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# DISEASES OF ANTARCTIC WILDLIFE

## A Report

**on the**

**"Workshop on Diseases of Antarctic Wildlife"**

**hosted by the Australian Antarctic Division**

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## EXECUTIVE SUMMARY

In response to concerns raised at Antarctic Treaty Consultative Meeting (ATCM) XXI Australia hosted a workshop on diseases in Antarctic wildlife. The workshop recognised that there was a significant risk of the introduction of disease into Antarctic wildlife species and that should it occur the consequences are likely to be serious and a response will be required.

A report of the *Workshop on Diseases of Antarctic Wildlife* summarising outcomes was presented to ATCM XXIII and considered by the Committee for Environmental Protection (CEP II). CEP II agreed that an open-ended contact group be formed when all Parties, SCAR and COMNAP have had the opportunity to consider the full report of the Workshop, and will operate under the following Terms of Reference:

*Prepare an initial report for presentation to CEP III outlining practical measures that might be implemented to:*

*a) diminish the risk of the introduction and spread of diseases to Antarctic Wildlife; and*
b) *detect, determine the cause, and minimise the adverse effects of unusual wildlife mortality and morbidity events in Antarctica.*

The attached document is the full report of the workshop prepared by the convenors. The workshop made a number of general recommendations (sections 4.0) to minimise the risk of the introduction and spread of disease. However, additional information and expertise are required before more specific recommendations are made.

**Recommendations to COMNAP and SCAR,**

1. request that the work of the open-ended contact group on disease in Antarctic wildlife established by CEP II should commence forthwith,

2. encourage managers of national Antarctic programs and national committees for Antarctic research to support participation by appropriate specialists in the open-ended contact group,

3. request managers of national Antarctic programs to consider the *Recommendations Arising from the Workshop* (section 4.0) and implement where practical,

4. encourage managers of national Antarctic programs and national committees for Antarctic research to support research targeted at providing information to reduce the risk of disease introduction to, or spread among, Antarctic wildlife by humans.

## GLOSSARY

| | |
|---|---|
| **Antibodies** | Products of lymphocytes that bind to specific antigens to prevent development of disease. |
| **Antigen** | Substance that stimulates antibody producing cells to produce specific antibodies. |
| **Arthropod** | Segmented invertebrate with jointed legs. |
| **Ataxia** | Loss of control over voluntary movements. |
| **Avirulent** | An avirulent organism is one that cannot induce disease even when present in large numbers. |
| **Bacteraemia** | Presence of bacteria in the blood but without the severe generalisation of infection characteristic of septicaemia. |
| **Carrier status** | When normally pathogenic organisms are living in host tissues without causing disease. This may occur following recovery from disease. |
| **Clinical disease** | Expression of a set of symptoms of disease that are peculiar to a particular disease agent. |
| **Disease** | Deviation from normal health. Occurs when parasites, viruses or bacteria have invaded the host tissues to visibly and deleteriously affect the latter. |
| **Endemic, enzootic** | Constant presence of a disease agent in the community, generally at low incidence. Endemic refers to human populations; enzootic refers to animals. |
| **Epidemic, epizootic** | Outbreak of disease affecting large numbers of the population in a certain area. Epidemic refers to human populations; epizootic refers to animals. |
| **Epidemiology** | The study of environmental and host-related factors that determine the incidence and spread of disease. |
| **Haemolysis** | Release of haemoglobin from red blood cells following their break-up, or due to osmotic effects. |
| **Helminths** | Worms, commonly parasitic. Include nematodes (round worms), cestodes (tapeworms) and trematodes (flukes) |
| **Host** | Any organism upon which another organism can live in a parasitic manner. |
| **Immune response** | The production of antibodies in response to antigenic stimulation which results in the rapid destruction and removal of the antigen and prevention of disease. |
| **Immunity** | The state of resistance of an animal to a specific disease. |
| **Incidence** | Frequency of occurrence or range of influence of a disease. |
| **Incubation period** | The time between entry of an organism into a host and the onset of symptoms. |
| **Infection** | When parasites, viruses or bacteria have bypassed or penetrated the defensive mechanisms of a host and gained access to its tissues. Infection may or may not result in disease. |
| **Leukocytes** | White blood cells, of which there are a number of types involved in the body's response to infection. |
| **Lymphocytes** | White blood cells involved in antibody production and the development of immunity. |
| **Morbidity** | The state of being diseased. |

| | |
|---|---|
| **Mortality rate** | Death rate. |
| **Non-pathogenic** | Non-pathogenic organisms are incapable of causing tissue damage or disease. |
| **Parasite** | Any organism that lives on another organism (the host) at the expense of the latter. Parasites include viruses, bacteria, fungi, protozoa, helminths and arthropods. In common language the term is often used to refer to the last three groups of organisms only. |
| **Pathogen** | An organism capable of invading host defence mechanisms and causing tissue damage. |
| **Pathogenic organism** | One capable of invading host defence mechanisms and causing tissue damage or symptoms of disease. |
| **Pathogenicity** | Degree of tissue damage or disease that an invading organism is able to cause. The pathogenicity of an organism depends on its ability to produce or initiate production of enzymes and toxins. |
| **Septicaemia** | Invasion of the bloodstream by large numbers of bacteria which multiply in it and spread around the body. |
| **Serology** | The study of the serum component of blood that contains antibodies, with emphasis on the specific nature of the latter. |
| **Subclinical disease** | When, despite no obvious pathological changes, organisms are present and multiplying in the host to produce minor changes that are not clinically apparent. |
| **Titre** | The extent to which an antibody-containing substance (eg serum) can be diluted before losing its ability to react specifically against an antigen. |
| **Vector** | An organism which carries other organisms from one place to another. |
| **Virulence** | This term relates to the number of pathogenic organisms required to initiate disease. Organisms capable of initiating disease with few numbers are said to be highly virulent. Those requiring large numbers to produce disease are of low virulence. An avirulent organism is one that cannot induce disease. |
| **Zoonosis** | A disease that can affect both man and animals, in which animals are usually the main reservoir of infection. |

# 1.0 INTRODUCTION

## 1.1 Information Paper at ATCM XXI

Australia presented an information paper at Antarctic Treaty Consultative Meeting (ATCM) XXI entitled *Introduction of Disease into Antarctic Birds.* This paper reported Australian research at Mawson which suggested that on serological evidence Adélie and emperor penguins at some locations had been exposed to Infectious Bursal Disease Virus (IBDV). This virus causes serious disease in poultry. Whilst the presence of IBDV is of specific concern, its discovery highlighted other more general issues,

- there is an increasing risk of disease being introduced into the fauna because of the increase in the numbers of people travelling to and within Antarctica, and.
- there is a need to develop measures to limit disease introduction and to control outbreaks.

As a result of these concerns Australia offered to host a workshop where these issues could be discussed and to report the outcomes of workshop at ATCM XXIII.

## 1.2 Organisation of the workshop

A steering committee to organise the workshop was established comprising Dr. Knowles Kerry, Australian Antarctic Division (Convenor), Dr. Martin Riddle, Australian Antarctic Division, Dr Mike Nunn, Bureau of Resource Sciences, Dr Harvey Westbury, Australian Animal Health Laboratories, CSIRO and Prof Geoff Shellam, Dept Microbiology, University of Western Australia.

The terms of reference for the workshop were,

- identify the potential for disease incursion into Antarctica's wildlife
- develop a series of recommendations to reduce the risk of such introductions and to limit the consequences of any disease establishment and spread
- report to the XXIII Antarctic Treaty Consultative Meeting (ATCM)

## 1.3 Workshop on Diseases of Antarctic Wildlife

The *Workshop on Diseases of Antarctic Wildlife* was held at the headquarters of the Australian Antarctic Division in Hobart on 25-28 August 1998 under the convenorship of Dr Knowles Kerry and Dr Martin Riddle.

The meeting was open to all interested parties and was attended by 52 registered participants from Australia, Brazil, Italy, Japan, New Zealand, Sweden, Netherlands, and the USA. Professor Albert Osterhaus of the Institute of Virology, Erasmus University, Rotterdam, Netherlands and Dr Joseph Geraci from the National Aquarium in Baltimore USA were invited as experts in disease of wildlife. Mr Greg Mortimer represented the International Association of Antarctic Tourist Operators (IAATO) to

whom a special invitation had been extended. A full list of participants is appended (Appendix 1).

The focus of the workshop was disease of birds and seals breeding within the Antarctic Treaty area however consideration was extended to sub-Antarctic animals and migratory species and to both endemic and exotic diseases.

The Workshop was conducted in three parts,

- review papers to provide background information,
- research papers on wildlife disease including case-studies on disease outbreaks in non-Antarctic wildlife, and
- working group sessions

A list of all reviews, research papers and case studies presented at the workshop is given at Appendix 1.

## 1.4 Working Group Sessions

The working group sessions were structured around the following four topics,

- Risks – what are the risks of disease introduction and spread in Antarctica?
- Monitoring – what should be done to ensure early detection of disease?
- Prevention – what procedures could reduce the risk of disease?
- Response – what should be done if introduced disease is suspected?

Workshop participants were assigned to one of four working groups and each working group was assigned primary responsibility for one of the four topics. To ensure all topics were considered widely, two working groups considered each topic. The working group with primary responsibility for a topic collated the notes from both groups and reported back to the meeting.

Reports of each working group, based on rapporteur's notes, are provided as Section 3.0. Working groups were asked to identify any information needs that could be addressed by research. These were collated and are included as a single, combined section on future research (Section 5.5). The outcomes agreed by workshop participants as the basis for reporting back to ATCM XXIII are provided in Appendix 1.

## 1.5 Presentation of Report to ATCM XXIII

The report of the *Workshop on Diseases of Antarctic Wildlife* was presented as Working Paper XXIII ATCM/WP32 to ATCM XXIII at Lima in May 1999 and considered by the Committee for Environmental Protection (CEP II) under Agenda Item 5b: *Compliance with the Protocol on Environmental Protection: Matters covered by Annex*

*II (Conservation of Antarctic Flora and Fauna)*. Paragraphs 59-61 of the report from CEP II records the Committee's response to the report of the workshop as follows,

*(59) The Committee agreed that an open-ended contact group be formed to present to CEP III an initial report on matters arising from the Workshop on Diseases of Antarctic Wildlife.*

*(60) The group will be formed when all Parties, SCAR and COMNAP have had the opportunity to consider the full report of the Workshop, and will operate under the following Terms of Reference:*

*Prepare an initial report for presentation to CEP III outlining practical measures that might be implanted to:*

*a) diminish the risk of the introduction and spread of diseases to Antarctic Wildlife; and*

*b) detect, determine the cause, and minimise the adverse effects of unusual wildlife mortality and morbidity events in Antarctica.*

*(61) The Committee accepted Australia's offer to convene the group under the leadership of Dr Martin Riddle (Australia) (martin.riddle@antdiv.gov.au)*

*(62) IUCN noted that worldwide it is considered that introduced organisms, including those causing disease, account for more loss of species than does loss of habitat. It suggested that the Global Invasive Species Programme (GISP) coordinated by the Scientific Committee on Problems in the Environment (SCOPE), and IUCN could provide valuable input.*

## 2.0 DISEASE IN ANTARCTICA: SUPPORTING INFORMATION

*Emerging diseases are usually not 'new', just freed from obscurity by acts of man* Morse (1990)

### 2.1 Introduction

This paper presents knowledge of the disease status of Antarctic wildlife, the risks of introduction of disease into Antarctica, precautions that could be taken to prevent their introduction and the measures necessary in the event of an outbreak of disease. It is written against the background provided by the *Workshop on Diseases of Antarctic Wildlife* held in Hobart in August 1998, and includes general information on the epidemiology of disease necessary for a full appreciation of the issue.

The geographic focus is on the region south of 60°S latitude ie the region covered by the Antarctic Treaty. However, reference is also made to the sub-Antarctic Islands because of their isolated nature, their location in relation to the South Polar Front and the relationship of their vertebrate fauna to that of Antarctica.

The following definition of disease, provided by Wobeser (1981) as quoted by Spalding and Forrester (1993), is used,

*Disease is considered to be any impairment that interferes with or modifies the performance of normal functions, including responses to environmental factors such as nutrition, toxicants, and climate; infectious agents; inherent or congenital defects, or combinations of these factors.*

Consistent with this definition the focus is on infectious agents from the perspective that for a disease outbreak to occur there needs to be the appropriate interaction between the host, the pathogen and the environment. Changes such as the increased density of host, the introduction of a new pathogen or a change in the environment can all trigger the outbreak of a disease. As a consequence the overall disease process must be considered in ecological terms.

Knowledge of infectious and parasitic diseases among Antarctic species is meagre and fragmentary even for species maintained in captivity and for those that have been extensively studied in the wild, such as the Adélie penguin. The following is a summary of known disease among Antarctic vertebrate species and is an updated version of the information presented at the Workshop (Appendix 3). An extensive review covering all species of penguins by Clarke and Kerry (1993) is available.

### 2.2 Isolation of Antarctic Wildlife

It is likely that many of the diseases found even on the southern continents will not be found in Antarctic species because of the geographical isolation of Antarctica. The

nearest continent, South America, is 1500 km to the north, South Africa is 4100 km and Australia 3600 km. This isolation which has existed for long periods of geological time coupled with epochs of glacial advance and retreat has limited the colonisation to those vertebrate species (seals and birds) which are able to cross the Southern Ocean by swimming or flying.

The Southern Ocean forms a barrier across which very few avian species cross; a consequence of this separation is that seals and birds in the Antarctic and on sub-Antarctic islands have evolved in relative isolation. Distance limits contact with disease and inhibits the introduction of vectors or intermediate hosts, this factor may well have protected these species from the major diseases found in birds and other species to the north. However, the speed of modern transport to Antarctica and the sub-Antarctic and the numbers of people visiting the south polar regions as scientists or tourists have increased enormously and these changes may increase the possibility of unwitting introduction of diseases. The nature of travel within the Antarctic is also changing. Transport *around* the Antarctic continent has increased, particularly from tourist ventures, and this increases the risk of disease spread regardless of whether the disease is endemic or introduced.

The sub-Antarctic Islands are similarly isolated. They form a ring approximately half way between the Antarctic Continent and the other southern continents and straddle the south polar front. Some Islands are heavily glaciated eg South Georgia and Heard Island but all share a maritime climate and are to some degree vegetated with flowering plants which on the northermost Islands are woody. All support large colonies of sea birds and seals which range widely to forage particularly during the non-breeding season. Several species of birds, for example the wandering albatross and the giant petrel, feed in the coastal waters of the continents to the north. The milder climate means that arthropod vectors of infectious disease such as ticks and fleas are likely to be present and are able to complete their life-cycle. It is possible therefore that disease may be introduced into the wildlife of these Islands more readily than to Antarctica and that they in turn may also play a role in the transmission of disease into Antarctic wildlife.

## 2.3 The Antarctic Treaty System and Disease

The possibility of disease introduction into Antarctic wildlife has been recognised since the start of the Antarctic Treaty and was identified as a concern at the first meeting of the Biology Working Group of the Scientific Committee for Antarctic Research (SCAR) in 1962 (Murray 1964). However the issue has received scant attention and very little has been achieved towards implementing practical procedures to protect Antarctic wildlife from introduced disease. The Agreed Measures for the Conservation of Antarctic Fauna and Flora 1964 prohibit the bringing into the Antarctic Treaty Area any species of animal and plant not indigenous to that area except in accordance with a permit. Annex D of the Agreed Measures lists *Precautions to prevent accidental introduction of parasites and diseases into the Treaty Area*. These required all dogs imported into the Treaty Area to be inoculated against distemper, contagious canine

hepatitis, rabies and leptospirosis and prohibited the transport of live poultry into the Treaty Area.

These measures now have been subsumed by the Protocol on Environmental Protection to the Antarctic Treaty, 1991 (Madrid Protocol) which came into force in 1998. Under Annex II of the protocol, *Conservation of Antarctic Fauna and Flora*, signatories are no longer permitted to take dogs to Antarctica and those already present were required to be removed. The annex also includes (Appendix C) *Precautions to prevent introductions of Micro-organisms* including a ban on the import of live poultry and other birds, the requirement to inspect dressed poultry for evidence of disease including Newcastle's disease, tuberculosis, and yeast infection and the instruction to avoid the importation to Antarctica of non-sterile soil to the maximum extent possible.

Apart from these measures there is little in place in international law to limit the introduction and spread of exotic diseases to Antarctica. Further there is nothing analogous to the strict quarantine laws established to restrict the importation across national borders of living plants and animals and products derived from them. However many countries including Australia impose strict quarantine control on the importation of materials from Antarctica. This implies that there is a risk that alien species, including disease, may be imported. In reality, the risk is more heavily weighted in the opposite direction.

Antarctica is the last continent to have any consideration given to limit human mediated introduction of organisms pathogenic to the indigenous wildlife and no measures are in place to limit the spread of any disease should an outbreak occur.

## 2.4 Endemic (enzootic) diseases

Endemic diseases are those that are widespread, although they may be of low incidence, in a population. Exotic diseases are those that are not present in the population or are sufficiently localized to be eradicable. The terms *endemic* (or more correctly *enzootic* as endemic refers to human populations) and *exotic* do not convey any information on whether humans were involved in their introduction or spread of disease. Those diseases which may be present in a population, but whose introduction or spread were not mediated by humans, are referred to as native or indigenous diseases.

Virtually nothing is known of endemic diseases in Antarctic populations of seals and birds and or whether any non-indigenous diseases are present. There have only been two cases of mass mortalities of Antarctic wildlife reported in the literature, one of crabeater seals and one of Adélie penguins. A few cases of bacterial infections leading to death have also been reported. This low incidence of reported disease does not mean that Antarctic wildlife is disease free. It is likely that agents capable of causing disease are present among the population but their expression in terms of diseased individuals has not often been observed because of the small human presence in Antarctica.

Endemic disease may be present in a population although it may not be manifest in a clinically recognisable form. However, outbreaks may occur if the animals are compromised by environmental or other stress. Animals not previously exposed to a pathogen may be immunologically naive and thus may succumb more readily to its presence. There is no evidence that naturally occurring ie. enzootic disease can bring about the extinction of a species. However, there is evidence that unnaturally introduced disease may cause population decline and could possibly lead to the extinction of a species. An often-cited example is the effect on the Hawaiian land birds of the introduction of avian pox and malaria (Warner 1968, van Riper III 1986).

## 2.5 Outbreaks of disease in Antarctica and the sub-Antarctic Islands

### 2.5.1 Birds

There has been only one instance of an infectious disease in Antarctic birds where the cause has been identified. This is an occurrence of avian cholera *Pasteurella multocida* (strain 1-X73) in the brown skua *Catharacta lonbergi* reported by Pamerlee (1980) on Livingston Island in the Antarctic peninsula region. Pure cultures of *Pasteurella multocida* have also been isolated from dead rockhopper penguins on sub-Antarctic Campbell Island where the disease has been observed on more than one occasion (de Lisle *et al*., 1990).

An Adélie penguin captured in Antarctica was reported to have Newcastle disease on arrival in the USA (Pierson and Pflow 1975) however, there is some doubt whether the bird contracted the disease in Antarctica.

