º30'E), and Tygerberg Nature Reserve (33°52'S 18°36'E), from February 1993 to September 1995. For a description of the study sites, see Chapter 3.

Birds were ringed, measured, and mass taken in accordance with recommendations for standard practice by SAFRING (de Beer *et al.* 2000). Wing length was measured to the nearest 1.0 mm using maximum chord method (flattened, straightened wing). Body mass was measured to the nearest 0.5 gram using spring balances. Sex and age of each bird were determined when possible.

Blood was obtained from birds by venipuncture to the brachial vein in the wing to obtain blood to spread over a microscope slide for a thin blood smear which was subsequently air-dried. Blood smears were fixed with absolute methanol or May Grünwald's Giemsa and stained with 4% Giemsa solution for 60 minutes. Blood smears were examined under light microscopy at 1000X magnification using oil immersion. Avian haemosporidian parasites were identified to species level. Intensity of infections was not assessed.

For each species and site the number of blood smears examined and the number of those with avian haemosporidian parasites was determined. The appropriate statistical model for these data is the binomial distribution, and therefore modelled the occurrence of parasites in relation to explanatory variables using a generalized linear model with a binomial distribution and logistic link function (McCullagh & Nelder 1989, Crawley 1993, Underhill & Kalejta-Summers 1995). Two explanatory variables, site and genus were used. Site was fitted to explore whether there were statistically significant differences in the prevalences of avian haemosporidian parasites infection between sites. However, the species composition between sites varied considerably, and the data at some sites consisted of large proportions of species known to have either low or high proportions of avian haemosporidia parasites. In order to remove the effect of this variable, genus was used as a second explanatory variable. The models were fitted using GENSTAT 8 (Genstat 8 Committee 2005), with site and genus as "factor" variables, so that the effect of each site was estimated separately. Models were fitted for all avian haemosporidian parasites and for Haemoproteidae and Leucocytozoidae infections separately. The Akaike Information Criterion (AIC) was used to assist model

selection. A model for Plasmodiidae infections was not fitted because of the low rate of infection (see Results).

RESULTS

In total, 9 304 birds of 77 species representing 32 avian families were examined for avian haemosporidian parasites (Table 4.1, Appendices 1—10). Avian haemosporidia parasites were found in 1 980 birds of 46 species from 22 avian families. For 29 species, less than 10 birds were trapped and examined. The *Leucocytozoon* species occurred in 61.8% of infected birds, followed by *Haemoproteus* species in 45.2%, and *Plasmodium* species in 3.2%. The double infection rate in Brimstone Canary *Crithagra sulphuratus* was 20.0%, followed by Cape White-eye *Zosterops virens* with 16.8%, Cape Turtle Dove *Streptopelia capicola* with 16.6% and Southern Boubou *Laniarius ferrugineus* with 12.5%. Species that had the greatest double infection rates also recorded the greatest haemosporidian parasite prevalence: *Streptopelia capicola* (43.9%), *Zosterops virens* (40.8%), *Laniarius ferrugineus* (38.0%). Conversely only 0.9% of Common Waxbill *Estrilda astrild*, 1.1% of Southern Double-collared Sunbird *Cinnyris chalybeus* and 2.2% of Cape Robin Chat *Cossypha caffra* were infected. Surprisingly, none of the 66 Levaillant's Cisticola *Cisticola tinniens* and 37 Little Rush Warbler *Bradypterus baboecala* was infected. The most trapped species Cape Weaver *Ploceus capensis* (n=2 086) was predominantly infected with *Haemoproteus queleae* and *Leucocytozoon bouffardi* and Cape White-eye *Zosterops virens* (n=1 597) with *H. killangoi, H. zosterops* and *L. zosteropis.*

Avian haemosporidian parasites from three genera were found in 22 avian families, consisting of 41 species (Table 4.2). *Leucocytozoon* species was the most prevalent occurring in 22 avian species, followed by *Haemoproteus* in 12 species and *Plasmodium* in seven species. Avian species (n=236) consisting of the family Columbidae which formed the largest part of the sample size at Mowbray were infected predominately by *Haemoproteus columbae*. The bird families Ploceidae and Sylviidae were infected by all three avian haemosporidian genera. Ploceidae was the most heavily infected with nine species. Motacillidae and Ploceidae were the only avian families to be infected by four species of *Plasmodium*, while Motacillidae was infected solely by the genus *Plasmodium* and by no other avian haemosporidia species (Table 4.2). Sylviidae and Muscicapidae were infected with two species of *Plasmodium* while the remaining Plasmodiidae infected avian families were infected with one species of *Plasmodium* each. The most prevalent Plasmodiidae species infection was *Plasmodium relictum*, occurring in six avian families: Turdidae, Sylviidae, Motacillidae, Promeropidae, Ploceidae and Fringillidae. Plasmodiidae which is regularly recorded to cross infect host avian orders and families (Garnham 1966, Beier & Stoskopf 1980, Waldenström *et al.* 2002), only infected the avian order Passeriformes as encountered in this study.

Based on the actual observations, rates of infections at the 10 study sites varied greatly, from 4.5% at Patryskraal to 28.0% at Tygerberg Nature Reserve (Table 4.3). The generalized linear modelling took into account the variability in species composition between sites. This was typified at Mowbray, were 87.7% of birds examined were Columbidae (Appendix 7), which on average had an infection rate of 33.6%, considerably higher than the overall average infection rate of 21.2% (Table 4.1). Thus the observed infection of 14.8% rate at Mowbray (Table 4.3) is biased upwards by the predominance of Columbidae at this site.

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Table 4.2 Species of avian haemosporidian parasites found in birds from the 10 study sites within the greater Cape Town area, South Africa.

Avian Order	Avian Family	Species of haemosporidian parasites
Piciformes	Lybiidae	*Leucocytozoon capitonis* [1], *L. squamatus*.
Upupiformes	Upupidae	*Leucocytozoon communis*.
Coliiformes	Coliidae	*Leucocytozoon colius*.
Cuculiformes	Centropodidae	*Leucocytozoon centropi*
Columbiformes	Columbidae	*Haemoproteus columbae*, *Leucocytozoon marchouxi*.
Passeriformes	Malaconotidae	*Haemoproteus cubiae*, *Leucocytozoon balmorali*
	Laniidae	*Haemoproteus lanai*.
	Hirundinidae	*Leucocytozoon whitworthi* [2].
	Pycnonotidae	*Haemoproteus otocompsae*, *H. sanguinis*, *Leucocytozoon bouffardi* [3], *L. brimonti* [4], *L. pycnonoti* [5].
	Sylviidae	*Haemoproteus sylvae* [6], *Leucocytozoon phylloscopus* [7], *Plasmodium vaughani*, *P. relictum*.
	Zosteropidae	*Haemoproteus killangoi*, *H. Zosterops*, *Leucocytozoon zosteropis* [8], *Plasmodium rouxi*
	Cisticolidae	*Haemoproteus sylvae*, *Leucocytozoon phylloscopus*.
	Muscicapidae	*Leucocytozoon shaartusicum* [9], *L. dubreuili*, *Plasmodium relictum*, *P. vaughani*.
	Sturnidae	*Haemoproteus pastors*, *Leucocytozoon stumi* [10].
	Nectariniidae	*Haemoproteus sequeirae*, *Leucocytozoon nectariniae* [11].
	Promeropidae	*Leucocytozoon deswardti* [12], *Plasmodium relictum*.
	Ploceidae	*Haemoproteus queleae*, *Leucocytozoon bouffardi* [3], *Plasmodium circumflexum*, *P. relictum*, *P. polare*, *P. vaughani*.
	Estrildidae	*Leucocytozoon roubaudi* [13], *Plasmodium* species.
	Viduidae	*Leucocytozoon* species.
	Passeridae	*Haemoproteus passeris*, *Leucocytozoon gentili* [14], *L. monardi* [15], *Plasmondium relictum*
	Motacillidae	*Plasmodium vaughani*, *P. subpraecox*, *P. cathemerium*, *P. relictum*.
	Fringillidae	*Haemoproteus chloris*, *Leucocytozoon dutoiti* [16], *Plasmodium relictum*.

Taxonomic revision according to Valkiūnas (2005): [1] *Leucocytozoon capitonis* is a synonym of *L. squamatus*; [2] *Leucocytozoon whitworthi*, [3] *L. bouffardi*, [4] *L. brimonti*, [10] *L. stumi*, [12] *L. deswardti*, [13] *L. roubaudi*, [14] *L. gentili*, [15] *L. monardi*, and [16] and *L. dutoiti* is a synonym of *L. fringillinarum*; [5] *L. pycnonoti*, [7] *L. phylloscopus* and [9] *L. shaartusicum*, is a synonym of *L. majoris*; [8] *L. zosteropis* and [11] *L. nectariniae* is a synonym of *Leucocytozoon dubreuili*. [6] *Haemoproteus sylvae* is a synonym of *H. belopolskyi*.

Table 4.3 Prevalence of avian haemosporidian parasites from 10 study sites within the greater Cape Town area, South Africa. Figures in parentheses represent percentages of birds infected.

Sites	Total birds		*Haemoproteus*		*Leucocytozoon*		*Plasmodium*	
	Exam.	Infected	Total	%	Total	%	Total	%
Bettys Bay [1]	438	98(22.3)	37	8.4(37.7)	81	18.4(82.6)	3	0.6(3.0)
Durbanville Nature Reserve [2]	1023	144(14.0)	48	4.6(33.3)	92	8.9(63.8)	9	0.8(6.2)
Goedeontmoeting [3]	2944	742(25.2)	527	17.9(71.0)	229	7.7(30.8)	41	1.3(5.5)
Glencairn [4]	405	68(16.7)	17	4.1(25.7)	48	8.9(63.8)	—	—
Koeberg Nature Reserve [5]	71	7(9.8)	4	5.6(57.1)	4	5.6(57.1)	1	1.4(14.2)
Kirstenbosch Botanical Gardens [6]	394	80(20.3)	30	7.6(37.5)	72	18.2(90.0)	3	0.7(3.7)
Mowbray	269	40(14.8)	32	11.8(80.0)	8	2.9(20.0)	—	—
Patryskraal [7]	218	10(4.5)	4	1.8(40.0)	6	2.7(60.0)	1	0.4(10.0)
Rondevlei Nature Reserve [8]	991	73(7.3)	12	1.2(16.4)	63	6.3(86.3)	1	0.1(1.3)
Tygerberg Nature Reserve [9]	2551	716(28.0)	183	7.1(25.5)	575	22.5(80.0)	6	0.2(0.8)
Total	**9 304**	**1 978(21.2)**	**894**	**9.6(45.1)**	**1178**	**12.6(59.5)**	**65**	**0.7(3.2)**

Multiple infections excluded from infected totals.
[1] double infections = 23; [2] double infections = 5; [3] double infections = 59; [4] double infections = 7; [5] double infections = 2; [6] double infections = 25; [7] double infections = 1; [8] double infections = 3; [9] double infections = 73.

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FAMILY *Species*	Total birds Examined	Total birds Infected	*Haem.*	*Leuco.*	*Plasm.*
SYLVIIDAE	**181**	**13(7.1)**	**7**	**3**	**3**
Sphenoeacus afer Cape Grassbird	17	1(5.8)	—	1	—
Sylvietta rufescens Long-billed Crombec	7	1(14.2)	—	1	—
Bradypterus baboecala Little Rush Warbler	37	0	—	—	—
Acrocephalus baeticatus African Reed Warbler	37	7(18.9)	7	—	—
Acrocephalus gracilirostris Lesser Swamp Warbler	79	2(2.5)	—	—	2
Parisoma subcaeruleum Chestnut-vented Tit Babbler	3	2(66.6)	—	1	1
Sylvia borin Garden Warbler	1	0	—		

Table 4.1 Summary of family and species prevalence data of avian haemosporidian parasites of birds trapped at 10 sites within the Greater Cape Town area between February 1993 and February 1995. *Haem* = *Haemoproteus; Leuco.* = *Leucocytozoon; Plasm.* = *Plasmodium.* Figures in parentheses represent percentages of birds infected. (Prevalence of infection for each species at each site are given in Appendices 1—10)

FAMILY *Species*	Total birds Examined	Total birds Infected	*Haem.*	*Leuco.*	*Plasm.*
PHASIANIDAE	**1**	**0**	**—**	**—**	**—**
Pternistis capensis Cape Spurfowl	1	0	—	—	—
INDICATORIDAE	**20**	**0**	**—**	**—**	**—**
Indicator minor Lesser Honeyguide	20	0	—	—	—
LYBIIDAE	**47**	**2(4.2)**	**—**	**2**	**—**
Tricholaema leucomelas Acacia Pied Barbet	44	2(4.5)	—	2	—
Lybius torquatus Black-collared Barbet	3	0	—	—	—
UPUPIDAE	**1**	**1(100.0)**	**—**	**1**	**—**
Upupa africana African Hoopoe	1	1(100.0)	—	1	—
ALCEDINIDAE	**5**	**—**	**—**	**—**	**—**
Alcedo cristata Malachite Kingfisher	5	—	—	—	—
DACELONIDAE	**1**	**—**	**—**	**—**	**—**
Megaceryle maximus Giant Kingfisher	1	—	—	—	—
COLIIDAE	**309**	**8(2.5)**	**—**	**8**	**—**
Colius colius White-backed Mousebird	169	5(2.9)	—	5	—
Colius striatus Speckled Mousebird	92	2(2.1)	—	2	—
Urocolius indicus Red-faced Mousebird	48	1(2.0)	—	1	—
CUCULIDAE	**10**	**0**	**—**	**—**	**—**
Chrysococcyx klaas Klaas's Cuckoo	10	0	—	—	—
CENTROPODIDAE	**2**	**1(50.0)**	**—**	**1**	**—**
Centropus superciliosus White-browed Coucal	2	1(50.0)	—	1	—
APODIDAE	**1**	**0**	**—**	**—**	**—**
Apus caffer White-rumped Swift	1	0	—	—	—
COLUMBIDAE	**539**	**172(31.9)**	**142**	**45**	**—**
Columba livia Rock Dove	8	4(50.0)	4	—	—
Columba guinea Speckled Pigeon	78	16(20.5)	16	—	—
Streptopelia senegalensis Laughing Dove [1]	394	133(33.7)	110	35	—
Streptopelia capicola Cape Turtle Dove [2]	41	18(43.9)	11	10	—
Streptopelia semitorquata Red-eyed Dove	17	1(5.8)	1	—	—
Oena capensis Namaqua Dove	1	0	—	—	—
RALLIDAE	**1**	**0**	**—**	**—**	**—**
Gallinula chloropus Common Moorhen	1	0	—	—	—
CHARADRIIDAE	**1**	**0**	**—**	**—**	**—**
Charadrius tricollaris Three-banded Plover	1	0	—	—	—
ARDEIDAE	**1**	**0**	**—**	**—**	**—**
Ixobrychus minutus Little Bittern	1	0	—	—	—
MONARCHIDAE	**4**	**0**	**—**	**—**	**—**
Terpsiphone viridis African Paradise Flycatcher	4	0	—	—	—
MALACONOTIDAE	**35**	**8(22.8)**	**4**	**4**	**1**
Laniarius ferrugineus Southern Boubou [3]	21	8(38.0)	4	4	1
Telophorus zeylonus Bokmakierie	7	0	—	—	—
Batis capensis Cape Batis	7	0	—	—	—
LANIIDAE	**39**	**1(2.5)**	**1**	**—**	**—**

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Coliiformes	Coliidae	*Leucocytozoon colius*.
Cuculiformes	Centropodidae	*Leucocytozoon centropi*
Columbiformes	Columbidae	*Haemoproteus columbae*, *Leucocytozoon marchouxi*.
Passeriformes	Malaconotidae	*Haemoproteus cublae*, *Leucocytozoon balmorali*.
	Laniidae	*Haemoproteus lanai*.
	Hirundinidae	*Leucocytozoon whitworthi* [2].
	Pycnonotidae	*Haemoproteus otocompsae*, *H. sanguinis*, *Leucocytozoon bouffardi* [3], *L. brimonti* [4], *L. pycnonoti* [5].
	Sylviidae	*Haemoproteus sylvae* [6], *Leucocytozoon phylloscopus* [7], *Plasmodium vaughani*, *P. relictum*.
	Zosteropidae	*Haemoproteus killangoi*, *H. Zosterops*, *Leucocytozoon zosteropis* [8], *Plasmodium rouxi*.
	Cisticolidae	*Haemoproteus sylvae*, *Leucocytozoon phylloscopus*.
	Muscicapidae	*Leucocytozoon shaartusicum* [9], *L. dubreuili*, *Plasmodium relictum*, *P. vaughani*.
	Sturnidae	*Haemoproteus pastoris*, *Leucocytozoon sturni* [10].
	Nectariniidae	*Haemoproteus sequeirae*, *Leucocytozoon nectariniae* [11].
	Promeropidae	*Leucocytozoon deswardti* [12], *Plasmodium relictum*.
	Ploceidae	*Haemoproteus greleae*, *Leucocytozoon bouffardi* [3], *Plasmodium circumflexum*, *P. relictum*, *P. polare*, *P. vaughani*.
	Estrildae	*Leucocytozoon roubaudi* [13], *Plasmodium* species.
	Viduidae	*Leucocytozoon* species.
	Passeridae	*Haemoproteus passeris*, *Leucocytozoon gentili* [14], *L. monardi* [15], *Plasmondium relictum*
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Goedeontmoeting [3]	2944	742(25.2)	527	17.9(71.0)	229	7.7(30.8)	41	1.3(5.5)
Glencairn [4]	405	68(16.7)	17	4.1(25.7)	48	8.9(63.8)	—	—
Koeberg Nature Reserve [5]	71	7(9.8)	4	5.6(57.1)	4	5.6(57.1)	1	1.4(14.2)
Kirstenbosch Botanical Gardens [6]	394	80(20.3)	30	7.6(37.5)	72	18.2(90.0)	3	0.7(3.7)
Mowbray	269	40(14.8)	32	11.8(80.0)	8	2.9(20.0)	—	—
Patryskraal [7]	218	10(4.5)	4	1.8(40.0)	6	2.7(60.0)	1	0.4(10.0)
Rondevlei Nature Reserve [8]	991	73(7.3)	12	1.2(16.4)	63	6.3(86.3)	1	0.1(1.3)
Tygerberg Nature Reserve [9]	2551	716(28.0)	183	7.1(25.5)	575	22.5(80.0)	6	0.2(0.8)
Total	**9 304**	**1 978(21.2)**	**894**	**9.6(45.1)**	**1178**	**12.6(59.5)**	**65**	**0.7(3.2)**

Multiple infections excluded from infected totals.
[1] double infections = 23; [2] double infections = 5; [3] double infections = 59; [4] double infections = 7; [5] double infections = 2; [6] double infections = 25; [7] double infections = 1; [8] double infections = 3; [9] double infections = 73.

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Table 4.4 Results of the generalized linear model, showing site factors for the prevalence of avian haemosporidian parasites at 10 sites in the Greater Cape Town area, South Africa. The site factors are on the logit scale, and are computed relative to Patryskraal as a baseline. The sites are ordered by estimated overall relative abundance of parasite infections, with the confounding effect of genus removed (see text).

Site	Overall	*Haemoproteus*	*Leucocytozoon*
Patryskraal	0	0	0
Rondevlei Nature Reserve	0.209	-0.577	0.638
Mowbray	0.320	0.453	0.026
Koeberg Nature Reserve	1.200	2.047	1.243
Durbanville Nature Reserve	1.477	1.087	1.622
Glencairn	1.919	1.219	2.376
Tygerberg Nature Reserve	2.046	1.226	2.414
Goedeontmoeting	2.250	2.644	1.547
Kirstenbosch National Botanical Garden	2.574	2.480	2.952
Bettys Bay	2.646	2.692	2.878

Table 4.5 Modelled proportions of overall infections with avian haemosporidian parasites for three example genera: *Colius, Acrocephalus and Zosterops,* at the study sites in the Greater Cape Town area, South Africa.

Site	*Colius*	*Acrocephalus*	*Zosterops*
Patryskraal	0.0035	0.026	0.108
Rondevlei Nature Reserve	0.0043	0.032	0.130
Mowbray	0.0048	0.035	0.143
Koeberg Nature Reserve	0.0116	0.081	0.287
Durbanville Nature Reserve	0.0152	0.104	0.347
Glencairn	0.0235	0.153	0.453
Tygerberg Nature Reserve	0.0266	0.170	0.484
Goedeontmoeting	0.0324	0.201	0.535
Kirstenbosch National Botanical Garden	0.0442	0.259	0.614
Bettys Bay	0.0474	0.272	0.631

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The generalized linear model for all infections with site and bird genus as explanatory variables accounted for 62.2% of the deviance, and provided a satisfactory fit to the data. Table 4.4 shows the estimates of infection rates for each site on the logit scale within the generalized linear model. Table 4.5 shows these logit-scale values transformed back into conventional proportions for three example genera: *Acrocephalus, Colius* and *Zosterops*. These are the modelled proportions of infections of each genus at each site. The values in Table 4.4 may be interpreted as standardized infection rates for each site with the effect of genus removed.

For all infections, further modelling showed that the 10 sites can be classified into four groups (Fig. 4.1). The AIC of the fitted model with 10 groups was 764. The lowest AIC, 761, was obtained when the sites were allocated to four groups, so that within each group, the infection rates were not significantly different from each other: Patryskraal, Rondevlei and Mowbray had low infection rates; Koeberg and Durbanville had intermediate infection rates; Glencairn, Tygerberg and Goedeontmoeting had high infection rates; and Kirstenbosch and Bettys Bay formed a group with very high infection rates.

From the perspectives of *Haemoproteus* and *Leucocytozoon*, the grouping of sites into those with similar infection rates differed from each other, and from the overall pattern (Fig. 4.1). For *Haemoproteus*, the AIC suggested that there were four groups of sites. The groups were the same as for the groups for all infections, except that Tygerberg and Glencairn moved from the high rate group to the intermediate rate group, Goedeontmoeting moved from the high rate group to the very high rate group,

and Koeberg moved from the intermediate rate group to become the only member of the high rate group. For *Leucocytozoon*, the AIC criterion suggested five groups of homogeneous sites; these groups were fairly different from those for *Haemoproteus*. The very low rate group consisted of only two sites, Patryskraal and Mowbray. There was a low rate group consisting of a single site, Rondevlei. The intermediate rate group consisted of Koeberg, Goedeontmoeting and Durbanville. The high and very high rate groups consisted of Tygerberg and Glencairn, and of Kirstenbosch and Bettys Bay, respectively.

