BLOOD PARASITES IN PENGUINS, AND THEIR POTENTIAL IMPACT ON CONSERVATION

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SUMMARY

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This paper reviews the reported blood parasites from wild and captive penguins, discusses the dynamics of haematozoan infection, considers factors which could alter the present equilibrium, draws attention to potential risks from exposure to intensified or introduced infection, and suggests standardized methodologies to increase understanding and facilitate timely detection of any changes in infection dynamics.

INTRODUCTION

Birds may be host to a number of species of blood-inhabiting protozoa and nematode worms transmitted by haematophagous arthropods. Protozoan parasites include haemosporidia in the genera *Plasmodium*, *Haemoproteus*, *Leucocytozoon*, *Hepatozoon,* and *Babesia,* and haemoflagellates in the genus *Trypanosoma*. Most families of birds are susceptible to blood protozoal infection in the wild (Bennett *et al.* 1982) and prevalence, especially in the tropics, may be high (>30%, Jones 1985). Nematode worms in the family Filariidae occur in many species of birds, but they have not been reported from penguins, and will not be considered further in this paper.

Haematozoa vary in their host specificity, both for the vertebrate host and for the arthropod vectors. Some are able to survive and reproduce in a wide range of birds and of arthropods, others are confined to a narrow range of host species. The arthropod vector plays an integral role in the parasites' life cycles. Vectors frequently have host preferences, but may feed on other hosts when the preferred one is unavailable. Pathology of infection with haematozoa in wild birds is difficult to study, and there are few reports of mortality in wild birds (Bennett *et al*. 1993).

Blood-inhabiting parasites of penguins are of particular interest for several reasons. Although predominantly sub-Antarctic in distribution, several species breed at low latitudes in temperate environments, and may come into contact both with a number of potential arthropod vectors, and with flying bird species which may provide a reservoir of infection. Although few parasites have been recovered from penguins in their natural habitats, there are many reports of infection with *Plasmodium* spp. from penguins species in captivity, where they may be exposed to new pathogens which may result in high morbidity or mortality (Stoskopf & Beier 1979, Fix *et al.* 1988). The potential of haematozoa to cause disease in endangered or localized isolated populations of birds has been discussed by Peirce (1989).

BLOOD PARASITES IN WILD PENGUINS

Five species of protozoan parasite have been reported from penguins in their natural habitats (reviewed by Clarke & Kerry 1993) – *Leucocytozoon tawaki* in Fiordland Crested Penguins *Eudyptes pachyrhynchus* (Fallis *et al*. 1976), *Plasmodium relictum* in Fiordland Crested Penguins and Yellow-eyed Penguins *Megadyptes antipodes* (Laird 1950), African *Spheniscus demersus* and Rockhopper *Eudyptes chrysocome* Penguins (Fantham & Porter 1944), *Babesia peircei* in African Penguins (Earlé *et al.* 1993), and *Trypanosoma eudyptulae* in Little Penguins *Eudyptula minor* (Jones & Woehler 1989). Intensity and prevalence of these infections have usually been low. All these reports have been from temperate localities in South Africa, Australia and New Zealand, and in the South Atlantic (Gough Island). There have been reports of negative blood examinations from 12 penguin species in the wild, totalling more than 700 birds, from 14 localities (Jones & Shellam 1999). All records from penguins in the Antarctic or sub-Antarctic have been negative.