Two mass mortality events have been recorded in Antarctica birds where infectious disease is suspected. A disease resembling the virus disease puffinosis, which occurs in Manx shearwaters (*Puffinus puffinus),* was reported in gentoo penguins at Signey Island, Antarctica, where several hundred chicks were found dead (MacDonald and Conroy, 1971). 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. Kerry et al (1996) reported the death of Adélie penguin chicks which occurred at Low Tongue approximately 40 km west of Mawson Station on 2 February 1972. About 65% of the chicks had died recently and freshly dead chicks littered the area. Many of those still alive were found face down and could not stand on their own. When assisted to stand they would stagger a few steps and limply fall forward. These chicks, both alive and dead, were of uniform size, plump and apparently well nourished. The cause of death remains unknown.

Twenty-three skuas that had died at Esperanza station were examined for the presence of bacterial and fungal pathogens. Lesions were consistent with fungal infection, and fungi were subsequently isolated. There was no evidence of concurrent bacterial infection (Leotta *pers. com*.). The most common fungal disease in birds is aspergillosis. Mortality, however, is rare in wild birds unless predisposing factors such as starvation,

concurrent disease or other causes of stress are present. No bacteria were isolated from the skuas that died and virus isolation was not attempted. The existence of other potential causes of stress is unknown.

### 2.5.2 Seals

Only one mass mortality event has been reported involving seals in Antarctica where disease is the most likely cause. Laws and Taylor (1957) reported the death of 1500 or more crabeater seals *Lobodon carcinophagus* in the Crown Prince Gustav Channel in the Antarctic Peninsula in 1955. The cause was suspected to be a highly contagious viral disease possibly exacerbated by stress from crowding and partial starvation as a result of the animals having been trapped by ice and deprived of access to the ocean.

A mass mortality occurred among the sea lions, *Phocarctos hookeri*, of the New Zealand sub-Antarctic Auckland Islands in January-February 1998. At least 1600 pups and an unrecorded number of adults died. The deaths of unweaned pup could have been a consequence of the deaths of the females. Two hypotheses have been proposed to explain these deaths,

*a) The deaths were caused by a previously unknown- or difficult to identify-gram negative pleomorphic bacterium which may be a highly pathogenic organism in its own right that has been recently introduced into a naive population, or it may be a normal commensal that became pathogenic because of some change in the normal host/pathogen relationship, swinging the balance in favour of the pathogen. Or,*

*b) some event predisposed the sea lions to a suite of bacterial infections. This could have been infection by a previously unknown virus, a marine biotoxin or a drastic environmental change associated with the El Nino/Southern Oscillation phenomenon. (Department of Conservation, New Zealand 1999)*

## 2.6 Exposure to Infectious Agents

There are a number of studies which provide evidence that birds and seals have been exposed to agents that can cause disease. However, in the following cases there is no evidence to suggest that any clinical expression of these diseases has occurred.

### 2.6.1 Exposure to Viral Diseases

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

Non-pathogenic paramyxovirus strains have been isolated from the cloacae of royal and king penguins, and antibodies to them have been 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 and it is therefore not surprising to find antibodies in penguins, however, the significance is uncertain since some forms cause disease while others do not. Serological evidence for the presence of infectious bursal disease virus (IBDV) in Adélie penguins at Mawson has been reported recently (Gardner et al 1997) however the virus has not been isolated.

### 2.6.2 Exposure to Bacterial and Fungal Diseases

Antibodies to the *Chlamydia* group of bacteria have been isolated from Adélie and emperor (*Aptenodytes forsteri*) penguins in Antarctica and from rockhopper, royal and gentoo penguins at Macquarie Is (Moore and Cameron, 1969; Cameron, 1968). The wide distribution of positive specimens indicates the probable circumpolar distribution of the *Chlamydia* group. 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). Antibodies to the spirochete *Borelia berdorferi*, which causes Lyme disease, were recently found in king penguins and the bacteria itself was found in the cosmopolitan tick *Ixodes uriae* infesting these birds in the Crozet Archipelago (Gauthier-Clerc et al 1999). This disease apparently does not cause serious disease in bird populations. It is however a zoonosis with symptoms in humans which may be severe.

Surveys of the intestinal flora of several penguin species in the wild have identified the presence of various apparently non-pathogenic bacterial species in healthy birds (Soucek and Mushin, 1970). Five *Salmonella* species have been isolated from Adélie penguins at Ross Island (Oelke and Steiniger, 1973) but they were not associated with disease. *Salmonella enteritidis* phage type 4, which accounts for 80% of clinical *S. enteritidis* isolates in the western world, has been isolated from one of thirty Gentoo penguins sampled from Bird Island, South Georgia (Olsen, Bergstrom, McCafferty,

Sellin and Wistrom, 1996). It is not known whether the bacteria were brought to these locations by other bird species or by man.

Aspergillosis, usually caused by *Aspergillus fumigatus* and rarely by *A. flavus*, is a common fungal disease in captive penguins where it is usually a secondary result of stress or other diseases (Stoskopf and Beall, 1980). Although aspergillosis is rarely seen in wild penguins, the pulmonary form has been diagnosed *post mortem* in skuas (Montaldi pers comm).

## 2.7 Detection of disease in Antarctic and sub-Antarctic species

Penguins and seals in the wild are susceptible to an array of infectious and parasitic diseases (J. Clarke and K.Kerry, A. Osterhaus this symposium) but clinical signs of disease are rarely obvious and disease is generally diagnosed *post mortem*. The diagnosis of disease in penguins in the wild is hampered by lack of information on both the diseases themselves and on what is normal or alternatively, what is pathological. There have been very few clinical investigations on healthy birds, although some biochemical and haematological data are available there is no matching pathological data.

The incidence of disease observed in wild populations of sea birds and seals is expected to be low since many species are seen only during their breeding seasons and sick birds are likely to die at sea away from their breeding colonies. The fact that there are very few visits to most aggregations of wildlife along the Antarctic coastline further reduces the probability of finding instances of disease. However the increase in tourist visits to penguin colonies around the continent and increased awareness of the value of reporting disease incidents will perhaps increase the numbers of outbreaks reported.

Incidence of disease is difficult to assess even using immunological methods of survey. Although the presence of antibodies suggests that exposure to pathogenic microorganisms may have occurred, it may indicate only the presence of serologically related non-pathogenic microorganisms. The presence of particular pathogens does not necessarily indicate the presence of clinical disease, however, symptoms may occur under the influence of other contributing disorders, including stress. The possibility also exists that these non-pathogenic species may provide cross immunity to the pathogenic strains.

Studies in zoos show that penguins and seals are susceptible to a wider variety of diseases than those that have been detected or reported in birds in the wild. Many of the disorders associated with captivity are due to locally occurring pathogens that may not be experienced at present by birds in the wild but could be introduced in the future. The introduction of exotic diseases may have a particularly devastating effect on penguin populations that live in isolated or remote colonies and may have little natural immunity.

## 2.8 Factors Influencing the Introduction and Spread of Disease

Environmental conditions in the Antarctic and biological adaptations of the Antarctic fauna to the environment may all influence the processes of disease introduction and spread.

### 2.8.1 Animal Behaviour

Migratory birds or vagrants, either of which could be carriers of infectious disease, may be potent sources of infection for Antarctic seabirds although this has not yet been proven. As albatrosses and the shearwaters are able to sustain flights of 50 km/hr for a day or more, it is possible, though not necessarily probable, for an infected animal to reach Antarctica from South America and possibly from Australia within 3-7 days.

The avifauna of Antarctica consists of only 11 species comprising Adelie, chinstrap, gentoo and emperor penguins, the Antarctic fulmar, Antarctic petrel, snow petrel, cape petrel, southern giant petrel, Wilson's storm petrel and the south polar skua. All of these are sea birds and most spend their entire life south of the Antarctic convergence within, or close to, the pack ice zone.

The emperor penguin breeds on the sea ice, while all other species nest on land. The penguins and Antarctic petrels form dense aggregations that tend to be separated by very large distances around the coast of continental Antarctica, although the separation is less between colonies on the northern part of the Antarctic Peninsula. Penguin colonies can exceed 100,000 breeding pairs and separation between colonies ranges from 10s to 100s of kilometres depending the availability of suitable land, sea ice conditions and access to open water at critical times in the breeding season. Little is known about movements of individuals between breeding colonies and the genetic isolation or relatedness between colonies is largely unknown.

Some Antarctic species do leave the Southern Ocean on their migrations and could be exposed to disease agents which they could bring with them on their return to Antarctica. Giant petrels migrate to the coast of the southern continents and Wilson's storm petrels migrate annually to the north Atlantic and north Pacific but do not visit land. The south polar skua has been recorded in Greenland and the Aleutian Islands and has been recorded feeding at sewage outfalls. The skua could be a particularly effective carrier of disease because it moves around the coast of Antarctica and comes into close contact with many other species as an opportunistic predator of Adelie penguins and other birds and as a carrion feeder on birds and seals.

Antarctic birds are not entirely isolated from sea birds breeding on the sub-Antarctic Islands and on the southern continents. The light mantled sooty albatross from Macquarie Island and the black browed albatross from Diego Rameriez Island (Chile) are known to forage for their chicks in Antarctic waters some 2000 km distant. Recent tracking and diet studies have shown that short tailed shearwaters from southeast Australia forage for krill in Antarctic waters. While none of these species intermingle

with Antarctic species on land, the opportunity for interactions at sea are possible. Arctic terns migrate to Antarctica each year and are occasionally observed on land near penguin colonies. Dominican gulls, which visit the Antarctic peninsula, are scavengers at refuse dumps and are capable of moving between the southern continents and Antarctica. Gulls (Hatch 1966) are known to carry a range of bacteria for example *Campylobacter* and *Salmonella* pathogenic to man and other species.

| **Species** | **Breeding locations** | **Migration** |
|---|---|---|
| Wilson's storm petrel | Antarctic continent | Trans equatorial migrant in Atlantic ocean where it feeds in offshore coastal regions. Occurs rarely on land. |
| Arctic tern | Breed in north Atlantic region | Observed on sub Antarctic Islands, moults on ice floes in Antarctica and occasionally found ashore on Antarctic Peninsula |
| Antarctic tern *Sterna vittata* | Sub Antarctic Islands, Antarctic Peninsula | North Atlantic |
| Giant petrel *Macronectes giganteus* | Breeds in small numbers around continent of Antarctica with larger concentrations in the Antarctic Peninsula region. Common breeding species on the sub-Antarctic Islands | Highly migratory species. Opportunistic feeder on offal, carrion. Often seen feeding at the sewage outfalls along coast of southern Australia. Whether on not Antarctic breeding individuals visit the southern continents is problematical. |
| Short tailed shearwater *Puffinus tenuirostris* | Breeds on offshore islands of south eastern Australia. | Feeds on zooplankton in coastal waters of Australia. Regular migrant to Antarctica to feed on krill in breeding season. Has opportunity to overlap foraging range with Antarctic breeding species but never observed close to shore. Migrates to the northern Pacific Ocean in the non breeding season. |
| Southern elephant seal Mirounga leonina | Breeds sub Antarctic Islands and Argentina | Young males migrate to Antarctica eg from Heard and Kerguelen islands to Davis and Macquarie Island to Casey region. Vagrant to Australia. Some suggestion that individuals breeding in Argentina migrate to the Antarctic Peninsula. |

**Table 1.** Some species that migrate between Antarctica and other continents.

### 2.8.2 Human Activity

Human activity in Antarctica could be the cause of disease outbreaks by a number of direct and indirect mechanisms. People could act as vectors for infectious agents, either by bringing non-indigenous pathogens into the region or by translocating indigenous pathogens. In addition stress caused by human activity could reduce immunity, increase pathogenicity and could cause the expression of a disease that might otherwise not have revealed itself. Stress may be the result of direct human disturbance, food shortage (perhaps caused by fisheries competing for the same food stocks), exposure to pollutants and possibly, in the longer term, as a result of climate change. Of course

humans are not the only mechanism for disease incursion to Antarctica. New diseases could be carried to the continent by migratory species and by atmospheric events.

Poultry and poultry products, including eggs, egg powder and frozen or freeze dried meat, may be a source of bacteria pathogenic to humans unless carefully prepared. It is believed also that these organisms are capable of infecting live poultry and other birds. Poultry meat has been linked to the transmission of Newcastle disease to other poultry. There is a possibility therefore that transmission of this and other viral disease such as infectious bursal disease virus to Antarctic birds could occur through this route. There is a long history of feeding kitchen scraps including poultry and eggs to skuas at Antarctic stations and there is a recent report of skua nests containing many chicken bones.

Although the importation of live birds to Antarctic is now prohibited under the environmental protocol pigeons have been taken to the Antarctic for release and caged birds kept as pets on stations. The presence of such species may pose a disease threat through direct contact with indigenous species or through the disposal into the environment of material from the cages.

Sewage is often discharged into the ocean from Antarctic Stations and sometimes illegally by ships. Untreated sewage is likely to contain some organisms pathogenic to humans and may also pose a threat to susceptible indigenous wildlife. Smith and McFeters (this Workshop) suggest there is the potential for transfer of virulence genes from pathogenic microorganisms which may be present in untreated sewage to indigenous microbiota, with unknown effects on susceptible indigenous wildlife.

Vehicles, equipment or clothing used in Antarctica could carry pathogens with them if they are used for recreation, training or work in other locations before being deployed in Antarctica.

The release of captive animals into the wild may carry with it some risk that these animals may harbour disease acquired while in captivity. The possibility exists therefore that they may introduce disease into wild populations (Spalding and Forrester, 1993; Viggers, this symposium).

### 2.8.3 Environment

Stress is a major contributing factor to the outbreak of disease and may have many causes. It may be induced by climatic conditions beyond the normal range, food shortage, catastrophic natural events, habitat destruction and fragmentation, environmental contaminants, direct human disturbance and other more subtle forms of human interference. Antarctic species are well adapted to survive under conditions that would be extreme to species that have evolved elsewhere and they exhibit many remarkable features that enable them to occupy their particular niches. However, their success is at least in part attributable to the absence of land based mammalian predators or niche competition from more competitive species. Although tolerant of the natural environment they are not necessarily immune to environmental stress by particularly

severe conditions particularly those that limit access to food such as unusually extensive, or limited, sea ice and unusual snow cover on nest sites.

Species breeding at the northern edge of their range at the Antarctic Peninsula region may at times also suffer from thermal stress while on land. This stress will increase in the advent of further global warming when temperatures on land are higher and less sea ice is present. The absence of predators makes species less prepared for humans; when they are disturbed by people the response is a rise in heart rate indicative of mild to severe stress and behavioural changes.

### 2.8.4 Disease Vectors

Fleas, sucking lice and particularly ticks (Nuttall 1984) are all capable of transmitting a wide range of disease causing organisms. Ticks and fleas live part of their life cycle away from the host and are then exposed to the extremes of climate. The continuous dry cold environment limits their occurrence in continental Antarctica, however, the Antarctic Peninsula has a more maritime climate and infestations do occur. No outbreak of tick borne disease has been recorded in the Antarctic or sub-Antarctic. If temperatures on the Antarctic Peninsula continue to rise conditions during winter months will become more favourable for the survival of ticks. The infestation of ticks on Antarctic species in itself compounds the stress of increased temperatures and makes these hosts more susceptible to bacterial and viral diseases as well as to cestodes and nematodes.

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# 3.0 REPORTS OF WORKING GROUPS

## 3.1 Risks

### 3.1.1 Risks from Infectious Agents

Disease has the potential to affect all components of the Antarctic biota including the plants and animals of Antarctic marine, freshwater and terrestrial ecosystems. Disease can affect the health and reproductive success of Antarctic wildlife and may be the product of either infectious or non-infectious agents. Infectious agents include exotic, emerging and indigenous agents. Agents considered to be the greatest potential risk to the health of Antarctic wildlife are the highly contagious viral diseases (such as morbillivirus, Newcastle disease and influenza), the immunosuppressant diseases (such as infectious bursal disease, morbillivirus and retrovirus) and agricultural and zoonotic diseases (such as brucellosis, tuberculosis and leptospirosis).

### 3.1.2 Risks from Non-infectious Agents

Non-infectious agents such as pollutants and toxins discharged from stations and ships may be the cause of unusual mortality events and altered health status of biota or they might be just one contributory factor. Indigenous infectious agents that may otherwise remain dormant can cause significant disease when environmental stressors effect host immunity or increase pathogenicity. Increased stress can be the result of natural processes but it can also be caused by human activity.

### 3.1.3 Awareness of Risk

In the absence of definitive information on the possibility of human mediated disease introduction, spread or expression the workshop recommended some simple steps that could be taken now to reduce the risks without causing significant disruption to established activities. Education to increase awareness of the possibility that people could be the cause of disease in the wildlife of Antarctica should, in itself, reduce the risks by causing people to be more vigilant. The implementation of simple surveillance and reporting procedures will increase the chance that a disease event will be known about and the acceptance of quarantine strategies should reduce the chance that a disease agent once reported would be spread. Compliance with the requirements of the Protocol on Environmental Protection to the Antarctic Treaty to eliminate untreated waste streams from Antarctic stations and ships should reduce the chance that disease agents are introduced.

## 3.2 Monitoring

### 3.2.1 Objectives of Monitoring

The goals of monitoring in this context should be to detect unusual mortalities in Antarctic wildlife, to identify the causal agent or agents and to determine whether the causal agent is indigenous or exotic.

### 3.2.2 Parameters for Monitoring

The workshop recommended that parameters for monitoring should include health, morbidity and mortality, population dynamics and external, non-infectious stressors such as environmental contaminants. Routine monitoring should be based upon standardised protocols for selection of species and sites, for collection, storage and transport of samples, for post-mortem techniques and reporting procedures. Serum and specimen banks will be required to house samples and these need to be established.

### 3.2.3 Priorities for Monitoring

Priorities for monitoring should be frequently visited sites and sites where human impact is high. Sites of existing monitoring programs and anywhere that species are known to be threatened should also be monitored. Appropriate control sites, away from human activity, will be essential for reliable interpretation of monitoring results. Monitoring should be reported on regularly and coordinated by a central clearing-house responsible for receiving all results and for making the collated information available to assist in an emergency response.

## 3.3 Prevention

### 3.3.1 Factors Influencing Preventative Measures

There are a number of factors intrinsic to the Antarctic and the type of activity undertaken there that suggest that it should be possible to reduce the chance of a human mediated disease outbreak in Antarctica. The geographic isolation of the continent has meant that until relatively recently it has been naturally quarantined from human activity. The isolation is no longer complete though passage between Antarctica and the other continents is still limited. The good will that exists among both Governmental and non-Governmental operators towards the Antarctic environment and its wildlife is the most important factor contributing to the opportunity to establish sensible measures which will reduce the probability of introduction of serious diseases. It also provides the foundation on which can be established a variety of measures to limit the spread of naturally occurring disease by people.

### 3.3.2 Principles for Preventative Measures

The workshop agreed to the general principles that preventive measures should be based on scientific understanding of the risk of disease introduction and spread and that a precautionary approach should be adopted that recognises that we do not have all the scientific information we need and that builds on existing standards and procedures.

