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Diseases and Parasites of Penguins

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The study of diseases in wild penguins is important for the identification of endemic diseases and the detection of exotic diseases should these occur. It is also important in the understanding of the degree to which disease may be expected to influence the results of biological studies. Results may be confounded and interpretation made difficult by both the transient and long term presence of disease particularly if it is at the sub clinical level. We present here a compilation of diseases and parasites recorded for all species of penguins present both in the wild and in captivity. Normal values for blood biochemistry and haematology are given as an aid to the identification of illness in penguins.

Key words: diseases, parasites, penguins

INTRODUCTION

There appear to be few studies and no statistics on the incidence of disease among penguin populations in the wild despite extensive biological studies of all species. The reports by Harrigan (1988) and Cunningham et al (1993) on little penguins (*Eudyptula minor*) are the most comprehensive investigations of disease for any penguin species in the wild. Disease in wild birds is generally diagnosed *post mortem*. There are however reports of individual diseases, parasites and pathogens scattered throughout the literature which enable a broad picture of the pathology of penguins in the wild to be assembled.

Much of the pathology of penguins has been reported from birds maintained in captivity for which there is an extensive literature, for example Stosfkopf *sic* and Beall (1980), Davis (1967), Fiennes (1967), Todd (1978), Gailey-Phipps (1967, 1978). Many of the disorders associated with captivity are due to locally-occurring pathogens which may not be experienced by birds in the wild.

This paper attempts to bring together knowledge on the diseases and parasites of penguins both in the wild and in captivity for use in conservation and management. Penguins in zoos provide very popular displays; but these are provided at considerable cost (Olney 1978). Keeping captive birds healthy is obviously very important and in the long term may have implications for survival of some species which could become extinct in the wild. The study of diseases in wild penguins enables the identification of endemic disease and the detection of exotic pathogens, and provides information on which to fashion regulations to protect both penguins and other avian species. It is important in the monitoring of possible impacts of human activity especially fishing (*see* below). The results of monitoring may be confounded by the occurrence of disease particularly at the subclinical level.

We present also a summary of constituents of blood of normal birds in the wild which may be of use in identification and diagnosis of disorders. Information is drawn from the literature and supplemented with data from our study of Adélie penguins (*Pygoscelis adeliae*) near Mawson, Antarctica.

PARASITES AND DISEASES

The following compilation provides a broadly based summary and discussion of parasites and diseases reported from all species of penguin and for which information is available readily in the literature. The list is comprehensive rather than exhaus-

Table 1. Ectoparasites of penguins. Most references are taken from Murray *et al.* (1991). This work should be consulted for the primary references

Ectoparasites	Penguin species affected	References
Ticks		
Ixodes kohlsi	little	Harrigan (1992), Obendorf & McColl (1980)
Ixodes uriae	rockhopper, royal, king, macaroni, Magellanic	Hawkey *et al.* (1989),Murray & Vestjens (1967), Zumpt (1952)
Ixodes uriae	emperor, gentoo, Adelie, Snares crested, little	Murray *et al.* (1991)
Ixodes eudyptidus	little	Murray *et al.* (1991)
Ixodes kohlsi	little	Murray *et al.* (1991)
Ixodes percavatus	little	Reilly & Balmford (1975)
Ornithodoros carpensis	little, African	Murray *et al.* (1991), Hoogstraal *et al.* (1985)
Ornithodoros spheniscus	Humboldt	Hoogstraal *et al.* (1985)
Ornithodorus yunkai	Galapagos	Hoogstraal *et al.* (1985)
Biting lice		
Austrogoniodes antarcticus	Adelie	Murray *et al.* (1991)
Austrogoniodes breviceps	king	Murray *et al.* (1991)
Austrogoniodes bicornutus	macaroni	Murray *et al.* (1991)
Austrogoniodes concii	rockhopper, Fiordland, Snares crested, Erect-crested, yellow-eyed	Murray *et al.* (1991)
Austrogoniodes cristata	rockhopper, Fiordland, Snares crested, Erect-crested, macaroni, royal	Murray *et al.* (1991)
Austrogoniodes strutheus	erect-crested	Murray *et al.* (1991)
Austrogoniodes mawsoni	emperor	Murray *et al.* (1991)
Austrogoniodes gressitti	gentoo, chinstrap	Murray *et al.* (1991)
Austrogoniodes hamiltoni	rockhopper, royal	Murray *et al.* (1991)
Austrogoniodes macquariensis	rockhopper, macaroni,royal	Murray *et al.* (1991)
Austrogoniodes waterstoni	little	Crockett & Kearns (1975), Harrigan (1992), Murray (1964), Obendorf & McColl (1980)
Nesiotinus demersus	king	Murray *et al.* (1991)
Mites		
	little	Crockett & Kearns (1975)
Fleas		
Listronius robertsianus	Magellanic	de Meillon (1952)
Parapsyllus longicornis	little, rockhopper, gentoo, macaroni, Magellanic	de Meillon (1952), Obendorf & McColl (1980)
Parapsyllus longicornis	yellow-eyed	Murray *et al.* (1991)
Parapsyllus australiacus	little	de Meillon (1952), Obendorf & McColl (1980)
Parapsyllus heardi	rockhopper, macaroni	Murray *et al.* (1991)
Parapsyllus jacksoni	little	Murray *et al.* (1991)
Parapsyllus taylori	little	Reilly & Balmford (1975)
Parapsyllus magellanicus heardi	rockhopper	Murray & Vestjens (1967)
Parapsyllus magellanicus	Magellanic	de Meillon (1952)

tive. Symptoms, diagnosis and treatment are not discussed and the reader is referred to the literature. Additional information from the early literature may be obtained from the bibliography compiled by Williams *et al.* (1985). There are many areas of taxonomic difficulty, particularly in relation to the identity of invertebrate parasites listed in the various research and taxonomic papers. Data are derived from a wide variety of sources and for this reason taxonomic inferences should not be

drawn. So far as possible synonyms have been avoided and the most recent nomenclature used to identify invertebrate species and other micro-organisms. Where a major revision of the group of organisms has occurred reference is made to both the original paper and the revision.