The four study sites Bettys Bay, Goedeontmoeting, Kirstenbosch and Koeberg had sufficient data to enable month patterns to be displayed (Figure 4.2). At all sites the seasonal patterns for all infections (Figures 4.2—4.4) were significant; the strongest patterns were at Goedeontmoeting (χ^2_{11} = 103.4, P<0.001) and at Tygerberg (χ^2_{11} = 71.8, P<0.001), with somewhat weaker patterns at Durbanville (χ^2_{11} = 24.7, P=0.010), and at Glencairn (χ^2_{11} = 22.5, P=0.021). At all sites the tendency was for infection rates to be highest at some stage during the summer period (the spike in August at Durbanville was based on a small sample size (n=28) (Figure 4.2)). Those study sites which display a seasonal dominance of prevalence of infections by one avian haemosporidian species, display a corresponding subordination by the other species at the same site (Figures 4.3 & 4.4).

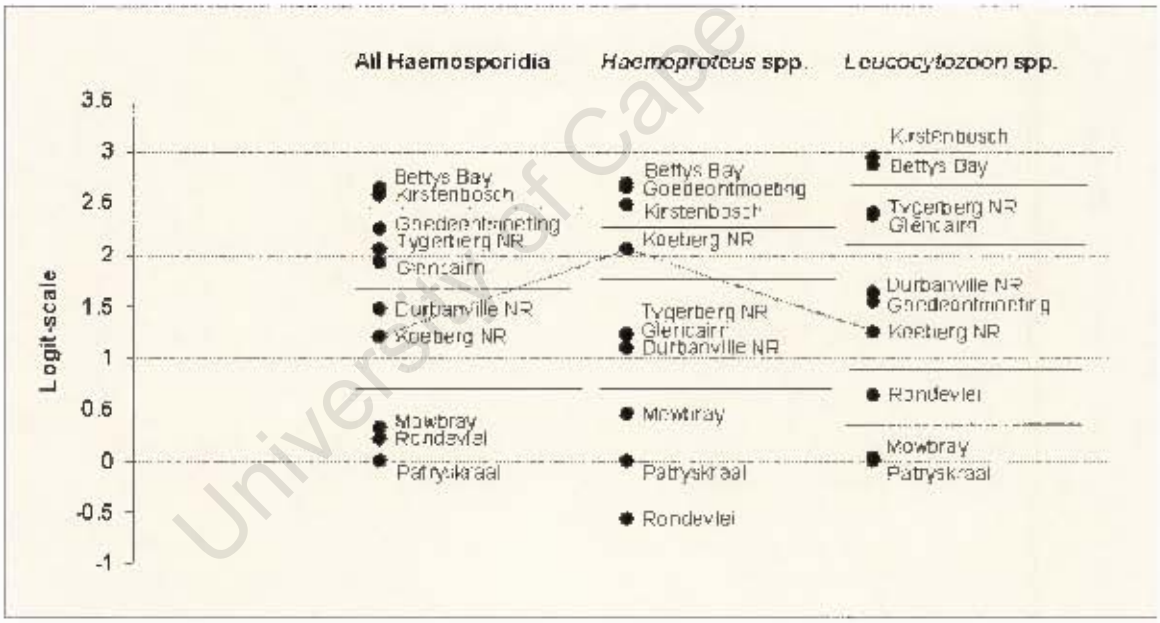

Figure 4.1 Relationship between sites of avian haemosporidian parasites from February 1993 to February 1995, using the logit scale index. The lines indicate groups of sites within which differences between infection rates are not statistically significant (see text).

DISCUSSION

Infection prevalence due to three families of avian haemosporidian parasites at each of the 10 study sites varied greatly (Table 4.3). Earlé *et al.* (1991) also obtained such varied results in northern South Africa. High prevalence of Haemoproteidae at Mowbray is biased towards the sample size of Columbidae, being their main hosts, of which Hippoboscid flies are the vector, spending much time on the birds (Klei & De Giusti 1975). A relatively warm climate is conducive to high transmission of these parasites (Markus & Oosthuizen 1972, Ayala *et al.* 1977). Overall Haemoproteidae infections occurred in 9.6% of birds examined (Table 4.1) which is similar to the

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figure of 11% found in northern South Africa (Earlé *et al.* 1991), but below that of the Afrotropical Region at 19.4% (Bennett *et al.* 1992a) and 50% for birds investigated world wide (Valkiūnas 2005). Prevalence of Plasmodiidae infections occurred in 0.7% of the sample, which concurs with low infections of 1.25% for northern South Africa (Earlé *et al.* 1991) and 3.5% from the Afrotropical Region (Bennett *et al.* 1992a). This low infection rate contrasts with the 30% in birds investigated worldwide (Valkiūnas 2005) and 59.6% in birds sampled from the Eastern Cape Province (Schultz & Whittington 2005). World climate change, brought about by global warming modifies habitat (Hill *et al.* 2001, Masters & Brown 2001, Parmesan 2001), creates climatic conditions favourable for initiating new foci for vectors, thus increasing transmission and prevalence (Tadei *et al.* 1998) as found in later studies.

The high prevalence of Leucocytozoidae infections in birds from Bettys Bay, Kirstenbosch National Botanical Gardens, Tygerberg Nature Reserve, and Glencairn results from species of Simuliidae (blackfly) adaptation to fast-flowing water. The exceptions to this are *Simulium ruficorne,* which has been recorded from small trickles of water, temporary, mineralized streams and agricultural seepage waters, and the widespread *S. adersi* from highly saline and brackish estuarine waters (de Moor 2003), which could account for the prevalence of Leucocytozoidae at Rondevlei Nature Reserve and Patryskraal. Female *S. ruficorneare* are ornithophilic (Freeman & de Meillon 1953) being pest to poultry and sparrows on Mauritius (Orian 1962). Adult female *S. adersi* are primarily ornithophilic (Palmer & de Moor 1998) are suspected of spreading *Leucocytozoon struthionis* in the Common Ostrich *Struthio camelus* in the Oudtshoorn district (Bennett *et al.* 1992e). The highest prevalence of Leucocytozoidae infections were found in Cape Sparrow at Patryskraal and in Cape Bulbul and Cape White-eye at Rondevlei Nature Reserve. The mountainous topography of the study sites in both regions with predominant Leucocytozoidae infections, have numerous perennial streams ideal for the sedentary existence of Simuliidae larvae. Although a distinct biome and climatic difference exists between these regions, the link for the high prevalence of 29.3% of infected birds sampled in the Lydenberg (25°06'S 30°27'E) area (Earlé *et al.* 1991), could be *S. adesi, S. nyaslandicum* and *S. vorax*. These vectors were identified to transmit *L. schoutedeni* and *L. neavi* to guineafowl and domestic chickens in Tanzania (Fallis *et al.* 1993), and they occur in both regions (Palmer & de Moor 1998). Earlé *et al.* (1991) speculated as to their existence in the Lydenburg area.

Three species within the family Sylviidae, Little Rush Warbler, African Reed Warbler and Lesser Swamp Warbler, and Levaillant's Cisticola from the newly formed family Cisticolidae occupy habitats in and near wetlands which are also occupied by avian haemosporidian vectors. Despite the coexistence within the same habitat of both vectors and avian species it was Little Rush Warblers and Levaillant's Cisticola which were recorded as having negative infections. While at the same time 2.5% of trapped Lesser Swamp Warbler and 18.9% of African Reed Warbler were recorded as being infected. Birds of the same species sampled at Mondplaas (33° 24'E 24° 59'S, [A.B. Schultz unpublished data]) and in the Afrotropical Region (Ashford *et al.* 1976, Bennett *et al.* 1992a) recorded similar results, also indicating no infections for the Little Rush Warbler and Levaillant's Cisticola. Once these species are infected they may not be capable of surviving the acute stage of infection, thus only healthy birds are trapped because weak individuals are eliminated by predators (Holmes 1982).

The resting and foraging behaviour of the Little Rush Warbler and Levaillant's Cisticola are confined to low down and upper stratum of dense vegetation boarding water bodies, where they are rather sedentary, as opposed to the migratory nature of the African Reed Warbler and the mobile and colonizing nature of the Lesser Swamp Warbler (Berruti *et al.* 1997). Although they share the same habitat with the Little

Rush Warbler and the Levaillant's Cisticola, it is their movement across biomes (Berruti *et al.* 1997) which brings them into contact with vectors.

Adult vector species of haemosporidia have a tendency to rest away from vegetation boarding wetlands, preferring habitats which provide protection from wind, sunlight, herbage and heavy dews on ground-level, thus resting in elevated areas, or in animal burrows, pits or inside dwellings (Crosskey 1990, Service 1995, Jupp 1996). Protection is provided to the Little Rush Warbler and Levaillant's Cisticola in their dense vegetated habitat, while African Reed Warbler and Lesser Swamp Warbler are exposed to aerial populations of unfed female vectors which are host seeking, when moving about. Although Levaillant's Cisticola does move away from the wetland vegetation to drier vegetation during the nonbreeding season (Berruti *et al.* 1997) this occurs after the vector breeding season (Valkiūnas 2005).

When avian systematics of the highly controversial family Sylviidae (Erard *et al.* 1997, Ryan 2002) which has been based upon the work of Sibley and Monroe (1990) has been reshaped, and the confounding relationships of the warbler and cisticola groups (Hockey *et al.* 2005) established, a structure might emerge indicating that Little Rush Warbler and Levaillant's Cisticola represent an evolutionary lineage which began earlier in evolutionary history than other warblers. This could explain an ancient evolutionary association with the vectors, of having evolved mechanisms for avoiding or reducing vector contact (Mendes *et al.* 2005); ornithophilic Simuliidae have evolved the adaptation of a tarsal claw for feeding on birds (Shewell 1955, Bennett 1960, Crosskey 1990). The negative prevalence of infection in these two species also coincides with findings of Logie *et al.* (1998) regarding Dippers *Cinclus cinclus* in Scotland, which also has negative infections, is sedentary, but inhabitat fast-flowing streams which provide ideal habitat for Simuliidae. These differences could reflect variations in the immune system, or protective behaviour, or the frequency with which high infection-risk habitats are used (Mendes *et al.* 2005)

The transmission of avian haemosporidia comprises a set of complex factors which relate to the biological properties of the avian host and vector, which includes certain abiotic factors. This frequently results in species inhabitating similar conditions but having different parasitological indices (Valkiūnas 2005). This is discernible in the Willow Warbler *Phylloscopus trochilus* with lower prevalence of infections when compared to the Blackcap *Sylvia atricapilla,* while both inhabit the same specific region (Valkiūnas 2005), comprising woodland and scrub with a deciduous element (Cramp 1992). Valkiūnas (2005) is of the opinion that the extent of contact between bird and vector is directly related to numerous peculiarities relating to a bird's biology. He concludes that deeper analyses of these processes which take place in nature are necessary to solve the problem.

Three of the four sites selected for analysis (Figure 4.2) were characterized by indigenous vegetated landscapes. Although the site at Glencairn is a small suburban garden, it is situated within a climatic and ecological setting comparable to the other three sites and is situated within the broader activity habitat of the birds. The first peak coincided with the end of the rainy season and the start of spring in September, when ambient temperatures increase. This peak is associated with increased vector activity by simuliid flies, resulting from increased water run-off after winter rains, coupled with increasing ambient temperatures, which also create favourable environmental conditions for both hippoboscid flies and mosquitoes. The second peak occurred in January prior to peaks in average ambient temperature and humidity in February, and when bird reproduction from spring has increased population size, resulting in large numbers of immature birds, which are highly susceptible to infection (Levine and Hanson 1953, Thul *et al.* 1980, Valkiūnas 2005),

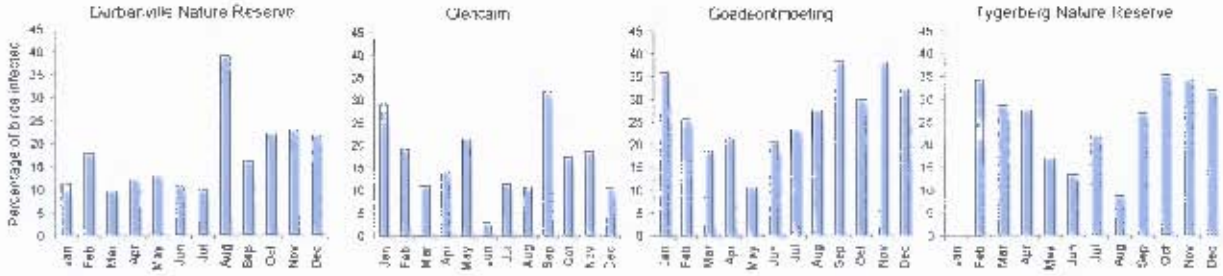

Figure 4.2 Seasonal pattern of inclusive avian haemosporidian parasite prevalence at each of the four study sites within the Greater Cape Town area (Summarized from appendices 2, 3, 4, 10 and 21).

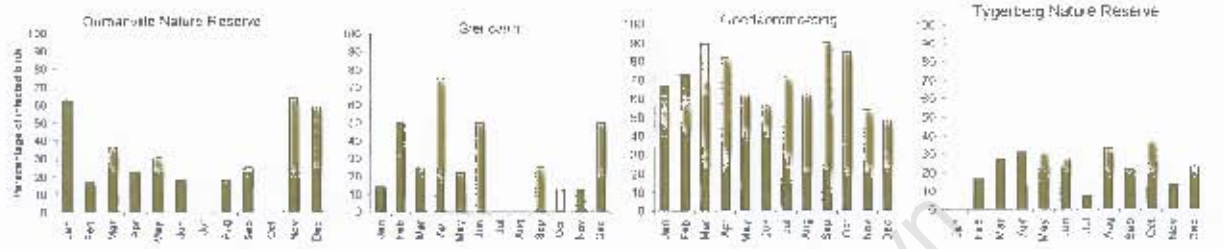

Figure 4.3 Seasonal pattern of *Haemoproteus* species prevalence in birds from the four study sites within the Greater Cape Town area (Summarized from appendices 2, 3, 4, 10 and 21).

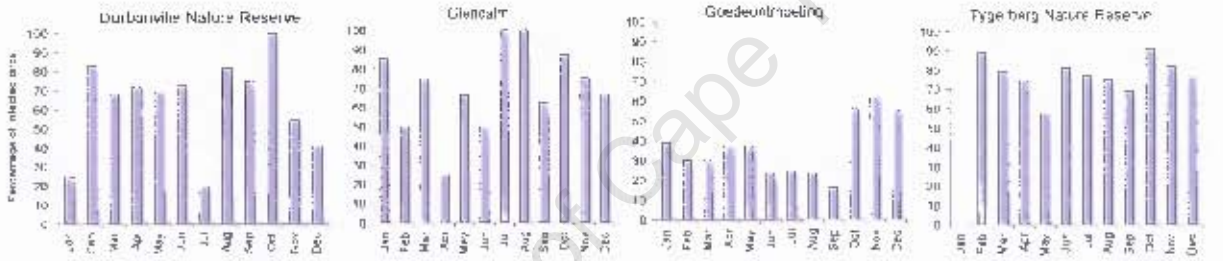

Figure 4.4 Seasonal pattern of *Leucocytozoon* species prevalence in birds from the four study sites within the Greater Cape Town area (Summarized from appendices 2, 3, 4, 10 and 21).

thus enhancing rapid parasite transmission (Valkiūnas 2005). Earlé *et al.* (1991), found in the summer rainfall region of northern of northern South Africa that prevalence of infections peaked at the beginning of winter in May to June, when temperatures decrease and the rains cease. This contrasts with the present study having two peaks, the main peak occurring with the commencement of summer.

Prevalence of infections also indicated two seasonal lows without a complete winter interruption. The first occurred in late autumn/early winter during May to July, when winter rain resumed and ambient temperatures reach seasonal lows in addition to extreme windy conditions which reduce vector numbers and haemosporidian transmission (Bennett *et al.* 1992f). The second low period occurred at the beginning of summer, during December, which was less of a decrease than the first. This period of decrease in prevalence of infection rates, which occurs during spring, is a natural seasonal phenomena known as "spring relapse" (Valkiūnas 2005). It results from acute primary parasitemia, which naturally follows a peak in haemosporidian infection which has been synchronized to the bird's breeding season, coinciding with the natural increase in vector activity and parasite transmission. Spring relapse, unknown at the time, also occurred in the Earlé *et al.* (1991) study. This decrease in prevalence of infections is dissimilar to the autumn decrease, which results from a natural seasonal vector decrease. The Earlé *et al.* (1991) study was characterized by a single seasonal low in prevalence of infections, coinciding with the second low of the present study—occurring with the commencement of summer.

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Haemoproteidae prevalence remained active throughout the year without any winter interruption, although there was an overall decrease, Tygerberg Nature Reserve recording the lowest prevalence in contrast to Goedeontmoeting which recorded the highest transmission rate over the same period (Figure 4.2). In Florida, *H. mansoni* and *H. meleagridis* was also recorded throughout the year (Forrester *et al.* 1974, Atkinson 1988). The Earlé *et al.* (1991) study showed similar results at Bloemfontein, recording a peak over winter and early spring, while Lydenburg recorded a peak in seasonal prevalence during spring and summer, almost similar to Durbanville Nature Reserve. The overall seasonal pattern of prevalence differs between the two regions due to two peaks being recorded in the present study corresponding with the same results recorded in Uganda (Bennett *et al.* 1974).

Leucocytozoidae transmission, as with Haemoproteidae, remained active throughout the year without any winter interruption (Figure 4.3) as was also found by Earlé *et al.* (1991). This seasonal pattern also occurs in South Carolina where *Leucocytozoon smithi* transmission takes place throughout the year (Noblet *et al.* 1975), although winter interruptions in haemosporidian transmission do occur in regions where seasonal changes are notably expressed (Noblet *et al.* 1975, Bennett *et al.* 1992e).

Seasonal prevalence patterns at all study sites recorded active transmission by vectors of Haemoproteidae and Leucocytozoidae throughout the year (Figure 4.1, 2, 3). This pattern is typically encountered at medium latitudes with mild climates and warm seasons (Noblet *et al.* 1975, Gabaldon & Ulloa 1980, Atkinson 1988, Atkinson *et al.* 1988, Atkinson & van Riper 1991). These weather conditions create relatively stable vector populations, resulting from favourable temperature and the necessary humidity for development and subsequent transmission of infection (Valkiūnas 2005). Thus a correlation of prevalence with temperature exists, which is also reflected in the Earlé *et al.* (1991) study. Irrespective of the present study being situated within a winter rainfall region and the Earlé *et al.* (1991) study done in a summer rainfall region, prevalence was recorded throughout the year in both regions negating a correlation with rainfall as put forward by Earlé *et al.* (1991), although Lydenburg was the only site to record an increase (± 7%) in prevalence with the onset of summer rainfall. These findings confirm that in temperate environments resident passerine birds maintain levels of avian haemosporidian infection prevalence throughout the year, in spite of seasonal variation in rainfall (Fallon *et al.* 2004)

For the past two decades all species of avian haemosporidian parasites were described as new species when infecting birds belonging to separate families (Bennett *et al.* 1991a, 1991b, 1992a). This was taken as the maximum level of specificity for the practice of avian haemosporidian taxonomy. From this followed the principle "one host-one parasite" (Bennett *et al.* 1992a, Valkiūnas 2005) resulting in many publications describing new species (Laveran & Marullaz 1914, Fallis & Bennett 1960, Bennett & Earlé 1992, Bennett & Peirce 1992, Bennett & Squires-Parsons 1992, Bennett *et al.* 1992b, 1992c, 1992d, 1993, 1995, Bennett 1993). Description of new species was based upon morphology of gametocyte development in the peripheral blood of the host. This did not distinguish between morphologically identical gametocytes from different avian families as being identical species. In this study *L. fringillinarum* is a synonym of *L. whitworthi, L. bouffardi, L. brimonti, L. sturni, L. deswardti,* and *L. gentili,* recorded nine times as a new species from the families Hirundinidae, Pycnonotidae, Sturnidae, Promeropidae, Ploceidae, Estrildidae and Fringillidae (Table 4.2) all within the order Passeriformes. In all cases gametocytes of these parasites were morphologically indistinguishable from *L. fringillinarum* although gametocyte size varies within the same species of leucocytozoids due to development in different vertebrate hosts (Valkiūnas 2005). Character differences of this nature are unsuitable for naming a new species of *Leucocytozoon* or

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Haemoproteus species. Given the duplication of identical haemosporidian species with different names parasitizing birds within avian families confined to the same avian order, warrants a revision of Haemoproteidae species taxonomy, where avian order becomes the maximum level of specificity as put forward by Valkinūnas (2005). Confirmation for such a taxonomic system through experimental studies can be found, by cross infection between avian families (Wenyon 1926, Fallis & Bennett 1960, Atkinson 1986, Križanauskienė 2005), including field studies (Malchevsky & Pukinsky 1983, Valkiūnas 1997). Also, through mitochondrial DNA amplification it has be shown that *Haemoproteus* species shift not only between species within the same genus, but also between species in different families (Bensch *et al.* 2000). Thus *Haemoproteus* and *Leucocytozoon* species (Table 4.2) encountered in this study are considered to be relegated to synonymy with earlier described morphologically similar valid species in avian hosts of other families.

Further reasons for dismantling the old paradigm of the family-level structure is the adoption of the new classification of birds as listed in the 7th edition of Roberts (Hockey *et al.* 2006). Numerous species have been placed in newly created family structures, while others are taken out of their existing families and assigned to new placing to which the population species are more related too. An example in this study is the family Sylviidae. Bar-throated Apalis, Grey-backed Cisticola, and Karoo Prinia have been placed in Cisticolidae, a newly formed family, while Cape Grassbird, Long-billed Crombec, Little Rush Warbler, African Reed Warbler, Lesser Swamp Warbler, Chestnut-vented Tit Babbler and Garden Warbler remain placed within Sylviidae (Table 4.1). Avian haemosporidian species *H. sylvae* and *L. phylloscopus* become common denominators to both avian families (Table 4.2), nullifying the family as maximum level of specificity. Thus the present position of describing *Haemoproteus* and *Leucocytozoon* species at the level of family and subfamily level cannot be accepted as the basis of taxonomy as it ignores the facts.