### 3.3.3 Preventative Measures

The workshop recommended that quarantine practices should be applied to both inter- and intra-continental travel and that established management measures such as the protected area system should be used to limit actions which may introduce or spread disease. *Gateway states* could be an effective mechanism for applying disease prevention measures but in the absence of enforced use of gateway states all operators should adopt standard procedures. Uniform practices based on existing and familiar standards and procedures should be applied at point of departure, point of landing, point of dispersal to field sites and in the marine environment. A code of behaviour should be developed and adopted by all operators to ensure that all visitors to Antarctica understand the risk and so that they are motivated to assist in disease control. The free exchange of information on disease should also increase understanding of the risks and could help prevent the transmission of disease agents.

The environmental protocol includes many measures that would reduce the chance of disease introduction and full implementation is to be encouraged. The observation, inspection and reporting requirements of the environmental protocol could all help. Improved waste handling systems in compliance with, or exceeding, the requirements of the protocol could also help.

Non-government organisations such as Antarctic tourist operators have a particularly important role in preventing disease incursion because they tend to focus their activities around aggregations of wild life and frequently visit many colonies on a single voyage. In many locations they will be the most frequent visitors to colonies and in these areas are likely to be the main source of information and data. They can assist with prevention by observing reasonable precautions before arriving at the continent, when near wildlife and between landings. They can also assist by reporting any observed outbreaks.

## 3.4 Response

### 3.4.1 Generic Disease Response Plan

The workshop recommended that a generic response plan should be developed in preparation for possible future disease outbreaks and should be implemented as required for unusual mortality events. The plan should be designed to minimise anthropogenic amplification of the event and should include procedures to help identification of the species involved, the extent of effected animals and the possible causative agents. The response plan should also assist with decisions on whether control actions are appropriate.

### 3.4.2 Readiness and Coordination

The response plan should include elements that assist with readiness for a disease event, administration and coordination, communication, identification of resources and should provide access to information from any precedents. In readiness for any disease event a basic 'disease kit' should be available, with equipment and instructions to facilitate the collection and treatment of material that would permit the identification of the responsible agent(s) with no danger to the sample collector. Administration and coordination should include procedures that assist with the formation of an Incident Control Structure (ICS), the identification of lead agencies and national coordinators, and ensure that the specific needs associated with necessary permits and quarantine are satisfied. Communication procedures should be established to ensure contact with advisory agents, ATCPs, IAATO, other appropriate operations and the media. Resources necessary to respond to a disease event include expertise, equipment, training and funding. Similar animal disease emergencies in other parts of the world can provide precedents for contingency plans and should be used where appropriate.

### 3.4.3 Graduated Response

The workshop recommended that the response strategy should be graduated and tailored to the circumstances of the event. The initial response should be containment by temporary restriction of access to the area and communication to all ATCPs, IAATO, international and national agencies and Antarctic operators. Each event should be carefully documented and reported through a central coordinating body so that the Antarctic community can learn from the event. The workshop strongly discouraged the use of live vaccines in Antarctica.

## 3.5 Research Requirements

### 3.5.1 Research to Understand the Risks

More information on many aspects of disease and Antarctic wildlife is required if the risk of disease introduction is to be managed reliably. A coordinated program of research will be required to provide this information. There is currently little information available on which to base reliable risk assessment. Research is required to develop appropriate risk assessment procedures and to create an inventory of currently known introductions.

Little is known about the survival of pathogens on food stuff, vehicles and clothing or the effectiveness of Antarctic sewage treatment to remove pathogens; the likelihood of transfer of pathogens to wild populations is largely unknown and the susceptibility of Antarctic wildlife to common disease causing agents has not been studied and for ethical reasons would be very difficult to study except during naturally occurring incidents. Carefully considered research targeted at identifying the major risk activities and providing alternatives that reduce the possibility of a disease event should be undertaken as a priority.

### 3.5.2 Research to Understand Disease Status of Antarctic Wildlife

A structured program of scientific research is required to provide information on what is normal and what is aberrant in the health of wildlife species. The research should test hypotheses about the distribution and abundance of potential pathogens and so indicate what occurrences are natural. Clarification of processes for the introduction of infectious disease agents including migration and adventitious introduction such as aeolian plankton will help to ensure that human activity does not contribute to these processes.

Research to understand the processes that lead to flare-up of disease due to indigenous pathogens will also assist with their prevention. These may include large-scale processes such as global climate change that may threaten entire ecosystems or local phenomena such as sewage effluent and contaminants associated with waste disposal. Studies on indicators of general ecosystem health (such as the effects of sewage effluent disposal or contaminants associated with abandoned waste disposal sites) are required to understand the effects of external stressors on the expression of disease that might otherwise go un-exhibited. Research should not be limited to simply understanding these processes but should also be targeted at reducing their effects such as developing improved methods for treating sewage and other wastes.

### 3.5.3 Research in Support of Monitoring

Research must include acquisition of baseline information on disease agents so that native and exotic disease agents can be distinguished and to understand regional and seasonal variation of disease. Baseline information is required on populations at risk including regional and seasonal variations in the size, health status and mortality rates of non-diseased vertebrate populations, their natural occurrence, migratory behaviour and dynamics.

Research will also be required to develop the specific procedures required for maintaining and coordinating serum and specimen banks for Antarctic wildlife.

### 3.5.4 Research in Support of Response Plans

Research should be targeted at providing practical methods for on-site investigation of disease incidents including the development of kits and protocols. Species-specific immunological reagents are required and research is needed to develop new diagnostic tests. Greater understanding of the immunology and antibody responses in Antarctic wildlife are needed for diagnosis. Research should also contribute to the development of standard methods for sampling and post mortem.

Response to a disease incident should be based on the best available information and in general this information is only available for other, very different environments. Research is required to ensure that procedures are modified appropriately to account for the specific conditions found in Antarctica. Response will include a decision on whether

attempts to contain a disease incident are appropriate. An important factor to consider when deciding on containment is the degree of connectivity and interaction between the affected population and other populations and hence the risk of transfer among population groups. Research using genetic procedures should be targeted at understanding the interactions among animals from different colonies and locations.

### 3.5.5 Coordination of Research

Review and analysis of the lessons learnt from disease incidents is crucially important, as is the communication of these lessons. Disease incidents must be used as an opportunity to test hypotheses that, for ethical reasons, could not otherwise be tested and to develop new hypotheses that could be addressed by research. To avoid duplication of effort and to facilitate the rapid exchange of information a scientific working group with expertise in disease should be established to coordinate the whole disease investigation process.

# 4.0 RECOMMENDATIONS ARISING FROM THE WORKSHOP

## 4.1 Introduction

Although there are more unknowns than knowns concerning the possibility of human mediated disease in Antarctic wildlife there are some actions that could be implemented immediately. They will all help to reduce the chance of people causing or contributing to a disease event and in general they can be achieved with relatively little effort. They will not restrict other activities in the region and will contribute information that will assist management of the risk of introduced disease in the future.

There has not yet been a disease outbreak in Antarctic that has been positively attributed to human activity and with some luck and a few sensible precautions this will continue to be the case. This should not lead to complacency however. If these precautions are successful, we will still not have positive proof that people can introduce or spread disease among Antarctic wildlife. This is not evidence that the risk is not present or that these precautions are unnecessary. There have been many cases of wildlife disease caused by human activity in other parts of the world. The following recommendations have been drawn from the foregoing outcomes of the Workshop.

## 4.2 Awareness of Risk

The risk of disease introduction to or spread among Antarctic wildlife by humans has been recognised by some specialists for decades however many involved in Antarctic activities are still unaware of the possibility.

**Recommendation 1:** Managers of national Antarctic programs should raise awareness of the possibility of disease introduction particularly among station leaders and voyage leaders.

**Recommendation 2:** Pre-departure environmental briefings to all expeditioners should include an explanation of the potential for disease introduction and translocation and the simple procedures that should be adopted to reduce the possibility.

**Recommendation 3:** National Antarctic programs should encourage the production and exchange of educational material such as posters and videos.

## 4.3 Information exchange

Exchange of information is important to ensure that if disease is suspected other parties that might visit the area are alerted so that accidental spread can be avoided. A better estimate of the actual incidence of disease will be obtained if all suspected disease occurrences are reported to a central agency.

**Recommendation 4:** A central clearing-house should be established for information on suspected disease occurrences.

**Recommendation 5:** All operators should provide to the central information clearing-house a contact address to receive information.

**Recommendation 6:** The central information clearing-house should report annually to the Antarctic Treaty System through the CEP as a standing item, including negative reports.

## 4.4 Response to suspected disease occurrence

Procedures for recording disease events in Antarctic birds have been published by CCAMLR (CEMP Standard Methods, 1997) and seals by the Department of Conservation, New Zealand and should be used as the basis for a standard response plan.

**Recommendation 7:** All government and non-government organizations operating in the Antarctic should be alerted to these publications and should nominate someone to be familiar with the procedures.

**Recommendation 8:** If disease is suspected the first response should be to stand back, view widely, photograph (preferably digitally) and count dead and dying.

**Recommendation 9:** Access to the site should be restricted to reduce the risk of transfer to uninfected populations.

**Recommendation 10:** If expert support (veterinarian, medical officer, biologist) is available, record symptoms and conduct sampling according to the procedures outlined by CCAMLR or Department of Conservation, New Zealand.

**Recommendation 11:** The minimum information that should be provided to the central clearing-house is,

1. location including coordinates
2. species involved
3. description of event including percentage and total number of animals affected
4. symptoms
5. contact person

## 4.5 Preventative measures

Disease could be introduced or spread by various mechanisms including on clothing, equipment or vehicles; by transfer with contaminated food or by human carriers through sewage disposal systems.

**Recommendation 12:** Operators should be made aware of the potential for disease transfer on clothing, equipment and vehicles particularly if used for other activities such as field training prior to their use in Antarctica.

**Recommendation 13:** Clothing, equipment and vehicles used in Antarctica should be carefully cleaned before being dispatched to Antarctica

**Recommendation 14:** Biocides, such as sodium hypochlorite or iodine solutions that are not persistent environmental contaminants, should be used for washing boots and other equipment when moving between locations; if a biocide is not available repeated washing with water is better than doing nothing.

**Recommendation 15:** Operators should source food supplies free of known diseases.

**Recommendation 16:** The potential for disease introduction from sewage treatment and effluent disposal procedures should be recognised and addressed.

**Recommendation 17:** Live vaccines should not be used as preventative treatments.

## 4.6 Research and monitoring

Research to understand diseases endemic to Antarctic wildlife and the archiving of tissue and blood samples in serum banks are long-term commitments that should be the responsibility of government agencies rather than driven by the interests of individual researchers.

**Recommendation 18:** Managers of national Antarctic programs should note the importance of serum banks and support the establishment of repositories for archival material.

**Recommendation 19:** Fundamental research on disease in Antarctic wildlife including immunology, pathology and preventative measures is needed and should be supported.

# APPENDIX 1 - PROCEEDINGS OF WORKSHOP

## PROGRAM

Day one Tuesday 25 August

| Morning session | Chair | Dr Knowles Kerry |
|---|---|---|
| 0830-0900 | | Registration |
| 0900-0910 | Dr Knowles Kerry | Introduction |
| 0910-0930 | Senator Ian Macdonald | Opening address by Senator Ian Macdonald, Parliamentary Secretary for the Antarctic |
| 0930-0945 | Dr Durno Murray | The development of studies on wildlife diseases in the Antarctic |
| 0945-1045 | Prof. Albert Osterhaus | Viral diseases in seals |
| 1045-1115 | | Morning tea |
| 1115-1140 | Dr Judy Clarke | Occurrence of diseases in Antarctic penguins |
| 1140-1200 | Prof. Geoff Shellam | Previous Australian studies of viruses in penguins on Macquarie Island and Antarctica |
| 1200-1245 | Prof. Geoff Shellam | Bacterial and viral infections of Antarctic penguins |
| 1245-1400 | | Lunch |
| Afternoon Session | Chair | Dr Martin Riddle |
| 1400-1420 | Dr Knowles Kerry | Ecological factors influencing disease in Antarctic wildlife |
| 1420-1445 | Dr David Adams | Disease host/parasite relationships |
| 1445-1530 | Dr Michael Pook | Environmental factors influencing disease in Antarctic wildlife |
| 1530-1600 | | Afternoon Tea |
| 1600-1700 | Workshop | Development of a report strategy; report back to meeting |
| Evening | | |
| 1800-2000 | | Reception at the Antarctic Adventure |

Day two Wednesday 26 August

| Morning session | Chair | Dr Alan Hemmings |
|---|---|---|
| 0830-0900 | Dr David Adams | Summing up of day one |
| 0900-0945 | A/Prof. Don Rothwell | The international and domestic legal framework for the regulation of disease among Antarctica's wildlife. |
| 0945-1030 | Mr Greg Mortimer | Antarctic tourism- size and scope of the industry and standard industry precedures for interacting with wildlife. |
| 1030-1100 | | Morning tea |
| 1100-1145 | Mr Alastair Graham Dr Louise Crossley | Changing risks for the introduction of disease into Antarctica |
| 1145-1215 | Dr PamelaYochem | Health and pathogen seroprevalence and health of Weddell Seals in McMurdo Sound Yochem PK.(presenter),Stewart BS, Gelatt T. and Siniff DB. |
| 1215-1245 | Dr Heather Gardner | Serological evidence for the presence of infectious bursal disease virus in Antarctic penguins. |
| 1245-1400 | | Lunch |
| Afternoon Session | Chair | Prof. Geoff Shellam |
| 1400-1430 | Dr Helena Palmgren to be presented by Dr Mats Sellin | Salmonella isolated from sub-Antarctic animals at Bird Island, South Georgia |
| 1430-1500 | Dr Anna-Christina Broman | Campylobacter species in sub-Antarctic birds |
| 1500-1530 | Ms Jo Gallagher | Investigations of bacterial, viral and parasitic infections in Antarctic penguins |
| 1530-1600 | | Afternoon tea |
| 1600-1630 | Dr Gary Miller | Screening for disease in penguins and skuas |
| 1630-1800 | Workshops | Risks (WG C&D)Monitoring (WG A&B) |
| Evening | | |
| 1900 for 1930 | Reception-Dinner | Royal Tennis Centre (Hobart Tennis Club) 45 Davey Street, Hobarts |

Day three Thursday 27 August

| Morning session | Chair | Prof. Albert Osterhaus |
|---|---|---|
| 0830-0900 | Working group chairman | Reports from workshops |
| 0900-0930 | Dr James Smith | Microbiological Issues of Sewage Disposal from Antarctic Bases: Dispersion, Persistence, Pathogens, and "Genetic Pollution" |
| 0930- 0950 | Dr Heather Gardner Presented by K.R.kerry | Adelie penguins chick deaths- case studies |
| 0950-1010 | Dr Diego Montalti | Mortality of Brown and South Polar skuas by hyphomycetes at Hope Bay Antarctica |
| 1010-1030 | Mr Andrew Jackson | Summing up of Rothwell/Mortimer/Graham papers |
| 1030-1100 | | Morning Tea |
| 1100-1130 | Prof. Geoff Shellam/ Ms Jo Gallagher | Summing up of microbiological papers |
| 1130-1230 | Dr Joseph Geraci | US Marine Mammal Commission experience in handling disease out breaks in pinnipeds in US waters |
| 1230-1400 | | Lunch |
| Afternoon Session | Chair | Dr Joseph Geraci |
| 1400-1500 | Dr Nick Gales/ Dr Padraigh Duignan | New Zealand Sea lion mass mortality event: lessons learnt |
| 1500-1530 | | Afternoon Tea |
| 1530-1545 | Dr Karen Viggers | Potential disease problems associated with reintroduction of seals rehabilitated in captivity |
| 1545-1600 | Dr Ro McFarlane | Morbidity and mortality of the Weddell seals, Vestfold Hills East Antarctica 1990-1991s |
| 1600-1700 | Workshops | Prevention (WG B&D)Response (WG A&C) |
| Evening | | Free |

Day four Friday 28 August

| Morning session | Chair | Prof. Pat Quilty |
|---|---|---|
| 0830-0900 | Working group chairman | Reports from workshops |
| 0900-0945 | Dr David Adams | Methods for controlling the introduction and spread of infectious disease: preparedness and response plans |
| 0915-1000 | Dr Alan Hemmings | NZ management of disease risk in Antarctica |
| 1000-1030 | | General discussion |
| 1030-1100 | | Morning Tea |
| 1100-1300 | Working group sessions | Working group discussions take out put of previous sessions to produce a submission to the ATCM |
| 1300-1400 | | Lunch |
| Afternoon session | Chair | Dr Martin Riddle |
| 1400-1500 | Working group sessions | |
| | Dr Durno Murray | Final report from Working Group A Risks |
| | Mr Andrew Jackson | Final report from Working Group B Prevention |
| | Dr David Adams | Final report from Working Group C Response |
| | Prof. Geoff Shellam | Final report from Working Group D Monitoring |
| 1600-1630 | Afternoon Tea | |
| 1630-1700 | Dr Knowles Kerry | Close of workshop |

## OUTCOMES AGREED BY PARTICIPANTS (REPORTED TO ATCM XXIII)

The workshop **recognised** that there was a significant risk of the introduction of disease into Antarctic wildlife species and that should it occur the consequences are likely to be serious and a response will be required. The recently reported mass mortality of the New Zealand sea lion, *Phocarctos hookeri,* on the sub-Antarctic Auckland Islands in 1998 strongly reinforced these points.

*Baker A. (1999) Unusual mortality of the New Zealand sea lion, Phocarctos hookeri, Auckland Islands, January-February 1998. Dept of Conservation. P.O Box 10-420, Wellington, New Zealand*

The workshop made a number of general recommendations (highlighted in bold) that relate to major issues of minimising the risk of the introduction and spread of disease. However, it was considered that additional information and expertise were required before more specific recommendations were made. The workshop **agreed** that the best way to progress would be the establishment of expert groups in the field of risk, prevention, response and monitoring and that these groups should report ultimately to the CEP.

The following presents the report of the Workshop sessions as agreed by the participants. Information is presented under the title of each of the workshop sessions namely Risks, Prevention, Monitoring and Response.

## Risks What are the risks of disease introduction and spread in Antarctica?

Infectious and non infectious agents may effect the health and reproductive success of biota of Antarctic marine, freshwater and terrestrial ecosystems.

Infectious agents include exotic, emerging and indigenous agents. Indigenous infectious agents may cause significant disease when environmental stressors effect host immunity or increase pathogenicity. Non infectious agents may contribute to unusual mortality events and altered health status of biota. Obligations exist under of the Madrid Protocol to prevent the introduction of non native species, parasites and diseases (Annexe 2, Article 4).

The workshop agreed that there is a risk to the health of Antarctic wildlife from the following causative agents:

- Exotic microbial agents
- highly contagious viral diseases [morbillivirus, Newcastle disease, influenza]
- immunosuppressant diseases [infectious bursal disease, morbillivirus, retrovirus];
- agricultural and zoonotic diseases [brucellosis, tuberculosis and leptospirosis].
- Discharges from ship and station, human movements, migratory species and atmospheric events.