A distinction is made between birds found in the wild exhibiting disease and those held in captivity. Further, birds which were captured in the wild (wild caught) but exhibited symptoms of disease or the presence of infectious agents soon after being taken into captivity are identified. The source of infection in these cases is not known.

Ectoparasites

Fleas and ticks are both common ectoparasites on penguins in the wild (Table 1).

The tick, *Ixodes uriae*, has a wide distribution, being found on most subantarctic islands and the Antarctic Peninsula (Murray, 1967). It is found also associated with other species of seabird in the northern hemisphere. At Macquarie Island *I. uriae* are found most commonly on royal (*Eudyptes chrysolophus schlegeli*) and rockhopper (*Eudyptes crestata*) penguins. Chicks are most frequently infested. The ticks are found around the eyes, at the commissures of the mouth, on the webs between the toes and around the cloaca. Gentoo penguins (*Pygoscelis papua*) are less commonly infested due to the non-permanent nature of their nests. Royal penguins nest densely thus providing the best food source for ticks. Heavy infestations may kill or contribute substantially to the death of penguin chicks and some adults. Ticks require the nests of their hosts to be sheltered and well drained.

Ticks of the genus *Ornithodorus* are found on penguins from tropical and temperate regions, including Humboldt (*Spheniscus humboldti*), Galapagos (*Spheniscus mendiculus*), African[1] (*Spheniscus demersus*) and little penguins (*Eudyptula minor*). It has been suggested (Hoogstraai *et al.*, 1985) that irritation produced by the feeding of numerous *O. (Alectorobius) spheniscus* upon Humboldt penguins may cause the birds to abandon their eggs.

Parapsyllus species of fleas are found on many penguins. The flea, *P. magellanicus heardi,* breeds on Macquarie Island in sheltered nests of the rockhopper penguin; only the adult flea visits the bird to breed. These fleas are seen easily on the brood patches of affected penguins (Murray and Vestgens, 1967).

Fleas have not been found on Antarctic penguin species since they spend parts of their life cycles off the host and thus need suitable nest environments with shelter from the weather.

Biting lice have been found on all subantarctic and Antarctic penguins except the gentoo and chinstrap (*Pygoscelis antarctica*), and live permanently on the birds. Penguin lice are similar to other bird lice and live and breed on the bird. They do not suck blood but rather eat feathers or skin debris (Murray, 1964).

Endoparasites

Nematodes, cestodes and trematodes have been reported in many species of penguin (Table 2). It should be noted that the taxonomy of some species is under revision and the identification of others is problematical since they have been identified from juvenile stages only. Some species may also be accidental occurrences due to being ingested in the food. The developmental cycle for any species of cestode in penguins has not been determined nor the intermediate hosts identified (Zdzitowiecki, 1993).

Endoparasite burdens tend to be heaviest in juvenile birds and, in all penguins for example little penguins (Norman *et al.*, 1992), contribute to mortality when combined with starvation and other forms of stress.

Nematodes are the most commonly found gastrointestinal parasites, and several species have been documented. *Stegophorus paradeliae* has been found in Adélie, rockhopper, gentoo and macaroni penguins (*Eudyptes chrysolophus*). *Contracaecum heardi* parasitises rockhopper, gentoo, king (*Aptenodytes patagonicus*) and macaroni penguins, and *Stomachus* species are found in gentoos (Mawson, 1953).

The trematode *Mawsonotrema eudyptulae* is known to contribute to mortalities in little penguins through damage to the liver. *Cardiocephaloides physalis* was reported to cause death in the chicks

[1] Also known as the black-footed (blackfooted) or jackass penguin

Table 2. Internal parasites of penguins

Internal parasites	Penguin species	Part of body affected	Location	References
Cestodes				
Tetrabothrius sp.	little	intestine	Southern Australia, Tasmania	Harrigan (1992), Norman *et al.* (1992),Oberdorf &McColl(1980)
	Magellanic	intestine		Fix *et al.* (1988)
Tetrabothrius ? *wrighti*	Emperor	intestine		Prudhoe (1969)
Tetrabothrius pauliani	king	intestine	Heard Is.	Prudhoe (1969)
Tetrabothrius pauliani	gentoo	intestine	Heard Is., Kerguelan Is.	Prudhoe (1969)
Tetrabothrius lutzi	magellanic	intestine	Brazil, Juan Fernandez Is.	Prudhoe (1969)
Tetrabothrius lutzi	little	intestine	Tasmania	Prudhoe (1969)
Parochites zederi	Adelie, emperor		Antarctic pack ice zone	Prudhoe (1969)
Parochites zederi	rockhopper		Macquarie Is.	Johnston (1938)
Nematodes				
Stegophorus macronectes (syn.: *S. paradeliae*: see Zdzitowiecki & Drodz(1980))	Adelie	GIT	Antarctica	Johnston & Mawson (1945)
Stegophorus macronectes	rockhopper, gentoo, macaroni	GIT	Heard Is.	Mawson (1953)
Stomachus spp.	gentoo, royal	stomach	Macquarie Is.	Mawson (1953)
Contracaecum heardi	rockhopper, gentoo, macaroni	GIT		Mawson (1953)
Contracaecum antarcticum (larvae only, adult unknown)	gentoo, emperor			Johnston & Mawson (1945)
Contracaecum eudyptes	rockhopper, yellow-eyed			Mawson *et al.* (1986)
Contracaecum eudyptulae	little			Mawson *et al.* (1986)
Contracaecum rudolphii (syn.: *C. spiculigerum*)	little	stomach		Harrigan (1992), Norman *et al.* (1992), Oberdorf & McColl (1980)
Contracaecum spp.	king, royal	GIT	Heard Is./Macquarie Is.	Johnston & Mawson (1945)
Anisakis spp.	rockhopper	GIT	Southern Australia, Tasmania	Mawson *et al.* (1986)
Filariodea	little	heart	Southern Australia	Mawson *et al.* (1986)
Corynosoma	little	GIT	Southern Australia	Mawson *et al.* (1986)
Cosmocephalus obvelatus	rockhopper	Oesophagus	Zoo Japan	Azuma *et al.* (1988)
Tetrameres wetzeli	rockhopper	proventriculus	Kerguelen Is.	Schmidt (1965)
Trematodes				
Galactosomum angelae	little	intestine, liver	South Eastern Australia	Oberdorf & McColl (1980)
Renicola sp.	little	liver	South Eastern Australia	Oberdorf & McColl (1980), Harrigan (1988)
Mawsonotrema eudyptulae	little	bile duct, liver	South Eastern Australia	Harrigan (1992), Norman *et al.* (1992)
Fam. Echinostomatidae possibly *Echinostoma* or *Hydrodermia*	little	kidney		Crockett & Kearns (1975)
Cardiocephaloides physalis (syn.: *C. szidati* Hartwick, 1954)	Magellanic			Lutz (1926)
Cardiocephaloides physalis (syn.: *Ditto*)	Humboldt			Dubois & Rausch (1960)
Cardiocephaloides physalis (syn.: *Ditto*)	African	intestine	South Africa	Randall & Bray (1983)