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CHAPTER FIVE

Infection prevalence, and absence of positive correlation between avian haemosporidian parasites, mass, and body condition in the Cape Weaver *Ploceus capensis*

May I never lose you, oh, my generous host, oh, my universe. Just as the air you breath, and the light you enjoy are for you, so you are for me.
Primo Levi, *Man's Friend*

INTRODUCTION

The body condition of a bird influences its physiological state, affects its performance, and its response to its environment (Brown 1996). Investigations have revealed that regardless of evolutionary coexistence between parasite and host, virulent strains can influence host population dynamics (Anderson & May 1978, 1979, May & Anderson 1978, 1979, Ewald 1983). These investigations have lead to numerous studies on avian haemosporidian parasite interactions: sexual selection (Hamilton & Zuk 1982, Read 1987, Pruett-Jones *et al.* 1990, Zuk 1991), ecology, behaviour and evolution (Loye & Zuk 1991, Clayton & Moore 1997, Møller 1997), and parasite influence on host fitness (Schall 1983, Pruett-Jones *et al.* 1990, Hudson & Dobson 1991, Korpimäki *et al.* 1993, Allander & Bennett 1995, Oppliger & Clobert 1997). However, effects of avian haemosporidia parasites on host body condition lacked complete attention in the studies reviewed by Brown (1996). Since then the only study to investigate avian haemosporidian parasite impact on body condition has been by Hatchwell *et al.* (2001) and Edler *et al.* (2004), although numerous studies have looked at the association of avian haemosporidian parasites and body mass (e.g. Ashford 1971, Smith & Cox 1972, Peirce 1984, Atkinson *et al.* 1988, Bennett *et al.* 1988).

Correlational studies have found associations between *Leucocytozoon* infections and body mass (Karastad 1965, Peirce 1984, Hatchwell *et al.* 2001, Valkiūnas 2005) while heavy infections with *Haemoproteus* species lead to a decrease of migratory fat (Valkiūnas 2005). Smith and Cox (1972) found a negative influence on body mass of Palearctic migrants, while Bennett *et al.* (1988) failed to confirm the influence of *Haemoproteus* and *Leucocytozoon* species on the body mass of passerines. Due to contrasting influences avian haemosporidian parasites exert on their avian hosts, coupled with the variables of body mass to food consumption, time of day, weather, moult, age, sex, body fat, variation in size and geographical origin, the use of mass alone to assess body size and condition is unreliable (Beer & Boyd 1962, Owen & Cook 1977, Summers 1988). Also, for many species body mass varies regionally (e.g. Hockey *et al.* 2006).

This study considers the Cape Weaver *Ploceus capensis*, on the farm Goedeontmoeting (33°41'S 18°36'E) situated in the Swartland agricultural region 30 km north of Cape Town, South Africa (see Chapter 3). There are regional differences in body mass for this species; for example there is considerable local variation extending across their range, being clinal (Fry & Keith 2004), with the Western Cape recording the largest overall bird size and Eastern Cape the smallest females (Craig

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2004). The selection of a single study site within the Western Cape eliminates the problems of cline. The Cape Weaver was present at the study site in numbers in all months of the year; at the overall retrap rate of 21% during a five-year period, indicating the sedentary nature of the population—a characteristic of the species (Oatley & Underhill 2001, Craig 2005). At the study site sedentariness can be ascribed to the availability of winter crops, large scale feeding of livestock during summer (grain wastage), and availability of summer grass seeds, in conjunction with reed roosting sites in close proximity along the Mosselbank River.

In this study, parasitism of Cape Weaver by three genera of avian haemosporidian parasites (*Haemoproteus, Leucocytozoon* and *Plasmodium*) is examined in relation to body mass, condition and time, including the pattern of infection prevalence.

MATERIALS AND METHODS

Birds were trapped in mist-nets at Goedeontmoeting (33°28'S, 18°44'E), from March 1993 to February 1995. Birds were ringed, measured and mass taken in accordance with recommendations for standard practice made by SAFRING (de Beer *et al.* 2000). Birds were aged and sexed using the procedures defined by Elliott 1973); only birds aged as adults were considered in this study. Wing length was measured to the nearest 1.0 mm using the maximum chord method (flattened, straightened wing). Body mass was measured to the nearest 0.5 g using spring balances. Blood smears were obtained from Cape Weavers by venipuncture to the brachial vein in the wing to obtain a thin blood smear, which was subsequently air-dried. Blood smears were fixed with absolute methanol or May Grünwald's and stained with 4% Giemsa solution for 60 minutes. Blood smears were examined under light microscopy at 1000X oil immersion magnification for avian haemosporidian parasites, which were identified to species level. Intensity of infections was not assessed. Sex and age of each bird were determined where possible. Immature birds were excluded from the analysis if sex could not be determined.

In order to examine the relationship between prevalence of infection and mass, an analysis of covariance was performed. This was done in two ways; firstly using mass with sex and month as covariate. Secondly, a mass index standardized for wing length was applied, and then sex was used as a covariate. The standardization used was that of Summers (1988) based on computing the allometric relationship between wing length and mass of the form $p=aw^b$, where a and b are estimated from the sample using log-log regression on the observed values for mass m (g) and wing length w (mm). The mean index of a particular bird of observed mass, m and wing length w was calculated as $i=m/p$, where p (g) is the predicted mass of a bird with wing length w. The mass index is the observed mass divided by the predicted mass for a bird of given wing length. A bird with the index $i>1$ has a larger mass than predicted for its wing length, and a bird with $i<1$ has a smaller mass than predicted for its wing length (because mass only is not a true reflection of body size, neither of the bird's condition) (Owen & Cook 1977, Summers 1988).

RESULTS

Blood smears from 945 adult Cape Weavers were acquired of which 632 were male and 308 were female (Table 5.1), of which 58.79% of the males and 61.90% of the females were infected. Mass and wing length were also available for birds sampled in all months of the year (Table 5.1 & 5.2).

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Table 5.1 Mean mass (g ± SD) of Cape Weaver *Ploceus capensis* by sex class captured at Goedemoeting from March 1993 to January 1995. Data are pooled from all months across years with sample sizes shown in brackets.

Month	Male		Female	
	Uninfected	Infected	Uninfected	Infected
January	46.65 ± 2.788 (40)	46.33 ± 3.456 (36)	40.55 ± 2.736 (20)	41.74 ± 4.351 (23)
February	46.33 ± 1.680 (18)	47.05 ± 1.477 (20)	40.00 ± 2.665 (12)	39.62 ± 2.873 (13)
March	46.38 ± 2.960 (47)	46.53 ± 1.581 (15)	40.04 ± 2.475 (23)	40.25 ± 2.315 (8)
April	48.27 ± 2.935 (40)	47.94 ± 1.799 (16)	40.71 ± 3.296 (7)	38.83 ± 4.997 (6)
May	48.32 ± 2.950 (63)	48.74 ± 1.944 (19)	41.07 ± 1.939 (15)	39.33 ± 0.577 (3)
June	46.91 ± 2.863 (69)	46.94 ± 3.326 (18)	39.74 ± 2.623 (31)	40.11 ± 2.848 (9)
July	47.77 ± 3.287 (30)	47.14 ± 2.506 (7)	40.44 ± 2.268 (9)	40.71 ± 1.799 (7)
August	46.97 ± 2.146 (29)	47.65 ± 2.368 (31)	40.56 ± 2.893 (32)	41.13 ± 2.532 (15)
September	47.31 ± 2.301 (29)	47.51 ± 2.738 (39)	43.19 ± 4.109 (16)	46.73 ± 3.495 (11)
October	47.33 ± 1.966 (6)	48.31 ± 2.435 (13)	39.75 ± 1.797 (8)	39.00 ± 3.742 (7)
November	51.00 ± 1.000 (3)	46.83 ± 1.140 (6)	40.40 ± 3.371 (5)	39.67 ± 2.082 (3)
December	47.00 ± 4.170 (24)	48.86 ± 4.446 (14)	44.82 ± 4.167 (11)	43.21 ± 4.492 (14)
Total	**47.41 ± 2.925 (398)**	**47.43 ± 2.945 (234)**	**40.80 ± 3.085 (189)**	**41.36 ± 3.974 (119)**

Table 5.2 Mean wing length (mm ± SD) of Cape Weaver *Ploceus capensis* by sex class captured at Goedemoeting from March 1993 to January 1995. Data are pooled from all months across years with sample sizes shown in brackets.

Month	Male		Female	
	Uninfected	Infected	Uninfected	Infected
January	91.10 ± 2.010 (40)	91.06 ± 1.943 (36)	85.75 ± 1.689 (20)	85.41 ± 2.323 (22)
February	91.72 ± 1.934 (18)	92.20 ± 1.528 (20)	85.83 ± 2.118 (12)	86.15 ± 1.068 (13)
March	93.17 ± 1.982 (47)	91.67 ± 2.210 (15)	86.39 ± 1.718 (23)	85.62 ± 1.768 (8)
April	93.20 ± 1.924 (40)	92.62 ± 1.265 (16)	87.00 ± 1.528 (6)	86.00 ± 2.683 (6)
May	93.03 ± 1.992 (63)	92.13 ± 1.668 (15)	86.93 ± 1.598 (15)	87.00 ± 2.000 (3)
June	93.10 ± 2.438 (69)	92.39 ± 1.820 (18)	86.61 ± 1.501 (31)	86.67 ± 1.871 (9)
July	92.47 ± 2.013 (30)	92.86 ± 3.300 (7)	87.00 ± 1.773 (10)	86.57 ± 0.787 (7)
August	92.72 ± 2.103 (29)	92.74 ± 2.385 (31)	86.00 ± 1.966 (33)	87.13 ± 1.642 (15)
September	93.13 ± 2.123 (29)	93.56 ± 1.799 (39)	86.33 ± 2.490 (15)	86.60 ± 1.955 (10)
October	92.00 ± 1.414 (6)	91.85 ± 2.563 (13)	86.00 ± 2.230 (8)	86.14 ± 0.900 (7)
November	93.33 ± 3.215 (3)	92.50 ± 0.894 (6)	86.40 ± 1.049 (5)	84.00 ± 1.000 (3)
December	90.40 ± 2.000 (25)	90.64 ± 2.006 (14)	86.75 ± 1.277 (12)	86.43 ± 1.742 (14)
Total	**92.53 ± 2.230 (399)**	**92.24 ± 2.059 (230)**	**86.35 ± 2.051 (190)**	**86.20 ± 1.844 (117)**

Six species of avian haemosporidian parasites from four genera were recorded (Table 5.3). The complete data set is contained in appendix 22.

Overall, *Haemoproteus queleae* was the most commonly encountered infection, being 69.45% of the infected sample, followed by *Leucocytozoon bouffardi* occurring in 23.91% of the infected sample. The remaining species of avian haemosporidian parasites occurred in substantially smaller number of infections than the previous two species, with *Plasmodium* species infections occurring in 5.76%, and *Trypanosoma everetti* in 0.28% (n=1) of the infected sample. Double infections occurred in 40 birds and 11.52% of the infections, with females having the most double infections occurring during summer (Table 5.3). Infections between sexes varied significantly (t-test: t=2.88, df=22, P=0.009) with infection prevalence patterns for both sexes mimicked each other from March to June (Figure 5.1).

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Table 5.3 Monthly prevalence data of avian haemosporidian parasites of 945 Cape Weavers *Ploceus capensis* captured at Goedemoeting from March 1993 to January 1995. *H. quel=Haemoproteus quelcae, L. bouf=Leucocytozoon bouffardi, P. reli=Plasmodium relictum P. vaug=P. vaughani, P. circ=P. circumflexum* and *T. ever=Trypanosoma everetti*. Figures in parentheses represent percentages of birds infected.

Month	Male Infected						Female Infected					Overall Total
	H. quel	*L. bouf*	*P. reli*	*P. vaug*	*T. ever*	Total	*H. quel*	*L. bouf*	*P. reli*	*P. circ*	Total	
January	24	12	—	—	—	36[9](16.0)	12	7	1	—	22[16](18.8)	58
February	12	7	—	—	1	20[10](9.1)	9	3	—	1	13[17](11.1)	33
March	11	4	—	—	—	15[1](6.8)	4	2	2	—	8(6.8)	23
April	11	4	1	—	—	16[2](7.3)	5	1	—	—	6[1](5.1)	22
May	10	2	3	—	—	15[3](6.8)	3	—	—	—	3(2.5)	18
June	9	6	1	2	—	18(8.2)	6	2	1	—	9(7.6)	27
July	3	2	2	—	—	7[4](3.1)	6	—	1	—	7(5.9)	14
August	22	9	—	—	—	31[5](14.1)	11	3	1	—	15[12](12.8)	46
September	29	7	3	—	—	39[8](17.8)	8	2	—	—	10(8.5)	49
October	13	—	—	—	—	13(5.9)	6	1	—	—	7[13](5.9)	20
November	4	2	—	—	—	6[7](2.7)	3	—	—	—	3[14](2.5)	9
December	10	3	—	1	—	14[3](6.3)	10	4	—	—	14[15](11.9)	28
Total	**158**	**58**	**10**	**3**	**1**	**219(66.2)**	**83**	**25**	**6**	**1**	**117(33.7)**	**347**

[1]double infection=1, [2]double infections=2, [3]double infection=1, [4] double infections=2, [5]double infections=3, [6]double infections= 4. [7]double infections=2, [8]double infections=3, [9]double infections= 5, [10]double infection=1, [11]double infection=1, [12]double infection=1 [13]double infection=1, [14]double infections=2, [15]double infections= 5. [16]double infections=3, [17]double infections=3.

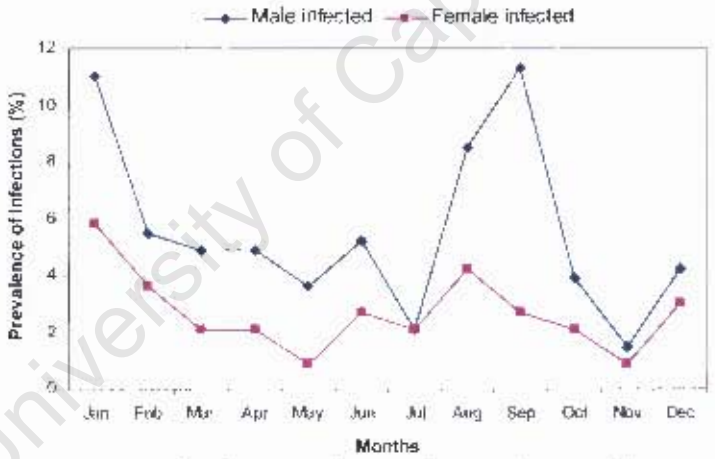

Figure 5.1 Monthly prevalence of infections of 347 Cape Weaver *Ploceus capensis* captured at Goedeonmoeting from March 1993 to January 1995.

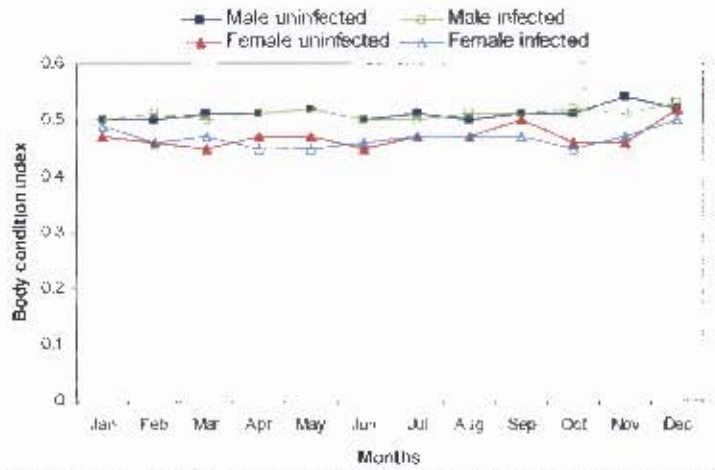

Figure 5.2 Condition index (m/p) of 945 Cape Weaver *Ploceus capensis* captured at Goedeonmoeting from March 1993 to January 1995. Values indicate monthly means ± se.

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The effect on mass was not significant with infected birds on average 0.10 g heavier than uninfected birds (two-tailed test: t_{938}=0.37, P=0.74). However, males were on average 6.40 g heavier than females variance in mass (t_{942}=29.5, P<0.001). A regression model including only sex as a covariate accounted for 48.1% of the variance in mass; the addition of month as a covariate, with a level for each month, increased the percentage of variance explained to 50.2%. Although the month effects were formally statistically significant ($F_{11,927}$=4.49, P<0.001) they were relatively small and showed no seasonal pattern (range of effects 2.2 g (Figure 5.2)), and the statistical significance is probably an artifact of the large sample size and not biological consequential. In regression models including sex, and both sex and month, the effect of infection was not significant (P=0.35 and P=0.82, respectively). Likewise, models including interaction terms were not significant, so there was no tendency for infections to impact the sexes differently (P=0.20).

The allometric relationship to predict mass (g) from wing length w (mm) was p=0.02793$w^{1.640}$. In the allometric regression, 44% of the variance in mass was explained by wing length. The analysis of covariance was repeated with the body condition index rather than observed mass. Sex now accounted for only 4.2% of the variance, indicating that the allometric relationship was largely successful in taking wing length into account. The effect of month on the body condition index was significant ($F_{11,918}$=5.87, P<0.001), but the effects were relatively small (Figure 5.2). The effect of infection on the body condition index was not significant (t_{929}=1.53, P=0.127), nor was it significant when sex or month, or interactions, were included in the model. There was therefore no impact of avian haemosporidian parasites on the masses of Cape Weavers at Goedeontmoeting.

DISCUSSION

Sample size of Cape Weavers was biased towards male birds during each month of the study period with the exception of July when the catch had balanced sex ratios. In a sample of 3 500 Cape Weavers trapped in the Western Cape a bias towards males also occurred with some individual catches strongly biased in favour of one or the other sex (Elliott 1980). Heÿl (1980) suggested that these differences were due to dispersal and catchability of age classes, but a greater variation in the female Cape Weaver diet (Elliott 1973) could also contribute to the dispersal and catchability bias.

The mass and wing length of the Cape Weaver reported here are within the range of measurements given by Elliott (1973) for the Western Cape although the mean wing length is slightly longer (1.0 mm for males and 1.2 mm for females). During the Elliott (1973) study, the recommended method for measuring wing length already ought to have been the flattened straightened wing method, giving maximum chord, which had only recently become the norm in South Africa (Ledger 1969); this method has remained in use (de Beer *et al.* 2000). The apparent increase in wing length may be due to inter-observer differences; in the 1990s observers were trained to flatten and straighten the primary wing feathers more diligently than was the case two decades earlier (e.g. Redfern 2006).

The pattern of infection prevalence over the entire study period confirms earlier studies showing that differences in infection prevalence to birds in males and females can differ (Figure 5.1) (Markov & Chernobai 1968, Applegate 1971, Burtikashvili 1978, Valkiūnas 1987). This study also corroborates the findings that decreased activity increases the probability of infection by avian haemosporidian parasites, due to inactive birds at nests (Valkiūnas 1984, 1987). At the start of the breeding season from July in the Western Cape (Elliott 1973), the male Cape Weaver becomes territorial and less mobile at the nest site due to nest construction and courtship

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display. Being polygynous and having up to seven females (Elliott 1973) nest building and courtship display continue until September thus the period of high prevalence in the males (Figure 5.1). In contrast, the female has a short period for of mate-searching and nest selection before starting egg laying and incubation. This results in a decline of prevalence as the female is shielded within a nest having a tunnel shaped entrance, which decreases the probability of access by vectors and of parasite transmission (Ashford 1974, Kučera 1981a, 1981b, Valkiūnas 1987). Correspondingly, the male becomes increasingly mobile from September on completion of nest building and courtship display, with subsequent decrease in prevalence.

Commencement of the breeding season for the Cape Weaver coincides with increased prevalence for both sexes in this study. Previous studies have also found correlations between reproductive effort and increased prevalence because of reproductive investment in future young (Loye & Zuk 1991, Norris *et al.* 1994, Richner *et al.* 1995, Sheldon & Verhulst 1996, Møller 1997, Newton 1998, Nordling *et al.* 1998). Also, the breeding period may have much in common with heavy endurance training in humans for marathon running and the intense sustained endurance of long-distance migratory flights of shorebirds—both activities being demanding on the immune system (Piersma 1997). After intense exercise the immune system is preoccupied with removing macrophages and phagocytic cells from injured muscle cells, thereby not being capable of responding fully to new infections (Lochmiller & Deerenberg 2000). Hence the reason for the increased possibility of "exercised" as opposed to "non-exercised" humans contracting a cold, and of shorebirds being susceptible to parasitic infection after demanding migratory flights (Piersma 1997). This explains the trade-off between reproductive effort and immune system maintenance which results in positive associations between reproductive effort and avian haemosporidian prevalence, also found in similar studies relating to various species (Norris *et al.* 1994, Allander 1997, Oppliger *et al.* 1997, Siikamäki *et al.* 1997, Nordling *et al.* 1998). Despite continual exposure to an environment containing parasites, which naturally results in heavy investment to the immune response system (Glick 1986), the polygynous Cape Weaver male still has a higher increase of avian haemosporidian infections.

The seasonal spring relapse as observed in this study is a natural phenomenon synchronized with the period of bird breeding when vector intensity and activity peak, with a corresponding peak in parasite transmission and prevalence (Valkiūnas 2005). Contributing to the increase in prevalence of infections during this period is the high stress level, relating to the reproductive cycle (Mendes *et al.* 2005). This high stress level also increase susceptibility to parasitism (Mendes *et al.* 2005), as stress induces immunosuppression (Sapolsky 1992, Apanius 1998). This process started and ended simultaneously for both male and female Cape Weavers in November and peaked in January.

Numerous studies investigating mass dynamics in various bird species show an increase in body mass from a low point after the breeding season which results from reproductive energetic demands (Summers *et al.* 1992, Smith & Metcalfe 1997, Swaddle & Witter 1997, Cresswell 1998, Browne & Aebischer 2003). This characteristic build up in body mass has been well demonstrated in northern latitudes, particularly in Green-winged Teal *Anas crecca* (Baldassarre *et al.* 1986, Fox *et al.* 1992), which is a pattern of "adaptive winter-fattening hypothesis" essential to the birds survival during temporary food shortages or cold weather (Lehikoinen 1987, Rogers & Rogers 1990, Smith & Metcalfe 1997). Due to the temperate climate of the Afrotropical Region were seasonality is minimal, body mass variation is not as apparent (de Swardt 1992) as for the northern equivalent. Most mass increases do occur during winter (June-August) (Cooper 1975, Cooper & Underhill 1991, Underhill

& Underhill 1997, Underhill *et al.* 1999, de Swardt *et al.* 2003) with the exception of Cape Robin Chat which has peak mass in summer—probably all cases are related to food availability (Bonnevie *et al.* 2003). Newton (1998) suggested that body mass can be seen as a measure of food availability, but can be misleading as many factors such as season, dominance, predation risk and breeding status do also affect body mass (Newton 1998).

