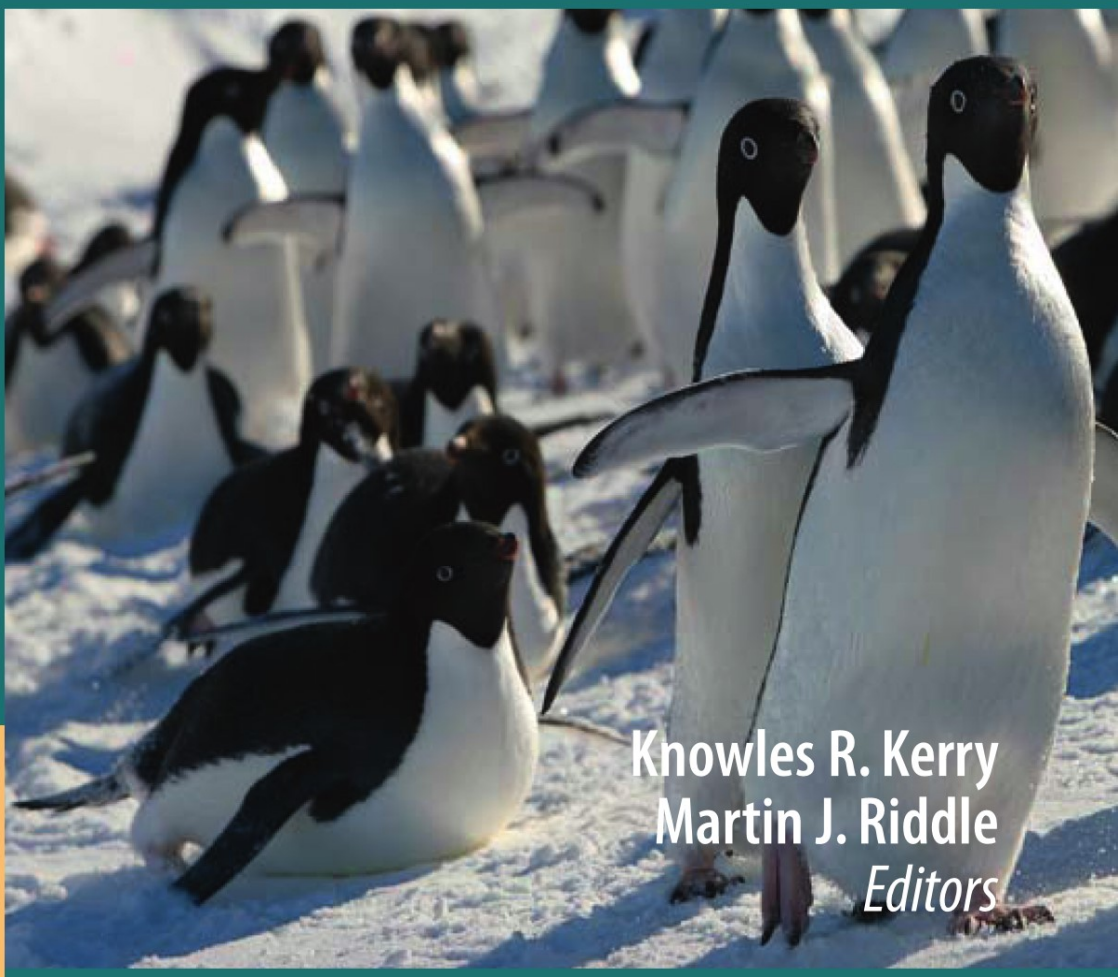

Knowles R. Kerry
Martin J. Riddle
Editors

Health of Antarctic Wildlife

A Challenge for Science and Policy

Springer

Health of Antarctic Wildlife

Moribund and dead Adelie penguin chicks, Low Tongue near Mawson, 10 Febraury 1972. “The chicks were almost fully grown but had not started to moult their down. Both those alive and recently dead were plump and appeared well fed. Field notes and photo K.R. Kerry

Dead crabeater seals Prince Gustav Channel, Antarctic Peninsula, October 1955. “All affected seals, whether dead or comatose, had oddly swollen necks and a trickle of blood running from their mouths. On dissection their guts were empty, their livers palid in colour, and pus oozed from the neck glands when an incision was made.” Dr. P.M. Massey as quoted by Sir Vivian Fuchs, Of Mice and Men. Photo A.F. Lewis

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Editors
Dr. Knowles R. Kerry
Australian Antarctic Division
Channel Highway
Kingston 7050
Tasmania, Australia
e-mail: knowles.kerry@keypoint.com.au

Dr. Martin J. Riddle
Australian Antarctic Division
Channel Highway
Kingston 7050
Tasmania, Australia
e-mail: martin.riddle@aad.gov.au

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Cover illustration: Adélie penguins *Pygoscelis adeliae* returning across the sea ice to their breeding colony near Casey Station, Antarctica. Photo © Chris Wilson 2007

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Dedicated to the memory of Durno Murray who, in the early 1960s, was among the first scientists to draw attention to the possible impact of human activity on the health of Antarctic wildlife. We acknowledge his continued efforts to undertake, promote and encourage research in this field.

Preface

This book provides a broad assessment of the health of Antarctica's birds and seals. It is set against the background of available scientific and environmental information and the political and administrative processes in place. It is intended for use by veterinary and biological scientists, policy makers and administrators whose job it is to protect the health of Antarctica's wildlife. It also provides readily accessible information through reviews and case studies for research into related health and disease issues. The term health is used in its widest sense to encompass the normal state and those factors which detract from it including both infectious and non-infectious causes.

Health is a condition to which we aspire for ourselves and for the wildlife with which we come in contact. Ill health through disease however is an integral part of life and diseases caused by macro parasites (nematodes, cestodes and arthropods) and microorganisms (bacteria, viruses, fungi, protozoans) are as much a part of ecology and the ecosystem as are the hosts themselves. There are many causes of ill health which include poisoning by chemical or biological toxins, trauma, starvation, behaviour modification, genetic predisposition and interaction with the physical environment. These may be natural or caused or exacerbated by human activity.

We deal with health in this book in the sense of its maintenance but also through an understanding of the state of ill health and those factors which cause it. We give little consideration to what constitutes good health in individuals and populations of wildlife as this is the purview of biologists who investigate "normal" populations.

This book has its origins in a Workshop on Diseases of Antarctic Wildlife held in Hobart, Australia in August 1998. This workshop which attracted 52 participants from eight countries was convened in response to concerns raised at the Antarctic Treaty Consultative Meeting (ACTM) XXI. The Workshop recognised "there was a significant risk of the introduction of (exotic) diseases into Antarctic wildlife species and should it occur the consequences are likely to be serious and a response will be required". The workshop at its conclusion requested that the conveners prepare and edit a book based on the papers presented and the outcomes of discussions held there. A task easier set than done!

Ten years have elapsed since this book was first conceived. A long gestation indeed. We are fortunate that all authors have assisted us in keeping the manuscripts

comprehensively up to date. We are most grateful to these authors and to those we recruited to write the additional chapters necessary to fill the significant gaps. We acknowledge also the sacrifice of some who presented results of original research and have thus suffered a longer time than usual to publication. The timing of publication however has provided the opportunity to highlight recent discussions and resolutions made within the Antarctic Treaty forum to protect wildlife against disease and to include responses by Government and non-Government operators in Antarctica. These developments mostly followed from the Workshop on Diseases of Antarctic Wildlife.

The book comprises 17 chapters presented in two parts. *Wildlife disease* consists of reviews, case studies and health assessments, and *External factors* covers the environmental, administrative and legal aspects. Each chapter is complete and contains all references. Six important documents are provided as Appendices. These present methods, reviews and other documents which are referred to in one or more chapters but are not readily available.

There are many related topics we have been unable to cover that would enhance the understanding of health and disease processes in Antarctica. While we acknowledge their importance they are outside the scope of the present volume. Such topics include epidemiology, new and emerging infectious diseases and the effects of climate change. These topics are referred to in the various chapters where references to source material are given.

We have attempted to provide a coherence among chapters that differs from the presentation of papers in scientific journals. We were assisted initially by comments from reviewers and then in conjunction with the authors sought assistance, often from the reviewers, to prepare the manuscript in a more suitable form. Manuscripts in some cases were amalgamated and in others additional material added and co-authorship given. Where additional chapters were requested editorial assistance was given where needed to these authors.

Several conventions were adopted while editing. We have accepted papers with both English and American forms of spelling as long as they are consistent within chapters. Species names have been hard to standardise. There have been some recent taxonomic revisions, many of which are controversial. Where taxonomy is important to the substance of the chapter, we have accepted the authors' decision but required that the taxa in question be cross referenced to previous names. The scientific and common names of birds in general use in the Antarctic literature have been accepted. Aggregations of seals and birds are recorded as being in colonies not rookeries and we have assumed that the readership of a book on Antarctic wildlife will understand the terms *summer* and *winter* refer to southern hemisphere seasons, without the necessity to specify that they are austral summer or winter.

We believe we have covered all the essential issues necessary for the understanding of health and disease relating to Antarctic wildlife to provide for wise council in the management of human activities in Antarctica. The challenge is now for scientists in concert with policy makers to ensure the executive wings of their Governments are fully briefed and are able to act proactively to protect against the introduction or spread of disease by human activities.

Acknowledgements We sincerely thank all who contributed to this book and in particular the authors and those who assisted in the review process. The editorial process has taken a long time and we hope this publication is sufficient reward for their work and their patience. We give special thanks to Genevieve Tanner who undertook the technical editing and Clodagh Jones for providing the index. We thank Steve Candy for advice on statistical analyses, Judy Clarke for scientific and veterinary advice, Angela Bender, David Smith and Henk Brolsma for providing maps and Jessica Fitzpatrick and Pauline de Vos for illustrations. The Australian Antarctic Division underwrote the cost of production and provided additional assistance to the editors. We acknowledge the contributions of the participants of the Workshop on Diseases of Antarctic Wildlife who paved the way leading to this volume and Durno Murray for providing an historical perspective to the considerations of wildlife disease by SCAR and the Antarctic Treaty System and particularly for his encouragement to pursue the subject matter leading to this book. Finally we wish to thank Dr Andrea Schlitzberger of Springer for her unfailing help over the long period of time needed to bring this book into print.

Australia
January 2009

Knowles Kerry
Martin Riddle

Contents

Contributors

Arthur Beggs
Australian Antarctic Division, Channel Highway, Kingston, TAS 7050, Australia

Paul J. Canfield
Faculty of Veterinary Science, The University of Sydney, NSW 2006, Australia
and
Australian Marine Mammal Research Centre, Zoological Parks Board of NSW, Mosman, NSW 2088, Australia

Thomas S. Gelatt
Department of Fisheries and Wildlife, University of Minnesota, St. Paul, MN, USA

Joseph R. Geraci
National Aquarium in Baltimore, Baltimore, MD 21202-3194, USA
and
University of Maryland School of Medicine, Comparative Medicine Program, Baltimore, MD 21201-1192, USA
jrgeraci@sbcglobal.net

Rachael B. Gray
Faculty of Veterinary Science, The University of Sydney, NSW 2006, Australia
gray@vetsci.usyd.edu.au
and
Australian Marine Mammal Research Centre, Zoological Parks Board of NSW, Mosman, NSW 2088, Australia

Carolyn J. Hogg
Evolution and Ecology Research Centre, School of Biological, Earth and Environmental Sciences, University of New South Wales, Sydney, NSW 2052, Australia
c.hogg@unsw.edu.au
and
Australian Marine Mammal Research Centre, Zoological Parks Board of NSW, Mosman, NSW 2088, Australia

Lyn Irvine
Australian Antarctic Division, Channel Highway, Kingston, TAS 7050, Australia
Lyn.Irvine@cwr.org.au

Julia Jabour
Institute of Antarctic and Southern Ocean Studies, University of Tasmania, Private Bag 77, Hobart, TAS 7005, Australia
julia.jabour@utas.edu.au

Hugh I. Jones
Microbiology and Immunology, School of Biomedical, Biomolecular and Chemical Sciences, University of Western Australia, WA 6009, Australia
hjones@cyllene.uwa.edu.au

Knowles R. Kerry
Australian Antarctic Division, Channel Highway, Kingston, TAS 7050, Australia
knowles.kerry@keypoint.com.au

Valerie J. Lounsbury
National Aquarium in Baltimore, Baltimore, MD 21202-3194, USA
vlounsbury@aqua.org

Ro. A. McFarlane
National Centre for Epidemiology and Population Health, Australian National University, ACT, Australia
romcfarlane@bushlink.net.au

Gary D. Miller
Biology Department, University of New Mexico, Albuquerque, NM 87131, USA

Greg Mortimer
Aurora Expeditions, 182 Cumberland Street, The Rocks, Sydney, NSW 2000, Australia
greg@auroraexpeditions.com.au

Richard J. de B. Norman
Ministry for Agriculture and Fisheries, 4J/51 Webb Street, Wellington 6011, New Zealand
richard.norman@maf.govt.nz

Michael Pook
CSIRO Marine and Atmospheric Research, Castray Esplanade, Hobart, TAS 7000, Australia
Mike.Pook@csiro.au

Elaine Prior
Aurora Expeditions, 182 Cumberland Street, The Rocks, Sydney, NSW 2000, Australia

Martin J. Riddle
Australian Antarctic Division, Channel Highway, Kingston, TAS 7050, Australia
martin.riddle@aad.gov.au

Wendi Roe
New Zealand Wildlife Health Centre, Institute of Veterinary Animal and Biomedical Sciences, Massey University, Palmerston North, New Zealand
W.D.Roe@massey.ac.nz

Tracey L. Rogers
Evolution and Ecology Research Centre, School of Biological, Earth and Environmental Sciences, University of New South Wales, Sydney, NSW 2052, Australia
tracey.rogers@unsw.edu.au
and
Australian Marine Mammal Research Centre, Zoological Parks Board of NSW, Mosman, NSW 2088, Australia

Donald R. Rothwell
Sydney Centre for International and Global Law, Faculty of Law, University of Sydney, NSW 2006, Australia
RothwellD@law.anu.edu.au

Geoff R. Shellam
Microbiology and Immunology, School of Biomedical, Biomolecular and Chemical Sciences, University of Western Australia, WA 6009, Australia
gshellam@cyllene.uwa.edu.au

Donald B. Siniff
Department of Ecology, Evolution and Behavior, University of Minnesota, St. Paul, MN, USA

James J. Smith
International Laboratory for Air Quality and Health, Institute for Sustainable Resources, Queensland University of Technology, Brisbane, QLD 4001 Australia
jj.smith@qut.edu.au

Brent S. Stewart
Hubbs-Sea World Research Institute, San Diego, CA, USA
BStewart@hswri.org

Joanne Watts
Microbiology and Immunology, School of Biomedical, Biomolecular and Chemical Sciences, University of Western Australia, WA 6009, Australia
djawatts@bigpond.net.au

Rupert Woods
Australian Wildlife Health Network, Mosman, NSW 2088, Australia
RWoods@zoo.nsw.gov.au

Pamela K. Yochem
Hubbs-Sea World Research Institute, San Diego, CA, USA
PYochem@hswri.org

Health of Antarctic Wildlife: An Introduction

K.R. Kerry and M.J. Riddle

1 Background to the Book

Human occupation of the Antarctic continent commenced in February 1899 with the arrival of the Southern Cross Expedition to establish the first winter camp at Cape Adare. The wintering party comprised 10 men and 75 sledge dogs. Until that time, Antarctica had been isolated by the vast encircling Southern Ocean. Following the Southern Cross expedition, there was a steady stream of expeditions to locations around the Antarctic Continent during what was known as the 'heroic era' of Antarctic exploration. Each expedition took with them animals in the service of man, many brought dogs and Scott brought ponies. Cats and caged birds were brought as pets, and on one Antarctic station pigs were maintained for food. The introduction of alien species of mammals and birds continued with little thought given to micro-organisms, including agents of disease, they might carry and introduce to the native wildlife. Geographic exploration was a main driving force for expeditions, but each was supported by a base located on or close to the coast. These for the most part were located close to large concentrations of seals and sea birds. Little care was taken in the disposal of food waste and so discarded chicken carcases, eggs and meat and other items were added to the diet of scavenging skuas.

The success of the International Geophysical Year (1957–1958) brought a change in Antarctic Activities and led to the establishment of the Antarctic Treaty, which entered into force in 1961. Scientific research in the physical sciences was accorded highest priority. Biological research, initially neglected, progressively increased in importance. The Scientific Committee on Antarctic Research (SCAR) was established in 1958 by the International Council of Scientific Unions and was charged with initiating, developing and coordinating high quality international scientific research in the Antarctic region.

K.R. Kerry and M.J. Riddle
Australian Antarctic Division, Channel Highway, Kingston, TAS 7050, Australia
e-mails: knowles.kerry@keypoint.com.au; martin.riddle@aad.gov.au

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Animal health was on the agenda for Antarctic scientific research almost from the beginning of SCAR. In a list of scientific priorities recommended by SCAR at its third meeting in Canberra in 1959, it was noted that 'unique opportunities exist in Antarctica for studying man's impact upon a relatively uncontaminated environment…..A survey is required of diseases already present in Antarctica's isolated or semi-isolated fauna, and their potential threat to man' (SCAR 1959). At the first SCAR Biology Symposium in 1962, Sladen (1964) noted that 'There is also an urgent need to find out what pathogenic organisms occur naturally in Antarctic populations before man introduces new ones.' At the same meeting several papers were presented on the micro-organisms and parasites of Antarctic seals and seabirds (Margni and Castrelos 1964; Dunnet 1964; Murray 1964).

The Consultative Parties to the new Antarctic Treaty were at the same time formulating their position on environmental protection in the region. The first Antarctic Treaty Consultative Meeting in Canberra in 1961 recommended interim measures for preservation and conservation of living resources in Antarctica pending broader consultation leading to the establishment of internationally agreed measures (ATCM 1961). These interim measures included the requirement that 'Alien forms of flora and fauna should not be deliberately introduced except when rigidly controlled….' In 1964, when the Agreed Measures for the Conservation of Antarctic Flora and Fauna (Agreed Measures) were established at the third Antarctic Treaty Consultative Meeting in Brussels (ATCM 1964), they were very much stronger on the topic of introduced disease. They recommended '….that all reasonable precautions shall be taken to prevent the accidental introduction of parasites and diseases into the Treaty Area' (Article IX, paragraph 4) and included an annex on precautions to prevent accidental introduction of parasites and diseases into the Antarctic. These precautions included the requirement that dogs taken to Antarctica should be inoculated against a variety of diseases and that living poultry should not be brought to Antarctica.

The Agreed Measures negotiated in 1964 finally come into force in 1982, but were soon superseded by the Protocol on Environmental Protection to the Antarctic Treaty, 1991. This protocol, known as the Madrid Protocol, was designed to provide comprehensive protection to the Antarctic environment and entered into force in 1998. Since then, the Committee for Environmental Protection (CEP), established by the Madrid protocol, has become the main advisory body to the Antarctic Treaty Consultative Meetings on all matters relating to environmental protection.

Despite the early recommendations of SCAR, no systematic and internationally coordinated studies into animal health and disease have yet been undertaken. However, since the 1970s, several independent investigations have looked for evidence that seals and birds may have been exposed to serious diseases. Antibodies to Newcastle disease and avian influenza virus in birds and to phocine distemper virus, canine distemper virus and herpes virus in seals have been found. Although antibodies have been identified, the actual virus in question has not been isolated. We are not sure if this means there are wild strains of these viruses endemic to the population or that they have been introduced. No outbreaks of these diseases have been reported. Despite these investigations, our knowledge base on health and disease among Antarctic species is slim. Further research is required.

The importance of protecting Antarctic species through limiting the introduction of alien organisms is central to both the Agreed Measures and the Madrid Protocol. The pool of scientific data available to support the obligations arising from these conservation measures is limited and growing only slowly. Until such data are obtained, management must be based primarily on information gained and practices applied on other continents.

This book brings together, through reviews and case studies, a compendium of information on health and disease and factors such as environmental, administrative and social, affecting the state of health of Antarctic birds and seals. We hope through these means to provide information in a consolidated form to the Antarctic Treaty Consultative Parties who, through domestic legislation and regulation, are responsible for implementing the environmental protection regime for Antarctica. Further, it is intended for use by veterinary and biological scientists, policy makers and administrators whose job it is to protect the health of Antarctica's wildlife.

2 Health and Disease

Health and disease in reality are part of a continuum. In its broadest sense, disease is any process, infectious or otherwise, which interferes with or modifies an animal's normal function (Environment Canada 2004). The term is often used in a more limited way to mean the deleterious effects on the host caused by parasites, viruses and bacteria. Healthy animals, nonetheless, are often able to carry low levels of potentially harmful organisms without succumbing to disease. In addition, subclinical disease may exist, which is not obvious and is thus difficult to detect, although it may have various effects, including generalised ill thrift, decreased breeding success and a reduced resistance to other diseases. As a consequence, subclinical disease may be confused with other conditions such as starvation caused from shortage of prey.

Diseases are normal ecological processes affecting all plants and animals and it must be expected that animals in the Antarctic would have experienced a wide variety of disease-causing agents, some of which may be restricted to Antarctic species, while others may be more widespread. However, outbreaks of infectious diseases have rarely been observed or suspected among seals and birds in Antarctica and in only a few of these instances have the infective agent been identified or isolated. The presence of disease agents in an animal population does not necessarily mean that clinical symptoms will develop. Similarly, the identification of a known pathogen in an animal showing clinical symptoms does not necessarily mean that the pathogen is the underlying cause of the disease. Several studies have used serology to show the presence of antibodies to a number of viruses and bacteria known to cause infectious disease. Again, the presence of antibodies does not mean that the animal has suffered from clinical disease, but it does indicate that it may have been exposed in the recent past to the specific disease agent or to an antigenically related agent.

3 Review of Contents

The first part of this book looks at the range of diseases reported in Antarctic animals, including reviews of disease in birds and seals, case studies of adverse health events and population health assessments. Together, these chapters form a reference that will provide essential background for investigating unusual wildlife mortality events in Antarctica. The second part looks at the external factors that may be imposed on the health of Antarctic animals, including environmental, administrative and societal.

4 Part 1. Wildlife Disease: Reviews, Case Studies and Health Assessments

Lessons learnt from 30 years studying diseases in wild populations of seals and cetaceans in temperate and tropical regions are reviewed by Geraci and Loundsbury in Chapter 1 and the implications to the Antarctic are discussed. The overwhelming insight is that even in relatively accessible locations, it is remarkably difficult to conclusively determine the cause of a mass mortality and this can be expected to be all the more difficult in the remote conditions of Antarctica. They also conclude that there is little that can be done to mitigate a large-scale mortality event wherever it occurs and whether caused by pathogenic disease or by other agents such as pollutants, natural biotoxins (algal blooms), or starvation. The remoteness of Antarctica adds what is probably an insuperable barrier to the challenge of mounting a successful rescue operation. The priority must be to ensure that human activity does not exacerbate naturally occurring disease by introducing or spreading disease agents or by creating additional stressors such as contamination or food shortage from over-fishing.

Diseases observed in captive animals may provide some perspectives on the type of diseases occurring in the wild, but this is no substitute for direct observations of wild populations in Antarctica, since environmental and biological conditions for the introduction, transmission and spread in captivity are considerably different. Reviews of the literature on diseases in the wild of Antarctic flying sea-birds (Chap. 2) by Woods et al. complements a previously published review of the literature on diseases in penguins (Clarke and Kerry 1993). McFarlane (Chap. 3) provides a similar review for Antarctic and sub-Antarctic seals. While acknowledging that research in this area is limited, these reviews confirm that Antarctic wildlife is susceptible to a wide variety of parasitic, viral, bacterial and fungal diseases.

Investigations into three apparent outbreaks of disease are reported as case studies in Chap. 4–6. Antibodies to infectious bursal disease virus (IBDV) have previously been found among Adélie and emperor penguins near Australia's Mawson Station, and fears were expressed that the virus may have been introduced through the disposal of poultry carcases (Gardner et al. 1997). Subsequent investigations by Watts

et al. (Chap. 4) found that the virus causing the antibody reaction was probably a nonpathogenic strain occurring naturally in the population. Kerry et al. in Chap. 5 present a cautionary tale. An unusual mortality event among adult Adélie penguins near Mawson Station occurred, which presented in a manner suggestive of infectious disease, although a physical cause was revealed after further investigation. Roe (Chap. 6), on the other hand, reports a mass mortality of New Zealand fur seals on Antipodes Island, which almost certainly has an infectious etiology. All these cases illustrate the need for very careful and thorough investigations to determine the cause of mass mortality events. Methods to investigate wildlife deaths where disease is suspected are provided in Appendices 1, 2 and 6. It is emphasised that in any such investigations, precautions must be taken to stop the spread of infection and to protect human health.

Health assessments of two geographically separated populations of Weddell seals are reported by Yochem et al. in Chap. 7 (McMurdo Sound) and Macfarlane in Chap. 8 (Vestfold Hills). Gray et al. (Chap. 9) provide a similar health assessment for leopard seals in the Vestfold Hills. These chapters summarise current knowledge on disease and trauma in these populations and provide reference values for blood biochemistry and haematology. Yochem et al. emphasise the need for information on normal mortality in order to better recognise and determine the cause of unusual mortalities. McFarlane points out that previous studies reporting ill health in seals generally do not give estimates of the proportion of the population affected or the rate of occurrence of new cases of diseases. Without this information, it is difficult to make comparisons between populations or to identify whether disease amplification is occurring. She suggests that future studies could benefit from taking an epidemiological approach. Gray et al. suggest that major changes in environmental conditions over large gradients, such as from the coast of Antarctica to the coast of Australia, can be associated with significant differences in body condition and other indicators of health. They also stress the importance of data from apparently healthy populations as the essential baseline for understanding the true significance of data and observations when illness is suspected.

5 Part 2. External Factors: Environmental, Administrative and Legal

The chapters in this section concentrate on those aspects of the Antarctic, including the natural environment and its administrative processes and social characteristics that distinguish it from other regions and may have a bearing on animal health.

The health of an animal is determined not just by the presence or otherwise of infectious disease agents, but by its complex interactions with the external environment. Indeed, ill health may be caused or exacerbated by a wide range of noninfectious agents such as physical trauma, starvation, and many others. In addition, whether or not an infectious disease causes ill health in a particular animal will be determined by its overall fitness and this in turn will be determined by many factors,

including prior exposure to other diseases and the external environment. The Antarctic environment differs from the environments of the more populated parts of the world in many ways that may influence the health of its native animals.

There is a growing concern that a combination of global warming, changes in atmospheric circulation patterns, and rapidly accelerating human activity is contributing to an increased risk of the introduction of exotic organisms to Antarctica and the sub-Antarctic islands and that their survival in these novel environments is becoming more likely (Frenot et al. 2005). An understanding of weather and climate is thus important for assessing the effects of climate change on the health of wildlife and also the way it can influence the introduction of carriers of disease from elsewhere. However, there are many potential mechanisms for environmental change to influence the health of Antarctic wildlife, some direct and some indirect. Rising temperatures coupled with extended periods of altered atmospheric circulation in the vicinity of the Antarctic Peninsula seem likely mechanisms for increasing the risk of transmission of diseases and parasites from lower latitudes and for increasing their chances of surviving on arrival. However, both climate and ecological systems are complex and, if in future there is a major Antarctic wildlife health incident associated with global climate change, it would not be surprising if the causal mechanism was previously unsuspected.

An overview of the climate and weather patterns of Antarctica and the Southern Ocean is presented by Pook (Chap. 10), who makes the point that, 'Climate and weather are the defining characteristics of Antarctica and to a large degree are what set it apart from other regions of the world'. The large-scale atmospheric pressure systems determine the isolation, or otherwise, of the Antarctic continent, at least for airborne particles, and also contribute to the geographical isolation of Antarctica in reducing opportunities for potential colonising species to cross the Southern Ocean. Antarctica is not completely isolated, however, as several mammal and bird species migrate to and from Antarctica annually and may carry micro-organisms with them. Elephant seals move between the Antarctic Peninsula region and Patagonia. Arctic tern breed in the northern hemisphere during the boreal summer but are found in Antarctic waters in the southern summer, and Wilson's storm petrel breed in Antarctica and migrate to the northern hemisphere in the Antarctic winter. Occasionally some species are transported to the Antarctic by wind streams, for example, a Kerguelen duck was found at Mawson Station in 2001 during a period of unusual northerly winds.

Local conditions of temperature, humidity and solar irradiance can all influence the survival of hosts, vectors, parasites and pathogens in the environment and, of course, control or severely limit the actions of people. However, the climate on the Antarctic Peninsula has changed, with the average temperature now 1°C higher and winter temperatures now just above zero. This may be conducive to the establishment of vectors of diseases, such as mosquitoes and ticks, and may cause heat stress in species of wildlife that have evolved to the cold conditions of Antarctica. It is important to note that seals and birds are well adapted for survival in the Antarctic environment, and descriptions, such as harsh or extreme, used to describe the environment in which they live may apply to human comfort and perhaps to species from other continents but are not applicable to the native fauna.

Chapters 11 and 12, describe the character and scale of modern human activity in Antarctica. They build a picture of the potential for people to be involved in the introduction or spread of wildlife disease and also their ability to mount an effective response to a disease event. Jabour (Chap. 11) describes the government sponsored national Antarctic programs, while Mortimer and Prior (Chap. 12) describe non-government tourist expeditions. The patterns of activity of these two groups are very different. Many more people visit Antarctica each year as tourists than with national Antarctic programs, but because tourist visits are generally short, the number of people days south of the Antarctic Circle from each type of expedition is approximately the same and there are many more person-days ashore associated with the national programs. Government expeditions tend to be concentrated on few localities, principally where support is provided from scientific stations operated year round, and activities radiate out from these using an array of over-snow vehicles and aircraft. In contrast, tourist expeditions are mostly ship based but make shorter visits to a wide variety of locations using inflatable rubber boats and helicopters. Tourists also tend to visit concentrations of wildlife, particularly penguin colonies. Given the two differing modes of Antarctic operation, how different is their impact on the Antarctic environment and wildlife health likely to be?

There is no evidence to suggest that human activity has been involved in the introduction of diseases into Antarctica wildlife or the translocation of endemic disease. This is despite a long history of introducing domestic animals. On the other hand, there is clear evidence that human activity has impacted on the health of Antarctic wildlife in a number of ways, reviewed by Riddle in Chap. 13. Some impacts have been direct and obvious while others are more subtle. In the past, habitat has been destroyed, whole penguin colonies have been moved, and eggs destroyed to make way for research stations and seals have been hunted for meat for sledge dogs. These activities are no longer permitted under internationally agreed measures to protect the Antarctic environment. However, other activities, such as major increases in the scale of fisheries in the Southern Ocean, have the potential for significant detrimental impacts on the health of wildlife populations, either directly through by-catch and entanglement in fishing gear or indirectly by removing food resources such as krill. During the investigation of an unusual health event in Antarctic wildlife, even if pathogenic disease agents are identified, it is worth considering whether other extrinsic factors, such as pollution, algal biotoxins, stress from physical disturbance, could have played a part in triggering the event.

Stress may well be a contributing factor in unusual health events, as it is known to depress immunity in many species. Stress is normal in animal populations but physiological stress can be exacerbated by human activity to levels that cause pathology. However, it is difficult to untangle the degree to which stress in animals is caused by human activities rather than being imposed by the natural environment. In Chap. 14, Gray and Rogers review techniques for objectively evaluating stress in seals using biochemical metabolites such as cortisol as biomarkers. Their conclusion that non-invasive techniques, such as analysis of faeces and urine, can provide useful data on the levels of biomarkers of stress is particularly important as this overcomes the potentially confounding problem of sampling using invasive techniques involving restraint of the animal, which in itself is likely to be highly

stressful. They provide a very practical summary of factors to consider when using non-invasive sampling, with particular emphasis on the stability of stress biomarkers to environmental conditions found in Antarctica, such as repeated cycles of freezing and thawing.

Environmental management regulations for Antarctica explicitly prohibit the introduction of non-native species and include measures to prevent wildlife being exposed to food waste, particularly poultry, which may carry pathogens. Sewage disposal is the only activity routinely undertaken in Antarctica knowing that it will result in the release of non-native species, including pathogens, to the environment. Smith and Riddle in Chap. 15 review the treatment and disposal of sewage and show that sewage treatment techniques commonly used at Antarctic research stations cannot guarantee the removal of all pathogens before effluent is discharged to the sea and that exposure of wildlife to effluent is likely. However, their conclusion that they could find no published accounts of sewage effluent being the cause of disease in any wildlife population, even in the most populated parts of the world, is both surprising and reassuring. Even if they have missed something in the literature, it is clear that exposure to sewage effluent is not a common cause of disease in wildlife, despite disposal of effluent to the environment being a world-wide phenomenon. That such a direct mechanism of exposure of wildlife to pathogens is not a widespread cause of disease is reassuring in itself. It is also reassuring as it suggests that wildlife are remarkably resilient to zoonotic transfer of disease from humans and may not be easily susceptible to pathogens introduced by people to Antarctica by other, less direct mechanisms, such as on dirty boots, clothing or equipment.

The legal and regulatory framework for protecting the health of Antarctic wildlife, derived principally from the complex regime of the Antarctic Treaty System (ATS) but also including other legal instruments in international law, is reviewed in Chap. 16 by Rothwell. In recent years, protection of the environment has become the major issue of concern for the ATS, with its Committee for Environmental Protection (CEP) being the prime advisory body to the ATS on matters related to environmental protection and management. Rothwell concludes that, while the legal structures for protection of the Antarctic wildlife are impressive, gaps do remain. The enactment of obligations arising from the ATS is the responsibility of individual consultative parties and acceding states through their own domestic legislation but, as with any international regime, much depends on the consistency and adequacy of implementation of individual states. The Madrid Protocol has a focus on *State-actors*, which means that its principal impact is upon the national Antarctic programs. For any Antarctic Treaty Party wishing to take a strong environmental stand, there is sufficient justification in the Madrid Protocol to implement tough measures for disease control. The emerging role of the CEP as 'watchdog' over the implementation of the Madrid Protocol is crucial for ensuring that all Parties accept at least some minimum standards for disease control. Tourists and the commercial operators who bring them to Antarctica are subject to the national legal regimes, which in the case of Antarctica are variable in their content and enforcement, supported by guidelines developed within the Antarctic tourist industry, which assist in reinforcing the intent of the Madrid Protocol.

Work on wildlife disease undertaken by the Antarctic Treaty Consultative Parties, principally through the CEP, is the subject of the final chapter by Riddle (Chap. 17). It recounts the recent process of developing awareness within the scientific community and the Antarctic Treaty System of the risk of human-mediated introduction and spread of disease among Antarctic wildlife. It includes an examination of the reports and recommendations from the Workshop on Diseases of Antarctic Wildlife, Hobart 1998, which focused on the need for risk assessment, monitoring, prevention and response. The Antarctic tourism industry responded quickly to advice from the workshop by introducing simple, practical methods for boot cleaning to reduce the risk of moving pathogens from one location to another (Curry et al 2005). Within the ATS, this workshop led to a request from the CEP for reports on the risk of human involvement in disease introduction and spread, and on practical measures to reduce risk. Some national programs responded by introducing their own precautionary procedures, such as plans for organisational response to the discovery of unusual animal mortalities in Antarctica. Among other objectives, these plans are designed to ensure that if infectious disease is responsible for animal deaths, it is not exacerbated by the very process of visiting neighbouring colonies to determine the spatial extent of the mortality event.

Six appendices to this volume were included because they each contain highly relevant, practical information that has been published before but is not readily accessible. These appendices include procedures for collecting samples for pathological analysis (Appendix A) and for toxicological analysis (Appendix B) established by the Commission for the Conservation of Antarctic Marine Living Resources. The recommendations arising from the Workshop on Diseases of Antarctic Wildlife held in Hobart in 1998 are included as Appendix C. Two reports prepared at the request of the Committee for Environmental Protection and referred to above are also included: one is a review and risk assessment of the introduction and spread by human activity of infectious disease causing agents in Antarctica (Appendix D) and the other is on practical measures to diminish the risk (Appendix E). An example of a response plan for organisational response to the discovery of unusual animal mortalities in Antarctica is included as Appendix F. Together, these appendices form a valuable resource for anyone faced with unusual wildlife mortalities either in Antarctica or elsewhere, whether investigating an actual event or preparing for potential events in future.

6 Conclusion

Diseases are present as a part of the natural environment of Antarctica, but their origins are as yet unknown. There is no evidence to suggest human involvement in their introduction or translocation, indeed natural pathways through animal migrations or the movement of vagrants may be more likely, although these are as yet unquantified. However, there is clear evidence that human activity has impacted on wildlife health in a number of ways both obvious and subtle. Mechanisms are now

in place within the Antarctic Treaty System and particularly the Madrid Protocol to protect the health of Antarctica's wildlife through informed debate and the regulation of human activity. What is required now is the will of all nations involved in field activities in Antarctica to work to the letter, and above all, to the spirit of these regulations. For our part we hope that this book will provide essential information needed by national policy makers and administrators for this purpose and, looking forward, will provide a stimulus for scientific research into health and disease to provide information to better support decision making and ultimately to ensure the maintenance of good health among Antarctica's wildlife.

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Part I
Wildlife Disease: Reviews, Case Studies and Health Assessments

Chapter 1
Risk of Marine Mammal Die-Offs in the Southern Ocean

J. R. Geraci and V. J. Lounsbury

1.1 Introduction

Compared to the vast amount of data on the northern polar and sub-polar marine mammals, there is relatively little information about the species in the Southern Ocean. These waters have been estimated to contain about 50% of the world's seal population and 80% of the world's biomass of pinnipeds (Laws 1984). Four species of seals – Weddell *Leptonychotes weddellii*, Ross *Ommatophoca rossii*, crabeater *Lobodon carcinophagus*, and leopard *Hydrurga leptonyx* – inhabit the pack-ice. In addition, the southern elephant seal *Mirounga leonina* and Antarctic fur seal *Arctocephalus gazella*, which breed farther to the north, forage southward into the marginal ice zone (Costa and Crocker 1996). Although these species are well adapted to the harsh polar environment, reproductive success depends on predictability of both ice conditions and food resources. Antarctic waters also are critical summer feeding grounds for many species of cetaceans, including six species of baleen whales and several species of odontocetes (Brown and Lockyer 1984; Costa and Crocker 1996; Moore et al. 1999).

Marine mammal die-offs appear to have increased in frequency in the past two decades (Geraci et al. 1999). With a few notable exceptions, such as mortalities due to oil spills, these events have been associated with outbreaks of infectious disease, harmful algal blooms, or oceanographic anomalies or unusual weather conditions that result in large-scale starvation or trauma. We can assume that these same factors are most likely to underlie future events in the Southern Ocean. However, we must be cautious when using information gained from conditions or events in the northern hemisphere, where contiguous coastlines may promote unusual migrations of

J.R. Geraci and V.J. Lounsbury
National Aquarium in Baltimore, Baltimore, MD, 21202-3194, USA
e-mail: jrgeraci@sbcglobal.net, vlounsbury@aqua.org

J.R. Geraci
University of Maryland School of Medicine, Comparative Medicine Program, Baltimore, MD, 21201-1192, USA

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marine animals as well as passive transport of algae and other organisms from one area to another, and where input of nutrients, contaminants, and pathogens from terrestrial sources is significant.

1.2 What Have We Learned?

Thirty years ago, studies on marine mammal health focused on the progression of disease in individuals, and on how marine species respond to specific disease agents in comparison with terrestrial mammals. A review of the literature reveals many observational studies: accounts of parasites and their effects; descriptions of tumors and other uncommon conditions; surveys of pathogens isolated from pinniped carcasses recovered from breeding grounds; and case studies of stranded dolphins and seals. Using this mechanistic approach, we learned much about disease processes. For some pinnipeds, for example, any disease or stress is likely to result in hyponatremia (low blood sodium), an incapacitating and sometimes fatal electrolyte disorder (St Aubin and Geraci 1986). For cetaceans and other pinnipeds, however, the same stressors may lead to failure of the protective mechanisms that prevent them from drinking salt water, and they develop hypernatremia (high blood sodium), or salt overload (St Aubin and Dierauf 2001; Walsh et al. 1990).

From these studies, we also learned that most marine mammals harbour a wide range of parasites by the time they are weaned or shortly thereafter. The species of parasites are frequently predictable in their occurrence and severity (Geraci and St Aubin 1987). A robust, apparently healthy dolphin or seal can carry a parasite load – for example, nematodes that appreciably reduce lung capacity – that would be fatal or at least debilitating to most terrestrial mammals. This apparent tolerance to heavy infestations of certain parasites led us to realize that marine mammals have evolved a substantial buffer capacity, i.e., a functional reserve that allows them to survive, if not thrive, in the harsh marine environment despite often seemingly serious health conditions.

We also learned that the line between health and debilitation can change quickly. In other words, once the functional reserve is exceeded, the animal's health declines rapidly. The rate of that decline depends largely on the thermal environment: debilitated marine mammals in high latitudes rarely survive long enough to become emaciated or develop the intensity of illnesses and infections that may be observed in animals in warmer waters.

Once we began to study large-scale mortalities, our thinking had to include other elements. We needed to consider the life history of the animals, whether the species was solitary or colonial, restricted in range, or migratory. We had to consider whether a reduction in traditional food sources might drive animals to feed on different prey, which could in turn expose them to nutrient deficiencies, parasites, or biological toxins for which they might have little tolerance; or whether the animals would respond to food shortage by moving into other habitats where they might face different risks, such as exposure to a novel pathogen. We realized that in order to unravel the

complex role of the physical environment in promoting disease, we had to consider oceanographic and meteorological conditions, water quality, changes in prey distribution and abundance, and the health of other species sharing their habitat.

Although we have witnessed an apparently unprecedented number of marine mammal mass mortalities since the late 1980s, we have also learned that similar events occurred in the past. Thus, we must consider historical evidence in our evaluation of current die-offs. As one example, during 1994, more than 100,000 Cape fur seals (*Arctocephalus pusillus*) along the coast of Namibia died from starvation; this followed a drastic reduction in prey availability due to long-term intrusion of oxygen-poor water onto the continental shelf (Geraci et al. 1999). Although some carcasses showed evidence of morbillivirus infection (Anselmo et al. 1995), the region's documented history of marine animal die-offs associated with harmful algal blooms and oceanographic anomalies (Wyatt 1980) reinforces the evidence that environmental factors were the underlying cause of the 1994 event. In 1828, Captain Benjamin Morrell, the master of the schooner *Antarctic*, visited this coast in search of fur seals and reported: '*it was evident that…not less than half a million had perished here at once, and that they had all fallen victims to some mysterious disease or plague*' (Wyatt 1980). Clearly, mass mortalities are not a new phenomenon.

1.3 Infectious Diseases

1.3.1 Infectious Diseases: Historical Perspectives

In retrospect, 30 years ago we knew little about infectious diseases in marine mammals, their transmission, and their effect on populations. First recognized in the late 1960s and considered insignificant to marine mammal health through the late 1970s, viral infections have emerged as a leading cause of large-scale mortality. The first event demonstrated to be of viral origin involved about 450 harbour seals (*Phoca vitulina*) that died along the New England coast during the winter of 1979–1980 (Geraci et al.1982). The cause was found to be an influenza virus of avian origin that had infected the seals, probably as they were hauled out along the shore of Cape Cod. Seals of all ages developed pneumonia, which forced many out of the water and onto crowded beaches where the virus could easily be spread from seal to seal by aerosol transmission. We now know that influenza continues to occur in harbour seals in that region, though in a mild form and on a small scale (Callan et al.1995), and occasionally in other North Atlantic phocids as well (Stuen et al.1994). Until the late 1980s, however, this event appeared to be an aberration in the normal patterns of marine mammal mortalities.

Successive outbreaks of morbillivirus infections — canine distemper (CDV), phocine distemper (PDV), porpoise morbillivirus (PMV), and dolphin morbillivirus (DMV) — in Baikal seals *Phoca sibirica*, European harbour seals and harbour

porpoises *Phocoena phocoena* and Mediterranean striped dolphins *Stenella coeruleoalba* from 1987 to 1992 showed that viral diseases can seriously affect marine mammal populations (Kennedy 1998; Van Bressem et al. 2001a). The die-off of striped dolphins forced us to reevaluate our views on the susceptibility of cetaceans to pathogens transmitted by aerosols. These events also resulted in reinvestigation of earlier events, including the 1987–1988 US mid Atlantic coastal bottlenose dolphin *Tursiops truncatus* die-off (Duignan et al. 1995a, 1996; Lipscomb et al. 1994) and a previously unexplained die-off of about 2,500 crabeater seals along the Antarctic Peninsula in 1955 (Laws and Taylor 1957). The discovery of antibodies to morbilliviruses in the crabeater seal population (Bengtson et al. 1991) and in stored serum samples from the bottlenose dolphins suggests that the viruses played some role in each of these events. Indeed, retrospective studies of the dolphin samples indicated that morbillivirus outbreaks have occurred sporadically in southeastern US coastal bottlenose dolphin populations since the early 1980s (Duignan et al.1996).

Through these studies we have learned valuable lessons about the epidemiology of viral infections in marine mammal populations. To spread rapidly, a virus requires a naïve host population of a minimum density, which in marine mammals can arise either through population growth or changes in social behaviour. Once infected, a migrating or wandering animal may carry the virus into new habitats. An epidemiological study of a viral infection in a marine mammal population must bring together information on the nature of the virus, its effect on the individual, its mode of transmission, and the behaviour and demographics of the species affected (Duignan et al.1995b, c; Hall et al.1992a; Heide-Jørgensen et al.1992). In some cases, animals infected with morbillivirus die after only a short period of illness and show clear evidence of pneumonia and encephalitis consistent with distemper infections in other species (Daoust et al.1993; Duignan et al. 1993; Kennedy et al. 1988, 1989). In other die-offs in which morbillivirus has been implicated, such as in the 1987–1988 Atlantic bottlenose dolphin event (Lipscomb et al.1994), the conditions apparent by the time the animals died or were stranded were associated with debilitation and immunosuppression, i.e., emaciation and overwhelming infections by bacteria, fungi, and other viruses (Geraci 1989). In such cases, the investigative process becomes complicated by the wide and variable range of observed health conditions and the possible involvement of more than one causative factor (e.g., a biotoxin and a pathogen).

Morbilliviruses have, not surprisingly, been a suspect in almost every subsequent marine mammal die-off – and with justification. Outbreaks of CDV in Caspian seals (*P. sibirica*) in 1997 (Forsyth et al. 1998) and 2000 (Kennedy et al. 2000), and of PDV in European harbour seals in 2002 (Harding et al. 2002) each killed thousands of seals. We are finding, too, that morbillivirus infection without recognized illness is common in many marine mammal populations (Kennedy 1998; Van Bressem et al. 2001a). In large populations in which the virus is endemic, such as pilot whales and dusky dolphins (Duignan et al. 1995b; Van Bressem et al. 1998), infection is presumably widespread but generally harmless because animals develop immunity through frequent exposure. Outbreaks occur when the virus is introduced into previously unexposed populations or into those that have lost immunity over a period of years. For example, evidence suggests that the morbillivirus

that caused the 1988 European harbour seal die-off may have been introduced into that population by infected, migrating harp seals (*Pagophilus groenlandicus*) (Markussen and Have 1992).

Many other viruses have been isolated from marine mammals. Some, such as pox viruses and caliciviruses, generally cause clinical illness only in animals already weakened by stress or disease (Geraci et al.1979; Kennedy-Stoskopf 2001); others, such as herpes viruses, have been linked to fatal infections (Kennedy et al. 1992), particularly in harbour seal pups (Borst et al.1986; Gulland et al. 1997). With the exception of influenza and morbilliviruses, none has been associated with marine mammal die-offs.

Even fewer bacteria have been shown to cause large-scale mortality of marine mammals. The spirochete *Leptospira interrogans* (serovar *pomona*) has been recognized since the early 1970s as a cause of periodic outbreaks of disease and increased mortality in California sea lions *Zalophus californianus* along the US Pacific coast (Dierauf et al. 1985; Gulland et al. 1996; Vedros et al. 1971). The condition has not been reported in marine mammals of the southern hemisphere. After eliminating viruses and biotoxins as possible causes of the 1998 die-off of New Zealand sea lions *Phocarctos hookeri*, investigators focused on a member of the genus *Campylobacter* (Duignan 1999). Its role as a primary pathogen in the outbreak was never established.

Certain pathogens or parasites of terrestrial origin are being found in marine mammals. Infection with the protozoan *Toxoplasma gondii* (sometimes in conjunction with *Sarcocystis*) causes fatal encephalitis in sea otters along the US Pacific coast, although infection also appears to be widespread among healthy animals (Lindsay et al. 2001). Domestic or feral cats are the presumed source, with oocysts shed in the faeces entering coastal waters in runoff or sewage (Miller et al. 2002).

As techniques to isolate and identify micro-organisms continue to improve, the list of marine mammal pathogens will undoubtedly grow, as will our understanding of their effects. There is, at the same time, growing evidence that changing environmental conditions can influence the prevalence or virulence of existing pathogens (Harvell et al. 1999), and that diseases can spread far more rapidly in the marine environment than in terrestrial populations (McCallum et al. 2003). Thus, when investigating an unusual event, we cannot ignore the possibility that a previously unknown agent, one never reported in marine mammals or in a particular region, or an organism traditionally not associated with serious disease, could be involved. One of the most important lessons of the past 25 years is that we must approach every investigation with an open mind.

1.3.2 Risks of Infectious Disease in the Southern Ocean

Compared to species from the northern polar waters, relatively little is known about diseases in the Antarctic marine mammals. We do know that resident seal populations are susceptible to the normal range of opportunistic pathogens (Baker and Doidge 1984; Baker and McCann 1989) and to certain viral infections, as demonstrated by

antibodies to herpes viruses in Weddell seals (Stenvers et al.1992) and to canine distemper (CDV) in crabeater and leopard seals (Bengtson et al. 1991). The discovery that one-third of the crabeater seals tested carried antibodies to CDV suggests that this virus, perhaps introduced by Greenland sledge dogs prior to use of effective CDV vaccines, is now endemic in the Antarctic Peninsula crabeater seal population (Bengtson et al. 1991). In distemper outbreaks in northern phocids, virus transmission was likely to have been promoted by crowded conditions on haul-out sites. The apparent lack of outbreaks in other Antarctic seals suggests that such close contact is infrequent, that populations may be too widely dispersed to support pathogen transmission, or that they are less susceptible to infection. A few leopard seals, the species perhaps most likely to have close encounters with crabeater seals, also showed evidence of CDV infection (Bengtson et al. 1991).

The Southern Ocean pinnipeds at greatest risk from infectious disease may be the southern elephant seal and the Antarctic fur seal. These species are highly gregarious during the breeding season and, for the elephant seal, during the moulting period (King 1983; Laws 1984). The potential for outbreaks of vector-borne disease (i.e., arboviruses) in southern elephant seals is suggested by the discovery of a new alphavirus carried by the elephant seal louse *Lepidophthirus macrorhini* (Linn et al. 2001). About 95% of Antarctic fur seals breed on South Georgia Island (Costa and Crocker 1996). An introduction of a virulent pathogen into that colony could be catastrophic, particularly if it coincided with a period of nutritional stress.

Several species of pinnipeds range widely in the Southern Ocean and frequently travel beyond their normal range to South America, New Zealand, Australia and South Africa. Like the harp seals in the North Atlantic, such vagrants could introduce disease to other populations, or acquire an infection that they introduce into their own population. Elephant seals utilize a particularly broad range and routinely travel long distances (McConnell et al. 2002). A satellite-tagged female seal from South Georgia was tracked more than 2,600 km to feeding areas along the northwestern coast of the Antarctic Peninsula (McConnell et al.1992); other elephant seals have been observed as far from home as South Africa, Peru, Angola, and Oman (Johnson 1990; King 1983). Leopard and crabeater seals are also known to stray far from their normal range, as are sub-Antarctic fur seals *Arctocephalus tropicalis*. The latter is also known to haul out with other species of *Arctocephalus* (Garrigue and Ross 1996; King 1983), increasing the potential for interspecies disease transmission.

Only within the past two decades have we recognized the significance of transmissible diseases in cetacean populations. To date, serious outbreaks have occurred only in social odontocetes. Other odontocetes and baleen whales tend to occur alone or in loose aggregations, conditions that would not promote disease transmission. Thus, although humpback whales, *Megaptera novaeangliae,* and fin whales, *Balaenoptera physalus,* migrate long distances between winter breeding grounds and summer feeding areas in the Antarctic, and may intermix with individuals from other stocks in both areas – including perhaps individuals from the northern hemisphere populations (Acevedo and Smultea 1996; Gambell 1985) – the risk of a die-off caused by any known pathogen seems low.

Other than the growing evidence of widespread exposure of Pacific Ocean odontocetes to morbilliviruses (Duignan 2000; Reidarson et al. 1998; Van Bressem et al. 1998), we know little about the incidence and severity of infection in cetaceans of the southern hemisphere. However, we can make some predictions based on knowledge gained from past events. Since the outbreak in Mediterranean striped dolphins in 1990–1992, evidence of morbillivirus infection has been found in the majority of odontocete species tested in the North Atlantic (Duignan et al.1995a). In pilot whales, *Globicephala melas* and *G. macrorhynchus*, infection is believed to be endemic, i.e., widespread but of little consequence because the whales have developed immunity through frequent exposure. However, these species commonly associate with other odontocetes and thus may act as vectors, spreading the virus to other Atlantic cetacean populations (Duignan et al. 1995b, 1996).

There are a number of pelagic, migratory, highly social species of odontocetes in the southern hemisphere that could play a similar role as carriers of pathogens, including both short-finned and long-finned pilot whales. Although their range does not extend into the Antarctic waters, long-finned pilot whales do enter the sub Antarctic regions where they may mix with other social species, such as dusky dolphins, *Lagenorhynchus obscurus,* and hourglass dolphins, *L. cruciger*, which venture farther southward. Long-finned pilot whales, at least in New Zealand waters, may be commonly infected with morbillivirus (Duignan 2000). Sperm whales, *Physeter catodon,* and killer whales, *Orcinus orca,* might also be good candidates for transmitting diseases from one region to another. Social tendencies aside, cetaceans in pack-ice may find themselves confined with other individuals or species. Taylor (in Laws and Taylor 1957) reported that an Arnoux's beaked whale *Berardius arnuxi* was found trapped in an ice pool, along with 120 minke whales *Balaenoptera acutorostrata* and 60 killer whales, in the Prince Gustav Channel in the winter of 1955 – in the same area as the concurrent die-off of crabeater seals.

Other than morbilliviruses, influenza viruses are the only viruses known to have caused large-scale mortalities of marine mammals. Aquatic birds worldwide serve as reservoirs for influenza A viruses, which are spread by faecal–oral transmission. Transmission from birds to mammals involves mutation or recombination; infection is then spread through aerosols. The outbreak in New England harbour seals showed that some strains can be fatal to marine mammals. The recurrent outbreaks in that population suggest that the virus is not maintained within the population, and that influenza infections in marine mammals probably represent independent introductions from aquatic birds (Mandler et al.1990; Webster et al. 1992). In the Ross Sea Dependency, serum antibodies to influenza A viruses have been detected in Adélie penguins, *Pygoscelis adeliae,* and Antarctic skuas, *Catharacta maccormicki*, but not in Weddell seals (Austin and Webster 1993). Introduction of a mutant or recombinant virus into naïve pinniped populations could be serious, particularly if coincident with conditions that increase contact rates, such as high population density or reduced ice cover (i.e., haul-out space).

The approximately 20,000 tourists (2005/06) who visit Antarctica each year (IAATO 2007), as well as the scientists and other personnel working there, represent another potential route for disease introduction and spread. The discovery of antibodies

to infectious bursal disease virus (IBDV) in emperor *Aptenodytes forsteri* and Adélie penguins near Australia's Mawson Station led to concerns that the virus may have been transmitted to scavenging birds from poultry scraps and then spread through droppings to penguins (Gardner et al. 1997). Whether IBDV was introduced or the findings reflect the presence of a closely related, endemic virus has yet to be demonstrated and highlights the difficulty of establishing with any certainty the origin of new – or newly discovered – pathogens.

While perhaps improbable, there are other possible mechanisms for introducing infectious agents into Antarctic animal populations. Studies have shown that airborne terrestrial algae and cyanobacteria originating in South America are transported to the South Orkney Islands (Marshall and Chalmers 1997); we cannot preclude the possibility that resistant forms of some pathogenic micro-organisms might have the same potential to spread from one region to another (Hughes 2003). The bacterium *Burkholderia pseudomallei,* which causes melioidosis in humans and several other species of mammals in Southeast Asia and northern Australia, is one example of a pathogen that appears capable of surviving in a dormant state in water and soil (Kanai and Kondo 1994). Humans and marine mammals may contract infection through wounds and by inhaling soil dusts (Liong et al. 1985). Might marine debris provide yet another means of introducing animal vectors to the Southern Ocean (Barnes and Fraser 2003), particularly in areas of continued regional warming?

Infectious agents could contribute to a die-off by rendering animals more vulnerable to toxins or other pathogens, or less able to cope with the stress of prey depletion or unusual environmental conditions. Some organisms generally associated with only mild or nonfatal illness, for example, seal herpes virus, may be relatively common in certain wild populations. In 1990 and 1991, a number of Weddell seals from the Vestfold Hills area showed purulent nasal discharge and watery diarrhoea (McFarlane 1996, also McFarlane this volume), signs similar to those attributed to herpes virus infection in Weddell seals in the eastern Weddell Sea (Stenvers et al. 1992). Of perhaps greater concern is the increased occurrence in marine mammal populations of several diseases known previously only in humans or domestic animals. Some organisms, such as *Salmonella* sp., have been implicated in disease in marine mammals but also have been isolated from apparently healthy animals (Baker et al.1995; Banish and Gilmartin 1992; Gilmartin et al. 1979). Several *Salmonella* serotypes were isolated from the New Zealand sea lions during the 1998 die-off, and a number of deaths were attributed to salmonellosis (Duignan 1999). Salmonellae also have been isolated from Antarctic fur seals and gentoo penguins *Pygoscelis papua* on Bird Island. In these populations, the observed increase in prevalence between 1996 and 1998, and low heterogeneity of isolates, may indicate that the organisms were recently introduced (Palmgren et al. 2000).

Campylobacter jejuni (Broman et al. 2000) and *Chlamydophila abortus* (Herrmann et al. 2000) also have been isolated from macaroni penguins, *Eudyptes chrysolophus,* and brown skuas, *Catharacta antarctica*, respectively, on Bird Island. Antarctic fur seals tested as part of this study showed no evidence of infection with *C. jejuni*, yet this bacterium does infect other mammals and is a common cause of enteritis in humans (Broman et al. 2000). The strain of *Chlamydophila* isolated is

similar to those that cause abortion or enteritis in sheep and cattle (Hermann et al. 2000). Penguins and other seabirds thus may serve as important vectors or reservoirs for a variety of pathogens of potential concern for local pinnipeds.

Brucella seems to be one bacterium (or a group) that can cause primary disease in some marine mammals, as it does in terrestrial species. First reported in harbour seals, a harbour porpoise, and a common dolphin from Great Britain (Ross et al. 1994) and in captive dolphins in California in 1994 (Ewalt et al. 1994), infection is widespread in marine mammals (Foster et al. 2002; Nielsen et al. 2001; Van Bressem et al. 2001b). Though the full range of pathogenicity is unknown, *Brucella* has been associated with reproductive failure and other lesions in some cetaceans (Miller et al. 1999; Ohishi et al. 2003), and isolates from seals have caused abortion in experimentally infected cattle (Rhyan et al. 2001). Antibodies to *Brucella* have been reported in a Weddell seal and several Antarctic fur seals from the South Shetland Islands (Retamal et al. 2000).

Mycobacteria of the complex associated with tuberculosis (*Mycobacterium bovis, M. tuberculosis*) have a peculiar niche in the Southern Ocean. First discovered in captive fur seals and seal lions in Australia (Forshaw and Phelps 1991), infection has now been reported in free-ranging sea lions and fur seals from Australia (Cousins et al. 1993; Woods et al. 1995), New Zealand (Hunter et al. 1998) and Argentina (Bernardelli et al.1996). Eventual transmission of this pathogen to Southern Ocean pinnipeds – particularly fur seals – seems inevitable.

1.4 Harmful Algal Blooms

Of the approximately 5,000 known species of marine phytoplankton, at least 40 are capable of producing toxins that can be harmful to humans and other top predators (Hallegraeff 1993; Van Dolah et al. 2003). Only in the past 20 years have we begun to realize the potential impact of biotoxins on marine mammal populations. These compounds are difficult to detect and may leave little evidence of their presence. Thus their role in marine mammal mortality is often uncertain and may have gone unrecognized in the past.

1.4.1 Harmful Algal Blooms: Historical Perspectives

Between June 1987 and March 1988, more than 740 bottlenose dolphin carcasses washed ashore along the US mid-Atlantic coast. Based on tissue and stomach samples, environmental evidence, and lack of evidence pointing to any single, common pathogen, investigators concluded that exposure to brevetoxin, produced by the dinoflagellate *Karenia brevis* (formerly *Gymnodinium breve*), had weakened the dolphins, leaving them susceptible to overwhelming infection by a variety of opportunistic pathogens (Geraci 1989; Geraci et al. 1999). Although this conclusion was

controversial – and the role of the identified algal toxins in this event uncertain – the case for natural toxins as a cause of marine mammal mortality was strengthened during the winter of 1987–1988, when 14 humpback whales died in Cape Cod Bay after eating fish contaminated with saxitoxin (Geraci et al.1989), the same toxin that causes paralytic shellfish poisoning in humans.

These were not the first marine mammal mortalities for which algal toxins had been proposed as the underlying cause. Earlier, there had been suspected poisonings of bottlenose dolphins in the Gulf of Mexico, Hawaiian monk seals *Monachus schauinslandi*, northern sea otters *Enhydra lutris kenyoni*, northern fur seals *Callorhinus ursinus*, and Florida manatees *Trichechus manatus latirostris* (Geraci et al.1999). In these earlier events, the numbers of animals involved were either unknown or relatively small, and the evidence circumstantial.

In the 1990s, harmful algal toxins emerged as a significant threat, especially to small, isolated populations. In spring 1996, a red tide killed an estimated 150 manatees; this occurred in the same area of southwestern Florida as a 1982 event in which about 37 manatees died (O'Shea et al.1991). The 1996 deaths were attributed not only to ingestion of toxins but also to inhalation of toxic aerosols. For that die-off, advances in laboratory techniques provided conclusive evidence of brevetoxin in tissue samples from the affected animals (Bossart et al.1998). Periodic red tides in southwestern Florida continue to account for significant manatee mortality in that region.

Toxic blooms were also implicated in the 1997 die-off of Mediterranean monk seals, *M. monachus,* at Cap Blanc, Mauritania (Hernandez et al.1998), and more recently in recurrent events along central California involving primarily California sea lions, and also northern fur seals, cetaceans, and sea otters (Lefebvre et al. 1999; Van Dolah et al. 2003). While the role of biotoxins in the monk seal die-off remains controversial (Harwood 1998), the link in the California die-offs was clearly demonstrated in 1998 (Scholin et al. 2000). The sea lions became ill after ingesting anchovies and sardines contaminated with domoic acid, following a bloom of the toxic diatom *Pseudo-nitzschia australis*.

Saxitoxin, domoic acid, and brevetoxin can be transferred through zooplankton and/or planktivorous fish to marine mammals (Geraci et al. 1989; Scholin et al. 2000). Brevetoxin can also poison through inhalation of aerosolized toxin (Bossart et al. 1998). Sublethal or chronic exposure to toxins may impair the immune function (Trainer and Baden 1999) or cause debilitation (Bargu et al. 2002; Durbin et al. 2002), leading to electrolyte imbalance, emaciation, hypothermia, overwhelming opportunistic infections and death.

Scientists generally agree that the frequency and distribution of harmful algal blooms have increased in the past two decades (Hallegraeff 1993; Van Dolah et al. 2003). How much of this increase is due to human activities is unknown. In some areas of the world, such as the Inland Sea of Japan and the Aegean Sea, the growing frequency of toxic blooms is related to coastal eutrophication and in other areas to oceanographic anomalies (Hallegraeff 1993; Tester and Fowler 1990; Yin et al. 1999). Toxic species can also be introduced in ballast water or through translocation of shellfish stocks (Hallegraeff et al.1990).

1.4.2 Risk of Harmful Algal Blooms in the Southern Ocean

The lack of reports of toxic blooms in Southern Ocean waters does not preclude their presence. Blooms causing paralytic and amnesic shellfish poisoning have been reported in sub Arctic and Arctic waters; and species of *Alexandrium* or *Gymnodinium* associated with paralytic shellfish poisoning are known to occur in southern Australia, New Zealand, and southernmost Africa and South America (Cordova et al. 2002; Hallegraeff 1993). Diatoms (*Pseudo-nitzschia* sp.) associated with amnesic shellfish poisoning also occur in New Zealand and along the southwestern coast of South America (Van Dolah et al. 2003). *Karenia brevis* was identified as a cause of neurotoxic symptoms in humans during harmful algal blooms in New Zealand in 1993 (O'Hara 1993).

Many species of algae thrive, at least seasonally, in Antarctic waters. The coastal regions of the Antarctic Peninsula, the waters around South Georgia, and the receding zone of the seasonal pack-ice are particularly productive (Karl et al.1992; Korb and Whitehouse 2004). Toxic species might already be present, or might be introduced from sub Antarctic waters by oceanographic anomalies associated with climate change. In a warming Antarctic Ocean, blooms may be promoted as the melting ice increases bioavailable iron and other nutrients (Sedwick and DiTullio 1997). Species of nontoxic algae may suddenly become toxic, for example, due to a change in available nutrients (Hallegraeff 1993).

The physical isolation of the Southern Ocean, normal current patterns, and temperatures might reduce the chances of introducing water-borne toxic algae. However, such organisms could conceivably be carried southward from southeastern Australia, southern New Zealand, or Argentina and, once entering the Antarctic Circumpolar Current, reach areas that support high primary productivity, such as north of the Ross Sea, along the western coast of the Antarctic Peninsula, or north of South Georgia Island, where marine mammals concentrate to feed.

As in other populations, pinnipeds and cetaceans ingesting contaminated prey or breathing aerosolized toxins might experience acute or chronic health effects, including impaired immune function. Harmful algal blooms might also affect animals in ways that have yet to be identified. The toxic dinoflagellate *Alexandrium catenella*, for example, may carry a variety of bacteria, including *Mannheimia* (formerly *Pasteurella*) *haemolytica* (Cordova et al. 2002), a known mammalian pathogen. Animals already compromised by toxin exposure during massive blooms might face added risk of infection from organisms released into the water during algal cell lysis.

1.5 Contaminants

Most of the die-offs of the 1980s and 1990s raised serious concerns about the health of the environment and the possible role of contaminants in these events. This was especially true in the 1988 US Atlantic coast bottlenose dolphin die-off

and in the outbreaks of morbillivirus in European and Mediterranean waters (Aguilar and Borrell 1994; Geraci et al.1999; Hall et al. 1992b). Contaminant burdens in some carcasses were high, particularly of certain organochlorines. Although studies of those events failed to demonstrate a link between contaminant levels and mortality (Geraci et al. 1999; O'Shea and Tanabe 2003), growing field and experimental evidence supports a causal relationship between chronic exposure to certain organochlorines and impaired immune function in at least some species of marine mammals (Jepson et al. 1999; Lahvis et al. 1995; Ross et al. 1996).

Relatively few studies have been conducted on contaminant burdens in pinnipeds of the Southern Ocean. Existing data indicate that although these species are exposed to anthropogenic contaminants, primarily from atmospheric sources, tissue levels are far lower than those commonly found in species in more industrialized areas (Hidaka et al. 1984; Kawano et al. 1984; Tanabe et al.1983). Contaminant levels in species feeding primarily on krill – baleen whales, crabeater seals, and Antarctic fur seals – should be even less (O'Shea and Brownell 1994). Although areas of human habitation, such as McMurdo Station (Lenihan et al.1990), may be highly contaminated with hydrocarbons, metals, and debris from waste disposal, such localized pollution would not be expected to affect large segments of any population. Thus, for these species, contaminant burdens are unlikely to play a contributing role in large-scale mortality caused by infectious disease.

The risk of a more localized die-off due to a toxic spill is greater. The diesel oil spilled from the Argentine supply and cruise ship *Bahia Paraiso* near the tip of the Antarctic Peninsula in 1989 had an immediate effect on krill and killed penguins from nearby colonies (Anon. 1989); significant mortality of marine mammals was not reported (St Aubin 1990). However, as demonstrated by the *Exxon Valdez* spill in Alaska in 1989 and the *San Jorge* spill in Uruguay in 1997, spills near pinniped breeding colonies can have severe effects (Levine et al.1997; Loughlin 1994). Deaths of harbour seals in Alaska were attributed to the toxic effects of inhalation of volatile hydrocarbons (Spraker et al. 1994); oiled fur seals in Uruguay may have also died from hypothermia. In polar waters, where biodegradation and volatilization are diminished, oil will persist and concentrate along ice edges, in leads, and between ice floes – areas where pinnipeds are most likely to be exposed. Thick, viscous oil can mat hair, prevent limbs from moving freely, and cause death from drowning or exhaustion (Geraci and St Aubin 1990).

Pinnipeds and cetaceans are generally unlikely to ingest significant amounts of oil. Those preying on contaminated krill might be at risk of ingesting hydrocarbon compounds for a few days or weeks, and species feeding on benthic invertebrates for a considerably longer time (Geraci 1990). A leopard seal that eats oiled penguins or other seabirds might ingest a harmful quantity of oil (St Aubin 1990).

Although a spill can have a devastating impact on a local environment, only rarely have such events caused large-scale mortality of marine mammals. In any area with significant vessel traffic, oil exploration or drilling, or along tanker routes, an accident, although rare, is probably inevitable, and history tells us that it could affect marine mammals in the vicinity.

1.6 Prey Depletion

Animals can die of starvation, especially the very young and the old. Widespread starvation usually results from prey depletion associated with sudden changes in oceanographic conditions. Examples include high mortality among Galapagos pinnipeds during the 1982–1983 El Niño (Trillmich 1985) and of Cape fur seals in Namibia in 1994 (Geraci et al. 1999). Starving animals eventually die – some quickly, as would a pup deprived of milk. Others die after a period of illness triggered by malnutrition and mediated by factors such as hypothermia, dehydration and electrolyte imbalance, stomach ulcers, hormonal disturbances, and infection by parasites and opportunistic pathogens (Baker 1984; Banish and Gilmartin 1992; St Aubin and Geraci 1986).

In prey-depleted waters, the search for new food sources can be risky or have unexpected consequences. Unusual migrations of harp seals into Norwegian waters in the mid 1980s coincided with dramatic declines in Barents Sea fish stocks; in 1987, an estimated 100,000 seals drowned in coastal fishing nets (Geraci et al.1999). In North Atlantic fin whales, an increased incidence of serious kidney infections with the nematode *Crassicauda* sp. coincided with apparent declines in zooplankton stocks; the presumed link is lowered resistance to infection due to nutritional stress (Lambertsen 1992). In California sea otters, the increased prevalence of peritonitis caused by the acanthocephalan *Profilicollis* sp. has been linked to ingestion of certain species of sand crabs, which may serve as intermediate hosts for these seabird parasites (Estes et al. 2003). In Alaska, killer whales facing reduced abundance of pinniped prey may have turned to sea otters, contributing to – or perhaps causing – the dramatic decline of that population in the past few decades (Estes et al. 1998).

Marine mammals feeding in Antarctic waters rely on predictable food resources, i.e., regional areas of high productivity, perhaps coupled with oceanographic features that act to concentrate prey. Such conditions are found along the southern boundary of the Antarctic Circumpolar Current (Costa and Crocker 1996; McConnell et al. 2002). The unusually dense krill stocks north of South Georgia Island, which depend on advection from spawning grounds along the western Antarctic Peninsula and the Weddell Sea (Thorpe et al. 2002), support abundant regional populations of seabirds and Antarctic fur seals. Any conditions that reduce the biomass of krill, the foundation of the Southern Ocean food chain, will affect marine animal populations.

Low krill years, which have been observed after El Niño events, e.g., 1977–1978, 1983–1984, 1990–1991 and 1993–1994, were years of population decline or reduced fitness for several seabird species and fur seals on Bird Island (Costa and Crocker 1996; Croxall et al. 1999; Reid and Croxall 2001). After a period of stability in the 1980s, numbers of Antarctic fur seals on Bird Island declined in the 1990s, despite increases at other breeding colonies (Reid and Croxall 2001). In 1994, unusual premature pupping, low birth weight, and high pup mortality were attributed to poor condition of adult females following a year of record low krill availability (Croxall et al. 1999; McCafferty 1999). Evidence suggests that more frequent years of prey shortages have left top predators at South Georgia operating at the limits of food resources (Reid and Croxall 2001). Other krill specialists, such as crabeater

seals and baleen whales, might experience comparable, although perhaps less observable, effects.

Indeed, changes in the population demographics of Antarctic phocid seals observed over the past few decades indicate that large-scale oceanographic variations may be an important factor in regulating top predator populations (Costa and Crocker 1996; Testa et al. 1991).

1.7 Oceanographic Anomalies and Climate Change

In the past few decades, we have witnessed dramatic and sometimes catastrophic effects of El Niño events on many Eastern Pacific pinniped populations (Trillmich et al. 1991). The impact of these events is widespread and is evident in populations of the Southern Ocean (Costa and Crocker 1996; Testa et al. 1991). The influences of climate change in this region are difficult to quantify due to normal inter-annual variability and larger-scale temporal patterns (Thorpe et al. 2002). Overall, models predict that global warming will have less impact on Antarctic ecosystems than those in the Arctic; changes observed to date include an increase in sea-ice cover in some areas and a decrease in others (Clarke and Harris 2003).

Climate models, however, did not predict the dramatic regional warming trends already observed along the Antarctic Peninsula over the past 50 years, an area that encompasses key krill breeding and nursery grounds (Clarke and Harris 2003; Croxall et al. 2002). The rising temperatures in this region coincide with a dramatic decline of krill stocks, which rely on sea-ice algae for food and on ice cover for juvenile survival (Atkinson et al. 2004; Reid and Croxall 2001). The ramifications of this change extend far beyond local marine animal populations, since krill from this area are transported by currents and concentrated in the waters around Bird Island and South Georgia (Atkinson et al. 2004; Thorpe et al. 2002). Nutritional stress may be evident as a slow decline in numbers, decreased birth rate, or reduced juvenile survival – impacts already observed in fur seal and some seabird populations. Reduced fitness also opens the door to pathogens and infection by normally harmless opportunistic organisms.

Models also failed to predict that temperatures within the Antarctic Circumpolar Current would rise faster than those in the Global Ocean, underscoring the complex relationships between tropical and subtropical climate variations and conditions in the Southern Ocean (Clarke and Harris 2003). Relatively small shifts in the Antarctic Circumpolar Current, for example, may contribute significantly to the inter-annual variation in krill stocks around South Georgia (Thorpe et al. 2002). Major shifts in ocean circulation are believed to cause rapid changes in climate (Kerr 1998), which can lead to restructuring of plant and animal communities (Walther et al. 2002). What such oceanographic changes might entail for the Southern Ocean is impossible to predict with certainty. However, increases in frequency or severity of climatic or oceanographic anomalies would be expected to lead to even greater disruption of populations through influences on distribution and survival.

Some species might benefit from decreased competition for prey or disappearance of predators. For most populations, however, negative impacts are likely to prevail, at least in the short term.

Sea-ice is a key feature of the polar environment and vital for reasons other than food production. Stable ice is crucial to successful breeding and rearing of polar phocids. An early thaw, or ice break-up due to storms, can be devastating to pinnipeds, especially pups (Geraci et al.1999). Among Antarctic species, mortality levels of 30–80% have been reported for Weddell seal pups (Mansfield in McFarlane 1996) and southern elephant seal pups at Signy Island (Laws 1960) during years of unusual ice conditions.

Although the mechanisms are not clear, and in some cases the association may be coincidental, unusually warm weather has been associated with several marine mammal die-offs (Geraci et al. 1999), including those involving harbour seals in New England (winter 1979–1980) and Europe (1988), bottlenose dolphins in the US mid-Atlantic (1987–1988), and striped dolphins in the Mediterranean (1990). For seals, warm conditions may increase haul-out time and thus opportunities for aerosol transmission of pathogens. In polar waters, reduction of available ice could lead to crowding or increased inter-species contact, conditions that further enhance the risk of disease transmission. The 1955 crabeater seal die-off occurred in spring after an abnormally warm winter, when the seals in the area were tenfold the usual number (Laws and Taylor 1957).

On a larger scale, evidence from ocean sediments indicates that the 'conveyor belt' current that moves water around the globe, warming northern Europe and Asia, has repeatedly slowed and even stopped during the past 100,000 years, causing colder temperatures in the north but warming southern waters; this is one potential effect of global warming (Kerr 1998). Events of this magnitude develop over longer periods of time and can influence evolutionary patterns. The disappearance of the cetacean taxa during the Oligocene, for example, is believed to have resulted from decreased availability of upwelling zones, which was associated with changes in thermal gradients (Lipps and Mitchell 1976). History reminds us that such changes, and their impacts on species viability, are largely beyond human control.

1.8 What Can We Do?

Realistically, there is currently little we can do to mitigate a large-scale mortality event caused by pathogens, biotoxins, or starvation. Some countries have the resources to rescue and rehabilitate a few cetaceans, and perhaps even hundreds of pinnipeds. Except for highly endangered species, the benefit of rescue and release at this level is negligible to wild populations. Immunization strategies for large populations are impractical, probably impossible. Even vaccines that might be administered from the air would reach only a small segment of a population. Once a die-off has begun, the more logical approach would be to let nature take its course. When a disease outbreak occurs, we expect that many animals will be infected; some will

die, while others will become immune and perhaps produce healthier offspring. Still, we can and must play a role to ensure that human activities do not contribute to the spread of disease to other populations. In areas where contamination and other forms of habitat degradation are factors in animal health, no matter how nebulous, we can promote habitat preservation and restoration. This includes measures to prevent introduction of alien species – from algae to mammals – into new waters. When over-fishing may be an underlying factor in marine mammal mortality, we can promote more responsible fishing methods or changes in quotas.

Beyond these measures, we can be prepared to respond to events that might occur. A logical first step is to collect and analyze serum samples from as many species as possible in order to determine the range of pathogens to which various populations have been exposed. Considered together with any observable patterns of exposure, the micro-organisms themselves might suggest an origin and possible route of transmission. We might then be able to take preventive measures to protect other populations or limit exposure to additional pathogens or toxins, especially those linked to human activities. Collecting serum and tissue samples for banking will allow for retrospective studies when new technologies may provide answers to previously unexplained events. Most importantly, the information we gain from die-off investigations and studies of baseline health conditions in animal populations will help us to improve environmental policies and animal management programs in ways that could help protect and sustain viable marine mammal populations.

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Chapter 2
Diseases of Antarctic Seabirds

R. Woods, H.I. Jones, J. Watts, G.D. Miller, and G.R. Shellam

2.1 Introduction

Seabirds are the approximately 300 avian species that feed mainly at sea (Harrison 1983). Nearly all Antarctica's bird species are seabirds. Of the 43 species breeding south of the Antarctic Convergence, only five live entirely on land. The marine birds include seven species of penguins, 24 of petrels, 2 of cormorants and 5 species of gulls, skuas and terns. Antarctic seabirds that breed on the peripheral islands also fly far south of their breeding latitudes in summer and the Antarctic waters are visited by sub-Antarctic and subtropical breeding species, and also by some species of Arctic seabirds (Stonehouse 1972).

The diseases and veterinary management of seabirds has been reviewed (Pokras 1996; Vogelnest 2000). There is, however, very little information about diseases in the Antarctic seabirds. This chapter limits itself primarily to those diseases reported among Antarctic seabirds, with the emphasis on flying birds. Diseases of penguins have been reviewed by Clarke and Kerry (1993, 2000), however, where relevant for comprehensiveness, research on penguin diseases since these publications, and some earlier research, have also been included.

Only 2% of Antarctica is free from ice; so nesting seabirds have limited space for their breeding activities. This important feature, together with the absence of land-based predators, leads to the aggregation of hundreds of thousands, and some-

R. Woods
Australian Wildlife Health Network, PO Box 20, Mosman, NSW, 2088, Australia
e-mail: RWoods@zoo.nsw.gov.au

H.I. Jones, J. Watts, and G.R. Shellam
Microbiology and Immunology, School of Biomedical, Biomolecular and Chemical Sciences, University of Western Australia, WA, 6009, Australia
e-mail: hjones@cyllene.uwa.edu.au
e-mail: djawatts@bigpond.net.au
e-mail: gshellam@cyllene.uwa.edu.au

G.D. Miller
Biology Department, University of New Mexico, Albuquerque, New Mexico, 87131, USA
e-mail: gdmiller@iinet.au

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times millions, of individuals during the breeding season. This crowding habit makes the seabirds and their offspring particularly vulnerable to endo- and ectoparasites (Clifford 1979; Olsen et al. 1995; Rothschild and Clay 1952).

2.2 Parasites and Diseases

2.2.1 General

The border between 'infection' and 'disease' caused by the infecting organism can be an artificial one; concomitant infections, stresses caused by poor nutrition or human impact and parasite burden are among the factors that can change an asymptomatic infection to a true disease state. For this reason, we include records of many infectious agents – viruses, bacteria, protozoa and helminths – that did not appear to be affecting the health of the bird, but which under certain conditions, may do so. Furthermore, taxonomists are not usually pathologists, and information on the state of health of the host may not have been noted.

2.2.2 Ectoparasites

2.2.2.1 General

Ectoparasites of Australian, New Zealand and Antarctic birds have been reviewed by Murray et al. (1991).

2.2.2.2 Ticks

Two different families of ticks feed on seabirds: the Ixodidae or hard ticks, and the Argasidae or soft ticks (Nuttall 1984). The various species of ticks that feed on seabirds, and their geographical distribution, have been reviewed by Clifford (1979) and those of Antarctica by Wilson (1970) and Zumpt (1952). All of the ticks found associated with seabirds, (either on them or in their nests or burrows) in the Antarctic and sub-Antarctic, are members of the genus *Ixodes*. The commonest species, *Ixodes uriae*, has a circumpolar distribution on the sub-Antarctic Islands, and has been found on the Antarctic Peninsula (Gressitt 1965; Miller unpublished data). It is found on a wide range of hosts (Murray et al. 1991). *Ixodes pterodromae* is also circumpolar, and is found in the nests of prions and diving petrels. *Ixodes kerguelensis* has been found on prions and petrels on the Kerguelen Archipelago and Heard Island, and *I. diomedeae* on the yellow-nosed albatross *Diomedea chlororhynchos* on Nightingale Island, Tristan da Cunha.

The taxonomy of the other species of the *I. auritulus* and *I. percavatus* groups has caused difficulty, and there may be several species involved (Arthur 1960; Murray 1967).

The seabird-associated tick *I. uriae* (sub-genus *Ceratixodes*) is probably the most important of all the ectoparasites of seabirds. It has a large and unique bi- and circumpolar distribution (see Fig. 2.1 in Clifford 1979) and infests more than 50 species of seabirds that colonise regions of high latitudes in both hemispheres, including black-browed albatrosses, *Diomedea melanophris,* at Campbell Island (Duffy 1989; Mehl and Traavik 1983; Murray et al. 1991; Olsen et al. 1993, 1995). It is important as a host for *Borrelia* spirochetes in seabirds (Olsen et al. 1995), and at least 31 tick-borne arboviruses have been isolated from it worldwide (Chastel 1988). Because *I. uriae* feeds on almost any available seabird host, including penguins, gulls, petrels, cormorants and shearwaters (Clifford 1979), it is reasonable to assume that its presence on a seabird is also likely to be associated with the presence of one or more of these parasites. Its life cycle has been summarised by Clifford (1979).

Colony abandonment by the sooty tern *Sturna fuscata* has been attributed to virus-infected ticks (Feare 1976). Though this has not been demonstrated in Antarctic birds, Gauthier-Clerc et al. (1998) attributed mortality in some king penguins, *Aptenodytes patagonicus,* on Crozet to hyperinfestation with *I. uriae.*

2.2.2.3 Mites

Mites are common inhabitants of nests of sub-Antarctic seabirds (Dunnet 1964). Many species of feather mites may be found, but at present only a few specimens have been collected (see Murray 1967; Murray et al. 1991). Host lists and biology of astigmatic feather mites from a number of Antarctic seabirds have been discussed by Atyeo and Peterson (1967, 1970). The nasal mite, *Rhinonyssus schelli,* has been recorded in the Adélie penguins, *Pygoscelis adeliae* (quoted in Watson 1975).

2.2.2.4 Lice

There are more species of Mallophaga biting lice in the Antarctic than of any other insect orders, and they, of all insects, are the most committed to parasitism (Askew 1972), with most species exhibiting high host-specificity. Clay and Moreby (1967) list 42 species, with their hosts and localities, in the Antarctic. A list of lice from 58 species of birds from sub-Antarctic islands is found in Clay and Moreby (1970). Overall, south polar birds are hosts to at least 60 species (Block 1984). A number of species of lice in the genus *Austrogoniodes* are found in the feathers of penguins (Clay 1967). However, despite heavy infestations on some birds, e.g. skuas, (Schaefer and Strandtmann 1971; Spellerberg 1971), these do not appear to be a cause of ill health and are not known to transmit infectious agents.

2.2.2.5 Fleas

The only species of flea known to occur on the Antarctic continent is *Glaciopsyllus antarcticus* (Murray et al. 1967; Murray 1967). *Parapsyllus* species of flea are found on many species of sub-Antarctic seabirds (Dunnet 1961, 1964; Murray et al. 1991). According to Dunnet (1964), the fleas of Antarctic and sub-Antarctic birds have evolved from stock parasitising birds in Australia and South America. Some species from each region have become circumpolar in their distribution whereas the distribution of others is restricted, and there is evidence of speciation of several of the species throughout their ranges. The Pygiopsyllidae is a predominantly Australian family although it occurs in Africa and South America. It is represented in the sub-Antarctic by the genus *Notiopsylla*, of which *N. kerguelensis* is circumpolar whereas *N. encirari* is found on sub-Antarctic islands in the New Zealand sub-region. The family of Rhopalopsyllidae has South American affinities, and is represented by the genus *Parapsyllus*. *Parapsyllus magellanicus heardi* is circumpolar and is found on a wide range of hosts whereas *P. cardinis* has only been found on Macquarie Island, but there may be speciation of this type throughout the islands of the New Zealand sub-region (Murray 1967).

There is evidence for the transmission of other micro-organisms (e.g. *Borrelia*; see below) by polar birds, but the paucity of evidence for the transmission of micro-organisms harmful to the birds themselves by these widespread and prevalent arthropods may be related to their ecological isolation, as well as to the lack of research on these groups. Ticks, mites, lice and fleas can be important vectors of protozoal, bacterial and viral agents in temperate and tropical regions. They could play an important role in the establishment, transmission and maintenance of disease should infectious arthropod-borne pathogens be introduced into Antarctic and sub-Antarctic ecosystems.

2.2.3 Endoparasites

Internal parasites are a ubiquitous, yet usually invisible, component of animal communities. There are many ways in which they influence the population dynamics of the host species (Dobson and Hudson 1986; Minchella and Scott 1991). Parasites may move through the food web, and can form a major component of the biomass of individual organisms. They place energetic demands on their hosts, and thus affect host nutritional status and host growth. They may also alter host behaviour and thus increase host susceptibility to predation, and increase host death rates and decrease host birth rates. Furthermore, parasites have been shown to alter the outcome of intra-specific and inter-specific competition, and they can influence mate choice and alter the sex ratio of the host population. The impact of parasites on Antarctic seabird populations is as yet unknown.

2.2.3.1 Helminths

The parasitic fauna of seabirds is vast and all species of seabird are natural hosts to a wide variety of metazoan endoparasites. Tapeworms (Cestoda), flukes (Trematoda), roundworms (Nematoda) and thorny-headed worms (Acanthocephala) are all well represented in Antarctic seabirds. The first descriptions of parasitic helminths from Antarctic birds (Leiper et al. 1914) were from birds collected on the British Antarctic Expedition of 1910–1913. This included the tapeworms, *Anomotaenia zederi,* from an emperor penguin *Aptenodytes forsteri*, *Tetrabothrius wrighti* from an Adélie penguin, *Tetrabothrius cylindraceus* from the south polar skua *Catharacta maccormicki*, and the nematode *Kathleena scotti* from the black-browed albatross. The following year Leiper et al. (1915) published a more comprehensive account, demonstrating the extensive nature of helminth parasitism in Antarctic vertebrates. However, most of these birds were collected opportunistically, often for food (see Shaughnessy 1990 for an account of birds collected from the drifting SY *Aurora* in 1915–1916), and their state of health was not commented upon. A report on parasitic worms recovered during the Discovery expeditions between 1925 and 1936 (Markowsky 1971) similarly made no mention of their effects on the hosts. In most cases, these parasites appear to be of no consequence to the bird. Massive parasitic infections are often encountered in wild, apparently healthy, birds as well as sick birds (Vogelnest 2000). Jones (1988) found 17 of 17 Antarctic petrels, *Thalassoica Antarctica,* heavily infected with tetrabothriid tapeworms. A filarioid nematode, *Eulimdana rauschorum*, was recovered from beneath the skin of nine of 21 kelp gulls, *Larus dominicanus,* at Anvers Island (Hoberg 1986). Hoberg (1986) suggested that infections were probably not acquired in the breeding grounds, but from arthropods in their more northerly wintering ground. Such a possibility could have implications if such arthropods were to extend their range to the Antarctic Peninsula. No filarial parasites were found in an additional 13 bird species from the same area. A checklist of helminths from Australian birds that includes many Antarctic seabirds has been produced by Mawson et al. (1986). This reference includes citations and readers are referred to this source for listings of species and hosts.

2.2.4 Protozoa

Miller et al. (1993) examined faecal samples from Adélie penguins (n = 44) and south polar skuas (n = 116) for coccidian parasites at Cape Bird, Ross Island. All the samples except one (taken from a breeding adult male skua) were negative for oocytes. The one positive sample had a heavy infection, as evidenced by tens of thousands of oocytes in the sample, suggesting that infection was not incidental. The genus and species of parasite were not determined, however the authors considered it to be a single species, the unsporulated oocytes of which did not match any coccidian oocytes described from birds in the literature.

Becker and Holloway (1968) failed to find any blood parasites in 11 snow petrels, *Pagodroma nivea*, 17 south polar skuas and one Wilson's storm petrel, *Oceanites oceanicus,* from McMurdo Sound and Hallett Station. Jones et al. (2002) did not detect any blood parasites in 125 south polar skuas from the Vestfold Hills, and Jones (1988) did not find any blood parasites in 143 penguins of four species (king penguins, *Aptenodytes patagonicus*, macaroni penguins, *Eudyptes chrysolophus*, royal penguins, *E. schlegeli* and gentoo penguins, *Pygoscelis papua*) on Macquarie Island. Laird (1961) attributed this reported lack of blood parasites to the absence of suitable vectors in the Antarctic. However, Peirce and Prince (1980) recorded a new species of hematozoan (*Hepatozoon albatrossi*, Eucoccida: Hepatozoidae) in the blood of three species of albatross (grey-headed albatross, *Diomedea chrysostoma*, wandering albatross, *D. exulans* and black-browed albatross) at South Georgia. They suggested that the vector of *H. albatrossi* was probably shared by penguins and albatrosses and was likely to be the tick *I. uriae* or a laelapid mite.

Although *Plasmodium* species are known to infect other species of seabird, there is only one report of this parasite in the Antarctic and sub-Antarctic seabirds: the brown skua, *Catharacta lonnbergi* (Bennett et al. 1993a; Vogelnest 2000).

A wide-ranging review of the literature on avian haematozoa (Bennett et al. 1993b) concluded that in their natural environments haematozoa cause little direct mortality to birds, though when concomitant infections with other disease agents occur, they may become pathogenic. The exception to this was when a new parasite is introduced to a naïve population or species, such as the introduction of malaria to Hawaii (Van Riper et al. 1988). Normally, the infrequency with which 'ill' birds are found in the wild indicates how much more difficult it would be to detect serious disease in oceanic birds away from their nesting areas.

2.2.5 Viral Diseases

2.2.5.1 General

Only a small number of viral diseases have been reported in seabirds. In most cases these have been identified based on serological evidence only and their significance is unknown (Vogelnest 2000; Wallensten et al. 2006). Most studies into viral diseases of Antarctic seabirds have focused on influenza A viruses, paramyxoviruses, birnaviruses and arboviruses.

2.2.5.2 Paramyxoviruses

Avian paramyxoviruses (APMV) are widespread among wild birds worldwide and consist of nine recognised serotypes. Strains of paramyxoviruses vary in virulence, i.e. in their capacity to cause clinical disease in infected birds, with the most

virulent strains being from the serotype AMPV –1, the causative agent of Newcastle disease (ND). This highly contagious, potentially devastating disease causes respiratory and nervous signs in domestic chickens.

APMVs have been isolated from penguins from the sub-Antarctic Macquarie Island and from Antarctica. Six isolates of APMV were obtained from five royal penguins and a king penguin at Macquarie Island (Morgan et al. 1981). Isolates of APMV from Adélie penguins have been obtained at various locations in Antarctica. Two isolates were obtained from penguins adjacent to Casey Station (Morgan and Westbury 1981), three from penguins from the Vestfold Hills (Morgan and Westbury 1988) and a further isolate from Cape Bird on Ross Island (Austin and Webster 1993). A single haemagglutinating virus isolate was obtained from Adélie penguin samples from Welch Island, near Mawson Station (Watts and Shellam unpublished data). This virus was classified as a paramyxovirus on the basis of its morphology under the electron microscope.

One APMV isolated from Macquarie Island was identified as a lentogenic ND virus (NDV) (Alexander et al. 1989). Antibodies to NDV have been found in Adélie penguins at the Béchervaise Island (3 of 73; Morgan and Westbury 1988), and Casey Station (2 of 285; Morgan and Westbury 1981), in Royal penguins on Macquarie Island (31 of 499; Morgan et al. 1978, 57 of 133; Miller and Shellam unpublished data), in King penguins on Macquarie Island (24 of 123; Miller and Shellam unpublished data), in sub-Antarctic skuas on Macquarie Island (2 of 149; Miller and Shellam unpublished data) and in south polar skuas in the Vestfold Hills (11 of 105; Miller et al. 2008). While a number of other studies have been unable to detect NDV antibodies in a variety of Antarctic seabird species (Morgan 1988; Morgan and Westbury 1988; Gauthier-Clerc et al. 2002), including an extensive study involving more than 1,000 Adélie and emperor penguins from various locations over a number of austral summers (Watts and Shellam unpublished data), it is clear that NDV occurs around Antarctica, but its distribution is patchy and unpredictable. Clinical Newcastle disease has been reported in a captive king penguin, and has also occurred in Adélie penguins following their capture from the wild (Pierson and Pfow 1975). However, outbreaks of clinical disease in wild birds are rare and there is no evidence of disease in Antarctic seabird populations.

Investigations of other isolated APMVs have found them to be unrelated to recognised avian paramyxovirus groups (Austin and Webster 1993; Watts and Shellam unpublished data). Antibodies to a number of these isolated viruses have been shown to be present in skuas and penguin species from different locations in the Antarctic and sub-Antarctic (Austin and Webster 1993; Morgan and Westbury 1981, 1988). Serological testing with the APMV isolated from Welch Island found specific antibody in 3% (21 of 639) of adult Adélie penguin sera tested (Watts and Shellam unpublished data). No antibody was detected in emperor penguin chicks or south polar skuas. Seropositive Adélie penguins were found over five austral summers and in various locations (Watts and Shellam unpublished data).

These results indicate that APMVs are widespread and endemic in Antarctic seabird populations. While the effect of these viruses on Antarctic seabirds has not been determined, paramyxoviruses have been isolated from birds in a wide variety of environments and are generally non-pathogenic.

2.2.5.3 Birnaviridae

In domestic poultry, the Birnavirus infectious bursal disease virus (IBDV) infects the bursa of Fabricius causing transient immunodeficiency in young birds. Clinically infection is associated with weight loss and a failure to thrive. The disease is not apparent in infected adult chickens (Lukert and Saif 1997).

In the first study of infection of Antarctic penguins with IBDV, Gardner et al. (1997) found antibodies (titres of 1:80 or more) to IBDV serotype 1 in 65.4% (34 of 52) of emperor penguin chicks and more than 2% (5 of 269) of Adélie penguin chicks from two colonies near Mawson Station on the Antarctic Continent. They found no antibodies (0 of 43) in Adélie penguin chicks from Edmonson Point, a more remote and rarely visited location on the Ross Sea. The authors postulated that human activity, including disposal of poultry products, might have been responsible. Although no disease was evident in the penguins, the presence of this virus could pose a threat should the birds come under other stresses.

As a result of that study, there have been additional attempts to find IBDV in Antarctic seabirds. Antibodies to both serotype 1 (12 of 30) and serotype 2 (2 of 104) IBDV were present in King penguins at Crozet (Gauthier-Clerc et al. 2002). Testing of emperor penguin chicks at Edmonson Point revealed 95.9% (47 of 49) of individuals with antibodies to IBDV serotype 1 (Watts et al. this volume). Similar high prevalence of antibodies to IBDV were found in emperor penguin chicks at Auster Rookery and Amanda Bay (29 of 31 and 17 of 17 respectively; Watts et al. this volume). Intensive sampling of Adélie penguins from widely dispersed breeding sites, including rarely visited remote sites, over five austral summers resulted in the detection of antibodies in 7.7% of adult birds and 0.8% of chicks with positive samples from all locations and time points (Watts et al. this volume.) These results contradict the notion of Gardner et al. (1997) that IBDV is less prevalent in more isolated colonies of penguins and indicate that IBDV is widespread in Antarctic penguin populations. Antibody to serotype 1 IBDV has also been detected in adult south polar skuas from the Vestfold Hills in three separate years of sampling, with a prevalence of 8–17% (Miller et al. 2008; Miller and Shellam unpublished data).

Infectious bursal disease virus has not been isolated from wild Antarctic seabirds, but it is known that IBDV can infect penguins in captivity. Gough et al. (2002) isolated an avian Birnavirus from captive African black-footed penguins, *Spheniscus demersus,* and macaroni penguins. Those isolates were later identified as IBDV serotype 2 (Jackwood et al. 2005).

2.2.5.4 Avian Influenza Virus

The presence of avian influenza (AI) virus in wild bird populations has become an important worldwide issue. The focus is primarily on understanding the ecology of the virus in wild populations and the risk posed to agriculture and human health (Clark and Hall 2006). Because waterbirds (including seabirds) are commonly

considered hosts, much of the effort to understand AI virus in wild populations has focussed on those species (Muzaffar et al. 2006).

Serological evidence of AI virus has been found in south polar skuas from the Ross Sea (Austin and Webster 1993) and Antarctic Peninsula (Baumeister et al. 2004); in Adélie penguins in Eastern Antarctica (Morgan and Westbury 1981), the Ross Sea (Austin and Webster 1993) and the Antarctic Peninsula (Baumeister et al. 2004); in gentoo penguins from South Georgia (Wallensten et al. 2006) and the South Shetland Islands (Baumeister et al. 2004); and in chinstrap penguins, *Pygoscelis antarctica,* and giant petrels from the South Shetland Islands (Baumeister et al. 2004). Antibody has also been found in occasional south polar skuas in the Vestfold Hills (Miller et al. 2008; Miller and Shellam unpublished data). These results from seabirds in different locations across many years indicate that AI virus is either endemic in the Antarctic or repeatedly introduced by migratory birds (Wallensten et al. 2006).

No evidence of clinical disease has been observed to date, although high mortality rates among Adélie penguin chicks were noted at one location where antibodies were detected (Morgan and Westbury 1981). Despite extensive isolation attempts, no AI viruses have been isolated from Antarctic seabirds (Wallensten et al. 2006).

2.2.5.5 Arboviruses (Arthropod-Borne Viruses)

A large group of viruses are transmitted by insects and other arthropods. These replicate in a blood-sucking arthropod (the virus vector) and are transmitted to a vertebrate host (e.g. seabird) when the arthropod takes a blood meal. The virus then replicates in the infected vertebrate and is transmitted to another arthropod when it feeds (Nuttall 1984). Despite intensive work on these viruses, many questions remain unanswered or poorly understood. These questions include the actual or potential pathological effects of tick-borne virus infections on the health of marine birds, especially that of young birds and on the population dynamics of avian species (Chastel 1988).

Flaviviruses have been isolated from *I. uriae* ticks found in close association with penguin colonies at Macquarie Island on a number of occasions. Doherty et al. (1975) isolated 16 strains of flavivirus, with the names Nugget and Taggert proposed for two newly identified viruses. Twenty-three flavivirus strains were isolated by Morgan et al. (1978), with three identified as Saumarez Reef virus, Taggert virus and Nugget virus. A further 33 isolated strains once again yielded Nugget and Taggert viruses along with two previously undescribed flaviviruses named Gadgets Gully and Precarious Point (St George et al. 1985).

With flaviviruses isolated from ticks found near penguin colonies, it is not surprising that flavivirus antibodies have been detected in penguins from Macquarie Island. Antibodies have been found in royal (9 of 384), king (7 of 218) and rockhopper *Eudyptes, chrysocome* (7 of 101) penguins (Morgan et al. 1981). There was no evidence of antibodies to Saumarez Reef virus in the royal penguin sera (0 of 42), but there were antibodies to Nugget virus (14 of 42) and Taggert virus (4 of 31) in royal penguin sera from Macquarie Island (Morgan 1988).

Flavivirus antibodies have also been detected in a brown skua (n = 30) from Macquarie Island (Morgan 1988) and south polar skuas (10 of 36 in 1999; 27 of 184 in 2001) in the Vestfold Hills, Antarctica (Miller 2008). The presence of antibodies in these migratory birds, together with the isolation of antigenically related flaviviruses from ticks in sub-Antarctic and sub-Arctic habitats (Doherty et al. 1975), suggests that these migratory seabirds carrying ticks may be responsible for the spread and introduction of arboviruses.

2.2.5.6 Adenoviruses (Egg Drop Syndrome 1976)

Egg Drop Syndrome 1976 (EDS 76) is caused by an avian adenovirus. Normally found in chickens and quail, it is characterised by a decrease in egg production and egg quality and therefore lowered breeding success. Antibody to EDS 76 was found in a small number of adult Adélie penguins (Watts and Shellam unpublished data). Antibody was detected in penguins from a number of locations and austral summers, suggesting that this virus is widespread and persistent in Adélie penguin populations. Further research is required to determine whether the virus is responsible for any reduction in breeding success in these birds. No antibody was detected in Adélie penguin chicks or emperor penguin chicks (Watts and Shellam unpublished data), or in south polar skuas from the Vestfold Hills (Miller unpublished data).