BLOOD PARASITES IN CAPTIVE PENGUINS

Susceptibility to patent infection may differ between penguins which have been transferred to a new environment with the potential of exposure to parasites for the first time, and those which are in captivity within their natural environment, such as birds being rehabilitated after oiling. In the latter case, stress may make patent a previously undetectable parasitaemia (Brossy 1992, Graczyk *et al*. 1995). Nine species of penguins (King *Aptenodytes patagonicus*, Rockhopper, Macaroni *Eudyptes chrysolophus*, Little, Chinstrap *Pygoscelis antarctica,* Gentoo *P. papua*, African, Humboldt *S. humboldti* and Magellanic *S. magellanicus*) have been recorded in captivity infected with *Plasmodium relictum, P. elongatum or P. cathemerium* (references in Bennett *et al*. 1982). In addition, the African Penguin has been reported infected with *Leucocytozoon tawaki* in captivity (Earlé *et al.* 1992), and *Babesia* sp. reported from Little Penguins held in a research station in Australia (Cunningham *et al*. 1993). However, the number of negative examinations undertaken on captive penguins is unknown, and we can locate no reports of examinations of Emperor *A. forsteri*, Fiordland Crested, Snares Crested *E. robustus*, Royal *E. schlegeli*, Erect Crested *E. sclateri*, Yellow-eyed *Megadyptes antipodes*, Adélie *P. adeliae*, or Galapagos *S. mediculus* Penguins in captivity. Infection of

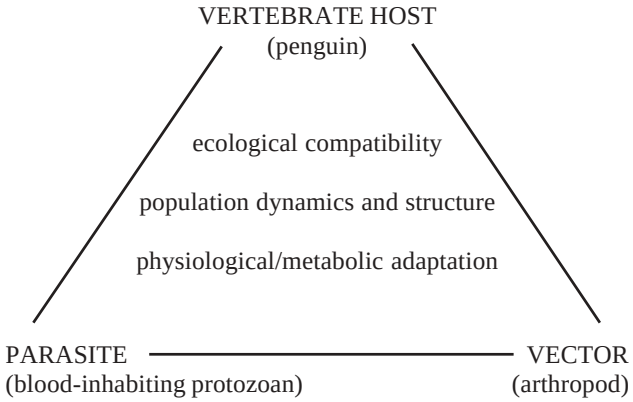

Fig. 1. *Dynamics of haematozoan infection.*

captive penguins is probably more widespread than published records indicate; histological material can be difficult to interpret, and malarial schizonts in histopathological material from captive penguins have frequently been misdiagnosed as being those of *Toxoplasma* (M. Peirce pers. comm.).

DYNAMICS OF HAEMATOZOAN INFECTION

Parasitic infection depends on several factors: the presence of suitable vectors, the presence of compatible parasites within the ecosystem, feeding preferences of vectors, the population density of penguin hosts, and opportunities for vectors to feed. Parasites have varying degrees of specificity both for their vertebrate hosts and for the invertebrate vectors, and the feeding preferences of the vectors will influence their suitability in transmitting a parasite. Availability of suitable arthropods within the range and habitat of the vertebrate hosts, the numbers and life span of both vertebrate and invertebrate hosts, and the presence or absence of reservoir hosts of other species can affect the probability of infection occurring (Fig. 1).

Insects in the Order Diptera and ticks and mites (Acarina) are the only known vectors and intermediate hosts of avian blood-inhabiting protozoa. *Plasmodium* spp. infecting birds are transmitted by *Aedes* and *Culex* mosquitoes, *Leucocytozoon* spp. by simuliid blackflies and ceratopogonid midges (*Culicoides* spp.), *Babesia* spp. by ticks, and *Trypanosoma* spp. by simuliid blackflies, ceratopogonid midges, mosquitoes, hippoboscid flies and *Dermanyssus* mites. However, no life cycles of blood parasites in penguins have been fully elucidated, nor have the vectors of most species infecting penguins been confirmed. The vectors referred to above are most widespread in tropical and temperate regions. No biting flies are known from sub-Antarctic islands or from Antarctica (Block 1984), although the tick *Ixodes uriae* is found on many polar birds (Zumpt 1952), including penguins, and is the putative vector of *Hepatozoon albatrossi* in three species of albatrosses (*Diomedea* spp.) at South Georgia (Peirce & Prince 1980).

The specificity of blood-inhabiting protozoa for their avian hosts exercises crucial constraints on infection. The host-specificities of *Plasmodium* species are variable, though *P. relictum* has a very low specificity and is known to infect a large number of bird species (Garnham 1966). *Leucocytozoon* species however are host family specific, and *Babesia* species, which are principally parasites of mammals, are also probably family specific. *Trypanosoma* are pleomorphic, so that one species may exist in several different forms. Their taxonomy is therefore difficult to study using purely morphological criteria, but they probably have low specificity (Baker 1976). *Haemoproteus* is the most widespread genus of avian blood protozoan, but it has never been reported from penguins either in the wild or in captivity.