Although the greater proportion of the infected sample in this study consisted of *Haemoproteus queleae,* no negative impacts could be accounted for. Confirmation that *Haemoproteus* species have no effect on their hosts is supported by numerous studies (Levine 1973, Fallis & Dresser 1977, Kemp 1978, Bennett *et al.* 1982, Bennett *et al.* 1993), although other studies have found deleterious effects (O'Roke 1930, Karastad 1965, Khan & Fallis 1968, Oosthuizen & Markus 1968, Markus & Oosthuizen 1972, Peirce 1984, Atkinson & Forrester 1987, Hartley 1992). No negative influence on body mass by avian haemosporidian parasites occurred in a sample of 148 birds in Zambia, the only exception being a single sick Emerald-Spotted Wood-Dove *Turter chalcospilos* with a double infection; it had high *Leucocytozoon marchouxi* and low *Haemoproteus columbae* parasitaemia (Peirce 1984). Studies found a positive correlation between body mass and avian haemosporidia from 3 739 passerines trapped and examined on Newfoundland Island, Canada (Bennett *et al.* 1988). Valkiūnas (2005) is of the opinion that Bennett *et al.* (1988) failed to obtain data confirming the influence of *Haemoproteus* and *Leucocytozoon* species infections on body mass as a result of not taking into account the confounding effect of a positive correlation between body mass and migratory fat in the 3 739 birds examined. Studies confirm that *Haemoproteus fringillae* in Chaffinch *Fringilla coelebs* results in significant body mass loss, but only during periods of peak parasitemia making it impossible to confirm negative impact on body mass loss in long-term investigations as presented by Bennett *et al.* (1988) (Valkiūnas 2005). Thus in this study there was no indication that avian haemosporidian parasites impacted negatively in the long term, or of significant seasonal variation on the body mass of the Cape Weaver, which could be associated with constant food availability throughout the year.

The analysis undertaken in this study provided no evidence of a decline in body condition of the Cape Weaver resulting from vector transmission of avian haemosporidia. In addition, given that the apparent increase in wing length is probably an artifact of measurement technique, it is likely that the wing length has remained constant over time. In conjunction with the lack of change in body mass, this supports interpretation that body condition of the Cape Weaver has remained constant over 22 years (Elliott 1973 to present study).

Although the no positive correlation between avian haemosporidia and body condition in this study is supported by Edler *et al.* (2004), it does contrast with other findings (Hatchwell *et al.* 2001). In the Common Blackbird *Turdus merula* there was significant seasonal variation in body condition which could have been due to time of sampling (breeding period) while a progressive decline in body condition was found to be associated with increased avian haemosporidia genera (Hatchwell *et al.* 2001). The *Leucocytozoon* genera were also correlated to negative adult body condition and wing length, which could also be related to time of sampling during chronic *Leucocytozoon* infections, (Hatchwell *et al.* 2001). Valkiūnas (2005) found that the influence of avian haemosporidia on the body mass (and body condition) only manifests it self within short periods (several days) during the peak of parasitemia (chronic phase), where after the intensity decreases, becoming problematic to detect changes in the pattern of body mass over an extended period of investigation as experienced in this study.

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APPENDICES

The following set of 22 appendices contains data sampled from 10 study sites within the Greater Cape Town Area on which this thesis is based.

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Appendix 1 Summary of prevalence data of haemosporidian parasites of birds trapped at Bettys Bay from February 1993 to January 1994. *Haem.=Haemoproteus; Leuco=Leucocytozoon; Plasm=Plasmodium.*

Species	Total individuals		Individuals with		
	Examined	Infected	*Haem*	*Leuco*	*Plasm*
Anthobaphes violacea Orange-breasted Sunbird	5	1	—	1	—
Apalis thoracica Bar-throated Apalis	1	0	—	—	—
Cisticola subruficapilla Grey-backed Cisticola	1	1	—	1	—
Cinnyris chalybeus Southern Double-collared Sunbird	11	1	—	1	—
Colius striatus Speckled Mousebird	17	1	—	1	—
Cossypha caffra Cape Robin-chat	18	0	—	—	—
Crithagra sulphuratus Brimstone Canary	5	2	—	2	—
Euplectes orix Southern Red Bishop	5	0	—	—	—
Hirundo fuligula Rock Martin	4	0	—	—	—
Hirundo rustica Barn Swallow	3	0	—	—	—
Motacilla capensis Cape Wagtail	2	1	—	—	1
Nectarinia famosa Malachite Sunbird	3	0	—	—	—
Onychognathus morio Red-winged Starling	4	1	1	—	—
Ploceus capensis Cape Weaver	67	12	5	6	1
Prinia maculosa Karoo Prinia	7	0	—	—	—
Promerops cafer Cape Sugarbird	202	22	—	22	—
Pycnonotus capensis Cape Bulbul[1]	19	11	—	12	—
Serinus canicollis Cape Canary	4	1	1	—	—
Sigelus silens Fiscal Flycatcher	4	1	1	—	—
Sphenoeacus afer Cape Grassbird	1	0	—	—	—
Streptopelia capicola Cape Turtle Dove[2]	1	1	1	1	—
Turdus olivaceus Olive Thrush	2	0	—	—	—
Urocolius indicus Red-faced Mousebird	1	0	—	—	—
Zosterops virens Cape White-eye[3]	51	42	28	34	1
Total	**438**	**98**	**37**	**81**	**3**
Percentage	—	**22.3**	**37.7**	**82.6**	**3.0**

[1] double infections = 1
[2] double infections = 1
[3] double infections = 21

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Appendix 2 Summary of prevalence data of haemosporidian parasites of birds trapped at **Durbanville Nature Reserve** from December 1992 to April 1995. *Haem.=Haemoproteus; Leuco=Leucocytozoon; Plasm=Plasmodium.*

Species	Total individuals		Individuals with		
	Examined	Infected	*Haem*	*Leuco*	*Plasm*
Acrocephalus baeticatus African Reed Warbler	1	0	—	—	—
Apalis thoracica Bar-throated Apalis	1	0	—	—	—
Bradypterus baboecala Little Rush Warbler	1	0	—	—	—
Chrysococcyx klaas Klaas's Cuckoo	4	0	—	—	—
Cinnyris afra Greater Double-collared Sunbird	2	0	—	—	—
Cinnyris chalybeus Southern Double-collared Sunbird	46	0	—	—	—
Cisticola tinniens Levaillant's Cisticola	13	0	—	—	—
Colius colius White-backed Mousebird	17	0	—	—	—
Columba livia Rock Dove	8	4	4	—	—
Columba guinea Speckled Pigeon	25	7	7	—	—
Cossypha caffra Cape Robin Chat	41	3	—	2	1
Crithagra albogularis White-throated Canary	3	1	—	1	—
Crithagra flaviventris Yellow Canary	5	0	—	—	—
Crithagra sulphuratus Brimstone Canary	9	2	—	2	—
Estrilda astrild Common Waxbill	3	0	—	—	—
Euplectes capensis Yellow Bishop [1]	15	7	4	3	1
Euplectes orix Southern Red Bishop [2]	11	5	4	1	1
Hirundo abyssinica Lesser Striped Swallow	1	0	—	—	—
Indicator minor Lesser Honeyguide	2	0	—	—	—
Lagonosticta rubricata African Firefinch	1	0	—	—	—
Lanius collaris Common Fiscal	9	0	—	—	—
Motacilla capensis Cape Wagtail	1	0	—	—	—
Nectarinia famosa Malachite Sunbird	56	0	—	—	—
Oena capensis Namaqua Dove	1	0	—	—	—
Passer melanurus Cape Sparrow	60	20	—	20	—
Ploceus capensis Cape Weaver	218	19	9	9	1
Ploceus velatus Southern Masked Weaver	95	8	—	4	4
Prinia maculosa Karoo Prinia	12	2	—	2	—
Promerops cafer Cape Sugarbird	86	3	—	2	1
Pycnonotus capensis Cape Bulbul	21	3	—	3	—
Riparia paludicola Brown-throated Martin	1	0	—	—	—
Sigelus silens Fiscal Flycatcher	12	0	—	—	—
Streptopelia capicola Cape Turtle Dove	10	4	2	2	—
Streptopelia senegalensis Laughing Dove	9	3	1	2	—
Sylvietta rufescens Long-billed Crombec	3	1	—	1	—
Telophorus zeylonus Bokmakierie	1	0	—	—	—
Tricholaema leucomelas Acacia Pied Barbet	17	1	—	1	—
Turdus olivaceus Olive Thrush	1	0	—	—	—
Urocolius indicus Red-faced Mousebird	21	0	—	—	—
Zosterops virens Cape White-eye [3]	180	51	17	37	—
Total	**1023**	**144**	**48**	**92**	**9**
Percentage	—	**14.0**	**33.3**	**63.8**	**6.2**

[1] double infections = 1
[2] double infections = 1
[3] double infections = 3

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Appendix 3 Summary of prevalence data of haemosporidian parasites of birds trapped at Glencairn from March 1993 to September 1995. *Haem.=Haemoproteus; Leuco=Leucocytozoon; Plasm=Plasmodium.*

Species	Total individuals		Individuals with		
	Examined	Infected	*Haem*	*Leuco*	*Plasm*
Andropadus importunus Sombre Greenbul	1	0	—	—	—
Anthobaphes violacea Orange-breasted Sunbird	5	1	—	1	—
Cinnyris chalybeus Southern Double-collared Sunbird	16	1	—	1	—
Colius colius White-backed Mousebird	3	0	—	—	—
Colius striatus Speckled Mousebird	32	1	—	1	—
Columba guinea Speckled Pigeon	3	0	—	—	—
Cossypha caffra Cape Robin Chat	31	0	—	—	—
Emberiza capensis Cape Bunting	12	0	—	—	—
Estrilda astrild Common Waxbill	17	0	—	—	—
Euplectes capensis Yellow Bishop	1	0	—	—	—
Hirundo cucullata Greater Striped Swallow	2	0	—	—	—
Hirundo fuligula Rock Martin	1	0	—	—	—
Laniarius ferrugineus Southern Boubou	12	7	3	4	—
Lanius collaris Common Fiscal	2	0	—	—	—
Monticola rupestris Cape Rock Thrush	1	0	—	—	—
Nectarinia famosa Malachite Sunbird	1	0	—	—	—
Onychognathus morio Red-winged Starling	69	4	2	2	—
Passer melanurus Cape Sparrow	6	0	—	—	—
Ploceus capensis Cape Weaver [1]	30	4	1	4	—
Ploceus velatus Southern Masked Weaver	2	0	—	—	—
Prinia maculosa Karoo Prinia	13	0	—	—	—
Promerops cafer Cape Sugarbird	21	2	—	2	—
Pycnonotus capensis Cape Bulbul	39	18	—	18	—
Riparia paludicola Brown-throated Martin	2	0	—	—	—
Serinus canicollis Cape Canary	5	0	—	—	—
Sphenoeacus afer Cape Grassbird	2	0	—	—	—
Streptopelia capicola Cape Turtle Dove	3	1	1	—	—
Streptopelia semitorquata Namaqua Dove	2	0	—	—	—
Streptopelia senegalensis Laughing Dove [2]	8	5	5	1	—
Sturnus vulgaris Common Starling	3	0	—	—	—
Telophorus zeylonus Bokmakierie	1	0	—	—	—
Turdus olivaceus Olive Thrush	2	0	—	—	—
Zosterops virens Cape White-eye [3]	57	26	6	25	—
Total	**405**	**70**	**18**	**59**	—
Percentage	—	**17.2**	**25.7**	**84.2**	—

[1] double infections = 1
[2] double infections = 1
[3] double infections = 5

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Appendix 4 Summary of prevalence data of avian haemosporidian parasites of birds trapped at **Goedeontmooting** from March 1993 to May 1995. *Haem=Haemoproteus, Leuco=Leucocytozoon; Plasm=Plasmodium.*

Species	Total individuals		Individuals with		
	Examined	Infected	*Haem*	*Leuco*	*Plasm*
Acrocephalus baeticatus African Reed Warbler	8	3	3	—	—
Apus caffer White-rumped Swift	1	0	—	—	—
Charadrius tricollaris Three-banded Plover	3	0	—	—	—
Chrysococcyx klaas Klaas's Cuckoo	2	0	—	—	—
Cisticola tinniens Levaillant's Cisticola	10	0	—	—	—
Cinnyris chalybeus Southern Double-collared Sunbird	3	0	—	—	—
Colius colius White-backed Mousebird	84	4	—	4	—
Colius striatus Speckled Mousebird	22	0	—	—	—
Columba guinea Speckled Pigeon	41	8	8	—	—
Cossypha caffra Cape Robin Chat	9	1	—	—	1
Cercotrichas coryphaeus Karoo Scrub Robin	5	0	—	—	—
Estrilda astrild Common Waxbill	46	1	—	1	—
Euplectes capensis Yellow Bishop [1]	32	9	3	5	2
Euplectes orix Southern Red Bishop [2]	337	80	62	23	—
Hirundo fuligula Rock Martin	1	0	—	—	—
Indicator minor Lesser Honeyguide	1	0	—	—	—
Lanius collaris Common Fiscal	4	1	1	—	—
Lybius torquatus Black-collared Barbet	2	0	—	—	—
Motacilla capensis Cape Wagtail	22	8	—	—	8
Nectarinia famosa Malachite Sunbird	3	1	—	1	—
Passer domesticus House Sparrow	184	23	1	21	1
Passer melanurus Cape Sparrow [3]	183	89	75	21	1
Ploceus capensis Cape Weaver [4]	1189	405	302	120	20
Ploceus velatus Southern Masked Weaver [5]	511	29	12	13	6
Prinia maculosa Karoo Prinia	2	1	—	1	—
Pycnonotus capensis Cape Bulbul	19	4	—	4	—
Riparia paludicola Brown-throated Martin	1	0	—	—	—
Serinus canicollis Cape Canary	8	1	—	—	1
Sigelus silens Fiscal Flycatcher	1	0	—	—	—
Spreo bicolor Pied Starling	22	1	—	1	—
Streptopelia semitorquata Red-eyed Dove	11	1	1	—	—
Streptopelia senegalensis Laughing Dove [6]	99	66	59	13	—
Sturnus vulgaris Common Starling	33	2	—	2	—
Sylvietta rufescens Long-billed Crombec	1	0	—	—	—
Tricholaema leucomelas Acacia Pied Barbet	10	0	—	—	—
Vidua macroura Pin-tailed Whydah	5	0	—	—	—
Zosterops virens Cape White-eye	29	4	—	3	1
Total	**2 944**	**742**	**527**	**233**	**41**
Percentage	—	**25.2**	**71.0**	**31.4**	**5.5**

[1] double infections = 1
[2] double infections = 5
[3] double infections = 8
[4] double infections = 37
[5] double infections = 2
[6] double infections = 6

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Appendix 5 Summary of prevalence data of haemosporidian parasites of birds trapped at **Koeberg Nature Reserve** from March 1994 to April 1995. *Haem.=Haemoproteus; Leuco=Leucocytozoon; Plasm=Plasmodium.*

Species	Total individuals		Individuals with		
	Examined	Infected	*Haem*	*Leuco*	*Plasm*
Apalis thoracica Bar-throated Apalis	2	0	—	—	—
Cisticola subruficapilla Grey-backed Cisticola	1	0	—	—	—
Colius striatus Speckled Mousebird	1	0	—	—	—
Cossypha caffra Cape Robin Chat	23	0	—	—	—
Crithagra flaviventris Yellow Canary	3	0	—	—	—
Emberiza capensis Cape Bunting	5	0	—	—	—
Estrilda astrild Common Waxbill	2	0	—	—	—
Euplectes orix Southern Red Bishop	3	0	—	—	—
Lanius collaris Common Fiscal	2	0	—	—	—
Parisoma subcaeruleum Chestnut-vented Tit Babbler	2	1	—	—	1
Ploceus capensis Cape Weaver	3	0	—	—	—
Prinia maculosa Karoo Prinia	2	0	—	—	—
Pycnonotus capensis Cape Bulbul [1]	6	4	2	4	—
Riparia paludicola Brown-throated Martin	1	0	—	—	—
Sigelus silens Fiscal Flycatcher	1	0	—	—	—
Tricholaema leucomelas Acacia Pied Barbet	1	0	—	—	—
Vidua macroura Pin-tailed Whydah	2	0	—	—	—
Zosterops virens Cape White-eye	11	2	2	—	—
Total	**71**	**7**	**4**	**4**	**1**
Percentage	—	**9.8**	**57.1**	**57.1**	**14.2**

[1] double infections = 2
[2] double infections = 2

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Appendix 6 Summary of prevalence data of haemosporidian parasites of birds trapped at **Kirstenbosch National Botanical Garden** from March 1993 to April 1995. *Haem.=Haemoproteus; Leuco=Leucocytozoon; Plasm=Plasmodium.*

Species	Total individuals		Individuals with		
	Examined	Infected	*Haem*	*Leuco*	*Plasm*
Andropadus importunus Sombre Greenbul	27	4	—	4	—
Anthobaphes violacea Orange-breasted Sunbird	58	0	—	—	—
Batis capensis Cape Batis	5	0	—	—	—
Chrysococcyx klaas Klaas's Cuckoo	1	0	—	—	—
Cinnyris chalybeus Southern Double-collared Sunbird	16	0	—	—	—
Colius colius White-backed Mousebird	1	0	—	—	—
Colius striatus Speckled Mousebird	5	0	—	—	—
Cossypha caffra Cape Robin Chat	34	1	—	1	—
Crithagra scotops Forest Canary	11	0	—	—	—
Crithagra sulphuratus Brimstone Canary	11	2	—	2	—
Crithagra totta Cape Siskin	5	0	—	—	—
Laniarius ferrugineus Southern Boubou [1]	7	1	1	—	1
Lanius collaris Common Fiscal	1	0	—	—	—
Muscicapa adusta African Dusky Flycatcher	1	0	—	—	—
Nectarinia famosa Malachite Sunbird	1	0	—	—	—
Ploceus capensis Cape Weaver	3	1	—	1	—
Prinia maculosa Karoo Prinia	9	0	—	—	—
Promerops cafer Cape Sugarbird	109	14	—	14	—
Pycnonotus capensis Cape Bulbul	2	0	—	—	—
Serinus canicollis Cape Canary	1	0	—	—	—
Sphenoeacus afer Cape Grassbird	6	0	—	—	—
Streptopelia capicola Cape Turtle Dove [2]	3	2	2	1	—
Streptopelia senegalensis Laughing Dove	1	0	—	—	—
Sturnus vulgaris Common Starling	4	0	—	—	—
Turdus olivaceus Olive Thrush	10	7	1	6	—
Zosterops virens Cape White-eye [3]	62	48	26	43	2
Total	**394**	**80**	**30**	**72**	**3**
Percentage	—	**20.3**	**37.5**	**90.0**	**3.7**

[1] double infections = 1
[2] double infections = 1
[3] double infections = 23

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Appendix 7 Summary of prevalence data of haemosporidian parasites of birds trapped at **12 Roseberry Road, Mowbray** from April 1993 to March 1994. *Haem.*=*Haemoproteus*; *Leuco*=*Leucocytozoon*; *Plasm*=*Plasmodium*.

Species	Total individuals		Individuals with		
	Examined	Infected	*Haem*	*Leuco*	*Plasm*
Columba guinea Speckled Pigeon	7	1	1	—	—
Gallinula chloropus Common Moorhen	1	0	—	—	—
Passer melanurus Cape Sparrow	3	0	—	—	—
Streptopelia capicola Cape Turtle Dove	5	0	—	—	—
Streptopelia semitorquata Redeyed Dove	2	0	—	—	—
Streptopelia senegalensis Laughing Dove	222	36	31	5	—
Sturnus vulgaris Common Starling	29	3	—	3	—
Total	**269**	**40**	**32**	**8**	**—**
Percentage	**—**	**14.8**	**80.0**	**20.0**	**0**

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Appendix 8 Summary of prevalence data of haemosporidian parasites of birds trapped at **Patryskraal** (Bredasdorp District) from May 1993 to February 1995. *Haem.=Haemoproteus; Leuco=Leucocytozoon; Plasm=Plasmodium.*

Species	Total individuals		Individuals with		
	Examined	Infected	*Haem*	*Leuco*	*Plasm*
Columba guinea Speckled Pigeon	2	0	—	—	
Cossypha caffra Cape Robin Chat	5	0	—	—	—
Crithagra albogularis White-throated Canary	2	0	—	—	—
Euplectes orix Southern Red Bishop [1]	36	1	1	1	—
Hirundo cucullata Greater Stripped Swallow	1	0	—	—	—
Hirundo fuligula Rock Martin	2	0	—	—	
Lanius collaris Common Fiscal	5	0	—	—	—
Nectarinia famosa Malachite Sunbird	3	3	2	1	—
Passer domesticus House Sparrow	17	0	—	—	—
Passer melanurus Cape Sparrow	65	2	—	2	—
Ploceus capensis Cape Weaver	40	2	—	1	1
Pternistis capensis Cape Spurfowl	1	1	—	—	—
Sigelus silens Fiscal Flycatcher	2	0	—	—	—
Spreo bicolor Pied Starling	4	0	—	—	—
Streptopelia capicola Cape Turtle Dove	1	0	—	—	—
Streptopelia senegalensis Laughing Dove	13	1	1	—	—
Sturnus vulgaris Common Starling	16	0	—	—	—
Telophorus zeylonus Bokmakierie	1	0	—	—	—
Upupa africana African Hoopoe	1	1	—	1	—
Zosterops virens Cape White-eye	1	0	—	—	—
Total	**218**	**10**	**4**	**6**	**1**
Percentage	**—**	**4.5**	**40.0**	**60.0**	**10.0**

[1] double infections = 1

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Appendix 9 Summary of prevalence data of haemosporidian parasites of birds trapped at **Rondevlei Nature Reserve** from February 1993 to January 1995. *Haem.=Haemoproteus; Leuco=Leucocytozoon; Plasm=Plasmodium.*

Species	Total individuals		Individuals with		
	Examined	Infected	*Haem*	*Leuco*	*Plasm*
Acrocephalus baeticatus African Reed Warbler	14	2	2	—	—
Acrocephalus gracilirostris Lesser Swamp Warbler	72	1	—	—	1
Alcedo cristata Malachite Kingfisher	4	0	—	—	—
Apalis thoracica Bar-throated Apalis	1	0	—	—	—
Bradypterus baboecala Little Rush Warbler	11	0	—	—	—
Centropus superciliosus White-browed Coucal	1	0	—	—	—
Chrysococcyx klaas Klaas's Cuckoo	1	0	—	—	—
Cinnyris chalybeus Southern Double-collared Sunbird	37	0	—	—	—
Cisticola subruficapilla Grey-backed Cisticola	3	0	—	—	—
Cisticola tinniens Levaillant's Cisticola	16	0	—	—	—
Colius colius White-backed Mousebird	28	0	—	—	—
Colius striatus Speckled Mousebird	6	0	—	—	—
Cossypha caffra Cape Robin Chat	21	0	—	—	—
Crithagra flaviventris Yellow Canary	11	0	—	—	—
Crithagra sulphuratus Brimstone Canary	21	1	1	—	—
Estrilda astrild Common Waxbill	6	0	—	—	—
Euplectes capensis Yellow Bishop	1	0	—	—	—
Euplectes orix Southern Red Bishop	1	0	—	—	—
Hirundo rustica Barn Swallow	3	0	—	—	—
Indicator minor Lesser Honeyguide	2	0	—	—	—
Laniarius ferrugineus Southern Boubou	2	0	—	—	—
Lanius collaris Common Fiscal	3	0	—	—	—
Lybius torquatus Black-collared Barbet	1	0	—	—	—
Passer melanurus Cape Sparrow	10	0	—	—	—
Ploceus capensis Cape Weaver	121	6	1	5	—
Ploceus velatus Southern Masked Weaver	44	1	—	1	—
Prinia maculosa Karoo Prinia	24	0	—	—	—
Pycnonotus capensis Cape Bulbul	201	18	1	17	—
Serinus canicollis Cape Canary	1	0	—	—	—
Sphenoeacus afer Cape Grassbird	4	0	—	—	—
Streptopelia capicola Cape Turtle Dove	3	0	—	—	—
Streptopelia senegalensis Laughing Dove	6	2	2	—	—
Sturnus vulgaris Common Starling	2	0	—	—	—
Sylvietta rufescens Long-billed Crombec	1	0	—	—	—
Tricholaema leucomelas Acacia Pied Barbet	2	0	—	—	—
Zosterops virens Cape White-eye [1]	306	42	5	40	—
Total	**991**	**73**	**12**	**63**	**1**
Percentage	—	**7.3**	**16.4**	**86.3**	**1.3**