2.2.6 Bacterial and Fungal Diseases

2.2.6.1 General

It is rare that primary bacterial and fungal diseases are diagnosed in wild seabirds. These diseases are far more common in captive, particularly recently hospitalised seabirds (Vogelnest 2000).

2.2.6.2 *Chlamydophila*

Chlamydophila psittaci has been isolated from seabirds in the northern hemisphere (Haagen and Mauer 1938; Mykytowycz et al. 1955), mites parasitising seabirds (Terkikh et al. 1961) and serosurveys have found antibodies to *C. psittaci* and *Coxiella burnettii* in seabirds (Flint pers. comm. cited in Chastel et al. 1993). There are, however, no reports of Chlamydiales or Rickettsiales in Antarctic seabirds. A rickettsia-like micro-organism (‘Mayes’ agent) was isolated from *I. uriae* ticks collected from penguins breeding on Mayes Island, Kerguelen Archipelago, French sub-Antarctic Territories. The organism was not isolated from *I. uriae* ticks collected from a Kerguelen cormorant chick *Phalacrocorax verrucosus* or from a

black-browed albatross chick at Port-aux-Francais and Canyon des Sourcils Noirs in the Kerguelen Archipelago (Chastel et al. 1993).

2.2.6.3 *Pasteurella multocida*

Pasteurella multocida has been reported as the cause of acute death of four pairs of adult brown skuas at Litchfield Island in Palmer Station area (Parmelee et al. 1979). Few other details were given. The recent worldwide spread of avian cholera (caused by *P. multocida*) raises concerns for Antarctic birds, and is probably the major cause in the decrease of the yellow-nosed albatross on Amsterdam Island, where Erysipelas (caused by *Erysipelothrix rhusiopathidae*) was also diagnosed in two dead chicks (Weimerskirch 2004). *P. multocida* may be threatening the Amsterdam albatross, *Diomedea amsterdamensis,* and the sooty albatross, *Phoebetria fusca* on the same island with extinction (Weimerskirch 2004). Avian cholera has also been reported from the southern giant petrel, *Macronectes giganteus,* on King George Island, South Shetland (Leotta et al. 2003), and was the cause of death in 86 birds (kelp gulls *L. dominicanus*, skuas *Catharacta* sp., and Adélie penguins) in Hope Bay on the Antarctic Peninsula (Leotta et al. 2006). Furthermore, in late 2004, 500–700 chinstrap penguins died from an outbreak of avian cholera at Cooper Bay on South Georgia (South Georgia Government Report 2005). These reports of a fatal infectious disease affecting several species of birds at a number of Antarctic sites, while still an uncommon occurrence, indicates that the Antarctic is not isolated from epizootics.

2.2.6.4 Gastrointestinal Bacteria

A number of studies have reported gastrointestinal flora in Antarctic seabirds (Ekelof 1908; Bunt 1955; Gazert 1912; Harvey Pirie 1912; Levin 1899; McBee 1960; McLean 1919; Soucek and Mushin 1970; Sieburth 1959) and the gastrointestinal flora of penguins has been reviewed in Clarke and Kerry (1993, 2000).

Bunt (1955) isolated a number of different bacteria from the faeces of a range of Antarctic seabirds. Samples from a diving petrel contained the most diverse flora. No bacteria could be isolated from the faeces of a white-headed petrel, *Pterodroma lessonii,* and an Antarctic prion, *Pachyptila desolata*. However, there were recognised practical difficulties associated with unavoidably imperfect laboratory conditions resulting in the loss of many of the organisms originally isolated.

Sieburth (1959) presented gastrointestinal flora of eight Antarctic seabirds with scavenging and predatory habits. It was suggested that because most descriptions of bacteria from Antarctic birds are morphological and incomplete, it is almost impossible to compare bacterial types observed by the various workers.

Soucek and Mushin (1970) carried out an investigation of the aerobic gram-negative intestinal flora of 40 skuas (brown and south polar) and penguins from the Antarctic and sub-Antarctic. No birds were devoid of aerobic gram-negative

microflora; of 236 specimens examined, 65% were positive. The ratio of coliforms to paracolons and non-lactose-fermenting bacteria was 13:1. *Escherichia coli* was predominant. *Alcaligenes faecalis* and paracolons appeared in both species of skuas, and *Proteus vulgaris* appeared in south polar skuas.

Eighteen percent of faecal samples ($n = 16$) from south polar skuas collected at Ross Island by Oelke and Steiniger (1973) contained *Salmonella* sp.: *S. blockley* from two chicks at Cape Crozier, and *S. typhimurium* from one adult at Cape Royds. The significance of these findings is unknown.

However, 9 *Salmonella enterica* serovar Enteriditis, phage types PT4, PT8 and PT23, which are prevalent in humans and domesticated animals, were isolated from Adelie penguins at Cape Denison (Iveson et al. 2008), suggesting human impacts. That *Salmonella* serovars of human and animal origin are already in the food chain of Antarctic wildlife is suggested by the isolation of 20 such *Salmonellae* from fish meal processed in the Southern Ocean and South Pacific regions (Iveson et al. *ibid*).

In an extensive study on the natural bacterial flora of Antarctic seabirds, cloacal swabs from Adélie penguins and south polar skuas were used for culture of aerobic and anaerobic bacteria, including fastidious organisms, to establish normal flora (Watts, Miller and Shellam unpublished data). All penguin and south polar skua cloacal samples grew bacteria under both aerobic and anaerobic conditions. Bacterial isolates obtained were grouped according to morphological and basic biochemical characteristics. In all, 62 groups of aerobic bacteria and seven groups of anaerobes were isolated. The majority of isolates were gram-positive rods that were not identifiable by conventional phenotypic methods. Isolates that were identified included *E. coli*, *Enterococcus faecalis*, *Bacillus* sp., *Streptococcus* sp. and *Staphylococcus* sp., all of which are common commensals of many animal species.

Investigations of bacterial isolates from Antarctic penguins, principally Adélie penguins which were sampled near Mawson and Casey Stations, have revealed hitherto unknown species. Among aerobic isolates, *Corynebacteria* were very common, while among the anaerobes, members of the genera *Actinomyces* or *Clostridium* were well represented (Watts and Shellam unpublished data). No *Salmonella* sp. or *Campylobacter jejuni* were isolated from penguin or skua samples (Watts, Miller and Shellam unpublished data). These bacteria can be pathogenic in other avian species and if present in the Antarctic could have been introduced.

The gastrointestinal bacterium *Edwardsiella tarda* was recovered from cloacal swabs taken from royal, macaroni, gentoo, king, rockhopper and Adélie penguins from a number of Antarctic and sub-Antarctic locations (Macquarie Island, Heard Island, Kerguelen, Crozet and Cape Denison) during studies between 1982 and 1986 (Iveson et al. unpublished data). No pathology was noted in infected birds. No evidence of infection was found in two Antarctic prions, one cape petrel, *Daption capense,* and three common diving petrels.

The antibiotic resistance pattern of enteric bacterial isolates was also determined as a measure of possible human impact (Watts, Miller and Shellam unpublished data). No acquired antibiotic resistance was detected in any bacterial isolates from penguins or skuas although large numbers of antibiotic resistant bacteria were isolated from around the sewage outfalls at Antarctic stations. These results suggest

that Antarctic birds have not been colonised by bacteria of human origin despite their entry into the environment.

2.2.6.5 *Borrelia*

The presence of Lyme disease *Borrelia* spirochetes in *I. uriae* ticks from seabird colonies in both the southern and northern hemispheres (including black-browed albatrosses from Campbell Island, and in ticks collected from the Crozet Islands) demonstrates the significant role of seabirds in the global transmission cycle of *Borrelia*. DNA was isolated from *I. uriae* ticks and from cultured spirochetes. Sequence analysis of a conserved region of the flagellin (*fla*) gene revealed that the DNA obtained was from *B. garinii* regardless of geographical origin of the sample. Identical *fla* gene fragments in ticks obtained from different hemispheres indicated a transhemispheric exchange of Lyme disease spirochetes. A marine ecological niche and a marine epidemiological route for Lyme disease borreliae were proposed (Olsen et al. 1995).

The finding of *Borrelia* DNA in *I. uriae* ticks obtained from the Crozet Islands and Campbell Island suggest that Lyme disease enzootic foci are present in that part of the world. Olsen et al. (1995) concluded that the importance of seabirds in the global distribution of Lyme disease must be considered one of the major factors in the worldwide dispersal of pathogens causing this disease and warned that persons working with colonial seabirds may be exposed to Lyme disease. So far, the only evidence of infection with *Borrelia* is the presence of antibodies in king penguins on the Crozet archipelago (Gautier-Clerc et al. 1999).

2.2.6.6 Aspergillosis

Aspergillosis, caused by the fungus, *Aspergillus fumigatus,* is invariably a disease associated with seabirds in captivity (Vogelnest 2000). It is rare in wild birds. When it occurs the bird is often compromised for some other reason such as starvation or exhaustion. There are no published reports of aspergillosis in wild Antarctic seabirds although one south polar skua at Ross Island probably died from complications associated with aspergillosis (Miller unpublished data).

2.2.7 Other Diseases and Trauma

2.2.7.1 General

Other diseases of seabirds include trauma, foreign bodies, intoxication, oiling and misadventure. Their impact at a population level is not known. Other conditions such as plumage damage, cloacal impaction, decubital and tarsometatarsal ulcers, nutritional disorders and pododermatitis (bumblefoot) are primarily diseases associated with captivity (see Vogelnest 2000).

2.2.7.2 Toxicities

Toxicities in seabirds are rarely reported or diagnosed (Vogelnest 2000). A wide range of toxic compounds both natural and anthropogenic (pollutants) may be found in marine environments. These may affect seabirds by either contact or ingestion. Long-term exposure to pollutants may have serious consequences for seabird populations, and over time, a gradual decline in numbers may go unnoticed. Pollutant toxins include metals and metalloids (lead, cadmium, copper, nickel, zinc, mercury, selenium and arsenic), pesticides (organophosphates, carbamates and organochlorines), industrial chemicals (polychlorinated biphenyls, polybrominated biphenyls, dioxins and dibenzofurans) and petroleum products (aromatic hydrocarbons) (Fairbrother 1999a, b; Fairbrother et al. 1996). Polychlorinated biphenyls have been known in Antarctic wildlife for some time (Risebrough et al. 1968, 1976) and have increased over the years (Court et al. 1997). The levels of pollutants in skuas on Ross Island were not sufficient to be responsible for high chick mortality (Court et al. 1997), but we cannot be confident about the future. South polar skuas probably accumulate much of their burden of organochlorines outside the Antarctic ecosystem while on their migration (Bustnes et al. 2006), but at least some of the pollutants are the result of contamination of the food supply (Corsolini et al. 2003).

Natural toxins include those produced by the red tide organism, *Gonyaulax tamaensis*, and the botulinum toxin. There are no reports of acute intoxication in Antarctic seabirds, and the long-term impacts of these compounds is unknown.

2.3 Clinical Pathology

Few data are available on Antarctic seabird haematology and biochemistry (ISIS 2002). From the little that is known, it appears that the response of seabird blood cells and biochemical changes are similar to those seen in other bird species (Vogelnest 2000).

Marked leucocytosis occurs with inflammatory processes such as bacterial, fungal or protozoal infections, tissue trauma, stress and blood loss (bone marrow rebound). Bacterial infections are usually characterised by heterophilia and occasionally a lymphocytic response. Fungal infections also cause marked heterophilia, frequently lymphopenia and, when chronic, a monocytosis. The lymphopenia associated with these infections may be a primary response to the fungal infection or a response to increased cortisol levels due to stress or nutritional and metabolic aberrations that predispose birds to secondary fungal infections. Protozoal infections, particularly *Plasmodium*, result in marked lymphocytosis (Stoskopf and Kennedy-Stoskopf 1986).

No enzymatic studies of the tissues of seabirds have been carried out. Biochemical responses seen in seabird blood must therefore be interpreted based on what is known about other species. Normal total serum protein or plasma protein levels in

seabirds are low (3–5 g dL^{-1}) as is the case with most bird species. Hyperproteinemia is seen with dehydration and antibody production. Hypoproteinemia occurs with malnutrition, inanition, severe hepatic disease, parasitism, haemorrhage and chronic disease. Uric acid is the major catabolic product of purine and other amino acids in birds. Seabirds, being piscivorous, tend to have higher uric acid levels than birds on lower protein diets, and post-prandial elevations are common. Marked elevations are seen in birds with impaired renal function or gout. Significant tubular nephrosis must occur before elevations are seen. Uric acid is largely removed by tubular secretion, so dehydration only causes mild elevations. Urea levels are usually very low, but may be elevated with severe dehydration. Measuring creatinine is of limited use in assessing avian renal function. Aspartate aminotransferase (AST), although not liver specific, seems to increase with liver disease in seabirds as it does in other bird species. Elevations are also seen with soft tissue trauma. Bile acids are a sensitive indicator of liver function. Elevated levels indicate impaired liver function; however, food consumption may affect values. Currently, plasma bile acids are the only method to confirm hepatic disease in birds with elevated AST levels. Reference ranges for seabirds are not available and interpretation of results may be difficult. Glucose is the major circulating carbohydrate in birds. Although commonly measured it generally provides little diagnostic information. True hypoglycaemia is rarely seen except in severely ill birds. Birds have higher glucose levels than mammals and elevated levels may be seen with stress and post-prandial sampling. Diabetes mellitus has not been diagnosed in seabirds. It is not necessary to use fluoride oxalate to prevent glycolysis in avian blood. Avian erythrocytes consume very little glucose and it seems that they depend more on fatty acid metabolism. Creatine kinase (CK) is a specific and sensitive indicator of muscle damage in seabirds as it is in other bird species. CK levels fall more rapidly than AST, which is also elevated with muscle damage (Vogelnest 2000).

2.4 Discussion

Despite growing evidence that Antarctic seabirds are host to a large and diverse group of potential disease-causing agents, almost nothing is known about the impact and importance of disease in this avifauna, and reports of disease events in Antarctic seabirds are still uncommon. Nevertheless, birds may be carriers of various pathogens, such as different viruses, *Borrelia* sp., and enteropathogenic bacteria. Antarctic and sub-Antarctic birds may host a diverse range of arthropod ectoparasites and harbour heavy worm burdens, whose cumulative effects may produce more pathology than has hitherto been recognised; ‘parasites affect the life and death of practically every other living organism’ (Price 1980). However, with so many unknowns and insufficiently-tested theories, we can say little about the effect of parasites on their hosts when we know only the mean parasite burden or proportion of infected individuals (Toft 1991). An additional risk to Antarctic seabirds is the warming climate at high latitudes, and therefore the increased

possibility of the introduction of vectors or pathogens to immunologically naïve populations of birds. There is good evidence that climate change is already affecting seabird populations in Antarctica (Croxall et al. 2002), and that those effects due to climate change may derive from changes in vector-borne disease (Kovats et al. 2001). Ticks already occur around the sub-Antarctic islands and the Antarctic Peninsula (Wilson 1970; Zumpt 1952), but so far they have not been found at continental sites in east Antarctica (Miller and Watts unpublished data). The geographic ranges of some Arctic vectors such as ticks have expanded with the warming climate (Lindgren et al. 2000). With the climate becoming more suitable for arthropod ectoparasites, and the fact that seabirds fly long distances, and readily cross national and international borders, seabirds are possible long-range vectors for human infectious agents (Palmgren et al. 1997). It has also been concluded that infection and disease will be an important determinant of the health and wellbeing of animal populations, and as such must be considered in the design of conservation policy (Scott 1988). Furthermore, wildlife managers lack a scientifically sound basis from which to formulate management policy regarding many host–parasite interactions. One contributing factor is the paucity of research concerning the ecological consequences of host parasite interactions (Peterson 1991).

Clearly more research is required into infectious diseases of Antarctic seabirds, and baseline data should be acquired against which future changes can be measured. Nonetheless this research is difficult and costly. Accordingly, emphasis needs to be placed on clearly identifying and articulating the needs and priorities of research and management. Encouraging and enterprising research and policy initiatives are happening in wildlife health in Antarctica, however such activities, taking into account the unique wildlife, organisational structure and governance, could benefit from improved coordination, better commitment to collaboration and integration into existing national and international animal health management systems.

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Chapter 3
Diseases and Parasites of Antarctic and Sub-Antarctic Seals

R.A. McFarlane, R.J. de B. Norman, and H.I. Jones

3.1 Introduction

Antarctic phocids (or true seals) include the crabeater seal *Lobodon carcinophagus*, the Ross seal *Ommatophoca rossii*, leopard seal *Hydrurga leptonyx*, the Weddell seal *Leptonychotes weddellii* and the southern elephant seal *Mirounga leonina*. While the first three species breed and spend most of their lives in the pack-ice, the fourth breeds on the coastal fast-ice of the Antarctic and makes foraging trips from there that may extend into the pack-ice. Leopard seals also exploit continental penguin colonies in summer. The southern elephant seal breeds predominantly on sub-Antarctic islands (some breeding occurs on the Antarctic Peninsula and Valdes Peninsula, Argentina) and some bachelor males haul out to moult in summer on the Antarctic continent. All (although more rarely the Ross seal) have been recorded as vagrants from the southern continents.

All otarid (or eared) seals, including the Antarctic fur seals *Arctocephalus gazelle*, breed north of 65°S. Sub-Antarctic fur seals *A. tropicalis* breed on sub-Antarctic islands north of the Antarctic Convergence and other otarids may breed on sub-Antarctic islands (e.g. the New Zealand fur seal *A. forsteri*) as well as further north. Opportunities exist for interaction and disease transmission between the pinnipeds, although limited by ecological niches, behaviour, population homogeneity and density.

R.A. McFarlane
National Centre for Epidemiology and Population Health, Australian National University, ACT, Australia
e-mail: romcfarlane@bushlink.net.au

R.J. de B. Norman
Ministry for Agriculture and Fisheries, 4J/51 Webb Street, Wellington 6011, New Zealand
e-mail: richard.norman@maf.govt.nz

H.I. Jones
Microbiology and Immunology, School of Biomedical, Biomolecular and Chemical Sciences, University of Western Australia, WA, 6009, Australia
e-mail: hjones@cyllene.uwa.edu.au

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Information on diseases and parasites of marine mammals in Antarctica and the sub-Antarctic is sparse and we have a limited understanding of the role of disease in regulating wildlife populations in this region, as in other regions. Without an understanding of a species' age and sex-specific survival rates and the dynamics that regulates them, the significance of disease is very difficult to assess and unusual mortality difficult to identify. Of the Antarctic seals, only the southern elephant seal has been observed to undergo a recent decline in population size, with some populations declining by as much as 80% since the 1950s. Commercial harvesting had reduced numbers in the nineteenth and up until the mid-twentieth centuries, and a rebound response to population recovery is a possible contributing factor to these declines. Disease, although not discounted, is considered less likely a causal factor than inter-specific competition for prey or environmental changes affecting prey availability (McMahon et al. 2005) and has not been seriously examined. Antarctic and sub-Antarctic fur seals were almost made extinct by commercial sealing for their fur in the eighteenth and nineteenth centuries, leaving perhaps only a few hundred of them. Populations are continuing to recover and new sites are being colonised. This chapter endeavours to summarise available information on disease and parasites of Antarctic and sub-Antarctic seals, much of it recorded from clinically normal animals or apparently healthy, reproductively successful populations. Further innovative work is required to provide insight into the significance of these diseases or in the recognition of novel diseases.

3.2 Descriptions of Ill Health and Clinical Disease

3.2.1 General

Some descriptions of clinical disease and ill health in seals have been published, predominantly concerning the Weddell seal. Often, no attempt has been made to elucidate the cause of disease but in some cases there is a logical mechanism of injury or a specific investigation has been undertaken.

3.2.2 Trauma and Wounds

It is likely that intra-specific and inter-specific fight wounds account for some morbidity and mortality in Antarctic seals. In a 2-year study of the cause of death of 102 male Antarctic fur seals on South Georgia, Baker and McCann (1989) found that fight wounds were the major cause (58 and 82% in the 2 years of study). Although the external wounds appeared relatively minor, massive cellulitis and abscess formation was apparent on post-mortem. One such case is described in the sub-Antarctic fur seal on Amsterdam Island (Paulian 1964).

Male Weddell seals have a significantly higher mortality than females and this has been speculated to be due to fighting. Stirling (1971) and Lindsey (1937) record Weddell seal bulls 'bleeding to death' after fighting. Biochemical and haematological investigations at McMurdo Sound have demonstrated that adult male Weddell seals sampled in summer differ from all other groups for parameters associated with infection and inflammation (Yochem et al., this volume). McFarlane (this volume) demonstrates a positive association between wounds and other clinical diseases in Weddell seals. Bertram (1940) described fist-sized abscesses, both draining and non-draining, in the axillae and between tail and hind flukes of slaughtered Weddell seals some of which were low in body condition and blubber thickness. He also described a female seal with distal body paralysis and multiple draining suppurating abscesses, which could apparently still swim. Lugg (1966) describes large suppurating body wounds in Weddell seals due to marine predators 'similar to those seen in crabeater seals' (i.e. probably due to killer whale *Orcina orca* or leopard seal attacks). McFarlane (1996) describes multiple observations of a Weddell seal with a traumatically amputated hind fluke and three other Weddell seals with large, open wounds (approximately 200 × 200 × 40 mm) on their dorsums which were granulating and contracting without complication. King (1969) noted several scars of up to 35 cm in length in the skin of a male Ross seal.

Elephant seals of all age classes have been observed on shore at Macquarie Island (Carrick and Ingham 1962) with serious wounds. These are attributed primarily to attacks by killer whales and sleeper sharks, *Somniosis antarcticus* (Van den Hoff and Morrice 2007), which leave characteristic bite marks. Few animals with such wounds are observed, and it is inferred that few animals survive attacks by killer whales at least. Seals with bite wounds from lesser predators and wounds from intra-specific fighting are also observed.

Carrick and Ingham (1962) state that the elephant seal can recover from an extensive wound if it does not penetrate through the blubber and describe a wound on a 2-m long seal measuring 300 × 250 × 75 mm, attributed to a killer whale, that healed completely in 3 months. They state also that nearly all males that die in the breeding season are bachelors apparently in good condition without visible external injury and that fight wounds between evenly matched bulls rarely result in more than superficial injuries with the occasional loss of an eye. Occasional death of cows during mating occurs from crushing. Tierney (1977) describes injuries from inter-specific trauma as the most common and spectacular disorders observed in (predominantly bachelor male) southern elephant seals visiting the Vestfold Hills on the continent of Antarctica and notes the rapid infection of all wounds. These include lacerations, eye injuries, and sub-dermal abscesses. The cause of death in three recently deceased seals in this study was attributed to ice entrapment and drowning. Laws (1953a) describes minimal mortality from intra-specific fighting among adults despite extensive injuries. However, numerous deaths from ice entrapment at Signy Island and entrapment and drowning in evaporating mud wallows on the Falkland Islands have been described.

The presence of infection of other wounds such as branding burns, some unhealed 2 years later (Stirling 1971), and infection due to ill-fitting tags (McFarlane

1996) have been described in Weddell seals. The effect of hot iron branding on 5,000 of the 14,000 southern elephant seal pups branded on Macquarie Island has been examined, with 98% of brands healed by the first moult. Some (<1%) of healed brand wounds were seen to subsequently break down on a repeat observation. No difference in survival probability was found among seals with poor quality or good quality brands, or no brands (van den Hoff et al. 2004), but public concern over the appearance of some wounds has resulted in the cessation of this form of identification in study animals.

Osteological evidence has contributed to the knowledge of Antarctic seal disease and injury. King (1969) recorded erosive lesions that could be termed degenerative joint disease in the temperomandibular joint in two Ross seal skulls. Hamilton (1939) described the skull of a leopard seal with bite wounds evident, and a second similar but less conclusive skull. Cave and Bonner (1987) speculated that a leopard seal skull demonstrating marked asymmetry could have been caused by the animal being crushed between ice floes. Vagrant juvenile leopard seals dispersing to temperate waters strand regularly on the southern Australian coast. Reddacliff (1988a) described 5 of 13 stranded leopard seals on the New South Wales coast during 1978–1987 with 3–6 cm diameter skin wounds thought to be the result of squaloid shark attacks. These lesions, referred to as 'cookie cutter' shark bites, were also observed in one southern elephant seal and one of three New Zealand fur seals in the same case series. A leopard seal died at Phillip Island, Victoria, in July 1992 from peritonitis and a parasplenic abscess secondary to intestinal perforation by a stingray spine (Norman 1995). Alimentary tract accidents recorded in other *Arctocephalus* spp. include oesophageal perforation with secondary pericarditis in the Australian fur seal *A. pusillus doriferus* (Obendorf and Presidente 1978) and intestinal torsion recorded from zoo captives of the same species (Reddacliff 1988b). Rock slides were an unusual cause of deaths of adult and juvenile sub-Antarctic fur seals of Amsterdam Island reported by Paulian (1964). Predation by killer whales is highlighted as the greatest danger for sub-Antarctic fur seals on Amsterdam Island (Paulian 1964). Misadventure in the form of accidental entrapment in rabbit burrows was described for Hooker's sea lion pups (Marlow 1975), but asphyxiation of pups during adult male's forced copulation attempts was more frequently encountered.

3.2.3 Dental Disease

A contributing factor to the natural population regulation of Weddell seals appears to be excessive teeth wear and dental disease (Bertram 1940; Stirling 1969, 1971). Maintaining open breathing holes in the ice by sawing back and forwards with the horizontally projecting upper incisors and canine teeth is critical survival behaviour. Large numbers of skulls from slaughtered seals have been examined that demonstrate dental abscesses, the incidence increasing with age and increased tooth wear (Stirling 1969). Tooth root abscess, sinuses discharging from the external surface of the maxillae, osteomyelitis and fracture of the mandible subsequent to breakage

of a lower canine were also reported in Weddell seals by Bertram (1940), and the increase in periodontal disease with age has been described by McFarlane (this volume). Worn, loose and broken teeth with associated infection and even fracture of underlying bone occur in other individual seals including Antarctic fur seals (Baker and McCann 1989; Erb et al. 1996), leopard seal (Gray et al. this volume, Hamilton 1939; Junin and Castello 1995; Rounsevell and Pemberton 1994) and the southern elephant seal (Carrick and Ingham 1962). Erb et al. (1996) report observations of fully mature Antarctic fur seal males on Heard Island with damaged canines and incisors. Several live males were seen with orofacial lesions associated with osteomyelitis, and several dead seals with dental alveolar osteomyelitis without fractures. The authors suspected surf trauma, not fighting, to be the cause. Dental anomalies comprising variations in the number, size, shape or position of teeth may be of no pathological significance, but merit attention as disorders of development. Miles and Grigson (1990) catalogue minor dental anomalies in Antarctic pinnipeds, but more significantly describe an erosive lesion affecting the cervical enamel in sea lions including the southern sea lion and Hooker's sea lion.

3.2.4 Tumours

Neoplasia is reported in many wildlife species in the veterinary and medical literature. Tumour, neoplasia and cancer are often, but erroneously, used as synonyms. A tumour is any swelling, but if disordered growth of cells is involved in its pathogenesis, a tumour may also be termed neoplastic. Mawdesley-Thomas (1974) described seven neoplasms in seven different pinniped species. A single case was from a Southern Hemisphere species, a malignant granulose-cell neoplasm weighing over 2 kg in the left ovary of a southern elephant seal (Mawdesley-Thomas 1971, 1974). Other reports of tumours include two adult Weddell seals each with a single tumour-like mass on their flank, 10–12 cm high and 25–32 cm in diameter (Stirling 1971). It appears no tissues were collected for histological examination, so it is not possible to determine whether this report refers to similar neoplastic masses in two separate animals or perhaps the more likely situation that these are subcutaneous abscesses. An 11-ft (3.4 m) male southern elephant seal had a 68.5 lb (31.07 kg) tumour originating in a thoracic vertebra (Carrick and Ingham 1962). Gwynn (1953) records the results of two necropsies on leopard seals, one with a 'malignant tumour obstructing one bronchus' and the other with a carcinoma of the stomach. No histological details are available, and it is possible, at least in the second case, that a parasitic gastritis may have been over-interpreted.

3.2.5 Skin Diseases

Seal parapox has been confirmed in a single Weddell seal (Tryland et al. 2005) and is discussed below under viral diseases. Csordas cited in Carrick and Ingham (1962)

reports southern elephant seals with skin diseases including large areas (75 × 38 cm^2) of alopecia and loss of epidermis. These authors report that pups in crowded harems develop non-fatal skin disease. Tierney (1977) lists exfoliative dermatitis as a disorder observed in a female southern elephant seal in the Vestfold Hills. Laws (1953a) described, as a frequent observation, southern elephant seal cows hauling out to pup on South Georgia with 'a scattering of brilliant orange spots' about the nose, eyes, axillae, flippers and back that appeared to be fungal and 'dry(ied) up quickly after the seals are on land'. Barnacles are also described. Gwynn (1953) described a variety of skin lesions in leopard seals, including alopecia of the head and flippers. Bonner (1968) described and illustrated a partial alopecia in Antarctic fur seals from South Georgia which he termed 'mange,' noting a comparable condition referred to as 'rub' in the South African fur seal pelt trade.

3.2.6 Cardiovascular Disease

A range of gross and microscopic degenerative lesions have been observed in the coronary arteries and the aortic arch of Weddell seals and southern elephant seals, with aortitis, fatty streaking of the aorta and thrombotic occlusion of aortic vasa vasorum the most dramatic in the Weddell seal (Prathap et al. 1966).

3.2.7 Renal Disease

A 1.5-in. (38-mm) diameter kidney stone lodged in the ureter of an adult female Weddell seal was reported by Bertram (1940). Non-fatal interstitial nephritis was a common finding as a secondary lesion in adult male Antarctic fur seals (Baker and McCann 1989).

3.2.8 Respiratory Disease

Pneumonia was the second most common cause of death in adult male Antarctic fur seals (9–44%) and was characterised by polymorphonuclear infiltrations, necrosis, abscess formation and fibrinous exudative pleurisy (Baker and McCann 1989). Nematodes were only seen in the lung tissue of minor cases (Baker and McCann 1989). The German Weddell Sea Expeditions in the spring of 1986 and the summer of 1990 reported 'large numbers of Weddell seal adults and pups at Drescher Inlet coughing and bringing up phlegm with nostrils surrounded by whitish coloured foamy layers' without evidence of generalised ill-health or death (Harder et al. 1991). They speculated that these were clinical signs of respiratory disease associated with a phocine herpes virus (PHV) for which they had serological evidence.

McFarlane (1996) describes a solitary Weddell seal sub-adult in respiratory distress with an approximately 15-cm diameter mass of frothy material across its nose and mouth and purulent nasal discharge in a 1990/91 study of the Vestfold Hills population. Other seals were observed with purulent ocular or respiratory discharges or expectorating white foam suggestive of irritated or obstructed airways without observed dypsnoea. The prevalence, necropsy findings and causal associations of respiratory disease in this population are examined further by McFarlane (this volume). Gray et al. (this volume) describes Leopard seals from the same location with respiratory discharge, including foam with and without coughing. Ocular discharges are also described. Pathological nodules on the turbinals of the nasal passages are said to be common in leopard seals (King 1983), which could be the result of irritation from respiratory mites although these have not been described.

3.2.9 Reproductive Pathology

Post-partum genital discharges have been described in the Weddell seal by McFarlane (1996) but are uncommonly recorded in wild seals. Mansfield (1958) recorded several Weddell seal cows dying shortly post-partum without elucidation of cause of death, and Stirling (1971) also recorded a dead female in a lactating group and thin pups attempting to suckle other cows. Abortion and stillbirth have been recorded in the Weddell seal (Lugg 1966; Lindsey 1937; McFarlane 1996). Multiple uterine fibroids, the largest being 5 cm in diameter, in a non-pregnant Weddell seal with a large apparently functional corpus luteum were recorded by Bertram (1940). Pathology of an Antarctic fur seal cow following death due to dystokia has been described by Baker and Doidge (1984). Carrick and Ingham (1962) describe two southern elephant seal cows unable to return to the ocean after weaning their pups and five cows dying during prolonged labour/dystokia. Csordas (1966) reported malformation of the penis in five southern elephant seals.

3.2.10 Neonatal Death and Pup Ill Health

Abortions, stillbirth, starvation and exposure, drowning and crushing have been described as causes of mortality in Weddell seal pups (Lugg 1966; Lindsey 1937; McFarlane 1996; Stirling 1971). Further, debilitating cow–calf aggression and abandonment of underweight pups has been observed in Weddell seals (Lindsey 1937; Mansfield 1958). Quality of ice is also a factor, and Mansfield (1958) recorded a Weddell seal pup mortality of 30–50% following early break-up of spring ice. However, the greater social spacing of pack-ice and fast-ice seals contributes to a much lower perinatal mortality rate due to aggression and crushing than in densely populated pupping colonies of the Antarctic and sub-Antarctic otarid seal species (Doidge et al. 1984; Laws 1953a; Harcourt 1992).

Starvation is a major cause of neonatal death in southern elephant seals, and death due to trampling, crushing, drowning, stillbirth and, after weaning, molestation by bachelor bulls is less common (Carrick and Ingham 1962). Laws (1953b) has described conjoined twins in the southern elephant seal. Laws (1953a) estimated that first-year mortality in southern elephant seals was up to 50% but very few died on land, the majority being early in the season when melting snow in difficult terrain led to drowning, separation and starvation. At Signy Island, Laws (1953a) describes years in which pupping takes place on sea-ice and very high pup mortality associated with early break-up of sea-ice.

Pup mortality in Antarctic fur seals increased with colony density and was predominantly due to abandonment and starvation, skull injuries and crushing. Stillborn pups and those with septicaemia were occasionally seen (Doidge et al. 1984). Similar patterns and causes have been elucidated in the New Zealand fur seal (Mattlin 1978) and the South American fur seal (Harcourt 1992). Reid and Forcada (2005) contrasted starvation and trauma as causes of Antarctic fur seal pup deaths in South Georgia with an argument between the mothers' ability to provision pups due to reduced prey availability and incidence of injury due to colony density of occupation. Paulian (1964) noted storms as a cause of sub-Antarctic fur seal pup mortality on Amsterdam Island. Georges and Guinet (2000) reported stillbirth and starvation as the main causes of death of sub-Antarctic fur seal pups at the same location.

Juvenile and sub-adults of sub-Antarctic seal species appear on coasts of temperate countries as vagrants, live stranded or dead beach-washed animals. Anecdotal and incidental pathological findings in such animals may be of little value in determining population characteristics of health and disease in their populations of origin. Nosocomial disease and exposure to organisms to which a vagrant animal may be naive increase the likelihood that data derived from distant stranded animals may be spurious. A sub-Antarctic fur seal stranded at Phillip Island, Victoria, in August 1991, was in poor condition with the lens of one eye luxated, complicated by an anterior synechiae (Norman, unpublished observation). Presumably, the lesion compromising sight in one eye would have impaired the animal's foraging ability, predisposing it to stranding. During rehabilitation, it had an episode of watery and mucoid diarrhoea associated with unidentified spirochaetal bacteria.

3.3 Parasites

3.3.1 Arthropod Parasites

Much of the parasitological work reported here, especially for southern hemisphere otarid species, predates the host nomenclature revisions of Repenning et al. (1971) and of Warneke and Shaughnessy (1985). Consequently, the names attributed to the otarid host species before these dates should be treated cautiously. Some recent publications in seal parasitology have compounded or added to these errors (Norman 2005).

3.3.1.1 Lice

Several species of blood-sucking lice (Anoplura) occur on Antarctic and sub-Antarctic seals, particularly on pups. The blood-sucking louse *Antarctophthirus ogmorhini* was described from the Weddell seal by Murray et al. (1965), who also made observations on its ecology. This species also occurs on the leopard seal (King 1983). *A. microchir* has been recorded in all five genera of sea lion (Kim et al. 1975). *Lepidophthirus macrorhini*, described by Murray and Nicholls (1964), occurs on southern elephant seals at Macquarie Island, but has not been found on this host at the Antarctic continent (Tierney et al. 1977), and it appears that *L. macrorhini* is unable to survive lower host-surface temperatures or breed below 25°C. *A. lobodontis* is recorded on crabeater seals and *A. mawsoni* on Ross seals (King 1964). *Proechinophthirus zumpti* is reported from the South African fur seal, but no sucking lice are reported from other species of *Arctocephalus* (Kim et al. 1975; Warneke and Shaughnessy 1985).

None of these blood-sucking lice has been reported to cause morbidity or mortality, and their ability to transmit other pathogens is unknown.

3.3.1.2 Mites

Respiratory mites, *Halarachne* spp., are reported from Weddell seals and southern elephant seals (King 1964). Domrow (1962) considered the species of respiratory mite infecting both northern and southern elephant seals to be *H. miroungae*, on the basis of material from seals near the type locality in California (Ferris 1925) and from South Georgia. Laws (1953a) described them as small, white mites that attach to the mucosa of the nasal passages, where they may cause obstructive nodular lesions on the turbinates. They also occur in the trachea, bronchioles and even in the lungs, affecting respiration and causing pulmonary lesions (King 1983). Transmission occurs via the active larvae, which either crawl to the nostrils or are sneezed from infected seals. Larvae do not feed and will survive in moist environments until they reach the nostrils of a new host, probably attracted by the raised carbon dioxide gradients from exhaled air (Furman and Smith 1973).

Mites of the genus *Orthohalarachne* parasitise the respiratory tract of otarid seals and of the walrus *Odobenus rosmarus*. The two species of mite most commonly involved are *O. attenuata* and *O. diminuata*; the females of *O. attenuata* are up to 4 mm long with an elongate abdomen (Banks 1910), whereas *O. diminuata* is smaller, with a mean length of the female of 0.9 mm (Doetschman 1941). The two species are ecologically separated; Till (1954) reported that *O. attenuata* is found in the upper and *O. diminuata* in the lower respiratory tract. This was confirmed by Norman (unpublished observations), who showed from necropsies of adult and pup Australian fur seals in Victoria that *O. attenuata* occurred only in the nasopharynx attached to the nasal septum and that *O. diminuata* occurred only in the trachea and the bronchial tree.

Mites were reported from an Australian fur seal with the cephalothorax buried in the mucus membrane of the nasal passages and the nasopharynx, and with their

elongate abdomen protruding into the lumen of the nasal passage (Tubb 1937). The species reported by Tubb was described as *H. reflexa*, but this was subsequently shown to be synonymous with *O. attenuata* (Newell 1947). Seawright (1964) described pulmonary collapse, oedema and excess mucus secretion associated with the presence of mites in the trachea and bronchi of an Australian fur seal pup which died after capture. Seawright identified the mites as *O. attenuata*, but in view of the observations of Till (1954) and Norman (unpublished observations), they were probably *O. diminuata. O. attenuata* has also been reported from the Australian sea lion, but not from Hooker's sea lion *Neophoca hookeri* (Marlow 1975).

O. diminuata was found in the sub-Antarctic fur seal on Gough Island (Bester 1989), at a prevalence of more than 40% ($n = 201$), and *O. magellanica*, an elongate mite, in the nasopharynx of the southern sea lion (Finnegan 1934). A small number of other species of *Orthohalarachne* occur in seals in other parts of the world (Newell 1947).

Demodex zalophi was found in the hair follicles of captive California sea lions *Zalophus californianus* in North America and Australia, associated with alopecia (Dailey and Nutting 1980).

3.3.1.3 Crustacea

The whale copepod *Penella balaenoptera* has been reported on one occasion in a northern elephant seal *Mirounga angustirostris*, probably by the invasion of a pre-existing skin wound (Dailey et al. 2002). Superficial wounds in beach-washed dead seals on the Australian coast are sometimes found infested with sea lice (copepods; Argulidae) (Norman, unpublished observation).

Adventitious attachment to the skin by barnacles *Lepas australis* has been reported from the Antarctic fur seal and the southern elephant seal (Bonner 1968).

3.3.2 Helminths

Dailey and Brownwell (1972) have provided a useful list of helminth parasites of seals known at that time, but Delyamure's monograph (1955) remains the most comprehensive treatment of marine mammal helminths. Some of the species quoted in these studies have undergone subsequent taxonomic revision, and later studies must be consulted to follow the congruence of names of species. Anderson (2000) provides summaries of known nematode life cycles. Many helminth parasites utilise intermediate and paratenic hosts (a carrier host where no development or reproduction occurs), and immature and larval forms observed in fish are collated in the checklist of Beumer et al. (1983).

Research in helminth taxonomy utilizing recent biochemical and molecular techniques (e.g. Mattiucci et al. 2003; Sardella et al. 2005) has illustrated the complexities of host specificity and revealed cryptic species among helminths infecting marine mammals (Dailey 1975; Hoberg 1989).

Studies of endoparasites in the past have usually involved slaughter of the host. This practice has lost favour with many investigators and attention has turned to non-lethal methods and opportunistic sampling of seals which have died or been killed for other reasons. Although such sampling presents opportunities to investigate relationships between these hosts and their parasites, confounding factors such as concurrent illness or potential cross-infection in rehabilitation reduce the epidemiological value of the resulting data. Large sample sizes from episodic mortality events may also be biased by the factors contributing to death or recovery of the animal (see Lucas 1899; Stiles and Hassall 1899). Data derived from by-catch sampling and from culling operations (Bester 1989) may also have inherent selection bias, and without random sampling statistical validity may be questioned.

3.3.2.1 Cestodes (Tapeworms)

A key to the cestodes of Antarctic seals has been published (Wojciechowska and Zdzitowieski 1995). Delyamure (1955) lists hosts of cestodes, and Khalil et al. (1994) provide a key for identifying cestodes to the genus level. Host–parasite associations of cestodes, infection rates and the peculiarities of their locations in Antarctic phocids have been described by Jurachno (1989) and Jurachno and Maltsev (1994, 1997).

Adult cestodes of Antarctic seals belong to several genera in the family Diphyllobothriidae (Order Pseudophylliidea) (Bray et al. 1994). Representatives of the genus *Anophryocephalus* (Order Tetrabothriidae), which occur as adults in holarctic phocids and otarids, have not been found in Antarctic seals, although many species of *Tetrabothrius* are present in Antarctic whales and seabirds (Hoberg 1994).

Mass infestations of cestodes in the gastrointestinal tract are common in many seals but appear to cause little pathology (King 1983). Maltsev (1995) provides further data for the very high prevalence and intensity of cestodes in some Antarctic seals: 89.5% ($n = 67$) of leopard seals were infected with two species of cestode at a mean intensity of 1.8×10^5 worms; 100% of Weddell seals ($n = 28$) were infected with five species at a mean intensity of infection of 1.3×10^6 worms; and 100% ($n = 14$) of Ross seals were infected with four species at a mean intensity of 1.4×10^5 worms. These figures corroborate earlier findings; Johnston and Mawson (1953) reported heavy tapeworm infections in leopard seals, apparently without observable effect on their body condition. Markowski (1952) reported that Weddell seals and elephant seals are frequently and heavily infected with cestodes, though the crabeater seal is rarely infected. *Diphyllobothrium* spp. occurred at a prevalence of 16.6% ($n = 201$) in sub-Antarctic fur seals on Gough Island (Bester 1989). Laws (1953a), however, stated that Weddell and leopard seals are more heavily parasitised with helminths than southern elephant seals despite their less gregarious natures and that crabeater and Ross seals are the least heavily parasitised; probably both diet and behavioural ecology are relevant. The levels of infection of three species of cestode (*Diphyllobothrium lashleyi*, *D. mobile* and *Glandicephalus perfoliatus*), two species of trematode and one species of nematode in seven Weddell seals were

quantified by Beverley-Burton (1971). These observations reported by different workers indicate that infection with adult diphyllobothriid cestodes is often heavy and widespread in both Antarctic and sub-Antarctic species of seals, but that there is no direct evidence for ill-effects on the health of the hosts.

The dipyllobothriid tapeworm life cycle involves at least two hosts (a crustacean and a fish), and many pinnipeds share cestode species, though some cestode species are recorded only from a single seal host; *Baylisiella tecta* for instance is known only from the southern elephant seal, where it provokes a nodular reaction at the site of scolex attachment in the wall of the rectum, and Baylisia baylisiis known only from crabeater seals (Markowski 1952).

Larval cestodes are often recovered in the blubber of seals. Rennie and Read (1912) found cysticerci of *Phyllobothrium* spp. in the subcutaneous blubber of Weddell seals, leading them to suggest that the final hosts were predatory killer whales and various scavenging birds which might ingest them, and noting that *Phyllobothrium* sp. was normally found as an adult in sharks. Tapeworm scolices, identified as *Phyllobothrium delphini*, were found in the abdominal blubber of carcasses of southern elephant seals on Macquarie Island (Morgan et al. 1978), and *P. delphini* occurred at a prevalence of 95.5% in sub-Antarctic fur seals over 1 year of age on Gough Island (Bester 1989). Paulian (1964) noted that it was normal to find a large number of cysticerci in the subcutaneous fat of fur seals on Amsterdam Island.

Morgan et al. (1978) describe tapeworms of great length, which could be *Monorygma grimaldii*, encysted in the mesentery of an elephant seal; this species is more commonly found at this site in dolphins, and is thought to mature in sharks (Norman 1997). Bester (1989) records another aberrant occurrence of *M. grimaldii* encysted in the testis of an 8-year-old fur seal on Gough Island, and De Graaf et al. (1980) report cystic stages of the human cestode *Taenia solium* in the South African fur seal.

3.3.2.2 Trematodes (Flukes)

Jones et al. (2005) provide keys for the identification of some trematode species occurring in pinnipeds. Trematodes are usually found in the liver, gall bladder, bile and pancreatic ducts or small intestine of seals. Large burdens have usually been associated with inflammation and even death but low numbers appear to be well tolerated (King 1983). Numerous species are described from the Northern Hemisphere (Price 1932). Two species of trematode, *Ogmogaster antarcticus* and *Orthosplanchnus* sp., were identified and quantified from the Weddell seal by Beverley-Burton (1971). *Mesostephanus neophocae* was described from the intestine of the Australian sea lion (Dubois and Angel 1976), in which *Hadwenius* sp., *Galactosomum angelae* and *Stictodora diplacantha* were also present. The latter two genera are more commonly associated with avian hosts. *M. neophocae* was also recorded in a captive southern elephant seal in South Australia (Dubois and Angel 1976). However, Bester (1989) found no trematodes in a large sample of sub-Antarctic fur seals from Gough Island, and remarked that trematodes might be overlooked with the standard necropsy techniques limited to opening the gut lumen

and a superficial examination of viscera. It is thus likely that the prevalence of trematodes in Antarctic and sub-Antarctic seals may be higher than has hitherto been realised.

3.3.2.3 Nematodes (Roundworms)

Dollfus (1948) published a checklist of records of *Anisakis* species in marine mammals that provides a useful record of the early taxonomic literature. A number of nematode species have been reported from Antarctic and sub-Antarctic seals, and there have been many records of pathology caused by these infections. High gastrointestinal burdens of both nematodes and cestodes have been reported from Weddell seals (Bertram 1940; Beverley-Burton 1971; King 1983), and Dearborn (1965) states that heavy infections with nematodes and cestodes are normal for Weddell seals in the Antarctic. Beverley-Burton (1971), however, recorded only one species of nematode, *Contracaecum osculatum*, in a sample of seven Weddell seals; Johnstone et al. (1973) describe regurgitation of a mass of this species by a bull Weddell seal. Bester (1989) reported a prevalence of gastric nematodes of 41.3% ($n = 201$) in sub-Antarctic fur seals on Gough Island, comprising larval and adult *Anisakis simplex* and *C. osculatum*. *C. ogmorhini* was originally described from a leopard seal from South Australia (Johnston and Mawson 1941), and this species was redescribed by Fagerholm and Gibson (1987). *C. mirounga* was described from the southern elephant seal from Balleny Island (Nikolskii 1974). However, recent studies have demonstrated cryptic species complexes among the *Contracaecum* species of seals, and their nomenclature will require revision (Mattiucci et al. 2003).

Gastrointestinal nematodes can cause inflammation and ulcers of the stomach wall, which may lead to peritonitis and death (King 1983). McFarlane (2004) describes nodular hyperplasia and ulceration in the stomach and small intestines of a female Weddell seal heavily infected with both nematodes and cestodes. Gastric ulcers in southern elephant seals impacted with nematodes, predominantly *A. simplex* and *C. osculatum*, were reported by Morgan et al. (1978), and Cattan et al. (1976) also described gastric ulcers in the South American sea lion associated with *Anisakis* sp. infection.

Hookworms in the genus *Uncinaria* occur in southern elephant seals (see Dailey and Brownwell 1972), and nematodes in this genus are known to cause anaemia and death of northern fur seal pups, *Callorhinus ursinus*, when transmitted through milk from infected mothers (Keyes 1965). Hookworms occur in all five genera of sea lions: Steller's sea lion *Eumetopias jubata*, Californian sea lion *Zalophus californianus*, southern sea lion *Otaria byronia*, Australian sea lion *Neophoca cinerea* and Hooker's sea lion *Phocarctos hookeri* (Morgan et al. 2000). Hookworms have also been found in the Australian fur seal and in the New Zealand fur seal *Arctocephalus forsteri* in the Australian region (Norman 1995). The hookworm *Uncinaria hamiltoni* was very numerous in the intestines of six elephant seal pups from the Crozets, though only females were collected (Johnston and Mawson 1945).

Thus ascarid nematodes (*Contracaecum* spp. and *Anisakis* spp.) and hookworms (*Uncinaria* spp.) are the predominant gastrointestinal nematodes infecting seals; they may occur in very large numbers and can cause obstruction, ulceration and anaemia in their hosts.

Lungworms in the genus *Parafilaroides* have been reported from the leopard seal in South Australia (Mawson 1953) and in the Australian sea lion *N. cinerea* (Nicholson and Fanning 1981). *Parafilaroides* spp. are well-known lung parasites in sea lions in the north Pacific Ocean, and their life cycle involves a coprophagous fish as intermediate host (Dougherty and Herman 1947; Dailey 1970). There are records of *Parafilarioides* spp. in a number of seal species – Australian fur seal *A. pusillus doriferus* (Cape Woollamai and Ocean Grove, Victoria), leopard seal (Geelong, Victoria), sub-Antarctic fur seal *A. tropicalis* (Cape Town), and New Zealand fur seals *A. forsteri* from Taranaki Bight, New Zealand (Norman, unpublished observations). Lungworms are a common underlying cause of respiratory disease (verminous pneumonia) in many pinnipeds (Measures 2001), but their prevalence or significance to the health of sub-Antarctic and Antarctic seals remains unclear. The possibility that *Parafilaroides* spp. could act as vectors for Calciviruses of seals in the north Pacific Ocean was investigated by Smith et al. (1980), but no positive serological or viral isolates have been reported.

Filarial tissue and blood parasites have been reported from the southern elephant seal (King 1964); Mawson (1953) described a fragment of a filarial nematode from a blood vessel, and referred to it as *Filaria sensu lato*. Laws (1953a) described very large numbers of nematodes in the cardiac atria of a male elephant seal. Filarial worms have also been reported from the hooded seal *Cystophora cristata*, but their precise identification is uncertain (Cobbold 1879; Anderson 1959). *Dipetalonema* spp. and *Dirofilaria immitis* are well-known tissue and blood filarial parasites in northern phocid and otarid seals (Dunn and Wolke 1976). The ectoparasitic seal louse *Echinophthirus horridus* acts as the intermediate host for *D. spirocauda* (Geraci and St. Aubin 1987). Thus, although filarial nematodes occur in southern seals, their prevalence and significance remain uncertain.

3.3.2.4 Acanthocephala (Thorny-Headed Worms)

Acanthocephalans or thorny-headed worms are widespread in pinnipeds, although little is known of their effects (King 1983). They shed larvae whose intermediate hosts are usually amphipods or fish. Infected cystacanths encysted in the mesenteries of fish are ingested by seals, where the adult worms attach to the mucosa of the stomach or intestine with a retractable spinous proboscis. This provokes a localised inflammatory response, usually limited to the mucosa. Large numbers of individuals may be present, appearing as small, white-domed or comma-shaped nodules 5–10 mm in diameter, scattered over the surface of the mucosa of the infected viscous (Norman, unpublished observation).

Morphometric and allozyme electrophoretic analyses demonstrate very high levels of genetic diversity among acanthocephalans in marine mammals (Sardella et al. 2005). Nine species of acanthocephalan have been identified from a number of different species of seal from Antarctic and sub-Antarctic sites (Table 3.1), and there is no evidence for any marked degree of host specificity. It is possible that this taxonomy will have to be revised as molecular and other techniques are applied. None were found in Antarctic fur seals on Gough Island (Bester 1989). Studies by Aznar et al. (2004) on the localisation of *Corynosoma australe* in the intestine of South American fur seals in Uruguay revealed differences in density, maturity and reproductive stages at different levels of the intestine.

Thus there is still much to learn about the biology of these widespread and unusual worms and their effects, if any, on the health of their hosts.

Table 3.1 Acanthocephalans identified in Antarctic and sub-Antarctic seals

Acanthocephalan species	Host	Location	Reference
Corynosoma arctocephali	Crabeater Leopard Antarctic fur seal	South Georgia South Georgia	Zdzitowiecki (1984a, 1987)
C. bullosum	Crabeater, Weddell Southern elephant	South Shetland Island NZ sub-Antarctic Islands, Heard Island, Macquarie Island, Crozet Island, South Shetland Islands	Zdzitowiecki (1984a) Johnston and Edmonds (1953), Edmonds (1954, 1957), Zdzitowiecki (1984b)
C. evae	Leopard seal	South Shetland Island	Zdzitowiecki (1984a)
C. hamanni (C. antarcticum)	Crabeater Leopard Weddell Ross Weddell	South Shetland Island Commonwealth Bay	Zdzitowiecki (1984b) Johnston and Best (1937)
C. pseudohamanni	Crabeater Leopard Ross Weddell Southern elephant Antarctic fur seal	South Shetland Island	Zdzitowiecki (1984b)
C. semerne	Hookers sea lion A. australis Southern elephant	Argentina Argentina	Johnston and Edmonds (1953)
C. australe	Leopard Hookers sea lion Australian fur seal A. australis	Uruguay	Smales (1986) Aznar et al. (2004)
C. cetaceum			
Polymorphus arctocephali	Australian fur seal		Smales (1986)

3.3.2.5 Protozoa

A number of species of parasitic protozoa have been recorded from marine mammals, although there are no records of their effects on the health of the seal hosts in southern waters. Examination of faecal samples from Weddell seals in the South Shetland Islands revealed *Eimeria weddelli* (21.5%), *E. arctowski* (15.3%) and two other *Eimeria* spp. at lower prevalences. One *Eimeria* sp. was seen in the faeces of one in six crabeater seals, but no coccidians were seen in the faeces of either leopard or Antarctic fur seals (Drozda 1987). *Isospora miroungae* occurred in four in six southern elephant seals (Drozda 1987), and *Sarcocystis hydrurgae* was found in the leopard seal (Dubey and Odening 2001). Coccidian schizonts were found in the lamina propria of the intestine of a fur seal (Norman, unpublished observation). Other than a report of *Cystoisospora* provoking an enteritis in a South African fur seal (Kuttin and Kaller 1992), the only reports of pathology caused by protozoa are from Northern Hemisphere seals. Toxoplasmosis was identified as a cause of encephalitis in a northern elephant seal (Dubey et al. 2004). *Giardia* spp. have been recorded from several species of Arctic phocid species (Olson and Buret 2001), and antibodies to *Toxoplasma gondii* and *Neospora caninum* have been reported from several Arctic seals (Dubey et al. 2003). No haematozoa were seen in the bloods of 28 elephant seals examined at Macquarie Island (Laird 1952), perhaps due to the scarcity or absence of suitable arthropod vectors.

From the small number of studies reported to date on protozoa in southern seals, it cannot be ascertained with certainty whether pathology is caused; further studies are required to determine whether, and under what conditions, intestinal protozoa adversely affect their hosts.

Thus reports on ectoparasites and endoparasites from Antarctic and sub-Antarctic seals undertaken over a long time and in many locations have revealed the range of species of parasites that occurs, and often important aspects of their biology. From these studies, gastrointestinal nematodes appear to cause the most significant morbidity in these animals, though pathology caused by lungworms is also important. Further research, taking into account such factors as concurrent infections and other potential sources of stress, may reveal more ill effects on health than have hitherto been reported. In view of the many changes now taking place in the Antarctic and sub-Antarctic environment, systematic prospective studies are urgently required.

3.4 Laboratory Evidence of Disease Agents

3.4.1 Viral Diseases

3.4.1.1 General

A suspected viral epidemic caused an 85–97% mortality of approximately 3,000 crabeater seals in King Gustav Channel on the western Antarctic Peninsula in 1955 (Laws and Taylor 1957). The crabeater seals were of mixed age, their females aborted near-term foetuses, and they had non-verminous pneumonia and nephritis,

but neither the nearby Weddell seals nor sled dogs that were fed meat from these seals died or displayed illness. Bengtson et al. (1991) suggested that this epidemic might be attributed to the canine distemper virus (CDV), possibly introduced by sled dogs, and speculated on the role that viral infections may have in regulating Antarctic seal populations. No similar observation of mass mortality has been reported since in any species of Antarctic pinnipeds. Serological surveys have established the presence of some viruses, and some association with clinical disease been made. Unfortunately, a good summary of positive and negative results by species and location cannot be presented, and sample sizes are rarely large enough to give an indication of prevalence.

3.4.1.2 Morbilliviruses

Evidence for the presence of morbillivirus infection in crabeater and leopard seals has been demonstrated. Bengston et al. (1991) tested sera from 96 crabeater seals, 3 leopard seals and 5 Weddell seals collected in 1989 from three areas on the Antarctic Peninsula. Antibodies to CDV (or a closely related virus) were demonstrated in 35% of crabeater sera and in two of the leopard seal sera. No seals demonstrated antibodies to phocine distemper virus (PDV), and Weddell seals were negative to both. Osterhaus et al. (1988) found no evidence of CDV in Weddell seals ($n = 19$) either. Neither Weddell ($n = 25$) or crabeater seals ($n = 3$) sampled in the Weddell Sea in 1990 were positive to either CDV or PDV (Harder et al. 1991). Serological evidence for a morbillivirus was demonstrated in sera from 3 of 13 crabeater seals tested (in two of these animals CDV was suspected) in the pack-ice of Eastern Antarctica in 1997 (Lynch et al. 1999). Only 5 of 56 Weddell seals from the Vestfold Hills demonstrated low (equivocal) titres to PDV, and all were negative to CDV (McFarlane, this volume). However subsequent to this, 4 Weddell seals ($n=7$) at McMurdo Sound and 26 southern elephant seals at Macquarie Island ($n=192$) tested positive to PDV (R.Slade personal communication 2008).

Morbilliviruses require a critical population size of up to 400,000 individuals to be endemic, possibly explaining the positive findings in crabeater seals, the most numerous of the Antarctic phocids, and for which genetically distinct populations have not been demonstrated despite their circumpolar distribution (Davis et al. 2000). CDV is believed to be responsible for a mass mortality of Lake Baikal seals, *Phoca sibirica*, in 1987 (Grachev et al. 1989). Weakened seals crawled onto the ice and died, many with paralysed hind extremeties and opthalmitis. The authors had examined a seal with acute diarrhoea, opthalmitis and hind body convulsions, and had noted dogs dying nearby with typical signs of canine distemper in the same location the previous year.

PDV has displayed age and species differences in morbidity and mortality in Northern Hemisphere marine mammals following its emergence as the cause of the deaths of > 60% (18,000) of harbour seals, *Phoca vitulina*, in the late 1980s (Jensen et al. 2002) and killed 22,500 seals (again mainly harbour seals) in 2002/03 (Reineking 2003). Grey seals, *Halichoerus grypus*, suffered a relatively low mortality in both events. These epidemics may be explained by an initial introduction by harp

seals from the Atlantic, organochlorine pollution in European waters compromising immune function, the largely susceptible northern European seal population (initially naïve and subsequently with diminished antibody levels) and the migratory behaviour of harbour seals, which can travel hundreds of kilometres within days (Harwood and Grenfell 1990). Phocine distemper virus is endemic in the western Atlantic, and epidemics do not occur except in small, isolated populations of harbour seals (Duignan 2000). Infection is characterised by elevated body temperature, watery or haemorrhagic diarrhoea 3–6 days after infection, respiratory distress, cyanosis, ocular and nasal discharge, central nervous system disturbance such as head tremors, convulsions and seizures, and death. Weight loss with pressure necrosis of skin is observed when debilitated animals remain ashore, and abortion may occur in pregnant seals. Secondary parasitic as well as bacterial and viral infections are common. Transmission is horizontal, through respiratory discharges, and immunity is medium to long term (Duignan 1999). At-sea transmission does occur in the related porpoise morbillivirus infections of striped and bottlenose dolphins, so direct transmission could occur on ice or at breathing holes.

3.4.1.3 Herpes Viruses

Neutralising antibodies against known European phocine herpes virus (PHV) isolates were present in all 25 Weddell seals (some with high titres) and the three crabeater seals sampled at Dresher Inlet in the eastern Weddell Sea, and described above under respiratory disease (Harder et al. 1991; Stenvers et al. 1992). This is the first report of a herpes virus infection in Antarctic pinnipeds. These seals were negative to PDV and CDV. An unidentified herpes virus was observed by transmission electron microscopy in the liver of a sub-Antarctic fur seal which died with enteritis after stranding at Apollo Bay on the Victorian coast in September 1990 (Norman 1995). Lynch et al. (1999) demonstrated virus-neutralising antibodies to PHV in 1 of 13 crabeater seal samples in eastern Antarctica in 1997. Only 8 of 56 Weddell seals demonstrated low (equivocal) titres to PHV1, all were negative to PHV2 in the Vestfold Hills in 1999 and none demonstrated a positive association with respiratory disease (McFarlane, this volume). Of 7 Weddell seal samples from McMurdo, none were positive for PHV, but 62 southern elephant seals ($n = 192$) were positive for PHV on Macquarie Island, 11 of which were also positive to PDV (R.Slade personal communication 2008).

The clinical syndrome associated with phocine herpes virus 1 (PHV1) observed at the Dutch Seal Rehabilitation and Research Centre lasts 1–6 days and includes an elevated body temperature greater than 38°C, inflammation of the oral mucosa, nasal discharge, coughing, vomiting, diarrhea, anorexia and lethargy. Outbreaks have been associated with pupping, and the disease is potentially fatal in pups less than 1 month old, self limiting in pups 1–12 months old and mild or asymptomatic in sub-adults and adults (Harder 1997). Mortality with PHV1 occurs in neonates (up to 50%) and where there is concurrent acute phocine morbillivirus or immunosuppression (Osterhaus et al. 1988). No reliable information on the effect in wild populations of pinnipeds in the absence of morbilliviruses exists. Virus is shed in nasal and

ocular discharges in naturally and experimentally infected animals. Herpes viruses perpetuate in small populations (unlike morbilliviruses) owing to persisting latent infections in susceptible hosts that are periodically reactivated from latency, and the virus is often asymptomatically shed. Seropositive seals appear to be life-long carriers of virus and responsible for asymptomatic shedding (Harder et al. 1997). While terrestrial herpes virus infections may have been introduced by sled dogs or cats from the nearby Marion Island, the extensive movements of crabeater seals as well as the occasional visit to southern continents by all Antarctic pinnipeds represents an equally likely route for the virus (Harder et al. 1991). Phocine herpes virus 2 (PHV2) is a serologically distinct virus isolated from pinnipeds in North America and Europe but no disease has been associated with infection (Harder et al. 1996).

3.4.1.4 Avian Influenza Virus

Positive titres to influenza A virus were not present in sera from 237 Weddell seals although positive titres were found in Adélie penguins, *Pygoscelis adeliae*, and south polar skuas, *Catharacta maccormicki*, tested at the same time in Antarctica (Austin and Webster 1993). Negative titres to avian influenza have subsequently been measured in crabeater seals (Lynch et al. 1999, $n = 13$) and Weddell seals (McFarlane, this volume, $n = 56$) in East Antarctica. Virological examination of penguins undertaken in the Vestfold Hills and elsewhere in east Antarctica demonstrated the presence of influenza A and paramyxoviruses in the 1980s (Morgan and Westbury 1981, 1988) but not in 1999 (Gallagher et al. 2001). Birds are the primary source of avian influenza in infections of other species including marine mammals. Influenza A virus was first recognised as an infection of marine mammals in the 1980s in epizootics of harbour seals in North America in association with Mycoplasma respiratory infections (Geraci et al. 1982; Hinshaw et al. 1984; Lang et al. 1981). The affected seals developed frothy white (or bloody) respiratory discharge, dypsnoea, lethargy and incoordination, many with subcutaneous emphysema of the neck. Pneumonia was characterised by necrotising bronchitis, bronchiolitis, haemorrhagic alveitis, mixed aerobic and anaerobic infections and positive isolation of Mycoplasmas (Geraci et al. 1982).

3.4.1.5 Poxvirus

A solitary skin lesion approximately 3 cm in diameter with proliferative papilloma-like structures was found on the neck of a Weddell seal in Queen Maud Land in 2001 (Tryland et al. 2005). A seal parapox was identified, and is the first such report in an Antarctic pinniped. This virus rarely causes disease in multiple animals except where an underlying stress or disease is involved but can cause cutaneous lesions in people handling affected seals. Poxvirus was diagnosed in two juvenile South American sea lions imported to North America from Peru (Wilson and Poglayen-Neuwall 1971). The animals had been held for 4 weeks in the Netherlands during transhipment by the dealer providing the animals. The lesions were extensively

distributed over the animals' skin, which then pustulated, and both the animals died after episodes of enteritis. Confirmation of the poxvirus was by light and transmission electron microscopy.

3.4.1.6 Calicivirus

Although the San Miguel sea lion virus is a well-known agent isolated from the Californian sea lion *Zalophus californianus* (Sawyer 1976), testing in Southern Hemisphere seal species has been limited, and no positive serological or virus isolation reports have been published. Southern elephant seals on Macquarie Island were negative (Morgan et al. 1978). A small-scale investigation of Australian fur seals at Seal Rocks, Victoria, also in the 1970s, yielded no serological or virus isolation evidence of calicivirus (Norman 1995).

3.4.1.7 Arbovirus

A novel alphavirus has been isolated from the southern elephant seal louse *Lepidopthirus leonine*, and nearly all seals tested on Macquarie Island had neutralising antibodies against the virus. Although no virus-associated pathology has been identified, this represents the first report of an arbovirus in marine mammals and is most closely related to Australasian and African alphaviruses which utilise mammals as their favoured enzootic host (Linn et al. 2001).

3.4.2 Bacterial and Fungal Diseases

3.4.2.1 General

Baker and McCann (1989) report organisms isolated from lesions and normal tissue in their post-mortem examinations of 102 adult male Antarctic fur seals dying at South Georgia. *Streptococcus* and *Corynebacterium* spp. were the most common organisms isolated from fight wounds, and *Acinetobacter calco, Moraxella phenylpyruvica* and *Streptococcus* spp. (including *S. agalactiae, S. bovis, S. uberis, S. faecalis, S. morbillorum,* and *S. lactis*), *Bordetella bronchiseptica, Neisseria elongata*, yeasts and *Proteus* spp. were isolated from lung tissue in animals that died of pneumonia, the two most common causes of death in their study. No comparable study exists for other Antarctic species but some survey and necropsy bacteriology have been described for Weddell seals (McFarlane, this volume). Baker's extensive work (e.g. Baker 1984, 1987) on grey seals, *Haliochoerus grypus*, discusses in detail bacteriology and pathology of a range of primary and secondary lesions and is strongly recommended.

Scattered reports of microbiological isolates from dead, sick and healthy marine mammals often describe Northern Hemisphere species and refer to small numbers of captive or rehabilitating animals (e.g. Hicks et al. 2000; Higgins 2000). Captive Southern Hemisphere seal species have yielded some reports (Nakagaki et al. 2000; Oxley et al. 2004). The application of genetic tools to the investigation of microbial isolates has revealed apparently seal host-specific strains of organisms such as *Arcanobacterium phocae*, now recognised to be a common organism associated with infected wounds, pneumonia and septicaemia in North American and European seals (Johnson et al. 2003). Similarly novel *Streptococcus* spp. have been isolated from the same region (Lawson et al. 2004).

Fungal pathogens are rarely reported in seals (Wilson et al. 1974), and no reports derive from free-living Southern Hemisphere species. Dunn et al. (1984) refer to a report from Germany of candidiasis of the skin and mucus membranes of three captive southern elephant seals. Nakagaki et al. (2000) isolated *Malassezia pachydermatis* from a captive southern sea lion with dermatitis.

Some important livestock pathogens (or related strains) have received particular attention and are briefly discussed below. Fenwick et al. (2004) have suggested that *Salmonella* spp. cycled between feral pigs on the Auckland Islands and Hooker's sea lions.

3.4.2.2 Tuberculosis

Mycobacteria belonging to the tuberculosis group have been found in opportunistic post-mortem studies of dead and moribund wild otarids, including the Australian sea lion and New Zealand fur seal from Western Australia (Cousins et al. 1993), the Australian fur seal from Tasmania (Woods et al. 1995), the New Zealand fur seal in New Zealand (Hunter et al. 1998), the sub-Antarctic fur seal *Arctocephalus tropicalis* and the South American sea lion *Otaria flavescens* from South America (Bastida et al. 1999). Although there are no specific reports from Antarctic species, this highlights the potential for spread between continental, sub-Antarctic and Antarctic seal populations (Bernardelli et al. 1996; Duignan 2000; Liebana et al. 1996). There is also evidence of zoonotic potential (Thompson et al. 1993). The organism involved has now been shown to belong to the novel seal-specific species *Mycobacterium pinnipedii* (Cousins et al. 2003).

3.4.2.3 Brucellosis

Exposure to bacteria of the genus *Brucella* has been demonstrated in 6 of 17 Antarctic fur seals *Arctocephalus gazella* and a single Weddell seal in the South Shetland Islands in Antarctica (Retamal et al. 2000). In the Vestfold Hills, 97% of Weddell seals tested ($n = 56$) were seropositive to *Brucella* spp. (McFarlane, this volume). One of three vagrant captive leopards seals and one of 15 rehabilitated Australian fur seals *Arctocephalus pusillus doriferus* handled by Melbourne Zoo

demonstrated positive titres to *Brucella*, as did (9 of 12) wild Australian sea lions *Neophoca cinerea* from Kangaroo Island (Dawson 2005). New Zealand fur seal pups at a South Island colony (n = 101) did not test positive for *Brucella abortus* (Mackereth et al. 2005). These studies have used multiple diagnostic tests, including a competitive enzyme linked immunosorbant assay (cELISA), but Brucella organisms have not been cultured to date. There is serological evidence of Brucella infections in cetaceans from Peru (van Bressem et al. 2001).

In the Northern Hemisphere, positive titres to *Brucella* have been found in many species of marine mammals, e.g. 8% of harbour seals and 10% of grey seals (Jepson et al. 1997), 19% harbour seals, 3% Californian sea lions (Payeur et al. 1998), 35% hooded seal *Cystophora cristata,* 10% ringed seal *Phoca hispida,* 2% harp seal *Phoca groenlandica* (Tryland et al. 1999), 4% ringed seals, 12% walrus (Nielsen et al. 1996), using multiple diagnostic tests. *Brucella* organisms have been recovered from tissues of some seals and cetaceans and cultured and sequenced as distinct pinniped and cetacean strains (Cloeckaert et al. 2003; Foster et al. 1996; Jahans et al. 1997; Payeur et al. 1998; Tryland et al. 1999), but the clinical significance of the infection is still incompletely understood. Associated disease and/or reproductive pathology has been reported in cetaceans (Foster et al. 2002; Ohishi et al. 2003), direct contact and community acquired disease has been seen in humans (e.g. Brew et al. 1999; Sohn et al. 2003) and experimental infection has resulted in disease including abortion in livestock and experimental animals (Perrett et al. 2004; Rhyan et al. 2001). While the pinniped strain of *Brucella* appears to be widespread, we do not currently know the origins of the *Brucella* infection to which Antarctic fur seals and Weddell seals have demonstrated antibodies, nor whether some Antarctic species are naïve and hence susceptible to clinical disease.

3.4.2.4 Klebsiella

Klebsiella pneumoniae was a consistent isolate in a syndrome of pup mortality of Hooker's sea lions on the Auckland Islands in 2002. More than 30% of 126 pups necropsied died because of a primary systemic bacterial infection causing suppurative polyarthritis, necrotizing fasciitis, myositis, serositis and meningitis (Duignan et al. 2003). *Salmonella* spp. were also frequently isolated in this event.

3.4.2.5 Leptospirosis

Serological evidence of *Leptospira interrogans* serovars Canicola and Pomona and *Leptospira borgpetersenii* serovar Hardjo was found in unweaned pups of the New Zealand fur seal at a South Island, New Zealand, colony (Mackereth et al. 2005). *Leptospirosa interrogans pomona* is the cause of recurrent epidemics in Californian sea lions every 3–4 years since the 1970s (Gulland 1999) in a pattern suggestive of a link to the El Niño Southern Oscillation. The source and maintenance of this infection in seals is unknown but coexistence with livestock and visitation by vagrants are possible sources.

3.4.3 Toxicities, Pollution and Climate

Human-mediated impacts on Antarctic wildlife are considered more generally by Riddle (this volume). The following discussion relates specifically to Antarctic seals.

3.4.3.1 Local Anthropogenic Effects

The mass dying of crabeater seals reported by Laws and Taylor (1957) alerted science to the possibility of disease introduction to seals from non-native terrestrial carnivores, such as sledge dogs. Human sewerage has been considered a potential source of biological pollution to Antarctic seals, and other wildlife, and is discussed further by Smith and Riddle (this volume). Screening enteric bacteria of wildlife for evidence of genetic pollution from sewerage outfall was undertaken by Howington et al. (1993) at McMurdo Sound. In that study, coliform cultures from seals and penguins were exposed to a range of antibiotics to examine the possibility that human enteric bacteria with its high incidence of antibiotic-resistant flora may colonise the gut of animals in the vicinity of outfalls. No antibiotic resistance was demonstrated in that study.

3.4.3.2 Persistent Organic Pollutants (POPs)

The presence of chemical pollutants, chlordane, polychlorinated biphenyls (PCBs) and dichlorodiphenyltrichloroethane (DDT) residues in Weddell seal tissues at levels lower than in the industrialised Northern Hemisphere was established in the 1980s but has not been associated with pathology (Hidaka and Taksukawa 1981; Hidaka et al. 1984; Kawano et al. 1984). Levels of polychlorinated dibenzo-*p*-dioxins, dibenzofurans and coplanar polychlorinated biphenyls have been measured in blubber samples of Antarctic fur seals, and in crabeater seals, and again have been found to be much lower than Arctic species (Oehme et al. 1995). Levels of POPs in krill and Antarctic fish were found to be 1–2 orders of magnitude lower than those in fish of the Northern Hemisphere, and evidence of bioaccumulation in top-level predators, including Weddell seals and southern elephant seals, was demonstrated (Goerke et al. 2004; Focardi et al. 1995). This suggests that POPs are continuing to increase from low levels in the Antarctic as a result of global redistribution and the recent increases in uses in the Southern Hemisphere. POPs are suspected to have increased the susceptibility of seals in Europe to primary and secondary pathogens such as phocine distemper virus (de Swart et al. 1996), lowered fertility and reproductive success (Luckas et al. 1990; Reijinders 1986; Bergman and Olsson 1985), skull lesions and adrenal hyperplasia (Bergman and Olsson 1985; Bergman et al. 1992). Evidence of health effects of Arctic marine animals and traditional hunting communities associated with contamination levels has not been conclusive, but the expectation that contamination levels in the high latitudes

will continue to rise for several decades even if source pollution ceases is of considerable concern (Bard 1999).

3.4.3.3 Heavy Metals

Heavy-metal distribution in Antarctic and sub-Antarctic seals and other biota has been examined (e.g. de Moreno et al. 1997; McClurg 1984; Nodat et al. 1993; Szefer et al. 1993; Yamamoto et al. 1987). These have been studied predominantly in terms of physiological adaptations and as baseline data for global trends.

3.4.3.4 Plastic Debris

Plastic debris represents another potentially fatal source of marine pollution. A study conducted at Bird Island, South Georgia, in 1988/89 indicated that several thousand Antarctic fur seals were entangled in plastic debris, much of it from fishing vessels (Croxall et al. 1990), and the majority were expected to die from these entanglements. Despite active campaigning through international conventions, this problem appears to be persisting and an incidence of entanglement of 0.3–1.4% is estimated at this site (Arnould and Croxall 1995). Plastic debris accounted for 51–88% of all debris collected on Antarctic and sub-Antarctic islands (Derraik 2002) and therefore also represents a threat by entanglement or ingestion to other species.

3.4.3.5 Climate

Climatic periodicity expressed by the Antarctic Circumpolar Wave (White and Peterson 1996) and the El Niño Southern Oscillation increasingly has been recognised as having an important effect on the reproductive rate and pup survival of Antarctic seals (e.g. Burton 1998; Testa et al. 1991). It is also suspected to influence the occurrence of algal blooms and biotoxins potentially toxic to marine mammals in sub-Antarctic and temperate waters (Duignan 2000). Changes in the frequency or magnitude of these effects could have significant effects on the population dynamics and community associations of many species. Increased exposure to UV-B radiation following atmospheric ozone depletion has been shown to diminish primary production in Antarctic waters although the responses are complex (Hader et al. 1998).

While ozone depletion has stabilised, the effects of global warming are becoming more widely recognised. Associated with declines in winter sea-ice extent and duration, krill production has declined by up to 80% in the southwest Atlantic sector of the Southern Ocean in the last 30 years (Atkinson et al. 2004). As the backbone of Antarctic and many sub-Antarctic food chains, this has profound repercussions for all species in this region. In addition to effects on food resources, adaptation to sea-ice habitats is also being disrupted. The rapid warming around the Antarctic Peninsula and cooling around the Ross Sea have affected ice concentration in those

regions, and Weddell seals have declined in number or gone from sections of the Ross Sea where ice thickness has increased. It has been predicted (Siniff et al. 2006) that of the Antarctic species, the highly specialised crabeater seal may be the most affected by global warming as krill abundance and preferred ice flows for breeding decline. Beaches may become very important to leopard, southern elephant seals and Weddell seals as fast-ice and pack-ice declines. The effect of climate on the health and success of all species of Antarctic wildlife may become a major challenge to biodiversity in the Antarctic and sub-Antarctic as well as to science seeking to understand and predict these complex interactions.

3.4.3.6 Haematology and Serum Chemistry

There are several published accounts of the haematology and serum chemistry of Antarctic and sub-Antarctic species (Table 3.2), including references to other

Table 3.2 Haematological and seum chemistry parameters available from published literature for Antarctic and sub-Antarctic seals

Species	Reference	Parameters measured
Southern elephant seal	Engelhard et al. (2002)	Serum chemistry of cows and pups including the effects of human visitation (stress)
	Fayolle et al. (2000)	Lipid composition of blood platelets and erythrocytes
	Lane et al. (1972)	Haematology
	Ramadohr et al. (1998)	Lipoproteins during the breeding season
	Seal et al. (1971)	Serum chemistry and protein polymorhism
	Ferreira et al. (2005)	Serum immunoglobulin G, sexual and adrenal steroids
Weddell seals	Seal et al. (1971)	Serum chemistry and protein polymorhism
	Schumacher et al. (1992)	Serum biochemistry including lipids, enzymes, serum proteins, thyroid hormones
	Margini et al. (1972)	Serum proteins, lipoproteins, glycoproteins
	Meiselman et al. (1992)	Haemorheological behaviour of blood
	Yochem (this volume)	Haematology and biochemistry
	McFarlane (this volume)	Haematology and biochemistry
Crabeater seals	Seal et al. (1971)	Serum chemistry and protein polymorhism
Leopard seal	Brown (1957)	Haematology
	Williams and Bryden (1993)	Haematology and biochemistry
	Gray et al. (2005)	Serum proteins
	Gray et al. (this volume)	Haematology and biochemistry
Antarctic fur seal	Fayolle et al. (2000)	Lipid composition of blood platelets and erythrocytes
	Baker and McCann (1989)	Serum proteins
Antarctic, sub-Antarctic phocid and otarids species	Clark (2004)	Haematology

accounts in this volume (Yochem et al., McFarlane et al. Gray et al. and all this volume). Most of these papers are primarily concerned with physiological responses rather than health and disease.

3.5 Discussion

While the information presented above is not exhaustive, it does represent an overview of the state of our knowledge on diseases of Antarctic and sub-Antarctic seals. A greater volume of knowledge exists for wild pinnipeds from temperate and northern regions and this should be consulted in any disease investigation. Much useful information has been assembled above by collating the anecdotal findings and incidental reports of numerous researchers. However, this also highlights the lack of coordinated baseline health information for most species, on the lack of information on causation (for observed clinical disease) or affect (of agent of interest in serological surveys) or on the standard epidemiological data for almost all diseases discussed. There has been little research to date examining the role disease may have in the declining southern elephant seal populations or into the unique features of population-level and multi-species disease of this marine system.

While metazoan parasites lack the charisma of their hosts, they are a powerful agency in linking levels of the trophic pyramid which potentially yields information on prey distribution and evolutionary relationships. Patterns of parasitism mapped out through research on North American and European pinniped host species have been applied to less accessible Southern Hemisphere species. Pathogenic microbes may use metazoan parasites as vectors or reservoirs. Biodiversity among helminths of Antarctic seals requires further exploration through the wider application of molecular genetic techniques to the known morphological forms. Host–parasite relationships are not static, and cyclic changes in their significance to maternal investment strategies, prey selection, impacts of changing climatic patterns and multifactorial disease causation are yet to be elucidated. Resourcing of curation of collections of parasites of Antarctic seals, and the accompanying host biological data, is an important practical issue for facilitating investigation of long-term phenomena.

An increased rate of epidemics and disease in marine species has been highlighted elsewhere (Harvell et al. 1999), and the Antarctic is unlikely to be isolated from this effect despite its apparently protected status to date. Understanding the continuum extending between ecological associations of seals and their normal commensal and parasitic flora and fauna and the observable disease states they may provoke is pivotal in designing surveillance and investigative programmes. Mass mortalities may be missed in this remote region because of the isolation of events, but trends in health and reproductive status should not. Interdisciplinary research involving biologists, ecologists, climatologists, oceanographers and other experts with animal health specialists is essential if significant advances in our understanding of the drivers of health and disease of Antarctic and sub-Antarctic seals and other wildlife are to be made.

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Chapter 4
Infectious Bursal Disease Virus and Antarctic Birds

J.M. Watts, G.D. Miller, and G.R. Shellam

4.1 Introduction

The geographic isolation of the Antarctic continent coupled with the extreme climate has historically been assumed to protect the indigenous Antarctic wildlife from exposure to infectious agents found among animals in more temperate regions. However, with the number of tourists visiting the region more than doubling in the last 10 years (IAATO 2004) and climate change predicted to enhance the success of alien micro-organisms (Frenot et al. 2005), the threat of introduced disease to Antarctic wildlife has become a concern (Kerry and Clarke 1995; Kerry et al. 1999). Emergence of disease in naïve bird populations can reduce both population abundance and geographical distribution (Friend et al. 2001).

This concern is highlighted by recent evidence of introduction of infectious organisms to isolated wildlife populations in the sub-Antarctic and southern Indian Ocean. The isolation of *Salmonella* serotypes normally associated with human activity from penguins, fur seals and albatross on sub-Antarctic Bird Island (Olsen et al. 1996; Palmgren et al. 2000) and *Campylobacter jejuni* subsp. *jejuni* with high genetic similarity to Northern Hemisphere strains from macaroni penguins *Eudyptes chrysolophus* at the same location (Broman et al. 2000) suggests recent introduction of these bacteria. The introduction of avian cholera, caused by *Pasteurella multocida,* to Amsterdam Island is thought to be a major cause of population decline in the now-endangered large yellow-nosed albatross *Diomedea chlororhynchos* (Weimerskirch 2004). Avian cholera has also been implicated as the cause of death in rockhopper penguins *Eudyptes chrysocome* on sub-Antarctic Campbell Island (de Lisle et al. 1990); brown skuas *Catharacta lönnbergi* on Litchfield Island, Antarctica (Parmelee et al. 1979); and a single southern giant petrel *Macronectes giganteus* on King George Island, Antarctica (Leotta et al. 2003).

J.M. Watts, G.D. Miller, and G.R. Shellam
Microbiology and Immunology, School of Biomedical, Biomolecular and Chemical Sciences, University of Western Australia, WA 6009, Australia
e-mail: djawatts@bigpond.net.au; gshellam@cyllene.uwa.edu.au

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However, the identification of introduced disease is difficult without baseline information on infectious disease and organisms native in a population. While both Antarctic and sub-Antarctic penguins in captivity are susceptible to a range of pathogenic avian diseases (Clarke and Kerry 1993; Penrith et al. 1996; Fielding 2000), to date very little is known of the disease status or the impact of disease on free-living Antarctic penguin populations (Clarke and Kerry 1993; Kerry and Clarke 1995). There is serological evidence of exposure of free-living penguins to potentially pathogenic avian paramyxo- and influenza viruses (Morgan and Westbury 1981,1988; Austin and Webster 1993), and *Chlamydia* species (Moore and Cameron 1969) and *Salmonella* serotypes have been isolated from Adélie penguins, *Pygoscelis adeliae*, on Ross Island (Oelke and Steiniger 1973) although no associated disease has been described. There are two instances in which an infectious agent has been suspected as responsible for an observed mass mortality in Antarctic penguins in the wild. Symptoms resembling those of the viral disease puffinosis were observed in gentoo penguins, *Pygoscelis papua*, on Signy Island where several hundred chicks died, although no infectious agent was isolated (MacDonald and Conroy 1971), and large numbers of apparently well-nourished Adélie penguin chicks were found dead at a colony near Mawson Station in 1972 (Kerry et al. 1996). Approximately 65% of chicks were recently dead and many surviving chicks were ataxic and unable to stand. The cause of these deaths remains unknown.

In 1997 Gardner et al. reported the presence of antibodies to a pathogenic poultry virus, infectious bursal disease virus (IBDV), in two Antarctic penguin species breeding in the vicinity of the Australian Antarctic research station of Mawson (67°36′S, 62°52′E). High-titre neutralising antibodies to serotype 1 virus were detected in up to 2.6% of 133 adult Adélie penguins from two breeding colonies and 65.4% of 52 emperor penguin chicks (*Aptenodytes forsteri*) from a single breeding site in the 1995/96 summer. Antibody was also found in 1.5% (n = 136) of adult Adélie penguins from the same area in 1991. The absence of antibodies in 26 adult and 17 chick Adélie penguins from a remote colony (74°21′S, 165°03′E) in the Ross Sea area of Antarctica led these authors to suggest that the virus may have been introduced into penguin populations in the Mawson region by human activity. A possible mechanism of introduction proposed was the inappropriate disposal of chicken products, allowing infection of migratory scavenging birds such as the south polar skua *Catharacta maccormicki* and subsequent transmission to penguin colonies (Gardner et al. 1997).

IBDV was identified in 1962 (Winterfield et al.) as the causative agent of a newly described, acute, highly infectious disease of chickens (Cosgrove 1962). The virus has subsequently been found to be ubiquitous within the poultry industry worldwide (Leong et al. 2000). IBDV is a non-enveloped virus with a bi-segmented double-stranded RNA genome of approximately 6,300 base pairs (Kibenge et al. 1988) and is classified as a member of the family Birnaviridae, genus *Avibirnavirus*. The Birnaviridae family also contains viruses in other genera which infect fish (infectious pancreatic necrosis virus, *Aquabirnavirus*), bivalve molluscs (tellina and oyster virus, *Aquabirnavirus*) and insects (*Drosophila* X virus, *Entomobirnavirus*)

but there is no serological cross-reaction between these and avian birnaviruses (Kibenge et al. 1988). Two serotypes of IBDV are recognised, designated serotype 1 and 2, and are differentiated by the virus neutralisation assay (McFerran et al. 1980), a specific and sensitive functional assay that utilises antibodies to inhibit virus infection of cells. IBDV has been isolated from chickens, ducks and turkeys, although clinical disease has been described only in chickens and is associated exclusively with serotype 1 strains (Lukert and Saif 1997). Serological studies have shown that antibodies to serotype 2 IBDV are widespread in turkeys, chickens and ducks although all strains isolated are non-pathogenic (Lukert and Saif 1997).

IBDV is a very stable virus that is highly resistant to disinfection and adverse environmental conditions (Kibenge et al. 1988). It can persist for months in poultry pens, feed and faeces (Lukert and Saif 1997). Once introduced to a susceptible flock, transmission is via the faecal–oral route, with virus excreted in the faeces for up to 14 days post infection (Fenner et al. 1987). There is no known vertical transmission via eggs or carrier state in recovered birds (Lukert and Saif 1997).

The most striking feature of virulent IBDV infection in chickens is the selective targeting of lymphoid tissue resulting in B lymphocyte depletion, particularly in the bursa of Fabricius. Infection in young chicks (0–3 weeks) is subclinical but results in severe immunosuppression, leaving the birds highly susceptible to secondary infection with a wide range of avian pathogens (Sharma et al. 2000). Acute disease is seen in 3–8-week-old chicks, the time of maximal bursal development, and commonly results in 100% morbidity and up to 30% mortality. Clinical signs include diarrhoea, anorexia, trembling and prostration (Lukert and Saif 1997). Infection in older chickens is generally subclinical; however, a broader age range of susceptibility to acute disease and increased mortality rates has been seen with the emergence of very virulent IBDV strains (vvIBDV) (van den Berg 2000). Phylogenetic studies have shown that vvIBDV strains belong to the same genetic lineage as the classical serotype 1 virus, although mortality rates following infection with vvIBDV can reach 100% (van den Berg 2000). The high mortality and morbidity along with worldwide distribution has led the Office International des Epizooties (OIE) to consider infectious bursal disease (IBD) a disease of considerable socio-economic importance (van den Berg 2000).

Although IBDV has been isolated only from chickens, turkeys and ducks, there is now accumulating serological evidence of widespread infection in wild bird populations. Antibody to IBDV has been detected by the agar gel immunodiffusion (AGID) test in a number of free-living wild bird species in Nigeria (Nawathe et al. 1978) and Western Australia (Wilcox et al. 1983). Common eiders *Somateria mollissima* and herring gulls *Larus argentatus* in the Baltic Sea, along with spectacled eiders *Somateria fischeri* in Alaska, displayed neutralising antibody to serotype 1 IBDV (Hollmen et al. 2000), while virus-neutralising antibody against both serotype 1 and 2 viruses has been found in 14 species of wild birds in Japan (Ogawa et al. 1998) and king penguins, *Aptenodytes patagonicus*, from sub-Antarctic Possession Island (Gauthier-Clerc et al. 2002). Most of these studies sampled birds at only a single time point in each location; however, antibody was found to persist across years in king penguins breeding on Possession Island (Gauthier-Clerc et al. 2002).

With the exception of the isolated spectacled eider colony in Alaska (Hollmen et al. 2000), exposure of the studied populations or closely associated scavenging birds to poultry or poultry waste was thought to be a possible source of infection in each case.

4.2 Case Study

The aim of this study was to investigate IBDV in Antarctic birds by establishing both the prevalence and persistence of the virus in populations of emperor penguins, Adélie penguins and migratory south polar skuas breeding in regions around three Antarctic research stations: the Australian stations of Mawson and Davis (68°34′S, 77°58′E) in East Antarctica and the Italian station Terra Nova Bay (74°41′S, 164°07′E) in the Ross Sea. Blood samples were collected from birds over five summers (Table 4.1). Adult Adélie penguins and south polar skuas were sampled across the breeding season, from November to February. Chicks of both species were sampled in February when 2–3 months of age. Adélie penguin samples were obtained from breeding colonies within a maximum of 80 km of each research station. All south polar skuas sampled were breeding in the vicinity of Adélie penguin colonies in the Vestfold Hills region surrounding Davis Station. Emperor penguin chick sera were obtained at three different colonies: Auster Rookery (50 km east of Mawson Station), Amanda Bay Rookery (90 km south west

Table 4.1 Serum samples collected from Antarctic bird species

Species	Age	Location	Sampling season	Number
Emperor penguin (*Aptenodytes forsteri*)	Chick	Auster	1996/97	31
		Amanda Bay	1997/98	17
		Cape Washington	2000/01	49
				Total 97
Adélie penguin (*Pygoscelis adeliae*)	Adult	Mawson coast	1996/97	127
			1997/98	66
		Davis coast	1997/98	369
			1999/00	186
			2001/02	40
		Terra Nova Bay	2000/01	84
				Total 872
	Chick	Mawson coast	1996/97	44
			2000/01	24
		Davis coast	2001/02	56
				Total 124
South polar skua (*Catharacta maccormicki*)	Adult	Vestfold Hills, Davis	1999/00	118
			2001/02	128
				Total 246
	Chick	Vestfold Hills, Davis	2001/02	56

of Davis) and Cape Washington Rookery (40 km east of Terra Nova Bay). Birds were bled in November/December of each year when approximately 4–5 months old.

Antibody titres to IBDV serotype 1 in serum were measured using a standard virus neutralisation test (Westbury and Fahey 1993). IBDV serotype 1 strain GT101 was used as antigen. Antibody titres are expressed as the reciprocal of the highest dilution that completely inhibited virus. Antibody titres of ≥16 were regarded as positive, with titres >64 deemed highly significant (Giambrone 1980). Results were compared using the chi-square test with statistical significance determined as $P < 0.05$.

Sera from 1,395 birds were tested, with 189 (13.5%) exhibiting neutralising antibodies to serotype 1 IBDV. Results of the virus neutralisation tests for each species are summarised in Table 4.2. The seroprevalence in adult Adélie penguins was 7.7%. There was no significant difference in prevalence between locations or between years at each location (data not shown). Highly significant titres were recorded in 1.8% of birds, with a maximum neutralising antibody titre of 512 and a geometric mean titre of 57.6. A single Adélie chick (prevalence 0.8%) possessed IBDV antibody with a titre of 16.

Neutralising antibody was detected in a total of 11.8% of adult south polar skuas in the Vestfold Hills region but there was a significant difference ($P < 0.05$) in prevalence between sampling periods. In the 1999/2000 summer, 16.9% of birds were antibody positive with only 7% prevalence in 2001/02. This difference was reflected in the antibody titres, with a maximum titre of 256 in 1999/2000 and highly significant titres in 9.3% of birds. In 2001/02, a maximum titre of 80 was recorded with highly significant titres in only 3.1%. All south polar skua chicks tested in 2001/02 were negative for antibody, although it should be noted that chicks were tested only in a year of low prevalence and antibody titres in adults.

A very different pattern was found in emperor penguin chicks, with an overall seroprevalence of 95.9%. There was no significant difference in prevalence between each of the three locations, with 93.5, 100 and 95.9% recorded at Auster, Amanda Bay and Cape Washington, respectively. Antibody titres were higher than in the other two species of birds (Table 4.2), with highly significant titres detected in 100% of chicks at Amanda Bay, 85.7% at Cape Washington and 67.7% at Auster,

Table 4.2 Prevalence of IBDV serotype 1 antibodies in Antarctic bird species. Antibody titres ≥ 16 positive

Species	Age	Number tested	Number positive	Prevalence (%)	Max. titre	Geometric mean titre
Emperor	Chick	97	93	95.9	10,240	1,757.4
Adélie	Adult	872	67	7.7	512	57.6
	Chick	124	1	0.8	16	16.0
South Polar skua	Adult	246	29	11.8	256	68.3
	Chick	56	0	0	0	0

and a maximum titre of 10,240. Results at Auster are consistent with the 65.4% prevalence (titres ≥ 80) recorded previously at this location (Gardner et al. 1997).

The detection of IBDV RNA in emperor penguin chick tissue samples was attempted by reverse transcriptase polymerase chain reaction (RT PCR). Bursa of Fabricius, spleen, kidney, liver and intestinal tissue were extracted from 23 emperor penguin chick carcasses collected frozen off the ice at Auster Rookery throughout the 2000 winter. While the ages of chicks collected are unknown, the weights of the carcasses ranged from 302 g to 1.5 kg, indicating that all chicks were below the creching age of 45–50 days (Williams 1995). A nested RT PCR was performed as described by Liu et al. (1998), using primers flanking the hypervariable region of the VP2 structural protein of IBDV between nucleotides 703 and 1,193. Bursae from IBDV type 1 vaccine strain V877 infected chickens was used as a positive control. Products of the predicted size were amplified from three emperor penguin bursae samples. All other organ samples were RT PCR negative. On sequencing, the RT PCR products from each penguin were found to be identical at the nucleotide level both to each other and to the positive control strain V877, a classical Australian strain of low pathogenicity used as a live vaccine in Australia, Europe and Asia (Firth 1974; Proffitt et al. 1999).

Isolation of IBDV from RT PCR positive penguin bursae samples was attempted in specific pathogen free (SPF) chicks. Intraocular inoculation of 3–4-week-old and 1-day-old chicks was performed with bursal homogenates prepared as described by Westbury and Fahey (1993). Three chicks from each group were killed, and the bursae examined on days 3, 5, 7, 14 and 21 post infection. Throughout the experiments no evidence of clinical disease was seen in inoculated SPF chicks. All bursae appeared normal, displaying no signs of atrophy, and there was no significant difference in bursal to body weight ratios between infected and control chicks. RT PCR on all bursal samples was negative. Serum was also extracted from day 14 and 21 chicks, and virus neutralisation tests performed as previously described. IBDV neutralising antibody was not detected in any of the SPF chicks.

4.3 Discussion

The presence of neutralising antibody in all three species of birds, from all locations and time points, indicates that IBDV serotype 1 is endemic and widespread in Antarctic bird populations. The large distances between the sampling sites (Terra Nova Bay is approximately 4,500 km via the coast from Davis, the closest Australian station) suggest that the virus would not spread readily between these sites by either direct contact between penguins or movement of migratory birds. It is therefore unlikely that IBDV serotype 1 has been introduced into Antarctic bird populations by human activity in the Mawson Station region. Detection of neutralising antibodies in king penguins at Possession Island (Gauthier-Clerc et al. 2002) suggests that the virus may also be endemic in sub-Antarctic bird populations, although rockhopper penguins in Argentina (Karesh et al. 1999) and southern giant

petrels from Patagonia (Uhart et al. 2003), two species which frequent the sub-Antarctic, were tested negative for IBDV antibodies by AGID. AGID is less sensitive than virus neutralisation, however (Weisman and Hitchner 1978); so further investigation of sub-Antarctic birds is required.

The generally low titres of neutralising antibody in Adélie penguin and south polar skua adults are similar to titres reported in other adult wild bird species. A maximum titre of 128 was recorded in common and spectacled eiders (Hollmen et al. 2000), 256 in king penguins (Gauthier-Clerc et al. 2002) and 269 in wild birds from Japan (Ogawa et al. 1998). While the prevalence recorded in adult birds varies widely between studies, the results in Adélie penguins compare well with those previously recorded in this species, where a high-titre antibody (≥80) was found in 1.5–2.6% of birds (Gardner et al. 1997), and with the 4% prevalence in adult king penguins (Gauthier-Clerc et al. 2002).

The high prevalence and titre of antibody recorded in emperor penguin chicks is greater than reported in any other wild bird species. Up to 96% prevalence was recorded in common eider adults but high-titre antibody (≥128) was seen in only 15% of these birds (Hollmen et al. 2000). Two studies have investigated chicks of other bird species; in the closely related king penguin, only 3% of 3-month-old and 5% of the 1-year-old chicks were positive with a maximum titre of 64 (Gauthier-Clerc et al. 2002), while 45% prevalence was seen in herring gull chicks from the Baltic Sea (Hollmen et al. 2000). Interestingly, rapid transmission of virus through entire flocks of domestic chickens resulting in seroconversion of 100% of birds and very high antibody titres is a common pattern following field exposure to IBDV (Lukert and Saif 1997).

The almost complete absence of neutralising antibody in Adélie and south polar skua chicks is in stark contrast with results recorded in emperor penguin chicks and suggests a very different epidemiology among these species. While neutralising antibody results indicate that Adélie penguin and south polar skua adults are exposed to IBDV during their life cycle, the lack of antibody in chicks from both species suggests that infection of adults occurs remote from breeding sites with no subsequent transmission to chicks. There are a number of possible routes of infection in each species. South polar skuas are known predators of weakened emperor penguin chicks (Williams 1995), which have a high seroprevalence of IBDV serotype 1, and may also have contact with infected birds or material during their annual northern migration. Adélie penguins are also occasional visitors to emperor penguin colonies, and while adult penguins generally spend the winter non-breeding months dispersed within the Antarctic pack-ice, they are occasional vagrants to South America, Australia, New Zealand and sub-Antarctic islands (Williams 1995) where exposure may occur.

The high antibody prevalence in emperor penguin chicks indicates that infection occurs each year at the breeding sites. While IBDV can survive for extended times in the environment (Kibenge et al. 1988), emperor penguins breed on sea-ice which often melts during summer, and so persistent contamination of breeding sites is not possible. Introduction of virus from an outside source into each breeding colony every year is unlikely, especially since migratory birds are absent during winter

when chicks are young. These results therefore suggest that emperor penguins are carriers of IBDV. As virus transmission in domestic chickens is via the faecal–oral route, emperor chicks are most likely infected by contaminated faecal material while brooding on the feet of adults or drinking snow. Vertical transmission through the egg cannot be excluded. The possibility of species of wild birds acting as carriers or reservoirs and playing a role in the epidemiology of IBDV has previously been suggested. Detection of antibodies in migratory birds led to the speculation that they may have been responsible for the introduction and spread of very virulent IBDV among domestic chickens in Japan (Ogawa et al. 1998), whereas scavenging herring gulls are the suspected source of infection in eider ducks in the Baltic Sea (Hollmen et al. 2000). Persistence of virus in spectacled eiders was suggested following the detection of antibody in 70% of adults in a remote Alaskan colony where recent introduction of virus from poultry waste or migratory birds was unlikely (Hollmen et al. 2000).

While the presence of neutralising antibody implies exposure to the virus, it does not necessarily indicate the presence of disease. IBDV serotype 1 strains differ markedly in virulence in chickens and, although all strains appear non-pathogenic in turkeys and ducks (Lukert and Saif 1997), very little is known of the effect on wild bird species. The presence of IBDV neutralising antibody in common and spectacled eiders raises concern as the populations of both species are in decline with low duckling survival rates a primary cause, particularly in the Baltic Sea common eider colonies where IBDV antibody prevalence in adult birds was highest (Hollmen et al. 2000). The presence of IBD with a pathology similar to that seen in chickens would directly affect chick and fledgling survival. However, no evidence of immunosuppression was found in herring gull chicks following sheep red blood cell immunisation, despite increasing antibody titres indicating active IBDV infection (Hollmen et al. 2000). The lack of any observed clinical symptoms along with the detection of a classical Australian low pathogenic strain of IBDV in emperor penguin bursal tissue suggests that IBDV does not cause disease in Antarctic bird species.