Since many species of penguin breed in temperate latitudes, they have the chance of coming into contact with potential vectors. The low prevalence and intensity of infection probably reflects aspects of the penguins' ecology, such as long absence at sea, and is in contrast to the many reports of *Plasmodium* spp. infection in captive penguins, with much morbidity and mortality. This raises the question of the origin or acquisition of the penguins' natural parasites.

Penguins are an ancient stock of marine birds which probably originated in cool southern regions, inimical to biting arthropods, their evolution coinciding with modern Southern Ocean circulation patterns (Fordyce & Jones 1990). Thus as a group they would have been relatively isolated from exposure to the diversity of avian parasites at lower latitudes. The ancestors of the four genera of haematozoa which infect penguins, now usually occurring at low prevalence and intensity, and usually sporadically, were probably acquired from contact with unrelated sympatric birds, and they evolved to become penguin-specific. Cross-infection experiments would be needed, however, to assess the extent of host-specificity of haematozoa recorded in penguins. It is probable that penguins never experienced the intense and continuous exposure to the diversity of parasites and vectors which occurs in temperate and tropical birds, and thus had limited opportunity to develop resistance. When they were exposed through transport to places in which there were reservoirs of infected birds and thus the opportunity for more intense transmission, this absence of inherited resistance resulted in often overwhelming infection with low host-specific *P. relictum*; the fact that a similar scenario does not occur with the other three parasite genera reflects the degree of their host-specificity, and consequent inability to cross host boundaries.

POTENTIAL RISKS OF PARASITIC INFECTION TO HEALTH OF WILD PENGUINS

Massive killing of many species of penguin for oil or food occurred during the Nineteenth Century and earlier this century, seriously affecting local populations (Cumpston 1968). However, most penguin species are now subject to legal protection, and in some colonies numbers have been increasing steadily since protection (e.g. Budd & Downes 1965, Budd 1968, 1970). As well as reduced predation, other factors such as food availability may have been involved (Boersma *et al*. 1990). However, there are now other insidious anthropogenic threats to the well-being and stability of wild penguin populations, and one such threat to be considered, and which has been hitherto been insufficiently studied, relates to arthropod-borne diseases.

There is ample evidence of the effects of infection with *Plasmodium* spp. on captive penguins (Fix *et al*. 1988). There have been records of unexplained mass deaths of wild penguins (Kerry *et al.* 1996, Splettstoesser 1997), but protozoal infections have not been implicated. Although there have been no reports of pathology from parasitic infections in wild penguins, pathology from infections is uncommonly reported from wild birds, presumably because such birds are readily taken by natural predators (Bennett *et al*. 1993).

In the absence of adequate data, the extent of the risk of introduced or exacerbated arthropod-borne protozoal infections to penguin populations as a result of environmental and other changes is not known. We consider several possible factors, directly or indirectly anthropogenic, which have the potential to produce or increase exposure to, or decrease resistance to, exotic pathogens.