[1] double infections = 3

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Appendix 10 Summary of prevalence data of haemospondian parasites of birds trapped at **Tygerberg Nature Reserve** from February 1993 to June 1995. *Haem.=Haemoproteus; Leuco=Leucocytozoon; Plasm=Plasmodium.*

Species	Total individuals		Individuals with		
	Examined	Infected	*Haemo*	*Leucoc*	*Plasm*
Alcedo cristata Malachite Kingfisher	1	0	—	—	—
Acrocephalus baeticatus African Reed Warbler	14	2	2	—	—
Acrocephalus gracilirostris Lesser Swamp Warbler	7	0	—	—	—
Andropadus importunus Somber Greenbul	10	2	—	2	—
Apalis thoracica Bar-throated Apalis	9	1	—	1	—
Batis capensis Cape Batis	2	0	—	—	—
Bradypterus baboecala Little Rush Warbler	25	0	—	—	—
Centropus superciliosus White-browed Coucal	1	1	—	1	—
Chrysococcyx klaas Klaas's Cuckoo	2	0	—	—	—
Cinnyris chalybeus Southern Double-collared Sunbird	49	0	—	—	—
Cisticola subruficapilla Grey-backed Cisticola	2	0	—	—	—
Cisticola tinniens Levaillant's Cisticola	27	0	—	—	—
Colius colius White-backed Mousebird	36	1	—	1	—
Colius striatus Speckled Mousebird	9	0	—	—	—
Cossypha caffra Cape Robin Chat	82	1	—	1	—
Crithagra albogularis White-throated Canary	15	6	—	6	—
Serinus canicollis Cape Canary	26	1	1	—	—
Crithagra flaviventris Yellow Canary	3	3	—	3	—
Cercotrichas coryphaeus Karoo Scrub Robin	1	0	—	—	—
Estrilda astrild Common Waxbill	138	1	—	—	1
Euplectes capensis Yellow Bishop [1]	147	55	7	50	—
Euplectes orix Southern Red Bishop [2]	40	24	13	15	—
Hirundo cucullata Greater Stripped Swallow	23	1	—	1	—
Indicator minor Lesser Honeyguide	15	0	—	—	—
Ixobrychus minutus Little Bittern	1	0	—	—	—
Lagonosticta rubricata African Firefinch	1	0	—	—	—
Lanius collaris Common Fiscal	13	0	—	—	—
Megaceryle maximus Giant Kingfisher	1	0	—	—	—
Motacilla capensis Cape Wagtail	1	0	—	—	—
Muscicapa adusta African Dusky Flycatcher	3	0	—	—	—
Nectarnia famosa Malachite Sunbird	11	0	—	—	—
Passer melanurus Cape Sparrow [3]	125	30	2	30	1
Ploceus capensis Cape Weaver [4]	415	60	7	52	3
Ploceus velatus Southern Masked Weaver	87	7	—	6	1
Prinia maculosa Karoo Prinia	53	3	1	2	—
Promerops cafer Cape Sugarbird	8	-	—	—	—
Pycnonotus capensis Cape Bulbul [5]	78	37	1	37	—
Riparia paludicola Brown-throated Martin	1	-	—	—	—
Sigelus silens Fiscal Flycatcher	12	0	—	—	—
Crithagra sulphuratus Brimstone Canary	5	0	—	—	—
Sphenocacus afer Cape Grassbird	4	1	—	1	—
Streptopelia capicola Cape Turtle Dove [6]	15	10	5	6	—
Streptopelia semitorquata Red-eyed Dove	2	0	—	—	—
Streptopelia senegalensis Laughing Dove [7]	36	20	11	14	—
Sturnus vulgaris Common Starling	7	1	—	1	—
Sylvia borin Garden Warbler	1	0	—	—	—
Sylvietta rufescens Long-billed Crombec	2	0	—	—	—
Telophorus zeylonus Bokmakierie	4	0	—	—	—
Terpsiphone viridis African Paradise Flycatcher	4	0	—	—	—
Tricholaema leucomelas Acacia Pied Barbet	14	1	—	1	—
Turdus olivaceus Olive Thrush	33	7	—	7	—
Urocolius indicus Red-faced Mousebird	26	1	—	1	—
Vidua macroura Pin-tailed Whydah	5	1	—	1	—
Zosterops virens Cape White-eye [8]	900	438	133	360	—
Total	**2 551**	**716**	**183**	**600**	**6**
Percentage	—	**28.0**	**25.5**	**83.7**	**0.8**

[1] double infections = 2; [2] double infections = 4; [3] double infections = 3; [4] double infections = 2; [5] double infections = 1; [6] double infections = 1; [7] double infections = 5; [8] double infections = 55

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Appendix 11 Monthly prevalence of avian haemosporidian parasites of birds trapped at **Bettys Bay** from September 1993 to December 1994. Figures in parentheses represent percentages of birds infected.

Year	Month	Total examined	Total infected	Average max (°C)	Average min (°C)	Average rain (mm)	Average wind speed (ms^{-1})	Prevailing wind direction
1993	September	13	5(38.4)	19.1	11.5	23.3	27.9	W/N
	October	**112**	**14**(12.5)	20.7	13.0	42.2	21.7	SW/E
	November	107	22(20.5)	23.1	15.1	50.1	22.5	SW
	December	**112**	**31**(27.6)	23.7	15.9	112.5	19.8	SSW
1994	January	18	3(16.6)	23.8	16.2	21.1	18.3	S
	February	0	—	23.3	15.6	27.9	17.3	S/E
	March	0	—	22.3	15.5	89.0	18.3	S
	April	25	10(40.0)	21.4	13.9	38.7	17.6	S
	May	0	—	17.7	10.7	89.2	21.3	SW/E
	June	0	—	16.4	10.3	370.9	26.7	W
	July	0	—	17.6	9.5	120.0	26.8	W
	August	0	—	17.9	10.3	67.7	19.4	S/W
	September	0	—	19.6	11.9	75.4	21.3	SW/E
	October	0	—	21.3	13.3	74.5	17.6	S
	November	24	5(20.8)	22.8	15.0	25.5	21.1	SW/E
	December	27	8(29.6)	23.4	15.2	83.5	19.8	SSW

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Appendix 12 Monthly prevalence of avian haemosporidian parasites of birds trapped at **Durbanville Nature Reserve** from December 1992 to April 1995. Figures in parentheses represent percentages of birds infected.

Year	Month	Total examined	Total infected	Average max (°C)	Average min (°C)	Average rain (mm)	Average wind speed (ms^{-1})	Prevailing wind direction
1992	December	26	9(34.6)	25.9	14.8	4.1	24.0	WSW
1993	January	7	2(28.5)	26.7	16.8	4.7	21.0	SW/E
	February	0	—	26.3	14.3	38.0	24.5	WSW
	March	80	8(10.0)	26.2	15.5	3.8	22.5	SW
	April	39	8(20.5)	21.4	12.3	178.9	29.0	WNW
	May	108	15(13.8)	18.6	11.0	136.9	21.1	SW/E
	June	81	8(9.8)	17.7	8.3	78.5	22.5	SW
	July	26	2(7.6)	18.3	9.3	142.5	30.5	NW
	August	14	8(57.1)	18.6	8.3	63.2	24.2	SW/W
	September	19	4(21.0)	19.6	10.1	9.1	25.0	WSW
	October	0	—	22.2	11.2	3.8	21.5	SW/F
	November	32	7(28.8)	24.7	13.4	2.3	24.0	SW/W
	December	41	6(14.6)	25.7	15.1	29.4	24.5	WSW
1994	January	48	3(6.2)	26.9	15.5	14.6	23.5	SW/W
	February	60	11(18.3)	28.0	15.4	0.7	23.5	SW/W
	March	118	8(6.7)	25.9	15.4	3.6	21.5	SW/F
	April	55	4(7.2)	24.3	12.8	35.0	22.5	SW
	May	67	8(11.9)	19.3	9.1	39.5	22.5	SW
	June	22	3(13.6)	17.2	8.3	229.4	35.5	N
	July	25	3(12.0)	17.5	6.8	68.9	27.5	W
	August	14	3(21.4)	18.1	7.0	30.9	22.5	SW
	September	6	0(0)	19.7	10.1	39.5	22.5	SW
	October	9	2(22.2)	22.6	11.0	14.1	19.5	S/W
	November	16	4(25.0)	23.5	12.5	7.9	23.0	SW
	December	11	2(18.1)	26.2	14.5	3.4	23.5	SW/W
1995	January	15	3(20.0)	26.8	16.0	9.9	25.5	W/S
	February	6	1(16.6)	27.8	16.3	1.0	21.5	SW/F
	March	25	6(24.0)	27.0	14.6	5.8	21.0	SW/E
	April	53	6(11.3)	22.4	10.6	17.9	23.5	SW/W

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Appendix 13 Monthly prevalence of avian haemosporidian parasites of birds trapped at **Glencairn** from September 1993 to December 1995. Figures in parentheses represent percentages of birds infected.

Year	Month	Total examined	Total infected	Average max (°C)	Average min (°C)	Average rain (mm)	Average wind speed (ms^{-1})	Prevailing wind direction
1993	May	11	1(9.0)	18.6	11.0	112.8	21.1	SW/E
	June	19	1(5.2)	17.7	8.3	106.4	22.5	SW
	July	15	3(20.0)	18.3	9.3	116.2	30.5	NW
	August	21	2(9.5)	18.6	8.3	68.5	24.2	SW/W
	September	22	7(31.8)	19.6	10.1	10.2	25.0	WSW
	October	41	6(14.6)	22.2	11.2	4.0	21.5	SW/E
	November	19	2(10.5)	24.7	13.4	17.2	24.0	SW/W
	December	12	0(0.0)	25.7	15.1	24.0	24.5	WSW
1994	January	22	5(22.7)	26.9	15.5	10.3	23.5	SW/W
	February	12	1(8.3)	28.0	15.4	0.6	23.5	SW/W
	March	21	0(0.0)	25.9	15.4	3.2	21.5	SW/E
	April	20	2(10.0)	24.3	12.8	22.5	22.5	SW
	May	30	8(26.6)	19.3	9.1	60.2	22.5	SW
	June	10	0(0.0)	17.2	8.3	301.5	35.5	N
	July	9	0(0.0)	17.5	6.8	92.5	27.5	W
	August	0	—	18.1	7.0	43.6	22.5	SW
	September	0	—	19.7	10.1	51.1	22.5	SW
	October	5	2(40.0)	22.6	11.0	19.0	19.5	S/W
	November	24	6(25.0)	23.5	12.5	29.4	23.0	SW
	December	7	2(28.5)	26.2	14.5	1.9	23.5	SW/W
1995	January	26	9(34.6)	26.8	16.0	6.0	25.5	W/S
	February	19	5(26.3)	27.8	16.3	6.4	21.5	SW/E
	March	15	4(27)	27.0	14.6	6.2	21.0	SW/E
	April	8	2(26.6)	22.4	10.6	14.5	23.5	SW/W
	May	1	0(0.0)	20.9	10.4	66.3	25.5	W/S
	June	4	0(0.0)	18.2	7.7	91.4	28.5	W/N
	July	2	0(0.0)	15.7	6.9	132.5	132.5	W/S
	August	7	1(14.2)	17.6	7.8	83.3	83.3	SW
	September	3	1(33.03)	19.4	9.9	30.1	30.1	SW

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Appendix 14 Monthly prevalence of avian haemosporidian parasites of birds trapped at **Goedeontmoeting** from March 1993 to May 1995. Figures in parentheses represent percentages of birds infected.

Year	Month	Total examined	Total infected	Average max (°C)	Average min (°C)	Average rain (mm)	Average wind speed (ms^{-1})	Prevailing wind direction
1993	March	34	7(20.5)	30.7	14.9	3.0	20.3	SSW
	April	69	25(36.6)	23.6	11.6	148.8	10.0	E/S
	May	43	0(0.0)	19.0	8.2	63.6	36.0	N
	June	202	48(23.7)	17.8	6.6	0.0	17.8	S
	July	233	61(26.1)	18.8	8.0	142.8	18.4	S
	August	202	67(33.1)	19.7	6.8	53.2	15.9	SSE
	September	135	63(46.6)	22.2	8.2	4.0	15.7	SSE
	October	61	20(32.7)	25.5	10.1	0.8	13.5	SE
	November	0	—	28.0	11.6	4.2	15.6	SSE
	December	139	45(32.3)	29.0	13.7	16.0	17.7	S
1994	January	218	80(36.6)	31.1	15.2	7.6	24.5	WSW
	February	179	46(25.6)	32.3	14.9	13.0	16.4	S/E
	March	188	32(17.0)	29.3	14.5	0.0	14.8	SE/E
	April	146	29(19.8)	26.8	12.0	41.0	15.7	SSE
	May	255	33(12.9)	21.3	6.9	21.4	15.6	SSE
	June	123	19(15.4)	17.6	6.9	166.0	23.6	SW/W
	July	113	20(17.6)	18.1	4.5	38.0	18.1	S
	August	149	30(20.1)	19.3	5.5	16.0	13.3	SE
	September	165	52(31.5)	21.3	8.4	49.0	15.1	SE/S
	October	82	23(28.0)	25.6	10.0	0.2	16.5	S/E
	November	21	8(38.0)	25.7	11.4	2.4	14.8	SE/S
	December	0	—	29.5	13.2	7.6	17.8	S
1995	January	38	12(31.5)	30.7	14.8	7.0	17.3	S/E
	February	0	—	32.5	15.1	2.0	15.3	SSE
	March	23	7(30.4)	30.0	14.8	15.4	0	0
	April	98	13(13.2)	25.7	9.3	6.2	17.2	S/E
	May	28	2(7.1)	23.7	9.5	40.6	15.4	SSE

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Appendix 15 Monthly prevalence of avian haemosporidian parasites of birds trapped at **Koeberg Nature Reserve** from March 1994 to April 1994. Figures in parentheses represent percentages of birds infected.

Year	Month	Total examined	Total infected	Average max (°C)	Average min (°C)	Average rain (mm)	Average wind speed (ms^{-1})	Prevailing wind direction
1994	March	45	6(13.3)	22.9	15.6	9.2	24.1	SW/W
	April	26	1(3.8)	22.2	14.4	13.6	30.5	NW/N

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Appendix 16 Monthly prevalence of avian haemosporidian parasites of birds trapped at **Kirstenbosch National Botanical Gardens** from March 1993 to April 1994. Figures in parentheses represent percentages of birds infected.

Year	Month	Total examined	Total infected	Average max (°C)	Average min (°C)	Average rain (mm)	Average wind speed (ms^{-1})	Prevailing wind direction
1993	March	44	4(9.0)	25.0	15.5	23.1	23.1	SW
	April	117	17(14.5)	20.5	12.3	218.8	218.8	WNW
	May	24	10(41.6)	18.4	10.5	238.7	21.1	SW/E
	June	37	18(48.6)	17.6	9.0	180.3	22.5	SW
	July	39	11(28.2)	18.4	9.8	424.5	30.5	NW
	August	37	5(13.5)	18.5	8.2	190.1	24.2	SW/W
	September	18	4(22.2)	19.7	9.8	20.8	25.0	WSW
	October	0	—	21.2	11.8	22.5	21.5	SW/E
	November	0	—	22.6	13.8	21.8	24.0	SW/W
	December	0	—	23.1	14.9	36.2	24.5	WSW
1994	January	18	1(5.5)	24.2	15.9	35.8	23.5	SW/W
	February	16	2(12.5)	25.6	15.9	5.5	23.5	SW/W
	March	37	7(18.9)	24.5	15.6	25.4	21.5	SW/E
	April	7	1(14.2)	23.1	13.7	74.8	22.5	SW

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Appendix 17 Monthly prevalence of avian haemosporidian parasites of birds trapped at **Mowbray, 12 Roseberry Road**, from April 1993 to March 1994. Figures in parentheses represent percentages of birds infected.

Year	Month	Total examined	Total infected	Average max (°C)	Average min (°C)	Average rain (mm)	Average wind speed (ms^{-1})	Prevailing wind direction
1993	April	16	6(37.5)	21.4	12.3	178.9	29.0	WNW
	May	22	3(13.6)	18.6	11.0	136.9	21.1	SW/E
	June	19	2(10.5)	17.7	8.3	78.5	22.5	SW
	July	11	0(0.0)	18.3	9.3	142.5	30.5	NW
	August	25	3(12.0)	18.6	8.3	63.2	24.2	SW/W
	September	20	5(25.0)	19.6	10.1	9.1	25.0	WSW
	October	35	7(20.0)	22.2	11.2	3.8	21.5	SW/E
	November	34	2(5.8)	24.7	13.4	2.3	24.0	SW/W
	December	51	11(21.5)	25.7	15.1	29.4	24.5	WSW
1994	January	10	0(0.0)	26.9	15.5	14.6	23.5	SW/W
	February	13	1(7.6)	28.0	15.4	0.7	23.5	SW/W
	March	13	0(0.0)	25.9	15.4	3.6	21.5	SW/E

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Appendix 18 Monthly prevalence of avian haemosporidian parasites of birds trapped at **Patryskraal (Bredasdorp District)** from May 1993 to February 1995. Figures in parentheses represent percentages of birds infected.

Year	Month	Total examined	Total infected	Average max (°C)	Average min (°C)	Average rain (mm)	Average wind speed (ms^{-1})	Prevailing wind direction
1993	May	18	0(0.0)	18.6	12.0	51.8	24.5	WSW
	June	22	3(13.6)	17.6	9.6	42.6	21.2	SW/E
	July	0	—	17.8	10.3	131.2	23.4	SW/W
	August	26	1(3.8)	17.3	9.7	39.0	17.9	S
	September	21	0(0)	18.6	12.0	25.0	21.5	SW/E
	October	29	1(3.4)	19.3	13.7	4.6	20.9	SW/E
	November	0	—	21.9	16.1	5.8	23.2	SW
	December	0	—	22.6	16.9	26.6	22.1	SW
1994	January	0	—	24.0	18.0	9.4	18.0	S
	February	0	—	24.0	18.4	7.2	16.9	S/E
	March	0		22.6	17.5	5.2	16.9	S/E
	April	0		21.4	15.2	40.0	18.3	S
	May	0	—	18.9	10.9	46.4	17.6	S
	June	0	—	17.2	9.5	137.6	25.0	WSW
	July	0	—	17.5	8.0	32.6	21.2	SW/E
	August	18	2(11.1)	16.7	10.0	32.0	17.4	S
	September	0	—	18.8	12.0	32.0	21.0	SW/E
	October	0		20.7	13.6	33.8	18.3	S
	November	0		22.3	14.3	6.0	23.4	SW
	December	0		23.3	16.8	121.4	20.3	SSW
1995	January	41	2(4.8)	24.3	18.1	8.0	20.3	SSW
	February	43	1(2.3)	24.4	19.6	6.8	20.9	S

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Appendix 19 Monthly prevalence of avian haemosporidian parasites of birds trapped at **Rondevlei Nature Reserve** from June 1993 to January 1995. Figures in parentheses represent percentages of birds infected.

Year	Month	Total examined	Total infected	Average max (°C)	Average min (°C)	Average rain (mm)	Average wind speed (ms^{-1})	Prevailing wind direction
1993	June	115	6(5.2)	17.7	8.3	105.8	22.5	SW
	July	212	12(5.6)	18.3	9.3	209.7	30.5	NW
	August	159	25(15.7)	18.6	8.3	91.5	24.2	SW/W
	September	59	6(10.1)	19.6	10.1	12.5	25.0	WSW
	October	0	—	22.2	11.2	2.9	21.5	SW/E
	November	83	6(7.2)	24.7	13.4	11.5	24.0	SW/W
	December	49	3(6.1)	25.7	15.1	21.9	24.5	WSW
1994	January	39	2(5.1)	26.9	15.5	10.8	23.5	SW/W
	February	45	0(0.0)	28.0	15.4	0.9	23.5	SW/W
	March	19	0(0.0)	25.9	15.4	12.7	21.5	SW/E
	April	0	—	24.3	12.8	22.0	22.5	SW
	May	12	0(0.0)	19.3	9.1	45.4	22.5	SW
	June	0	—	17.2	8.3	344.0	35.5	N
	July	0	—	17.5	6.8	76.0	27.5	W
	August	82	4(4.8)	18.1	7.0	42.4	22.5	SW
	September	57	6(10.5)	19.7	10.1	51.8	22.5	SW
	October	19	2(10.5)	22.6	11.0	8.9	19.5	S/W
	November	0	—	23.5	12.5	7.2	23.0	SW
	December	0	—	26.2	14.5	6.9	23.5	SW/W
1995	January	41	1(2.4)	26.8	16.0	7.9	25.5	W/S

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Appendix 20 Monthly prevalence of avian haemosporidian parasites of birds trapped at **Tygerberg Nature Reserve** from February 1993 to June 1995. Figures in parentheses represent percentages of birds infected.