of African penguins through infestation of the small intestine (Randall and Bray, 1983). Renal flukes of unknown species have been found in healthy little penguins and appear not to cause serious disease (Crockett and Kearns, 1975).

Protozoans

A variety of protozoan species have been record-

ed infecting penguins (Table 3) but of these only a few produce symptoms of disease at the clinical level. Most do not appear to be serious pathogens but may render birds more susceptible to secondary infections or sensitive to stress factors such as weather or poor nutrition. All are spread by arthropod vectors particularly ticks, and in the case of malaria by mosquitos.

No blood parasites have been found in smears from penguins in Antarctic or subantarctic regions. Species investigated include Adélies, emperors (*Aptenodytes forsteri*), chinstraps, gentoos, kings, royals, rockhoppers and macaronis (Jones, 1988). These negative findings are attributed to lack of suitable vectors in cold climates since blood parasites are known to occur in penguins from more temperate regions.

Jones and Woehler (1989) demonstrated the presence in low numbers of the trypanosome, *T. eudyptula*, in the blood of little penguins. The actual vector of these parasites is unknown but potential organisms include mites, mosquitoes, flies and ticks. Avian trypanosomes rarely cause harm to their hosts, and no evidence of ill effects have been observed in infected penguins.

Malaria due to *Plasmodium relictum* has been reported in a wide variety of captive penguins and *P. elongatum* has been observed in wild and captive African and Humboldt penguins. Both *Plasmodium* species have been found together in some captive African penguins (Beier and Stoskopf, 1980). *Plasmodium cathemerium* has also been recorded in a king penguin (Luera-Carbo, 1965 cited by Bennett *et al.*, 1993). Avian malaria is transmitted by mosquitos and is an important cause of mortality in penguins in zoos in North America and Europe where it is maintained in the wild bird-mosquito cycle (Cranfield *et al.*, 1991). *Plasmodium* species appear to be absent from birds in the Australasian region (Bennett *et al.*, 1993). Penguins become infected when interjected into an area of endemic transmission (Beier and Stoskopf, 1980). Malaria been successfully treated with primaquine and chloroquine in zoo environments and birds which survive rarely develop symptoms thereafter; however, some individuals carry the parasite for many years after initial infection and treatment (Cranfield *et al.*, 1991).

The avian blood parasite, *Leucocytozoon tawaki*, has been found in wild Fiordland crested penguins (*Eudyptes pachyrhynchus*) in New Zealand (Fallis *et al.*, 1976). The vector is a simuliid fly *Austrosimulium ungulatum*. A little penguin also became infected when exposed to simuliid flies alongside an infected Fiordland penguin. *Leucocytozoon tawaki* does not appear to be a serious pathogen of these penguins. A leucocytozoon also believed to be *L. tawaki*, has been recorded in blood smears from African penguins (Earlé *et al.*, 1992).

Lethal toxoplasmosis (*Toxoplasma gondii*) has been reported in a little penguin held for rehabilitation in Australia (Mason *et al.*, 1991). Toxoplasmosis is a common parasitic disease of domestic sheep in Australia and it is likely that infection occurred through the sheep meat which was fed to the penguin for several weeks (Mason, pers. comm.). Toxoplasmosis was also reported in penguins by Ratcliff and Worth (1951). However Fleischman *et al.* (1968) suggest that these birds may in fact have had malaria since the organisms are difficult to separate at certain stages in their life cycles.

Babesiosis, *Babesis pieircei*, is endemic in African penguins but does not cause clinical signs or morbidity. However, it may contribute to illness in combination with other agents such as malaria or leucocytozoonosis (Brossy, 1993). A piroplasmid, possibly *Babesia*, has also been found in little penguins in Australia. It is tick borne and causes mild regenerative anaemia in juveniles (Cunningham *et al.*, 1993; Cunningham, pers. comm.).

Viral Diseases (Table 3)

Avian paramyxoviruses (APMV) are widespread among Adélie penguins in Antarctica and also subantarctic royal penguins (Morgan and Westbury, 1988; Morgan *et al.*, 1978). Antibodies to Newcastle disease virus (NDV) have been demonstrated in serum from Adélie, royal, and little penguins (Morgan and Westbury, 1981; Morgan *et al.*, 1978). The significance of these antibodies is uncertain; however, it is known that penguins are susceptible to pathogenic virus strains since disease has occurred in Adélie penguins believed to have become infected in the wild (Pierson and Pfow, 1975) and in a captive king penguin (Krauss *et al.*, 1963).