All Australian IBDV strains isolated are genetically distinct from strains isolated in other parts of the world (Proffitt et al. 1999; Ignjatovic and Sapats 2002). The presence of a classical Australian strain of IBDV serotype 1 in emperor penguin chick samples therefore suggests a common evolution or source of Australian and Antarctic IBDV strains, although this has yet to be confirmed. The detection of IBDV RNA in bursal samples by RT PCR but a failure to isolate the virus in SPF chicks is not unusual, as IBDV RNA persists in bursal tissue from domestic chickens for substantially longer than infectious virus (Abdel-Alim and Saif 2001); however, 100% nucleotide similarity with the positive control virus means that contamination of samples cannot be conclusively ruled out.

These results contribute to the understanding of the health and survival of Antarctic penguins by establishing that IBDV serotype 1 is endemic in Antarctic bird populations and has not been introduced by recent human activity. However, as the threat to the Antarctic wildlife is amplified through increasing human activity and environmental change, this study also highlights the need for further information

on the endemic microflora of Antarctic birds and surveillance to allow the identification of introduced disease events. This is also the first study to demonstrate persistence of IBDV neutralising antibody in wild bird populations across both geographic location and time and to establish the role of a species as a carrier of the virus. Further work is now required to fully understand the epidemiology of IBDV in emperor penguins, with the prevalence of antibodies in adult emperor penguins to be determined, a virus isolate obtained and mode of transmission examined.

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Chapter 5
An Unusual Mortality Event Among Adélie Penguins in the Vicinity of Mawson Station, Antarctica

K. R. Kerry, L. Irvine, A. Beggs, and J. Watts

5.1 Introduction

An unusual mortality event among adult Adélie penguins was observed during November 2001 at breeding colonies in the vicinity of the Australian Antarctic Station of Mawson (67°36′17″S, 62° 52′15″E). The circumstances surrounding this event originally suggested infectious disease as the cause. Although this turned out to be not the case, investigations proceeded as if it were.

This case study sets out the field and clinical investigations undertaken and examines the likely cause of death. Reference is made to steps taken by the Australian Antarctic Division to be better prepared for and respond to any future unusual mortality event among Antarctica's bird and seal populations.

5.2 Penguin Colonies in the Mawson Region

Adélie penguins breed on the near-shore islands along the coast to the east and west of Mawson (Fig. 5.1). A colony of Adélie penguins at Béchervaise Island 3 km west of Mawson has been the subject of a long-term research and monitoring program since the summer of 1990/91 (Kerry et al. 1993) as part of the Commission for the Conservation of Antarctic Marine Living Resources (CCAMLR) Ecosystem Monitoring Program (CEMP) (CCAMLR 2004). Studies on Béchervaise Island are conducted annually over the entire breeding season. Related studies are conducted

K.R. Kerry, L. Irvine, and A. Beggs
Australian Antarctic Division, Channel Highway, Kingston, TAS 7050, Australia:
e-mail: knowles.kerry@keypoint.com.au, Lyn.Irvine@cwr.org.au

J. Watts
Microbiology and Immunology, School of Biomedical, Biomolecular and Chemical Sciences, University of Western Australia, WA 6009, Australia
e-mail: djawatts@bigpond.net.au

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Fig. 5.1 Map of the coastline in the vicinity of Mawson Station showing the locations of Adélie penguin colonies and Auster Rookery, the emperor penguin colony

on other nearby islands including Welch Island (Fig. 5.1). Occasional visits are made to colonies further afield. Knowledge gained from these activities became important for the understanding of the mortality event described below.

Adélie penguins arrive at their breeding colony in the second half of October at a time when the sea-ice extends 200 km or more to the north. Egg laying occurs in November, with a peak between the 19th and 25th. Hatching occurs from approximately 20th December. The full breeding season at Mawson has been described by Kerry et al. (1993).

5.3 Description of Mortality Event

The breeding season of 2001/02 commenced and was progressing normally. However, on 23 November, 10 adult penguins were found dead in a group on the sea-ice close to Kirton Island 40 km east of Mawson. Some had died recently and were still warm. Two days later, on 25 November, a large number of penguins were found dead on Welch Island 6 km northeast of Mawson. The following day, 89 dead and 6 obviously ill birds were counted. The penguin colony on Welch Island is large containing 15,000 pairs. The dead birds, however, were found grouped at the shore and up the broad valley and into the lower edge of the colony some 150 m distant.

Dead birds were not found on the nearby islands of Klung, Peterson, Verner or Béchervaise.

Surveys carried out in the following weeks revealed additional groupings of dead birds. Overall, 148 dead adult Adélie penguins were found along 80 km of coast line at locations identified in Fig. 5.1. The dead birds were all found close to breeding colonies in discrete areas interspersed with areas of unaffected birds. Most dead birds were located in the rafted sea-ice at the land–sea-ice interface.

Dead adult penguins are found sporadically through the early part of the breeding season. However, the finding of groups of dead penguins during egg laying was in our experience unusual, and as such made it of sufficient interest to warrant a detailed investigation. The circumstances surrounding these deaths initially suggested disease as a possible cause and investigations proceeded on this assumption.

At the time of the event, Mawson Station was isolated from the outside world and investigations were conducted by the resident ornithologist (L. Irvine) with the assistance of the Station medical officer (A. Beggs) with additional support as required from the staff at Mawson Station. Investigations were carried out and samples collected according to the protocols established by CCAMLR (see Appendix A, this volume). Sampling equipment was limited to materials at Mawson and some medical supplies. Equipment for microbiological investigations was not available.

Unless part of the investigation team, the personnel at Mawson Station were requested not to visit any of the penguin colonies for the remainder of the breeding season (to March 2002). Strict precautions were taken to protect the investigators from possible infection and to avoid possible spread of infectious disease to other colonies. Those in charge of the investigation wore clean clothing and boots. Vehicles were restricted to travelling on the sea-ice and vehicle treads were washed clean and sanitised with bleach (dilute sodium hypochlorite) before and after visiting each penguin colony.

5.4 Investigations at Welch Island

Although dead penguins were found at a number of locations along the Mawson Coast (see Fig. 5.1), it was decided to concentrate investigations at Welch Island. The largest cluster of dead birds was found at this site and, being closest to Mawson Station, it was easier to visit given the difficult environmental conditions at the time.

After the initial visit to Welch Island on 25 November, four additional visits were made between then and 24 December. A total of 117 carcasses were found – one was on the sea-ice, 50 were among the broken sea-ice at the land–sea-ice interface, 5 on land between the sea-ice and the breeding colony and 61 spread among the lower reaches of the colony. Some birds were found dead on their nests and others dead beside an active nest with an incubating bird. All dead birds appeared to be in good body condition with no external injuries, and there was no

obvious discharge from the eyes, mouth or cloaca. Dead birds were marked with dye when first observed and any newly dead bird that had not be attacked by skuas was collected, stored frozen at −20°C at Mawson and returned to Australia in March 2002.

Post-mortem investigations were carried out on 34 Adélie penguins from three locations along the Mawson coast. Initially, these were done under maximum security at the Australian Animal Health Laboratories (AAHL), Geelong, Victoria, because of the risk of these birds carrying an infectious agent. Later, when the cause of death was determined, additional investigations were made under lower biological security at the Headquarters of the Australian Antarctic Division, Kingston, Tasmania.

The birds investigated comprised 26 females, 6 males and a further 2 undetermined. Death in all but three birds was due to severe traumatic injuries consistent with crushing by a powerful external force. These injuries included fractured bones, dislocated limbs, internal haemorrhage and bruising. Many had multiple injuries. Twelve females had hard-shelled eggs in the oviduct, all of which were broken. Sixteen birds had fractures and/or dislocations, 13 experienced abdominal haemorrhages and five showed evidence of head injury. Severe injuries such as fractured vertebrae, keel and pelvis were also evident. The force required to inflict such injuries is substantial. There was no evidence from histo pathology or microbiological investigations to suggest disease was the primary cause of death.

Three birds exhibited no evidence of trauma. Two of these had been extensively scavenged, presumably by skuas (*Catharacta maccormicki*) and all viscera removed. The third bird had a prolapsed cloaca. Three carcasses collected at Kirton Island on 23 November and one at the Rookery Islands on 7 December showed evidence of death from traumatic injury similar to the birds from Welch Island.

5.5 Discussion

5.5.1 Cause of Death

No disease process was identified in the investigation, and noting the severe injuries obviously sustained through crushing it is concluded that most birds died as a direct result of these injuries. Given that most dead birds were found at the land–sea-ice interface, it seems most likely that these injuries occurred there. Other penguins with severe injuries were found within the breeding colony at their nest site or at varying distances between these locations. It is suggested that these birds survived the initial impact but later succumbed to their injuries, or possibly died of secondary infection caused by the initial trauma and exacerbated by poor weather.

The first birds were found dead on 23 November, and by 30 November, 106 birds were dead. There was a severe storm on 15/16 November during which the winds at Mawson averaged 100 km h^{-1} for 48 h. Further blizzards occurred on

27/28 November and 4/5 December and nine additional birds were found dead on 11 December. Details of meteorological conditions at the time are given in M. Pook (this volume).

The main point of arrival for penguins at Welch Island and the location of the dead penguins are at the northern end, i.e. at the seaward side. The bay shoals rapidly and the beach is narrow and often covered with rafted and broken ice. It is postulated that the storm of 15/16 November or any other meteorological/oceanographic event at much the same time, resulted in a swell which swept into the bay and broke up the sea-ice close to shore. Those penguins on the ice close to the shoreline at the time probably fell between the floes where they were crushed and killed or severely wounded. A similar explanation may be applied to other sites where crushed birds were found. The uneven distribution of these sites along the coast may be due to differing sea-ice conditions and surrounding bathymetry. A shallow approach to an island, as found on the seaward side of Welch Island, will magnify the effect of a large swell and will move the ice substantially.

The mortality event as described may occur from time to time along the Antarctic coast but, given the sparsity of human habitation and movement around the Antarctic coastline, it is not surprising that such an event has not been observed before.

Since the event occurred at the peak of egg laying, this offers a possible explanation as to why the dead birds were predominantly female. Some of the females may have just returned from their pre-laying exodus while others were trapped while on the sea-ice to ingest snow or ice. The female penguins close to laying frequently have been observed leaving their nest guarded by the male to eat snow at a nearby snow bank.

The death of 147 penguins, from a population of 80,000 breeding pairs of Adélie penguins, at first sight may seem unimportant or insignificant. However, in our experience the occurrence of clusters of dead adult penguins in the vicinity of Mawson is uncommon, particularly at egg laying (November) and thus warranted investigation. The death of chicks later in the breeding season is common, however. Dead chicks are observed frequently in their natal colonies and in some seasons in large numbers. The causes of death are considered natural and include predation by skuas, poor parenting and starvation arising from the absence of food within the foraging range of the parents (Irvine et al. 2000).

5.6 General Conclusions

As it transpired, infectious disease was not the cause of death among the penguins. However, the possibility existed and we believe it a reasonable precaution that all investigations of unusual mortality events should start with this premise and from that point be investigated in a safe, systematic and thorough way.

The process by which our investigations were carried out was developed on an ad hoc basis. Apart from the field investigations, many other practical issues emerged relating to decision making, allocation of priorities, notification to appropriate

authorities and agencies, identification of expert assistance, the requirement of permits to enter protected areas, and quarantine issues, both within Antarctica and on the entry of samples into Australia and other Sovereign States and Territories.

Investigations in Antarctica are carried out often under difficult circumstances. The field team recognised the urgent need to carry out investigations and to obtain samples for pathology before the carcases deteriorated, became covered with snow or were scavenged by skuas. This requirement often conflicted with the availability of support staff and the occurrence of poor weather or sea-ice conditions which precluded sea-ice travel. Other problems included the absence of trained investigators to provide advice and the lack of readily available and appropriate sampling equipment.

As a consequence of the experience gained during this investigation, Australia, through the Australian Antarctic Division (Australia 2002), has developed a contingency plan for action should unusual mortality events occur in the future (reproduced as Appendix F, this volume). The plan is designed to provide guidance for people working in Antarctica on the collection of information and samples to assist with determination of the cause, while reducing the likelihood of accidental spread by people if an infectious disease is involved. Central to the plan is the provision to all Australian Antarctic stations and ships of a kit for the 'investigation of any unusual mortality'. These kits contain both detailed instructions on how to conduct an investigation, and all the sampling equipment and protective clothing required to carry out the investigations. The plan also provides guidance to head-office staff in Australia on how to manage the incident.

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Chapter 6
Investigation of the 1998 Mass Mortality Event in New Zealand Sea Lions

W. Roe

6.1 Introduction

The New Zealand (Hooker's) sea lion, *Phocarctos hookeri*, is New Zealand's only endemic pinniped, and, with a population of less than 15,000 (Gales and Fletcher 1999) and a limited breeding range, is one of the world's most endangered pinnipeds. While the range of the NZ sea lion extends from Macquarie Island in the south to the South Island of New Zealand in the north (Childerhouse and Gales 1998), breeding occurs in a much more restricted area. The main breeding sites are in the sub-Antarctic islands, with between 86% (Wilkinson et al. 2003) and 95% (Cawthorn 1993) of pups born at four breeding colonies in the Auckland Islands. A further breeding population is present on Campbell Island, and recent studies suggest that the importance of this colony is increasing (Childerhouse et al. 2005; Childerhouse and Gales 1998; Chilvers et al. unpublished data). Occasionally pups are born on Stewart Island (Childerhouse and Gales 1998) and the New Zealand mainland (McConkey et al. 2002). Currently, the NZ sea lion is classified as 'Vulnerable' by the International Union for Conservation of Nature (IUCN) (Reijnders et al. 1993), and as 'Threatened' under the New Zealand Marine Mammal Protection Act 1978 (Hitchmough 2002).

Since the early 1980s, survey teams have visited the Auckland Islands to carry out population monitoring studies. Visits take place during the summer breeding season, and are based at Sandy Bay on Enderby Island (Fig. 6.1). While Dundas Island holds the largest breeding population, topographical and tidal conditions make landing at this location difficult and unpredictable. In contrast, Enderby Island has a more protected landing site and a supply of fresh water (Cawthorn 1993), and hence the Enderby breeding colonies can be more regularly and closely monitored.

W. Roe
New Zealand Wildlife Health Centre, Institute of Veterinary Animal and Biomedical Sciences, Massey University, Palmerston North, New Zealand
e-mail: W.D.Roe@massey.ac.nz

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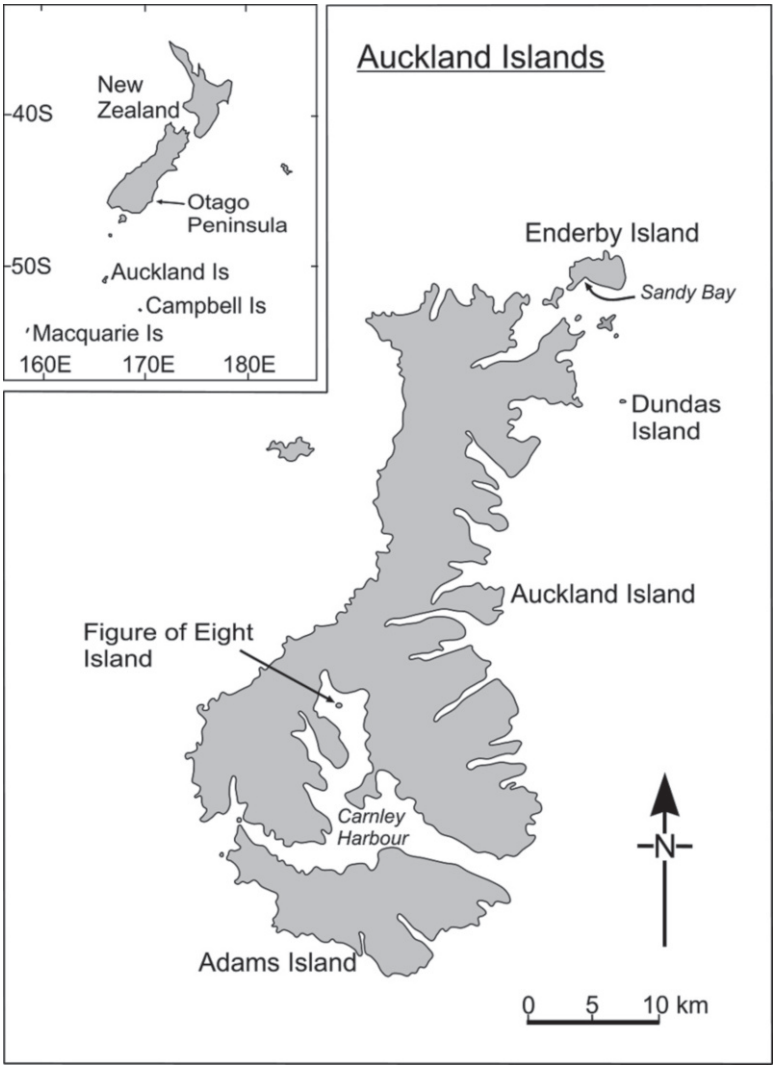

Fig. 6.1 Auckland Islands map (from Baker 1999). With permission of the New Zealand Department of conservation

Most published information regarding the reproductive behaviour of the NZ sea lion has been gained from observations made on Enderby Island (Cawthorn 1993; Gales 1995). Briefly, adult males land and establish territories in late November, while pregnant females begin to arrive in early December. Pupping begins within a few days of the females' landing, and continues until the second week in January, with about half the pups being born by 25 December. Females undergo a post-partum oestrus within 2 weeks of pupping, and mating continues until mid-January, after which males leave the colony. Following mating, females alternate between

foraging trips to sea and periods on land to suckle pups. From the third week in January, females at Sandy Bay move their pups away from the beach, often into areas of dense scrub where observation is difficult (Cawthorn 1993; Gales 1995).

The New Zealand sea lion population is susceptible to a number of potential threats, including predation, environmental change, infectious disease, biotoxins and anthropogenic influences. Little is known about natural mortality and overall disease status of the species, and the behavioural ecology and remote nature of major breeding and haul-out sites mean that an appreciable amount of undetected mortality is likely occurring. There have, however, been three documented unusual mortality events since 1998; the earliest of these is described here.

6.2 Case Study: The 1998 Mortality Event

6.2.1 Field Observations

The following information is derived from a report written by Gales and Childerhouse (1999).

On 13 January 1998, an expedition team from the New Zealand Department of Conservation (DOC) arrived at the Sandy Bay research base in order to carry out mark–recapture studies as part of an annual population monitoring study. Observations and tagging of pups at Sandy Bay and Southeast Point began on 14 January, at which time the colonies at these sites appeared normal, with 11 dead pups noted at Sandy Bay and 14 dead pups at the smaller colony at Southeast Point. By 17 January all pups at Sandy Bay had been caught and tagged, with a total of 471 live pups and 11 dead (2% mortality). The mortality rates observed were assessed as being somewhat lower than usual for the Sandy Bay colony, and within the expected range for Southeast Point, where the mortality rate can vary from 15 to 40% (Chilvers, unpublished data). No evidence of illness was present in pups or in adults at either site.

A landing was attempted at Dundas Bay on 20 January, but adverse sea conditions led to the attempt being aborted. The following day, 97 live and 23 dead pups were observed at the Figure of Eight Island, a normal rate of mortality for this site. A successful landing was made at Dundas Island late the same afternoon, and it was quickly apparent that a large number of dead pups were present. On the basis of the level of decomposition of the bodies, it was estimated that many of these pups had died within the previous few days. A few adult females were also found dead, but their numbers did not appear to be excessive. On 22 January, 400 Dundas pups were captured and tagged. Many of these pups showed clinical signs of illness, including purulent ocular discharges, abscesses over the head and ulceration of the genital mucosa. A mark–recapture study carried out the next day resulted in an estimate of 1,748 live pups, and a total of 625 dead pups were counted (36% mortality). This level of mortality is approximately 3 times higher than expected on the basis of

surveys from previous years, and the survey team began to be concerned that an unusual mortality event had taken place. The fact that mortality was low or normal on Enderby and Figure of Eight Islands led them to suspect that the causative agent was restricted to Dundas Island.

Two days later, Dundas Island was again visited, this time in order to capture, anaesthetise and extract teeth from adult females as part of an ageing study. Eight of 38 females captured had enlarged swellings in the throat area, although each appeared in good body condition. One dead female was seen on the rocks, but could not be recovered for necropsy.

During this same period, several dead adult females were seen at Sandy Bay. One was necropsied, and had a small amount of purulent exudate in the region of the sub-mandibular lymph node. Tissue samples were collected and fixed in formalin. Pup mortality at Sandy Bay was within normal limits for that stage of the breeding season, and no ill pups were observed. However, on 27 January, 6 days after the first landing at Dundas, 20 dead pups were found on Sandy Bay beach, and many other pups appeared weak and lethargic. Nearly all females had left the beach, but a single dead female and two dead adult males were seen. Necropsy of the female was undertaken. She had numerous small raised skin lesions along her flank and swelling of the sub-cutaneous tissues of the neck. Tissues were collected for histopathological analysis. External lesions on adults are shown in Fig. 6.2.

The survey team was now convinced that a mortality event was occurring at Dundas and Enderby Islands, representing a major threat to the population, given that these sites represented up to 95% of the annual pup production for the species. The Department of Conservation's Southland Conservancy was contacted, and a helicopter carrying sampling equipment and protocols was despatched the next day. All other scientific investigations on the islands were suspended in order to minimise stress and to decrease chances of disease transmission between sites.

The supply helicopter landed at Sandy Bay on 28 January, and over the next 24 h post-mortems were carried out on three adult females, one sub-adult female and four pups. The bodies of three additional pups and samples collected from the field post-mortems were sent to Massey University for further analysis.

By the time the scientific team left the Auckland Islands on 22 February, the mortality event was effectively over. During a period of 20 days, from 20 January to 8 February, a total of 1,606 pups and 74 adults were observed to have died. The actual adult mortality is likely to have been much higher than this figure; however, since lactating females spend almost half their time foraging at sea, and most adult males leave the islands by late January, deaths in these animals would not have been observed.

6.2.2 Diagnostic Investigation

The post-mortem findings and results of tests for infectious agents are summarised from Duignan (1999).

Half of the 10 pups necropsied were dehydrated and in poor body condition, while the rest were in fair to moderate condition with few gross lesions. One pup

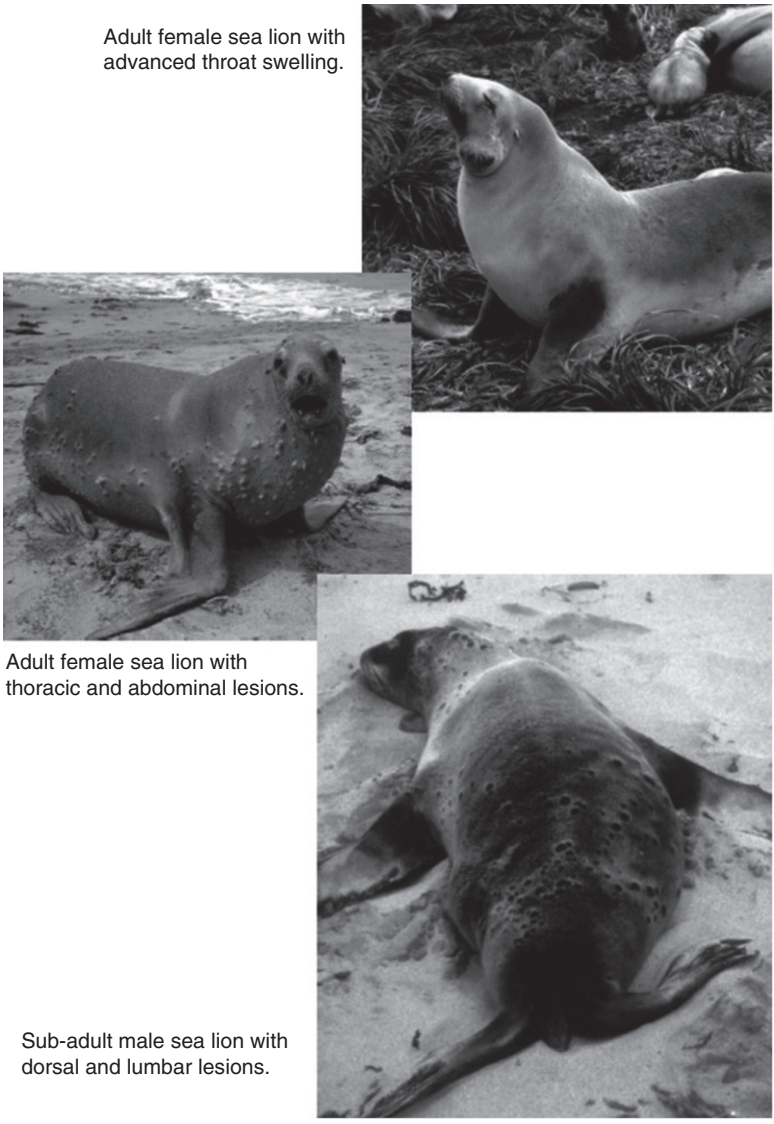

Fig. 6.2 Gross appearance of affected adults. All images by Nick Gales, Simon Childerhouse and Nadine Gibbs (from Baker 1999). With permission of the New Zealand Department of conservation

had ulceration of the peri-anal tissue, and one had suppurative arthritis of the stifle joint.

One-third of the adults subjected to post-mortem examination were in poor condition. A few of them had cellulitis involving the soft tissues of the neck, and several had discrete raised red skin lesions that oozed blood when incised. One subadult female was in good body condition but had multifocal areas of necrosis in the liver.

Most pups examined had little microscopic evidence of disease. In the few that did have lesions, bacterial infection was the most likely cause of death, while some had evidence of other concurrent disease (e.g. hookworm infection) that would likely have played a role in their mortality. In adults, the most notable histopathological lesions were in the skin, peripheral lymph nodes and lungs, and were characterised by fibrinoid necrosis of arterioles with suppurative inflammation and colonies of Gram-negative bacteria. These lesions are consistent with a systemic bacterial infection causing septicaemia and vasculitis. A Gram-negative organism was isolated from multiple organs collected at necropsy, and sequencing of the 16S rRNA was consistent with a member of the *Campylobacter* genus. Several other pathogenic bacteria were isolated from the affected animals, including a number of *Salmonella* species.

Skin lesions and cell cultures were negative for viral particles using electron microscopy, and viral culture, polymerase chain reaction (PCR) and ELISA tests were negative for morbilliviruses, herpesviruses and phocine distemper virus. Serological testing on serum collected from 28 convalescent adult females showed no evidence of exposure to canine distemper virus, although 11% had low or borderline titres against phocine distemper virus. The low prevalence of exposure to this virus, however, in conjunction with the low titres and the absence of histological lesions consistent with viral infection means that distemper was unlikely to have played a role in this event.

Climatic conditions around New Zealand waters during the summer of 1997/98 were influenced by an El Niño/Southern Oscillation (ENSO) event. Surface water temperatures in the sub-Antarctic region were below average over this period, but sea temperatures off the coast of northern New Zealand at this time were higher than average, and resulted in a number of toxic phytoplankton blooms between January and March. These blooms were caused by *Gymnodinium* sp. and were associated with fish and shellfish die-offs, which also casused respiratory signs in humans swimming in affected areas (Chang 1998a, b, c). Despite low water temperatures in the sub-Antarctic area, an aerial image taken on 8 February 1998 indicated increased phytoplankton levels in the region of the Auckland Islands. As biotoxins were considered a possible causal factor in the mortality event, part of the early response of the Southland Conservancy was to contact toxicologists at AgResearch, New Zealand. Sampling protocols were established and transmitted to field workers by radio. Samples of blood, tissue, milk and stomach contents were collected and transported back to AgResearch (Ruakura, Hamilton, New Zealand) by helicopter.

Mucilaginous slime was reported to be present in the water column in the vicinity of the Auckland Islands at around the same time. A sample of this slime was collected from the trawl net of a scampi fishing vessel and was analysed at AgResearch (Murdoch 1999) and at the Cawthron Institute in Nelson, New Zealand (Mackenzie 1999). Shellfish, which are known to accumulate and store biotoxins, were collected from Sandy Bay in late February and analysed for toxins. Samples were analysed for the presence of known marine biotoxins using specific assays, and for general toxicity using mouse bio-assays. No toxins were identified. Blubber samples collected

in the field were also analysed for the presence of organochlorine and polychlorinated biphenyl (PCB) residues, which were detected at low levels in the three samples examined (Jones 1999).

6.2.3 Conclusions from Diagnostic Investigations

On 8 and 9 June 1998, a workshop took place at the Department of Conservation Science and Research Unit in Wellington, New Zealand, in order to review and discuss aspects of this mortality event. Participants concluded that no single agent could be identified as being solely responsible for the event. While most of the adults examined died as a result of bacterial septicaemia caused by an organism likely to be from the *Campylobacter* genus, there were also a significant number of severe bacterial infections caused by other pathogens, including organisms likely to be part of the normal environmental flora (Duignan 1999). It is most likely that the cause of the dramatic increase in mortality during this time was multi-factorial, with some unidentified external event, or combination of events, causing increased susceptibility to infectious disease.

6.3 Discussion

This case study highlights many of the difficulties faced in recognising and responding to mortality events in wild populations, particularly those in remote areas. Recognition of unusual mortality events requires both the ability to detect and quantify mortalities, and a knowledge of previous mortality rates. Difficulties in accessing some NZ sea lion breeding sites, particularly Figure of Eight Island, Campbell Island and Dundas Island, have meant that regular, reliable estimates of population size and pup mortality have been lacking (Wilkinson et al. 2003). Since 1995, however, increased population monitoring on the Auckland Islands (Gales and Fletcher 1999; Wilkinson et al. 2003) and Campbell Island (Childerhouse et al. 2005) has greatly increased the amount of information available. Identification of significant mortality events also requires that dead animals can be observed. While the seasonal breeding behaviour of NZ sea lions means that close observation of pups and females can occur over the mating and early breeding period, deaths that occur outside this period, as well as those that occur in males and juveniles, are more difficult to detect. During the 1998 epidemic for example, it was impossible to estimate adult mortality; lactating females spend almost half their time foraging at sea and adult males begin to leave the breeding colonies in mid-January (Cawthorn 1993). For adult females, the only likely manifestation of significant levels of mortality would be in decreased pup production during subsequent breeding seasons.

When an unusual mortality event is able to be detected in a remote location, logistical difficulties in terms of access and communication between affected sites and the

mainland impose constraints on our ability to respond to such events. Limited permanent facilities are available on Enderby Island, and most supplies for the scientific team are carried in each year. A key event in the response to the 1998 epidemic was rapid notification of the DOC Southland conservancy and prompt initiation of contact between the Department and advisors in other scientific organisations. This in turn enabled rapid development of diagnostic strategies and tissue sampling protocols, thereby greatly increasing the chances of identifying causal factors.

Possibly the most important practical outcome of this event was the development of the sub-Antarctic Pinniped Mortality Event Contingency Plan (Baker 1999). This document sets out criteria for defining, notifying and responding to an unusual mortality event, and details a chain of command and external support networks. Specific details of necropsy and sampling protocols are included in the document, as are methods for carcass disposal, minimisation of inter-site disease transmission, and mitigation of human health risks.

Currently, scientific expeditions to the Auckland Island sea lion breeding colonies include a veterinarian trained in marine mammal necropsy and tissue sampling techniques, and the annual research program incorporates routine necropsy of all accessible dead animals. Two further mortality events have occurred in recent years, one in the 2001/02 breeding season and a second in 2002/03 causing 33% and 21% pup mortality, respectively (Wilkinson et al. 2006). In these cases, systems developed during and following the 1998 event were able to be rapidly implemented, and allowed early diagnosis of the causal agent *Klebsiella pneumoniae* (Castinel, unpublished data). While the immediate effect of these epidemics was on the pup population, the effect on the total NZ sea lion population, particularly with respect to recruitment of adult females into the breeding pool, is currently unknown, but is likely to be ongoing (Wilkinson et al. 2006).

While recognition of and response to mortality events that occur in remote locations in the Antarctic and sub-Antarctic region is fraught with difficulties, the investigative efforts detailed here show that robust protocols and networks can be set in place. In addition, these events emphasise the fragile nature of some of our Antarctic wildlife populations, and reiterate the importance of efforts to understand and protect these species.

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Chapter 7
Health Assessment of Weddell Seals, *Leptonychotes weddellii*, in McMurdo Sound, Antarctica

P.K. Yochem, B.S. Stewart, T.S. Gelatt, and D.B. Siniff

7.1 Introduction

The demography of Weddell seals in eastern McMurdo Sound, Antarctica, has been well studied during the past three decades (e.g. Stirling 1971; Siniff et al. 1977; Testa and Siniff 1987; Hastings and Testa 1998; Gelatt et al. 2001). Detailed life-history data are available on thousands of seals tagged as pups in McMurdo Sound, making this population a rich resource for wildlife health studies because health parameters can be evaluated in the light of reproductive histories and genetic relationships of several generations of tagged seals.

Recently, evidence of exposure to diseases generally associated with domestic animals and feral wildlife has been detected in Antarctic wildlife (Austin and Webster 1993; Olsen et al. 1996; Gardner et al. 1997; Retamal et al. 2000; Foster et al. 2002) and this has generated concern and debate regarding the risks of disease introduction to Antarctic wildlife. Antibodies to viruses that have caused large die-offs in phocids in other areas of the world have been detected in Weddell seals (Bengtson et al. 1991), and there is a historical report of a mass die-off of crabeater seals that may have had a viral etiology (Laws and Taylor 1957).

We examined and collected biomedical samples from Weddell seals (*Leptonychotes weddellii*) during studies of post-breeding-season foraging behaviour of adults and movements of weaned pups as a complement to ongoing studies on the ecology and population dynamics of the McMurdo seals (Stewart et al. 2000, 2003). Here we report on Weddell seal health assessments conducted during the 1996/97, 1997/98 and 1998/99 breeding seasons at the Delbridge Islands (77.68°S,

P.K. Yochem and B.S. Stewart
Hubbs-Sea World Research Institute, San Diego, CA, USA
e-mail: PYochem@hswri.org; BStewart@hswri.org

T.S. Gelatt
NOAA Fisheries, NMML, Seattle, WA, USA

D.B. Siniff
Dept. Ecology, Evolution & Behavior, University of Minnesota, St. Paul, MN, USA

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166.50°E), McMurdo Sound, Antarctica. Our objectives were to compile baseline biomedical data for Weddell seals in McMurdo Sound, and to identify infectious and non-infectious diseases affecting the population. Development of such a database, including information on normal background morbidity and mortality, is an important first step in evaluating natural versus anthropogenic impacts on population health (Geraci et al. 1999; Reddy et al. 2001). These data will be integral to international studies of southern ocean pinnipeds that seek to evaluate the influence of biotic and abiotic factors on the ecology of these apex predators.

7.2 Methods

Seals that appeared to be healthy were observed for several minutes while resting prior to initiation of physical restraint (Fig. 7.1). Respiratory rate was obtained by counting chest excursions for 2–5 min; heart rate was obtained, where possible, by observing the left lateral chest wall just behind the fore flipper for the slight movement associated with each heartbeat. The body was scanned for obvious abnormalities such as fresh wounds or nasal discharge. Animals were captured and handled using standard techniques (Stirling 1966; Cline et al. 1969); head bags (Fig. 7.2) were used for all adults (some animals were first captured in a hoop net) and for some larger pups. Seals were examined by a veterinarian and were measured (standard length and axillary girth). Physical examinations included evaluation of the musculoskeletal system (symmetry, lesions) and cardiopulmonary system (heart rate, mucous membrane colour, respiratory rate and character). The integument was examined for evidence of moult, wounds or other lesions. Eyes, ears, nares and oral cavity were inspected for discharges or lesions such as corneal ulcers and broken teeth. Hydration was estimated by visual inspection of mucous membranes, skin turgor and tears. The urogenital and gastrointestinal systems were evaluated opportunistically by examining urine or faeces and by visual inspection for lesions at urogenital openings. Examination of the nervous system consisted of a limited evaluation of animals' vision, hearing, cranial nerve function (e.g. blink reflex) and peripheral nerve function (e.g. flinching when rear flippers were touched) in response to the activities of handlers. Blood samples were collected from the extradural vein or from the interdigital vessels of the rear flippers of all seals; faecal samples were collected opportunistically from a subset of seals (17 adults, 3 weaned pups). Satellite-linked radio-transmitters and other telemetry instruments were glued to the dorsal pelage of some seals (Fig. 7.2). A subset of animals was sedated with intra-vascular injections of diazepam (mean dosage = 0.07 mg kg^{-1}; s.d. = 0.02 mg kg^{-1}) immediately following blood sample collection to facilitate attachment of instruments.

Forty-six adults (14 males, 32 females), 15 weaned pups (8 males, 7 females) and 20 nursing pups (8 males, 12 females) were examined and sampled. In addition, a random sample of 214 seals was surveyed for lesions (as described by McFarlane 1996) without being handled.

Preliminary haematological evaluation was conducted at the Crary Laboratory at McMurdo Station, Antarctica, and included determination of haematocrit and

Fig. 7.1 Resting Weddell seal female and pup being examined prior to capture for biomedical sample collection

description of serum quality. Red and white blood cell counts were obtained using the Unopette system and a haemacytometer. Haemoglobin was evaluated with a BMS haemoglobinometer. Blood smears were air-dried and preserved for transport to the Hubbs-SeaWorld Research Institute laboratory in San Diego, California (USA), where they were stained for microscopic examination and differential count of white blood cells (band neutrophils, mature neutrophils, lymphocytes, monocytes, eosinophils and basophils), morphology of red and white blood cells and subjective evaluation of platelet number. In all, 11 haematological parameters were measured.

Fig. 7.2 Satellite-linked radio-transmitter being glued to the dorsal pelage of a physically restrained Weddell seal

Serum was stored in ultralow freezers (−70 to 80°C) for several weeks and transported on dry ice prior to U.S. laboratory evaluation. Biochemical assays were performed by the SeaWorld San Diego Animal Care Laboratory, San Diego, California. Twenty parameters were measured, including markers for liver, kidney and muscle disease (alanine aminotransferase, aspartate aminotransferase, total bilirubin, gamma glutamyltransferase, blood urea nitrogen, creatine kinase, lactate dehydrogenase), body condition and nutritional status (glucose, cholesterol, triglycerides,

total protein), inflammation/infection (albumin, globulin, alkaline phosphatase, serum iron) and electrolytes (sodium, potassium, chloride) and other ions (calcium, phosphorus). Haematological and serum biochemical parameters were evaluated for age/sex class differences using Kruskal–Wallis ANOVA; differences are reported as significant if $p < 0.05$.

Faecal examinations (consistency, colour, presence of blood or mucous, presence and identity of parasites) by direct smear and faecal flotation were conducted at the Crary Laboratory, McMurdo Station.

Serological assays for antibodies to selected bacterial and viral pathogens were conducted by the California Animal Health and Food Safety Laboratory System, Davis, California (*Brucella* sp., *Leptospira* sp.) and the Okalahoma Animal Disease Diagnostic Laboratory, Stillwater, Oklahoma (canine distemper virus, phocine distemper virus, dolphin morbillivirus, porpoise morbillivirus).

7.3 Results

Abnormal findings from physical examination were recorded for 21 of 81 (26%) animals handled during the course of this study (Table 7.1a); some seals had more than one lesion. Abnormalities included 1 adult that began shivering within 5 min of capture, 8 adults with very worn or broken teeth, 1 pup with an inspiratory wheeze and bilateral nasal discharge, and 11 seals (8 adults, 3 pups) with fresh puncture wounds. Ocular lesions were observed in three adults. There were no significant ($p < 0.05$) differences in serum biochemical parameters between animals with normal versus abnormal physical findings; however, animals with abnormal physical findings had significantly more band neutrophils and mature neutrophils, and fewer lymphocytes than normal seals. Clinically healthy pups were significantly larger (length and girth) than pups that were abnormal on physical examination.

Haematologic and serum chemistry values recorded by age and sex are presented in Table 7.2. Adults differed significantly from pups (weaned and nursing) for 14 parameters. Adults had more band neutrophils and eosinophils, as well as higher creatinine, total bilirubin, alanine aminotransferase, sodium and chloride levels. Pups had more lymphocytes and higher glucose, cholesterol, calcium, phosphorus and serum iron. There was no significant difference in haematocrit between adults and pups, but weaned pups had higher haematocrits than nursing pups. Although there was no significant difference in triglycerides between weaned pups and adults, nursing pups had significantly higher triglyceride levels than either weaned pups or adults. Nursing pups also had higher total protein levels and globulin levels than adults or weaned pups; albumin levels were higher in weaned pups than in either adults or nursing pups.

No sex differences were detected for any of the biochemical parameters measured. However, statistically significant differences were noted for some haematological parameters, and these differences were present in both adults and pups: males had lower haematocrits, lower red blood cell counts and more band neutrophils than females.

Table 7.1 Lesions and other sources of morbidity recorded during visual inspections (214 seals) or veterinary medical examinations (81 seals) of Weddell seals in McMurdo Sound

(a) Lesions and other abnormalities detected during veterinary medical examinations of 81 seals handled for biomedical sampling. Abnormalities were recorded from 21 of 81 (26%) seals; some seals had more than one lesion

	Adults		Pups	
Lesion/abnormality	Males (n = 14)	Females (n = 32)	Males (n = 16)	Females (n = 19)
Fresh skin lesion(s), minor (e.g. puncture wound)	5 (36%)	3 (9%)	2 (13%)	1 (5%)
Worn or broken teeth	4 (29%)	4 (13%)	0	0
Ocular pathology (corneal opacity)	2 (14%)	1 (3%)	0	0
Inspiratory wheeze	0	0	0	1 (5%)
Purulent bilateral nasal discharge	0	0	0	1 (5%)
Shivering	1 (7%)	0	0	0

(b) Lesions and other abnormalities recorded during visual surveys of 214 randomly selected Weddell seals. Abnormalities were noted for 75 of 214 (35%) seals; some seals had more than one lesion

Lesion/abnormality	Adult males (n = 42)	Adult females (n = 119)	Weaned pups (n = 53)
Pup ill-health	0	0	13 (25%)
Fresh skin lesion(s), minor	9 (21%)	23 (19%)	2 (4%)
Fresh skin lesion(s), major	10 (24%)	7 (6%)	0
Worn or broken teeth	11 (26%)	9 (8%)	0
Ocular pathology (e.g. corneal opacity, punctured globe)	3 (7%)	7 (6%)	1 (2%)
Respiratory disease	1 (2%)	2 (2%)	3 (6%)
Firm nodule on thorax	0	1 (1%)	0
Healed fracture, foreflipper	1 (2%)	0	0

No parasites were detected during examinations of blood smears or during direct examinations of fresh faecal smears. Faecal floatations revealed ova from both anisakid nematodes and diphyllobothrid cestodes in 11 of 17 (65%) adult seals. Anisakid nematode ova were found in only 3 of 17 (18%) adults. Diphyllobothrid cestode ova were found in only 3 of 17 (18%) adults and 2 of 3 (67%) weaned pups. No parasite ova were detected in 2 of 17 (12%) adults and 1 of 3 (33%) weaned pups.

Visual surveys of 214 randomly selected Weddell seals (42 adult males, 119 adult females, 53 weaned pups; Table 7.7b) revealed the following lesions or other sources of morbidity: pup ill-health (lethargy, malnourishment; 13 animals), fresh skin lesions/wounds (34 minor, 17 major), severely worn or cracked teeth (20 animals), ocular pathology (11 animals), and respiratory disease (6 animals). One animal had a firm nodule on its thorax, and one appeared to have a healed fracture of one foreflipper. Some seals had more than one lesion.

No serum antibodies were found in any seal for *Brucella* sp., *Leptospira* sp. or the four morbilliviruses (canine distemper virus, phocine distemper virus, dolphin morbillivirus, porpoise morbillivirus).

Table 7.2 Haematologic and serum chemistry values for 81 Weddell seals sampled in McMurdo Sound expressed as mean (± standard deviation)

	Adults		Weaned pups		Nursing pups	
(a) Haematology	Males	Females	Males	Females	Males	Females
Haematocrit (%)	60.0 (4.4)	63.0 (2.5)	62.0 (3.0)	63.4 (3.1)	59.7 (4.2)	58.3 (1.0)
Red blood cells (no. × 10^6)	4.5 (0.7)	4.7 (0.5)	4.5 (0.5)	4.7 (0.6)	4.4 (0.8)	5.0 (0.6)
White blood cells (no.)	10,235.0 (2,752.1)	8,275.0 (1,052.3)	7,956.3 (1,429.6)	8,371.4 (1,326.3)	7,800.0 (2,271.6)	6,362.5 (1,949.1)
Band neutrophils (no.)	297.9 (344.1)	67.1 (101.9)	12.5 (35.4)	26.1 (44.9)	90.0 (106.8)	13.5 (27.0)
Neutrophils (no.)	5,353.4 (2347.7)	4,486.1 (719.2)	3,969.9 (812.8)	4,557.9 (896.6)	4,390.0 (1561.3)	3,555.5 (1140.8)
Lymphocytes (no.)	2,051.1 (417.9)	1,887.8 (514.6)	1,976.8 (425.5)	2,276.6 (693.9)	1,602.7 (1,426.3)	1,789.3 (1,030.4)
Monocytes (no.)	1,124.5 (714.3)	879.2 (216.3)	938.0 (491.5)	975.1 (664.4)	642.7 (359.7)	514.0 (205.4)
Eosinophils (no.)	1,237.1 (778.6)	1,008.3 (461.9)	1,052.3 (529.1)	536.4 (223.7)	505.3 (250.3)	489.0 (135.4)
Basophils (no.)	33.0 (72.0)	8.8 (27.8)	6.8 (19.1)	0.0 (0.0)	0.0 (0.0)	0.0 (0.0)
(b) Serum chemistry (SI Units)	Adults		Weaned pups		Nursing pups	
	Males	Females	Males	Females	Males	Females
Glucose (mmol L^{-1})	5.8 (0.7)	5.5 (1.0)	6.6 (0.5)	6.9 (0.5)	6.9 (0.3)	7.6 (0.8)
Blood urea nitrogen (mmol L^{-1})	6.7 (2.7)	7.9 (2.4)	4.7 (1.7)	5.5 (1.5)	5.8 (0.7)	5.3 (1.4)
Creatinine (mmol L^{-1})	114.9 (26.5)	123.8 (26.5)	88.4 (17.7)	79.6 (17.7)	79.6 (8.8)	70.7 (44.2)
Total bilirubin (mmol L^{-1})	8.6 (5.1)	10.3 (5.1)	5.1 (1.7)	3.4 (1.7)	5.1 (1.7)	5.1 (1.7)
Cholesterol (mmol L^{-1})	5.7 (1.5)	7.9 (2.2)	15.2 (1.9)	14.1 (2.6)	13.4 (0.8)	12.6 (1.5)
Triglycerides (mmol L^{-1})	1.0 (0.5)	0.9 (0.6)	1.4 (1.0)	1.0 (0.4)	4.3 (1.2)	2.3 (1.7)
Total protein (g L^{-1})	80.0 (10.0)	74.0 (8.0)	73.0 (10.0)	70.0 (5.0)	87.0 (9.0)	76.0 (14.0)
Albumin (g L^{-1})	25.0 (2.0)	25.0 (3.0)	28.0 (2.0)	28.0 (1.0)	25.0 (1.0)	23.0 (2.0)
Globulin (g L^{-1})	58.0 (10.0)	49.0 (6.0)	45.0 (11.0)	42.0 (11.0)	62.0 (9.0)	53.0 (12.0)

(continued)

Table 7.2 (continued)

(b) (serum chemistry (SI Units)	Adults		Weaned pups		Nursing pups	
	Males	Females	Males	Females	Males	Females
Alkaline phosphatase (IU L^{-1})	278.6 (140.2)	480.8 (302.6)	478.8 (110.9)	506.7 (116.1)	869.3 (207.6)	484.3 (345.8)
Alanine amino transferase (IU L^{-1})	26.5 (6.3)	25.7 (6.7)	18.8 (5.8)	19.3 (9.4)	22.0 (3.5)	27.3 (7.2)
Aspartate amino transferase (IU L^{-1})	42.1 (14.3)	46.4 (26.4)	38.3 (20.8)	39.6 (16.5)	34.3 (8.0)	57.8 (10.7)
Gamma glutamyl transferase (IU L^{-1})	3.9 (2.1)	7.8 (15.1)	5.3 (5.3)	3.0 (2.4)	4.5 (3.8)	3.9 (4.2)
Creatine kinase (IU L^{-1})	343.4 (494.9)	501.6 (397.9)	509.5 (558.4)	450.4 (264.6)	393.3 (178.0)	718.3 (239.0)
Lactate dehydrogenase (IU L^{-1})	500.8 (138.2)	608.0 (375.0)	611.3 (139.2)	696.4 (329.7)	639.3 (113.1)	855.5 (243.0)
Calcium (mmol L^{-1})	4.8 (0.4)	4.9 (0.6)	5.2 (0.3)	5.1 (0.3)	5.4 (0.3)	4.9 (1.2)
Phosphorus (mmol L^{-1})	2.0 (0.6)	1.9 (0.5)	2.7 (0.4)	2.5 (0.5)	3.2 (0.1)	2.8 (0.2)
Sodium (mmol L^{-1})	155.7 (3.6)	154.4 (4.9)	152.0 (1.7)	151.4 (1.6)	152.0 (1.5)	152.0 (2.8)
Chloride (mmol L^{-1})	111.2 (4.7)	109.6 (3.3)	107.7 (2.8)	108.4 (2.2)	111.0 (2.5)	106.5 (0.7)
Potassium (mmol L^{-1})	5.3 (0.6)	5.1 (0.5)	4.8 (0.3)	4.7 (0.5)	4.6 (0.5)	4.7 (0.9)
Iron (mmol L^{-1})	34.7 (14.6)	39.2 (13.1)	40.1 (13.1)	55.7 (22.0)	59.1 (35.3)	74.5 (13.8)

7.4 Discussion

7.4.1 Haematology and Serum Chemistry

Seal et al. (1971) and Schumacher et al. (1992) have presented data for a number of biochemical parameters in Weddell seal blood; the values we measured were consistent with those reported by these authors (exceptions noted below) and were generally within reference ranges known for other phocid seals. Variability in haematology and serum or plasma biochemistry in pinnipeds, including Weddell seals, has been correlated with many factors, including age (Bryden and Lim 1969; Seal et al. 1971; Lane et al. 1972; Horning and Trillmich 1997; Hall 1998; Sepulveda et al. 1999; Lewis et al. 2001; Nordøy and Thoreson 2002), sex (Poulin et al. 1994; Lewis et al. 2001), diet (Kuiken 1985; Thompson et al. 1997), habitat (e.g. coastal vs. offshore dolphins, Duffield et al. 1983), diving ability (e.g. deep-diving vs. shallow-diving species; Vallyathan et al. 1969; Hedrick and Duffield 1991), air temperature (Poulin et al. 1994), physiologic state (e.g. pregnancy, lactation, malt, fasting vs. feeding; Worthy and Lavigne 1982; Poulin et al. 1994; Lewis et al. 2001; Englehard et al. 2002), handling (St Aubin et al. 1979) and condition (Geraci et al. 1979; Roletto 1993).

Some of the age and sex class differences in haematological and serum biochemical parameters we observed in Weddell seals have been reported previously in Weddell seals or other pinnipeds. Seal et al. (1971) also measured higher creatinine levels in adult versus young Weddell seals. Nordøy and Thoresen (2002) reported higher sodium levels and lower calcium levels in harp seal adults than in pups; we observed the same differences in Weddell seals. Although Hall (1998) did not sample adults, she did report higher glucose levels in pups than in yearling grey seals; we observed higher glucose levels in pups than in adults. We observed higher haematocrits in weaned than in nursing pups; this has been reported by other researchers for Galapagos fur seals (Horning and Trillmich 1997) and southern elephant seals (Lewis et al. 2001). Packed cell volume is often used interchangeably with haematocrit (Willard et al. 1999); Sepulveda et al. (1999) reported higher packed cell volume in juveniles than in pups (Juan Fernandez fur seals). Although we did not detect significant differences in haematocrits between adults and pups, others have reported conflicting data on these differences: Sepulveda et al. (1999) found that packed cell volumes of adult females were lower than those of juveniles (Juan Fernandez fur seals), whereas Bryden and Lim (1969) and Lane et al. (1972) found higher haematocrits in adults than in pups.

The nursing Weddell seal pups we sampled had higher total protein levels and globulin levels than adults or weaned pups. Seal et al. (1971) and Nordøy and Thoresen (2002), however, reported higher globulin levels in adults than pups for Weddell seals and harp seals, respectively. Sepulveda et al. (1999) reported higher plasma proteins in juveniles than pups for Juan Fernandez fur seals.

No sex differences were detected for any of the biochemical parameters we measured. However, we did find statistically significant differences for some haematological parameters, and these differences were present in both adults and pups:

males had lower haematocrits, lower red blood cell counts, and more band neutrophils than females. Although male Weddell seals had more neutrophils than females, there was no significant difference between the sexes in the total white blood cell count. Poulin et al. (1994), however, did report higher white blood cell counts in males than in females. Lewis et al. (2001) reported lower haemoglobin levels in males than in females. While there was no significant difference in haemoglobin levels in the males and females we sampled, males did have lower values for two other measures of oxygen-carrying capacity of the blood, haematocrit and red blood cell count.

7.4.2 Macroparasites

A wide variety of endoparasites and ectoparasites have been reported in Weddell seals and other Antarctic phocids (Beverley-Burton 1971; Drozda 1987; Orecchia et al. 1994; Wojciechowska and Zdzitowiecki 1995; Mehlhorn et al. 2002). We did not detect any previously unreported nematodes or cestodes in the Weddell seals we examined, and none of these animals had clinical signs consistent with gastrointestinal or respiratory parasitism (e.g. diarrhea, cough). Parasite infections in marine mammals, even when infestations are heavy, often do not have a significant impact on otherwise healthy hosts and may be considered incidental or secondary findings (Baker 1989; Duszynski et al. 1998 cited in Dailey 2001; Geraci et al. 1999; Dailey 2001; Gulland et al. 2001). Parasites may cause morbidity and even mortality in some cases (Gerber et al. 1993; Roletto 1993; Geraci et al. 1999; Dailey 2001), although this may be due primarily to concurrent secondary bacterial infection (as in verminous pneumonia, Dailey 2001). Gastrointestinal protozoa have been reported in Antarctic and sub-Antarctic pinnipeds (Drozda 1987; Duszynski et al. 1998 cited in Dailey 2001), but we did not detect them in the fresh faecal smears we examined. However, protozoal parasite ova and cysts are frequently missed with a single faecal smear (Willard et al. 1999); examination of multiple samples, collected 1–2 weeks apart, is generally recommended before an animal is considered negative for protozoal parasites.

7.4.3 Bacteria and Viruses

As with macroparasites (nematodes, cestodes, trematodes), the presence of bacteria and viruses is not always correlated with clinical disease. However, bacteria and viruses may have been responsible for mass die-offs, particularly where large populations of social or seasonally gregarious species have been exposed to new pathogens to which they have no herd immunity. Although no Weddell seals tested positive for *Brucella* sp. or morbillivirus in our study, others have reported the presence of antibodies to these and other pathogens in Antarctic phocids

(Bengtson et al. 1991; Stenvers et al. 1992; Retamal et al. 2000). We did not detect leptospirosis in any of the Weddell seals we tested. Although leptospirosis has not been reported to date in pinnipeds in the Southern Hemisphere, it has caused epizootics among pinnipeds in other areas (Vedros et al. 1971). Other serosurveys have demonstrated little exposure to other viruses in Weddell seals and other Antarctic phocids. Truyen et al. (1995) surveyed over 200 adult and 10 juvenile Weddell seals for influenza viruses and paramyxovirus but found no positives. Stenvers et al. (1992) reported no antibodies against phocine distemper virus in Weddell seals, although they did detect high neutralizing titres to seal and feline herpes virus. No antibodies to either herpes virus or distemper virus were detected in crabeater seals by Stenvers et al. (1992).

Our seroprevalence data suggest that the McMurdo Weddell seal population is relatively naïve with respect to pathogen exposure, suggesting that it may be vulnerable to disease outbreaks such as those that have caused large die-offs of marine mammals in other regions. The die-off of crabeater seals reported by Laws and Taylor (1957) affected over 85% of the local population (over 1,500 seals in the Antarctic Peninsula region). Barrett (1999) noted that the population size of Antarctic crabeater seals (several million) would be adequate to maintain a morbillivirus in circulation once introduced; it has been estimated that at least 300,000 individuals are necessary to maintain the human morbillivirus, measles, in the population (Black 1991). Such large numbers are needed because the virus is maintained by infecting new susceptible hosts. Grenfell et al. (1992), examining the long-term consequences of the 1988 morbillivirus epizootic in North Atlantic phocids, stated that the seal population would have to recover for at least 10 years before another outbreak was likely; it appears that this was an accurate calculation, as the most recent (2002) outbreak occurred 13–14 years after the previous one. Jensen et al. (2002) noted that testing in the 10 years leading up to the 2002 phocine distemper outbreak in Europe demonstrated 95% seronegativity in the harbour seal population; all positives were newly-weaned pups, suggesting that these were passive/maternal antibodies. The authors noted that this was an indication that the virus had not been circulating in the population and that the recent (2002) reappearance of phocine distemper virus into this largely susceptible population could allow rapid spread with devastating consequences, as was observed during the 1988 outbreak that killed over 18,000 animals. Van Bressem et al. (2001) noted that decreased seroprevalence to morbillivirus detected over time in two species of odontocete cetaceans made them susceptible to new epizootics, as the virus was not becoming endemic in the population and animals were losing their humoral immunity.

Harwood and Hall (1990) reviewed marine mammal epizootics and their population effects. The factors responsible for emergence or resurgence of a viral disease are often difficult to determine (Miller et al. 2001); often a change in host range is implicated (Truyen et al. 1995). Innate differences among species in immune response (and therefore resistance) to morbilliviruses have been reported in phocids (Duignan et al. 1997). Duignan et al. (1995) proposed that enzootic infection may become established in one species (grey seals), facilitated by factors such as large population size, high annual recruitment, and innate resistance to the virus, and

then be maintained in another less abundant and/or less resistant species (harbour seals) through casual contact with the first species.

7.4.4 Starvation and Physical Injury

Common and important causes of morbidity and mortality in pinnipeds include starvation and malnourishment (particularly of neonates and newly weaned pups), conspecific trauma, and predation (Tierney 1977; Baker and McCann 1989; Steiger et al. 1989; Banish and Gilmartin 1992; Cooper 1996; McFarlane 1996; Baker et al. 1998; Geraci et al. 1999; Gulland et al. 2001; Lucas et al. 2003). Physical injury caused by inter-specific and intra-specific interactions has long been recognised as an important source of non-infectious disease in free-ranging wildlife, including marine mammals (Cooper 1996). The clinical significance of a physical injury to an individual animal depends upon the severity of the injury (including the area of the body affected) and the presence or absence of secondary conditions (e.g. haemorrhage, infection; Cooper 1996; Gulland et al. 2001). Cooper (1996, p. 163) noted that '…clinical signs [of trauma] can range from coma to an almost complete absence of abnormalities.' In the Weddell seals we examined, pup ill health (lethargy, malnourishment) and physical trauma were the most common findings. However, most of the wounds we examined in Weddell seals appeared to be the result of interactions with conspecifics (e.g. male fighting) rather than inter-specific or predatory wounds. The Delbridge Islands in McMurdo Sound are surrounded by fast-ice during the breeding season, when our disease surveys were made. Predators such as leopard seals and killer whales generally do not move into the area until the ice begins to break up and/or icebreakers have opened up a channel into McMurdo Station several months after the peak of the breeding season. Ocular pathology (e.g. corneal oedema, cataracts) was observed in 5% of the seals we examined, and is commonly reported in other stranded and free-ranging pinnipeds (Stoskopf et al. 1985; Schoon and Schoon 1992; Gerber et al. 1993). Severely worn or cracked teeth were observed in 6% of the seals we examined (all adults), and this has been identified as a mortality factor in Weddell seals (Stirling 1969); Weddell seals' teeth are important for maintaining breathing holes in the ice.

7.5 Conclusions

Baseline biomedical data such as those reported here, combined with information on life history and behaviour (e.g. migratory behaviour), are important for elucidating patterns of infection and risk within and between populations and for interpreting toxicological data (Duignan et al. 1995; Gelatt et al. 1999; Geraci et al. 1999). Geraci et al. (1999, p. 368) noted that 'only by understanding the causes and patterns of normal mortality can we recognise unusual events and determine their cause and impacts on a population….'

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Chapter 8
Health Assessment and Diseases of the Weddell seal, *Leptonochotes weddelli*, in Vestfold Hills, East Antarctica

R.A. McFarlane

8.1 Introduction

The Weddell seal, *Leptonochotes weddelli*, is the most southerly breeding of the Antarctic seals. Small groups remain for extended periods over the summer months on the fast-ice of the inlets and shorelines of the Antarctic continent for parturition, lactation and moulting. Those breeding in areas close to Antarctic research stations are uniquely accessible for study, but this may bring them in close contact with human activity and pollution derived from human habitation. Antarctic Treaty nations have undertaken to prevent the accidental introduction of parasites and diseases (Article IX of the Agreed Measures 1964 and The Protocol on Environmental Protection to the Antarctic Treaty, 1991) into Antarctica, but our understanding of existing diseases and their effects are limited. At two sites, the Vestfold Hills, East Antarctica, and at McMurdo Sound, Ross Sea, Weddell seals have been tagged over many years for population and feeding ecology studies. This provides two unparalleled opportunities to investigate health and disease in well-described populations of Antarctic wildlife. Health assessment of the population at McMurdo is reported by Yochem et al. (this volume). It is the population of Weddell seals in the Vestfold Hills that is the subject of this chapter.

There are a few reports of pathology in Weddell seals (Lugg 1966; Johnstone et al. 1973; McFarlane 1996) from the Vestfold Hills but none involved the collection of diagnostic samples. The literature from elsewhere is extensive. These include dental disease (Bertram 1940; Stirling 1969, 1971a), reproductive pathology (Mansfield 1958), gastrointestinal parasites (Bertram 1940; Beverley and Burton 1971; Dearborn 1965; Johnstone et al. 1973; King 1983; Laws 1953; Ridgeway 1972) and ectoparasites (Murray et al. 1965), fight

R.A. McFarlane
National Centre for Epidemiology and Population Health, Australian National University, ACT, Australia
e-mail: romcfarlane@bushlink.net.au

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wounds (Lindsey 1937; McFarlane 1996; Yochem et al. 1999), internal abscesses (Bertram 1940; Stirling 1971b.), and cardiovascular pathology (Prathap et al. 1966). Neonatal death has been investigated by carcass examination (Lindsey 1937; Lugg 1966; McFarlane 1996; Stirling 1971b). Serological surveys have been undertaken for antibodies to morbilli, herpes and influenza viruses (Austin and Webster 1993; Bengston et al. 1991; Harder et al. 1991; Osterhaus et al. 1988) and to *Brucella* spp. bacteria (Retamal et al. 2000). Haematology and biochemical profiles have been developed (Seal et al. 1971; Schumacher et al. 1992; Yochem et al. 1999), and the presence of chlordane, polychlorinated biphenyls (PCBs) and dichlorodiphenyltrichloroethane (DDT) residues have been demonstrated in Weddell seal tissues at low levels not associated with pathology (Hidaka et al. 1983; Kawano et al. 1984).

From these reports it appears that gastrointestinal and external parasitism and non-infectious disease, specifically wounds from con-specific fighting and damage to the teeth used to maintain breathing holes, are the most common disease processes in Weddell seals. The fast-ice habitat gives protection from marine predators until the sea-ice melts in late summer. There are no terrestrial predators. A specific cause for the observed internal abscesses has not been determined, but fight wounds and dental disease could provide adequate sources of anaerobic bacteria. Antibodies to *Brucella* spp. (Retamal et al. 2000) and phocine herpes virus (PHV) (Harder et al 1991) have been reported, but without evidence of mortality or morbidity, although observations of a foamy respiratory discharge are discussed in the latter report.

Investigations of clinical disease by McFarlane (1996) in the Vestfold Hills and Yochem et al. (1999) at McMurdo Sound suggest that respiratory disease, including the production of foamy respiratory discharge, is more common in the Vestfold Hills population. Other clinical findings described in these studies included ocular pathology, post-partum discharges, inter- and con-specific wounds and trauma. However, neither study was designed to enable the accurate estimation of population parameters such as prevalence or incidence of disease syndromes. This limits the usefulness of information from these earlier studies for comparisons over time or between populations, or to identify disease amplification and consider risks of disease introduction.

This study approaches the task of developing baseline health data for Weddell seals using an epidemiological approach (e.g. Thrushfield 1995). This enables population parameters to be developed and disease causation to be considered in the absence of opportunities to contain or observe individuals repeatedly. Three complementary procedures were employed. Non-invasive observations of a large cross-sectional sample of the population were used to describe clinical presentations of disease. A smaller case–control study of restrained seals enabled detailed clinical examination. Animals with and without clinical disease were selected. Samples were collected to determine whether disease agents were present and for haematology and serum biochemistry. In addition, opportunistic post-mortem examinations and necropsy of recently dead seals were undertaken.

8.2 Methods

8.2.1 Study Location and Population

Davis Station (68°31′S, 78°12′E) in the Vestfold Hills is inhabited year round, with approximately 20 people in winter and 80–100 in summer. A policy of minimal impact is followed with the objective of preventing disturbance and harm to wildlife and the environment.

The Vestfold Hills is an ice-free coastal area of ~400 km^2, much of it deeply dissected by fjord-like inlets. A detailed description of the region and its biology has been provided by Johnstone et al. (1973). The coastal sea-ice as well as that of the inlets is stable and provides a platform for a large population of breeding Weddell seals. Diet and foraging behaviour of Weddell seals in the Vestfold Hills region is described by Lake et al. (2003), and the annual breeding cycle by Lugg (1966) and Green et al. (1995a). Females give birth from September to November. Males compete for mating opportunities from this time and this is followed by the annual moult. After weaning at about 6 weeks, pups are rarely seen until they reappear at breeding colonies 4–6 years later. Stewart et al. (2000) suggest that some may spend time in the pack-ice.

The Weddell seals investigated were part of a known-age population established through annual tagging of pups over a 30-year period. Re-sighting of known-age animals has shown a high breeding fidelity to the site of birth (Green et al. 1992; Green et al. 1995a; Lugg 1966). Pup survival to 1 year, over 24 years of study, varied in the range 21.9–90.0% (mean 50.6% ± 4.2%) with a periodicity that suggests a possible relationship to a cyclic event such as the Antarctic Circumpolar Wave and the Southern Oscillation (Burton 1998; White and Peterson 1996). Juvenile and sub-adult seals up to the age of 6 years have an average survivorship from birth of 20% after which survivorship slowly declines. Survival beyond 15 years is thought to be unusual but some seals in this population have been re-sighted into their late 20s (Burton, unpublished data). The population has a stable rate of pup production (ca. 150–170 pa) with apparent fluctuations in adult populations attributed to variation in foraging effort required in some seasons (Green et al. 1995b). The study was carried out in November–December 1999, by which time most pups had been born and were 0–4 weeks old.

8.2.2 Study Design

Prior re-sight information was used to ensure that the sample was not biased towards an age class or location, and helicopters were used to ensure that less accessible areas of sea-ice were represented. Re-sight data for the region indicated that there were approximately equal numbers of seals in inlets and offshore locations, and approximately equal numbers of males and females, with juveniles and

males being mostly offshore and females mostly inshore. Survivorship curves indicate three approximately equal age groups – pups, sub-adults (1–6 years) and adults (≥7 years).

The non-invasive observational part of the study was conducted as a cross-sectional study (Thrushfield 1995) with a systematic random sampling design. The study aimed to collect data from at least 120 animals to enable an estimate of prevalence for a presentation ≤10% to ±5% (at the 95% confidence level) with 80% power (Cannon and Roe 1982; Thrushfield 1995). To achieve this, observational data were required from every fifth seal encountered, based on an average population of 700 seals. To avoid under-representation of males and juveniles that occur singly or in small groups, the first seal at each location was sampled. In the event, a much larger sample was actually achieved, comprising 342 adults (205 females, 137 males) and 156 pups, making it possible to estimate age-group and sex-specific prevalences to this level of confidence and power.

The more detailed clinical observations and sampling of restrained animals were designed as a case–control study (Thrushfield 1995) matching approximately for age, sex and location. The case–control approach was chosen to enable testing whether there was an association between laboratory and clinical evidence of disease. Cases were initially defined as any inflammatory disease present on the basis of the non-invasive observations and included advanced periodontal disease. Sizes of case and control categories were calculated to provide 80% power and 5% precision so that an exposure of interest with an odds ratio of 10 (i.e. a difference of this order) could be detected between cases and controls (Thrushfield 1995). This required sampling from at least 24 case–control pairs. Subsets of the original case category, respiratory foam present (7) and other respiratory discharges present (12), were examined further, increasing the power of the study to 99.9% and 91.5%, respectively (Schlesselman 1982). Some additional samples were available from necropsied animals. The reference population was stratified into the three age classes described above (pups, juveniles and adults up to 6 years of age, adults 7+ years), sex and within and outside Long Fjord (the most favoured haul-out location).

Twenty-five cases (8 pups, 4 sub-adults and 13 adults) of observed inflammatory disease (respiratory discharges and/or foam, ocular discharge, periodontal disease or external wounds) and 30 controls (12 pups, 12 sub-adults and 6 adults) were clinically examined in detail.

8.2.3 Non-Invasive Clinical Observations

Tags were used to obtain age and non-invasive observations to provide information on sex, reproductive status, body condition, attitude and respiratory pattern and discharges. Dental, ocular, coat and skin, genitourinary, gastrointestinal and musculoskeletal abnormalities were also noted (Fig. 8.1).

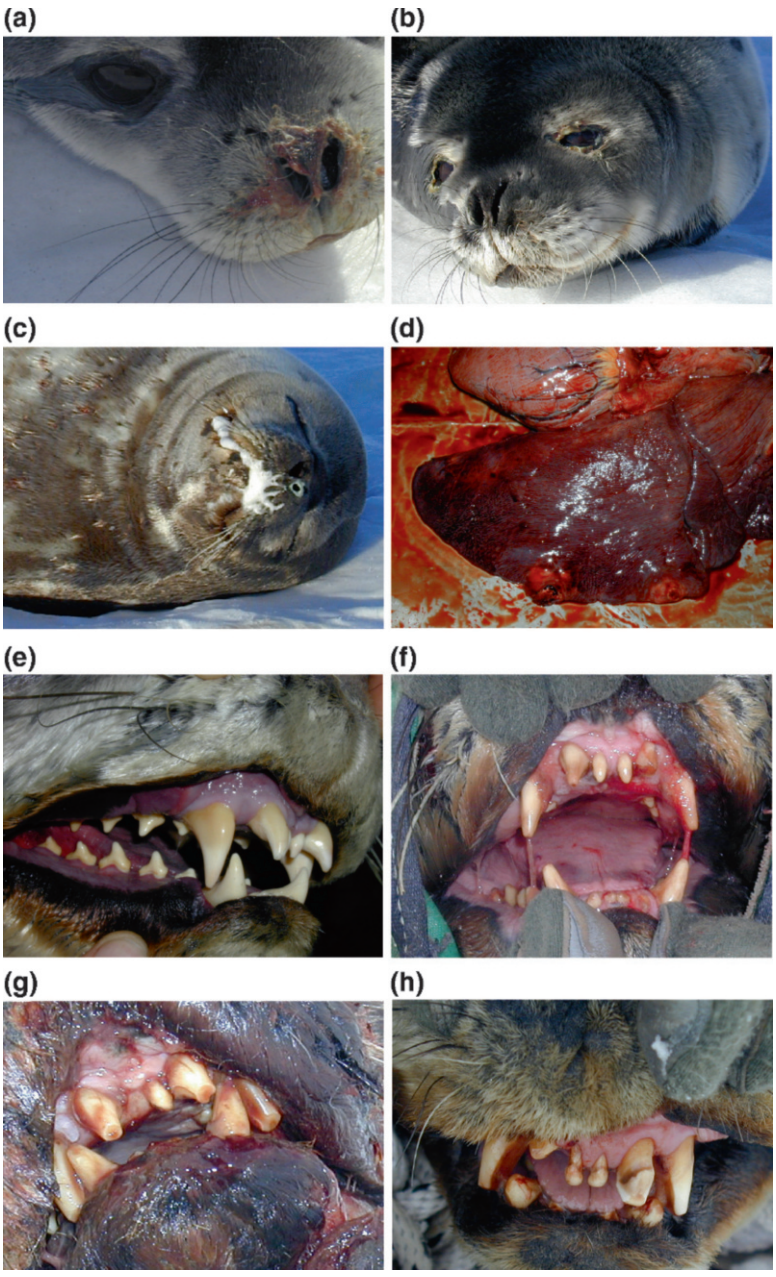

Fig. 8.1 Plates of pathology observed in Weddell seals in Vestfold Hills region during November–December 1999: (**a**) blood-stained purulent nasal discharge (and normal lacrimation); (**b**) muco-purulent ocular discharge; (**c**) seal producing foam discharge at mouth and nose; (**d**) pulmonary abscesses in necropsied 9-year-old cow; (**e**) 9-year-old cow: minor wear of tooth tips, staining; (**f**) 13-year-old male: teeth staining, worn tips, black spots and ulceration and lower central incisors also worn; (**g**) 15-year-old male: receding ulcerated gums with worn tips and facets created by opposing teeth, pulp cavities exposed; (**h**) 21-year-old female: gum recession, exposed tooth roots, lower incisors worn to gum, greater overall wear on incisors on the left side

Multiple observations of system-specific pathology were grouped as system-specific disease syndromes. Animals displaying purulent and sanguineous respiratory discharges were recorded as having respiratory discharge. Production of foam was noted separately because of its possible association with phocine herpes virus (PHV1) (Harder et al. 1991). Seals showing external signs of infection or trauma to the eye were recorded as having ocular disease. Eyelid spasm was also recorded. Although it may represent only a mild transient irritation of the eyes, it may also be the only external evidence of a painful pathological process. Multiple observations of affected seals in this study confirmed that it precedes purulent conjunctivitis and corneal scarring.

Non-invasive observations of dental disease, made when seals yawned or vocalised, were possible only in 31% of seals investigated. Wounds were recorded as present or absent with no attempt to identify cause or distinguish between minor or major wounds. Purulent vaginal discharges were differentiated from serous, serosanguinous post-parturient and mucoid pre-mating discharges.

8.2.4 Detailed Clinical Examination and Sampling of Restrained Seals

Seals were restrained with a canvas head bag as described by Stirling (1966). Large and active seals (n = 14) were hand-injected with midazolam (Roche) (0.15–0.30 mg kg^{-1} i.m. as necessary). Lower doses were used for aged or debilitated animals, including those with signs of respiratory disease. On two occasions, ketamine hydrochloride (Parnell) at 0.2 mg kg^{-1} was also administered to achieve the desired level of sedation. No problems were encountered, and this level of sedation was adequate for sample collection and for general examination. Heart rate and respiratory rate were monitored pre-restraint, by counting thoracic movements and the clearly visible apex beat, and throughout the procedure for chemically restrained animals. Care was taken to prevent anaesthetised seals from re-entering water until coordinated, purposeful forward movement was made and the head could be raised.

Tag number, age (from tag database), sex, body weight, axial girth and standard length were recorded for all restrained animals. Mouths were examined for teeth wear, fractures, exposed pulp cavities, gingivitis and periodontal disease, and photographs were taken of pathology. Detailed visualisation of internal structures of the eye was not possible in the field conditions. The coat was examined with particular attention for ectoparasites around the tail and hind flukes. Blood was collected from the extradural vein in the lumbar spine using a 19G 3.5 in. spinal needle and 20 mL syringes. Microbiological swabs were taken from the deep nasal and wound sites and from the rectum. An additional deep nasal swab and fresh faeces per rectum were collected for parasitology.

To avoid transmission of pathogens between animals, head bags were soaked in the virocidal disinfection solution Virkon (Antec International) after each use and a new pre-prepared sampling kit was used for each animal.

8.2.5 Post-Mortem Examinations

A total of 11 seals were found dead during the study. Two lactating cows were discovered shortly after death in nursing colonies in Long Fjord, one of which had not been attacked by scavengers and was transported to Davis Station for post-mortem examination. A weanling was found in good condition on melting ice in Long Fjord but was not recovered. Five of eight neonate carcasses were well preserved, although frozen, and were examined post-mortem.

Sections of tissue were preserved in 10% buffered formalin; microbiological material was either frozen, or swabs were placed in vials of glycerol broth before freezing at −80°C. Heart blood was collected where possible, centrifuged and also stored at −80°C. Tissue for virus isolation and toxicology was also collected.

8.2.6 Laboratory Procedures

Because many tests have not been validated for wildlife species, samples were sent to laboratories that had had previous experience investigating and reporting specific disease agents in marine mammals, and, where possible, multiple diagnostic tests were used.

8.2.6.1 Bacterial Culture

Microbiological samples were placed in glycerol broth with 10% serum immediately after collection in the field and frozen to −80°C. Fifty-six rectal swabs, 54 respiratory swabs and 9 wound swabs from the study population, and 10 abscess and tissue swabs from a necropsied adult seal with pulmonary abscesses were cultured and isolates identified using standard methods (Cowan 1993) by Veterinary Pathology Services, now IDEXX Laboratories, Adelaide, South Australia.

Rectal swabs were cultured using the following selective and non-selective media and conditions:
Campylobacter isolation:

- CAMP plate incubated at 42°C under microaerophilic conditions for 2 days.

Yersinia isolation:

- SYN plate incubated at 30°C under aerobic conditions for 2 days.

Salmonella isolation:

- Direct inoculation onto XLD agar at 37°C under aerobic conditions for 2 days.
- Rappaport's and Selenite broth enrichment under aerobic conditions at 37°C for a day followed by inoculation on XLD.

Coliforms and non-selective culture:

- Sheep blood agar incubated at 37°C under aerobic conditions for 2 days.
- McConkey agar incubated at 37°C under aerobic conditions for 2 days.

Respiratory swabs were cultured using the following media and conditions.

Gram-negative organisms and non-selective culture:

- Horse blood Agar/McConkey plate incubated at 37°C aerobically for 2 days.

Gram-positive organisms:

- CNA (blood agar + nalidixic acid) incubated at 37°C aerobically for 2 days.

Fastidious organisms:

- Chocolate plate incubated 2 days at 37°C with CO_2.

API Coryne was used for identifying the *Coryne* and *Gardnerella* spp. organisms. Necropsy tissues and abscess samples were cultured on

- Selective anaerobe agar (anaerobic) at 37°C for 2 days.
- Sheep blood agar (aerobic and anaerobic) at 37°C for 2 days.
- CNA (blood agar + naladixic acid) incubated at 37°C aerobically for 2 days.
- McConkey agar incubated at 37°C aerobically for 2 days.

Mycobacterial culture was undertaken on 56 rectal swabs and 9 wound swabs from the study population, and abscess and tissue swabs (10) from the necropsied adult seal with pulmonary abscesses. Before analysis, samples were stored in glycerol broth at −80°C without specific mycobacterial preserving additives. Pulmonary abscess and lung tissue were submitted to the Australian Reference Laboratory for Bovine Tuberculosis, Agriculture Western Australia, Perth (ARLBTB). Radiometric (BATEC) culture and conventional culture were undertaken for *Mycobacterium bovis* and *Mycobacterium* spp. using standard techniques (Corner 1993). Samples were macerated, decontaminated with 0.075% hexadecylpyridinum and centrifuged. The supernatant was inoculated onto egg-based media and incubated at 37°C aerobically for 12 weeks. Smears made of bacteriological samples collected during necropsy were stained with Ziehl–Neelsen (acid fast) and examined at Veterinary Pathology Services, (now IDEXX Laboratory), and histological sections of pulmonary lesions and lung tissue, also stained with Ziehl–Neelsen, were examined at the Australian Registry of Wildlife Pathology.

Brucella spp. culture was undertaken on pulmonary abscesses and lung, liver, kidney, pancreas, thoracic lymph node and spleen from the necropsied adult by the Australian Animal Health Laboratory, Geelong, Victoria (AAHL) and ARLBTB. Each sample was cultured using standard procedures (Corner and Alton 1993) for the presence of *Brucella* spp. as follows.

- Serum dextrose agar incubated at 37°C aerobically for 2 days.
- Diphasic media incubated at 37°C aerobically with 10% CO_2.

Other pathogens:

- Horse blood agar incubated at 37°C aerobically for 2 days.
- McConkey agar incubated at 37°C aerobically for 2 days.

8.2.6.2 Bacterial Serology

Sera from 35 sub-adult and adult seals and from the necropsied adult cow were tested at ARLBTB for Mycobacteria by TB enzyme-linked immunosorbent assay (ELISA) using commercial bovine purified protein derivatives (PPD) and avian PPD as antigen and protein A conjugate. Each sample was tested in duplicate against the two PPD antigens.

Adult sera (35 samples) were tested for Brucella at ARLBTB using the Rose Bengal plate test (RBPT) and complement fixation test (CFT) using standard methods (O.I.E. 1996) and the serum agglutination test (SAT) (Corner and Alton 1993). Because of the high number of reactors in the SAT, the tests were repeated using incubation at 56°C rather than at the standard 37°C to validate the results.

Fifty-seven samples, representing sera from all sampled seals and the necropsied adult, were tested at AAHL using CFT at 1:2 dilution and the *Brucella abortus* competitive ELISA at a 1:10 dilution (Nielsen et al. 1995). This protocol has been used in other serological studies of marine mammals (Jepson et al. 1997; Nielsen et al. 1996, 2001; Retamal et al. 2000), as it allows the detection of *Brucella*-specific antibodies in a number of animal species and also distinguishes between *Brucella* spp. and other related Gram-negative bacteria.

8.2.6.3 Antibiotic Resistance

Isolates from swabs were tested for resistance to selected antibiotics using antibiotic susceptibility discs for ampicillen, amoxycillen, clavulonic acid, cefotaxime, cephalothin, chloramphenicol, co-trimazole, doxycycline, enrofloxacillen, erythromycin, gentamycin, lincospectin, naladixic acid, neomycin, penicillin, sulphur–trimethoprim and tetracycline.

8.2.6.4 Virology

Virological examinations were conducted by the Institute of Virology, Erasmus University, Rotterdam, on sera from 61 seals including heart blood collected during necropsies. Two-fold dilutions of serum samples were tested for their ability to neutralise 60 $TCID_{50}$ of canine distemper virus (CDV, Bussel strain), phocine distemper virus (PDV) and PHV1. After incubation for 4 days, virus-neutralising antibodies were determined microscopically on the basis of cytopathic changes and expressed as the reciprocal of the highest serum dilution still giving 100% reduction of cytopathic changes. Sera were also tested with an ELISA specific for PHV2 and influenza virus A (A/Nanchang).

Lung tissue from the necropsied seal was examined for morbillivirus and PHV1 nucleic acid by reverse transcriptase polymerase chain reaction (RT-PCR) at the Institute of Virology, Erasmus University, using techniques described by Jensen et al. (2002).

8.2.6.5 Haematology

Haematocrits and blood smears were made in the laboratory at Davis Station, and serum and plasma were separated and frozen to −80°C. Whole blood preserved with calcium EDTA was made into smears, air-dried and stained with Diff Quick (Lab-Aid). Packed-cell volume was measured using 9-µl heparinised microhaematacrit tubes, and samples were centrifuged at 16,000 rpm for 120 s. Preserved blood smears were read at Veterinary Pathology Services (IDEXX Laboratories), and white cell counts estimated from the haematocrit. Cells were also examined for haemoparasites.

8.2.6.6 Serum Biochemistry

A limited biochemical profile including total protein, albumin, serum electrophoresis, urea, creatinine, aspartate transaminase (AST), alkaline phosphatase (ALP) and tetraiodothyronine (T4) was performed by Veterinary Pathology Services (IDEXX Laboratories) on 30 samples. Half were from animals with no evidence of inflammatory disease and half were animals that had wounds, periodontal disease, respiratory or ocular discharges and viral titres. Approximately equal numbers of animals in each age class and gender were selected. Tests chosen were potential indicators of inflammation, necrosis and organ dysfunction and for which other literature from Weddell seals existed (Seal et al. 1971; Schumacher et al. 1992; Yochem et al. 1999). Electrophoresis was performed using a Helena Laboratories Titan Gel Serum Protein System Kit and a Quick Scan Junior TLC Densitometer. T4 was measured using a Biomedia Immulite Total T4 Test Kit. Other biochemistry was performed with an Olympus AU400 analyser. At the time of analysis, serum samples had been frozen for almost 18 months, and some samples investigated by electrophoresis did not yield reliable results.

8.2.6.7 Parasitology

Deep nasal swabs were examined as wet preparations under a cover slip as soon as possible, specifically for evidence of mites or ascarids. Zinc sulphate flotation of faecal samples (Coles 1986) was performed within 24 h. Ova and protozoa were measured and photographed, and parasitological samples were preserved in 2% potassium dichromate, ethanol or 10% buffered formalin as required. Ectoparasites were preserved in ethanol and necropsy material in 10% buffered formalin.

8.2.7 Epidemiological and Statistical Analysis

The required sample-sizes were calculated using the epidemiological software Win Episcope 2.0 and FreeCalc (Cameron 1998) and the tables of Cannon and Roe (1982). Calculations of prevalence and incidence, measures of association (odds ratio and relative risk) and agreement between tests (kappa) followed Thrushfield (1995). Execution of case–control studies with uneven case:control ratios was as described by Schlesselman (1982). Descriptive statistics were calculated using Number Cruncher Statistical Systems (NCSS) 2000 (Hintze 1998).

Two-factor analysis of variance using the generalised linear model for binomial data was performed using S-plus (Mathsoft Inc. Cambridge MA, USA) to examine the effects of time (weeks into the study) and age on the incidence of clinical presentations of disease and between age and sex on biochemical and haematological parameters. A Chi-squared test was used to analyse frequencies of observations with Yates correction applied to calculations relating to associations between disease syndromes and risk factors. The significance of the measures of associations (odds ratio) was tested using a two-tailed Fisher's exact test at 95% confidence limits. A multiple linear regression model was developed to describe the relationship between (1) respiratory discharge and (2) respiratory foam and their potential predictors in both the cross-sectional and case–control studies. In the former, time was categorised into the 8 weeks of the study and the categorical data was transformed with logistic transformation. Mixed multiple linear regression was also used to examine the effect of multiple and simultaneously confounding and interacting risk factors as indicated by the variation in odds ratio within summary tables for respiratory discharges and foam production in the case–control study. Factors under consideration were both continuous (biochemical and haematological parameters, body weight) and categorical (pathogen present or absent in laboratory results, concurrent clinical signs present or absent). To simultaneously consider both types of data, categorical data was transformed with a logistic transformation.

8.3 Results

8.3.1 Evidence of Clinical Disease from Non-Invasive Observations

During the time of the study, males and females favoured different locations ($\chi_2^2 = 46.62$, $p < 0.001$). Males were more numerous on sea-ice further offshore, and females were more numerous in Long Fjord and Tryne Fjord.

The most frequently observed disease syndrome was external wounds (Table 8.1), which affected 35% of the total adult population (59% of adult males and 20% of females affected). Pups had much fewer wounds than adults. Approximately 21% of adults showed obvious signs of tooth wear. Respiratory signs were apparent in about 15% of the adult population and 18% of pups.

Table 8.1 Prevalence (and 95% confidence interval) of observed disease syndromes in Weddell seals in the Vestfold Hills region, Antarctica, November–December 1999

Observed disease syndrome		Adults Males (n = 137) Females (n = 205)		Pups (n = 156[a])	
All respiratory signs		15.49%	(±3.84)	18.47%	(±6.07)
Respiratory discharge only	Total	12.57%	(±3.29)	13.37%	(±5.32)
	Male	10.22%	(±5.07)		
	Female	14.15%	(±4.77)		
Foam production only	Total	11.69%	(±3.41)	5.09%	(±3.44)
	Male	14.6%	(±5.91)		
	Female	9.6%	(±4.06)		
Respiratory discharge and foam production	Total	3.5%	(±1.95)	0.006%	(±1.21)
All ocular disease	Total	4.97%	(±2.31)		
	Male	5.11%	(±3.69)	6.4%	(±3.83)
	Female	4.88%	(±2.95)		
Purulent conjunctivitis	Total	2.3%	(±1.59)	5.73%	(±3.64)
Dental condition					
Obvious tooth wear	Total	21.5%	(±7.78)		
Periodontal disease	Total	2.8%	(±3.13)		
	Male	4.93%	(±9.49)		
	Female	2.31%	(±3.16)		
External wounds	Total	35.38%	(±5.07)		
	Male	59.0%	(±8.15)	3.2%	(±2.75)
	Female	20.0%	(±5.48)	1.3%	(±1.77)
Purulent vaginal discharge	Female	0.0075%	(±1.04)		

[a]Note that this is approximately the entire pup population

Different clinical disease syndromes occurred concurrently in some animals and the associations are statistically significant for the combinations of respiratory discharge and foam ($\chi_2^2 = 12.599$, $p < 0.001$), ocular pathology ($\chi_2^2 = 10.817$, $p < 0.001$) and wounds ($\chi_2^2 = 3.192$, $p < 0.05$); and for foam production and wounds ($\chi_2^2 = 26.269$, $p < 0.001$).

The factor, Location, was highly significant for respiratory discharge (Fig. 8.2) in adults ($\chi_2^2 = 42.35$, $p < 0.001$), with greater than expected numbers of cases in Long Fjord and less than expected in the sea-ice locations. Location was also highly significant for ocular discharge in adults ($\chi_2^2 = 12.13$, $p < 0.001$), with greater than expected numbers of cases in Long Fjord and also in Tryne Fjord. Location was not significant for foam production or external wounds in adults. Location was not significant for any clinical signs in pups.

The incidence of respiratory discharge (Fig. 8.3) changed significantly during the period of study ($p < 0.001$): for adults it fell throughout the study, while for pups it rose and then fell. The incidences of foam production ($p < 0.001$) and external wounds ($p < 0.001$) were higher in adults than in pups throughout the study. There was a significant interaction between the age class and week of study for incidence

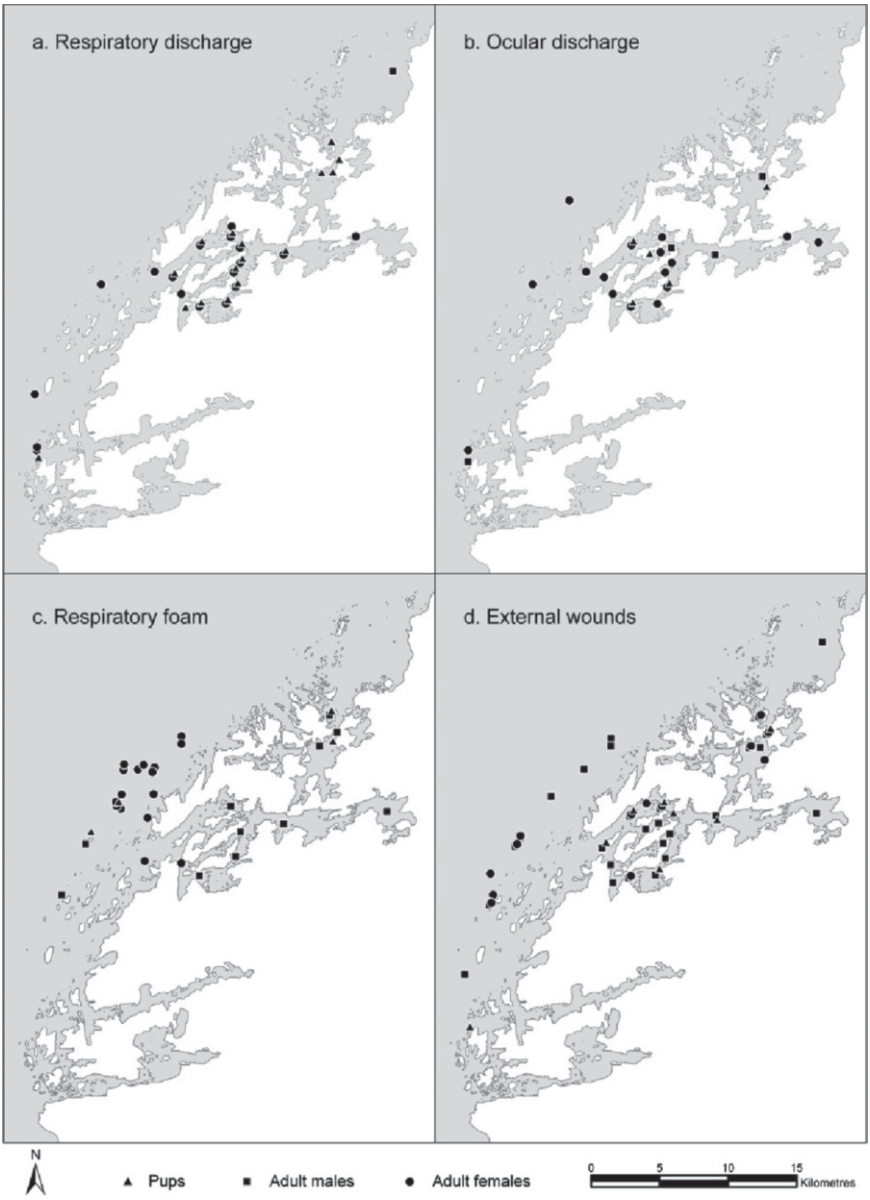

Fig. 8.2 The distribution of Weddell seals with respiratory discharge, ocular discharge, foam and external wounds examined in the Vestfold Hills region, Antarctica, November–December 1999

of ocular discharge ($p < 0.001$), indicating that the incidence of ocular discharge in adults and pups varied differently during the study.

Mixed linear regression analysis of the cross-sectional study indicates that age class (adults) and wounds are good predictors for respiratory foam ($R^2 = 0.90$;

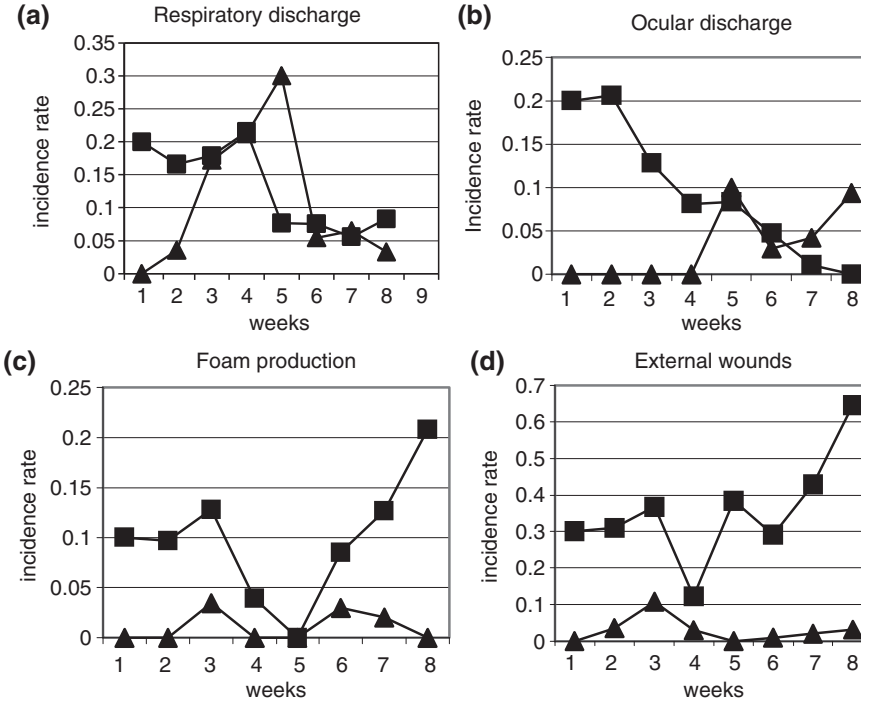

Fig. 8.3 Weekly incidence of (**a**) respiratory discharge, (**b**) ocular discharge, (**c**) foam production and (**d**) external wounds in Weddell seal adults (*filled square*) and pups (*filled triangle*) in the Vestfold Hills region, Antarctica, November–December 1999. At the beginning of the study pups were approximately 2 weeks old ± 2 weeks

F-statistic = 22.7361; *P*-value = 0.0031). The best predictor for respiratory discharge is ocular disease; however, it was not significant (R^2 = 0.22, *F*-statistic = 1.6947, *P*-value = 0.2407) in this study.

8.3.1.1 Detailed Clinical Examination

No animal observed or examined was in poor body condition. The relationships between body weights and known age (n = 34) are shown in Fig. 8.4. A full description of the relationship between standard length, axial girth measurements and mass of sub-adult and adult seals is given elsewhere.

The range of conditions used to define animals with ocular, respiratory discharges and foam production is illustrated in Fig. 8.1. Seals with respiratory and

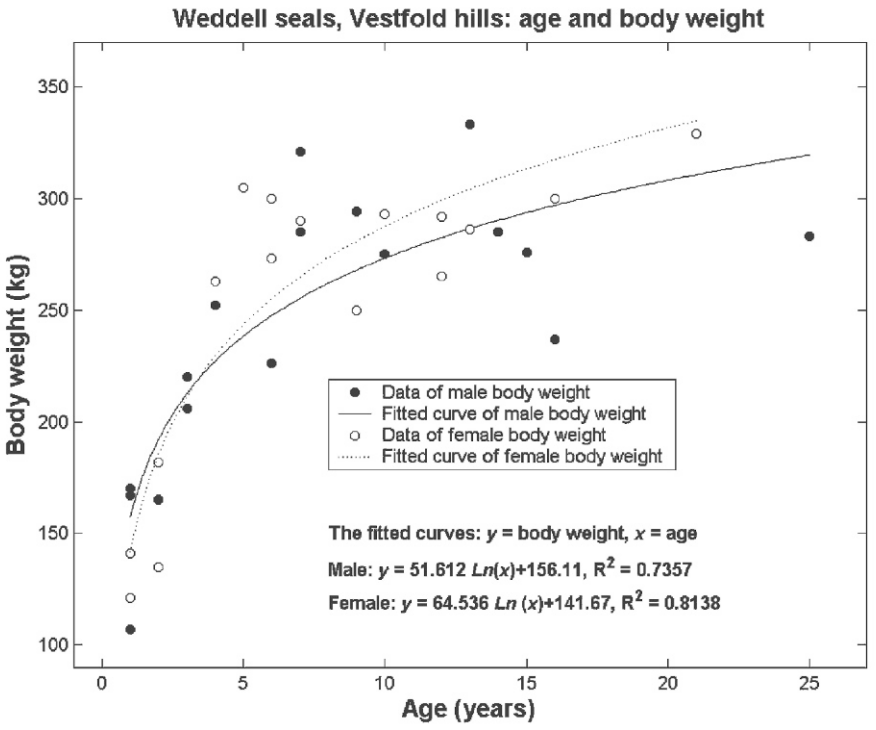

Fig. 8.4 Body weights of sub-adults and adult Weddell seals of known age in the Vestfold Hills region Antarctica, November–December 1999

ocular discharges did not demonstrate respiratory distress post restraint or sedation, although restraint was sometimes followed by further expectoration of foam or respiratory discharge.

The progression of tooth wear and periodontal disease observed in this study is illustrated in Fig. 8.1. Flattening of the tips of the upper canines and second incisors was evident in seals as young as 2 years of age. Staining, the wearing of facets by opposing teeth and exposure of pulp cavities or reparative dentine was notable on animals from 7 to 10 years of age. Gingivitis and gingival ulceration, gum recession and loosening of teeth became obvious in animals over 9 years of age. All animals seen over 13 years of age had some dental disease. Two ‘aged’ animals were examined. A non-lactating 21-year-old female had more notable periodontal disease than teeth wear, but advanced teeth wear was evident in the 25-year-old male. Wearing of the lower and central incisors in advance of the longer ice-abrading teeth, presumed solely due to feeding and catching prey, occurred in some animals but was not apparent in all older animals.

8.3.1.2 Laboratory Tests

No bacteria of potential human origin or of zoonotic potential were isolated. No positive culture results were obtained for *Mycobacterium* spp., *Salmonella* spp., *Campylobacter* spp. or *Brucella* spp. There was no evidence of atypical antibiotic resistance from any of the bacterial isolates. Rectal swabs were unintentionally omitted from antibiotic sensitivity testing.

Isolates from rectal samples include haemolytic *E.coli* ($n = 37$), *Streptococcus* of groups C ($n = 7$), D ($n = 2$) and G ($n = 1$) and *Yersinia* spp. ($n = 1$; note, however, *Y. ruckeri*, a notifiable disease of fish, was positively excluded). No significant bacteria were cultured from 12 samples. Deep nasal swabs recovered *Corynebacterium* spp. ($n = 37$), *Staphlococcus aureus* ($n = 15$), Group C β-haemolytic *Streptococci* ($n = 2$), Group F β-haemolytic *Streptococci* ($n = 6$) and haemolytic *E. coli* ($n = 1$). No growth occurred from seven samples. One vaginal swab grew haemolytic *E. coli* and *Actinomyce pyogenes.*

A. pyogenes was cultured from all wound samples, and *Enterococcus* spp. also grew from one sample. *Fusobacterium necophorum*, *Bacteroides* spp. and *Bacillus* spp. were cultured from three of five samples of lung abscesses from the necropsied adult. No acid-fast bacteria were detected in the smears from any of the specimens.

All adult sera tested were negative for *Mycobacterium* spp. (i.e. optical density was above the cut-off level of 0.095 for bovine PPD serology).

Serological screening for *Brucella* spp. at two laboratories both indicated high numbers of positive reactions (Table 8.2), with only one individual (a male pup) negative to both CFT and ELISA suggesting substantial agreement between tests above that expected by chance (kappa = 0.654). Neither laboratory was able to culture the organism from necropsy material.

Viral neutralizing titres (VNTs) of less than 20 were considered to be negative in serology tests for PHV2, CDV or AI. One adult, an unrelated pup and two dead pups had a VNT of 20 against PDV. Two pups, a sub-adult and five adults had a VNT of 20 against PHV1. These are low, equivocal titres. The 9-year-old cow necropsied with pulmonary pathology was negative to all five viruses, and the PCR of tissues was negative for herpes or morbillivirus.