Year	Month	Total examined	Total infected	Average max (°C)	Average min (°C)	Average rain (mm)	Average wind speed (ms^{-1})	Prevailing wind direction
1993	February	39	13(44)	26.3	14.3	38.0	24.5	WSW
	March	38	10(34)	26.2	15.5	3.8	22.5	SW
	April	0	—	21.4	12.3	178.9	29.0	WNW
	May	0	—	18.6	11.0	136.9	21.1	SW/E
	June	0	—	17.7	8.3	78.5	22.5	SW
	July	0	—	18.3	9.3	142.5	30.5	NW
	August	0	—	18.6	8.3	63.2	24.2	SW/W
	September	84	25(33)	19.6	10.1	9.1	25.0	WSW
	October	1	0(0.0)	22.2	11.2	3.8	21.5	SW/E
	November	0	—	24.7	13.4	2.3	24.0	SW/W
	December	0	—	25.7	15.1	29.4	24.5	WSW
1994	January	0	—	26.9	15.5	14.6	23.5	SW/W
	February	261	96(37)	28.0	15.4	0.7	23.5	SW/W
	March	206	59(31)	25.9	15.4	3.6	21.5	SW/E
	April	237	85(38)	24.3	12.8	35.0	22.5	SW
	May	86	7(9)	19.3	9.1	39.5	22.5	SW
	June	75	11(21)	17.2	8.3	229.4	35.5	N
	July	59	13(31)	17.5	6.8	68.9	27.5	W
	August	137	12(20)	18.1	7.0	30.9	22.5	SW
	September	205	53(28)	19.7	10.1	39.5	22.5	SW
	October	307	109(37)	22.6	11.0	14.1	19.5	SW
	November	64	22(42)	23.5	12.5	7.9	23.0	SW
	December	106	34(37)	26.2	14.5	3.4	23.5	SW/W
1995	January	0	—	26.8	16.0	9.9	25.5	W/S
	February	160	56(34)	27.8	16.3	1.0	21.5	SW/E
	March	175	51(30)	27.0	14.6	5.8	21.0	SW/E
	April	218	41 (20)	22.4	10.6	17.9	23.5	SW/W
	May	66	19(32)	20.9	10.4	68.0	25.5	W/S
	June	7	0(0.0)	18.2	7.7	109.8	28.5	W/N

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Appendix 22 Monthly mass (g) and Wing length (mm) of *Ploceus capensis* Cape Weaver by sex and age class trapped at Goedeontmoeting from March 1993 to January 1995. Uninfect=Uninfected; Infect=Infected; ♂=males, ♀=females; A=adult

Year	Month	Ring number	Sex	Age	Mass		Wing Length		Avian haemosporidian parasites
					Uninfect	Infect	Uninfect	Infect	
1993	March	CC 08314	♂	A	49	—	95	—	—
		CC 08315	♀	A	42	—	80	—	—
		CC 08316	♂	A	48	—	96	—	—
		CC 08317	♂	A	—	41	—	91	*Leucocytozoon bouffardi*
		CC 08318	♀	A	41	—	90	—	—
		CC 08319	♂	A	46	—	92	—	—
		CC 08320	♂	A	—	51	—	93	*L. bouffardi*
		CC 08321	♂	A	—	47	—	90	*L. bouffardi*
		CC 08323	♂	A	49	—	93	—	—
		BD 06544	♀	A	39	—	89	—	—
		BD 06545	♀	A	41	—	86	—	—
		BD 06546	♀	A	40	—	88	—	—
		CV 03402	♂	A	48	—	96	—	—
1993	April	BD 06554	♀	A	—	39	—	89	*Haemoproteus queleae*
		BD 06345	♂	A	—	47	—	92	*L. bouffardi, H. quelcae*
		BD 06375	♂	A	47	—	93	—	—
		BC 17056	♂	A	43	—	96	—	—
		BC 20555	♂	A	40	—	94	—	—
		BB 99933	♀	A	—	39	—	85	*L. bouffardi, H. quelcae*
		CC 04412	♂	A	—	50	—	94	*L. bouffardi*
		CC 08330	♂	A	45	—	91	—	—
		CC 08331	♂	A	—	49	—	92	*L. bouffardi*
		CC 08329	♂	A	48	—	94	—	—
		CV 02370	♂	A	—	50	—	94	*L. bouffardi*
		CV 03319	♂	A	52	—	94	—	—
		CC 08193	♂	A	—	47	—	92	*L. bouffardi, H. queleae*
		CC 08320	♂	A	—	51	—	93	*H. quelcae*
		CC 04340	♂	A	53	—	96	—	—
		CC 08337	♂	A	—	46	—	92	*H. quelcae*
		CC 08338	♂	A	—	54	—	97	*H. queleae*
		CC 08339	♂	A	45	—	91	—	—
1993	May	BC 17160	♂	A	48	—	95	—	—
		BC 17014	♂	A	47	—	95	—	—
		BC 20495	♂	A	40	—	89	—	—
		CC 08375	♂	A	47	—	91	—	—
		CC 08383	♂	A	43	—	94	—	—
		CC 08384	♂	A	45	—	91	—	—
		BB 99973	♂	A	—	47	—	92	*H. quelcae*
		BD 06304	♂	A	47	—	92	—	—
		BC 20464	♂	A	46	—	90	—	—
		BC 03896	♂	A	—	47	—	94	—
		CC 03333	♂	A	42	—	87	—	—
		CC 08082	♂	A	51	—	93	—	—
		CC 08120	♂	A	46	—	93	—	—
1993	June	CC 08398	♂	A	—	42	—	94	*H. queleae*
		CC 08399	♂	A	44	—	95	—	—
		CC 08400	♂	A	46	—	92	—	—
		CC 08401	♂	A	44	—	92	—	—
		CC 08402	♂	A	—	44	—	92	*H. quelcae*

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Year	Month	Ring number	Sex	Age	Mass		Wing Length		Avian haemosporidian parasites
					Uninfect	Infect	Uninfect	Infect	
1993	June	CC 08403	♂	A	—	48	—	95	*H. queleae*
		CC 08404	♂	A	44	—	91	—	—
		CC 08406	♂	A	47	—	94	—	—
		CC 08408	♂	A	43	—	98	—	—
		CC 08409	♂	A	46	—	93	—	—
		CC 08410	♀	A	39	—	86	—	—
		CC 08411	♂	A	48	—	93	—	—
		CC 08412	♂	A	49	—	90	—	—
		CC 08413	♂	A	—	51	—	93	*P. vaughani*
		CV 03395	♂	A	49	—	98	—	—
		CV 02329	♂	A	48	—	96	—	—
		BB 99872	♂	A	49	—	93	—	—
		BD 04921	♀	A	40	—	86	—	—
		CC 08194	♂	A	46	—	93	—	—
		CV 03389	♂	A	47	—	96	—	—
		BD 06360	♂	A	46	—	90	—	—
		BD 12076	♀	A	—	37	—	85	*H. queleae*
		BD 12082	♀	A	37	—	83	—	—
		BD 12084	♀	A	40	—	87	—	—
		BD 12085	♂	A	46	—	90	—	—
		BD 12090	♀	A	37	—	85	—	—
		CC 08428	♂	A	43	—	92	—	—
		CC 08430	♂	A	49	—	92	—	—
		CC 08433	♂	A	—	46	—	92	*L. bouffardi*
		CC 08435	♂	A	36	—	86	—	—
		CC 08436	♂	A	44	—	90	—	—
		CC 08437	♂	A	50	—	96	—	—
		CC 08438	♂	A	44	—	91	—	—
		CC 08439	♂	A	47	—	91	—	—
		CC 08440	♂	A	52	—	97	—	—
		CC 08442	♂	A	45	—	93	—	—
		CC 08443	♂	A	48	—	91	—	—
		CC 08444	♂	A	47	—	93	—	—
		CC 08445	♂	A	48	—	95	—	—
		BB 99817	♂	A	—	46	—	90	*H. queleae*
		BD 06287	♀	A	—	40	—	85	*H. queleae*
		BD 06260	♂	A	—	45	—	93	*H. queleae*
		BD 06303	♀	A	—	41	—	86	*H. queleae*
		BB 99934	♂	A	49	—	95	—	—
		BD 06389	♀	A	36	—	88	—	—
		BD 06724	♀	A	42	—	86	—	—
		BD 06726	♀	A	43	—	89	—	—
		BD 06727	♀	A	41	—	86	—	—
		BD 06728	♀	A	45	—	89	—	—
		BD 06729	♂	A	46	—	90	—	—
		BD 06432	♂	A	48	—	90	—	—
		BD 06735	♀	A	36	—	86	—	—
		BD 06736	♀	A	45	—	90	—	—
		BD 06737	♀	A	39	—	87	—	—
		BD 06738	♀	A	40	—	87	—	—
		BD 06739	♀	A	39	—	87	—	—
		BD 06740	♀	A	39	—	87	—	—

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Year	Month	Ring number	Sex	Age	Mass		Wing Length		Avian haemosporidian parasites
					Uninfect	Infect	Uninfect	Infect	
1993	June	BD 06741	♀	A	—	43	—	86	*P. relictum*
		CC 08442	♂	A	45	—	93	—	—
		BD 06743	♀	A	38	—	86	—	—
		BD 06744	♀	A	38	—	87	—	—
		BD 06459	♂	A	—	47	—	91	*L. bouffardi*
		BD 06554	♀	A	37	—	86	—	—
		CV 04078	♂	A	47	—	94	—	—
		BD 06417	♀	A	—	39	—	87	*H. quelcae*
		CC 08434	♂	A	—	48	—	91	*P. vaughani*
		CC 08441	♂	A	49	—	89	—	—
		CC 08432	♂	A	—	45	—	92	*L. bouffardi*
		BC 22540	♂	A	45	—	91	—	—
		BB 99954	♀	A	40	—	90	—	—
		BB 99774	♂	A	—	48	—	92	*H. queleae*
		BB 99854	♀	A	—	44	—	91	*H. quelcae*
		CV 02383	♂	A	—	47	—	91	*H. queleae*
		CV 04171	♂	A	51	—	94	—	—
		BD 06784	♀	A	—	39	—	86	*L. bouffardi*
		BD 06786	♀	A	37	—	85	—	—
		BD 06789	♀	A	38	—	85	—	—
		BD 06791	♀	A	42	—	90	—	—
		BD 06795	♀	A	—	36	—	86	*L. bouffardi*
		BD 06799	♀	A	38	—	85	—	—
		CC 08464	♂	A	45	—	93	—	—
		CC 08465	♂	A	54	—	97	—	—
		CC 08466	♂	A	49	—	93	—	—
		CC 08467	♂	A	—	49	—	92	*L. bouffardi*
		CC 08468	♂	A	45	—	92	—	—
		CC 08469	♂	A	45	—	90	—	—
		CC 08470	♂	A	44	—	92	—	—
		CC 08471	♂	A	45	—	88	—	—
		BD 06601	♀	A	32	—	82	—	—
		BD 06604	♀	A	47	—	87	—	—
		CC 08473	♂	A	—	44	—	91	*L. bouffardi*
		CC 08475	♂	A	46	—	93	—	—
		BB 99992	♂	A	44	—	91	—	—
		BD 04920	♀	A	46	—	86	—	—
1993	July	CC 04394	♂	A	48	—	93	—	—
		CC 08509	♂	A	51	—	96	—	—
		CC 08512	♂	A	51	—	96	—	—
		CC 08541	♂	A	46	—	92	—	—
		CC 08544	♂	A	—	45	—	93	*H. queleae*
		CC 08546	♂	A	39	—	90	—	—
		BD 12347	♂	A	47	—	91	—	—
		BD 12356	♀	A	46	—	86	—	—
		BD 12358	♂	A	—	46	—	91	*H. queleae*
		BD 12360	♀	A	—	38	—	87	*H. queleae*
		BD 12362	♀	A	—	41	—	86	*H. queleae*
		BD 12363	♀	A	—	40	—	86	*H. queleae*
		BD 12376	♀	A	42	—	86	—	—
		CC 08572	♂	A	45	—	90	—	—

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Year	Month	Ring number	Sex	Age	Mass		Wing Length		Avian haemosporidian parasites
					Uninfect	Infect	Uninfect	Infect	
1993	July	CC 08573	♂	A	43	—	91	—	—
		CC 08575	♂	A	—	49	—	96	*L. bouffardi*
		CC 08576	♂	A	45	—	93	—	—
		CC 08577	♂	A	46	—	90	—	—
		CC 04321	♂	A	—	47	—	93	*H. queleq*
		CV 03397	♂	A	49	—	91	—	—
		BD 12229	♀	A	39	—	85	—	—
		BD 06412	♂	A	46	—	90	—	—
		BD 06501	♀	A	38	—	85	—	—
		BD 06637	♂	A	44	—	89	—	—
		BD 06655	♀	A	—	41	—	88	*H. queleq*
1993	August	BC 17006	♂	A	—	50	—	94	*L. bouffardi, H. queleae*
		BB 99878	♀	A	—	40	—	89	*L. bouffardi*
		BD 12289	♀	A	—	36	—	86	*H. queleae*
		BB 99849	♀	A	40	—	83	—	—
		BC 22598	♂	A	48	—	89	—	—
		CC 04301	♂	A	—	48	—	95	*L. bouffardi*
		CC 04380	♂	A	—	48	—	96	*H. queleae*
		CC 08375	♂	A	—	45	—	93	*L. bouffardi*
		BD 12415	♀	A		43	—	86	*L. bouffardi*
		BD 12416	♀	A	40	—	86	—	—
		BD 12417	♀	A	37	—	86	—	—
		BD 12418	♀	A	45	—	86	—	—
		BD 12424	♀	A	39	—	84	—	—
		BD 12425	♀	A	34	—	85	—	—
		BD 12432	♀	A	39	—	89	—	—
		BD 12433	♀	A	45	—	89	—	—
		BD 12434	♀	A		40	—	84	*L. bouffardi*
		BD 12435	♀	A	40	—	87	—	—
		BD 12442	♀	A	43	—	90	—	—
		BD 12443	♀	A	42	—	87	—	—
		BD 12451	♀	A	—	43	—	86	*H. queleae*
		BD 12457	♀	A	—	43	—	89	*H. queleae*
		BD 12458	♀	A	39	—	87	—	—
		BD 12460	♀	A	43	—	89	—	—
		BD 12466	♀	A	—	45	—	87	*H. queleae*
		CC 08578	♂	A	50	—	93	—	—
		CC 08584	♂	A	47	—	92	—	—
		CC 08585	♂	A	45	—	93	—	—
		CC 08586	♂	A	43	—	90	—	—
		CC 08587	♂	A	47	—	91	—	—
		CC 08588	♂	A	—	51	—	93	*H. queleae*
		CC 08589	♂	A	47	—	92	—	—
		BD 12113	♀	A	—	39	—	90	*H. queleae*
		BD 12114	♀	A	—	40	—	85	*H. queleae*
		BD 12116	♀	A	41	—	87	—	—
		BD 12193	♀	A	41	—	87	—	—
		BD 12194	♀	A	41	—	83	—	—
		BD 12200	♀	A	43	—	89	—	—
		BD 12494	♀	A	34	—	78	—	—
		BD 99882	♂	A	—	43	—	90	*H. queleae*

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Year	Month	Ring number	Sex	Age	Mass		Wing length		Avian haemosporidian parasites
					Uninfect	Infect	Uninfect	Infect	
1993	August	BD 99925	♂	A	44	—	92	—	—
		BC 17124	♂	A	—	49	—	96	*H. quelcae*
		CV 03222	♂	A	—	48	—	90	*H. quelcae*
		CV 02368	♂	A	48	—	95	—	—
		CV 04089	♂	A	45	—	90	—	—
		CV 04090	♂	A		46	—	91	*L. bouffardi*
		CV 04091	♂	A	45	—	89	—	—
		CV 04092	♂	A	48	—	95	—	—
		CV 04093	♂	A	—	44	—	93	*H. quelcae*
		CV 04094	♂	A	46	—	96	—	—
		CV04096	♂	A	—	51	—	94	*H. queleae*
		CV 04098	♂	A	—	51	—	96	*H. queleae*
		CV 04099	♂	A	—	46	—	95	*L. bouffardi, H. quelcae*
		CV 04100	♂	A	—	55	—	94	*L. bouffardi, H. quelcae*
		CV 04102	♂	A	—	51	—	93	*H. quelcae*
		BB 99975	♂	A	—	44	—	93	*H. quelcae*
		BC 20381	♂	A	—	51	—	90	*H. queleae*
		BD 12194	♀	A	41	—	83	—	—
		BD 12225	♀	A	39	—	86	—	—
		BC 22549	♂	A	—	45	—	89	*H. queleae*
		CC 04379	♂	A	—	47	—	95	*H. quelcae*
		CC 08436	♂	A	43	—	90	—	—
		BD 12596	♀	A	39	—	86	—	—
		BD 12541	♀	A	—	41	—	89	*H. queleae*
		BD 12542	♀	A	39	—	86	—	—
		BD 12543	♀	A	43	—	88	—	—
		BD 12550	♀	A	—	46	—	87	*H. quelcae*
		BD 12553	♀	A	—	41	—	87	*H. queleae*
		BD 12595	♀	A	40	—	87	—	—
1993	September	CV 04113	♂	A	—	50	—	90	*H. quelcae*
		CV 04117	♂	A	46	—	90	—	—
		CV 04118	♂	A	—	48	—	88	*H. quelcae*
		CV 04126	♂	A	—	47	—	94	*L. bouffardi*
		CV 04128	♂	A	49	—	95	—	—
		CC 08411	♂	A	48	—	92	—	—
		BB 99973	♂	A		44	—	91	*L. bouffardi*
		BB 98559	♂	A	—	51	—	96	*H. quelcae*
		BB 99942	♂	A	—	42	—	91	*L. bouffardi*
		BC 20794	♀	A	—	44	—	87	*H. queleae*
		BD 06523	♀	A	—	44	—	84	*H. queleae*
		BD 12737	♀	A	—	36	—	85	*H. queleae*
		BD 12761	♀	A	—	38	—	85	*H. quelcae*
		BD 12748	♀	A	—	38	—	89	*H. queleae*
		CC 08601	♂	A	—	50	—	97	*L. bouffardi, H. queleae*
		CC 08602	♂	A	—	50	—	95	*H. queleae*
		CC 08603	♂	A	—	48	—	95	*H. quelcae*
		BB 99967	♂	A	—	46	—	90	*H. queleae*
		BB 99832	♂	A	45	—	94	—	—
		BC 16785	♂	A	—	50	—	95	*H. quelcae*
		BC 03862	♂	A	—	48	—	93	*H. queleae*
		BD 06501	♀	A	—	40	—	85	*L. bouffardi*

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Year	Month	Ring number	Sex	Age	Mass		Wing length		Avian haemosporidian parasites
					Uninfect	Infect	Uninfect	Infect	
1993	September	BD 12250	♀	A	39	—	88	—	—
		BD 12757	♀	A	41	—	87	—	—
		BD 04849	♂	A	—	53	—	92	*L. bouffardi, H. queleae*
		CV 02331	♂	A	50	—	96	—	—
		CV 03317	♂	A	—	49	—	93	*H. queleae*
		CV 04303	♂	A	—	51	—	95	*H. queleae*
		CV 02381	♂	A	—	51	—	94	*H. queleae*
		CV 02321	♂	A	—	52	—	98	*H. queleae*
		CC 04377	♂	A	—	48	—	97	*H. queleae*
		CC 08445	♂	A	—	48	—	96	*H. queleae*
		BD 12762	♀	A	—	46	—	90	*H. queleae*
		BD 12766	♀	A	40	—	82	—	—
		BD 12767	♀	A	46	—	90	—	—
		CC 08604	♀	A	—	44	—	88	*H. queleae*
		CC 08605	♂	A	48	—	91	—	—
		CV 03410	♂	A	—	50	—	94	*L. queleae*
		CV 03408	♂	A	—	48	—	92	*H. queleae*
		BC 17120	♂	A	—	51	—	94	*H. queleae*
1993	October	CC 08720	♂	A	—	49	—	91	*H. queleae*
		CC 08721	♀	A	—	41	—	87	*H. queleae*
		CC 08722	♂	A	—	45	—	89	*H. queleae*
		CC 08723	♂	A	48	—	94	—	—
		CC 08724	♂	A	50	—	92	—	—
		BD 12834	♀	A	—	41	—	86	*H. queleae*
		BD 12835	♀	A	—	40	—	87	*H. queleae*
		BD 12836	♀	A	39	—	82	—	—
		BD 06663	♀	A	38	—	87	—	—
		BD 06665	♀	A	40	—	88	—	—
		BD 06388	♂	A	—	49	—	90	*H. queleae*
		BB 99617	♂	A	—	46	—	89	*H. queleae*
1993	December	CC 08871	♂	A	45	—	91	—	—
		CC 08872	♂	A	49	—	92	—	—
		BC 22549	♂	A	43	—	88	—	—
		BD 06259	♂	A	47	—	90	—	—
		BD 12824	♂	A	45	—	92	—	—
		BD 12875	♀	A	—	35	—	83	*L. bouffardi*
		BD 12885	♀	A	—	37	—	87	*H. queleae*
		CV 04143	♂	A	—	44	—	90	*H. queleae*
		BD 12968	♀	A	—	47	—	88	*L. bouffardi, H. queleae*
		BD 12973	♀	A	45	—	89	—	—
		BD 12986	♂	A	—	47	—	90	*L. bouffardi, H. queleae*
		BD 12990	♀	A	43	—	85	—	—
		BD 12996	♀	A	37	—	87	—	—
		BD 13000	♀	A	40	—	87	—	—
		BD 15016	♀	A	—	45	—	88	*L. bouffardi*
		BD 15017	♀	A	—	42	—	85	*L. bouffardi, H. queleae*
		BD 15018	♀	A	—	43	—	88	*H. queleae*
		BD 15020	♀	A	41	—	88	—	—
		CC 08846	♂	A	44	—	92	—	—
		CC 08849	♂	A	—	46	—	91	*H. queleae*
		CC 08854	♂	A	—	43	—	90	*L. bouffardi, H. queleae*