Nonpathogenic paramyxovirus strains have been

Table 3. Micro-organisms isolated or identified through the presence of antibodies in penguins. The terms "wild" and "captivity" indicate where the birds were living when the disease was manifest or evidence of disease was detected. Birds designated "wild-caught" indicates that the disease or infective agent was present or caught by the bird while in the wild state and that the disease became manifest shortly after the birds was taken into captivity. The identification of a pathogenic organism does not necessarily mean that symptoms of disease are manifest

Microorganism	Disease	Penguin species affected	Wild or captive	Vector	Part of body infected	References
Protozoan						
Coccidia	coccidiosis	little			kidney ,small intestine	Harrigan (1988), Harrigan (1992), Obendorf & McColl (1980)
Sarcocystis spp.		gentoo, chinstrap	wild		skeletal muscle	Ippen *et al.* (1981)
Plasmodium elongatum	Malaria	Humboldt	wild caught	Mosquitos	parenchymal organs and blood	Huff & Shiroishi (1962)
Plasmodium elongatum	Malaria	African, Humboldt, Magellanic, macaroni	captive	Mosquitos	parenchymal organs and blood	Fleischman *et al.* (1968),Herman *et al.*. (1968), Stoskopf & Beier (1979), Beier & Stoskopf (1980), Bak *et al.* (1984)
P. relictum	Malaria	African, Humboldt, little, rockhopper, Fiordland, chinstrap, king, macaroni	captive	Mosquitos	parenchymal organs and blood	Fantham & Porter (1944), Fix *et al.* (1988), Griner & Sheridan (1967), Beier & Stoskopf (1980), Rodhain & Andrianne (1952), Earle *et al.* (1992), Sladen *et al.* (1979)
P. relictum	Malaria	African, yelloweyed, rockhopper, chinstrap	wild	Mosquitos	parenchymal organs and blood	Fantham & Porter (1944), Fix *et al.* (1988), Laird (1952), Earle *et al.* (1992)
		Magellanic	wild caught	Mosquitos	parenchymal organs and blood	Fix *et al.* (1988)
P. carthemerium		king				Bennett *et al.* (1993), Luera-Carbo (1965)
Aegyptianella		African	captive (?)			Berson (1964)
Leucocytozoon tawaki		Fiordland, African	wild	Simuliid flies		Fallis *et al.* (1976), Allison *et al.* (1978), Earle *et al.* (1992)
Babesia pierci	Babesiosis	African	wild	ticks	Alone none,but may aggravates other disease eg malaria	Brossy (1993), Earle *et al.* (1993)
Piroplasmid possibly *Babesia* spp.		little	wild	ticks	none	Cunningham *et al.* (1993)
Trypanosoma eudyptulae		little			blood	Jones & Woehler (1989)
Toxoplasma gondii		little	wild/ rehabilitation		blood, liver spleen. Possibly derived from meat fed to bird	Mason *et al.* (1991)
Toxoplasma					Possibly malaria misdiagnosed. See Fleishman *et al.* (1968)	Ratcliff & Worth (1951)

(continued)

Table 3. *(continued)*

Microorganism	Disease	Penguin species affected	Wild or captive	Vector	Part of body infected	References
Viral						
Newcastle disease virus	Newcastle disease	Adelie	wild-caught		serum antibodies	Pierson & Pfow (1975)
		royal	wild			Morgan *et al.* (1985)
avian influenza virus		Adelie	wild		serum antibodies	Morgan & Westbury (1981)
avian paramyxoviruses		Adelie, little, royal, king	wild		serum antibodies, cloacal swabs	Morgan & Westbury (1981), Morgan *et al.* (1985)
flavivirus		little	wild		serum antibodies	Morgan *et al.* (1985)
herpes-like virus		African			respiratory tract	Kincaid *et al.* (1988)
Puffinosis like virus		gentoo			blisters on feet, ataxia, death	Macdonald & Conroy (1971)
Bacterial						
gram-negative aerobes		Adelie, chinstrap, emperor, little, rockhopper, royal, gentoo, king			gut	Bunt (1955), Soucek & Mushin (1970)
Bacillus spp.		rockhopper, royal, gentoo			faeces	Bunt (1955)
Salmonella spp.		Adelie	wild		faeces	Oelke & Steiniger (1973)
Salmonella typhimurium		African	captive		blood, faeces	Cockburn (1947)
Pseudomonas pseudomallei		macaroni	captive			MacKnight *et al.* (1990)
Edwardsiella tarda		rockhopper			small intestine (enteritis)	Cook & Tappe (1985)
Chlamydia spp.		Adelie, emperor, rockhopper, royal, gentoo			serum antibodies	Moore & Cameron (1969), Sladen (1962)
Pasteurella multocida	Avian cholera					
Pasteurella anatipestifer		little				Mason (pers. comm.)
Acid fast bacillus ?Mycobacterium	Tuberculosis	African			localised in metatarsophalangeal joints	Hamerton (1936,1937) cited Fiennes (1967), Lensink & Dekker (1978)
Erysipelothrix spp.		African				Lensink & Dekker (1978)
Plesiomonas shigelloides		Humboldt	captive		heart, liver	Glünder (1988)
Fungal						
Aspergillus spp.	Aspergillosis	little	wild		lung	R. Norman (pers. comm.)
Aspergillis flavus	Aspergillosis	little, African, Magellanic	captive		lung	Fiennes (1962,1967)
Aspergillus fumigatus	Aspergillosis	little, African, Magellanic	captive		lung, CNS	Fix *et al.* (1988), Kincaid *et al.* (1988), Obendorf & McColl (1980), Lensink & Dekker (1978)

isolated from the cloacas of royal and king penguins and antibodies detected in serum from Adélie and little penguins (Morgan and Westbury, 1981). The pathogenicity of these viral isolates was low in chickens and, although the effects on penguins are unknown, they are likely to be asymptomatic. Paramyxoviruses may cause disease if in combination with other agents and may also help produce immunity to NDV. Antibodies to NDV and APMV-1M have been shown to be only transiently present in little penguins after infection (Morgan and Westbury, 1988). There may be some factor that prevents spread of virus infection until after chick hatching since more antibodies are detected late in the breeding season than earlier.