Nasal cytology and histopathology of lung tissue did not reveal any respiratory parasites. Twelve of 32 faecal samples had at least one species of nematodes present,

Table 8.2 Results of serological tests and culture for *Brucella* organisms from this sample of Weddell seals, Vestfold Hills region, November–December 1999

Test	Laboratory	Result
Competitive-ELISA (cELISA)	a.	55/57 Positive
Compliment Fixation Test (CFT)	a.	56/57 Positive
Compliment Fixation Test (CFT)	b.	35/35 (all adults) Positive
Serum Agglutination Test (SAT)	b.	24/35 Positive
Rose Bengal Plate Test (RBPT)	b.	22/35 Positive
Bacterial culture	a.,b.	Negative

[a]Australian Animal Heal Laboratory (AAHL)

[b]Australian Reference Laboratory for Bovine Tuberculosis (ARLBTB)

some with and some without protozoa, 4 demonstrated protozoa only and 16 samples were negative. One animal was infested with lice, similar to *Antarctophithirus ogmorhini* described by Murray et al. (1965).

Table 8.3 summarises the estimated white cell counts (EWCC) in relation to clinical or serological presentation. Table 8.4 summarises haematological and biochemical parameters in relation to age class.

The distribution of these parameters in the case and control groups did not demonstrate a significant effect of inflammation. Analysis of variance was used to examine the different effects of age class and sex and their interaction. There was significant variation between age classes for PCV (p = 0.009), urea (p = 0.030), ALP (p = 0.0001), total globulins (p = 0.001), α-globulins (p = 0.050) and γ-globulins (p = 0.0006).

There was significant variation between the sexes for PCV (p = 0.0014) and albumin (p = 0.0363) only. Females had higher PCV values than males (0.63 ± 0.06; 0.57 ± 0.06). Males had higher albumin values than females (31.7 ± 4.7; 29.0 ± 5.4). There was significant interaction between age class and sex for ALP (p = 0.0364) and AST (p = 0.0219) and for γ-globulins (p = 0.019). Mean AST values were highest in sub-adult males and lowest in male pups, while values for females remained relatively stable. ALP values declined steadily with age in males, but sub-adult values were lowest for females. While γ-globulins decreased sharply from adults to pups for males, levels in female declined less rapidly.

8.3.1.3 Post-Mortem Examinations

Adults The tagged cow found dead in a nursing colony in Long Fjord was 9 years old, and although she was recorded to have given birth, she had no pup beside her. The seal weighed 250 kg, with an axial girth of 144 cm and a standard length of 238 cm. There were no external wounds or evidence of scavenging and the carcass was not frozen. White foam was present around the chin and throat but no other discharges

Table 8.3 Estimated white cell counts associated with clinical or laboratory conditions identified in Weddell seals of the Vestfold Hills region, November–December 1999

		Estimated white cell count × 10^{-9} WBC L^{-1}		
Clinical or serological presentation	*N*	Mean	s.d.	Range
Wounds	13	11.4	4.2	6.4–20.8
Respiratory discharge	12	9.1	3.9	6.0–12.6
Foam	7	11.5	5.0	6.0–20.8
Ocular disease	3	11.2	7.3	6.4–19.6
Periodontal disease	5	12.48	6.5	6.8–22.4
Positive faecal flotation ($ZnSO_4$)	8	10.75	5.9	6–22.4
PDV1[a]	2	16.4	2.3	16.8–20.0
PHV1	5	12.72	6.6	5.6–20.8

[a]Only 2 samples with VNT ≥ 20 were ante-mortem

Table 8.4 Summary of biochemical and haematological parameters measured from Weddell seals of the Vestfold Hills region, Antarctica, November–December 1999

Numbers of seal samples = (adults/ sub-adults/pups)	Total mean ± s.d. range	Adults mean ± s.d.range	Sub-adults mean ± s.d. range	Pups mean ± s.d. range
EWCC × 10^{-9} WBC/L (19/15/20)	11.704 ± 5.686 4.8–26	14.1 ± 5.8 6.4–26	11.3 ± 6.4 4.8–26	9.9 ± 4.5 5.6–19.2
PCV (20/15/20)	0.595 ± 0.069 0.43–0.70	0.59 ± 0.06 0.5–0.6	0.64 ± 0.06 0.52–0.7	0.56 ± 0.06 0.43–0.64
Total protein g/L (10/10/10)	83.2 ± 17.303 4–120	91.4 ± 17.7 62–120	84.9 ± 12.9 58–101	73.3 ± 4.1 47–106
Albumin g L^{-1} (10/10/10)	30.367 ± 5.162 21–40	27.5 ± 4.5 21–33	29.9 ± 4.3 21–36	33.7 ± 5.1 23–40
α-Globulins g L^{-1} (3/6/8)	11.853 ± 2.067 8–16.5	9.8 ± 1.3 8–11	13.0 ± 2.2 10–16.5	12.1 ± 1.5 10–14
β-Globulins g L^{-1} (3/6/8)	15.088 ± 2.195 12–20	16.0 ± 2.7 14–20	15.5 ± 2.8 12–18	14.2 ± 1.8 13–16
γ-Globulins g L^{-1} (3/6/8)	21.353 ± 14.438 3–54	39.5 ± 14.4 22–54	23.5 ± 6.9 16.5–35	9.2 ± 3.8 3–15
Albumen:globulin (10/10/10)	0.973 ± 0.980 0.166–4.333	0.279 ± 0.128 0.166–0.455	0.607 ± 0.243 0.285–1.0	1.683 ± 1.2 0.8333–4.333
Urea mmol L^{-1} (10/10/10)	9.727 ± 4.054 4.1–21.3	11.9 ± 4.3 6.6–21.3	9.9 ± 3.4 4.9–15.2	7.3 ± 3.3 4.1–14
Creatinine mmol L^{-1} (10/10/10)	0.122 ± 0.021 0.09–0.17	0.137 ± 0.02 0.11–0.7	0.11 ± 0.02 0.09–0.13	0.119 ± 0.02 0.09–0.06
Alkaline phosphatase U L^{-1} (10/10/10)	163.267 ± 98.379 43–414	116.1 ± 58.9 43–204	111.1 ± 65.4 44–249	262.6 ± 83.6 161–414
Aspartate transaminase U L^{-1} (10/10/10)	5.533 ± 7.176 0–31	4.9 ± 3.8 0–11	9.0 ± 10.4 0–31	2.7 ± 4.4 0–14
T4 nmol L^{-1} (9/10/10)	29.466 ± 8.169 17–59	27.0 ± 5.6 18–35	26.8 ± 6.5 17–36	34.6 ± 9.8 26–59

EWCC Estimated White Cell Count, *WBC* White Blood Cell, *PCV* Packed Cell Volume

were present at time of necropsy. The nipples were recessed, and 2 mL of milk was collected from the base of one nipple. Teeth were in good condition with points worn on upper canines and lateral incisors and minimal wear on points of lower incisors and lateral canines.

Multifocal acute to sub-acute pulmonary abscesses and multifocal chronic pleural abscesses with diffuse pulmonary consolidation, oedema and pleuritis were present in both lungs. No parasites or viral inclusion bodies were observed in any of the sections, small numbers of bacteria were present in the eosiniphillic material and cell debris was present within the collapsed and congested pulmonary parenchyma. There was a very early connective tissue response surrounding the largest lung abscess examined, suggesting that this lesion was roughly 2–7 days old. The liver had a pattern of periacinar congestion evident throughout the tissue, and the splenic

parenchyma was congested and contained scattered discrete lymphoid follicles. Adrenal glands were mildly congested, and there was mild focal nodular cortical hyperplasia. Stomach and intestines were extensively parasitised with cestodes and nematodes causing nodular hyperplasia and ulceration.

Heart blood serology was negative to PHV1 and 2, PDV, CDV and avian influenza (AI) as well as avian and bovine tuberculosis but reacted positively to RBPT, SAT and CFT for *B. abortus*. No bacteria were cultured from heart blood. Viral PCR was performed on lung tissue and was negative for PHV1 and PDV, the only two viruses for which positive titres had been detected in the population. *F. necophorum* was cultured from three of five lung abscess swabs, but no bacteria grew from swabs of lymph nodes or other organs. Tissue samples preserved at −80°C were re-cultured 12 months later to examine specifically for *Brucella* spp. organisms. No *Brucella* organisms were isolated, but large numbers of anaerobes present in the lung abscesses were identified as *Bacteroides* spp. and *Bacillus* spp. No mycobacteria were cultured from either swabs or tissue.

This seal most likely died as a result of the spread of infection from the thoracic and pulmonary abscesses. On the basis of the microscopic examination of the tissues, a bacterial infection was suspected; however, primary periacinar hepatic congestion and parasitic, viral and fungal infections could not be completely ruled out. The airways were clear, suggesting a haematogenous rather than aerogenous spread of bacteria.

Pups

Heart blood from only two of the dead neonatal pups was collected and submitted for viral serology. Both had a VNT of 20 against PDV1 and were negative for other viruses. The lungs in one of these pups had been inflated, but acute interlobular oedema indicative of a failure of vascular integrity shortly before death was evident. The pup appeared to have only partially inflated its lungs, and marked congestion and consolidation of lungs were evident without evidence of inflammation. Of the other pups, one appeared to be stillborn; one had depleted fat reserves and its lungs congested, consolidated and interlobular spaces distended with eosinophilic fluid; the other appeared to have failed to inflate its lungs at birth and there were no inflammatory infiltrates.

8.3.1.4 Case–Control Study

No significant differences were apparent between the case (inflammatory disease observed) and control (no inflammatory disease observed) groups. Positive associations (odds ratio > 10) between potential risk factors and respiratory foam and other respiratory discharges were investigated to examine the effects of multiple and simultaneously confounding factors and interactions. Sex, age and location were matched in the design of the original case–control study and therefore were not considered independent factors in the analyses of risk factors for respiratory disease.

Mixed linear regression of the case–control study indicates that wounds, body weight, γ-globulins and AST in combination were good predictors for respiratory foam ($R^2 = 0.343$, F-statistic = 6.644, P-value = 0.0002). The maximum conditional probability p under the data is 61% and minimum 21%.

Other respiratory discharges were best predicted by respiratory foam, ocular discharge and positive β haemolytic *Streptococci* culture in respiratory samples ($R^2 = 0.153$, F-statistic = 3.128, P-value = 0.0334). The maximum conditional probability p under the data is 59% and minimum 24%.

8.4 Discussion

This study found that respiratory disease is common in Weddell seals in the Vestfold Hills, and may be fatal. Ocular disease, dental disease, parasitism and wounds are also common, this finding is consistent with previous studies (Bertram 1940; Beverley and Burton 1971; Lindsey 1937; Stirling 1969, 1971a).

The population appears to be free from most infectious diseases tested, with the exception of low and equivocal titres to PHV1 and PDV, and antibodies to *Brucella* spp. The finding of rapid development of pulmonary abscess and death in one lactating seal as well as the deaths of another lactating female and a weanling male in very good condition suggests that death is not restricted to misadventure and that disease may account for a significant proportion of the small annual mortality of mature animals. The consequences of even mild disease may be more significant for pups. Despite their limited diving experience, pups quickly extend their diving depths until they are similar to those of adults (Burns and Schreer 2000), and even mild respiratory disease could seriously compromise them while diving.

Pulmonary diseases are the most frequently encountered systemic disease of pinnipeds, usually occurring secondarily to some other problem such as parasitism or viral disease (Sweeney 1974). Excess respiratory mucous and productive cough due to a parasitic inflammatory or obstructive airway disease have been recorded in other pinnipeds (Sweeney 1978) where they were attributed to upper and lower respiratory mites (*Orthohalarachne* spp.) and true lungworms (*Otostrongylus* spp. and *Parafilaroides* spp.). Although no evidence of respiratory parasitism was found in this study, halarachne mites were recorded for the Weddell seal by King (1964).

Respiratory disease was considered here to have two presentations, foam and other respiratory discharges, although a primary infectious aetiology was not demonstrated for either presentation. Respiratory foam was linked statistically to wounds and adult age class in the cross-sectional study and to wounds, body weight, γ-globulins and AST in the case–control study. The post-mortem findings of fatal, mixed anaerobic bacterial abscesses without evidence for a primary parasitic or viral disease suggest a bacterial cause. Histological examination of tissues suggests haematic spread from other lesions such as fight wounds, dental abscesses or a heavily parasitised and ulcerated gastrointestinal tract (the latter was evident at post mortem). Gross and histopathological findings similar to those described in this

study were present in two seals found dead in Long Fjord the following year, 2000 (Gray and Rose, personal communication).

Other respiratory discharges were statistically linked to respiratory foam, ocular discharge and positive β-haemolytic *Streptococci* cultured from respiratory samples in the case–control study. Both Group C and F β-haemolytic *Streptococci* were isolated from eight respiratory swabs and an α-haemolytic *Streptococcus* was isolated from a single respiratory swab, but they were not cultured from the pulmonary abscess or lungs of the necropsied seal. Streptococcal (α-haemolytic, β-haemolytic and non-haemolytic) bacteria are reported to cause pneumonia and conjunctivitis (also peritonitis and septicaemia) in seals (Baker et al. 1980; Baker and Doidge 1984; Baker 1988; Baker and McCann 1989; Baker and Ross 1992; Higgins 2000; Skaar et al. 1994; Steiger et al. 1989) and β-haemolytic *Streptococci* cause septicaemia, bronchopneumonia, pneumonia and abscesses in harbour porpoises (Swensholm et al. 1998).

Sera tested in this study gave low positive titres (VNT = 20) for PDV and PHV1. If these represent true positives, they indicate exposure to the virus, a previous infection or the presence of maternal antibodies in the seropositive animals. However, the association between clinical signs of respiratory foam and older age groups or larger bodyweights is not necessarily suggestive of PHV1 infection, because, where it is endemic, PHV1 is expressed primarily in pups and young seals (Harder et al. 1997). The associations between clinical signs and viral titres were not statistically significant, and in future studies clarification of virus status would be better achieved using a herpes virus PCR assay on conjunctival and other swabs.

No evidence of an association between PDV and clinical signs of disease was found. The PDV results (VNT = 20) are also difficult to interpret, occurring as they did in both the dead pups sampled and a single live adult and pup. The ability to neutralise 60 $TCID_{50}$ of virus (as used for this study) is less rigorous than those using 100 $TCID_{50}$ as used in North American studies (Duignan, personal communication 2001) and previously in Antarctica (Bengston et al. 1991). Sampling from further dead pups is required, preferably using PCR examination of tissues, to investigate the involvement of virus and to establish whether there is a causal relationship between PDV and pup mortality. If the viral titres in these pups reflect only maternal antibodies associated with illness in the mother, then the virus could have a secondary role in causing pup abandonment and starvation. This is the first evidence for this virus in Weddell seals to our knowledge and requires confirmation. If confirmed, it is still uncertain what the presence of the disease may mean to the population. In some cases PDV can have devastating effects, such as in the epidemics in Northern European seals in 1988 and 2002. In contrast, retrospective surveys of North American marine mammals have indicated that the virus has emerged elsewhere without catastrophic mortality (Duignan 1999, 2000).

The rate of new cases of respiratory foam increased towards the end of the study period, corresponding to and statistically associated with the expected increase in wounds caused by aggressive contact during mating and competition between males. Incidence of foam appeared to display two peaks, also mirrored in pups,

which, if due to an infectious disease, could indicate an incubation period following contact with primary cases. The incidence of other respiratory discharges in adults (Fig. 8.3a) indicates that respiratory disease peaks before or early in the study period and then declines. Incidence of respiratory discharge in pups peaks 4–5 weeks after the beginning of the study at a time that coincides with loss of maternal antibody protection for most pups. This pattern is consistent with an infectious aetiology influenced by the increased concentration of seals in the Vestfold Hills region from October. Ocular disease in adults also appears to decline from the beginning of the study period, but in pups the first cases appeared 5 weeks (Fig. 8.3b) into the study. The higher than expected occurrence of respiratory and ocular disease at Long Fjord suggests that the breeding aggregations there may be important for disease amplification.

Serological and microbiological evidence of *Brucella* spp. infection in marine mammals has been reported in the Northern Hemisphere and Arctic since 1989 and in the Antarctic since 1998 (Retamal et al. 2000). Our study suggests that it is endemic in this population of Weddell seals; however, the clinical significance of this is as yet unknown. Evidence of antibodies to *Brucella* spp. in other Antarctic pinniped species has not been reported, and a scenario in which increased competition for residual ice and beaches (such as might occur from a continuing trend in increased global temperatures) resulting in greater transmission opportunities intra- and inter-species could be envisaged. Experimental studies on marine strains of Brucella have demonstrated low transmissibility and low pathogenicity in sheep (Perrett et al. 2004) and abortion in cattle (Rhyan et al. 2001) indicating a moderate risk to domestic stock from vagrants. Zoonotic infection in a laboratory worker in Britain (Brew et al. 1999) and two cases of community acquired human neurobrucellosis caused by marine mammal *Brucella* spp. (Sohn et al. 2003) suggest that contact with Weddell seals and their excreta must be considered a risk factor for human Brucellosis. No cases of Brucellosis have been reported in Australian Antarctic personnel to date (Ayton, personal communication 2008).

The potential for spread of *Mycobacterium tuberculosis* between continental, sub-Antarctic and Antarctic seal populations has been highlighted (Bastida et al. 1999) following the isolation of *M. tuberculosis* complex from pulmonary granulomas and other pulmonary pathology in the sub-Antarctic fur seal *Arctocephalus tropicalis*. No evidence of *M. tuberculosis* was found from 56 rectal swabs and 35 sera from the Vestfold Hills Weddell seal populations. Diagnosis of *Mycobacterium* spp. in live animals is difficult, and no single test including tissue culture is 100% sensitive (Forshaw and Phelps 1991; Cousins et al. 1993). Although we cannot say that it is definitely not present in this population, if *M. tuberculosis* is present, it is not common.

The upper canines and secondary incisors, the teeth that are most important for maintaining breathing and haul-out holes, were worn by ice abrasion in a manner similar to that described by Stirling (1969). Our examination of live seals of known age showed that periodontal disease and interdontic problems become evident after maturity, as seen in other pinniped species (Amand and Tinkkelman 1985; Anderson et al. 1979; Baker et al. 1980; Baker 1987; Hinshaw et al. 1996). Although the

aetiology in wild marine mammals is not clear, captive fur seals may develop periodontal disease from impaction of fish scales and hair within periodontal pockets (Coles, personal communication 2000). Pathology associated with periodontal disease that was obvious in these live animals would not have been evident in preserved skulls and also may contribute to mortality.

The lack of significant difference in serum biochemistry between seals within the case and control categories is consistent with the observation that all animals were in good condition and not debilitated by the range of conditions observed. However, it is also consistent with animals being wrongly classified as healthy controls if inflammatory conditions such as gastrointestinal parasitism, asymptomatic viral infections and internal abscesses were missed during field examination.

Our biochemical data are similar to those of Seal et al. (1971) and Schumacher et al. (1992) except for creatinine, ALP, AST and γ-globulins which in our study are at least twice those previously reported. Values other than AST are in the range described by Gray from Weddell seals also from the Vestfold Hills and using the same commercial laboratory (Gray, personal communication 2003). Mean globulins, total protein, urea and creatinine are higher in both adults and sub-adults, and EWCC and haematocrit are higher in adults in this current study. This may reflect the effect of specifically targeting seals with inflammatory disease in the study design, even though there was no statistical difference between the case and control categories. Gray's values of AST are greater by a factor of 10 and ALP is more than double that of this study. These values are likely to vary depending on the gender, age class and physiological state of seals in the samples (e.g. fasting/non-fasting; lactating/non-lactating) and will therefore be influenced by the choice of animals sampled.

The association of γ-globulins with respiratory foam is consistent with the hypothesis that respiratory foam is a clinical presentation of respiratory disease because γ-globulins are immunoglobulins indicative of bacterial, viral or parasitic infections. Raised levels of aspartate transaminase are generally indicative of heart, liver or muscle necrosis. Significant differences in levels of ALP, creatinine, urea and total protein were reported by Roletto (1993) between healthy harbour seals and those with respiratory disease and heart failure. Elephant seals with skin diseases and parasites also differed significantly from unaffected seals in their levels of PCV, AST, creatinine, urea and albumin values (Roletto 1993). Although these comparisons are between different species, values are of the same order with the exception of AST, for which values are 30–60 times greater than in our results. Given the differences also between our results and those of Gray (Gray, unpublished results), it would be prudent to treat the absolute values of AST in our study with caution.

Investigations of disease presentations and pathogens in the McMurdo population by Yochem et al. (1999, this volume) suggest that the frequency of skin lesions and ocular disease from the McMurdo study are approximately the same, when not stratified by age or sex, but the frequency of respiratory disease is lower than that of the Vestfold Hills population (McFarlane 1996, this study) and viral titres and antibodies to *Brucella* spp. may be low or absent.

8.5 Conclusion

This study found no evidence of direct human-mediated pathogen introduction; however, the clinical prevalence and seroprevalence data reported here for Weddell seals of the Vestfold Hills provide a robust baseline for assessing future change in this population. The role of disease and parasites in the dynamics of marine wildlife populations is being increasingly recognised (Harvell et al. 1999). Health observations need to be included in any long-term population study if trends are to be correctly interpreted and understood. Conversely, studies of disease status in populations require ecological information; for example, simple random sampling of a population may miss subtle signals of disease found only in sub-sets of the population.

The life history of Weddell seals, such as their relative isolation, may give them a degree of protection from disease, but because infection is likely to be most serious in naïve populations, this isolation also makes them to some degree more vulnerable. The movement of juvenile Weddell seals into the pack-ice (Burns et al. 1999; Stewart et al. 2000) may increase exposure to other species of pinniped and potentially to other disease agents. In addition, leopard seals, *Hydrurga leptonyx*, bachelor southern elephant seals, *Mirounga leonina*, and crabeater seals, *Lobodon carcinophagus*, are all present in the coast and fast-ice of the Vestfold Hills in summer. Weddell seals from other locations also congregate here in late summer (Green et al. 1995b), and any reduction of pack-ice and fast-ice habitats due to climate change is likely to increase multi-species contact in places such as this.

Simple precautions to reduce the chance of accidental introduction or translocation of disease agents to Antarctic wildlife should be the highest management priority. Genetic studies of Weddell seals (Davis et al. 2000) suggest that the Vestfold and McMurdo populations of Weddell seals are genetically distinct despite the circumpolar distribution of the species. Information such as this is invaluable for designing effective precautionary quarantine procedures. Beyond this, much could be learned from a network of monitored sentinel wildlife populations, but this will take the commitment of some resources. Our finding that disease was more prevalent in the lactating aggregation of cows in Long Fjord suggests that surveillance of Weddell seals elsewhere should target similar aggregations, as they could provide early warning of new and expanding infectious diseases in the population.

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Chapter 9
Health Assessment of the Leopard Seal, *Hydrurga leptonyx*, in Prydz Bay, Eastern Antarctica and NSW, Australia

R.B. Gray, T.L. Rogers, and P.J. Canfield

9.1 Introduction

The leopard seal *Hydrurga leptonyx* is the most widely distributed of the Antarctic pinnipeds, ranging from the Antarctic coastline to the sub-Antarctic (Bonner 1994) and less frequently, temperate and sub-tropical areas, including the Australian coast. The main population of leopard seals remains within the circumpolar pack-ice (Bonner 1994) however, there is some north–south movement of individuals, the majority of which are immature, non-breeding seals (Gwynn 1953; Brown 1957; Csordas 1963; King 1983; Rounsevell and Eberhard 1980; Rounsevell 1988; Walker et al. 1998). A periodicity in peak abundance has been observed for this northward dispersal and it is suggested that this could be related to cyclic climatic variation within the region (Harris et al. 1988; Testa et al. 1991; Croxall 1992).

As a large-bodied and long-lived upper trophic species, the leopard seal is considered a useful indicator of change within the Antarctic ecosystem. As such, baseline information on health status for the detection of disease, population dynamics, spatial distribution and foraging behaviour of the leopard seal can be vital to understanding the status of the ecosystem they inhabit.

Developing baseline data and reference intervals for health parameters such as body condition, haematology and serum biochemistry are essential prerequisites for monitoring the impact of disease on a wild population and enables determination of the effects of natural fluctuations and anthropogenic influences on the population within the ecosystem they inhabit.

R.B. Gray and P.J. Canfield
Faculty of Veterinary Science, The University of Sydney, NSW, 2006, Australia
e-mail: rgray@vetsci.usyd.edu.au

T.L. Rogers
Evolution and Ecology Research Centre, School of Biological, Earth and Environmental Sciences
University of New South Wales, Sydney, NSW, 2052, Australia
e-mail: tracey.rogers@unsw.edu.au

R.B. Gray, T.L. Rogers, and P.J. Canfield
Australian Marine Mammal Research Centre, Zoological Parks Board of NSW, Mosman, NSW 2088, Australia

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Leopard seals are difficult to sample due to their location in the pack-ice, their solitary distribution, logistical difficulties associated with their capture, and complications arising during chemical immobilisation. As such, baseline data for leopard seal health and subsequent disease identification is relatively poorly described. Haematological and biochemical values for two captive leopard seals have been reported (Williams and Bryden 1993) and haematological values have been reported for a leopard seal at Heard Island (Brown 1957). Few studies document the incidence of disease in the leopard seal; however, several reports identify the presence of intestinal parasites (Gwynn 1953; Markowski 1952; Green and Williams 1986; Andersen 1987; Wojciechowska and Zdzitowiecki 1995) and the presence of antibodies to canine distemper virus (Bengtson et al. 1991).

Whilst the health and disease status of the leopard seal is poorly documented, the general biology of the species has been better described. Standard length measurements of leopard seals have been determined (Hamilton 1939; Gwynn 1953; Laws 1957; Hofman et al. 1977 and others) and comparisons made of standard length measurements of individuals from various localities. Breeding biology has also been reported. Pupping occurs between late October and mid-November (Siniff and Stone 1985), lactation extends for approximately 4 weeks (Laws 1984), and the breeding season extends from December to early January (Siniff and Stone 1985).

As part of an integrated study of the leopard seal, data were collected on the spatial distribution, acoustic behaviour, foraging behaviour and the health status of a population of leopard seals in Prydz Bay, Eastern Antarctica. This paper focuses on the health status of the leopard seal and describes body condition, body weight, morphometric measurements, haematology and serum biochemistry. It also describes parasitic burden and the results of clinical examination of a population of leopard seals in Prydz Bay. In addition, archival records of leopard seals hauling out along the NSW coast, Australia, have been collated, to ascertain the health status of these seals, as well as the seasonal and annual abundance of the leopard seal in Australian waters.

9.2 Methods

9.2.1 Study Areas

9.2.1.1 Antarctica

Male and female leopard seals were sampled during the summer seasons of 1997/98 and 1999–2002 along the fast-ice edge or on ice floes off Davis Station, Eastern Antarctica (68°36′S 78°02′E). Leopard seals were located either from the air during daily helicopter reconnaissance or by surveys of the ice edge using all-terrain vehicles and their positions determined using the Global Positioning System (GPS). Chemical restraint was attempted in 56 seals. Six seals were sampled in successive seasons 1999/2000 and 2000/01. Observations on sex and body condition, and the

collection of scats and hair off the ice, were also undertaken for seals not regarded suitable for capture (see below).

9.2.1.2 NSW, Australia

Thirty-eight leopard seals that hauled out along the NSW coast between 1972 and 2003 were examined by Veterinary and Quarantine Centre staff at the Taronga Zoo, Zoological Parks Board of NSW, in association with the National Parks and Wildlife Service (NSW), Department of Environment and Conservation (DEC). Information on seal sex, age and location of haul-out, as well as results of clinical examination, blood analysis, faecal analysis and necropsy of a number of seals was obtained from the archival records of Taronga Zoo and from observations and analysis by the present authors.

9.2.2 Immobilisation and Restraint

Leopard seals in Antarctica were sampled under chemical restraint. Prior to immobilisation, seal sex was determined, the presence/absence of tags noted and a general health appraisal was undertaken which included an assessment of body condition and any evidence of obvious clinical disease, for example, the presence of oral and nasal discharges. Seals were also assessed to determine their suitability for immobilisation and subsequent recovery. The factors considered when selecting a seal for capture included the suitability of the ice floe for a procedure for both seal and personnel safety, proximity of the seal to the water, the general behaviour of the seal, the overall health of the animal as determined by initial clinical appraisal, absence of obvious clinical disease, and observation of normal breathing patterns prior to immobilisation. During immobilisation, other information such as the presence or absence of noticeable scars and wounds was also collected.

Seals were darted (CO_2 powered Telinject G.U.T 50 rifle Telinject Australasia) from a distance of 12–15 m. Initial procedures ($n = 16$) employed a combination of midazolam (0.18–0.27 mg kg^{-1}, Roche Products Pty Ltd, Australia) and pethidine (1.0–1.5 mg kg^{-1}, Sigma Pharmaceuticals, Clayton, Victoria). Another anaesthetic regime was subsequently developed employing a 1:1 ratio of 0.5–1.5 mg kg^{-1} tiletamine/zolazepam (Telazol 100 mg ml^{-1}, Fort Dodge, Australia; or Zolatil 100 100 mg ml^{-1}, Virbac Australia Pty Ltd) because of inadequacies of the midazolam/pethidine combination initially used (Higgins et al. 2002). Atropine 0.015 mg kg^{-1} (16 mg ml^{-1}, AstraZeneca Pty Ltd, Australia, reconstituted by Royal Hobart Hospital, Tasmania) was administered with the immobilising drugs.

The combination of tiletamine/zolazepam in the leopard seal produced faster induction of sedation (19 ± 3 min), more reliable response to dose, improved pulmonary ventilation and faster return of cognitive function compared to the midazolam/pethidine combination (Higgins et al. 2002). The dose rate employed for the majority

of the procedures in the 2000/01 season was 1.3 mg kg^{-1} based on the regime reported by Higgins et al. (2002) and this was found to be optimal for the requirements of the present study for sample collection. For the majority of the seals immobilised, the combination of tiletamine/zolazepam provided generally safe chemical restraint. However, three seals died during chemical restraint using tiletamine/zolazepam due to inadvertent intravenous administration or anaesthetic complications during recovery. Therefore extreme care and vigilance are mandatory during administration of immobilising drugs in this species, and attentive monitoring of anaesthetic procedures needs to be continued throughout the recovery period.

Due to the young age and generally poor condition of the leopard seals sampled in NSW, manual restraint using a hoop net and head bag or light sedation was often sufficient for the purposes of physical examination, blood collection, and collection of morphometric measurements and weighing. If these techniques were not sufficient for restraint, sample collection did not occur.

9.2.3 Sample and Data Collection

9.2.3.1 Age, Morphometric Measurements, Body Condition and Body Weight

The age of seals sampled was estimated by length (Laws 1957). Standard length was measured in 44 seals in Prydz Bay, axillary girth in 19 seals, and neck girth, pelvic girth and the length of the pectoral flipper in nine seals. Standard length measurements of 26 female and 18 male leopard seals in Prydz Bay in the present study, including repeat measurements of two female seals sampled in successive seasons (1999/2000 and 2000/01), were compared with the lengths of nine female and nine male leopard seals from Palmer Station on the Antarctic Peninsula (64°46′S 64°03′W) (Hofman et al. 1977, Fig. 9.3) and from 11 female and 12 male leopard seals sampled in the pack-ice (Erickson et al. unpublished in Fig. 9.3 of Hofman et al. 1977). Standard length was also measured in seven leopard seals hauling out in NSW, and axillary, neck and pelvic girth, and length of the pectoral flipper, measured in three of these seals.

The standard length was taken to be the straight-line distance from the snout to the tip of the tail and the axillary girth was measured around the surface of the body underneath the fore flippers to the nearest cm (Bonner and Laws 1993). The neck and pelvic girth were also measured. Blubber thickness, excluding skin thickness, was measured over the sternum on a line between the axillae to the nearest mm using a tape measure or ruler in two seals in Antarctica and in three seals hauling out in NSW.

Body condition was graded qualitatively into four classes prior to chemical or physical restraint for the seals sampled in Prydz Bay in the 1999/2000, 2000/01 and 2001/02 seasons and for leopard seals in NSW. The classes used were poor, fair/thin, good and excellent and examples of each class are illustrated in Fig. 9.1. Body weight was estimated in leopard seals prior to immobilisation in order to calculate the required dose. Two seals in Prydz Bay were also weighed using a tripod and

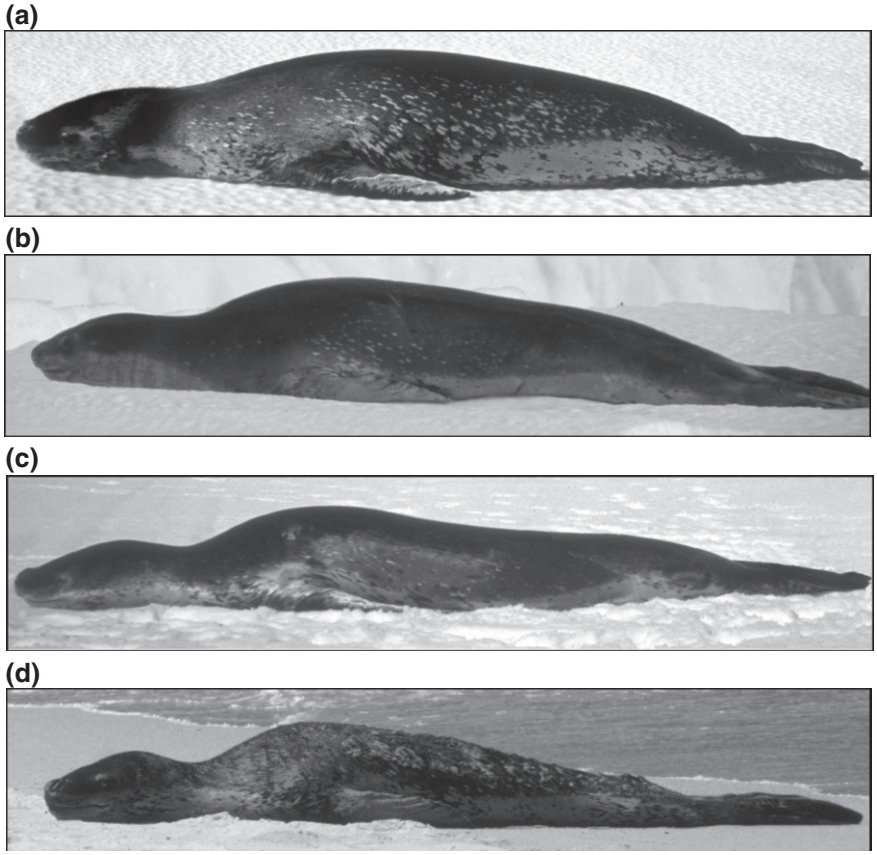

Fig. 9.1 Body condition classes of leopard seals: (**a**) excellent (**b**) good (**c**) fair/thin (**d**) poor

electronic scales (1T Dillon Electronic Dynamometer, Model # ED-2000–1 Division of Weigh-Tronix Inc. USA). The estimated weight differed from true weight by 0.9% and 6.9% in these two seals. A small number of seals examined at the Veterinary and Quarantine Centre at Taronga Zoo were also weighed upon arrival.

9.2.3.2 Clinical Examination

Clinical examination was undertaken during chemical restraint of leopard seals in Prydz Bay. Observations on the condition of the teeth including presence of chipped or worn teeth, gingivitis, and tooth wear; the presence of nasal and ocular discharges; and the presence of wounds and scars were noted. The level of immobilisation attained in each procedure imposed limitations on the number of seals for which these data could be obtained, thus the sample size varies for each observation. Clinical examinations, where possible, were undertaken for seals hauled out along the NSW coast.

Data collection focused primarily on assessment of body condition, presence or absence of wounds and scars, and evidence of clinical disease.

9.2.3.3 Blood Collection

Blood was collected from 29 leopard seals in Prydz Bay between 1999–2002 and from 21 leopard seals hauled out along the NSW coast. It was taken from the extradural, intravertebral vein (Harrison and Tomlinson 1956) in the lumbar region by the method described by Geraci and Smith (1975) using 3¼ in. spinal needles (Yale® Spinal Needle B-D Becton Dickinson Spain). Blood samples were collected into potassium EDTA, plain serum and lithium heparin tubes (BD Vacutainer Systems, Bellivier Industrial estate, Plymouth, UK). Serum and lithium heparin tubes were centrifuged at 10,000 rpm and serum and plasma stored at −80°C or in liquid nitrogen prior to analysis.

9.2.3.4 Haematology

Haematology was performed on whole blood preserved in potassium EDTA within 1–6 h of collection. Manual packed cell volume (PCV) was performed by the micro-haematocrit method by centrifuging a sample of blood in a micro-haematocrit tube for 120 s at 15,800 rpm (Statspin-Multi-Purpose, StatSpin, USA) and estimating the volume of red blood cells to total volume in L/L. PCV measurement was performed in duplicate and the mean of the two readings used for the statistical analysis. Total leukocyte (white) cell counts were performed using the improved Neubauer technique. Differential white cell counts were performed on air-dried blood smears stained with the Diff Quik® stain with 100 cells counted. Red blood cell indices, mean corpuscular volume (MCV), mean corpuscular haemoglobin concentration (MCHC) and mean corpuscular haemoglobin (MCH) were also measured for a number of leopard seals hauling out in NSW.

9.2.3.5 Biochemistry

Serum samples were utilised for the determination of 24 biochemical analytes by a commercial wet chemical analyzer (Olympus AU 400, IDEXX Veterinary Pathology Services, IDEXX Laboratories Pty Ltd Australia). As commercial laboratory facilities were not available in Antarctica, frozen stored serum was used for commercial biochemistry analysis within 3–6 months of collection for leopard seals sampled in Prydz Bay. Serum samples collected from leopard seals in NSW were stored frozen for shorter periods prior to analysis. The biochemical analytes determined were glucose, urea, creatinine, total protein, albumin, globulin, total bilirubin, alkaline phosphatase (ALP), aspartate aminotransferase (AST), alanine aminotransferase (ALT), creatinine kinase, cholesterol, calcium, phosphate, sodium, potassium, chloride, bicarbonate, anion gap, amylase and lipase. Plasma fibrinogen levels were determined by the microhaematocrit heat precipitation method (Millar et al. 1971) using thawed frozen-stored plasma samples.

9.2.4 Parasite Examination

9.2.4.1 Necropsy

Parasites were identified in the stomach and small intestine in three leopard seals necropsied in Prydz Bay and from seals hauled out in NSW.

9.2.4.2 Scat Parasites

Scat samples ($n = 73$) were collected off the ice from known and unknown leopard seals in Prydz Bay. Scats were washed through a series of sieves ranging from 425 μm to 4.75 mm for another study (Hall-Aspland and Rogers 2004) and parasites collected into 10% buffered formalin for subsequent identification. Observations of scat parasites were also made for seals hauled out in NSW.

9.2.4.3 Faecal Flotation

Faecal floatation using saturated salt solution (NaCl; specific gravity 1.18–1.20) was performed on 52 frozen and fresh scat samples from seals in Prydz Bay and in scat samples from seals hauled out in NSW.

9.2.5 Statistical Analysis

Data was analysed using Minitab (Minitab Inc., Philadelphia, USA) and GenStat® 6th Edition (Rothamsted Experimental Station 2002) statistical packages. A two-sample unpaired *t*-test was used to test for differences in standard length between male and female leopard seals. For leopard seals sampled in Prydz Bay, haematology and biochemistry data were analysed using a general linear model ANOVA (year of sampling, seal sex and moult status as factors). Backwards elimination was employed to sequentially eliminate non-significant factors from the general linear model. Six individuals were sampled in successive seasons (1999/2000 and 2000/01). It was not possible to include seal identification as a factor in the statistical model due to co-linearity with other terms, thus seal-to-seal variation was not explicitly taken into account. This is not expected to impact on the statistical results due to the small number of repeat samples employed, and the use of sampling times sufficiently far apart such that serial correlation would not be expected for the substances being measured. The assumptions of ANOVA (normal distribution and homogeneity of variances) were tested by assessment of the histogram of residuals and the fitted-value plot. Where these assumptions were not met by untransformed and natural log transformed data, the non-parametric Mann Whitney U test was employed and each factor (seal sex, year and moult status) was tested individually for each variable. Reference intervals were developed for each haematological and biochemical value using the estimated 2.5th and 97.5th percentiles. Where these values were log normally distributed or where non-parametric

statistical tests were employed, geometric means and 95% confidence intervals are reported instead of means and standard deviations. Band neutrophils were not analysed statistically due to the large number of zero values. Values more than three standard deviations from the mean were excluded from the analysis.

9.3 Results

9.3.1 Immobilisation and Restraint

The combination of tiletamine/zolazepam provided up to 35 min of immobilisation. Optimal results were attained at dose rates of 1.2–1.4 mg kg^{-1}. Immobilisation in leopard seals was characterised by good analgesia (assessed by the response to sample collection) and an inability to move the caudal region despite the seal being able to move the head in the later stages of the procedure, facilitating the collection of blood, biopsies and tagging. Recovery was rapid and uneventful during which there was movement of the head and attempted vocalisations, with seals generally mobile within 1.5 h post-darting. No noticeable difference in behavioural response to approach was observed in seals that had undergone a procedure and seals that had not.

During immobilisation with tiletamine/zolazepam, there was an initial decrease in respiratory rate from 5 to 15 min post-darting (Fig. 9.2). The respiratory rate subsequently increased between 15 and 30 min post-darting and then stabilised at approximately 10 breaths per minute. Further increases in respiratory rate were seen after 70 min post-darting with respiratory rate presumably attaining pre-dart levels at around this time. Figure 9.2 displays the mean respiratory rate in breaths/minute of 11 seals immobilised with tiletamine/zolazepam. Mean respiratory rate and standard deviation are displayed for 5-min intervals during the 5–90 min post-drug administration. Minimum mean respiratory rate was seen at 15 min post-darting.

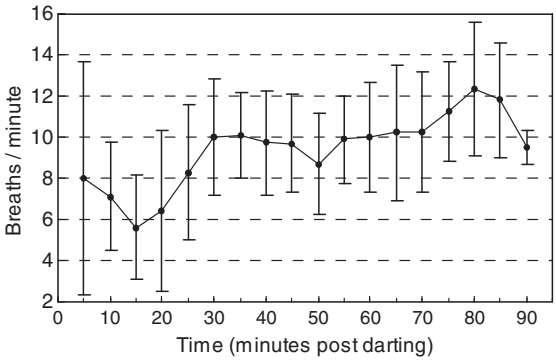

Fig. 9.2 Mean respiratory rate (breaths/minute) and standard deviation of leopard seals immobilised with tiletamine/zolazepam

9.3.2 Age, Morphometric Measurements, Body Condition and Body Weight

9.3.2.1 Antarctic Seals

For those seals in which standard length was measured, the majority (36 out of 44) were classed as adults (>4 years of age). Four seals (two female and two male) were determined to be between 3 and 4 years of age, and another four seals (two females and two males) were determined to be less than 3 years of age.

No significant difference in length was seen for male and female adult leopard seals (*t*-test: $t = 2.00$; df = 1,34; $p = 0.054$). The mean standard length for males > 4-years-old was 2.94 ± 0.19 m and the mean standard length for females > 4 years of age was 3.07 ± 0.20 m. Morphometric measurements of leopard seals in Prydz Bay are shown (Table 9.1). A comparison between standard length measurements for both male and female leopard seals in the present study with those of leopard seals around Palmer Station and in the pack-ice are shown (Fig. 9.3).

The majority of the seals sampled in Antarctica (44 out of 55 seals) were in good body condition (Table 9.2). The sternal blubber depth determined in two seals from Prydz Bay was 35 mm for both seals.

The mean estimated body weight of leopard seals > 4 years of age in each season was 494 kg ($n = 9$) in 1997/98, 352 kg ($n = 17$) in 1999/2000 and 362 kg ($n = 15$) in 2000/01. Seals sampled in 1997/1998 were observed to be generally in excellent condition and larger than the seals sampled in 1999/2000 (D Higgins personal communication).

9.3.2.2 Leopard Seals Hauling Out in NSW

Age was estimated for 28 of the 38 seals sighted during 1972–2003. Eighty-six percent of seals were determined to be <3-years-old and 14% were classed as adult (>4 years). Sex was determined in 30 of the 38 seals reported, with 57% female ($n = 17$) and 43% male ($n = 13$). Of the 26 seals for which information other than sex, age and location of haul-out are available, 11 seals were in poor body condi-

Table 9.1 Morphometric measurements (m) of adult leopard seals in Prydz Bay

Measurement	*n*	Mean ± s.d.	Observed range
Standard length – male	14	2.94 ± 0.19	2.76–3.50
– female	22	3.07 ± 0.20	2.80–3.42
Standard (axillary) girth	19	1.46 ± 0.33	1.04–2.06
Neck girth	9	1.23 ± 0.20	1.00–1.60
Pelvic girth	9	1.16 ± 0.19	0.79–1.43
Pectoral flipper length	9	0.58 ± 0.11	0.48–0.85

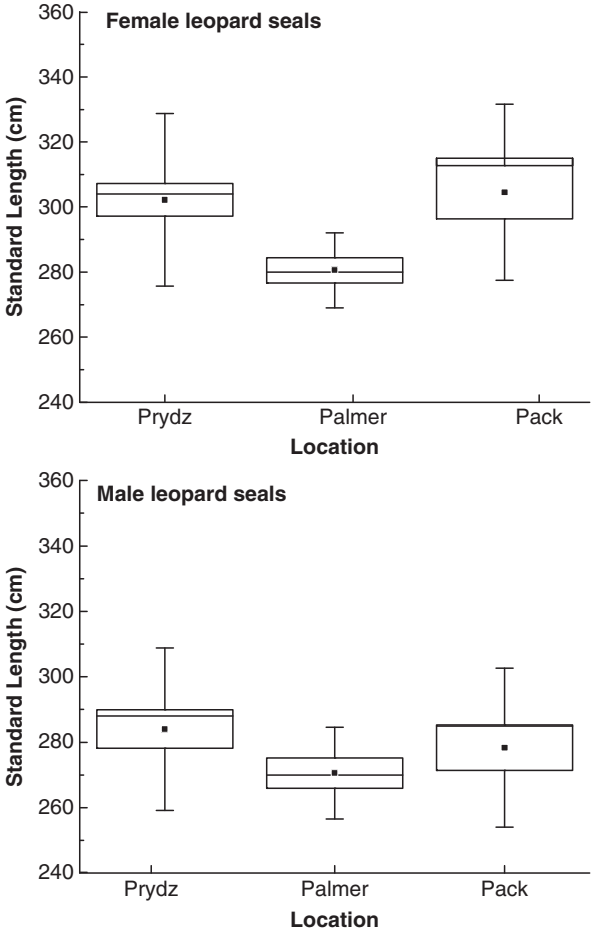

Fig. 9.3 Standard length measurements of female (*top*) and male (*bottom*) leopard seals in Prydz Bay, Palmer Station (Antarctic Peninsula) and the pack-ice. The *filled in square* denotes the mean, the *box* denotes the standard error and the *horizontal line* is the median. The *whiskers* denote the standard deviation

Table 9.2 Body condition of leopard seals examined in Prydz Bay in 1999/2000, 2000/01 and 2001/02

Season	Number of seals	Poor (%)	Fair/thin (%)	Good (%)	Excellent (%)
1999/2000	27	0	18.5	74.1	7.4
2000/2001	23	0	8.7	87.0	4.3
Combined[a]	55	0	14.6	80.0	5.4

[a]Includes five seals from 2001/02

Table 9.3 Morphometric measurements (m), sex, month of haul-out, weight and estimated age of leopard seals in NSW. Measurements as at haul-out unless otherwise indicated

Sex	Month of haul-out	Standard length	Axillary girth	Neck girth	Pelvic girth	Front flipper	Weight [kg]	Estimated age [years]
M	July	2.40					–	1–2
M	July	2.05	1.08	0.8	0.68	0.47	78	<1
M	August	2.20					–	1–2
M	August	–					168	
M	September	2.25					89.5	<1
	February (6 months post haul-out)		1.28	0.91	0.92	0.67	160	1–2
F	July	2.20					100	1–2
F	August	2.46	1.11	0.79	0.62	0.66	96.2	2–3
F	August	–					102.2	<1[a]
F	August	2.70					240	3–4

[a]Visual estimate of age

tion/thin/emaciated, whilst three seals were in good condition and these were generally the older seals sighted. No record of body condition was available for the remaining 12 seals. Six out of 26 seals were dead on arrival, or were euthanased due to poor body condition or died within 1 week of captivity (two of these seals were classed as being in poor body condition when first sighted). Sternal blubber depth in three leopard seals hauled out along the NSW coast were 16 mm, 16 mm and 17 mm. Sex, month of haul-out, morphometric measurements, weight and estimated age of nine leopard seals hauled out in NSW are shown (Table 9.3).

9.3.3 Clinical Examination

Antarctic Seals

9.3.3.1 Ocular Discharge

In the 1999/2000 season, seven of the leopard seals examined ($n = 33$) had a bilateral or unilateral ocular discharge and reddening of conjunctiva (conjunctivitis). In some seals this was manifested as crusty material around the eye/s. In the 2000/01 and 2001/02 seasons, no seals were observed with ocular discharge ($n = 26$).

9.3.3.2 Nasal Discharge

In the 1999/2000 season, 12 seals ($n = 33$) were observed with unilateral or bilateral nasal discharge, ranging from a muco-purulent discharge to white stable foam discharging from the nostrils. Four of the seals examined were also coughing. In the 2000/01 season, one seal ($n= 21$) was observed with nasal discharge seen as froth

from the left nostril, whilst no seals in the 2001/02 season (n = 5) were observed with a nasal discharge.

9.3.3.3 Oral Examination

Thirty-nine leopard seals were examined to enable assessment of the condition of the teeth. Thirty-one seals had good teeth (Fig. 9.4 left photograph); five seals had broken, worn, chipped or missing teeth (Fig. 9.4. right photograph). A number of combinations of conditions were seen: one seal had gingivitis and dermatitis around the mouth and lips, another had gingivitis, dermatitis around the lips and mouth, and broken teeth, and another seal had gingivitis, broken, chipped or missing/worn teeth and exposure of the tooth roots (periodontitis).

9.3.3.4 Wounds/Scars

Of the seals sampled in the 1999/2000, 2000/01 and 2001/02 seasons (n = 38), 13 had obvious scars or wounds. Of these 13 seals, lesions ranged from a few small scars (n = 4), to rake marks along the body (n = 3), multiple puncture wounds (n = 2), large bite wounds (n = 1), missing portions of flippers (n = 2) and multiple scars and healing wounds with purulent discharges over the entire body (n = 1). In the latter individual, one eye appeared as a pale fibrous mass, thought to be a resolved penetrating wound to the anterior chamber. Some seals were observed with combinations of these injuries.

Leopard Seals Hauling Out in NSW

The main clinical problems identified on examination of seals hauling out along the NSW coast (n = 26) were poor body condition, weakness, debilitation, emaciation and dehydration. Skin lesions or wounds of varying degrees were reported in 15 seals with possible causes including bites from cookie cutter sharks, *Isistius*

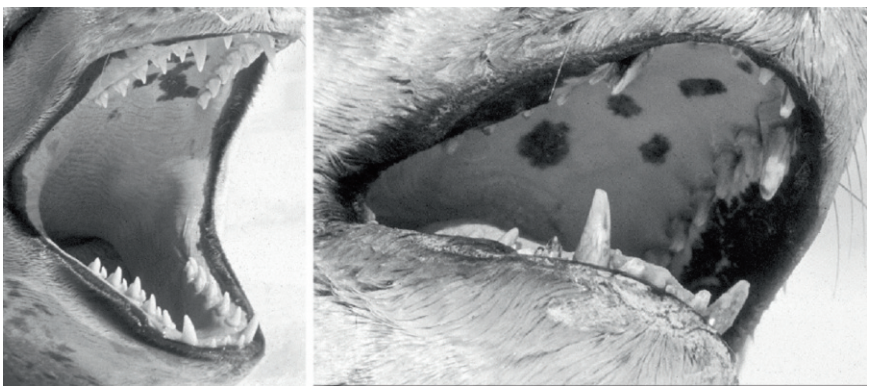

Fig. 9.4 Examples of leopard seal teeth: good condition (*left*), poor condition with staining, chipped teeth and tooth wear (*right*)

brasiliensis, and injuries from stingray spines, *Urolophus paucimaculatus*, and the latter often progressed to abscess formation, particularly around the head and neck. Two seals exhibited clinical signs indicative of respiratory disease including nasal and ocular discharges, coughing, conjunctivitis and laboured breathing. Two seals also suffered seizures and subsequently died. One young seal (1 to 2-years-old) also presented with a swollen face and was found on further examination to have significant wearing of the teeth, multiple draining sinuses into the mouth, as well as numerous exposed pulp cavities.

9.3.4 Haematology and Serum Biochemistry

9.3.4.1 Antarctic Seals

Significant differences were seen for a small number of haematological and biochemical values when moult, sex and season of sampling were compared and these are shown in Table 9.4. Table 9.5 displays descriptive statistics and calculated reference intervals for haematological values of 28 leopard seals sampled in Prydz Bay in 1999/2000 and 2000/01 and Table 9.6 displays descriptive statistics and developed reference intervals for serum biochemical values and plasma fibrinogen of 29 leopard seals sampled in Prydz Bay in 1999/2000 and 2000/01.

9.3.4.2 Leopard Seals Hauling Out in NSW

Table 9.7 displays descriptive statistics for haematological values of 21 leopard seals hauled out along the NSW coast. Results are shown for blood collected at the time of haul-out.

Table 9.4 Haematological and biochemical values in which there were significant differences seen when season of sampling, moult status and sex are compared for the leopard seals in Prydz Bay

Variable	Factor	Mean ± s.d	Factor	Mean ± s.d	Statistic
Eosinophil count ($\times 10^9$ L^{-1})	Moult	1.12 (n = 18)	Non-moult	2.05 (n = 8)	ANOVA: F = 4.82; df = 1,23; p = 0.039
Eosinophil count ($\times 10^9$ L^{-1})	Female	1.68 (n = 16)	Male	0.96 (n = 10)	ANOVA: F = 4.55; df = 1,23; p = 0.044
Anion gap	1999/2000	21.9 (n = 16)	2000/01	17.3 (n = 13)	ANOVA: F = 21.04; df = 1,27; p = 0.00
Fibrinogen (g L^{-1})	1999/2000	1.79 (n = 13)	2000/01	1.25 (n = 13)	ANOVA: F = 6.27; df = 1,24; p= 0.019
Total Protein (g L^{-1})	Female	70.7 (n = 19)	Male	77.4 (n = 10)	ANOVA: F = 5.25; df = 1,27; p = 0.03
Albumin (g L^{-1})	Female	29.5 (n = 19)	Male	32.4 (n = 10)	ANOVA: F = 7.46; df = 1,27; p = 0.011
Calcium (mmol L^{-1})	Female	2.37 (n = 19)	Male	2.57 (n = 10)	ANOVA: F = 6.61; df = 1,27; p = 0.016

Table 9.5 Descriptive statistics and reference intervals for haematological values of leopard seals in Prydz Bay (n = 28). Mean ± standard deviation (median) (when parametric statistics used), or geometric mean and 95% confidence interval (when $\log_e$ transformed data or non-parametric statistics used), developed reference interval (estimated 2.5th and 97.5th percentiles) and observed range (from Gray 2005)

Variable	Mean ± s.d. (median)	Reference intervals	Observed range
Packed cell volume (l/l)	0.47 ± 0.03 (0.48)	0.41–0.53	0.42–0.52
Total leukocyte count ($\times 10^9$ L^{-1})[a]	8.37 (7.34, 9.55)	4.21–16.6	3.85–14.85
Neutrophils (%)[a]	59.2 (55.6, 63.0)	43–82	43–75
Neutrophils ($\times 10^9$ L^{-1})[a]	4.95 (4.29, 5.72)	2.35–10.5	2.31–10.3
Band Neutrophils (%)[b]	0.33 ± 0.62 (0)	0–2	0– 2
Band Neutrophils ($\times 10^9$ L^{-1})[b]	0.03 ± 0.05 (0)	0–0.13	0–0.21
Lymphocytes (%)[a]	16.6 ± 5.30 (16)	6–27	10–29
Lymphocytes ($\times 10^9$ L^{-1})[a]	1.42 ± 0.58 (1.23)	0.22–2.62	0.62–2.63
Monocyte (%)[a]	2.87 (2.18, 3.76)	1–12	0–9
Monocyte ($\times 10^9$ L^{-1})[a]	0.24 (0.18, 0.31)	0.06–0.99	0–0.60
Eosinophil (%)[b]	16.5 (13.9, 19.6)	7–40	4–33
Eosinophil ($\times 10^9$ L^{-1})[b]	1.35(1.08, 1.69)	0.43–4.20	0.37–3.11
Basophil (%)[a]	2.12 (1.42, 3.16)	0–11	0–8
Basophil ($\times 10^9$ L^{-1})[a]	0.19 (0.13, 0.28)	0.04–0.88	0–0.63

[a]n = 27; [b]n = 26

Table 9.6 Descriptive statistics and reference intervals for biochemical values and plasma fibrinogen of leopard seals in Prydz Bay (n = 29). Mean ± standard deviation (median) (when parametric statistics used), or geometric mean and 95% confidence interval (when $\log_e$ transformed data or non-parametric statistics used), developed reference interval (estimated 2.5th and 97.5th percentiles) and observed range (from Gray 2005)

Analyte	Mean ± s.d. (median)	Reference intervals	Observed range
Glucose (mmol L^{-1})[c]	6.74 ± 0.57 (6.80)	5.56–7.92	5.70–8.10
Urea (mmol L^{-1})	6.65 ± 3.07 (6.40)	0.35–13.0	1.80–15.1
Creatinine (μ mol L^{-1})	93.4 ± 19.1 (90.0)	54.3–133	60–130
Total Protein (g L^{-1})	73.0 ± 8.05 (73.0)	56.5–89.5	56.0–92.0
Plasma Fibrinogen (g L^{-1})[a]	1.52 ± 0.61 (1.55)	0.26–2.78	0.20–2.80
Albumin (g L^{-1})	30.5 ± 3.04 (30.0)	24.2–36.7	24.0–37.0
Globulin (g L^{-1})	42.5 ± 6.01 (42.0)	30.2–54.8	32.0–55.0
Total bilirubin (μ mol L^{-1})	3.56 (2.98, 4.25)	1.36–9.29	1.50–9.30
Alkaline phosphatase (U L^{-1})	75.0 (65.5, 85.8)	36.2–155	41.0–177
Aspartate aminotransferase (U L^{-1})	71.1 (58.8, 86.0)	25.5–199	19.0–158
Alanine aminotransferase (U L^{-1})	83.6 (60.0, 117)	14.0–500	11.0–301
CK (U L^{-1})[c]	242 (192, 306)	70.2–836	86–1823
Cholesterol (mmol L^{-1})	6.67 ± 1.62 (6.50)	3.35–10.0	4.00–10.7
Calcium (mmol L^{-1})	2.44 (2.36, 2.52)	2.03–2.93	2.00–3.00
Phosphate (mmol L^{-1})	2.49 ± 0.53 (2.30)	1.39–3.58	1.70–3.40
Sodium (mmol L^{-1})[b]	152 ± 5.23 (153)	141–163	137–160
Potassium (mmol L^{-1})	4.21 (4.09, 4.33)	3.61–4.89	3.60–4.80
Chloride(mmol L^{-1})[c]	112 (110, 114)	101–125	99.0–135
Bicarbonate (mmol L^{-1})	25.7 ± 4.51 (25.0)	16.4–34.9	14.0–35.0
Anion gap	19.6 (18.3, 20.9)	13.6–28.1	13.4–25.7
Amylase (U L^{-1})[c]	1.38 (0.92, 2.05)	0.26–7.20	0.00–5.80
Lipase (U L^{-1})	353 (266, 467)	77.4–1,609	47.0–1,150
Sodium:Potassium[c]	36.6 ± 2.29 (36.5)	31.9–41.3	32.1–41.1
Calcium:Phosphate	1.00 (0.93, 1.08)	0.68–1.48	0.72–1.41
Albumin:Globulin[c]	0.73 (0.70, 0.76)	0.59–0.91	0.57–0.91

[a]n = 26; [b]n = 27; [c]n = 28

Table 9.7 Descriptive statistics for haematological values of leopard seals in NSW ($n = 21$). Mean ± standard deviation (median) and observed range (from Gray 2005)

Variable	N	Mean ± s.d. (median)	Observed range
Packed cell volume (l/l)	21	0.45 ± 0.07 (0.46)	0.27–0.54
Red blood cell count ($\times 10^{12}$ L^{-1})	6	5.37 ± 1.40 (5.75)	3.6–7.09
Haemoglobin (g L^{-1})	12	186 ± 30.3 (193)	128–230
Mean corpuscular haemoglobin concentration (g L^{-1})	8	387 ± 57 (380)	353–438
Mean corpuscular haemoglobin (pg)	3	–	32–42.3
Mean corpuscular volume (fl)	3	–	83.3–120
Total leukocyte count ($\times 10^{9}$ L^{-1})	21	9.90 ± 3.99 (10.0)	3.2–19.0
Neutrophils (%)	20	69 ± 14 (71)	43–89
Neutrophils ($\times 10^{9}$ L^{-1})	20	7.01 ± 3.89 (5.88)	2.34–16.9
Band neutrophils (%)	20	4.30 ± 7.12 (1.0)	0–30
Band neutrophils ($\times 10^{9}$ L^{-1})	20	0.45 ± 0.90 (0.13)	0–4.02
Lymphocytes (%)	20	20.7 ± 11.2 (20.0)	8–44
Lymphocytes ($\times 10^{9}$ L^{-1})	20	1.93 ± 1.10 (1.59)	0.41–4.04
Monocyte (%)	20	4.70 ± 5.00 (3.00)	0–19
Monocyte ($\times 10^{9}$ L^{-1})	20	0.47 ± 0.52 (0.28)	0–1.9
Eosinophil (%)	20	1.20 ± 3.40 (0)	0–12
Eosinophil ($\times 10^{9}$ L^{-1})	20	0.11 ± 0.33 (0)	0–1.21
Basophil (%)	20	0.05 ± 0.22 (0)	0–1
Basophil ($\times 10^{9}$ L^{-1})	20	0.01 ± 0.03 (0)	0–0.13

Table 9.8 Descriptive statistics for biochemical values of leopard seals hauled out in NSW ($n = 8$). Mean ± standard deviation (median) and observed range (from Gray 2005)

Analyte	Mean ± s.d. (median)	Observed range
Glucose (mmol L^{-1})	6.06 ± 1.91 (6.80)	1.90–7.48
Urea (mmol L^{-1})	8.25 ± 5.68 (6.85)	3.3–21.6
Creatinine (μmol L^{-1})	77.5 ± 38.5 (65.0)	50.0–170
Total Protein (g L^{-1})	73.6 ± 8.14 (76.5)	62.0–86.0
Albumin (g L^{-1})	31.6 ± 8.35 (30.5)	21.0–45.0
Globulin (g L^{-1})	42.0 ± 13.7 (42.0)	19.0–65.0
Total bilirubin (μmol L^{-1})	6.04 ± 3.94 (5.0)	1.40–13.0
Alkaline phosphatase (U L^{-1})	51.3 ± 16.7 (48.0)	30.0–80.0
Aspartate aminotransferase (U L^{-1})	74.4 ± 42.2 (68.0)	15.0–137
Alanine aminotransferase (U L^{-1})	50.5 ± 21.2 (42.5)	31.0–91.0
Cholesterol (mmol L^{-1})	7.50 ± 2.17 (7.40)	4.99–11.6
Calcium (mmol L^{-1})	2.49 ± 0.16 (2.50)	2.30–2.75
Phosphate (mmol L^{-1})	2.06 ± 0.34 (2.00)	1.70–2.80
Sodium (mmol L^{-1})	153 ± 3.59 (153)	146–158
Potassium (mmol L^{-1})	4.34 ± 0.78 (4.05)	3.40–5.90
Chloride (mmol L^{-1})	106 ± 3.51 (105)	101–110
Bicarbonate (mmol L^{-1})	25.3 ± 5.98 (25.8)	14.0–34.1
Anion gap[a]	–	22.8–33.9
Amylase (U/L)[a]	–	0–4.00
Lipase (U/L)[a]	–	5.00–58.0
Sodium:Potassium	36.2 ± 6.12 (37.8)	25.8–45.9
Calcium:Phosphate	1.23 ± 0.18 (1.30)	0.90–1.41
Albumin:Globulin	0.91 ± 0.62 (0.70)	0.32–2.26

[a] $n = 4$

Table 9.8 displays descriptive statistics for serum biochemical values for leopard seals hauled out along the NSW coast. The biochemical data are from five female and three male juvenile and sub-adult seals. Results are shown for blood collected at the time of haul-out.

9.3.5 Parasite Examination

9.3.5.1 Antarctic Seals

Large numbers of cestodes, and to a lesser extent nematodes, were seen in the stomach and small intestine of leopard seals during necropsy. Of the leopard seal scats examined, 64% (n = 73) contained parasites (cestodes and nematodes). Seventy-five percent of scats examined by faecal flotation were positive for *Diphyllobothrium* eggs and 2% of scats were positive for both *Diphyllobothrium* and ascarid eggs. Only 23% of leopard seal scats were negative for worm eggs on faecal flotation.

9.3.5.2 Leopard Seals Hauling Out in NSW

Intestinal parasitism, as evidenced by positive faecal flotation, presence of parasites in scats or on necropsy, was recorded for only 13 leopard seals. Of these seals, one seal was negative on faecal flotation and scat examination for worms, one had nematodes in the scat, nine seals were positive for *Diphyllobothrium* infection, one seal had both *Diphyllobothrium* and nematodes on necropsy and another seal had *Diphyllobothrium* and strongyle larvae observed in faecal flotation. Copepods were seen on the flippers in 2 (n = 26) seals and barnacles were seen on the flippers of one seal.

9.3.6 Seasonal and Annual Abundance of Leopard Seals in NSW, Australia

Seventy-six percent of leopard seals hauling out along the NSW coast (n= 45) between 1950 and 2003 were sighted from July to September, with a peak of sightings in August (Source: Elliot 1982 and Taronga Zoo archival records). One leopard seal was sighted hauled out in NSW in 2004 and two in 2005 (Source: NSW Department of Environment and Conservation, 2006, Atlas of New South Wales Wildlife Database as of October 2006). Figure 9.5 displays the month of haul-out of leopard seals along the NSW coast from 1950 to 2003. Figure 9.6 displays leopard seal sightings along the NSW coast grouped by year from 1950 to 2005.

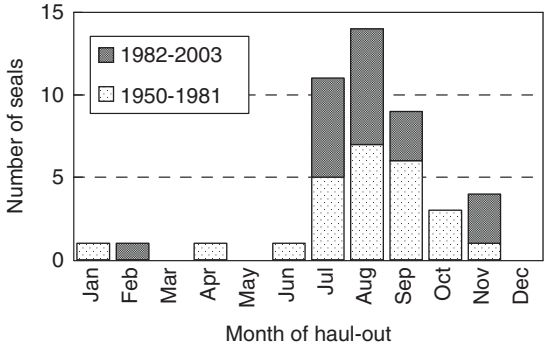

Fig. 9.5 The number of leopard seals hauling out each month along the coast of NSW from 1950 to 2003. Data for 1950–1981 (Elliot 1982) and 1982–2003 (present study)

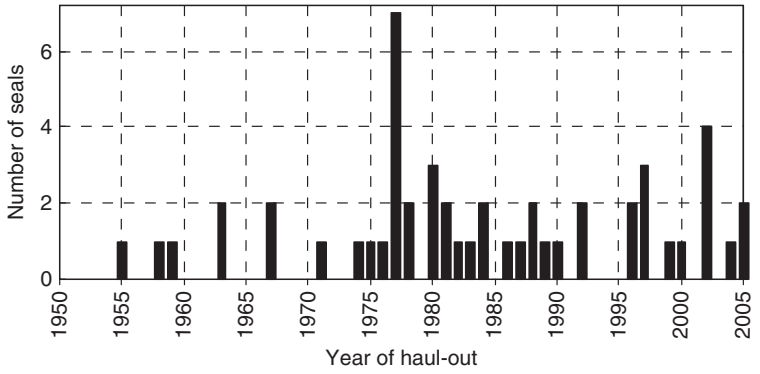

Fig. 9.6 The number of leopard seals hauled out each year along the NSW coast from 1950 to 2005. Data for 1950–1981 from Elliot (1982) and 1982–2005 (present study)

9.4 Discussion

9.4.1 Antarctic Seals

9.4.1.1 Morphometric Measurements, Body Condition and Body Weight

The mean standard length of male and female leopard seals determined in the present study are similar to those previously reported for adult leopard seals (Hamilton 1939; Laws 1957). Previous reports in the literature suggest that leopard seals are spatially segregated by age, with leopard seals at Palmer Station, for example, being of intermediate age (3–9 years) compared to the adults seen in the pack-ice (Hofman et al. 1977). The comparison of standard length measurements for both male and female leopard seals in the present study, with those of leopard seals at

Palmer Station and in the pack-ice, indicates that leopard seals sampled in Prydz Bay were similar in length to those sampled in the pack-ice, and generally larger than those sampled at Palmer Station.

The body condition of the majority of the seals examined in the present study was determined to be good. However, the data on body condition may be skewed due to the selection of seals for capture based primarily on their suitability for chemical immobilisation. In addition, one of the general aims of the overall study was to obtain data on leopard seals that were free of obvious clinical disease. In the 1999/2000 season, a large number of seals were reported to have nasal and ocular discharges, were observed coughing and appeared to have a higher proportion of wounds present. Early in the 1999/2000 season, these seals were not selected for procedures. As a consequence, the body condition scores for the 1999/2000 season may be biased toward seals in good body condition. In the 2000/01 and 2001/02 seasons, the majority of the seals were deemed suitable for capture except for one seal noted with voluminous diarrhoea.

Body condition was assessed qualitatively in the present study, however, various quantitative indicators of body condition have been reported in the literature for pinnipeds (including Ryg et al. 1988, 1990; Nilssen et al. 1997; Pitcher et al. 2000; Chabot and Stenson 2002) and a body condition index has been developed for the leopard seal (Van Den Hoff et al. 2005). These quantitative indicators may be useful in future studies of this species.

9.4.1.2 Clinical Examination

Although thorough clinical examination was not possible for all seals due to the varying levels of immobilisation attained during protocol development, the majority of the seals observed appeared to be free of obvious clinical disease. A greater incidence of oral and nasal discharges, wounds and scars were observed in leopard seals examined in the 1999/2000 season than those from the 2000/01 and 2001/02 seasons. In addition, more seals were classed as being in fair/thin body condition in the 1999/2000 season than in the 2000/01 season. However, the differences between seasons apparent from clinical examination and body condition assessment were not reflected in significant differences between seasons in the blood values measured (see below).

9.4.1.3 Haematology and Biochemistry

Few significant differences in haematological and biochemical values were observed when seal sex, moult status and season of sampling were compared, however, the limitations of the small sample size used for this analysis, such as the influence of individual seals, needs to be recognised when interpreting the results of the statistical comparisons.

The only significant difference seen for season of sampling were anion gap and fibrinogen concentration. As anion gap is a derived value, it is difficult to attribute importance to the finding of significant differences between seasons for this analyte. Fibrinogen is an acute phase protein and a non-specific indicator of tissue damage and inflammation. Its concentration is thought to increase rapidly in inflammatory conditions thus the significantly higher fibrinogen concentrations determined in the 1999/2000 season may be reflecting the higher incidence of respiratory disease, including ocular and nasal discharges, observed in 1999/2000. However, it is important to note that although a significant difference was seen in fibrinogen concentration between years, the values are in fact low and within normal limits for reference intervals in other seal species.

The absence of significant differences with seal sex for the majority of haematological and biochemical variables is not unexpected and similar findings have been reported in previous haematological (Vallyathan et al. 1969; Lane et al. 1972; Goldstein et al. 1998; Hall 1998) and biochemical (Vallyathan et al. 1969; Geraci et al. 1979; Hall 1998) studies in pinnipeds. The significant differences determined with seal sex for a small number of values has no apparent implications for health status.

In the present study, significant differences in haematological and biochemical values were generally not observed with differing moult status, except for a higher eosinophil count in non-moulting compared to moulting seals. The moult in the leopard seal is relatively mild and only the hair is shed and during the moult leopard seals are still going into the water daily and presumably feeding. The moult was not reported to cause interference with the normal activities of leopard seals (Gwynn 1953) thus it is not unexpected that alterations in blood values were not seen with the moult. However, in other species such as in the harp seal, *Pagophilus groenlandicus,* moult was observed to be a stressful time associated with anorexia, ocular opacity, lethargy and irritability (Ronald et al. 1969, 1970) and decreases in red blood cell counts, haemoglobin concentrations and PCV have been reported during the moult in the harp seal (Ronald et al. 1969).

The development of reference intervals for haematological and biochemical values and other parameters of health in the leopard seal is an essential prerequisite for the utilisation of the leopard seal as an indicator species of change within the Antarctic ecosystem. Reports in the literature of blood values for leopard seals are scant and include haematological values in one leopard seal at Heard Island (Brown 1957) and haematological values and bicarbonate concentration in two captive leopard seals (Williams and Bryden 1993). The reference intervals for the majority of the haematological values determined for the leopard seal in the present study were similar to those previously reported in other seal species (including Lane et al. 1972; Geraci and Smith 1975; Needham et al. 1980; McConnell and Vaughan 1983; Bossart and Dierauf 1990; Roletto 1993; Horning and Trillmich 1997; Sepúlveda et al. 1999; Bossart et al. 2001; McFarlane this volume). An exception is the mean eosinophil count ($1.35 \times 10^9\ l^{-1}$) which is considerably higher than the values reported in other seal species (Geraci and Smith 1975; Banish and Gilmartin 1988; Roletto 1993; Nielsen 1995) except for yearling grey seals, *Halichoerus grypus,* ($1.33 \times 10^9\ l^{-1}$) (Hall 1998) and Australian sea lions, *Neophoca cinerea,* in

early lactation (1.63×10^9 l^{-1}) (Needham et al. 1980). In the absence of other causes of elevations in eosinophil count in the seals studied, such as skin and mucosal disease and allergic conditions, and with no evidence of obvious ectoparasitism, internal parasitism with prolonged exposure is thought to be responsible for the high eosinophil counts seen in the present study. Similarly, the reference intervals developed for the majority of the serum biochemical analytes in the present study are within the ranges reported for other seal species (including Hunter and Madin 1976, 1978; Geraci et al. 1979; Ronald and Kay 1982; McConnell and Vaughan 1983; Bossart and Dierauf 1990; Schumacher et al. 1992; Roletto 1993; Hall 1998; Nordøy and Thoresen 2002; McFarlane this volume). Where differences exist between the reference intervals determined in the present study with those of previous studies in pinnipeds, they may reflect true differences between species due to a number of factors including differing environments, diet and physiology, as well as methods of sample collection, storage and analytical technique.

9.4.1.4 Parasite Examination

The majority of the leopard seals in the present study exhibited evidence of internal parasitism. However, most of the seals were regarded as being in good body condition and it was thought that their parasite burdens did not affect their body condition. Heavy infestations of both gastric nematodes and intestinal cestodes have been reported in the leopard seal; however, the infestations were not thought to affect the general health of the animal (Gwynn 1953). An earlier study of the leopard seal in Prydz Bay found 100% of leopard seal scats examined in August and October and 60% of scats examined in September, contained nematodes (Green and Williams 1986). In addition, several species of cestodes of the family *Diphyllobothrium* Luehe, 1910 have been identified in the small intestine of leopard seals with reports of massive infection in the seals examined (Markowski 1952; Andersen 1987; Wojciechowska and Zdzitowiecki 1995).

9.4.2 Leopard Seals Hauling Out in NSW

9.4.2.1 Health Status

Many of the leopard seals hauled out along the NSW coast were thin and too weak to return to sea and a number of these seals were euthanased due to poor body condition which was evident in their sternal blubber thickness, which was markedly less than that observed in two adult leopard seals in Antarctica. These differences in blubber thickness may also reflect, in part, age differences as blubber thickness is generally increased in larger/older seals (Pitcher 1986; Castellini et al. 1993), as well as seasonal and physiological influences such as breeding status and moult status (Pitcher 1986; Nilssen et al. 1997; Chabot and Stenson 2002).

Differences were seen in haematological values when comparing leopard seals hauled out in NSW to those in Prydz Bay. Coupled with their poor body condition, the higher values for neutrophils and monocytes could indicate alterations in the health status of leopard seals hauled out in NSW including increased stress or inflammatory demand as well as the effects of manual compared to chemical restraint. Differences in biochemical values were also seen between the NSW and Prydz Bay seals, perhaps reflecting alterations in the health status of the seals hauling out in NSW, as well as changes arising from differing environmental, dietary and physiological influences. The biochemical data obtained from leopard seals in Prydz Bay were from frozen samples whilst fresh samples, or samples stored at −80°C for short periods of time, were generally employed for leopard seals in NSW. Studies assessing the effects of freezing on biochemical analyte concentrations in marine mammals (including Hunter and Madin 1978; McConnell and Vaughan 1983; Tryland and Brun 2001) report alterations in the concentrations of some analytes, most commonly decreases in enzyme concentrations including alkaline phosphatase and alanine aminotransferase. In a previous study in which the effects of long-term freezing (−80°C) of serum samples on biochemistry values was assessed in leopard and Weddell seals, *Leptonychotes weddellii* (Gray 2005), minor (although significant) changes in concentrations of a small number of analytes were seen with frozen storage, however, these changes would generally not be considered to be of clinical importance. For those analytes in which significant alterations in concentration were seen, such as amylase in the leopard seal, caution needs to be employed when interpreting the results of the analysis of frozen samples (Gray 2005).

Similar to the findings in NSW, leopard seals hauled out in other Australian States present with highly variable health status and body condition. For example, in Western Australia, some leopard seals were euthanased due to poor body condition and emaciation whilst other seals were reported to be resting prior to returning to sea (Mawson and Coughran 1999). In contrast to the findings in NSW, 53% (n = 34) of leopard seals in Tasmanian waters were reported to be in good condition, whilst the remainder of the seals observed were thin, found dead, shot illegally, or presented with other injuries and a higher proportion of these seals were regarded as being in poorer body condition than those on sub-Antarctic islands (Rounsevell and Pemberton 1994).

9.4.2.2 Seasonal and Annual Abundance

Similar to the findings in the present study, the majority of leopard seals sighted in Victoria, South Australia and Western Australia were seen from July to October, with a peak of sightings in August (Victoria and South Australia), September (Western Australia) (data obtained from the Wildlife Atlas Database, Museum Victoria, South Australian Museum and Mawson and Coughran 1999) and August and September in Tasmanian waters (Rounsevell and Pemberton 1994). Similarly, immature leopard seals congregate at sub-Antarctic islands such as Macquarie Island and the Kerguelen Islands, during June to December with the peak numbers

in August and September (Rounsevell and Eberhard 1980; King 1983; Rounsevell 1988; Borsa 1990; Walker et al. 1998). Presumed adults are sighted throughout the year at the Kerguelen Islands (Borsa 1990). At Heard Island, leopard seals are present all year round, with the lowest numbers observed during November and early December when it is assumed the adults leave for the pack-ice (Gwynn 1953). Some of the seals observed on the sub-Antarctic islands were found dead, were wounded, ill, thin, tired and unable to escape when disturbed (Gwynn 1953; Rounsevell and Eberhard 1980; Borsa 1990). The poor condition of the majority of these seals has been attributed to inexperienced prey catching ability and/or intra-specific competition for food (Rounsevell and Eberhard 1980). However, similar to the findings from Tasmania, apparently healthy seals were seen on Macquarie Island (Rounsevell and Eberhard 1980) and leopard seals generally appeared to be in good condition on Bird Island, South Georgia (Walker et al. 1998).

Numerous reasons are postulated for the northward dispersal of leopard seals into the sub-Antarctic and temperate regions, including changes in the extent of the pack-ice (Gwynn 1953; Rounsevell and Eberhard 1980; Kooyman 1981) creating a shortage of particular resources, such as krill, at the outer edge of the advancing ice front (Rounsevell and Eberhard 1980). It is thought that krill might be more heavily utilised by juvenile seals (Hofman et al. 1977) and the majority of the seals hauling out in NSW were less than 3-years-old and 77% of seals hauling out in Tasmanian waters were juveniles less than 2-years-old (Rounsevell and Pemberton 1994). Intra-specific aggression or food shortage (Rounsevell and Eberhard 1980; Siniff and Stone 1985) and dispersion occurring as part of the northerly migration (Gwynn 1953; Csordas 1963) are also suggested reasons for leopard seal dispersal.

Large inter-annual variation in leopard seal abundance has been reported in the sub-Antarctic (Rounsevell and Eberhard 1980; Walker et al. 1998). A periodicity in peak abundance of 3–4 years was observed at South Georgia (Walker et al. 1998) and 4–5 years at Macquarie Island (Rounsevell and Eberhard 1980; Rounsevell 1988) with peak years of abundance of leopard seals on Macquarie Island coinciding with the highest counts seen on the coasts of Australia and New Zealand sub-Antarctic islands (King 1983; Rounsevell 1988). Leopard seal sightings along the NSW coast between 1950 and 1981 showed a similar pattern in numbers, age and sex to those seen during the same period at Macquarie Island, again with an observed periodicity of 4–5 years (Elliot 1982). The Taronga Zoo archival records examined here, demonstrate that there is variability in the number of leopard seals hauling out annually in NSW, although there is little evidence of regular periodicity (Fig. 9.6). A larger data set collected over a longer time period would be necessary to determine if there is regular periodicity in leopard seal annual abundance in NSW. In a study of Tasmanian sightings, no apparent periodicity was observed for leopard seal sightings (Rounsevell and Pemberton 1994).

Local shortages in prey availability in the pack-ice may regularly act to produce the inter-annual variation (periodicity) in leopard seal numbers at Macquarie Island (Rounsevell and Eberhard 1980). It has also been suggested that the periodicity is a consequence of long-term climatic changes, specifically El Niño Southern Oscillation (ENSO) events (Harris et al. 1988; Testa et al. 1991) with peaks in leopard seal

dispersal to Macquarie Island most likely as a result of poor conditions for the population in the pack-ice (Testa et al. 1991). Regardless of the reason for the variation in annual abundance of the leopard seal in Australian waters, the majority of the seals observed are in poor body condition when compared to those seen in Antarctica and may have important implications for the management of these seals in Australian waters.

9.5 Conclusion

This paper has reported our efforts to establish reference intervals for a number of health parameters for leopard seals in Prydz Bay, Antarctica and the coast of NSW, Australia. Leopard seals in Prydz Bay were generally in good body condition and free of obvious clinical disease when compared to leopard seals found in NSW waters.

The differences observed in body condition, clinical disease and haematological and biochemical values between leopard seals sampled in Prydz Bay and those hauled out along the coast of Australia, suggest that leopard seals can be sensitive to changes imposed upon them by their environment. On a finer scale, seasonal variations in observed clinical signs and body condition were evident in leopard seals sampled in Prydz Bay, indicating that leopard seals respond to changes within their environment and that these responses can be determined by simple observation and by the measurement of commonly utilised variables.

In order to effectively monitor this species, baseline data for a number of variables needs to be obtained. In the present study, baseline data for haematological and serum biochemical values have been determined, and information on body condition, body weight, morphometric measurements and clinical examination of leopard seals in Prydz Bay has been presented. The development of baseline data from an apparently healthy, free-ranging population is essential to enable the effect of natural fluctuations and anthropogenic influences on the population to be studied, to understand the effect of the introduction of disease on the population as a whole, and to determine the health status of individuals.

Further investigations are required to clarify the role of the leopard seal as an indicator of change within the Antarctic ecosystem. Monitoring of parameters of health, as well as disease status, should also be undertaken to enhance knowledge of this species and the ecosystem it inhabits.

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Part II
External Factors: Environmental, Administrative and Legal

Chapter 10
Antarctic Climate, Weather and the Health of Antarctic Wildlife

M. Pook

10.1 Introduction

Climate and weather are the defining characteristics of Antarctica and to a large degree are what set it apart it from the other regions of the world. The interactions of the geography of Antarctica with large-scale climatic processes and the local weather conditions they generate influence all facets of the natural environment. Consequently, they will affect many aspects of disease in Antarctic wildlife and the way that humans can respond to disease events.

Climate and weather exert their influence on wildlife at a range of scales. The large-scale atmospheric pressure systems determine the isolation, or otherwise, of the Antarctic continent, at least for airborne particles. More locally, conditions of temperature, humidity and solar irradiance can all influence the survival of hosts, vectors and pathogens in the environment and can control or limit the actions of people. The extreme environmental conditions that characterise Antarctica may well mean that accepted rules of disease epidemiology may not apply here or that disease response procedures that are accepted as normal in the rest of the world may not be appropriate or even possible. For these reasons an understanding of Antarctic climate processes and weather conditions is of a very direct practical relevance to any consideration of health and disease of Antarctic wildlife.

As if the weather of Antarctica were not a sufficient challenge, there is evidence that the climates of the polar regions, including Antarctica and the Southern Ocean, are changing faster than many other regions as a consequence of global atmospheric changes. There is a growing concern that a combination of global warming, changes in atmospheric circulation patterns and rapidly accelerating human activity is contributing to an increased risk of the introduction of exotic organisms to Antarctica and the sub-Antarctic islands and their subsequent survival in these

M. Pook
The Centre for Australian Weather and Climate Research, CSIRO, Hobart Tasmania 7000, Australia
e-mail: Mike.Pook@csiro.au

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novel environments (Frenot et al. 2005). Yet, even without the effects of these longer-term trends, there is sufficient seasonal and year-to-year variability in the atmospheric system to suggest the possibility of a major regional climate anomaly providing suitable conditions for such an event over time.

Although the sub-Antarctic islands are located for the most part well to the north of the Antarctic continent, they are still very isolated from the major southern hemisphere continents and may be vulnerable to the introduction of new diseases and the spread of indigenous diseases in more favourable climatic conditions. Consequently, the sub-Antarctic region is included in this discussion for its own interest and also because the presence of new diseases in the sub-Antarctic may increase the threat to wildlife on the Antarctic continent itself.

10.2 Background Climate

The detailed climatology and meteorology of Antarctica and the sub-Antarctic islands has been presented by a range of authors (see for example, Astapenko 1964; Schwerdtfeger 1970; Schwerdtfeger 1984; van Loon and Shea 1988; King and Turner 1997; Phillpot 1997; Bromwich and Parish 1998) and only a brief outline will be given here. Many sources of real-time and historical data are available through the internet and a selection of these is provided at the end of this chapter.

10.2.1 Geography and Physical Considerations

The climate of the Antarctic region is strongly influenced by geography, the earth's orbital characteristics and the topography of Antarctica, itself. The Earth's axis of rotation is inclined by approximately 23.5° from the normal to the plane of the ecliptic and this imposes strict limitations on the receipt of direct solar radiation (short-wave) by the poles. It ensures one period of virtually continuous sunlight within the Antarctic Circle during summer and a 'polar night' during the winter months. Additionally, the southern hemisphere summer occurs during the period when the distance between the sun and earth is at a minimum (perihelion) while the austral winter occurs when the sun–earth distance is at a maximum (aphelion). Hence, the Antarctic receives a greater share of solar insolation in January than the Arctic region does in July during its summer.

The ice-covered Antarctic continent and surrounding sea-ice have a high albedo or reflectivity with respect to solar radiation. Approximately 75% of the total flux of incident solar energy is reflected from high concentrations (>85%) of sea-ice and as much as 90% from snow surfaces on the plateau (King and Turner 1997). The radiation received at the surface (global radiation) is the sum of the direct solar radiation and the diffuse or sky radiation. As the shallow dry atmosphere over the high Antarctic plateau is virtually transparent to the incident and reflected short-wave radiation, the

ratio of direct to diffuse radiation is higher in the Antarctic during summer than at lower latitudes.

The topography of the Antarctic continent plays a major role in the climate and weather of the region. The average elevation of Antarctica is about 2,500 m but the plateau in East Antarctica rises to about 4,000 m at its highest point. This extreme elevation and the cold surface contribute to the formation of katabatic winds – winds created as cold dense air falls under gravity from the high inner parts of the continent towards the coast. Furthermore, the combination of elevation and extreme cold results in continental Antarctica experiencing some of the driest atmospheric conditions known on earth.

One of the most important considerations in the climate of the Antarctic region is the annual cycle of sea-ice. Although the total area of Antarctica including its islands and ice shelves is approximately 14 million sq km, sea-ice contributes an additional 4 million sq km in February and approximately 22 million sq km in September (SCAR 2004).

10.2.2 The Broad-Scale Features

The climates of the sub-Antarctic islands are strongly influenced by the location of the oceanic Antarctic Polar Front (Antarctic Convergence). The position of this feature varies considerably around the hemisphere (Streten and Zillman 1984) but is generally found within the latitude zone of 47°S to 61°S (King and Turner 1997). There are distinct changes in ocean temperature across the frontal boundary and variations in the location of the front can result in marked climatic changes on sub-Antarctic islands. Within the atmosphere, a front representing strong north–south gradients of temperature is usually found between 40°S and 50°S, but it is not normally a well-defined continuous feature around the globe (King and Turner 1997). This front is an active region where atmospheric cyclonic systems frequently develop (cyclogenesis). The majority of these low pressure systems subsequently move polewards.

The broad-scale control on the climate of coastal Antarctica is the so-called circumpolar trough of low pressure which is located at an annual mean latitude of 65°S–66°S (King and Turner 1997; Schwerdtfeger 1984) but has a seasonal cycle of changing intensity and position (Fig. 10.1). The trough intensifies and moves closer to Antarctica in autumn and spring, while the average pressure is higher in summer and winter when the trough is located further from the coast. The circumpolar trough not only represents the integrated effect of the synoptic cyclonic systems that develop and decay within this region but also reflects the fact that this is the region to which extratropical cyclones migrate after developing and intensifying in the mid latitudes (Jones and Simmonds 1993). Most cyclones follow south-easterly tracks towards the Antarctic coast. On the northern side of the circumpolar trough the mean isobaric pattern indicates that westerly winds predominate, while on its southern flank the winds are generally easterlies. The sub-Antarctic islands mainly lie within the belt of westerlies while close to the Antarctic coast katabatic winds flowing out from the continent contribute to the easterly winds.

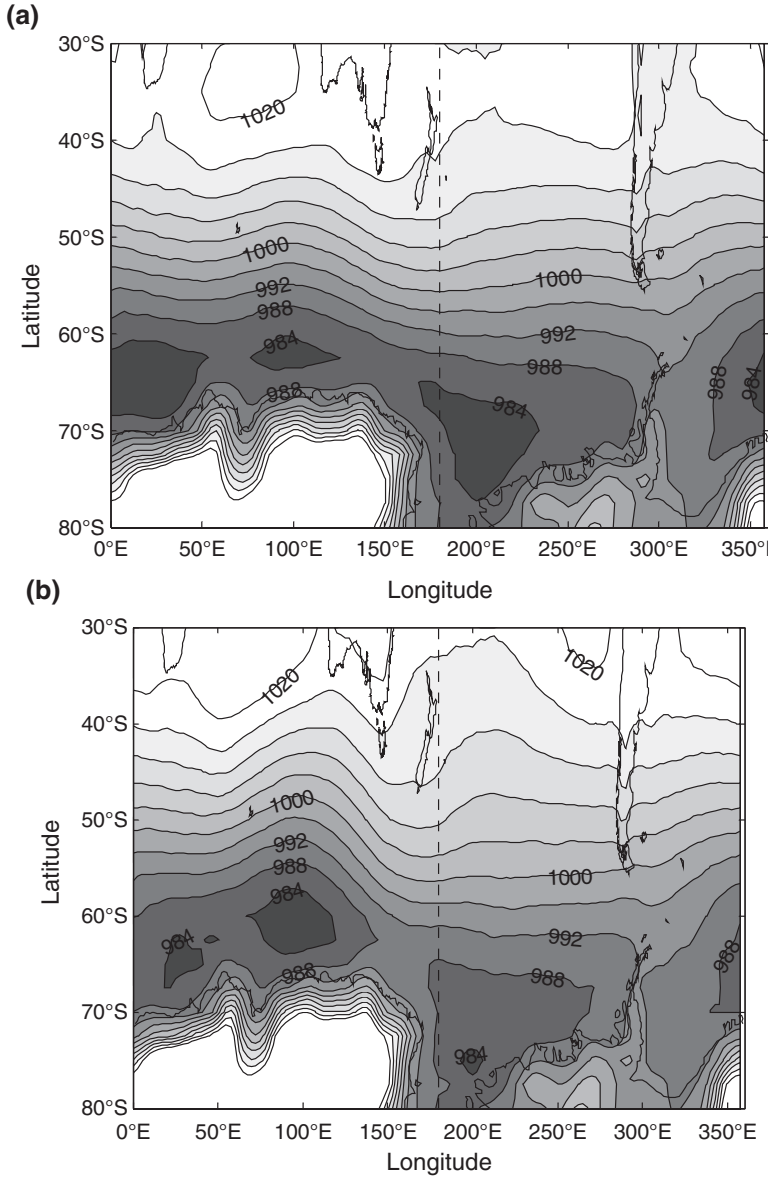

Fig. 10.1 Seasonal means of mean sea-level pressure from NCEP/NCAR reanalysis data (Kalnay et al. 1996; Kistler et al. 2001) averaged over the period 1970–2004 for (**a**) autumn (Mar–May), (**b**) winter (Jun–Aug), (**c**) spring (Sep–Nov), and (**d**) summer (Dec–Feb). *Darkest shading* indicates lowest pressures

The synoptic low pressure systems (extratropical cyclones) constantly move through the region of the trough, predominantly from west to east, so that there are regular changes in the direction and strength of wind. Most of these episodes do not persist for more than a few days, at most. However, the circumpolar trough is occa-

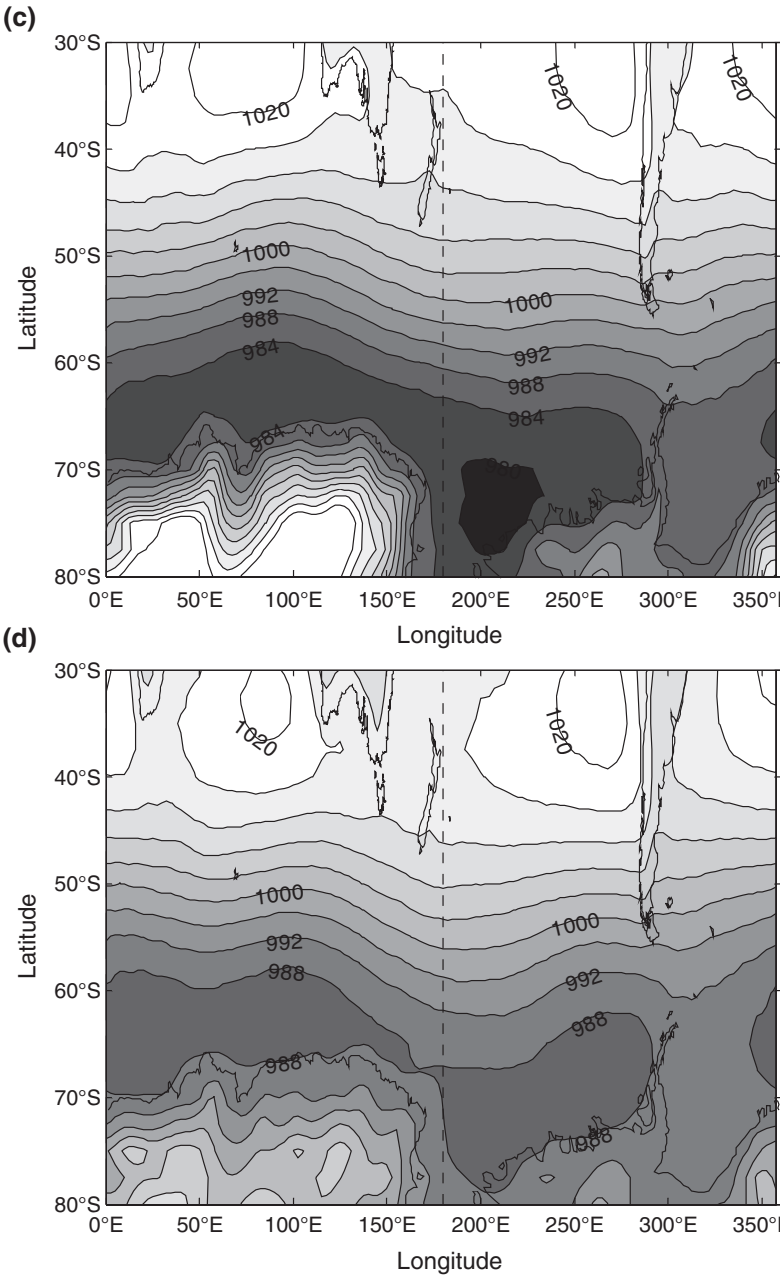

Fig 10.1 (continued)

sionally interrupted by the development of high pressure systems (anticyclones) at mid and occasionally high southern latitudes. These slow-moving anticyclones are commonly known as blocking highs because they can have significant effects on

the behaviour of cyclones such as impeding their progress towards the east and steering them polewards. A configuration of synoptic weather systems of this type frequently results in warmer and more humid air moving over large distances from the north towards the Antarctic coast (Goodwin et al. 2003; Pook and Cowled 1999; Pook and Gibson 1999; Turner et al. 2002). Such a physical process may have biological and ecological consequences if the movement of air from the north causes the translocation of organisms into the Antarctic region.

Atmospheric blocking is a significant feature of the winter circulation in the southern hemisphere with highest frequencies of occurrence in the Pacific sector but instances of blocking can occur throughout the hemisphere at any time of the year (Lejenäs 1984). Schwerdtfeger (1984) has reported a significant change in wind conditions from strong westerly winds to moderate northeasterlies which occurred during a 10-day period in June 1952, when an intense blocking high persisted between the Falkland and South Shetland Islands. Sinclair (1996) has found that there are two regions in the southern hemisphere where intense and persistent blocks tend to occur. Both of these regions are in the Pacific Ocean, one to the southeast of New Zealand and the other southwest of South America, near 110°W.

Atmospheric blocking has been observed to be a major influence on the paths followed by cyclones in the Pacific sector of the Southern Ocean during winter (Pook and Gibson 1999; Pook and Cowled 1999), apparently contributing to the crossing of the Antarctic coast by vortices on several occasions and causing warmer air and precipitation on the eastern side of these systems to penetrate well inland. Advection of cloud and precipitation over the East Antarctic Plateau has been associated with these and other synoptic events involving atmospheric blocking to the southeast of Australia (Massom et al. 2004).

Variability in snow accumulation in Wilkes Land, East Antarctica, has provided evidence of the effect of periodic incursions of high pressure systems into the Antarctic Circumpolar Trough during the period 1930–1985 (Goodwin et al. 2003). Decadal fluctuations and the overall trend of increasing accumulation since the mid-1960s suggest a poleward shift and intensification of the circumpolar trough. Year-to-year variability of snow accumulation rate throughout Wilkes Land has been found to be dependent upon shifts in the preferred tracks of cyclones along or across the coast, demonstrating how local weather conditions are influenced by large-scale regional atmospheric processes.

Clearly, atmospheric blocking is a key factor in the advection of mid-latitude air into the sub-Antarctic and Antarctica itself and is likely to form an important component of any conceptual model which attempts to explain the introduction of exotic organisms to the region by natural means.

10.2.3 Temperature Distribution and Variability

A detailed description of the temperature distribution over latitudes south of 40°S has been given by Van Loon and Shea (1988) and Schwerdtfeger (1970). Schwerdtfeger (1984) has investigated the temperature variation over the Antarctic

plateau at the surface and throughout the lower portion of the atmosphere. In particular, he has drawn attention to two significant features of the annual march of temperature on the high plateau in the interior of Antarctica. The 'pointed' summer refers to the brief period of maximum temperatures around the summer solstice in late December and early January. By way of contrast, the so-called 'coreless' winter is the name given to the long drawn-out period of up to 6 months between April and September during which annual minimum temperatures occur. Despite the absence of solar radiation during this period, monthly mean temperatures vary only slightly, indicating that exchange of air with lower latitudes continues during winter (King and Turner 1997).

The form of the annual cycle of temperature alters significantly with movement towards the coast (King and Turner 1997) so that the locations on the coast of East Antarctica and on the Antarctic Peninsula have a broad summer maximum and a minimum around July or August.

Both inland and coastal stations display a surface inversion in the temperature–height profile in the winter months but this feature is much more defined over the high plateau than it is near the coast. In summer the inversion is only weakly present on the plateau and almost disappears near the coast.

Monthly temperature ranges for Antarctic and sub-Antarctic stations are available from many sources, including Schwerdtfeger (1984), King and Turner (1997), Phillpot (1997) and Turner and Pendlebury (2004). Broadly speaking, mean air temperatures during summer range from near 0°C around the coastal margin of Antarctica to about −40°C on the plateau. In winter, mean coastal temperatures are generally within the range of −18 to −29°C and mean temperatures on the plateau are around −68°C (SCAR 2004). In contrast, monthly mean temperatures for the northern section of the Antarctic Peninsula exceed 0°C in summer (King and Turner 1997).

The temperature ranges at the sub-Antarctic islands depend largely on their location relative to the oceanic Antarctic Polar Front. Macquarie Island, at approximately 54.5°S 159°E, is located to the north of the Polar Front and has winter mean maximum temperatures of about 5°C and summer mean maxima of about 8°C. In contrast, Heard Island at approximately 53°S 73.4°E, lies to the south of the front and has winter mean maximum temperatures of about 1°C and summer mean maxima of about 5°C. Grytviken, on South Georgia Island (approximately 54°S 36.5°W) is also located south of the front but, unlike Heard Island, lies near the northern extent of Antarctic sea-ice and experiences a mean temperature in June of about −1.5°C (Turner and Pendlebury 2004).

10.2.4 The Surface Wind and Its Effects

One of the most outstanding features of the surface wind field in the Antarctic region is its constancy of direction (Schwerdtfeger 1984; Bromwich and Parish 1998). This is a result of the winds being largely generated by the strong radiative

cooling of the air in contact with the sloping surface of the ice. The direction of the flow is controlled by the slope of the underlying surface and concentrated by topographic features such as glacial valleys. The flow down the slope is acted on by friction and the Coriolis force due to the earth's rotation and the wind is deflected to become a generally easterly wind. As these katabatic winds blow towards the coast, the wind in the vicinity of the steeper coastal slopes frequently reaches gale or storm force, a fact to which many an explorer's diary will attest (e.g. Mawson 1915; King 1982; Cherry-Garrard 2003). According to Bromwich and Parish (1998), the steep ice slopes along the coast of East Antarctica are associated with the strongest surface winds found anywhere on earth. Cape Denison, the site of Mawson's base in 1911–1914, is credited with the highest annual mean wind speed (19.4 ms^{-1}) ever recorded (King and Turner 1997).