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Year	Month	Ring number	Sex	Age	Mass		Wing length		Avian haemosporidian parasites
					Uninfect	Infect	Uninfect	Infect	
1993	December	CC 08856	♂	A	—	47	—	91	*H. queleae*
		CC 08861	♂	A	46	—	90	—	—
		CC 08862	♂	A	44	—	93	—	—
		CC 08867	♂	A	—	49	—	89	*H. queleae*
		CC 08868	♂	A	46	—	91	—	—
		CC 08869	♂	A	—	48	—	90	*H. queleae*
		CC 08870	♂	A	—	49	—	91	*L. bouffardi*
		BD 15051	♀	A	-	38	---	87	*L. bouffardi, H. queleae*
		BD 15053	♀	A	—	48	—	86	*H. queleae*
		BD 15056	♀	A	49	—	86	—	—
		BD 15059	♀	A	—	48	—	84	*L. bouffardi*
		BD 15060	♀	A	—	48	—	87	*H. queleae*
		BD 15061	♀	A	45	—	84	—	—
		BD 15062	♀	A	—	48	—	88	*L. bouffardi, H. queleae*
		BD 15064	♀	A	—	45	—	84	*L. bouffardi, H. queleae*
		BD 15058	♀	A	46	---	83	---	—
		CC 08877	♂	A	—	48	—	92	*H. queleae*
		CC 08878	♀	A	56	—	89	—	—
		CC 08879	♂	A	54	—	91	—	—
		CC 08880	♂	A	47	—	91	—	---
		CC 08881	♂	A	49	---	89	—	—
		CC 08882	♂	A	45	—	89	—	—
		CC 08883	♂	A	44	—	91	—	—
		CC 08886	♂	A	52	—	92	—	—
		CC 08887	♂	A	—	54	—	93	*P. vaughani*
		CC 08888	♂	A	57	—	92	—	—
		CC 08889	♂	A	—	57	—	93	*H. queleae*
		CC 08890	♀	A	51	---	88	—	—
		CC 08891	♀	A	46	—	89	—	—
		CC 08892	♂	A	46	—	91	—	—
		CC 08893	♂	A	---	54	—	89	*H. queleae*
		CC 08894	♂	A	52	—	90	—	---
		CC 08896	♂	A	47	—	91	—	—
		CC 08897	♂	A	43	—	90	—	—
		BD 02893	♂	A	61	—	91	—	—
		BD 12762	♂	A	45	—	88	—	—
		BD 12885	♀	A	—	40	—	88	*H. queleae*
		BD 12979	♀	A	-	41	—	87	*H. queleae*
		BD 12998	♀	A	51	—	88	—	—
		CC 08495	♂	A	46	—	92	—	—
		CC 08738	♂	A	—	45	—	90	*L. bouffardi, H. queleae*
		CC 08858	♂	A	—	53	—	90	*L. bouffardi*
		CC 08865	♂	A	53	—	90	—	—
		CV 03390	♂	A	38	—	83	—	—
1994	January	CC 08947	♂	A	47	—	93	—	—
		CC 08948	♂	A	45	—	90	---	—
		CC 08949	♂	A	—	47	—	91	*L. bouffardi, H. queleae*
		CC 08950	♂	A	—	46	—	90	*L. bouffardi*
		CC 08951	♂	A	46	---	93	—	—
		CC 08952	♂	A	---	44	—	91	*H. queleae*
		CC 09853	♂	A	—	47	—	92	*H. queleae*

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Year	Month	Ring number	Sex	Age	Mass		Wing length		Avian haemosporidian parasites
					Uninfect	Infect	Uninfect	Infect	
1994	January	CC 08954	♂	A	43	—	90	—	—
		CC 08955	♂	A	44	—	94	—	—
		CC 08956	♂	A	44	—	90	—	—
		CC 08957	♂	A	—	46	—	90	*H. queleae*
		CC 08958	♂	A	—	43	—	92	*H. queleae*
		CC 08959	♂	A	—	49	—	89	*H. queleae*
		CC 08960	♂	A	46	—	90	—	—
		CC 08961	♂	A	—	45	—	94	*H. queleae*
		CC 08962	♂	A	40	—	93	—	—
		CC 08963	♂	A	—	43	—	91	*L. bouffardi*
		CC 08964	♂	A	—	41	—	91	*L. bouffardi, H. queleae*
		CC 08965	♂	A	—	48	—	90	*L. bouffardi*
		CC 08966	♂	A	—	45	—	91	*H. queleae*
		CC 08967	♂	A	44	—	90	—	—
		CC 08969	♂	A		43	—	91	*H. queleae*
		CC 08971	♀	A	—	39	—	88	*H. queleae*
		BD 15118	♀	A	38	—	88	—	—
		BD 15119	♀	A	—	48	—	88	*L. bouffardi*
		BD 15133	♀	A	—	45	—	87	*H. queleae*
		BD 15134	♀	A	39	—	84	—	—
		BD 15136	♀	A	38	—	86	—	—
		BD 15137	♀	A	—	38	—	85	*L. bouffardi*
		BD 15139	♀	A	39	—	86	—	—
		BD 15140	♀	A	38	—	85	—	—
		BD 15141	♀	A	—	37	—	85	*H. queleae*
		BD 15142	♀	A	—	36	—	85	*P. relictum*
		BD 15147	♀	A	39	—	86	—	—
		BD 15150	♀	A	—	45	—	87	*L. bouffardi, H. queleae*
		BD 15151	♀	A	37	—	85	—	—
		BD 15153	♀	A	—	37	—	80	*H. queleae*
		BD 12766	♀	A	47	—	81	—	—
		CC 08976	♂	A	—	49	—	92	*L. bouffardi, H. queleae*
		CC 08977	♂	A	48	—	93	—	—
		CC 08978	♂	A	47	—	91	—	—
		CC 08979	♂	A	48	—	90	—	—
		CC 08980	♂	A	50	—	91	—	—
		CC 08991	♂	A	52	—	94	—	—
		CC 08992	♂	A	49	—	89	—	—
		CC 08983	♂	A	—	50	—	92	*L. bouffardi*
		CC 08984	♂	A	47	—	92	—	—
		CC 08985	♂	A	50	—	92	—	—
		CC 08986	♂	A	49	—	95	—	—
		CC 08987	♂	A	47	—	93	—	—
		CC 08988	♂	A	46	—	90	—	—
		CC 08989	♂	A	48	—	91		—
		CC 08990	♂	A	—	49	—	94	*L. bouffardi*
		CC 08991	♂	A	—	43	—	90	*L. bouffardi*
		CC 08992	♂	A	48	—	92	—	—
		CC 08993	♂	A	46	—	91	—	—
		CC 08994	♂	A	—	48	—	91	*L. bouffardi*
		CC 08995	♂	A	48	—	90	—	—

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Year	Month	Ring number	Sex	Age	Mass		Wing length		Avian haemosporidian parasites
					Uninfect	Infect	Uninfect	Infect	
1994	January	CC 08996	♂	A	—	49	—	89	*L. bouffardi, H. queleae*
		BD 15160	♀	A	—	39	—	85	*L. bouffardi*
		BD 15161	♀	A	49	—	86	—	—
		BD 15162	♀	A	43	—	86	—	—
		BD 15163	♀	A	39	—	85	—	—
		BD 15164	♀	A	—	49	—	89	*H. queleae*
		BD 15166	♀	A	—	49	—	88	*H. queleae*
		BD 15167	♀	A	—	39	—	85	*L. bouffardi*
		BD 15170	♂	A	49	—	90	—	—
		BD 15171	♂	A	46	—	90	—	—
		BD 15172	♀	A	46	—	87	—	—
		BD 15174	♀	A	38	—	89	—	—
		BD 15175	♀	A	—	40	—	83	*L. bouffardi, H. queleae*
		BD 15176	♀	A	—	35	—	85	*L. bouffardi, H. queleae*
		BD 15178	♂	A	—	49	—	95	*H. queleae*
		BD 15179	♀	A	41	—	83	—	—
		BD 15180	♀	A	37	—	84	—	—
		BD 15181	♀	A	39	—	85	—	—
		BD 15182	♀	A	—	49	—	89	*L. bouffardi*
		BD 15183	♀	A	—	46	—	84	*L. bouffardi*
		BD 15184	♂	A	46	—	89	—	—
		BD 15185	♀	A	41	—	86	—	—
		BD 15186	♂	A	50	—	89	—	—
		BD 15187	♂	A	45	—	91	—	—
		BD 15190	♀	A	—	40	—	84	*L. bouffardi*
		BD 15191	♀	A	39	—	86	—	—
		BD 12999	♂	A	—	44	—	90	*L. bouffardi, H. queleae*
		CC 10218	♂	A	—	48	—	90	*H. queleae*
		CC 10219	♂	A	—	46	—	91	*H. queleae*
		CC 10220	♂	A	—	41	—	89	*H. queleae*
		CC 10221	♂	A	—	48	—	94	*H. queleae*
		CC 10222	♂	A	46	—	92	—	—
		CC 10223	♀	A	—	40	—	86	*H. queleae*
		CC 10224	♂	A	41	—	88	—	—
		CC 10225	♂	A	39	—	94	—	—
		CC 10226	♂	A	48	—	93	—	—
		CC 10227	♂	A	—	46	—	93	*L. bouffardi*
		CC 10228	♂	A	49	—	92	—	—
		CC 10229	♂	A	—	42	—	88	*H. queleae*
		CC 10230	♀	A	—	43	—	87	*H. queleae*
		CC 10232	♀	A	—	41	—	82	*H. queleae*
		CC 10233	♂	A	—	47	—	90	*H. queleae*
		CC 10234	♂	A	—	42	—	90	*H. queleae*
		CC 10235	♂	A	47	—	92	—	—
		CC 10236	♀	A	—	42	—	84	*H. queleae*
		CC 10237	♂	A	48	—	91	—	—
		CC 10238	♂	A	44	—	89	—	—
		CC 10239	♀	A	44	—	88	—	—
		CC 10240	♂	A	—	49	—	89	—
		CV 04113	♂	A	50	—	94	—	—
		CV 04136	♂	A	45	—	91	—	—
		CC 08958	♂	A	—	49	—	91	*H. queleae*

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Year	Month	Ring number	Sex	Age	Mass		Wing length		Avian haemosporidian parasites
					Uninfect	Infect	Uninfect	Infect	
1994	January	CC 08959	♂	A	—	50	—	92	*H. queleae*
		CC 08867	♂	A	—	50	—	89	*H. queleae*
		CC 08877	♂	A	—	45	—	92	*L. bouffardi*
		CC 08968	♂	A	—	48	—	94	*H. queleae*
		BB 99948	♂	A	46	—	92	—	—
		BC 03896	♂	A	48	—	94	—	—
		BC 17207	♀	A	38	—	87	—	—
		BC 17006	♂	A	50	—	98	—	—
		BD 06659	♂	A	50	—	93	—	—
		BD 15004	♀	A	39	—	84	—	—
		BD 15018	♂	A		45		90	*H. queleae*
1994	February	CC 10250	♂	A	48	—	93	—	—
		CC 10252	♂	A	—	48	—	91	*L. bouffardi*
		CC 10253	♂	A	—	49	—	93	*H. queleae*
		CC 10254	♂	A	—	48	—	92	*H. queleae*
		CC 10255	♂	A	—	50	—	91	*H. queleae*
		CC 10256	♀	A	—	41	—	86	*H. queleae*
		CC 10257	♂	A	46	—	93	—	—
		CC 10258	♂	A	47	—	93	—	—
		CC 10259	♂	A	—	51	—	90	*L. bouffardi*
		CC 10260	♀	A	—	40	—	87	*H. queleae*
		CC 10261	♂	A	—	47	—	90	*H. queleae*
		CC 10262	♀	A	—	38	—	87	*H. queleae*
		CC 10263	♂	A	43		89		—
		CC 10264	♀	A	—	41	—	85	*L. bouffardi*
		CC 10265	♀	A	—	45	—	85	*H. queleae*
		BC 22770	♀	A	—	37	—	87	*L. bouffardi*
		BD 06732	♂	A	—	47	—	94	*H. queleae*
		BD 12991	♂	A	46	—	89	—	—
		BD 15056	♀	A	40	—	85	—	—
		BD 15058	♀	A	40	—	87	—	—
		CV 03435	♂	A	—	46	—	95	*H. queleae*
		CC 08267	♂	A	45	—	94	—	—
		CC 08383	♂	A	44	—	94	—	—
		CC 08977	♂	A	—	48	-	92	*H. queleae*
		CC 10251	♂	A	45	—	90	—	—
		BC 22799	♀	A	41	—	86	—	—
		BD 15256	♀	A	—	41	—	87	*H. queleae*
		BD 15257	♂	A	—	45	—	96	*H. queleae*
		BD 15258	♂	A	—	51	—	92	*H. queleae*
		BD 15259	♀	A	—	38	—	85	*H. queleae*
		CC 10268	♂	A	—	46	—	94	*L. bouffardi*
		CC 10269	♀	A	42	—	84	—	—
		CC 08962	♂	A	—	40	—	90	*H. queleae*
		CC 10235	♂	A	—	50	—	95	*H. queleae*
		CC 10252	♂	A	46	—	90	—	—
		CC 10259	♂	A	49	—	91	—	—
		CV 03337	♂	A	50	—	96	—	—
		BC 17119	♂	A	—	48	—	96	*H. queleae*
		BD 12999	♂	A	—	44	—	90	*Trypanosoma everetti*
		BD 15150	♂	A	46	—	91	—	—
		BD 15266	♀	A	42	—	85	—	—

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Appendix 21 Monthly avian haemosporidian prevalence of birds trapped at the 10 study sites within the Greater Cape Town Area from February 1993 to February 1995. Avian haemos.=Avian haemosporidian parasites; Tot.=Total number of blood smears examined; Inf.=Total number of infections including multiple infections; *Hae.=Haemoproteus* species; *Leu.=Leucocytozoon* species; *Pla.=Plasmodium* species.

Months	Bettys Bay (34°22'S 18°56'E)					Durbanville Nature Reserve (33°50'S 18°38'E)					Glencairn (34°09'S 18°25'E)					Goedeontmoeting (33°41'S 18°36'E)					Koeberg Nature Reserve (33°40'S 18°26'E)					Kirstenbosch National Botanical Gardens (33°48'S 18°25'E)					12 Roseberry Road Mowbray (33°55'S 18°18'E)					Patryskraal (34°26'S 20°1'E)					Rondevlei Nature Reserve (34°04'S 18°30'E)					Tygerberg Nature Reserve (33°52'S 18°36'E)				
Avian haemos.	Tot.	Inf.	Hae.	Leu.	Pla.	Tot.	Inf.	Hae.	Leu.	Pla.	Tot.	Inf.	Hae.	Leu.	Pla.	Tot.	Inf.	Hae.	Leu.	Pla.	Tot.	Inf.	Hae.	Leu.	Pla.	Tot.	Inf.	Hae.	Leu.	Pla.	Tot.	Inf.	Hae.	Leu.	Pla.	Tot.	Inf.	Hae.	Leu.	Pla.	Tot.	Inf.	Hae.	Leu.	Pla.
January	18	3	1	1	1	70	8	5	2	1	48	4	2	12	—	256	92	68	36	1	0	0	—	—	—	18	1	—	1	—	10	0	—	—	—	41	2	0	2	—	80	3	2	—	—	0	0	—	—	—
February	0	2	—	2	—	66	12	2	10	—	31	5	3	3	—	179	45	31	14	2	0	0	—	—	—	16	2	3	3	—	13	1	—	1	—	43	1	1	0	—	45	0	—	1	—	480	165	27	147	—
March	0	0	-	—	—	223	22	8	15	1	38	4	1	3	—	245	46	41	14	3	45	6	3	4	—	81	11	—	9	—	13	0	4	1	—	0	0	0	0	—	18	0	—	—	—	419	120	33	95	1
April	25	10	6	4	—	147	18	4	13	—	28	4	3	1	—	313	67	42	25	5	26	1	1	—	1	124	18	9	21	1	18	5	3	—	—	0	0	0	0	—	0	0	—	—	—	455	126	39	94	1
May	0	0	-	—	—	175	23	7	16	1	42	9	2	6	—	326	95	29	13	8	0	0	—	—	—	24	10	3	7	1	22	3	1	1	—	18	1	0	1	—	12	0	—	—	—	152	26	8	15	—
June	0	0	—	—	—	103	11	2	8	1	33	2	1	1	—	325	67	38	16	6	0	0	—	—	—	37	18	12	16	—	19	2	—	1	—	22	1	0	1	—	116	6	4	2	1	82	11	5	6	
July	0	0	—	—	—	51	6	—	1	4	26	1	—	1	—	346	81	59	15	3	0	0	—	—	—	39	11	2	12	1	11	0	—	1	—	0	0	0	0	—	242	12	12	12	—	59	13	1	10	—
August	0	0	—	—	—	28	11	2	9	—	28	1	—	1	—	351	97	48	23	5	0	0	—	—	—	37	5	—	9	—	25	3	4	1	—	44	5	3	0	1	241	29	27	29	—	137	12	4	8	—
September	42	5	1	4	—	25	4	1	3	—	25	5	2	5	—	300	115	80	15	3	0	0	—	—	—	18	4	—	—	—	20	5	4	1	—	21	1	0	1	—	116	12	11	11	—	289	78	17	64	3
October	112	14	4	10	—	9	2	—	2	—	46	5	1	7	—	145	43	39	24	3	0	0	—	—	-	36	7	3	1	—	29	1	0	1	—	19	2	2	1	—	308	109	40	89	1					
November	137	27	8	25	—	48	11	7	6	1	43	5	1	6	—	21	6	10	5	—	0	0	—	—	—	0	0	—	—	—	34	2	3	—	—	0	0	0	0	—	83	5	0	6	—	64	22	3	16	—
December	139	39	17	34	2	78	17	10	7	—	19	3	1	2	—	139	45	30	25	2	0	0	—	—	—	0	0	—	—	—	51	11	10	—	—	0	0	0	0	—	49	3	1	1	—	106	34	8	25	—
Total	438	98	37	81	3	1023	144	49	92	9	405	68	17	46	—	2944	742	527	229	41	71	7	6	4	1	398	80	30	72	3	269	40	22	8	—	278	19	6	6	1	991	73	12	63	1	2551	715	163	575	6
Percentage	—	22.3	37.7	82.6	3.0	—	14.0	33.3	63.8	6.2	—	16.7	25.0	70.5	—	—	25.2	71.0	30.8	5.5	—	9.8	57.1	57.1	14.2	—	20.1	37.5	90.0	3.7	—	14.8	80.0	30.0	—	—	6.8	40.0	60.0	10.0	—	7.3	16.4	86.3	1.5	—	28.0	25.5	80.3	0.8

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Year	Month	Ring number	Sex	Age	Mass		Wing length		Avian haemosporidian parasites
					Uninfect	Infect	Uninfect	Infect	
1994	February	BD 15268	♀	A	40	—	83	—	—
		BD 15269	♀	A	—	39	—	87	*H. queleae, P. circumflexum*
		BD 15271	♀	A	38	—	86	—	—
		BD 15272	♀	A	—	39	—	85	*L. bouffardi, H. queleae*
		BD 15273	♀	A	40	—	85	—	—
		BD 15275	♀	A	—	33	—	88	*H. queleae*
		BD 15276	♀	A	37	—	88	—	—
		BD 15278	♀	A	39	—	88	—	—
		BD 15280	♀	A	40	—	86	—	—
		BD 15281	♀	A	—	42	—	86	*L. bouffardi, H. queleae*
		CC 10285	♂	A	48	—	93	—	—
		CC 10286	♂	A	—	45	—	90	*L. bouffardi, H. queleae*
		CC 10287	♂	A	—	47	—	90	*H. queleae*
		CC 10288	♂	A	46	—	90	—	—
		CC 10289	♂	A	—	44	—	91	*H. queleae*
		CC 10290	♂	A	46	—	92	—	—
		CC 10291	♂	A	46	—	92	—	—
		CC 10292	♂	A	—	47	—	92	*L. bouffardi*
		CC 10293	♂	A	47	—	90	—	—
		BD 12989	♀	A	41	—	87	—	—
		BD 12994	♂	A	46	—	91	—	—
		BD 15059	♀	A	—	41	—	85	*H. queleae*
1994	March	BD 15306	♀	A	39	—	85	—	—
		BD 15317	♀	A	38	—	84	—	—
		BD 15319	♀	A	—	41	—	84	*H. queleae*
		BD 15323	♀	A	42	—	88	—	—
		BD 15325	♀	A	39	—	83	—	—
		BD 15327	♀	A	—	40	—	86	*H. queleae*
		BD 15328	♀	A	39	—	86	—	—
		BD 15324	♀	A	38	—	86	—	—
		CC 10325	♂	A	46	—	92	—	—
		CC 10326	♂	A	46	—	92	—	—
		CC 10327	♂	A	51	—	93	—	—
		CC 10328	♂	A	50	—	94	—	—
		CC 10330	♂	A	44	—	96	—	—
		CC 10331	♂	A	47	—	92	—	—
		CC 10332	♂	A	—	43	—	93	*H. queleae*
		CC 10333	♂	A	44	—	93	—	—
		CC 10334	♂	A	46	—	94	—	—
		CC 10335	♂	A	—	45	—	90	*H. queleae*
		CC 10336	♂	A	43	—	92	—	—
		CC 10337	♂	A	48	—	91	—	—
		CC 10338	♂	A	50	—	92	—	—
		BB 99292	♀	A	37	—	88	—	—
		BC 17014	♂	A	48	—	95	—	—
		BD 06528	♀	A	—	36	—	85	*H. queleae*
		CC 08954	♂	A	44	—	91	—	—
		CC 10240	♂	A	—	47	—	91	*H. queleae*
		CV 04103	♂	A	51	—	96	—	—
		BD 15350	♀	A	31	—	87	—	—
		BD 15359	♂	A	49	—	91	—	—
		CC 10352	♂	A	46	—	92	—	—

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Year	Month	Ring number	Sex	Age	Mass		Wing length		Avian haemosporidian parasites
					Uninfect	Infect	Uninfect	Infect	
1994	March	CC 08138	♂	A	—	49	—	93	*H. queleae*
		BC 06858	♂	A	—	48	—	92	*H. quelcae*
		BD 15187	♂	A	47	—	92	—	-
		BD 15362	♀	A	—	38	—	83	*P. relictum*
		BD 15363	♂	A	—	46	—	87	*L. bouffardi, H. quelcae*
		BD 15364	♀	A	40	—	88	—	-
		BD 15365	♀	A	41	—	88	—	-
		BD 15366	♀	A	38	—	84	—	-
		BD 15367	♀	A	42	—	88	—	-
		BD 15368	♀	A	42	—	86	—	-
		BD 15369	♀	A	41	—	87	—	-
		BD 15370	♀	A	40	—	87	—	-
		CC 10353	♂	A	48	—	93	—	-
		CC 10354	♂	A	47	—	91	—	-
		CC 10355	♂	A	48	—	94	—	-
		CC 10356	♂	A	48	—	92	—	-
		CC 10357	♂	A	—	48	—	93	*H. queleae*
		CC 10358	♂	A	43	—	94	—	-
		CC 10359	♂	A	41	—	91	—	-
		CC 10360	♂	A	50	—	92	—	-
		CC 10361	♂	A	48	—	94	—	-
		CC 10362	♂	A	—	45	—	92	*H. queleae*
		CC 10363	♂	A	46	—	93	—	-
		CC 10364	♂	A	47	—	94	—	-
		BD 12478	♀	A	—	42	—	86	*L. bouffardi*
		BD 15004	♀	A	—	40	—	85	*H. queleae*
		BD 10560	♀	A	—	42	—	88	*H. queleae*
		CC 08319	♂	A	48	—	90	—	-
		CC 08337	♂	A	—	46	—	93	*H. queleae*
		CC 08845	♂	A	—	49	—	91	*H. queleae*
		CC 20552	♂	A	41	—	89	—	-
		BD 04923	♀	A	39	—	85	—	-
		BD 06349	♂	A	—	46	—	93	*H. quelcae*
		BD 12362	♀	A	—	43	—	88	*H. quelcae*
		BD 12968	♂	A	—	47	—	93	*H. queleae*
		BD 15367	♀	A	42	—	88	—	—
		CC 04327	♂	A	51	—	99	—	—
		CC 04408	♂	A	47	—	94	—	—
		CC 08127	♂	A	49	—	93	—	—
		CC 08383	♂	A	44	—	96	—	—
		CC 08845	♂	A	51	—	92	—	—
		CC 08882	♂	A	45	—	92	—	—
		CC 08969	♂	A	49	—	95	—	—
		CC 08982	♂	A	42	—	92	—	—
		CV 02322	♂	A	53	—	95	—	—
		CV 02390	♂	A	46	—	94	—	—
		CV 03321	♂	A	54	—	96	—	—
		BB 99920	♂	A	49	—	94	—	—
		BD 15058	♀	A	42	—	86	—	—
		CC 08965	♂	A	52	—	90	—	—
1994	April	BD 06288	♂	A	49	—	94	—	—
		BD 04617	♀	A	40	—	85	—	—