- Indigenous pathogens. Indigenous organisms may become pathogenic when animals are subjected to additional environmental stress such as food shortage and human disturbances and perhaps, in the longer time, as a result of climate change.
- Infection introduced through the presence of exotic and feral biota and non-infectious agents such as pollutants and toxins.

The workshop **recommended** that steps be taken to reduce the risk through,

- enhanced vigilance
- the implementation of surveillance and quarantine strategies
- the elimination of untreated waste streams from bases and ships

# Monitoring **What should we be doing to ensure early detection?**

The workshop identified the following goals for monitoring,

- to detect unusual mortalities in Antarctic wildlife
- to determine whether the causal agent is indigenous or exotic

The workshop **recommended** the following procedures,

- A working group of expertise in disease should be established to coordinate the whole disease investigation process. Parameters monitored should include population dynamics, environmental contaminants, health, morbidity and mortality.
- Routine monitoring should be based upon standardised protocols for selection of species and sites, for collection, storage and transport of samples, for post-mortem techniques and reporting procedures.
- Serum and specimen banks will be required and need to be established.
- Priorities for monitoring should be at frequently visited sites, at sites where human impact is high, at appropriate control sites, at the sites of existing monitoring programs and where threatened species are involved.
- Monitoring should be coordinated, reported on regularly and the results should be readily available to assist in an emergency response.
- Research is required to distinguish between native and exotic disease agents, to understand regional and seasonal variation of disease and the size, health status and mortality rates of non-diseased vertebrate populations. New tests and greater understanding of the immunology and antibody responses in Antarctic wildlife are needed for diagnosis.

# Prevention **What procedures could reduce the risk of disease?**

The workshop agreed that the following general principles should be applied to disease prevention,

- preventive measures should be based on scientific understanding of the risk of disease introduction and spread,
- precautionary approach should be adopted which recognises that we do not have all the scientific information we need and builds on existing standards and procedures.

The workshop **recommended** the following procedures should be considered.

- Apply quarantine practices to intra-continental travel, use the protected area system and related measures to limit actions which may introduce or spread disease
- Use *gateway states* to apply agreed procedures
- Apply uniform practices at point of departure, point of landing, point of dispersal to field sites and in the marine environment
- Base procedures on existing and familiar standards and practices
- Adopt a code of behaviour to ensure that all visitors to Antarctica understand the risk so that they are motivated to assist in disease control
- Freely exchange information to increase understanding of risks
- Maintain and improve waste handling systems
- Amend Environmental Protocol to resolve inconsistencies, implement existing protocol and ensure compliance through observation, inspection and reporting requirements

## Response **What should we do if introduced disease is suspected?**

The workshop **recommended** that a generic response plan should be developed in preparation for possible future disease outbreaks and should be implemented as required for unusual mortality events. The plan should include procedures to,

- identify the extent, cause and source of the event
- minimize anthropogenic amplification of the event
- consider whether control actions are appropriate

The response plan should also include the following elements,

- readiness: A basic 'disease kit' should be available, with equipment and instructions to facilitate the collection and treatment of material that would permit the identification of the responsible agent(s) with no danger to the sample collector.
- administration: including the formation of Incident Control Structure (ICS), the identification of lead agencies and national coordinators, and the specific needs associated with the required permits and quarantine.
- communication: with advisory agencies, Antarctic Treaty Consultative Parties (ATCPs), the International Association of Antarctic Tour Operators (IAATO), other appropriate operations and the media.
- resources: expertise, equipment, training and funding.
- precedents: contingency plans developed for similar animal disease emergencies.

- live vaccines: their use in the Antarctic is strongly discouraged.

The workshop **recommended** that the response strategy should be graduated and tailored to the circumstances of the event. The initial response should be containment by temporary restriction of access to the area and communication to all ATCPs, IAATO, international and national agencies and Antarctic operators.

Each event should be carefully documented and reported to a central coordinating body so that response procedures can be modified in the light of experience from the event.

# ABSTRACTS

**Of Papers Presented**
**at the**
**Workshop on Diseases of Antarctic Wildlife**

## CAMPYLOBACTER SPECIES IN SUB-ANTARCTIC BIRDS

**Tina Broman[1], Mats Sellin[1], Helena Palmgren[1], Dominic Mccafferty[2], Sven Bergström[1] and Björn Olsen[1]**

[1]Departments of Microbiology, Clinical Bacteriology and Infectious Diseases, Umea University, Sweden,
[2]British Antarctic Survey, Cambridge, UK

Campylobacteriosis is a widely spread zoonosis of great importance, causing enteritis or reproductive disorders in a number of mammalian species. Wild living and domesticated birds often harbour the bacteria and thereby constitute reservoirs for infection.

Until now there has been little knowledge on Campylobacter spp. In Antarctica. We have investigated faecal samples from birds, collected at Bird Island, South Georgia. Campylobacter were found in samples from Gentoo penguins (Pygoscelis papua), Macaroni penguins (Eudyptes chrysolopus), albatross species (Diomedea spp.) and skuas (Catharacta loennbergi). In order to clarify if the strains found are unique to the area, we used different pheno- and genotyping characterising methods. A selection of the samples was sequenced in respect of the 16S rRNA gene, thus enabling comparisons to database strains.

Among the investigated samples we found strains with 16S rRNA sequences exhibiting only minor differences compared to C. jejuni and C. Lari respectively.

We discuss the results of our investigations, the methods used and the possible impact of different Campylobacter species in this area with its vast populations of birds and seals.

## OCCURRENCE OF DISEASE IN ANTARCTIC PENGUINS

**Judy Clarke**

Australian Antarctic Division, Channel Highway, Kingston 7050

Penguins are susceptible to a number of infectious and parasitic diseases but clinical signs of disease have rarely been observed in wild populations. There have been very few studies carried out to examine the incidence of disease among penguin populations, and only a limited number of reports of individual diseases in any other than captive penguins. This paper presents a summary of all disease agents which have been found and documented in Antarctic and sub-Antarctic species of penguin with an emphasis on those agents most likely to cause disease outbreaks if introduced in virulent forms into naïve colonies.
Disease agents are discussed under the headings of

- Parasites (ectoparasites, endoparasites and protozoa)

- Viruses
- Bacteria and fungi

Three of the important avian viral diseases are discussed in detail as they have the potential to pose the greatest disease threat to Antarctic bird life. These viruses are:

- Newcastle disease virus and other avian paramyxoviruses
- Avian influenza viruses
- Infectious bursal disease virus

Antibodies to all these viruses have been detected in Antarctic or sub-Antarctic penguins, although clinical signs of disease have not so far been observed. The significance of the presence of these antibodies remains uncertain, especially as virus particles themselves have been less commonly isolated. The viruses in all these groups occur in a number of serotypes which vary in pathogenicity. It is possible that birds infected with viruses of low pathogenicity may thus develop resistance to related but more dangerous serotypes. However, it is still important that we ensure that humans do not facilitate the introduction of virulent strains of these diseases into Antarctica as we cannot be sure what the outcome of such an introduction could be.

## NEW ZEALAND SEA LION MASS MORTALITY EVENT: LESSONS LEARNT

**Nick Gales[1] And Padraig Duignan[2]**

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An unusual mass mortality of New Zealand sea lions *Phocarctos hookeri* occurred at the remote subantarctic Auckland Islands in January/February 1998. *P. hookeri* is a threatened New Zealand endemic that breeds almost exclusively at the Auckland Islands (>95% pup production); its total population is estimated to be 10-12,000. At least 53% of the pups born during the breeding season (early Dec '97- mid Jan '98) had died by 20th February '98. Carcasses of 74 adult and juvenile females and 11 males were found over the same period. However, the full extent of non-pup mortality is unknown. At the onset of the mortality event, pups were in good condition and within normal body mass ranges.

The initial phase of the event was primarily characterised by the deaths of large numbers of apparently healthy pups and a sudden desertion by most adult females. The proportion of adult females that died at this time is unknown. Most of the pups that died during the initial phase of the event did not show signs of illness. However, a few had lesions consistent with superficial bacterial infections. Later in the event, starvation was a contributing factor to pup mortality. Clinical signs among non-pup age classes included one or more of the following: unilateral swellings of the cranial cervical

region; raised focal lesions in the skin and superficial blubber on the ventral abdomen and flanks; body stiffness, difficulty in moving and in some cases dragging of hind quarters. Based on a survey conducted on 30th January, 18% of adult females had one or more of these signs while on 8th February approximately 9%were affected.

No principal cause for the mortality event has been identified. Gross pathology and histopathology indicated that bacterial septicaemia was the cause of death for most animals examined. Several potentially pathogenic bacteria were isolated including five *Salmonella* serotypes, and an unidentified pleomorphic gram-negative organism associated histologically with necrotizing arteritis. No viruses have been isolated from tissues and there were no lesions suggestive of viral infection. However, four of 28 serum samples had low titers against phocine distemper virus suggesting previous exposure to this, or a related morbillivirus. Biotoxins were not detected in tissue, milk, stomach contents or environmental samples but sampling for this cause was probably inadequate. Seawater temperatures at the time of the mortality event were 2°C colder than usual and may be indicative of some unusual environmental conditions.

We conclude that most mortalities were caused by a variety of highly pathogenic bacteria (principally several serotypes of *Salmonella* and an unknown organism). It is likely that these organisms occur normally in the environment, but that morbidity and morality were acutely increased during January and February 1998 due to an undefined suite of environmental factors that stressed the sea lion population and decreased its immunity.

This investigation was compromised by the remote location in which it occurred and by a general lack of preparation for such an event. Many useful lessons were learnt and these are discussed.

## INVESTIGATIONS OF BACTERIAL, VIRAL AND PARASITIC INFECTIONS IN ANTARCTIC PENGUINS

**J.M. Gallagher[1], K. Kerry[2], H.I. Jones[1] And G.R. Shellam[1]**

[1]Dept. of Microbiology, The University of Western Australia, Nedlands, Perth, Western Australia, 6907.
[2]Australian Antarctic Division, Dept. of Environment, Kingston, Tasmania, Australia, 7050.

Penguins breeding in the Antarctic have evolved in the most isolated ecosystem in the world receiving only minimal exposure to infectious agents from other regions. However the ease and speed of modern transport has brought changes that may have serious effect on the penguins health and survival. Yearly expansion of human activity in the region, in both tourism and science, is increasing the risk of introduction of infectious agents. It also causes stress and disruption to the normal activities of penguins which can result in increased susceptibility to infection. While much research has been carried out into the breeding biology and foraging ecology of Antarctic penguin species little is known about the disease status of wild populations. This lack of baseline information on what is the normal endemic microflora of these animals makes

it difficult to evaluate any changes which may occur and therefore is required for the development of future monitoring schemes. We have obtained faecal and blood samples from over 1000 Adelie (*Pygoscelis adeliae)* and Emperor *(Aptenodytes forsteri)* penguins at frequently visited and remote sites in Eastern Antarctica and representing each stage of the Adelie breeding season. Analysis has revealed the presence of aerobic and anaerobic bacteria in all faecal samples, with a number appearing to be new bacterial species. A dominant example of this is a coryneform bacterium isolated from >90% of birds. We have also found that the complexity of the aerobic flora increases as the birds come ashore after six months at sea and begin egg incubation. The majority of arriving birds yielded only a single bacterial isolate. However after only 2-4 weeks in the colony 38% had 3 or more isolates with the number yielding a single isolate dropping to 19%. This suggests that egg incubation is a period of heightened susceptibility to infection and that human disturbance should be minimised during this time. Screening for antibiotic resistance among the bacterial isolates from the penguins has revealed all to be susceptible to antibiotics, in contrast to bacteria isolated from the sewage outfalls at both Mawson and Davis stations where significant resistance was detected. The screening of sera for antiviral antibodies and the examination of faecal samples for infectious virus and blood smears for the presence of parasites is in progress.

## DISEASE IN PENGUINS IN ANTARCTICA: THE USE OF RISK ANALYSIS AS AN APPROACH

**Heather Gardner**

Australian Quarantine and Inspection Service, Department of Primary Industries and Energy, Canberra, ACT 2601

Little is known about disease in penguins in Antarctica, There is a perception that Antarctica is a pristine, harsh and cold environment in which pathogens do not survive. The potential for diseases to be introduced into Antarctica has been acknowledged by the Environmental Protocol to the Antarctic Treaty. However, the pattern and level of human activity has changed since this was developed.

There has been an increase in the movement of people to and within Antarctica Tourist and expeditioner numbers have been increasing in recent years and air travel quickly takes people, equipment and food to distant localities. These factors directly contribute to an increase in the risk of importing an exotic organism and or spreading of endemic disease. The issue of exotic disease in wildlife in Antarctica, and specifically penguins is raised in this paper.

The risk analysis process is a structured method of examination of the issues pertaining to the introduction of disease into Antarctica. While the lack of knowledge of diseases in penguins and an understanding of the processes of diseases in this environment may preclude the development of firm guidelines, the risk analysis process will highlight areas for further investigation and lead to subsequent preventative and management strategies.

**The Risk Analysis Process**

The risk of introduction of exotic disease into penguins is examined through the process of qualitative risk analysis. This is a 4 stage process entailing the identification of the risk, assessment of the risk, its management and communication.

The risk identification relates to a knowledge of the disease that are endemic and exotic to penguins. Whilst a number of viral and bacterial pathogens have been identified in penguins, further investigation is required to establish what the status of disease is in penguins. Until this occurs, those pathogens not recorded in penguins can be assumed to be exotic. The disease status of other seabirds may assist in identifying potential organisms that may be pathogenic to penguins.

In assessing the risk of introduction and establishment of disease in penguins, pathway of introduction is first examined. Some of the factors to consider are the country of origin of the expedition, source of food and equipment and the level of contact with penguins and their environment. The significance of these factors could vary from region to region.

An assessment of the risk of disease becoming established in a susceptible population would be difficult to obtain. A lack of knowledge of disease and its epidemiology exists in penguins. The ecology of micro-organisms and their survival in Antarctica conditions and the impact of disease amongst penguins can only be speculated upon.

Management of the risk of an exotic disease is addressed at 3 levels: local, the Australian government and international levels. The field and station personnel apply basic quarantine procedures. The Australian Antarctic Division addresses the operational liaison and policy issues and liaises at the international level to inform Antarctic Treaty members and other international stakeholders.

In undertaking a risk analysis, communication with the stakeholders is essential for the acceptance of any management strategies. Communication and consultation through all stages of the risk analysis is essential.

**Conclusion**

The use of risk analysis approach to examine the issue of importation of disease into penguins shows gaps in the knowledge and understanding of the processes of disease in this environment.

## ECOLOGICAL FACTORS INFLUENCING THE INTRODUCTION OF DISEASE INTO ANTARCTIC BIRDS.

**Knowles Kerry, Rupert Summerson and Eric Woehler**

Australian Antarctic Division, Channel Highway, Kingston 7050, Tasmania, Australia.

Antarctica is geographically isolated from the continents to the north by a vast encircling ocean. The nearest continent South America is 1500 km to the north. South Africa is 4100 km and Australia 3600 km. Its avifauna consists of only 11 species comprising Adelie, chinstrap, gentoo and emperor penguins, the Antarctic fulmar, Antarctic petrel, snow petrel, cape petrel, southern giant petrel and Wilson's storm petrel and the south polar skua. All of these are sea birds and most spend their entire life south of the Antarctic convergence within or close to the pack ice zone. This paper will explore the possibility of Antarctic species contracting an exotic disease given this degree of isolation, the climate of Antarctica and the absence of known vectors.

The emperor penguin breeds on the sea ice, while all other species nest on land. The penguins and Antarctic petrels form dense tend to be separated often by very large distances around the coast of eastern Antarctica but less so at the northern part of the Antarctic Peninsula. Penguin colonies can exceed 100000 breeding pairs. Separation between colonies ranges from 10s to 100s of kilometres depending the availability of suitable land, sea ice conditions and access to open water at critical times in the breeding season. Little is known about movements of individuals between breeding colonies and genetic isolation may be present.

Wilson's storm petrels migrate annually to the north Atlantic and north Pacific but do not visit land. Giant petrels migrate to the coast of the southern continents; the south polar skua has been recorded in Greenland and the Aleutian Islands. and is recorded feeding at sewage out falls. The skua moves around the coast and is an opportunistic predator of Adelie penguins and other birds.

Further, Antarctic birds are not entirely isolated from sea birds breeding on the sub-Antarctic Islands. The light mantled sooty albatross from Macquarie Island and the black browed albatross from Diego Rameriez Island (Chile) are known to forage for their chicks in Antarctic waters some 2000 km distant. Recent tracking and diet studies have shown that short tailed shearwaters from south east Australia forage for krill in Antarctic waters. While none of these species intermingle with Antarctic species on land, the opportunity for interactions at sea are possible. Arctic terns migrate to Antarctica each year and are occasionally observed on land near penguin colonies.

It remains to be determined whether migratory birds are able to introduce disease into Antarctic species. However, given that albatrosses and the shearwaters are able to sustain flights of 50 km/hr for a day or more, it is possible, though not necessarily probable, for an infected to reach Antarctica from South America and possibly from Australia within an incubation period of 3-7 days. Migratory birds or vagrants which are

carriers of infectious disease may be a more potent source of infection of Antarctic seabirds.

## MORTALITY OF BROWN AND SOUTH POLAR SKUAS BY HYPHOMYCETES AT HOPE BAY, ANTARCTICA

**G.A. Leotta[1], M.A. Petruccelli[1], D. Montalti[2] and E.H. Reinoso[3]**

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Mortality of nineteen skuas, thirteen Brown skuas Catharacta antarctica lonnbergi and six South Polar skuas Catharacta maccormicki, at Hope Bay, (63¯24'S, 56¯59'W) at the northermost end of the AntarcticPeninsula is described. Dead birds were found and collected within a period of fifteen days in February 1997. They were kept in plastic bags at -20¯C and necropsy was performed. Samples of heart, trachea, lung, intestine, proventricle and liver were taken. Necropsy showed that in nine birds pericardium and peritoneal bags were thicker, with a fibrinous aspect and a whitish colour. In four birds tracheal mucous membrane was removed and light was obstructed with a fibrinous and yellowish substance. In five birds there were nodular wounds of one to two centimeters on the costal surface.

Histopathological study was carried out. Samples were stained with H-E and PAS technique. In eleven cases lungs showed pneumonia, intra-alveolar edema, congestion, and the presence of fungus mycelium in the parenchima. An abundant quantity of fungous elements was obstructing traqueal light.

Micological studies in nineteen samples of trachea and lung were performed. They included microscopic observation, culture and taxonomic identification. The presence of hyphae and talconides was microscopically demostrated. Eumycetes strains were isolated in all organs with wounds histopathologically described.

Taking into account previous reports describing a total number of 96 skuas (Brown and South Polar) at Hope Bay, it is possible to infer that mortality of micotic origin was 11 %.