Serum antibodies to avian influenza virus H7 were detected in six of 285 sera from Adélie penguins at Casey (Morgan and Westbury, 1981). Many 4-5 wk old Adélie chicks died from an unknown cause on Petersen Is. (Casey) in the same year, but no clinical disease was observed and post mortems were not carried out. Antibodies to avian influenza A viruses have been detected in Adélie penguins in the Ross Sea Dependency (Austin and Webster, 1993). Avian influenza viruses are common in free-flying birds; thus it is not surprising to find antibodies in penguins. However, the significance is uncertain since some forms cause disease while others do not.

Antibodies to flaviviruses have been demonstrated in little penguins and a pathogenic flavivirus strain was isolated from ticks on Macquarie Island (Morgan *et al.*, 1985). Antibodies have also been detected in occasional serum samples from royal, king and rockhopper penguins (Morgan *et al.*, 1981). The low prevalence suggested that either infection rates are low or that disease is rapidly fatal and few birds survive.

A disease resembling the virus disease puffinosis in Manx shearwaters (Puffinus puffinus) was reported in Gentoo penguins at Signey Island, Antarctica by MacDonald and Conroy (1971). Several hundred chicks were found dead. Although they appeared in good bodily condition all had multiple ulcers, 2-4 mm in diameter, on the dorsal surfaces of both feet. The infectious agent was not proven, however, and Adélie and chinstrap penguins in adjacent colonies were unaffected.

A herpes-like virus infection in an African penguin was reported by Kincaid *et al.* (1988). The infection was characterised by debilitation and respiratory distress.

Bacterial and Fungal Diseases (Table 3)

Surveys of the intestinal flora of various penguins in the wild have identified the presence of various apparently non-pathogenic bacterial species in healthy birds. These include *Escherichia coli*, *Alcaligenes faecalis*, *Citrobacter freundii*, *Enterobacter* spp., *Paracolon* spp., *Bacillus* spp. and *Pseudomonas* strains (Soucek and Mushin, 1970). Some gut samples were sterile for aerobic gram-negatives, but all contained micro-organisms (eg. *Staphylococcus epidermis* and *Streptococcus faecalis*), and all species harboured *E. coli*. *Pseudomonas* was only found in two gentoo penguins. Diet affected the numbers of anaerobes found; birds eating mainly crustaceans were most likely to give negative results, probably due to the antibacterial properties of the algae found in the krill (Sieburth, 1959). Bacterial growth from faecal samples tended to be greater towards the end of the birds' breeding season than at the beginning. Enteritis due to *E. coli* and *Klebsiella* spp. has been described in captive penguins, but not reported in the wild (Stoskopf and Beall, 1980).

Death attributed to a disseminated *Pseudomonas pseudomallei* infection was reported for a captive macaroni penguin in Hong Kong at a time when the bird was stressed by exceptional temperatures and humidity (MacKnight *et al.*, 1990). *Pseudomonas pseudomallei* is present in water and soils.

Five *Salmonella* species have been isolated from Adélie penguins at Ross Island (Oelke and Steiniger, 1973), but not associated with disease. It is not known whether the bacteria were brought into Antarctica by other bird species or by man. *Salmonella typhimurium* and *S. anatis* have been known to cause disease in captive penguin species (Stoskopf and Beall, 1980). Chronic enteritis due to *Edwardsiella tarda* infection has been reported in captive rockhopper penguins (Cook and Tappe, 1985).

Antibodies to the *Chlamydia* group of bacteria have been isolated from Adélie and emperor penguins in Antarctica and from rockhopper, royal and gentoo penguins at Macquarie Island (Moore and Cameron, 1969; Cameron, 1968). The wide distrib-

ution of positive specimens indicates the probable circumpolar distribution of the *Chlamydia* group which may have been brought south by flying birds. The significance of the above findings is unknown, but it is possible that psittacosis (*C. psittaci*) may contribute to chick mortality in penguin colonies (Cameron, 1968).

Pure cultures of *Pasteurella multocida* (avian cholera) have been isolated from dead rockhopper penguins on Campbell Island where the disease has been observed on more than one occasion (de Lisle *et al.*, 1990).

Two cases of localised tuberculosis (acid fast bacilli, possibly *Mycobacteria*) in the metatarsophalangeal joints of two captive penguins were reported by Hamerton (1936, 1937). The organisms may have entered through severe bumblefoot lesions (Stoskopf and Beall, 1980). Tuberculosis as the cause of death of an African penguin was reported by Lensink and Dekker (1978) but no additional details were given.

Death from *Erysipelothrix* septicaemia of a captive African penguin was reported by Lensink and Dekker (1978). The infection may have been the result of contaminated fish (Stoskopf and Beall, 1980).

Aspergillosis (due to *Aspergillus fumigatus* and rarely to *A. flavus*) is a common fungal disease in captive penguins where it is usually secondary to stress or other diseases (Stoskopf and Beall, 1980). It is however one of the most common causes of death in penguins and is usually manifest shortly after capture or arrival at the zoo. There are two forms: respiratory (most common) and central nervous system aspergillosis. Healthy penguins are normally able to resist the disease and the most effective treatment of sick birds is with endotracheal amphotericin B (Fix *et al.*, 1988). Although aspergillosis is rarely seen in wild penguins, the pulmonary form has been diagnosed in dead beachwashed little penguins (Obendorf and McColl, 1980). Beachwashed little penguins (usually juveniles) are frequently found along the southern coastlines of Australia. The most common cause of mortality in such birds is starvation exacerbated by heavy parasite burdens, but other lesions including coccidiosis and aspergillosis are occasionally seen (Obendorf and McColl, 1980).

Other Diseases and Trauma

Several diseases of unknown aetiology have been recorded including a nephritis of young chicks of African and Humboldt penguins which is rapidly fatal (Stoskopf and Beall, 1980). African penguins have also succumbed to hepatitis and salpingitis (Lensink and Dekker, 1978). Renal stones successfully treated by extracorporeal shockwave lithotripsy were reported in a captive Magellanic penguin by Machado *et al.* (1987). Various carcinomas have been observed including a cholangiocarcinoma in a Humboldt penguin (Shigemi, 1979) and a lymphocytic sarcoma/mixed cell lymphoma in little penguins (Reece, 1992). Cataracts have been found in a little penguin (Reece *et al.*, 1992). We have observed otherwise healthy Adélie penguins with uni- or binocular serous discharge in breeding colonies in some seasons. We also have archival film which shows a colony of Adélie penguins near Mawson in 1973 where there were large numbers (possibly hundreds) of well grown chicks dead and others ataxic.