The often violent wind events around the Antarctic coast are accompanied by phenomena which have important implications for human activities such as outside field work and maintenance, aircraft operations and shipping. The onset of wind events can be very sudden and may be difficult to forecast because they are often conditioned by the local terrain. The winds can be associated with sharp changes in atmospheric pressure and there may often be rotor clouds evident in the vicinity. These vertically overturning cloud fragments are indicators of severe turbulence. Extremely strong winds at coastal stations normally result from interactions of the katabatic winds with strong pressure gradients created by low pressure systems moving around the Antarctic coast, sometimes centred hundreds of kilometres offshore.

It was demonstrated in the previous section that the large synoptic systems moving to the north of Antarctica could bring mild maritime air from the low latitudes to the Antarctic coast and it was suggested that these airstreams could convey exotic organisms to the continent, given the right conditions. However, the winds near the Antarctic coast are generally flowing outward from the continent. Their role is more a factor affecting how humans are able to deal with outbreaks of disease. The strength of the wind is not the only factor to consider. The combination of even moderate wind strengths with the intense cold can result in severe heat loss from the human body, the so-called wind-chill effect (Schwerdtfeger 1984), leading to rapid damage to exposed skin and risk of hypothermia if adequate precautions are not taken.

Furthermore, the wind is capable of raising ice particles from the surface (known as drift) even at moderate speeds of about 8 ms^{-1} (Turner and Pendlebury 2004). This drift can affect visibility and as the wind speed increases the height of the drift also increases, reducing visibility significantly. When drift is raised above eye level it is reported as blowing snow in weather observations. A blizzard is said to occur when the temperature is at or below freezing, the wind speed exceeds 17 ms^{-1} and visibility drops to 100 m or less in blowing snow. It is normally impossible to tell in these circumstances whether snow is also falling from cloud.

Onshore flow of maritime air leads to the formation of fog and low cloud as the air is cooled from below by the ice surface and by orographic lifting by the coastal slopes. Offshore flow, on the other hand, normally involves extremely dry air which can occasionally produce a form of 'steaming' fog where the cold air moves over open water 'leads' in the sea-ice. This fog can lead to local reductions in visibility but is usually quite unstable and tends to ascend in a similar manner to steam; hence the name.

While winds around the Antarctic coast generally blow from the east, there are regions where topography acts on the cold dense air to create special effects. On the eastern side of the Transantarctic Mountains and the Antarctic Peninsula cold, persistent winds from a southerly or southwesterly direction are often experienced and are known as barrier winds (Schwerdtfeger 1984; King and Turner 1997).

On the sub-Antarctic islands the wind directions are generally from the west as the islands are located to the north of the circumpolar trough of low pressure. Intense low pressure systems move regularly from west to east through these latitudes and create strong pressure gradients which result in regular, strong wind events. These systems can be accompanied by heavy precipitation in the form of snow, rain and sleet, making field operations very difficult. The periods of strongest winds are normally encountered in late winter and spring as the circumpolar trough reaches its peak intensity at that time. The belt of strongest westerly winds around the hemisphere is found in the Indian Ocean sector between about 45°S and 55°S. Crozet Island (approximately 46.7°S 52°E) has a monthly mean wind speed of 11.5 ms^{-1} in July while Kerguelen Island (49.35°S 70.3°E) has a mean wind speed of 10.7 ms^{-1} for the months of August and September (Turner and Pendlebury 2004).

10.2.5 Climate Trends

Mean annual temperature records for many Antarctic stations exhibit a warming trend with the strongest signal appearing over the Antarctic Peninsula (King and Turner 1997; Marshall et al. 2002). According to Marshall et al. (2002) the near-surface warming observed on the western side of the Antarctic Peninsula is among the largest observed across the globe during the last 50 years (1951–2000) with the greatest warming trend (more than 5°C) occurring in the winter and a weaker trend (approximately 1°C) in summer. Paradoxically, the smaller summer increase has been implicated in the decay of some of the ice shelves about the northern Peninsula and recent changes in the distribution of penguin species according to whether their winter habitat tends to be in the pack-ice or open water (Marshall et al. 2002).

Thompson and Solomon (2002) argue that the warming over the Peninsula region and slight cooling of the Antarctic interior are consistent with a systematic bias towards a strong polar vortex in the lower stratosphere and troposphere, as measured by the Southern Annular Mode (SAM) index. They postulate that this trend towards positive values of the SAM index is linked to stratospheric changes induced by decreasing ozone concentrations. The instrumental record for the Antarctic region is relatively short and a definitive answer to this question is yet to be found. However, Goodwin et al. (2004) have found recently that a proxy record of the SAM in an ice core drilled at Law Dome near Casey from 1300 to 1995 exhibits a pronounced decadal scale variability and that a weak positive mean is evident in the past 500 years, suggesting that longer-term influences are present.

Walther et al. (2002) report that rapid environmental warming has been reported over the last 30–50 years at a number of stations in the Antarctic, particularly in the Antarctic Peninsula region and on sub-Antarctic islands, and this trend has been

accompanied by changes in precipitation patterns. In the Antarctic terrestrial ecosystems, there are many examples of biological changes in response to climatic warming with an increasing number of human-mediated imports of non-native species, particularly to the sub-Antarctic region (Walther et al. 2002).

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, most notably in the northern hemisphere (Cubasch et al. 2001). The consensus of increases in annual mean temperature for the Antarctic region from a selection of climate models is in the range of 1–4°C by 2100 (Cubasch et al. 2001). These estimates are from broad-scale simulations and high-resolution regional simulations can be expected to show finer detail of warming and possibly, some regional cooling trends.

The projected changes in mean temperatures are unlikely to significantly affect the environmental conditions favourable to disease in the Antarctic in the short term but may become significant on the mid to longer time scales. When coupled with extended periods of altered atmospheric circulation in the vicinity of the Peninsula, increasing mean temperatures could increase the risk of translocation of diseases and parasites from lower latitudes and assist in the survival of these organisms. Climate change, leading to environmental conditions that are outside the normal range, may cause stress to some animals, which can in turn lead to increased susceptibility to disease. In the long-term this could be particularly significant in the Antarctic where, for some coastal populations, there may be no option to move further south to escape a warming environment.

Climate change leading to changing patterns of sea-ice distribution and duration could also have indirect detrimental effects on those species that are dependent on sea-ice as a place to breed, feed or travel. Warming could reduce the habitat available to those species that are dependent on the productivity of annual sea-ice for their survival.

Large icebergs can significantly change sea-ice patterns by restricting the movement of ice and preventing it from breaking up. They can also result in the creation of ice-free polynas on their down-wind sides. An increase in the number of large icebergs created by the break-up of ice-shelves increases the chances of icebergs of sufficient size becoming grounded in locations that change sea-ice conditions such that primary productivity is altered or that animals can no longer access feeding grounds or breeding sites (Arrigo et al. 2002).

10.3 Extreme Regional Climate Anomalies and Their Biological Consequences

10.3.1 Antarctic Peninsula and the Weddell Sea Region

Turner et al. (2002) report that an anomalous atmospheric pressure pattern prevented the re-supply of the UK base, Halley, in December 2001 when heavy ice conditions persisted in the eastern Weddell Sea. This was the first occasion since the International

Geophysical Year (1957–1958) that the supply ship had been unable to reach the base. Apart from severely altering ice conditions in the region, the intense long-lived atmospheric blocking episode over the South Atlantic Ocean coupled with anomalously low surface pressure over the southwest Weddell Sea, the Antarctic Peninsula and the Bellingshausen Sea also resulted in the advection of relatively warm mid-latitude air into the interior of the continent. Referring to the same climatic anomaly, Massom et al. (2006) assert that exceptional sea-ice conditions also extended to the West Antarctic Peninsula region with an unusually compact marginal ice zone and a major increase in ice thickness as a result of deformation and over-rafting. Ecological effects were dramatic. The largest recorded population decrease between successive seasons and the lowest reproductive rate were recorded in an Adélie penguin population that had been continuously studied for 30 years. This rare event can be explained in terms of the natural variability of the atmospheric system but longer-term trends cannot be dismissed. It provides an example of how the opportunity for a foreign organism to be carried to the Antarctic Peninsula and the Antarctic continent by natural means may develop and it also indicates a mechanism by which introduced species could experience favourable climatic conditions for an extended period.

10.3.2 East Antarctica

The weather conditions caused by atmospheric blocking have been associated with unusual biological events.

A Kerguelen pintail duck, *Anas eatoni*, a species not recorded previously from Antarctica, was discovered alive but died shortly after at the Mawson Station (67°35′S 62°52′E) on 6 December 2001 (Johnstone and Irvine 2004). This species has a distribution restricted to the vicinity of the Kerguelen Archipelago (Port-Aux-Français 49°21′S 70°13′E) and is rarely recorded as a vagrant elsewhere. It is suggested that it was caught in the interaction between an intense depression and a blocking anticyclone in the region east of Kerguelen Island and was transported about 2,000 km southward.

Similar episodes of blocking and associated intense depressions in late 2001 may have caused the strong oceanic swell from the north which resulted in the breaking up of the sea-ice at the shore, also in the vicinity of the Mawson Station which trapped, injured or killed Adélie penguins among blocks of floating ice at Welch Island and elsewhere along the coast of Mac.Robertson Land (Kerry et al, this volume).

The climatological situation in the southern Indian Ocean late in 2001 was part of an enhanced hemispheric three-wave pattern of atmospheric blocking anticyclones separated by low pressure troughs. According to Turner et al. (2002) the magnitudes of some of the 2001 atmospheric circulation anomalies are unprecedented in the 1971–2000 reanalysis period.

Figure 10.2a shows the synoptic situation at mean sea level on 28 November 2001 when an extensive high-pressure system became established at high latitudes just to the east of the Kerguelen Island. The blocking anticyclone had become evident on 26 November but steadily intensified on 27 November and remained almost

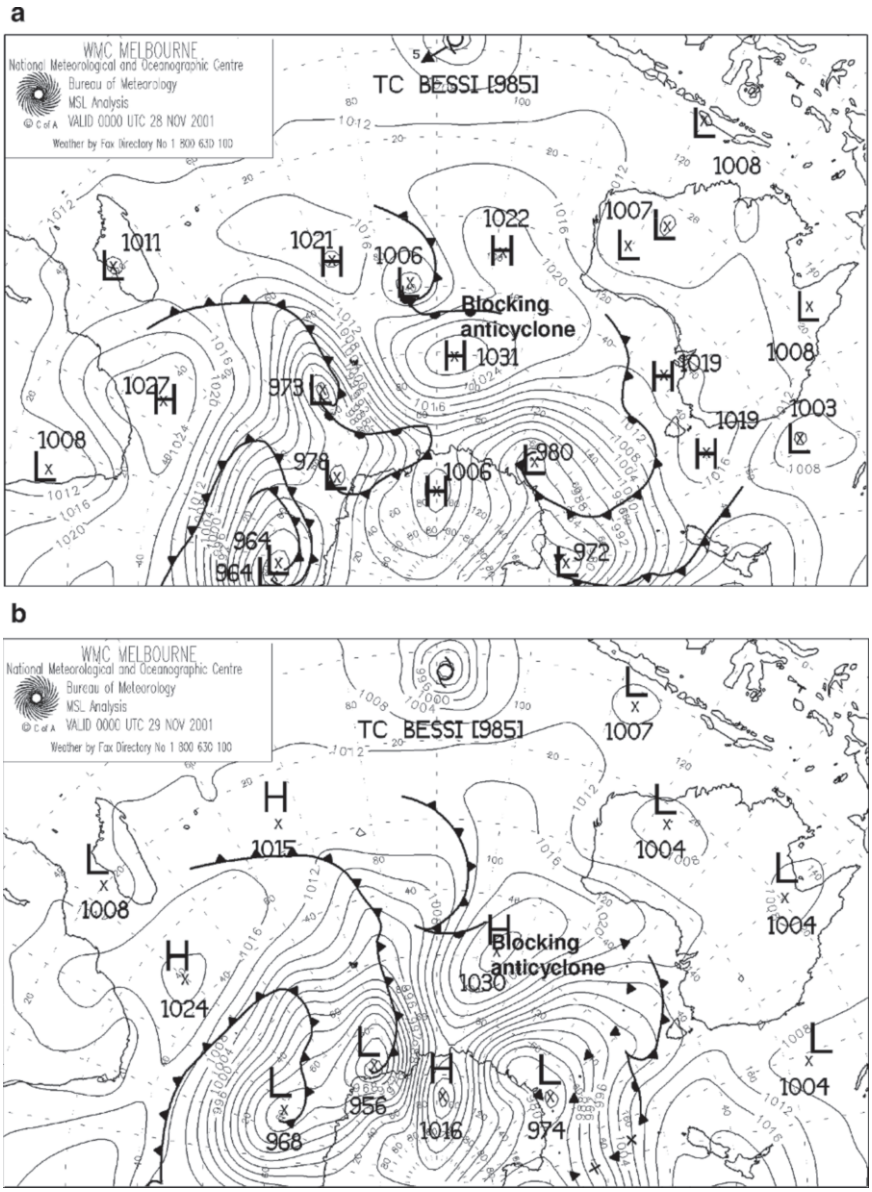

Fig. 10.2 Mean sea-level pressure analyses for the Indian Ocean and East Antarctica at (**a**) 00 UTC on 28 November 2001, and (**b**) 00 UTC on 29 November 2001, showing the intense blocking anticyclone (*labelled*) to the southwest of Australia (Australian Bureau of Meteorology routine analyses)

stationary on 28 November before drifting eastwards during the following day. Figure 10.2b indicates the location and intensity of the anticyclone at 00 UTC on 29 November 2001. The slow movement of this high-pressure system appears to have

been a critical factor in producing an extended period of strong northerly winds as a depression approaching from the west was steered southwards and intensified near the Antarctic coast. Long-lived blocking highs in the southern hemisphere are particularly associated with the southwest and southeastern regions of the Pacific Ocean (Sinclair 1996) but a secondary maximum of occurrence of positive pressure anomalies persisting for at least 5 days has been found near 50°S 80°E in summer (Trenberth and Mo 1985).

10.4 Conclusion

The climate and weather of Antarctica make it a unique environment which has remained relatively isolated from other regions of the world. However, the global climate is changing and most predictions are that the climate of polar regions, including Antarctica, will change faster than other places on earth. Such changes could bring about profound and, as yet, unpredicted changes to the ecology of the Antarctic region. Climate and weather studies are thus fundamental to the future understanding of the ecology of species and their health

The weather affects every aspect of the lives of the animals and plants that live in the Antarctic region. It defines the habitat in which the native species live, the range of conditions they must survive and the conditions that introduced pathogens and their vectors must withstand if they are to establish and spread. It also sets limits on what people can do in Antarctica and how they could respond to a wildlife health emergency. Activities that can be done very easily in the temperate world can become major challenges because of the weather of Antarctica. Simple tasks can be very difficult when working at very low temperatures in bulky clothing and wearing gloves. Response plans for animal health emergencies must, above all, recognise and accommodate the realities of working in Antarctic weather if they are to be of practical use.

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Sources of Meteorological Data

1. The READER (Reference Antarctic Data for Environmental Research) project of the Scientific Committee on Antarctic Research (SCAR) provides ready access to high quality long-term data of mean surface and upper air meteorological measurements from in situ Antarctic observing systems. The primary sources of data are the Antarctic research stations and automatic weather stations operated by the national Antarctic research organisations.
   A background to the project together with data and metadata can be found at: www.antarctica.ac.uk/met/READER/
2. The Antarctic Meteorological Research Center (AMRC) at the University of Wisconsin–Madison's (UW–Madison) Space Science and Engineering Center (SSEC) makes available automatic weather station (AWS) data in real time and archives data from AWS. It also provides composite satellite imagery. (see http://ice.ssec.wisc.edu/)
3. Detailed treatments of typical weather conditions, climatological data and forecasting requirements for a large number of locations in Antarctica and the sub-Antarctic islands have recently become available with the publication of the International Antarctic Weather Forecasting Handbook (Turner and Pendlebury 2004). The International Antarctic Weather Forecasting Handbook is available as a single portable document file (PDF) of approximately 26 Mb from the following sources:
   Australian Bureau of Meteorology (www.bom.gov.au/weather/ant/)
   Office of the Council of Managers of National Antarctic Programs in Hobart, Australia (www.comnap.aq/publications/manuals/)
   British Antarctic Survey in Cambridge, UK (www.antarctica.ac.uk/met/jtu/ftpinst.html)

Chapter 11

National Antarctic Programs and Their Impact on the Environment

J. Jabour

11.1 Introduction

This chapter describes the nature and scope of government operations in the Antarctic, touching briefly on the sub-Antarctic islands and also on the growing non-government tourism industry. Its aim is to provide the context for estimating the overall size and impression of the human footprint (i.e. the spatial extent of disturbance) and the degree to which human activity might pose a risk to the health of Antarctic wildlife. The consequences of the introduction and/or spread of disease, which is the subject of several other chapters in this book, largely depend on the nature of those activities.

Information about national operations has been obtained from a number of sources. This chapter relies on information exchanged by each Contracting Party in accordance with the requirements of Article VII.5 of the Antarctic Treaty and Article 17 of the Madrid Protocol (ATS 2009) and collated by the Antarctic Treaty Secretariat and the Council of Managers of National Antarctic Programs (COMNAP). The Antarctic Treaty Secretariat was established in 2004 and is hosted by the Government of Argentina in Buenos Aires to, inter alia, facilitate the exchange of information between the Parties and to maintain databases of information. Notification about national program activities is available either directly through the Secretariat's website (www.ats.aq) or through its links to the National Antarctic Programs. Three categories of information are collected by the Secretariat: pre-season, annual (end of season) and permanent, in compliance with Resolution 6 (2001) of ATCM XXIV. The Antarctic Treaty Parties established COMNAP in 1988 and its primary function and activities are related to '…the exchange of practical operational information with a view to improving the way all National Programs can fulfill their various missions, together or independently. That includes mutual support in the design, ongoing improvement and operation of Antarctic facilities and transport infrastructure' (COMNAP 2009).

J. Jabour
Institute of Antarctic and Southern Ocean Studies, University of Tasmania, Private Bag 77, Hobart 7005, Australia
e-mail: julia.jabour@utas.edu.au

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Reports from teams of observers, carrying out the inspection functions prescribed in the Antarctic Treaty Article VII and Madrid Protocol Article 14 will be used to estimate and interpret national program activities and their impact. Although inspections are rare and until recently were not systematically organized, they produce official documents which make a useful contribution to the store of knowledge about national program activities. Two inspections carried out during 2005 are used here as examples. A team from Australia inspected Scott Base and McMurdo Station in the Ross Sea region in January 2005 (referred to here as Inspection Report 1). The United Kingdom, Australia and Peru conducted joint inspections during February and March 2005 of 32 facilities, including stations (both occupied and unoccupied), a station under construction, historic sites and monuments, and vessels in the Antarctic Peninsula (referred to here as Inspection Report 2). The purpose of inspections is to assess consistency between exchanged information and the actual situation on the bases and to assess the level of compliance with the Madrid Protocol. Before the report is made public, each Party whose facilities were inspected has a right of reply.

For primary information relating to tourism, this chapter draws heavily on the International Association of Antarctica Tour Operators (IAATO), which claims to be the largest non-government operator in Antarctica (IAATO 2009). IAATO collects and collates information on tourism operations from the 100 companies that currently comprise its membership. In addition, it has useful insights into companies and individuals who operate outside its realm, as not all Antarctica tour operators are members of the IAATO.

Because all proposals for human activity in Antarctica are subject to environmental evaluation, the primary source of information about national scientific projects, significant logistic activities and tourism ventures is sourced from Initial and Comprehensive Environmental Evaluations stored on the Antarctic Treaty Secretariat's database (ATS 2009).

11.2 Antarctica: From Terra Incognita to Terra Vitalis

Throughout its short social history, the Antarctic has been *discovered* and many of its important secrets *unlocked* but it is fair to say that it has never been *conquered*. No indigenous human societies have evolved to live in Antarctica and no communities in Antarctica are self-sustaining. Instead, an artificial environment supported from lower latitudes is required to keep all who visit or work there safe and secure in the extreme climatic conditions. With acknowledgement that 'the Antarctic derives much of its scientific importance from its uncontaminated and relatively undisturbed condition' (ATCM Recommendation XII-3 1983), comes the imperative that, in the establishment of infrastructure essential to supporting their research projects, the National Antarctic Programs should coordinate and collaborate with each other. Furthermore, each activity is to be conducted with the least environmental impact possible under the circumstances. Starting from a fundamental base of scientific interest, national Antarctic programs have expanded, and their numbers have

increased, with the growing realization that Antarctica is a keystone in the earth's climate system. But with this expansion has come a growing footprint that has the potential to degrade Antarctica's scientific and cultural importance.

The discovery of *terra incognita* evolved over many centuries as piece by piece the human world was traced out, bordered and eventually transcribed onto maps (Murray 2005). The last piece of the puzzle was incorporated less than 200 years ago and while the discoverers – Bransfield, Bellingshausen and Palmer – remain relatively obscure, the explorers – Cook, Scott, Amundsen, Shackleton and Mawson, among others – leave an enduring legacy of stories, place names and inspiration. Their stories transport us back to a place in time that is both romantic and engaging from our contemporary armchairs, and inspires us to perpetuate the myths and preserve the icon so that its status will never be compromised. Today, maintaining that perspective and managing the impression that human activity in Antarctica is innocuous go hand in hand. The reality is, unfortunately, somewhat different.

The first wave of recorded human activity, beginning with Captain Cook's voyages, led to a regrettable state of affairs: the unfettered exploitation of wildlife, resulting in the destruction or near collapse of populations of whales, seals and some penguin species and the introduction and the long-term survival of a number of alien species of flora and fauna to the newly-discovered sub-Antarctic islands, many of which were used as bases for harvesting operations. Ironically it was this devastation that motivated some early scientific research (albeit for commercial and nationalistic purposes). Later, expeditions to and within the Antarctic continent were driven by inexorable human curiosity, with scientific endeavours conducted more intensely over the 50 years following the International Geophysical Year 1957/58. This event was a significant display of Cold War posturing in scientific clothing, but it also became a proving ground for international cooperation that led indirectly to the negotiation of the Antarctic Treaty of 1959 (Beck 1986) and inspired the contemporary face of Antarctica as a continent of peace and science. Subsequent human activity (scientific research and support, tourism and to a lesser extent fishing) heralded the modern era, in which regulation now prescribes what humans can and cannot do. That Antarctica is now *terra vitalis* is without question thanks to important scientific discoveries, and the protection of its integrity has become the primary aim of the Antarctic Treaty Contracting Parties today.

11.3 National Antarctic Programs – Overview

Scientific inquiry was, and still is, the superficial driver of national interest in Antarctica but it serves the dual purpose of also giving legitimacy to sovereign interests. Science and national interests are so thoroughly interconnected that trying to tease them apart is hardly useful in this context. In the 50 years since its adoption, the number of nations that are parties to the Antarctic Treaty has increased from 12 in 1959 to 47 today (ATS 2009). The original signatories all had Consultative Party (decision-making) status under the terms of the Treaty (Article IX), so the Treaty

System began with 12 Consultative Parties and this has now grown to 28. For Acceding States, Consultative Party status is achieved when they demonstrate 'substantial scientific research activity [there], such as the establishment of a scientific station or the dispatch of a scientific expedition' (Article IX.2). Taking the condition in Article IX literally, common interpretation was that becoming a Consultative Party required the establishment in Antarctica of infrastructure to support its scientific activities. However, this is not a necessity; for example, the Netherlands have achieved Consultative Party status without establishing facilities but instead by undertaking substantial scientific research using the infrastructure of other parties.

Building a station is still considered a prestigious activity despite modern interpretation of Article IX.2 being broader and including collaborative projects and expeditions with existing programs as evidence of substantial scientific research activity. In 2007 there were 36 year round facilities in Antarctica (Fig. 11.1). Infrastructure at a typical national research station may include buildings for accommodation, recreation, medical services, laboratories, kitchen and ablution

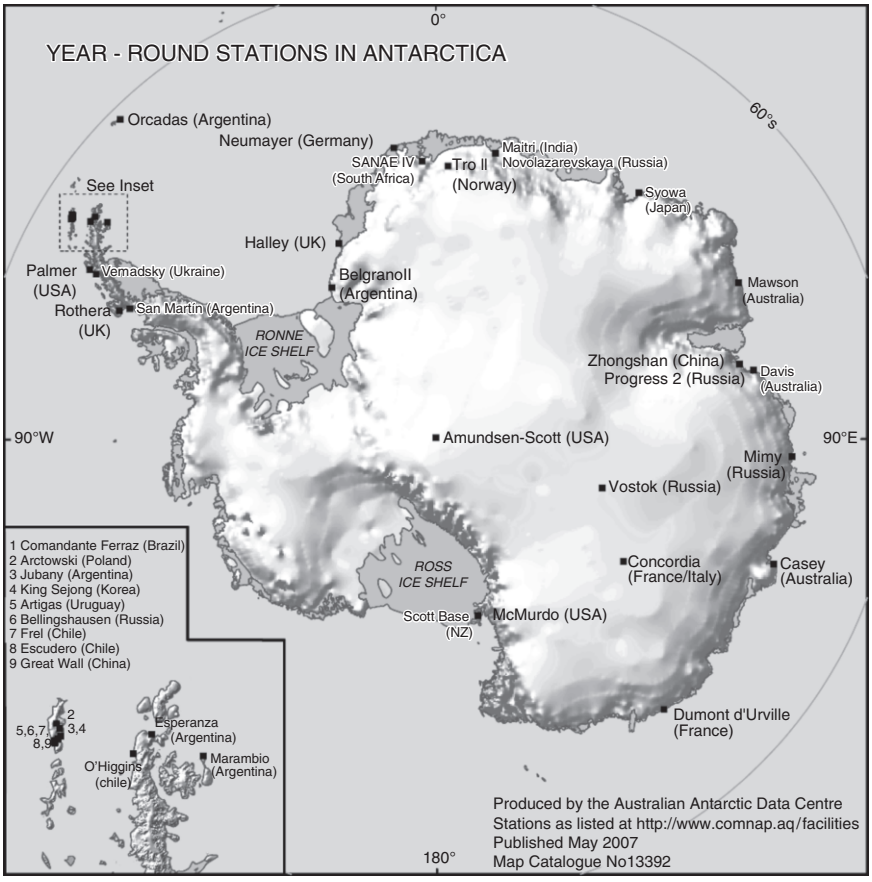

Fig. 11.1 Year-round stations in Antarctica

facilities, workshops, garages, hangars and storehouses. Within station limits there are also likely to be facilities for power generation (including fuel storage areas), communications and waste treatment (including incinerators, compactors and rubbish dumps). Roadways, helicopter landing sites and aircraft runways are also commonly needed. Service routes may extend well beyond station limits, as with the roadways connecting McMurdo Station to Scott Base, Bellingshausen to Frei and from Casey Station to its Wilkins airstrip, 75 km away.

Many Antarctic Treaty Consultative Parties, especially the claimants (Argentina, Australia, Chile, France, New Zealand, Norway and the United Kingdom) have stations of long standing, both on the continent proper and on sub-Antarctic islands (particularly Crozet, Heard, Kerguelen, Macquarie, Marion and South Georgia). Argentina, for example, established its continental Orcadas station in 1904 and it is the longest continuously operating scientific station in Antarctica. A research station was established on Marion Island (South Africa) in 1947 and at Heard Island (Australia) the same year. Most of the stations (both continental and island) of the original signatories to the Antarctic Treaty date from around the time of the post-war International Geophysical Year and many of these parties now have more than one research station (Table 11.1). In addition, the two States that reserve the right to make a claim under Article IV of the Antarctic Treaty, the United States and the Russian Federation, have both maintained stations since 1956. Over time, the States acceding to the Treaty have added their own infrastructure, creating a proliferation of

Table 11.1 Antarctic stations and their populations (COMNAP 2009)

Name of facility	Operated by	Station first opened	Station current status	Winter population	Station peak population
Aboa	Finland	1989	Seasonal	n/a	20
Amundsen-Scott	USA	1956	Year-round	75	250
Arctowski	Poland	1977	Year-round	12	40
Artigas	Uruguay	1984	Year-round	9	60
Arturo Prat	Chile	1947	Seasonal	n/a	27
Belgrano II (1)	Argentina	1955	Year-round	12	12
Bellingshausen	Russia	1968	Year-round	25	38
Casey	Australia	1969	Year-round	20	70
Comandante Ferraz	Brazil	1984	Year-round	12	40
Concordia (2)	France and Italy	1997	Year-round	13	45
Davis	Australia	1957	Year-round	22	70
Dome Fuji	Japan	1995	Seasonal	n/a	15
Druzhnaya 4	Russia	1987	Seasonal	n/a	50
Dumont d'Urville	France	1956	Year-round	26	100
Escudero	Chile	1994	Year-round	2	33
Esperanza	Argentina	1952	Year-round	55	90
Frei	Chile	1969	Year-round	70	120
Gabriel de Castilla	Spain	1990	Seasonal	n/a	14
Great Wall	China	1985	Year-round	14	40
Halley	United Kingdom	1956	Year-round	15	65
Juan Carlos Primero	Spain	1989	Seasonal	n/a	14
Jubany	Argentina	1982	Year-round	20	100
King Sejong	Korea	1988	Year-round	15	60

(continued)

Table 11.1 (continued)

Name of facility	Operated by	Station first opened	Station current status	Winter population	Station peak population
Kohnen	Germany	2001	Seasonal	n/a	28
Law – Racovita	Australia and România	1987	Seasonal	n/a	13
Macchu Picchu	Peru	1989	Seasonal	n/a	28
Maitri	India	1989	Year-round	25	65
Maldonado	Ecuador	1990	Seasonal	n/a	22
Marambio	Argentina	1969	Year-round	55	150
Mario Zucchelli	Italy	1986	Seasonal	n/a	90
Mawson	Australia	1954	Year-round	20	60
McMurdo	USA	1955	Year-round	250	1,000
Mirny	Russia	1956	Year-round	60	169
Neumayer	Germany	1981	Year-round	9	50
Novolazarevskaya	Russia	1961	Year-round	30	70
O'Higgins	Chile	1948	Year-round	16	44
Ohridiski	Bulgaria	1988	Seasonal	n/a	15
Orcadas	Argentina	1904	Year-round	14	45
Palmer	United States	1965	Year-round	12	43
Progress 2	Russia	1989	Year-round	20	77
Rothera	United Kingdom	1976	Year-round	22	130
San Martín	Argentina	1951	Year-round	20	20
SANAE IV (3)	South Africa	1962	Year-round	10	80
Scott Base	New Zealand	1957	Year-round	10	85
Signy	United Kingdom	1947	Seasonal	n/a	10
Syowa	Japan	1957	Year-round	40	110
Tor	Norway	1985	Seasonal	n/a	4
Troll (4)	Norway	1990	Year-round	7	40
Vernadsky	Ukraine	1996	Year-round	12	24
Vostok	Russia	1957	Year-round	13	25
Wasa	Sweden	1989	Seasonal	n/a	20
Zhongshan	China	1989	Year-round	15	30

scientific stations, especially in the Antarctic Peninsula region (Fig. 11.2). For example, the stations of Esperanza (Argentina) and T/N Ruperto Elichiribehety (Uruguay) are only 500 m apart, with General Bernardo O'Higgins (Chile) 45 km away.

The sub-Antarctic islands are often stopping off points for expeditions travelling further south to the continent (tourism and marine science cruises and re-supply voyages) and this chapter will include a brief consideration of the contribution that human activity on these islands makes to the total human footprint in the Antarctic.

11.3.1 National Antarctic Programs in the Sub-Antarctic

The sub-Antarctic can be defined in a number of different ways, but here it is considered to be the area between the Sub-Tropical Convergence and the Polar Frontal Zone. The major islands located within this narrow zone are Crozet, Heard, Kerguelen,

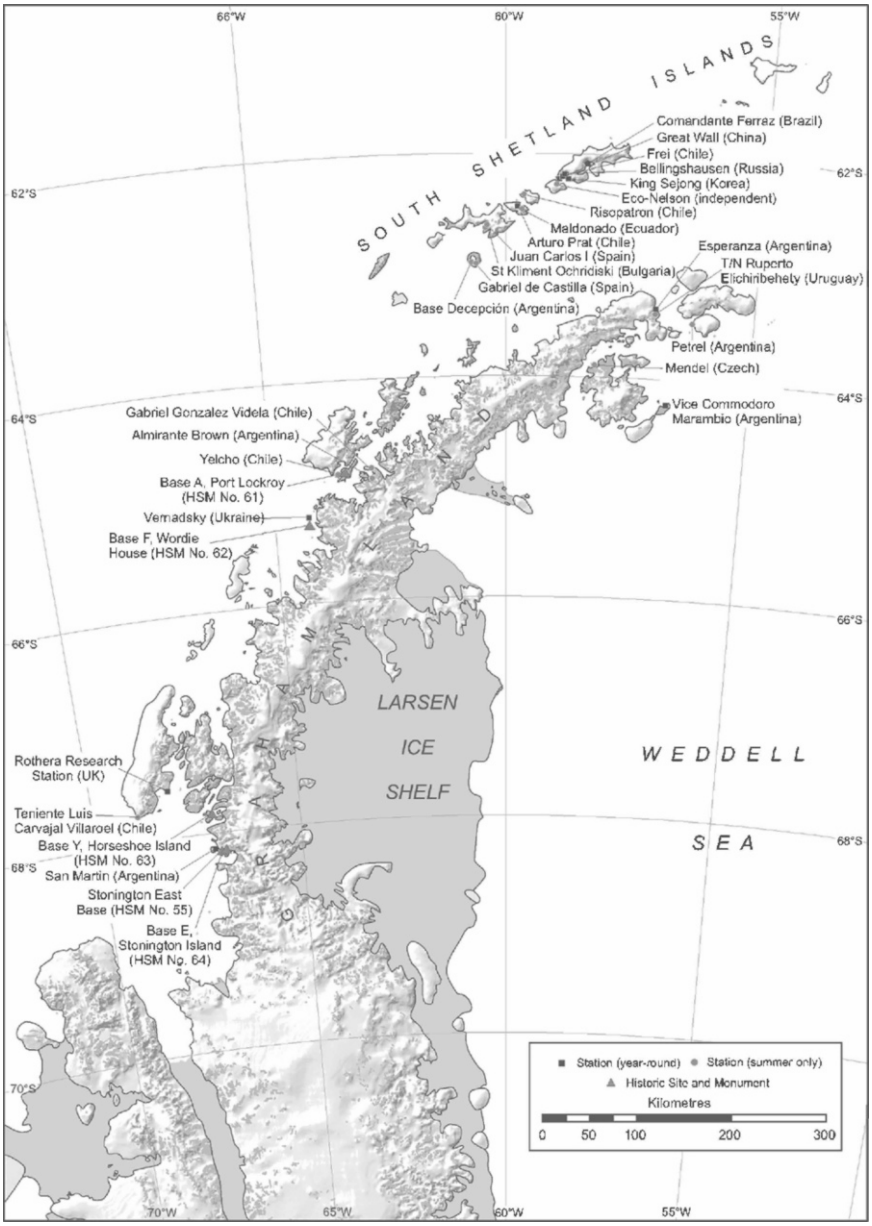

Fig. 11.2 Antarctic Peninsula region

Macquarie, Marion and South Georgia and all have research stations which were established from the post-war period onwards. Most are permanently staffed with the exception of Heard Island which is only intermittently occupied (Frenot 2006).

The scientific value of the sub-Antarctic islands is principally related to their geographic isolation, which kept them relatively free from human impact until the establishment of permanent or semi-permanent stations (Frenot 2006). The climate, which is cold, wet and windy, both constrains biodiversity and increases endemism in these isolated places, making them significant in ecological terms. Their location, in the highly productive Southern Ocean, makes them important breeding sites for many marine predators such as seals, penguins and other seabirds. Some islands are also particularly important geologically and Macquarie Island, for example, was granted World Heritage status partly because it is composed entirely of oceanic crust and rocks from the earth's mantle and is the only island in the world where the earth's mantle is at the surface. This status effectively means that Macquarie Island must be managed in accordance with the principles of the World Heritage Convention, with particular attention to ensuring that its values are identified, conserved, protected, presented, transmitted to future generations and, if appropriate, rehabilitated. Its values are maintained through various environmental management processes, including limiting access to a prescribed number of visitors each year, environmental impact assessment and monitoring of permitted human use (EPBC Regulations 2000).

Research activities on the sub-Antarctic islands cover a range of scientific disciplines, including both terrestrial and marine ecology, geology and glaciology. Low diversity, high endemism and unique morphological adaptations lure scientists to the sub-Antarctic terrestrial ecosystems, while the exceptionally high productivity of the marine environment attracts scientists to study the range of marine predators that use sub-Antarctic islands to breed. Glacial retreat is also being measured on Heard, Kerguelen and South Georgia Islands. It is thought that the change in these glaciers is linked to increased annual air temperatures of at least 1°C over a 30–50 year time-span at some locations (Frenot 2006) which may in some part be attributed to global climate change. The warming trend and decreasing precipitation in the region is likely to have important biological consequences for those species in the sub-Antarctic at the limit of their distribution and for others introduced from outside.

All of the sub-Antarctic-islands are beyond the reach of the Antarctic Treaty Parties' environmental protocol and are not subject to the same reporting or evaluation requirements applied to the continent. Many of the islands are, however, managed under separate management plans that provide differing levels of protection depending on the status of the island (e.g. World Heritage) and the general standards applied by the country administering the island. These plans may contain various restrictions on human activity, such as prohibiting the introduction of alien fauna and flora, limits on the number of tourists and human-use zoning, as well as quarantine procedures.

Many of the islands have long-standing problems with introduced species and at many there are ongoing programs aimed at the eradication of pests and remediation of the environment. When expeditions visit these islands on their way to the continent, extra care should be taken to minimize the possibility of the translocation of alien species already established in the sub-Antarctic into the Antarctic.

11.3.2 National Antarctic Programs on the Antarctic Continent

In contrast to the sub-Antarctic, the Antarctic, or at least the Antarctic Treaty Area can be very precisely defined as the area south of 60°S latitude. All countries currently operating in Antarctica are Parties to the Antarctic Treaty and are subject to a uniform approach to environmental management – established by the Protocol on Environmental Protection to the Antarctic Treaty, known as the Madrid Protocol.

Today there are about 50 scientific stations in the Antarctic (Table 11.1) being operated by 28 countries, and well over half of the infrastructure is used all the year-round. There are stations scattered throughout the continent, with the largest concentration in the Peninsula. In total size, these workplaces might only cover, at most, one hundred km^2 – probably a lot less. On a continent of over 14 million km^2 it might seem at first glance that the stations would hardly make an impression, however, these workplaces are not evenly distributed over the entire continent. Most are on ice-free land near the open sea to allow solid foundations for construction and access to shipping for re-supply. Ice-free land makes up about 0.5% of the continent of the Antarctic but most of this is inaccessible mountainous terrain, for example in the Transantarctic Mountains. Ice-free land within 5 km of open sea makes up only 0.04% of the continent (Poland et al. 2003) and this is the same land that is used as breeding sites for nesting birds and seals. Thus wildlife, operational facilities, scientists and tourists are all attracted to the same locations, leading to the conclusion that most of the impact is, in fact, occurring in the areas that can sustain it the least. The extent of the human 'footprint' in Antarctica (used here to mean the spatial extent of disturbance) is unknown.

Typical station facilities might consist of year-round and seasonal stations, airstrips and regularly visited field camps. Many of the locations have a ship anchorage and a 'port' facility and/or landing strips for aircraft. Most government expeditions establish a station in a coastal location that is suitable for ship anchorage (and/or aircraft operations) and the construction of buildings, even if their primary research focus is inland or on the plateau. The site of a station may also be convenient for scientific research projects that investigate phenomena close at hand, or is a useful central location for the deployment of field parties during the summer months when the scientific population is at its peak. These parties may erect temporary camps – most likely ones that are completely dismantled and returned to station on completion of the project (such as ski-mounted accommodation modules or 'apple' huts) – or they may make use of existing refuge huts and small camps as work sites during the Antarctic summer period. This means the footprints are often centralised and near wildlife concentrations or special scientific sites that are the object of their research.

Antarctica's largest station, McMurdo (USA) has a winter population of about 200 and has a capacity for 1,100 people in summer, housed and working in 110 different buildings. It has a new waste-water treatment plant, an ice pier, 14 gravel roads and several ice and snow roads (one of which is 27 km long), two helipads and four airstrips; nearly 250 ground vehicles, 18 aeroplanes and over 37 million litres of various types of fuel. All electricity generators are diesel-driven and emissions are neither filtered nor monitored. However, a generic environmental monitoring

program was begun in 2003/04 to 'study variability in natural and anthropogenic disturbance in and around McMurdo Station's terrestrial and marine environment' (Inspection Report 1 2005, p. 17).

All other Antarctic stations are much smaller than McMurdo. For example, the three Australian stations, Mawson, Davis and Casey, each have a winter population of 18–20 and can support 45–70 people over summer. Irrespective of the size of a station, there are several essential services that must be provided. All stations have living accommodation, including bedrooms, bathrooms, kitchen and recreational facilities. They also have power generators, communications facilities, including aerial farms and satellite receivers, and workshops, and most have laboratories. All need a source of drinking water and systems for waste-water treatment and disposal, and systems for managing other wastes, including incinerators and holding facilities for waste that is to be returned to the country of origin for disposal. Storage facilities must be available with capacity to hold enough food, fuel, equipment and spares to maintain the operation for the longest planned period between re-supply visits with enough in reserve for unplanned events. Stations also have various types of transport infrastructure including roads, jetties, landing pads for helicopters and runways for fixed-wing aircraft, depending on their location and role.

Activities of the National Antarctic Programs are diverse, and while they are primarily concentrated in the Peninsula region, they do, in fact, occur throughout the continent. The infrastructure is sometimes extensive, sometimes small, with the numbers of people involved related to station size and operational requirements. One way to evaluate on-station activity is to examine the official reports of observer teams. Two such reports are used here as case studies to illustrate the range of environmental concerns identified.

Representatives of the Australian government spent from the 21st to the 25th of January 2005 in the Ross Sea region observing facilities and activities at Scott Base (New Zealand) and McMurdo Station (USA). Their report (Inspection Report 1 2005) contains factual details about the stations, the personnel, station operation, scientific and other observations of an environmental nature. There were some minor comments about dusty roads and permafrost melt undermining building foundations on the 0.2 km^2 Scott Base, and the presence of polystyrene (a prohibited substance) at the McMurdo Station. But essentially the observer team reported its satisfaction with the very high level of compliance with the Madrid Protocol, scientific research and cooperation and collaboration. In particular it singled out 'state of the art' performance with regard to waste handling, site remediation and energy efficiency on both stations.

The report of a second inspection team, this one to the Antarctic Peninsula (Inspection Report 2 2005) was more critical, in part because it inspected a range of stations very close together, as well as unoccupied stations. This team, comprising representatives from the United Kingdom, Australia and Peru, visited sites and facilities during the period from the 10th of February to the 5th of March 2005. Their inspection route also gives a good indication of the concentration of research facilities within the relatively narrow confines of the Peninsula – and remembering that these are also locations of significant tourism interest. While the team reported about

individual stations, it also made recommendations of a general nature about issues common to many, or a number of, stations. One comment was that 'Although some stations were undertaking world-class scientific research into a wide variety of disciplines (though many geared to climate change), a larger number of stations appeared to have relatively modest, or even rudimentary, scientific facilities' (Inspection Report 2 2005, p. 9). They called on the Scientific Committee on Antarctic Research (SCAR) to undertake an audit of the Antarctic scientific research. Furthermore, very little cooperation or scientific interchange was evident. This was significant criticism, considering scientific cooperation and collaboration are fundamental tenets of the Antarctic Treaty. The point was made that sharing resources and conducting joint operations would reduce the human footprint (Inspection Report 2 2005, p. 9). About one quarter of the stations visited or overflown were abandoned or unoccupied and the inspection team asked Parties to consider what the future of these locations might be - a historic site, a lease to another Party or to be removed altogether.

Fuel handling and storage was another prominent issue raised by the Peninsula inspection team. A number of stations did not have a formal oil spill contingency plan, despite COMNAP advice, guidelines and checklists and despite warnings that oil spills pose the major hazard to the Antarctic terrestrial and marine ecosystems.

Significantly, it appeared to the inspection team that protection of the flora and fauna was accorded high priority at most stations, and there was good knowledge of the regulations, however, there were few measures to prevent the accidental introduction of non-native species in evidence (Inspection Report 2 2005, p. 11). Furthermore monitoring of the impact of station activities was inconsistent and ad hoc (Inspection Report 2 2005, p. 12).

The report noted that there were generally inconsistent approaches to even the presence and information requirements of the team and that this is a matter that needed attention by the Parties and by COMNAP in the Antarctic Treaty Consultative Meetings (Inspection Report 2 2005, p. 13).

Part of the brief of the inspection teams is to observe and comment on tourism and non-government activities and in this instance it was noted that due to the wide disparity between the Parties' attitudes to tourism, they should each set out and make publicly available their policies on visits to their stations (Inspection Report 2 2005, p. 12).

11.4 Antarctic Tourism

Antarctica now has over 73,000 non-governmental visitors a year (tourists, staff and crew; IAATO 2009), in addition to about 4,000 government personnel. The peak time for the visits of both scientists (and their support staff) and tourists is during the summer from about December to March. This coincides with the breeding season for the continent's vertebrates. The main attraction for tourists is predominantly wildlife viewing, therefore the main sites of interest are those with the greatest accessible concentrations of seals, penguins, and other iconic bird species such as

albatrosses. These sites are also where the majority of scientific stations are located, intensifying the pressure on the environment for a short time each year.

IAATO collects data on tourism activities each season. This information is publicly-accessible on their website at www.iaato.org.

A typical site visit will last for approximately 2 hours, with IAATO by-laws requiring that operators only have 100 tourists ashore at one site at one time. This number does not include expedition staff and crew. Using these calculations, one site, Almirante Brown (an unused Argentine research station) is subject to over 100,000 people-hours ashore in one season. How significantly this might impact on the environment is not known. However, Almirante Brown is not one of the sites for which site-specific guidelines have been developed, indicating that it is not considered at risk of significant impact.

Antarctic tourism activity comes under heavy scrutiny from the Antarctic Treaty Parties, not all of whom are in favour of tourism per se. As a result, a number of sites now have site-specific guidelines, which were trialled recently. Included in the guidelines or discussed for proposed guidelines were criteria that help determine management options for the sites, such as preferred landing areas; zoning (e.g. closed areas, guided walking areas, and free roaming areas); daily and seasonal limits; control of size of ships, number of ship visits and time and length of stay; voluntary closure during peak incubation periods; visitor codes of conduct, including acceptable approach distances to wildlife; rest periods for wildlife; and the uniqueness of each site from which its sensitivity could be calibrated (UK 2006). It was acknowledged, however, that more information is required, such as when the most sensitive stages for Antarctic wildlife are and that SCAR biology experts should be consulted (UK 2006, p. 14).

11.5 The Developing Antarctic Environmental Management Regime

In the early years (the 1950s to the late 1980s), scant attention was paid to the consequences of the presence and operation of stations, and to the behaviour of humans visiting and working in Antarctica. For example, a common practice at most coastal stations, was to dump refuse onto the sea-ice in winter so that when the sea-ice broke up during the summer melt it would sink to the bottom of the ocean or be carried out on the ice only to sink somewhere along the coast. As the Antarctic Treaty legal regime developed, in tandem with the progression of international environmental law, a series of environmental protection measures were generally agreed upon. First came the protection for fauna and flora through specially protected species and area regulations (Agreed Measures). These conditions prescribed, among others, the processes for obtaining permits for the collection of specimens of living organisms or the entry into areas that were set aside because of intrinsic scientific value. At this time the human footprint was relatively soft in terms of numbers but could still leave an impression because some environmentally damaging

practices were accepted as the norm. For example, wildlife could be taken for human consumption and to feed dogs or as live specimens to be kept in zoos. Even then, however, the introduction of alien species was prohibited, except in accordance with a permit.

Then came rules for the management of commercial harvesting of living resources: seals first in 1972 (the Convention for the Conservation of Antarctic Seals) and then, marine living resources such as krill and fish in 1980 (the Convention for the Conservation of Antarctic Marine Living Resources). Messages from these conventions were atypical of resources harvesting protocols of the time. For example, the principal objective of CCAMLR is the conservation of Antarctic marine living resources, including rational use and the maintenance of ecological relationships between harvested and dependent species, with underpinning scientific management decisions.

These pieces of international law comprising the Antarctic Treaty System, which are described in greater detail by Rothwell (this volume) continued to give legitimacy to scientific research in the Antarctic. However, at this time (1980s) little or no attention was paid to quarantine measures for movements into Antarctica or to the risks of introductions through vectors such as vessels carrying ballast water from other oceans, ship hulls fouled by parasitic organisms, or crew departing from ports around the world potentially bringing with them seeds or insects in clothes and equipment (Convey et al 2006).

Finally, when the Protocol on Environmental Protection to the Antarctic Treaty was adopted in 1991, wide-ranging environmental regulations were put in place to ensure that all human activity was carried out in accordance with strict and comprehensive environmental principles. The message was then clear: being careless about environmental matters would no longer be tolerated. But how well does the rhetoric match practical application of its principles?

The Madrid Protocol requires the Parties, and persons over whom they have jurisdiction (such as tourism operators) to conduct environmental evaluations of all planned activities (including scientific research, logistics and tourism) before their commencement. At the intermediate level of assessment – the initial environmental evaluation – proponents are asked to supply information on the nature of the activity and to predict the likely impacts of the activity on the Antarctic environment and on dependent and associated ecosystems (Article 8 and Annex I). A more detailed assessment – the comprehensive environmental evaluation – is triggered when impact is predicted to be more than minor or transitory. Initial Environmental Evaluations (IEEs) are generally circulated to Antarctic Treaty Parties for interest but this is not a requirement and other Parties play no part in the formal appraisal process. Comprehensive Environmental Evaluations (CEEs) are more significant; draft CEEs are made public (Annex I Article 3), are circulated to the Parties for comment and are discussed in the Committee for Environmental Protection (CEP) at the Antarctic Treaty Consultative Meetings. Ultimately, though, other Parties cannot veto an activity covered under a CEE as this responsibility rests solely with the Party concerned.

The Antarctic Treaty Secretariat's database of IEEs and CEEs is a valuable resource for examining the types of activities undertaken by the Parties and the perceived level of impact. Scientific research projects, logistics, expeditions,

station refurbishment, airstrip construction, tourism, fishing and marine science cruises all come under the scope of environmental evaluation, most of them at the intermediate level. Some components of larger activities are assessed separately at the intermediate level and it is not always possible to view these documents. All CEEs, however, are readily available on the database as complete documents.

The requirement for a CEE is relatively uncommon, for example only two CEEs were posted in 2005 and both relate to the construction of research stations, one by Belgium and one by Germany (Neumayer III, replacing Neumayer II). There were many IEEs however, and they included evaluation of activities as diverse as road building and adventure tourism.

11.6 The Human 'Footprint'

In the academic literature, 'footprint' is often tied to the estimation of a total ecological impact and has been defined as 'a measure of the 'load' imposed by a given population on nature'. It represents 'the land area necessary to sustain current levels of resource consumption and waste discharge by that population' (Wackernagel and Ress 1996, p. 5). It is modelled conceptually on the notion that resources are extracted from the environment by an economy that utilizes those resources, resulting in an end product that is waste and that a specific area of land is required to support extraction, use and final disposal. In this chapter the concept of ecological footprint has been amended to cover the range of environmental pressures from activities undertaken in Antarctica, with particular emphasis on their potential to add to the risk of human mediated introduction or transfer of disease to wildlife.

There are many parameters that could be used to compare trends in environmental pressure on Antarctica over time or from different sources. Perhaps the closest literal analogy to a footprint would be to calculate the total area impacted upon by such activities. On investigation it has not been possible to estimate even the actual area occupied by all the national scientific research facilities in the Antarctic today and we are a long way from being able to report the total area impacted by the facilities and the activities undertaken at these facilities. This is because the information held by the Antarctic Treaty Secretariat and COMNAP is incomplete and fundamental details such as the size of each station are in many cases not recorded. In fact, a survey of the Member States of the Committee for Environmental Protection has confirmed that not only is the area of each research station largely unknown, but different operators interpret the area covered by a station in different ways. For example, it has been reported that Great Wall Station (China) covers an area of 0.7 km^2 compared with McMurdo Station (USA) 0.4 km^2 (Inspection Report 2 2005, p. 54, Inspection Report 1 2005, p. 4 respectively). Yet, it is known that McMurdo is the larger of the two, with more than 110 buildings housing 1,100 people, compared with Great Wall's 12 buildings for ~50 people. This gross disparity shows the difficulties faced in trying to measure even something as fundamental as the area of a station. There is no clear information on whether the areas reported

include only the floor area of the buildings, or whether they also include the areas bounded by all other structures, such as service routes and communications facilities including aerial farms. In addition, the total area impacted is not limited to the area physically covered by the facilities but also extends to the receiving environment for emission plumes or sonic or vibratory dispersal. Without accurate fine-scale mapping of each scientific establishment, including the station limits and outwards to the receiving environment, it is impossible to present an accurate picture of the total area occupied by National Antarctic Program facilities and this is unlikely to happen unless the Parties set standard parameters and put significant pressure on each other to carry out the work.

Area occupied by infrastructure and the associated impacts would, however, only present part of the picture. For example, to date, Antarctic tourism operates largely without the establishment of significant on-ground facilities, but does this mean that the impacts of Antarctic tourism are that much lesser than those of the national Antarctic programs? An alternative method for gauging the relative pressures might be to consider the total number of people visiting the continent. In 2007/08 over 73,000 people visited as part of a tourism operation, either as tourists, staff or ships' crew (IAATO 2009), and about 4,000 people participated in national Antarctic operations. Again these simple statistics may be misleading and probably do not represent the relative environmental pressure exerted by the two sectors. By far the majority of Antarctic tourism is ship-based using no permanent infrastructure within the region beyond the occasional jetty for landings, few tourists spend nights ashore, even in tented accommodation, and few spend more than 2 or 3 weeks in the Antarctic Treaty area. In contrast, most people visiting as part of National Antarctic Programs live in permanent onshore infrastructure and spend several months, and some for more than a year, in the Antarctic Treaty area. Perhaps a more realistic comparison would be to calculate the number of person-days in the Antarctic Treaty area or the number of person-days spent on the continent (Table 11.2). These indicative figures illustrate that although many more people visit Antarctica as tourists than as part of a national program, the number of person-days ashore in Antarctica associated with the National Antarctic Programs (~675,795) is very much greater than as tourists (~32,263).

Indicators of environmental pressure that concentrate only on the scale of activity, such as numbers of people involved or area occupied by stations, overlook the fact that it is not just the scale of presence that influences the risk of environmental impacts, but also the types of activities and where they occur. A large station supporting many people at a location hundreds of kilometers inland is less likely to create risks to Antarctic wildlife than one in a coastal location adjacent to wildlife breeding colonies. Activities that specifically target wildlife aggregations, further add to the risk of disease spread through human mediation. Thus a research program that involves repeated visits to different wildlife aggregations carries an increased risk of transferring disease-causing agents between sites and populations. For the same reason, tourism ventures, which typically visit a succession of wildlife aggregations, are inherently more likely to be the medium for the spread of disease-causing agents than activities that seldom go near wildlife. Similarly, an operation

Table 11.2 Indicative comparison of visitor pressure from tourist and national operators (2007/08). Time within the Antarctic Treaty area and duration ashore are broad estimates based on typical activities

	Total number of visitors	Person-days in the Antarctic Treaty Area	Person days ashore
Ship-based tourism operations	73,710 Includes tourists, staff and crew (IAATO 2009)	737,100 Assumption: each person spends an average of 10 days in the Antarctic Treaty area	17,263 Assumption: 197,286 tourist landings (IAATO 2009) plus 9,864 staff estimated at ratio of pax:staff 20:1 with each landing of 2 hours' duration
Air/land-based tourism operations	1,500 1,050 passengers as reported by IAATO (2009) plus 500 staff, guides and aircrew based on an assumed average 2:1 ratio	15,000 Assumption: each person spends 10 days in the Antarctic Treaty area	15,000 Assumption each person spends 10 days on land in the Antarctic
National Antarctic Programs year-round positions only	1,088 Includes scientists, station and support staff (www.comnap.aq/facilities) (COMNAP 2009)	393,105 Assumption: each wintering position represents one person in the Antarctic Treaty area for the complete year	393,105 Assumption: each wintering position represents one person on land in the Antarctic for the complete year
National Antarctic Programs summer-only positions	3,141 Based on reported peak station capacity of 4,229 minus 1,088 year-round positions (www.comnap.aq/facilities) (COMNAP 2009)	314,100 Assumption: each position spends 100 days in the Antarctic Treaty area	282,690 Assumption: each position is occupied at the station for an average of 90 days per year

that involves many transits (voyages or flights) between low latitudes and the Antarctic, or that which carries a great number of different people, as does the tourism industry, creates more opportunities for the introduction of non-native species, including disease-causing agents, than one involving few transits. Thus, although the number of person-days on the Antarctic continent associated with tourism operations may not be as great as that associated with the National Programs and tourism does not have much in the way of established infrastructure, the risks of tourism involvement in an event of wildlife disease may be as great, if not greater, than that of the national operators.

11.7 Discussion

Antarctic Treaty Consultative Meeting reports are littered with recommendations regarding exchange of information about National Program activities, assessment of the environmental impacts of these activities, a code of conduct and facilitating cooperation in the setting up of stations (e.g. Meeting I Recommendation 1, indicated as I-1, VI-4, VII-1, VIII-13, IX-5, XII-3, XIV-2, XV-17 – ATS 2009). Notwithstanding early acknowledgment of the potential impacts of National Programs, today the Parties still have a tendency to act as individuals, with very few examples of collaboration in either science or logistics. Furthermore, there has been no in-situ audit of scientific research to investigate priorities, duplication of data collection, or identification of research areas needing to be strengthened (Inspection Report 2 2005, p. 9). It appears that in some cases the national operators site their stations according to political motives and the search for appropriate scientific research programs comes later. In this regard, at the 2006 Antarctic Treaty Consultative Meeting, the Parties once again heard of concern about the potential environmental consequences of an excessive concentration of stations. The Meeting recalled Recommendation XV-17 establishing the measures which Parties were urged to take when considering the establishment of new stations or facilities, and it reiterated that the construction of a station or base in Antarctica was not a pre-condition for attaining Consultative Party status (ATCM 2006). At Maxwell Bay on King George Island, King Sejong Station (Republic of Korea) has Teniente Jubany (Argentina) 5 km to the southeast and Artigas (Uruguay) 7 km to the northwest, with Bellingshausen (Russian Federation) and Frei (Chile) virtually merged together, and Great Wall (China). A system of roadways and vehicle tracks join many of these locations, extending the area of impact of the individual stations. The major thrust of the recommendations in Inspection Report 2 concerns collaboration and cooperation in the conduct of scientific research and the operation of Antarctic stations.

Notwithstanding the fact that the Parties decided a number of years ago to discourage the establishment of new infrastructure to support scientific research, many still believe that as science is the ‘currency of credibility’ (Davis 1990), having a scientific station also adds legitimacy to a Party’s standing within the Antarctic Treaty Consultative Meetings. Therefore despite the entreaty to new Parties to collaborate with countries that have existing infrastructure, new stations continue to spring up in the Antarctic (e.g. those of India and the Czech Republic). Some of these new buildings use highly sophisticated materials and embrace state-of-the-art technology for heating, waste-water management, design and portability, thereby reducing their footprint (e.g. Neumayer Station III).

Tourism also attracts its share of criticism. There is a heavy concentration of visits to the Peninsula at peak times and the risk of accidental introduction of non-native species is heightened. In terms of impact, it could be argued that built infrastructure has a permanent or ongoing footprint, whereas tourism has little or

no infrastructure in situ and therefore its impact is less than minor or transitory. However, there are weaknesses to both arguments. A medium-sized station such as Ferraz (Brazil) uses about 300,000 l of fuel per year (Inspection Report 2 2005, p. 36) and it is reported that one average over-flight from Australia to East Antarctica and return will use half that amount. However, there may be as many as nine over-flights of this kind each year (Maggs, Personal communication).

11.8 Conclusion

This chapter began by describing both the historical and current human impact on the sub-Antarctic and continental environment with a view to estimate the total human footprint and how this might pose a risk to the health of Antarctic wildlife. It soon became apparent that there was a problem: there is insufficient information on the human footprint in Antarctica to make any meaningful judgment about that footprint and its risk. All humans *have* an impact, of this there is no doubt, and being a scientist with a 'legitimate' reason for being there does not legitimate the impact. But, neither does being a tourist visiting by the good grace of some Antarctic Treaty Parties make the impact worse. If anything, national operators could take a leaf from the book they have written for the tourism industry and adopt for their stations site-specific guidelines with environmental codes of conduct, long-term monitoring programs, agreed terminology, fine-scale maps and the input of organizations like SCAR to help with calibrations on the sensitivity of the receiving environment.

The first step in developing site-specific guidelines is to collect standardized information on each station precinct, each roadway, runway and port facility, and each vessel and aircraft. The Antarctic Treaty Secretariat and COMNAP should take joint responsibility for designing a database that will serve as the receptacle for this information. The requirement to submit standardized information to the database could be achieved through an ATCM Measure, which brings with it a legal obligation. This is not something the Parties should find onerous; there is already a legal requirement for them to submit information on the nature of expeditions under their flag or departing their shores. Furthermore, the environmental evaluation process within the Madrid Protocol could serve as the substantive basis of the Measure by securing from each Party with continental infrastructure a comprehensive environmental audit of that place/those places using a template jointly developed by the CEP, the Secretariat and COMNAP and adopted by the Parties through an ATCM. This is not very different from the inspection process now, except that it would be a legal obligation, using a standard template, and with agreed parameters such as terminology and units of measurement. Such a template is currently under development; a recent desktop study identified likely parameters, which were then tested on Australia's Davis Station and verified with in situ observations at both Davis and Casey Stations (Brooks 2009). With the assistance of the CEP, and through its institutional processes, the database – once complete – would be the

basis upon which site-specific guidelines are developed. Many stations now have some form of site guidelines so the process of designing a standard template would also not be onerous. Assuming that it would take a number of years to collect the data, the inadequacies of the tourism site guidelines would have been fine-tuned and the process streamlined. In the future, as new projects are developed the database could be updated accordingly. Without further urgent action by the Parties, the whole notion that they are committed to the environmental wellbeing of the Antarctic and the health of its wildlife will be seriously undermined.

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Chapter 12

Antarctic Tourism: An Operator's Perspective

G. Mortimer and E. Prior

12.1 Introduction

The aim of this chapter is to describe the history, management, scope and style of Antarctic ship-borne tourism, and to outline the daily operating procedures of a typical voyage to Antarctica. It describes some of the operating procedures designed to ensure that the impact of tourism on the Antarctic environment and its wildlife is minimised.

12.2 Tourism History and Statistics

Human involvement in Antarctica and the Southern Ocean has evolved from the heroic era and exploitative sealing and whaling in the nineteenth and early twentieth centuries, to the precedence of science in the mid- to late twentieth century. Today we recognise the importance of this region to the health of the global environment and its intrinsic value as a huge wilderness area. Under the Antarctic Treaty System, priority is given to understanding its ecosystems and to careful environmental stewardship.

As awareness of the research and environmental values of Antarctica and the Southern Ocean has grown through the late twentieth and into the twenty-first century, an ecotourism industry has also developed (reviewed by Bauer 2001; Hall and Johnston 1995; Snyder and Stonehouse 2007).

The modern Antarctic tourism industry was born with the launching of a specialist polar tourist ship, the *Lindblad Explorer*, by Lars-Eric Lindblad of Sweden in 1969. By 1991 the industry was well established, and with encouragement in particular from the U.S. Government (National Science Foundation – NSF), an association of international Antarctic tour operators was established to advocate, promote and practice safe and environmentally responsible private-sector travel to the Antarctic.

G. Mortimer and E. Prior
Aurora Expeditions, 182 Cumberland Street, The Rocks, Sydney NSW 2000, Australia,
e-mail: greg@auroraexpeditions.com.au

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Founded by seven private tour operators, the International Association of Antarctica Tour Operators (IAATO) had more than 100 members by 2009 from Argentina, Australia, Belgium, Canada, Chile, Germany, Netherlands, New Zealand, Norway, UK, USA, the Falkland Islands (Islas Malvinas) and elsewhere. IAATO introduced guidelines designed to minimise the impact of tourism on the Antarctic environment, and these guidelines continue to be further developed.

Visiting Antarctica and the Southern Ocean is an opportunity to experience and value a unique and wonderful environment that relatively few people have enjoyed. Until the early 1990s, some 60,000 people had visited Antarctica as tourists. During the 1990s, Antarctic tourism grew steadily (Fig. 12.1). The rate of growth increased progressively to 2007/08, however in 2008/9 there was reduction of 14% from the previous year. This may indicate that the market is reaching saturation or it may be a temporary lull, perhaps reflecting the world-wide economic slow-down during 2008. The majority of tourists who land in Antarctica come from the USA, followed by Germany and the UK (Table 12.1). Because these voyages are reasonably expensive, these tourists are generally well educated, financially comfortable people from around the globe, with a particular concentration from a few affluent nations.

The Antarctic tourism season runs from November to March, and is mainly ship-based. Passengers are taken ashore using small inflatable boats, generally for periods of 1–3 h. Commercial sailing vessels and private yachts also visit Antarctica, and in recent years a small number of tourists have been flown to Antarctica and are involved in land-based expeditions. Tourist activities include small boat cruising, most frequently in inflatable rubber boats and shore landings, and to a lesser extent

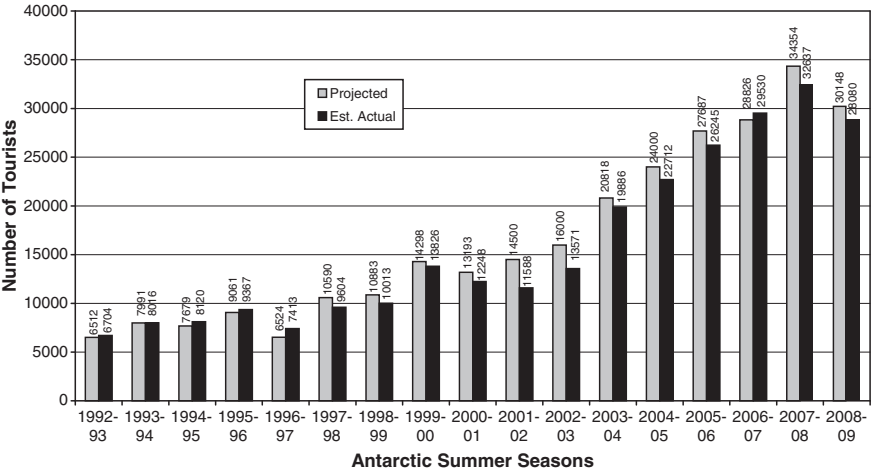

Fig. 12.1 1992–2008 Antarctic tourist trends – landed personnel. Includes ship and land-based passenger numbers. 1997–98 onwards includes some commercial yacht activity (IAATO 2009)

Table 12.1 Partial comparison of nationalities for seaborne passengers who landed in Antarctica in 2007/08 (available on IAATO website)

2007/08	Tourists	%
USA	10,020	30.3
UK	5,542	16.8
Germany	4,851	14.7
Australia	3,053	9.2
Canada	1,169	3.5
Switzerland	1,062	3.2
Netherlands	990	3
France	860	2.6
Others	5,507	16.7
Total	33,054	100.00%

kayaking, mountain climbing, scuba diving, skiing, snowboarding, camping, marathon running and helicopter operations. Large ships with passenger capacities of over 1,000 visit the region for sightseeing. However, under IAATO membership rules, vessels carrying over 500 passengers do not offer landings. Over-flights from Australia and Chile offer 'flight-seeing'.

Figures reported to IAATO (IAATO 2008) indicate that in the 2007/08 Antarctic season 32,198 passengers were landed from vessels in the Antarctic Treaty Area from 47 commercially organised tour vessels and small sailing vessels. IAATO members operated all but two (*Discovery* and *Marco Polo*) of these tour vessels.

Ship-based tourists and accompanying staff made 220,210 person-landings at a total of 145 sites on the Antarctic Peninsula (www.iaato.org/tourism_stats.html accessed 25/03/09). The most frequently visited site was Goudier Island which had more than 18,000 person-landings. The eight most visited sites each received more than 10,000 person-landings. A further 2,208 person-landings were made to 16 sites in the Ross Sea and Continental area of Antarctica, with the three most visited sites there receiving more than 200 landings each. This was a reduction by more than half from the 5,755 person-landings in the Ross Sea and Continental area in 2005/06. The Peninsula is the easiest part of the Antarctic coastline to reach. It is the closest part of Antarctica to any other continent, which makes the voyage shorter in duration and less expensive. Another reason for the concentration of tourist visits to the Peninsula is its abundant wildlife and magnificent scenery.

Six large cruise vessels operated by IAATO members (*Prinsendam, Star Princess, Artemis, Azamara Journey, Topaz* and *Rotterdam*) each carrying more than 500 passengers conducted cruise only operations, carrying a total of 13,015 passengers to the Antarctic Peninsula area. Two large cruise vessels capable of carrying more than 500 passengers operated by non-IAATO members landed passengers, one of these vessels, on one voyage only, landed more than 500 passengers (IAATO 2008).

In 2007/08 261 people participated in air-supported, land-based expeditions with IAATO Member companies (IAATO 2008), significantly less than the 1,074 who participated in similar operations in 2005/06 (IAATO 2007). In addition, IAATO

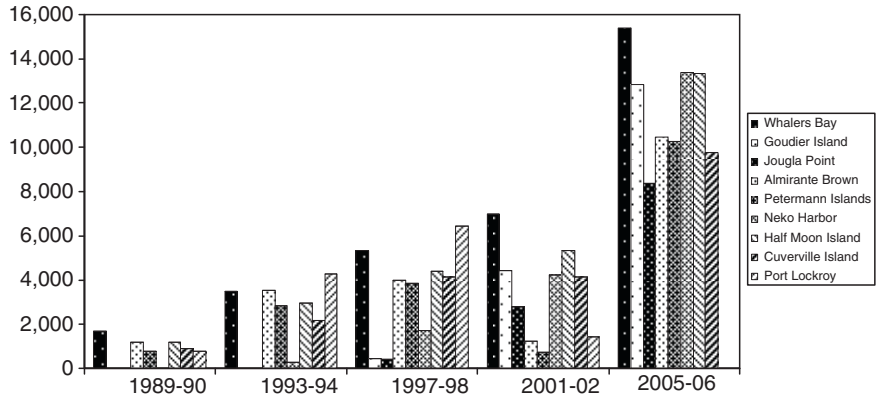

Fig. 12.2 Sites most frequently visited by tourists in the Antarctic

reported that multiple non-IAATO operators were providing land-based expeditions to the Antarctic Peninsula region and to the continent, departing from South America and South Africa respectively, and that the numbers of passengers carried were unknown to IAATO (IAATO 2008). There is one dedicated land-based tourist facility operated by IAATO members – a tent-based summer camp at Patriot Hills in the interior (Ellsworth Mountains), which is dismantled at the end of each summer season.

The U.S. NSF and IAATO have compiled statistics, particularly detailed from the 1990s onwards, showing that ship-based tourists have visited over 150 different sites in the Antarctic Peninsula. However, the majority of visits are concentrated on a few sites (identified in Fig. 12.2), all of which are on the Antarctic Peninsula (Naveen 2005). The western side of the Peninsula between the Antarctic Circle and the northern tip of the Peninsula and the South Shetland Islands is the area of Antarctica that is most visited by tourists. The eastern side of the Peninsula to the northern end of the Weddell Sea is visited to a lesser extent. The least visited area of the Peninsula is the south side, south of the Antarctic Circle. The Continental sites visited are very largely in the Ross Sea area. Visits to East Antarctica have so far been minimal.

Private-sector tourist activities still account for a relatively small part of all human activity in Antarctica. There are around 80 government stations with some sort of permanent structures (Jabour, this volume). Infrastructure associated with this full-time presence includes buildings, fuel storage facilities, roads, research drilling rigs, over-snow and other vehicles, aircraft landing facilities and field camps. National Antarctic programs rely on shipping for most of their re-supply and expedition support needs, as well as for providing support platforms for a range of marine science activities.

There are now many more people visiting Antarctica as tourists than with government-supported research programs. However, tourist days on land in Antarctica are estimated to be less than 6% of the people-days associated with national programme activities (Jabour, this volume). Tourist programmes do not have permanent infrastructures on the Antarctic continent.

12.3 Environmental Guidelines, Procedures and Regulations

Ship-based tourism in Antarctica is focused on the Antarctic Peninsula, and in particular on a number of sites with wildlife, historical and scenic values. There is potential for frequent visits to these sites to create a cumulative impact on the environment. However, after 35 years there are very few discernible and significant environmental impacts from tourist activities. This can be attributed to a large extent to the policies and procedures established by IAATO and the tourist industry to minimise and mitigate environmental impacts. These policies and procedures are outlined below.

Human activity in Antarctica is managed under the umbrella of the Antarctic Treaty System. The Environmental Protocol to the Antarctic Treaty (known as the Madrid Protocol) requires environmental impact assessment and environmental monitoring of all human activities, including both private-sector and government operations. All IAATO members and some other tour operators and private expeditions provide environmental impact assessments to their government authorities. The Protocol, however, does not provide an easy mechanism to monitor, assess and manage potential cumulative impacts of all human activities (science and tourism) across a number of countries and/or operators.

Recognising the potential cumulative environmental impacts of tourism, the Antarctic tourist companies, through IAATO, have established a range of common procedures and guidelines. Visitor guidelines developed originally by tour operators in the 1980s were subsequently modified and strengthened by IAATO, which then provided the basis for a comprehensive Antarctic Treaty Recommendation (XVIII-1) on tourism and visitor activities adopted by the Antarctic Treaty countries in 1994. IAATO, in conjunction with the U.S. National Science Foundation, maintains records of all IAATO member activities and reports annually to the Antarctic Treaty Consultative Meetings.

IAATO has developed a number of other guidelines and procedures to assist its members in self-regulation. These include guidelines on numbers ashore, wildlife watching, small boat and helicopter operations, activity reporting, passenger, crew and staff briefings; contingency and emergency medical evacuation plans; and communication procedures to coordinate site visits. IAATO provides guidance for its members on environmental impact assessment and supports a site inventory programme to monitor impacts at commonly visited sites. Site-specific guidelines were introduced for the 2003/04 season and are being further developed.

In addition to the Antarctic Treaty System and IAATO guidelines and procedures, tour operators' activities are regulated by international air and maritime regulations. In particular, SOLAS (the International Convention for the Safety of Life at Sea) and MARPOL (the International Convention for the Prevention of Pollution from Ships), associated with Port State inspections, impose stringent safety and environmental requirements. Insurance needs also encourage high standards. Tour operators must also comply with appropriate national legislation, in addition to the legislation implementing the Antarctic Treaty.

Within the frameworks described above, a number of precautions specifically mitigate against the potential introduction or spread of wildlife diseases.

- Food is rarely taken ashore by tourists, and chicken products are never taken ashore or on to the ice. Food is not fed to the wildlife. Poultry products are separated from other food wastes on board ship and are either frozen for disposal in port or are incinerated north of 60°S.
- Ships' ballast water is not discharged, nor are tanks washed, south of 60°S.
- Passengers are advised to clean clothing and footwear before joining their cruise. Boots are scrubbed before the first excursion from the ship, and after each shore excursion. Other personal gear is washed if it has come into contact with the ground.

12.4 Shore Visits: The Tourist Experience

This section outlines a typical tourist experience from the environmental management perspective. It is based on the operations and practices of the Australian-based tour operator, Aurora Expeditions, and is representative of the practices of many Antarctic tour operators.

The process of educating Antarctic tourists starts well before they leave home, and helps to prepare visitors for an experience that is far removed from their normal circumstances.

Generally 6–9 months prior to departure for Antarctica, intending visitors have their first exposure to the more formal side of their voyage. It might be a commercial slide show to convince them to participate in a voyage, and which also introduces the nature of Antarctica and the associated environmental sensitivities. Or it may be a conversation between the operator and potential client, in which more detail about the nature of Antarctica is discussed. Before the voyage, every client receives a package of information including a primer that outlines Antarctic history, the Antarctic Treaty System, Madrid Protocol issues and important environmental considerations. They also receive a copy of Antarctic Treaty Recommendation XVIII-1, a set of guidance rules for onshore visits.

During the voyage to Antarctica, there is a more formal education process, involving a number of briefings and lectures. These cover safety procedures, using small boats or helicopters where applicable, and – more importantly – what they will find when they go ashore and how to approach wildlife. A connection is made between the basis of Environmental Protocol considerations and people's actions on shore. In the case of Australian citizens, their legal responsibilities are spelled out – i.e. that they are required by law to behave in an environmentally responsible manner when they are in the Antarctic Treaty area.

Visitors are informed that eating, smoking, littering, taking souvenirs and going to the toilet are not permitted during most shore operations. There are some exceptions to this rule, and these are discussed in detail later in the chapter.

This program aims to instil an understanding of the effect that a visit to a penguin colony, for example, may have. Visitors are advised that, in Aurora's case, 5 m from the edge of a colony is close enough. However, more importantly, they are advised

how to recognise behavioural responses of any of the wildlife with which they make contact, in order to understand when their presence is causing wildlife to become agitated or change their behaviour.

Inflatable rubber boats, carrying up to 15 people each, are used for excursions from the ship. Excursions involve both shore visits and cruising in ice, around icebergs and in front of glacier snouts.

Prior to the first landing on each voyage, passengers are reminded that Antarctica is an isolated continent, free of diseases as far as we know, and that we must ensure that it remains so. Therefore, all shore visitors are instructed to check their clothing and equipment carefully for soil, seeds and other plant material, and that special care must be taken with clothing or equipment used for hiking or around farm animals. Particular attention is paid to Velcro fastenings and inside pockets. Before proceeding down the gangway, all boots – irrespective of their condition – are scrubbed in a footbath.

IAATO has laid down as a guideline that no more than 100 passengers should be onshore at one place at any given time. The passenger carrying capacity of ships now being used for Antarctic tourism ranges from 46 to upwards of 400, but largely falls into the category of 50–120 passengers. The commonly adopted practice among tour operators is that for every 10–20 passengers there is one experienced staff member accompanying them ashore.

The first landing is important and can set a precedent for behaviour on future landings on that voyage. On arrival at each landing site, each boatload of passengers is briefed about particular sensitivities of the place. They are told what they might see, where they might walk safely without falling into a crevasse or walking to the edge of a cliff, where there may be a concentration of nesting giant petrels to be avoided, and so on. In general, when the uninitiated first make contact with a large penguin colony, they are fearful of doing the wrong thing.

Staff distributed around the area keep an eye on activities, as per the guidelines. They communicate with each other and with the ship by hand-held radios, so there is a network of people overseeing activities and enabling passengers to wander relatively freely ashore.

Most landings are at previously visited sites. There are also opportunistic landings, as some operators visit new locations. In such circumstances, staff members will first go ashore to evaluate the site against specific criteria with respect to particular sensitivities, before passengers are landed.

Procedures onshore vary during the penguin breeding season. For example, early in the season it is particularly important to avoid disturbing penguins tending eggs or young chicks. As the season progresses at some sites, people may be able to walk further afield as chicks become mobile in the 'créche' stage.

On all tourist ships the passengers sleep every night on board and eat all their meals on the ship. Generally food is not taken ashore, apart from during all-day climbing activities run by a few operators, and emergency supplies during helicopter operations in case a change in weather strands a party away from the ship. Poultry products are never taken ashore. Generally, people spend 2–6 h onshore at once, and in the Antarctic Peninsula they will visit two or three different places

each day. On each voyage they will, on average, visit 10–12 different sites, most of which will have some wildlife importance.

12.5 Adventure Activities

In recent years, some operators such as Aurora Expeditions have become more adventurous as they have developed a better understanding of the Antarctic Peninsula. Aurora Expeditions was the first operator to offer climbing, sea kayaking, scuba diving and overnight camping as optional activities on its voyages. These activities are part of the changing face of Antarctic tourism.

Passengers have the option to camp ashore for a single night on several voyages. Each campsite is on snow or smooth, clean granite above the shoreline. Sites are away from concentrations of wildlife and from moss beds or small lakes, to avoid potential contamination of water or impact on flora or fauna.

Camping takes place only when the weather appears settled, and arrangements are in place to evacuate the party back to the ship quickly should conditions change. On each occasion, the campers go ashore after having dinner on the ship, then set up camp in small 2–3 person tents for the night, and return to the ship for breakfast the following morning. A portable toilet is taken onshore and toilet waste returned to the ship. No food is taken onshore. Each camper is briefed before going ashore for the night.

Equipment used in kayaking and scuba diving activities is cleaned before use to avoid introduction or transfer of non-indigenous materials or organisms.

12.6 Ship Operations

The increasing number of Antarctic tourists in the 1990s was catalysed by the collapse of the Soviet Union and resultant availability of ice-strengthened vessels. Increased awareness of Antarctic and environmental issues also resulted from media reports around the development of the Madrid Protocol.

In the 2003/04 season, around 30 commercially operated vessels took tourists to Antarctica. Before each vessel leaves its home port, the operator of that vessel completes an Environmental Impact Assessment (EIA) for their respective authority. Very importantly, this details sewage disposal, bilge water procedures and disposal of food in the Antarctic Treaty area.

The ships' chefs are well briefed on the need to separate food materials. No scraps are thrown overboard in the Treaty area. Most of the Russian ships have high-grade incinerators that are capable of melting glass and burning most things. It is believed that no tourist ships discharge sewage within the Antarctic Treaty area, although this is permitted under the terms of the Antarctic Treaty, subject to regulations regarding minimum distance from land and speed of vessel. Any hard waste that is not incinerated is returned to port for disposal.

Ship operators are conscious of the need to avoid introducing foreign organisms into Antarctic waters. Normal ship practice is not to take northern ballast water into Antarctica because ships go to Antarctica with full fuel tanks and take on Antarctic ballast water for the return northwards passage.

12.7 East Antarctica

East Antarctica is a very different case with heavier ice conditions. There have been several tourist visits to the Ross Sea, Commonwealth Bay and to Australian and other Antarctic bases in East Antarctica. In addition, the Russian icebreaker, *Kapitan Khlebnikov*, has completed two circumnavigations of the continent, visiting many of the stations in East Antarctica and emperor penguin colonies in the Weddell Sea area. Travelling to this area requires a bigger, more sophisticated vessel – icebreaker class generally. While visiting East Antarctica is particularly attractive to potential tourists, it costs about twice as much as going to the Antarctic Peninsula and it is likely in the foreseeable future that the numbers visiting East Antarctica will remain small.

Essentially the procedures for passengers visiting East Antarctica are the same as for the Antarctic Peninsula, except that the use of helicopters requires a new level of operating procedures. Between each landing, the underside of the helicopter's skids are washed down on the ship's decks. Visits to emperor penguin colonies and whale watching are key attractions of these voyages.

12.8 The Future

Concerns have been expressed at the potential for further growth in Antarctic tourism resulting in unacceptable environmental impacts. Having developed from the Lars-Eric Lindblad model, the initial small group of Antarctic tourist operators were people with previous Antarctic experience who had a strong emotional attachment to the place. It appears that the next phase is likely to increasingly involve entrepreneurial money.

The initial challenge of increased numbers is in the logistics of visit management, and good operational procedures are critical for managing this growth. Monitoring of key sites is critical to assess any impacts of increased visitation. Governments need to ensure that operators from their countries are aware of the requirements of the Antarctic Treaty and encourage them to join IAATO or adopt IAATO or similar operating practices.

Both large ship and smaller sailing vessel-based tourism are likely to continue to grow. It appears that ship-based tourism will continue to be the major form of visitation to Antarctica. While air-supported, land-based tourism may increase, expense, logistics and weather variability will always pose a constraint. Any significant new land-based development is likely to require a comprehensive environmental evaluation

for consideration by the Antarctic Treaty Consultative Meeting. This will enable extensive opportunity for review before any infrastructure is established.

To date, private-sector Antarctic tourism has developed as a remarkably low-impact and cooperative model. Thousands of people have been able to experience and appreciate the Antarctic wilderness with much less environmental impact than has been seen from tourist activities in many other parts of the globe. Most people who visit Antarctica become ambassadors for protecting its environment, and as more people see and experience Antarctica in an environmentally responsible way, the chances are improved that it will be well managed for future generations.

A pristine environment is critical to the future of the Antarctic tourism industry. Tour operators, IAATO and Antarctic Treaty governments continue to work together to ensure that future generations have the opportunity to experience an Antarctic environment that is not impaired by current and future human activities in the region.

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Chapter 13
Human-Mediated Impacts on the Health of Antarctic Wildlife

M.J. Riddle

13.1 Introduction

Disease-causing pathogens are only one of a number of potential causes of ill-health in wildlife. Many other factors may have harmful effects on animal health and should be considered when investigating an unusual wildlife mortality or health event. In addition, infection may take hold of a stressed animal that could otherwise have resisted. This chapter will provide an overview of animal health problems that could be caused or exacerbated by human activity, such as physical injury and exposure to environmental pollution.

The Antarctic is undoubtedly among the least impacted parts of the planet; however, even this remote region has suffered from the activities of humanity occurring both locally and elsewhere in the world. For most of the time since its discovery 200 years ago, the natural resources of Antarctica have been exploited for commercial reasons, such as sealing and whaling. Only relatively recently has it been formally recognised as a place worthy of very high standards of environmental protection. The Protocol on Environmental Protection to the Antarctic Treaty, 1991, commonly known as the Madrid Protocol, now provides comprehensive protection to the Antarctic environment. This agreement and its annexes prohibit harmful interference with the native fauna and flora of Antarctica and significantly reduce the chances of severe pollution or other human impacts but do not remove the risks entirely.

There is increasing evidence from non-polar regions that environmental stressors, such as pollution, abnormal temperature ranges, increased UV radiation, food shortage, parasites and others, have additive or synergistic detrimental effects on animal health and welfare when present in combination and particularly in combination with the presence of pathogens. There have been few studies of this phenomenon from the Antarctic; however, what little evidence there is confirms that it also occurs in Antarctic species (Leiss et al. 2001). When investigating an unusual

M.J. Riddle
Australian Antarctic Division, Channel Highway, Kingston Tas, 7050, Australia,
e-mail: martin.riddle@aad.gov.au

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health or mortality event, if the underlying cause is not immediately obvious, it may be advisable to consider the combined effects of multiple stressors.

13.2 Physical Disturbance

Wilful injury to the wildlife of Antarctica is prohibited by the Madrid Protocol. This prohibition is given effect by the implementing legislation required of all Parties as a condition of ratification of the Protocol. Most, if not all, Parties to the Antarctic Treaty also have other domestic legislation prohibiting wilful injury to wildlife. The Madrid Protocol includes provision for issuing permits for taking or interfering with Antarctic fauna and flora for scientific research or educational purposes, with strict limits to ensure that only small numbers are taken so that collections do not impact on the viability of populations.

13.2.1 Disturbance by Visitors, Vehicles and Aircraft

Accidental disturbance or injury may result from the careless, rather than wilful, actions of people visiting Antarctica, either as tourists or as part of a national Antarctic program. Guidelines including minimum separation distances for approaching Antarctic wildlife on foot (Giese 1998) and in vehicles including aircraft overflights (Harris 2005) have been developed for national operators (ATCM 2004) and for tourism operators by the International Association of Antarctica Tourism Operators (IAATO 2007). Those guidelines that are based on scientific information, such as observations of animal behaviour during controlled experimental approaches (Giese 1998; Giese and Riddle 1999; Holmes et al. 2005), have generally taken a precautionary approach, with separation distances selected to ensure that animals will respond only minimally to the presence of people. It is therefore expected that injury to wildlife, including stress reactions, will not occur as a consequence of visitor disturbance where these guidelines are observed. The long-term cumulative effects of repeated visits to wildlife aggregations are much more difficult to discern than the immediate, short-term responses. Some studies have indicated a negative association between visitor disturbance and factors such as breeding success in the Adélie penguin, *Pygoscelis adeliae* (Giese 1996), fledging weight in yellow-eyed penguins, *Megadyptes antipodes* (McClung et al. 2003), and population growth in Adélie penguins (Woehler et al. 1994); however, other studies report no negative effects of visitation, for example, population change in Adélie penguins (Fraser and Patterson 1997), breeding success in gentoo penguins, *Pygoscelis papua* (Cobley and Shears 1999; Holmes et al. 2006), or in some cases higher breeding success of Adélie penguins in locations subject to high disturbance (Carlini et al. 2007), suggesting that wider environmental influences are exerting greater effects than local human disturbance. Habituation, whereby animals that have been exposed previously to human activity may become less sensitive (Nisbet

2000; Walker et al. 2005, 2006), can also influence the response of individual animals, breeding groups or populations to visitors.

Disturbance caused by a low over-flight during an airdrop at Macquarie Island has been suggested as the cause (Cooper et al. 1994) of perhaps the largest recorded, single-event, mass mortality of wildlife attributable to human activity in the Antarctic and sub-Antarctic in recent times. In 1990, about 12 days after an airdrop and flypast by a Royal Australian Air Force (RAAF) Lockheed C-130 Hercules, approximately 7,000 dead king penguins, *Aptenodytes patagonicus*, including about 1,000 adults and 6,000 chicks, were found in the Lusitania Bay colony (Anon 1990). Corpses were piled up to 10 deep in an area of about 30 m by 6–10 m against a barrier of rock and tussock, suggesting that they had panicked and stampeded (Cooper et al. 1994). Post-mortem examination of five adults indicated that they had died of asphyxiation (Rounsevell and Binns 1991). Although the actual event that caused the deaths of the penguins was not observed and an internal RAAF enquiry exonerated the flypast as the cause, airdrops on Macquarie Island were discontinued. Subsequent observations of king penguin behaviour during airdrops at Marion Island in 1992 confirmed that low passes by aircraft caused panic and scattering, but on these occasions no deaths were observed (Cooper et al. 1994).

13.2.2 Collision and Strike Injuries

Collisions by flying birds against buildings, communications towers and support guys are not uncommon, and in the developed regions of the world frequently cause injury or death. In the US, estimates for the annual mortality from birds striking windows range from 3.5 million (Banks 1979) to between 98 and 976 million (Klem 1990), with reflective plate glass, which is commonly used in Antarctica, identified as a particular hazard (Banks 1976).

Lights from stations and ships attract birds and a low level of mortality has been described as ‘an almost nightly occurrence’ on vessels operating in the Southern Ocean (Black 2005). There have also been several occasions when birds have been found dead on the decks of ships in large numbers. One morning in January 2004, 215 dead birds were found on the deck of the MV *Dorada*, a trawler involved in research in the South Georgia Maritime Zone, and a further 684 were collected live and released (Black 2005). Most of the dead birds were diving petrels, *Pelecanoides* spp., blue petrels, *Halobaena caerulea*, and Antarctic prions, *Pachyptila desolata*. A similar event involving 200 dead birds, including prions and diving petrels occurred in January 1992 (Woehler pers. comm. reported in Black 2005) on the RV *Aurora Australis* while anchored in Atlas Cove Heard Island. Common to both these incidents is that most of the species involved were small burrow-nesting petrels, the vessels were operating at night with lights at the time and weather conditions were calm and foggy. Many of the live birds had become water-logged following prolonged immersion in water-filled cavities on deck and were held in cardboard fish boxes in sheltered but unheated parts of the deck long enough to dry and recondition their feathers before release (Black 2005). Recommendations for

minimising bird strikes all involve reducing external lights, such as search lights used for ice navigation, and deck lights and using blackout blinds to prevent light escaping from portholes (Black 2005). Calm, foggy conditions appear to increase the risk of bird strikes.

Wind turbines are increasingly being used as alternative sources of power at Antarctic stations to reduce the use of fossil fuels, but despite their environmental advantages, they are also known to be a cause of bird deaths in other regions (Drewitt and Langston 2006). The environmental impact assessment for the installation of wind turbines at Australia's Mawson Station identified collision with rotating vanes as a potential risk to flying birds and regular monitoring for bird strikes is required as a condition of the environmental approval. In the 5 years since the wind turbines were established, five snow petrel, *Pagodroma nivea*, carcasses were reported from the area; one of these was seen to hit a blade confirming that the death was caused by the turbine (Lesley Frost, pers. comm.).

Injuries to animals caused by collisions with vehicles or boats are possible, but the Madrid Protocol and the national legislation of most countries include provisions to reduce this happening. Adherence to the guidelines for minimum approach distances for people on foot, vehicles and boats designed to reduce disturbance should help also reduce the risk of collisions. The most likely accidental injury involving a vehicle is the striking of penguins with the propeller of light boats such as inflatable rubber boats, which are widely used by tourist operators and by national Antarctic programs. These boats can travel at 20–30 knots, and at these speeds drivers can suddenly find themselves in the middle of a group of swimming penguins. Injuries from propellers are likely to be severe.

Both seabirds and marine mammals can also receive deep wounds from unsuccessful attacks by predators including leopard seals, *Hydrurga leptonyx*, killer whales, *Orcinus orca* (Maniscalco et al. 2007), and, in the sub-Antarctic, sharks (van den Hoff and Morrice 2007). A mortality event involving a high incidence of physical trauma in a colony of Adélie penguins was attributed to birds being crushed by lumps of floating ice at the land/sea-ice interface (Kerry et al., this volume).

13.2.3 Injuries from Energy Sources

Some radio transmitters used for communications and scientific research create high-energy electromagnetic radiation (microwaves) that is sufficient to cause internal injuries if animals are exposed for long enough. Because the length of exposure to radio waves and their power are the main factors that influence health risks, research transmitters, which are required to operate continually, are regulated more stringently than communications transmitters, which typically operate intermittently. Until recently, regulation of non-ionising radiation, such as by the International Commission for Non-Ionizing Radiation Protection (ICNIRP), has been based on their acute thermal effects (ICNIRP 1998); however, it is now becoming apparent that there is also the potential for a diverse range of non-thermal adverse effects (Banik et al. 2003; Belyaev 2005), including nerve cell damage (Salford et al. 2003), DNA breakage (Diem

et al. 2005), effects on gene experession (Czyz et al. 2004) and others (Belyaev 2005). Reports of population-level effects on non-Antarctic wildlife of exposure to electromagnetic fields from mobile phone base stations are beginning to appear in the literature, including a reduction in breeding success of white stork, *Ciconia coconia* (Balmori 2005), reduction in abundance of house sparrows, *Passer domesticus* (Everaert and Bauwens 2007), and the suggestion that radiofrequency radiation may be contributing to the decline of amphibians (Balmori 2006). No cases of detrimental effects of radio frequency radiation on Antarctic wildlife have yet been reported.

Thermal effluent from power stations is known to have ecological effects (Gallup and Hickman 1975) and has been implicated in both beneficial (Janssen and Giesy 1984) and detrimental impacts on populations in the receiving environment (Mustard et al. 1999). Some apparently beneficial short-term effects, such as that observed in manatees which congregate near outfalls discharging heated effluent (Packard et al. 1989), may prove to be detrimental in the long term if the population develops a reliance on artificial sources of heat (Laist and Reynolds 2005). In addition, thermal stress caused by exposure to heated effluent can make animals more susceptible to disease-causing agents such as parasites (Esch et al. 1976). Antarctic stations can cause thermal changes to the local physical environment, particularly where small natural water bodies are used for the supply of cooling water for engines such as electrical generators. Changes to the protistan ecology of simple Antarctic lake systems have been attributed to waste heat (Ellis-Evans et al. 1997); however, there have been no reports yet of detrimental effects on Antarctic vertebrates.

A number of acoustic technologies commonly employed in the marine environment for navigation, survey and research can use high-energy soundwaves, including seismic survey equipment, depth sounders, acoustic doppler current profilers and acoustic releases. It is possible for soundwaves at sufficiently high intensity to cause physical injury to animals including auditory damage (Finneran et al. 2002), formation of gas bubbles in tissues (Crum and Mayo 1996) leading to decompression-like effects (Piantadosi and Thalmann 2004) and injury to organs caused by resonance effects in body cavities (Balcombe and Claridge 2001 cited in Boebel et al. 2005). A recent qualitative assessment of the risks posed by a range of acoustic scientific instruments in Antarctic waters (Boebel et al. 2005) concluded that there was insufficient evidence of injury to justify a ban on these technologies but that there was also insufficient evidence to confirm that all equipment and surveys were harmless. They recommend that hydroacoustic activities should be documented to allow retrospective assessment of the likely causes of any future observations of changes to potentially affected species and populations.

13.2.4 Entanglement and Ingestion of Litter and Debris

Litter, particularly plastic debris (Thompson et al. 2004), is a growing environmental problem throughout the world both on land and in the sea (Coe and Rogers 1997). It can be harmful to wildlife by various mechanisms, such as entanglement which

restricts the animal's abilities to move and can cause lesions as the animal grows, ingestion which can be detrimental to normal feeding and digestive processes, and physical trauma when blown by strong winds. On land in Antarctica, most debris is created by poor storage of materials at stations allowing dispersal by wind, wind damage to abandoned or poorly maintained buildings (Burgess et al. 1992) and from old waste disposal sites (Snape et al. 2001). Although there are apparent risks to wildlife from this material, there are few reported cases of injury to animals directly attributable to this type of debris; one documented example is of a juvenile Adélie penguin found with its feet wrapped in metal wire which proved to be 50 m long (Woehler 1990).

The release of meteorological balloons used to measure wind speeds at height through the atmosphere is a relatively low-level but ongoing source of litter around many Antarctic stations. These consist of a large latex balloon, a battery, some electronic circuitry, cardboard and polystyrene packaging and several metres of string. At each station from which meteorological observations are carried out, balloons would be launched daily, and at some stations two or three times a day resulting in about 400–1,000 launches per station per year. Despite the large number of releases, the balloons and their cargos are seldom found; however, on one occasion known to the author, a pair of Adélie penguins was found tangled together in the string of a meteorological balloon. Had they not been caught and released, it is almost certain that they would have been unable to feed properly and would soon have died from starvation.

Although land-based sources may be locally important, marine debris is very much more widely dispersed (Derraik 2002) and is the cause of a significant number of injuries to wildlife each year (Arnould and Croxall 1995). With the possible exception of the Antarctic Peninsula region, the ocean around Antarctica is effectively separated from the other oceans of the world by a series of fronts across which only limited exchange is possible. As a consequence, marine debris found there is mostly of local origin, with the fishing industry as the major source (Walker et al. 1997). At some locations, such as South Georgia, the dominant components of marine debris correspond to the nature and intensity of local fishing practices (Convey et al. 2002), with most items being either discarded or lost fishing equipment or domestic waste illegally dumped from fishing vessels. At other locations, such as the South Sandwich Islands (Convey et al. 2002) and Bouvetøya (Hofmeyr et al. 2006), debris is of more distant origin. As interest in Antarctic marine living resources increases and the number of fishing vessels operating in the Southern Ocean grows, the amount of marine debris in the Southern Ocean is also likely to increase unless efforts to ensure compliance with the MARPOL (International Convention for the Prevention of Pollution from Ships, 1973) provisions on waste disposal at sea by the Commission for the Conservation of Antarctic Marine Living Resources (CCAMLR) are successful (Arnould and Croxall 1995).

Entanglement of seals and seabirds is a particular risk with fishing equipment such as hooks, lines, ropes and nets, but is also a risk with packing case straps, polythene bags and plastic sheeting (Croxall et al. 1990; Arnould and Croxall 1995; Kock 2001). The incidence of entanglement varies with species, location and the

amount of fishing debris present, but in some places has been as high as 0.4% of the population (Antarctic fur seals at Bird Island, South Georgia, reported by Croxall et al. 1990), representing thousands of individual animals effected each year. Entanglement in large items may cause rapid death by drowning or may cause more prolonged harm or death by restricting normal movement (Campagna et al. 2007). Smaller items such as polypropylene packaging bands may have little immediate effect but, as the animal grows, can tighten and cut into the flesh causing deep lesions. In some areas, packaging bands have represented more than 50% of the cases of entanglement (Croxall et al. 1990). A campaign by CCAMLR to publicise the harmful effects of packaging bands and to promote adherence to the MARPOL requirements for correct disposal of waste from vessels may have triggered a substantial reduction in the incidence of entanglement in packaging bands reported for the period 1989–1994 (Arnould and Croxall 1995). Fishing net fragments around the necks of seals were the second most frequent type of entanglement (20%) reported by Arnould and Croxall (1995) and were probably caused by the seal being cut from the net without fully releasing it from the collar formed by the remaining net fragment. Capturing the animal and removing the article is the only effective treatment but, to be done safely, requires people experienced in handling large animals.

There are a far fewer reports of seabirds living with fishing gear or marine debris entangled around them than there are for seals. This is because birds are less likely to survive the initial entanglement and probably die at sea as 'fishery by-catch' (discussed later). Occasionally, seabirds will survive entanglement long enough to get ashore; for example, a long-dead south polar skua, *Catharacta maccormicki*, entangled in fishing line was found near Casey Station (Woehler 1990).

Ingestion of plastics by seabirds has been widely reported from non-Antarctic regions (Derraik 2002) and is common in Antarctic seabirds (Kock 2001). Items ingested range in size from whole plastic bags to small worn-down pellets of plastic and polystyrene (Copello and Quintana 2003). The incidence of plastic ingestion varies among the different species depending on factors such as foraging strategy, diet and migrations (Ryan 1987; Azzarello and Van Vleet 1987) and within a species on location (Spear et al. 1995). Surface feeding seabirds, such as storm petrels and prions, have a higher incidence of ingested plastics than those that feed subsurface (Ainley et al. 1990) and birds that forage in places with a high incidence of floating marine debris have a higher incidence of ingested plastics. Plastic particles have been found to be more common in seabirds that breed on the Antarctic continent but migrate north during winter, such as Wilson's storm petrels, *Oceanites oceanicus*, and Cape petrels, *Daption capense* (van Franeker and Bell 1988), than in species which spend their entire lives in the Antarctic region such as snow petrels, although ingested plastics have been found even in these. Ingested plastics can be passed from foraging adults to their unfledged young. Of the southern giant petrel chicks from Isla Arce on the Patagonian Shelf, Argentina, 73% had some type of marine debris, including plastics, in their stomachs (Copello and Quintana 2003), and in the Antarctic, chicks of Wilson's storm petrels that had died before fledging contained more plastic particles than were found in the stomachs of dead adults (van Franeker and Bell 1988).