## MANAGEMENT OF DISEASE RISK IN THE ROSS SEA REGION

**Alan D. Hemmings**

Environmental Assessment and Review Panel
(Ministry of Foreign Affairs and Trade, New Zealand)

The Ross Sea region is, after the Peninsula, the most visited region of Antarctica, and the risk that human activity may introduce, or spread, disease organisms needs to be addressed in environmental management of the region. Activities directly involving New Zealand, which may provide vector mechanisms include: the national Antarctic programme operated by Antarctica New Zealand, supported by the airbridge from Christchurch to McMurdo involving aircraft of the RNZAF, which deploys field parties widely and moves between a number of potentially sensitive biological sites; and tourism expeditions mounted from (or terminating at) New Zealand ports, which make landings in both the subantarctic and Antarctic Treaty Area, and which may entail semi-circumpolar voyages between biologically sensitive sites. For New Zealand management agencies, avian or seal disease organisms may pose particular concerns, given similar taxa (eg penguins) found in the Antarctic, New Zealand subantarctic and New Zealand mainland. Present precautionary measures adopted by New Zealand for Antarctic operations are described and questions posed concerning future steps that may need to be taken to minimise the risk of introduction of new potential disease organisms or spread of existing (and possibly natural) disease organisms.

## MORBIDITY AND MORTALITY OF THE WEDDELL SEAL, VESTFOLD HILLS, EAST ANTARCTICA 1990–1991: DIFFICULTIES IN ASSESSING THE HEALTH OF ICE SEALS

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The Weddell seal *(Leptonychotes weddelli)* inhabits the fast ice of the Antarctic continent and whole population processes can be examined in detail relatively easily. Despite this *Green* et *al (1992)* documented an 80% decline over 12 years in aerial counts of Weddell seals in the Vestfold Hills region near Davis (63 31 'S, 78 1 2'E*)* made during the moulting haul out. Pup production did not decline over this period.

This population of Weddell seals presents an opportunity to make multiple observations of tagged individuals ( and during the most stressful period of the annual cycle ) which may provide information about morbidity and mortality factors in the species and detailed observations were made during the 1990-1991 season. Ocular, skin, respiratory. gastrointestinal and reproductive disease were described and pup mortality was investigated by post mortem examination (McFarlane 1996).

The diseases observed in this study did not appear to present an important challenge to the overall health and regulation of the population although it did provide a baseline of morbidity. Green et al (1995) proposed that the apparent declines in the Weddell seal numbers were related to interannual differences in the time seals spent foraging and prey availability. The limited number of carcasses available in this and other studies for post mortem examination makes direct associations between morbidity and mortality difficult and it must be concluded that the heaviest mortality occurs at sea or during winter.

Baseline understanding of morbidity and mortality are an important part of our understanding of health factors that regulate popuIations and can be compared over time or examined in more detail experimentally. Despite the isolation of Antarctica and its wildlife increasing human activity in the region raises the likelihood not just of epizootics of exotic pathogens but of the more subtle effects of decreased survivorship and fecundity through clinical and subclinical disease. In monitoring the heath of these populations an awareness of environmental and human associated stresses (eg tourism, pollution, increased ultraviolet light effects on the food chain, commercial fisheries) which may contribute to increases in frequency of disease or immunosuppression in addition to the level of quarantine necessary for protection from novel pathogens is recommended.

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## SCREENING FOR DISEASES IN PENGUINS AND SKUAS

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The recent discoveries of certain diseases in Antarctica have created concern for the future of Antarctic wildlife. Introduced microorganisms may have severe negative consequences for immunologically naïve wildlife and novel epidemics may be especially disastrous for rare or endangered species. In this paper, I present information on the presence of an internal parasite, Coccidia, in the Adelie penguin, the Galapagos penguin, and the south polar skua. In addition, I have screened the penguins for Marek's Disease Virus. I used standard parasitological techniques to search for Coccidia in fecal samples from 116 skuas from Ross Island, 44 Adelie penguins from Ross Island, and 24 Galapagos penguins. No Coccidia were found in any of the penguin samples and only one skua sample indicated infection. I used DNA amplification

techniques (PCR) to screen blood (or tissue) samples from Adelie penguins (n=29) from around the Antarctic peninsula and >from Galapagos penguins (n=109).

Many other pathogens can be identified from the samples and I will discuss plans for continuing work.

The methodology used to screen the samples will necessarily vary according to the disease in question. Because Marek's disease virus is a DNA virus and found in lymphocytes it is easily identified in blood samples using DNA amplification techniques (PCR). Likewise, Plasmodium, found in erythrocytes, lends itself well to PCR as a means of detection. Because of the absence of insect vectors, avian malaria is not likely to concern us in Antarctica, but may be present in other penguins. In contrast, a disease such as Newcastle disease, owing to its transitory nature in the infected host, is less amenable to molecular techniques. In this case we must rely on well established serological screening techniques.

Finally, I will discuss additional pathogens and some of the issues related to the search for diseases in Antarctic avifauna.

## ANTARCTIC TOURISM - SIZE AND SCOPE OF THE INDUSTRY, AND STANDARD INDUSTRY PROCEDURES FOR INTERACTING WITH WILDLIFE

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Approximately 7,000 to 10,000 people visit Antarctica as tourists each austral summer. Tourists are a highly mobile population in Antarctica, visiting a number of sites within a short space of time. Antarctic tour operators collaborate via the highly successful industry organisation, IAATO (The International Association of Antarctic Tour Operators), which is dedicated to appropriate, safe and environmentally sound private sector travel to the Antarctic. With due regard to the transfer of exotic organisms between visitor sites, IAATO members have developed a series of standard operating procedures to avoid translocation of exotic organisms.

This paper examines the size and scope of Antarctic tourism in the late 1990's, offers projections for future growth in Antarctic tourism and describes standard operating procedures of IAATO members when interacting with Antarctic wildlife, including measures taken to avoid the translocation of exotic organisms.

## THE DEVELOPMENT OF STUDIES ON WILDLIFE DISEASES IN THE ANTARCTIC

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At the 3rd meeting of the Scientific Committee on Antarctic Research (SCAR), March 1959, I expressed the need for recommendations for the conservation of Antarctic fauna and flora, a task which became a responsibility of the permanent SCAR Working Group for Biology. The lst SCAR Symposium on the Antarctic Biology in 1962 sought contributions to assist this task, and the concerns about the risks of introduction of diseases were incorporated into the Agreed Measures for the Conservation of Antarctic Flora and Fauna within Article IX and Annex D. The risks remain today exacerbated by the increasing number of visits which regularly link annually the previously isolated stations and pristine coastlines.

Interests in diseases have followed trends of developing competence. Earlier, they were of organisms that could be seen easily. The upsurge of microbiological techniques in the 60's and 70's led to a probing interest. Nowadays, with the increased studies of diseases of wildlife there is an active interest. It is now possible to preserve the microbiological present for the future, to recapture the past, particularly where sera banks exist, and, with the increased understanding of the ecology of Antarctic wildlife, to contemplate epidemiological studies.

## METHODS FOR CONTROLLING THE INTRODUCTION AND SPREAD OF INFECTIOUS DISEASE: PREPAREDNESS AND RESPONSE PLANS

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Epidemiology is the study of patterns of diseases that exist under conditions in the field. Its practical purpose is to provide rational grounds for the prevention and control of disease in animal populations. Epidemiological knowledge underwrites the preparedness and response plans formulated for disease emergencies in domestic animals in Australia. As a discipline, epidemiology merges ideas from pathology, the study of disease, and ecology which deals with the interrelationships between organisms and their environment. It depends absolutely on the processes of diagnosis (1) to make sense out of the interactions between host organisms, infectious disease agents (microparasites and macroparasites) and the environment and (2) to identify primary and secondary causative factors in outbreaks of disease.

By and large, epidemiological knowledge has come from experience in the diagnosis, control and therapy of disease in populations of people and domestic animals and has

been applied to these same populations. However, an understanding of the ecological concepts that guide epidemiology has come from studies with populations of wild animals. To what extent can the methods of disease diagnosis, control and therapy used for domestic animals be applied to populations of wild animals? Are the reasons for disease control the same for domestic animals and wild animals? If they are different, do they make a difference to methods for disease control? What other ideas from biology can be invoked for their utility in making decisions about disease control in wild animals?

This paper looks to the Australian veterinary emergency plans (AUSVETPLANs) as examples of planning for disease preparedness and response. The AUSVETPLANs are based on experience with domestic animals and on the possibilities for managing populations that may apply to domestic animals but not wild animals. What guidance can be had from these plans for managing disease in wild animal populations in Antarctica? What are the strictures? One comes from the uniqueness of any given host-parasite relationship. As a consequence, knowledge about disease incubation times and the like for one host-pathogen relationship does not extend to other hosts species infected by the same pathogen. So, the uncertainties are even greater for the management of disease in wild animals. Procedures for risk analysis are designed to cope with uncertainty. These are being applied to quarantine issues in Australia. What are these procedures and how can they be applied to diseases of wild animals in Antarctica?

## VIRAL DISEASES IN SEALS

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During the last decade, we and others have identified infections with previously known and newly recognised morbilliviruses, as the primary cause of mass mortalities among wild pinniped and cetacean species inhabiting coastal waters. Due to the immunosuppression induced by these, infections with several other agents, including viruses, could also be identified. This eventually led to the identification of viruses belonging to at least eleven families of aquatic mammals. Due to their pathogenic significance some of these infections have a major impact on population dynamics, whereas others are limited to small numbers of animals within a population. Interspecies transmission of viruses has been shown to be at the basis of many major outbreaks of viral diseases of aquatic and terrestrial mammals in recent years. In addition we have shown that immunosuppression due to the effects of environmental pollutants accumulated via the aquatic food chain, may have contributed to the severity and extent of outbreaks of viral diseases among aquatic mammals.

Collectively our data show that, although viral infections should be considered a natural component of the ecosystem of aquatic mammals, human activities often significantly

contribute to the development or triggering of conditions which lead to viral disease outbreaks in these animals.

## SALMONELLA ISOLATED FROM SUB-ANTARCTIC ANIMALS AT BIRD ISLAND, SOUTH GEORGIA.

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Human activity in Antarctica poses a threat of introducing non-native microorganisms. The basic information on what is to be considered normal native microbiological flora and data on the prevalence of infectious diseases in Antarctic wildlife is scarce. This study was undertaken to determine the occurrence of *Salmonella* and thermophilic *Campylobacter* spp. in wild populations of seabirds and seals on Bird Island, South Georgia (54°S 38°W).

In 1996 and 1998 approximately 700 faecal swabs were collected from gentoo (*Pygoscelis papua*), and macaroni (*Eudyptes c. chrysolophus*) penguins; grey-headed (*Diomedea chrysostoma*), and black-browed (*D. melanophrys*) albatrosses; Antarctic scuas (*Catharacta loennbergi*) and Antarctic fur seals (*Arctocephalus gazella*) at the British Antarctic Survey base at Bird Island. This presentation will focus on the findings of *Salmonella*. The results on *Campylobacter* are prepared as a separate presentation.

## ENVIRONMENTAL FACTORS INFLUENCING DISEASE IN ANTARCTIC WILDLIFE

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Most descriptions of the climate of the Antarctic region emphasise the cold, dry and windy nature of the continent but these descriptions can mask the tremendous variety of weather experienced across the vast expanse of Antarctica and adjacent ocean throughout the year. Increasingly, studies are demonstrating links between the Antarctic region and the atmospheric and oceanic circulations of lower latitudes.

The study of the climatology of the Antarctic is limited by the period of record for which data are available and the sparse network of the climatological stations. Many meteorological stations on the Antarctic mainland date from the International Geophysical Year (IGY) of 1957-58, giving a maximum record of around 40 years. Despite the patchy nature of the climate record, the period since the IGY has seen a

growth in understanding of the processes whereby Antarctica interacts with the Southern Hemisphere and the global atmosphere-ocean system. Among these are the major effects of the sea ice formation each year, the advection of heat and water vapour over the continent from lower latitudes and the outflow of cold, continental air from Antarctica to lower latitudes. Recent research has focussed on possible teleconnections between the Antarctic and El Niño signals in the tropical Pacific Ocean (Smith and Stearns, 1993).

Mean annual temperature records for most Antarctic stations exhibit a warming trend with the strongest signal appearing over the Antarctic Peninsula. Furthermore, numerical models of the atmosphere have emphasised that the greatest temperature changes in a warmer global climate due to anthropogenic effects are likely to occur in the polar regions. These changes in mean temperatures are considered unlikely to significantly affect environmental conditions favourable to disease in Antarctica in the short to medium term but when coupled with extended periods of altered atmospheric circulation in the vicinity of the Peninsula could increase the risk of transmission of organisms from lower latitudes.

Studies of historical data sets have revealed cases of significant circulation anomalies associated with atmospheric blocking events in the Pacific and southwest Atlantic Oceans. These events have been shown to advect relatively warm and humid air over Antarctica from middle latitudes during the winter months and could play a vital role in the survival of exotic organisms.

**Reference:**

Smith, S. R. and C. R Stearns, 1993. Antarctic climate anomalies surrounding the minimum in the Southern Oscillation Index. In Antarctic Meteorology and Climatology: Studies based on Automatic Weather Stations (D. H. Bromwich and C. R. Stearns, eds.). Antarctic Research Series 61, p149-174. American Geophysical Union, Washington D.C.

## BACTERIAL AND VIRAL INFECTIONS OF ANTARCTIC PENGUINS

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Antarctic microbiology has a long history, dating from basic studies carried out during exploration in the early years of this century.

Studies of bacteria isolated mainly from faeces have revealed the presence of a range of aerobic and anaerobic bacteria, many of which resemble organisms encountered in human or veterinary clinical practice. Most studies have focused on aerobic bacteria and have identified organisms which in man are harmless commensals as well as *Salmonella* sp, *E. coli* and *Edwardsiella* sp which are potential pathogens.

Sequential studies during a single breeding season have revealed an increasing complexity to the microflora as the birds proceed through egg incubation and chick rearing, suggesting that there may be periods of heightened susceptibility to infection. Using the presence of plasmid-mediated antibiotic resistance amongst coliforms as a surrogate marker for exogenous bacteria (human or animal origin), little evidence of human impacts on the microflora of penguins has been obtained so far, although resistant bacteria are present in sewage outfalls at bases.

However, bacteriological studies are often confounded by inadequate methodologies for collection and transport of specimens, and by the difficulty of identifying bacteria, many of which appear to be new species.

Viral infections have been less well studied. Antibodies to avian influenza virus have been detected in Adelie penguins, and antibody studies and virus isolations have revealed that avian paramyxovirus infections (including Newcastle disease virus) are widespread in Adelie penguins. Recently, serological evidence of infection of Emperor and Adelie penguins with infectious bursal disease virus has been reported. While this gives cause for concern, the role of any of these viruses in disease causation in Antarctic penguins in unknown.

Virological studies suffer from inadequate reagents for viral diagnosis and from the lack of knowledge of the immune responses of penguins to viral antigens, which makes the timing of sampling and the interpretation of results difficult.

Exciting challenges lie ahead in our attempts to understand the normal micro flora of penguins and the effect of human impacts. Greater co-ordination of effort and provision of research funds would be very beneficial.

## THE INTERNATIONAL AND DOMESTIC LEGAL FRAMEWORK FOR THE REGULATION OF DISEASE AMONG ANTARCTICA'S WILDLIFE

**Donald R Rothwell**

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International law has during the past 40 years developed a range of mechanisms to regulate activities in Antarctica with a progressive focus on the protection and conservation of Antarctic fauna and flora. This process commenced in earnest with the 1964 Agreed Measures for the Conservation of Antarctic Flora and Fauna and has recently culminated in the entry into force of the 1991 the Protocol on Environmental Protection to the Antarctic Treaty (the Madrid Protocol) to the Antarctic Treaty. In furtherance of the international obligations Antarctic Treaty parties are under, a range of domestic laws and policies have been adopted to give effect to these international laws adopted through the Antarctic Treaty System. This paper will assess these developments by first assessing relevant Antarctic Treaty law that has sought to deal with controls of disease, plus other relevant international laws which have addressed this problem such

as the 1992 Convention on Biological Diversity. It will then assess how Antarctic Treaty parties have responded to this issue by way of domestic legislation and review the obligations imposed upon Antarctic expeditioners and others travelling to Antarctica from Australia, New Zealand, the United Kingdom and the United States. The paper will conclude with some recommendation for reform in the law.

## MICROBIOLOGICAL ISSUES OF SEWAGE DISPOSAL FROM ANTARCTIC BASES: DISPERSION, PERSISTENCE, PATHOGENS, AND "GENETIC POLLUTION"

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Sewage is most often discharged untreated from polar bases due to logistical constraints associated with secondary and primary treatment. Untreated sewage is likely to contain some human pathogenic microorganisms, and thus may represent a risk for disease transmission to susceptible indigenous wildlife. The dispersion of untreated sewage from the McMurdo Station (Ross Dependency) marine outfall has been investigated, focusing on microbiological indicators of fecal pollution (coliforms, fecal coliforms), and degradation of organic material (biochemical oxygen demand [BOD]). The long-term persistence and physiological responses of several indicator (*Escherichia coli*, *Enterococcus faecalis*), pathogenic (*Salmonella typhimurium*, enterotoxigenic *E. coli*, *Yersinia enterocolitica*), and conjugative/antibiotic resistance plasmid-harboring (pUC19, pFamp) bacteria were also investigated *in-situ* in the local marine environment. Sewage plume dispersion indicating movement towards the drinking water intake for McMurdo Station, along shore tidal sea-ice cracks, and corresponding to an apparent current gyre in Winter Quarters Bay were observed. The presence of high numbers of fecal indicator bacteria in the annual sea-ice in the vicinity of the station was also observed. Our work demonstrated the longest survival times yet reported for the above bacteria in the marine environment, and significantly reduced rates of long-term organic material degradation (20-day BOD) compared to more temperate conditions used for standard assays (20$^{o}$C). Cold temperatures also produced sublethal injury and the “viable-but-nonculturable” (VBNC) response in all bacterial strains exposed, precluding accurate enumeration using standard techniques. Thus, standard techniques for assaying microbial and organic constituents of sewage in this environment represent *significant* underestimates, and should be modified accordingly. Activity of the above enteric bacteria (as measured by respiratory activity) was limited organic material availability, rather than temperature. Thus, discharge of organic material may increase the already significant persistence of these bacteria in this environment. In addition, bacterial strains harboring both conjugative, and antibiotic-resistance plasmids showed increased survival, the VBNC response, and maintained their transferable genetic material (plasmids) throughout *in-situ* exposures of 21-54 days in length. Together with increased persistence, these results indicate the potential for

transfer of virulence, and/or antibiotic resistance genes from pathogenic microorganisms, which may be present in untreated sewage, to indigenous microbiota, with unknown effects on susceptible indigenous wildlife. The term "genetic pollution" has been used to describe the introduction of new genetic material, or the transfer of genes in the environment, resulting from anthropogenic activities. More appropriate indicators of organic and microbial pollution have been proposed. However, the release of large volumes of untreated sewage into the Antarctic marine environment represents a significant possible source of disease-causing agents for indigenous wildlife.