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Year	Month	Ring number	Sex	Age	Mass		Wing length		Avian haemosporidian parasites
					Uninfect	Infect	Uninfect	Infect	
1994	April	BD 12396	♂	A	—	41	—	90	*H. queleae*
		BD 15171	♂	A	51	—	92	—	—
		BB 99832	♂	A	46	—	93	—	—
		CV 02316	♂	A	52	—	95	—	—
		CV 02379	♂	A	50	—	92	—	—
		CV 03395	♂	A	50	—	98	—	—
		CV 04104	♂	A	50	—	95	—	—
		CC 08477	♂	A	45	—	95	—	—
		CC 08957	♂	A	52	—	92	—	—
		CC 10396	♂	A	46	—	91	—	—
		CC 10397	♂	A	46	—	91	—	—
		CC 10400	♂	A	52	—	92	—	—
		CC 10402	♂	A	51	—	91	—	—
		BD 15433	♀	A	—	45	—	89	*H. quelcae*
		BD 15434	♀	A	42	—	88	—	—
		BD 15429	♀	A	43	—	88	—	—
		CC 10395	♂	A	50	—	94	—	—
		CC 10396	♂	A	51	—	91	—	—
		BD 12273	♂	A	43	—	91	—	—
		BD 15564	♀	A	—	31	—	85	*L. bouffardi*
		BD 15565	♀	A	—	36	—	82	*H. quelcae*
		BD 15569	♀	A	—	43	—	86	*H. queleae*
		CC 10417	♂	A	50	—	94	—	—
		BD 15583	♀	A	41	—	86	—	—
		BD 15588	♂	A	—	42	—	90	*H. queleae*
		CC 10410	♂	A	47	—	91	—	—
		CC 10411	♂	A	46	—	94	—	—
		CC 10412	♂	A	50	—	91	—	—
		CC 10413	♂	A	—	46	—	92	*H. quelcae*
		CC 10415	♂	A	48	—	93	—	—
		CC 10416	♂	A	—	50	—	93	*H. queleae*
		BB 99263	♀	A	38	—	87	—	—
		BB 99934	♂	A	50	—	97	—	—
		BB 99991	♂	A	—	45	—	93	*H. quelcae*
		BC 20495	♀	A	42	—	88	—	—
		BC 25948	♂	A	—	48	—	92	*P. relictum*
		BD 06304	♂	A	50	—	95	—	—
		BD 06453	♂	A	46	—	92	—	—
		BD 12737	♀	A	—	39	—	87	*H. quelcae*
		BD 15164	♂	A	49	—	92	—	—
		BD 15367	♂	A	52	—	91	—	—
		CV 01820	♂	A	49	—	94	—	—
		CV 04091	♂	A	48	—	91	—	—
		CV 04115	♂	A	49	—	95	—	—
		CC 08118	♂	A	48	—	95	—	—
		CC 08399	♂	A	46	—	95	—	—
		CC 08547	♂	A	46	—	92	—	—
		CC 08853	♂	A	—	49	—	93	*H. quelcae*
		CC 10240	♂	A	—	50	—	93	*H. queleae*
1994	May	BC 25934	♂	A	48	—	95	—	—
		BD 12987	♂	A	48	—	93	—	—
		BD 15363	♂	A	—	49	—	90	*L. bouffardi*

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Year	Month	Ring number	Sex	Age	Mass		Wing length		Avian haemosporidian parasites
					Uninfect	Infect	Uninfect	Infect	
1994	May	CC 08194	♂	A	51	—	94	—	—
		CC 08270	♂	A	54	—	97	—	—
		CC 08960	♂	A	48	—	90	—	—
		CC 10363	♂	A	—	49	—	90	*H. quelcae*
		CC 10461	♂	A	50	—	90	—	—
		CC 10221	♂	A	49	—	96	—	—
		CC 08969	♂	A	48	—	93	—	—
		CC 08853	♂	A	52	—	92	—	—
		CC 08144	♂	A	51	—	94	—	—
		BD 15602	♀	A	—	40	—	85	*H. quelcae*
		BD 15607	♀	A	—	39	—	89	*H. quelcae*
		BD 15610	♀	A	—	39	—	87	*H. quelcae*
		CC 10432	♂	A	47	—	92	—	—
		CC 10433	♂	A	47	—	93	—	—
		CC 10434	♂	A	53	—	93	—	—
		BD 15635	♀	A	39	—	85	—	—
		BD 15662	♀	A	39	—	87	—	—
		BD 15663	♀	A	42	—	89	—	—
		BD 15683	♀	A	41	—	86	—	—
		CC 10440	♂	A	—	47	—	91	*L. houffardi, P. relictum*
		CC 10441	♂	A	47	—	92	—	—
		CC 10442	♂	A	51	—	92	—	—
		CC 10443	♂	A	49	—	92	—	—
		CC 10444	♂	A	51	—	94	—	—
		CC 10445	♂	A	51	—	95	—	—
		CC 10446	♂	A	47	—	91	—	—
		CC 10447	♂	A	—	47	—	94	*H. quelcae*
		CC 10448	♂	A	53	—	95	—	—
		CC 10449	♂	A	52	—	94	—	—
		BB 99775	♂	A	47	—	91	—	—
		BC 17334	♀	A	39	—	88	—	—
		CC 08572	♂	A	—	47	—	90	*H. quelcae*
		BD 04855	♂	A	51	—	95	—	—
		BD 06653	♀	A	42	—	88	—	—
		BD 12968	♂	A	46	—	93	—	—
		BD 15359	♂	A	49	—	93	—	—
		CV 04140	♂	A	—	52	—	93	*H. quelcae*
		CC 04141	♂	A	50	—	97	—	—
		CC 08966	♂	A	50	—	94	—	—
		BD 15764	♀	A	44	—	87	—	—
		BD 15774	♀	A	41	—	88	—	—
		BD 15779	♀	A	40	—	87	—	—
		CC 10454	♂	A	46	—	94	—	—
		CC 10455	♂	A	—	46	—	94	*H quelcae*
		BB 98564	♂	A	48	—	93	—	—
		BB 99973	♂	A	—	50	—	92	*H. quelcae*
		BC 22551	♂	A	50	—	97	—	—
		BC 20507	♂	A	40	—	89	—	—
		BC 17332	♂	A	50	—	95	—	—
		BD 05686	♀	A	44	—	87	—	—
		BD 15020	♀	A	44	—	88	—	—
		BD 12853	♂	A	47	—	94	—	—

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Year	Month	Ring number	Sex	Age	Mass		Wing length		Avian haemosporidian parasites
					Uninfect	Infect	Uninfect	Infect	
1994	May	CC 04423	♂	A	47	—	93	—	—
		CC 08551	♂	A	—	46	—	90	*P. relictum*
		CC 08893	♂	A	—	51	—	93	*P. relictum*
		CV 01824	♂	A	51	—	94	—	—
		CC 10460	♂	A	48	—	95	—	—
		CC 10462	♂	A	49	—	91	—	—
		CC 10463	♂	A	51	—	93	—	—
		BD 15610	♀	A	41	—	82	—	—
		CC 10469	♂	A	53	—	92	—	—
		CC 10470	♂	A	—	49	—	94	*H. queleae*
		CC 10471	♂	A	48	—	93	—	—
		CC 10472	♂	A	50	—	95	—	—
		CC 10474	♂	A	49	—	90	—	—
		CC 10478	♂	A	45	—	92	—	—
		CC 10479	♂	A	45	—	93	—	—
		CC 10480	♂	A	49	—	93	—	—
		CC 10486	♂	A	50	—	92	—	—
		BB 98589	♂	A	49	—	95	—	—
		BB 99957	♂	A	48	—	94	—	—
		BC 20552	♀	A	42	—	88	—	—
		BC 20716	♀	A	38	—	87	—	—
		CC 08478	♂	A	49	—	94	—	—
		CC 08542	♂	A	49		93	—	—
		CC 08731	♂	A	—	48	—	93	*H. queleae*
		CC 10228	♂	A	—	50	—	92	*L. bouffardi*
		CV 03315	♂	A	43	—	92	—	—
		CC 10487	♂	A	48	—	95	—	—
		CC 10488	♂	A	47	—	94	—	—
		CC 10489	♂	A	49	—	96	—	—
		CC 10490	♂	A	51	—	90	—	—
		CC 10255	♂	A	52	—	94	—	—
		BD 12033	♀	A	40	—	87	—	—
		CV 03397	♂	A	52	—	94	—	—
1994	June	BD 15978	♀	A	43	—	86	—	—
		CC 10492	♂	A	47	—	93	—	—
		CC 10493	♂	A	48	—	94	—	—
		CC 10495	♂	A	—	53	—	96	*L. bouffardi*
		CC 10497	♂	A	49	—	93	—	—
		CC 10498	♂	A	51	—	96	—	—
		CC 08074	♂	A	43	—	92	—	—
		CC 08550	♂	A	48	—	95	—	—
		CC 08730	♂	A	46	—	91	—	—
		CC 08953	♂	A	53	—	96	—	—
		BC 03894	♂	A	49	—	95	—	—
		BD 06265	♂	A	49	—	96	—	—
		BD 06266	♂	A	50	—	93	—	—
		BD 15998	♀	A	36	—	88	—	—
		CC 10512	♂	A	51	—	97	—	—
		BB 94185	♀	A	42	—	86	—	—
		BB 98564	♂	A	47	—	92	—	—
		BC 03882	♂	A	49	—	92	—	—
		CC 08495	♂	A	50	—	94	—	—

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Year	Month	Ring number	Sex	Age	Mass		Wing length		Avian haemosporidian parasites
					Uninfect	Infect	Uninfect	Infect	
1994	June	CC 10249	♂	A	46	---	95	—	—
		BC 16778	♂	A	51	—	95	—	—
		CV 01819	♀	A	—	43	—	88	*P. relictum*
		CV 02390	♂	A	47	—	96	—	—
		CV 04143	♂	A	—	47	—	93	*P. relictum*
		CC 08550	♂	A	48	---	95	—	—
		BB 99813	♂	A	54	---	95	—	—
		BC 17309	♀	A	40	—	87	—	—
		BC 20744	♂	A	45	—	96	—	—
		BD 06736	♂	A	46	—	93	—	--
		BD 15226	♂	A	—	49	---	92	*H. queleae*
		CC 08962	♂	A	42	—	93	—	---
		CC 10363	♂	A	—	46	---	93	*H. queleae*
		CC 10473	♂	A	46	—	91	—	—
1994	July	BD 19092	♀	A	41	—	87	—	—
		CC 10528	♂	A	48	—	93	—	—
		CC 10536	♂	A	51	—	90	—	—
		CC 10541	♂	A	49	—	95	—	---
		BB 99834	♂	A	46	—	94	---	—
		BC 20531	♀	A	57	—	96	—	—
		BD 04880	♀	A	—	41	—	86	*P. relictum*
		BD 12994	♂	A	51	—	91	—	—
		BD 15323	♀	A	—	44	—	87	*H. queleae*
		CV 03317	♂	A	52	—	94	—	—
		CV 03365	♂	A	49	—	97	—	—
		CV 04143	♂	A	45	—	92	—	---
		CV 04147	♂	A	54	---	93	—	—
		CV 04158	♂	A	50	—	94		—
		CC 08144	♂	A	—	49	—	94	*L. bouffardi, P. relictum*
		CC 08867	♂	A	51	—	93	—	—
		CC 08955	♂	A	50	---	94	—	—
		CC 10466	♂	A	—	50	—	92	*L. bouffardi, P. relictum*
		CC 10607	♂	A	46	—	92	—	—
		CC 10612	♂	A	—	44	—	91	*H. queleae*
		BD 06250	♀	A	40	—	86	—	—
		BD 06663	♀	A	38	—	85	—	---
		BD 12442	♀	A	39	--	88	—	—
		BD 15137	♀	A	—	40	—	86	*H. queleae*
		BD 15185	♀	A	41	—	86	—	—
		CC 08977	♂	A	47	—	94	—	—
		CC 10395	♂	A	50	—	92	—	---
		CC 10499	♂	A	46	—	92	---	---
		CC 10463	♂	A	50	—	92	---	—
1994	August	BD 19305	♀	A	—	40	—	87	*H. queleae*
		BD 19319	♀	A	38	—	85	—	—
		BD 19320	♀	A	40	—	86	—	—
		BD 19321	♀	A	39	—	86	—	—
		CC 10614	♂	A	50	—	91	—	—
		CC 10618	♂	A	—	46	—	90	*H. queleae*
		CC 10620	♂	A	45	—	95	—	---
		CC 10621	♂	A	45	—	95	--	—
		BD 19323	♀	A	44	—	85	--	—

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Year	Month	Ring number	Sex	Age	Mass		Wing length		Avian haemosporidian parasites
					Uninfect	Infect	Uninfect	Infect	
1994	August	CC 10619	♂	A	50	—	93	—	—
		BB 99832	♂	A	47	—	94	—	—
		BC 20407	♀	A	41	—	87	—	—
		BC 22560	♀	A	40	—	85	—	—
		BD 04871	♂	A	—	49	—	94	*H. queleae*
		BB 99775	♂	A	49	—	92	—	—
		CC 08722	♂	A	47		91	—	—
		CC 08958	♂	A	46		93	—	—
		CC 10219	♂	A	—	46	—	93	*H. queleae*
		CV 02383	♂	A	48	—	92	—	—
		CV 03337	♂	A	50	—	97	—	—
		CV 04202	♂	A	46	—	93	—	—
		BB 99333	♀	A	44	—	88	—	—
		CC 08981	♂	A	—	52	—	94	*H. queleae*
		BD 19482	♀	A	40	—	86	—	—
		BD 19486	♀	A	—	41	—	87	*L. bouffardi P. relictum*
		CC 10655	♂	A	—	48	—	91	*H. queleae*
		CC 10656	♂	A	—	49	—	92	*L. bouffardi*
		CC 10657	♂	A	49	—	93	—	—
		CC 08134	♂	A	49	—	95	—	—
		CC 10224	♂	A	45	—	93	—	—
		BD 06521	♀	A	39	—	82	—	—
		CC 10393	♂	A	—	46	—	93	*H. queleae*
		BD 15133	♂	A	—	46	—	91	*H. queleae*
		BD 12981	♂	A	—	45	—	91	*H. queleae*
		BB 99793	♀	A	—	39	—	88	*H. quelcae*
		CC 10219	♂	A	—	43	—	93	*H. quelcae*
		CC 10288	♂	A	—	46	—	92	*H. quelcae*
		CC 08479	♂	A	50	—	95	—	—
		CC 10600	♂	A	—	48	—	91	*L. bouffardi*
1994	September	BD 19679	♀	A	38	—	85	—	—
		BD 19695	♀	A	42	—	86	—	—
		BD 19702	♀	A	45	—	86	—	—
		BD 19706	♀	A	—	42	—	86	*H. queleae*
		BD 19712	♀	A	—	50	—	87	*H. queleae*
		CC 10611	♂	A	45	—	91	—	—
		CC 10682	♂	A	47	—	92	—	—
		CC 10683	♂	A	—	47	—	95	*L. bouffardi, P. relictum*
		CC 10684	♂	A	—	49	—	96	*H. queleae*
		CC 10685	♂	A	—	51	—	95	*H. queleae*
		CC 10686	♂	A	—	44	—	93	*H. queleae*
		CC 10687	♂	A	49	—	94	—	—
		CC 10688	♂	A		47	—	90	*H. quelcae*
		CC 10689	♂	A	—	43		87	*H. quelcae*
		CC 10690	♂	A	—	50	—	93	*H. queleae*
		CC 10691	♂	A	49	—	95	—	—
		CC 10692	♂	A	48	—	96	—	—
		CC 10693	♂	A	—	50	—	93	*H. queleae*
		CC 10694	♂	A	46	—	90	—	—
		CC 10695	♂	A	—	49	—	94	*P. relictum*
		CC 10696	♂	A	46	—	92	—	—
		BB 99223	♂	A	44	—	86	—	—

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Year	Month	Ring number	Sex	Age	Mass		Wing length		Avian haemosporidian parasites
					Uninfect	Infect	Uninfect	Infect	
1994	September	BD 12672	♂	A	48	—	95	—	—
		BD 15268	♀	A	44	—	86	—	—
		CC 10269	♀	A	44	—	84	—	—
		CC 10369	♂	A	44	—	91	—	—
		CC 10389	♂	A	—	49	—	94	*L. bouffardi, P. relictum*
		CC 10398	♂	A	42	—	92	—	—
		CC 10452	♂	A	46	—	91	—	—
		CC 10485	♂	A	—	43	—	94	*H. quelcae*
		CC 10583	♂	A	47	—	92	—	—
		BD 19752	♀	A	44	—	86	—	—
		BD 19760	♀	A	43	—	87	—	—
		BD 19762	♀	A	49	—	87	—	—
		BD 19799	♀	A	44	—	87	—	—
		CC 10698	♂	A	50	—	92	—	—
		CC 10699	♂	A	50	—	92	—	—
		CC 10700	♂	A	51	—	93	—	—
		CC 13001	♂	A	50	—	91	—	—
		CC 13002	♂	A	50	—	90	—	—
		CC 10659	♂	A	47	—	94	—	—
		CV 03383	♂	A	—	53	—	96	*H. queleae*
		BD 19889	♀	A	43	—	87	—	—
		CC 13010	♂	A	43	—	92	—	—
		CC 13013	♂	A	49	—	94	—	—
		CC 10314	♂	A	46	—	92	—	—
		CC 13019	♂	A	—	48	—	91	*H. queleae*
		CC 13020	♂	A	—	49	—	94	*L. bouffardi*
		BB 99854	♂	A	—	49	—	92	*H. quelcae*
		BD 15368	♀	A	44	—	87	—	—
		CC 08602	♂	A	—	50	—	97	*H. quelcae*
		CC 10490	♂	A	48	—	92	—	—
		CC 10493	♂	A	—	47	—	95	*H. quelcae*
		CC 10676	♂	A	46	—	95	—	—
		CC 10681	♂	A	—	52	—	95	*H. queleae*
1994	October	BD 19144	♂	A	45	—	90	—	—
		CV 02345	♂	A	—	49	—	93	*H. queleae*
		CV 03407	♂	A	—	50	—	94	*H. queleae*
		CC 08413	♂	A	—	52	—	92	*H. quelcae*
		CC 08977	♂	A	—	47	—	93	*H. quelcae*
		CC 10580	♂	A	49	—	92	—	—
		CC 13027	♂	A	—	48	—	93	*H. queleae*
		CC 13028	♂	A	—	48	—	89	*H. queleae*
		CC 13029	♂	A	46	—	91	—	—
		CC 13031	♂	A	—	49	—	96	*H. queleae*
		CC 13035	♂	A	46	—	93	—	—
		BB 99967	♂	A	—	47	—	91	*H. queleae*
		CC 10564	♂	A	—	49	—	94	*H. queleae*
		BD 15602	♀	A	—	40	—	85	*H. queleae*
		BD 15564	♀	A	—	31	—	85	*L. bouffardi*
		BD 15276	♀	A	37	—	88	—	—
		CC 10262	♀	A	—	38	—	87	*H. quelcae*
		BD 15281	♀	A	—	42	—	86	*L. bouffardi, H. queleae*
		BC 17334	♀	A	39	—	88	—	—

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Year	Month	Ring number	Sex	Age	Mass		Wing length		Avian haemosporidian parasites
					Uninfect	Infect	Uninfect	Infect	
1994	October	BD 19702	♀	A	45	—	86	—	—
		BC 20407	♀	A	41	—	87	—	—
		BD 06521	♀	A	39	—	82	—	—
1994	November	CC 08870	♂	A	—	50	—	92	*H. queleae*
		BD 04871	♂	A	—	49	—	94	*H. queleae*
		CC 08983	♂	A	—	50	—	92	*L. bouffardi*
		CC 10686	♂	A	—	44	—	93	*H. queleae*
		BD 15325	♀	A	—	39	—	84	*L. bouffardi, H. queleae*
		BD 22514	♂	A	—	46	—	91	*L. bouffardi, H. queleae*
		BD 22515	♀	A	—	42	—	85	*L. bouffardi, H. queleae*
		BD 22518	♀	A	41	—	86	—	—
		BD 15191	♀	A	39	—	86	—	—
		BD 15280	♀	A	40	—	86	—	—
		BD 15364	♀	A	40	—	88	—	—
		BD 15058	♀	A	42	—	86	—	—
		BD 22525	♀	A	—	38	—	83	*H. queleae*
		CC 13121	♂	A	—	42	—	93	*L. bouffardi, H. queleae*
		BD 10400	♂	A	52	—	92	—	—
		BB 99934	♂	A	50	—	97	—	—
		BD 12994	♂	A	51	—	91	—	—
1995	January	CC 13227	♂	A	—	50	—	89	—
		CC 13237	♂	A	—	50	—	89	*H. queleae*
		CC 10504	♂	A	48	—	92	—	—
		BD 06452	♀	A	40	—	85	—	—
		CC 08965	♂	A	49	—	91	—	—
		CC 13240	♂	A	—	48	—	91	*L. bouffardi*
		CC 13243	♀	A	—	41	—	89	*H. queleae*
		BB 99877	♀	A	—	41	—	83	*L. bouffardi*
		CC 08439	♂	A	—	48	—	91	*H. queleae*

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Cape Weaver *Ploceus capensis* against a background depicting a microscopical view containing avian haemosporidian parasites: *Leucocytozoon majoris*, *L. smithi* and *Haemoproteus danilewskii*, interspersed among avian erythrocytes. In nature *Leucocytozoon* and *Haemoproteus* species are avian order specific. Therefore these parasites belonging to different orders would not be present within a single blood smear from a single individual avian host.