The harmful effects of ingested plastics include reduction of the storage volume of the stomach and reduction of the feeding stimulus leading to reduced food consumption (Ryan 1988) and lessening of the ability to lay down fat (Connors and Smith 1982; Spear et al. 1995). Plastics may also cause injury to the intestinal tract (Ryan and Jackson 1987) and block gastric enzyme secretion, and may increase body levels of pollutants such as polychlorinated biphenyls (PCBs) which are commonly found in plastics (Ryan et al. 1988). There is some evidence that mechanical lesions from ingested plastics are rarely the direct cause of death (Sievert and Sileo 1993). Vulnerability to the detrimental effects of plastic ingestion varies between species, with those that seldom regurgitate indigestible stomach contents most prone to adverse effects (Ryan 1990).

If plastic ingestion is suspected as a factor in an unusual wildlife health event, examination of the gut content and scats should indicate whether plastics are present; however, their presence does not confirm they were the cause of death. The risk to wildlife from ingestion of particles of expanded polystyrene is recognised within the Antarctic Treaty System, and for this reason polystyrene beads, chips or similar forms of packaging are listed in the Madrid Protocol as prohibited products that should not be taken to Antarctica.

13.2.5 Fishery by-catch

Without doubt the most serious cause of injury and death among Antarctic wildlife in recent years has been the incidental mortality associated with longline fisheries, which saw more than 6,000 seabirds taken as by-catch in the CCAMLR Convention Area in 1997 (CCAMLR 2007). More recently it was discovered that a great many seabirds (26,668, mostly white-chinned petrels, *Procellaria aequinoctialis*, between September 2001 and August 2003) were being killed by the legal Patagonian toothfish fishery in the French Exclusive Economic Zones (EEZs) of Crozet and Kerguelen Islands (Delord et al. 2005). Fortunately, once the problem was recognised, the quick introduction of a range of mitigation measures caused the mortality rate to fall dramatically so that in some sectors of the Southern Ocean mortality is now in the order of tens per year rather than thousands (CCAMLR 2007). However, despite the success of CCAMLR, high rates of seabird mortality also occur in some adjacent areas which are beyond the jurisdiction of CCAMLR, such as off South Africa where the annual mortality of the white-chinned petrel from the hake longline fleet was estimated at 58,800 birds (Barnes et al. 1997), and around the Falkland Islands where the trawl industry killed an estimated 1,500 birds, mostly black-browed albatross, *Thalassarche melanophris*, in 2002/2003 (Sullivan et al. 2006).

Incidental mortality from the longline industry is largely the result of seabirds being drowned after taking baited hooks while lines are being set. Accidental injury can also occur by foul-hooking seabirds when lines are being recovered. Mitigation measures, such as the use of streamers to deter birds and ensuring longline deployment and offal discharge points are separated on opposite sides of the vessel, have reduced

the incidence of this type of injury but foul hooking still occurs and may cause physical trauma such as skin tears, broken bones or birds with imbedded hooks with line attached. Seabirds can also be killed through drowning after entanglement in driftnets and trawl nets when at the surface, and through collisions with trawl cables (Sullivan et al. 2006). CCAMLR decided in 1990 that pelagic driftnet fishing should not be carried out in the CCAMLR Convention Area (CCAMLR 2007) effectively eliminating this risk from the region, at least from legal and regulated vessels. In 1994, trawl net sonde cables were banned by CCAMLR because of reported injuries to seabirds from collisions. Other measures to reduce incidental mortalities are the requirement to clean nets before they are set to reduce their attractiveness to birds and arrangement of deck lighting to minimise the illumination that is directed out of the vessel (CCAMLR 2007).

A persistent or sudden decline in numbers of a nesting population of seabirds in Antarctica may indicate that a new fishery has been established in their foraging area and should be reported to CCAMLR.

13.3 Chemical Pollution

Chemical pollutants may get to Antarctica either from activity in Antarctica (local sources) or from activities elsewhere in the world with the pollutants being transported to Antarctica (remote sources) in the atmosphere or ocean. When investigating an unusual wildlife event, if pollution is suspected as a cause there are more likely to be clues to pollution of local origin, if involved, such as oil slicks or abandoned waste disposal sites, than to pollution from remote sources, which as a consequence could easily be overlooked. Protocols for collecting samples for toxicological analysis have been developed by CCAMLR (reproduced as Appendix B this volume).

13.3.1 Pollution from Local Sources

Pollution from local sources may either be from deliberate management decisions, such as waste disposal to land-fill or sewage effluent disposal to the sea, or from accidents such as oil spills. Over recent years, improved environmental controls, particularly arising from the Madrid Protocol, have stopped many practices which in the past caused pollution. Within the Antarctic Treaty area, waste can no longer be dumped in open land-fill sites, there are restrictions on the disposal of sewage and sewage effluent and some chemicals are completely banned. However, past practices have left a legacy of contaminated sites in Antarctica and on the sub-Antarctic islands which wildlife may come in contact with. The practicalities of operating in Antarctica mean that some potentially polluting activities are still permitted, including sewage effluent disposal to the sea, incineration of some waste material, the use of anti-freeze chemicals in ice-core drilling and, of course, exhaust from the use of generators, vehicles and aircraft.

Abandoned waste disposal sites in Antarctica contain a range of materials similar to those found in any municipal waste tip. The material would have come from workshops, kitchens and laboratories, and could include anything used on station, such as food waste, batteries, electronic equipment, vehicle parts, paint tins, waste lubricants as well as laboratory and photographic chemicals. As a consequence, tips contain a variety of potentially toxic substances (Snape et al. 2001) including metals and metalloids, such as copper, lead, zinc, cadmium, silver and arsenic, and organic chemicals such as petroleum-derived hydrocarbons, anti-freeze and specialised materials such as the PCBs used as coolant oils for transformers and as plasticisers in some paints.

Despite the cold of Antarctica, contaminants at these sites are not permanently frozen in place. Many Antarctic stations are on the coast and it was common practice to locate waste tips near the shoreline, often in gullies that become stream beds during the summer melt. It was also not unusual to push rubbish onto the sea-ice or into the sea for disposal. There are therefore a number of mechanisms by which contaminants from land-based activities can be mobilised and dispersed in both terrestrial and marine environments (Snape et al. 2002) and enter the nearshore marine food chain (Duquesne and Riddle 2002). There is also a growing body of evidence indicating that at even relatively low environmental concentrations metals and hydrocarbon contaminants can cause changes to Antarctic marine benthic communities, such as loss of diversity (Lenihan and Oliver 1995; Stark et al. 2003).

Major oil spills are among the most serious pollutant threats to the health of Antarctic wildlife. They are also likely to be the easiest to trace back to source, as in many cases there will be visible signs of an oil spill and, in contrast with other potential environmental pollutants, the mechanism of harm is primarily through the direct effects of smothering, fouling of feathers and ingestion of oil during preening rather than through exposure to invisible toxic components such as the polycyclic aromatic components of oil dissolved in the water column (Boehn et al. 2007).

Fuel oil will be used to power ships, aircraft and research stations for the foreseeable future, bringing with it the possibility of major oil spills. Recognising that the likelihood and consequences of oil spill puts it among the highest environmental risks to the Antarctic (COMNAP 1999), many nations now have well-documented procedures for fuel handling and transfer, including continual monitoring to ensure early detection of spills. Most stations and ships also have oil-spill contingency plans and spill kits designed to reduce dispersion and environmental damage from a spill. Unfortunately, accidents still occur despite improved management practices. Handling and transport of fuel and oil is risky in any environment, but the particular conditions found in the Antarctic significantly increase the risks. Strong winds and extreme cold combine to make all operations more difficult and increase the chances of human error. Spills can also occur because of failure of storage facilities. Leaks from damaged pipe-work are a particular risk in a seasonally snow-covered environment because they may go unnoticed for many months.

In recent years several vessels have suffered hull damage in the Southern Ocean with resulting loss of fuel and oil to the environment; however, there appears to have been relatively few confirmed mortalities of seabirds or mammals as a direct effect of oiling caused by these spills. In December 1987, the re-supply vessel

Nella Dan working for Australia's national Antarctic program went aground on sub-Antarctic Macquarie Island and subsequently sank releasing 270,000 l of mostly light marine diesel. The oil was blown onshore for the first 26 h following the grounding, and was then blown out to sea. Although mortality among marine invertebrates was high with thousands of animals reported to be washed ashore in the first few days following the spill (Smith and Simpson 1995), little effect was noted on penguins and seals beyond slight oiling of a few rockhopper penguins, *Eudyptes chrysocome*, in the immediate vicinity of the grounding (J. Reeve, pers. comm.). In January 1989, the Argentine tourist and re-supply vessel *Bahia Paraiso* ran aground near the U.S. Palmer Station releasing approximately 600,000 l of diesel fuel (Kennicutt and Sweet 1992). Few seabirds appear to have died as a direct result of smothering or toxicity, although large numbers of birds were observed to be directly exposed to the oil both externally on their feathers (Fraser and Pattersen 1997) and internally through ingestion of large numbers of dead krill that were washed ashore (Barinaga and Lindley 1989). It is possible that the most significant impact of the spill was the complete reproductive failure of the population of south polar skuas. It has been suggested that sub-lethal oiling of adults disrupted parental guarding and allowed unattended chicks to be preyed on by other skuas resulting in the loss of all chicks from the local population (Eppley and Rubega 1990; Eppley 1992); however, an alternative explanation of natural variation in reproductive success has also been proposed (Trivelpiece et al. 1990).

There have been 6 recent marine incidents involving tourist vessels in the Southern Ocean, all of which were in the Antarctic Peninsula region. Three resulted in the loss of smaller quantities of oil and again there have not been reports of large-scale seabird or marine mammal mortalities. In November 2007 the tourist vessel MS *Explorer* struck an iceberg and sank off King George Island with 190,000 litres of diesel, 24,000 litres of lubricant and 980 litres of petrol on board. In February 2007, MS *Nordkapp* ran aground at Deception Island in the South Shetland Islands, spilling about 1,000 litres of oil. The MV *Ushuaia* grounded near Cape Anna in the Antarctic Peninsula region in December 2008 rupturing the fuel tanks releasing some marine gas oil, with initial reports of a 50 m x 500 m slick in the vicinity of the vessel. The other three incidents which occurred without reports of fuel or oil spill were the MV *Ocean Nova* which grounded in Marguerite Bay on the Antarctic Peninsula (February 2009), the MS *Fram* which drifted in to a glacier after a power failure (December 2007) and the MV *Lyubov Orlova* which grounded at Deception Island (November 2006).

It would appear that Antarctic wildlife have, until now, been lucky in avoiding the worse consequences of a major maritime oil spill, although the light diesel commonly used for Antarctic operations may reduce the risk. Experience in non-Antarctic regions indicates that penguins are particularly vulnerable to oil spills. The grounding of the ore carrier *Iron Baron* in northern Tasmania in July 1995 resulted in the release of 325 tonnes of bunker oil fuel and is estimated to have caused the death of between 10,000 and 20,000 penguins, *Eudyptula minor* (Goldsworthy et al. 2000a). One thousand eight-hundred and ninety-four penguins were treated for oiling, 95% of which survived rehabilitation and were released back into the wild; however, the post-release survival rate of rehabilitated oiled birds was estimated at just 44–59% (Goldsworthy et al. 2000b). The harmful effects on individuals continued

for several years after the spill, with breeding success of rehabilitated oiled female birds 2 years after the event being 22% lower than for non-oiled females (Giese et al. 2000). In South Africa, between 1970 and 2000, there were at least 14 major oil spills that threatened populations of African penguins, *Spheniscus demersus*, but only some of these caused large numbers of penguins to be oiled and they were not necessarily the spills involving the largest quantity of oil (Nel et al. 2003). The location of the spill and the prevailing weather conditions appear to be more important in determining the level of harm to wildlife than the size of the spill.

Rescue and rehabilitation of oiled birds requires significant resources and is costly; estimates range from US$90 per bird for the MV *Treasure* oil spill in South Africa (Nel et al. 2003) to about US$51,000 per bird for the *Exxon Valdez* spill in Alaska (Sharp 1996). It is questionable whether a large-scale rehabilitation program could be mounted anywhere in Antarctica and it is certainly not feasible anywhere but adjacent to one of the larger research stations.

13.3.2 Pollution from Remote Sources

Certain classes of pollutants, collectively known as persistent organic pollutants (POPs), such as the DDT group of organochlorine pesticides, polychlorobiphenyls and the brominated flame retardants, can be transported great distances by atmospheric or oceanic processes, including to Antarctica (Wania and Mackay 1993). The specific mechanisms of transport are determined by the volatility and water-solubility of each chemical (UNEP 2003). Very volatile substances, such as the chlorofluorocarbons, tend to remain in the atmosphere and do not condense out on the earth's surface. Chemicals that readily shift between gas and liquid phases, such as PCBs, toxaphene, dieldrin, chlordane and endosulphan, can be transported selectively to cold regions through the process of 'global distillation' in which volatile chemicals evaporate from warmer regions and condense in cooler places. Chemicals that are non-volatile and insoluble, such as Mirex (perchloropentacyclodecane) and decachlorobiphenyl, tend to move only in association with particulates, either as dust carried in air or as suspended particles in water. Highly water soluble chemicals, such as atrazine, can be transported in solution; however, although there is significant exchange of water between the Southern Ocean and the rest of the world ocean, the total quantity of pollutants transported in solution in the ocean is expected to be fairly small, in the range of 10 kg to 10 tonnes per year (UNEP 2002).

There is a strong asymmetry in the world-wide production and use of most of these chemicals, with greater quantities being used in the Northern Hemisphere, and the global distillation process tends to maintain the hemispheric differences. Recent assessments indicate that organochlorine levels in the Southern Hemisphere, and the Antarctic in particular, are significantly lower than the mid-latitudes of Europe and North America which show the greatest loads (Aguilar et al. 2002; UNEP 2003). However, as the use of some chemicals, such as DDT, has been banned by many countries, concentrations have tended to decrease in the regions where pollution was initially high; in contrast, concentrations in both polar regions

continue to rise as a consequence of global transport redistributing these chemicals to the cold regions (Aguilar et al. 2002; UNEP 2003). Once in the Antarctic, these chemicals bio-magnify up the food chain and can reach high levels in some top predators (Goerke et al. 2004).

Pollutants may also be transported to Antarctica in the tissues of migratory species (Wania 1998) that travel to more polluted regions at lower latitudes. Several studies have reported higher levels of POPs in tissues from migratory species than in those which spend their entire life in the Antarctic (Lukowski et al. 1987; Luke et al. 1989; Court et al. 1997), suggesting that most accumulation occurs during periods of higher environmental exposure north of the Antarctic convergence. On a regional basis, the total quantity of pollutants transported by this mechanism is unlikely to be very great compared to other processes such as global distillation, but it may be the most significant process in relation to the health of Antarctic wildlife. Many of the persistent toxic substances are lipid-soluble and tend to accumulate in fat reserves that may be used during migration, causing mobilisation of the pollutants and potentially triggering health effects which until then may not have been realised.

Although most studies indicate that levels of POPs in Antarctic species are generally lower than those measured in similar species from elsewhere (UNEP 2002), there have been some reports of surprisingly high levels, with the overall patterns varying among different species and different chemicals. For example, PCBs and *p*,*p*′-DDE were very low in the south polar skua compared to those in Arctic glaucous gull, *Larus hyperboreus*, and great black-backed gulls, *Larus marinus*, but Mirex levels were 3 to 26 times higher and among the highest reported for birds (Bustnes et al. 2006). In addition, levels of several POPs in skuas increased by 30–60% in a 2-week period after their return to Antarctica, suggesting that they were accumulating these compounds in the region (Bustnes et al. 2006). Higher levels of hexachlorobenzenes (HCBs) and volatile PCB congeners have been reported from several Antarctic seabird species than from a tropical species (van den Brink 1997) and have been attributed to directed transport of volatile compounds by global distillation.

There have been very few controlled exposure experiments using any seabirds or marine mammals that can be used to directly relate measured concentrations of POPs in tissues to health effects. The recent regional assessment of POPS in the Arctic (AMAP 2004) identifies this as a major limitation on the ability to interpret residues in Arctic animals and mentions only two such studies. Glaucous gulls fed a diet high in PCBs had an impaired ability to produce antibodies, and two juvenile harp seals, *Phoca groenlandica*, treated with increasing doses of PCB congeners and then starved had increased levels of serum cortisol, aldosterone and tumour necrosis factor alpha than controls (AMAP 2004). Data from common laboratory animals such as mink kits and rhesus monkeys were used as the main guide to determine whether concentrations measured in Arctic species were significant (AMAP 2004). It is unsurprising that there are no such studies using Antarctic species.

Most of the literature from non-polar regions on the health effects of POPs in seabirds and marine mammals is based on observed associations between high loadings of POPs in marine mammals and birds, and detrimental health events, such as strandings, parasite infestations and incidence of infectious disease.

The limitation with this approach is that the cause–effect relationship is not absolutely confirmed (Jepson et al. 2005) and it leaves unresolved the many potentially confounding factors such as age, sex, diet, condition of individuals, and individual and species-specific ability to metabolise and excrete pollutants (Evans 2003). Recently, however, new statistical treatments of large datasets are proving useful in separating the effects of contaminant loading from the confounding variables. For example, PCB levels in harbour porpoises were found to be higher in animals that had died from infectious disease than in those that had died from acute physical trauma (Jepson et al. 2005) and the risk of infectious disease increased by 2% with every 1 mg kg^{-1} increase in blubber PCBs (Hall et al. 2006).

Although there have been many investigations of POPs in Antarctic species since these chemicals were first discovered there in the 1960s (Sladen 1966), few accounts have been published of detrimental health effects to Antarctic birds or marine mammals that can be positively attributed to exposure to POPs. One recent study has demonstrated that eggs from female south polar skuas with high levels of organochlorines hatched later, and their chicks were in poorer condition, than those from females with low levels, suggesting that organochlorines may delay reproduction and reduce foetal growth (Bustnes et al. 2007). The suggestion that Antarctic birds are particularly at risk from POPs because of their extreme variability of physical condition during the breeding season (van den Brink et al. 1998) is supported by observations that concentrations of both PCB and DDE in fat tissues of Adélie penguins increased linearly as fat reserves shrink with advancing starvation during the breeding season (Subramanian et al. 1986).

On the basis of experience elsewhere, if high concentrations of POPs are found during the investigation of an unusual health event involving Antarctic birds or mammals species, it is likely that there will be other factors present, such as disease or starvation due to food shortage or stage in the breeding cycle. As a consequence, it is unlikely that it will be possible to conclusively attribute a cause to the presence of POPs.

13.3.3 Relative Sensitivity of Antarctic Species to Pollutants

A number of studies have reported potentially harmful anthropogenic substances, both locally sourced and globally transported, including metals, hydrocarbons and organochlorines, in tissues from Antarctic invertebrates (Duquesne and Riddle 2002), fish (McDonald et al. 1992; Weber and Goerke 2003), seabirds (Luke et al. 1989; Court et al. 1997) and marine mammals (Goerke et al. 2004). However, to date there have been very few controlled toxicological studies using Antarctic species, or indeed, using any polar species whether from the Arctic or the Antarctic (Chapman and Riddle 2003).

It is not possible to say with any degree of certainty whether, in comparison with temperate species, Antarctic species are more sensitive, less sensitive or about as sensitive to even the most common environmental pollutants (Chapman and Riddle 2005) because of the limited amount of toxicological data available. In addition, when investigating a wildlife health event in which pollution is thought to have played a

part, it is not possible to use Antarctic-specific data to determine whether pollutants are present at levels that are likely to be significant to the health of local animals. The best that can be done is to compare with levels known to cause detrimental effects in temperate areas. Recognising the limitations imposed by the paucity of data, overall there is very little direct evidence that contaminants from land-based sources in Antarctica are getting into vertebrate top predators in harmful concentrations.

Metabolic indicators of exposure to pollutants, or *biomarkers*, are beginning to be studied only recently in Antarctic species but are showing results consistent with those from other regions. An experimental exposure of gentoo and Adélie penguins to trimethyltin, aviation fuel and diesel showed marked changes in blood proteins and enzymes indicating significant toxic responses (Najle et al. 2006), suggesting that serum protein and enzyme levels could be used as indicators of exposure to contamination in these species. The cytochrome P450 (subfamily 3A) detoxifying system in the liver has been shown to operate on quinine at a much lower rate in Adélie penguins (Wanwimolruk et al. 1999) than in humans, suggesting that penguins are much less capable of eliminating toxins of this type. These results also demonstrate that the cytochrome P450 system could be a useful biomarker for exposure of Adélie penguins to xenobiotics, as it is for other species. One study has shown that the specific cytochrome P450 enzymes differ between Adélie penguin chicks and adults, suggesting that they may have different susceptibilities to environmental pollutants at different life stages (Numata et al. 2004).

13.4 Toxic Algal Blooms

Toxic algal blooms are reported to have increased globally in recent years (Hallegraeff 1993), and at least 200 species in a wide range of phytoplankton groups are known or suspected to be toxic (Landsberg 2002). Although toxic algae have been suggested as a possible cause of several unusual mortalities in Antarctic seabirds (Shumway et al. 2003) and marine mammals (Baker 1999), as yet there appears to be no conclusive evidence, such as the identification of specific algae-derived toxins in the stomach. However, blooms of algae from groups known to be toxic, such as the genus *Phaeocystis* (DiTullio et al. 2000), are common in Antarctic waters, and the deaths of captive king penguins fed with anchovies found later to contain algal toxins (Naar et al. 2002, cited in Shumway et al. 2003) suggest that penguins are susceptible to these toxins.

There is a wide range of sensitivities and symptoms to algal toxins, as both vary depending on the algae involved and the species affected. Low doses can impair swimming, flying and foraging through loss of motor coordination and may lead to starvation. Loss of coordination may also be observed as uncharacteristic gait and stance, tremors and rapid involuntary movement of the eyeball. Other effects may include the inability to lay eggs, vomiting and abnormal faeces. Higher doses can lead to impaired respiration, paralysis and death. Symptoms frequently include extensive inflammation or haemorrhaging of the internal organs, with blood vessels enlarged and possibly blood on feathers around the vent (Shumway et al. 2003).

13.5 Outlook for the Future

Since the end of large-scale commercial sealing and whaling, the most serious human-related impact on Antarctic wildlife has been the mortality of seabirds caused by commercial fisheries. Despite the success of CCAMLR in changing the practices, it is likely that illegal and unregulated vessels operating outside the CCAMLR Convention will continue to kill large numbers. With the growing global population, the ever-rising demand for protein and the failure of many of the world's fisheries, this mortality is likely to rise as more unregulated vessels search out new opportunities in the Southern Ocean. Increasing unregulated fishing pressure on fish, and possibly krill, could also ultimately lead to food shortages for the higher predators, with the potential for a range of health effects such as reduced breeding success, loss of fat reserves, reduced fitness, increased susceptibility to disease and parasites and even starvation.

In the longer term, anthropogenic climate change may become a significant pressure on the health of the region's birds and marine mammals (Harvell et al. 1999). Changes in the distribution and abundance of penguin species, consistent with warming-induced reductions in annual sea-ice extent, have already been seen in the western Antarctic Peninsula, with those species favoured by a reduction in sea-ice, gentoo and chinstrap, extending their ranges further south at the expense of the ice-dependent Adélie penguins (Smith et al. 1999).

There is still a great deal unknown about how climate change may affect the Antarctic ecosystem, both in the mechanisms of effects and their consequences. The direct effects of warming may cause some species to expand or contract their ranges; however, the potential variety of indirect effects of climate change is enormous, including interacting effects on distribution, abundance, reproductive success, fitness and vigour, susceptibility to disease and contaminants, as well as the cascading and interacting effects of all these factors on both prey and predator species (Learmonth et al. 2006). Despite the development of ever-more sophisticated models, with such inherent complexity, it is quite possible that science will fail to predict the exact mechanism of some serious future impacts. Past experience has shown that long-term datasets from carefully targeted and designed monitoring programs are the most reliable means of establishing trends and identifying changes that may raise the alarm alerting us to some shift in the system.

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Chapter14
Measuring Stress in Antarctic Seals

C. J. Hogg and T. L. Rogers

The term stress is used widely to describe the possible effects of external factors (stressors) on animals at both the individual and population levels. An important question in biology is how animals cope with their environment (Romero and Reed 2005). Assessing this coping mechanism can be done either behaviourally or physiologically or by a combination of both. Stressors include those aspects of everyday life such as energetic and physical demands as well as the more unpredictable events such as habitat loss, predation risk, loss of social status and impacts of human activities.

Antarctic animals live in weather conditions that would be considered extreme by most humans and are subject to changes in prey sources and, more recently, to localised disturbance by tourist activities. The Protocol on Environmental Protection to the Antarctic Treaty has highlighted the need to understand the impacts of human activities in Antarctica. An understanding of how Antarctic species cope with their environment is needed if we are to understand how human activities are impacting different species. In the past, human impact studies focused on changes in species' demographic patterns such as breeding success (e.g. Frederick and Collopy 1989; Lord et al. 2001), but more recently attention has been given to the physiological changes that occur in species which are subject to human disturbances (e.g. Romero and Wikelski 2002; Müllner et al. 2004; Walker et al. 2006). Behaviour, heart rate and physiological responses are modulated depending on the type of stressor (Nephew et al. 2003), and so in order to monitor human disturbances in Antarctica, both behavioural and physiological responses should be measured (Walker et al. 2006).

Physiological responses can be assessed by determining the concentration of stress hormones in samples collected either invasively (blood) or non-invasively (faecal or urine). The purpose of this chapter is to provide a basic understanding of

C.J. Hogg and T.L. Rogers
Evolution and Ecology Research Centre, School of Biological, Earth and Environmental Sciences, University of New South Wales, Sydney, NSW 2052, Australia
e-mail: c.hogg@unsw.edu.au; tracey.rogers@unsw.edu.au

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the physiology of stress and review the methods of assessment of stress hormones in Antarctic seals. Particular attention is given to non-invasive techniques that have been recently developed, as invasive methods impart their own level of stress on the animal and can confound results.

The stress response is an in-built mechanism designed to protect animals from external noxious stimuli (Romero 2002). It is a result of the activation of the hypothalamic–pituitary–adrenal (HPA) axis which releases glucocorticoids (cortisol, corticosterone) into the system a few minutes after the stressful event. These glucocorticoids then induce both physiological and behavioural responses in the animal. Cortisol is the dominant glucocorticoid in all marine mammals, although corticosterone concentrations may parallel those of cortisol in bottlenose dolphins (St Aubin and Dierauf 2001). Cortisol is produced by the adrenal cortex in mammals and is excreted in a diurnal rhythm as well as a response to stress (Norman and Litwack 1997). There is also evidence that many free-living species modulate their cortisol concentrations seasonally (Romero 2002). The excretion and maintenance of cortisol is through the HPA. During stressful events, corticotrophic releasing hormone (CRH) is released from the hypothalamus and in turn causes the release of adrenocorticotrophic hormone (ACTH) from the pituitary. ACTH binds to specific receptor cells on the adrenal cortex and influences the secretion of cortisol into the bloodstream. A corticosteroid-binding globulin (CBG) is present in the blood and binds cortisol with a relatively high affinity, and so cortisol circulates in a bound form with only small amounts of free hormone present. During a stressful event, cortisol and other glucocorticoids alter carbohydrate metabolism to increase available energy; permit catecholamines, such as adrenaline, to act on metabolic pathways and increase blood flow; and provide protective adaptations to distress by limiting immunological reactions, including inflammations, which minimise cell and tissue damage (St Aubin and Dierauf 2001).

There are two types of stressors: short-term stressors, which are also commonly known as the ‘alarm’ response, and long-term stressors. The alarm response suppresses unnecessary processes and promotes survival until the stressor passes (Sapolsky 1987). Generally reproduction and territorial behaviour are suppressed during stress (Free and Tillson 1973; Moberg 1985; Sapolsky 1987; Wingfield 1988) whereas energy mobilisation is elevated (Wingfield et al. 1997). Once the stress has passed, the glucocorticoid concentrations decline and reproduction and territorial behaviour are resumed. The adrenocortical response is important in the short term but can become harmful if it is repeatedly activated (Sapolsky 1992; Romero 2002). The term ‘modifying factor’ is generally preferred in reference to long-term stressors such as environmental conditions, as it is only (Table 14.1) under severe conditions that they are truly stressful (Wingfield et al. 1997). Mammals undergoing long-term stress will have higher amounts of circulating cortisol than unstressed mammals (Norman and Litwack 1997).

Many Antarctic species have unique physiological adaptations to survive in the extreme Antarctic climate. Knowledge of the baseline cortisol concentrations in Antarctic species will assist in understanding the impacts of different stressors, either anthropogenic or environmental. Antarctic seals are known to have some of

Table 14.1 Cortisol concentrations of Antarctic seals in different matrices. There are no values for cortisol concentrations in the Ross seal, *Ommatophoca rossii*, at this time

Species	Plasma	Faeces	Pure urine	Ice urine
Crabeater seal, *Lobodon carcinophaga*	48.91 ± 3.62 μg dL^{-1} (n = 3) (Liggins et al. 1993)	N/A	N/A	N/A
Southern elephant seal, *Mirounga leonina*	51.46 μg dL^{-1} (n = 2) (Liggins et al. 1993)	N/A	N/A	N/A
Leopard seal, *Hydrurga leptonyx*	61.95 ± 2.17 μg dL^{-1} (n = 4) (Liggins et al. 1993) 46.8–85.9 μg dL^{-1} (n = 10) (Rogers, unpublished data)	N/A	0.99 μg mg^{-1} (n = 1) (Rogers, unpublished data)	0.516 – 0.971 μg mg^{-1} (n = 7) (Rogers, unpublished data)
Weddell seal, *Leptonychotes weddelli*	86.26 ± 5.80 μg dL^{-1} (n = 3) (Liggins et al. 1993) 76.38 ± 1 1.73 μg dL^{-1} (n = 14) (Rogers, unpublished data)	1.38 ± 0.85 μg g^{-1} (n = 80) (Rogers, unpublished data)	1.48 ± 0.24 μg mg^{-1} Cr (n = 19) (Constable et al. 2006)	1.03 ± 0.09 μg mg^{-1} Cr (n = 126) (Constable et al. 2006)

the highest circulating cortisol concentrations of any species (Liggins et al. 1993; Constable et al. 2006) (Table 14.1). The reason for these high cortisol concentrations is unknown. Liggins et al. (1993) attributed these high concentrations to deep diving behaviour. However, the southern elephant seal, *Mirounga leonina*, is a deeper diver than the leopard seal, *Hydrurga leptonyx*, and yet leopard seals have higher circulating cortisol concentrations than southern elephant seals (Table 14.1). High cortisol concentrations in Antarctic seals may be a result of extreme weather conditions, as Weddell seals, *Leptonychotes weddelli*, have the highest cortisol concentrations of any marine mammal and are the most southerly dwelling mammal species (King 1983).

Obtaining baseline cortisol concentrations from free-living animals can be problematic, as cortisol concentrations increase during stressful events. There are two types of capture: physical restraint and chemical restraint. Physically restraining animals to collect blood samples can increase their cortisol concentrations. For most species that have been studied, cortisol concentrations begin to increase within 2–3 min of capture (Romero 2002; Romero and Reed 2005). So only blood samples

collected during this brief window (Sapolsky et al. 2000; Wingfield et al. 2001; Romero and Reed 2005) or prior to capture (Sanvito et al. 2005) represent pre-stress concentrations. Under chemical restraint, the drug is administered and then the animal is left for a period of time so the drug can take effect. So handling and blood sample collection usually occurs 10–15 min after the drug has been administered. At this time the impacts of chemical restraint on pinniped cortisol concentrations is unknown; however, caution should be used when determining cortisol concentrations using chemical sedation, as the administration of chemical sedatives has been shown to elevate serum cortisol concentrations in primates (Fuller et al. 1984; Walker et al. 1987; Udelsman and Chrousos 1988). As chemical or physical restraint is required to collect blood samples from Antarctic seals, it is unlikely that pre-stress cortisol concentrations will be obtained. This has led to an increase in the development of non-invasive sampling techniques for hormonal analysis. Reproduction and stress hormones have been studied in both terrestrial and marine species using faeces (Kirkpatrick et al. 1991; Wasser et al. 1993; Jurke et al. 1997; Goymann et al. 1999; Lynch et al. 2002; Huber et al. 2003; Nakagawa et al. 2003; Rolland et al. 2005); urine (Miller et al. 1991; Brown et al. 1995; Czekala and Sicotte 2000; Kjeld 2001; Stoinski et al. 2002; Constable et al. 2006), ice-urine (McLeod et al. 1996; Constable et al. 2006), saliva (Pietraszek and Atkinson 1994; Lutz et al. 2000; Iwata et al. 2003; Hogg et al. 2005) and cetacean blow (Hogg et al. 2005; Hogg et al. 2009). Blood samples from Antarctic seals have been commonly used to measure cortisol concentrations (Liggins et al. 1979, 1993); but more recently, assays using urine (Constable et al., 2006), ice-urine (Constable et al. 2006) and faeces (unpublished data) have been developed. Urine samples can be easily utilised with the current analytical techniques, as they do not require extraction. However, if faecal samples are to be used to determine cortisol concentrations, samples need to be extracted prior to analysis. It should be noted that because of the high lipid content of most Antarctic seal diets, faecal samples need to be extracted using diethyl ether to ensure maximum recovery of cortisol during the extraction process.

The most commonly used analytical techniques to determine cortisol concentrations are immunoassays, either radio-immunoassay (RIA) or enzyme immunoassay (EIA). Assay kits can either be commercially available kits, commonly used for blood and urine samples, or can be individually tailored for a specific matrix (faeces, urine or saliva). Typically it is advisable to use a commercial kit rather than one specifically developed in a laboratory for a certain species, as this allows comparative studies because other laboratories can replicate the work. Other biochemical analytical techniques used to study hormones include high-performance liquid chromatography (HPLC) and chromatography linked with a mass spectrometer, such as liquid chromatography-mass spectrometry (LC-MS-MS). The various analytical techniques have different advantages. Immunoassays will generally provide a greater sensitivity, whereas MS uses very small sample volumes and samples can be stored and re-used at a later date. Collecting samples from wildlife can be difficult and expensive, so re-using samples has a significant advantage.

Caution should be used when comparing hormonal concentrations between laboratories. Recent studies have shown that there are considerable variations between immunoassay results from different laboratories but there is sufficient reproducibility within a laboratory to be able to compare hormone concentrations of individuals (Gail et al. 1996; McShane et al. 1996). In addition, comparative studies have been conducted to highlight the differences between immunoassays and mass spectrometry. It has been shown that although the absolute concentrations between immunoassay and mass spectrometry may differ for some hormones, the two methods showed similar estimates of between-subject differences in serum concentrations of most steroid hormones (Dorgan et al. 2002).

Although non-invasive techniques hold a good deal of merit for studying wildlife, there are some pitfalls. An ACTH stimulation test (Feldman et al. 1978) is the most reliable way of assessing adrenal function in a species rather than simply measuring cortisol concentrations from a single blood sample (Gulland et al. 1999). Also, faecal passage rates and urine retention/turnover rates will influence cortisol concentrations, as these rates are species specific. Without knowing a species' faecal passage rate or urine retention time, it is difficult to determine which samples (hourly, daily, 3-day intervals) are the most suitable to be used when determining cortisol concentrations in relation to specific events. However, if faecal samples, for example, are to be used to determine changes in cortisol concentrations in relation to specific events, then samples 2–3 days post the 'stressful' event should be used. If urine samples are to be used, then samples should be collected within 12 h of the stressful event. This is an evolving field, and recently other factors have been identified that can influence faecal analyses including body condition, diet of the animal, reproductive status, season, sample mass and sample storage and treatment techniques (Millspaugh and Washburn 2004).

If considering the use of non-invasive samples to monitor baseline stress in Antarctic seals, it is particularly important to understand the stability of those samples. Owing to climatic conditions in the Antarctic, it is not always feasible to visit different colonies on a daily basis. Caution should be used when collecting faecal samples that have been on the ice for a number of days. Recently it has been shown that steroid hormones, such as cortisol, are not stable in non-invasive samples as was previously thought (Khan et al. 2002; Hunt et al. 2003; Lynch et al. 2003; Hogg et al. 2005). There are a number of factors that can influence the stability of steroid hormones: (1) different weather conditions can affect hormonal concentrations in the samples prior to collection (Washburn and Millspaugh 2002); (2) the presence of bacteria (Hogg et al. 2005; Whembolua et al. 2006) and enzymatic activity (Hogg et al. 2005) can alter hormone concentrations; (3) repetitive freezing and thawing of samples can change hormonal concentrations (Chattoraj and Watts 1987); and (4) the length of storage can affect concentrations. Changes in hormonal concentrations may be improved with the use of suitable inhibitors, by prompt extraction of the samples and suitable long-term storage of extracts, such as at –80°C. If the above issues are not addressed, then the observed changes in hormone concentrations may be an artefact of environmental changes, storage time and storage temperature, rather than an indication of an animal's biological activity.

In conclusion, non-invasive sampling methods using faeces, urine or ice-urine are particularly useful when studying the baseline cortisol concentrations in Antarctic seals. Weather permitting, these sampling methods allow daily or weekly collection which can be used to describe the seasonal changes in cortisol concentrations, particularly for colonial species such as the Weddell seal. It also allows the easy determination of baseline cortisol concentrations, so that different environmental or anthropogenic influences can be assessed.

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Chapter 15
Sewage Disposal and Wildlife Health in Antarctica

J.J. Smith and M.J. Riddle

15.1 Introduction

Sewage and its microbiology, treatment and disposal are important to the topic of Antarctic wildlife health because disposal of untreated sewage effluent into the Antarctic marine environment is both allowed and commonplace. Human sewage contains enteric bacteria as normal flora, and has the potential to contain parasites, bacteria and viruses which may prove pathogenic to Antarctic wildlife. Treatment can reduce levels of micro-organisms in sewage effluent, but is not a requirement of the Environmental Protocol to the Antarctic Treaty (the Madrid Protocol). In contrast, the deliberate release of non-native organisms for any other reason is prohibited. Hence, disposal of sewage effluent to the marine environment is the only activity routinely undertaken in Antarctica knowing that it will likely result in the release of large numbers of potentially non-native species.

When the Madrid Protocol was negotiated, the decision to allow release of untreated sewage effluent was considered the only pragmatic option, as a prohibition would have been costly, and may not have been achievable by many Antarctic operators. In addition, at that time the potential for transmission of pathogens to wildlife from sewage was not emphasised as a significant potential risk. Since then, the transmission of disease-causing agents between species is more widely recognised and it is now timely to consider the risks of continued discharge of sewage effluent in Antarctica and whether there are practical alternatives.

In this chapter we describe sewage treatment technologies used in Antarctica both in the past and currently, we summarise the regulations governing sewage disposal in

J.J. Smith
Institute for Sustainable Resources, International Laboratory for Air Quality and Health, Faculty of Science Queensland University of Technology, Brisbane QLD 4001, Australia
e-mail: jj.smith@qut.edu.au

M.J. Riddle
Australian Antarctic Division, Channel Highway, Kingston TAS 7050, Australia
e-mail: martin.riddle@aad.gov.au

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Antarctica and discuss aspects of the Antarctic environment that may constrain the implementation in Antarctica of new sewage treatment technologies. We then summarise the potential environmental impacts of sewage effluent, discuss the documented extent of sewage and faecal contamination in Antarctica and review studies of the survivability of faecal micro-organisms in the Antarctic environment. We review the range of pathogens known to be commonly associated with sewage and discuss Antarctic-specific factors that might influence which pathogens are present in sewage from Antarctic operations. To assess whether there is a link between exposure to sewage effluent and microbial colonisation, infection, or virulence acquisition, we review reports of sewage-associated pathogens in Antarctic wildlife and of uptake of faecal bacteria. Finally, we consider whether there is any evidence for a link between exposure of wildlife to sewage and ill health.

15.1.1 Sewage Disposal from Antarctic Bases

15.1.1.1 Sewage and Wastewater

Consumption of food and water and production of the by-products of human metabolism are unavoidable, absolute requirements for human survival. As a general approximation, each human discharges about 1–1.5 l of urine and about 500 g of faeces per day dependent upon climate, activity and intake.

For the purposes of this discussion, the term *sewage* refers to both human waste products (faeces and urine) and domestic greywater (wastewater from kitchens, showers, etc.) because in most Antarctic bases they are combined and treated as a single wastewater stream. Elsewhere in the world, sewage treatment plants may process a broader range of wastewater, including industrial waste (solvents, metals, desalinisation brine, etc.) and surface runoff, as well as domestic greywater and human excretory products (US EPA 1992, 1999), bringing a greater range of potential environmental risks.

This chapter focuses on the microbiological aspects of sewage disposal and wastewater components most likely to harbour micro-organisms of animal- or public-health concern (AWWA 1999; Bitton 2005; Gerardi 2004; Leclerc et al. 2002; Long and Ashbolt 1994; Morris 2003; US EPA 1999; WHO 1999, 2003). A diseased or stressed state may also result from exposure of indigenous wildlife to wastewater components other than sewage, such as metals, synthetic organics and disinfection by-products. Additionally, stressed populations may be at increased risk of infection and disease by microbiological agents (Daszak et al. 2001). As micro-organisms in human sewage are known to cause a number of diseases in exposed human hosts, the potential exists for introduction of disease via exposure of wildlife to sewage (Daszak et al. 2001, 2004; WHO 2003).

Relative to temperate environments, Antarctic studies of the impact(s) of sewage-associated or human-waste-associated *micro-organisms* on indigenous flora and fauna are few, and largely focus on detection of sewage-associated bacteria, rather than association with disease. With the exception of a study of an accidental

exposure of captive Antarctic marine fauna to sewage in an aquarium (Meyer-Rochow 1992), no studies have shown a direct causal link between station-sewage micro-organisms and pathogenic effects and/or active infection of indigenous macrofauna. Indeed, recognition of microbiological pathogenesis (whether symptomatic or asymptomatic) remains largely unstudied for a variety of Antarctic wildlife.

Most microbiological studies of sewage disposal in Antarctica focus on issues related to protection of human health rather than indigenous biota (Boyd et al. 1972; Harker 1989; McFeters et al. 1993; O'Neill et al. 1968; Tzabar and Pennington 1991). Additionally, little evidence presently exists directly linking microbial agents of human infection and disease with a diseased or infected state in Antarctic wildlife. For this reason, the information presented here is largely based on sewage-associated microbial agents known to produce infection (with the possibility of producing a diseased state) in humans and recognised animal hosts.

So little is known of the interactions and infection/disease susceptibility of Antarctic wildlife to many, if not most, human pathogens that the potential risk to Antarctic biota from sewage-associated microbial agents cannot yet be precisely quantified. Hence, assessment of risk of introduction of disease into Antarctic wildlife via station sewage is limited by the lack of information for quantitative microbial risk assessments (QMRA) (Haas 2002; Westrell et al. 2004).

15.1.1.2 The Basics of Sewage Treatment and Disposal

Treatment of domestic sewage is generally a successive multi-step process employing physical, biological and chemical treatment steps that may be classified into several 'levels'. Primary treatment includes physical separation of large materials via grating or screening, and often additional settling of suspended solids. Secondary treatment uses aerobic or anaerobic biological processes to reduce organic and inorganic content and pathogen load (APHA, AWWA, WEF 1998; Bitton 2005; US EPA 1992, 1999). Tertiary treatments are physicochemical processes, which may include precipitation, coagulation, filtration and disinfection to further reduce levels of suspended solids, nutrients and viable micro-organisms.

Historically, the priority for sewage disposal has been physical removal from human proximity to eliminate odour and reduce the presence of disease vectors such as flies (US EPA 1992, 1999). The additional need to move sewage away from human settlements to minimise the possibility of infection of human and domesticated animals by the faecal–oral route became a priority after it was realised that sewage was a source of disease. The ultimate disposal site varies with local conditions, but includes land and aqueous disposal.

Aqueous environments, such as rivers lakes and the sea, are particularly attractive for disposal because of their capacity to absorb significant quantities of liquid and solid wastes, and their often flowing nature which both carries away and dilutes sewage. In general, liquid wastes have been disposed of in aqueous environments, and sludge or dried solids have been incinerated or disposed of on land, where soils have appropriate absorptive capacity and biological activity.

The ultimate goals of sewage treatment are reduction of biochemical oxygen demand (BOD), total suspended solids (TSS), total solids (TS) and numbers of viable pathogenic micro-organisms. Significant reductions in all these parameters may be achieved through primary treatment (screening and settling) and removal of sludges (US EPA 1992, 1999). Drying, anaerobic digestion and composting of sludges can all lead to further reductions (USEPA 1999), although effluent quality is highly dependent on the type of system and how well it is operated. Further BOD, TSS and pathogen reductions in the remaining liquid sewage fraction are achieved through stimulation of aerobic heterotrophic microbial oxidation of sewage organics and competitive die-off of many pathogens, typically aided by active or passive aeration (Bitton 2005; Gerardi 2004).

In some processes, conditions are manipulated so that bacteria form aggregates which flocculate both biotic and abiotic particulates. These settle, or are filtered or screened, leading to further reductions in pathogen, TSS and BOD levels. Composting processes make use of a succession of largely aerobic heterotrophic micro-organisms, in which mesophilic bacteria are succeeded by thermophilic bacteria and fungi (largely streptomycetes and actinomycetes). During these processes, temperatures of 70°C may be obtained, killing many non-spore-forming pathogenic micro-organisms and reducing moisture content (Bitton 2005).

15.1.1.3 History of Sewage Disposal in Antarctica

Early Antarctic explorers disposed of shipboard human wastes overboard, while terrestrial huts were generally equipped with latrines that were emptied at sea or with pit latrines in which wastes were ultimately buried. These practices continued until the 1960s, with sewage and wastewater routinely buried or discharged untreated either into ice pits, the near-shore marine environment (when ice-free), or set upon seasonal sea-ice to be carried away (Bleasel et al. 1989; Boyd et al. 1972; Boyd and Boyd 1963; Holmes et al. 1983; Tyler 1972). The goal of these techniques was localised containment, minimisation of human contact and ‘out-of-sight’ disposal for aesthetic and sanitary purposes.

More recently, increasing station populations and commensurate increases in sewage and wastewater production have necessitated the installation of piped continuous discharges at some bases (ASCE 1989; Arcone et al. 1994; Huh et al. 1989; Lee and Oh 1997; Lori et al. 1993; Redvers 2000; Reed and Sletton 1889). At coastal bases these were, and largely remain, typically near-shore surface discharges onto the sea-ice or the intertidal zone (ASCE 1989; Bleasel et al. 1989; Bruni 1992; Holmes et al. 1983; Hughes 2004; Huh et al. 1989; Lee and Oh 1997; Lori et al. 1993; Redvers 2000; Reed and Sletton 1989). Inland bases and field camps have typically discharged wastewater and sewage into ice pits, subsurface ice-wells or nearby lakes (Bou et al. 1996; Ellis-Evans et al. 1997; Kryzyszowska 1991, 1993; Mellor 1969; Tyler 1972). More recently, waste from remote field camps has been collected for treatment and disposal at main bases or for transport out of Antarctica (ASCE 1989; Arcone et al. 1994; Bou et al. 1996; Flynn and Bubenheim 1997; Holmes et al. 1983; Ishizawa and Takahashi 1990; Nakawo 1985; Reed and Sletton 1989) (Table 15.1).

Table 15.1 Wastewater quantities and treatment and disposal practices at various Antarctic bases as of December 2003

Station/Base	[a]Population	[b]Quantity of waste-water generated per day (L)	Food waste included?	Treatment	Discharge	Method of solids or sludge disposal	Monitoring
ABOA (Finland)	10–15 (mid-Nov –mid Feb)	300–500 greywater, sewage shipped out	No	Greywater-biological, urine-evaporated	Greywater-snow/ ground surface	Shipped out	Starting 2003–04
Syowa (Japan)	110/40	4 400–12 100	No	Contact aeration biological	Submerged marine outfall	Incinerated	BOD, TSS, pH monthly
King Sejong (S. Korea)	70/16	<5 000	Yes	Biological (aeration), settling, chemical (HOCl)	Near-shore marine into tidal zone	Shipped out (sludge)	BOD, TSS, faecal coliform
Henryk Arctowski (Poland)	12–15	1 000	No	Settling and biological	Near-shore marine	Incinerated	None
Sanae (S. Africa)	60–80/9	2 000–15 000	Yes	Biological, UV disinfection	Surface	Shipped out (sludge)	NH3, nitrates, pH, COD, colour
Wasa (Sweden)	10–20	500–1 000	No	Settling, freeze-drying	Surface (ice-pit) greywater only	Shipped out	Periodic BOD, TSS
Academic Vernadsky (Ukraine)	15–30/13–15	2 000	Yes	None	Near-shore marine into tidal zone	Shipped out	Periodic
Rothera (UK)	140/22	15 000	Yes	Screening/settling, submerged aerated-biological filter, UV disinfection	High water marine	Dewatered and shipped out	Faecal coliform
McMurdo (US)	1 150/150	40 000–270 000	No	Aerobic, activated sludge secondary treatment plant, UV disinfection	Submerged marine	Shipped out for incineration	BOD, TSS, faecal coliform or faecal enterococci

(continued)

Table 15.1 (continued)

Station/Base	[a]Population	[b]Quantity of waste-water generated per day (L)	Food waste included?	Treatment	Discharge	Method of solids or sludge disposal	Monitoring
Palmer (US)	17–44	100 000–200 000	Yes (no raw poultry or eggs)	Macerated	Near-shore	BOD, TSS, NH_3 (periodic)	
Scott (New Zealand)	86/10–12	70 000	Yes	Biological (aerated submerged media)	Ocean surface	Shipped out	Monthly Faecal coliform, BOD, TSS
Mirny (Russia)	Unknown	1 000–2 000	Unknown	Unknown	Ice-shelf going to sea	Unknown	Unknown
Progress 2 (Russia)	30	4 000	Unknown	Electric impulse	Sea (Bukhta)	Unknown	Unknown
Casey (Australia)	80–100/20	1 600–4 300	No	[c]RBC	Near-shore marine	Sludge shipped out	BOD, TSS
Mawson (Australia)	30–35/20	2 900–7 600	No	[c]RBC	Near-shore marine	Sludge shipped out	BOD, TSS
Davis (Australia)	80–100/20	2 500–7 500	No	[c]RBC decommissioned 2005/06. Currently no treatment	Near-shore marine	Sludge shipped out	BOD, TSS
Terra Nova (Italy)	Up to ca. 173	Unknown	No	Unknown secondary	Near-shore marine	Filtered, pressed sludge shipped to Italy	Unknown
Gondwana (Germany)	Unknown	Unknown	No	Single chamber, activated sludge	Macerated, near-shore marine	Sludge + food waste dried and shipped out	BOD, TSS

Dumont D'Urville (France)	60/25	Unknown	No	Collected, compressed	Shipped out	Shipped out	None
Port-aux-Francais (France)	200/60	Unknown	No	Incinerated	Shipped out	Shipped out	None
Troll (Norway)	5–30 (summer only, considering year-round)	200	No	Composting toilets, greywater filtered	Incinerated	Ashes + food waste shipped out	None

[a]Station population summer/winter, where applicable.
[b]Note: Data for Davis station discharge based on monitoring of flow rate through effluent pump, most other station values based on potable water production/usage
[c]RBC = Rotating Biological Contactor.

Sewage treatment is a more recent phenomenon, with most stations now having installed, or planning to install, wastewater or sewage treatment systems (Bleasel et al. 1989; Flynn and Bubenheim 1997; Holmes et al. 1983; Hughes and Blenkharn 2003; Hughes 2004; Lee and Oh 1997; Lori et al. 1993; NSF 1990; Stephan 1991) (Table 15.1). This has largely been brought about by increased awareness of the need to protect the environmental values of Antarctica and recognition of localised adverse effects on indigenous biota, which in turn led to international agreement on the need for better environmental management in Antarctica and subsequently to obligations under the Antarctic Treaty System (Coughlin 1998; Greenpeace USA 1990). Improved practices have been made possible by the development and availability of logistically feasible, small-scale wastewater treatment technologies (Bou et al. 1996; Hughes 2004; Meyer-Rochow 1999; NSF 1990; Redvers 2000; US EPA 1992).

15.1.1.4 Regulations Governing Sewage Disposal in Antarctica

The disposal of sewage is now largely regulated under Annex III of the Protocol on Environmental Protection to the Antarctic Treaty (the 'Madrid Protocol'). Aspects of wastewater and sewage disposal are primarily dealt with in the following articles:

'*Article 2 (Waste disposal by removal from the Antarctic Treaty area)*

3. The following wastes shall be removed from the Antarctic Treaty area by the generator of such wastes, unless incinerated, autoclaved or otherwise treated to be made sterile:

   (a) Residues of carcasses of imported animals;
   (b) Laboratory culture of micro-organisms and plant pathogens; and
   (c) Introduced avian products.

Article 4 (Other waste disposal on land)

1. Wastes not removed or disposed of in accordance with Articles 2 and 3 shall not be disposed of onto ice-free areas or into fresh water systems.
2. Sewage, domestic liquid wastes and other liquid wastes not removed from the Antarctic Treaty area in accordance with Article 2, shall, to the maximum extent practicable, not be disposed of onto sea-ice, ice shelves or the grounded ice-sheet, provided that such wastes which are generated by stations located inland on ice shelves or on the grounded ice-sheet may be disposed of in deep ice pits where such disposal is the only practicable option. Such pits shall not be located on known ice-flow lines which terminate at ice-free areas or in areas of high ablation.
3. Wastes generated at field camps shall, to the maximum extent practicable, be removed by the generator of such wastes to supporting stations or ships for disposal in accordance with this Annex.

Article 5 (Disposal of waste in the sea)

1. Sewage and domestic liquid wastes may be discharged directly into the sea, taking into account the assimilative capacity of the receiving marine environment and provided that:

(a) such discharge is located, wherever practicable, where conditions exist for initial dilution and rapid dispersal; and (b) large quantities of such wastes (generated in a station where the average weekly occupancy over the summer is approximately 30 individuals or more) shall be treated at least by maceration.

2. The by-product of sewage treatment by the Rotary Biological Contacter process or similar processes may be disposed of into the sea provided that such disposal does not adversely affect the local environment, and provided also that any such disposal at sea shall be in accordance with Annex IV to the Protocol.'

Environmental impact monitoring is regulated under Article 3, section 2:

'(d) Regular and effective monitoring shall take place to allow assessment of the impacts of ongoing activities, including the verification of predicted impacts;

(e) Regular and effective monitoring shall take place to facilitate early detection of the possible unforeseen effects of activities carried on both within and outside the Antarctic Treaty area on the Antarctic environment and dependent and associated ecosystems.'

Article 3, section 2 also required the phasing out of open burning of wastes by the end of the 1998/99 field season.

These regulations effectively limit the available options for sewage treatment and disposal in Antarctica. Wastewater disposal is prohibited in freshwater environments or ice-free areas. Therefore, terrestrial sewage and wastewater disposal at in-land locations is restricted to ice pits and the ice subsurface, while disposal of sewage into aqueous environments is effectively restricted to marine discharge, and then only in locations where conditions exist for initial dilution and rapid dispersal.

As described above, the Treaty specifically identifies the need for regular and effective monitoring for assessment of the adverse environmental impacts of ongoing activities such as disposal of sewage effluent. For monitoring to be effective in testing predicted impacts, it is essential that it is directed towards measuring the impacts rather than simply recording the amount of treated or untreated effluent discharged. Microbiological analysis parameters comprise an important component of wastewater environmental impact monitoring schemes (APHA 2005; COMNAP 2005; EPHC/NRMMC 2005; US EPA 1992, 1999). However, microbiological monitoring of sewage discharges is not currently performed at all Antarctic stations or by tourist vessels (COMNAP 2005). Such data would be useful not only for monitoring environmental impact, but also for assessing treatment efficacy of different systems for review by all Antarctic operators. Additionally, microbiological data is essential as the basis for meaningful assessments of the risk of disease introduction to indigenous wildlife (Haas 2002; Westrell et al. 2004).

15.1.1.5 Current Sewage Management Practices in Antarctica

The most common forms of primary treatment at Antarctic stations are maceration or comminuation, screening and settling (Table 15.1). Aerobic biological secondary

treatment is the most common technology and is used at a number of stations. Formats include trickling filter, rotating biological contactor, fixed-media aeration, activated sludge and aerated submerged media processes.

At some coastal stations untreated or macerated sewage is still discharged directly to the sea (Table 15.1). Discharge points may either be submerged or above sea level. Some stations and field camps located on inland ice shelves, grounded ice sheets or permanent snow fields discharge wastewater untreated into ice pits or sub-surface boreholes, although small inland field camps are required (to the extent possible) to collect and transport human wastes back to main station facilities for disposal with station sewage. Shipboard wastewater (including food wastes) can also be discharged untreated other than grinding/maceration, but must be discharged more than 12 n miles from shore for vessels carrying more than 10 people (Protocol 1991; Knox et al. 2001; Harris et al. 2001).

At many Antarctic stations, kitchen and food wastes, particularly poultry and poultry products, are separated to prevent them from entering the wastewater stream (Table 15.1). The removal of poultry products complies with Annex III, Article 2 of the Madrid Protocol, which was intended to reduce the risk of introduction of pathogenic agents associated with animal products, such as Newcastle disease (avian paramyxovirus). As an additional benefit, removal of food waste reduces levels of nutrients and solids requiring treatment and/or discharge.

The cost and complexity of tertiary treatment are generally prohibitive for routine use at some Antarctic stations. However, disinfection of secondary effluent prior to discharge is currently being used in some locations. The most common form of effluent disinfection is ultraviolet (UV) irradiation (McMurdo, Rothera, Sanae) and chlorination (King Sejong) (Table 15.1).

Most sewage treatment processes ultimately result in both liquid and solid waste. One of the primary goals of biological treatment is reduction of solids. Solids reductions of 70–95% in efficiently operating systems are not uncommon (Bitton 2005; Morris 2003; US EPA 1992, 1999); however, some solids remain. At most stations solids are separated, often dewatered, and stored for shipment out of Antarctica for ultimate disposal. While awaiting shipment, these solids must be stored in such a way as to restrict access by indigenous fauna (Burger 1981; Müller-Schwartze et al. 1978). At some smaller stations such as Aboa (Finland) and Wasa (Sweden), urine is separated at source and the liquid fraction is evaporated or freeze-dried after settling. This latter approach is a low-energy method for reducing sewage bulk and is suitable for use at smaller stations (Sanin et al. 1994).

The largest wastewater treatment facility in Antarctica is at McMurdo Station (USA). It consists of a 457 kL/day capacity secondary activated sludge treatment facility with associated sludge dewatering and UV disinfection of the secondary-treated effluent. Additional features include raw sewage storage tanks for continuous flow regulation, and two Muffin Monster® grinder units to macerate pre-treatment solids. Prior to discharge, treated wastewater is mixed with seawater from the flow-through marine aquarium at ambient temperature (ca. –1.8°C) and with reject brine from the reverse osmosis drinking-water plant. The discharge point is about 50 m from shore at 17 m depth. To limit damage to the discharge pipe from seasonal

sea-ice, the pipe runs through a reinforced earthen quay. Dewatered sludge is shipped back to the USA for incineration.

Scott Base (NZ) operates an aerated, submerged-media biological treatment system, with the sludge shipped back to New Zealand for disposal (Harris et al. 2001; Redvers 2000). Rotating biological contactor secondary treatment systems are used at the three Australian stations (Casey, Mawson, Davis).

15.1.1.6 Practical Constraints on Sewage Treatment and Disposal in Antarctica

Sewage treatment in the Antarctic environment presents several particular challenges (Bleasel et al. 1989; McAneney 1998; Mellor 1969; Reed and Sletton 1989), many of which are caused by the low ambient temperatures.

Low temperatures reduce the efficiency of biological treatment (Bitton 2005; Mara 2003; McAneny 1998), and as a consequence heated facilities must be allocated for treatment equipment such as holding tanks, pumps and solids handling. Treatment also requires varying amounts of energy for heat, pumps (particularly for actively aerated systems) and control and ancillary equipment. Treatment facilities and equipment must also be isolated from general living and working quarters for sanitary and odour-control purposes. Additionally, insulated and possibly heated wastewater transfer lines are required. Large seasonal variations in station populations may also require significant adjustments to treatment parameters, particularly for those biological processes affected by large fluctuations in nutrient loading. Treatment problems related to such fluctations have been experienced at Terra Nova (Lori et al. 1993) and Casey Stations.

The formation of sea-ice creates difficulties for the disposal of effluent to the marine environment. The breakout of seasonal fast-ice and the scouring effects of icebergs and pack-ice make permanent submerged discharge points difficult to maintain (Bleasel et al. 1989; Holmes et al. 1983). Therefore, at some locations disposal is through pipes that terminate above sea level and effluent is discharged onto ice-cliffs (e.g. Casey), onto the shoreline above the sea-ice (e.g. Davis) or onto the sea-ice at pressure ridges and tidal cracks. Discharge above the ice in this way limits dispersion, as it must first permeate through the ice which tends to channel effluents, and may cause aesthetic and odour problems (McFeters et al. 1993; Redvers 2000.)

In temperate environments, small wastewater systems often employ open settling and oxidation ponds as forms of primary and secondary treatment (Bitton 2005; Mara 2003; US EPA 1992). The low mean temperatures at higher latitudes in Antarctica effectively rule out such systems because of freezing and logistical problems (McAnaney 1998). Open systems might be possible in the warmer, more northerly parts of the Antarctic Peninsula or on sub-Antarctic islands, but if used would create the possibility of direct access by indigenous wildlife, primarily birds (Burger 1981; Müller-Schwartze et al. 1978). Indeed, disposal of liquid or solid wastes in any manner that leaves them open to the environment increases the risk

of transmission of infectious diseases to wildlife. Similarly, standard septic systems with subsurface leach fields would face problems of restricted penetration of the leachate through permafrost layers and possible channelling and pooling (US EPA 1992). Both these technologies are effectively prohibited in the Antarctic Treaty Area because of the ban on the disposal sewage onto ice-free areas included in the Madrid Protocol.

Chlorine-disinfection processes are commonly used in other regions but they may produce chlorine residuals (both free and combined) which are toxic to aquatic organisms at low concentrations. Low temperatures, ice cover, and seasonal low light intensities also decrease rates of oxidation of the residual chlorine after discharge. Additionally, certain chlorination by-products formed during wastewater disinfection (trihalomethanes [THMs], chlorophenols, etc.) are carcinogenic and have been associated with significant adverse environmental effects (Bull et al. 1995; Leenheer et al. 2001; Stewart et al. 1996; Szal et al. 1991). The continual need to supply chlorination agents adds to the logistics of operating in Antarctica, although free chlorine could be generated electrolytically in wastewater streams after mixing with seawater.

Commercially available UV wastewater disinfection systems have been used for disinfection of secondary treated waste at some stations (McMurdo, Rothera, Sanae, Neumayer). However, the technology has not always been successfully applied. The failure of a recent UV-treatment trial at Casey Station was attributable to engineering factors rather than intrinsic problems associated with use of the technology in Antarctica. The flow rate used was too high, resulting in too short an exposure time, and the path length and turbidity too great, leading to insufficient UV exposure.

15.1.1.7 Future Developments

The increasing availability of efficient, reduced-maintenance, small-scale wastewater treatment technologies such as single and multi-chamber activated sludge, trickling filter, rotating biological contactors, etc. will likely continue to make the logistics and costs of more advanced and efficient sewage treatment more feasible. Exchange of information on new technologies is actively encouraged among the international Antarctic community, with the report of the XIVth meeting (1987) of the Antarctic Treaty Consultative Meeting (ATCM) suggesting that ‘Information on new and improved methods of waste disposal should be exchanged between national operating agencies, and their implementation and application should be encouraged’.

Static pile or in-vessel composting may be an attractive option for stations with smaller population owing to low capital costs and ease of maintenance. However, odour control through maintenance of uniform aerobic conditions in static piles (mixing and turning) and use of negative pressure with odour control practices are important for wastewater sludge composting (Bitton 2005; US EPA 1999). In addition, separation of urine is desirable in composting toilets, as it provides greater control over compost moisture. Considering the low ambient temperatures at many stations, the use

of ambient freezing and subsequent sublimation of solids, as well as source-separated liquids, may provide an effective alternative for reduction of sewage volume (Parker et al. 2000; Sanin et al. 1994). However, issues of containment and bioaerosol generation require investigation (Hughes 2006; Shuval et al. 1989, see below).

15.1.2 Environmental Impacts of Sewage Effluent

Sewage can impact the environment in a number of ways. Impacts include the aesthetic nuisance created by the sight and smell of effluent, the physical effects of releasing large quantities of particulate material, chemical effects of constituents of sewage such as nutrients and organic material, and the introduction of living, and potentially infectious or invasive, micro-organisms.

15.1.2.1 Physical and Aesthetic Impacts

The sight and smell of sewage has obvious aesthetic impacts and is the reason why removal from human contact is the minimum treatment for most societies, including Antarctic stations. It is the impact most likely to generate a remedial response because it is the most easily detected.

Large quantities of particulates in sewage can smother aquatic organisms (Lenihan et al. 1990, 1995; Long and Ashbolt 1994) and reduce light available for photosynthesis, particularly if discharged in still water bodies such as lakes or sheltered coastal locations. Limiting discharge of effluent to the sea to those locations where conditions exist for initial dilution and rapid dispersal, as stipulated in the Madrid Protocol, will go some way to preventing smothering. Some level of particulate reduction is achieved by settling of suspended solids, which is commonly part of even the simplest sewage treatment processes.

15.1.2.2 Chemical Impacts

Wastewater is a complex mixture containing many potentially toxic chemical components, such as metals, synthetic organics, estrogens, disinfection by-products, etc. (APHA 1998; Bickford 1996; Purdom et al. 1994) as well as human waste products, which may lead to reduced biological diversity or productivity near wastewater outfalls (Anderson and Chagué-Goff 1996; Bickford 1996; Crockett 1997; Ferguson et al. 1996; Lenihan et al. 1990). In addition, organisms stressed by chemical contaminants may be at increased risk of infection and disease by microbiological agents. Important differences between exposure to biological disease agents, as opposed to chemical stressors, include the capability of the former for *in vivo* (and possibly *in situ* environmental) amplification of the agent through replication.

Several studies have correlated a decrease in diversity of benthic in-fauna in the vicinity of an Antarctic station with concentrations of petroleum hydrocarbons and metals in sediments, although it was suggested that this was largely due to non-sewage-related sources (Conlan et al. 2004; Lenihan et al. 1995). Mortality of the amphipod *Heterophoxus videns* was high in 28-day bioassay exposures to sediments from near the McMurdo Station sewage outfall and to those from Winter Quarters Bay, compared to relatively uncontaminated sediments (Lenihan et al. 1995). Increased levels of petroleum hydrocarbons have been found in marine sediments in the vicinity of Davis Station (Green and Nichols 1995; Green et al. 1992), and increased levels of metals have been found to be centered around the sewage outfall at McMurdo Station (Anderson and Chagué-Goff 1996). Although the sources of the contaminants were postulated to be previous fuel spills, dumping and possibly some wastewater input, these studies illustrate the importance of preventing these wastes from entering wastewater discharge streams.

The high levels of nutrients in sewage can upset the balance of ecosystems, particularly those that are naturally nutrient-limited, a common characteristic of both Antarctic terrestrial and lake systems (Ellis-Evans et al. 1997). High levels of organic material in sewage have the capacity to consume oxygen from water (termed biological oxygen demand, BOD) largely due to respiration by heterotrophic bacteria, and can be detrimental to aquatic systems by reducing dissolved oxygen to levels that are insufficient to sustain life (APHA 2005; Bitton 2005; USEPA 1992). In a study of BOD of discharged sewage at *in situ* temperatures (−1.8°C) at McMurdo Station, Howington et al. (1994) found that rates of oxygen uptake were approximately 3-fold lower than at the standard test temperature of 20°C. This suggests that while oxygen limitation in mixed receiving water columns is unlikely, oxidation rates of organic material are significantly reduced. This may result in localised acute benthic impacts caused by the high BOD creating oxygen limitation in settled solids. Both nutrient levels and BOD can be reduced by settling, and most of the more advanced treatment technologies are designed to further reduce them (Bitton 2005; Gerardi 2004; US EPA 1999).

The accumulation of organic material from sewage in marine environments can create anaerobic conditions, largely due to heterotrophic microbial activity and associated oxygen consumption. This is typically associated with settling and accumulation of effluent solids over existing benthic environs. The resultant anoxic reducing environment can lead to microbially mediated sulphate reduction and concomitant generation of reduced sulphur compounds such as H_2S and mercaptans (Bickford 1996). Indeed, the presence of a microbial mat of *Beggiatoa* sp. (a chemolithotrophic sulphur-oxidising bacterium) on top of the zone of settled solids proximal to the McMurdo Station outfall suggests a source of reduced sulphur compounds (AWWA 1999; Lenihan et al. 1990, author's (Smith) unpublished data). These compounds can prove toxic to marine invertebrates and can inhibit their growth. Mitchell and Chet (1975) found evidence of both *Beggiatoa* and *Desulfovibrio* sp. involved in the destruction of stressed corals (*Platigyra* sp.), while Campos et al. (2006) noted possible wastewater enrichment of sub-Antarctic methanogenic microbial communities near Commandante Ferraz Station (Admiralty Bay, King George Island, South Shetlands).

The fatal effects of raw sewage on Antarctic benthic invertebrates (nemertean worms *Parborlasia corrugatus,* starfish *Diplasterias brucei*, *Perknaster* sp., sea spiders *Colossendeis* sp., *Ammotheo* sp.) and two fish species, *Pagothenia borchgrevinki, Trematomas bernacchii*, were observed when the accidental introduction of sewage to an aquarium resulted in the survival of only the giant Antarctic slater *Glyptonotus antarcticus* (Meyer-Rochow 1992). Negative effects and mortality were evident within 10 min. of exposure. Analysis of aquarium water found high levels of total nitrogen (12.2 mg l^{-1}), phosphorus (2.15 mg l^{-1}), BOD (21.5 mg l^{-1}) and faecal coliforms (>35,000 per 100 ml). Although thermal and/or osmotic stress could not be ruled out, and the exact cause of mortality could not be determined, the acute effects of short-term gross exposure to raw sewage on Antarctic marine fauna were evident.

The presence of chemicals in sewage that exert estrogenic or androgenic effects in exposed wildlife has become of growing concern in recent years. Commonly referred to as endocrine-disrupting compounds (EDCs), they have been associated with developmental and reproductive abnormalities in a variety of aquatic wildlife, including marine fishes and invertebrates (Deplege and Billinghurst 1999; Matthiessen 2003; Purdom et al. 1994). The most commonly reported EDCs are estrogenic reproductive steroid hormones and biodegradation products of alkylphenol ethoxylate surfactants (APEs, common in cleaning products, shampoos, household cleaners, contraceptives, etc.) The effects of EDCs in Antarctica have been very little studied. EDCs are particularly harmful to reproductive and early development phases, and because many fish and invertebrate species reproduce and undergo early development in the nearshore marine environment in Antarctica, it is possible that they could exert detrimental impacts. If EDCs do prove to be a major concern in Antarctica, they will be difficult to address, as advanced tertiary treatments such as reverse osmosis are required to effectively remove them.

15.1.2.3 Biological Impacts

The most significant potential environmental impact that could be caused by sewage effluent disposal, and among the most significant impacts by any cause in Antarctica, is the introduction and establishment of a serious disease to wildlife. In later sections we discuss the possibility of disease transmission by this route. However, disease is not the only impact that could be caused by introduced micro-organisms from sewage effluent. Even if the introduced micro-organisms do not cause any further detrimental effect beyond their own survival and establishment, that alone would be an environmental impact and would be contrary to the principles of the Madrid Protocol were it not for the specific exemption which allows disposal of sewage effluent.

Sewage and wastewater represent the largest and most obvious single-point sources of anthropogenically derived micro-organisms, but there are others. People carry with them and disperse their own microflora wherever they go, and as a consequence the human microflora are also found associated with vehicles, vessels, food, field equipment and clothing. Frenot et al. (2005) have suggested mitigation of such microbial propagules to decrease the risk of ecosystem disturbance, including spread of infectious agents to indigenous wildlife, whether macro or micro flora.

Antarctica harbours some of the earth's most isolated and unique microbial and plant communities, including unusual cryptoendolithic, geothermal, subglacial, as well as meromictic- and hypersaline-lake communities, mosses, liverworts, lichens and macrofungi (Fitzsimons et al. 2001). The effects of introducing anthropogenic microbial contaminants to Antarctica are almost wholly unknown and unexplored (Cowan and Lemese 2004), and the tools to even recognise the changes to indigenous microbial communities caused by introductions are only recently becoming available. New molecular biological analytical techniques for surveying microbial communities (Ah Tow and Cowan 2005; Deutschbauer et al. 2006; Xu 2006) and ecotoxicogenomics (Snape et al. 2004) provide valuable tools for studying possible anthropogenic impacts on these organisms and communities.

Non-microbial species introductions have also been associated with Antarctic sewage treatment facilities. Black fungus midges (*Lycoriella* spp.) have been found to be breeding in the Casey Station (Australia) sewage facilities (Hughes et al. 2005). Although these are not expected to survive outdoors in continental Antarctica, their presence associated with sewage and their reported ability to transmit fungal plant pathogens is of concern, particularly for sub-Antarctic stations where their survival outdoors is more likely.

It is possible that sewage may contain bacteria harbouring genes encoding virulence, such as toxin production, or antibiotic resistance factors, as has been found in temperate environments (Fontaine and Hoadley 1976; Kruse and Sørum 1994). Antibiotic-resistant strains are introduced to sewage from populations that have been exposed to antibiotics, such as humans treated with antibiotics for medical reasons and domestic animals given antibiotics for medical purposes and to increase productivity. Antibiotic-resistant strains can pass through treatment facilities; for example, secondary treatment of sewage using an aerobic lagoon without subsequent disinfection did not significantly reduce numbers of antibiotic-resistant faecal coliforms (Bell 1978). However, treatment and/or disinfection of sewage will reduce the numbers of *viable* sewage-derived potential donor bacteria, and hence reduce the rates of possible *conjugative* genetic transfer in the environment, which requires viable cells of both donors and recipients.

Genetic elements for antibiotic resistance or other characteristics may also be transferred between bacteria of diverse genera through transformation, conjugation or transduction (Arvanitidou et al. 1997; Lorenz and Wackernagel 1994; Young 1993). The transfer of non-native genetic elements into native microbial populations has been termed 'genetic pollution' (Gleckman and Madoff 1969; Paul et al. 1991; Anderson and Sandaa 1994). Genetic exchanges may transfer infectivity to a bacterium that was not previously pathogenic for a particular host (Goodman et al. 1993; Lorenz and Wackernagel 1994), or enhance the pathogenicity or infectivity of an autochthonous bacterial agent. Several studies have shown that natural genetic exchange takes place in marine environments, and that antibiotic resistance plasmids (extra-chromosomal DNA capable of replicating independently of the chromosomal DNA) are transferred from enteric (such as *Escherichia coli*) to marine bacteria (*Vibrio* spp.) as well as fish pathogenic species (*Aeromonas salmonicida*) (Goodman et al. 1993; Kruse and Sørum 1994; Paul et al. 1991).

Plasmids have been found in naturally occurring psychrophilic and psychrotrophic bacteria in McMurdo Sound (Kobori et al. 1984), with the highest percentage of plasmid-harbouring isolates from surface-associated environments (sediments, sea-ice, animals). Additionally, isolates containing plasmids conferring antibiotic resistance were all derived from sediments. No differences in plasmid incidence were noted between sites proximal and remote from McMurdo Station and Scott Base. Although it was concluded that plasmids are ubiquitous in autochthonous Antarctic bacteria (Kobori et al. 1984), mating and transformation experiments between indigenous bacteria that contain ampicillin resistance plasmids and *E. coli* were unsuccessful. Ray et al. (1991) found plasmids in 10 of 31 isolates belonging to the genera *Pseudomonas*, *Arthrobacter*, *Flavobacterium*, *Planococcus* and *Micrococcus* in soils of the Shirmacher Oasis (Queen Maud Land), including some *Flavobacterium* spp. isolates containing antibiotic resistance plasmids (R-plasmids). Smith et al. (1993) found that the faecal indicator bacterium *E. coli* maintained and was capable of expression of antibiotic resistance (R) and conjugative (F) plasmids during long-term exposure to the Antarctic marine environment.

In contrast to the relative frequency of antibiotic resistance in isolates from soils and sediments, there are few, if any, records of antibiotic resistant isolates from Antarctic animals. A study of antibiotic resistance profiles of intestinal bacteria isolated from Weddell seals, *Leptonychotes weddellii*, Adélie penguins, *Pygoscelis adeliae*, and emperor penguins, *Aptenodytes forsteri*, and local fish in the vicinity of the McMurdo Station sewage outfall, prior to installation of the treatment plant, did not find any antibiotic-resistant isolates in fauna (Howington et al. 1993) despite finding large numbers of antibiotic-resistant isolates in sewage from the station. Similarly, Palmgren et al. (2000) did not find antibiotic resistance in any of 50 *Salmonella* spp. isolates from Bird Island seabirds and fur seals. These studies suggest a lack of antibiotic-resistant bacteria in these animals and imply that they have not been colonised or infected by bacteria from the sewage. Alternatively, the absence of *in vivo* antibiotic-induced selective pressure may select against antibiotic-resistant strains in these animals.

Most studies of environmental transfer between species have focused on transfer of antibiotic resistance between bacteria, with few studies on the environmental transfer of virulence factors, and to our knowledge none has occurred in polar regions. Therefore, although the possibility exists for genetic exchange between sewage-derived bacteria and those in the Antarctic marine environment, this has yet to be conclusively demonstrated.

15.1.3 Environmental Indicators of Sewage and Faecal Material

Before considering the extent of sewage contamination in Antarctica, we will briefly review methods used for tracking environmental contamination by sewage. Meays et al. (2004) provide a more thorough review.

15.1.4 Indicators Based on Microbial Culturing Techniques

Most studies of faecal contamination in the environment use indicator micro-organisms, as direct measurement of the wide variety of individual pathogens is impractical using conventional culturing techniques. In addition, by focusing on individual pathogens, others of significance may be missed. Criteria for good indicator organisms are that they should (1) be universally present in large numbers in the faeces of humans and warm-blooded animals, (2) be readily detected by simple methods, (3) not be present in natural waters, (4) exhibit similar rates of inactivation by sewage treatment as pathogens, and (5) exhibit similar persistence in the environment as pathogens. Bacterial indicators are widely used for microbiological assessment of water used for drinking, recreational activities and seafood (particularly shellfish) harvesting (EC 1991; NSSP 1999; WHO 2003). Limits are based on indicator dose/disease response relationships from human epidemiological studies (Fleisher et al. 1998; Prüss 1998) and are unlikely to include data on disease risk to wildlife.

The most commonly used indicators of faecal pollution in near-shore marine environments are faecal and thermotolerant coliforms, *E. coli*, or enterococci (APHA 2005; Mara 2003; WHO 2003). Although total coliforms and faecal coliforms have been used as standard indicators of faecal pollution for many years, studies have demonstrated a number of deficiencies in their use as indicator organisms in marine waters compared to enterococci (Cabelli et al. 1982; Noble et al. 2003). Some epidemiological studies, for example, have shown poorer relationships between faecal coliform densities and illness rates in bathers than those obtained using enterococci (WHO 1999; Cabelli et al. 1982). Hence, microbiological monitoring guidelines generally recommend enterococci for marine recreational waters (APHA 2005; WHO 2003).

In some cases, indicators may be used as surrogates for specific classes of pathogens. The spore-forming sulphite-reducing *Clostridium perfringens* is commonly used as a surrogate for encysted protozoan parasites such as *Giardia* and *Cryptosporidium* in sediments (Constantina and Yanko 2001; Fujioka 2001; Hill et al. 1993; Payment et al. 1985; Payment and Franco 1993; Brookes et al. 2005). Somatic (DNA) and male-specific (F+) RNA coliphage have also been used as surrogates for enteric viruses; however, detection of coliphage in human sewage can be inconsistent (IAWPRC 1991; Lasobras et al. 1999; Leclerc et al. 2000; Payment and Franco 1993; Brookes et al. 2005) and the F+ coliphage is not particularly host specific (Schaper et al. 2002).

Several techniques have been proposed to distinguish human and non-human sources of faecal pollution, such as the ratio of faecal coliforms (FC) to faecal streptococci (FS) (Feachem 1974) and multiple antibiotic resistance (MAR) profiles. MAR is a relatively new method based on the observation that bacteria from wildlife species are generally lacking in antibiotic resistance, while strains from humans and domestic animals exhibit varying antibiotic resistance (Parveen et al. 1997; Harwood et al. 2000). FC/FS ratios have also been used to distinguish between human and animal faecal sources. Generally, FC/FS ratios of ≥ 4 are indicative of human faeces, whereas ratios of < 0.7 are indicative of animal wastes. However, as FS generally survive longer than FC, this ratio can be expected to change over time. The use of FC/FS

ratios is therefore discouraged unless data are derived from a site proximal to a source of contamination and within hours of the pollution discharge (Geldreich 1976).

15.1.4.1 Chemical Indicators of Sewage

The faecal sterol coprostanol (5β[H]-cholenstan-3β-ol) and its corresponding epimer epicoprostanol (5β[H]-cholenstan-3α-ol) have also been used as specific indicators of human-derived sewage contamination in the Antarctic marine environment (Edwards et al. 1998; Green et al. 1992; Green and Nichols 1995; Hughes and Thompson 2004; Venkatesan et al. 1986; Venkatesan and Mirsadeghi 1992; Venkatesan and Santiago 1989) and may be analysed in suspended solids, faeces or sediments. Faecal sterols may be useful for distinguishing between sewage and wildlife-derived organic material. Total coprostanol/epicoprostanol ratios have been suggested for discriminating between human and marine mammal faecal contamination (Green et al. 1992), but may be limited because of the high variability of the ratio. Indeed, Venkatesan et al. (1986) suggested that sterol composition in Antarctic sediments might be different from that in temperate environments and urged caution in the specific use of coprostanol for estimation of sewage-derived organic material. Hughes and Thompson (2004) used coprostanol + epicoprostanol:coprostanol + cholestanol (5α[H]-cholenstan-3β-ol) ratios (also termed 5β:5β + 5α ratios) to more specifically distinguish between these faecal sources. The relationship between faecal sterols and bacterial indicators was investigated by Leeming and Nichols (1996). These authors found better correlation between levels of coprostanol and enterococci than for thermotolerant coliforms in a temperate river estuary (Tasmania, Australia). Their correlation of faecal sterol data with bacterial indicator data in this environment indicated that levels of coprostanol of 76 and 499 ng l^{-1} corresponded to 35 and 230 enterococci per 100 ml, while levels of coprostanol of 60 and 400 ng l^{-1} corresponded to 150 and 1,000 thermotolerant coliforms per 100 ml, respectively.

15.1.4.2 Molecular Techniques for Tracing Sewage Micro-Organisms

Modern techniques for tracing microbiological agents of disease, including antibiotic resistance profiles and genotyping, can also be used for tracking sewage. Genotyping includes techniques such as restriction fragment length polymorphism (RFLP), amplified fragment length polymorphism (AFLP), pulsed field electrophoresis (PFGE), multi-locus restriction typing (MLRT), random amplified polymorphic DNA (RAPD), BOX-PCR fingerprinting and ribotyping, as well as enterobacterial repetitive intergenic consensus-PCR (ERIC-PCR), among others. Recent reviews of this rapidly advancing field include those by Olive and Bean (1999), Jannes and De Vos (2006) and others. Significant recent advances have also occurred in microbial community profiling techniques (Spiegelman et al. 2005). While isolation of identical genotypes from respective sewage populations and associated wildlife is not definitive evidence of sewage as the source of the organism, it does provide strong presumptive

evidence. Supporting evidence would include significantly reduced occurrence of the genotype (or microbe) in the same animals remote from the sewage source(s). Significant recent advances in molecular genotyping techniques will allow greater application for determination of wildlife disease epidemiology in relation to sewage disposal. Indeed, application of such techniques for genotyping of bacteria isolated from Antarctic wildlife is increasing (Broman et al. 2000; Leotta et al. 2006a).

With the possible exception of trematodes, cestodes, nematodes and some protozoan parasites, the normal intestinal biota of Antarctic wildlife has been little investigated (Clarke and Macleod 1982; Drozda 1987; Kloeser et al. 1992; Odening 1984; Palm et al. 1998; Pugh 1993; Raga et al. 1997; Skinner and Klages 1994; Wojciechowska and Zdzitowiecki 1995; Yurakhno and Mal'tzev 1995; Zdzitowiecki 1984, 1996; Zdzitowiecki and White 1992). This is not surprising considering the ongoing investigations into the considerable microbial diversity of the human gut. However, a survey of the dominant flora of selected wildlife in terms of faecal indicator bacteria (faecal enterococci, *E. coli*, *C. perfringens*), the Enterobacteriaceae, selected viral pathogens and indicators (Hepatitis A, Norovirus, somatic and male-specific (F+) coliphage) and fungi (*Aspergillus* sp., *Candida* sp.) would be of significant value as the basis for identifying possible changes to normal flora in response to sewage exposure. Indeed, knowledge of the normal enteric microbial flora of Antarctic wildlife is essential as the baseline for determining whether organisms may have been introduced by human activities (Broman et al. 2000).

15.1.5 Extent of Faecal Indicators in Antarctica

Because of its significance to human health, there have been many studies of the extent of faecal contamination in the environment, with quite a number in Antarctica. Most of the Antarctic studies have focused on the marine environment and the extent of contamination around sewage outfalls. There have also been a smaller number of studies of faecal indicators in the Antarctic terrestrial environment, either examining contamination associated with past human activity at sites, or documenting natural faecal indicators associated with the indigenous wildlife.

15.1.5.1 Faecal Contamination from Antarctic Marine Sewage Outfalls

In general, these studies have found faecal indicator organisms at high concentrations (ca. 10^3–10^6 colony forming units[CFUs] per 100 ml) close to sewage discharges (within 25–50 m), with concentrations progressively declining with distance. The extent of sewage plumes has been related to seasonal station populations, the presence or absence of sea-ice cover, as well as related wind-driven turbulence and localised currents (McFeters et al. 1993; Bruni et al. 1997; Delille and Dellile 2000; Redvers 2000; Delille and Geizon 2003; Hughes 2003; Lisle et al. 2004).

At McMurdo Station (USA), the largest station in Antarctica with a summer population of ca. 1,500, concentrations of up to 10^5 total coliforms per 100 ml

were found along a 1 km length of shoreline adjacent to the marine outfall, and concentrations of 100–1,000 total coliforms per 100 ml extended 200–300 m seaward prior to installation and operation of a wastewater treatment plant in 2003 (Howington et al. 1992; McFeters et al. 1993). The size of localised areas of high coliform densities (>1000/100 ml) reduced significantly (Fig. 15.1) in the near-shore area when the outfall was changed from surface to subsurface discharge (5 m depth, 15 m from shore) (Howington 1992; McFeters et al. 1993). An additional ca.

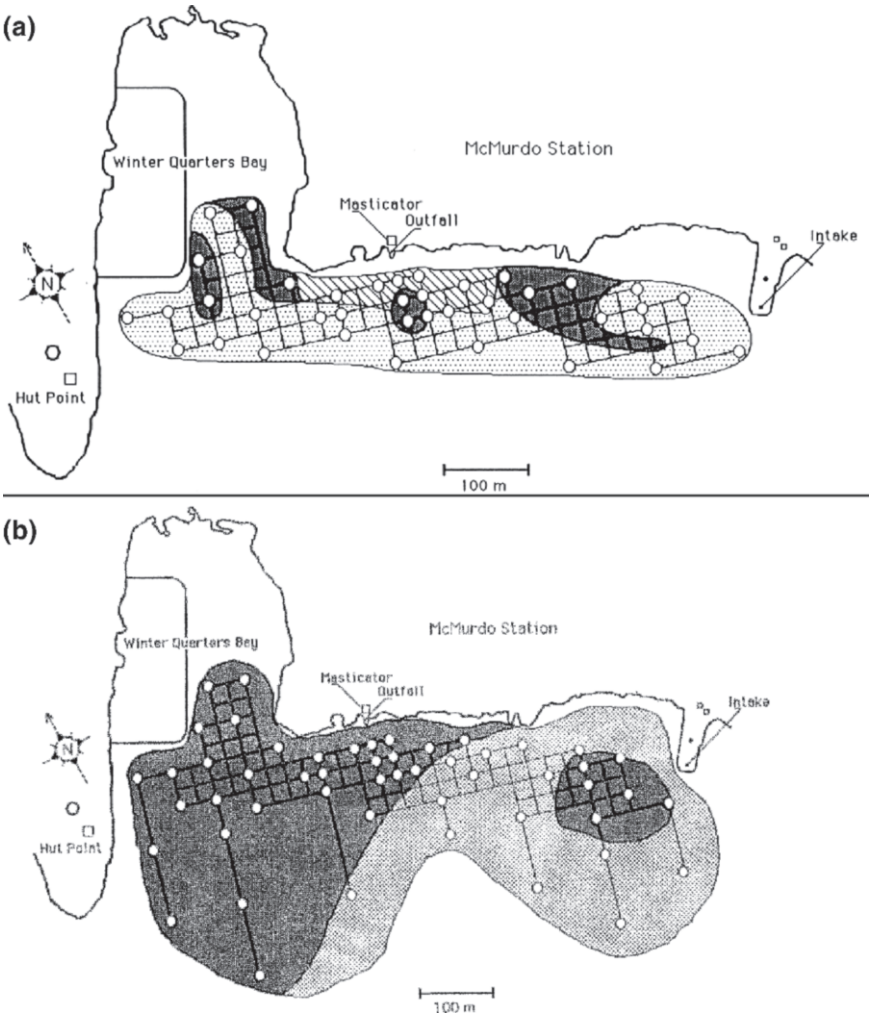

Fig. 15.1 Total coliform bacterial densities near McMurdo Station, Antarctica, (**a**) before (October 1990) and (**b**) after (October 1991) reconfiguration of outfall discharge from surface to submerged. Sampling locations are shown as circles. Total coliform densities are indicated as areas that are stippled (<100/100 ml), shaded (100–1000/100 ml) and cross-hatched (>1000/100 ml). Reprinted from Howington et al. 1992, with permission from Elsevier

10-fold reduction in coprostanol in sediments close to the outfall was observed (Edwards et al. 1998). The changes were thought to be due to better dispersion of sewage from the submerged outfall than when the outfall discharged directly onto the sea-ice. Localisation was thought to be due to restricted dispersion of sewage due to submerged shore-associated sea-ice tide cracks and pressure ridges, and tide-associated movement within these features.

In contrast to McMurdo, most other Antarctic stations have summer populations of 20–60 or less. Faecal indicator bacteria in marine receiving waters surrounding such stations have generally been found to be detectable for just a few hundred metres from outfalls (Bruni et al. 1997; Delille and Delille 2000; Hughes 2003). However, relocating outfalls to improve dispersion can still bring about environmental improvements. At Scott Base (NZ, population 10–100) relocation of the outfall from the foreshore (13 m from the shoreline) to 5 m offshore through a tide crack in the sea-ice increased dispersion and faecal coliform (>1 CFUs per 100 ml) plume area. Increased lateral dispersion along the near shore was noted, and is associated with an area of large tide cracks and pressure ridges. Several other studies have also noted restricted dispersion when effluent is discharged directly onto the foreshore or sea-ice (Delille and Dellile 2000; Hughes 2003). A study at Rothera Station (UK, population ~22 in winter and up to 120 in summer) indicated presumptive faecal coliform levels > 10 CFUs per 100 ml between 300 m (February) and 500 m (September) from the outfall (Hughes 2003). This study also emphasised the effects of variations in station population on presumptive faecal coliform densities, and that these were masked by the effects of changing environmental factors such as solar radiation, the summer algal bloom and sea-ice formation.

A study of the distribution of total coliforms *E. coli*, and enterococci from treated sewage discharged 50 m from the shoreline at Terra Nova Station (Italy), Ross Sea, found indicator concentrations of < 1 CFU per 100 ml at ca. > 200 m from shore (Bruni et al. 1997). Increased concentrations of faecal indicators were related to increases in station population and this was attributed to the inability of the wastewater treatment system to handle the increased volume of influent. A study of faecal coliform dispersion from untreated sewage discharged from Dumont d'Urville Station (France, population ~25–60) found levels < 5 CFUs per 100 ml at > 2 km from the point of discharge (Delille and Delille 2000). A similar study at the sub-Antarctic station Port-aux-Français (France, population ~60–200) on Kerguelen Island also found concentrations of *E. coli* ≥10 CFUs per 100 ml up to 2 km from the discharge (Delille and Gleizon 2003). Maxima of both *E. coli* and enterococci in samples taken near the outfall were strongly correlated with the presence of the supply ship and the concomitant increases in station population.

The spatial extent of faecal contamination also decreases with improvements to sewage treatment. At Rothera Station, regular flushing to the ocean of sewage holding tanks with cold seawater reduced the numbers of faecal indicator bacteria in discharge by 90%, and significantly reduced faecal coliform concentrations in the receiving bay (Hughes and Blenkharn 2003). In February 2003, Rothera Station installed a submerged aerated biological filter sewage treatment plant including UV sterilisation of effluent and removal of dewatered sludge for disposal outside

Antarctica (Hughes 2004, Table 15.1). This significantly reduced dispersion of faecal coliforms (>10 CFUs per 100 ml) from ca. 300 to 50 m when measured at the same time of year (February) (Hughes 2004; Hughes and Thompson 2004).

C. perfringens has been used as an indicator of human faecal contamination in Antarctica both in sediments and fauna. Both Hughes and Thompson (2004) and Edwards et al. (1998) showed that *C. perfringens* concentrations in sediments ca. > 50 CFUs g^{-1} dry wt were restricted to within 200 m of sewage discharges at both Rothera and McMurdo Stations, respectively. Lisle et al. (2004) subsequently found *C. perfringens* in sediments at levels of ca. 3.5×10^3 CFUs g^{-1} dry wt at ca. 800 m from the outfall (Cape Armitage). Edwards et al. (1998) also detected *C. perfringens* in sediments in this area, but at levels between ca. 10 and 50 CFUs g^{-1} dry wt. A general trend of reduced densities of *C. perfringens* in sediments with distance from outfalls was observed in all studies. Campos et al. (2006) also reported the presence of *C. perfringens* near the sewage outfall of Commandante Ferraz (Brazil) Station, Admiralty Bay.

15.1.5.2 Faecal Contamination in the Antarctic Terrestrial Environment

Although the microbiology of Antarctic soils has been well studied over many years (Block 1984; Vincent 1988; Wynn-Williams 1990; Cowan and Lemese 2004), there have been relatively few studies of the extent of faecal indicators at Antarctic terrestrial sites. Bacteria (*Corynebacterium-Rothia*, *Bacillus*, *Pseudomonas*, *Micrococcus* spp.) and fungi (*Cladosporium*, *Penicillium* spp.) reported to be of human origin (but not *E. coli* or other standard faecal indicators) were found in soils from 68 sites up to 100 m from Syowa (Showa) Station (Japan) (Toyoda et al. 1985). Non-culturing techniques (PCR amplification) were used to demonstrate the presence of *E. coli* in soils at an abandoned field site (Canada Glacier camp site), but they were not detected at a currently occupied site (Lake Fryxell camp site) (Sjöling and Cowan 2000) possibly due to improved environmental practices in recent years. Lemese and Cowan (2005) also used PCR to demonstrate the presence of *Staphylococcus epidermidis* in soils from heavily impacted sites in the DryValleys, but not in low-impact and pristine sites. Upton et al. (1997) used PCR to detect human commensals from soils around Halley Station (UK), but were unable to detect them using culture-based techniques. It should be emphasised that standard PCR (as opposed to reverse-transcriptase PCR) does not distinguish between viable and non-viable cells, and that viable cells are required for a microbial pathogen to infect a susceptible host. However, these results do demonstrate the environmental persistence of DNA from these organisms, and hence availability for transformation of indigenous bacteria under suitable conditions (Lorenz and Wackernagel 1994).

15.1.5.3 Faecal Contamination of Antarctic Air

Microbial agents of disease may also be transmitted as airborne particles termed *bioaerosols*. Aerosols can be created via sewage treatment and discharge processes,

and their subsequent dispersion has been described (Bitton 2005; Fannin et al. 1985; Shuval et al. 1989; WHO 2003). Surface discharge into surf or intertidal zones, and venting of wastewater treatment processes may be expected to generate bioaerosols. Generally, propagules of certain viruses as well as sporulated bacteria and fungi are more resistant to airborne environmental stressors (dessication, irradiation, etc.) than non-spore-forming micro-organisms. In a general study of fungal bioaerosols on Signy Island (sub-Antarctic) Marshall (1997) found concentrations of a common saprophytic fungal bioaerosol *Cladosporium* spp. tended to decrease with increasing latitude. Seasonal abundances were associated with the thaw, and transport from more northerly latitudes was suggested as a possible source. Using 16S rRNA PCR to analyse biodiversity of aerosols at Rothera Point (Antarctic Peninsula) Hughes et al. (2004) found that the majority of sequences obtained appeared to be of local or regional origin. These authors also noted the utility of this analytical technique over conventional microscopic morphologic or culture-based isolation/identification techniques. In a study of sewage-derived bioaerosols from an Antarctic marine discharge, Hughes (2006) found faecal coliforms up to 175 m downwind from the intertidal discharge point (which included a 1 m drop to the marine environment) and that dispersion appeared to be related to wind speed. Faecal coliform survival in relation to desiccation and UV irradiation indicated ca. 3–4 $\log_{10}$ reductions in this indicator within 1 h of exposure. However, these organisms retained viability and were deposited cumulatively under low UV irradiation when collected into a suitable growth medium. Hence, although environmental survival of non-spore-forming sewage-derived microbial bioaerosols appears limited, wildlife close to sources may be subjected to cumulative exposures.

15.1.5.4 Faecal Indicators at Sites Occupied by Antarctic Fauna

Faecal indicator bacteria have been found at several sites remote from station sewage outfalls. Typically, these sites were chosen as non-sewage-impacted controls during studies of indicator distribution in the marine environment. In some studies, indicators were found at sites frequented by indigenous macrofauna. Although not a faecal indicator *per se*, numbers of culturable heterotrophic and total bacteria correlated positively with proximity to Adélie and emperor penguin colonies (Dellile 1987). Delille and Delille (2000) detected total coliform bacteria in crowded Adélie penguin colonies in the vicinity of Dumont d'Urville Station. Hughes (2003) found increased presumptive faecal coliform levels (2.16×10^4 CFUs per 100 ml) in seawater proximal to an Adélie penguin colony at East Beach. Bruni et al. (1997) detected 1 CFU enterococci per 100 ml at a control site in the presence of Weddell seals. Hughes and Thompson (2004) found low levels of faecal coliforms (4–12 CFUs per 100 ml) in water collected from control sites near penguins and beached seals. Campos et al. (2006) found *C. perfringen*s at two reference sites and speculated as to its anthropogenic or indigenous faunal origin.

Lisle et al. (2004) detected low concentrations of *E. coli* and enterococci (1 and 3 CFUs per 100 ml, respectively) in water samples from control sites with localised

Weddell seal populations (Little and Big Razorback Islands). Significantly higher concentrations of total and faecal coliforms, *E. coli,* enterococci and *C. perfringens* were found in sediments at these sites than in the water column. Low levels of group II F+ RNA coliphage, presumed to be human-derived, were found in a single water sample (Little Razorback) and in sediment samples (Big Razorback). These were the same genotype, presumed to be human-derived, as that found in untreated sewage from McMurdo Station. Nearby sea-ice camps that dispose of faecal material into ice holes drilled into the annual sea-ice could not be ruled out as the source of these coliphage. Additionally, analysis of Weddell seal scats, as well as rectal swabs, indicated the presence of *C. perfringens*, faecal coliforms, *E. coli*, and enterococci at concentrations similar to that in sewage solids from sediments proximal to McMurdo Station outfall (Lisle et al. 2004). Interestingly, no enterovirus was detected in any samples in this study, including untreated station sewage.

Smith (2000) has suggested that the amount of faeces discharged in sewage from the small sub-Antarctic station at Macquarie Island (Australia, population ~14) was insignificant compared to that from indigenous seals and penguins. Although concentrations of faecal indicator bacteria in control sites near indigenous fauna were generally much lower than those found near sewage discharges, the presence of these organisms as apparently normal flora of indigenous wildlife complicates their use as definitive indicators of anthropogenic input, particularly at low levels, i.e. ca. 1–10 CFUs per 100 ml or 100 g dry wt. Several studies have suggested that enterococci (Bruni et al. 1997; Smith et al. 1994; Lisle et al. 2004) are optimal faecal indicators for water samples and *C. perfringens* for sediments (Edwards et al. 1998; Hughes and Thompson 2004; Lisle et al. 2004), because they are present in greater numbers and at greater distances from sewage outfalls, and may be more persistent in the Antarctic marine environment than alternative microbial indicators.

15.1.6 Survival of Faecal Bacteria in the Antarctic Environment

Studies of the spatial extent of culturable micro-organisms around a point source, such as an outfall, demonstrate that indicator species can survive in the environment, but provide little information on the duration of survival. When sewage effluent is discharged into the marine environment, the micro-organisms within it will be subject to two basic processes: dilution and mixing will reduce their concentrations, and exposure to environmental conditions may render them non-viable or reduce their culturability. Spatial studies using only culturing techniques cannot distinguish between these two processes and therefore provide little information on the survivability of micro-organisms in effluent.

Survivability of pathogens in the Antarctic environment is important to the assessment of the risk of disease transmission to wildlife from human sewage because persistence is a major factor in determining ultimate exposure dose. The next section will consider the likelihood of exposure to viable agents of disease.

15.1.6.1 Survival of Faecal Bacteria in the Antarctic Terrestrial Environment

A number of studies have used faecal material of known age to study survivability of faecal bacteria in the Antarctic environment. Spore- forming and non-spore-forming (including encapsulated *Pseudomonas* spp.) organisms were successfully cultured from 50-year-old faeces at Scott's hut (1910–1911) at Cape Evans (Meyer et al. 1963). Thirty years later, spore-forming *Bacillus* spp. and actinomycetes (*Micromonospora* spp.) were cultured in significant numbers from 80-year-old pony dung from Shackleton's (1907) and Scott's (1910–1911) huts (Nedwell et al. 1994). However, *Pseudomonas* spp., which do not form spores, were not cultured. Boyd and Boyd (1963) found that a single 1 g sample of 208 samples collected at Shackleton's waste disposal site at Cape Royds (1907) was positive for *E. coli*. However, total coliform bacteria were not otherwise isolated in either of these studies.