## DISEASES IN SEALS: A VETERINARY ECOLOGICAL PERSPECTIVE

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An understanding of the epidemiology of disease in free ranging populations of seals is required to identify species or populations at risk, as well as potential sources of infection. Information derived from studies following mass mortalities of seal populations in the northern hemisphere has provided insight into the behaviour of viral disease epidemics. However, current knowledge of disease status of free-ranging populations of Antarctic seals is limited. Speculation on the possible impact of specific diseases is not possible without prior knowledge of the disease agents that are endemic in seal populations. Suggestions are outlined of field studies to determine the disease status of Antarctic seal populations. This will help to clarify our understanding of the potential implications of disease introduced by various possible sources, as well as expanding our understanding of the fluctuations of seal populations in general.
In addition, issues associated with the reintroduction of seals that have been rehabilitated in captivity will be discussed. Zoos are often faced with decisions relating to the treatment of debilitated, weak seals that haul out in public places. Ethical questions concerning potential outcomes for rehabilitated seals, as well as perceived disease threats upon reintroduction and release will be discussed.

## AUSTRALIAN STUDIES OF VIRUSES IN PENGUINS ON MACQUARIE ISLAND AND ANTARCTICA

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Surveys of penguins on Macquarie island [Morgan, Westbury and others, Aust.vet J **57**:333-335 (1981)] and in Antarctica [Morgan and Westbury, Avian Dis **25**: 1019-1026 (1981) and FJ Austin (unpublished data)] during 1976-79 resulted in the isolation of nine avian paramyxoviruses. The strains could be divided into four distinguishable

groups, three of which have not been previously described [Alexander, Manvell, Collins, Brockman, Westbury, Morgan and Austin, Arch Virol **109**: 135-143 (1989)].

The recognisable virus was a member of avian paramyxovirus serotype 1 (Newcastle disease virus - NDV) and was isolated from a Royal penguin at Hurd point, Macquarie island. Serological studies revealed antibody to NDV in other Royal penguins at this location which suggests that the virus was endemic at the site during the time of sampling. Morgan and Westbury (1981) also detected NDV antibody in penguins in Antarctica, albeit at a low prevalence (2 positive of 164 tested), but sufficient to indicate that penguins in at least one area of Antarctica were infected with NDV. Thus it appears that infection with NDV occurs in penguins in the sub-antarctic and antarctic region though the disease significance of this infection is unknown. However it is known that penguins are susceptible to pathogenic strains of NDV as captive Adelie and King penguins have succumbed to the disease [Pierson and Pfow, J Amer vet med Assoc **167**:801-803 (1975), Kraus, Paulick Huchzermeyer and Gylstorff, Dtsch tierarzt Wochenshr **70**: 307-309 (1963)]. The other three avian paramyxovirus groups were clearly new though the small number of strains in each group precluded definitive categorisation with respect to the current classification of avian paramyxoviruses, except that they increase the complexity within the avian paramyxoviridae. Limited serological testing indicates that antibody to two of these new groups is present in three different species of penguins, and at widely separated sites in Antarctica so they probably are endemic and part of the normal ecology of penguins. Nothing is known of the pathogenicity of these viruses, or what part they may play in the few reports of unusual mortality in Antarctic penguins [Budd, Proc Zoo Soc London **139**:365-388 (1962), Nelson, p166 “Seabirds – their biology and ecology” Hamlyn Publishing Syd (1980)]

Testing of Antarctic penguins also showed the presence of serum antibody to Avian influenza virus with haemagglutinin 7 [Morgan and Westbury 1981]. This virus, together with avian influenza virus strains with haemagglutinin 5, is able to cause severe disease in gallinaceous birds, but not usually in water- and sea- birds though there is no data on the their impact in Antarctic birds,

Thus there is data from Antarctica and sub-Antarctica of infection of penguins with two viruses (ie NDV and Avian influenza) able cause severe disease in birds in other parts of the world. The impact these viruses have, and could have, in penguins and other birds there is not known. Likewise there is infection of penguins with a previously undescribed set of avian paramyxoviruses which have yet to be completely characterised. In particular there is no data on the pathogenicity of these viruses for penguins, or, indeed, for birds in other parts of the world.

# HEALTH AND PATHOGEN SEROPREVALENCE OF WEDDELL SEALS *LEPTONYCHOTES WEDDELLII* IN MCMURDO SOUND, ANTARCTICA

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Little is known about morbidity and mortality of Antarctic phocids, with a few notable exceptions (Laws and Taylor 1957, Stirling 1969, Seal et al. 1971, Liggins et al. 1979, Osterhaus et al. 1988, Bengtson et al. 1991, Schumacher et al. 1992, Stenvers et al. 1992, Castellini et al. 1996, McFarlane 1996). Development of biomedical reference ranges is an important first step in assessing wildlife population health (e.g., Fowler 1986, 1993; Dierauf 1990). Without these normal values, it is difficult to evaluate the relative impacts of natural environmental change and anthropogenic influences. The population of Weddell seals in McMurdo Sound is ideal for such study for two reasons: 1) demographic and ecological data, compiled for several decades, provide detailed histories on individual animals, a rarity in studies of wildlife health; and 2) its proximity to a major settlement makes it a good candidate for assessing the impacts of human activities on Antarctic wildlife (e.g., Howington et al.1993).

We conducted physical exams and collected blood samples from 39 clinically healthy Weddell seals (17 adults, 22 pups) and from14 seals (11 adults, 3 pups) with significant abnormalities on physical exam (e.g., suppurative wounds) during the 1996/97 and 1997/98 breeding seasons. Standard veterinary hematological and serum biochemical profiles were developed by age and sex class. We also surveyed all seals at two sites for lesions as described by McFarlane (1996) for the Vestfold Hills population. Our goals were to compile a baseline biomedical database for Weddell seals, including the prevalence of infectious and non-infectious disease.

Most blood parameters were within reference ranges reported for other phocid seals, though several varied with age and condition ($p < 0.05$). Total white blood cell counts, monocyte counts, serum urea nitrogen and creatinine were higher in adults; serum glucose, cholesterol, triglycerides and iron were higher in pups. Clinically healthy adults had higher red blood cell counts, lower white blood cell counts, and fewer bands and neutrophils than seals with abnormal physical exam findings. Clinically healthy pups were larger (length and girth), had higher hematocrits and serum glucose levels, had fewer bands and lower serum creatinine kinase and lactate dehydrogenase than pups that were abnormal on physical exam. Physical injury (e.g., conspecific trauma) was the most common non-infectious lesion observed. No previously unreported parasites were detected during examinations of faeces or blood smears. Finally, our preliminary pathogen seroprevalence data suggest that this is a relatively naive population, which has important implications regarding its susceptibility to epizootics (c.f., Duignan et al. 1995).

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## APPENDIX 2 – CCAMLR STANDARD METHODS

PROTOCOLS FOR COLLECTION OF SAMPLES FOR PATHOLOGICAL ANALYSIS IN THE EVENT OF DISEASE BEING SUSPECTED AMONG MONITORED SPECIES OF BIRDS

**Commission for the Conservation of Antarctic Marine Living Resources (CCAMLR)**

**CCAMLR Ecosystem Monitoring Program**

**Standard Methods**

# PROTOCOLS FOR COLLECTION OF SAMPLES FOR PATHOLOGICAL ANALYSIS IN THE EVENT OF DISEASE BEING SUSPECTED AMONG MONITORED SPECIES OF BIRDS

## INTRODUCTION

Disease and parasitism occur in all colonies of birds but in many cases they are not apparent. Instead, they can exist at subclinical levels and manifest in periods of stress or changes in circumstances in the colony. Overt disease, usually recognised by deaths, is obvious. Subclinical disease is unlikely to be recognised although it may be suspected in times of reduced chick production or generalised failure to thrive.

This section outlines the basis for a pathological assessment in the event of disease being suspected among monitored species of birds.

The following protocol is provided for collection of specimens in the field where primitive or no laboratory facilities are available and personnel undertaking the investigation may have little training in pathology. It is not expected that the cause of death or disease will be established at the time as microbiological analysis and follow-up investigations are generally required. Long delays in the diagnosis are expected. Detailed labelling and recording of the specimens, storage and description are of the utmost importance.

It is recommended that all field teams conducting CEMP programs receive instruction on the collection of specimens and on basic anatomy of birds and post-mortem techniques outlined in this document. Field teams should maintain stocks of sampling equipment at their monitoring site.

It is important to consult with a veterinary pathologist before going to Antarctica to ensure samples can be analysed. The laboratory may also have special requirements for the collection and storage of specimens.

## BACKGROUND INFORMATION

### Prevention of the Spread of Disease

While birds may die of non-infectious causes, the presence of an infectious agent should

always be assumed. The potential to spread disease from colony to colony is ever present; the consequences of this may be devastating. Bacteria and viruses can survive at low temperatures, some are extremely resistant to adverse conditions. Pathogens can be spread mechanically by adhering to clothes, equipment and vehicles.

While it is important to establish how widespread the suspected outbreak of disease is, it is critical not to spread pathogens. Visits to other colonies should not occur unless measures to reduce microbial contamination such as cleaning and disinfection of equipment and clothing, especially boots, has been carried out.

## Epidemiological Information

While a parasitic or microbial pathogen actually causes disease, there are usually many other factors that contribute to the outbreak of disease or the death of a bird. Factors such as stress, starvation, excessive predation, disruption by humans, inclement weather, etc. will contribute to the conditions in which clinical disease can occur. These factors are important in determining the cause of an outbreak, its epidemiology and understanding the implications of disease on the breeding performance of a population. Such data need to be recorded in addition to the collection of pathological samples and carcasses.

It is important therefore to record such factors as:

- demographic factors: species, sex, age, reproductive status, stage of breeding cycle, colony size;
- environmental factors: location, weather, time, date, geography of the colony, human access and intervention, presence and activity of predators;
- number of ill and dead birds, age of affected birds, the location of affected birds in the colony: proportion of birds which recover or are affected clinically; and
- description of the symptoms of the disease.

## Human Health and Hygiene

A number of avian diseases, some of which have been recorded in Antarctic birds, are contagious to humans and some can produce serious disease (e.g. *Chlamydia* spp. (Psittacosis), *Salmonella* spp., *Mycoplasma avium* (Tuberculosis) and avian influenza). On the other hand, a number of pathogenic and non-pathogenic organisms carried by humans cause disease in birds. Diseased or environmentally-stressed birds would be more susceptible to such pathogens and care should be taken to minimise the risk of introduction of diseases to birds. Precautions such as those listed below should be taken to prevent the transfer and spread of disease between humans and birds:

- wear rubber gloves;
- wear a surgical mask if in a poorly-ventilated room or if the person doing the dissection or in close contact with birds has a respiratory infection;
- wet down the feathers of the bird or fully dip the bird in water before examining the carcass or opening the abdomen;
- open the bird in a well-ventilated room or area, but not in windy conditions;
- cuts and scratches on personnel should be treated with disinfectants as soon as possible;
- wear protective clothing and change and wash clothes after handling ill or dead birds;
- observe sensible hygiene measures; and
- be aware of the occupational and safety measures applying to the use of formalin, liquid nitrogen and absolute alcohol.

The Investigation

An investigation of the death of a bird involves the observation and description of clinical symptoms (if present), the external examination of the bird and the collection of samples and performance and reporting of the post-mortem. It may not be necessary to conduct a detailed post-mortem, but the following steps will allow a systematic approach to the collection of tissue samples and other samples for microbiological and parasitic assessment.

Examination of the internal organs should be performed in a systematic manner so as to avoid microbial contamination of organs and ensure that all organs are examined. To reduce the risk of cross-contamination, swabs and impression smears for microbial analyses should be taken progressively before any organs are removed or samples of tissue are taken. Tissues can be taken from most organs when the gastrointestinal tract and the thoracic organs are *in situ.* The gastrointestinal tract should be removed in order to investigate for parasites, to collect stomach and intestinal contents for dietary and bacteriological analyses and to examine the kidneys and gonads which are obscured by the intestines.

Detailed records supported by colour photographs, taken progressively, particularly before the tissues are collected, will greatly assist the investigation of the cause of illness and mortality in a colony. Description on audio tape and video may also be of value to the investigation.

External Examination of a Bird

Examination of a bird should commence with palpation to feel for broken bones or any other abnormalities. *Rigor mortis* and freezing of the carcass may hinder detection of this. Injury and other lesions (e.g. tumours, lumps, areas of feather loss and discharges) should be described. The description should include colour, consistency and size.

A systematic examination of the bird should be followed as suggested below:

- measure body weight;
- morphometric assessment – beak length and depth, wing and mid-toe length;
- integument – condition of the skin and plumage, signs of trauma, scabby lesions of the skin, look for external parasites;
- head – eyes, nares, beak, oral cavity and ears, look for discharges, note colour and consistency, colour of mucous membrane – lesions in the mouth, swellings;
- neck – swellings and any injuries;
- body condition – fat, normal, emaciated, dehydrated;
- abdomen – distension indicates that the bird has fed recently, flat indicates that the bird has not fed recently;
- brood patch – presence of scabby lesions; red and vascular as seen when the bird is brooding;
- vent – cloaca: soiled or caked-up; diarrhoea, blood, colour of excreta;
- preen gland (above the base of the tail);
- wings – injuries, deformities; and
- legs – injuries, deformities.

## Collection of Samples

### Whole Bird Collection

In cases of serious outbreaks, with many birds sick and dying, entire carcasses should be collected. Specimens should represent a range of ages, sexes (if known), clinical symptoms and be as fresh as possible. Post-mortem changes reduce the quality of histological and microbiological analysis of the bird.

- Collect a minimum of three to five birds.
- Wrap individually in plastic if possible and place in plastic bags.
- Freeze (-20 to -70°C) as soon as possible.
- To identify individuals and relate samples to each other, number each bird.
- Label each bird with details of its number, its sex if known, age, from where it was collected, when and by whom.
- Prepare a full inventory of the specimens collected.

### Tissue Collection

Choose birds which exhibit a range of symptoms of disease before they die or are euthanised. Collect tissue samples from all major organs from dead birds, specifically from those which show macroscopic lesions (e.g. white spots on the liver).

- Tissue samples should be collected from the intestine, pancreas, liver, kidneys, spleen, lung, heart, brain, thymus and bursa (in chicks) and any abnormal lesions; in the case of small birds leave the intestines coiled, make sections across the coil and fix; this avoids handling the delicate tissue.
- Use a scalpel blade to remove tissue samples.
- Each sample should be labelled with the type of tissue, the number/identity of the bird, the place of collection, the date and time and name of the person who collected it.
- Prepare a full inventory of the specimens collected.

*Histopathology samples*:

- Store tissues in 10% buffered formalin.

- Cut pieces of tissue no greater than 1 $cm^3$.
- Store 1:10 (tissue-preserving fluid) for two to three days.
- Transfer fixed tissue to another container containing only a small amount of formalin to keep the tissue moist.
- Do not let fixed tissue freeze.

*Microbiology samples*:

- For each sample of tissue collected for histopathology, collect another sample for virus isolation and identification.
- Freeze and store at -70°C.

*Toxicology samples* – (see Part IV, Section 5)

*Intestinal contents*:

- Stomach contents should be collected and fixed in 70% alcohol.
- Samples should be well labelled on the container in pencil or alcohol- and water-resistant pen. An additional label written in pencil should be placed inside the container.

Egg Collection

The failure of eggs to hatch can have a significant impact on the recruitment of birds into the breeding colony in the future. Several bacterial and viral diseases can affect the viability of the chick in the egg.

- Collect fresh or incubated eggs.
- Store frozen at -70°C if possible.
- Label in pencil on the shell and on attached paper the identity number and type of egg, the place of collection, the date and time and name of the person who collected it.
- Prepare a full inventory of the specimens collected.

## Collection of Blood for Serology

An antibody titre indicates that a bird has, at some stage, been in contact with a specific disease. The level of the antibody titre can indicate whether there has been recent contact or active infection. Recently-infected birds will have the highest antibody titres.

- Using aseptic techniques collect 2 to 3 ml blood from the brachial or tibial veins into a glass tube or plain sterile blood collection tube.
- Avoid clotting of the blood during collection by obtaining a good blood flow.
- Avoid freezing of the blood during collection by performing venipuncture protected from the wind.
- Keep the blood collection tubes warm (e.g. on the inside of your jacket) and stand overnight in warm conditions to encourage clotting.
- Avoid freezing as this will lyse the red blood cells and discolour the serum.
- Blood samples can be spun down by centrifugation to obtain more serum.
- Pipette off the serum, avoid contamination with cell fraction.
- Store serum and cell fraction in cryotubes. Cryotubes should have the thread on the outside of the tube to minimise the loss of serum when the cap is removed.
- Freeze serum at -20°C to -70°C. Store cell fraction at -70°C if possible, as it can be used for microbiological investigations.
- Label serum and cells with place and date of collection, identification number of the bird, species, chick or adult, sex if known and who collected the specimen.
- Prepare a full inventory of the specimens collected.

## Collection and Preservation of Parasites

### Ectoparasites

Lice and ticks are usually found where they cannot be removed by preening eg. under the bill, in the ear canals, on top of the head and along the back. The brood patch also may provide an ideal site for ectoparasites.

- Take skin scrapings of scaly areas, in the centre and on the edges of the lesion.

- Preserve specimens in a 70% ethyl alcohol and 5% glycerol solution.
- Label each container with place of collection, date, species, approximate age of the bird and sex if known and who collected the sample.
- Prepare a full inventory of the specimens collected.

Endoparasites

Round and tape worms and flukes are found in the gut and organs. The intestinal tract is opened from the stomach to the cloaca after removal from the abdominal cavity. The general procedure for the collection and preservation of endoparasites is as follows:

- Wash off excess intestinal contents, fluids and debris and gently remove parasites from the lumen.
- Dissect and handle endoparasites gently as they are fragile.
- Fix in 10% formol saline or in warm to hot 70% ethyl alcohol for later examination.
- Preserve transverse sections of parasitised tissue in 10% formol saline.
- Label samples with details of the collection site, time, species, identification number of the bird, age, sex if known and who collected the specimen.
- Prepare a full inventory of the specimens collected.

*Nematodes (round worms)*

These worms are found in the trachea, oesophagus (even under the lining), stomach and small intestine. Check the subcutaneous and visceral lining tissues for any cysts or walled-off lesions. Larval nematodes can encyst. Parasites in the lumen of the intestine or trachea can be collected as described above.

- Hold parasites in warm normal saline (0.9% NaCl) solution for several hours before fixing.

*Trematodes (flukes)*

Flukes are found in the small intestine, lower intestine, cloacal antrum, kidneys, gall bladder and liver and caused damage to the associated tissues and organs. Check the

blood vessels of the gut mesentery and kidneys for vascular flukes if there are any abnormalities in these organs.

- Hold flukes in warm normal saline (0.9% NaCl) for several hours.
- If the flukes are very contracted, place in distilled water for a few hours. Osmosis will cause the fluke to swell and relax.

*Cestodes (tape worms)*

Adult tape worms are found in the lumen of the intestine. Larval cestodes can occur in subcutaneous fat or in body cavity as a cyst or a bladder-like sphere. The adults are fragile and often numerous.

- Collect a few whole specimens from head (scolex) to gravid terminal proglottis.
- Wash gently in distilled water for a few hours until they relax.