Various traumas are observed in penguins, both due to natural causes (especially predator attack) and human-induced. Injuries range from superficial abrasions to deeply-penetrating wounds and limb fractures. These derive from normal accidents as well as close encounters with predators such as leopard seals. We have occasionally observed other traumas such as cloacal prolapse and paralysis in Adélie penguins. Human-induced injuries include those caused by propellers, dogs, flipper band abrasion and stomach flushing.

Congenital disorders including malformed beaks and the absence of flippers have been observed in the little penguin by Reilly and Balmford (1975).

Nutritional disorders have been documented in captive penguins but little is known about the occurrence or importance of such disorders (other than general starvation) in wild populations. Nutritional problems that have been reported for penguins in zoos include thiamine deficiency (Harrigan, 1988), nutritional secondary hyperparathyroidism due to vitamin D3 deficiency and iodine-induced hyperplastic goitre (Russell, 1977).

A major problem in captive birds is bumblefoot caused by bacterial infection of the soles of the feet damaged by wet and possibly unsuitable surfaces in the zoo environment (Gailey-Phipps, 1978a,

Table 4. The cellular composition of whole blood-erythrocytes and haemoglobin. Erythrocyte count (number concentration of erythrocytes), MCH (mean corpuscular haemoglobin), MCMC (mean corpuscular haemoglobin expressed as the mass concentration of haemoglobin in erythrocytes), MCV (mean corpuscular volume ie. volume of one erythrocyte). Haematocrit is the volume of erythrocytes in relation to the volume of whole blood). Haemoglobin concentration is given as the concentration in whole blood

Species	Erythrocyte count ($x\ 10^{12}$/l)	Haemoglobin (g/100ml)	Haematocrit (%)	MCH (pg)	MCHC (%)	MCV (fl)	References
Adelie	1.90 - 2.17	16.2 - 17.6	44.4 - 50.3	81.6	35.0 - 37.6	235 - 239	Block & Murrish (1974), Clark & Nicol(1993), Douglas *et al.* (1976),Guard & Murrish(1973), Milsom *et al.* (1973), Myrcha & Kostelecka-Myrcha (1980)
Gentoo	1.60 - 2.24	16.0 - 17.2	43.0 - 52.6	92.5 - 99.4	36.1 - 38.0	206 - 284	Block & Murrish (1974), Douglas *et al.* (1976), Hawkey *et al.* (1945), Hawkey *et al.* (1989), Milsom *et al.* (1973), Myrcha & Kostelecka-Myrcha (1980)
Chinstrap	1.91 - 2.10	16.6 - 19.6	43.0 - 52.8	87.2	35.9 - 37.7	224 - 243	Block & Murrish (1974), Douglas *et al.* (1976), Milsom *et al.* (1973), Myrcha & Kostelecka-Myrcha (1980)
Little	1.66 - 2.05	13.8 - 14.4	41.1 - 43.3	70.3	32.1	217 - 229	Clark & Nicol (1973), Nicol *et al.* (1988), Spielman (pers. com.)
Rockhopper	2.36	16.4	45.0		36.6	195	Hawkey *et al.* (1989)
Magellanic	1.99	13.9	42.0		33.1	215	Hawkey *et al.* (1989)
African	1.57	15.4	46.7	103.0	32.8	315	Stoskopf *et al.* (1980)
Chicken	0.72 - 3.80	8.6 - 13.9	25.5 - 40.8	36.0 - 41	30.0 40.0	92 - 137	Clark & Nicol (1993), Hodges (1977)

Table 5. The cellular composition of whole blood-leucocytes

Species	Total leukocytes (10^9/l)	Heterophils (10^9/l)	%	Lymphocytes (10^9/l)	%	Monocytes (10^9/l)	%	Eosinophils (10^9/l)	%	Basophils (10^9/l)	%	Polychromatophils (10^9/l)	%	References
Adelie			38.5-86.0		7.8-51.8		2.5-5.5		1.8-7.5		0.8-6.0		1.0-2.0	Zinsmeister & van der Heyden (1987)
Gentoo	6.00	3.80	40.8-69.8	1.60	26.0-54.0		0.0-3.3	0.10	1.0-7.0		0.0-1.0		0.9-2.0	Hawkey *et al.* (1985, 1989) Zinsmeister & van der Heyden (1987)
Chinstrap			56.0-67.3		27.8-43.8		0-4.8		0.0		0-0.5		1.0-2.0	Zinsmeister & van der Heyden (1987)
Little penguin	7.02	4.38		2.12		0.44		0.27		0.01		4.79		D.Spielman (pers. comm.)
Rockhopper			29.0		63.0		1.8		6.4		0.6			Hawkey *et al.* (1989)
Magellanic			32.0		60.0		1.2		6.4		0.1			Hawkey *et al.* (1989)
African	11.57	8.23		4.19		0.37		0.11						Stoskopf *et al.* (1980)
Chicken	16.6-29.4		13.3-27.2		59.1-76.1		5.7-10.2		1.4-2.5		1.7-2.4			Hodges (1977)

Table 6. Total protein, fibrinogen and pH levels of whole blood of penguins and the chicken

Species	Total protein (g/l)	Fibrinogen (g/l)	Body Temp (deg C)	pH	References
Adelie	41.5-49.4		37.8-39.9	7.50	Block & Murrish (1974), Boyd & Sladen (1971), Clarke & Nicol (1993), Douglas *et al.* (1976)
Gentoo	41.7-59.8	3.1	38.8	7.51	Block & Murrish (1974), Douglas *et al.* (1976), Hawkey *et al.* (1985), Rosa *et al.* (1993)
Chinstrap	45.4-50.6			7.52	Block & Murrish (1974),Douglas *et al.* (1976), Rosa *et al.* (1993)
Emperor			38.1-39.0		Boyd & Sladen (1971)
Little	46.3-5.33				Clarke & Nicol (1993), D. Spielman (pers. comm.)
Rockhopper	25-64				Ghebremskel *et al.* (1989)
African	59.20				Stoskopf *et al.* (1980)
Magellanic	32-60				Ghebremskel *et al.* (1989)
Chicken	4.36				Clarke & Nicol (1993)