Viable enterococci and *C. perfringens* as well as anaerobic and spore-forming aerobic bacteria (*Bacillus* spp.) were also found in 30–40-year-old faecal material from the Fossil Bluff Field Station (UK) (Hughes and Nobbs 2004). However, a broad range of other potential bacterial pathogens, including *Staphylococcus aureus*, *Campylobacter, Salmonella* and *Vibrio* spp., as well as total coliforms and *E. coli,* were not detected. While some of these organisms may have been absent in the faecal material when deposited, the absence of the non-spore-forming total coliforms and *E. coli* indicators suggested that these organisms are particularly susceptible to damage by in situ freeze–thaw processes (Parker and Martel 2002). Boyd et al. (1970) found spore-forming bacteria (*Bacillus* sp.) significantly associated with high organic content and coal-contaminated soils, as well as station debris, in the area of Almirante Brown Station, Paradise Harbor, Antarctic Peninsula. These authors found thermophilic bacteria (capable of growth at > 55°C) specifically associated with the contaminated soils and station debris.

15.1.6.2 Survival of Faecal Bacteria in the Antarctic Marine Environment

Faecal material on land is subject to very different conditions from those experienced by sewage effluent discharged into the sea. For practical reasons, studies using historic material have typically sampled relatively large concentrations of faecal matter that may have been subjected to repeated freeze–thaw cycles and span decades (Hughes and Nobbs 2004). In contrast, faecal material in sewage effluent is diluted after discharge to the sea, and often during transfer and treatment. Once in the sea, the effluent is exposed to solar radiation and a saline environment and will not be subjected to temperatures ≤1.8°C or freeze–thaw cycles unless near the surface and frozen in sea-ice. In the Antarctic, most biological productivity and the greatest concentrations of wildlife are found in the marine environment. Therefore, anthropogenic introduction of allocthonous and potentially pathogenic agents into this environment is of particular concern.

The period of survival of faecal bacteria in seawater from non-polar regions is variable, ranging from less than an hour to weeks depending on environmental

conditions (Carluci and Pramer 1959), with low temperatures tending to favour survival (Halton and Nehlsen 1968; Baross et al. 1975). Solar radiation, dissolved oxygen levels, sea-ice, algal blooms, grazing and predation and salinity can also effect bacterial survival (Hughes 2003; Smith et al. 1994).

Very few direct experimental studies of the survival of faecal bacteria in Antarctica have been reported. Using both laboratory and field exposures, Statham and McMeekin (1994) demonstrated that faecal bacteria are rapidly inactivated (~50 min, 40 min and 2 h for 1 log reduction for *E. coli*, *Salmonella zanzibar* and a faecal *Streptococcus*, respectively) when exposed to artificial light (290–800 nm) under Antarctic conditions. *E. coli* showed a similar decline under ambient light. They concluded that as repair mechanisms are unlikely to operate *in situ*, resuscitation of sublethally damaged cells is improbable, and recommended that sewage should be discharged on or near the sea surface to maximise exposure to solar radiation. These authors also recommended against rapidly discharging large volumes, which could cause increased turbidity, lower light penetration and consequent accumulation of undamaged cells in sediments.

Rapid mortality (100% after 90 min exposure to 0.36 kJ of UV_{DNA} m^{-2}) of *E. coli* (exposed population concentration unknown) exposed to solar radiation at Rothera Station at mid-day (12:30–14:00 local time) during the summer (8 February) has also been reported by Hughes (2003). Observations of lower faecal coliform counts with seasonally increasing solar radiation led this author to suggest that solar radiation dose is the dominant factor controlling concentrations of this indicator in seawater near the station. A later study demonstrated that the bactericidal effects of solar radiation on *E. coli* progressively increased with shorter wavelengths < 345 nm, showing a ca. 3.5 $\log_{10}$ reduction at 280 nm over 60 min exposure (Hughes 2005). Additionally, die-off was greater in seawater compared to phosphate buffered saline, under UV_B compared to UV_A or photosynthetically-active radiation (400–700 nm), and in filtered sewage compared to raw, centrifuged sewage containing particulates. These results indicate the importance of solar radiation in reducing numbers of bacteria in sewage discharged into the marine environment.

When exposed to Antarctic marine conditions using in situ diffusion chambers, but without high levels of light, enteric bacteria (*E. coli*, *Salmonella typhimurium* and *Yersinia enterocolitica*) remained within 1% of inoculum values (measured by direct viable counts and respiratory activity) after 54 days of exposure (Smith et al. 1994). This was despite progressive relative decreases in recovery of these bacteria on selective media versus non-selective media, suggesting that these species may persist for extended periods in the Antarctic in a sub-lethally injured or viable but non-culturable (VBNC) state, particularly when shielded from solar radiation in sediments or by sea-ice cover (Smith et al. 1994). After 54 days exposure *E. coli*, *S. typhimurium* and *Y. enterocolitica* respiratory activity was found to be limited by nutrients rather than temperature, and these organisms had become markedly thermosensitive. These results suggest that reduced-temperature and/or reduced-selectivity resuscitation steps may increase recovery of these organisms from the Antarctic marine environment, and that counts obtained should be considered minima owing to the possibility of sub-lethal injury or VBNC responses. However,

the extended persistence observed in the absence of high light levels is in contrast to the rapid mortality observed when enteric bacteria are exposed to Antarctic ambient light (Hughes 2003, 2005; Statham and McMeekin 1994).

Field observations have confirmed that human enteric micro-organisms can persist in the long term in the marine environment, particularly at low temperatures. Counts of *C. perfringens* in benthic sediments at an abandoned deepwater sewage sludge dumping ground (~2,500 m depth) were 10-fold higher than at a reference location 1 year after the cessation of dumping (Hill et al. 1993). O'Neill et al. (1968) found that 10–20% of enterovirus remained infective in frozen sewage at −33°C over 4 months.

It is common practice in microbiology laboratories to store bacterial cultures at reduced temperatures. Indeed, one of the most common ways to preserve viable microbial cultures in the long term is by freezing, albeit often in the presence of a cryoprotectant. Hence, while the low *in situ* ambient temperatures in Antarctica may be expected to generally prolong microbial survival relative to more temperate regions, other factors, such as increased summer photoperiod, freeze/thaw, predation/grazing and salinity, may collectively or individually contribute to increased die-off of potential pathogens in human faecal waste and sewage. Considering these factors, the ability of potential pathogens to remain viable for periods of hours, days or months may be more relevant to the risk of disease transmission to marine wildlife than periods of tens of years.

15.2 Links Between Sewage Exposure and Ill Health in Wildlife

In order for an infectious disease to be produced in a host, the host must first be exposed to a sufficient *number* of sufficiently *infective* microbial cells (or virions) to which the host is sufficiently *susceptible*. Proof that an infectious agent causes disease in a particular host requires the satisfaction of one of the basic tenants of pathogenic microbiology, Koch's postulates: (1) The same pathogen must be present in every case of the disease, (2) the pathogen must be isolated from the diseased host and grown in pure culture, (3) the pathogen from the pure culture must cause the disease when inoculated into a healthy, susceptible laboratory animal and (4) the pathogen must be isolated from the inoculated animal and must be shown to be the original organism.

In practice, exceptions must often be made to Koch's postulates. These include cases where an infectious agent is not culturable outside the host (postulates 2, 4), or ethical/regulatory considerations prevent experimental host infection (postulate 3). Additional problems arise when a single organism may cause different or subclinical/asymptomatic disease(s) under different conditions. Diseases that may be caused by a community of micro-organisms rather than a single pathogen also require exceptions to Koch's postulates.

The authors found no published accounts of investigations of disease in Antarctic wildlife that satisfies Koch's postulates. The difficulty in obtaining and maintaining Antarctic wildlife, particularly birds and mammals, for experimental laboratory

exposure to infectious agents largely precludes these types of studies. This is particularly the case for vertebrate macrofauna. Instead, investigations of disease are likely to be limited to isolation of etiological agents from animals presenting symptoms, and correlation with absence of the disease and the presumptively associated etiological agent in control animals.

In the next sections we will consider the range of disease-causing agents likely to be present in Antarctic sewage and whether there is evidence that Antarctic fauna have been exposed to pathogens from sewage. We will also consider whether there is evidence that exposure to human sewage has caused disease in any wild animal populations.

15.3 Pathogenic Micro-Organisms in Sewage

Raw domestic sewage contains a wide range of pathogenic or potentially pathogenic micro-organisms (Table 15.2) (Long and Ashbolt 1994; WHO 1999, 2003; US EPA 1992, 1999). Quantities of individual pathogens and classes of pathogens per unit volume vary widely depending on a number of factors, including numbers and types of infections in the source population, climate and type of wastewater treatment. It should be noted that the diseases associated with the organisms listed in Table 15.2 are those in human hosts, although several are known to cause disease in non-human hosts, for example, *Giardia lamblia/intestinalis* giardiasis in canines.

During sewage treatment, efficiencies of removal and inactivation of pathogens vary in ways that are dependent on treatment process and the characteristics of individual pathogens. Some pathogens, particularly encysted protozoans and helminths, sporulated bacteria, and some encapsulated viruses, are more difficult to remove and/or inactivate than others. As a *broad* generalisation, primary treatment processes may result in ca. 0.5–3 $\log_{10}$ reductions in viable pathogen levels, and secondary treatment and disinfection in further ca. 0.5–4 $\log_{10}$ and 0.5–6 $\log_{10}$ reductions, respectively (Bitton 2005; EPHC/NRMMC 2005; Gerardi 2004; Long and Ashbolt 1994; WHO 1999, 2003).

Sewage may contain other pathogens in addition to the enteric and urogenital micro-organisms expected in faeces and urine. Respiratory, ocular and dermal pathogens may enter the sewage system via bathing, washing of affected materials, or on tissues, etc. disposed of into wastewater. In recognition of this, current World Health Organisation guidelines for classification of recreational waters using microbiological indicators (enterococci) are based on risk of both gastrointestinal disease as well as acute febrile respiratory illness (AFRI) (WHO 2003).

15.3.1 Factors that may Influence Pathogens in Antarctic Sewage

Pathogen incidence and concentrations vary regionally throughout the world (EPHC/NRMMC 2005; Gerardi 2004; Leclerc et al. 2002; Morris 2003; WHO 2003).

Table 15.2 Examples of known human pathogens in raw sewage and associated disease(s)

Micro-organism	Associated human diseases
Bacteria	
Pathogenic *Escherichia coli*	Gastroenteritis
Salmonella typhi	Typhoid fever
Various *Salmonella* spp.	Gastroenteritis
Shigella sp.	Bacillary dysentery
Vibrio cholerae	Cholera
Campylobacter spp.	Gastroenteritis
Clostridium perfringens	Gastroenteritis/gangrene
Legionella spp.	Legionellosis (respiratory illness)
Pseudomonas aeruginosa	Wound, skin and pulmonary infections
Staphylococcus aureus	Wound infections, gastroenteritis
Streptococcus sp.	Respiratory infections
Hellicobacter pylori	Peptic ulcers
Yersinia spp.	Gastroenteritis, septicemia
Campylobacter spp.	Gastroenteritis, Guillain–Barré syndrome
Atypical Mycobacteria	Respiratory illness
Fungi	
Candida albicans	Thrush, candidiasis
Aspergillus sp.	Aspergillosis
Viruses	
Adenovirus	Gastroenteritis
Astrovirus	Gastroenteritis
Calicivirus	Gastroenteritis
Coronavirus	Gastroenteritis
Enterovirus	Gastroenteritis, respiratory illness, nervous disorders, myocarditis
Hepatitis A	Infectious hepatitis
Norovirus G	Viral gastroenteritis
Poliovirus	Polio, diarrhoea
Rotavirus	Diarrhea, vomiting (emesis)
Protozoans	
Giardia lamblia/intestinalis cysts	Gastroenteritis
Cryptosporidium parvum/hominis oocysts	Gastroenteritis
Entamoeba histolytica	Amoebic dysentery
Naegleria fowleri	Amoebic meningitis
Helminths	
Ancyclostoma sp.	Anemia
Ascaris sp.	Ascariasis
Diphyllobothrium latum	Fish tapeworm
Taenia solium/saginata	Tapeworms
Trichuris	Diarrhoea, anemia, whipworm

Adapted from: Bitton 2005; EPHC/NRMMC 2005; Gerardi 2004; Leclerc et al. 2002; WHO 1999, 2003

In addition, the quantity and type of pathogens present in sewage from an Antarctic station may be different from those generated by the general population, although we are not aware of any comprehensive studies of pathogens (as opposed to faecal indicators) in wastewater from Antarctic stations that could be used to test whether this is the

case. Most Antarctic programs require participants to pass a physical examination prior to deployment, which usually includes screening for signs of disease. Some diseases are more common in children than in the adult population; consequently, these diseases are likely to be under-represented as a result of the absence of children from Antarctic communities. Thus, the overall health of people at Antarctic stations may be better than average, with some diseases reduced or excluded. These factors, together with the absence of many vectors of human disease common in temperate climates, such as domestic animals and insects, suggest a lower likelihood of disease and shedding of pathogens into wastewater in station populations.

Conversely, the relatively confined, largely indoor nature of life on Antarctic bases increases the risk of person-to-person disease transmission once an infectious disease is introduced (Allen 1973). Antarctic expeditioners undergo physiological and psychological stress and these can be expected to impact their health (Cosman and Brandt-Rauf 1987) including their immune status and consequent resistance to disease. Francis et al. (2002) found lowered mucosal immunity in some expeditioners, while Mehta et al. (2000) found diminished cell-mediated immunity during over-winter isolation. A study of *E. coli* transmission over 26 weeks within a small group of isolated expeditioners at Signy Station (UK, South Orkney Islands) using multi-locus electrophoresis allozyme typing and plasmid analysis (Tzabar and Pennington 1991) indicated spread between station personnel. In contrast, Shult et al. (1991) found low levels of transmission of adenovirus between personnel at McMurdo Station. Lowered immune status may also reactivate latent viruses in expeditioners, resulting in possible increased viral shedding (Mehta et al. 2000).

15.3.2 Sewage-Associated Pathogens in Indigenous Fauna

There are a number of records of isolation of organisms which may be found in sewage from marine mammals and avians; however, there is little conclusive evidence linking them to active disease processes in these hosts. For example, *Giardia* spp. cysts have been isolated from harp seals, *Phoca groenlandica*, grey seals, *Halochoerus grypus,* harbour seals, *Phoca vitulina*, and ringed seals, *Phoca hispida*, in the western Arctic and eastern Canada (Measures and Olson 1999; Olson et al. 1997). Similarly, Oelke and Steiniger (1973) isolated *Salmonella* spp. from 12% of Adélie penguins and 18% of south polar skuas, *Catharacta maccormicki*, on Ross Island (Cape Crozier). A survey of two species each of penguins and albatrosses, as well as Antarctic fur seals on Bird Island, South Georgia, in 1996 and 1998 isolated various *Salmonellae* species of very low genetic heterogeneity (Palmgren et al. 2000). The S. Enteritidis was thought to be introduced from land- or ship-based sewage, and migratory birds were suggested as possible sources (Olsen, et al. 1996). More recently a multi-year (2000–2003) study of avians at Hope Bay, Antarctic Peninsula (site of Esperanza Station), by Leotta et al. (2006b) isolated *Campylobacter lari* from the intestines from 1 of 58 Adélie penguins, 5 of 28 brown skuas, *Stercorarius antarctica lonnbergi*, 2 of 16 kelp gulls, *Larus dominicanus*, and 2 of 13 south polar skuas. All birds

were dead at time of examination; however, this coincided with an outbreak of avian cholera. A clonal isolate of *Campylobacter jejuni* was isolated from cloacal and/or rectal swabs of 3 of 100 macaroni penguins, *Eudyptes chrysolophus*, at Bird Island in the sub-Antarctic (Broman et al. 2000).

A New Zealand study identified an increase in fur seal pup mortality associated with poor adult feeding conditions which coincided with an observed 4-fold increase in *Salmonella* sp. (Connolly et al. 2001). They isolated a number of *Salmonella* sero-, and phage-types from New Zealand fur seals, *Arctocephalus forsteri*, and sea lions, *Phocarctos hookeri*, but none were isolated from single samples from Leopard seals, *Hydrurga leptonyx*, Elephant seals, *Mirounga leonina*, or sub-Antarctic fur seals, *Arctocephalus tropicalis*. Several of the fur seal isolates showed identical sero-, and phage-types to known New Zealand porcine isolates. One isolate of S. Enteritidis was isolated from a fur seal suffering from lesions consistent with *Salmonella* septicaemia; one from a seal also infected with *Mycobacterium* sp.; and several from fur seals at necropsy, but without typical Salmonellosis lesions.

These studies indicate that *Salmonella* and *Campylobacter* sp. are found in a number of diverse locations in indigenous Antarctic and sub-Antarctic birds. Although it is known that some *Giardia*, *Salmonella and Campylobacter* sp. can produce disease in wildlife, it is not known whether the organisms identified in these studies originated from sewage or other anthropogenic sources. Indeed, the presence of these organisms as normal or commensal flora cannot be discounted (Broman et al. 2000; Leotta et al. 2006a, 2006b; Murray 1991). As yet, no direct causal links have been established between sewage-derived micro-organisms and disease in these animals.

15.3.3 Uptake of Faecal Bacteria by Indigenous Fauna

As described above and elsewhere in this book, infection with *Salmonella* spp. and *Giardia* spp. has been described for several marine mammals and/or avians. However, the source of these infections is unknown. It is well known that faecal bacteria can be taken up by marine invertebrates. Indeed, the ability of groups such as bivalves to concentrate bacteria, viruses and parasites makes them promising bio-indicators (Faghri et al. 1984; Marino et al. 2005; Miller et al. 2005; Pommepuy et al. 2004). It is reasonable to consider that wildlife may also be exposed to sewage-associated disease agents through bioconcentration and subsequent transmission via the food chain.

In a sewage exposure experiment at Terra Nova Station, Bruni et al. (1997) showed that coliforms were concentrated by an indigenous bivalve mollusc *Laternula elliptica* and suggested possible transmission via the food chain. Edwards et al. (1998) demonstrated a high incidence of *C. perfringens* in invertebrate and fish species intestines collected near the McMurdo Station sewage outfall relative to distant locations, with the following rates of isolation: tunicates (*Cnemidocarpa verrucosa*) 100%; clams (*Laternula elliptica*) 90%; sea urchins (*Sterechinus neumayeri*) 83%; and starfish (*Odontaster validus*) 32%. *C. perfringens* was not detected in nemertean worms, *Parborlasia corrugatus*, or the most abundant local

fish species, *Trematomus* spp. There was a trend of decreasing *C. perfringens* incidence in tunicates and sea urchins with distance from outfall (Fig.15. 2), with 70% of overall tunicates and 40% of sea urchins harbouring *C. perfringens* in samples taken up to 822 m from outfall. *C. perfringens* was absent at the control site 3 km from outfall.

These results indicate that bacteria from station sewage can be ingested and harboured in the guts of a number of indigenous Antarctic benthic marine invertebrates. It is not known whether *C. perfringens* is capable of replication within these hosts, and therefore whether these data indicate active infection or simple ingestion of viable cells. The presence of *C. perfringens* in the guts of these invertebrates roughly corresponded with their presence in associated sediments (Edwards et al. 1998; Lisle et al. 2004). However, no disease symptoms were reported in any of the invertebrates and fish analysed.

15.3.4 Evidence that Sewage-Associated Pathogens Cause Disease in Wildlife

Throughout the world many large cities dispose of sewage effluent to the ocean, and at many such sites there are established populations of birds and marine mammals which likely come in close contact with effluent. We are therefore surprised that we have been unable to locate any reports, either from Antarctica or elsewhere, in which disease in wildlife has been definitively linked to exposure to pathogens from sewage effluent in the environment. One possible exception is the case of a human

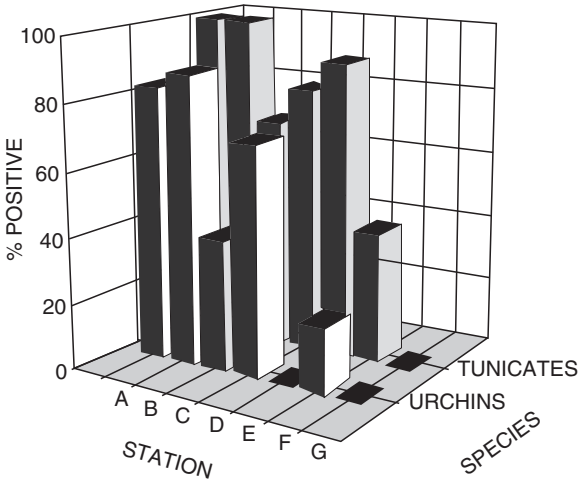

Fig. 15.2 Distribution of *Clostridium perfringens*-positive tunicates and sea urchins along a transect of increasing distance from the McMurdo Station sewage outfall. Station distances from sewage outfall are as follows: **a** (0 m), **b** (150 m), **c** (332 m), **d** (444 m), **e** (594 m), **f** (822 m), **g** (3,000 m). Reprinted from Edwards et al. 1998, with permission from ASM Press, Washington DC

faecal bacterium *Serratia marcescens* that has recently been found to be the causal agent of white pox disease in Carribean elkhorn coral, *Acropora palmata*, resulting in coral losses of up to 70% in the Florida Keys (Patterson et al. 2002). The source of *S. marcescens* is thought to be human faeces, but the link has yet to be confirmed.

We find this surprising because there is considerable historic evidence of human-to-human transmission of diseases such as typhoid, cholera, polio and hepatitis through direct contact with faeces, exposure to sewage or sewage effluent, or indirectly by consumption of sewage-contaminated water or foods such as shellfish. There are also many common examples of animal-to-human transmission of disease through contact with animal faeces that contain disease-causing agents such as Salmonellae (Murray 1991), *Giardia lamblia*, tapeworms and others.

We can offer no logical reason why there should be an absolute barrier preventing transmission from humans to wild animal populations, and suggest that a more likely explanation is that the link between human sewage and wildlife disease has not been demonstrated because it has not been widely investigated. Research effort has focused on health threats to humans *from* animals rather than the other way round. Indeed, the majority of wildlife disease literature is focused on zoonotic disease transmission to humans (Bengis et al. 2004), with some research effort directed towards transmission between animals (Daszak et al. 2004), or the impacts of environmental change on wildlife disease (Daszak et al. 2001; Williams et al. 2002). The extent and nature of a 'species barrier', in which pathogens show a degree of taxonomic specificity with regard to host infection and disease, remain poorly understood, particularly for wildlife as opposed to domesticated animals. However, host–parasite co-evolution and host adaptation into relatively taxonomically distinct pathogen species/genotypes has been described for pathogens causing disease in both human and wildlife (Xiao 2002).

15.3.5 Application of Microbiological Monitoring

Standardised procedures for monitoring of environmental impacts have been the subject of much discussion within the Antarctic Treaty System with the objective of agreeing on a set of common biological and non-biological parameters to be monitored (Kennicutt and Walton 2006). Even though the risk of infection of indigenous wildlife from sewage contamination is almost wholly unknown, the recognised risk of environmental contamination from sewage is such that monitoring of faecal contamination should be included in routine assessment and monitoring systems for Antarctic stations. The microbiological indicators described here offer cost-effective options for monitoring both marine and terrestrial environments.

To ensure that monitoring achieves its objectives, the overall approach and the details of sampling design and laboratory techniques should all be appropriate for use under the very particular conditions experienced in Antarctica. The sampling design should include sufficient replication to account for natural variability, and to provide sufficient statistical power to identify important differences. Reference locations are an essential component of any rigorous monitoring design and are

particularly important in this application because faecal indicators from non-human sources are known to occur at many locations in Antarctica well away from anthropogenic influences. Analytical techniques must be logistically feasible and suitable for use by a wide range of personnel, often with limited laboratory space. Microbiological samples have relatively short hold-times (8–48 h) within which analyses must be initiated. Therefore, samples cannot be routinely shipped out for analysis. Analyses should additionally minimise requirements for sub-culturing and/or confirmation of presumptive positive samples. Recent advances in defined substrate and chromogenic/fluorogenic media for the analysis of total coliforms, *E. coli, Salmonella* spp. enterococci and *C. perfringens* allow for such simplified analyses (Manafi 1996; Adcock and Saint 2001; Kinzelman et al. 2003; WHO 2003). Indeed, several studies have already used such media for enumeration of *E. coli* (Bruni 1992; Delille and Gleizon 2003; Lisle et al. 2004) and enterococci (Delille and Gleizon 2003) in Antarctic and sub-Antarctic marine environments. To increase the utility of monitoring data, it must be made available and disseminated (Kennicutt and Walton 2006), preferably using the network of national Antarctic data centres established under the Antarctic Treaty obligation to share scientific data.

Efforts expended in rigorous experimental design, careful sampling, accurate analysis and the most thorough systems for data collation and dissemination are wasted without good data assessment tools. Assessment of the risk to humans of sewage in the environment is commonly based on epidemiological models linking disease occurrence to exposure (Fleisher et al. 1998; Haas 2002; EPHC/NRMMC 2005; Prüss 1998; WHO 2003). As specific data on the risk to wildlife from exposure to sewage is lacking, human-derived risk assessment data may be the best available starting point for this purpose. Alternatively, selected animal-derived pathogen dose–response relationships may be used in conjunction with estimated indicator/pathogen ratios/correlations in order to estimate risk of disease using microbiological monitoring data (Haas 2002).

Regular monitoring and assessment against standards developed in other regions will encourage wastewater treatment practices that conform to national and international regulatory schemes established to protect people from contact with dangerous levels of sewage-derived pathogens through activities such as recreational bathing and the consumption of seafood (COMNAP 2005, EPHC/NRMMC 2005, NSSP 1999, WHO 2003). It is not yet certain whether these standards are appropriate to protect the wildlife of Antarctica.

15.4 Conclusions and Implications for the Future

Sewage treatment and disposal technologies currently in use at many Antarctic stations, particularly marine disposal of sewage effluent, introduce a great variety of micro-organisms, likely including human-disease-causing pathogens, to the Antarctic environment. Because of the need to keep Antarctic stations supplied with fuel, food, equipment and personnel, most are located on ice-free land adjacent to the coast so that they are easily accessible by ship. These ice-free coastal sites are also the major breeding habitats for many Antarctic wildlife species

including seals, penguins and other seabirds. As a consequence, exposure of wildlife to discharged effluent is likely at many locations.

Whether or not exposure of Antarctic wildlife to sewage effluent can result in disease remains unproven. Indeed, we were surprised that we could not find any definitive examples of transmission of disease to wildlife from sewage, not just in Antarctica but from any region, but we do not accept that this is evidence that such transfers cannot occur. Even without proof that sewage discharge to the Antarctic environment can lead to the transfer of disease, the practice stands out as the only activity permitted to occur in Antarctica which will inevitably lead to the introduction of non-native species and genetic material on a large scale.

Microbiological monitoring using standard indicators is essential for assessing the efficacy of sewage treatment, and may be used to trace dispersion in the environment. However, in isolation, dispersion investigations will only confirm what is already known – that effluent disposal results in introduction of a range of micro-organisms, detectable over a finite area around the discharge point. They will be of far greater value if combined with more targeted studies designed to provide information in support of risk assessment such as the occurrence of faecal indicators or pathogens in the Antarctic food chain (for example, benthic filter feeders) and epidemiological studies of wildlife.

Remote communities such as Antarctic stations represent unique environments for study of possible links between sewage effluent disposal and wildlife disease, as numbers of potential sewage sources are limited, and often geographically isolated. These factors may make it easier to definitively trace the origin of a pathogen. On the other hand, the probability of detecting pathogens derived from sewage in wildlife in general may be higher in studies undertaken in more densely populated regions where much larger volumes of effluent are discharged, with a greater variety of pathogens entering the wastewater stream and a greater diversity of wildlife in the receiving environment. The development of molecular genotyping methods for identifying and tracing particular strains of disease-causing agents provides a set of useful tools for such investigations.

The development and implementation of new sewage treatment facilities with a target of zero emissions to the Antarctic environment would very directly solve the known problem of introduction of non-native species through effluent discharge and would make the question of whether sewage associated pathogens can cause disease in Antarctic wildlife irrelevant. However, until zero-emission technologies are readily available for Antarctic use, we suggest that wastewater should receive a minimum of secondary treatment with subsequent disinfection prior to discharge into the marine environment.

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Chapter 16
The International Legal Framework for Protecting the Health of Antarctic Wildlife

D.R. Rothwell

16.1 Introduction

An assessment of the framework of the international law for protecting the health of Antarctic wildlife first requires an appreciation of the Antarctic Treaty System (ATS 1959). The ATS has rapidly developed since the adoption of the Antarctic Treaty in 1959. While initially designed to resolve tensions over sovereignty, the freedom of scientific research, and the potential militarisation of the continent during the Cold War, the Treaty parties soon began to direct their attention to the protection of the Antarctic environment. In 1964, the Agreed Measures for the Conservation of Antarctic Fauna and Flora were adopted as Recommendation III–VIII (Bush 1982a, pp. 146–169). These were followed in 1972 by the Convention for the Conservation of Antarctic Seals (CCAS 1972), and in 1980 by the Convention for the Conservation of Antarctic Marine Living Resources (CCAMLR). During the 1980s there was considerable debate amongst the Antarctic Treaty Consultative Parties (ATCPs) over the need for a minerals regime in Antarctica. This eventually resulted in the adoption of the Convention for the Regulation of Antarctic Mineral Resource Activities in 1988 (CRAMRA 1988); however, this Convention was quickly abandoned in favour of the 1991 Protocol on Environmental Protection to the Antarctic Treaty (Protocol 1991), commonly referred to as the Madrid protocol.

At the same time as the ATS has been developing, there has been a growth in international environmental law (Birnie and Boyle 2002). International environmental law has generally not sought to specifically deal with Antarctic issues, by and large leaving the ATS to develop its own specific responses to Antarctica. Nevertheless, there are a number of international environmental conventions which create general obligations for States which conduct operations in Antarctica which are relevant when considering issues associated with protecting the health of Antarctic wildlife. At the conclusion of this review, an attempt will be made to summarise the state of the current law on this topic.

D.R. Rothwell
ANU College of Law, Australia National University ACT 2600, Australia
e-mail: RothwellD@law.anu.edu.au

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16.2 The Antarctic Treaty System and Environmental Protection

16.2.1 The Antarctic Treaty

Formal negotiations between States with an interest in Antarctic affairs commenced in the late 1950s and these culminated in the 1959 Washington Conference which was convened by the United States. The major issues considered at the Conference were sovereignty and scientific research. Present at the Conference were all of the seven Antarctic territorial claimants (Argentina, Australia, Chile, France, New Zealand, Norway and United Kingdom) and five other States (Belgium, Japan, South Africa, United States and USSR) which had an active interest in Antarctic affairs, either through a historical association or as a result of the conduct of research activities during the 1957–58 International Geophysical Year. Notwithstanding the difficulty of some of the issues being considered, a treaty was quickly negotiated and on 1 December 1959 the Antarctic Treaty was concluded and signed by all of the participating States. Ratifications were received without undue delay and the Antarctic Treaty entered into force on 23 June 1961. In addition to the 12 States which attended the Washington Conference, Poland acceded to the Treaty early in June 1961, resulting in there being 13 original parties.

Compared to modern international legal instruments, the Antarctic Treaty seems a very straightforward document. Comprising of only 14 Articles, it combines some very basic measures dealing with demilitarisation and conduct of science, with some very sophisticated provisions dealing with sovereignty and Treaty review. The Treaty focuses on the critical issues of Antarctica's management and future as identified by the Antarctic States in the 1950s. Provisions dealing with demilitarisation, the importance of science, and the resolution of sovereignty claims in particular stand out. The significance of these issues is noted in the Treaty's Preamble (Auburn 1982; Triggs 1986).

The Treaty attempts to promote the continuation of the 'freedom of scientific investigation' which occurred during the International Geophysical Year (Antarctic Treaty review, Article II). In furtherance of this ideal of Antarctica being a 'continent of science', the Treaty provides for ways in which international scientific cooperation can continue (Antarctic Treaty, Article III). The difficult question of sovereignty in Antarctica is dealt with in Article IV of the Treaty. Existing claims, potential claims and future claims to Antarctica are neither recognised or prejudiced under the Treaty (Antarctic Treaty, Article IV(1)). No new claims, including the extension of existing claims, can be made while the Treaty is in force (Antarctic Treaty, Article IV(2)). The Treaty extends to the area south of 60° South latitude, including all ice shelves (Antarctic Treaty, Article VI). The effect of this somewhat artificial northern limit on the operation of the Treaty is that certain sub-Antarctic islands are excluded from the Treaty's operation. These include the Falkland Islands, the South Sandwich Islands, South Georgia, Prince Edward Island, Kerguelen, Marion Island, Macquarie Island, Heard and McDonald Islands, Campbell Island, Bouvet Island, Gough Island and Crozet Islands.

It was recognised during the negotiation of the Antarctic Treaty that there would be a need for the Treaty parties to meet on a regular basis and discuss further measures for regulating the Antarctic affairs. To that end, provision is made for regular 'Antarctic Treaty Consultative Meetings' (ATCMs) (Antarctic Treaty, Article IX) at which the original States that participated at the Washington Conference could meet in conjunction with others which had achieved membership by demonstrating substantial scientific commitment to Antarctica. The Treaty parties could agree upon recommendations and measures in furtherance of the principles and objectives of the Treaty. Recommendations and measures adopted at ATCMs have been the primary method by which the ATCPs have been able to deal with matters relating to the implementation and operation of the Antarctic Treaty, many of which have had a strong environmental focus (Rothwell 1996).

16.2.1.1 Assessment

The Antarctic Treaty contains a number of core provisions which are relevant to the health of Antarctic wildlife. The first relates to the fundamental freedom of scientific research in Antarctica. However, notwithstanding the considerable attention given to scientific research in Antarctica, this is not an unlimited freedom. The Treaty indicates that science is to be conducted 'subject to the provisions of the present Treaty' (Antarctic Treaty, Article II) and this opens the door for an interpretation which justifies research rights being read alongside provisions which also include those dealing with 'preservation and conservation of the living resources' (Antarctic Treaty, Article IX). The Treaty does, however, have a limited scope. It only extends to a defined geographical area and does not extend beyond 60°S. However, the Treaty has consequences for many operations conducted beyond Antarctica because if States wish to ensure that they meet the Treaty obligations they will ensure that they have in place various mechanisms – legal and non-legal – which control the activities of expeditioners and other visitors prior to reaching Antarctica. Finally, it must be remembered that the Antarctic Treaty contains sensitive provisions on sovereignty and the exercise of jurisdiction. Effectively, jurisdiction can only be exercised over nationals under the terms of the Treaty (Antarctic Treaty, Articles VII, VIII). There are exceptions to this, however, in relation to nationals of States which are not parties to the Treaty; this immediately raises very difficult questions as to the status of the Treaty and whether any recognition is granted to territorial (and associated maritime) claims.

The Antarctic Treaty therefore creates the framework within which activities effectively take place in Antarctica and much of the Southern Ocean. To this end, the most significant aspect of the Antarctic Treaty is how, through the adoption of recommendations and measures at the ATCMs, plus the negotiation and entry into force of a range of additional instruments, the Treaty has been expanded into the much wider ATS which does seek to address a range of environmental issues, including the question of the health of wildlife.

16.2.2 The 1964 Agreed Measures

The increasing attention given to environmental questions at the ATCMs has seen the ATS become an environmentally focused regime (Rothwell 1996, p. 446). This process commenced with the 1964 Agreed Measures which represented the first comprehensive attempt in the ATS to protect the Antarctic environment. They declared the Antarctic Treaty area to be a 'Special Conservation Area' and seek to bind not only the Antarctic Treaty parties but also to impact upon third parties (Bush 1982a, p. 146). Their main focus was to protect Antarctic flora and fauna from the impact of man's increasing activity on the Antarctic continent. To this end, Article VI imposed an obligation upon all participating governments to prohibit 'within the Treaty area the killing, wounding, capturing or molesting of any native animal or native bird, or any attempt at any such act, except in accordance with a permit'. Permits could only be issued in instances where they were necessary to provide 'indispensable food' for men or dogs, to provide specimens for scientific study, scientific information, or to provide specimens for museums and other related institutions. For issuing permits, it is the responsibility of the government to take into account the ability of the native mammals or birds being taken to reproduce and maintain the species, the variety of species, and balance of the natural ecological system. The Agreed Measures also prohibited harmful interference with 'the normal living conditions of any native mammal or bird' and in Article VII listed certain activities which were considered to be such.

It is interesting to reflect that as far back as the early 1960s, the ATCPs recognised the threat posed to Antarctica as a result of the introduction of non-indigenous species and disease, especially in the context of the impact upon the fauna and flora. To that end, Article IX of the Agreed Measures prohibited the bringing of non-indigenous species of animals and plants into Antarctica except by way of permit. Article IX(2) qualified the conditions under which a permit could be issued and as such guidelines did effectively exist for their implementation. To that end, when this provision was combined with Annex C of the Agreed Measures, the only animals and plants which could be imported were: sledge dogs, domestic animals and plants, and laboratory animals and plants. A later amendment to the Agreed Measures adopted at ATCM VII (1972) proposed that Annex C be extended so that it applies in the following instance:

> *laboratory animals and plants including viruses, bacteria, yeasts and fungi*.

This amendment resulted in some interesting responses by the ATCPs when it came to giving effect to the provision in their domestic legislation. A number of the ATCPs only approved Recommendation VII-5 after some delay, in some instances because of the need to take legislative action. The United States, for example, accepted Recommendation VII-5 only as a 'modification of the interim guidelines for the conservation of Antarctic fauna and flora.' (Bush 1982b, p. 282). Annex D of the Agreed Measures outlined precautions that were to apply in the case of the 'accidental' introduction of parasites and diseases. It provided for the inoculation of dogs against disease, and a prohibition on the introduction of live poultry

after 1 July 1966. However, notwithstanding that several governments sought to give effect to these provisions through domestic legislation, the uncertain legally binding nature of the Agreed Measures combined with the soft language contained within Article IX raised doubts as to how uniformly these measures were implemented amongst the ATCPs (Joyner 1998).

16.2.2.1 Assessment

The 1964 Agreed Measures were an important step in the ATS developing a response to the introduction of disease into Antarctica and its impact upon fauna and flora. Given the state of the development of the ATS, and international laws dealing with wildlife protection, the Agreed Measures were well ahead of their time. When the various aspects of the Agreed Measures are combined, especially the subsequent developments of the protected area system following ATCM Recommendations, a reasonably comprehensive system for Antarctic environmental protection was created. However, the biggest failing of the Agreed Measures was in their implementation. This partly resulted from the fact that they did not have an independent existence of their own but rather that they fell directly under the umbrella of the Antarctic Treaty where also the legal status of Recommendations has been gray. In the case of disease, an additional problem existed due to the soft language contained within Article IX. These provisions were also not comprehensive and had too narrow a focus. However, the Agreed Measures provided a basis for important aspects of the Protocol which eventually superseded them and accordingly the parallels between the two instruments are of interest.

16.2.3 CCAS and CCAMLR

Following the entry of the Antarctic Treaty into force, the need to protect seals was first raised at ATCM 1 (1961) and Recommendation I–VIII encouraged the Treaty parties to implement measures to conserve the living resources of the Treaty area. More particular action was taken to combat sealing activities in the 1964 Agreed Measures. However, the limitations in the Agreed Measures with respect to sealing were quickly realised when a 1964 sealing expedition revived concerns that full-scale commercial sealing could recommence. Various interim Recommendations were adopted by the ATCPs until the conclusion of CCAS in 1972. On its face, CCAS does not deal with the health of seals in Antarctica; rather its focus is directed at the commercial harvesting of Antarctic seals. As harvesting has never been undertaken during the life of the Convention it has never been fully tested.

The 1980 Convention for the Conservation of Antarctic Marine Living Resources (CCAMLR 1980) was concluded to address concerns over unregulated fishing in the Southern Ocean and the potential for significant impact upon the marine environment. The Convention entered into force in 1982 and in 2008 has 31 parties.

CCAMLR is directed specifically to the protection and conservation of a marine resource. Instead of having a species focus, it concentrates on a marine ecosystem. This follows from the Convention being negotiated as a direct response to concerns over the exploitation of krill. To implement an ecosystem approach in resource management CCAMLR adopted a number of features which make it distinctive from other component instruments in the ATS. The Convention's area of application extends to '…Antarctic marine living resources of the area south of 60° South latitude and to the Antarctic marine living resources of the area between that latitude and the Antarctic Convergence which form part of the Antarctic marine ecosystem' (CCAMLR, Article I(1)). The significance in this is that for the first time the ATS recognised the Antarctic Convergence and its importance for the purposes of delimiting the maritime area subject to Antarctic influence. CCAMLR does not however extend to all activities that take place within that area. Rather it only applies to Antarctic marine living resources. These are defined as 'the populations of finfish, molluscs, crustaceans and all other species of living organisms, including birds found south of the Antarctic convergence' (CCAMLR, Article I(2)). CCAMLR seeks to achieve the 'rational use' of the living resources of the Southern Ocean and this is how the Convention interprets the term 'conservation' (CCAMLR, Article II(2)). Article II provides guidelines as to how 'rational use' is to be implemented when harvesting of Antarctic marine living resources takes place. Through implementing these objectives to safeguard not only those marine living resources subject to potential harvesting, but also the wider marine and related environment, CCAMLR adopts the 'ecosystem' approach to marine living resource management (Edwards and Heap 1981).

The Convention is supported by the CCAMLR Commission which serves both a secretariat role and adopts and implements the ecosystem approach to marine living resource management (CCAMLR, Article VII). Membership of the Commission is limited to original parties plus Acceding States during such time as they are engaged in research or harvesting activities within the Convention Area. The Commission has assigned to it a variety of roles, including conducting research and gathering data on Antarctic marine living resources, analysis of catch statistics, identification of conservation needs, and the implementation of systems of inspection so as to monitor the effect of the Convention (CCAMLR, Article IX). The work of the Commission has also been assisted by permanent and ad hoc working groups.

CCAMLR operates via the implementation of 'Conservation Measures' adopted by the Commission at its annual meetings and which are binding on the Convention parties (CCAMLR, Article IX(6)). A broad mandate exists to adopt Conservation Measures dealing with the health of Antarctic wildlife. In particular, the Commission has the capacity to take:

> *such other conservation measures as the Commission considers necessary for the fulfilment of the objective of this Convention, including measures concerning the effects of harvesting and associated activities on components of the marine ecosystem other than harvested populations (CCAMLR, IX(2)).*

To date CCAMLR Conservation Measures have had a particular focus on fisheries management, especially certain fish stocks in and around South Georgia, and

Patagonian toothfish (*Dissostichus eleginoides*). Measures have been taken to also deal with the regulation of mesh size, the limitation of seabird mortality, the impact of marine debris upon living resources, requirements for the consideration of a new fishery, and improved catch reporting systems (Molenaar 2001).

16.2.3.1 Assessment

CCAS and CCAMLR together seek to regulate marine living resource management in the Southern Ocean, the only exception being whaling which falls under the 1946 Whaling Convention (Whaling Convention 1946). CCAS has never really been tested and therefore it is in only speculation to consider the possible impact it could have upon the health of seals in Antarctica. This has not been the case with CCAMLR, which in recent years has been tested and through its overseeing, the CCAMLR Commission is constantly monitoring Antarctic and Southern Ocean marine living resource management. CCAMLR is one of the most progressive ATS instruments in terms of adopting an ecosystem approach and the precautionary principle. While much of the Convention is driven by its focus on marine living resource management – particularly from the perspective of the harvesting of Antarctic marine living resources – the Convention also has concerns for the wider marine environment impacted upon by marine living resource management. A recent illustration of this concern has been the CCAMLR focus on the impacts of by-catch in the Southern Ocean, and its consequences for seabirds such as the albatross. CCAMLR therefore has considerable potential to respond to issues concerning the health of Antarctic marine life.

16.2.4 1991 Protocol

The 1991 Protocol on Environmental Protection to the Antarctic Treaty (Protocol) is the most significant addition to the ATS since the adoption of CCAMLR (Blay 1992; Redgwell 1994; Rothwell 2000). The entry of the Protocol into force in 1998 creates for the first time an integrated environmental protection regime in Antarctica, incorporating many of the mechanisms established under the 1964 Agreed Measures on the Conservation of Antarctic Fauna and Flora, and Recommendations subsequently adopted by the ATCPs. It also creates a new Antarctic institution – the Committee for Environmental Protection (CEP) – to oversee implementation of the Protocol and provide advice to the ATCPs on environmental issues.

The Protocol's Preamble confirms its close relationship with the Antarctic Treaty. It reconfirms the importance of ensuring that Antarctica is used for peaceful purposes, that it does not become the scene of international discord and that the global and regional importance of Antarctic science remain a primary consideration. The Preamble also acknowledges the existence of the ATS and the impact of other component parts of the system, such as CCAMLR. The Protocol does not clearly

define its area of application, instead reference is made throughout to 'Antarctica', the 'Antarctic environment and dependent and associated ecosystems', and the 'Antarctic Treaty area'. What this confirms is the bias inherent in the Protocol towards protecting the Antarctic continent rather than the greater Antarctic and Southern Ocean region. However, while the operation of the Protocol may be technically limited to the Antarctic Treaty area, there remains a strong argument that it was the intention of the Protocol's framers to extend its operation beyond 60°S so as to include 'dependent and associated ecosystems' which are beyond that limit. Given the use made of the Antarctic convergence boundary by CCAMLR, and the clear ecosystem focus of both that instrument and the Protocol, any interpretation of certain provisions of the Protocol which refer to Antarctica's ecosystems would justifiably include the area beyond the Treaty area up to the limit of the CCAMLR boundary. The connection between the Antarctic Treaty area and the dependent and associated ecosystems is clearly made in Article 3(1) which provides:

> *The protection of the Antarctic environment and dependent and associated ecosystems... shall be fundamental considerations in the planning and conduct of all activities in the Antarctic Treaty area.*

The desire on the part of the ATS parties to add a strong environmental focus to the operation of the Treaty system is clearly demonstrated in Article 2 of the Protocol which provides:

> *The Parties commit themselves to the comprehensive protection of the Antarctic environment and dependent and associated ecosystems and hereby designate Antarctica as a natural reserve devoted to peace and science.*

This provision, introduces for the first time the notion that Antarctica is a 'nature reserve' (Blay 1992; Redgwell 1994). The designation of Antarctica as a natural reserve is a significant development. It is certainly symbolic and may be seen as reflective of the campaign to have Antarctica declared as a 'World Park' (Joyner 1992a; Rothwell 1990). While it could be argued that this designation has purely political implications (Watts 1992, p. 227), it does place a greater responsibility upon the Antarctic Treaty parties to maintain, protect and preserve the Antarctic environment including the wildlife of the region (Francioni 1993, p. 61). This creates implications for the balancing of the pursuit of scientific freedom and research in Antarctica, with the need for environmental protection. One commentator has taken the view that the 'former importance of the freedom of scientific research has to be balanced with the priority accorded to the preservation of the environment' (De Cesari 1996, p. 422) and this would seem in accord with the Protocol's provisions.

The Protocol's principal provisions are those dealing with environmental protection, the key aspects of which provide:

1. That activities in the Antarctic Treaty area are to be planned and conducted so as to limit adverse impacts on the Antarctic environment and dependent and associated ecosystems
2. That activities in the Antarctic Treaty area shall be planned and conducted so as to avoid effects on weather patterns and air and water quality, significant changes in the environment, impacts on the populations of species of fauna and flora,

further jeopardy to endangered or threatened populations, and degradation or risk to areas of significance
3. That activities which are undertaken shall be based on prior assessments of their potential impact and of their value for scientific research
4. That monitoring of ongoing activities shall take place to allow for assessment of their impact and to facilitate early detection of possible unforeseen effects (Protocol, Article 3(2))

These environmental principles are wide-ranging and have the potential, if strictly implemented, to impact upon all activities undertaken in Antarctica and the Southern Ocean from the simplest biological research project on Antarctic lichen to the construction of new scientific stations. The result is that for the first time a standard for the assessment of all human activity has been created by the Treaty parties which will have the effect of overriding fragmentary Recommendations dealing with this topic and a variety of national standards which had been established (Blay 1992, p. 389). Ultimately, however, the interpretation of this key provision is very dependent upon the legislative regimes and policy mechanisms adopted by individual ATCPs and how the Committee for Environmental Protection interpret these provisions.

16.2.5 Committee for Environmental Protection

The Committee for Environmental Protection formally met for the first time in 1998 at ATCM XXII in Tromsø. The Committee is made up of representatives of all parties to the Protocol and has the function of monitoring the implementation of the Protocol and reporting on its progress at ATCMs. While not having any power to enforce the Protocol, the Committee is designed as an expert body which is to assist the ATCPs in ensuring the effectiveness of the Protocol. To that end, it can provide advice on the need to update or strengthen the Protocol, the need for additional Annexes, the application and implementation of EIA procedures, means of minimizing or mitigating environmental impact of activities in the Antarctic Treaty area, inspection procedures, and the collection and evaluation of information relating to environmental protection (Protocol, Article 12(1)).

16.2.6 Matters Covered by Annex I (Environmental Impact Assessment)

A major feature of the Protocol is the provision in Annex I for Environmental Impact Assessment (EIA) to be carried out on all activities undertaken pursuant to ‘scientific research programmes, tourism and all other governmental and non-governmental activities’ for which notice is required to be given under Article VII of the Antarctic Treaty (Protocol, Article 8). The EIA process classifies activities on the basis of

whether they have one of three degrees of impacts upon the Antarctic environment or dependent or associated ecosystems:

1. Less than a minor or transitory impact
2. A minor or transitory impact
3. More than a minor or transitory impact

To that end, as noted above, the interpretation of the terms 'minor' and 'transitory', are matters of considerable significance. EIA under the Protocol requires the active participation of each Treaty party whose nationals, expeditioners, or corporations who wish to engage in activities in the Antarctic. This requires the enactment of domestic laws and policies to give effect to the Protocol's EIA processes which in turn requires flexibility and transparency. This point was emphasized as long ago as in 1996, when it was noted that:

> the nature and significance of possible environmental impacts could be affected by a range of variables including the nature, scale, location and timing of the activity; the experience of the organization of individuals conducting the activity; and other activities that have been or are being conducted in or near the area of the activity in question.…identifications and considerations of possible cumulative impacts is an important part of environmental impact assessment (ATCM XX 1996, paragraph 137).

The ATCPs have shown an increased understanding of the central importance of the EIA process to the Protocol and Resolutions have been adopted dealing with the methodology of comprehensive environmental evaluations (ATCM XXI 1997, Resolution 2).

16.2.7 Matters Covered by Annex II (conservation of Antarctic fauna and flora)

Annex II of the Protocol provides for the conservation of Antarctic fauna and flora. It effectively repeals and replaces the Agreed Measures while also expanding their reach. Of particular relevance is Article 4 dealing with the 'Introduction of non-native Species, Parasites and Diseases', which broadly provide that:

1. Non-native animals or plants are not to be introduced to Antarctica without a permit as per Appendix B
2. Dogs shall not be introduced and those already in Antarctica to be removed by 1 April 1994
3. Plants or animals introduced by way of permit are to be removed or disposed of by means that eliminate risk to native fauna or flora. other non-indigenous plants and animals are also to be removed in a similar manner
4. An exception in the case of the importation of food, other than: (a) no live animals are imported; (b) all plants and animal parts and products are kept under carefully controlled conditions
5. Precautions are to be taken to prevent the introduction of micro-organisms not present in native fauna and flora

This Article proved to be especially sensitive at the time of its negotiation, especially the requirement concerning the removal of all dogs. This imposed a particular obligation upon Australia as the only ATCP that used sledge dogs at its Antarctic stations. However, consistent with the Protocol and in advance of the required deadline, Australia removed all the dogs under its control in 1993 (Rothwell and Davis 1997, p. 162).

Of ongoing concern however, is the contemporary introduction of non-native species by all visitors to Antarctica, including scientists and especially tourists. It should be recalled that this issue was first addressed in the 1964 Agreed Measures, while in 1995 it was recommended that all ATCPs examine their facilities to identify non-native species introduced by human activities, remove such species unless they are present in accordance with a permit, and take necessary action to ensure tourists and non-government activities do not result in the introduction of non-native species (ATCM XIX 1995). Further, in 1996 it was noted that non-native species were being introduced into Antarctica via certain non-native grass species and ATCPs were again encouraged to make thorough checks of their facilities (ATCM XX 1996, paragraph 125).

A number of comments can be made concerning Article 4 of Annex II and its focus on controlling the introduction of disease into Antarctica. First, it has taken a strict approach to the presence of dogs in Antarctica and provided for their complete removal. Secondly, non-indigenous domestic plants and laboratory animals and plants (including viruses, bacteria, yeasts and fungi) may only be introduced into Antarctica under permit. Permits can specify not only the number or type of such species, but also set precautions 'to prevent escape or contact with native fauna and flora' (Protocol, Annex II, Article 4(3)). The permits however are ultimately regulated by each ATCP and there is the potential for variable practices to develop. Thirdly, non-indigenous plants or animals which have entered Antarctica by permit are to be removed or disposed of to ensure that there is no risk to native fauna or flora. The obligation upon the ATCP to ensure that these requirements are met is unspecified. Fourthly, all other non-indigenous plants and animals which have been introduced into Antarctica (and are not dealt with by other provisions above), are to be removed from Antarctica so that the area is rendered sterile unless it is determined that they pose no risk. Who makes the determination that such animals or plants pose no risk is uncertain. Who also has the obligation to remove? Is it the ATCP or other State which introduced them? Does the claimant State have an inherent interest in this type of activity? Fifthly, food is exempt from the general provisions with the exception of live food. All plants, animal parts and products are subject to the provisions of Appendix C and Annex III – Waste Management. Appendix C contains particularly strict provisions for the importation of poultry products into Antarctica, including requirements of inspection for disease. However, it again remains unclear as to who has the responsibility for conducting these inspections. A further curiosity of the provision is that it does not make clear that diseased poultry is not to be brought into Antarctica – if that ever was the original intention of the framers of the Annex. Sixthly, precautions are to be taken to prevent the introduction of micro-organisms not present in native fauna and flora. Here the

choice of the word 'precautions' is curious. While each party to the Protocol is to 'require' that 'precautions' are undertaken, this does not suggest a strong legal obligation and is a reflection of the desire of parties to keep this commitment at the policy level only.

16.2.8 Matters Covered by Annex III (waste disposal and management)

Annex III deals with Antarctic waste disposal and management. This Annex, which applies to all activities undertaken in the Treaty area 'pursuant to scientific research programmes, tourism and all other governmental and non-governmental activities in the Antarctic Treaty area for which advance notice is required under Article VII(5) of the Antarctic Treaty' (Protocol, Annex III, Article 1(1)) provides that:

> *The amount of wastes produced or disposed of in the Antarctic Treaty area shall be reduced as far as practicable so as to minimise impact on the Antarctic environment and to minimise interference with the natural values of Antarctica, with scientific research and with other uses of Antarctica which are consistent with the Antarctic Treaty (Protocol Annex III, Article 1(2)).*

The Annex seeks to establish a comprehensive scheme for the removal of waste from Antarctica (Protocol, Article 2), the incineration of waste (Protocol, Annex III, Article 3), disposal of waste on land and at sea (Protocol, Annex III, Articles 4, 5), and waste management planning (Protocol, Annex III, Article 8). The provisions dealing with the incineration of waste are particularly significant in the context of food and other products dealt with under Article 4 of Annex II. The solid residue resulting from incineration is to be removed from the Antarctic Treaty area.

16.2.9 Matters Covered by Annex IV (marine pollution)

Annex IV relates to the prevention of marine pollution. It applies not only to each Protocol party but also to ships entitled to fly its flag and other ships engaged in supporting a party's operations while within the Antarctic Treaty area (Protocol, Annex IV, Article 2). The Annex seeks to implement standards similar to those which are found in the 1973/78 International Convention for the Prevention of Pollution from Ships (MARPOL 73/78 1973; Joyner 1992b, p. 174). Provisions deal with the discharge of oil (Protocol, Annex IV, Article 3), noxious liquid substances (Protocol, Annex IV, Article 4), garbage (Protocol, Annex IV, Article 5), and sewage (Protocol, Annex IV, Article 6). The application of this Annex extends to each party to the Protocol, ships that fly their flags, and ships which are engaged in or supporting their Antarctic operations (Protocol, Annex IV, Article 2). At ATCM XX in 1996 it was noted that non-native species were being introduced into Antarctica via ballast water (ATCM XX 1996, paragraph 125), and again at

ATCM XXII reference was made to this matter and the work of the International Maritime Organisation (IMO) in responding to the ballast water issue. At present, Annex III is not sufficiently wide in its scope to catch the discharge of ballast water in Antarctic waters notwithstanding the type of organisms that may be contained within that water. This is because Articles 3, 4, 5 and 6 of the Annex all address the discharge or disposal of substances which have been the subject of regulation under MARPOL. Ballast water has yet to reach that stage of control. To particularly address this issue the Annex requires amendment.

16.2.10 Matters Covered by Annex V (Antarctic Protected Area System)

Annex V was adopted at ATCM XVI (1991) shortly after the protocol was concluded. This Annex deals with the Antarctic Protected Area system and seeks to reorganise the previously existing system of area management under a single Annex. Two types of special areas are provided for: Antarctic Specially Protected Areas and Antarctic Specially Managed Areas. Antarctic Specially Protected Areas can be designated in order to protect any area, including a marine area, which has 'outstanding environmental, scientific, historic, aesthetic or wilderness values' (Protocol, Annex V, Article 3; Harris 1994). Areas which meet certain criteria are eligible for designation under this category, in addition to those areas that were previously designated as either Specially Protected Areas or Sites of Special Scientific Interest (Protocol, Annex V, Article 3). Entry into these areas is by permit only. Antarctic Specially Managed Areas are areas of the continent, including marine areas, where permissible activities have been and are being conducted. Such areas will probably have been subjected to heavy use resulting from interest in their scientific research potential or as a tourism destination. In order 'to assist in the planning and co-ordination of activities, avoid possible conflicts, improve co-operation between Parties or minimise environmental impacts' (Protocol, Annex V, Article 4) these sites can be designated as Specially Managed Areas. Entry into these sites is restricted to persons holding a permit. Before any site can be designated under either of these categories it is necessary for a Management Plan for the area to be submitted to an ATCM for approval. Management Plans are to include not only full details on the area, but also a clear description of the conditions under which permits for entry and codes of conduct for the use of the area may be issued (Protocol, Annex V, Article 5).

16.2.11 The CEP and the Health of Antarctic Wildlife

In addition to the range of matters under the Protocol with which the CEP must deal, it has a mandate to deal with matters related to the health of Antarctic wildlife (Protocol, Annex II). This was confirmed at the CEP's very first meeting in 1998

when it noted Australia's intention to host a workshop that year on the 'Introduction of Diseases to Antarctic Wildlife' and the interest amongst Committee members of the outcome of that process (ATCM XXII 1998, paragraph 34). The Australian-hosted workshop was held in August 1998 in Hobart and a report was duly prepared for CEP consideration in 1999 (Kerry et al. 1999; ATCM XXIII 1999). CEP II responded by creating an open-ended contact group to facilitate further dialogue on the issue between the ATCPs, SCAR and COMNAP with a view to consider the matter in further detail in 2000. The Terms of Reference for the group were:

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

1. *Diminish the risk of the introduction and spread of diseases to Antarctic wildlife*
2. Detect, determine the cause, and minimise the adverse effects of unusual wildlife mortality and morbidity events in Antarctica (CEP II 1999, paragraph 60)

The work of this group was carried out between 2000 and 2001 and a number of reports were prepared including submissions by SCAR and COMNAP (Australia 2000; CEP 2000). Australia coordinated a 2001 Report from the Intersessional Contact Group (Australia 2001) which was considered by CEP IV in 2001. The CEP's view was that 'the risk that human activities in Antarctica might introduce diseases was currently assessed to be very low' and it was considered that the work of the Intersessional Contact Group was complete (CEP IV 2001, paragraph 41). This seemingly brought the CEP's interest in this matter to an end for the time being.

However, since 2001 there have been some interesting developments which would suggest that the introduction of disease to Antarctic wildlife is not as comprehensively managed and regulated as first thought. First, in 2002 the CEP noted that differing approaches were being taken in protected area management plans with respect to the use of poultry products. While SCAR took the position that there was no causal link between poultry products and the introduction of Newcastle disease, nevertheless there was merit on a precautionary basis to place restrictions on the use of poultry products in areas which were being protected because of their value as sites for breeding birds (CEP V 2002, paragraph 77). In 2003 at ATCM XXVI/CEP VI consideration was given to the need for adjustment to the Protocol in terms of its internal consistency and adequacy in addressing the health of Antarctic wildlife. Particular attention was given to provisions dealing with the introduction of non-native species, the inspection of poultry products, parasite checks and the importation of non-sterile soil (CEP VI 2003, paragraphs 94–102). These matters remain under active consideration by the CEP.

16.2.11.1 Assessment

The Madrid Protocol sought to gather together many of the developments that had been taking place in the Antarctic environmental protection throughout the life of the ATS and bundle them together in one instrument. However, it also sought to update

the ATS with developments in international environmental law and to that end the endorsement of compulsory environmental impact assessment procedures is a very important step. In relation to the controls placed on the introduction of disease, Annex II of the Protocol reproduces many of the key elements found in the 1964 Agreed Measures. However, it does extend the reach of the Agreed Measures by seeking to completely eliminate dogs in Antarctica and to also more clearly deal with poultry. The Protocol also contains many other provisions which when combined place greater environmental obligations upon the Treaty parties to protect the Antarctic environment, and especially the fauna and flora. The potential operation of this obligation may also extend beyond the limits of 60°S. One aspect of the Protocol which may provide a further basis for protecting Antarctica against the introduction of disease may be the requirement of Article 8 for the conduct of EIA. There can be no question that the spread of disease amongst Antarctic fauna and flora would have 'more than a minor or transitory impact'; however, the difficulty with relying upon the EIA requirements is whether it is possible to develop EIA procedures which will be broad enough to catch disease. Nevertheless, for an ATCP wishing to take a strong environmental stand there is sufficient justification in the Protocol to implement tough measures for disease control. What is clear in this area is that the emerging role of the CEP as a 'watchdog' over the implementation of the Protocol is crucial.

16.3 International Environmental Law and Health of Antarctic Wildlife

A feature of international law during the past 30 years has been the growth in the development of international environmental law (Birnie and Boyle 2002). Many of these developments can be traced to the 1972 Stockholm Conference on the Human Environment, resulting in the adoption of the Stockholm Declaration. The more recent 1992 United Nations Conference on Environment and Development, which resulted in adoption of the Rio Declaration (Rio Declaration 1992), and the 2002 Johannesburg World Summit on Sustainable Development have politically sought to reinforce these developments. International environmental law has developed primarily by way of treaties and customary international law during this time. To that end, the many treaties negotiated during the past 30 years on species and habitat protection, pollution prevention, climate change and biological diversity have been especially significant.

16.3.1 Principles of International Environmental Law

As a result of the developments in international law, and particularly international environmental law, throughout the twentieth century, it is now possible to identify several core principles of international environmental law. These principles are

found in a variety of traditional international law sources (Birnie and Boyle 2002, pp. 79–82; Sands 1995a, pp. 104–126). The principles are:

- The obligation upon all States to conserve the environment and its natural resources
- The need for States to assess potential, and monitor actual environmental impact
- The need for international cooperation to conserve the environment both within and beyond areas of national jurisdiction (Kiss and Shelton 1991, pp. 145–154)

This list is not exhaustive. It may well be possible to identify other principles which are in a state of development, or which have particular application for specific environmental problems (Lang 1995; Birnie and Boyle 2002, pp. 97–150). However, the purpose here is to provide a general overview.

The obligation upon States to conserve the environment and natural resources has been recognised in numerous international instruments since the Stockholm Conference. Perhaps the most significant of these, given the number of participating States, is the 1982 United Nations Convention on the Law of the Sea (LOS Convention 1982). As a result of these provisions, it can be said that throughout international law it is now generally recognised that States have an obligation to prevent environmental harm (Rio Declaration, Principle 2). Having its foundation in the *Trail Smelter* case (Trail Smelter Arbitration 1939–1941), the effect of the principle is that States are not under a negative obligation to refrain from damaging the environment, but under a positive obligation to protect it. One manifestation of this obligation is the 'precautionary principle' or the 'precautionary approach'. While this approach has now found expression in a large number of international instruments, its scope is still indefinite. Principle 15 of the Rio Declaration is the most recent attempt to clarify the concept. It provides:

> *In order to protect the environment, the precautionary approach shall be widely applied by States according to their capabilities. Where there are threats of serious or irreversible damage, lack of full scientific certainty shall not be used as a reason for postponing cost-effective measures to prevent environmental degradation (Rio Declaration, Principle 15).*

However, while the adoption of Principle 15 is certainly an important step in universal recognition of the importance of the precautionary approach, there remains considerable scope for interpretation. Perhaps the most important issue is the degree of foreseeability. Are States under an obligation to protect the environment from all risks, whether they are foreseeable or not, or should a common law standard of foreseeability be applied? Kiss and Shelton argue that 'there is a duty to impose a regulatory regime on activities which could harm the environment of other States or areas outside the limits of their jurisdiction' (Kiss and Shelton 1991, pp. 129–130). Birnie and Boyle characterise this obligation as primarily one of 'diligent prevention and control' (Birnie and Boyle 2002, p. 115). While debate remains over how the concept is to be implemented, it is clear that as part of the obligation upon States to prevent harm, a State is also required to control an activity when the environmental consequences of that activity are unknown or uncertain. In the Antarctic context, the decision made in the Protocol to prohibit mining activities (Protocol, Article 7), is a clear illustration of the precautionary principle at work (Rothwell 1996, p. 401).

The second international environmental law principle is the duty to monitor the environment and to assess the risk of potential transboundary environmental damage from proposed or existing activities. States need to be aware of the state of the environment and the potential that their activities may have for environmental damage. This is especially the case with new activities which may not be considered environmentally harmful when first initiated. To a degree, these principles have developed from municipal law where environmental impact assessments began to be recognised as part of environmental and planning law in the 1960s (Kiss and Shelton 1991, p. 147). Initiatives developed through the United Nations Environment Programme (UNEP) have also seen environmental monitoring and prior impact assessment become essential features of international environmental risk management (Birnie and Boyle 2002, p. 132).

The third general principle of international environmental law, the need for greater international cooperation to deal with environmental problems both within and beyond areas of national jurisdiction, also has its roots in the *Trail Smelter* decision and the Stockholm Declaration (Stockholm Declaration, Principle 21). The United Nations has played an important role in implementing this principle. Through bodies such as UNEP it has facilitated and sponsored a number of international conferences dealing with a range of specific, regional and global environmental problems. This principle has found expression in a number of bilateral and multilateral conventions dealing with issues ranging from marine pollution (LOS Convention, Article 197) to nuclear accidents as well as in the Rio Declaration (Rio Declaration, Principles 5, 7, 9, 12, 14, 27).

An emerging norm of international environmental law is that of sustainable development. The definition of sustainable development most commonly used is that which is found in the report of the World Commission on Environment and Development, *Our Common Future* which provided that:

> *Sustainable development is development that meets the needs of the present without compromising the ability of future generations to meet their own needs. It contains within it two key concepts:*
>
> - *The concept of 'needs', in particular the essential needs of the world's poor, to which overriding priority should be given*
> - The idea of limitations imposed by the state of technology and social organization on the environment's ability to meet present and future needs (World Commission on Environment and Development 1987, p. 87)

Over 15 years since it was first proposed, debate still exists over the precise content and meaning of sustainable development. However, it is possible to identify four core components:

- The principle of intergenerational equity
- The principle of sustainable use
- The principle of equitable use
- The principle of integration (Sands 1995b, pp. 58–62)

The norm continues to be developed through the work of the Commission for Sustainable Development and by international conferences.

16.3.2 General International Environmental Conventions

There exist a number of international treaties which create potential obligations for States with interests in Antarctica that can be interpreted as relevant when dealing with disease control and the health of Antarctic wildlife. The 1972 Convention for the Protection of the World Cultural and Natural Heritage (World Heritage Convention 1972) creates a range of obligations upon State parties to protect areas considered to be a part of the world cultural and natural heritage. Particular obligations are imposed with respect to properties placed on the so-called 'World Heritage List' (which presently includes some sub-Antarctic islands such as Heard and McDonald Islands), but in addition there exist a range of general obligations upon State parties for the protection and conservation of natural heritage. There has been an ongoing speculation about the application of the World Heritage Convention to the Antarctic continent, however the basic principles that it promotes are clearly consistent with the goals of environmental protection and management found within the Antarctic Treaty System.

The 1973 Convention on International Trade in Endangered Species of Wild Flora and Fauna (CITES 1973) establishes a means for seeking to control the export of endangered species. However, while CITES assists in the protection of endangered species it does not allow for controls over activities which may be harmful to such species – such as the introduction of disease into Antarctic wildlife, or overfishing of endangered Southern Ocean fish stocks. Nevertheless, in recent years there have been attempts to achieve CITES listing of some Southern Ocean species in order to ensure their protection (Rothwell and Stephens 2004, p. 170).

16.3.3 Environmental Conventions Regulating Disease

There has been a long-standing interest in the international community throughout the course of the twentieth century to regulate disease affecting not only human health but also disease affecting the fauna, including farm animals, and the flora. For example, the 1924 International Agreement for the Creation of an International Office for Dealing with Contagious Disease of Animals at Paris (Contagious Disease Agreement 1924) was an early attempt by the international community to regulate this issue collectively and it was followed by several other initiatives (Convention Against Disease of Animals 1935; Convention Concerning Transit of Animals 1935).

More recently, the 1951 International Plant Protection Convention has sought to control and prevent the introduction of pests and diseases of plants and plant products (IPPC 1951). This Convention establishes some core obligations and encourages international cooperation with bodies such as the FAO, but ultimately relies upon national organisations for plant protection to ensure domestic implementation. In relation to animal diseases, the 1973 Agreement for the Establishment of a Regional

Animal Production and Health Commission for Asia, the Far East and the South-West Pacific also sought to control disease in livestock production amongst member States (Regional Animal Production Agreement 1973).

The most prominent recent international agreement which has implications for the control of disease and the health of Antarctic wildlife is the 1992 United Nations Convention on Biological Diversity (CBD 1992). This Convention has the primary purpose of attempting to create a number of core obligations upon the State parties to conserve biological diversity and ensure its sustainable use (CBD 1992, Article 1). Specific obligations are created for both in-situ and ex-situ conservation of biological diversity. Of these, the most significant for the purposes of controlling disease is found in Article 8 dealing with in-situ conservation. It provides that:

> *Each Contracting Party shall, as far as possible and as appropriate:*
> *…*
>
> (g) *Establish or maintain means to regulate, manage or control the risks associated with the use and release of living modified organisms resulting from biotechnology which are likely to have adverse environmental impacts that could affect the conservation and sustainable use of biological diversity, taking into account the risks to human health*
> (h) *Prevent the introduction of, control or eradicate those alien species which threaten ecosystems, habitats or species*

These provisions represent a clear obligation upon States to control the introduction of non-indigenous species and organisms that may result in disease that threatens ecosystems and species. It is a global obligation and its scope clearly extends to Antarctica. It is significant not only because it provides further support for some of the provisions of the Protocol, but also because its reach is much greater than the Protocol in terms of States which are bound by the obligation. While the provisions do not make express reference to 'disease' its language is sufficiently wide to allow for its inclusion. The clear intent in the Convention is to prevent the introduction of non-indigenous species that will cause environmental impact. Further provisions of the Convention also provide requirements for the conduct of environmental impact assessment and related obligations for the control of organisms (CBD, Articles 8, 9, 14).

16.4 Conclusion

Antarctica has a very complex legal regime which has developed throughout the past 50 years in response to concerns driven by the need to promote scientific research, set aside sovereignty disputes, and increasingly protect the environment. These are matters of interest to not only the Antarctic claimant States, or the members of the Antarctic Treaty, but to the whole international community. 'A case can be made for the proposition that Antarctica nevertheless has many of the features of a common heritage regime, but such a view remains controversial and does not take full account of the complex legal and political status of that continent, nor of the

absence of any scheme for sharing resources.' (Birnie and Boyle 2002, p. 144). Environmental protection and management, however, has become a significant priority for the ATCPs and this is reflected not only in the political decisions over the rejection of the CRAMRA regime and in its place support the Protocol, but in the reality of how the ATS operates post-1991 in a CEP era where environmental principles are paramount.

The legal structures for protection of the Antarctic environment, and for the health of Antarctic wildlife are impressive. Significant steps have been taken throughout the life of the ATS to increasingly focus on environmental conservation and management with the Protocol now seen as the 'state of the art' in the field. Developments in related areas such as international environmental law, and disease protection and management also assist to reinforce the regime. However gaps do remain. The Protocol has a focus on State-actors which means that its principal impact is upon governments and their scientific programs. Non-State actors, such as tourists and the commercial operators which bring them to Antarctica, are only subject to national legal regimes which in the case of Antarctica are variable in their content and enforcement. Rather, 'Codes of Conduct' and 'Guidelines' for tourism operations in Antarctica which seek to protect Antarctic wildlife assist to reinforce the spirit of the Protocol (IAATO 2002). The Protocol is also not comprehensive in its scope and recent suggestions for its amendment and adjustment need to be viewed seriously if it is going to be able to keep abreast of new challenges. Likewise, as with any international regime, much depends on the consistency and adequacy of implementation by individual States. That major outbreaks of disease in Antarctic wildlife have yet to be detected should not be taken as a sign that the problem does not potentially exist. Increasing tourism and expanded scientific expeditions will all result in greater potential for the introduction of disease in Antarctica. International law would suggest that the continued application of the precautionary approach will be fundamental in the protection and management of Antarctic wildlife.

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Chapter 17
The Antarctic Treaty System and Wildlife Health: Disease Awareness, Prevention and Response

M. J. Riddle

17.1 Introduction

The Antarctic Treaty System has long recognised the potential risk to Antarctic wildlife from human mediated introduction or spread of disease (reviewed by Rothwell, this volume). Precautions to reduce the risk were included in the first environmental measures adopted by the Treaty Parties in 1964 (ATCM III 1964), specifically, dogs imported into the Treaty Area were required to be inoculated against distemper, contagious hepatitis, rabies and leptospirosis. These precautions were strengthened by the Protocol on Environmental Protection to the Antarctic Treaty, 1991, which prohibits the introduction of non-native species, parasites and diseases, except with a permit, and then only for laboratory plants, animals and micro-organisms, and domestic plants. The Environmental Protocol also requires that imported poultry products be inspected for evidence of disease, such as Newcastle's disease, tuberculosis and yeast infection, and that all residues of imported animals, plants and micro-organisms should be removed from the Treaty Area or treated to make them sterile.

The Antarctic Treaty System's early response to the risk to wildlife of human-mediated disease was either to establish very general prohibitions, or to be surprisingly specific with the naming of a few particular diseases to guard against. What was missing until recently is a systematic, risk-based treatment. In 1998, with this gap in mind, Australia hosted an international workshop to identify the potential for disease incursion and spread in Antarctica's wildlife, to develop a series of recommendations to reduce the risk of such introductions and to limit the consequences of any disease establishment and spread. This chapter summarises the outcomes of that workshop and their subsequent progress in the Antarctic Treaty System.

M.J. Riddle
Australian Antarctic Division, Channel Highway, Kingston TAS, 7050, Australia
e-mail: martin.riddle@aad.gov.au

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17.2 Workshop on Diseases of Antarctic Wildlife

The sequence of events leading to the workshop on diseases of Antarctic wildlife, held in Hobart in 1998 is recounted by Rothwell (this volume). The outcomes of the workshop were reported in summary (Australia 1999) to the Second Meeting of the Antarctic Treaty's Committee for Environmental Protection (CEP II) under the following major headings.

- 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 we do if introduced disease is suspected?

CEP II responded to this summary report from the workshop (Australia 1999) by agreeing that when all Parties, SCAR and COMNAP have had the opportunity to consider the full report of the workshop, an open-ended contact group should be formed to present an initial report on matters arising from the workshop (CEP II 1999).

The full report of the workshop (Australia 2000) was presented by Australia at CEP III and included 19 specific recommendations (reproduced here as Appendix C). The Scientific Committee for Antarctic Research (SCAR) and the Council of Managers of National Antarctic Programs (COMNAP) also presented a joint working paper (SCAR and COMNAP 2000) in which they provided qualified support to most of the recommendations, at the same time stating that the scientific data available from the Antarctic, and used as the basis for the recommendations, are currently inadequate and do not in themselves justify any action.

CEP III (2000) noted that there has not yet been a disease outbreak in Antarctic wildlife directly attributed to human activity, but that this should not prevent the Parties from taking a precautionary approach to disease introduction. It was also stressed that it would be advisable to increase awareness and scientific knowledge about diseases in Antarctic wildlife, aimed at identifying possible risks so that appropriate measures could be taken to prevent them. The Committee agreed that the work of the intersessional open-ended contact group set up at CEP II should continue and the following revised terms of reference were agreed upon – that the contact group prepare an initial report for CEP IV which:

- Provides a review of the introduction and spread of infectious disease causing agents by human activity in Antarctica and provides a risk assessment of those activities which may introduce or spread disease causing agents in Antarctica
- Presents practical measures that might be implemented by the Parties to diminish the risk of the introduction and spread of infectious disease causing agents by human activity in Antarctic wildlife
- Presents practical measures that may be implemented to determine the cause of unusual wildlife mortality and morbidity events in Antarctica and to reduce the likelihood that human activity may exacerbate these events

17.3 Report to CEP on the Review and Risk Assessment of Disease Introduction and Spread

The work of the intersessional contact group on diseases of Antarctic wildlife was presented to CEP IV at St Petersburg, Russia, in 2001 by Australia in two reports. The first was a review and risk assessment addressing the first of the terms of reference for the intersessional contact group (Australia 2001a). The second considered practical measures to diminish risk (Australia 2001b). On receiving the reports, CEP IV determined that the risk that human activities in Antarctica might introduce diseases was assessed to be very low and agreed that the work of the intersessional contact group was complete (CEP 2001).

The review and risk assessment (Australia 2001a) is printed in full in this volume as Appendix D and is summarised below.

17.3.1 Risk Assessment Methodology

The report to CEP IV concluded that there is insufficient information available to conduct a reliable quantitative risk assessment of disease introduction and spread to Antarctic wildlife, but that a qualitative risk assessment approach should be sufficient to indicate priorities for precautionary measures. Historically, no diseases have been demonstrated to have been introduced to Antarctic wildlife or spread by human activities. However, no systematic studies of disease in Antarctica had been undertaken and it is therefore unlikely that conclusive evidence of human involvement in disease events would be available.

17.3.2 Historic Information on Disease

The report noted that several unusual mortality events in which disease was suspected have been recorded for Antarctic or sub-Antarctic wildlife but only one, a seal mass mortality on the Auckland Islands [Roe, in this volume] had been well investigated, and the causes of it and the others remain unknown. Serological evidence indicates that Antarctic wildlife has been exposed to a variety of agents that cause antibody reactions that are the same as or similar to those caused by known infectious disease causing agents, indicating that they are not completely naïve populations with respect to disease. Captive Antarctic birds [Woods et al this volume] and seals [McFarlane et al this volume] have exhibited symptoms of a variety of diseases known in other wildlife populations, indicating that they are susceptible to a range of diseases.

Disease is suspected in a significant number of the marine mammal mass mortality events reported in non-Antarctic regions. Most of the diseases on ‘List A of transmissible diseases with the potential for very serious and rapid spread’ of the Office International

des Epizooties (OIE), the world organisation for animal health, occur in countries that participate in Antarctic activities. This indicates that, despite the economic incentives to prevent them and the large preventive effort, serious transmissible diseases of animals occur in most countries. Most of the diseases in the OIE List A would not be transmissible to birds and seals; however, there is evidence that birds and seals are susceptible to some, such as Newcastle disease and avian influenza. In recent years, Newcastle disease has occurred widely in countries operating in Antarctica.

The report concluded that the diseases most likely to be at risk of introduction and spread by people are those that are established in the home countries of the people visiting Antarctica, can survive well without a host, do not require a vector that is not present and can infect different hosts; examples include Newcastle disease, avian influenza and the morbilliviruses causing canine and phocine distemper. It is not possible to identify all diseases with the potential for introduction and this is not necessary as a precursor to implementation of precautions.

17.3.3 Factors that Could Influence Disease Introduction or Spread

The report identified several factors that could influence disease introduction or spread in the Antarctic. Some places have environmental conditions that are similar to parts of the Antarctic and mechanisms for disease transfer that occur in those places may also occur in Antarctica. Animal behaviour will influence the likelihood of disease transmission within populations and between species. For example, several Antarctic species disperse widely, many to regions beyond the Antarctic where they could be in contact with disease-causing agents carried by other wildlife and in human waste at sewage effluent outfalls and waste disposal tips. Carrion feeders are most likely to be in direct contact with diseased or dying animals of other species and opportunist scavengers are most likely to feed on waste generated by human activity if precautions are not taken to prevent access. Skuas were identified as being among the most likely species to be the point of entry of disease from waste because they are not shy of people and they will scavenge on station waste given the opportunity. They are also among the most likely routes of transfer to other species because of their habit of associating with other species. The cold and lack of available water may make otherwise simple precautions difficult or impossible under some circumstances such as at remote field locations.

17.3.4 Human Activities Which may Introduce or Spread Disease

The report identified that activities undertaken before going to Antarctica, including precautions, will determine whether people bring infectious disease with them. Activities in Antarctica identified as being most likely to cause disease introduction or spread are those that involve close contact with wildlife or those that allow wildlife to come in contact with waste generated from human activities. Certain combinations

of activities may also significantly increase the risks and precautions should be prioritised to target the most likely pathways of disease introduction or spread. Human activities identified as priorities for practical measures to diminish risk were,

- Feeding of wildlife
- Actions following discovery of unusual mortality events
- Research that involves handling of Antarctic animals, particularly research on disease
- Import of food, particularly poultry products
- Waste disposal and sewage treatment
- Use of equipment and clothing before departure to Antarctica
- Serial visits to wildlife aggregations

17.3.5 Report to CEP on Practical Measures to Diminish Risk

The report on practical measures to diminish risk (Australia 2001b) by the intersessional contact group on diseases of Antarctic wildlife was presented as a draft to CEP IV (2001) but was not developed further. On receiving the two reports CEP IV determined that the work of the contact group was complete.

The following general approaches, originally suggested by the workshop on disease of Antarctic wildlife held in Hobart in 1998, were used by the Intersessional Contact Group as the framework for developing practical risk-reduction measures. The full report from the contact group is included in this volume as Appendix E.

17.3.6 Education and Awareness

The report noted that the success of all the other measures depends on their acceptance and adoption by people visiting Antarctica and that the measures will not be effective unless the requirement for them is disseminated. In addition, if people understand the reasons behind the concern they will be better prepared to make appropriate decisions if presented with an unpredicted situation which has implications for disease introduction or spread.

Antarctic operators were encouraged to include an explanation of the potential for disease introduction and translocation, and simple procedures that should be adopted to reduce the possibility in pre-departure or in-transit briefings.

17.3.7 Initial Response to Unusual Mortality Events

Unusual wildlife mortality events are by their nature unpredictable and it is unlikely that an event will be discovered by someone with previous experience of such occurrences. The report noted that it would be unwise to leave decisions on how to react to those

discovering a mortality event and that most people do not know normal mortality rates among Antarctic species and therefore may not recognise unusual mortality. For these reasons, information to help recognise unusual mortality is required as well as guidance on response. A probable first reaction to discovery of an unusual mortality event would be to quickly check other localities to determine the spatial extent of the event. Under these circumstances, moving from location to location without some precautions could cause translocation of infection agents.

If disease is suspected the first response should be to stand back, view widely, photograph (preferably digitally), count dead and dying, and note any obvious abnormal characteristics that can be seen from a distance, such as inability to stand, swellings or skin lesions. As soon as possible, this information should be sent to Antarctic wildlife experts with expertise sufficient to determine whether the number of dead and dying and the characteristics of affected animals are within normal limits. Access to the site should be restricted to reduce the risk of transfer to uninfected populations until advice is received on whether the mortality event is unusual or likely to be caused by disease.

17.3.8 Information Exchange

Exchange of information is an important aspect of most response plans for unusual wildlife mortality events developed for other regions. The Antarctic Treaty System and associated organisations (such as SCAR and COMNAP) have established structures for information exchange – the report recommended the use of these structures. To be effective in reducing the likelihood of human exacerbation of unusual mortality events, reporting to alert others must occur quickly using proven information networks such as the Antarctic Environmental Officers Network and the International Association of Antarctica Tourist Operators (IAATO) to disseminate information.

17.3.9 Cleaning and/or Sanitising of Equipment

Cleansing of clothing, equipment and vehicles is commonly used as a precaution against the transfer of disease-causing agents in other parts of the world, particularly when moving from a location in which a disease is known to be present. The report noted that simple cleaning of surfaces by steam cleaning or brushing with detergent solution is effective in removing viruses and is necessary for removing grease and organic dirt prior to any subsequent chemical decontamination, if this is required. Micro-organisms vary in their susceptibility to disinfectants and the best disinfectant will depend on characteristics of the disease-causing agent, the characteristics of the equipment or clothing, and the circumstances in which they will be used.

The report recommended cleaning of equipment and footwear shortly before departure to Antarctica, and in Antarctica under some circumstances, such as when

moving from the vicinity of one discrete population to another or if visiting from a ship, between each landing. Cleansing procedures after activities that involve close contact with wildlife, such as research, or when disease is suspected, should be more stringent and may require the use of stronger disinfectants.

17.3.10 Source of Food Supplies

The potential for introduction of disease causing agents through food products to Antarctica is recognised by the Antarctic Treaty System and precautions are included in the Madrid Protocol, including a requirement to inspect poultry products before shipment to Antarctica.

The report established that it was not within its scope to specify the details of meat industry inspection. Procedures are established and enforced by appropriate authorities in each country and the World Health Organisation, the World Trade Organisation and the Office International des Epizooties (OIE) advise on some international aspects of standards. It is important that meat that would not be accepted by other markets is not sent to Antarctica and that meat and animal products sent to Antarctica should be procured from industry certified suppliers with documented quality assurance procedures covering the entire supply chain from primary producers, through slaughter and meat processing to the wholesale and retail outlets. These quality assurance procedures should satisfy all the domestic sanitary regulations established to reduce transfer of disease causing agents of the country sending the products to Antarctica or the highest export standards achievable by the meat industry in the country, whichever is the higher.

The report also suggested that Antarctic operators, whether operators of national programs or tourist operators, should take steps to ensure that they are aware of animal disease outbreaks occurring within the area from which they procure meat and meat products.

17.3.11 Waste Management, Sewage Treatment and Effluent Disposal

The Antarctic Treaty System recognises the potential for transfer of pathogens to Antarctic wildlife from waste generated by Antarctic activities. The Madrid Protocol addresses the risk in its annexes that deal with conservation of fauna and flora, waste disposal and waste management, and prevention of marine pollution. These annexes provide directions on the disposal of imported animal and plant products and micro-organisms by procedures that render them sterile.

The report identified several activities that are associated with a greater risk of exposing Antarctic wildlife to potential pathogens in waste food. Feeding of food scraps to Antarctic wildlife is the most direct means by which pathogens could be

introduced by people and should be explicitly prohibited. Kitchen and field camp waste should be stored at all times in secure containers designed to prevent access by scavengers, such as skuas, and as a precaution, uncooked waste meat and meat scraps should be boiled for 20 min before disposal if there is any chance that scavengers can feed on the scraps. Melt water produced from thawing meat and meat products should be boiled before disposal to domestic sewage systems that discharge effluent into the Antarctic environment.

17.3.12 Research Priorities

The report noted that relatively little is known about disease and disease processes in Antarctic wildlife. Available information indicates that Antarctic wildlife species carry a diversity of potential pathogens and display immune reactions to many other disease-causing agents that have not yet been isolated. Further investigations of the spatial and temporal patterns of disease-causing agents (including serological evidence) within Antarctic species, comparisons of the type and diversity of disease-causing agents among animals that spend their entire life within the Antarctic region and those that migrate to other continents, and the development of a tissue bank that in the event of a disease incident could be used to do retrospective analyses for evidence of historic occurrences of disease causing agents, could all contribute to the understanding of the causes of unusual mortality events when they occur. Research priorities for helping reduce the risk of human involvement in disease introduction or spread include investigations of pathogen survival in the Antarctic environment, tests of the effectiveness of footwear, equipment and vehicle cleaning methods and tests of the effectiveness of sewage treatment and effluent disposal in reducing the pathogen load released into the environment.

17.3.13 Recent Practical Developments

IAATO responded quickly to the potential risk of disease introduction and spread. In 1998, IAATO reported to CEP I that it would continue their practice of requiring visitors to clean their boots and check clothing before and after each landing, and that boot-washing stations were standardized on all tour vessels (IAATO 1998). In 2000, IAATO reported that it had adopted a protocol to report any high mortality incidents and to avoid the introduction and translocation of alien diseases by decontamination of boots, clothing and equipment (IAATO 2000) and these initiatives were developed as part of their mandatory operational procedures for all members (IAATO 2005). In the absence of available information, IAATO supported research on the efficacy of chemical disinfection of boots (IAATO 2002) which concluded that boot washing with the addition of the disinfectant Virkon S was more efficient than using seawater alone (Curry et al. 2002, 2005).

In 2002 Australia reported to CEP V (2002) on a response plan (Australia 2002) developed to provide guidance for people working in Antarctica on what to do if sick or dead animals are discovered in unusually high numbers or with signs of disease (reproduced as Appendix F, this volume). The plan was designed to reduce the likelihood of people unwittingly spreading the infective agent if a disease is involved and to provide a clear organisational structure for managing the response. Central to the response plan is the provision of an Unusual Mortality Response Kit to each of Australia's Antarctic stations, which includes a copy of the response plan, personnel protective equipment, data recording equipment, sampling equipment and protocols for collection of samples for pathological analysis based on protocols developed by CCAMLR (reproduced as Appendix A this volume).

In 2002, CEP V established an intersessional contact group (CEP V 2002) to review Annex II (Conservation of Antarctic flora and fauna) of the Protocol on Environmental Protection to the Antarctic Treaty, 1991 which includes provisions to reduce the chances of the introduction of diseases. This review was still under way in 2007, with the main points of discussion being about whether the scope of the annex should be extended, and the legal and policy implications of such an extension (Russia 2007). As part of this review, the CEP recommended to the ATCM (CEP VII 2004) that the requirement to inspect poultry products for evidence of disease, such as Newcastle disease, tuberculosis and yeast infection should be replaced with a more general requirement to make all appropriate efforts to ensure that poultry or avian products imported into Antarctica are free from contamination by diseases which might be harmful to the native flora and fauna.

In 2005, Australia provided a working paper to CEP VIII on measures to address the unintentional introduction and spread of non-native biota and disease to the Antarctic (Australia 2005). At the same meeting, related information papers were provided by the International Union for the Conservation of Nature (IUCN 2005) and IAATO (IAATO 2005). A subsequent workshop on non-native species in the Antarctic (New Zealand 2006) reported back to CEP IX with the following six major recommendations that were strongly supported by the Committee.

- The issue of non-native species should be given the highest priority consistent with the high environmental standards set out in the Protocol; a 'zero tolerance approach'.
- CEP should take the lead on this issue.
- CEP should give consideration to sharing information with, and seeking advice from, other bodies, notably SCAR, CCAMLR, COMNAP, IAATO, IUCN and other organisations as appropriate (e.g. International Maritime Organisation).
- Dedicated research is required to improve the understanding of, inter alia, existing biological and genetic diversity, species distributions and bio-geographic zones, the potential implications of a warming climate and the identification of high-risk areas and ecosystems; particular research attention needs to be given to microbial communities and marine ecosystems.
- To the extent possible, concerns about non-native species issues should be built into existing procedures and practices; notably EIA procedures and the protected areas system.

- A set of comprehensive and standardised guidance and/or procedures should be developed, aimed at all operators in the Antarctic, based on a 'Prevention, Surveillance, Response' approach.

At the same meeting of the CEP an intersessional contact group (ICG) was established to develop a 5-year work plan (CEP IX 2006) for the CEP with the objective of prioritising its workload and to ensure it was well positioned to deal with Antarctica's future environmental challenges. As part of this process, the ICG used a risk-based approach to prioritise the environmental challenges and developed a timetable for addressing the issues based on the priority rating (New Zealand 2007). The introduction of non-native species was ranked among the highest priority environmental issues on a provisional listing accepted by CEP X (2007), indicating that the risk of introduced species, including disease-causing agents, is now well accepted.

17.4 Conclusions

Although there is as yet no conclusive evidence that human activity has been responsible for the introduction or spread of any disease among wildlife in Antarctica, the international Antarctic community has made significant progress developing and implementing precautionary measures. When the issue was first raised, the Antarctic tourism industry, through its representative body IAATO, moved quickly to introduce their own guidelines and procedures ahead of any externally imposed requirements in this respect, and at the time of writing, they were in advance of the Antarctic Treaty System. Some individual national programs have played an important leadership role by undertaking research, implementing their own precautionary measures, developing contingency plans and reporting regularly on the issue to the Antarctic Treaty System through the CEP. In this way, the potential risk to the flora and fauna of introduced species, including disease, has now become widely recognised as a priority for those responsible for environmental stewardship of the Antarctic.

Acknowledgements The recommendations reported here from the workshop on diseases of Antarctic wildlife, held in Hobart 1998, are the product of the participants at the workshop. Many people from the international Antarctic community contributed to the reports of the Intersessional Contact Groups reported here. I thank you all for your willingness to share your expertise. I accept full responsibility for any errors of transmission.

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Appendix A
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 sub-clinical levels and manifest in periods of stress or changes in circumstances in the colony. Overt disease, usually recognised by deaths, is obvious. Sub-clinical 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.

Reproduced from: CCAMLR (2004) CCAMLR Ecosystem Monitoring Program: Standard Methods for Monitoring Studies, Part IV, Section 6. CCAMLR, Hobart, Australia

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
- 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
- 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
- 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 2–3 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 Appendix B this volume]

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 2–3 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 0–4°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 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, 5, 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, 5, 10 ml*
Blood tubes holders*

Needles 21, 23, 27 gauge 1 in.*
Nunc tubes for storage of serum
Slide box
Syringes 3, 5, 10 cc*

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Appendix B
Protocols for Collecting Samples for Toxicological Analyses

The following procedure describes the methods for collecting and storing samples of animal tissues in the event that pollutants or toxic substances are suspected in species being monitored as part of CEMP.

Samples should be collected and analysed for organochlorine compounds such as polychlorinated biphenyls (PCBs), dichloro-diphenyl-trichloroethane (DDTs), lindane, polycyclic aromatic hydrocarbons (PAHs) and heavy metals (cadmium, mercury, lead, zinc and copper). It should also be appreciated that chemical content in seabirds may be related to diet and lifestyle and is naturally occurring.

It is recommended that all field teams conducting CEMP programs maintain stocks of sampling equipment at their monitoring site to allow adequate collection, storage and transport of samples for the following laboratory analyses.

The analyses of samples for contaminants involve sophisticated and expensive techniques and therefore require support from appropriate specialised centres.

Sampling Guidelines

Chlorinated Hydrocarbons

The body burden of chlorinated hydrocarbons can be evaluated from muscle and/or fatty tissue, skin biopsies, unhatched eggs, blood, preen gland oil and stomach contents. Collect a minimum of 2 g of tissue or skin and a few microlitres of preen gland oil. If the animal is dead, collect in addition liver, muscle and brain. Post-mortem sampling should be carried out on recently-dead individuals, with records of biometric parameters and times of death and sampling attached.

Reproduced from: CCAMLR (2004) CCAMLR Ecosystem Monitoring Program: Standard Methods for Monitoring Studies Part IV, Section 5. CCAMLR Hobart Australia

Heavy Metals

Ante-mortem collection of feathers, faeces and skin biopsies is suitable. Post-mortem sampling of recently-dead animals can also include liver and kidney.

Biochemicals

The modification of specific biochemical responses (i.e. enzymes and metabolites) may indicate the presence of pollutants in seabirds. These analyses can be correlated with those carried out on samples collected as described above. The following table summarises the biological samples suitable for specific biochemical tests:

Test	Sample
Porphyrin (COPRO-URO-PROTO)	Faeces, feathers, liver, blood (whole)
Mixed-function oxidases: Ethoxyresorufin-O-deethylase (EROD) Penthoxyresorufin-O-deethylase (PROD) Benzyloxyresorufin-O-deethylase (BROD) Benzopyrene-monooxygenase (BPMO) CYT-P450-reductase	Liver, skin biopsies
Esterases: Acetylcholinesterase (AChE) Butyrylcholinesterase (BChE)	Brain, blood (whole for mammals, and serum or plasma for birds and fish)

Collection and Storage of Samples

All samples should be collected into glass containers or tubes which can be sealed so they do not dehydrate in storage.

Samples for heavy metals and chlorinated hydrocarbon analyses should be stored as soon as possible at −20°C. Care should be taken to prevent contamination of samples – in the case of heavy metals, by metallic compounds in the sampling tubes (e.g. metal tops) and in the case of hydrocarbons, by plastics (e.g. plastic wrapping material).

Samples for biochemical analyses should be stored promptly in liquid nitrogen; it is very important for further successful laboratory analyses to freeze the samples immediately.

All samples should be labelled to provide details of sample, the identity of the individual animal and date of collection. It is important to ensure that tissue from the same animal may be matched in the laboratory. A detailed logbook should be maintained and forwarded with the samples.

Summary Table B.1

	Sample type (amount)	Collection	Storage	Important
OC	Fatty tissue, skin biopsies (>2 g) Preen gland oil (a few mL) Liver, brain (post mortem; >2 g)	Glass/PE Tubes/pots	−20°C (it is permitted to keep samples at about 0°C for >12 h until storage at −20°C)	Do not use tube/pots made with chlorine materials
HM	Feathers, faeces, skin biopsies (2 g) Liver, kidney (post mortem; 2 g)	Glass/PE Tubes/pots	−20°C (it is permitted to keep samples at about 0°C for >12 h until storage at −20°C)	Do not use aluminium foil or metal materials
BR:				
P	Feathers, faeces, liver, blood	Glass/PE Tubes/pots	Liquid nitrogen (after freezing they can be stored at −80°C)	Do not keep samples at room temperature
MFO	Liver, skin biopsies	Glass/PE Tubes/pots	”	”
E	Brain, blood	Glass/PE Tubes/pots	”	”

OC = organochlorine compounds; *HM* = heavy metals; *BR* = biochemical responses; *P* = porphyrin; *MFO* = mixed function oxidases system; *E* = esterases; *PE* = polyethylene

Appendix C
Recommendations Arising from the Workshop on Diseases of Antarctic Wildlife, Held in Hobart, Australia, on 25–28 August 1998[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.

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.

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[1]Editors' note: These recommendations are reproduced from the full report of the Hobart workshop, submitted by Australia to VII Special Antarctic Treaty Consultative Meeting/CEP III as an appendix to their working paper WP6.

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.

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.

Response to Suspected Disease Occurrence

Procedures for recording disease events in Antarctic birds have been published by CCAMLR (CEMP Standard Methods 1997)[reproduced as Appendix A this volume] 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 organisations 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.

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.

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 D
Report on the Open-Ended Intersessional Contact Group on Diseases of Antarctic Wildlife Report 1 – Review and risk assessment[1]

Background

CEP III agreed to the following terms of reference for the open-ended intersessional contact group (ICG) on diseases of Antarctic wildlife:

'That the contact group prepare an initial report for CEP IV which:

- *Provides a review of the introduction and spread by human activity of infectious disease causing agents in Antarctica and provides a risk assessment of those activities which may introduce or spread disease causing agents in Antarctica*
- *Presents practical measures that might be implemented by Parties to diminish the risk to Antarctic wildlife of the introduction and spread by human activity of infectious disease causing agents; and*
- *Presents practical measures that may be implemented to determine the cause of unusual wildlife mortality and morbidity events in Antarctica and to reduce the likelihood that human activity may exacerbate these events.'* (CEP III Report, paragraph 52)

This paper reports on the work of the ICG in response to the first of the terms of reference. The ICG's report is at Annex 1. Australia coordinated the process, with participation from AEON, ASOC, IAATO, Italy, Norway and Sweden.

The review and risk assessment were used by the ICG to identify those human activities that are a priority for practical measures to diminish the risk to Antarctic wildlife from the introduction and spread by human activity of infectious disease causing agents. The ICG seeks CEP endorsement of the list of activities identified as priorities and will then complete work on practical measures to diminish risk.

[1] Editors' note: This report on the work of the ICG is reproduced from Working Paper 10, submitted by Australia to XXIV Antarctic Treaty Consultative Meeting/CEP IV in July 2001 and includes the review originally produced as an annex to the working paper and an attachment to the annex which outlines the risk assessment process used.

A draft report prepared by the ICG in response to the second of the terms of reference on practical measures is submitted as an annex to a separate working paper. The ICG does not yet have a draft report in response to the third of the terms of reference.

Outcome of Review and Risk Assessment

The ICG reached a number of conclusions on the basis of the review and risk assessment.

Risk Assessment Methodology

1. There is insufficient information available to conduct a reliable quantitative risk assessment of disease introduction and spread to Antarctic wildlife.
2. A qualitative risk assessment approach should be sufficient to indicate priorities for precautionary measures.

Historic Information on Disease

3. No diseases have been demonstrated to have been introduced to Antarctic wildlife or spread by human activities.
4. No systematic studies of disease in Antarctica have been undertaken and it is unlikely that conclusive evidence of human involvement in disease events would be available.
5. There is recent evidence to indicate that some microorganisms have been introduced to Antarctic wildlife and spread as a consequence of human activity.
6. Seven unusual mortality events in which disease was suspected have been recorded for Antarctic wildlife. Only one was investigated and the causes of the others are not known.
7. A seal mass mortality event on the Auckland Islands in 1998 was well investigated but the causal agent is still not known with certainty, indicating that identification of the cause of a mortality event is not always possible.
8. Clinical and serological evidence indicates that many Antarctic and sub-Antarctic penguins and seals have been exposed to infectious disease causing agents, indicating that they are not completely naïve populations with respect to disease.
9. Captive Antarctic birds and seals have exhibited symptoms of a variety of diseases known in other wildlife populations, indicating that they are susceptible to a range of diseases.