Firstly, climate change may enable the range or density of invertebrate vectors to be increased. This may be particularly relevant where penguins breed on large land masses on which arthropod vectors occur at lower latitudes, and which with little climate amelioration could increase their range southwards without the necessity of crossing a sea barrier. Colonies of penguins which breed on the South American and African mainlands may be at particular risk in this respect. There is also the concomitant risk of their parasites' range increasing, since these organisms, like the parasites themselves, reproduce more rapidly at higher temperatures. It is possible for instance that the widespread ixodid ticks in polar birds could be potent vectors of parasites to which they are not normally exposed; Earlé *et al.* (1993) suspected that *I. uriae* may be implicated in the transmission of *Babesia peircei* in the African Penguin; however, *Ornithodorus* ticks are widespread on African Penguins in South Africa, and no ixodid ticks have been recovered from this host (J.-J. Brossy pers. comm.). Secondly, increased ultraviolet radiation from ozone depletion may depress immune responsiveness (Jeevan & Kripke 1993, Armstrong 1994). Thirdly, increasing exposure of penguins to environmental pollutants may compromise their immune systems and make them more susceptible to infective challenge (Luebke *et al.* 1997). Fourthly, food availability may be reduced by El Nino Southern Oscillation events (Schreiber & Schreiber 1984), or by more intense commercial fishing in their feeding grounds (Woehler 1995) and hence decrease resistance to infective challenges. Fifthly, increasing disturbance of their breeding habitats, whether by tourists or by aircraft (Cooper *et al.* 1994), engenders stress which may increase susceptibility to disease if the pathogens are present. If a penguin has subpatent *Plasmodium* infection, stress is probably a potent factor in increasing parasitaemia and hence pathogenicity in penguins soon after capture (Brossy 1992). It is possible that various of these factors could act together and reinforce one another, and it is therefore vital that sound base-line data are obtained on the host-specificity, infectivity, vectors and pathology of these parasites, so that potentially adverse changes to penguins' biotopes can be identified early, closely monitored, and the true risks from infective exposure accurately gauged.

METHODOLOGIES

In view of the susceptibility of captive penguins to *Plasmodium* spp. infections and the dearth of information on the effects of other haematozoan genera on the health of penguins, there is a need for more extensive sampling of wild penguin populations, especially those in temperate mainland Argentina and Chile. So that valid comparisons can be made between populations and over time, methods of collection, storage, preservation, and examination techniques ideally should be standardized. However, in reality the logistics of collection and storage from remote and inaccessible locations, varying experience and expertise of collectors, and of those examining stained preparations, make true standardization difficult. Methods of collection, storage, preservation and examination should be recorded in all publications. Adherence to the points detailed in Table 1 would result in the accumulation of more reliable data upon which to base future research and conservation strategies.

Penguins form a major component of the biomass of the Southern Ocean, and in the case of the genus *Pygoscelis* they dominate the Antarctic biomass (Prévost 1981). They thus have a central role in the ecology of the Southern Ocean, the effects of major perturbations of which are speculative. We therefore have a responsibility and an obligation to ensure that changes to their environment do not include the introduction of diseases to which penguins are not at present exposed or to which they demonstrate limited resistance.

CONCLUSIONS

Plasmodium, Babesia, Leucocytozoon and *Trypanosoma* have been recorded in five species of wild penguins, all from temperate sites. Infections generally have been of low prevalence and intensity, and there is no published evidence of ill-effects of these parasites in wild penguins. *Plasmodium* spp., *Leucocytozoon tawaki* and *Babesia peircei* have been recorded in captive penguins; infections with *Plasmodium* spp. are frequently heavy with commensurate morbidity or mortality, although deaths may occur before parasites can be detected in the erythrocytes. We conclude that infection in wild penguins is limited by the presence of suitable arthropod vectors and by their feeding preferences, and by the specificity of the parasites for the avian host. Knowledge of the evolution of penguins suggests that opportunities for adaptation of parasites to these birds has been limited by their cool climate origins. We draw

TABLE 1

Parameters to be recorded in studying penguin blood parasites

- Where sampled; free-living or captive
- Colony size
- Health, sex and age of bird if possible
- Date of sample collection
- Time between collection and staining
- Fixation and staining techniques used
- Magnification and number of microscope fields examined
- Prevalence of infection (or number examined if all negative)
- Intensity of infection (parasites per field, or per red blood cell)
- In captive penguins – size of captive population; length of time in captivity; origin of birds
- Positive specimens should be deposited in a reference collection

attention to a suite of possible or actual environmental changes, all of which are likely to intensify in the penguins' biotopes, and we make a case for the urgent need for more studies of penguin responses to arthropod-borne infection under natural and captive conditions, and for the need for geographically comprehensive baseline data. We suggest protocols to ensure more standardized methodologies for collection and preservation of blood specimens.

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