Haemoparasites

Haemoparasites can be identified in blood smears. The smears can be stored indefinitely and examined at a later date.

- Make a thin blood smear on a microscope slide. This may take some practice.
- Air dry. Avoid blowing heated air on the slide.
- Fix in 100% methanol.
- Store in a dry, dark place.
- Label slide with details of the collection site, time, species, identification number of the bird, age, sex if known and who collected the specimen.
- Prepare a full inventory of the specimens collected.

## Collection and Preservation of Material for Investigation of Bacterial, Viral and Fungal Infections

Infectious diseases occur in birds in Antarctica. Juvenile mortalities can be associated with infectious or opportunistic microbial diseases when birds are under stresses such as starvation, predation and crowding. Adults are rarely found dead in a colony. Viral or bacterial diseases may be suspected in cases of sudden death. Clinical symptoms may

not be apparent. However, in less acute illness or less rapid mortality some clinical symptoms may be apparent. These could include: discharge from the eyes or mouth, coughing, sneezing, laboured breathing, nervous signs, tremors, convulsions and diarrhoea. Viral infections should also be considered when scabby lesions occur on unfeathered regions or in the mouth.

It is important to collect blood for antibody analysis, and to take swabs for culture from the palatine fissure, trachea and cloaca. Birds displaying a range of symptoms as well as birds showing no evidence of disease should be sampled.

- Collect material on a sterile swab.
- Do not use swab with a wooden stick to collect *Chlamydia* spp. as the timber can be toxic to the organism.
- Place swab in a sterile cryotube.
- Add chilled transport medium to the swab as soon as possible.
- Store swabs for viral and bacterial isolation at -70°C.
- Bacterial samples collected in Ames charcoal transport media tubes should be stored at 0°C to 4°C.
- Do not allow these swabs to freeze.
- These samples should be cultured as soon as possible.
- Media for sample storage:

  viral sample –
  brain heart infusion broth containing antibiotics;

  bacterial sample –
  brain heart infusion broth without antibiotics;

  mycoplasma and *Chlamydia* sample –
  brain heart infusion broth without antibiotics.

## EXAMINATION AND DISSECTION OF DEAD BIRDS

Details of the dissection procedure (a post-mortem) are given as a guide to the collecting samples in such a way as to minimise contamination. This is not a priority and if undertaken is best conducted in a clean, comfortable environment. A detailed pathological assessment of a bird can take several hours to complete. Colour

photographs of the opened bird and the organs will assist in diagnosis of the cause of death.

Post-mortem Dissection Technique and Examination

The procedures for carcass dissection and examination of the organs follow.

- Wet bird down, in warm running water containing detergent if bird is soiled.

- Place bird on its back on a well lit, dissection board covered with a disposable surface such as paper or plastic. Support may be needed either side of the bird to keep it upright. Dislocate hips in all birds except penguins, if necessary.

- Part the feathers and make a skin incision over the sternum or keel. Extend incision to the midline of the beak and to the vent, taking care not to cut through the abdominal wall. Peel skin back with fingers until the neck, all the chest (pectoral) and abdominal muscles are exposed, and extend down the thighs and legs where possible. This is necessary to avoid contamination of the abdominal and thoracic cavities with feathers. Extreme care is needed in small birds and birds which have been dead for some time as pressure can rupture the abdominal musculature.

*Examination Note:*
*Make a subjective assessment of the bulk and the colour of the muscles; the presence of haemorrhage in the muscle and under the skin should be described. Haemorrhages can appear as red spots, splashes or bruises.*

- Open the abdominal cavity with scissors, cutting along the midline and the posterior border of the thoracic cavity while holding up the abdominal wall with rat-tooth forceps. Care must be taken not to pierce the gall bladder, liver or intestinal tract. Fold back the abdominal muscles so that the abdominal contents are exposed.

*Examination Note:*
*Colour of liver, size of gall bladder, presence of fluid in the abdominal cavity – quantity, colour and consistency; colour and distension of loops of intestine; the presence of food in the stomach and intestine.*

- Cut the pectoral muscles with a scalpel blade along either side of the sternum, across the surface of the ribs. Use bone cutters or sturdy scissors, depending on the size and maturity of the bird, to cut through the sternal ribs and lever the sternum up to expose the thoracic and anterior abdominal contents. The air sacs are exposed. Cut through the clavicles to remove the sternum.

- Alternatively, to maximise exposure of the thoracic cavity most of the ribcage can

be removed by cutting across the ribs as dorsal as possible. This will disturb the air sacs but examination of them is still possible.

*Examination Note:*
*Air sacs are transparent membrane sacs located in the thorax and abdomen and should contain no fluids. Note the presence of any fluid, its colour and consistency. Take swabs of fluid or material. Abnormalities in the membrane thickness and transparency of the air sac wall should also be recorded. Record the colour and consistency of fluid and other unusual material in the thoracic cavity and pericardial sac – the membranous sac containing the heart. In addition, describe any tumours or other lesions in the lungs.*

- Cut through the right mandible and hyoid apparatus and open the oral cavity. Examine and take samples and swabs from the tongue, palatine fissure, oropharynx, glottis, larynx and thymus (in young birds) where applicable. Open the oesophagus and take swabs and samples as necessary.

*Examination Note:*
*Look for evidence of swelling, discharges, discolouration, lesions, etc.*

- To remove the gastrointestinal tract, transect the oesophagus between two ties which occlude the lumen, low in the thoracic cavity and the large intestine, close to the cloaca. The intestine can be lifted out while gently breaking the mesentery and suspensory ligaments. Care should be taken not to rupture the gall bladder as the bile will discolour tissues.

- The bursa of Fabricius is located near the vent in young birds – examine and take samples.

- Open the trachea, syrinx and pericardial sac. It is generally not necessary to take out thoracic organs.

*Examination Note:*
*Note the presence of fluid, froth, its consistency and colour in the lumen of the trachea. The sac is normally translucent and has a shiny surface. Record any thickening of the membrane, the presence of any fluid and or material in the pericardial sac.*

- Skin the head before removing the brain. The head can be removed from the neck at this stage. Cut through the skull using scissors or bone cutters. The brain should be removed with minimal handling. Drop the brain out under gravity, tipping in an anterior to posterior direction.

- Open wing and leg joints.

*Examination Note:*
*The joint fluid is normally clear and the cartilage white and smooth. Take swabs of*

*fluid or material in any joint that is not clear.*

- Bone marrow can be obtained from the medullary cavities of the femur and tibia. If no marrow can be found, submit ribs for histology.

## LIST OF RECOMMENDED EQUIPMENT

The equipment listed below is sufficient to enable a detailed collection of samples. Items marked with a single asterisk are the minimum required to collect specimens from birds in the field for further study. Collection of samples for identification of microorganisms from sick birds is a priority.

### Ancillary equipment:

storage containers*
clothes – overalls*
hand warmers
hot-water bottles*
insulated containers or boxes to prevent freezing of material*
liquid nitrogen cylinder or freezer*
plastic sheeting for ground cover*
vacuum flasks
plastic bags – large and small*

### Chemicals:

alcohol – absolute
disinfectants*
10% buffered or saline formalin
formalin – chemicals to make up 10% buffered formalin
methanol – absolute
normal saline – 0.9% NaCl
stains – e.g. Diff-Quick, new methylene blue or giemsa
sterile water
glycerol

### Data recording equipment:

camera with 35 mm and 50 mm macro lenses*
cassette tape recorder
counters – to do counts in colonies
field note books – preferably water resistant paper*

film – colour slide is preferable*
freezer bag pens
labels for bodies*
labels for specimens*
pencils*
permanent marker pens*
rubber bands*
erasers
video camera

Microbiological equipment:

cryotubes – 2 ml, 5 ml, 10 ml*
culture media for bacteria culture and storage*
culture media for virus storage*
swabs – sterile plain wood stick, plastic stick for *Chlamydia**
transport media swabs – Ames charcoal transport tubes*

Post-mortem equipment:

adhesive tape*
alcohol or isopropanol tissues*
aluminium foil
bone cutters
bone saw
bottles 20, 50, 100 ml*
centrifuge
cover slips
diamond pencil
dissection boards – plastic*
disposable overalls*
drawing pins or tacks
forceps:  plain and rat-toothed
glass containers
glass slides*
gloves – latex, powder-free*
knives – plastic handles*
labels
large garbage bags*
needle disposal container*
paper towels
plastic bags – freezer resistant various sizes
rubber gloves – various sizes, long, thick*
ruler*

scalpels – blades and handles, number 22 and 11
scissors – fine and sturdy blade
screw top, wide-mouthed plastic containers*
string*
surgical masks
tape measure*

Serology and haematology:

blood collection tubes – heparin and plain, 2.5 ml, 5 ml, 10 ml*
blood tubes holders*
needles 21, 23, 27 gauge 1 inch*
Nunc tubes for storage of serum
slide box
syringes 3 cc, 5 cc, 10 cc*

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## APPENDIX 3 - DISEASES IN PENGUINS AND SEALS

Judy Clarke and Knowles Kerry

# DISEASES IN PENGUINS AND SEALS

Judy Clarke and Knowles Kerry

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## INTRODUCTION

Disease has been reported in penguins and seals in both wild and captive environments. This review concentrates on reports and investigations of wild populations. Diseases found in wild animals need to be differentiated from those observed in captive species since some conditions appear to be restricted to animals in captive environments.

Penguins are susceptible to a number of infectious and parasitic diseases but clinical signs of disease have rarely been observed in wild populations. There have been very few studies carried out to examine the incidence of disease among penguin populations, and only a limited number of reports of individual diseases in any other than captive penguins.

Seals are affected by a variety of diseases, both in the wild and in captivity. Most reports of disease outbreaks in seals are from populations in the Northern Hemisphere. The only documented mass die-off of seals in Antarctica occurred in 1955 (Laws and Taylor 1957). A number of serological investigations of seals in south polar regions have been carried out during more recent years (Bengtson and Boveng 1991; Harder *et al.* 1991; Austin and Webster 1993).

This paper presents a summary of all the major disease agents that have been found and documented in penguins and seals with an emphasis on those agents most likely to cause disease outbreaks if introduced in virulent forms into naïve colonies.

## AGENTS OF DISEASE

### 1. Parasites:

These can be divided into three categories:

- Ectoparasites which live on the skin.
- Endoparasites which live inside the body. These include various types of worms which generally parasitise the gastro-intestinal tract, but may also be found in other organs.
- Protozoa which are single-celled parasites living in either the bloodstream or the gastro-intestinal tract.

### 2. Viruses.

These are comprised of nucleic acids and protein. Viruses invade host cells and alter normal cell processes to cause the invaded cells to produce large amounts of virus particles that can then cause disease.

3. **Bacteria and Fungi.**
   These are single-celled organisms that multiply within the tissues of infected animals and birds to cause disease.

## RESISTANCE TO DISEASE

- The presence of an agent of disease in an animal or bird does not necessarily mean that clinical symptoms of disease will develop. Healthy animals are often able to carry low levels of potentially harmful organisms without succumbing to disease.

- Animals frequently develop immunity to disease agents (particularly viruses) following low levels of exposure or exposure to less virulent forms of the agent. Such immunity (in the form of antibodies) then prevents disease developing even when the animal is challenged with higher levels or more virulent forms of the disease agent.

- Detection of antibodies in wild penguin and seal populations provides evidence of previous exposure to disease agents. Presence of antibodies does not necessarily mean that an animal has suffered from clinical disease, but it does indicate that the animal has been exposed to the specific disease agent or to an antigenically related agent in the recent past.

- Disease may often be difficult to detect when it occurs at a subclinical level. This is particularly the case for wild populations in which symptoms are not necessarily obvious, and when signs may be confused with other conditions, for example, starvation. Subclinical disease can have various effects, including decreased breeding success and reduced resistance to other diseases.

## ECTOPARASITES

There are four groups of ectoparasites that have been described in penguins and seals: ticks, fleas, mites and lice.

- Ticks of the genus *Ixodes* are widely distributed around the sub-Antarctic and the Antarctic Peninsula (Zumpt 1952; Murray and Vestjens 1967; Hawkey *et al.* 1989; Murray *et al.* 1991), and affect penguins more commonly than seals. Ticks require a sheltered well-drained habitat and densely nesting hosts, and are found most commonly in tussocky areas. They tend to infest chicks more than adults, and are found around the eyes and mouth, the webs of the feet and the cloaca. Heavy infestations may kill chicks and debilitate adult birds. Ticks can also act as carriers of viral diseases and blood parasites.

- Fleas have only been found on penguins in sub-Antarctic regions since they spend large parts of their life cycle off the host and thus need sheltered, dry habitats such as caves for their survival (Murray *et al.* 1967; Murray *et al.* 1991).

- Nasal mites are common in young fur seals. Heavy infestation can cause respiratory disease (Kim *et al.* 1980).

- Biting lice have been found on most sub-Antarctic and Antarctic penguin species, and live and breed on their hosts (Murray 1964). They eat feathers and skin debris, but do not suck blood.

- Sucking lice are found on all Antarctic seal species. They infest mostly young seals; particularly those hauled out for long periods. The lice are confined to the tail and flipper areas and are adapted to reproduce at low temperatures (Murray *et al.* 1965; Murray 1967; Harder *et al.* 1991).

Emperor penguins are the least susceptible of penguin species to ectoparasite infestation since they breed in winter in cold conditions and do not have nests to provide a suitable habitat for ticks or fleas. Ticks are most commonly found on Royal penguins at Macquarie Island that breed densely on tussock grass and are ashore for a large proportion of the year. Heavy infestations have also been reported affecting gentoo penguins on the Antarctic Peninsula on rare occasions. Fleas are most common on rockhopper penguins that tend to nest in caves where the fleas are sheltered from wet weather. In general, ectoparasites do not cause severe disease in penguins or seals. However, environmental conditions may sometimes be suitable for heavy infestations to result in debilitation and death of affected animals.

## ENDOPARASITES

A number of endoparasites have been described in penguins and seals (Mawson 1953; Schmidt 1965; Prudhoe 1969; Beverley-Burton 1971; Dailey 1972; Arundel 1978; Obendorf and McColl 1980; Mawson *et al.* 1986; Azuma *et al.* 1988; Harringan 1992; Norman *et al.* 1992). Only nematodes and cestodes have been documented in Antarctic penguin species while nematodes, cestodes and trematodes have been found in Antarctic seals.

- Nematodes are roundworms that live in the gastro-intestinal tracts of many seal and penguin species. Gastric nematodes are the most commonly observed parasites in seals (Baker 1987; Baker 1989). Hookworms are common in young seals and can be major cause of pup mortality when animal densities are high, especially in combination with environmental or nutritional stress (Abegglen *et al.* 1958; George-Nascimento *et al.* 1992; Lyons *et al.* 1997). Lungworms have been reported as capable of killing young or starving seals (Ridgway *et al.* 1972).

- Cestodes are tapeworms that live in the intestines, and require intermediate hosts to complete their life cycles. Fish and crustaceans are common intermediate hosts. Tapeworms have been observed in Antarctic penguins and seals with no evidence of associated disease (Morgan 1978).

- Trematodes are flukes and have been found in Antarctic seals but have not been observed in penguins, nor associated with disease.

Endoparasite burdens tend to be heaviest in juvenile birds and animals and generally only contribute to mortality when combined with starvation and other forms of stress.

**Protozoa**

Protozoa are capable of invading various parts of the body including the gastro-intestinal tract, muscles and organs, and the bloodstream. A number of protozoan species have been recorded infecting penguins and seals, but the only reported isolations from Antarctic species have been from captive penguins.

- Malaria, caused by Plasmodium species is the most commonly reported protozoal disease in captive penguins where it is often fatal (Stoskopf and Beier 1979), but it is not found in the wild in Antarctica because mosquitoes are required for transmission.

- Coccidiosis is known to affect seal pups in captivity, but has not been found in wild seals (Munro and Synge 1991). Stress and poor hygiene predispose to disease.

- Giardia has been isolated from seals in Arctic regions (Olson 1997). Seals are thus a potential reservoir for this zoonosis.

- Microfilaria similar to those causing heartworm in dogs are able to cause gastro-enteritis in seals (Ridgway *et al.* 1972).

- Antibodies to the tick-transmitted spirochete that causes the zoonosis Lyme disease have been detected in king penguins (Gauthier-Clerc *et al.* 1999). The effect of this agent on the penguins is unknown, but the organism is likely to be widespread among tick-infested birds.

Blood smears from a number of wild Antarctic and sub-Antarctic penguin species have been examined for evidence of parasites. So far all findings, except the one above, have been negative. The reason for this is assumed to be a lack of suitable vectors, since blood parasites are found in wild penguins from temperate regions and can infect Antarctic species in captivity.

## VIRAL DISEASES: PENGUINS

There are three main groups of viruses that have been found in Antarctic and sub-Antarctic penguins.

- Avian paramyxoviruses are widespread among wild birds in general, and have been isolated from a number of penguin species (Morgan and Westbury 1981; Morgan *et al.* 1985). Strains vary in virulence, i.e.: in their capacity to cause clinical disease in infected birds. This group of viruses includes the potentially devastating Newcastle Disease virus which has been known to cause disease in captive penguins (Pierson and Pfow 1975), although it has not yet been observed in virulent form in the wild.

- Avian influenza viruses also come in a variety of strains that vary in virulence. These viruses are known to infect a wide range of bird species but do not generally cause severe disease in wild birds.

- Flaviviruses are carried by ticks and can infect the birds on which ticks feed. Antibodies to flaviviruses have been found in several sub-Antarctic penguin species, although associated disease has not been observed (Morgan *et al.* 1985).

There are two other viruses that have been associated with Antarctic penguins. A disease resembling puffinosis was reported affecting gentoo penguins on Signy Island in the early 1970s (MacDonald and Conroy 1971). Puffinosis is caused by an unclassified virus, and has previously been described affecting shearwaters. Antibodies to infectious bursal disease virus have recently been demonstrated in Emperor and Adélie penguins from the Mawson region (Gardner *et al.* 1997).

A recent survey of sub-Antarctic rockhopper penguins has provided the first evidence of exposure to other avian viruses in penguins (Karesh *et al.* 1999). Antibodies to avian adenovirus, avian encephalomyelitis virus, infectious bronchitis virus (a coronavirus) and avian reovirus were found. No symptoms of disease were apparent, although the blood biochemistry of birds with antibodies to infectious bronchitis virus indicated that some degree of physiologic response had occurred.

### Newcastle disease

Newcastle Disease virus is an avian paramyxovirus. Strains vary in virulence, with virulent strains occurring mainly in domestic poultry. Waterbirds are commonly associated with avirulent strains, and appear resistant to clinical signs following infection.

The disease is highly contagious, causing respiratory and nervous signs. A carrier status is likely to exist in waterbirds. The virus is easily inactivated by chemicals and sunlight, but difficult to vaccinate against effectively.

Antibodies to Newcastle Disease have been found in Adélie and royal penguins, and non-pathogenic paramyxovirus strains have been isolated from royal and king penguins (Morgan and Westbury 1981; Morgan *et al.* 1981). Clinical Newcastle disease has been reported in a captive king penguin, and has also occurred in Adélie penguins following capture from the wild (Pierson and Pfow 1975). Outbreaks of clinical disease in wild birds are rare.