Table 7. Normal levels of blood biochemical constituents

Species	Sodium (mM/l)	Potassium (mM/l)	Chloride (mM/l)	Phosphorus (mM/l)	Calcium (mM/l)	Magnesium (mM/l)	Total bilirubin (mM/l)	Creatinine (mM/l)	Cholesterol (mM/l)	Uric acid (mM/l)	Alkaline phosphatase (U/l)	Glucose (mM/l)	References
Adelie	144-153	2.8-5.3	106-121	0.39-1.28	2.36-3.01	0.76-1.2	7.0-19.0	7.0-40.0	5.37-8.41	125-817	22.0-171+	12.4-17.6	This study
Little	104-170	2.5-15.3	76-130	0.64-3.94		0.48-117		31-111			0.3-1093		D.Spielman (pers. comm.)
Gentoo	154-169	1.9-2.7	106-117	0.46-1.54	2.18-2.76		1.0-4.0	34-43		329-1138	31-91		Ghebremskel *et al.* (1989)
Rockhopper	138-162	10.3-16.7	103-126	28-176	1.46-3.31		1.0-5.0	29-68		71-494	28-176		Ghebremskel *et al.* (1989)
Magellanic	140-166	2.3-20.1	100-113	1.42-2.82	1.84-2.43		1.0-5.0	53-83		217-1780	57-266		Ghebremskel *et al.* (1989)

1978b; Stoskopf and Beall, 1980). Healing has been induced however by providing a dry soft surface (kitty litter or carpet) or dry decking (Todd, 1978) in the enclosure.

CLINICAL PATHOLOGY

The cellular, chemical and biochemical constituents of penguin blood are provided in tables 4-7. Values are provided for apparently normal birds captured in the wild and determined by comparable methods. Given the limited number of clinical studies on penguins, discussion on changes which may take place in illness must be inferred from investigations of other birds, particularly domestic fowl. The following information is provided as a guide but should be treated with caution in its application to penguins.

Haematology

The cellular components of whole blood are detailed in tables 4 (erythrocytes) and 5 (leucocytes). Table 4 includes the erythrocyte count (number concentration of erythrocytes), mean corpuscular haemoglobin (MCH), mean corpuscular haemoglobin expressed as the mass concentration of haemoglobin in erythrocytes (MCMC), and mean corpuscular volume (MCV) ie. the volume of one erythrocyte. Haematocrit is given as the packed cell volume (PCV) of erythrocytes in relation to whole blood and haemoglobin concentration (Hb) as the concentration in whole blood.

The total erythrocyte count in most bird species is higher in males than females, increases in autumn, increases with age, increases with altitude and latitude and decreases after bacterial infections such as *Salmonella gallinarum* and *E. coli* (Stoskopf *et al.*, 1983).

Haematocrit increases in summer, is greater in males than females, increases with age, increases in dehydration, drops slightly in moult, decreases in egg laying and decreases in anaemia and parasitism (Stoskopf *et al.*, 1983). Haemoglobin levels increase with age, are higher in males than females, increase at altitude, and are greater in winter (Stoskopf *et al.*, 1983).

Penguins have higher Hb and PCV values and larger erythrocytes than most other avian species. By the end of moult rockhopper penguins have lower Hb, RBC, PCV and MCHC, and higher MCV than before (Hawkey *et al.*, 1989).

Leucocytes

Heterophils are the most numerous white cells in normal penguin blood in contrast to many other bird species where the lymphocyte is most common. Eosinophils are the second most common leucocyte in penguins (Zinsmeister and VanDer-Heyden, 1987).

Heterophils in birds in general are elevated in stress, bacterial infections and inflammatory reactions; eosinophils are elevated in hypersensitivity reactions and haemoparasitism; lymphocytes numbers decrease in chronic stress and may change with season; and monocytes can increase in chronic and tissue destructive diseases. Polychromatophil numbers are related to rate of erythropoiesis and increased numbers may be associated with inflammation and haemorrhage (Zinsmeister and VanDerHeyden, 1987).

Heterophil/lymphocyte ratios vary between surveys. The absolute lymphocyte count is relatively stable in healthy adult birds while heterophil numbers are more susceptible to variation under to the influence of stress and microbial challenge (Hawkey *et al.*, 1989).

By the end of moult rockhopper penguins have lower heterophil values, and higher lymphocyte counts than before. In one survey wild rockhoppers and gentoos had lower heterophil numbers and higher lymphocyte, monocyte and eosinophil values than captive birds (Hawkey *et al.*, 1989).

Total leucocyte count, heterophil count and fibrinogen levels are raised in inflammation and infection (eg. bumblefoot). Monocytosis and basophilia may be associated with tissue necrosis (Hawkey *et al.*, 1985).

Captive African penguins show a wide range of total leucocyte values with heterophils predominating (Stoskopf *et al.*, 1980).

Bacterial infections are characterised by heterophilia and lymphopaenia. Total leucocyte numbers increase in renal disease, with age, with stress and in the evenings; and counts decrease in viral infections. Basophils contain histamine and are involved in acute inflammation and hypersensitivity reactions. Eosinophil numbers increase in parasitisms, especially blood parasitisms. Heterophil

numbers are elevated in bacterial infections and stress. Lymphocyte numbers decrease in stress and bacterial infections; and values increase in plasmodial infections (Stoskopf *et al.*, 1983).