10. Disease is suspected in a significant number of the marine mammal mass mortality events reported in non-Antarctic regions.
11. Most of the diseases on the Office International des Epizooties (OIE), the world organisation for animal health, List A of transmissible diseases with the potential for very serious and rapid spread occur in countries that participate in Antarctic activities. This indicates that, despite the economic incentives to prevent them and the large preventive effort, serious transmissible diseases of animals occur in most countries.
12. Most OIE List A diseases would not be transmissible to birds and seals, however, there is evidence that birds and seals are susceptible to some, such as Newcastle disease and avian influenza.
13. Newcastle disease has occurred widely in ATCP countries in recent years and may be the disease most likely to be a risk to Antarctic wildlife.
14. Diseases most likely to be of risk of introduction and spread by people are those that are established in the home countries of people visiting Antarctica, can survive well without a host, do not require a vector that is not present and can infect different hosts, examples include Newcastle disease, avian influenza and the morbilliviruses causing canine and phocine distemper.
15. It is not possible to identify all diseases with the potential for introduction and this is not necessary as a precursor to implementation of precautions.

Factors that Could Influence Disease Introduction or Spread

16. Environmental conditions in parts of the Antarctic are similar to conditions elsewhere and so mechanisms for disease transfer that occur in these places are likely to also occur in Antarctica.
17. The cold and lack of available water may make otherwise simple precautions difficult or impossible under some circumstances such as at remote field locations.
18. Animal behaviour will influence the likelihood of disease transmission within populations and between species.
19. Several Antarctic species migrate beyond the Antarctic to regions where they could be in contact with disease causing agents carried by other wildlife and in human waste at sewage effluent outfalls and waste disposal tips.
20. Carrion feeders are most likely to be in direct contact with diseased or dying animals of other species.
21. Opportunist scavengers are most likely to feed on waste generated by human activity if precautions are not taken to prevent access.
22. Skuas are among the most likely species to be the point of entry of disease from waste because they are not shy of people and they will scavenge on station waste given the opportunity. They are also among the most likely routes of transfer to other species because of their habit of associating with other species.

Human Activities Which may Introduce or Spread Disease

23. Activities undertaken before going to Antarctica, including precautions, will determine whether people bring infectious disease with them.
24. Activities in Antarctica most likely to cause disease introduction or spread are those that involve close contact with wildlife or those that allow wildlife to come in contact with waste generated from human activities.
25. Certain combinations of activities may significantly increase the risks.
26. Precautions should be prioritised to target the most likely pathways of disease introduction or spread.
27. Human activities identified as priorities for practical measures to diminish risk are,

    - Feeding of wildlife
    - Actions following discovery of unusual mortality events
    - Research that involves handling of Antarctic animals, particularly research on disease
    - Import of food, particularly poultry products
    - Waste disposal and sewage treatment
    - Use of equipment and clothing before departure to Antarctica
    - Serial visits to wildlife aggregations.

Recommendations

It is recommended that:

- The CEP accepts the attached report (Annex 1) from the ICG in fulfilment of the requirement to provide CEP with a review of the introduction and spread by human activity of infectious disease causing agents in Antarctica and to provide a risk assessment of those activities which may introduce or spread disease causing agents in Antarctica
- The CEP notes the conclusions of the ICG
- The CEP considers the list of human activities identified by the ICG as priorities for practical measures to diminish risk and, if appropriate, endorses these as the basis for further work by the ICG on practical measures to diminish the risk to Antarctic wildlife of the introduction and spread by human activity of infectious disease causing agents
- The CEP encourages Parties, COMNAP, SCAR, CCAMLR and other expert bodies such as IUCN to nominate relevant specialists to participate in the continued work of the open-ended contact group

ANNEX 1

Review of the Introduction and Spread by Human Activity of Infectious Disease Causing Agents and Risk Assessment of Those Activities Which may Introduce or Spread Disease Causing Agents in Antarctica

Contents

1 Methodology for Review and Risk Assessment

The review and risk assessment process included the following steps,

1. Agreement on the risk assessment approach to be used (a discussion of the risk assessment procedure used is included as Attachment 1)

Editors' note: This review was written by Martin J. Riddle with contributions from the Antarctic Environmental Officers Network, the Antarctic and Southern Ocean Coalition and the International Association of Antarctica Tourism Operators and representatives from Italy, Norway and Sweden. Enquiries should be directed to the author (MJR).

2. Review of historic information on wildlife diseases in Antarctica and elsewhere to determine if particular diseases should be a concern, including,
   a. diseases known to have been introduced to Antarctic wildlife or spread by human activity
   b. documented wildlife mass mortality events in Antarctica and the sub-Antarctic
   c. indications that Antarctic and sub-Antarctic wildlife have been exposed to infectious disease causing agents
   d. diseases considered a risk to wildlife in other regions
   e. characteristics of disease that influence their risk
3. Assessment of characteristics of the Antarctic environment and biota to determine,
   a. whether there are particular characteristics that increase the chance of disease introduction
   b. whether particular species are at greater risk
4. Assessment of human activities to determine whether there are particular activities that have an increased chance of causing introduction or spread of disease
5. Identification of combinations of activities (scenarios) that increase risk

2 Diseases that may be a Risk to Antarctic Wildlife

2.1 Diseases Known to have been Introduced to Antarctic Wildlife or Spread by Human Activity

No diseases have been demonstrated to have been introduced to Antarctic wildlife or spread among them as a consequence of human activity. The epidemiology of disease in Antarctic wildlife has been little studied and on the basis of information currently available, it is unlikely that past disease events could have been attributed unequivocally to the activities of people. To date there have been no concerted studies designed to determine the origin of disease agents in Antarctic wildlife or their mode of introduction. Recent evidence indicates that some microorganisms have been introduced to Antarctic wildlife and spread as a consequence of human activity (Broman et al. 2000, Palmgren et al. 2000).

In other regions of the world significant resources are directed towards determining the cause of disease outbreaks, often without success. However, despite the lack of direct proof of human involvement in many disease events, humans are recognised as potential disease vectors and appropriate precautions are taken. The absence of evidence for the past involvement of people in disease introduction in Antarctica is not evidence that people have not been involved or that they could not be involved in future.

2.2 Documented Wildlife Mass Mortality events in Antarctica and the Sub-Antarctic

Disease has been suspected in six recorded unusual mortality events of birds and one of seals in the Antarctic Treaty area. There have been few cases where a disease has been expressed and the cause identified. An exception is the case of avian cholera, *Pasteurella multocida* (strain 1-X73), in which four pairs of the brown skua, *Catharacta lonnbergi*, died suddenly on Livingston Island (Parmelee, 1979). The disease has also been observed on more than one occasion on sub-Antarctic Campbell Island where *P. multocida* has been isolated from dead rockhopper penguins (de Lisle et al. 1990). A 90% mortality of banded brown skuas at Admiralty Bay on King George Island in 1981 was reported as being similar to the mortality on Livingston Island (Trivelpiece et al. 1981) but no evidence for cause was reported.

38 adult sub-Antarctic skuas, *Catharacta antarctica*, were found dead at Hope Bay on the Antarctic Peninsula in 1990 (Montalti et al. 1996). The animals showed no unusual pathological signs but no analyses for disease agents were undertaken.

37 sheathbills, *Chionis alba*, were found dead in the vicinity of Factory Cove on Signey Island between July and October 1965 (Howie et al. 1968). Bacteriological, histological and parasitological examinations of three carcasses were negative. Extreme weather conditions could have contributed to some of the deaths and poisoning from chemical waste from a station was also suggested as a possible cause.

Several hundred gentoo penguin chicks were found dead on Signy Island, Antarctica (MacDonald and Conroy 1971). The symptoms were described as similar to the viral disease puffinosis that occurs in Manx shearwaters (*Puffinus puffinus*). Body condition appeared to be good, however, all had multiple ulcers, 2–4 mm in diameter, on the dorsal surfaces of their feet. Many were found lying face down and those that were still alive were unable to stand unaided. The causal agent was not identified. Adélie and chinstrap penguins in adjacent colonies were not affected.

Large numbers of plump and apparently well-nourished Adélie penguin chicks were found dead and dying at Low Tongue approximately 40 km west of Mawson in February 1972 (Kerry et al. 1996). 65% of chicks had died recently and many of those still alive were found face down and could not stand on their own. The cause of death was not investigated at the time and remains unknown.

At least 1,500 crabeater seals, *Lobodon carcinophagus,* were found dead in the Crown Prince Gustav Channel, Antarctic Peninsula in 1955 (Laws and Taylor 1957). All affected seals had swollen necks and blood running from their mouths, on dissection their guts were empty, their livers pallid and pus oozed from the neck glands when incised (Fuchs 1982). The cause was suspected to be a highly contagious virus possibly exacerbated by stress from crowding and partial starvation as a result of being trapped by ice. The cause of death was not investigated and remains unknown.

A mass mortality of New Zealand sea lions, *Phocarctos hookeri,* on the New Zealand sub-Antarctic Auckland Islands, in January–February 1998 (Gales and Childerhouse 1999) is better documented than any of the events that occurred in the Antarctic. About 1,600 pups and an unrecorded number of adults died. At the start

of the event dead pups were in good condition (plenty of fat) but as the event progressed more lean and apparently starving pups were found. Pups had few clinical signs of disease although some showed paralysis in the hind limbs that appeared to be associated with an abscess. Other clinical signs were noted but these could have been secondary. The most common symptom of the adults was swelling in the throat region that appeared to be caused by an extensive abscess in the tissue surrounding the salivary gland. Some animals also had a number of raised swellings, about 1 cm in diameter in the ventral region of the body. A few adults were apparently paralysed in the hind limbs as seen in the pups. Animals were autopsied and samples of tissue, serum, milk and faeces were collected. Examination included gross pathology, histopathology, virology, serology, parasitology and chemical analysis for organochlorine pesticides. Other investigations included analysis for algal biotoxins and documentation of oceanographic conditions. A previously unidentified bacterium (Campylobacter like) is thought to have been the primary pathogenic agent, however, despite the thorough investigation, the cause remains uncertain. This illustrates the difficulty in identifying causal agents for mass mortalities.

These events indicate that mass mortalities occur in Antarctic and sub-Antarctic wildlife and that unless samples are collected during or soon after the event there is very little likelihood of identifying the causal agent. The experience on the Auckland Islands demonstrates that even after intense sampling and investigation by skilled people with appropriate expertise the causative agent may not be identified. If the causative agent is not known it is not likely that humans could be implicated or disregarded with confidence as agents of introduction or spread of the causative agent.

2.3 Indications that Antarctic and Sub-Antarctic Wildlife have been Exposed to Infectious Disease Causing Agents

Evidence from clinical examination, pathology and serology indicates that Antarctic and sub-Antarctic wildlife have been exposed to a variety of infectious disease causing agents in the past (Table 1). Much of the evidence is based on antibody reactions and in most cases there were no clinical signs of disease. Serological evidence, such as antibody reactions, is not conclusive proof of past exposure to infectious disease causing agents. To confirm the presence of a disease causing agent it must be isolated, however, isolation of an agent does not prove that it has caused disease.

Serological evidence (Table 1) indicates that Antarctic wildlife have been exposed to a variety of agents that cause antibody reactions that are the same as, or similar to, those caused by known infectious disease causing agents. The presence of antibodies also indicates that these species have active immune systems and have survived exposure to these agents.

There are no published accounts of systematic studies designed to determine whether humans have been involved in the introduction or spread of infectious disease causing agents to Antarctica. As a consequence, there is no conclusive evidence that human activity has or has not been responsible for the introduction to

Table 1 Evidence for exposure of Antarctic and sub-Antarctic birds and mammals to infectious disease causing agents (based on Clark and Kerry 2000 and other sources)

Disease causing agent and associated disease	Host species and location	Type of evidence	Reference
Bacteria and fungi			
Borrelia burgdorferi sensu lato (Lyme disease)	King penguins (Crozet)	Antibodies	Gauthier-Clerc et al. (1999)
Salmonella	Adélie penguins (Ross Island) and south polar skuas	Isolated	Oelke and Steiniger (1973)
Salmonella enteritidis	Gentoo penguins (Bird Island)	Isolated	Olsen et al. (1996)
Salmonella enteritidis	Fur seals, black browed albatross, gentoo penguins	Isolated	Palmgren et al. (2000)
Chlamydia sp	Emperor penguin (Auster) and rockhopper, royal and gentoo penguins (Macquarie Island)Brown skua	Antibodies	Moore and Cameron (1969), Cameron (1968)
Chlamydia psittaci		DNA detection	Herman et al. (2000)
Pastuerella multocida (avian cholera)	Rockhopper penguins (Campbell Island)	Isolated	Lisle et al. (1990)
	Brown skua (Palmer)	Mortality, agent isolated	Parmelee et al. (1978)
Brucella sp (brucellosis)	Weddell and fur seals	Antibodies	Retamal et al. (2000), Blank et al. (2000)
Campylobacter jejuni	Birds and seals (South Georgia)	Isolated	Broman et al. (2000)
Mycobacterium (tuberculosis)	Fur seal	Pathology, isolated	Bastida et al. (1999)
Viruses			
Avian paramyxovirus (Newcastle disease)	Adélie and royal penguins	Antibodies	Morgan et al. (1978)
Non-pathogenic paramyxovirus strains	Royal and king penguins	Isolated	Morgan and Westbury (1988)
	Adélie penguins	Antibodies	Morgan and Westbury (1981)
Avian influenza (influenza A)	Adélie penguins (Casey)	Antibodies	Morgan and Westbury (1981)
	Adélie penguins and Antarctic skuas (Ross Sea)	Antibodies	Austin and Webster (1993)
Flaviviruses	Various penguins (sub-Antarctic)	Antibodies	Morgan et al. (1985)
Birnavirus (infectious bursal disease virus or Gumboro disease)	Shearwaters, Adélie and emperor penguins	Antibodies	Gardner et al. (1997)
Avian adenovirus	Rockhopper penguins	Antibodies	Karesh (1999)

(continued)

Table 1 (continued)

Disease causing agent and associated disease	Host species and location	Type of evidence	Reference
Avian encephalomyelitis virus	Rockhopper penguins	Antibodies	Karesh (1999)
Coronavirus (infectious bronchitis virus)	Rockhopper penguins	Antibodies	Karesh (1999)
Avian reovirus	Rockhopper penguins	Antibodies	Karesh (1999)
Unknown virus (puffinosis)	Gentoo penguins (Signy Island)	Clinical signs similar to those of puffinosis	MacDonald and Conroy (1971)
Morbilliviruses			
Canine distemper virus	Leopard and crabeater seals (Antarctic Peninsula)	Antibodies	Bengtson and Boveng (1991)
Herpesviruses			
European phocine herpesvirus	Weddell and crabeater seals (Weddell Sea)	Clinical signs of respiratory disease and antibodies to herpesvirus	Harder et al. (1991)
Phocine herpesvirus	Weddell seal	Antibodies	Stenvers et al. (1992)

the Antarctic region of the agents causing the antibody reactions or the pathological or clinical signs observed in Antarctic wildlife.

The occurrence of disease among captive representatives of Antarctic species (Table 2) indicates that, under certain conditions, these animals are susceptible to, and will express the symptoms of, diseases known from non-Antarctic regions.

2.4 Diseases Considered a Risk to Wildlife in Other Regions

There is an enormous literature reporting diseases and the occurrence of infectious disease causing agents among wild stocks of non-Antarctic species of seals and penguins and other sea mammals and sea-birds. It is not possible or necessary to undertake a complete review of this literature. Examples are provided to illustrate the range of diseases reported on the basis of clinical signs, pathology and antibody reactions (Table 3). Clearly elsewhere in the world many diseases are circulating actively among birds and marine mammals.

There have been suggestions that some of these diseases are linked to human activity, such as exposure of wildlife to domestic animals (Barrett et al. 1995), pollution (Harve et al. 1999), however, these links are very difficult to prove and there is little conclusive evidence for human involvement. One notable exception is a controlled experiment that indicated pollution might have contributed to the severity and extent of recent morbillivirus infections among seals (Osterhaus et al. 1995).

Of 22 marine mammal mortality events reported by the US National Marine Mammal Fisheries Service (of NOAA) for the period 1978–1996 (Wilkinson 1996) bacterial or viral diseases were implicated in 9, algal biotoxins were implicated in 5, environmental extremes (El Niño) were implicated in 2, oil spill or toxic discharge were implicated in 2, gun shot was the cause of 1 and the causes of 4 were not determined (disease and biotoxins were together implicated as the cause of one event). Bacterial or viral diseases have therefore been implicated in nearly half these mass mortality events. Two cases were identified as influenza A virus in seals, two as phocine distemper virus (a morbillivirus), 4 cases as an undetermined morbillivirus (3 in dolphins and 1 in seals) and one case as the bacterial disease, leptospirosis, in sea lions.

The Office International des Epizooties (OIE), the world organisation for animal health, has a list of 15 transmissible diseases (OIE List A) which have the potential for very serious and rapid spread, irrespective of national borders, which are of serious socio-economic or public health consequence and which are of major importance in the international trade of animals and animal products (Table 4). Reports on these diseases must be provided to the OIE when the disease first occurs and at monthly intervals until the area is declared free of the disease. The OIE also has a list of over 90 transmissible diseases (OIE List B) which are considered to be of socio-economic and/or public health importance within countries and which are significant in the international trade of animals and animal products.

Fourteen of the 15 OIE List A diseases have occurred within the 27 Antarctic Treaty Consultative Party (ATCP) countries during the period 1996–1999 for which

Table 2 Evidence of infectious disease in captive Antarctic birds and mammals

Disease causing agent and associated disease	Host species	Evidence	Reference
Protozoa			
Plasmodium (avian malaria)	Penguins	Histopathology	Stoskopf and Beier (1979)
	King penguin	Clinical signs, agent isolated	Penrith et al. (1996)
Coccidia (coccidiosis)	Common seals	Clinical signs, histopathology, agent isolated	Munro and Synge (1991)
Bacteria and Fungi			
Salmonella	Penguins		Cockburn (1947)
Pastuerella multocida (avian cholera)	Unspecified seals		Lynch (1999)
Non-specific bacterial infection (bumblefoot)	Penguins	Clinical signs	Gailey-Phipps (1978), Stoskopf and Beall (1980)
Clostridium Perfringens	King penguins	Clinical signs, agent isolated	Penrith et al. (1996)
	Gentoo penguins	Clinical signs, agent isolated	Fielding (2000)
Aspergillus (aspergillosis)	Penguins	Presence of spores	Stoskopf and Beall (1980)
	Gentoo penguins	Clinical signs, agent isolated	Fielding (2000), Flach et al. (1990)
Viruses			
Avian paramyxovirus	Adélie penguin	Clinical signs of Newcastle disease	Pierson and Pfow (1975)
(Newcastle disease)	King penguin	Isolated	Krauss et al. (1963)
Herpesvirus-like	Blackfooted penguins	Clinical signs, isolated and electron microscopy	Kincaid et al. (1988)

Table 3 Evidence of infectious disease in wild stocks of non-Antarctic seals, and penguin and other marine mammals and seabirds

Disease causing agent and associated disease	Host species	Evidence	Reference
Ectoparasites			
Nasal mites	Young fur seals	Presence	Kim et al. (1980)
Endoparasitic worms			
Nematodes – gastric	Seals	Presence	Baker (1987), Baker (1989)
Nematodes – hookworm	Seals	Presence	Abegglen et al. (1958), George-Nascimento et al. (1992), Lyons et al. (1997)
Nematodes – lungworm	Seals	Presence	Ridgeway et al. (1972)
Microfilaria			
Protozoa			
Giardia	Ringed seals (Arctic)	Antibodies	Olson (1997)
Bacteria and Fungi			
Vancomycin resistant Enterococci	Black headed gulls (Sweden)	Isolated	Sellin et al. (2000)
Brucella (Brucellosis)	Many marine mammals including seals, whales, dolphins	Antibodies, agent isolated	Tryland et al. (1999), Jensen et al. (1999), Garner et al. (1997)
Salmonella	Californian sea lions, northern fur seals	Isolated	Gilmartin (1979), Baker et al. (1995), Stroud and Roelke (1980)
Antibiotic resistant Salmonella	Black headed gulls (Sweden)		Palmgren et al. (1997)
Leptospirosis (meningoench ephalomyelitis)	California sea lions, northern fur seals	Isolated and antibodies	Dierauf et al. (1985), Smith (1977)
Mycobacterium tuberculosis (tuberculosis)	New Zealand and Australian fur seals, Australian sea lions	Isolated	Forshaw (1991), Cousins et al. (1993), Romano et al. (1995)
	Arctic marine mammals		Tryland (2000)
Mycoplasma	Northern hemisphere seals	Isolated and inoculation	Geraci et al. (1984)
Borrelia burgdorferi s.l.	Puffins (northern hemisphere)	Isolated	Gylfe et al. (1999)
Aspergillus	Little penguin (Australia)		Obendorf and McColl (1980)
Viruses			
Avian paramyxovirus (Newcastle disease)	Double crested cormorants	Clinical signs, agent isolated, antibodies	Meteyer et al. (1997), Glaser et al. (1999)

(continued)

Table 3 (continued)

Disease causing agent and associated disease	Host species	Evidence	Reference
	Little penguins	Antibodies	Morgan et al. (1985)
Avian influenza (influenza A)	Harbour seals (New England)	Pathology, isolation and inoculation	Geraci et al. (1982), Geraci et al. (1984), Callan et al. (1995)
	Ring billed gulls, common terns	Antibodies, isolated agent and electron microscopy	Graves (1992), Becker (1966)
Influenza B	Harbor seals (Dutch coast)	Antibodies and virus isolated	Osterhaus et al. (2000)
Birnavirus (infectious bursal disease virus or Gumboro disease)	Fleshy footed shear water, sooty tern, silver gull	Antibodies	Wilcox et al. (1983)
Calicivirus (San Miguel sea lion virus)	Grey Seals (North Atlantic)	Isolated and electron microscopy	Stack et al. (1993), Barlough et al. (1986)
(Vesicular disease)	White tern	Clinical signs, DNA probe	Poet et al. (1996)
Parapox virus	Grey Seals (North Atlantic)	Isolated and electron microscopy.	Stack et al. (1993), Simpson et al. (1994), Nettleton et al. (1995)
	Manx shearwater	Clinical signs, agent isolated, electron microscopy, inoculation	Nuttal et al. (1985)
Rabies virus	Ringed seals (Svalbard)	Isolated and inoculation	Odegaard (1981)
Adenovirus (viral hepatitis)	California seal lions	Isolated and electron microscopy	Brit et al. (1979), Dierauf (1981)
Herpervirus	Harbor seals	Serology, isolated and electron microscopy	Osterhaus et al. (1985)
Unknown virus (puffinosis)	Manx shearwaters	Clinical signs	Harris (1965)
Morbilliviruses phocine distemper virus and canine distemper virus	Lake Baikal seals	Mass mortality	Grachev et al. (1989), Mamaev (1995), Barrett et al. (1995)
	Harbour and grey seals (North and Baltic Seas)	Isolated	Osterhaus et al. (1988)
	Harp seals (Arctic)	Antibodies	Goodhart (1988), Dietz et al. (1989), Markussen and Have (1992), Barrett et al. (1995)
	Monk seals (west Africa)	Antibodies, agent isolated	Osterhaus et al. (1997)
	Caspian seals	Serology, PCR	Kennedy et al. (2000)

Table 4 The OIE List A of transmissible diseases with potential for very serious and rapid spread irrespective of national borders, which are of serious socio-economic or public health consequence and which are of major importance in the international trade of animals and animal products

Disease and causative agent	Hosts	Mode of transmission	Number of ATCP countries experienceing outbreaks (and number of outbreaks in ATCP countries) during 1996–1999
Foot and mouth disease Family: Picornaviradae Genus: Aphthovirus	Most livestock	Direct and indirect contact (droplets). Animate vectors (humans). Inanimate vectors (vehicles etc), airborne (60 km overland, 300 km over sea)	6 (6 691)
Vesicular stomatitis Family: Rhabdoviridae Genus: Vesiculovirus	Humans Domestic: horses, sheep and pigs Wild: white tailed deer and many small tropical mammals	Contamination by transcutaneous or transmucosal route Arthropod transmission	4 (866)
Swine vesicular disease Family: Picornaviradae Genus: Enterovirus	Humans Pigs	Direct contact or contact with excretions from infected animals Faecal contamination Meat scraps and swill	1 (62)
Rinderpest Family: Paramyxoviridae Genus: Morbillivirus	Cattle, sheep, goats and pigs Many species of wild animal	Direct or close indirect contact	1 (1)
Peste des petits ruminants Family: Paramyxoviridae Genus: Morbillivirus	Sheep and goats Captive wild ungulates	Direct contact between animals	1 (248)
Contagious bovine pleuropneumonia Bacterial, Mycoplasma Mycoplasma mycoides	Cattle, zebu and water buffalo Wild bovids are resistant	Aerial, mostly by direct contact: droplets from coughing, saliva and urine	0 (0)
Lumpy skin disease Family: Poxviridae Genus: Capripoxvirus	Cattle, zebu, domestic buffalo, oryx, giraffe and impala	Infected saliva. No specific vector identified but flies and mosquitos could play a role	1 (909)

(continued)

Table 4 (continued)

Disease and causative agent	Hosts	Mode of transmission	Number of ATCP countries experienceing outbreaks (and number of outbreaks in ATCP countries) during 1996–1999
Rift Valley fever	Cattle, sheep, goats, camels, rodents, wild ruminants	Haematophagous mosquitoes of many genera.	1 (1)
Family: Bunyaviridae	African monkees and domestic carnivores	Direct contact when handling infected animals and	
Genus: Phlebovirus	Humans very susceptible	meat	
Bluetongue	Sheep (as disease) also in cattle, goats,	Biological vectors Culicoides spp.	9 (1,973)
Family: Reoviridae	camels and wild ruminants as inap-		
Genus: Orbivirus	parent infection		
Sheep pox and goat pox	Sheep and goats	Direct contact, inhalation, subcutaneous inocula-	3 (2,148)
Family: Poxviridae		tion; indirect transmission by contaminated	
Genus: Capripoxvirus		implements, vehicles or products; insects as mechanical vectors	
African horse sickness	Reservoir host unknown; usual host	Not directly contagious; usually transmitted by	1 (259)
Family: Reoviridae	are horses, mules, donkeys, zebra;	Culicoides spp, occasionally by mosquitoes and	
Genus: Orbivirus	occasionally elephants, onager, camels, dogs	ticks	
African swine fever Unclassified DNA virus, with characteristics of Iridovirus and Poxvirus	Pigs including some wild pigs	Direct contact with sick animals; feeding on infected meat; soft ticks of the genus Ornithodoros; vehicles, implements, clothes	2 (140)
Classical swine fever	Pigs and wild boar	Direct contact with sick animals; vistors to infected	12 (1,506)
Family: Flaviviridae		areas and implements, vehicles; insufficiently	
Genus: Pestivirus		cooked waste food fed to pigs	
Highly pathogenic avian influenza	Isolated in chickens and turkeys; assumed all avians are susceptible	Direct contact with secretions especially faeces; contaminated feed, water, equipment, clothing;	2 (77)
Family: Orthomyxoviridae		carrier waterfowl and sea birds; eggs	
Genus: Influenzavirus A (subtypes H5 and H7)			
Newcastle disease	Many species of domestic and wild birds	Direct contact with secretions especially faeces;	19 (2,623)
Family: Paramyxoviridae		contaminated water, clothing, implements;	
Genus: Paramyxovirus		carriers in psittacine and other birds	

OIE notification data is currently available. The only List A disease not to have occurred in an ATCP country during the reporting period is contagious bovine pleuropneumonia. 22 of the 27 Antarctic Treaty Consultative Parties reported at least one of the List A diseases within this period. The OIE database records that there have been cases of at least one List A disease in the other five ATCP countries during the 50 years preceding the OIE notification period. Within the ATCP countries the most widely reported List A disease is Newcastle disease, which was reported by 19 ATCP countries in the period 1996–1999. 2,623 outbreaks of Newcastle disease were reported from ATCP countries in the period.

The occurrence of OIE List A diseases in ATCP countries indicates that infectious diseases of animals, with the potential for very serious and rapid spread, are occurring in domestic animal stocks and in wildlife populations in countries actively involved in Antarctic operations. Many of the List A diseases require the presence of specific vectors for transmission or for completion of their life cycle. Many of these vectors are not present in Antarctica and therefore these diseases are not likely to infect Antarctic animals. It is also likely that Antarctic wildlife would not be susceptible to many of the List A diseases even if they were to come in contact with them. For example, diseases that are known to be limited to particular animal groups, such as swine vesicular disease, may be less likely to make the switch to Antarctic species than diseases that are known to be capable of infecting diverse species. However, there is good evidence to indicate that Antarctic birds and seals could be susceptible to at least two of the List A diseases. Captive penguins have been diagnosed with the clinical signs of Newcastle disease (Pierson and Pfow 1975) and some non-Antarctic seals have been diagnosed with avian influenza (Geraci et al. 1982).

Many important wildlife diseases do not appear on the OIE lists because they are not significant in the international trade of animals and animal products. Occurrences of these diseases are not required to be reported and as a consequence their frequency of occurrence and worldwide regional distribution are not known. It will never be possible to identify in advance all diseases that could be introduced to Antarctic wildlife. Precautions implemented in response to known diseases may also reduce the risk from unknown diseases.

2.5 Characteristics of Disease that Influence Their Risk

The individual characteristics of diseases will influence whether they are more likely to be translocated and successfully introduced to previously naïve populations (Table 5). Among the most critical characteristics are the duration of survival of the pathogen in a potentially infective form and its means of transmission (Wilson 1995). Infectious Newcastle disease has been recovered from meat after 250 days at −14°C to −20°C and from skin and bone marrow after 250 days at −4°C (Asplin 1949). Avian Influenza virus can survive in faeces for at least 35 days at 4°C, virus is stable over a pH range of 5.5–8 and can remain infective in lake water for up to 4 days at 22°C and over 30 days at 0°C (Webster et al. 1978). Survival is

Table 5 Characteristics of diseases or their causative agents and implications for transmission in Antarctica

Characteristic of disease	Implications for transmission of disease in Antarctica	Examples
Present in animal populations of countries participating in Antarctic activities	Creates the possibility that people or equipment may be in contact with the disease before visiting Antarctica.	Newcastle disease, avian influenza
Requires intervention by vector	Disease cannot be transmitted if vector is not present; disease may become a risk if the vector extends its geographic range.	Blue tongue (OIE List A) is unlikely to be a risk to Antarctic wildlife because the vectors, Culicoides spp, are not present. Lyme disease spirochetes may be involved in enzootic cycles on sub-Antarctic islands involving seabirds and the sea-bird associated tick *Ixodes uriae*.
Able to survive well without host	Increases the chance of transmission on equipment, vehicles or clothing.	Newcastle disease, avian influenza, infectious bursal disease virus
Tendency to form new strains	Host switching.	Morbilliviruses
Ability to infect different hosts across taxonomic groups	Caliciviruses can infect mammals, birds, fish and maybe molluscs.	Caliciviruses (Smith et al. 1998)

prolonged by low relative humidity and low temperature in aerosols whereas low temperature and high moisture levels prolong survival in faeces. Avian Influenza virus survives only several days in carcases at ambient temperature compared with up to 23 days at refrigeration temperatures.

Disease agents that cannot remain viable without a host will not be successfully transferred to Antarctica by people on equipment such as vehicles and clothing. Diseases that require the direct transfer of body fluids are unlikely to be mediated by humans except under very particular circumstances, such as by some invasive scientific procedures. Diseases with an obligate relationship with a specific vector will not become established if the vector is not present.

Some pathogens, particularly viruses, are capable of infecting different host species. This may be because the pathogen is flexible, such as the caliciviruses, or it may be that it mutates rapidly to form new strains, for example, the morbilliviruses.

Many common diseases, including some of the OIE List A diseases, require a vector for transmission or for completion of their life cycle. Although many disease vectors are not present in Antarctica some, such as ticks of the genus *Ixodes*, have been recorded among parasites collected from Antarctic and sub-Antarctic seals and birds (Table 6).

Diseases most likely to be of risk of introduction and spread by people are those that are established in the home countries of people visiting the Antarctic, can survive

Table 6 Parasites recorded from Antarctic penguins and seals

	Host species and location	Reference
Ectoparasites		
Ticks – *Ixodes*	Penguins and seals – sub-Antarctic and Antarctic Peninsula	Zumpt (1952), Murray and Vestjens (1967), Hawkey et al. (1989), Murray et al. (1990), Bergström et al. (1999a, b)
Fleas	Penguins – sub-Antarctic only	Dunnet (1964), Murray et al. (1967, 1990)
Biting lice	Penguin – most sub-Antarctic and Antarctic species	Murray (1964), Murray et al. (1990)
Sucking lice	Seals – all species	Murray et al. (1965), Murray (1967), Harder et al. (1991)
Endoparasitic worms		
Nematodes	Penguins and seals	Mawson (1953)
Cestodes	Penguins and seals	Prudoe (1969)
Trematodes	Seals	

well without a host, do not require a vector that is not present and are able to infect different hosts. There are several diseases, common elsewhere, that are likely to result in the death of many animals if they are introduced successfully to Antarctic populations, examples include Newcastle disease, avian influenza and the morbilliviruses that cause canine and phocine distemper.

3 Factors that Could Influence the Introduction and Spread of Disease Among Antarctic Wildlife

3.1 Environmental Conditions

Environmental conditions will influence the chance of disease introduction and spread both directly and indirectly (Table 7). Factors such as temperature, humidity, wind, available water etc will directly influence the survival times of pathogens in the environment. There is little published work on the survivability of pathogens in Antarctica, however, the information available indicates that micro-organisms may survive in Antarctica at least as well as they do in other environments. Human enteric bacteria introduced to the Antarctic environment with untreated sewage effluent are able to persist for long periods (up to 54 days) in a viable but non-culturable state (Smith et al. 1994). The human bacterium *Clostridium perfringens* is known to persist in Antarctic marine sediments and be ingested by marine invertebrates (Edwards et al. 1998, Conlon et al. 2000). There is some indication that seals in the vicinity of a sewage outfall can be infected by *Clostridium perfringens* (McFeters and Edwards, in press, cited in Conlon et al. 2000). Environmental conditions may extend the viability of some disease causing agents and reduce the viability of others.

Table 7 Environmental conditions in Antarctica and implications for disease transmission

Environmental condition	Implication to survival or transmission of disease causing agents	Implication to precautions against transmission of disease causing agents
Temperature	Some infectious agents may be susceptible to low temperatures; others may survive well. Temperature controls availability of water (see below). Low temperatures may render introduced vectors immobile but may not effect indigenous vectors	Low temperature can make otherwise simple precautions difficult or impossible to implement. Warming of parts of Antarctica may increase the range of some vectors
Humidity	Low humidity may cause desiccation of some pathogens and reduce survival away from a host. Higher humidity in the maritime environment of the Antarctic Peninsula may aid transmission by droplets	
Availability of water	Shortage of water may cause desiccation of some pathogens and reduce survival away from a host	Lack of available water can make otherwise simple precautions difficult or impossible to implement
Winds	High winds may cause desiccation of some pathogens and reduce survival away from a host; wind may assist transmission of disease as aerosol	High winds can make otherwise simple precautions difficult or impossible to implement
Snow cover	Snow at colonies may protect debris, feathers, faeces from dispersion by wind; on melting it provides a source of water	
Sea-ice	Annual sea-ice is transient; a site that is infected by disease-causing agents will eventually be replaced	For some species replacement of annual sea-ice may provide an effective natural method for limiting disease transfer between years
Distance from other continents	May limit contact with some species including humans	Provides opportunity for quarantine procedures

Environmental conditions across the continent are not constant and regional differences may influence the likelihood of disease transmission. The maritime environment of the Antarctic Peninsula is warmer and more humid than eastern Antarctica. The distance separating Antarctica from other land masses also varies considerably depending on locality and will influence the frequency of interactions between Antarctic species and animals from other regions.

Indirectly the environment determines all aspects of animal behaviour and, as a consequence, can influence disease transfer between animals. The environment also affects the activities that people undertake, which in turn, can influence their role in disease transfer. In particular, the difficult nature of working in Antarctica can reduce human motivation to follow precautionary procedures and, in a practical sense, the scarcity of liquid water at some locations can make otherwise simple precautions a major burden.

3.2 Animal Behaviour

Animal behaviour will influence the potential for introduction and spread of infectious disease-causing agents in several ways (Table 8). The tendency to form aggregations will increase the opportunities for infectious agents to be spread within a population. The mode of feeding will influence the probability of coming into contact with the body fluids of other species. Of all feeding types, scavengers and carrion feeders are probably the ones most likely to be in contact with tissues of infected animals or human food. Migration patterns will affect the chance that a species may translocate a disease-causing agent. Many species travel between Antarctica and other regions and may be exposed to diseases by

Table 8 Behaviour of Antarctic wildlife and implications for disease transmission

Behaviour	Implications to disease transmission	Species
Solitary or colonial		
Solitary or small groups		Leopard seals
Dispersed colony	May have only limited intra-specific interactions within the colony but may form aggregations during the breeding season at other locations	Wilson's storm petrels, snow petrels
Dense colony on ice	Forms colony on 'fresh' ice at the start of each breeding season so no chance that infectious agents remaining from previous season will be transmitted to reformed colony	Emperor penguins
Dense colony on rock	Faeces, feathers etc from previous seasons will be exposed during the summer melt; opportunity for infectious agents to be transmitted from one breeding season to the next	Adélie, chinstrap, gentoo penguins, blue-eyed shag
Feeding type		
Carnivore – feeding on invertebrates or fish	In general, disease transfer between phyla is less likely than between more closely related species. However, some invertebrate species may act as an intermediate host	Penguin species, Weddell and crabeater seals
Carnivore – feeding on birds or mammals	May come in contact with diseases that use prey species as reservoir. In general, the more closely related the prey, the more likely it is that diseases carried will be transmissible to the predator. Identical isolates of campylobacter jejuni in prey and predator species within a food chain indicates that pathogens can be passed along the food chain (Olsen pers. comm.)	Leopard seals

(continued)

Table 8 (continued)

Behaviour	Implications to disease transmission	Species
Carnivore – scavenger or carrion feeder	Generalist scavengers are most likely to come into contact with disease causing agents, e.g. by feeding on dead and dying diseased animals, by feeding at sewage outlets. Wide ranging scavengers are a likely vector for translocation of disease causing agents. Scavengers and carrion feeders are likely to have evolved effective defence mechanisms against disease	Brown and southern polar skua, northern and southern giant petrel, kelp gull, sheathbill
Aggression		
Non-aggressive	May be the subject of aggression; wounding can create a route for disease transfer	
Displays inter-specific aggression	Aggression leading to wounding can create a route for disease transfer	
Displays intra-specific aggression	Greater opportunity of transfer of diseases requiring direct contact with bodily secretions (mucus, blood, urine, faeces)	
Migration patterns		
Does not migrate	May be local reservoirs of microrganisms	Sheathbills in sub-Antarctica
Travels widely within the Antarctic region	May provide a mechanism for translocation of disease within Antarctica	
Travels between Antarctica and other regions	May be in contact with disease carrying animals from other regions; may feed at rubbish disposal sites, sewage outfalls, abattoir effluent outfalls and other sites where the chances of coming in contact with infectious disease causing agents is high	Wilson's storm petrel, Southern giant petrel, brown skua, Arctic tern, Antarctic tern, Dominican gull, greater sheathbill, kelp gull, southern elephant seal, fur seals, fin whales, humpback whales, blue whales, minke whales, and possibly many species of dolphin

contact with wildlife or as a consequence of human activity, such as waste disposal, in these regions.

The animals most likely to come into contact with pathogens as a result of human activity are those that will feed on waste generated by people given the opportunity. Species that also scavenge at aggregations, such as breeding colonies, are most likely to be agents of disease transfer to other Antarctic species.

4 Human Activities Which may Introduce or Spread Disease

4.1 Human Activities and Their Implications for Disease Introduction or Spread

Common human activities undertaken in Antarctica and elsewhere that may lead to disease introduction and spread are listed in Table 9. The type of activities undertaken before going to Antarctica, their locations and subsequent precautions will determine whether people bring infectious disease causing agents with them to Antarctica. The types of activities and how they are undertaken within Antarctica will determine whether pathogens brought into the region could be transmitted to wildlife or whether people could translocate indigenous pathogens.

A recent assessment of the risk of disease to wildlife on the Antarctic Peninsula (Pfennigwerth 2001) developed a qualitative approach to assessing the likelihood that activities would cause a disease event. This method has been adapted and applied here to the activities identified in Table 9. The activities are considered in relation to each of the steps leading to a disease event (Table 10). Likelihood has been assessed on a simple relative scale of low, medium/low, medium, high and very high based on the responses to each of the questions.

4.2 Combinations of Activities and the Risk of Disease Introduction or Spread

This approach is useful for indicating the relative likelihood of disease events arising from individual activities, however, activities do not happen in isolation. Antarctic operations consist of many combinations of these activities. Some will operate synergistically to increase the likelihood, while others will be antagonistic and so reduce the chance of disease introduction. Activities will be combined in many complex ways and may have unpredictable effects on the probability of disease introduction.

Consideration of specific scenarios could assist in focussing attention on activities and combinations of activities that have a greater likelihood of bringing disease into Antarctica.

Scenario 1 – Scientists Working on Disease in Antarctic Wildlife

Among visitors to Antarctica, scientists involved in disease research are more likely than others to be in contact with diseased animals before travelling to Antarctica. Their equipment may be in close contact with animals both in Antarctica and elsewhere, creating opportunities for transfer of pathogens. Their research may entail

Table 9 Common human activities and implications for disease transmission in Antarctica

Human activity	Implications for disease transmission
Activities outside Antarctica	
International travel	Travel between countries is recognised as one of the major factors causing the rapid spread of disease around the globe; visits to different countries and different environments increase the chance of coming into contact with a variety of diseases
Visits to farms, abattoirs, food-processing plants, zoos, scientific animal houses etc.	Visits to locations where animals are held will all increase the chance of people coming in contact with diseased animals or their products (e.g. faeces)
Use of equipment in other regions (field training, scientific etc.)	Use of Antarctic equipment in other regions will increase the chance that it may be contaminated with disease causing agents
Release of captive animals	The risk of disease introduction associated with re-release to the wild of captive animals has been recognised. SCAR recommends against the release of captive animals, however, there is no specific ATS recommendation on this
Activities within Antarctica	
Logistics	
Import of equipment, vehicles and clothing	There is no specific AT requirement to clean vehicles, clothing or equipment before sending to Antarctica or moving between locations in Antarctica. However, import of non-sterile soil must be avoided to the maximum extent practicable
Import of non-indigenous plants and animals	Non-indigenous plants and animals (except food) cannot be introduced to Antarctica without a permit and after use must be disposed of by incineration or equally effective means. These requirements are in response to concerns about the potential for disease introduction with plants or animals, however this remains a risk as there has not been complete compliance
Import of food	No live animals can be imported for food. Precautions are required to prevent the introduction of micro-organisms (eg viruses, bacteria, parasites, yeasts, fungi) not present in the native fauna and flora. Poultry must be inspected for evidence of disease, such as Newcastle's Disease, tuberculosis, and yeast infection. These requirements are in response to concerns about the risk to wildlife of disease associated with food (poultry in particular)
Waste disposal	Human waste and food waste are the most likely sources of bacterial and viral introductions to Antarctica. Whether they contain infectious disease causing agents will depend on their source, treatment and subsequent method of disposal
Sewage treatment	Sewage and domestic waste may be discharged directly to the sea. Treatment, at least by maceration, is required for populations of 30 or more. People will carry many opportunist infectious agents and these will be shed in faeces. Sewage treatment techniques used in Antarctica by most operators are not designed to kill pathogens
Kitchen waste	Kitchen waste must be either incinerated or removed from the Antarctic. Stored waste needs to be in robust containers to prevent interference by scavengers. Frozen meat including poultry is commonly defrosted in kitchen sinks and the melted water passed through sewage treatment (if present) before disposal to the environment

(continued)

Table 9 (continued)

Human activity	Implications for disease transmission
Feeding wildlife	Feeding of wildlife is not permitted under many national regulations however feeding of wild life is not specifically prohibited under any AT measure. Feeding of waste food (particularly poultry products) to wildlife is among the most direct ways that disease could be introduced to wildlife
Field camps	The practicalities of living in field camps make some precautions that would be relatively simple to instigate at stations very difficult to follow
Storage of food	Scavengers may gain access to food or food waste unless precautions are taken
Waste disposal	Sewage and domestic liquid wastes from field camps cannot be disposed of to ice-free areas or fresh water systems. Waste may be disposed of in deep ice pits. To the maximum extent practicable waste should be removed to stations or ships for disposal. Handling of human waste from field camps can create hygiene and disease risk to people
Science	Permits from national authorities are required for any direct contact with wildlife. Permits are more likely to be given for scientific purposes than for other types of activities
Scientific observations	Scientific observations, such as surveys, may not require contact with the animals but may involve approaches closer than otherwise permitted. Surveys at more than one location may create the risk of translocation of microorganisms between sites
Scientific manipulations	Science involving manipulations of wildlife are the only planned activities in which contact between animals and people occur. Translocation of microorganisms between animals and sites will occur unless hygiene precautions such as cleansing of people and equipment are followed
Feeding of wildlife for dietary experiments such as the use of radio-labelled food or replacement of food after stomach flushing	Food provided to wildlife could contain disease causing agents
Recreation	Most visitors to Antarctica, whether as scientists, in support of science or as tourists, will visit breeding aggregations of wildlife, such as penguin colonies, if given the opportunity
Visits to wildlife aggregations	Recreational visitors to wildlife aggregations will not be in direct contact with animals if normal guidelines are followed. Footwear is likely to be in contact with animal faeces and this could be transferred among locations if precautions such as cleansing are not followed. Several tourism companies use the opportunity to visit multiple wildlife colonies in their marketing. Commercial tourists are usually supervised and return to ship between visits to wildlife colonies. Personnel from national Antarctic programs are more likely to visit wildlife aggregations unsupervised and may visit several colonies, at different locations, in a single day
Fishing	
Bait used for longline fishing	Fish used as bait for longline fishing could be infected with disease causing agents
Waste discharges from fishing boats	Waste discharged from fishing boats is the most significant attractor of wildlife in sub-Antarctic waters

Table 10 Qualitative assessment of those activities which may introduce or spread disease causing agents in Antarctica (based on Pfennigwerth 2001)

Activity	Are pathogens that could cause wildlife disease likely to be present?[a]	If present could pathogens be released during this activity?	If released could pathogens survive in the environment?	Could activity assist pathogens to multiply?	Could activity contribute to dispersal of pathogens?	Could wildlife come into contact with pathogens as a result of this activity?	Relative likelihood of causing disease
Import of equipment, vehicles and clothing	Possibly	Yes	Possibly	No	Yes	Possibly	Low
Import of non-indigenous plants and animals	Yes	Yes	Yes	Yes	Yes	Possibly	High
Import of food	Possibly	Yes	Yes	Possibly	Yes	Possibly	Medium
Waste disposal	Possibly	Yes	Possibly	No	Possibly	Possibly	Low
Sewage effluent disposal	Possibly	Yes	Possibly	Possibly	Yes	Yes	Medium
Kitchen waste disposal	Possibly	Yes	Yes	Possibly	Yes	Possibly	Medium
Deliberate feeding of wildlife	Possibly	Yes	Not necessary, direct transfer possible	Yes	Yes	Yes	Very high
Storage of food at field camps	Possibly	Yes	Possibly	Possibly	Possibly	Possibly	Medium–low
Waste disposal at field camps	Possibly	Yes	Possibly	Possibly	Possibly	Possibly	Medium–low
Scientific observations of wildlife	Possibly	Yes	Possibly	Unlikely	Yes	Possibly	Low
Scientific manipulations of wildlife	Possibly	Yes	Not necessary, direct transfer possible	Yes	Yes	Yes	Very high
Feeding of wildlife for dietary experiments	Possibly	Yes	Not necessary, direct transfer possible	Yes	Yes	Yes	Very high
Discovery of unusual mortality events	Possibly	Yes	Yes	Yes	Yes	Yes	Very high
Recreational visits to wildlife aggregations	Possibly	Yes	Possibly	Unlikely	Yes	Possibly	Low
Longline fishing using bait	Possibly	Yes	Not necessary, direct transfer possible	Possibly	Yes	Yes	High

[a]Based on the disease status of operator nations

visiting several sites including breeding aggregations, which creates the possibility of spreading disease-causing agents. All these factors will combine to increase the chance of disease introduction or spread. On the other side of the equation, scientists working in this field are likely to be aware of the risks, should know what precautions are necessary and should have their own procedures for ensuring their studies are not confounded by cross-contamination of samples. Scientists working with wildlife for reasons other than the study of disease will also be in contact with animals if their research involves direct manipulations.

Scenario 2 – Investigation of an Unusual Mortality Event

Unusual mortality events are by their nature unpredictable. It is unlikely that a wildlife mortality event will be discovered by someone with previous experience of such occurrences and it would be unwise to leave decisions on how to react to those discovering a mortality event. Most people do not know normal mortality rates among Antarctic species and may not recognise unusual mortality. A likely first reaction to discovery of an unusual mortality event would be to quickly check other localities to determine the spatial extent of the event. Moving from location to location without some precautions could cause translocation of infection disease causing agents.

Scenario 3 – Use of Poultry Products by Antarctic Personnel

The Madrid Protocol requires that dressed poultry should be inspected for disease before sending to Antarctica because of the perceived risk from diseases such as Newcastle disease, however, inspection is not a reliable method for detecting many diseases including Newcastle disease. Frozen chicken products are commonly thawed in kitchens and the resulting melted liquid discarded with other domestic grey water. Treatment of grey water is limited to the level of treatment available for sewage, which in most cases is not sufficient to kill pathogens. Disposal of sewage effluent is permitted to the marine environment.

Scenario 4 – Recreational Visits to Wildlife Aggregations

Members of national Antarctic programs will frequently take the opportunity to visit breeding aggregations of wildlife for recreational purposes. Those who enjoy outdoor activities are more likely to take the opportunity to visit several breeding sites while in Antarctica. As a generalisation, the type of person who enjoys outdoor pursuits may own their own footwear and use these in preference to footwear issued specifically for Antarctica. They may use their footwear before going to Antarctica, possibly in circumstances that could expose them to pathogens. Currently people visiting Antarctica do not necessarily receive advice to suggest that cleaning of footwear is a sensible precaution.

4.3 Human Activities Identified as Priority Risks

The following human activities are identified as the priority risks. Details of the precautions suggested to reduce these risks are to be developed as the second of the three Terms of Reference of the Intersessional Contact Group on Disease in Antarctic wildlife (Practical measures to diminish the risk to Antarctic wildlife of the introduction and spread by human activity of infectious disease causing agents),

1. Feeding of wildlife
2. Actions following discovery of unusual mortality events
3. Research that involves handling of Antarctic animals, particularly research on disease
4. Import of food, particularly poultry products
5. Waste disposal and sewage treatment
6. Use of equipment and clothing before departure to Antarctica
7. Serial visits to wildlife aggregations

5 Summary and Conclusions

The following conclusions are numbered sequentially but are grouped according to the section in the report from which they are derived.

Risk Assessment Methodology

1. There is insufficient information available to conduct a reliable quantitative risk assessment of disease introduction and spread to Antarctic wildlife.
2. A qualitative risk assessment approach should be sufficient to indicate priorities for precautionary measures.

Historic Information on Disease

3. No diseases have been demonstrated to have been introduced to Antarctic wildlife or spread by human activities.
4. No systematic studies of disease in Antarctica have been undertaken and it is unlikely that conclusive evidence of human involvement in disease events would be available.
5. There is recent evidence to indicate that some microorganisms have been introduced to Antarctic wildlife and spread as a consequence of human activity.
6. Seven unusual mortality events in which disease was suspected have been recorded for Antarctic wildlife. Only one was investigated and the causes of the others are not known.

7. A seal mass mortality event on the Auckland Islands in 1998 was well investigated but the causal agent is still not known with certainty, indicating that identification of the cause of a mortality event is not always possible.
8. Clinical and serological evidence indicates that many Antarctic and sub-Antarctic penguins and seals have been exposed to infectious disease causing agents, indicating that they are not completely naïve populations with respect to disease.
9. Captive Antarctic birds and seals have exhibited symptoms of a variety of diseases known in other wildlife populations, indicating that they are susceptible to a range of diseases.
10. Disease is suspected in a significant number of the marine mammal mass mortality events reported in non-Antarctic regions.
11. Most of the OIE List A of transmissible diseases with the potential for very serious and rapid spread occur in countries that participate in Antarctic activities. This indicates that, despite the economic incentives to prevent them and the large preventive effort, serious transmissible disease of animals occur in most countries.
12. Most OIE List A diseases would not be transmissible to birds and seals, however, there is evidence that birds and seals are susceptible to some, such as Newcastle disease and avian influenza.
13. Newcastle disease has occurred widely in ATCP countries in recent years and may be the disease most likely to be a risk to Antarctic wildlife.
14. Diseases most likely to be of risk of introduction and spread by people are those that are established in the home countries of people visiting Antarctica, can survive well without a host, do not require a vector that is not present and can infect different hosts, examples include Newcastle disease, avian influenza and the morbilliviruses causing canine and phocine distemper.
15. It is not possible to identify all diseases with the potential for introduction and this is not necessary as a precursor to implementation of precautions.

Factors that Could Influence Disease Introduction or Spread

16. Environmental conditions in parts of the Antarctic are similar to conditions elsewhere and so mechanisms for disease transfer that occur in these places are likely to also occur in Antarctica.
17. The cold and lack of available water may make otherwise simple precautions difficult or impossible under some circumstances such as at remote field locations.
18. Animal behaviour will influence the likelihood of disease transmission within populations and between species.
19. Several Antarctic species migrate beyond the Antarctic to regions where they could be in contact with disease causing agents carried by other wildlife and in human waste at sewage effluent outfalls and waste disposal tips.
20. Carrion feeders are most likely to be in direct contact with diseased or dying animals of other species.

21. Opportunist scavengers are most likely to feed on waste generated by human activity if precautions are not taken to prevent access.
22. Skuas are among the most likely species to be the point of entry of disease from waste because they are not shy of people and they will scavenge on station waste given the opportunity. They are also among the most likely routes of transfer to other species because of their habit of associating with other species.

Human Activities Which may Introduce or Spread Disease

23. Activities undertaken before going to Antarctica, including precautions, will determine whether people bring infectious disease with them.
24. Activities in Antarctica most likely to cause disease introduction or spread are those that involve close contact with wildlife or those that allow wildlife to come in contact with waste generated from human activities.
25. Certain combinations of activities may significantly increase the risks.
26. Precautions should be prioritised to target the most likely pathways of disease introduction or spread.
27. Human activities identified as priorities for practical measures to diminish risk are,

    - Feeding of wildlife
    - Actions following discovery of unusual mortality events
    - Research that involves handling of Antarctic animals, particularly research on disease
    - Import of food, particularly poultry products
    - Waste disposal and sewage treatment
    - Use of equipment and clothing before departure to Antarctica
    - Serial visits to wildlife aggregations.

Attachment 1 – Risk Assessment Process

Processes called risk analysis are used in many different fields and different terminologies have evolved. In veterinary medicine the phrase *risk analysis* is generally used as the term for the overall process for dealing with risks. In the framework established by the Office International des Epizooties (OIE), the world organisation for animal health, risk analysis consists of,

- Hazard identification – the process of identifying pathogenic agents that could be introduced
- Risk assessment – evaluation of the likelihood and consequences of introducing a pathogen

- Risk management – the process of identifying, selecting and implementing measures to reduce the level of risk, including determination of acceptable risk
- Risk communication – the interactive exchange of information on risk among interested parties

The process of hazard identification used here was to review historic information on wildlife diseases in Antarctica and from elsewhere to determine if particular diseases should be a concern. Risk was assessed using information on the nature of the pathogens, environmental conditions, the biology and behaviour of the animals of concern and the activities of people visiting Antarctica. Risk management and risk communication are the subject of the other terms of reference of this intersessional contact group.

Risk is the product of the likelihood of an event happening and the consequences of the event should it occur. The smallest risks are associated with activities that are unlikely to occur and are of little consequence; the greatest risks are those that are likely to occur and are of great consequence. Between these extremes are various combinations of likelihood and consequence (Table 11).

Risk assessments may be either quantitative or qualitative; both approaches will involve some degree of uncertainty. Qualitative risk assessments may appear to be more objective however this may be illusionary. If probability data are not available, but are estimated and the estimates are subsequently used as the basis for calculation of likelihood, the subjective nature of the assessment may be obscured. Any risk assessment should include an indication of the source and scale of uncertainty in the information on which it is based.

Risk management is based on the precept that risk cannot be eliminated completely but if the sources of greatest risk are recognised in advance they can be reduced. An important component of risk management is the decision on what constitutes an acceptable risk.

Likelihood

In a quantitative risk assessment, such as those for the importation of farm animals to a country (Hayes 1997), the risk assessment may commence with review of the prevalence of the infectious agent in the country of origin. The next step would be to assign a probability to each of the steps that must be completed if the disease is

Table 11 Level of risk based on assessment of likelihood of an event and consequences of the event

	Severity of consequences				
Likelihood	Extreme	Very high	Medium	Low	Negligible
Almost certain	Very severe	Severe	High	Major	Significant
Likely	Severe	high	Major	Significant	Moderate
Moderate	High	Major	Significant	Moderate	Low
Unlikely	Major	Significant	Moderate	Low	Very low
Extremely unlikely	Significant	Moderate	Low	Very low	Negligible

to be established in the importing country. For a disease to cause an epidemic a series of steps must each take place. In a quantitative assessment the overall probability for successful disease introduction is calculated as the product of the individual probabilities of each step. For this process to be applied to disease importation to Antarctic wildlife, the probability of each of the following would be required,

1. A piece of equipment, food or a person is infected with the disease causing agent
2. The agent survives handling, treatment and transit time
3. Wildlife are exposed to the agent
4. The agent is exposed to a portal of entry (e.g. a wound, inhaled etc)
5. The agent induces infection
6. The infection induces disease
7. The disease spreads

When this process is used in non-Antarctic regions probabilities are estimated on the basis of prior information. For Antarctic activities, sufficient information is not currently available to provide a meaningful estimate of probability for any of the steps. Because the method is based on the mathematical product of the probabilities of each step, the individual uncertainties associated with each step compound; as a consequence it is unlikely that a method based on the probabilities for a series of steps will be useful at this stage.

An alternative, qualitative approach used here is to consider the range of possible consequences of disease introduction and to assign a rough indication of the likelihood that each consequence could occur. This is used as the basis for determining whether any of the possible consequences are a sufficiently high risk that precautionary measures are warranted. The next step is to identify qualitatively which human activities are most likely to create exposure and transmission pathways, and which species are most vulnerable. This information may then be used as the basis for practical measures to reduce risk.

After the qualitative risk assessment, if it is still unclear whether the risks are sufficient to warrant preventative measures, it may be necessary to embark on an extensive information gathering process to acquire data sufficient for a quantitative risk assessment. This effort should not be necessary if the qualitative assessment clearly indicates that there are significant risks that could be prevented or that the risks are acceptable.

Consequences

Some potential consequences of taking pathogens to Antarctica, listed in increasing order of severity and reducing likelihood, are,

1. The pathogen is not exposed to a suitable host and dies
2. Transient sickness and distress to individual animals
3. Establishment of a non-native micro-organism

4. Loss of productivity or breeding success
5. Death of a few animals
6. Death of many animals
7. Eradication of local populations
8. Disruption of a component of the ecosystem
9. Extinction of a species

It is inevitable that people will take some pathogens with them when they visit Antarctica. Pathogens that are taken to Antarctica and subsequently die without infecting a suitable host will have a negligible impact. Their effects, if any, are of little consequence as they are both short-term and local. A pathogen that becomes established within a population without causing the outward signs of disease may have no ecological effect but may become a long-term addition to the biota of Antarctica. A pathogen that becomes established without causing disease may have a minor impact on the population and may have no wider ecological implications, however, if it is established, it is, by definition, not transitory.

Pathogens that cause sickness and distress to infected animals may have transient effects on individual animals and may have few, if any, wider ecological consequences. However, if the disease persists in the population and continues to infect other individuals, the consequences of the introduction are not transient. Diseases that cause the death of animals obviously have a permanent effect on the infected animals. Whether the disease causes lasting change to a population or has wider ecological implications will depend on a number of factors, including the number, age-cohort or sex of animals killed.

Extinction of a species is the most serious effect that any human activity could cause because it is both permanent and widespread. However, experience in other regions indicates that species extinction is a very unlikely consequence of disease introduction without the co-occurrence of other stress factors.

Overall Risk

It is impossible to accurately predict the likelihood and consequences of disease introduction to a population in which the disease has not previously occurred (Table 12). Both likelihood and consequences will vary according to characteristics of the pathogen and the affected species, including host range, means of transmission, degree of exposure, immune status and response to the potential hosts. In general, the consequences of disease are more severe in naïve populations than in populations previously exposed. Knowledge of the full consequences of introduction is not a necessary precursor to the implementation of methods to reduce the likelihood of an introduction. If establishment of a non-native pathogen is undesirable and precautions are taken to reduce the likelihood of this happening, these precautions will also reduce the likelihood of other, more serious consequences such as death of animals.

Table 12 Potential consequences of taking pathogens to Antarctica and their likelihood, severity and indication of the overall risk

Potential consequence	Likelihood	Severity of consequences	Overall risk
1. Pathogens are introduced but are not exposed to a suitable hosts and die	Certain	Negligible	Significant
2. Transient sickness and distress to individual animals	Moderate	Low	Moderate
3. Establishment of a non-native micro-organism	Moderate	Medium	Significant
4. Loss of productivity or breeding success	Moderate	Medium	Significant
5. Death of a few animals.	Moderate	Medium	Significant
6. Death of many animals	Unlikely	Very high	Significant
7. Eradication of local populations	Unlikely	Very high	Significant
8. Disruption of a component of the ecosystem	Unlikely	Extreme	Major
9. Extinction of a species	Extremely unlikely	Extreme	Significant

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Appendix E
Report on the Open-Ended Intersessional Contact Group on Diseases of Antarctic Wildlife Report 2 – Practical Measures to Diminish Risk (Draft)[1]

Background

CEP III agreed to the following terms of reference for the open-ended intersessional contact group (ICG) on diseases of Antarctic wildlife:

That the contact group prepare an initial report for CEP IV which:

- *Provides a review of the introduction and spread by human activity of infectious disease causing agents in Antarctica and provides a risk assessment of those activities which may introduce or spread disease causing agents in Antarctica*
- *Presents practical measures that might be implemented by Parties to diminish the risk to Antarctic wildlife of the introduction and spread by human activity of infectious disease causing agents; and*
- *Presents practical measures that may be implemented to determine the cause of unusual wildlife mortality and morbidity events in Antarctica and to reduce the likelihood that human activity may exacerbate these events.* (CEP III Report, paragraph 52)

This paper reports on the work of the ICG in response to the second of the terms of reference. The ICG's draft report is at Annex 1. Australia coordinated the process, with participation from AEON, Australia, IAATO, Italy and Sweden.

A report in response to the first of the terms of reference (Review and Risk Assessment) has been submitted to CEP IV as an annex to a separate working paper. The ICG does not yet have a draft report in response to the third of the terms of reference.

The report on practical measures to diminish risk is presented to CEP IV as a draft to encourage further participation in the work of the ICG. The implementation of practical measures is likely to have implications for the way that Antarctic activities are undertaken and the establishment of practical measures within the Antarctic

[1]Editors' note: This report is reproduced from Working Paper 11, submitted by Australia to XXIV Antarctic Treaty Consultative Meeting/CEP IV in July 2001. The working paper and its annex are reproduced in their entirety here. The report, presented as a draft, was accepted by CEP and the work of the ICG was determined to be complete.

Treaty System may involve Parties in formal *decisions, resolutions or measures* that could have implications for the domestic legislation of Parties. It is therefore important to the success of the practical measures that they are carefully considered.

The practical measures address human activities identified by the ICG as risks through the process of review and risk assessment. Before the report on practical measures is finalised it is important that the CEP indicates whether or not it endorses the list of human activities identified as a priority by the ICG.

The purposes of submitting the work of the ICG on practical measures to CEP IV as a draft are:

- To ensure they are discussed widely and to encourage the widest possible participation in their further development, and
- To ensure that the ICG has the opportunity to modify the practical measures to address priorities determined by CEP in its consideration of the review and risk assessment

Recommendations

It is recommended that:

- The CEP notes the draft report from the ICG on practical measures to diminish risk
- The CEP encourages Parties to continue participation in the work of the ICG to further develop the draft practical measures to diminish the risk to Antarctic wildlife of the introduction and spread by human activity of infectious disease causing agents in Antarctica
- The CEP asks the ICG to prepare a report for CEP V which:
  (a) Presents practical measures that might be implemented by Parties to diminish the risk to Antarctic wildlife of the introduction and spread by human activity of infectious disease causing agents; and
  (b) Presents practical measures that may be implemented to determine the cause of unusual wildlife mortality and morbidity events in Antarctica and to reduce the likelihood that human activity may exacerbate these events

Draft Report – Practical Measures to Diminish the Risk to Antarctic Wildlife of the Introduction and Spread by Human Activity of Infectious Disease Causing Agents

Contents

1 Introduction

The open-ended intersessional contact group on diseases of Antarctic wildlife has undertaken a review of the introduction and spread by human activity of infectious disease causing agents, and risk assessment of those activities which may introduce or spread disease causing agents in Antarctica. This process identified the following human activities as priorities for practical measures that might be implemented by Parties to diminish risk,

- feeding of wildlife
- actions following discovery of unusual mortality events

Editors' note: This review was written by Martin J. Riddle with contributions from the Antarctic Environmental Officers Network, the Antarctic and Southern Ocean Coalition and the International Association of Antarctica Tourism Operators and representatives from Italy, Norway and Sweden. It was submitted to XXIV Antarctic Treaty Consultative Meeting/CEP IV in July 2001 by Australia as an annex to Working Paper 11. Enquiries should be directed to the author (MJR).

- research that involves handling of Antarctic animals, particularly research on disease
- import of food, particularly poultry products
- waste disposal and sewage treatment
- use of equipment and clothing before departure to Antarctica
- serial visits to wildlife aggregations.

A workshop on disease of Antarctic wildlife held in 1998 identified the following as general approaches that could contribute to reducing the risk of disease introduction and spread by human activity,

- education and awareness
- initial response to unusual mortality events
- information exchange
- cleaning or sanitising of equipment
- source of food supplies
- waste management, sewage treatment and effluent disposal
- fundamental research on disease in Antarctic wildlife.

These approaches have been widely disseminated and discussed, and were in general endorsed in a joint SCAR and COMNAP working paper to CEP III (XII SATCM/WP20) (SCAR and COMNAP 2000). The approaches are used as the framework for developing practical measures to diminish the risk to Antarctic wildlife of disease introduction and spread from those human activities identified as priority risks.

2 Education and Awareness

2.1 Background

The success of all the other measures depends on their acceptance and adoption by people visiting Antarctica. Measures will not be effective unless the requirement for them is disseminated and they will be most effective if people understand the reasons for precautions. In addition, people that understand the reasons behind the concern about disease introduction will be better prepared to make appropriate decisions if presented with an unpredicted situation which has implications for disease introduction or spread.

2.2 Action Required

Encourage operators to include an explanation of the potential for disease introduction and translocation, and simple procedures that should be adopted to reduce the possibility in pre-departure or in-transit briefings.

Collate a list of educational material on the topic of wildlife disease currently available from national Antarctic programs and tourist operators to determine what is available and where the gaps are.

Prepare and make available to all national operators and Antarctic tourism operators standard educational material such as posters and video/CD-ROMS to convey the following information,

1. Antarctic wildlife could be susceptible to wildlife diseases that occur in other regions,
2. People could accidentally introduce infectious disease causing agents from other regions of the world to the Antarctic or could accidentally spread disease causing agents that occur naturally in the Antarctic between locations,
3. Activities judged to bring some risk of disease introduction or spread include,

    a. those that involve close contact with wildlife, such as disease research
    b. the discovery or investigation of unusual mortality events
    c. importation of meat, especially poultry products, to the Antarctic
    d. feeding Antarctic wildlife
    e. disposal of kitchen waste
    f. moving between aggregations of wildlife with footwear or other clothing, equipment and vehicles that are contaminated with animal faeces.

3 Initial Response to Unusual Mortality Events

3.1 Background

Unusual mortality events among Antarctic wildlife have occurred in the past and are by their nature unpredictable. It is unlikely that a wildlife mortality event will be discovered by someone with previous experience of such occurrences and it would be unwise to leave decisions on how to react to those discovering a mortality event. Most people do not know normal mortality rates among Antarctic species and may not recognise unusual mortality, as a consequence, information to help recognise unusual mortality is required.

A likely first reaction to discovery of an unusual mortality event would be to quickly check other localities to determine the spatial extent of the event. Under these circumstances, moving from location to location without some precautions could cause translocation of infection agents.

Development of a complete response plan for unusual wildlife mortality events in Antarctica is the 3rd of the three terms of reference for this Intersessional Contact Group and is not considered further here. However, ensuring the correct initial response to the discovery of a mortality event that may be caused by disease is an important practical measure to diminish the risk to Antarctic wildlife of spread by human activity of disease causing agents.

3.2 Action Required

If disease is suspected the first response should be to stand back, view widely, photograph (preferably digitally), count dead and dying, and note any obvious abnormal characteristics that can be seen from a distance, such as inability to stand, swellings or skin lesions. As soon as possible this information should be sent to Antarctic wildlife experts with expertise sufficient to determine whether the number of dead and dying and the characteristics of affected animals are within normal limits. Access to the site should be restricted to reduce the risk of transfer to uninfected populations until advice is received on whether the mortality event is unusual or likely to be caused by disease.

People discovering a suspected disease event should not visit other locations to determine the spatial extent of the disease without taking very careful precautions to ensure they do not transfer disease causing agents on footwear, clothing and equipment. Cleaning methods are discussed in Section 5.

If it is determined that an unusual mortality event has been discovered the response plan (to be developed) for unusual wildlife mortality events in Antarctica should be implemented.

4 Information Exchange

4.1 Background

Exchange of information is an important aspect of most response plans for unusual wildlife mortality events developed for other regions and is a key component of the response plan developed in response to the 3rd of the terms of reference for this Intersessional Contact Group. The Antarctic Treaty System and associated organisations (such as SCAR and COMNAP) has established structures for information exchange and the use of these structures is preferred. To be effective in reducing the likelihood of human exacerbation of unusual mortality events reporting to alert others must occur quickly. It is therefore not appropriate to use established annual information exchange mechanisms for this purpose. Rather, reporting should use proven information networks such as the Antarctic Environmental Officers Network and IAATO to disseminate information.

4.2 Action Required

A standard procedure for information exchange after the discovery of an unusual mortality among Antarctic wildlife is to be included in the response plan (to be developed) for unusual wildlife mortality events.

5 Cleaning/Sanitising of Equipment

5.1 Background

Cleansing of clothing, equipment and vehicles is commonly used as a precaution against the transfer of disease causing agents in other parts of the world, particularly when moving from a location in which a disease is known to be present. Priorities for cleansing should be,

1. clothing, equipment and vehicles that are to be brought into the Antarctic from a location that is experiencing an animal disease outbreak caused by an infectious disease causing agent,
2. clothing, equipment and vehicles that are to be moved from locations within the Antarctic region in which unusual wildlife mortality events have occurred or are suspected,
3. clothing, equipment and vehicles that have been in contact with Antarctic wildlife, particularly those used for activities such as disease research that involve close contact, and
4. clothing (particularly footwear), equipment and vehicles, that are likely to carry animal faeces, before moving from one distinct location to another (the term *distinct location* to be defined).

Simple cleaning of surfaces by steam cleaning or brushing with detergent solution is effective in removing viruses and is necessary for removing grease and organic dirt prior to any subsequent chemical decontamination, if this is required. Micro-organisms vary in their susceptibility to disinfectants. The best disinfectant will depend on characteristics of the disease causing agent (Table 1). Lipid containing viruses and vegetative forms of most bacteria are relatively susceptible. Fungi, acid-fast bacteria (*Mycobacterium* spp.) and non-lipid containing viruses are less susceptible, and bacterial spores are resistant to many disinfectants. Viruses cause most diseases of concern. The lipid content and size of viruses will determine whether they are susceptible to decontamination with detergents.

Table 1 The best disinfectants for use against different virus families

Category of virus	Virus families	Best disinfectants
Category A – Lipid containing viruses; intermediate to large size	Bunyaviridae, coronaviridae, flaviridae, herpesviridae, iridoviridae, orthomyxoviridae, paramyxoviridae, poxviridae, retroviridae, rhabdoviridae, togaviridae	Detergents, hypochlorites, alkalis, Virkon®, glutaraldehyde
Category B – No lipid in virus; small size	Caliciridae, picornaviridae	Hypochlorites, alkalis, Virkon®, glutaraldehyde
Category C – No lipid in virus; intermediate size	Birnaviridae, reoviridae	Hypochlorites, alkalis, Virkon®, glutaraldehyde

Both Newcastle disease (paramyxoviridae) and avian influenza (orthomyxoviridae) can be inactivated effectively with detergents.

Common bactericides like quaternary ammonium and phenolics are not effective against category B and C viruses.

5.2 Action Required

The requirement to avoid, to the maximum extent possible, the importation of non-sterile soil to Antarctica should be re-enforced (Protocol on Environmental Protection to the Antarctic Treaty, Annex II, Appendix C).

Shortly before departure to Antarctica, equipment and vehicles should be cleaned using steam or hot water if possible together with brushing to dislodge encrusted soil and organic matter. If only cold water is available then using cold water and brushing is better than not cleaning at all. Clothing supplied for use in Antarctica should be cleaned using normal laundry procedures prior to sending to Antarctica. Footwear should be cleaned with detergent and brushing on the ship during transit to Antarctica or just prior to boarding the aeroplane if flying. Stronger disinfectants should be used (Table 2) if there is reason to think that people, clothing, equipment or vehicles have been in contact with diseased animals, disease causing agents or have been in an area of known disease risk.

In the Antarctic appropriate cleansing procedures will depend on circumstances. Under normal circumstances (when disease is not suspected) when moving from the vicinity of one ‘discrete population’ to another, footwear should be rinsed with water, using several changes of water to achieve the effect of serial dilution (seawater or freshwater may be used), and should be brushed.

The definition of a discrete population will depend on the species and on the terrain. Cleansing with water and brushing should normally occur before moving between discrete ice-free areas or before moving between islands. Whether cleansing should be performed when moving within ice-free areas and islands will depend on their size and on the characteristics of wildlife populations supported. If the wildlife populations form discrete aggregations with limited opportunity for natural mixing then cleansing before moving between aggregations is recommended. If personnel are visiting Antarctica from a ship, boot washing, as described above, should be repeated after each landing.

Cleansing procedures following activities that involve close contact with wildlife, such as research, should be more stringent and may require the use of stronger disinfectants. Environmental impact assessment of such activities should include assessment of the possibility of disease transfer and if a risk is identified appropriate procedures for cleansing equipment and clothing should be specified as a precautionary condition for approval.

Disinfectants are by their nature biocides and their use can cause health or environmental problems (Table 3). Strong disinfectants should not be used in a manner or situation in which they could cause problems. Hydrochloric acid and the aldehydes should only be used when no alternatives exist and then only by experienced

Table 2 Recommended disinfectants and concentrations for inactivation of viruses and bacteria

Disinfectant group	Dilution/final strength/contact time	Method of application and virus category
Soaps and detergents	As normal/as normal/10 min	Thorough cleaning is essential before other decontamination methods can be used effectively; effective for Category A viruses
Oxidising agents		
Sodium hypochlorite	1:5/2–3% available chlorine/10–30 min	Categories A, B and C; not effective in presence of organic material; less stable in warm, sunny conditions (above 15°C)
Calcium hypochlorite	30 g/l/2–3% available chlorine/10–30 min	
Virkon®	20 g/litre/2% w/v/10 min	Active against all virus families
Alkalis		
Sodium hydroxide	20 g/l/2% w/v/10 min	Categories A, B and C; do not use in presence of aluminium and derived alloys (i.e. aircraft)
Sodium carbonate		
–Anhydrous	40 g/l/4%/10 min	Effective in presence of high concentrations of organic matter
–Washing soda	100 g/l/10%/30 min	
Acids		
Hydrochloric acid (10 molar)	1:50/2% v/v/10 min	Use only when better disinfectants are not available; corrosive
Citric acid	2 g/l/02% v/v/30 min	Safe for clothes and body; especially useful for foot and mouth disease virus
Aldehydes		
Glutaraldehyde	As appropriate/2% w/v/10–30 min	Categories A, B and C
Formalin (40%)	1:12/8% w/v/10–30 min	Releases irritating, toxic gas.
Phenols		
Polyphenolic complex	1:25/4%/??	All are effective anti-bacterials; not efficient against Category B viruses.
Chlorinated phenols	1:20/5%/??	
Quaternary ammonium compounds		
Benzalkonium chloride	1:10/10%/??	All are effective anti-bacterials; not efficient against Category B and C viruses
Chlorohexidine	1:1,000/0.1%/??	
Dioctyl dimethylammonium chloride	1:1,000/0.1%/??	
Iodines	??/0.4%/??	Organic matter reduces the activity; useful in areas used for food preparation

Table 3 Health and environment aspects of disinfectant use

Disinfectant	Health aspects	Environmental problems
Hypochlorites	Toxic for eyes and skin	Strong bleach; inhibited by organic matter; corrosive for metals
Virkon®	Reasonable care necessary	
Sodium hydroxide	Caustic for eyes and skin	Avoid contact with strong acids; cannot be used with aluminium or alloys (aircraft)
Sodium carbonate	Mildly caustic for eyes and skin	Avoid use with aluminium and alloys (aircraft)
Hydrochloric acid	Toxic for eyes, skin and respiratory tract	Corrosive; avoid contact with strong alkalis
Glutaraldehyde	Avoid contact with eyes and skin	Toxic to all living tissues
Formalin solution	Releases toxic gas; irritating for mucous membranes	Toxic to all living tissues

personnel with appropriate safety equipment. The environmental risks associated with the use of disinfectants in Antarctica should be considered as part of the environmental impact assessment for any activity for which strong disinfectants are deemed necessary.

6 Source of Food Supplies

6.1 Background

The potential for introduction to Antarctica of disease-causing agents with food products is recognised by the Antarctic Treaty System. In response, the Madrid Protocol includes the requirement to inspect dressed poultry for evidence of disease, such as Newcastle disease, tuberculosis and yeast infection, before it is packaged for shipment to the Antarctic Treaty area. The Protocol does not specify the type of inspection required. The Protocol also requires that non-sterile soil should not be imported to Antarctica to the maximum extent practicable. Vegetables sent to Antarctica often have non-sterile soil associated with them.

It is not for this group to specify the details of meat industry inspection. Procedures are established and enforced by appropriate authorities in each country and the World Health Organisation, the World Trade Organisation and the Office International des Epizooties (OIE) advises on some international aspects of standards. However, it is important that meat that would not be accepted by other markets is not sent to Antarctica. Normal meat food industry inspection standards should be applied and may include,

1. procedures to detect abnormal signs or death rates during animal production (the producer will have commercial reasons for establishing such procedures),

2. procedures to notify unusual disease during production (some diseases must be reported, such as those on the OIE lists),
3. antemortem inspection to ensure that each batch is in good health before slaughter,
4. inspection of carcasses and meat products for signs of disease,
5. inspection and registration of abattoirs, meat processing and packing establishments to ensure sanitary conditions, and
6. procedures for certification, documentation and labelling of meat and meat products.

Meat and all other animal products intended for human consumption sent to Antarctica should pass inspection to the standard normally applied for domestic consumption within the country or to the highest export standard achievable within the country by the meat processing industry, whichever is the higher. Meat and animal products that are not acceptable, for sanitary reasons, for consumption within the country or for export should not be sent to Antarctica.

Meat and animal products sent to Antarctica should be procured from industry certified suppliers with documented quality assurance procedures covering the entire supply chain from primary producers, through slaughter and meat processing to the wholesale and retail outlets. These quality assurance procedures should satisfy all the domestic sanitary regulations, established to reduce transfer of disease causing agents, of the country sending the products to Antarctica or the highest export standards achievable by the meat industry in the country, whichever is the higher.

6.2 Action Required

Managers of national Antarctic programs, members of the International Association of Antarctica Tourism Operators, other tourism operators arranging visits to the Antarctic Treaty area and all others organising visits to the Antarctic region should be asked to ensure that meat and other animal products intended for human consumption in Antarctica should,

1. be procured from industry registered suppliers with documented quality assurance procedures that satisfy standards for domestic consumption, or the highest export standards achievable by the meat industry in the country, whichever is the higher, and
2. pass inspection to the standard normally applied for domestic consumption within the country or to the highest export standard achievable within the country by the meat processing industry, whichever is the higher.

In addition, Antarctic operators, whether operators of national programs or tourist operators, should take steps to ensure that they are aware of animal disease outbreaks occurring within the area from which they procure meat and meat products. The Office International des Epizooties (OIE, the world organisation for animal health) website (http://www.oie.int) is the definitive source for information on

notifiable animal diseases. Operators should ensure that susceptible meat and meat products are not sourced from the area designated at risk for any notified disease outbreak.

7 Waste Management, Sewage Treatment and Effluent Disposal

7.1 Background

The Antarctic Treaty System recognises the potential for transfer of pathogens to Antarctic wildlife from waste generated by Antarctic activities. The Madrid Protocol addresses the risk in Annex II, Conservation of Fauna and Flora, Annex III, Waste Disposal and Waste Management, and Annex IV, Prevention of Marine Pollution.

Annex II, Conservation of Flora and Fauna, requires that domestic plants and laboratory animals, plants and micro-organisms brought to Antarctica under a permit, and any poultry or parts not consumed, should be disposed of by incineration or equally effective means that eliminates risk to native fauna and flora.

Annex III, Waste Disposal and Waste Management, requires that residues of carcasses of imported animals, laboratory cultures of micro-organisms and plant pathogens, and introduced avian products should be removed from the Antarctic Treaty area by the generator of the wastes, unless incinerated, autoclaved or otherwise treated to be sterile. Sewage and domestic liquid wastes may be discharged directly into the sea untreated (if the summer station population averages less than 30) or after maceration. The by-product of sewage treatment by the rotary biological contacter process or similar may be disposed of into the sea.

Annex IV Prevention of Marine Pollution, permits disposal of food wastes into the sea no less than 12 nautical miles from the nearest land or ice-shelf after the waste has been passed through a comminuter, provided the ground waste can pass through a screen with openings no greater than 25 mm.

Infectious Newcastle disease virus has been recovered from meat after 250 days at –14°C to –20°C and from skin and bone marrow after 250 days at –4°C (Asplin 1949). Viable virus remains in the carcase until decomposition is well advanced. It is stable in non-putrefying tissue and organs or faeces if not exposed to high temperatures and has been isolated from bone marrow held for several days at 30°C (Omojola and Hanson 1986). Frozen meat products have been a significant means of spread of Newcastle disease virus when uncooked poultry scraps have been fed to poultry. Packaging and the drip that develops during storage can also be contaminated with virus from infected carcasses (Lancaster and Alexander 1975).

Minimum core temperatures to kill avian influenza and Newcastle disease viruses in poultry are,

- 70°C for a minimum of 30 minutes
- 75°C for a minimum of 5 minutes
- 80°C for a minimum of 1 minute.

Activities associated with a greater risk of exposing Antarctic wildlife to potential pathogens in waste food include,

1. feeding food scraps to Antarctic wildlife,
2. allowing scavengers, such as skuas, access to kitchen and field camp waste stored in garbage bags, and
3. thawing frozen meat and meat products in kitchen sinks and disposing of the melt water on to land or to the sea via the sewage treatment system.

7.2 Action Required

Feeding of food scraps to Antarctic wildlife is the most direct means by which pathogens could be introduced by people and should be explicitly prohibited.

Kitchen and field camp waste should be stored at all times in secure containers designed to prevent access by scavengers. As a precaution, uncooked waste meat and meat scraps should be boiled for 20 minutes before disposal if there is any chance that scavengers can feed on the scraps.

Melt water produced from thawing meat and meat products should be boiled before disposal to domestic sewage systems that discharge effluent to the Antarctic environment.

8 Research Priorities

8.1 Background

Relatively little is known about disease and disease processes in Antarctic wildlife. Available information indicates that Antarctic wildlife species carry a diversity of potential pathogens and display immune reactions to many other disease causing agents that have not yet been isolated. Beyond the intrinsic scientific value of providing greater understanding of an aspect of Antarctic ecology, the practical benefits of research are that it may provide information to reduce the likelihood of human introduction or spread of disease causing agents, and that it may provide information to help explain the cause of disease events.

The results of research to reduce the likelihood of a human mediated disease event will be used to improve practical measures to diminish the risk to Antarctic wildlife. For example, if it can be shown that a cleaning technique is not effective at reducing the viability of specific pathogens then the technique could be abandoned. Results of research to help explain the cause of a disease event may be used to reduce the risk to Antarctic operators of adverse public reaction if an unusual mortality occurs. For example, if it can be shown that pathogens associated with an unusual mortality event were common among Antarctic wildlife in locations

remote from human activity prior to the event then it may be inferred that people did not recently introduce the pathogen. The growing body of information on immune reactions in Antarctic wildlife is already a valuable resource in this respect.

Questions raised during the process of developing practical measures to diminish risk to Antarctic wildlife that may warrant research include,

1. How well do potential pathogens survive as viable infectious agents in the Antarctic environment?
2. How effective for eliminating potential pathogens are the methods currently used or proposed for cleaning footwear, equipment and vehicles in Antarctic operations?
3. Do current methods of sewage treatment and effluent disposal reduce the risk of disease introduction sufficiently?

Research and other activities that may provide information to help explain disease events includes,

1. investigation of the spatial and temporal patterns of disease causing agents (including serological evidence) within Antarctic species,
2. comparison of the type and diversity of disease causing agents among animals that spend their entire life within the Antarctic region and those that migrate to other continents, and
3. development of a tissue bank that in the event of a disease incident could be used to do retrospective analyses for evidence of historic occurrences of disease causing agents.

Most of the research activities identified could be addressed by individual researchers working with the support of national programs without the need for establishing a formal structure within the Antarctic Treaty System or SCAR. The establishment of a tissue bank would benefit from international coordination however it does not follow that the most efficient mechanism would be to establish a single international facility to archive Antarctic material. Most countries involved in Antarctic activities already have properly curated facilities for archiving non-Antarctic animal tissue. At this stage the most efficient mechanism to establish a tissue bank for Antarctic animal tissue would be to develop cooperative arrangements with established archival facilities. Information on Antarctic material held by archival facilities could be made available using established Antarctic Treaty System processes for scientific data management and data exchange.

8.2 Action Required

Request SCAR to endorse the research priorities and other activities identified above and to disseminate these to SCAR representatives and to appropriate SCAR Working Groups.

Request the Joint Committee for Antarctic Data Management to advise on the development of procedures for sharing access to information on tissues stored in archival facilities located in different countries.

References

Asplin FD (1949) Observations on the viability of Newcastle disease. Vet Rec 61(13):159–160
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Omojola E, Hanson RP (1986) Collection of diagnostic specimens from animals in remote areas. World Anim Rev 60:38–40
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Appendix F
Unusual Animal Mortality Response Plan

This plan produced by Australia is provided as an example of a plan for an organisational response to the discovery of unusual animal mortalities in the Antarctic.

Reproduced from:

Australian Antarctic Division (2006) Unusual Animal Response Plan 2006. Registry File 03/712, Responsible Officer: Operations Safety and Environment Advisor. Appendix A to this document (Unusual Mortality Investigation Kits and Necropsy Tissue Collection Techniques) has not been reproduced here because it duplicates information published as part of the 'CCAMLR Ecosystem Monitoring Program: Standard Methods for Monitoring Studies' and reproduced in this volume as Appendix A.

1 Introduction

Unusual animal mortalities, although rare, have been observed amongst Antarctic wildlife and may occur again. An international workshop on diseases of Antarctic wildlife was held in Hobart in 1998 resulting in a number of recommendations to reduce the risk of disease introduction into Antarctica along with provision of guidelines for response to suspected disease occurrence. The workshop report[1] also contains a useful summary of all major disease agents previously documented in penguins and seals.

1.1 Purpose and Objectives

The purpose of this response plan is to provide guidance on what to do if sick or dead animals are discovered in unusually high numbers or with signs that suggest disease.

[1]Kerry K, Riddle M, Clarke J (1999) Diseases of Antarctic wildlife. Report for SCAR and COMNAP 1999, pp 104.

The objectives are to obtain information on the species involved, the extent of affected animals and the cause. A further objective is to reduce the likelihood of people spreading the infectious agent if disease is involved.

1.2 Target Audience

The plan provides information for people in the field who discover unusual numbers of sick or dead animals; the Station Leader (SL) or Voyage Leader (VL) responsible for the people in the field and people at the Australian Antarctic Division (AAD) headquarters at Kingston responsible for coordinating a response.

1.3 Geographic Scope

The plan covers the Australian Antarctic Territory (AAT) and the Southern Ocean.

1.4 Health and Welfare of Personnel

Protection of the health and welfare of personnel must be a priority in any response to the discovery of sick or dead animals.

2 Preparation and Planning

2.1 Awareness and Training

SLs, Field Leaders (FL) or VLs and Antarctic Medical Practitioners are to be briefed on the response plan and response kit as part of their pre-departure briefing by the Operations Safety and Environment Advisor (Ops SEA). During their briefing to new arrivals on station or ship, the SL/VL should instruct people to report the discovery of unusual numbers of sick or dead animals to them immediately.

2.2 Equipment

A response kit has been provided to each station and ship. It contains the equipment necessary to record an event, undertake post-mortem examinations and prepare samples for transport and subsequent analysis, together with instructions on procedures and safety precautions. Further information about the kit is included in

Appendix A. Station Leaders hold the key to the kits on station. [*Editors' note: The Appendix (A) referred to here is the document 'Unusual Mortality Investigation Kits and Necropsy Tissue Collection Techniques' available from the Operations Safety and Environment Adviser, Australian Antarctic Division, Channel Highway, Kingston, 7050 Australia. It is not reproduced in this volume because much of it is covered in the CCAMLR Protocol given in Appendix A in this volume.*]

The equipment is stored in a clearly labelled sealed yellow box in the following locations on stations:

Station	Storage location
Casey	Green Store – racking in the warm store section near the medical fire stock
Davis	Flammable Liquids Store (Chemicals section)
Macquarie Island	Obsolete darkroom in the Science Building (now being used for storage)
Mawson	Green Shed among the field equipment items

On ships, the kits are to be stowed in the Deputy Voyage Leader container. All kits must have appropriate dangerous goods labelling on the outside. Any goods inside that could spill or leak should be packed properly; e.g., in a sealed plastic bag with absorbent material.

2.3 Permits

In general, permits are required to collect specimens of animals in the Antarctic.

A permit to import samples to Australia is required under the *Quarantine Act 1908*.

2.4 Funding

Expenditure on equipment, analysis or consultants must be approved in advance by the AAD response team and funds will be provided from the Director's contingency budget.

3 Immediate Response

3.1 On Discovery

Upon discovery of unusually high numbers of sick or dead animals:

- Withdraw from immediate area
- Restrict access to the site to reduce the risk of transferring pathogens to uninfected populations

- Do not visit other colonies or sites with aggregations of animals without taking precautions to prevent transfer of pathogens on boots, clothes or equipment
- Immediately notify the SL, FL or VL that unusual numbers of dead or dying animals have been discovered. The Leader will refer to this response plan and provide instructions to the field party
- On Macquarie Island, immediately notify the Tasmanian National Parks Ranger on site and
- Notify the Antarctic Medical Practitioner on station or on board ship

3.2 Criteria for Determining an Unusual Mortality Event

The decision on what constitutes unusual numbers of sick or dead animals can be made only on the basis of experience of what is normal for the species and location. If you discover dead or dying animals in numbers, or in a condition which, in your experience, is unusual then report it.

For animals and birds, unusual behaviour that may indicate presence of disease includes:

- Staggering, falling, paralysis, inability to rise or disinclination to move when approached
- Coughing, sneezing, excessive nasal discharge or respiratory distress (note that panting is a normal response of penguins in warm weather)
- Ocular discharge, apparent blindness and/or
- Diarrhoea or bloody and fetid faeces

3.3 Initial Data Collection

Sufficient data should be collected initially to provide animal experts with information to determine whether something unusual has been discovered.

Stand back, view widely and record the following information from the periphery of the group of animals. *Do not walk* among the sick or dead animals.

Record the following information:

- location including coordinates (use a GPS if available);
- area affected;
- species involved and whether adults or chicks/pups;
- indication of the number of animals involved – if possible count the dead and dying and estimate the percentage of each among the colony;
- clinical signs;
- contact details of all people who were at the site;
- weather conditions now and, if known, over the previous week; and
- take lots of photographs, preferably with a digital camera and video.

Call the SL, FL or VL by radio again and report the information gathered during the initial data collection

3.4 Hygiene

Boots must be cleaned using sea water or snow before leaving the general area of the dead or dying animals. Repeated washing in sea-water will remove most pathogens.

The SL, FL or VL should arrange for boots, clothing, equipment and vehicles to be sanitised on return of the field party. Boots and equipment should be scrubbed in a 2% w/v solution (20 g l^{-1}) of Virkon, which is included in the response kit.

3.5 Notification

The field party must inform the SL or VL as soon as they suspect they have discovered unusual numbers of sick or dying animals and again when the initial data collection has been completed.

The SL, FL or VL will:

- Advise the Antarctic Medical Practitioner at station or onboard ship
- Advise personnel that access to the area is restricted to those authorised to investigate the event
- Inform the Support and Coordination Manager (SCM), at the AAD headquarters at Kingston and send the information from the initial data collection and the names of people on station who have relevant skills
- Lodge an incident report on the Incident Reporting System on the Intranet as soon as practicable

The incident report will automatically go to the following people:

- General Manager, Operations
- Manager, Environmental Protection and Policy Section
- Chief Medical Officer, Polar Medicine Unit, who will ensure that the Chief Scientist is notified

3.6 Preparation for Further Investigation

On notification of the incident the Station Leader and/or their delegate should open the response kit in anticipation that further investigation may be required. The Station Leader is to determine on station skills medical/veterinary skills available to assist in any required sample collection.

4 Administration (Incident Control Structure)

4.1 Response Team

The General Manager, Operations, shall form a response team to determine whether the mortality is in fact unusual or may involve disease and if so, will direct any further action that might be required.

The response team should include the following expertise and responsibilities:

- Convenor – Operational responsibility for management of the response
- Advisor – Station or ship operations (as appropriate)
- Advisor – Environmental management
- Advisor – Human health/disease implications
- Advisor – Permits
- Advisor – Quarantine implications
- Advisor – Occupational health and safety aspects
- Advisor – Animal ecology
- Advisor – Veterinary aspects and pathology
- Advisor – Treaty and Government, including Ministerial briefs and information to ATCM and other parties

Expertise across several of these areas may reside in a single person, in which case the overall size of the response team will be reduced. If avian flu is suspected, the Operations Branch Crisis Management and Response Team will form the basis of the response team.

The Australian Wildlife Health Network will be contacted to determine scientific/technical expertise available to assist in the response.

4.2 Assessment for Further Investigation

The convenor of the response team will determine whether further investigation is required on the basis of the information provided by the station/ship and any other information available to it. The response team may decide that:

- The number or nature of sick or dead animals is not unusual and that no further action is required
- It is uncertain whether the event is unusual and that the population should be observed, without sampling, to determine whether more animals are affected, or whether the symptoms worsen
- The event is unusual and that further investigation, including sampling, is required

Responsibility for further investigation of the incident in Antarctica will be assigned to the person on site with the most appropriate expertise, such as veterinary science, pathology, microbiology.

The priorities for further investigation are to determine the geographic extent of the event and to determine the cause.

4.3 Coordination of Further Investigation

General Manager, Operations, will assume overall responsibility for ensuring the event is managed correctly. This responsibility may be delegated to the convenor of the response team.

The SL, FL or VL will assume responsibility for ensuring the incident is managed correctly in Antarctica and should coordinate the response in the field. The Leader should liaise with the Medical Officer and other people with appropriate skills on station/ship.

4.4 Health and Safety

The Advisor – Human Health/Disease Implications will determine risks and mitigation techniques to prevent transmission of disease to humans.

The Antarctic Medical Practitioner on station or on board ship should ensure that all personnel who may come in contact with affected animals are aware of the need for appropriate hygiene practices and should provide instruction on the contents and use of the response kit.

4.5 Communication

The SL, FL or VL is responsible for informing their personnel of the possibility of a disease event. The convenor will liaise with all advisors to determine required statutory notifications that need to be undertaken.

Do not contact the media. Refer all media queries to the Media Liaison Officer. Dissemination of information to the public about the event is to be in accordance with the AAD Media Policy. Any external communication by expeditioners will require authorisation by the SL or VL.

The Director, AAD will determine whether other national operators or other organisations need be informed and will arrange for information to be passed on if required.

4.6 Station/Voyage and Field Personnel

Station, voyage and field personnel should be warned about potential human health risks. People in the field may be called on to determine the geographic extent of the event but must be cautioned about the risk of transferring disease causing agents on

boots and clothing when moving between locations. All personnel should be advised when hygiene precautions such as boot washing are required and of the procedures for doing this.

4.7 Legislation and Permits

Before authorising sampling the convenor of the response team is responsible for ensuring that all permits required under various acts of legislation (Sect. 2.3) are in place and up to date. All investigative activities, such as sampling, must be consistent with the conditions of permits.

5 Containment

5.1 Site Access

Access to the site should be restricted to reduce the risk of transferring pathogens to uninfected populations. Do not take vehicles close to a colony or aggregation that includes sick or dead animals. Park vehicles at least 250 m from the aggregation to reduce the chance of faeces, blood or other organic matter being trapped in vehicle treads and spread to other locations. Access routes may be specified to minimise disease spread.

5.2 Removal of Site Access Restrictions

Restrictions on site access will remain in force until the SL, FL or VL is advised that normal access may be resumed by the General Manager, Operations.

5.3 Disposable Overshoes and Protective Clothing

Disposable overshoes and personal protective equipment (PPE) are provided in the response kit and should be worn when visiting colonies or aggregations during the event. On leaving the colony, overshoes should be removed at least 250 m from the edge of the aggregation and then should be sealed in plastic bin liners for disposal in the station/ship incinerator. Overalls, masks etc. should be disposed of in a similar manner.

Protective clothing, including overshoes, must be changed before moving between discrete aggregations of wildlife, i.e. between colonies on separate islands or between colonies separated by at least 1,000 m of unoccupied ground.

5.4 Precautions During Authorised Sampling

All possible precautions are to be taken by people handling affected animals to avoid the geographical spread of disease to other colonies. If dead animals are seen on the sea-ice while travelling to a colony they should be collected on the return trip to minimise the risk of introducing or spreading disease within the colony. As far as practicable, the shortest route from the edge of an aggregation of animals is to be taken when collecting dead specimens. Only authorised people are permitted to approach and handle affected animals.

6 Necropsy and Tissue Collection

Protocols for examination and dissection, tissue collection, sample preservation and storage are contained in Appendix A.

7 Post-Event Activities

7.1 Post-Event Debrief

Post event debriefs should be held at the station/ship and at AAD headquarters at Kingston.

The station/ship debrief should include:

- full account of the discovery
- Collation of an event timetable with details of response actions implemented
- Report on contents of response kit, including items to be restocked, items that could be improved and new items that would improve the kit
- Suggestions for improvements to the response plan
- Collation of information on samples, photographs etc. including number, location, storage requirements etc
- record of the people responsible for ensuring all samples, photographs and records are transmitted to the AAD

AAD debrief at Kingston should include:

- Review of response to the event
- The effectiveness of the incident reporting process
- Collation of information on samples, photographs and records
- Review of the response plan
- Review of response kit
- Initiate restocking of response kit

- Outline of communication strategy
- Summary of information provided to Minister, ATCM, other Parties, national operators and other organisations (e.g. IAATO)
- Review of relevant parts of training programs

This could be facilitated by a professional organisation experienced in disease outbreak.

7.2 Reporting

7.2.1 SL, FL or VL

The following reports will be transmitted by the SL, FL or VL to the Support and Coordination Manager at AAD Headquarters, Kingston:

- Report by the people discovering the unusual mortality event – details of circumstances in the field including the initial data collection.
- Report by the person responsible for further investigation – details of further investigation, and samples including location and storage requirements.
- Report by the Station/Voyage/Antarctic Medical Practitioner – details of Occupational Health and Safety aspects and response kit restocking requirements.
- Report by the SL/VL - details of station/ship response and collation of all the above reports from station/ship personnel.

The reports will be passed to the convenor of the response team.

7.2.2 Convenor of the Response Team

The convenor of the response team will prepare a report for the AAD Director to include:

- Collated report on the incident and response from the SL/VL
- Review of the event and possible causes
- Review of response to the event
- Collated information on samples, photographs and records including location and storage requirements
- Post-activity reports where required by the ATEP or EPBC Acts
- Review of the response plan
- Review of response kit
- Schedule for restocking of response kit
- Publication strategy
- Summary of information provided to Minister, ATCM, other Parties, national operators and other organisations (e.g. IAATO)

The convenor will table the report of the response team at the next AAD Executive meeting and provide a copy to the Permits Officer in accordance with any permit requirements.

The convenor will also arrange preparation and distribution of reports as required by any external organisations.

7.3 Follow-Up Activities and Stand-Down

Restrictions on site access will remain in force until the SL/VL is directed to resume normal access by the General Manager, Operations. The convenor of the response team will advise the General Manager, Operations on the appropriate time to resume normal site access.

The response team shall direct follow-up activities until told to stand-down by the General Manager, Operations.

7.4 Access to Tissue Samples

The response team will determine the policy on access to tissue samples for each event. If an infectious disease is suspected, samples may be required to be lodged in a secure facility such as the Australian Animal Health Laboratory in Geelong.

7.5 Post-Event Monitoring

The response team will determine whether post-event monitoring is required and, if so, will identify who is responsible for monitoring design and implementation.

8 Review of This Plan

This plan will be reviewed every two years by the Operations Safety and Environment Advisor, the Chief Medical Officer, the Manager of the Environmental Protection and Change Program, the Environmental Manager and a representative of the Wildlife Health Network.

Subject Index

*Figures in *Italic*, Tables in **Bold**

Taxonomic Index