**Avian influenza (Influenza A)**

Avian influenza is an orthomyxovirus related to the influenza viruses found in other species. The various subtypes of influenza A viruses vary in pathogenicity. Non-pathogenic strains are ubiquitous in waterbirds and studies have revealed that aquatic birds are the original hosts of influenza outbreaks in mammalian species. Virulent strains affecting both birds and mammals can emerge following mutation or rearrangement of existing strains. Highly pathogenic strains cause oedema, haemorrhage and sudden death in domestic fowl. Avian influenza is highly contagious and difficult to vaccinate against.

Antibodies to avian influenza have been found in Adélie penguins at Casey and in penguins and skuas in the Ross Sea, but no actual virus particles have yet been isolated from penguins (Morgan and Westbury 1981). Many Adélie penguin chicks died from an unknown cause at Casey in the same year that antibodies were detected in adults, but no clinical signs were observed and post-mortems were not carried out. The widespread presence in Adélie penguins and skuas of antibodies to both avian influenza virus and paramyxoviruses are evidence that the myxoviruses are widespread and persistent in the Antarctic (Austin and Webster 1993).

There has been one report of mass death due to avian influenza in wild birds -- this occurred in terns in South Africa in 1961 (Webster *et al.* 1992). It is worth noting that strains of influenza closely related to avian influenza have caused at least three mass mortality events in seals in the Northern Hemisphere over the past two decades (Geraci *et al.* 1982; Geraci *et al.* 1984; Callan *et al.* 1995).

**Infectious Bursal Disease Virus (IBDV)**

Infectious bursal disease virus is a birnavirus. The disease, also known as Gumboro disease, occurs in two serotypes, one of which is highly pathogenic. Low pathogenicity strains are widespread among birds. The disease is highly contagious and the virus difficult to chemically inactivate, therefore allowing it to be easily transmitted via contaminated clothing and equipment. IBDV causes haemorrhages, diarrhoea and

ataxia in its virulent form, and immunosuppression in the sub-clinical form. Thus effects of infection may be subtle.

Antibodies to IBDV have been detected in shearwaters and penguins, although clinical disease has not so far been documented in these species. Recent studies have demonstrated the presence of antibodies to IBDV in emperor penguin chicks at Auster rookery near Mawson (Gardner *et al.* 1997). It is not known how these birds became exposed to the virus, or whether any degree of disease or immunosuppression has occurred as a result. There were certainly no signs of ill health in the colony.

IBDV affects only birds with functional bursae (i.e. chicks). The bursa is the organ responsible for the production of B-lymphocytes in birds, and regresses by adulthood. The epidemiology of the disease is poorly understood, as theoretically only chicks are affected and adults are presumed not to transmit the virus. However, in the laboratory situation virus replication is able to take place in bursectomised birds, and it is therefore possible that a carrier status could exist.

## VIRAL DISEASES: SEALS

Seals are known to be susceptible to a number of viral diseases in their natural environment and outbreaks of three of these have been observed over the past two decades in various species of seals. The viruses that have been isolated from seals to date include:

- Morbilliviruses, which result in distemper, and have been the cause of a number of outbreaks of disease in pinnipeds and cetaceans over the past two decades (see below).

- Influenza A viruses, which cause outbreaks of pneumonia under suitable environmental conditions and appear to derive from avian strains (see below).

- Herpesviruses, which can cause respiratory disease and hepatitis and affect young animals with greatest severity (see below).

- A calicivirus (San Miguel sea lion virus) appears to be enzootic in some Northern Hemisphere pinniped species. The pathogenic role of this virus in seals is unclear, but the same virus is known to cause Vesicular Exanthema in pigs. Seals (and fish) have been implicated as carriers (Stack *et al.* 1993).

- Parapox virus has been isolated from seals with seal pox in the Northern Hemisphere (Stack *et al.* 1993; Simpson *et al.* 1994; Nettleton *et al.* 1995). The infection, which affects mainly young seals and is rarely fatal, is capable of being transmitted to man.

- Rabies virus has been diagnosed in ringed seals in Arctic regions (Odegaard 1981). This virus is a zoonosis.

- Adenovirus infection causing hepatitis has been reported in California sea lions (Brit 1979; Dierauf 1981). Symptoms are similar to those of canine infectious hepatitis in dogs. The origin and distribution of the virus in seals in unknown.

**Phocine Distemper**

Distemper in seals is caused by a morbillivirus, phocine distemper virus (PDV) which is closely related to that which causes canine distemper (CDV). Several outbreaks of phocine distemper have occurred in recent decades in the Northern Hemisphere after the virus was first introduced into susceptible populations. The origin of these outbreaks is not certain, but it appears likely that the disease was enzootic in harp seals in Arctic regions prior to its appearance in seals further south. Mass deaths were first observed in harbour and grey seals in the North and Baltic Seas in 1988 (Osterhaus *et al.* 1988). Serology showed that prior to the outbreak seals in these regions did not have antibodies to morbillivirus, although seals from more northern regions did. It is likely that the disease was transmitted from harp to harbour seals in 1987 when large numbers of harp seals migrated further south than usual (Goodhart 1988; Dietz *et al.* 1989; Markussen and Have 1992; Barrett *et al.* 1995). A mild winter and an algal bloom preceded the 1988 epizootic. Seals hauled out in large numbers that year allowing rapid transmission of the virus. High organochlorine burdens in the animals may have contributed to immunosuppression also.

Antibodies to morbillivirus have persisted at low levels in Northern Hemisphere seal populations since the initial outbreak (Barrett *et al.* 1995). A less severe outbreak occurred along the north-east coast of the US in 1991-92 (Duignan *et al.* 1995). Morbilliviruses have also been identified in dolphins and porpoises (DMV and PMV respectively) (Duignan *et al.* 1996). A virus related to DMV caused an outbreak of distemper in the endangered monk seal population off West Africa in 1996 (Osterhaus *et al.* 1997).

Seals are susceptible to CDV as well as PDV. This first became apparent following an outbreak of distemper in Lake Baikal seals in Siberia in 1987 (Grachev *et al.* 1989). It was presumed that the seals became infected from diseased dogs (Barrett *et al.* 1995). Antibodies to PDV have not been detected in Antarctic seals. However, antibodies to CDV have been found in leopard and crabeater seals (but not in Weddell seals) at the Antarctic Peninsula (Bengtson and Boveng 1991). The significance of this finding is unknown; however, it is possible that a mass death of crabeater seals observed in 1955 at the Antarctic Peninsula (Laws and Taylor 1957) could have been due to morbillivirus infection.

It is probable that PDV, DMV and PMV infections of seals and cetaceans have been enzootic in these animals for many years. When these viruses have not been present in a particular population for some time levels of immunity become low and introduction by, for example, interspecies transmission may lead to severe outbreaks. Stressful environmental conditions, high population densities and impairment of immune functions due to pollution may contribute to severity and extent of disease (Osterhaus 1995).

**Influenza A**

There have been three recorded outbreaks of pneumonia due to influenza A in harbour seals along the New England coast between 1979 and 1992 (Geraci *et al.* 1982; Geraci *et al.* 1984; Callan *et al.* 1995). Young seals were mostly affected, and abortions among pregnant females were also observed. All events were transient. The viral strains isolated from each outbreak were of different subtypes, but all were closely related to avian variants. Birds represent a major reservoir of influenza A viruses in nature, and it is likely that avian viruses are frequently transmitted to marine mammals (Hinshaw *et al.* 1984).

Experimental infection of susceptible seals with influenza A results only in mild disease. For an epizootic to occur there seems to be the need for concurrent biological and/or environmental factors to be synchronised with the virus to produce disease (Geraci *et al.* 1984). The first recorded outbreak involved concurrent mycoplasma infection, high population density and potentially adverse environmental conditions, i.e. unseasonably warm temperatures (Geraci *et al.* 1982). Conditions were similar to those coinciding with the mass mortality of crabeater seals in 1955 (Laws and Taylor 1957). It has been suggested that the crabeater deaths could have been due to an outbreak of influenza A (as opposed to distemper, which has also been postulated as a cause).

No antibodies to influenza A have yet been detected in Antarctic seals, despite their presence in Antarctic birds. The spread of these viruses may be facilitated by skua migratory and feeding habits. Actual virus particles have not yet been isolated in any Antarctic vertebrate species, but this failure may be due to timing of sample collection since the distribution of influenza viruses in birds is generally seasonal (Austin and Webster 1993).

**Phocine Herpesvirus**

Phocine herpesvirus (PHV) has been isolated from harbour seals showing respiratory disease, hepatitis and/or encephalitis. Herpesviruses in seals fall into 2 types: PHV-1 and PHV-2. The former is most commonly isolated, being an alphaherpesvirus related to feline herpesvirus-1. The host restriction of alpha herpesviruses is variable. Some

can cross species barriers but most are fairly host specific. Disease in the natural host is commonly self-limiting. In aberrant hosts symptoms may be more severe.

The first isolation of PHV-1 occurred following an outbreak of disease characterized by liver necrosis and interstitial pneumonia in juvenile harbour seals in a Dutch seal orphanage in 1985 (Borst *et al.* 1986). Neonate seals are most susceptible to PHV-1. Older seals will generally develop only mild respiratory signs unless other stresses are involved (Harder *et al.* 1997). Antibodies against European phocine herpesvirus were detected in Weddell and crabeater seals in the Weddell Sea in 1990 (Harder *et al.* 1991). The seals were suffering from a respiratory disease at the time, and titers in some animals were high suggesting that the symptoms may have been due to the virus. The herpesvirus was considered likely to be endemic, and may have originated from temperate zones via individuals that traveled further north than most.

Herpesviruses should be considered as important widespread pathogens in seals, which may cause severe illness, especially in stressed or immuno-suppressed hosts (Stenvers *et al.* 1992).

**Bacterial Infections**

A number of bacterial species have been isolated from wild penguins and seals, and some of these are known to have caused disease.

- A number of Salmonella species were isolated from Adélie penguins at Ross Island in the '70s, but were not associated with disease (Oelke and Steiniger 1973). The origin of these bacteria is unknown; it is possible that they were brought into Antarctica by man. Salmonella species are known to cause disease in captive penguins (Cockburn 1947). Salmonella species are commonly isolated from seals, both diseased and healthy (Gilmartin 1979; Baker *et al.* 1995). Clinical disease is most likely to occur in young animals under conditions of stress, crowding or poor hygiene (Stroud and Roelke 1980).

- Antibodies to the Chlamydia group of bacteria have been isolated from a number of Antarctic and sub-Antarctic penguin species, including a dead emperor chick at Auster rookery (Moore and Cameron 1969). The significance of these findings is unknown, but it is possible that psittacosis (the disease caused by *Chlamydia psittaci*) may contribute to chick mortality in penguin colonies. Chlamydia are most likely to have been introduced to Antarctica by migratory birds.

- *Pasteurella multocida*, the causal agent of **avian cholera**, has been isolated from rockhopper penguins on Campbell Island where mortalities have been observed on more than one occasion (Lisle *et al.* 1990). *Pasteurella multocida* has also been isolated from brown skuas at Palmer where it caused deaths among the brown skua population, but did not affect penguins nor Antarctic skuas (Parmelee *et al.* 1978).

Avian cholera is known to cause mass mortalities in wild waterfowl as well as sub-acute and chronic infections, and can also be carried by rats and cats. It could therefore pose a threat to sub-Antarctic penguin species on islands with feral cat populations, and may possibly cause outbreaks of disease in Antarctica following introduction via migratory birds or contaminated poultry products. Antarctic skuas breed in Antarctica in the summer and migrate to the Northern Hemisphere in the austral winter. Due to their migratory behaviour they have the potential to carry disease agents such as pasteurella, avian influenza virus and avian paramyxoviruses into Antarctica. *Pasteurella multocida* has also been known to cause septicaemia and mortality in captive seals (Lynch 1999).

- Tuberculosis has been diagnosed in fur seals in the Southern Hemisphere. The causal agents have been shown to belong to the Mycobacterium tuberculosis complex, and are more similar to human than other animal strains (Forshaw 1991; Cousins *et al.* 1993; Romano *et al.* 1995).

- Antibodies to Brucella species have been found in a number of seal species in the Northern Hemisphere (Nielsen *et al.* 1996). Brucella organisms differing from recognised species have been isolated from seals along the Scottish coast (Foster *et al.* 1996).

- Leptospirosis is endemic in California sea lions among which it has caused at least one outbreak of disease (Dierauf *et al.* 1985). The organism has not been reported in other seal species, or in the Southern Hemisphere (Lynch 1999). Disease is mnifest as abortion in adults and multiple haemorrhagic syndrome in neonates (Smith 1977).

- Mycoplasma organisms have been isolated from Northern Hemisphere seals suffering from pneumonia, but have not been shown to cause disease unless combined with other pathogens such as influenza virus (Geraci *et al.* 1984).

- Campylobacter species are present in birds and seals at South Georgia. Origin and pathogenicity are unknown, but a human source is possible and the organism may be implicated in some seal mortalities (A. Broma, pers comm.).

Other bacterial and fungal diseases to which penguins and seals are susceptible but which have only been observed in captive animals include bumblefoot and aspergillosis. Bumblefoot is a bacterial disease of the feet of captive penguins related to the housing of birds on hard wet surfaces. It does not occur in seals or wild penguins (Gailey-Phipps 1978; Stoskopf and Beall 1980). Aspergillosis is a fungal infection that usually affects the respiratory system and can be fatal. It has only been observed in captive penguins and seals in association with stress or other diseases (Obendorf and McColl 1980; Stoskopf and Beall 1980). Aspergillus species are widespread in the environment, and have been detected in Antarctic soils. Healthy animals can resist the disease, but aspergillosis could potentially present problems in association with stress or starvation.

## MASS MORTALITIES: PENGUINS

There have been a few documented reports of mass mortalities, presumed due to disease, in wild penguin populations over the years. All have been localised to single colonies and single species, and appear to have been self-limiting. Causal agents were not identified in most cases.

- Many 4-5 week old Adélie chicks died at Petersen Is near Casey in the late 1970s (Morgan and Westbury 1981). No clinical signs were observed and no post mortems carried out. Antibodies to avian influenza were detected in adults the same season, but the significance of this finding is unknown.

- Several hundred gentoo chicks died at Signy Island in the late 1960s with symptoms resembling puffinosis (MacDonald and Conroy 1971). However, no infectious agent was isolated.

- Many crèche-age Adélie chicks were found dead and dying at Low Tongue near Mawson in the early 1970s (Kerry *et al.* 1996). All were in good body condition, and ataxia was observed prior to death. No samples were taken, and the cause remains unknown.

- Mass mortality of rockhopper penguin chicks has been observed on Campbell Island on more than one occasion (Lisle *et al.* 1990). *Pasteurella multocida* (avian cholera) was shown to be the cause of these outbreaks.

## MASS MORTALITIES: SEALS

Viral diseases are most likely to spread rapidly when large numbers of susceptible animals are congregated together over a period of time. The majority of documented mass deaths of seals have had a viral or suspected viral aetiology. Reported mortality events include:

- Mass mortality of crabeater seals on the north-eastern tip of the Antarctic Peninsula in the spring of 1955 (Laws and Taylor 1957). Most seals affected were young. Crowding and starvation probably increased the effects of disease, as seals were congregated in larger numbers than normal that winter. The winter was warmer than average also. Disease was presumed contagious and likely to be viral. Pathology included congestion of the lungs with leukocyte infiltration, enlarged spleens with evidence of haemolysis, evidence of acute nephritis, and numerous abortions. No other seal species were affected although Weddell and leopard seals were both hauled out nearby. Meat from live seals was eaten by dogs and men with no ill effects. The symptoms and environmental conditions were similar to those affecting harbour seals that died along the New England coast in 1979-80 from

pneumonia associated with influenza A and mycoplasma (Geraci *et al.* 1982). However, symptoms of disease were also consistent with an outbreak of phocine distemper (Bengtson and Boveng 1991).

- Mass mortality of harbour seals due to pneumonia associated with influenza A virus H7N7 subtype in 1979-80 (Geraci *et al.* 1982). The isolated virus had avian characteristics, though it replicated best in mammals (Lang 1981). Isolates caused only mild respiratory disease in experimentally infected seals. The outbreak involved concurrent mycoplasma infection, high population density and unseasonably warm environmental temperatures. Environmental conditions and crowding were similar to the circumstances surrounding the crabeater seal deaths in 1955 (Geraci *et al.* 1982).

- An outbreak of distemper occurred in Lake Baikal seals in Siberia in 1987-88 killing thousands of seals (Grachev *et al.* 1989). Dogs were also affected after contact with seals. Serology showed CDV-neutralizing antibodies in most animals, and PCR showed that the virus was indistinguishable from CDV (Mamaev 1995). Within a year all the seals in the population had developed antibodies, suggesting that they should be immune to further infection. No further outbreaks occurred.

- Mass abortions and high mortality occurred following an outbreak of a new morbillivirus (phocine distemper virus) in European harbour seals in 1988 (Osterhaus *et al.* 1988). The epizootic was due to introduction of PDV into a highly susceptible population. Its origin was unknown, but serological evidence of prior PDV infection was found in arctic seal populations (Markussen and Have 1992).

- An outbreak of morbillivirus occurred in endangered monk seals off West Africa in 1996 killing half the population (Osterhaus *et al.* 1997). Symptoms were consistent with previous morbillivirus infections of seals. The morbillivirus isolated was shown to be different from both PDV and CDV, and more closely related to the dolphin morbillivirus. It was thus named monk seal morbillivirus (MSMV).

- High mortality occurred among monk seals in 1997. Pulmonary congestion was present, but no evidence of morbillivirus. The cause was eventually found to be saxitoxin intoxication due proliferation of toxic dinoflagellates in the waters.

- Mass mortality of fur seals occurred on Auckland Island in early 1998 (Gales pers. comm.). Deaths were consistent with septicemia; however, no definitive cause was determined despite extensive tests. Salmonella species were isolated in some samples. Tests for viruses were negative. Involvement of haemophilus species was suspected, but these bacteria are not easy to culture and were not isolated.

**SUMMARY**

In summary, our knowledge of the incidence and natural exposure to disease in Antarctic penguins and seals is still meagre and fragmentary. Studies of captive birds

show that penguins are susceptible to a wider variety of diseases than have been detected or reported in birds in the wild. Wild seal populations have been shown to be susceptible to outbreaks of viral diseases in the Northern Hemisphere at least. The presence of antibodies to various viral agents suggests that recent exposure to pathogenic viruses has occurred, or that serologically related non-pathogenic viruses are present. The presence of non-pathogenic viruses may provide cross immunity to pathogenic strains.

Animals and birds breeding in the Antarctic and on sub-Antarctic islands have evolved in relative isolation. Distance reduces contact with disease and inhibits the introduction of vectors or intermediate hosts. The speed of modern transport to Antarctica and the increase in the numbers of people visiting south polar regions may increase the possibility of unwitting introduction of diseases. Changes in global climate may also facilitate the spread of disease through altered migratory habits of animals and birds and the introduction of vectors such as ticks.

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