Blood Biochemistry (Tables 6, 7)

Total blood protein levels in birds are low in malnutrition and acute infections. Values lower than 20 g/l indicate poor prognosis. Serum protein increases in egg laying, dehydration and chronic infections. Glucose levels vary widely with season, decrease in fasting, tend to be higher in females than males and increase in hyperthermia and excitement. Alkaline phosphatase increases in growth, rickets, primary hypoparathyroidism, fracture repair, osteomyelitis and aspergillosis. Cholesterol levels increase with age and tend to be higher in females. Uric acid increases in ovulation, starvation, trauma and gout. Calcium levels increase 2 weeks prior to laying and decrease in fasting, hyperthermia, and renal failure. Phosphate levels increase 2 weeks prior to laying and decrease during hyperthermia and fasting. Potassium levels increase in hyperthermia while sodium and magnesium levels decrease (Stoskopf *et al.*, 1983).

Heat stress in pygoscelid penguins leads to increased body temperature. If body temperature exceeds 40 °C then blood pH increases (alkalosis) and arterial pCO_2 decreases (hypocapnia). Ventilation rate also increases significantly (Douglas *et al.*, 1976).

DISCUSSION

This review shows that penguins in the wild are susceptible to an array of infectious and parasitic diseases but that clinical signs of disease are rarely obvious and usually not reported. However our knowledge as shown from this review is meagre and fragmentary even for species subject to extensive scientific studies. Incidence of disease even using immunological methods of survey is difficult to assess. The presence of antibodies suggests that exposure to pathogenic viruses may have occurred although this may indicate only the presence of serologically related non pathogenic viruses. The possibility exists that these non pathogenic species may provide cross immunity to the pathogenic strains. The presence of particular pathogens also does not necessarily indicate the presence of clinical disease, although symptoms may occur under the influence of other contributing disorders including stress.

Studies in zoos show that penguins are susceptible to a wider variety of diseases than those that have been detected or reported in birds in the wild. Penguins spend most of their life at sea and breed on the Antarctic continent and on islands isolated from other areas of human habitation. They are thus to a marked degree isolated from other avian species and vectors of disease. The absence of appropriate vectors and the ability of infectious agents to survive under maritime or polar conditions may have limited the occurrence of disease. The observed incidence of disease in wild populations can be expected to be low since many species are observed only during their breeding seasons, and sick birds are likely to die at sea away from their breeding colonies.

The diagnosis of disease in penguins in the wild is hampered by the lack of information on both the diseases and on what is normal and what is pathological. There have been very few clinical investigations on healthy birds although some biochemical and haematological data such as that contained in tables 4-7 are available. Matching pathological data are not available, however.

The treatment of illness in penguins in zoos has economic, ethical and aesthetic importance. It is also important should species become endangered and ultimately confined to zoos. The large percentages of birds which die during transport or in captivity, particularly during their first weeks, attest to the difficult problems to be solved. Fortunately progress has been made on the understanding and treatment of aspergillosis and malaria which are the major causes of death of captive penguins (Stoskopf and Beall, 1980).

Treatment of injuries and illness in wild birds is difficult and rehabilitation is often unsuccessful (Harrigan, 1988). Ethical problems arise on euthanasia. We have adopted a policy of leaving such birds alone unless the trauma is caused by a human agent in which case we may treat or kill the bird.

The study of diseases in wild penguins is important for the identification of endemic diseases and for the detection of exotic diseases should these

occur. It is also important in the understanding of the degree to which disease may be expected to influence the results of biological studies. Results may be confounded and interpretation made difficult by both the transient and long term presence of disease particularly if it is at the sub clinical level.

The detection and estimation of disease is of particular importance to long term studies such as those taking place under the CCAMLR Ecosystem Monitoring Program (CEMP) (CCAMLR, 1991). This program, as a first step, seeks to determine the natural variation in selected variables (relating to feeding ecology and reproductive success) in order to evaluate possible effects of the harvest of krill on its predators. Predator species include Adélie, chinstrap, gentoo and macaroni penguins. It is likely that changes in predator performance outside the norm will be difficult to detect and that disease may mask early subtle changes caused by fishery activities.

Knowledge of infectious diseases is also necessary for quarantine and conservation purposes. The introduction of exotic diseases may have a devastating effect on penguin populations which live in isolated or remote colonies and have little natural immunity. Similarly species of penguins may carry diseases which could infect wild or domestic birds, particularly poultry, if introduced.

Penguin populations at most breeding locations, particularly in the Antarctic and on subantarctic islands, have evolved in relative isolation. The Southern Ocean forms a barrier across which very few avian species cross. This factor may well have protected the birds from the major diseases found in birds and other species to the north. Distance prohibits contact with disease and inhibits the introduction of vectors or intermediate hosts. The speed and volume of modern transport to Antarctica and the sub Antarctic and the number of people visiting the south polar regions as expeditionary personnel or as tourists has increased enormously; this may increase the possibility of unwitting introduction of diseases. Good hygiene and good quarantine must be practised. The introduction of poultry products except for food, and their disposal into the Antarctic environment is prohibited under the Agreed Measures for the Conservation of Antarctic Flora and Fauna, and the Protocol on Environmental Protection to the Antarctic Treaty provides additional protection.

APPENDICES

Additional information below has been provided by the authors.

Tetrameres wetzeli (a species of nematode) was first isolated from the proventriculus of rockhopper penguins at the Kerguelen Islands (Schmidt, 1965). The bacterium, *Plesiomonas shigelloides*, was isolated as a secondary pathogen from two captive spheniscid penguins. One of these was a Humbodt penguin which had died from a clostridial infection and the other an unidentified species which died from aspergillosis (Glünder, 1988). *Salmonella typhimurium* has been isolated from captive African penguins (Cockburn, 1947). Surviving penguins can become carriers of this disease and transfer infection to other birds. Malignant melanomas are rarely found in birds; however, one such tumour was diagnosed in a captive macaroni penguin (Kufuor-Mensah and Watson, 1992). Ruptured uropygial glands have been observed in captive gentoo penguins, and can be treated surgically (MacCoy and Campbell, 1991). Substantial mortality among yellow-eyed penguins (*Megadyptes antipodes*) was observed during one summer and, despite extensive culture of tissue specimens, no causal organism was detected. A diatom toxin was suspected but not positively identified (Gill and Darby, 1993).

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